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I am submitting herewith a thesis written by Zachary Clay Wilcox entitled "The Influence of Sport Competition on Mood and Secretory Immunoglobulin A." I have examined the final copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Psychology.

Dr. Debora Baldwin, Major Professor

We have read this thesis and recommend its acceptance:

Accepted for the Council:

Dr. C.W. Minkel
Associate Vice Chancellor and
Dean of the Graduate School

THE INFLUENCE OF SPORT COMPETITION ON MOOD AND SECRETORY IMMUNOGLOBULIN A

A Thesis
Presented for the
Master of Arts
Degree
The University of Tennessee, Knoxville

Zachary Clay Wilcox
August 1997

DEDICATION

This thesis is dedicated to my parents, Mr. Charles Wilcox and Mrs. Dorothy Wilcox, who have provided endless support to me in all my endeavors.

ACKNOWLEDGMENTS

I owe gratitude to a number of individuals who have guided and assisted me in the completion of this master's thesis. Foremost, I would like to thank Dr. Debora Baldwin, my major professor and committee chairperson. Her advice and encouragement were much appreciated through the entire thesis process. Dr. Baldwin provided endless enthusiasm about the project and always provided instructive advice.

I appreciate the assistance of my other committee members: Dr. Kathleen Lawler and Dr. James Lawler. Dr. Kathleen Lawler's mentoring during my early graduate school days will always be cherished. Dr. James Lawler made crucial recommendations for the planning of the research and important corrections to the manuscript.

I owe a special thanks to William S. Wire and Nelly Kuklin for training me in the ELISA procedure. In addition, I would like to thank Siva Prakash Gangapappa, Ila Davis, Zhiya Yu, Elanchezhiyan Manickan and Johnson Thomas in the laboratory of Barry T. Rouse for their willingness to answer my immunology questions. I would also like to thank Megan Wilson and Howard Rasey for their assistance during data collection. Keith Kline, Francis Craig and Jenelle Krishnamoorthy assisted me with the final product. Cartoon work in Appendix C was done by Dustin Runyan, a good friend.

ABSTRACT

Research has shown that individuals exhibit a variety of immunological and emotional changes with sport participation. For example, exhaustive aerobic exercise tends to reduce secretory immunoglobulin A (S-IgA) whereas aerobic activity of moderate intensity typically enhances or has little effect on this same parameter. With regard to mood, exercise has been found to reduce tension, depression and anxiety levels in trained and untrained individuals. However, there is little understanding of possible mediating factors such as competitive outcome (winning vs. losing) and mood status on S-IgA modifications. The purpose of this study was to examine the influence of sport competition on S-IgA and mood. Twenty-six adult tennis players (21 males, 5 females) involved in tournament matches served as participants in the study. Before and after play, individuals completed the Profile of Mood States (POMS) and rendered saliva samples. S-IgA concentraton was determined using the enzyme-linked immunosorbent assay (ELISA). Data analyses revealed thant males exhibited no significant change in S-IgA concentration from pre to postmatch. However, females displayed significant increases in S-IgA concentration from pre to postmatch. For males, there was a

trend toward a main effect for competitive outcome on S-IgA, with winners showing higher levels than losers. In addition, there was a significant negative correlation between S-IgA level and total mood disturbance (TMD), as measured by the POMS. Also, losers reported significantly greater anger, tension and depression scores (on the POMS) than winners. The findings from this study support the notion that sport competition has immunological and emotional effects on participants. The clinical significance of enhanced S-IgA concentration with sport competition in females has not been determined.

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CHAPTER 1

INTRODUCTION

Research has shown that individuals exhibit a variety of immunological and emotional changes with sport participation (Mackinnon, 1992; Yeung, 1996). However, there is limited understanding of the specific factors associated with such changes. With regard to the exercise component of sport, it is generally accepted that moderate exercise training has beneficial effects or very little influence on immune parameters. For example, Kaufman, Harris, Higgins and Maisel (1994) gradually swim-trained eighteen Sprague-Dawley rats once a day over a 4-week period, after injection with a harmless antigen. At the end of the training period, all trained and nine untrained animals underwent a short-term exhaustive swim. Blood samples were obtained two days per week throughout exercise training, and antigen-specific Immunoglobulin G (IgG) and Immunoglobulin M (IgM) levels were determined. It was revealed that trained rats exhibited higher IgM and IgG responses during early and late training than untrained controls. With regard to the influence of moderate exercise on mood, the findings are mixed. However, mood enhancement is generally reported with aerobic exercise of various

intensities.

Intense, exhaustive exercise, however, often produces detrimental effects on the immune system. For example, Mackinnon, Chick, Van As and Tomasi (1989) studied eight male competitive bicyclists on a laboratory fitness test requiring them to pedal a bicycle until completely exhausted. The athletes exercised at 70-75% of VO_2 max for about 2 hours. Saliva samples were taken before and after testing. Secretory Immunoglobulin A (S-IgA) showed a significant decrease from pre to postexercise session. Mackinnon and Jenkins (1993) studied the influence of exercise on S-IgA in twelve male college students undergoing an eight-week, three days per week ergometer training program. Before training began, the participants were given an interval exercise test on an ergometer. In five, sixty-second bouts, each separated by a five-minute rest period, participants were instructed to pedal as many times as possible. The same test was administered at the conclusion of the training program. Saliva samples were obtained before and after both exercise tests. Interestingly, it was revealed that S-IgA concentrations decreased significantly from pre to postexercise test. This effect was observed before as well as after the eight-week training program, as training did not alter the effect of acute exercise on S-IgA.

Exercise, however, is merely one component of sport competition. Other factors such as competitive outcome (i.e., winning or losing) may also have important consequences. For example, in a study by Abadie (1989), fifteen women and four men were randomly assigned to two volleyball teams. All individuals participated in a game for twenty-five minutes; and the team with the highest score after this period was declared the winner. All individuals completed a state anxiety inventory immediately before and after the game. Following competition, the winning team exhibited a significant decrease in state anxiety. However, the losing team showed a nonsignificant increase in state anxiety.

Epidemiological reports have found higher rates of upper respiratory illness among elite athletes compared with untrained individuals (Peters & Bateman, 1983). Research shows that S-IgA is an important immunological agent against upper respiratory infection. The purpose of this study was to examine the influence of tennis competition on S-IgA and mood.

An overview of the basic components of the immune system will precede examining the specific topic of this study. It is intended to provide the reader with an immunological context in which to place S-IgA, the immune parameter of interest in the present study.

General Immunology

The immune system is responsible for protecting organisms against infection from foreign substances (antigens) such as viruses, fungi, bacteria and parasites, and also serves to prevent malignant tumor growth. It can be divided into two basic divisions: innate (natural) immunity and acquired (specific) immunity.

Innate Immunity

According to Wilson (1994), innate immunity: "(1) is present from birth, (2) is not enhanced by prior exposure to an infectious agent, (3) lacks immunologic memory, and (4) does not display antigenic specificity" (p. 8). Innate immunity includes physical barriers such as the skin, epithelium and mucus that discourage the entry of antigens into the interior of the organsim. Chemical barriers are also a component of innate immunity. For example, pH and other soluble factors provide an inhospitable environment for microorganisms. In addition, the presence of phagocytic immunological cells (e.g., macrophages, natural killer cells) confer the ability to detect and lyse antigens without prior contact (Mackinnon, 1992).

Acquired Immunity

Typically, if antigenic invasion is not effectively prevented by innate immune mechanisms, an acquired immune response will ensue. According to Wilson (1994), acquired immunity is characterized by "(1) exquisite specificity for the offending antigen(s), (2) memory (a rapid and heightened response following subsequent encounter with the same or a closely related antigen), (3) diversity (the capacity to respond to millions of different antigens present in the environment), and (4) self- versus nonself recognition" (p. 8). Acquired immunity can be functionally divided into two main types of responses: cell mediated and humoral. Lymphocytes are the basic cellular units of both types of acquired immunity.

T-lymphocytes and Cell-mediated Immunity. T-lymphocytes, or sometimes referred to as T-cells, originate in the bone marrow and differentiate in the thymus. They play a key role in cell mediated immunity. They initiate and regulate the immune response against antigens. Each T-cell has a very selective receptor on its surface that can recognize and bind with a particular antigen. A given T-lymphocyte has many receptor sites, but they are all specific for the same antigen. T-lymphocytes consist of several basic subpopulations. For example, T-helper cells regulate much of the immune response by secreting substances called

lymphokines. Lymphokines secreted by T-helper cells cause T-cytotoxic cells to proliferate. In addition, T-cytotoxic cells directly lyse antigens via the release of toxic substances (Mackinnon, 1992).

B-lymphocytes, Humoral Immunity and Salivary Immunoglobulin A (S-IgA). B-lymphocytes (B-cells) originate and mature in the bone marrow. They are the effector cells of humoral immunity. When a B-cell binds with an antigen, it divides, and its progeny differentiate into plasma cells and memory B cells. Plasma cells secrete soluble immunoglobulin products called antibodies, which bind to antigens. Each plasma cell secretes a single type of antibody to a particular antigen. The process of binding the antigen is sometimes sufficient to eliminate it. Alternatively, binding the antigen can activate a system called complement which can destroy the antigen. The process of generating antibody usually takes five or more days. If the antigen is encountered again in the future, the antigen-specific memory cells can produce a much more rapid and potent reaction (Maier, Watkins & Fleshner, 1994).

Immunoglobulins are found in serum and in secretory fluids such as saliva, breast milk, and nasal, gastointestinal, bronchial and urogenital secretions (Roitt, 1994). Antibody molecules are Y-shaped and consist of two identical heavy and two identical light polypeptide chains

held together by interchain disulfide bonds. The two ends of the molecule have different functions. One end of the molecule (Fab) binds to antigens and the other end (Fc) binds to receptors on immune cells. Based on the structure of their heavy chain regions, immunoglobulins are categorized into five main clases: immunoglobulin G (IgG), IgA, IgM, IgD and IgE. These antibodies differ in their amount and distribution throughout various regions of the body. For example, IgG is the predominant antibody in serum whereas IgA is the most abundant antibody in mucosal fluids (Roitt, 1994).

The mucosal immune system functions somewhat separately from the systemic immune system. Its primary function is to provide for host defense at mucosal surfaces. The predominant antibody class in mucosal secretions is secretory immmunoglobulin A (S-IgA). The presence of S-IgA in the oral cavity is due to production by plasma cells located in salivary glands. The ductal and acinar cells of these glands contain receptors called secretory component which are responsible for the transport of IgA to the lumen and into the oral cavity as a constituent of saliva (Mestecky, 1993). S-IgA differs from IgA in that it contains the secretory component, which also protects the antibody from degradation (Strober & James, 1994).

Since many infectious agents enter the body through

mucosal surfaces, the presence of S-IgA antibodies in the fluids bathing these surfaces provides a key initial defense against infection. In saliva, the primary effector functions of S-IgA are thought to include the following: 1) virus neutralization, 2) neutralization of toxins or enzymes, 3) inhibition of adherence (or growth) of microorganisms on epithelial cells or teeth, 4) antigen trapping and antigen exclusion by preventing the access of antigen to the systemic immune system, and 5) interaction of IgA with nonspecific defense mechansisms (Challacombe & Shirlaw, 1994). There is overwhelming evidence that S-IgA interferes with bacterial and viral adherence to mucosal surfaces and consequently limits colonization of these surfaces by pathogens (Roitt, 1994). Of course, a suppression in one aspect of immunity (i.e., S-IgA) does not produce illness. Many other factors play a role, including presence or absence of a pathogen, amount of pathogen exposure, virulence of the pathogen, and competence of other immune and even non-immune factors. Hence, S-IgA concentration might be best thought of as a risk factor for developing infection. Indeed, S-IgA levels have been shown to be extremely low in infection-prone children compared with healthy controls (Lehtonen, Tenovuo, Aaltonen, & Vilja, 1987).

In a prospective study, Rossen, Butler, Waldman,

Alford, Hornick, Togo and Kasel (1970) measured IgA concentration in nasal secretions of fifteen healthy adult males prior to inoculating them with a live influenza virus. During the one-month study, the men lived in relatively isolated conditions. Nasal secretions were obtained and symptoms of influenza were monitored every day. Interestingly, a negative correlation was found between S-IgA concentration before inoculation and number of symptoms of infection. In addition, it was noted that the virus specific antibody was detected earlier and rose to higher levels sooner among men who had higher total S-IgA levels prior to infection.

Immunological Measurement

An array of laboratory techniques is available to assess nearly every component of the immune response. These procedures can be divided into two main approaches: enumerative and functional.

Enumerative

Enumerative techniques involve counting specific immune cells. The procedures vary with respect to the type of cell under consideration. Microscopic viewing can be used to determine total lymphocyte number and percentage. However, T-lymphocytes can only be specifically counted by detecting surface molecules which serve as markers (Hyde, 1995). The most universal marker on T cells is cluster of

differentiation (CD)3, a cell surface protein which is a component of their antigen receptors. Flow cytometry is the method of choice for counting lymphocytes. Using florescently labeled monoclonal antibodies, the technique uses light scatter and Coulter volume measurements. This procedure also allows for subpopulations of T cells (eg., T-helper) to be identified. For B cells, a variety of surface markers (CD19 or CD20) can be detected by monoclonal antibodies using flow cytometry. Natural killer (NK) cells can also be counted by detecting specific cell surface markers (CD56 and CD16). Either microscopy or flow cytometry can be used to count NK cells (Stites, 1994).

Functional

A host of techniques allow investigators to assess the functioning (responsiveness) of the immune system. These procedures are a more direct test of immunocompetence than merely counting cells.

Mitogen Assays. Several plant lectins and other substances can be used to assess lymphocyte activation. These substances induce proliferation of lymphocytes and are called mitogens. Concavalin A (con A) and phytohemagglutinin (PHA) are the predominant T cell mitogens. For B cells, pokeweed serves as a mitogen.

In a mitogen assay, blood serum (which contains lymphocytes) and mitogens are incubated together for about

72 hours, allowing mitogen to produce effects of DNA synthesis. An increase in DNA synthesis is a necessary event for cell division (ie., lymphocyte proliferation). DNA synthesis is measured by pulse-labeling the serum-mitogen culture with tritiated thymidine (3H-Tdr), a nucleoside precursor that is incorporated into newly synthesized DNA. The amount of 3H-Tdr incorporated relative to the rate of DNA synthesis is determined by scintillation counting using a beta counter. Greater lymphocyte responsiveness is indicated by higher counts per minute (Stites, 1994). Generally, it can be inferred that higher counts per minute indicate a more competent response.

Antibody Titers. The antibody titer is used to assess the competence of the immune system against a particular antigen (Benjamini & Leskowitz, 1988). The reaction of antibodies with insoluble particle antigens (e.g., foreign red blood cells) results in the cross-linking of the various antigen particles by the antibodies. The cross-linking eventually results in the clumping, or agglutination, of the antigen particles by the antibodies. The agglutination of an antigen by antibodies is dependent on the appropriate proportion of antigen to antibody.

First, the same volume of an insoluble antigen is added to multiple (typically ten) test tubes. Second, serial dilutions of samples (containing antiserum) are performed

such that the concentration of the antibody being measured is decreased by one-half in each successive tube. The highest dilution of sample that still causes agglutination, but beyond which no agglutination occurs, is termed the titer. A relatively high titer is an indication of greater specific antibody concentration.

Enzyme-linked Immunosorbent Assay (ELISA). ELISA is another common procedure which is used to determine antibody concentration. In the common sandwich variation of the assay, the desired antibody is used as an antigen. An antibody that binds the antigen is first immobilized upon a solid surface. Second, the sample containing the antigen is delivered in serial dilutions to the wells containing the antibody, allowing the antigen being measured to bind the antibody. Third, an enzyme-labeled antibody directed against the antigen to be detected is added. Fourth, the substrate is added, which reacts with the enzyme to produce a colored product. The antigen concentration is inferred from the amount of color change (optical density) that occurs. Optical density is measured by a spectrophotometer (Rodgers, 1994).

Natural Killer (NK) Cell Activity. NK cells, a component of innate immunity, are believed to provide defense against viral infections and possibly some tumors. Functional assays for NK cells take advantage of the fact that these

cells lyse NK sensitive target cells (eg., erythroleukemia cell line K562) *in vitro*. In order to determine NK cell activity, target cells are labeled with chromium and mixed with effector (NK) cells. Lysis of the target cells by NK cells causes chromium to be released from the target cells. The amount of chromium release is used as an index of NK cell activity (Stites, 1994) and is measured in a gamma scintillation counter. High counts are an indication of greater NK cell activity.

CHAPTER II

LITERATURE REVIEW

Exercise is a form of leisure physical activity that is usually repeated over an extended period of time. The major reasons for participating in an exercise program usually involve improved fitness, appearance or health. Sport, in North America, implies a form of physical activity that involves competition. However, in Europe the term sport may also embrace exercise and recreation (Bouchard & Shepard, 1994).

Regular physical exercise has long been regarded as an important component in a healthy lifestyle. Recently, this impression has been reinforced by new scientific evidence linking physical activity to a wide array of physical and mental health benefits (Pate, Pratt, Blair, Haskell, Macera, Bouchard, Buchner, Ettinger, Heath, King, Kriska, Leon, Marcus, Morris, Paffenbarger, Patrick, Pollock, Rippe, Sallis, & Wilmore, 1995). However, exercise has also been shown to be potentially detrimental to the individual depending on the intensity and duration of the session (Mackinnon, 1992).

Exercise is often classified into basic types. For example, aerobic exercise (e.g., running) consists of physical activity whose primary fuel source is fat, whereas

anaerobic exercise (e.g., weight lifting) depends primarily on carbohydrate. Generally, as the intensity and duration of exercise increases, fat becomes the primary fuel source and the activity is considered aerobic (Nieman, 1990). Acute exercise generally refers to a single bout of physical activity, and chronic exercise describes repeated sessions, often associated with changes in cardiovascular function (Wilmore & Costill, 1994).

Acute Physiological Effects of Exercise and Sport

Exercise and participation in sports have a wide range of biological effects. The following is an overview of the primary influences of aerobic exercise on three important physiological systems of the body: cardiovascular, endocrine and immune.

Cardiovascular Response

Physical exercise provides a great challenge to the cardiovascular system, which is met by a complex integration of the local regulation of blood flow to the active skeletal muscles and the neural regulation of the hemodynamic response. In exercise the active muscle must receive a blood supply appropriate to its increased metabolic needs, while the brain, heart, and other organs receive an adequate blood flow to maintain function (Mitchell & Raven, 1994).

Heart rate increases with exercise and reflects the amount of work the heart must do to meet the demands of the

activity. It increases in proportion to the intensity of the exercise (Wilmore & Costill, 1994).

Stroke volume, the amount of blood ejected from the left ventricle during contraction of the heart muscle, also usually increases during exercise to allow the heart to work more efficiently. Most researchers agree that stroke volume increases with increasing rates of work, but only up to exercise intensities between 40% and 60% of maximal capacity. Stroke volume is thought to plateau at higher intensities (Wilmore & Costill, 1994).

Cardiac output, the total volume of blood pumped by the heart per minute, is the product of heart rate and stroke volume. Since heart rate and stroke volume increase with exercise intensity, cardiac output does as well. It increases primarily to match the need for increased oxygen supply to working muscles (Wilmore & Costill, 1994).

With regard to blood pressure changes associated with exercise, systolic and diastolic pressure show differential responses. Systolic pressure is the pressure on the arteries when the heart contracts. With endurance, whole-body activity, systolic blood pressure increases in direct proportion to increased exercise intensity. Increased systolic blood pressure results from increased cardiac output. It helps drive the blood quickly through the vasculature. Also, blood pressure determines how much fluid

leaves the capillaries, entering the tissues and carrying needed supplies. Thus, increased systolic pressure facilitates the delivery of nourishment to tissues (Wilmore & Costill, 1994). Even though systolic blood pressure increases as work intensity increases, it can sometimes decrease with prolonged exercise. This decrease is a result of increased arteriole dilation in the active muscles. Diastolic pressure, which reflects the pressure in the arteries when the heart is at rest, changes little, if any, during endurance exercise (Wilmore & Costill, 1994).

Resistance exercise (e.g., weight lifting) produces exaggerated increases in systolic and diastolic blood pressure, often due to the Valsalva maneuver. This maneuver occurs when an individual attempts to exhale while the mouth, nose and glottis are closed. This action produces an enormous increase in intrathoracic pressure. Much of the subsequent blood pressure increase results from the body's effort to overcome the high internal pressures created during the Valsalva maneuver (Wilmore & Costill, 1994).

Endocrine Response

Endocrine glands release hormones directly into the blood. The blood stream carries hormones to various tissues where they bind to specific receptors and exert their effects (Powers & Howley, 1990).

A single bout of exercise is characterized by increased

plasma concentrations of a large variety of hormones, and decreased concentrations of only a few hormones. Hormonal changes play a vital role in homeostasis, and include increasing the fuels necessary for muscular exercise, such as muscle glycogen and fat, plasma glucose, free fatty acids and amino acids (Powers & Howley, 1990). Although only studied for some hormones, large changes in plasma hormone concentration during exercise are generally the result of changes in secretion rate rather than changes in rate of clearance of the hormone. Exercise induced decreases in plasma volume also tend to increase plasma concentrations of all hormones (Richter & Sutton, 1994).

The adrenal medulla increases its production of the catecholamines, epinephrine and norepinephrine, during exercise. With respect to energy metabolism, epinephrine primarily acts as a stimulator of glycogen breakdown in liver and muscle and lipolysis in adipose and muscular tissue during exercise. Norepinephrine is a powerful stimulator of lipolysis in adipose tissue (Richter & Sutton, 1994).

Cortisol, the primary glucocorticoid produced by the adrenal cortex, also plays a role in restoring homeostatic balance during exercise. Cortisol promotes the maintenance of plasma glucose during exercise in a variety of ways. For example, it breaks down tissue protein so that necessary

amino acids can be formed so that the liver can synthesize new glucose. The plasma concentration of cortisol seems to increase or decrease as a function of acute exercise, depending on exercise intensity. In a classic study, Davies and Few (1973) found that when exercise intensity exceeded 60% of VO_2 max, cortisol concentration increased. However, exercise of any lesser intensity decreased cortisol levels. During the less intense exercise, cortisol was removed faster than the adrenal cortex could produce it. However, the more intense exercise resulted in a secretion rate of cortisol much higher than the removal rate.

The plasma concentration of reproductive hormones is also influenced by bouts of exercise. For example, acute exercise has been found to increase plasma testosterone levels (Powers & Howley, 1990). Testosterone is secreted from the testes and is responsible for the appearance of secondary sex characteristics, sperm production, and is a stimulator of protein synthesis. Estrogen, actually a group of hormones consisting of estradiole, estrone and estriol, is secreted by the ovaries and normally increases slightly with acute exercise. Estrogen is involved in fat deposition, the appearance of secondary sex characteristics and ovum development (Powers & Howley, 1990).

General Immune Response

Acute exercise has a wide range of effects on innate and acquired immunity. When evaluating the influence of an exercise bout on the immune system, the intensity of the session is crucial: moderate endurance exercise tends to have little effect or may enhance a host of immune system indices. However, exhausting exercise may have a deleterious effect on these same parameters (Mackinnon, 1992; Shephard & Shek, 1994).

Alterations in lymphocytes have been observed with exercise participation. According to Mackinnon (1992), acute exercise usually results in an increase in the number of plasma lymphocytes during and following the session. Nieman, Nehlsen-Cannarella, Donohue, Chritton, Haddock, Stout and Lee (1991) studied the influence of acute moderate exercise on leukocytes in twelve women. Participants were required to engage in regular aerobic exercise for six weeks prior to the study. In addition, baseline testing was conducted and VO_2 max was determined prior to the study via maximal graded treadmill exercise. On two consecutive Sundays, participants either engaged in laboratory exercise or rested. Half of the group exercised on each Sunday and the other half rested. Exercise consisted of forty-five minutes of 60% VO_2 max treadmill exercise. Blood samples were taken fifteen mimutes before, immediately after and at

four other times subsequent to exercise. In addition, blood samples were taken from resting individuals at these same times. Participants in the exercise group exhibited significantly higher total leukocytes, lymphocytes and granulocytes than resting subjects. However, neutrophils and monocytes were not significantly different between the two groups. Also, significant increases in total leukocytes, lymphocytes and neutrophils were observed from pre to postexercise. In addition, total leukocytes and neutrophils remained significantly elevated over preexercise levels three hours postexercise. For lymphocyte subpopulations, the exercise group exhibited significantly higher CD4 (helper) and CD8 (cytotoxic/suppressor) T-cells than resting participants. CD8 T-cells were revealed to be responsible for the lymphocytosis resulting from exercise.

MacNeil, Hoffman-Goetz, Houston and Arumugam (1991) studied the influence of exercise intensity and duration on lymphocyte proliferation. The participants in their study consisted of thiry-two males placed into one of three fitness groups (high, moderate and low) based on VO_2 max tests. The three different fitness level groups consisted of eight individuals and eight additional males were assigned to a control group. Each participant in the exercise groups performed four bicycle ergometer exercise tests consisting of 30 minutes at 65% VO_2 max, 60 minutes at

30% VO_2 max, 60 minutes at 75% VO_2 max and 120 minutes at 65% VO_2 max. One test was completed per week in random order. For each exercise session, six blood samples were taken at the following times: 24 hours and 3 minutes preexercise, 3 and 30 minutes postexercise and 2 hours and 24 hours postexercise. The low fitness group exhibited significantly lower lymphocyte proliferation (LP) levels than control individuals. LP levels were also lower in the high and moderate fitness groups than in controls. In addition, for all groups, LP was reduced after exercise and did not change (or recovered slightly) by 30 minutes postexercise. The greatest decreases in LP were observed 24 hours after exercise. However, almost full recovery occurred by 24 hours postexercise. With regard to the intensity of the exercise, comparing the two 60-minute tests of 30 and 75% VO_2 max revealed an insignificant decrease in LP with higher intensity exercise. In addition, comparing the two exercise tests of the same intensity (65%) but of different duration (30 vs 120 minutes) revealed no differences in terms of LP.

Immunoglobulins, products of B-cells, also generally increase with brief exercise (Nieman & Nehlsen-Cannarella, 1991). However, S-IgA may decrease with exercise, particulary if the session is highly intense. A review of the influence of exercise and sport participation on this

mucosal immune system parameter will follow.

Secretory Immunoglobulin A (S-IgA) and Exercise. Perhaps the first examination of the effect of exercise on S-IgA was conducted by Tomasi, Trudeau, Czerwinski and Erredge (1982). Saliva samples were obtained from eight Nordic skiers before and after national cross country races. It was revealed that the athletes exhibited significantly lower S-IgA levels than age-matched controls. In addition, the skiers experienced a significant reduction in S-IgA levels pre to postcompetition. The researchers speculated that temporary S-IgA deficiency, especially during the interval immediately following competition, may be associated with enhanced risk of mucosal infection.

Tharp and Barnes (1990) studied the effect of training at various intensities in twenty-one competitive swimmers. Saliva samples were obtained before and after 2-hour training sessions on four different days with various exercise intensities (light, moderate, heavy-intensity and very light). The Profile of Mood States (POMS) was also administered to participants before training in order to assess their overall mood that day. The saliva samples were used to determine S-IgA and cortisol levels. S-IgA concentration was reduced after training on each day. However, the decrease was significant for the moderate training session only. Cortisol levels showed little change

from pre to posttraining except for a significant increase with the heavy-intensity session. No significant correlations were found between S-IgA, cortisol and total mood disturbance on the POMS.

In a study by McDowell, Chaloa, Housh, Tharp and Johnson (1991), laboratory exercise did not reduce S-IgA concentration. In study one, nine subjects engaged in three treadmill runs of 15, 30 and 45 minute duration at a heart rate which corresponded to approximately 60% of VO_2 max. In study two, nine subjects participated in three twenty-minute treadmill runs at a heart rate which corresponded to approximately 50, 65 and 80% of VO_2 max. Saliva samples were given before and immediately after, as well as one and two hours post exercise. Neither group exhibited significant changes in S-IgA concentration during the course of the study.

Findings appear to be mixed with regard to the effect of exercise on S-IgA. Since actual sport competition can be very different from exercise in a laboratory setting, it is important to consider field studies involving competitive games as well. In one of the few studies on this topic to date, Mackinnon, Ginn and Seymour (1992) followed elite athletes in training in order to determine if changes in IgA concentration occur. Saliva samples were taken from twelve female hockey players before and after three training and

four tournament matches. Analyses of the saliva showed that IgA concentrations were significantly lower during competition compared to training. Also, it was revealed that S-IgA levels decreased significantly after each match during training and competition. Interestingly, the decreases in S-IgA concentration were largest after the most difficult competition matches. S-IgA modifications with exercise are of interest due to the potential health consequences of such changes. Indeed, engagement in exercise or sport may influence risk for diseases such as respiratory infections.

Exercise and Risk for Respiratory Infection

Understanding the relationship between exercise, sport participation and respiratory infections has obvious public health implications (Nieman & Nehlsen-Cannarella, 1992). Indeed, upper respiratory infection (URI) may cause more acute disability among athletes than all other diseases combined (Schouten, Verschuur, & Kemper, 1988). Various groups of athletes exhibit an abnormally high incidence of URI. For example, Douglas and Hanson (1978) compared conditioned male crew athletes with unconditioned controls on frequency and severity of URI symptoms over a nine week period. The crew participants exhibited significantly more frequent and severe symptoms than the control group. Peters and Bateman (1983) examined the incidence of URI in marathon

runners as compared with matched controls. The runners were questioned the day preceding as well as two weeks after running a marathon. Thirty-three percent of marathoners and only 15.3 % of controls exhibited symptoms of upper respiratory infection. Interestingly, with regard to the runners, those who had slower race times showed no greater incidence of infection than controls. However, faster runners exhibited a much higher incidence of infection, indicating that sport intensity may be an important risk factor for infection.

Mackinnon, Ginn and Seymour (1991) followed elite athletes over a period of training in order to determine if changes in IgA levels are related to risk for URI. Saliva samples were obtained from fourteen squash and nineteen hockey athletes before and after training sessions. For hockey athletes, samples were taken daily over a ten day period. Samples were taken weekly over a 10-week period for squash participants. As diagnosed by a physician, five of 14 squash and five of 19 hockey athletes exhibited URI during the study. All but one of the athletes who displayed URI showed decreases in S-IgA levels after exercise within two day preceding the illness. Interestingly, S-IgA concentration decreased 22 and 27% from pre to posttraining session for squash and hockey athletes, respectively. In contrast, when athletes were not developing URI, S-IgA

concentration changed only slightly (within 10%) from pre to posttraining session.

Nieman and Nehlson-Cannarella (1992) proposed a J-shaped curve model to explain the relatioship between exercise and respiratory infection. According to the model, moderate exercise decreases risk, below that incurred by no exercise. However, excessive exercise puts one at heightened risk for infection.

The higher incidence of upper respiratory infection in athletes cannot be solely attributed to the detrimental effects of intense exercise. For example, sport participation also produces close physical contact with others, increasing the likelihood of virus contact. In addition, URI may be spread by indirect contact involving objects such as athletic equipment (Weidner, 1994). Sport competition also often produces intense emotional responses, which could alter immune system functioning via stress-immunomodulation.

Effects of Exercise on Mood

Many attempts have been made to study the effects of exercise and sport participation on mood. A recent review of the past two decades of literature on exercise and mood found at least some improvement in mood after acute exercise participation in over 85% of studies on the topic (Yeung, 1996).

Berger and Owen (1983) examined the influence of exercise on mood in one hundred college students enrolled in swim classes. Mood was assessed using a self-report scale, the Profile of Mood States (POMS), before and after the swim class. It was found that tension, depression, anger, confusion and vigor scores decreased significantly from pre to postexercise.

In a classic study on exercise and mood, McCann and Holmes (1984) randomly assigned forty-three depressed, undergraduate women to one of three conditions for a ten week period: aerobic exercise, placebo and no treatment. Aerobic capacity was assessed and the Beck Depression Inventory was completed before the study began. The aerobic exercise condition involved training in class twice weekly for a one hour interval. In addition, participants were required to exercise outside of class time to meet a minimum standard. Individuals assigned to the placebo condition were instructed to practice progressive muscle relaxation for 15-20 minutes per day for four days per week. They were also told to take leisurely walks of at least five-minute duration before each relaxation session. Participants in the no treatment group were simply told that their treatment had been temporarily postponed. Post-treatment results revealed that participants in the aerobic exercise condition showed greater improvements in aerobic capacity and reduced

depression as compared to individuals in the other two conditions.

In an attempt to understand the differential effects of high and low intensity exercise on mood, Steptoe and Cox (1988) admininisterred a modified POMS before and after exercise to thirty two female medical students. Each participant performed eight-minute bouts of both high and low intensity exercise. High intensity exercise produced significant increases in tension and decreases in vigor. Interestingly, however, low intensity exercise increased vigor scores.

The mechanism(s) reducing negative mood as a function of acute exercise participation are little understood. It has been proposed that elevations in endorphins mediate these changes (Markoff, Ryan, & Young, 1982). Even though much attention has been given to the endorphin-induced mood improvement with exercise hypothesis, attempts to correlate endorphin levels with mood changes have generally been unsuccessful (Yeung, 1996). Psychological theories have also been put forth to explain the finding of mood improvement with acute exercise. For example, according to the distraction hypothesis, exercise may reduce negative mood by providing time away from worrisome thoughts and daily stressors (Raglin & Morgan, 1985). In addition, according to the mastery hypothesis, mood improvement with

exercise results from a sense of achievement or mastery when an important task such as exercise is completed (Simons, McGowan, Epstein, Kupfer, & Robertson, 1985).

Competitiveness is an important factor to consider with regard to understanding emotional changes associated with athletic participation. In a study by Kerr and Svebak (1994), 109 male students were assigned to participate in a sport of low, medium or high antagonistic physical interaction, as determined by the investigators. Low, medium and high antagonistic interaction consisted of easy running in small groups, basketball, and rugby, respectively. Before and after the sport sessions, participants completed 2 stress inventories which measured arousal and tension. The students showed non-significant increases in self-reported arousal in all three conditions. Unpleasant mood scores decreased in the low antagonistic interaction group, but did not change in the medium group and actually increased in the high group, indicating that level of antagonistic interaction is a crucial variable affecting sport competition mood states.

Determining the emotional changes associated with exercise and sport may give researchers a more complete understanding of exercise-immune interactions. Indeed, the changes in mood as a consequence of exercise and sport participation may be of critical importance in clarifying

how immune system parameters such as S-IgA are altered.

Mood and S-IgA

Mood may be a crucial factor in terms of understanding how athletic participation alters S-IgA. Dillon, Minchoff and Barker (1985) assessed the relationship between mood and S-IgA levels in an experimental investigation. Ten college students were assigned to view humorous and didactic control videotapes. Each videotape was viewed for thirty minutes, with a ten minute session between viewing each tape. Counterbalancing was used to control for the order effects of tape viewings. Saliva samples were taken immediately before and after both viewings. S-IgA concentration was significantly greater after viewing the humorous videotape than before. However, there were no significant difference between pre and post measures of S-IgA for the control tape viewing.

Stone, Cox, Valdimarsdottir, Jahdorf and Neale (1987) monitored daily mood and S-IgA in thirty male dental students. The researchers began exposing participants to a harmless, albumin, orally-administerred antigen two weeks before the collection of saliva and questionnaire data. Antigen capsules were taken every morning over a ten week period. Saliva samples from parotid glands and self-report mood data were collected at three sessions per week. Saliva samples were analyzed for antigen specific S-IgA response to

the orally-administerred albumin as well as total S-IgA protein. Interestingly, the specific antibody response was higher on days with relatively high positive mood. The same response was lower on days with relatively high negative mood. However, total S-IgA protein was not significantly related to mood.

Kugler, Hess and Haake (1992) studied the relationship between mood state and S-IgA in thirty-nine male and forty-five female medical students. Mood was assessed by a short version of the Adjective List including fifteen dimesions: concentration, activation, dreaminess, anxiety, depression, good mood, sensitivity, irritation, excitement, self-reliance, numbness, sociability, tiredness, introversion and passiveness. Whole, unstimulated saliva samples were taken and assayed for total S-IgA protein. There was a significant positive correlation between excitement and S-IgA concentration. No other mood dimensions were significantly related to S-IgA levels.

In a study of S-IgA concentration and mood, Evans and Bristow (1993) asked twelve college students to render saliva samples and complete the Nowlis Mood Adjective Checklist over a 2-week period. Positive mood was found to correlate positively with IgA concentration.

Stumpf-Curio, Curio and Scholz (1994) monitored stress, mood and S-IgA concentration in two male and two female

healthy volunteers. Participants reported stress and mood once a day over a 100-day period. Unstimulated saliva samples were collected each morning throughout the duration of the study and assayed for S-IgA levels. The researchers found only three significantly correlations between S-IgA concentration and affect. With respect to males, S-IgA and bad mood were negatively correlated on the same day. In addition, a negative correlation was found between mood uplifts and S-IgA levels on the same day for one of the females. Also, a negative correlation was observed between mood uplifts and S-IgA concentration on the following day for one of the males. However, overall, no consistent relationship was found between mood and S-IgA in the study.

As stated previously, exercise is only one component of sport participation. Other factors, such as competitive outcome, are in need of consideration when examining the physiologial and emotional consequences of engagement in sporting activities.

Competition

According to Martens (1976), "Competition is a process in which the comparison of an individual's performance is made with some standard in the presence of at least one other person who is aware of the criterion for comparison and can evaluate the comparison process" (p. 14). Competition permeates the lives of humans and nonhuman

animals. In the animal literature, social competition has been treated as a means by which organisms arrange themselves in dominance hierarchies. The winners of agonistic encounters, usually referred to as dominants, have preferential access to resources over losers (subordinates). Similary, sport psychologists have viewed competition as an interaction involving the unequal distribution of rewards based on winning and losing (Martens, 1976).

Physiological Outcomes of Competition in Nonhumans

A plethora of studies have been conducted to understand the physiological consequences of competitive interactions in animals. It has been recognized that dominance rank and physiological measures are often related, especially when a rank order is initially being contested. Indeed, in a variety of animals neuroendocrine reactivity and cardiovascular reactivity have been shown to be associated with dominance rank. Since neuroendocrine reactivity (Henry & Grimm, 1990) and cardiovascular reactivity (Krantz & Manuck, 1984) have been implicated as important mediators in disease etiology, a dominance-subordinance model may be valuable for linking animal models to human research on risk factors for illness.

Cardiovascular Response. Support for the notion that social position has an influence on heart rate was obtained in a study by Cherkovich and Tatoyan (1973) on macaques and

baboons. The researchers assigned combinations of same-species animals into various groupings and measured heart rate via radiotelemetry. Heart rates were recorded at thirty minutes, one hour, two hours, three hours, and four hours after the introduction of group members to each other. Initially, a male and female macaque were paired. After thirty minutes, the female, which was subordinate to the male, showed a dramatically higher heart rate than the male. After one hour, the heart rate of the male seemed to decline to a resting level, whereas the heart rate of the female gradually decreased but remained significantly higher than that of the male. Then, the investigators removed the female from the cage and replaced her with a very strong male. Interestingly, the original male, who now became subordinate, exhibited HR levels similar to those of the female in the previous trial. It was now the new male who quickly declined to resting levels in terms of heart rate. The researchers performed a slightly different procedure with baboons. They formed an aggregation of two males and two females. Similar to the previous study, the heart rate of the dominant male soon recovered to what appeared to be a resting level. In this context it was the subordinate male (not either female) who showed the highest heart rate across all time intervals. The females initially displayed significant heart rate increases, which decreased up to the

four hour interval; however, their heart rates were never lower than that of the dominant male. Finally, the researchers removed the dominant male from the cage; predictably, the originally subordinate male assumed the highest dominance rank. Accordingly, his heart rate decreased, while the females reacted to the different grouping with a slight increase in heart rate at first, that soon declined to a point not quite as low as that exhibited by the male. The health implications of these findings were not addressed by the researchers.

Kaplan, Manuck, & Gatsonis (1990) found strikingly similar results with male cynomolgus monkeys. Twenty-nine animals were housed in groups of five that were periodically reorganized in a manner in which each monkey was grouped with either three or four new animals each time they were reorganized. Those animals tending to attain high dominance status across social groupings exhibited significantly lower resting (nocturnal) heart rates than low ranking animals. The researchers then examined the heart rate changes in four monkeys, which clearly changed social status from the first part of the study to the second. These monkeys showed inverse changes in heart rate: monkeys showing downward mobility exhibited higher resting heart rates and those rising in status showed lower rates, again supporting the idea that social factors may be of primary importance in

determining this measure.

Endocrine Response. The influence of dominance status on hormone levels has also been examined. Brain (1973), using mice as subjects, housed adult males in pairs for 17 days. Dominance status was determined near the end of this period. On the eighteenth day, the mice were sacrificed and various physiological measurements were made. Those mice classified as subordinate had heavier adrenals but lighter ventral prostates and preputial glands than dominants. The author interpreted this as meaning the dominants showed higher gonadal activity and lower adrenocortical stimulation than subordinates.

A study by von Holst, Fuchs, & Stohr (1983) on tree shrews produced very dramatic and intriguing results. A pair of tree shrew adult strangers placed in a laboratory cage will vigorously attack one another until a victor emerges. The loser then cowers in a corner, motionless, constantly watching the winner. Even though further altercations are rare between two such conspecifics, the submissive animal normally dies in fewer than twenty days. It is not the damage done during fighting that ultimately leads to the doom of the subordinate; rather, it is the constant "anxiety." With this in mind, the researchers designed their experiment to determine the biochemical changes of tree shrews involved in such encounters. After

careful observation, it was noted that losers could be classified as either submissive or subdominant. The former, subsequent to defeat, sat in a corner of the cage and made very little or no response to external stimulation. The latter remained active, at first trying to escape the cage and also responding to the dominant by either fleeing or fighting. Submissives died in less than eight days, presumably from kidney dysfunction. Biochemical tests revealed that cortisol increased markedly in submissives but not in dominants or subdominants. Also, testosterone levels were lowest in submissive animals and highest in their dominant counterparts. Interestingly, norepinephrine levels were greatest in subdominants, with dominants and submissives having similar levels.

Immune Response. The relationship between social status and immune parameters has not received much research attention. There have been a few non-human studies on this topic, however. For example, Fauman (1987) assigned mice to either pairs or groups and observed their dominant and submissive behaviors. Subsequently, all animals were exposed to a harmless antigen. Blood samples were obtained 7 days after immunization and specific antibody levels were determined for each mouse. The dominant mice exhibited reduced antibody responses as compared to subordinates and isolated controls. Also, submissive animals displayed antibody

responses equivalent to or significantly above that of isolated controls. Thus, dominants produced weaker immune responses than submissives and controls.

Fleshner, Laudenslager, Simons and Maier (1989) compared the antibody responses of male Sprague-Dawley rats assigned to either resident or experimental conditions. Resident animals were housed in pairs and experimental (intruders) individuals were housed singly. Experimental animals were exposed to residents in five successive ten-minute sessions twice during the course of the experiment. The time period between exposures was one week. Experimental rats were further divided into two groups: control and aggressive. Individuals in the control group were placed into the residents' boxes, with air-permeable, Plexiglass barriers between residents and controls. Animals assigned to the aggressive group were placed directly into the resident's quarters (the Plexiglass barrier was removed).

All experimental (intruder) animals were injected with a foreign protein, keyhole limpet hemocyanin (KLH), immediately before the first exposure to resident dyads. Blood samples were obtained one, two and three weeks after antigen exposure. Serum levels of KLH-specific IgG antibodies were determined after all samples were obtained. As expected, all animals showed elevated KLH-specific

antibody levels over the three week sampling period. However, experimental animals placed directly into the resident' quarters exhibited significantly fewer serum antibodies to KLH than controls two and three weeks after antigen exposure. Interestingly, animals that spent the most time in submissive postures (at week one) showed the most reduced antibody responses. These results indicate that submissive behavior may compromise the antibody response to antigens.

Physiological Outcomes of Competition in Humans

If social interaction in nonhuman animals parallels that found in humans in fundamental ways, we should expect similarities in their physiological profiles. However, comparisons should be made cautiously, with an appreciation for differences between species and for the importance of the contexts in which the social interactions occur.

Cardiovascular Response. Rejeski, Gagne, Parker, and Koritnik (1989) examined the cardiovascular reactivity of dominant and submissive males. Dominance and submissiveness were determined by extreme scores on the Adjective Check List on these two personality dimensions. Each subject participated in a debate with a trained confederate on the legitimacy of abortion, while being monitored for heart rate, systolic, diastolic and mean arterial blood pressure. Subjects were told that their interpersonal dominance/power

would be judged during the task via their biological responses, and that biofeedback (actually bogus) would be given to them to inform them of their dominance status. The biofeedback was simply an attempt to make the subjects exhibit a high level of dominance behavior. Participants were given one minute to decide their position on the issue and then the four minute debate was begun. The results for males were inconsistent with those found for females. Submissive males had significantly higher heart rates than dominants during the middle portion of the task, and there were no significant differences between dominants and submissives on systolic, diastolic and mean arterial blood pressure. It should be noted, however, that a female confederate was used to challenge both male and female subjects. Hence, dominant males may not have responded to the debate task as they would have with a male experimenter. The researchers speculated that submissive males may have showed greater heart rate reactivity due to the stress associated with maintaining their self-image while in the presence of the female confederate. It is also possible that submissive males would display greater heart rate reactivity in many interpersonally stressful situations, as research with nonhuman animals has shown (Cherkovich et al. 1973).

Smith, Allred, Morrison, and Carlson (1989) studied

male undergraduate pairs (who were not previously acquainted) during discussions. Heart rate and blood pressure were recorded during a baseline period, and then throughout the discussion task involving assigned topics. Each pair consisted of one subject assigned to a primary role and one subject in a secondary role. Also, the primary participants were put in either a contingent or noncontingent condition. All students were told they would be given one to six entries in a drawing for a $50 prize. Subjects in the secondary role condition were told that following the task the experimenter would roll a die to determine their number of entries. For participants in the primary role, the noncontingent condition consisted of instructions identical to those received by the secondary subjects: his number of entries in the lottery would be determined randomly. However, primary subjects in the contingent condition were told that their number of entries would be determined by the degree of attitude change they were able to induce in their partners. The discussion task included a randomly assigned pro or con position on each of four topics. Each subject was asked to speak for two 30 second periods and listen for two 30 second periods during the task. Subjects in the contingent condition showed higher HRs, SBP, and DBP than participants in the noncontingent condition. These findings demonstrate the

effects of effortful attempts at interpersonal influence on cardiovascular reactivity. If the attempts at persuasion can be interpreted as the striving for interpersonal dominance, the results of the study suggest of the importance of dominance on the stress response in humans.

Rejeski, Parker, Gagne and Kortnik (1990) studied the cardiovascular responses of ten dominant and ten submissive female undergraduates using the same procedure as that used with males. It was found that dominant females displayed significantly greater systolic, diastolic and mean arterial blood pressure as well as heart rate during the task than submissive women. But at the end of a ten minute recovery period, there were no significant differences between dominants and submissives on the cardiovascular measures. Although the sample size was small, these results suggest that females with a dominant personality type respond to interpersonal disagreements with enhanced cardiovascular reactivity compared to those with a submissive personality.

Endocrine Response and Possible Implications for S-IgA.

Support for the notion that hormonal changes are a function of competitive outcome is revealed in an intriguing study by Mazur, Booth and Dabbs (1992). Saliva samples were rendered from sixteen male chess players the day before and immediately before and after competing in tournaments. Testosterone was found to be higher in winners than losers

after the matches.

In a study by Gladue, Boechler and McCaul (1989) competitive outcome was controlled by the experimenters. Saliva samples were taken throughout a non-athletic reaction time task. Winners showed higher post-competition salivary testosterone levels than losers. The authors concluded that there is specificity to the hormonal changes associated with competition. Indeed, these modifications may be related to status or mood alterations.

Booth, Shelley, Mazur, Tharp and Kittok (1989)also examined the influence of competitive outcome on testosterone. Six university male tennis players were studied during six matches. Saliva samples were taken the day before, fifteen minutes before, immediately after and the day after or two days after each match. Salivary testosterone concentration rose dramatically between the preceding day and fifteen minutes prematch. From pre- to postmatch, however, salivary testosterone increased for winners but decreased for losers. Interestingly, winners exhibited higher testosterone levels than losers before their next matches.

Indeed, victory or defeat may produce differential endocrinological modifications. Since hormones regulate the immune system (Stanisz, et al., 1994; Weicker & Werle, 1991), competitive outcome could differentially affect

immune functioning. With regard to mucosal immunity, there is some evidence that testosterone may be immunoenhancing. Although androgen receptors have yet to be discovered on lymphocytes, testosterone on lacrimal glands has been shown to increase S-IgA secretion (Sullivan & Hann, 1989). Also, it is documented that females, who have lower testosterone levels than males, are generally more at risk for diseases involving the mucosal surfaces (Stanisz, Kataeva & Bienenstock, 1994). By integrating these findings, one might suspect that, for males, winning a competition could enhance S-IgA secretion due to enhanced testosterone levels.

Control

Few attempts have been made to explain why dominant and subordinates animals have differential stress responses. According to Henry (1986), "The basic question facing any mammal is the degree of control that it can exert over its environment" (p. 37). Of central importance is Henry's notion of neuroendocrine differentiation of the stress response, which depends on whether or not an animal perceives that it has control. When an organism perceives something as easy to deal with, it may respond actively and then the sympathetic adrenal medullary (SAM) system becomes activated. Many physiological changes occur as a result, including an increase in the release of norepinephrine. When an event becomes less easy to handle, more passive

coping techniques are employed as the animal begins to lose control. Under these conditions, the SAM system remains active and increases its release of epinephrine, among other biochemicals. Finally, if the event becomes even less certain, distress sets in and the hypothalamic pituitary adrenal cortical (HPAC) axis becomes activated. One result of this is an increase in the release of cortisol from the adrenal cortex. The significance of this psychoneuroendocrine model in terms of dominance/subordinance is that dominant animals often exert more control over their environments than subordinates. Therefore, we should expect physiological differentiation in the stress responses of dominants and subordinates. Specifically, dominants should show more SAM axis activation and less HPAC system activation than subordinates. Since risk for particular diseases may be differentially affected by varying patterns of stress physiology, dominance status in a competitive encounter may be predictive of disease risk as well.

Power and Status

Winning a competition may confer status or power gain whereas losing may result in status or power loss. According to Kemper (1991), power may be understood as "a form of relationship through which one actor can get his or her way in a social action, even when another resists or is

opposed" (p. 332). Thus, competition can be regarded as a contest for power between competitors. Status, on the other hand, has been viewed as "a form of relationship in which one actor complies voluntarily with the wishes, desires, interests, and needs of another.... It is characterized by acceptance, respect, congeniality, friendship, sociability, helpfulness, inclusion and, in contexts where normatively permitted, intimacy" (Kemper, 1991, p.332). Indeed, competitions are not normally characterized by status-accord between competitors. However, others may accord status to winners of competitions. Kemper (1991) generated hypotheses about the emotional outcome of status and power gains and losses. They are as follows:

1) Power gain by self or power loss by other instigates happiness/security.

2) Power loss by self or power gain by other instigates fear/anxiety.

3) Status gain by self (where self is the responsible agent) instigates pride.

4) Status gain by self (where other or circumstance is agent) instigates joy or happiness.

5) Status loss by self (where self is agent) instigates shame.

6) Status loss by self (where self is agent in an irremedial sense, or where circumstance - fate, God, Life, or other

intractable force - is responsible) instigates sadness-depression.

7) Status loss by self (where other is agent) instigates anger.

8) Status gain by other instigates happiness (in self) if one likes the other, and unhappiness if one doesn't.

9) Status loss by other (where self is agent) instigates guilt/shame (in self) if one likes the other, happiness if one doesn't.

10) Status loss by other (where other or circumstance is agent) instigates unhappiness (in self) if one likes the other, happiness if one doesn't.

Assuming that winning a sport competition confers power and status to the individual and losing is associated with power and status loss, the physiological consequences of sport may be differentially affected by competitive outcome. These differences may have important influences on health.

CHAPTER III

RATIONALE AND HYPOTHESES

The specific physiological demands of actual tennis match play is poorly understood. The most comprehensive examination of this topic was conducted by Bergeron, Maresh, Kraemer, Abraham, Conroy, & Gabaree (1991). The researchers monitored heart rate, hematocrit, hemoglobin, blood glucose, and plasma concentrations of cortisol and testosterone in ten male university tennis players during near match play conditions. All matches consisted of 85 total minutes of play, divided into 15 minute segments. Each segment was followed by physiological sampling. Some physiological measures (e.g., heart rate) were taken continuously throughout play.

Heart rate increased from a pre-exercise value of 75.6 to 144.6 beats per minute during play. However, plasma lactate and blood glucose concentrations did not change significantly during play and recovery. However, blood glucose did increase by 23% between the post-warmup value and the second 15 minute segment, but remained steadily elevated above the pre-exercise value subsequently. Plasma cortisol levels gradually decreased during play; and postmatch levels were significantly lower than preexercise values. However, plasma testosterone concentration

gradually increased throughout the warmup and match play; and postexercise concentration was significantly greater than prematch levels. Based on the findings, the researchers suggested that "Although tennis is characterized by periods of high-intensity exercise, the overall metabolic response resembles prolonged moderate-intensity exercise" (p. 474). However, the authors acknowledged that "real" competition may have produced different changes. For example, cortisol levels may have been higher, due to psychological factors such as anxiety.

The study of the relationship of sport competition, mood and S-IgA is a promising area of inquiry. The current study examined the influence of tennis competition on mood and S-IgA. In addition, the effect of competitive outcome on mood and S-IgA was assessed.

The hypotheses of this study are as follows: (1) Participants will exhibit a decrease in S-IgA concentration from pre to postmatch; (2) Male winners will exhibit higher postcompetition S-IgA levels than losers; (3) Losers will report higher postmatch total mood disturbance (TMD), anger (A), depression (D) and tension (T) levels than winners; and(4) Total mood disturbance (TMD) will correlate negatively with S-IgA concentration pre and postmatch.

CHAPTER IV

METHODS

Participants

Twenty-six tennis players (21 males, 5 females) competing at four Knoxville, Tennessee area summer tournaments (Ruby Tuesday Tournament, Falcons For Food Tennis Classic, Harriet Tubman Championships and Holston Hills Tennis Classic) participated in this study. The mean age of the participants was 35.8 years and the range was 16-64 years. The tournaments were played outdoors, except for the Ruby Tuesday event which was held in an indoor facility. All play occurred between 8:16 A.M. and 10:23 P.M. The mean length of play was 74.8 (S.D.= 31.1) minutes and the range was 39-186 minutes. However, due to a rain delay, one match was interrupted for 112 minutes. Each participant received a packet containing a brief description of the requirements for participation, a consent form, questionnaires (See Appendix C) and two test tubes.

Instruments Used and Procedures

Profile of Mood States (POMS)

Upon signing the consent form, all participants completed the 65-item POMS (McNair, Lorr and Droppleman, 1992) 10-30 minutes before tournament play. The POMS has achieved wide acceptance as a measure of psychological

distress in a variety of healthy, physically ill and psychiatric populations. In addition, it has been used extensively in sport research (Renger, 1993). The POMS was scored through the used of overlay stencils provided by the Educational and Industrial Testing Service. Standard scoring of the POMS was used to yield a global distress score, referred to as Total Mood Disturbance (TMD), as well as scores for six mood factors: Tension-Anxiety (T), Depression-Dejection (D), Anger-Hostility (A), Vigor-Activity (V), Fatigue-Inertia (F) and Confusion-Bewilderment (C). Factor T is defined by adjectives descriptive of musculoskeletal tension. Factor D represents a mood of depression accompanied by a sense of personal inadequacy. Factor A represents a mood of anger and antipathy towards others. Factor V is defined by adjectives suggesting a mood of vigorousness, ebullience and high energy. Factor F represents a mood of weariness, inertia and low energy levels. Factor C appears to be characterized by bewilderment and muddleheadedness (McNair, Lorr and Droppleman, 1992).

The directions for completing the scale were as follows: "Below is a list of words that describe feelings people have. Please read each one carefully. Pleas place a mark under the answer to the right which best describes How You Feel Right Now." The items were responded to on a 5-

point adjective rating scale: 0 = Not at all; 1 = A little; 2 = Moderately; 3 = Quite a bit; 4 = Extremely. Completion of the scale took 3-6 minutes. At the conclusion of play (withing 5-15 minutes), the POMS was readministered using the same instructions as before.

Scores were obtained for each mood factor by summing responses made for the adjectives defining the particular factor. The TMD scores were obtained by summing the scores on the six mood factors (with V weighted negatively). The internal consistency of the POMS is acceptable and has been calculated for each subscale, as administered to adults: r(T) = .90; r(D) = .92; r(A) = .92; r(V) = .90; r(F) = .91 and r(C) = .83 (r = Cronbach's alpha).

In order to assess the importance and expected enjoyability, as well as confidence in winning the upcoming match, a short three-item, five-point questionnaire was constucted. The questions were administered upon completion of the POMS. Additionally, gender and age were requested (see Appendix A).

Saliva Collection and Analyses

Upon completion of the questionnaires, unstimulated saliva samples were collected. After rinsing their mouths with water, participants provided approximately 3 ml of saliva by spitting into a centrifuge test tube up to a marked volume. The test tubes were immediately capped,

wrapped with Parafilm, and placed on ice. All samples were transferred to a freezer within 3 hours of collection and stored at -20 C until assayed. After completion of the tennis match and second administration of the POMS, participants rendered another saliva sample using the same procedure as before.

The saliva samples were analyzed for total immunoglobulin A (IgA) concentration by Enzyme Linked Immunosorbent Assay (ELISA) (Voller & Bidwell, 1986). The samples were thawed and centrifuged at 2000 revolutions/minute for ten minutes and checkerboard assays were initially performed to determine appropriate saliva, mouse anti-human IgA and human IgA standard concentrations. ELISA (Dynatech, Immulon 4) flat-bottom plates were coated with 100 ul of mouse monoclonal anti-human IgA (Sigma Chemical Company, St. Louis, MO) diluted in carbonate buffer (pH 9.6 - 9.8) and allowed to incubate overnight in the refrigerator at 4 C. Following three washes with phospate-buffered saline (PBS) containing 0.05% Tween 20, ph 7.4 (PBST), plates were blocked with 200 ul/well PBS with 3% dehydrated milk, incubated at 37 C for two hours, and washed with PBST as before. Two-hundred microliters of human IgA standard (Sigma Chemical Company), St. Louis, MO) of a known concentration in PBST were added to duplicate wells coated with anti-human IgA and serially diluted. One standard (in

duplicate) was used per plate, with saliva concentration identical on all plates. Two-hundred microliters of saliva sample (diluted in PBST) were added in duplicate to wells coated with anti-human IgA and diluted similarly. The plates were incubated for two additional hours at 37 C. After three washed with PBST, 100 ul of anti-human IgA peroxidase conjugate (Sigma Chemical Company, St. Louis, MO)(diluted in PBST) were added to each well. Next, the plates were incubated at 37 C for one hour. After three additional washes with PBST, 100 ul of 2,2-azino-bis-3-ethylbenz-thiazoline-6-sulfonic acid substrate were added to each well. The plates incubated at room temperature for a period of appoximately 20 minutes. The color intensity was measured by specrophotometer (Biotek Instruments, Burlington, VT) using wavelength 405 nm. All samples were assayed in duplicate and the average of the absorbance values was used a the representative value. The concentration of IgA was determined from the standard curve formula (Cricket Graph Version 1.3, Malvern, PA).

CHAPTER V

RESULTS

Secretory IgA (S-IgA)

In order to examine the influence of the tennis competition on S-IgA, the data were analyzed by a 2 (won vs lost) X 2 (pre vs postmatch) analysis of variance (ANOVA) with repeated measures. Figure 1 displays the S-IgA levels for males, and figure 2 illustrates S-IgA levels for females. Because of the small number of female players, competition outcome comparisons were not made with respect to S-IgA. For males, there was a trend toward a main effect for outcome (Appendix A, Table 1), [$F(1,15) = 3.01$, $p < .10$], as male winners showed a trend toward greater S-IgA levels than losers. However, there was no main effect for time [$F(1,15) = .09$, $p > .05$], or time by outcome interaction [$F(1,15) = .01$, $p > .05$]. For females, there was no main effect for outcome [$F(1,2) = .20$, $p > .05$]. However, a main effect for time of sample was obtained, as postmatch S-IgA concentrations were significantly higher than prematch levels [$F(1,2) = 18.17$, $p < .05$]. However, there was no interaction effect ($p > .05$). Missing data for S-IgA are indicated in tables B-6 through B-9.

Figure 1

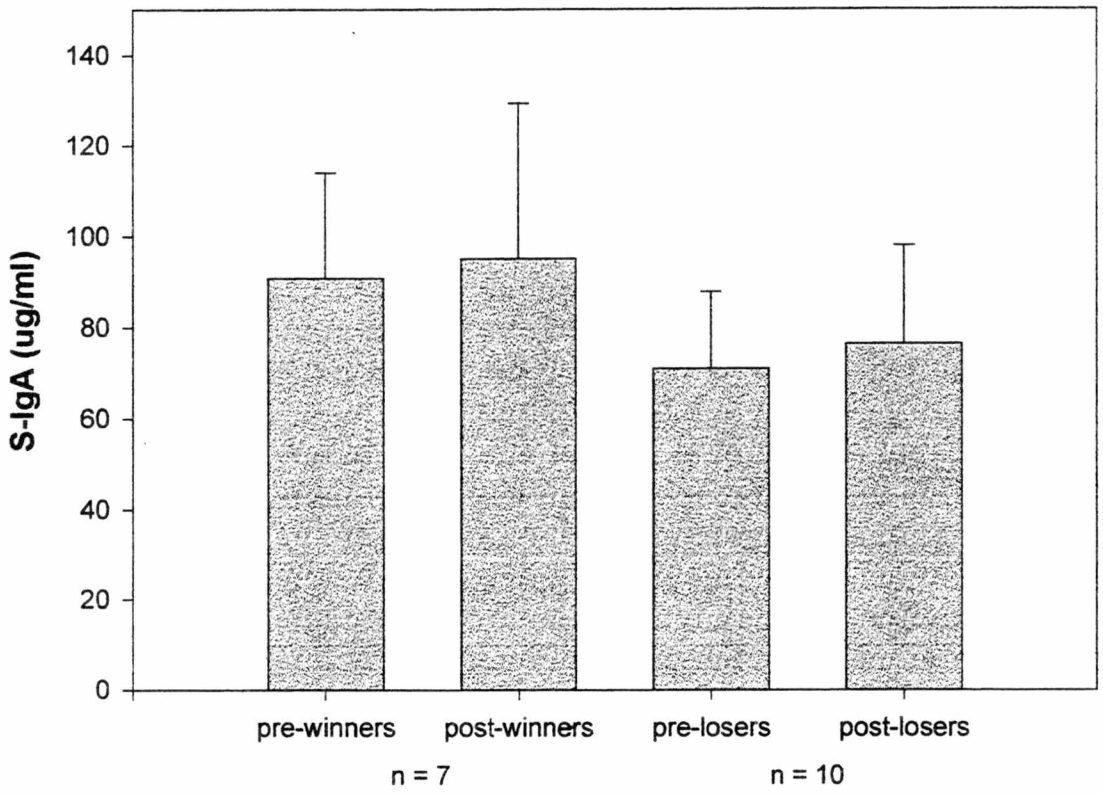

Figure 1. Salivary IgA response to tennis competition in males. Bars represent SEM.

Figure 2

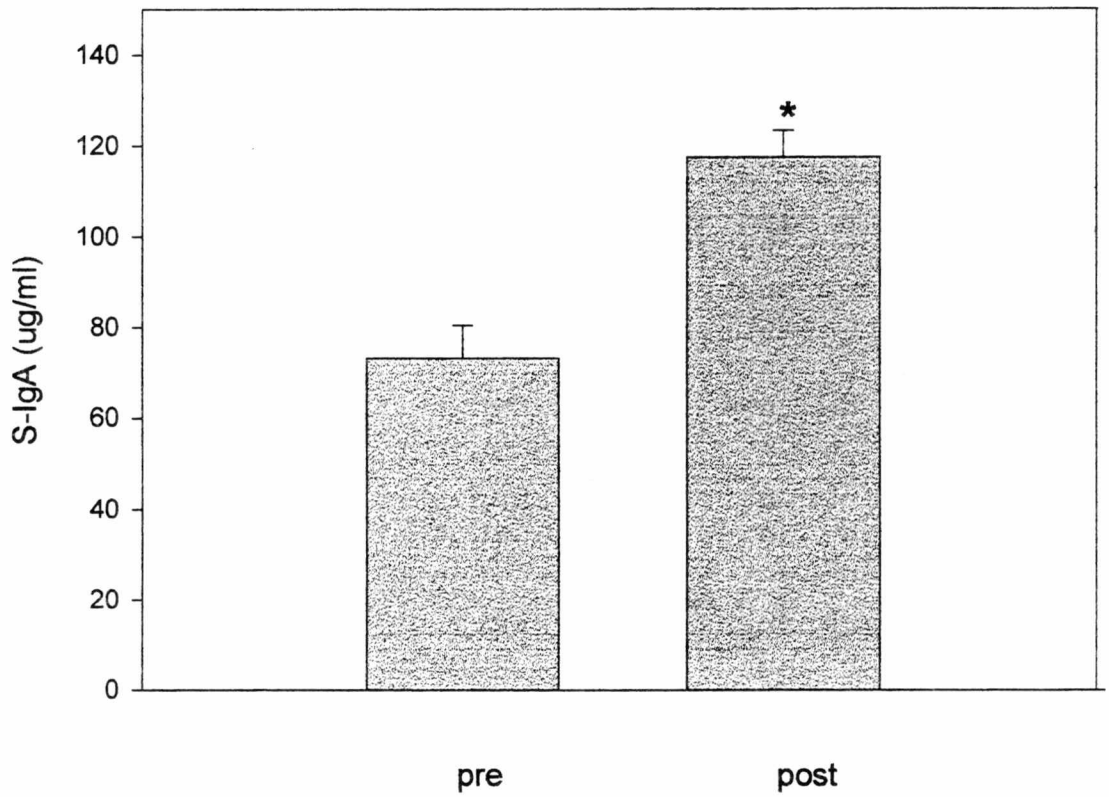

Figure 2. Salivary IgA response to tennis competition in females. Bars represent SEM. * $p < .05$

Mood

Table 1 presents the means and SEMs for the POMS. In order to determine the effects of time of sample and competitive outcome outcome on mood and their interactions a 2 (outcome) X 2 (time) repeated measures multivariate analysis of variance (MANOVA) was performed on the data, with total mood disturbance (TMD), anger (A), depression (D) and tension (T) scores as dependent variables. A multivariate main effect for competitive outcome [Wilk's Lambda = .59, $F(4,21) = 3.60$, $p < .05$], time [Wilk's Lambda = .41, $F(4,21) = 7.62$, $p < .05$], and their interaction [Wilk's Lambda = .62, $F(4,21) = 3.19$, $p < .05$] occurred. (See Appendix A, Table 2 for all analyses)

Following the significant multivariate effects, univariate analyses were performed on each dependent variable. With regard to TMD, losers were significantly higher than winners [$F(1,24) = 12.68$, $p < .05$]. However, there was not a main effect for time [$F(1,24) = 2.02$, $p > .05$]. In addition, there was not a significant outcome by time interaction [$F(1,24) = 5.91$, $p < .05$]. (See Appendix A, Table 3 for all analyses) Tukey's Honestly Significant Difference (HSD) post hoc procedure indicated that losers exhibited significantly higher TMD scores than any other group after the match (HSD = 16.67).

Losers displayed significantly higher anger (A) scores

Table 1. Means and standard errors for POMS in winners and losers. T1 = prematch; T2 = postmatch

	Winners		Losers	
	T1	T2	T1	T2
TMD	3.4(3.4)	-2.0(2.8)	12.1(4.2)	32.6(8.2)
Anger	1.7(.5)	1.2(.8)	3.3(.9)	8.3(2.5)
Depression	3.7(1.5)	.7(.5)	2.7(1.2)	8.6(1.9)
Tension	8.8(4.7)	4.7(1.1)	13.4(1.4)	9.7(1.7)

than winners, [$F(1,24) = 6.88$, $p < .05$]. However, there was no main effect for time [$F(1,24) = 1.98$, $p > .05$]. In addition, there was a trend toward an outcome by time interaction [$F(1,24) = 3.06$, $p < .10$].

Losers also reported marginally higher depression (D) scores than winners [$F(1,24) = 3.78$, $p < .10$], but there was no main effect for time [$F(1,24) = 1.42$, $p > .05$]. However, there was a significant outcome by time interaction [$F(1,24) = 13.58$, $p < .05$]. Tukey's HSD post hoc analysis revealed that losers, postmatch, reported significantly higher depression scores than any other group (HSD = 3.77).

In addition, losers reported significantly greater tension (T) scores than winners [$F(1,24) = 6.69$, $p < .05$]. There was also a main effect for time [$F(1,24) = 13.55$, $p > .05$], as tension scores were significantly higher pre than postmatch. There was no outcome by time interaction for tension [$F(1,24) = .04$, $p > .05$].

Correlational Data

In order to assess the relationship between S-IgA, mood, and competitive outcome, correlational analyses were performed on the data. Table 2 presents the results. A significant negative correlation was found between S-IgA and TMD before the match, $r = -.42$, $p < .05$. However, S-IgA levels and TMD scores did not correlate significantly after play, $r = .04$, $p > .05$. Competitive outcome (CO) did not

Table 2. Correlation matrix--total mood disturbance(TMD), secretory immunoglobulin A (S-IgA) and competitive outcome (CO). (N = 26 for all variables except S-IgA where N = 22). * significant $p < .05$

	S-IgA	TMD	CO
		Prematch	
S-IgA	-	-.42*	.19
TMD		-	-.22
CO			-
		Postmatch	
S-IgA	-	.04	.11
TMD		-	-.54*
CO			-

correlate significantly with S-IgA prematch, r = .19, p > .05 or postmatch, r = -.22, p > .05. All other (unplanned) correlations can be found in Appendix B.

Additional Analyses

The responses of the following three items were used for descriptive information and were not used in any statistical analyses due to the low reliability of one-item construct measures (John Lounsbury, personal communication).

stongly disagree = 1; disagree = 2; undecided = 3; agree = 4; stongly agree = 5

1. I perceive this match to be very important as compared to other matches I have played this summer.

The mean response on this item was 3.54 (S.D.= .90).

2. I expect this match to be enjoyable.

The mean response on this item was 4.00 (S.D.= .89).

3. I expect to win this match.

The mean response on this item was 3.77 (S.D. = .91).

CHAPTER VI

DISCUSSION

The purpose of the study was to investigate the influence of sport competition on mucosal immunity and mood. It was hypothesized that tennis participants would show decreased S-IgA levels from pre to postmatch due to the moderately intense exercise requirements of match play (Bergeron, et al., 1991). Furthermore, it was predicted that winners would exhibit significantly higher postmatch S-IgA levels than losers. Overall, there was not a main effect for time (pre versus postmatch) on S-IgA. However, for females (but not for males) postmatch levels of S-IgA were significantly higher than prematch concentrations. The finding of a gender difference in S-IgA response to the tennis competition warrants further investigation. Since only a small number of females participated in the study, a larger sample is needed in order to make valid gender comparisons. In terms of competitive outcome, there was no significant difference found between winners and losers on S-IgA. However, losers reported significantly greater mood disturbance than winners.

The mechanisms involved in S-IgA modifications are not well established. However, several factors are known to influence S-IgA changes. For example, exercise

participation produces dehydration, a condition associated with reduced saliva flow. Although the findings are mixed, saliva flow rate is sometimes negatively correlated with S-IgA concentration (Bosch, Brand, Ligtenberg, Bermond, Hoogstraten & Amerongen, 1996). Thus, the reduction in saliva flow with sport participation could increase S-IgA levels without exerting any influence on the plasma cells responsible for antibody production. Furthermore, the experience of psychological stress and exercise participation, which are associated with increased sympathetic nervous system activation, may also reduce saliva flow (Bosch, et al., 1996; Mackinnon, et al., 1993). In the present study, flow rate was not measured. Thus, S-IgA concentration increases in females may have been due to reduced saliva flow rate.

High S-IgA levels due to reduced saliva flow are doubtful to enhance protection against pathogens since a decrease in saliva flow hinders another function of saliva: washing away harmful microorganisms (Dawes, 1987). In terms of the competence of the mucosal immune system, it is probably important to have a sufficient concentration of S-IgA as well as adequate saliva flow rate. S-IgA secretion rate, which can be determined by multiplying saliva flow rate by S-IgA concentration, is an index of the total amount of S-IgA available in the oral cavity. Assuming that a

relatively large amount of S-IgA in the oral cavity enhances mucosal immune system effector functioniong, antibody secretion rate should be a valid measure of mucosal immunity. However, several longitudinal studies have supported the predictive utility of S-IgA concentration (Isaacs, Webster & Valman, 1984; Mackinnon et al., 1991; Rossen et al., 1970), whereas S-IgA secretion rate is unproven in this regard.

Smith and Weidemann (1990) proposed that exercise-induced immunological changes may be due to hormonal alterations. Moderate exercise may increase resistance to infection via an increase in the release of certain immunostimulatory factors such as growth hormone, prolactin, certain cytokines and possibly substance P. However, with strenuous exercise (when a threshold of work intensity is reached) the beneficial effects could be negated by an elevation in immunosuppressive factors such as glucocorticoids and opioids. The mechanisms by which these hormones affect the mucosal immune system specifically are not well understood.

In terms of competitive outcome, it was predicted that male winners would exhibit higher postmatch S-IgA levels than losers due to testosterone increases in male winners relative to losers. Although testosterone levels were not measured in the present study, it was found that male

winners showed a trend toward higher S-IgA levels from pre to postmatch than losers. Testosterone has been shown to suppress systemic immune parameters, but it seems to have the opposite effect on the mucosal immune system (Stanisz, et al., 1994). For example, it has been found to increase the accumulation of IgA in lacrimal glands and tears (Sullivan & Hann, 1989). In addition, receptors for testosterone have been located on IgA-secreting plasma cells in the mucosal immune system of mice (Parr, Ren, Russell, Prins & Parr, 1992), suggesting that IgA secretion may be directly influenced by testosterone. Male winners in competition tend to show increases in testosterone relative to losers; and the effect has been observed in a variety of contexts including tennis competition (Booth et al., 1989), wrestling matches (Elias, 1981) and even non-athletic competitions such as chess (Mazur, et al., 1992) and reaction time tasks (Gladue, et al., 1989). Moreover, male winners also tend to display greater testosterone levels before competitions than losers (Mazur, et al, 1992; Booth et al., 1989). Although not statistically significant, the higher S-IgA concentration in male winners from pre to postcompetition could have been due to the relatively high testosterone levels in these individuals.

Since competitive outcome was not officially determined until the end of the match, there was only a mean time

interval of 9.7 (S.D.= 4.1) minutes between the time competitive outcome was determined and postmatch saliva sample collection. Ten minutes may have been too short to produce S-IgA modifications. It is possible, however, that decisive control over a match functionally lengthened the aformentioned time interval. In other words, domination of an opponent may have occurred well before the end of a match. Hence, in these cases, the time interval may have been long enough for S-IgA changes to occur. Indee, significant S-IgA changes have been shown to occur in as little as twenty minutes (Green, Green, & Santoro, 1988).

In the present study, female players displayed an elevation in S-IgA levels from pre to postmatch. Currently, there is no evidence that acute S-IgA increases confer enhanced protection against infection. However, in a study on female squash athletes average S-IgA concentration decreased 22% (to 30.5 ug/ml) after exercise on days preceding illness. However, on days not preceding illness the mean S-IgA level increased to 47.3 ug/ml after execise (Mackinnon et al., 1992). Pretraining levels were the same for females who subsequently became ill and those who did not. Thus, in the present study the tennis prematch-postmatch increases in S-IgA in females is consistent with a pattern associated with protection against respiratory infection. The expected negative correlation between mood

disturbance and S-IgA was only found prematch. It is difficult to determine whether or not mood changes precipitate S-IgA modifications or vice versa. Perhaps mood and S-IgA covary as a function of a third variable, yet to be determined. Since the present study was correlational, the possibility that immune system changes influenced mood cannot be ruled out. Some support for the notion that mood alters S-IgA was found by Green et al., (1988). These researchers assigned twenty-four individuals to either a twenty-minute per session relaxation training group or a waiting list control condition. Saliva samples were collected before and after relaxation training sessions at the beginning and end of a three-week period. S-IgA secretion rates increased significantly from pre to post relaxation period, supporting the idea that mood can influence S-IgA. One possible explanation for the enhanced secretion rate of S-IgA during periods of relaxation involves the neurotransmitter acetylcholine. With relaxation, there is an increase in parasympathetic nervous system activation, which increases the release of acetylchloline into mucosal areas containing S-IgA-secreting plasma cells. Acetylchloline administration has been demonstrated to increase rat intestine S-IgA secretion rates (Wilson, Soltis, Olson & Erlandsen, 1982) and it is suggested that a similar effect of acetylcholine on S-IgA

secretion into the oral cavity in humans may occur.

The clinical significance of S-IgA levels and their modifications is not established. A longitudinal study by Rossen et al.(1970) revealed that S-IgA total protein concentration in nasal washings was lower in individuals who later developed infection after experimental virus exposure. Moreover, S-IgA total protein before inoculation correlated positively with specific antibody upon subsequent experimental antigen exposure. The average initial S-IgA values for individuals who did not succumb to infection was .28 mg/ml, whereas the initial value for individuals who later exhibited infection was .10 mg/ml. In a study comparing children prone to respiratory infections with age-matched healthy controls, salivary IgA were on average approximately 14 ug/ml in the former and 28 ug/ml in the latter (Lehtonen, Tenovuo, Aaltonen, & Vilja, 1987). The difference between groups was only appoximately 14 ug/ml. However, the infection-prone group also had twice as much antibody as the control group.

Research is needed to provide standards for immune system parameters in order to interpret risk probabilities at given levels. However, one must recognize that single parameters should be interpeted in a global immunological context. For example, a competent systemic immune system could compensate for a deficiency in S-IgA.

The changes in mood associated with the tennis competition cannot be purely attributed to the exercise alone. Indeed, the anticipation of the match, the antagonistic interaction associated with competing, judgments about performance and the outcome of the competition are all important factors which may influence mood. Acute exercise generally results in a reduction of negative mood (Yeung, 1996). In a study by Lichtman and Poser (1983), POMS anger (A), depression (D) scores were significantly reduced from before to after physical exercise. In the current study, postmatch tension (T) scores were significantly lower than prematch values. The reduced T scores may be a result of exercise itself. Alternatively, prematch T may have been elevated due to anticipation of the upcoming competition. According to Kemper (1991) fear/anxiety is associated with the anticipation of power loss by self or power gain by others. In addition, high prematch tension/anxiety may be attributable to uncertainty in meeting performance expectations. Tension scores were also higher in losers than winners, collapsed across time. Elevated prematch tension scores in losers may be attributable to the anticipation of power loss by self or power gain by others due to losing.

In contrast to the findings of Lichtman and Poser

(1983), A and D increased, but not significantly, from pre to postmatch. However, these findings can be attributed to overrepresentation of losers in the study (losers = 15; winners = 11). Indeed, there was a main effect for competitive outcome on mood in the current study, corroborating the idea that mood is influenced by competive outcome. For example, Abadie (1989) found that losing a sport competition resulted in an increase in state anxiety whereas losers exhibited a tendency for elevated values. Also, In a non-athletic competition, winners reported being less anxious, depressed and hostile following the task, and losers exhibited the opposite pattern.

Explanations of the differential influence of competitive outcome on mood were not offered in previous work. Perhaps Kemper's (1991) theory of emotion can be used as a framework for interpreting the emotional outcomes of tennis competition, assuming tennis matches are contests for power and status. Losers reported higher anger scores than winners, possibly due to status loss when another person is seen as responsible (Kemper, 1991). Perhaps prematch anger scores in losers were elevated relative to winners due to the anticipation of status loss, whereas the postmatch discrepancy can be attributed to perceived status loss resulting from the outcome of the competition. Lastly, the elevated depression in losers may be attributable to blaming

oneself or having no control over the outcome of the match (Kemper, 1991).

It should be noted that Kemper's hypotheses were not specifically tested. Stronger support for his theory could have been obtained if attributions of performance outcomes had been ascertained. For example, depression scores would be predicted to increase in losers if they attributed their loss to themselves. However, anger scores would not be expected to increase unless the loss was attributed to another.

In summary, the present study found no significant overall effect for tennis competition on S-IgA levels in saliva. However, the female players displayed an elevated level of S-IgA from pre to postmatch. Further work is needed to determine the neuroendocrine role of immunity as a function of sport competition and gender. Additionally, future studies should examine the clinical significance of altered immunity as a function of competitive outcome and mood.

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APPENDICES

APPENDIX A

ANOVA and MANOVA Summary Tables

Table A-1. ANOVA Summary Table for S-IgA

Source	Degrees of freedom	Sum of squares	Mean square	F value	Significance of F
2 X 2 repeated measures ANOVA--Dependent variable = S-IgA(ug/ml)--males (n=17)					
Outcome	1	17372.72	17372.72	3.01	.10
Time	1	216.56	216.56	.09	.77
Outcome*Time	1	17.99	17.99	.01	.93
2 X 2 repeated measures ANOVA--Dependent variable = S-IgA(ug/ml)--females only (n=4)					
Outcome	1	48.48	48.48	.20	.70
Time	1	2090.85	2090.85	18.17	.05
Outcome*Time	1	283.80	283.80	2.47	.26

Table A-2. MANOVA Summary Table for POMS

2 X 2 repeated measures MANOVA--Dependent variables = TMD, A, D and T--all participants (n=26)

Source	Wilk's Lambda	Degrees of freedom	Exact F value	Significance of F
Outcome	.59	4,21	3.59	.022
Time	.41	4,21	7.62	.001
Outcome*Time	.62	4,21	3.19	.034

Table A-3. Follow-up ANOVA Summary Table for POMS

Source	Degrees of freedom	Sum of squares	Mean square	F value	Significance of F
2 X 2 repeated measures ANOVA--Dependent variable = TMD--all participants (n=26)					
Outcome	1	5968.34	5968.34	12.68	.002
Time	1	723.78	723.78	2.02	.168
Outcome*Time	1	2117.09	2117.09	5.91	.023
2 X 2 repeated measures ANOVA--Dependent variable = Anger (A)--all participants (n=26)					
Outcome	1	236.01	236.01	6.88	.015
Time	1	62.96	62.96	1.98	.173
Outcome*Time	1	97.58	97.58	3.06	.093
2 X 2 repeated measures ANOVA--Dependent variable = Depression (D)--all participants (n=26)					
Outcome	1	150.14	150.14	3.78	.064
Time	1	26.08	26.08	1.42	.245
Outcome*Time	1	249.46	249.46	13.58	.001
2 X 2 repeated measures ANOVA--Dependent variable = Tension (T)--all participants (n=26)					
Outcome	1	291.69	291.69	6.59	.017
Time	1	190.96	190.96	13.55	.001
Outcome*Time	1	.57	.57	.04	.842

APPENDIX B

Table B-4. Correlation matrix--prematch (N=26) for all variables except S-IgA where N=22). *significant $p < .05$

	S-IgA	TMD	A	C	D	F	T	V	CO
S-IgA	-	-.42*	-.36	-.07	.02	-.29	-.33	.10	.19
TMD		-	.59*	.39*	.32	.56*	.64*	-.22	-.22
A			-	.21	.30	.42*	.25	.11	-.29
C				-	.26	.12	.40*	-.06	-.01
D					-	.20	.03	.01	.12
F						-	.28	-.24	-.37
T							-	.02	-.29
V								-	-.19
CO									-

Table B-5. Correlation matrix--postmatch (N=26 for all variables except S-IgA where N=22). *significant $p < .05$

	S-IgA	TMD	A	C	D	F	T	V	CO
S-IgA	-	.04	.09	.30	-.11	.03	.19	.20	.11
TMD		-	.81*	.72*	.88*	.74*	.88*	-.69*	-.54*
A			-	.46*	.81	.34	.77*	-.31*	-.40*
C				-	.52*	.54*	.67*	-.42*	-.37
D					-	.51*	.71*	-.48*	-.52*
F						-	.56*	-.64*	-.46*
T							-	-.44*	-.37
V								-	.41*
CO									-

Table B-6. Prematch S-IgA, TMD, and POMS subscale scores for winners

Subject number	sex	S-IgA (ug/ml)	TMD	A	C	D	F	T	V
1	male	183.40	-6	3	5	2	6	3	23
2	male	17.59	-4	2	1	0	1	7	15
3	male	135.30	1	3	5	4	0	8	19
4	female	79.27	15	3	9	2	0	22	21
7	male	78.21	2	5	0	1	3	12	19
11	male	119.58	13	0	4	8	0	3	20
12	male	--	11	0	1	0	0	9	22
16	female	93.54	-7	0	22	1	0	9	18
19	male	99.93	-11	0	2	0	5	6	24
24	male	8.85	25	3	9	17	10	12	15
25	male	176.66	-2	0	2	6	0	6	13
	N	10	11	11	11	11	11	11	11
	Mean	99.23	3.4	1.7	5.5	3.7	2.6	8.8	19
	SD	58.22	3.4	1.8	6.3	5.1	3.3	5.3	3.5

Table B-7. Prematch S-IgA, TMD, and POMS subscale scores for losers

Subject number	sex	S-IgA (ug/ml)	TMD	A	C	D	F	T	V
5	female	64.13	33	8	8	9	7	12	11
6	male	43.67	34	8	12	9	8	19	22
8	male	44.24	24	1	7	0	11	17	12
9	male	49.66	28	5	10	0	5	18	25
10	male	--	12	5	8	15	4	16	29
13	male	--	9	5	5	1	5	11	17
14	male	78.78	4	1	3	0	2	8	20
15	male	53.22	2	1	4	0	3	12	19
17	male	31.50	-4	0	2	0	2	8	16
18	male	--	-10	0	2	0	2	6	20
20	male	92.80	33	1	10	0	4	27	16
21	male	131.90	-9	2	5	7	0	9	25
22	female	90.71	12	2	3	0	12	15	20
23	male	1.72	24	10	5	0	7	9	24
26	female	58.76	-10	0	1	0	0	14	27
	N	12	15	15	15	15	15	15	15
	Mean	108.28	-2.0	1.2	3.6	.73	6.6	4.7	18.8
	SD	86.29	2.8	3.0	1.8	3.7	3.6	3.6	4.9

Table B-8. Postmatch S-IgA, TMD, and POMS subscale scores for winners

Subject number	sex	S-IgA (ug/ml)	TMD	A	C	D	F	T	V
1	male	205.50	-2	1	6	0	6	3	18
2	male	57.60	4	0	3	1	7	3	10
3	male	284.40	-4	0	6	0	7	8	7
4	female	102.85	18	9	11	6	2	12	22
7	male	117.94	-2	3	2	0	9	6	22
11	male	75.91	-17	0	4	0	0	1	22
12	male	--	-15	0	0	1	9	0	25
16	female	--	0	0	2	0	6	7	15
19	male	12.07	-1	0	2	0	14	1	18
24	male	84.33	-5	0	2	0	5	5	17
25	male	33.88	2	0	2	0	7	6	13
	N	9	11	11	11	11	11	11	11
	Mean	108.28	-2.0	1.2	3.6	.73	6.6	4.7	18.8
	SD	86.29	9.3	2.8	3.0	1.8	3.7	3.6	4.9

Table B-9. Postmatch S-IgA, TMD, and POMS subscale scores for losers

Subject number	sex	S-IgA (ug/ml)	TMD	A	C	D	F	T	V
5	female	128.55	17	4	6	0	19	10	22
6	male	6.06	58	7	8	21	20	12	10
8	male	72.65	57	7	14	5	22	18	9
9	male	53.07	-14	2	1	6	1	4	28
10	male	--	109	36	12	26	20	22	7
13	male	--	45	7	11	12	13	12	10
14	male	112.93	35	5	5	7	17	6	5
15	male	50.01	23	2	7	4	11	9	10
17	male	23.75	16	16	1	5	2	11	19
18	male	6.33	-5	0	2	0	7	1	15
20	male	171.33	50	21	7	15	10	12	15
21	male	67.81	12	0	2	5	11	3	9
22	female	125.29	61	12	5	13	20	19	8
23	male	2.27	1	4	3	6	6	3	21
26	female	113.03	24	1	11	4	16	4	12
	N	13	15	15	15	15	15	15	15
	Mean	71.78	32.6	8.3	6.3	8.6	13.0	9.7	13.3
	SD	54.75	31.7	9.7	4.2	7.4	6.9	6.4	6.5

Table B-10. Prematch means and standard deviations for males

Variables*	Mean	Standard Deviation
IgA (ug/ml)	79.24	55.67
TMD	8.4	14.6
Anger(A)	2.6	2.8
Confusion(C)	4.9	3.4
Depression(D)	3.3	5.2
Fatigue(F)	3.9	3.2
Tension(T)	10.8	5.9
Vigor(V)	19.8	4.4

*The value of N was 21 for all variables except IgA where N was 17.

Table B-11. Postmatch means and standard deviations for males

Variables*	Mean	Standard Deviation
IgA (ug/ml)	79.88	75.77
TMD	16.52	31.8
Anger(A)	5.3	9.0
Confusion(C)	4.8	3.9
Depression(D)	5.4	7.4
Fatigue(F)	9.7	6.2
Tension(T)	7.0	5.8
Vigor(V)	15.6	6.6

*The value of N was 21 for all variables except IgA where N was 18.

Table B-12. Prematch means and standard deviations for females

Variables*	Mean	Standard Deviation
IgA (ug/ml)	77.28	15.53
TMD	8.6	17.6
Anger(A)	2.6	3.3
Confusion(C)	8.6	8.2
Depression(D)	2.4	3.8
Fatigue(F)	3.8	5.5
Tension(T)	14.4	4.8
Vigor(V)	19.4	5.8

*The value of N was 5 for all variables except IgA where N was 4.

Table B-13.Postmatch means and standard deviations for females

Variables*	Mean	Standard Deviation
IgA (ug/ml)	117.43	11.80
TMD	24.0	22.5
Anger(A)	5.2	5.2
Confusion(C)	7.0	3.9
Depression(D)	4.6	5.4
Fatigue(F)	12.6	8.1
Tension(T)	10.4	5.7
Vigor(V)	15.8	6.2

*The value of N was 5 for all variables except IgA where N was 4.

APPENDIX C

Sport Competition, Mood and Immunity

We are recruiting players involved in singles matches to participate in a study. Participation involves completing paper and pencil questionnaires as well as rendering saliva samples. The information gained from te study will remain confidential and will be coded (no names used after the consent form is signed). If you wish to participate, it will take about 18 minutes total, approximately 10 minutes before and 8 minutes after your match. The steps are as follows:

Before your match

1) Give your consent by reading and signing a consent form (approx. 1 minute).

2) Fill out the designated questionnaires (approx. 6 minutes).

3) Render a saliva sample (approx. 3 minutes)

After your match

4) Fill out the designated questionnaires (approx. 5 minutes).

5) Render a saliva sample (approx. 3 minutes).

Sport Competition, Mood and Immunity

Consent Form

This research focuses on the effects of athletic participation on physiological and psychological variables. This study involves filling out two paper and pencil questionnaires, which take a total of 10 minutes to complete. The Profile of Mood States (POMS) and 3 additional questions will comprise the self-report aspect of this study. In addition, two salivas samples will be obtained. All measures (self-report and saliva sample) will be collected before and after the competition and used as an index of stress.

Participation in this study is purely voluntary, and anyone can withdraw at any time without penalty. The information gathered from the questionnaires will remain confidential and will be coded (no names will be used) in order to protect the participant's indentity. Participation in this study involves no risk. It offers the possible long-term benefit of aiding coaches in understanding the psychological states of their athletes; and will also provide data on the relationship between sport competition, stress and health. Should you have any questions, please contact Dr. Baldwin at 974-2531 or Mr. Zac Wilcox at 579-2819.

I volunteer to participate in the research conducted by Dr. Baldwin and Mr. Zac Wilcox. I understand that I may withdraw from this study at any time, and that I have the privilege to ask any questions at any time concerning the procedures. In addition, I am under no obligation to answer any question. I have read the above description and agree to participate in this study.

Participant's signature:_____ Date:_____

Researcher's signature: _____ Date:_____

_____ Date:_____

Sex M_____ F_____ Age_____

1. I perceive this match to be very important as compared to other matches I have played this summer.

strongly disagree	disagree	undecided	agree	strongly agree
1	2	3	4	5

2. I expect this match to enjoyable.

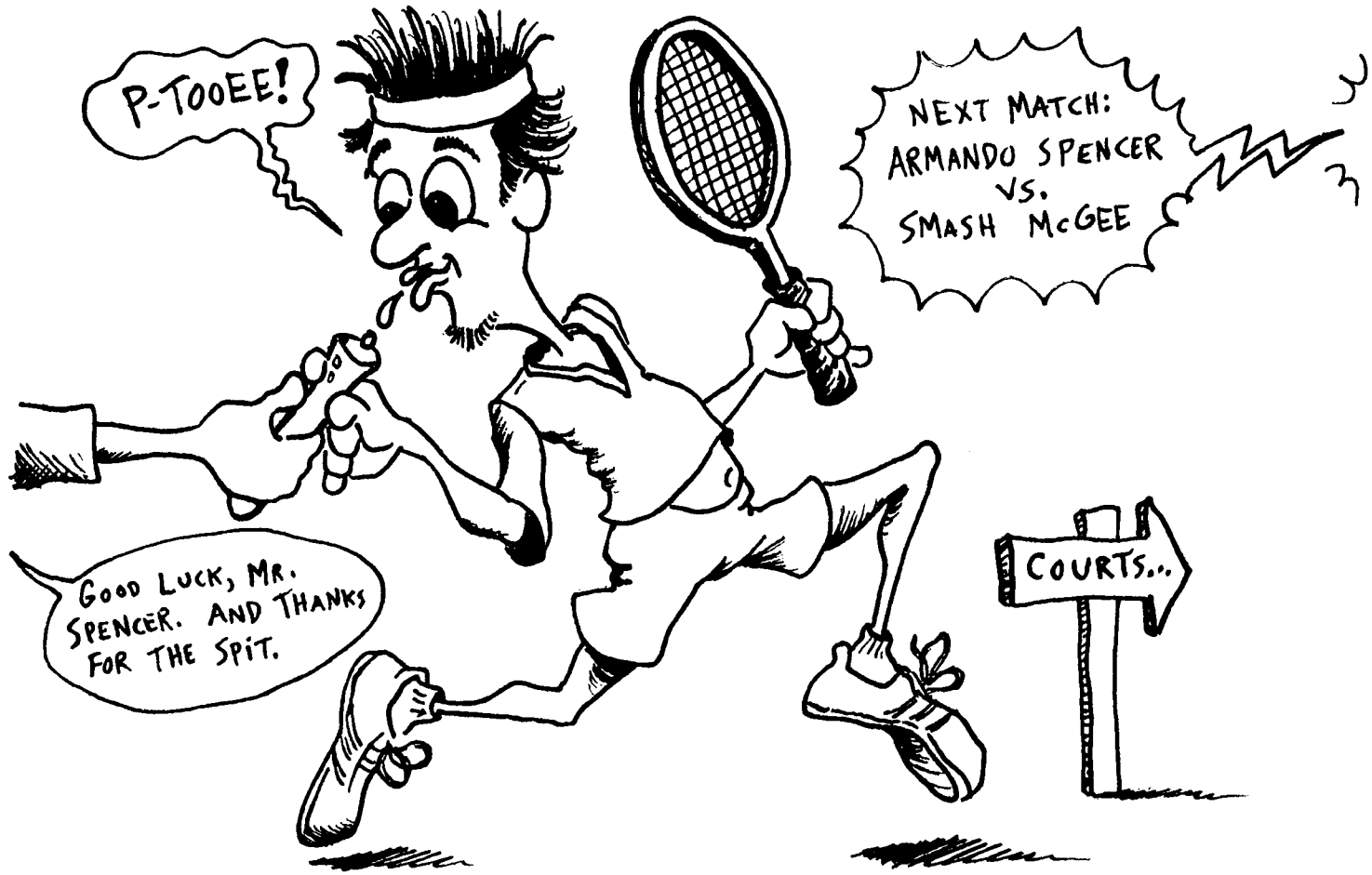
strongly disagree	disagree	undecided	agree	strongly agree
1	2	3	4	5

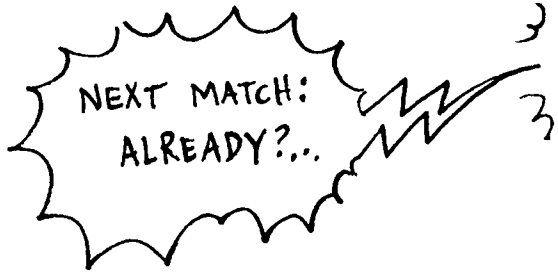
3. I expect to win this match.

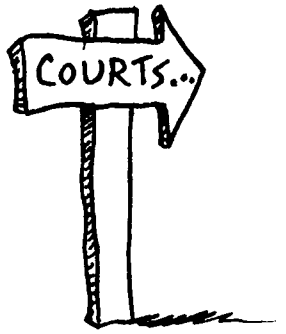
strongly disagree	disagree	undecided	agree	strongly agree
1	2	3	4	5

PROFILE OF MOOD STATES (POMS)

The POMS was used in this study. Permission to reproduce the scale was sought and denied. Copies of the scale can be purchased from EdITS/Educational and Industrial Testing Service, San Diego, California 92167.


P-TOOEE!
NEXT MATCH:
ARMANDO SPENCER
VS.
SMASH McGEE
GOOD LUCK, MR.
SPENCER. AND THANKS
FOR THE SPIT.
COURTS...





DUSTIN E KUNYAN

VITA

Zachary C. Wilcox was born in Killeen, Texas on June 16, 1968. He lived in nearby Belton, Texas through the third grade, where he attended public school. He moved to Tucumcari, New Mexico before the beginning of his fourth grade year. Zachary attended public school in Tucumcari through high school and received his diploma in May, 1987. He entered Eastern New Mexico University in fall, 1987, intending to become an instructor in Special Education. Zachary finally settled upon Psychology as his major during his junior year and graduated with a Bachelor of Science degree in May, 1991. He decided to pursue a Doctor of Philosophy degree in Experimental Psychology at the University of Tennessee and enrolled in fall, 1992. He completed the requirements for his Master of Arts degree in Psychology in August, 1997.

Zachary is currently continuing his doctoral work in Psychology at the University of Tennessee. His doctoral research will investigate the influence of stress and relaxation on salivary IgA and total protein secretion. The long-term aim of the research is to delineate the mechanisms by which stress and relaxation may alter disease risk.