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Stress Resilience in Male and Female Long-Evans Rats"

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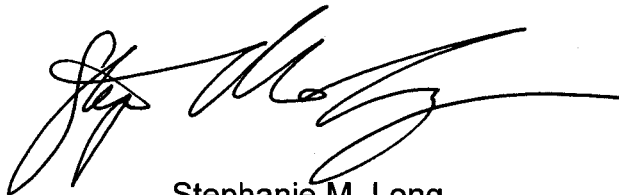
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ABSTRACT

Title of Thesis: Effects of exercise training and social environment on stress resilience in male and female Long-Evans rats

Author: Stephanie Marie Long, Doctor of Philosophy, 2010

Thesis directed by: Neil E. Grunberg, Ph.D., Professor
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The purpose of this doctoral dissertation research project was to determine if exercise training or social enrichment could enhance stress resilience in rats. The specific aims of this experiment were to evaluate: (1) how combined sleep disturbance and predator stress affect biological and psychological components of the stress response; (2) if exercise training attenuates the biological and psychological components of the stress response and promotes recovery following exposure to sleep disturbance and predator stress; (3) if social support attenuates the biological and psychological components of the stress response and promotes recovery following exposure to sleep disturbance and predator stress; and (4) sex differences in the effects of sleep disturbance and predator stress on biological and psychological components of the stress response.

The independent variables were: (1) exercise training (yes, no), (2) social enrichment (pair, individual housing), (3) sex (female, male), and (4) stress period (pre-stress, stress, post-stress). A combined sleep disturbance and predator stressor was administered over a 2 week period. The sleep disturbance stressor was administered daily during the animals' sleep period. The predator stress was administered intermittently during the animals' active period. This

stressor was designed to be analogous to conditions that military personnel experience during combat deployments. Biological and psychological variables were measured before, during, and after the stress period. The biological dependent variables were corticosterone (fecal and serum) and body weight. The psychological dependent variables were open field activity (including center time, a behavioral index of anxiety), ultrasonic vocalizations (a behavioral index of affect), forced swim test (a behavioral index of depression), home cage activity, and food consumption.

Female and male rats responded differently to the combined stressor used in this experiment. Females displayed higher serum corticosterone values, greater anxiety-like behavior, and more ultrasonic vocalizations at approximately 20kHz than did males. Exercise training reduced ultrasonic vocalizations at approximately 20kHz in females. Social enrichment reduced fecal corticosterone levels in males during the post-stress period. Based on these findings, exercise training appears to be important for reducing the stress response in females and social enrichment appears to be important for promoting quicker recovery following stress exposure for males.

Effects of Exercise Training and Social Environment on Stress Resilience
in Male and Female Long-Evans Rats

by

Stephanie M. Long

Doctoral Dissertation submitted to the Faculty
of the Department of Medical & Clinical Psychology
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Introduction

Everyone experiences stress. For some individuals, stress results in mental and physical health problems. Some individuals may struggle with these problems for a prolonged period of time before recovering, while others may never fully recover. In contrast, some individuals exposed to the same stressor may recover fully after an abbreviated period of time, while others may become stronger than they were before the stressor occurred. Those individuals who fully recover are labeled as “resilient” (Luthar, 1991; Luthar & Cicchetti, 2000; Masten, 2001; Rutter, 1993). For those individuals who become stronger post-stress, the process is often labeled as “posttraumatic growth” (Calhoun, Cann, Tedeschi, & McMillan, 2000; Christopher, 2004; Cryder, Kilmer, Tedeschi, & Calhoun, 2006; Ho, Chan, & Ho, 2004; Maercker & Zoellner, 2004; Shakespeare-Finch & Enders, 2008; Tedeschi & Calhoun, 1996, 2004; Zoellner & Maercker, 2006).

Resilience is a characteristic that is often admired and sought after (Bartone, 1998, 2006; Defense Centers of Excellence [DCoE], 2009; Eisold, 2005; Fonagy, Steele, Steele, Higgitt, & Target, 1994; Newman, 2005; Rutter, 2006). It is greatly valued among military personnel because of the stressful nature of military life. The DCoE for Psychological Health & Traumatic Brain Injury (PH/TBI) includes a Resilience and Prevention Directorate with a mission, in part, to “assist the Services and the Department of Defense (DoD) to optimize resilience” (DCoE, 2009). Military personnel are exposed to a variety of stressors, including sleep disturbances, separation from friends and family, threat

of death, and so on (Bartone, 1998, 2006; Bray, Hourani, Rae, Dever, Brown, Vincus, et al., 2006; Dolan & Ender, 2008; Hoge, Castro, Messer, McGurk, Cotting, & Koffman, 2004; Wilcove & Schwerin, 2008). Whereas some individuals are able to effectively cope with these stressors, others are unable to do so. Individuals who cannot cope effectively with the multitude of stressors inherent to military life may develop mental health problems, including anxiety and depression (Tanielian & Jaycox, 2008). For personnel deployed to a war zone, 6-16% meet criteria for generalized anxiety disorder, 12-31% for post-traumatic stress disorder, and 5-38% for depression (Hoge et al., 2004; Lapierre, Schwegler, & LaBauve, 2007; Seal, Bertenthal, Miner, Sen, & Marmar, 2007). Prevalence rates for any mental health disorder after deployment are approximately 30% (Hoge et al., 2004; Seal et al., 2007). These mental health problems can have deleterious effects on the careers and lives of military personnel, as well as on military operations (Tanielian & Jaycox, 2008).

Some individuals, however, do not suffer mental health problems after experiencing stressors that are characteristic of military life. These individuals are able to continue to perform their duties following exposure to a stressor. Continuing to perform, physically and mentally, during and after repeated exposures to stress is critical to military operations (Kennedy & Zillmer, 2006; Steinberg & Kornguth, 2009).

As a topic of study, resilience often inspires excitement and fascination because of its potential implications for clinical treatment of health problems resulting from stress and because of its potential to improve the human condition.

Despite the excitement that surrounds the topic and the flurry of research it has stimulated, the concept of resilience is poorly understood. The etiology of resilience has not yet been determined. As such, it is unclear whether it can be developed or is an inborn characteristic. If resilience can be developed or enhanced, then research has yet to demonstrate which factors may cultivate resilience. Further, it is unclear if there exist individual differences, such as sex, race, ethnicity, age, etc., which also may contribute to resilience.

This doctoral dissertation project used an animal model to examine two factors currently viewed in the human literature as contributing to reducing stress: exercise behavior and social environment. Animal models are a valuable part of the research process and allow experimentation that may not be feasible or ethical to perform in humans. They also allow for basic exploration of topics and subject matter that are not well studied before conducting more targeted or refined research in humans. The present experiment investigated whether exercise training and social enrichment attenuate the deleterious psychological and biological effects of stress and enhance resilience to stressors. This doctoral dissertation research project reviews relevant background to key topic areas: stress, resilience, exercise, and social environment. Next, the procedures used in this research project are presented, followed by the results. Finally, this paper discusses the findings of this research project including its clinical and military implications.

Stress

Stress causes numerous negative mental and physical health consequences. Mental health consequences include anxiety and depression (Bryant et al., 2000; Daley, Hammen, & Rao, 2000; Haller et al., 2003; Hammen, 2005; LeBlanc et al., 2007; Lenze et al., 2008; Mazure et al., 2002; Muscatell et al., 2009; Olf et al., 2007; Orth, Robins, & Meier, 2009; Rygula et al., 2008; Slawewski, 2005). Physical health consequences include hypothalamic-pituitary-adrenal (HPA) axis dysfunction, which may contribute to a variety of other problems (Lynn, Prince, & Phillips, 2009; McEwen, 2000; Sapolsky, Romero, & Munck, 2000; Tsigos & Chrousos, 2002). These health consequences, however, are influenced by a variety of factors. Type of stressor, coping techniques, and individual differences in stress responsiveness (including sex) all play a role in how stress affects the individual.

According to the 2008 American Psychological Association (APA) annual report "Stress in America," 30% of Americans rate their average stress level as extreme (classified as a rating of 8 - 10 on a ten-point scale). Further, 50% of Americans rate their stress as average (classified as a rating of 4 - 7 on a 10-point scale). Only 20% of Americans rate their average stress level as low (classified as a rating of 1 - 3 on a 10-point scale). During periods of high stress, these percentages shift to a greater proportion reporting extreme stress.

Stress is a general term used commonly in the vernacular as well as in the scientific community. Although this word is widely used, it can have several different, but related meanings. Stress has been defined as: an overall state, a

response to a stimulus, a stimulus that causes a response, and as a process.

The most pertinent definition to this doctoral dissertation is the one offered by Baum, Gatchel, and Krantz (1997). These authors defined stress as “the process by which environmental events (stressors) challenge or threaten us, how these threats are interpreted, and how they make us feel” (page 63). Stress is a general term, whereas stressor and stress response are more specific terms.

A stressor is an event or a series of events that provoke a response within an individual, specifically the stress response (Alleva & Santucci, 2001; Dayas, Buller, Crane, Xu, & Day, 2001; Dickerson & Kemeny, 2004; Tsigos & Chrousos, 2002). Stressors may be classified by source (e.g., psychological or physical), type (e.g., eustress or distress), duration (e.g., acute or chronic), frequency (e.g., singular or repeated occurrences), or intensity (e.g., mild, moderate, severe, or extreme). Psychological stressors consist of perceived challenges to well-being, but do not contain physical threat (Alleva & Santucci, 2001; Dayaset al., 2001; Dickerson & Kemeny, 2004; Tsigos & Chrousos, 2002). Physical stressors, in contrast, involve actual threat (Alleva & Santucci, 2001; Dayas et al., 2001; Dickerson & Kemeny, 2004; Tsigos & Chrousos, 2002). A stressor may be interpreted as positive (eustress) or negative (distress) (Goldstein & Kopin, 2007; Selye, 1976; Steel, 2005). An acute stressor has a defined beginning and end, occurs within a proscribed period of time, and does not alter basal physiological functioning (Lynn et al., 2009; Paris et al., 2009). It is essentially a short-term event. An acute stressor may occur once or may be episodic. If an acute stressor occurs repeatedly, without allowing the individual sufficient time to

recover, then it may be considered to be a chronic stressor (Cyr, Earle, Tam, & Romero, 2007; Paris et al., 2009). In contrast, a chronic stressor may or may not have a defined beginning and end, but occurs over a period of time and alters basal physiological functioning (Cyr et al., 2007; Paris et al., 2009). It is essentially a long-term event. The specific parameters of these factors and how they interact within an individual determine the stress response.

The stress response is composed of psychological and biological factors. Our bodies are equipped to deal with acute stressors and a prolonged stress response can have deleterious effects (Buckingham, 2006; Carrasco & Van de Kar, 2003; Dedovic et al., 2009; Dickerson & Kemeny, 2004; Goldstein & Kopin, 2007; Goldstein & McEwen, 2002; McEwen, 1998, 2000; Sapolsky et al., 2000; Selye, 1976; Steel, 2005; Tsigos & Chrousos, 2002). Negative health consequences may occur when the individual is either unable to recover fully before encountering another acute stressor (repeated acute) or experiencing continuous levels of stress (chronic) (Goldstein & Kopin, 2007; Goldstein & McEwen, 2002; McEwen, 1998, 2000; Sapolsky et al., 2000). To understand the various parameters relevant to the concept of stress, a historical understanding is useful.

Historical Context of Stress

The concept of stress has evolved over the past century (Faraday, 2005; Goldstein & Kopin, 2007; Goldstein & McEwen, 2002). Notable figures in the history of stress include Walter B. Cannon, Hans Selye, John W. Mason, David

C. Glass, Jerome E. Singer, Richard Lazarus, Peter Sterling, Joseph Eyer, and Bruce McEwen. These investigators have all contributed greatly to the study and understanding of stress. The focus of stress research has changed from investigating basic mechanisms to include questions of application and prevention. Enhancing stress resilience as a preventive technique is the next step in the field of stress research.

The historical context of stress as a biomedical topic began with Walter B. Cannon in the early 1900's. Cannon expanded Claude Bernard's (1872) concept of the *milieu interieur*, which was that organisms maintain a constant pattern of physiological functioning, and considered stress in terms of a biopsychological model (Goldstein & Kopin, 2007; Goldstein & McEwen, 2002). Cannon (1929; Goldstein & Kopin, 2007; Goldstein & McEwen, 2002) defined stress in terms of homeostasis; stress occurs when a threat (either external or internal) causes the body to move outside of the normal range of functioning. Cannon allowed for these threats to be both physical and psychological, and considered stress to be a process. When the individual's physiological responses move outside the normal functioning range, the body produces a "fight-or-flight" response to permit the individual to deal with the stressor and return to baseline. Some aspects of the "fight-or-flight" response include increases in heart rate, blood pressure, respiration rate, and blood flow to large muscle groups. For Cannon, the stress response could be represented as increased physiological activity during the stressor followed by a return to pre-stress levels. Cannon's conceptualization

appears to hold true for males, but may not be as applicable to females (Taylor, Klein, et al., 2000).

Hans Selye is the next notable figure in the history of stress. Selye introduced the term “stress” into the vernacular. Selye (1946, 1956, 1976) described the stress response as a process, named the general adaptation syndrome (GAS). The GAS is a non-specific stress response that includes three phases: alarm, adaptive-resistance, and exhaustion. The alarm phase is similar to Cannon’s fight-or-flight response. Like Cannon, Selye described the alarm stage as an attempt to immediately deal with an acute stressor. The adaptive-resistance phase is the stage in which the individual attempts to return to normal functioning. The exhaustion phase is also known as burnout, and occurs when the individual no longer has resources to deal with the stressor. The sequelae of chronic stress may be considered a part of Selye’s exhaustion phase. This non-specific stress response, however, does not appear to occur similarly across individuals. Selye allowed for individual differences in stress response, determined by numerous psychological and biological factors (Selye, 1946, 1956, 1976). Among these factors are experiences: experiential processes can either increase or decrease the magnitude of the stress response to a stimulus. Factors such as the environment (e.g., social influences) and learned behaviors (e.g., exercise), may affect the stress response.

John W. Mason proposed a psychobiological approach to the study of stress (Mason, 1968, 1975a, 1975b) by suggesting that psychological stressors provoke the biological stress response, and that this stress response is further

moderated by psychological factors (Faraday, 2000, 2005). These psychological factors may be either environmental (e.g., social) or individual (e.g., exercise training) influences. He also highlighted the importance of individual differences in the stress response. If environmental and individual factors moderate the stress response, then they may alter stress resilience. Mason's work, while revolutionary because of his integration of psychological and biological aspects of the stress response, considered the relationship between psychology and biology to be unidirectional (Mason, 1968, 1975a, 1975b). Specifically, he believed that psychological processes could influence biological processes, but did not believe that biological processes could influence psychological processes. The fact that medications are often used to treat mental illness, such as anxiety and depression (National Institute of Mental Health, 2008), indicates that mental health providers recognize this relationship to be bidirectional.

David C. Glass and Jerome E. Singer (1972) contributed to the stress literature in their classic study of the impact of predictability and perceived control in the stress response, as well as the after-effects of the stress response. Glass and Singer found that predictability and perceived control are important in the stress response: both are associated with decreased stress responsivity. Glass and Singer (1972) also found that a stressor may continue to impact an individual's functioning after termination of the stressor itself. For Glass and Singer, the stress response could be represented as a disruption of physiological activity after cessation of the stressor with a subsequent, gradual return to pre-

stress levels. Glass and Singer did not, however, consider the potential implications of the after-effects of stress in a clinical setting.

Stress management is often employed in a clinical setting to treat a variety of conditions, including anxiety and depression. Stress management techniques are commonly employed after experiencing a stressor to manage the stress response. Prolonged stress responses may occur after experiencing an extreme and/or chronic stressor, which may then result in psychological disorders (i.e., anxiety and/or depression). If stress management techniques are employed before the stressor is experienced and attenuate the magnitude of the stress response, then they may prevent the negative sequelae that can occur after experiencing an extreme stressor. If a stressor has continued effects on the individual even after the stressor has terminated, then it is likely that a stress management intervention also will have continued effects on the individual after the intervention has been terminated. If indeed there are aftereffects of an intervention, then it may be possible to use stress management techniques in a preventive fashion to allow an individual to better handle an extremely stressful situation.

Richard Lazarus and Susan Folkman (1984) built upon Mason's work and highlighted the importance of appraisal in the stress response. These investigators proposed that stress occurs when an individual's expectations and reality do not match. After appraising the event, if it is deemed stressful, then individuals go through the process of reappraisal whereby they assess if they have the resources to deal with the stressor. Reappraisal is influenced by past

experience. Past experience impacts what resources are available and how effective the individual finds those resources to be. Past experience of stress resilience may then decrease future stress responses through reappraisal. Lazarus and Folkman acknowledged the potential impact of psychological processes on biological processes throughout the stress response. Like Mason, Lazarus and Folkman considered this relationship to be unidirectional – that only psychological processes may influence biological processes.

Peter Sterling and Joseph Eyer (1984) introduced the concept of allostasis. Allostasis is the process of maintaining stability through change. Sterling and Eyer described allostasis in terms of biological processes, but the concept may be applied to psychological processes as well. In essence, it is the process of maintaining individual psychological and biological functioning through flexibility in physiological functioning. Allostasis promotes adaptation to stressors. Successful adaptation to stressors is also labeled resilience. Allostasis may be the mechanism by which stress resilience occurs. If certain factors, such as the social environment and exercise, increase allostasis, then they may increase resilience.

Bruce McEwen (1998, 2004) expanded the concept of allostasis to differentiate between allostatic state, allostatic load, and allostatic overload. Allostasis is a process by which stress produces “wear and tear” on the body. An allostatic state occurs when the mediators of allostasis, such as stress hormones, are out of balance (e.g., during an acute stressor). Allostatic states are adaptive in the short-term and allow the individual to return to homeostatic

functioning. Allostatic load is the amount of “wear and tear” generated and, over time, can accumulate and lead to allostatic overload, when the individual is no longer able to effectively adapt to stressors. This process occurs in the context of repeated acute or chronic stress. Allostatic load and overload may prevent stress resilience because the individual may then not have resources to respond effectively to a stressor. Like Sterling and Eyer, McEwen (1998, 2004) focused on the biological processes affecting the stress response. He acknowledged the psychological sequelae of the stress response, but did not consider how psychological processes affect the biological processes.

The concepts of allostasis and allostatic load are rooted in biology, but they also may be conceptualized psychologically. If the individual does not return to baseline psychological functioning, then the resulting wear and tear is likely to lead to psychological distress. This psychological distress may manifest as changes in affect, subclinical levels of anxiety and depression, or even full-blown psychiatric disorders such as major depressive disorder and generalized anxiety disorder. These psychological effects are likely to occur in conjunction with biological effects. It is likely that some factors can prevent allostatic load and, therefore, prevent the negative sequelae of events that occur as a result of allostatic load.

Exercise and the social environment are two factors that have beneficial effects on the stress response and are, therefore, likely candidates to prevent allostatic load (Hamer & Steptoe, 2007; Hamer, Taylor, & Steptoe, 2006; Nausheen et al., 2007; Rejeski, Thompson, Brubaker, & Miller, 1992; Schnohr,

Kristensen, Prescott, & Scharling, 2005; Tsatsoulis & Fountoulakis, 2006).

Exercise is often recommended as a stress management technique and also may train the body to better adapt to stressors. The social environment influences how an individual copes with or manages response to a stressor. The benefits of exercise and social environment on the stress response are widely recognized and are therefore often recommended to increase stress resilience (American Psychological Association [APA], 2010; de Kloet, 2008; DeVries, Glasper, & Detillion, 2003; McEwen, 2007; Southwick, Vythilingam, & Charney, 2005). Research, however, has not yet supported these recommendations. Little literature exists that has manipulated either exercise or the social environment to increase stress resilience. The recommendations, to this point, are pure conjecture. This doctoral dissertation research aims to combat this dearth of experimental research regarding exercise and the social environment as interventions to increase stress resilience.

Biological Effects of Stress

The stress response is a complex cascade of events that occurs after first encountering a stressor (Charney, 2004; Lynn et al., 2009; McEwen, 1998, 2000; Sapolsky et al., 2000; Tsigos & Chrousos, 2002). After a stressor is perceived, the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS) are activated. The hypothalamus releases corticotropin-releasing hormone (CRH, also known as corticotropin-releasing factor), which causes the pituitary gland to release adrenocorticotrophic hormone (ACTH, also known as

corticotropin). ACTH in turn causes the adrenal glands to release both cortisol and dehydroepiandrosterone (DHEA). Cortisol mobilizes glucose from energy stores; increases arousal, vigilance, and attention; enhances memory formation; and inhibits immune system functioning (Charney, 2004; Lynn et al., 2009; McEwen, 1998, 2000; Sapolsky et al., 2000; Tsigos & Chrousos, 2002). This cascade of events is controlled by a negative feedback system (Goldstein & McEwen, 2002).

Cortisol in particular is an important biochemical in the stress response. It is the end biochemical responsible for many of the physiological effects of stress. As such, it is an ideal biological marker of the stress response. In humans, cortisol can be measured in numerous ways, including in the urine, saliva, and blood (Daughters et al., 2009; Gozansky et al., 2005; Kim et al., 2008; McRae et al., 2006; Paramastri et al., 2007; Paris et al., 2009; Stroud, Salovey, & Epel, 2002; van Stegeren, Wolf, & Kindt, 2008). Urinary cortisol is a non-invasive measure and is not immediately sensitive to acute stress. Saliva cortisol is a non-invasive measure and is immediately sensitive to acute stress. Blood cortisol is invasive and the measurement itself often produces a stress response. Blood cortisol also provides the total amount of cortisol, including both bound and free-circulating cortisol.

The biochemical most similar to cortisol in rats is corticosterone. Several methods for corticosterone measurement exist. Most commonly, blood is drawn to ascertain corticosterone levels in a rat. Blood may be collected by nicking the tail vein, inserting a catheter into a vein, cardiac puncture, or collecting trunk

blood after euthanasia (Bhatnagar et al., 2006; Consoli et al., 2005; Dalla et al., 2005; Konkle et al., 2003; Panagiotaropoulos et al., 2004; Retanta-Marquez et al., 2003). Rats, because of their much smaller size, have less blood than do humans, which limits the amount of and frequency with which samples may be collected. Blood can often be collected only once, and typically at the end of the study. In addition, in stress research, blood collection from rats is a problem because the procedure itself is invasive and often acts as a stressor. Measuring corticosterone through blood in animals is, therefore, limited because of the stressful nature of collection, and the limited number of times blood can be collected. Alternative methods, such as urinary and fecal corticosterone measurements, are not limited in the same way as blood measurements.

Urine samples may be collected to evaluate corticosterone levels in rats (Eriksson, Royo, Carlsson, & Hau, 2004; Gomez-Sanchez & Gomez-Sanchez, 1991; Krohn, Hansen, & Dragsted, 2003; Paramastri et al., 2007). However, urinary corticosterone does not reflect acute stress responses and is more appropriately an index of stress responses over time. Urinary corticosterone measurement requires housing the rats in a metabolic cage, which is essentially a small metal cage with a wire grid floor. This type of cage is more stressful than housing a rat in a standard polycarbonate cage with bedding (Gomez-Sanchez & Gomez-Sanchez, 1991; Krohn et al., 2003; Paramastri et al., 2007). Use of a metabolic cage, therefore, may confound results in stress research. Fecal samples, however, can be collected in a standard cage without the same limitation.

Corticosterone metabolites are found in the feces. Cavigelli and colleagues (2006) reported that levels of fecal corticosterone metabolites are reflective of circulating corticosterone levels. Fecal corticosterone, therefore, can be used as a non-invasive, non-stressful technique to collect repeated measures of corticosterone levels in the rat.

Sympathetic nervous system (SNS) activation occurs in conjunction with HPA axis activation. SNS activation leads to a variety of outcomes, including: increased heart rate, increased blood pressure, increased respiration rate, increased blood flow to large muscle groups and the brain, increased glucose release, and pupil dilation (Baum et al., 1997; Guyton & Hall, 2000; Kolb & Whishaw, 2009). SNS activation also decreases blood flow to the digestive tract and to the reproductive organs. All of these outcomes prepare an individual to deal more effectively with a stressor.

Regardless of whether the stressor is biological, psychological, or social, the same cascade of events occurs. The purpose of the stress response is to enable the individual to deal effectively with the stressor (Carrasco & Van de Kar, 2003; Cyr et al., 2007; McEwen, 1998, 2000; Sapolsky et al., 2000; Tsigos & Chrousos, 2002). Once the stressor or threat is resolved, the individual should resume normal functioning (i.e., return to baseline). The duration and intensity of the stressor influences whether the individual is able to return to baseline and, if so, how quickly the individual regains normal functioning (McEwen, 1998, 2004). Chronic stress can result in prolonged return to baseline, which may have deleterious effects.

Psychological Effects of Stress

Stress has numerous psychological effects. These effects vary in type, duration, frequency, and intensity. These effects depend on the stressor, the individual, and the environment. Arguably, the individual and the environment are more important in the stress response than is the stressor *per se*. The individual's past experience and homeostatic range influence the stress response, as does the individual's environment. Based on these factors, some individuals are stress resilient, whereas other individuals are not. Those individuals who are not stress resilient suffer multiple negative psychological consequences that affect quality of life and possibly their careers. (Resilience is discussed in detail below.)

Most Americans recognize that stress contributes to illness, including depression and insomnia. Further, Americans list irritability and anger; fatigue; anxiety; sadness; and lack of interest, motivation, and energy as common psychological symptoms of stress. Psychological effects of stress also include behavioral attempts to cope with a stressor. Sometimes these coping attempts are beneficial to health, but often they are detrimental. For example, 47% of Americans report using exercise and 41% report engaging social support from friends or family as stress management techniques (APA, 2008). It is not clear whether these health-promoting behaviors reduce stress, enhance resilience, or simply co-vary with stress. In addition, some of the behaviors that accompany stress can pose health risks. For example, 34% of people under stress report increased food consumption, 18% drink alcohol, and 16% smoke tobacco

cigarettes. These behaviors have detrimental health effects and may exacerbate the negative effects of stress.

In addition to the co-morbidity of stress and health-related behaviors, stress may affect psychological disorders. For example, acute and chronic stressors may both precipitate the occurrence of a major depressive episode (Daley et al., 2000; Lenze et al., 2008; Mazure et al., 2002; Muscatell et al., 2009; Orth et al., 2009; Rygula et al., 2008). Acute and chronic stressors also may precipitate the occurrence of an anxiety disorder (Bryant et al., 2000; Haller et al., 2003; LeBlanc et al., 2007; Olf et al., 2007; Slawewski, 2005), including post-traumatic stress disorder.

Two of the most important negative psychological consequences of stress relevant to the present research are anxiety and depression. Anxiety and depression are common psychological problems with great potential negative implications for quality of life. Anxiety disorders have a lifetime prevalence rate of 28.8% and depressive disorders have a lifetime prevalence rate of 20.8% (Kessler et al., 2005; Kessler & Wang, 2008). Another potential negative psychological consequence of stress is a change in affect. Affect, the behavioral display of emotion, changes in response to stressors and provides a measure of well-being that may capture sub-clinical symptoms (Bolger, DeLongis, Kessler, & Schilling, 1989; Folkman & Moskowitz, 2000; McKinzie, Altamura, Burgoon, & Bishop, 2006). This change may take the form of decreases in positive affect and/or increases in negative affect. In addition to being clinical indicators of

stress in their own right, positive and negative affect are symptoms of anxiety and depressive disorders.

Anxiety, depression, and affect can all be studied in a laboratory setting using an animal model. Anxiety can be studied using the open field chamber, elevated plus maze, acoustic startle reflex, and light-dark box (Dalla et al., 2005; Marin, Cruz, & Planeta, 2007; Mineur, Belzung, & Crusio, 2006; Padilla et al., 2009; Pohl et al., 2007; Slawecki, 2005; Strekalova et al., 2005). Depression can be studied using immobility in the forced swim test, sucrose preference, and dexamethasone suppression test (Baker et al., 2006; Consoli et al., 2005; Dalla et al., 2005; Konkle et al., 2003; Mineur et al., 2006; Pohl et al., 2007; Strekalova et al., 2005). Positive and negative affect can be studied using ultrasonic vocalizations (Brudzynski, Ociepa, & Bihari, 1991; Burgdorf & Panksepp, 2006; Panksepp, 2007; Panksepp & Burgdorf, 2000, 2003; Rosa et al., 2005). All of these behavioral indices have been validated throughout numerous experiments and allow for examination of these constructs in a controlled laboratory environment. Of these possible measures, the ones used in the present research included center time in an open field chamber (anxiety), immobility in the forced swim test (depression), and ultrasonic vocalizations (positive and negative affect). The present research examined all of these variables as stress responses in conjunction with physiological measures of stress.

Sex Differences in Stress Response

Research has shown inconsistent sex differences in the stress response. In terms of the biological stress response, some researchers have reported that females have higher basal levels of physiological functioning than males, whereas others have demonstrated the opposite (Baker et al., 2006; Consoli et al., 2005; Dalla et al., 2005; Paris et al., 2010). Still others have found no sex differences in stress response at all (Konkle et al., 2003; Padilla et al., 2009; van Stegeren et al., 2008). Further, researchers have reported conflicting findings with regard to sex differences in the magnitude of the physiological stress response. It appears that these differences may depend on the type of stress and how it is measured (Stroud et al., 2002; Wang et al., 2007).

Taylor, Klein, and colleagues (2000) proposed that females have a different dominant stress response than males which they have termed “tend and befriend.” These investigators proposed that the female stress response is based in the attachment-caregiving system and is modulated primarily through oxytocin and endogenous opioid peptides. These researchers argued that it is in the best interest of the species for females to care for their offspring and affiliate with others during a stressor because these behaviors increase the likelihood of survival of the species.

Sex differences in the stress response have implications for interventions to enhance stress resilience. It is possible that, because of these sex differences, interventions will be differentially effective in females and males. Based on the “tend and befriend” stress response, social interventions may be

more effective in females than males. In addition to implications for the effectiveness of different stress interventions, sex differences in stress response have implications for stress resilience. No one, however, has examined whether resilience to stressors differs between females and males. The present research compares the stress responses of females and males in an aim to provide further insight into stress resilience.

Laboratory Models of Stress

Stress is often studied in a laboratory setting (Akinboboye, Krantz, Kop, Schwarz, Levine, et al., 2005; Cohen, Liberzon, & Richter-Levin, 2008; Faraday, Blakeman, & Grunberg, 2005; Faraday, O'Donoghue, & Grunberg, 2003; Heinrichs & Koob, 2006; Kop, Gottdiener, Patterson, & Krantz, 2000; Korte & De Boer, 2003; Korte, De Boer, & Bohus, 1999; Krantz, Quigley, & O'Callahan, 2001; Rabat, 2007; Zakowski, Cohen, Hall, Wollman, & Baum, 1994).

Laboratory stressors provide a standardized stressor that may be reproduced with multiple individuals which may or may not be possible with naturalistic stressors. Responses to laboratory stressors are often easier to study than responses to naturalistic stressors because they afford experimental control. Experimental control permits researchers to design an experiment to answer a specific question with regard to a specific topic (e.g., stress). Further, manipulation of independent variables is possible in laboratory studies, such that individuals are exposed to the same type of stressor of a similar duration and frequency. This manipulation of independent variables allows for determination

of causality. Further, laboratory stressors allow for easier replication of experiments. Several laboratory models of stress exist, some designed for use with humans and some with animals.

Human models of stress include both psychosocial and physiological tasks (Akinboboye et al., 2005; Kajantie & Phillips, 2006; Kop et al., 2000; Krantz et al., 2001; Zakowski et al., 1994). Examples of psychosocial tasks include public speaking and performing mental calculations under time pressure (i.e., the Trier Social Stress Test). Examples of physiological tasks include the cold-pressor test, exercise testing, insulin tolerance tests, and the dexamethasone test. Although human laboratory models of stress are often used and provide valuable information about an individual's stress response, they can be difficult to conduct. Strict ethical requirements, recruiting and scheduling subjects, and a plethora of psychosocial influences can make data collection and interpretation of findings exceedingly difficult. Fortunately, several laboratory stressors have been designed for use with animals to avoid some of these difficulties.

Multiple animal models of stress exist, including: inescapable shock, social defeat, swim stress, restraint stress, predator stress, and sleep disruption stress (Baran et al., 2008; Berger, 2009; Cohen et al., 2008; Cui et al., 2008; Faraday et al., 2003, 2005; Heinrichs & Koob, 2006; Kikuchi et al., 2008; Kinn et al., 2008; Korte & De Boer, 2003; Korte et al., 1999; Perry, 2009; Rabat, 2007; Rabat et al., 2004, 2005, 2006; Valentine et al., 2008; Zoladz et al., 2008). Many are behavioral, although some pharmacological models exist. Sleep disturbance and predator stress are two behavioral models that are of particular interest

because of their relevance to military and clinical psychology and have been used successfully in the Grunberg laboratory (Berger, 2009; Perry, 2009; see Table A below).

Military Stressor	Laboratory Stressor	Symptoms/Behaviors
Separation from family	Individual housing	↑ anxiety, ↑ depression, ↑ fear, ↑ startle reflex, ↑ ACTH, ↑ corticosterone
Not enough sleep	Sleep disturbance	↑ anxiety, ↑ depression, ↓ cognition
Life-threatening situation (being attacked or ambushed; receiving small arms fire; and receiving incoming artillery, rocket, or mortar fire)	Predator stress	↑ anxiety, ↑ depression, ↑ fear, ↑ startle reflex, ↓ body weight, ↓ cognition, ↑ corticosterone

Table A. Deployment stressors and their laboratory correlates. Information compiled from: Berger (2009), Bray et al. (2006), Brenes and Fornaguera (2008), Cohen et al. (2008), Hoge et al. (2004), Perry (2009), Weiss et al. (2004), Zoladz et al. (2008).

Sleep disturbance stress is a face valid model of chronic stress. Sleep disturbance is often associated with psychological disorders and this relationship appears to be bidirectional (Gupta, Dahiya, & Bhatia, 2009; Lemke, Puhl, & Broderick, 1999; Lewis, Creamer, & Failla, 2009; Meerlo et al., 2008; Perry, 2009; Uhde, Cortese, & Vedeniapin, 2009). Military life also is associated with sleep disturbance, both in peace and war. Peacetime operations often require shiftwork, which disrupts circadian rhythms and leads to sleep disturbances.

Wartime operations lead to sleep disturbances for a variety of reasons, including nighttime operations, hyperarousal, and uncomfortable sleeping conditions.

Predator stress is a face valid model of traumatic stress (Apfelbach et al., 2005; Berger, 2009; Cohen et al., 2008; Perry, 2009; Takahashi et al., 2005; Zoladz et al., 2008). Predator stress mimics life-threatening situations that many military personnel experience in a deployed setting. These life-threatening situations often occur in combination with chronic sleep disturbance stress.

Sleep disturbance stress alone has significant effects on the stress response of rats (Rabat, 2007; Rabat et al., 2004, 2005, 2006). Predator stress also has significant effects on the stress response of rats (Apfelbach et al., 2005; Berger, 2009; Cohen et al., 2008; Perry, 2009; Takahashi et al., 2005; Zoldaz et al., 2008). Recent work in our laboratory has demonstrated that combined sleep disruption and predator (fox urine) stress has significant effects on the stress response of rats (Perry, 2009). A stressor that combines sleep disturbance stress and predator stress is a face valid representation of stressors that military personnel experience while deployed. It is of critical importance to examine interventions that reduce an individual's response to combined sleep disturbance and predator stress because of the implications, particularly for military populations. This doctoral dissertation project examined the effects of exercise training and the social environment on resilience to combined sleep disturbance and predator stress in an effort to determine if these interventions will increase resilience to this military-appropriate stressor.

Stress Management

The Yerkes-Dodson principle (1908) states that moderate levels of arousal result in optimum performance. Both too little arousal and too much arousal can have detrimental effects on performance. Most individuals, however, struggle with levels of stress (which affect arousal) that are too high rather than too low. Stress management techniques may reduce their stress to moderate levels, thereby resulting in optimum performance.

Stress management techniques may be categorized as behavioral, cognitive, and pharmacological. Behavioral techniques include diaphragmatic breathing, muscle relaxation, biofeedback, exercise, and music therapy (Lehrer, Woolfolk, & Sime, 2007; Robins, McCain, Gray, Elswick, Walter, & McDade, 2006; Suinn, 2005). Cognitive techniques may be further divided into problem-focused or emotion-focused techniques (Bond & Bunce, 2000; Collins, Baum, & Singer, 1983; Lazarus & Folkman, 1984; Nyklicek, Poot, & van Opstal, 2010). Problem-focused techniques include removing, managing, or altering the stressor (Bond & Bunce, 2000; D'Zurilla & Goldfried, 1971; Hill, Hall, & Appleton, 2009). Emotion-focused techniques include avoiding, reappraising, or minimizing the stressor (Bond & Bunce, 2000; Collins et al., 1983; Huth, Broome, & Good, 2004; Lazarus & Folkman, 1984). Pharmacological techniques include benzodiazepines, antidepressants, neuroleptics, beta-adrenergic receptor antagonists, anticonvulsants, buspirone, antihistamines, and d-cycloserine (Muriel, Hwang, Kornblith, Greer, Greenberg, Temel, et al., 2009; Papp, 2007).

Techniques from these different categories can be used alone or in combination to reduce stress.

Despite the large numbers of available stress management techniques, the appropriate technique will depend on the individual and the situation (Simpson-McKenzie, 2008). For example, cognitive techniques may be difficult for some individuals (e.g., individuals with neurological problems or deficits, mentally retardation, or thought disorders) and pharmacological techniques have numerous undesirable side effects (e.g., dietary restrictions, weight gain, sexual dysfunction). Behavioral techniques do not have the same restrictions and may, therefore, have the broadest range of applicability with minimal risk. The situation and the specific stressor, however, may limit the behavioral techniques that may be applied. Military service members, particularly those who are deployed, may not be able to employ many of the common techniques recommended to civilians (i.e., listening to music, diaphragmatic breathing, progressive muscle relaxation) depending on the situation in which they are engaged.

The stress response prepares individuals to deal effectively with acute, physical stressors. While deployed, military personnel are likely to encounter acute physical stressors, including being ambushed, engaging in hand-to-hand combat, and participating in demining operations (Bartone, 2006; Hoge et al., 2004; Kolkow, Spira, Morse, & Grieger, 2007). Military personnel must be able to continue to function while experiencing these extreme stressors and their stress responses, provided that they are not too extreme, will allow them to continue

functioning. In these situations, it is beneficial for individuals to maintain their stress responses until the stressor has ended. An intervention that prevents an individual from an extreme stress response resulting in dysfunction, therefore, would be ideal. One possibility is to enhance stress resilience through exercise.

Exercise is one stress management technique with great potential to enhance stress resilience (Blumenthal et al., 2005; Bruning & Frew, 1987; Byrne & Byrne, 1993; Gauvin & Spence, 1995; Kerr & Kuk, 2001; McCain, Gray, Elswick, Robins, Tuck, Walter, et al., 2008; Milani & Lavie, 2009; Salmon, 2001). Exercise has numerous advantages over other stress management techniques for a military population. It is a required aspect of military life and fits within the military culture (Taylor, Markham, Reis, Padilla, Potterat, Potterat, Drummond, et al., 2008). Exercise is encouraged and is non-stigmatizing. Exercise allows the individual to withstand more physical stress and it also may allow the individual to withstand more psychological stress (Taylor et al., 2008). Individuals who engage in exercise behavior more often may be better prepared to perform effectively in high-stress situations. Acevedo and colleagues (2006) reported that individuals who have high fitness levels have attenuated cardiovascular responses while experiencing both mental and physical stress. In addition, Traustadóttir, Bosch, and Matt (2004) reported that individuals who have high fitness levels have attenuated HPA axis responses compared with individuals who have low fitness levels.

Other techniques (e.g., diaphragmatic breathing, meditation, progressive muscle relaxation) may help reduce the stress response in the moment, which

may or may not be beneficial. It is unclear, however, if these other techniques have lasting effects on the stress response, including cardiovascular system and HPA axis functioning. In contrast, exercise appears to have lasting beneficial effects on the stress response. It may attenuate negative consequences of extreme stress exposure and enhance resilience. Exercise, because of its military relevance and its beneficial effects on the stress response, is an ideal intervention for stress resilience. The present research, therefore, examined exercise training as one intervention to enhance stress resilience.

Resilience

Resilience is commonly defined as an ability to "bounce back" after experiencing an extreme stressor (Luthar, 1991; Luthar & Cicchetti, 2000; Masten, 2001; Rutter, 1993). In accordance with this definition, resilience can be defined as a return to pre-stress baseline levels of psychological and biological functioning. Others, however, have defined resilience as an improvement upon previous levels of psychological functioning after experiencing an extreme stressor (Flach, 2004; Lyons & Parker, 2007; Polk, 1997). This phenomenon also has been labeled "thriving" and "post-traumatic growth." Another definition of resilience is maintaining a stable equilibrium with no significant disruption in functioning (Bonanno, 2004; Mancini & Bonanno, 2006). Bonanno (2004; Mancini & Bonanno, 2006) has highlighted the distinction between child and adult resilience, and has focused on adult resilience. This work has been based on studies of grief. Bonanno distinguishes between resilience and recovery, with a

definition of recovery that is strikingly similar to Luthar and Cicchetti's (2000) definition of resilience. In fact, Bonanno's definition of resilience is similar to Kobasa and Maddi's concept of hardiness (Kobasa, 1979; Kobasa et al., 1982a, 1982b, 1985). Bonanno's definition fails to consider adults who experience repeated traumatic events and, therefore, may not be applicable to military members who have served in combat.

Many researchers use the terms resilience, hardiness, stress buffering, and stress resistance interchangeably. Hardiness was originally conceived as a personality construct, based on a study of business executives (Kobasa, 1979). The first studies of hardiness were designed to investigate whether or not hardiness altered the stress-illness relationship. Kobasa and colleagues (1982a) suggested that hardy individuals may be more likely to engage in health-protective behaviors, such as exercise. Kobasa and colleagues (1982b; 1985), however, found that hardiness and exercise are discrete moderators in the stress-illness relationship and each buffers the stress-illness relationship independently of the other. Although Kobabsa and colleagues did not find a relationship between hardiness and exercise, it is unclear if resilience is related to exercise. More recently, hardiness has been described as a "pathway" to resilience, indicating that they are related, but distinct constructs (Maddi, 2005). Some mental health professionals now offer training programs to enhance hardiness which suggests that hardiness either is not a personality construct or that the term is being used to describe stress resistance and responses, rather than to describe an innate trait.

Stress buffering derives from research on social support (Cohen & Wills, 1985). A stress buffer is a moderator and reduces the magnitude of the relationship between a stressor and the stress response, such that the stressor does not produce as great a stress response when a stress buffer is present as when a stress buffer is not present. Stress resistance is a mediator and alters the impact of the stressor on the stress response. The constructs of resilience, hardiness, stress buffering, and stress resistance are related, but appear to be distinct from one another. Resilience, hardiness, and stress resistance are most commonly considered as individual factors, whereas stress buffering may be best characterized as an external or environmental factor. Hardiness, resilience, and stress resistance all overlap and are, at the same time, influenced by stress buffers such as social support. (see Figure A below).

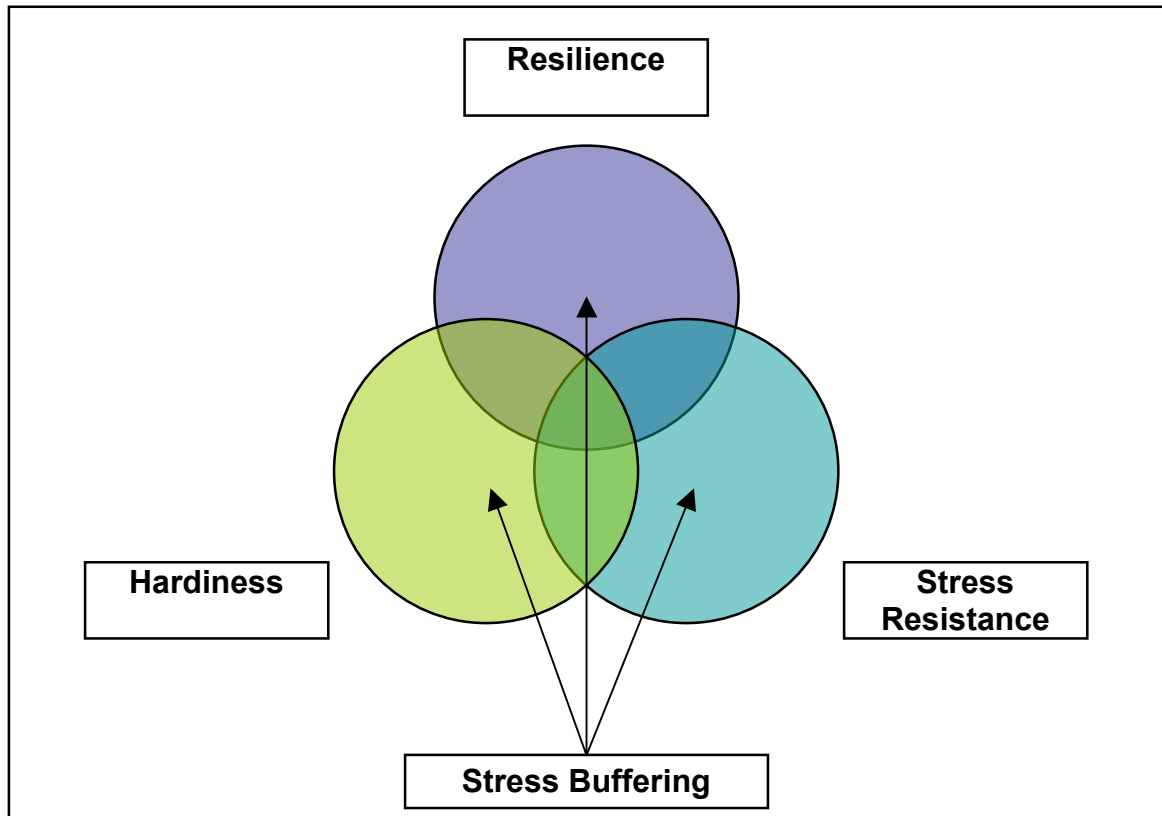


Figure A. The inter-related constructs of resilience, hardiness, stress resistance, and stress buffering.

There exists a divide in the field as to whether resilience is a personality construct, similar to hardiness, or is a process. Resilience must exist before the challenge occurs, but manifests subsequent to the challenge. It appears that the outcome of resilience is an interaction between trait and state.

In addition to the lack of consensus for a single definition of resilience and the confusing use of multiple terms (i.e., hardiness, stress buffering, and stress resistance) when discussing resilience, the construct of resilience itself is poorly understood. Based on the literature, several models of resilience exist.

One model of resilience involves the time course of recovery from a stressful experience. Morgan and colleagues (2000) reported that more resilient individuals display similar physiological responses as less resilient individuals in reaction to an acute stressor, but have a quicker return to baseline physiological functioning. If their findings hold true, then it is possible that stress resilience is associated with a shorter time course of the physiological reaction to stress. Alternatively, it is also possible that stress resilience is associated with decreased magnitude in physiological reaction to stressors.

Another model of resilience involves the trajectory of functioning after a stressful experience. Similar to the phenomenon commonly referred to as post-traumatic growth, thriving, and/or flourishing, some researchers consider resilience to be an improvement on pre-stressor functioning. An individual who displays resilience, therefore, is at an improved level of functioning after a stressful experience compared with before the stressful experience (Flach, 2004; Lyons & Parker, 2007; Polk, 1997).

The concept of allostasis provides a valuable framework for understanding stress resilience. Allostasis is the process of maintaining homeostasis. When an individual encounters a stressor, the individual mobilizes psychological and biological resources to deal with the stressor. After the stressor has been effectively dealt with, the individual returns to baseline functioning. This return to functioning, or allostasis, also can be conceptualized as stress resilience. McEwen (2000, 2007) described four conditions that may

lead to allostatic load: repeated acute stressors, lack of adaptation to a stressor, prolonged stress response, and inadequate stress response.

It is unclear if parameters of the stress response, including baseline, magnitude of the response, reset period, and the differences between responses to acute and chronic stressors, differ in resilient and non-resilient individuals. It is also unclear if resilience can be manipulated or even enhanced by other factors, specifically exercise and the social environment.

Resilience is clearly relevant to both military and clinical psychology. Resilient individuals are able to function normally after experiencing a stressor and perform their duties, whereas individuals who are not resilient may develop psychological disorders that prevent them from performing their duties. In 2005, approximately 13% of military personnel screened positive for generalized anxiety disorder and 22% for depression (Bray et al., 2006). In addition, approximately 8% reported experiencing serious psychological distress and 18% of military personnel reported a perceived need for mental health counseling. In troops returning from deployment in Iraq, approximately 30% screened positive for depression, generalized anxiety disorder, or posttraumatic stress disorder (Hoge et al., 2004). Preventing and/or limiting the negative effects of psychological distress could have great benefits for all individuals. If resilience can be better understood and enhanced, then fewer individuals would suffer from psychological distress. Enhancing resilience is essential for military members most likely to encounter extreme stressors and those limited in stress management techniques. The present research used exercise training and the

social environment as interventions to increase stress resilience because of their beneficial effects on the stress response.

Exercise

Exercise has physical and mental health benefits. Physical health benefits include: reduced risk of chronic disease (i.e., coronary heart disease, stroke diabetes mellitus, and colon cancer), decreased total blood cholesterol and triglycerides, increased high-density lipoproteins, decreased hypertension, and enhanced bone, muscle, and joint health (Centers for Disease Control and Prevention, 2008a). Mental health benefits include: improved mood, increased positive and reduced negative affect; reduced anxiety and depression; increased self-esteem; improved memory, concentration, attention, and information processing; and decreased hallucinations (Blumenthal et al., 2005; Bruning & Frew, 1987; Byrne & Byrne, 1993; Gauvin & Spence, 1995; Hays, 1999; Kerr & Kuk, 2001; McCain et al., 2008; Milani & Lavie, 2009; Salmon, 2001; Sandlund & Norlander, 2000). Exercise's beneficial effects on some of these variables may result from exercise's ability to decrease stress.

Despite the health benefits of physical activity and exercise, more than 50% of American adults do not engage in recommended levels of physical activity (CDC, 2008b). Further, approximately 25% of the American population is sedentary. Even within the military, only approximately 60% of service members engage in recommended levels of physical activity (Bray et al., 2006). Physical activity is necessary to health and lack of physical activity leads to a variety of

chronic health conditions, including cardiovascular disease, obesity, diabetes, and metabolic syndrome.

Physical activity is defined as any bodily movement produced by skeletal muscle which results in energy expenditure (American College of Sports Medicine [ACSM], 2000; CDC, 2008a). Exercise is a subset of physical activity. Exercise is defined as physical activity that is both planned and structured with the intention to improve or maintain physical fitness (ACSM, 2000; CDC, 2008a). Exercise may be aerobic or anaerobic, and involve muscular strength and/or endurance. Aerobic exercise involves the supply and use of oxygen to maintain physical activity (ACSM, 2000; CDC, 2008a). Aerobic exercise improves cardiovascular fitness and requires the cardiovascular and pulmonary systems to work harder in order to supply oxygen to the muscles. Common examples of aerobic exercise include cycling, running, swimming, and walking. In contrast, anaerobic exercise does not rely on a continuous supply of oxygen to maintain physical activity. Common examples of anaerobic exercise include weight lifting and sprinting. Muscular strength is the ability of a muscle to exert force during an activity, whereas muscular endurance is the ability of a muscle to maintain performance without fatigue (ACSM, 2000; CDC, 2008a).

Regular exercise decreases the cardiovascular response to acute psychological stress. In addition, acute exercise promotes cardiovascular recovery from acute psychological stress (Chafin, Christenfield, & Gerin, 2008). A quicker return to normal functioning, compared with an average recovery time, results in decreased allostatic load and decreased negative health effects. If

exercise can promote quicker recovery times in terms of cardiovascular function, then it is likely that exercise can promote quicker recovery times in terms of other variables. These other variables may include: increased positive affect, decreased negative affect, decreased anxiety, and decreased depression. In addition, if acute exercise has beneficial effects on recovery from a stressor, then regular exercise is likely to have a more profound effect. Exercise training may, therefore, promote stress resilience by decreasing the magnitude of the stress response and by decreasing recovery time and therefore added strain on the individual.

Exercise training is promoted as a healthy lifestyle behavior and encouraged and valued within the military. It is a valuable stress management technique that does not provoke stigma, a huge barrier to mental health care in the military. In addition to exercise training, environmental variables may be used to enhance resilience and decrease the stress response in individuals. This doctoral dissertation research project used both exercise and the social environment to increase stress resilience.

Environment

The environment greatly influences individual psychological and biological functioning. The social environment impacts how individuals respond to and cope with stress. Nearly two-thirds of Americans use social support to cope with stress (APA, 2008). Three-fourths of military service members use social support to cope with stress (Bray et al., 2006). Women in the military are more

likely than men to use social support as a coping mechanism (87% versus 71.8%). Altering the environment, therefore, may have a substantial effect on individual functioning.

Environmental manipulation is easily accomplished in a laboratory setting. One type of environmental manipulation in the laboratory setting is environmental enrichment. Mark Rosenzweig (1966) introduced the paradigm of environmental enrichment with laboratory rats and found that enrichment causes structural brain changes.

The classic enrichment paradigm involves two forms of enrichment: physical and social. Physical enrichment involves adding objects to the home cage to allow for tactile stimulation and physical activity (Rosenzweig et al., 1972; Woodcock & Richardson, 2000a, 2000b). Social enrichment involves housing animals in groups of two or more to allow for social interaction. Standard housing conditions in rats involve limiting one animal to a cage without toys or other objects (Varty et al., 2000). The present research used social enrichment as an environmental manipulation to increase stress resilience.

The environment has psychological and biological effects. Biological effects include increased neurogenesis (Diamond et al., 1972; Fernandez-Teruel et al., 2002; Johansson, 2003; Kleim et al., 2003; Kramer et al., 2002; Mohammed et al., 2002; Rosenzweig et al., 1972; Sutoo & Akiyama, 2003; Van de Weerd et al., 2002) and wound healing (Detillion et al., 2004; Glasper & DeVries, 2005). Psychological effects include improved attention and performance on learning and memory tasks, and decreased anxiety, food

consumption, and body weight (Benarova-Milshtein et al., 2004; Chapillon et al., 1999; Daniel et al., 1999; Elliott & Grunberg, 2005; Friske & Gammie, 2005; Pham et al., 1999; Pietropaolo et al., 2004; Schrijver et al., 2002; Tomchesson, 2004, 2006; Williams et al., 2001; Zimmermann et al., 2001).

The social environment alters the stress response of individuals (Brown & Grunberg, 1995; Detillion et al., 2004; DeVries et al., 2003, 2007; Ditzen et al., 2007; Glasper & DeVries, 2005; Heinrichs et al., 2003; Hennessy, Kaiser, & Sachser, 2009; Plante et al., 2007). The social environment may either exacerbate or attenuate the stress response. Social defeat and having a lower status in the social hierarchy may exacerbate the stress response (McCormick et al., 2009; Strekalova et al., 2005). For males, it appears that too much social enrichment (e.g., too many cage mates) may be stressful (Brown & Grunberg, 1995). The social environment, however, also may attenuate the stress response.

The social environment can be divided into several levels. At the most basic level is the mere presence of others. Group housing includes two or more individuals in a housing situation, and may attenuate the stress response. For example, pair housing alone can facilitate wound healing in laboratory animals (Detillion et al., 2004; Glasper & DeVries, 2005). At a more complex level is social support.

Social support can attenuate the stress response. The stress-buffering hypothesis states that social support benefits health by protecting individuals from the negative effects of stressors (Cohen & Wills, 1985). Social support

attenuates the stress response to acute stressors (Thorsteinsson & James, 1999). It appears that central oxytocin is the mechanism by which social support buffers the stress response (DeVries et al., 2003). Oxytocin promotes affiliative behaviors and decreases HPA axis functioning. Conversely, social isolation reduces behavioral indices of depression and increases biological responses to an acute stressor (Grippe, Cushing, & Carter, 2007). It appears that the effect of social support on the stress response, however, may be sex-specific.

Kirschbaum and colleagues (1995) reported that men exposed to an acute psychosocial laboratory stressor had attenuated stress responses (as measured by salivary cortisol) when supported by a significant other, whereas women exposed to the same stressor had increased stress responses when supported by a significant other.

The social environment clearly can have a positive impact on the stress response. It is unclear, however, if social support is the mechanism by which the social environment alters the stress response. It is possible that social facilitation and the mere presence of others are responsible for attenuating the stress response (Zajonc, 1965; Zajonc & Sales, 1966). It is clear, however, that a socially-enriched environment has numerous benefits for mental and physical health. In addition, the social environment is of particular relevance to the military.

Military personnel are often required to work in teams. The entire military structure, regardless of branch of service, is based on groups of individuals who work together to accomplish specific tasks or missions. Much research in the

military has been devoted to group dynamics: group cohesion, leadership, etc. (Bartone, Johnsen, Eid, Laberg, & Brun, 2002; Ng, Ang, & Chan, 2008; Rona, Hooper, Jones, Iversen, Hull, Murphy, et al., 2009; Thunholm, 2009; Zohar & Tenne-Gazit, 2008). Another reason why the social environment is of particular relevance to the military is that military personnel are often exposed to changing social environments, both at work and at home. For those living in on-base housing and during deployment, personnel may be housed with others or individually. During deployments, personnel also are separated from their friends and family back home. During permanent changes of station, personnel are exposed to a novel social environment. Military personnel are exposed to multiple social environments, but it is yet unclear which environment may be the most valuable.

Humans are social animals, and social affiliation is natural and beneficial to survival (Schachter, 1959; Taylore et al., 2000). Altering the social environment to promote social affiliation may have beneficial effects. In the military in particular, which may be limited in the number of stress management techniques available, altering the social environment may be advantageous for personnel health and military readiness. Comparing different social environment conditions may provide insight into which conditions may be most beneficial to mental health. This doctoral dissertation research manipulated the social environment to examine its effects on stress resilience.

Summary

Stress is an essential part of life. Stress in moderation is helpful, but in excess can be harmful if it exceeds an individual's capacity to handle the experience. Military service members especially are struggling to cope with the stressors inherent to repeated combat deployments, including separation from family and friends, environmental stressors (i.e., noise, heat), sleep difficulties, repeated life threatening situations (i.e., mortar attacks, improvised explosive device blasts, fire fights). These stressors may lead to a variety of negative sequelae, problems in an individual's biological and psychological functioning. Exercise and the social environment have positive effects on the stress response and, therefore, have great potential for increasing stress resilience. The purpose of this doctoral dissertation research project was to use a rodent laboratory model to determine if exercise and the social environment could be manipulated to increase stress resilience.

Value of Animal Models

Animal models in research have tremendous value. In general, they allow increased experimental control which may permit researchers to determine causality. In addition, animal models permit researchers to conduct experiments that would not be logistically possible or ethical in human research. Animal models, however, also lack face validity and do not include unique aspects of the human experience. They are, therefore, one step in the research process albeit an important one. Animal research provides valuable information that later can be used to improve the human condition.

An animal model was particularly valuable for this doctoral dissertation research project for several reasons, including manipulation of the independent variables and measurement of the dependent variables. The present research utilized a combined sleep disturbance and predator stress paradigm.

Manipulating a chronic stressor in humans would be unethical, particularly one that combined a stressor with known mental and physical health consequences (i.e., sleep disturbance) with a stressor that involved repeated exposure to a potentially traumatic event (i.e., predator stress). In addition, in humans, manipulating sleep disturbance as a stressor is difficult. Manipulating sleep disturbance in an animal model is more feasible than doing so in humans.

Another advantage of using an animal model in this research project concerns the experimental manipulation of exercise training. Behavioral compliance with a prescribed regimen is difficult to achieve in human studies, especially when the prescribed regimen includes exercise. Assigning subjects to

exercise training is more easily done in animals than in humans. Another advantage concerns the experimental manipulation of the social environment. It would be difficult in humans to keep some subjects housed in isolation and others in group housing. In animals, manipulation of housing condition is a well-accepted and valuable aspect of experimental design.

Utilization of an animal model in this research also had benefits in terms of the dependent variables. In humans, requiring repeated measurements is taxing for the subjects and often results in missing data. In an animal study, the subjects are always available. Further, the data collection procedure can be carefully controlled, including time and environmental conditions (e.g., temperature, humidity, exposure to sounds, and other environmental exposures), which may influence the results. Time is a particularly important factor for biological stress measures because of diurnal variation. In the present research, for example, all biological stress measures were collected at approximately the same time of day.

Animal research is clearly an important part in the research process. Depending on the research area and the specific questions the researcher chooses to address, it may be more appropriate to select an animal model than a human one. In the present research, an animal model was more appropriate because of the manipulation of independent variables and the measurement of the dependent variables.

Overview of the Research Experiment

The present research examined the effects of exercise training and social environment on biological and psychological responses to sleep disturbance and predator stress in young adult, male and female Long-Evans rats. The specific biological variables included: fecal corticosterone, serum corticosterone, and body weight. The specific psychological variables included: a behavioral index of anxiety (time spent in the center of an open-field chamber), a behavioral index of depression (immobilization when forced to swim in an inescapable cylinder of water), behavioral indices of positive and negative affect (ultrasonic vocalizations), home cage activity, and food consumption.

Specific Aims

The specific aims of the proposed research were to:

- (1) Determine how combined sleep disturbance and predator stress affect biological and psychological components of the stress response;
- (2) Determine if exercise training attenuates the stress response and enhances resilience to the effects of sleep disturbance stress on biological and psychological components of the stress response;
- (3) Determine if social support attenuates the stress response and enhances resilience to the effects of sleep disturbance stress on biological and psychological components of the stress response;
- (4) Evaluate sex differences in the effects of sleep disturbance stress on the biological and psychological components of the stress response.

Independent Variables

The independent variables included stress phase, exercise training, social environment, and sex. The subjects were young adult male and female Long-Evans rats, all exposed to combined sleep disturbance and predator stress. Long-Evans rats were appropriate subjects for this study because of the sensitivity of their stress responses. Long-Evans rats are stress-sensitive in comparison with Sprague Dawley rats (Faraday, 2000; Padilla et al., 2009). A stress-sensitive rat is appropriate for this research project because so little research has been conducted on interventions to increase stress resilience in an animal model. Any intervention to increase stress resilience was likely to be more effective with stress-sensitive subjects than stress-resistant subjects. Because there is a dearth of animal and intervention studies in the resilience literature, it was beneficial to use subjects with which an animal intervention study is likely to be most effective. Long-Evans rats, therefore, were used as subjects because of their heightened stress responsiveness.

The stressor used in this experiment was a complex stressor designed to model sleep disturbance and possibility of attack experienced by military service members during deployments. The stressor included: (1) sleep disruption and fragmentation induced by noise in periodic intervals throughout the inactive 12-hour period of each day for 14 days, and (2) intermittent exposure to fox urine paired with various environmental mild stressors (e.g., noise, flashing lights, cage shaking) during the stress period. Details of these stressors are presented in the Methods section. Combined sleep disturbance and predator stress was a within-

subject variable and was presented in an ABA design: pre-stress, stress, post-stress. An ABA design is valuable to study stress resilience. Resilience is a return to baseline levels of functioning and, as such, requires an understanding of pre-stress, stress, and post-stress functioning. Sleep disturbance is relevant to many individuals, particularly deployed service members (Hancock, 2009; McLay & Spira, 2009; Morin & Hu, 2007). Further, sleep difficulties are related to anxiety, depression, reduced job performance, and impaired cognition (Giam, 1997; Krakow et al., 2002; Luine et al., 2007; Meltzer & Moore, 2008; Scott & LaDou, 1990). Predator stress also is relevant to deployed service members. Predator stress is a face-valid model of the stress which many deployed military personnel experience (Apfelbach et al., 2005; Cohen et al., 2006; Takahashi et al., 2005; Zoldaz et al 2008). Military personnel in deployed settings often experience both chronic sleep disturbance and episodic stressful events. These experiences may lead to decreased job performance, interpersonal relationship problems, and psychological disorders (Bray et al., 2006; Taylor et al., 2008). Combined sleep disturbance and predator stress was, therefore, a face valid model of military conditions. Sex, exercise training, and social environment were all between-subjects variables.

Sex is a variable relevant to military operations and to stress because females and males are deployed to war zones. In addition, 35.5% of females report high levels of stress related to being a woman in the military and females report higher levels of mental health problems than do men (Bray et al., 2006). Females and males also exhibit different stress responses (Luine et al., 2007;

Taylor, Klein, et al., 2000). Sex was, therefore, an important variable to include in this experiment.

Exercise training is relevant to mental health and the military. Exercise prevents multiple chronic diseases, including depression and emotional distress (Kruk, 2007). In addition, exercise is an effective, non-stigmatizing stress management technique (Hays, 1999; Lehrer et al., 2007) because it is not perceived as a mental health treatment. Individuals who engage in regular exercise and, therefore, have high fitness levels have decreased cardiovascular and HPA axis responses to stress (Acevedo et al., 2006; Traustadóttir et al., 2004). Exercise also decreases the impact of psychological stressors (Callaghan, 2004; Dubbert, 2002; Phillips, Kiernan, & King, 2001; Rahe, 1988; Taylor et al., 2008; Warburton, Nicol, & Bredin, 2006). Exercise training has numerous benefits and is, therefore, a valid stress intervention that may increase resilience.

Manipulating the social environment provides a face valid model of overall human and specific military conditions. Social support is an effective stress management technique, and the majority of Americans and service members seek social support to manage their stress (APA, 2008; Bray et al., 2006). In addition, interpersonal stressors influence the stress response of males and females, but appear to have a greater impact on females (Vogt et al., 2005). Further, approximately 15% of service members separate from their significant other during or after deployment (Bray et al., 2006). Social environments, therefore, may either reduce or increase stress responses. Specifically, the

presence of another may lead to decreased stress responses, whereas isolation may lead to increased stress responses. Manipulating the social environment is a valid intervention that may increase resilience to chronic stressors.

Dependent Variables

The dependent variables included biological and psychological measures of the stress response. The biological measures included fecal corticosterone, serum corticosterone, and body weight. The psychological measures included open field activity, ultrasonic vocalizations, immobilization during the forced swim test, home cage activity, and food consumption.

Corticosterone is a biochemical released throughout the stress response.

Fecal corticosterone is reflective of circulating blood corticosterone levels and provides a non-invasive technique to repeatedly measure corticosterone levels within a subject (Cavigelli et al., 2006; Kim et al., 2008; Paramastri et al., 2007).

Serum corticosterone provides a biological measure of the stress response and predator stress reliably produces increases in serum corticosterone levels (Apfelbach et al., 2005; Blanchard et al., 1998; Day et al., 2004; Dias Soares et al., 2003; Figueiredo et al., 2003; Zoladz et al., 2008). Berger (2009) reported robust effects of predator stress on serum corticosterone levels in female and male Sprague Dawley rats. Perry (2009) reported robust effects of sleep disruption stress, predator stress, and combined sleep disruption and predator stress to increase serum corticosterone levels in female and male Long-Evans and Sprague Dawley rats.

Open field activity provides a measure of **center time activity**, an index of anxiety (Faraday, 2000; Hlavacova & Jezova, 2008; Pohorecky, 2008). This paradigm is based on the rat's natural tendency to remain on the perimeter of a novel environment because the walls offer some degree of protection from an external threat. As the rat moves into the center of the open field, it is hypothesized that it is less anxious because of the lack of protection from an external threat. Center time activity in the open field chamber is commonly used to determine if a biochemical or drug is anxiolytic or anxiogenic (Calabrese, 2008; Cohen et al., 2009; Frye et al., 2008; Lin et al., 2009; Liu et al., 2009). Open field activity also records **horizontal activity**, a measure of general movement. Horizontal activity is important to include whenever dependent variables depend on movement (e.g., swim test, center time activity) to be able to determine if any changes in these activity-dependent measures reflect the presumed psychological construct that is operationalized (e.g., swim test as index of depression) or, instead, are the result of changes in general movement. In addition, horizontal activity provides a general measure of health. When animals are ill, they move less overall (Bauhofer et al., 2002; Engeland, Kavaliers, & Ossenkopp, 2006; Hart, 1988, 1991; Swain et al., 1998).

Ultrasonic vocalizations provide an index of positive and negative affect. Rats emit ultrasonic vocalizations at different frequencies in response to different stimuli. Stimuli such as maternal grooming and play cause rats to emit ultrasonic vocalizations at approximately 50 kHz (Burgdorf & Panksepp, 2006; Panksepp, 2007; Panksepp & Burgdorf, 2000, 2003). Aversive stimuli such as foot shock

and novelty cause rats to emit ultrasonic vocalizations at approximately 22 kHz (Brudzynski et al., 1991; Rosa et al., 2005). Ultrasonic vocalizations around 50kHz are considered to be a measure of positive affect, whereas ultrasonic vocalizations around 22kHz are considered to be a measure of negative affect.

Forced swim immobilization provides an index of depression (Dalla et al., 2005; Dayas et al., 2001; Mineur et al., 2006; Strekalova et al., 2005). Porsolt and colleagues' (1977) forced swim test is based on the learned helplessness paradigm developed by Seligman (1974; Seligman, Maier, & Geer, 1968). The rats are placed into an inescapable cylinder filled with water for a short period of time. The proportion of time spent immobile to time spent mobile is considered a measure of learned helplessness and, therefore, an index of depression.

Home cage activity is a measure of general movement. In this experiment, because numerous dependent measures are movement-based (i.e., center time, forced swim test), a general measure of movement is necessary to control for differences in movement. Past research in the Grunberg laboratory (Simpson-McKenzie, 2008; Tomchesson, 2006) has found that home cage activity provides a general measure of activity different from that found in novel environments. Novelty may alter activity levels. Home cage activity provides a measure of general activity that is not confounded by novelty.

Body weight and **food consumption** are outcomes related to the stress response. Specifically, stress appears to decrease body weight, particularly in males (Bhatnagar et al., 2006; Bowman et al., 2009; Duncko et al., 2001; Ely et

al., 1997; Faraday, 2000; Kim et al., 2008; Panagiotanopoulos et al., 2004).

Stress appears to have variable effects on food consumption. The majority of studies indicate that stress has no effect on food consumption (Ely et al., 1997; Gamaro et al., 2003; Pettenuzzo et al., 2008), some studies indicate that stress decreases food consumption (Badiani et al., 1996; Pecoraro et al., 2004; Rybkin et al., 1997), and some studies indicate that stress increases food consumption (Levine & Morley, 1981, 1982; Morley, Levine, & Rowland, 1983; Youngblood, Ryan, & Harris, 1997).

Experiment

This experiment was designed to evaluate effects of stress, exercise training, and social environment on biological and psychological stress responses of male and female Long-Evans rats. Biological responses were measured using fecal samples and blood serum. Psychological responses were measured using predominately behavioral techniques. All measures were piloted to insure that they worked and to increase the investigator's experience with each measure. This experiment lasted eight weeks. The experimental protocol was approved by the USUHS Institutional Animal Care and Use Committee (IACUC) and was conducted in full compliance with the National Institutes of Health Guide for Care and Use of Laboratory Animals (National Institutes of Health [NIH], 1996).

Hypotheses

1. Combined sleep disturbance and predator stress will have significant effects on biological and psychological measures in both female and male rats.

(a) Combined sleep disturbance and predator stress will decrease center time in the open field chambers, body weight, and food consumption.

(b) Combined sleep disturbance and predator stress will increase fecal corticosterone levels, ultrasonic vocalizations at lower frequencies, and forced swim immobility. This hypothesis is based on Perry (2009) who reported that combined sleep disturbance and predator stress significantly affected the stress response, including serum corticosterone levels, open field activity, and forced swim immobility. Although Perry (2009) did not measure fecal corticosterone, it was anticipated that fecal corticosterone levels would increase in a similar fashion to serum corticosterone levels based on Cavigelli and colleagues (2005) who reported that fecal corticosterone levels reflected serum corticosterone levels.

(c) No *a priori* hypothesis is offered for the effects of combined sleep disturbance and predator stress on home cage activity. No literature was found to suggest how combined sleep disturbance and predator stress will affect this dependent variable.

2. Exercise training will attenuate stress' effects on biological and psychological measures. This hypothesis is based on research demonstrating that exercise is an effective stress management technique in humans (Blumenthal et al., 2005;

Bruning & Frew, 1987; Byrne & Byrne, 1993; Gauvin & Spence, 1995; Kerr & Kuk, 2001; McCain et al., 2008; Milani & Lavie, 2009; Salmon, 2001; Sandlund & Norlander, 2000).

3. Social housing will attenuate stress' effects on biological and psychological measures. This hypothesis is based on research demonstrating that social housing decreases the stress response in rodents (Brown & Grunberg, 1995; Detillion et al., 2004; DeVries et al., 2003, 2007; Ditzen et al., 2007; Glasper & DeVries, 2005; Heinrichs et al., 2003; Hennessy et al., 2009; Plante et al., 2007; Thorsteinsson & James, 1999) and in humans (Kulik, Mahler, & Moore, 1996; Lepore, 1992).

4. Stress responses to sleep disturbance and predator stress will differ between females and males. This hypothesis is based on research that reported females and male rats differ in their stress responses (Bhatnagar et al., 2006; Bowman et al., 2009; Duncko et al., 2001; Ely et al., 1997; Faraday, 2000; Kim et al., 2008; Panagiotanopoulos et al., 2004; Perry, 2009).

(a) No *a priori* hypothesis is offered for sex differences in the effects of exercise training on the stress response. No literature was found to suggest that exercise training may have differential effects on stress responsiveness and reactivity in females and males.

(b) Social housing will attenuate stress' effects on behavioral and biological measures more in males than in females. This hypothesis is based on

research that reported social housing was more effective in reducing stress responses in males than in females (Beck & Luine, 2002; Kirschbaum et al., 1995; Westenbroek, Snijders, den Boer, Gerrits, Fokkema, & Ter Horst, 2005).

Methods

Experimental Design

The present research examined the effects of exercise training and social environment on psychological and biological responses to sleep disturbance and predator stress in female and male Long-Evans rats. This experiment was a 3 x 2 x 2 x 2 mixed design ([within-subject factor: pre-stress, during stress, post-stress] x [between-subjects factor: female, male] x [between-subjects factor: exercise training, no exercise training] x [between-subjects factor: pair housing, individual housing]) with eight subjects assigned to each cell. The within-subject variable of stress phase was used in this experiment because resilience is a process that occurs within an individual after encountering a stressor. A pre-stress baseline was required in order to determine if the individual returned to baseline post-stress. The stressor was required because resilience occurs only after experiencing a stressor. All three aspects of this variable, pre-stress, stress, and post-stress phases, were therefore required. The between-subjects variables were required to determine if: (1) exercise training as an intervention could increase stress resilience, (2) social environment as an intervention could increase stress resilience, and (3) sex differences in stress resilience and in response to the two interventions exist.

A sample size of eight subjects per cell was selected to allow for detection of main effects and interactions within this experiment. The sample size was determined based on two factors: past research and a power analysis. This sample size is based on past experiments within the Grunberg laboratory that

have demonstrated that a sample size of eight animals per cell provides sufficient power to detect interactions in behavioral variables (e.g., Berger, 2009; Brown & Grunberg, 1995; Faraday, 2000; Grunberg & Bowen, 1985; Grunberg, Bowen, & Morse, 1984; Perry 2009; Winders & Grunberg, 1990). Power analysis software (GPower, version 3.0.3) was used to determine sample size for this experiment (Faul et al., 2007). Preliminary data collected were used to compute a sample size of seven subjects per cell to achieve a power level of 0.8. The sample size was increased by one subject per cell (for a total of eight subjects per cell and 64 subjects overall) in case of disease or death. The dependent variables included biological and behavioral measures (biological: fecal corticosterone, serum corticosterone, and body weight; behavioral: open field activity [center time and horizontal activity], ultrasonic vocalizations, forced swim immobility, home cage activity, and food consumption). All of these variables are relevant to the effects of stress.

All animals were exposed to behavioral testing before, during, and after a 14 day stress period. The 14 day stress period consisted of chronic sleep disturbance throughout the sleep period and repeated acute predator stress during the waking period. The duration of the entire experiment was eight weeks. Several dependent variables were measured repeatedly throughout the course of the experiment (e.g., open field activity, body weight, food consumption) to determine any effects of stress over time. (See Appendix A for experimental timeline.)

Subjects

Subjects were 32 female and 32 male Long Evans rats (Charles River Laboratories). They arrived at approximately 50 days of age, which roughly corresponds to young adulthood in the developmental trajectory of rats. Adolescence in rats is typically described as lasting from 28-42 days of age, but occasionally extends up to 55 days in males because they develop at a slower rate than do females (Spear, 2000; Spear & Brake, 1983). Subjects were immediately placed into experimental housing conditions upon arrival.

Housing

Upon arrival, subjects were randomly assigned to one of four treatment conditions: (1) no exercise training and individually housed; (2) exercise training and individually housed; (3) no exercise training and pair housed; or (4) exercise training and pair housed. All subjects resided in the same housing room and were housed in standard polycarbonate rat cages (40 x 20 x 20 cm). The polycarbonate cages and bedding were changed weekly by animal husbandry staff.

A hardwood chip bedding (Pine-Dri) was used in all cages. All subjects had continuous access to food (Harlan Teklad 4% Mouse/Rat Diet 7001) and water. The housing room was maintained at approximately 23°C and approximately 50% relative humidity on a 12-hour reversed light/dark cycle (lights off at 0600 hours). Rats are nocturnal animals, and reversing the light cycle permitted experimenters to conduct behavioral measures during the animals'

active phase. An overhead red light provided dim illumination for personnel working in the housing room. This light was used when experimenters, veterinary technicians, and animal husbandry staff were in the housing room during the dark cycle. These housing conditions were designed to provide optimal levels of comfort to the rats within their home cages.

Procedures

See Appendix A for Experimental Timeline. On the first day of the experiment, subjects were assigned to one of the four conditions. On each of the subsequent three days, subjects were briefly gentled (by handling about 3 minutes each) to attenuate or prevent stress responses due to handling. Handling was required to measure body weight and place animals into the behavioral equipment. Further, three days provided adequate time for the subjects' circadian rhythms to adapt to their new light cycle based on previous research in our laboratory. The exercise training began one week into the experiment and continued for the duration of the experiment. The baseline phase lasted for two weeks, after which the stress phase began and also lasted for two weeks. The post stress phase lasted for four weeks.

Fecal corticosterone was measured once during the pre-stress phase, once during the stress phase, and three times during the post-stress phase for a total of five times throughout the experiment. Open field activity was measured once during the pre-stress phase, once during the stress phase, and three times during the post-stress phase for a total of five times throughout the experiment.

Ultrasonic vocalizations were measured once during the pre-stress phase, once during the stress phase, and three times during the post-stress phase for a total of five times throughout the experiment. Forced swim immobility was measured once during the pre-stress phase, once during the stress phase, and three times during the post-stress phase for a total of five times throughout the experiment. Home cage activity was measured once during the pre-stress phase, once during the stress phase, and three times during the post-stress phase for a total of five times throughout the experiment. Body weight and food consumption were measured throughout the experiment. (Please see timeline in Appendix A for exact days when measures were taken.)

Sleep Disturbance

Sleep disturbance was manipulated for 14 consecutive days from experiment day 15 through 28 (Perry, 2009; Rabat et al., 2004, 2005, 2006). Sleep disturbance was manipulated in the housing room by exposing all rats to environmental sounds during the light-phase or sleep period. Compact discs (CDs) were recorded with numerous sounds of various duration, frequency, and quality. The shortest sound played for 6 seconds, and the longest sound played for 70 seconds. The shortest period of silence was 2 minutes and the longest period of silence was approximately 17 minutes. Sound duration and frequency were altered after seven days to adjust for habituation. A clock/radio/CD player (Sony Dream Machine, Model # ICF-CD843V) was used to produce the environmental noises causing sleep disturbance. The CD player was connected

to a timer that was programmed to play intermittently throughout the animals' 12-hour light (sleep) period. Ambient sound levels in the housing room were approximately 59 decibels (db). Recorded sounds ranged from 65 db to 80 db. Total hourly sound exposure did not exceed 10 minutes at any time during the experiment.

Predator Stress

Predator stress was manipulated three times per week during the sleep disturbance stress phase on experiment days 16, 18, 21, 23, 25, and 28, for a total of six exposures (Berger, 2009; Perry, 2009). Predator stress was manipulated between 1200 and 1400 hours. Rats were transported from the housing room to a nearby procedure room with negative pressure so that the predator scent did not contaminate the animal facility. The investigators turned on the overhead fluorescent lights upon entering the procedure room and the bright light remained on throughout the procedure. Animals were placed into clear Plexiglas standard mouse size cages (27 cm x 15 cm x 13 cm) without bedding. The smaller cage size and lack of bedding both increased the stressful nature of the environment. Jumbo-sized cotton balls were soaked with 15mL of synthetic fox urine (Buck Stop, Stanton, MI) and placed into these cages. The placement of the cotton balls changed during each exposure to prevent habituation. The predator stress was paired with aversive, unpredictable stimuli (e.g., cage-shaking, loud noises, flashing light) to also prevent habituation and to increase the unpredictability of the stressor. On experiment day 16, fox urine

was presented alone without an additional stimulus. On experiment day 18, fox urine was paired with flashing lights six times presented 5 minutes into the stress procedure. On experiment day 21, fox urine was paired with a loud alarm presented 3 minutes into the stress procedure. On experiment day 23, fox urine was paired with cage shaking presented 3 and 7 minutes into the stress procedure. On experiment day 25, fox urine was paired with a loud whistle presented 1 and 8 minutes into the stress procedure. On experiment day 28, fox urine was paired with shaking coins in a metal container presented 90 seconds and 7 minutes into the stress procedure. The stress procedure occurred over a 10-minute period and was performed during the rats' active phase. The order in which rats were exposed to predator stress was counterbalanced across the different treatment conditions. The cages were cleaned with a 35% isopropyl alcohol solution between animals.

Exercise Training (Ex)

Activity in exercise wheels was recorded five times a week for 60 minutes between 0700 and 1200 hours beginning in the second week and continuing through the end of the experiment. The equipment is in a dedicated procedure room that is separate from, but nearby, the housing and open field activity rooms. The temperature, humidity, and lighting conditions were the same as those conditions in the open field activity room. The eight activity wheels (35.6 cm diameter) consist of stainless steel grid rods (4.8 mm diameter) spaced 1.6 cm apart (Med Associates, Inc., St. Albans, VT) connected to separate plastic cages

(48.26 cm L x 26.67 cm W x 20.32 cm D) with stainless steel wire covers. Each cage has a 7.2 cm W x 10.2 cm H opening that allows voluntary access to the running wheel or cage. This opening can be closed to ensure that the animal remains in the activity wheel. Each activity wheel has 12 grams of drag. Rats were placed singly into the exercise wheels and remained in the exercise wheel portion of the equipment. Revolutions of each activity wheel are recorded automatically on a dedicated computer that is interfaced with the activity wheels during a 60-minute period. The data (number of quarter revolutions of the activity wheel) were electronically recorded in 60 1-minute bins. After 60 minutes of data collection, animals were returned to their home cages and the exercise wheels were cleaned using a 35% isopropyl alcohol solution. Cleaning the exercise wheels with this solution prevents transmission of disease, prevents odors from animals previously tested in the chamber influencing activity of subsequent animals, and helps maintain the equipment.

Fecal Corticosterone

Fecal corticosterone was measured once during the pre-stress phase, once during the stress phase, and three times during the post-stress phase on experiment days 6, 20, 34, 41, and 48, for a total of five times throughout the experiment. Fecal samples were collected after open field activity measurements. All samples were collected in the same time period (between 0700 and 1300 hours) with animals counterbalanced by treatment group to minimize differences between the groups due to diurnal variations in

corticosterone metabolites found in the feces. Fecal samples were collected from the open field chamber and placed into Ziploc freezer storage bags. The storage bags were labeled with the animal number and date of collection. Fecal samples were then placed in the -80°C freezer until they were ready to be processed.

This fecal corticosterone procedure was based on the work of Cavigelli and colleagues (2005). Samples were thawed, placed into 12 x 75 mm polyurethane tubes, and weighed using a Sartorius electronic balance (Model BP 4100S; Sartorius Corporation; Edgewood, NY). After weighing, samples were placed into a speedvac (Model AS160 Automatic Speed Vac; Savant Instruments, Inc; Holbrook, NY) to remove moisture from the samples. The dried samples were then reweighed and crushed into a fine powder/dust. 0.20 g of the dust was placed into 15mL Corning centrifuge tubes. 10 mL of 100% ethanol was added to the dust in the centrifuge tubes and the samples were boiled in a water bath (Model IsoTemp 210; Fisher Scientific; Pittsburgh, PA) for 20 minutes. After boiling, the samples were centrifuged at 2000 rpm at 20°C for 15 minutes (Model Allegra 6R Centrifuge; Beckman Coulter, Inc.; Fullerton, CA). After centrifuging, the supernatant was poured off into 16 x 100 mm glass culture tubes. An additional 5 mL of 100% ethanol was poured into the centrifuge tubes and the samples were centrifuged again at 2000 rpm at 20°C for 15 minutes. The supernatant was poured off again and combined with the previous supernatant in the 16 x 100mm glass culture tubes. The fecal matter in the centrifuge tubes was discarded and the supernatant was placed under an air

dryer. After the supernatant was dried, it was stored in a -80°C freezer until ready to be assayed. When the samples were ready to be assayed, 1 mL of methanol was added to reconstitute the supernatant.

Fecal corticosterone was assayed by an ImmuChem Double-Antibody radioimmunoassay (RIA) kit using ¹²⁵I-labeled corticosterone (MP Biomedicals, Irvine, CA). All samples and standards were run in duplicate.

Serum Corticosterone

Serum corticosterone was collected upon completion of the experiment on experiment day 56. Animals were first anesthetized by carbon dioxide inhalation and then decapitated with a rat guillotine. After decapitation, trunk blood was collected and placed into 15 mL polystyrene centrifuge tubes. The tubes were centrifuged and serum was transferred to 1.5 mL microcentrifuge tubes with a transfer pipette. A new transfer pipette was used for each animal's blood sample. Serum samples were then placed into a -80°C freezer until they were ready to be assayed. Serum corticosterone was assayed by an ImmuChem Double-Antibody radioimmunoassay (RIA) kit using ¹²⁵I-labeled corticosterone (MP Biomedicals, Irvine, CA). All samples and standards were run in duplicate.

Open Field Activity (OF).

OF was measured once during the pre-stress phase, once during the stress phase, and three times during the post-stress phase on experiment days 6, 20, 34, 41, and 48, for a total of five times during this experiment using an

Omnitech/Accuscan Electronics Digiscan infrared photocell system. OF was measured between 0700 and 1300 hours. This procedure was performed in a behavioral testing room near the housing room and has been used extensively in the Grunberg laboratory (Berger, 2009; Cook, 2001; Elliott, 2004; Faraday, 2000; Perry, 2009; Tomchesson, 2006). The investigator turned on red overhead lights while placing rats into and removing rats from the activity chambers. Lights were turned off during the testing procedure.

Animals were placed individually into a clear Plexiglas chamber (40 x 40 x 30 cm) for one hour. Horizontal activity was measured by a photocell array containing 32 total pairs of infrared photocells placed 2 cm above the floor of the Plexiglas chamber. These 32 pairs of infrared photocells created a grid with the photocells spaced 2.5 cm apart from each other. Vertical activity was measured by an additional photocell array placed 10.5 cm above the floor of the chamber. This additional array contained 16 pairs of infrared photocells which were placed in parallel and were also spaced 2.5 cm apart. Data were electronically recorded in 12 5-minute bins using an Omnitech Model DCM-I-BBU analyzer.

Subjects were returned to their home cages after data collection and the locomotor chambers were cleaned using a 35% isopropyl alcohol solution. Cleaning the chambers with this solution prevents transmission of disease, minimizes the influence of odors from animals previously tested in the chamber on the activity of subsequent animals, and helps maintain the equipment.

Ultrasonic Vocalizations (USV)

Ultrasonic vocalizations were measured once during the baseline phase, once during the stress phase, and three times during the post-stress phase on experiment days 7, 21, 35, 42, and 49, for a total of five times throughout the experiment. Ultrasonic vocalizations were measured between 1200 and 1600 hours using a Med Associates, Inc. Ultrasonic Vocalization Detector (ANL-937-1, Med Associates, Inc., St. Albans, VT). Animals were transported from their housing room to a nearby procedure room. An overhead red light was turned on upon entry to the procedure room. The red light remained on during testing to permit the investigator to see. Rats were placed individually in large polycarbonate rat cages (46 cm x 36 cm x 20 cm) with hardwood chip bedding (Pine-Dri). The vocalization detector was placed on top of the cage lid with the microphone facing downwards. Ultrasonic vocalizations were recorded electronically for 2 minutes. The high frequency range was set to 45 kHz and above (Burgdorf & Panksepp, 2006; Panksepp, 2007; Panksepp & Burgdorf, 2000; Panksepp & Burgdorf, 2003). The low frequency range was set to 15 to 35 kHz (Brudzynski, Ociepa, & Bihari, 1991; Rosa et al., 2005). After testing, the animals were returned to their home cage. The order of testing was counterbalanced across conditions to minimize differences due to order effects.

Forced Swim Test (FST)

Forced swim test was performed once during the baseline phase, once during the stress phase, and three times during the post-stress phase on

experiment days 12, 26, 33, 40, and 47, for a total of five administrations throughout the experiment. Forced swim test was measured between 0700 and 1300 hours. This procedure consists of a training stage followed by a testing stage (Berger, 2009; Carlezon et al., 2002; Perry, 2009; Porsolt, 1977). The training stage was performed on experiment day 11, the day before the first testing day. The training and testing stages were identical with the exception of the length of the procedure. Rats were placed individually into Plexiglas cylinders (65 cm height x 25 cm diameter) filled with approximately four gallons of water. The water temperature was approximately 27°C. Rats remained in the cylinders for 15 minutes during the training stage and 5 minutes during the testing stage. Rats were then removed from the cylinders, dried with towels and warmed under heat lamps for 15 minutes.

A video camera mounted on the ceiling recorded the data throughout the procedure. Data were analyzed by AnyMaze software (Stoelting Co., Wood Dale, IL) for immobility during the forced swim test. Cylinders were cleaned of debris and feces between trials with a fish net. After testing, cylinders were cleaned using a 35% isopropyl alcohol solution. Cleaning the chambers with this solution prevents transmission of disease and helps maintain the equipment. A greater amount of immobility was interpreted as increased helplessness or depression. Animals were monitored throughout the procedure for signs of distress.

Home cage activity (HCA)

HCA was measured once per week on experiment days 2, 9, 16, 23, 30, 37, 44, and 51. HCA was rated between 0700 and 0800 hours by two experimenters who have been trained in this measure. The HCA rating sheet is included in Appendix C. Experimenters were trained in this method by experienced lab members who have previously used the HCA rating sheet.

This home cage activity procedure is based on the work of Tomchesson (2005), Long (2008), and Simpson-McKenzie (2008). The two experimenters quietly entered the darkened housing room and turned on an overhead red light (so that the room is dimly lit to allow experimenters to see). Both experimenters independently observed the movement of each subject for two 30-second intervals. At the end of each observation, each experimenter then used a 7-point Likert scale to rate level of activity. The order of subjects observed changed with each observation period.

Body weight (BW)

BW was measured once per week on experiment days 2, 9, 16, 23, 30, 37, 44, and 51, between 0700 and 1300 hours by using a Sartorius electronic balance (Model LC 4200, Sartorius Corporation, Edgewood, NY). The balance was programmed to take 10 measurements rapidly and provide the mean weight. This programming prevented inaccurate weight measurements based on movement artifacts.

Food consumption (FC)

FC was measured once per week on experiment days 2, 9, 16, 23, 30, 37, 44, and 51, between 0700 and 1300 hours by using a Sartorius electronic balance (Model LC 4200, Sartorius Corporation, Edgewood, NY). Cage lids with food were weighed and the weight recorded. Twenty-four hours later, the cage lids with food were re-weighed to determine the amount of food consumed over a 24 hour period. The amount of food consumed was calculated based on the change in weight. For animals that were pair housed, the change in weight was divided by the number of animals within the cage. When new food was added, the lids plus food pellets were weighed and recorded.

Data Analytic Strategy

Subjects were randomly assigned to housing conditions upon arrival. After conducting the experiment, all data were entered into electronic files and checked for entry errors. Once the data were verified as having been entered correctly, the data were examined for outliers. Data that were more than three standard deviations from the treatment group mean were excluded.

Once the outliers were excluded, exploratory analyses were conducted. The purpose of these exploratory analyses was to determine the best data analytic strategy. These exploratory analyses included multivariate analyses of variance (MANOVAs) at each time point and consideration of covariates in analyses. Although some variables were related, repeated measures analyses of variance (rmANOVAs) were chosen because the conceptual variable of

interest was resilience which, by definition, is a process that occurs over time. The available statistical software does not allow for repeated-measures multivariate analyses of variance and, even if such software was available, it is not clear that such analyses would provide additional information relevant to the hypotheses.

Exercise was used as a covariate in the exploratory analyses, but did not affect the results of the analyses and was, therefore, excluded from consideration as a covariate to simplify the statistical tests and their interpretation. Baseline measures also were used as covariates in the exploratory analyses, but also did not affect the results and were, therefore, excluded from the final analyses. The one baseline measure that did affect the results was body weight and was, therefore, included in the final statistical analyses.

In addition to consideration of multiple types of analyses and of several covariates, another step in the data analyses was to examine the differences in variance between treatment groups. Variance did not differ greatly between treatment groups. When sphericity (a type of variance across repeated measures) was not met, the Greenhouse-Geisser correction was used to statistically control for differences in variance.

Analyses of variance (ANOVA) were used for most of the final data analyses. Pair-wise comparisons were used for repeated-measures analyses where warranted. Chi-square tests were used to analyze social interaction data. The data analysis allowed assessment of the magnitude and time course of stress responses.

Fecal corticosterone was analyzed using a three-way ANOVA (sex x exercise training x social environment) to examine the effects of stress, exercise training, and social environment on the stress response. In addition to an overall repeated-measures ANOVA, a three-way ANOVA during the stress period was used to analyze stress effects. A repeated-measures ANOVA during the post-stress period with the stress period values as a covariate was used to analyze resilience and recovery during the post-stress period. Serum corticosterone was analyzed using a three-way ANOVA to examine the effects of stress, exercise training, and social environment on the stress response. For both of these analyses, sex, exercise training, and social environment were the between-subjects factors.

Open-field activity was analyzed using three-way repeated-measures ANOVAs (sex x exercise training x social environment) to examine the effects of stress, exercise training, and social environment on center time activity, an index of anxiety. Home cage activity was not used as a covariate to control for general movement because it was not correlated with either horizontal activity or the center time ratio. For all open-field activity analyses, stress phase was the within-subject factor and sex, exercise training, and social environment were the between-subjects factors. In addition to an overall repeated-measures ANOVA, a three-way ANOVA during the stress period was used to analyze stress effects. A repeated-measures ANOVA during the post-stress period with the stress period values as a covariate was used to analyze resilience and recovery during the post-stress period.

Ultrasonic vocalizations were analyzed using three-way repeated-measures ANOVAs (sex x exercise training x social environment) to examine the effects of stress, exercise training, and social environment on an index of positive and negative affect. Positive and negative affect analyses were conducted separately. For all ultrasonic vocalization analyses, stress phase was the within-subject factor and sex, exercise training, and social environment were the between-subjects factors. In addition to an overall repeated-measures ANOVA, a three-way ANOVA during the stress period was used to analyze stress effects. A repeated-measures ANOVA during the post-stress period with the stress period values as a covariate was used to analyze resilience and recovery during the post-stress period.

Social interaction data were analyzed using an overall χ^2 test at each time point to determine if the quality of social interaction differed between socially-housed animals. A χ^2 test crosstabulation test was used at each time point to examine the effects of sex on quality of social interaction. A χ^2 test crosstabulation test was used at each time point also to examine the effects of exercise on quality of social interaction.

Forced swim procedure data were analyzed using three-way repeated-measures ANOVAs (sex x exercise training x social environment) to examine the effects of stress, exercise training, and social environment on immobility, an index of depression. Home cage activity was not used as a covariate to control for general movement because it was not correlated with the forced swim test. For all forced swim immobility analyses, stress phase was the within-subject

factor and sex, exercise training, and social environment were the between-subjects factors. In addition to an overall repeated-measures ANOVA, a three-way ANOVA during the stress period was used to analyze stress effects. A repeated-measures ANOVA during the post-stress period with the stress period values as a covariate was used to analyze resilience and recovery during the post-stress period.

Body weight and food consumption were analyzed using three-way repeated-measures ANOVAs (sex x exercise training x social environment) to assess changes over time throughout the experiment. Each significant main effects or interaction was examined using a separate ANOVA following the procedures of Keppel (1991). In addition to an overall repeated-measures ANOVA, a three-way ANOVA during the stress period was used to analyze stress effects. A repeated-measures ANOVA during the post-stress period with the stress period values as a covariate was used to analyze resilience and recovery during the post-stress period.

Several strategies were used to minimize the probability of Type I and Type II error. First, only if overall analyses revealed a significant main effect or interaction were subsequent analyses performed. This strategy reduces the number of statistical tests (Keppel, 1991; Cohen & Cohen, 1983). All tests were two-tailed with significance determined by $p \leq 0.05$. In addition, the experiment was designed to provide adequate power (0.80), reducing the probability of Type II error (Keppel, 1991). Power is determined by the following factors: sample size, alpha, effect size, and variance. As the sample size, alpha, and effect size

increase, power also increases. As variance increases, power decreases.

Power may therefore be altered by changing the sample size, adjusting the alpha level, or by choosing a treatment with a larger effect size. This experiment was adequately powered to detect main effects and two-way interactions. In some instances, perhaps when the effect size was larger or the variance less, the experiment was adequately powered to detect three-way interactions.

Results

Experimental results are organized by hypothesis. Within each section, results are presented in the following order: fecal corticosterone, serum corticosterone, open field activity, ultrasonic vocalizations, forced swim test, home cage activity, social interaction, body weight, and food consumption. Within each dependent variable, overall analyses are presented first, followed by stress period and post-stress period analyses. Results for internal analyses (females and males analyzed separately) are presented only when they yielded significant findings. Figures are presented in Appendix D and are organized conceptually by dependent variable. Only statistically significant findings are presented within the body of this report with the F-value and p-value in parentheses and reference to the corresponding figure. The particular type of main effect or interaction is in bold type within these parentheses for clarity of presentation. All values from statistical analyses are presented (including non-significant findings) in tables in Appendix E.

Hypothesis I

Combined sleep disturbance and predator stress will have significant effects on biological and psychological measures over time.

(a) Combined sleep disturbance and predator stress will decrease center time in the open field chambers, body weight, and food consumption.

(b) Combined sleep disturbance and predator stress will increase fecal corticosterone levels, ultrasonic vocalizations at lower frequencies, and forced swim immobility.

(c) No *a priori* hypothesis is offered for the effects of combined sleep disturbance and predator stress on home cage activity.

Fecal corticosterone (Figures 1- 8; Tables 1- 20). Fecal corticosterone was collected from the open field chambers after one hour of testing: once during the pre-stress period, once during the stress period, and three times during the post-stress period for a total of five measurements throughout the experiment.

Overall, fecal corticosterone levels changed over time (**Time:** $F[4,16] = 4.910$, $p = 0.009$; see Figure 2 and Table 2). Fecal corticosterone values increased during the stress period and decreased during the post-stress period.

Stress period analyses. During the stress period, after covarying for pre-stress period values, time did not significantly affect fecal corticosterone.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, time did not significantly affect fecal corticosterone.

Serum corticosterone (Figures 9 - 11; Tables 21 – 22). Serum corticosterone was collected at the end of the experiment. Serum corticosterone

could not be analyzed for stress effects over time because all data were collected at one time point.

Open field center time activity (Figures 12 -16; Tables 23 - 33). Open field center time activity provides a behavioral index of anxiety and was measured once during the pre-stress period, once during the stress period, and three times during the post-stress period for a total of five measurements throughout the experiment.

Center time changed over the course of the experiment (**Time:** $F[4,224] = 20.919$, $p < 0.001$, see Figures 12 and 13; Table 24). Center time values remained similar from pre-stress to stress, increased from stress to the first post-stress measurement, increased from the first to the second post-stress measurement, and decreased from the second to the third post-stress measurement.

Stress period analyses. During the stress period, after covarying for pre-stress period values, time did not significantly affect center time activity.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, time did not significantly affect center time activity.

Open field horizontal activity (Figures 17 - 23; Tables 34 - 46). Open field horizontal activity was measured once during the pre-stress period, once

during the stress period, and three times during the post-stress period for a total of five measurements throughout the experiment.

Horizontal activity changed over time (**Time**: $F[4,224] = 14.943$, $p < 0.001$, see Figures 17 and 18; Table 35). Horizontal activity increased from the pre-stress to the stress period, decreased from the stress period to the first post-stress measurement, decreased from the first to the second post-stress measurement, and increased from the second to the third post-stress measurement.

Stress period analyses. Treatment groups did not differ significantly during the stress period after covarying for pre-stress period values.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, horizontal activity decreased from the first post-stress measurement to the second and increased from the second post-stress measurement to the third (**Time**: $F[1.805,99.263] = 4.547$, $p = 0.016$, see Figure 19; Tables 42 and 43).

Ultrasonic vocalizations – Negative affect (Figures 24 - 27; Tables 47 - 58). Low frequency (approximately 20kHz) ultrasonic vocalizations provide an index of negative affect and were measured once during the pre-stress period, once during the stress period, and three times during the post-stress period for a total of five measurements throughout the experiment. There were no significant effects of time on negative affect.

Stress period analyses. During the stress period, after covarying for pre-stress values, there were no significant effects of time on ultrasonic vocalizations at approximately 20kHz.

Post-stress period analyses. During the post-stress period, after covarying for stress values, there were no significant effects of time on ultrasonic vocalizations at approximately 20kHz.

Ultrasonic vocalizations – Positive affect (Figures 28 - 31; Tables 59 - 70). High frequency (approximately 50kHz) ultrasonic vocalizations provide an index of positive affect and were measured once during the pre-stress period, once during the stress period, and three times during the post-stress period for a total of five measurements throughout the experiment. There were no significant effects of time on ultrasonic vocalizations at approximately 50kHz.

Stress period analyses. During the stress period, after covarying for pre-stress period values, time did not significantly affect ultrasonic vocalizations at approximately 50kHz.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, time did not significantly affect ultrasonic vocalizations at approximately 50kHz.

Home cage activity (Tables 71 - 73). Home cage activity provides an overall measure of activity in a familiar environment, which is different from activity in the open field chamber in a novel environment. Home cage activity

was measured once weekly for the duration of the experiment. Of the eight home cage activity time points, five (i.e., one pre-stress, one stress, and three post-stress) were analyzed to make the statistical analyses comparable across dependent variables. The measurement of home cage activity was intended to be used as a covariate for other activity-based measures, specifically open field activity and forced swim test, to control for baseline differences in movement. Home cage activity was not consistently correlated with either activity-based measure (open field activity or forced swim test) and therefore was not used as a covariate.

Social interaction (Tables 74 - 95). Social interaction was included in the home cage activity measurement to account for differences in the social environment within the social housing condition. Social interaction was assessed once weekly for the duration of the experiment. Of the eight social interaction time points, five (i.e., one pre-stress, one stress, and three post-stress) were analyzed to make the statistical analyses comparable across dependent variables. Social interaction data were analyzed separately at each time point rather than over time. Results of these statistical analyses are presented in hypothesis II (exercise training), hypothesis III (social housing), and hypothesis IV (sex differences).

Forced swim test (Figure 32; Tables 96 - 106). Forced swim test provides a behavioral index of depression and was measured once during the

pre-stress period, once during the stress period, and three times during the post-stress period for a total of five measurements throughout the experiment.

Immobility in the forced swim chamber may be defined by the percentage of the animal's body that is not moving. The sensitivity of immobility detection may be set anywhere between 50% and 100%. The forced swim data were analyzed at several cut-off points: 50%, 60%, and 65%. At all three cut-off points, treatment groups did not differ significantly in time spent immobile in the forced swim test. The only significant finding from this measure, present at all three cut-off points and presented here at 65% sensitivity, was that animals increased forced swim immobility over time (**Time:** $F[4,220] = 7.268$, $p < 0.001$; see Figure 32; Table 97). Forced swim immobility remained similar from the pre-stress to the stress period, and again from the stress period to the first post-stress measurement, then increased from the first to the second post-stress measurement, and increased again from the second to the third post-stress measurement.

Body weight (Figures 33 - 36; Tables 107 - 120). Body weight was measured once weekly for the duration of the experiment. Of the eight body weight time points, five (i.e., one pre-stress, one stress, and three post-stress) were analyzed to make the statistical analyses comparable across dependent variables. Time did not significantly affect body weight.

Stress period analyses. During the stress period and after covarying pre-stress period values, time did not significantly affect body weight.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, time did not significantly affect body weight.

Food consumption (Figures 37 - 40; Tables 121 - 134). Food consumption was measured once weekly for the duration of the experiment. Of the eight food consumption time points, five (i.e., one pre-stress, one stress, and three post-stress) were analyzed to make the statistical analyses comparable across dependent variables. Time did not significantly affect food consumption.

Stress period analyses. During the stress period and after covarying for pre-stress period values, time did not significantly affect food consumption.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, food consumption increased from the first post-stress measurement to the second, and then decreased from the second post-stress measurement to the third (**Time**: $F[1.709, 93.996] = 5.996$, $p = 0.005$; see Figure 37; Tables 130 - 131).

Summary of hypothesis I findings. Fecal corticosterone values changed over time, with values increasing during the stress period. Center time ratio values changed over time, with values increasing during the post-stress period. Horizontal activity changed over time, with values increasing during the stress period and the third post-stress measurement. Ultrasonic vocalizations values did not change over time. Forced swim test immobility values changed over time, with values increasing during the second and third post-stress

measurements. Body weight and food consumption values did not change over time. A summary of the significant findings related to hypothesis I is presented in Table B below.

Variable	Time
fCort	Increase during stress
sCort	N/A
CTR	Increase post-stress
HZA	Increase during stress and post-stress
USV 20kHz	---
USV 50kHz	---
FST	Increase post-stress
BW	---
FC	---

Table B. Hypothesis I significant effects. fCort = fecal corticosterone; sCort = serum corticosterone; CTR = center time activity; HZA = horizontal activity; USV = ultrasonic vocalizations; FST = forced swim test; BW = body weight; FC = food consumption.

Hypothesis II

Exercise training will attenuate stress' effects on biological and psychological measures.

Fecal corticosterone (Figures 1- 8; Tables 1- 20). Animals that did or did not receive exercise training differed in fecal corticosterone values over time (**Time x Exercise:** $F[4,16] = 3.392$, $p = 0.034$; see Figure 3; Table 2). More specifically, animals that did not receive exercise training displayed higher fecal corticosterone values during the stress period than during the pre-stress or post-stress periods. In contrast, animals that did receive exercise training displayed

higher fecal corticosterone values during the pre-stress measurement than during the first and second post-stress measurements.

Stress period analyses. During the stress period, after covarying for pre-stress values, exercise training did not significantly affect fecal corticosterone values.

Post-stress period analyses. During the post-stress period, after covering for stress period values, exercise training did not significantly affect fecal corticosterone values.

Pre-stress analyses. During the pre-stress period, exercise training did not significantly affect fecal corticosterone values.

Stress analyses. During the stress period, exercise training did not significantly affect fecal corticosterone values.

Post-stress I analyses. During the first post-stress measurement, exercise training did not significantly affect fecal corticosterone values.

Post-stress II analyses. During the second post-stress measurement, exercise training did not significantly affect fecal corticosterone values.

Post-stress III analyses. During the third post-stress measurement, exercise training increased fecal corticosterone levels (**Exercise:** $F[1,36] = 4.081$, $p = 0.051$; see Figure 3; Table 19).

Serum corticosterone (Figures 9 - 11; Tables 21 - 22). Exercise training did not significantly affect serum corticosterone levels.

Open field center time activity (Figures 12 -16; Tables 23 - 33).

Exercise training decreased center time activity overall (**Exercise:** $F[1,56] = 8.091$, $p = 0.006$; see Figure 12; Table 23).

Stress period analyses. During the stress period, after covarying for pre-stress values, exercise training did not significantly affect center time activity.

Post-stress period analyses. During the post-stress period, after covarying for stress values, animals that did not receive exercise training exhibited greater center time activity than did animals that did receive exercise training (**Exercise:** $F[1,55] = 7.619$, $p = 0.008$; see Figure 12; Table 30).

Open field horizontal activity (Figures 17 - 23; Tables 34 - 46).

Exercise training decreased horizontal activity (**Exercise:** $F[1,56] = 5.582$, $p = 0.022$; see Figure 17; Table 34). Exercise changed the pattern of horizontal activity over time (**Time x Exercise:** $F[4,224] = 23.818$, $p < 0.001$; see Figure 17; Table 35). In animals that did not receive exercise training, horizontal activity increased from the pre-stress to the stress period, remained similar from the stress period to the first post-stress measurement, remained similar from the first to the second post-stress measurement, and decreased from the second to the third post-stress measurement, whereas in animals that did receive exercise training, horizontal activity increased from the pre-stress to the stress period, decreased from the stress period to the first post-stress measurement,

decreased from the first to the second post-stress measurement, and increased from the second to the third post-stress measurement.

Stress period analyses. During the stress period, after covarying for pre-stress period values, exercise training did not significantly affect horizontal activity.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, animals that received exercise training engaged in less horizontal activity than did animals that did not receive exercise training overall (**Exercise:** $F[1,55] = 4.649$, $p = 0.035$; see Figure 17; Table 41). Exercise training changed the pattern of horizontal activity in the post-stress period (**Time x Exercise:** $F[1.805,99.263] = 46.250$, $p < 0.001$; see Figure 17; Tables 42 - 43). In animals that did not receive exercise training, horizontal activity did not change significantly from the first post-stress measurement to the second and decreased from the second post-stress measurement to the third. In animal that did receive exercise training, horizontal activity decreased from the first post-stress measurement to the second and increased from the second post-stress measurement to the third.

Ultrasonic vocalizations – Negative affect (Figures 24 - 27; Tables 47 - 58). Exercise training decreased ultrasonic vocalizations at approximately 20kHz overall (**Exercise:** $F[1,56] = 10.321$, $p = 0.002$; see Figure 24; Table 47). Exercise training dramatically reduced the expression of ultrasonic vocalizations at approximately 20kHz in females overall and moderately reduced the

expression of ultrasonic vocalizations at approximately 20kHz in males overall (**Sex x Exercise:** $F[1,56] = 4.775$, $p = 0.033$; see Figures 26 and 27; Table 47).

Stress period analyses. During the stress period, after covarying for pre-stress values, exercise training did not significantly affect ultrasonic vocalizations at approximately 20kHz.

Post-stress period analyses. During the post-stress period, after covarying for stress values, exercise training decreased expression of ultrasonic vocalizations at approximately 20kHz (**Exercise:** $F[1,55] = 9.978$, $p = 0.003$; see Figure 24; Table 55). In females, exercise training decreased ultrasonic vocalizations at approximately 20kHz, whereas in males exercise training had no significant effect (**Sex x Exercise:** $F[1,55] = 11.199$, $p = 0.001$; see Figure 26 for females and Figure 27 for males; Table 55).

Ultrasonic vocalizations – Positive affect (Figures 28 - 31; Tables 59 - 70). Exercise training decreased ultrasonic vocalizations at approximately 50kHz overall (**Exercise:** $F[1,56] = 18.535$, $p < 0.001$; see Figure 28; Table 59).

Exercise training dramatically reduced the expression of ultrasonic vocalizations at approximately 50kHz in females overall and moderately reduced the expression of ultrasonic vocalizations at approximately 50kHz in males overall (**Sex x Exercise:** $F[1,56] = 9.063$, $p = 0.004$; see Figures 30 and 31; Table 59).

Stress period analyses. During the stress period, after covarying for pre-stress period values, exercise training did not significantly affect ultrasonic vocalizations at approximately 50kHz.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, exercise training decreased expression of ultrasonic vocalizations at approximately 50kHz (**Exercise:** $F[1,55] = 25.038$, $p < 0.001$; see Figure 28; Table 67). In females, exercise training decreased expression of ultrasonic vocalizations at approximately 50kHz, whereas in males exercise training had no significant effect on ultrasonic vocalizations at approximately 50kHz (**Sex x Exercise:** $F[1,55] = 25.905$, $p < 0.001$) see Figures 30 and 31; Table 67).

Social Interaction (Tables 74 - 95). Social interaction data were analyzed separately at each time point for the effects of exercise on type or quality of social interaction.

Pre-stress analyses. Exercise did not significantly affect social interaction.

Stress analyses. Exercise did not significantly affect social interaction.

Post-stress I analyses. Exercise did not significantly affect social interaction.

Post-stress II analyses. Exercise did not significantly affect social interaction.

Post-stress III analyses. Exercise did not significantly affect social interaction.

Forced swim test (Figure 32; Tables 96 - 106). There were no significant effects of exercise training on immobility in the forced swim test.

Body weight (Figures 33 - 36; Tables 107 - 120). Exercise training decreased body weight overall (**Exercise:** $F[1,55] = 15.524$, $p < 0.001$; see Figure 33; Table 107). Animals receiving exercise training gained less body weight over time than did animals not receiving exercise training (**Time x Exercise:** $F[1.740,95.674] = 12.845$, $p < 0.001$; see Figure 33; Tables 108 - 109). Exercise training attenuated body weight gain only in males and had no overall effect on body weight in females (**Sex x Exercise:** $F[1,55] = 28.939$, $p < 0.001$; see Figure 35 for females and Figure 36 for males; Table 107).

Stress period analyses. During the stress period and after covarying pre-stress period values, animals receiving exercise training weighed less than those animals not receiving exercise training (**Exercise:** $F[1,55] = 27.897$, $p < 0.001$; see Figure 33; Table 113). Exercise training decreased body weight only in males and had no overall effect on body weight in females (**Sex x Exercise:** $F[1,55] = 40.090$, $p < 0.001$; see Figure 35 for females and Figure 36 for males; Table 113).

Post-stress period analyses. During the post-stress period, after covarying for stress period values, exercise training had no effect on body weight.

Food consumption (Figures 37 - 40; Tables 121 - 134). Exercise training decreased food consumption overall (**Exercise:** $F[1,53] = 8.523$, $p = 0.005$; see Figure 37; Table 121). Animals that received exercise training ate less than did those animals that did not receive exercise training throughout the experiment, except in the last post-stress measurement when there was no significant difference between the two groups (**Time x Exercise:** $F[3.106,164.592] = 4.835$, $p = 0.003$; see Figure 37; Tables 122 - 123). Exercise training decreased food consumption only in males and had no overall effect on food consumption in females (**Sex x Exercise:** $F[1,54] = 10.228$, $p = 0.002$; see Figure 39 for females and Figure 40 for males; Table 121).

Stress period analyses. During the stress period and after covarying for pre-stress period values, animals that received exercise training ate less than did those animals that did not receive exercise training (**Exercise:** $F[1,53] = 9.039$, $p = 0.004$; see Figure 37; Table 127).

Post-stress period analyses. During the post-stress period, after covarying for stress period values, exercise training decreased food consumption in males but had no significant effect in females (**Sex x Exercise:** $F[1,55] = 8.681$, $p = 0.005$; see Figure 39 for females and Figure 40 for males; Table 129). Overall, food consumption increased from the first post-stress measurement to the second, and then decreased from the second post-stress measurement to the third but the decrease from the second to the third post-stress measurement was greater in animals that did not receive exercise training (**Time x Exercise:** $F[1.709,93.996] = 13.382$, $p < 0.001$; see Figure 37; Tables 130 - 131).

Summary of hypothesis II findings. Exercise training changed the pattern of fecal corticosterone values over time by decreasing values from the pre-stress period to the post-stress period. Exercise training had no effect on serum corticosterone values. Exercise training decreased both center time ratio and horizontal activity values. Exercise training decreased ultrasonic vocalizations at both 20kHz and 50kHz, but more in females than in males. Exercise training had no effect on forced swim test immobility or social interaction. Exercise training decreased body weight and food consumption values overall, but this decrease occurred in males only and had no effect in females. A summary of significant findings related to hypothesis II is presented in Table C below.

Variable	Exercise (Ex)			
	Overall	Female	Male	Time x Exercise
fCort	---	---	---	Ex decrease pre- to post-stress
sCort	---	---	---	--
CTR	No Ex > Ex	No Ex > Ex	No Ex = Ex	--
HZA	No Ex > Ex	No Ex > Ex	No Ex = Ex	Ex increase during stress and post-stress 3
USV 20kHz	No Ex > Ex	No Ex >> Ex	No Ex > Ex	--
USV 50kHz	No Ex > Ex	No Ex >> Ex	No Ex > Ex	--
FST	---	---	---	--
BW	No Ex > Ex	No Ex = Ex	No Ex > Ex	Ex decrease over time
FC	No Ex > Ex	No Ex = Ex	No Ex > Ex	Ex decrease over time

Table C. Hypothesis II significant effects. fCort = fecal corticosterone; sCort = serum corticosterone; CTR = center time activity; HZA = horizontal activity; USV = ultrasonic vocalizations; FST = forced swim test; BW = body weight; FC = food consumption.

Hypothesis III

Social housing will attenuate stress' effects on biological and psychological measures.

Fecal corticosterone (Figures 1- 8; Tables 1- 20). Individually- and socially-housed animals differed in fecal corticosterone values over time (**Time x Social**: $F[4,16] = 4.879$, $p = 0.009$; see Figure 4; Table 2). Individually-housed ("No Social") animals displayed no significant differences in fecal corticosterone values over time. Socially-housed ("Social") animals displayed higher fecal corticosterone values during the stress period than during the pre-stress or post-stress periods.

Stress period analyses. During the stress period, after covarying for pre-stress values, socially-housed animals had greater levels of fecal corticosterone than did individually-housed animals (**Social**: $F[1,24] = 5.248$, $p = 0.031$, see Figure 4; Table 5). When fecal corticosterone was analyzed separately for males and females, the social housing difference appeared only for females (**Social**: $F[1,10] = 5.574$, $p = 0.040$; see Figure 5; Table 6) and not males (see Figure 6; Table 6).

Post-stress period analyses. During the post-stress period, after covarying for pre-stress values, social housing did not significantly affect fecal corticosterone values.

Pre-stress analyses. During the pre-stress period, social housing decreased fecal corticosterone levels (**Social**: $F[1,30] = 4.619$, $p = 0.040$, see

Figure 4; Table 11). When females and males were analyzed separately, this difference held true only for females (**Social**: $F[1,15] = 4.424$, $p = 0.053$, see Figure 5 for females and Figure 6 for males; Table 12).

Stress analyses. During the stress period, social housing increased fecal corticosterone levels (**Social**: $F[1,42] = 9.152$, $p = 0.004$; see Figure 4; Table 13), but this held true only for females when analyzed separately from males (**Social**: $F[1,18] = 11.182$, $p = 0.004$, see Figure 5 for females and Figure 6 for males; Table 14).

Post-stress I analyses. During the first post-stress measurement, social housing did not significantly affect fecal corticosterone values.

Post-stress II analyses. During the second post-stress measurement, social housing did not significantly affect fecal corticosterone values.

Post-stress III analyses. During the third post-stress measurement, social housing did not significantly affect fecal corticosterone values.

Serum corticosterone (Figures 9 - 11; Tables 21 - 22). Social housing tended to increase serum corticosterone levels overall (**Social**: $F[1,55] = 3.668$, $p = 0.061$; see Figure 9; Table 21). For animals without exercise training, those that were socially-housed had greater levels of corticosterone than those that were individually-housed (**Exercise x Social**: $F[1,55] = 9.123$, $p = 0.004$, see Figure 10; Table 21). For animals with exercise training, there were no differences between housing conditions.

Open field center time activity (Figures 12 -16; Tables 23 - 33). Social housing increased center time activity overall (**Social:** $F[1,56] = 10.241$, $p = 0.002$; see Figure 13; Table 23). Animals in the social housing condition increased time spent in the center of the open field chamber over the course of the experiment more than did animals in the individual housing condition (**Time x Social:** $F[4,224] = 7.057$, $p < 0.001$; see Figure 13; Table 24).

Stress period analyses. During the stress period, after covarying for pre-stress values, socially housed animals exhibited greater center time activity than did individually housed animals (**Social:** $F[1,55] = 5.197$, $p = 0.027$; see Figure 13; Table 28). When males and females were analyzed separately, social housing increased center time only in females (**Social:** $F[1,27] = 5.233$, $p = 0.030$; see Figure 15; Table 29) but not in males (see Figure 16; Table 29).

Post-stress period analyses. During the post-stress period, after covarying for stress values, animals that were pair-housed exhibited greater center time activity than did animals that were individually-housed (**Social:** $F[1,55] = 9.302$, $p = 0.004$; see Figure 13; Table 30). Social housing changed the overall pattern of center time activity during the three post-stress measurements (**Time x Social:** $F[2,110] = 6.096$, $p = 0.003$; see Figure 13; Table 31). Animals that were individually housed did not change their pattern of center time activity significantly across any of the post-stress measurements. Animals that were socially housed increased their center time activity from the first post-stress measurement to the second and decreased their center time activity from the second post-stress measurement to the third.

Open field horizontal activity (Figures 17 - 23; Tables 34 - 46). Social housing decreased horizontal activity (**Social:** $F[1,56] = 9.928$, $p = 0.003$, see Figure 18; Table 34).

Stress period analyses. During the stress period, after covarying for pre-stress period values, social housing did not significantly affect horizontal activity.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, animals that were socially housed engaged in less horizontal activity than did animals that were individually housed (**Social:** $F[1,55] = 9.102$, $p = 0.004$; see Figure 18; Table 41).

Ultrasonic vocalizations – Negative affect (Figures 24 - 27; Tables 47 - 58). Social housing did not significantly affect ultrasonic vocalizations at approximately 20kHz.

Stress period analyses. During the stress period, after covarying for pre-stress values, social housing did not significantly affect ultrasonic vocalizations at approximately 20kHz.

Post-stress period analyses. During the post-stress period, after covarying for stress values, social housing did not significantly affect ultrasonic vocalizations at approximately 20kHz.

Ultrasonic vocalizations – Positive affect (Figures 28 - 31; Tables 59 - 70). Social housing did not significantly affect ultrasonic vocalizations at approximately 50kHz.

Stress period analyses. During the stress period, after covarying for pre-stress period values, social housing did not significantly affect ultrasonic vocalizations at approximately 50kHz.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, social housing did not significantly affect ultrasonic vocalizations at approximately 50kHz.

Social interaction (Tables 74 - 95). Social interaction data were collected only from socially-housed animals and, therefore, were analyzed separately at each time point for differences in type or quality of social interaction.

Pre-stress analyses. There appeared to be no overall differences in types of social interaction the subjects displayed.

Stress analyses. Subjects displayed more passive and dominant/submissive social interaction than expected, and less co-aggressive and co-supportive behavior than expected (**Stress:** $\chi^2 [3, N = 32] = 15.000$, $p = 0.002$; Tables 74 and 75).

Post-stress I analyses. Subjects displayed more passive social interaction than expected, and less dominant/submissive, co-aggressive, and co-supportive behavior than expected (**Post-Stress 1:** $\chi^2 [3, N = 32] = 43.750$, $p < 0.001$; Tables 74 and 75).

Post-stress II analyses. Subjects displayed more passive and dominant/submissive social interaction than expected, an expected amount of

co-aggressive social interaction, and less co-supportive behavior than expected (**Post-Stress 2:** $\chi^2 [3, N = 32] = 12.250, p = 0.007$; Tables 74 and 75).

Post-stress III analyses. Subjects displayed more passive and dominant/submissive social interaction than expected, and less co-aggressive and co-supportive behavior than expected (**Post-Stress 3:** $\chi^2 [3, N = 32] = 21.25, p < 0.001$; Tables 74 and 75).

Forced swim test (Figure 32; Tables 96 - 106). There were no significant effects of social housing on immobility in the forced swim test.

Body weight (Figures 33 - 36; Tables 107 - 120). Social housing had no significant effect on body weight.

Stress period analyses. During the stress period and after covarying pre-stress period values, social housing had no effect on body weight.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, social housing had no effect on body weight.

Food consumption (Figures 37 - 40; Tables 121 - 134). Social housing decreased food consumption overall (**Social:** $F[1,53] = 19.116, p < 0.001$; see Figure 38; Table 121). Animals that were socially housed ate less than did animals that were individually housed throughout the experiment, except in the pre-stress period when there was no significant difference between the two

groups (**Time x Social**: $F[3.106,164.592] = 5.701$, $p = 0.001$; see Figure 38; Tables 122 and 123).

Stress period analyses. During the stress period and after covarying for pre-stress period values, animals that were socially housed ate less than did animals that were individually housed (**Social**: $F[1,53] = 43.044$, $p < 0.001$; see Figure 38; Table 127). This effect of social housing on food consumption was greater in males than in females (**Sex x Social**: $F[1,53] = 6.589$, $p = 0.013$; see Figure 39 for females and Figure 40 for males; Table 127). This effect of social housing on food consumption was greater in animals that did not receive exercise training than in animals that did (**Exercise x Social**: $F[1,53] = 7.252$, $p = 0.009$; see Figure 39 for females and Figure 40 for males; Table 127).

Post-stress period analyses. During the post-stress period, after covarying for stress period values, socially housed animals ate less overall than did individually housed animals (**Social**: $F[1,55] = 4.330$, $p = 0.042$; see Figure 38; Table 129).

Summary of hypothesis III findings. Social housing changed the pattern of fecal corticosterone values over time by increasing values during the stress period. Social housing increased serum cortisosterone and center time ratio values. Social housing decreased horizontal activity, and had no effect on ultrasonic vocalizations or forced swim test immobility. The quality of social interaction at each time point was different during the stress period and during each post stress measurement. Social housing had no effect on body weight

and decreased food consumption values. A summary of significant effects related to hypothesis III is presented in Table D below.

Variable	Social Housing (Soc)			
	Overall	Female	Male	Time x Social
fCort	---	---	---	Soc increase during stress
sCort	No Soc < Soc	---	---	---
CTR	No Soc < Soc	---	---	Soc increase over time
HZA	No Soc > Soc	No Soc > Soc	No Soc = Soc	---
USV 20kHz	--	---	---	---
USV 50kHz	---	---	---	---
FST	---	---	---	---
BW	---	---	---	---
FC	No Soc > Soc	---	---	Soc decrease over time

Table D. Hypothesis III significant effects. fCort = fecal corticosterone; sCort = serum corticosterone; CTR = center time activity; HZA = horizontal activity; USV = ultrasonic vocalizations; FST = forced swim test; BW = body weight; FC = food consumption.

Hypothesis IV

These stress responses to sleep disturbance stress will differ between females and males. Specifically, females will be more stress responsive than males.

(a) Social housing will attenuate stress' effects on behavioral and biological measures more in males than in females.

(b) No *a priori* hypothesis is offered for sex differences in the effects of exercise training on the stress response.

Fecal corticosterone (Figures 1- 8; Tables 1- 20) . Males tended to have higher levels of fecal corticosterone than did females (**Sex:** $F[1,4] = 6.598$, $p = 0.062$; see Figure 1; Table 1). This sex difference was significant during the stress measurement ($F[1,42] = 44.185$, $p < 0.001$; Table 13), the first post-stress measurement ($F[1,22] = 5.059$, $p = 0.035$; Table 15), and the third post-stress measurement ($F[1,36] = 11.747$, $p = 0.002$; Table 19), but not during the pre-stress measurement or the second post-stress measurement.

Stress period analyses. During the stress period, after covarying for pre-stress values, males had greater levels of fecal corticosterone than did females (**Sex:** $F[1,24] = 20.787$, $p < 0.001$, see Figure 1; Table 5).

Post-stress period analyses. During the post-stress period, after covarying for stress values, males had higher levels of fecal corticosterone than did females (**Sex:** $F[1,9] = 9.629$, $p = 0.013$, see Figure 1; Table 7). Exercise training and social housing together altered fecal corticosterone levels differently in females and males (**Sex x Exercise x Social:** $F[1,9] = 6.488$, $p = 0.031$; see Figure 7 for females and Figure 8 for males; Table 7). In females, animals that had not received exercise training and were socially housed had greater levels of fecal corticosterone than did animals that had not received exercise training and were individually housed, whereas in males the opposite was true. In females, animals that had received exercise training and were individually housed animals had greater levels of fecal corticosterone than did animals that were socially housed, whereas in males there were no differences between housing groups. The effects of exercise training and social housing on fecal corticosterone levels

in males and females changed over time during the post-stress period (**Time x Sex x Exercise x Social**: $F[2,18] = 3.987$, $p = 0.037$, see Figure 7 for females and Figure 8 for males; Table 8).

Internal analyses for resilience (post-stress period). When males and females were analyzed separately, females did not differ by treatment group. In males only, animals that were individually-housed increased their fecal corticosterone levels over the post-stress period, whereas animals that were socially-housed decreased their fecal corticosterone levels over the post-stress period (**Time x Social**: $F[2,14] = 4.533$, $p = 0.030$; see Figure 8; Table 10). Exercise training and social housing changed the pattern of male fecal corticosterone values over time (**Time x Exercise x Social**: $F[2,14] = 6.961$, $p = 0.008$; see Figure 8; Table 10). Male animals that did not receive exercise training and were individually housed maintained similar levels of fecal corticosterone throughout the post-stress period, whereas male animals that did not receive exercise training and were socially housed displayed an increase in fecal corticosterone levels from the first to the second post-stress measurement. Male animals that did receive exercise training and were individually housed maintained similar levels of fecal corticosterone throughout the post-stress period, whereas male animals that did receive exercise training and were socially housed displayed a decrease in their fecal corticosterone from first to the second post-stress period.

Pre-stress analyses. During the pre-stress period, sex did not significantly affect fecal corticosterone values.

Stress analyses. Males had higher levels of fecal corticosterone than did females (**Sex:** $F[1,42] = 44.185$, $p < 0.001$; see Figure 1; Table 13). Exercise training and social housing together altered fecal corticosterone levels differently in females and males (**Sex x Exercise x Social:** $F[1,42] = 4.024$, $p = 0.051$; see Figure 7 for females and Figure 8 for males; Table 13). In female animals that did not receive exercise training, there were no differences between housing groups, whereas in male animals that did not receive exercise training, socially housed animals displayed greater fecal corticosterone values than did individually housed animals. In female animals that did receive exercise training, socially housed animals displayed greater fecal corticosterone values than did individually housed animals, whereas in males that did receive exercise training, there were no differences between housing groups.

Post-stress I analyses. During the first post-stress measurement, males had higher levels of fecal corticosterone than did females (**Sex:** $F[1,22] = 5.059$, $p = 0.035$; see Figure 1; Table 15). Exercise training and social housing together altered fecal corticosterone levels differently in females and males (**Sex x Exercise x Social:** $F[1,42] = 7.534$, $p = 0.012$; see Figure 7 for females and Figure 8 for males; Table 15). In both females and males, there were no differences between social housing groups in animals that had not received exercise training. In females that did receive exercise training, individually housed animals displayed greater fecal corticosterone levels than did socially housed animals, whereas in males that did receive exercise training, socially housed animals displayed greater fecal corticosterone levels than did individually

housed animals. When females and males were analyzed separately, there were no differences between treatment groups for females. In males, social housing increased fecal corticosterone levels overall (**Social**: $F[1,15] = 9.674$, $p = 0.007$; see Figure 6; Table 16), but this difference was due solely to animals that had received exercise training and were socially housed (**Exercise x Social**: $F[1,15] = 23.448$, $p < 0.001$; see Figure 8; Table 16).

Post-stress II analyses. During the second post-stress measurement, sex did not significantly affect fecal corticosterone values.

Post-stress III analyses. During the third post-stress measurement, males had higher overall levels of fecal corticosterone than did females (**Sex**: $F[1,36] = 11.747$, $p = 0.002$; see Figure 1; Table 19). Social housing decreased fecal corticosterone levels in males but not females (**Sex x Social**: $F[1,36] = 6.694$, $p = 0.014$; see Figure 5 for females and Figure 6 for males; Table 19).

Serum corticosterone (Figures 9 - 11; Tables 21 - 22). Females had greater corticosterone levels than males (**Sex**: $F[1,55] = 40.222$, $p = 0.004$; see Figure 9; Table 21). Exercise training and social housing together changed serum corticosterone levels differently in females and in males (**Sex x Exercise x Social**: $F[1,55] = 5.350$, $p = 0.024$; see Table 21). In females without exercise training, social housing increased serum corticosterone levels, whereas in males without exercise training, there were no differences between housing conditions. In females with exercise training, social housing decreased serum corticosterone

levels, whereas in males with exercise training, there were no differences between housing conditions.

When females and males were analyzed separately, social housing increased corticosterone levels in animals without exercise training in females (**Exercise x Social**: $F[1,27] = 8.346$, $p = 0.008$; see Figure 10; Table 22) but not males (see Figure 11; Table 22). For females with exercise training, there were no differences between housing conditions. There were no differences between treatment groups in males.

Open field center time activity (Figures 12 -16; Tables 23 - 33). Males spent greater amounts of time in the center of the open field chamber than did females (**Sex**: $F[1,56] = 12.150$, $p < 0.001$; see Figure 14; Table 23). Males increased time spent in the center of the open field chamber over the course of the experiment more than did females (**Time x Sex**: $F[4,224] = 2.637$, $p = 0.035$; see Figure 14; Table 24). When males and females were analyzed separately, exercise training decreased center time only in females (**Exercise**: $F[1,28] = 7.074$, $p = 0.013$; see Figure 15; Table 25) and not in males (see Figure 16; Table 25).

Stress period analyses. During the stress period, sex did not significantly affect center time activity.

Post-stress period analyses. During the post-stress period, after covarying for stress values, males exhibited greater center time activity than did females (**Sex**: $F[1,55] = 13.533$, $p = 0.001$; see Figure 14; Table 30).

Internal analyses for post-stress period. When males and females were analyzed separately, exercise training decreased center time activity only in females (**Exercise:** $F[1,27] = 5.635$, $p = 0.025$; see Figure 15; Table 32) but not in males (see Figure 16; Table 32). Social housing increased center time activity only in males (**Social:** $F[1,27] = 7.829$, $p = 0.009$; see Figure 16; Table 32) but not in females (see Figure 15; Table 32). Social housing changed the pattern of center time activity during the post-stress period only for females (**Time x Social:** $F[2,54] = 3.720$, $p = 0.031$; see Figure 15; Table 33) and not males (see Figure 16; Table 33). In females that were individually housed, there were no differences in center time activity across the post-stress period, whereas in females that were socially housed, center time activity increased from the first to the second post-stress measurement and decreased from the second to the third post-stress measurement.

Open field horizontal activity (Figures 17 - 23; Tables 34 - 46). Males had lower levels of horizontal activity than did females (**Sex:** $F[1,56] = 20.816$, $p < 0.001$; see Figure 19; Table 34). The pattern of horizontal activity over time was different for females and males (**Time x Sex:** $F[4,224] = 2.689$, $p = 0.032$; see Figure 19; Table 35). In females, horizontal activity increased from the pre-stress to the stress period and remained similar across the post-stress period, whereas in males, horizontal activity increased from the pre-stress period to the stress period, decreased from the stress period to the first post-stress measurement, remained similar from the first to the second post-stress

measurement, and increased from the second to the third post-stress measurement.

When males and females were analyzed separately, exercise training decreased horizontal activity in females (**Exercise**: $F[1,28] = 10.414$, $p = 0.003$; see Figure 20; Table 36) but not in males (see Figure 21; Table 36). Social housing decreased horizontal activity in females (**Social**: $F[1,28] = 4.873$, $p = 0.036$; see Figure 22; Table 36) but not in males (see Figure 23; Table 36).

Stress period analyses. Treatment groups did not differ significantly during the stress period after covarying for pre-stress period values.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, females engaged in more horizontal activity than did males (**Sex**: $F[1,55] = 9.366$, $p = 0.003$; see Figure 19; Table 41).

Internal analyses for post-stress period. When males and females were analyzed separately, exercise training decreased horizontal activity for females (**Exercise**: $F[1,27] = 4.685$, $p = 0.039$; see Figure 20; Table 44) but not males (see Figure 21; Table 44).

Ultrasonic vocalizations – Negative affect (Figures 24 - 27; Tables 47 - 58). Females exhibited more ultrasonic vocalizations at approximately 20kHz than did males overall (**Sex**: $F[1,56] = 16.072$, $p < 0.001$; see Figure 25; Table 47). Females increased the expression of ultrasonic vocalizations at approximately 20kHz over the course of the experiment, whereas males showed no significant changes over the course of the experiment (**Time x Sex**:

$F[2.876,161.069] = 2.933$, $p = 0.037$; see Figure 25; Tables 48 - 49). This increase over time was due almost entirely to females without exercise training (**Time x Sex x Exercise**: $F[2.876,161.069] = 3.814$, $p = 0.012$; see Figure 26; Tables 48 - 49).

Stress period analyses. During the stress period, after covarying for pre-stress values, females expressed higher levels of ultrasonic vocalizations at approximately 20kHz than did males (**Sex**: $F[1,55] = 4.066$, $p = 0.049$; see Figure 25; Table 53).

Post-stress period analyses. During the post-stress period, after covarying for stress values, females expressed more ultrasonic vocalizations at approximately 20kHz than did males (**Sex**: $F[1,55] = 11.558$, $p = 0.001$; see Figure 25; Table 55).

Ultrasonic vocalizations – Positive affect (Figures 28 - 31; Tables 59 - 70). Females exhibited more ultrasonic vocalizations at approximately 50kHz than males overall (**Sex**: $F[1,56] = 40.333$, $p < 0.001$; see Figure 29; Table 59). Females increased the expression of ultrasonic vocalizations at approximately 50kHz over the course of the experiment, whereas males showed no significant changes over the course of the experiment (**Time x Sex**: $F[3.417,191.332] = 2.641$, $p = 0.043$; see Figure 29; Tables 60 - 61). This increase over time was due almost entirely to females without exercise training (**Time x Sex x Exercise**: $F[3.417,191.332] = 4.534$, $p = 0.003$; see Figure 30; Tables 60 - 61).

Stress period analyses. During the stress period, after covarying for pre-stress period values, sex did not significantly affect ultrasonic vocalizations at approximately 50kHz.

Post-stress period analyses. During the post-stress period, after covarying for stress period values, females expressed ultrasonic vocalizations at approximately 50kHz more frequently than did males (**Sex:** $F[1,55] = 35.903$, $p < 0.001$; see Figure 29; Table 67). In females, exercise training decreased expression of ultrasonic vocalizations at approximately 50kHz, whereas in males exercise training had no significant effect on ultrasonic vocalizations at approximately 50kHz (**Sex x Exercise:** $F[1,55] = 25.905$, $p < 0.001$; see Figures 30 and 31; Table 67). When males and females were analyzed separately, pair housing decreased expression of ultrasonic vocalizations at approximately 50kHz in males (**Social:** $F[1,27] = 7.043$, $p = 0.013$; see Figure 30; Table 69) but not in females (see Figure 31; Table 69).

Social interaction (Tables 74 - 95). Social interaction data were analyzed separately at each time point for the effects of sex on type or quality of social interaction.

Pre-stress analyses. Females displayed more co-aggressive behavior than expected, whereas males display more dominant/submissive behavior than expected (**Sex:** $\chi^2 [3, N = 32] = 8.029$, $p = 0.045$; Tables 66 - 67).

Stress analyses. Females displayed less passive social interaction than expected and more dominant/submissive and co-aggressive social interaction,

whereas males displayed more passive social interaction than expected and less dominant/submissive and co-aggressive social interaction (**Sex:** $\chi^2 [2, N = 32] = 9.143$, $p = 0.01$; Tables 80 - 81).

Post-stress I analyses. Females displayed more passive and co-supportive social interaction than expected and less dominant/submissive and co-aggressive social interaction, whereas males displayed less passive and co-supportive social interaction than expected and more dominant/submissive and co-aggressive social interaction (**Sex:** $\chi^2 [3, N = 32] = 8.667$, $p = 0.034$; Tables 84 - 85).

Post-stress II analyses. Sex did not significantly affect social interaction.

Post-stress III analyses. Sex did not significantly affect social interaction during the third post-stress measurement.

Forced swim test (Figure 32; Tables 96 - 106). There were no significant effects of sex on immobility in the forced swim test.

Body weight (Figures 33 - 36; Tables 107 - 120). Males weighed more than females overall (**Sex:** $F[1,55] = 19.735$, $p < 0.001$; see Figure 34; Table 107).

Stress period analyses. During the stress period and after covarying pre-stress period values, males weighed more than females (**Sex:** $F[1,55] = 10.678$, $p = 0.002$; see Figure 34; Table 113).

Post-stress period analyses. During the post-stress period, after covarying for stress period values, sex had no effect on body weight.

Food consumption (Figures 37 - 40; Tables 121 - 134). Males consumed more food than did females overall (**Sex:** $F[1,53] = 46.822$, $p < 0.001$; see Figure 39 for females and Figure 40 for males; Table 121).

Internal analyses. In females, exercise training slowed the rate of increase in food consumption over time (**Time x Exercise:** $F[3.222,80.5777] = 3.559$, $p = 0.016$; see Figure 39; Tables 125 - 126). Social housing decreased food consumption overall (**Social:** $F[1,25] = 8.659$, $p = 0.007$; see Figure 39; Table 124) and slowed the rate of increase in food consumption over time (**Time x Social:** $F[3.222,80.5777] = 3.744$, $p = 0.012$; see Figure 39; Tables 125 - 126).

In males, exercise training decreased food consumption overall (**Exercise:** $F[1,27] = 14.631$, $p = 0.001$; see Figure 40; Table 124) and slowed the rate of increase in food consumption over time (**Time x Exercise:** $F[2.545,68.703] = 3.536$, $p = 0.025$; see Figure 40; Tables 125 - 126). Social housing decreased food consumption overall (**Social:** $F[1,27] = 9.573$, $p = 0.005$; see Figure 40; Table 124) and slowed the rate of increase in food consumption over time (**Time x Social:** $F[2.545, 68.703] = 3.609$, $p = 0.023$; see Figure 40; Tables 125 - 126).

Stress period analyses. During the stress period and after covarying for pre-stress period values, males ate more than did females (**Sex:** $F[1,53] = 39.846$, $p < 0.001$; see Figure 39 for females and Figure 40 for males; Table 127). When females and males were analyzed separately, exercise training

decreased food consumption only in males (**Exercise**: $F[1,27] = 10.483$, $p = 0.003$; see Figure 40; Table 128) and had no effect on food consumption in females (see Figure 39; Table 128). In males, social housing decreased food consumption more in animals without exercise training than in animals with exercise training (**Exercise x Social**: $F[1,27] = 5.453$, $p = 0.027$; see Figure 40; Table 128) but not in females (see Figure 39; Table 128).

Post-stress period analyses. During the post-stress period, after covarying for stress period values, males ate more overall than did females (**Sex**: $F[1,55] = 37.204$, $p < 0.001$; see Figure 39 for females and Figure 40 for males; Table 129).

Summary of hypothesis IV findings. Males had higher fecal corticosterone values than did females, whereas females had higher serum corticosterone values than did males. Males exhibited greater center time ratio values than did females, whereas females exhibited greater horizontal activity values than did males. Females displayed greater ultrasonic vocalizations at both 20kHz and 50kHz than did males. Females and males displayed different patterns of social interaction at each of the time points analyzed. Males had higher body weight and food consumption values than did females. A summary of the significant main effects of sex is presented in Table E below. A summary of the significant main effects of the experiment is presented in Table F below.

Variable	Sex		
	Overall	Female (F)	Male (M)
fCort	F < M	---	---
sCort	F > M	No Ex: Soc > Soc Ex: Soc < No Soc	No Ex: Soc = No Soc Ex: Soc = No Soc
CTR	F < M	Increase over time	Greater increase over time
HZA	F > M	Increase during stress and remain elevated	Increase during stress and post-stress 3
USV 20kHz	F > M	Increase over time due to Ex	No changes over time
USV 50kHz	F > M	Increase over time due to Ex	No changes over time
FST	---	---	---
BW	F < M	---	---
FC	F < M	Ex decrease over time	Ex decrease over time

Table E. Hypothesis IV significant effects. Ex = exercise; Soc = social housing;

fCort = fecal corticosterone; sCort = serum corticosterone; CTR = center time

activity; HZA = horizontal activity; USV = ultrasonic vocalizations; FST = forced

swim test; BW = body weight; FC = food consumption.

Variable	Exercise (Ex)			Social Housing (Soc)			Sex
	Overall	Female	Male	Overall	Female	Male	
fCort	---	---	---	---	---	---	F < M
sCort	---	---	---	No Soc < Soc	---	---	F > M
CTR	No Ex > Ex	No Ex > Ex	No Ex = Ex	No Soc < Soc	---	---	F < M
HZA	No Ex > Ex	No Ex > Ex	No Ex = Ex	No Soc > Soc	No Soc > Soc	No Soc = Soc	F > M
NA	No Ex > Ex	No Ex >> Ex	No Ex > Ex	--	---	---	F > M
PA	No Ex > Ex	No Ex >> Ex	No Ex > Ex	---	---	---	F > M
FST	---	---	---	---	---	---	---
BW	No Ex > Ex	No Ex = Ex	No Ex > Ex	---	---	---	F < M
FC	No Ex > Ex	No Ex = Ex	No Ex > Ex	No Soc > Soc	---	---	F < M

Table F. Summary of significant main effects. F = females; M = males; fCort = fecal corticosterone; sCort = serum corticosterone; CTR = center time activity; HZA = horizontal activity; USV = ultrasonic vocalizations; FST = forced swim test; BW = body weight; FC = food consumption.

Hypotheses Revisited

This doctoral dissertation project had four major hypotheses.

1. Combined sleep disturbance and predator stress will have significant effects on biological and psychological measures in both female and male rats.

(a) Combined sleep disturbance and predator stress will decrease center time in the open field chambers, body weight, and food consumption. **Not supported.** Instead, center time activity increased during the post-stress period, suggesting decreased (rather than increased) anxiety. There were no effects of stress on body weight and food consumption.

(b) Combined sleep disturbance and predator stress will increase fecal corticosterone levels, ultrasonic vocalizations (USV) at lower frequencies, and forced swim immobility. **Partially supported.** Stress increased fecal corticosterone. Forced swim immobility increased during the post-stress period. Low frequency USV increased in females over time, but only in the no exercise group.

2. Exercise training will attenuate stress' effects on biological and psychological measures. **Partially supported.** Exercise training decreased low frequency USV, especially in females. In contrast, exercise training decreased center time activity and high frequency USV.

3. Social housing will attenuate stress' effects on biological and psychological measures. **Partially supported.** Social housing increased center time activity. In contrast, social housing increased serum corticosterone.

4. Stress responses to sleep disturbance and predator stress will differ between females and males. **Mostly supported.** Females and males differed on fecal corticosterone, serum corticosterone, center time activity, horizontal activity, low and high frequency ultrasonic vocalizations, body weight, and food consumption. Exercise decreased center time activity and horizontal activity in females but not in males; decreased USV in females more than in males; decreased body weight and food consumption in males but not in females. Social housing decreased center time activity during the post-stress period in males but not in females.

Discussion

The purpose of this doctoral dissertation research project was to use a rodent model to determine if exercise training or social enrichment could enhance stress resilience. Resilience is the return to baseline functioning after encountering an adverse event (Luthar, 1991; Luthar & Cicchetti, 2000; Masten, 2001; Rutter, 1993). Overall, the findings suggest that exercise training is particularly useful to enhance stress resilience in female rats, whereas social housing is particularly useful to enhance stress resilience in male rats.

There were several findings that are most relevant to the overall conclusion. For female rats, exercise training reduced low frequency ultrasonic vocalizations (USV) suggesting decreased negative affect, reduced stress responses, or enhanced stress resistance and resilience. However, exercise training also reduced female rats' high frequency USV suggesting decreased positive affect or decreased communication. For male rats, social housing increased center time (suggesting decreased anxiety or greater stress resistance or resilience) during the post-stress period but did not have this effect on female rats. Other effects of stress, exercise, and housing conditions were found and are summarized in the Results and Hypothesis Revisited sections, but do not address the overarching question of which independent variables affected stress resilience.

Consistent with existing literature, sleep disturbance and predator stress increased fecal corticosterone levels (Apfelbach et al., 2005; Berger, 2009; Cohen et al., 2008; Perry, 2009; Rabat, 2007; Rabat et al., 2004, 2005, 2006;

Takahashi et al., 2005; Zoladz et al., 2008). This combined stressor, designed to be analogous to conditions service members experience during combat deployments, is effective at producing stress responses within a laboratory setting.

Fecal and serum corticosterone, although intended to be measuring the same biological stress response, had a sex difference in the opposite direction. Males had higher fecal corticosterone levels than did females, and females had higher serum corticosterone levels than did males. The serum corticosterone findings are consistent with existing literature (e.g., Berger, 2009; Faraday, 2000; Doremus-Fitzwater, Varlinskaya, & Spear, 2009). It is unclear why the direction of the sex difference appeared to have been reversed for fecal corticosterone. A recent study by Thanos and colleagues (2009) reported substantial circadian variations in fecal and serum corticosterone values that follow different time courses. The fecal sampling in the present research project occurred in 1 to 7 hours after the beginning of the active phase when fecal corticosterone values are relatively stable. The serum corticosterone sampling occurred within the same window of time but when there can be substantial within-subject variations. Therefore, future research should more tightly control times of sampling or could collect samples over the course of 24 hours to determine whether and when there are sex differences in corticosterone values.

The effects of exercise training on different aspects of the stress response were largely consistent with previous literature. Partially consistent with existing literature in humans, exercise decreases negative affect in females (Abrantes,

Strong, Cohn, Cameron, Greenberg, Mancebo, et al., 2009; Stathopoulou, Powers, Berry, Smits, & Otto, 2006). No literature is currently available on the effects of exercise on ultrasonic vocalizations in rodents. Current literature indicates that exercise decreases anxiety and depressive symptoms, but sex differences have not yet been investigated. Fully consistent with existing literature, exercise decreased body weight gain and food consumption in males (Katch, Martin, & Martin, 1979; Reith & Larue-Achagiotis, 1997).

Consistent with previous literature, social housing decreased anxiety-like behavior and food consumption (Benarova-Milshtein et al., 2004; Chapillon et al., 1999; Elliott & Grunberg, 2005; Friske & Gammie, 2005; Schrijver et al., 2002; Tomchesson, 2004, 2006; Zimmermann et al., 2001). Also consistent with previous literature was the finding that there exist sex differences in the stress response (Baker et al., 2006; Consoli et al., 2005; Dalla et al., 2005; Paris et al., 2010).

With regard to the examination of sex differences, it is relevant to consider that sex hormones may alter the stress response of females (Kajantie & Phillips, 2006). Because of this effect, measurement of the estrous cycle in females was carefully considered as a dependent variable in the present research. This measurement was rejected, however, because the process of measuring estrous is considered inherently stressful to the rat. An additional stressor that occurred throughout the study would have confounded the results. Further, no comparable procedure exists for males, and any sex differences founds would have been confounded by handling differences related to this measurement.

Given these considerations, this measurement was rejected as being appropriate to this doctoral dissertation project. Future studies of stress resilience that examine sex differences should consider this measurement as an option.

Interpretation of results

The present findings can be interpreted in the context of four different conceptual models of the stress response (see Figure B). Model I derives from Cannon's line of thinking that stress causes a disruption in homeostasis and the body attempts to regain homeostasis. The rise in physiological activity with stress followed by a gradual return to pre-stress levels also is consistent with the work of Selye and Mason (summarized in the Introduction section). Model II is quite different and follows Glass and Singer's findings that the individual is able to withstand stress during the stressor itself, but that the individual suffers the disruption of functioning post-stressor when the threat to the individual no longer exists (the so-called "after-effect" of stress). Model III is most consistent with stress resilience as disruption occurs during the stressor but the individual is able to rapidly return to pre-stress levels. Model IV captures the phenomenon of thriving or post-traumatic growth as the individual is disrupted during the stressor but "over-recovers" or improves in function following cessation of the stressor. It is noteworthy that the biological overshoot accompanying post-traumatic "growth" either could reflect an improvement in function or it could result from an altered biological system that responds differently following stress experiences.

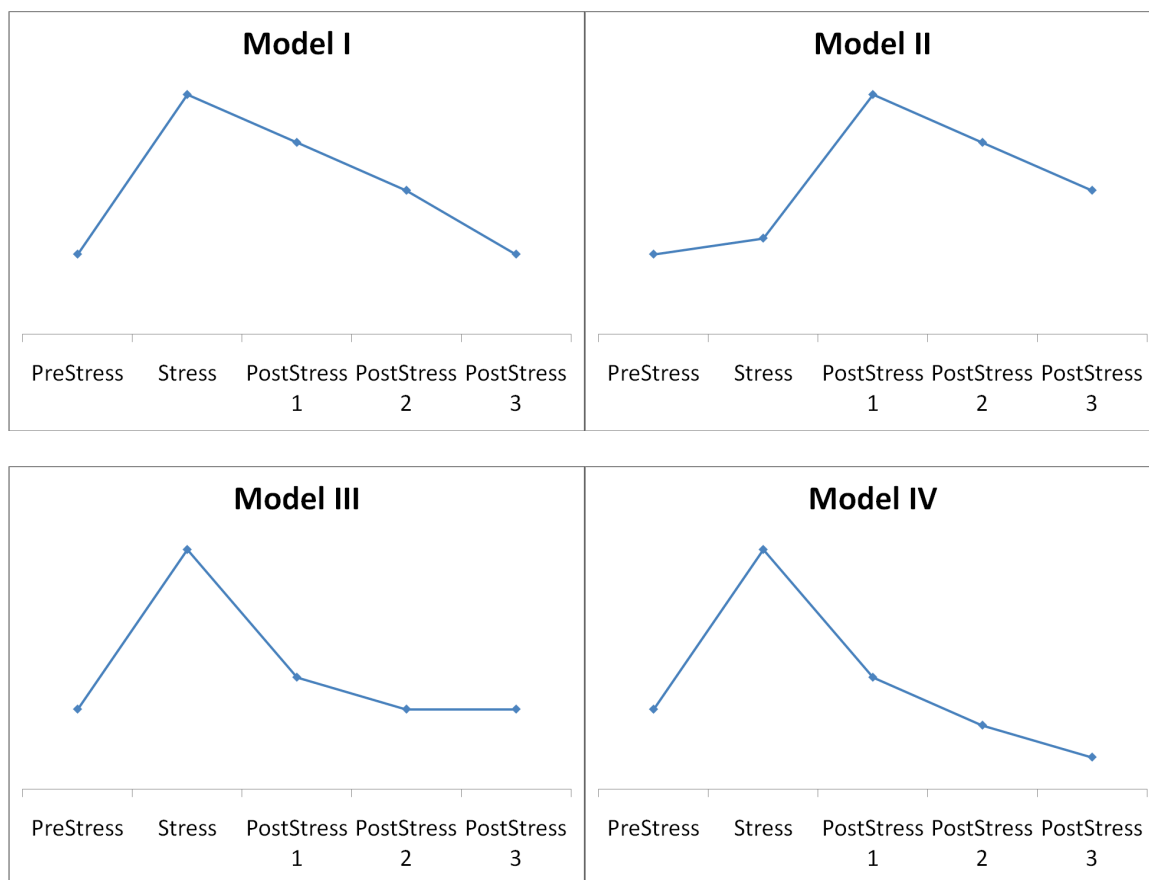


Figure B. Idealized graphs of stress exposure on functioning over time. Model I: Stress disruption followed by post-stress gradual return (based on Cannon). Model II: Little or no stress disruption followed by post-stress disruption (based on Glass & Singer). Model III: Stress disruption followed by post-stress rapid return (resilience). Model IV: Stress disruption followed by post-stress improvement (thriving or post-traumatic growth).

None of the findings of this project support Model I, Cannon's homeostasis. It appears that a combined chronic and intermittent stressor may not produce results consistent with Cannon's homeostasis. Homeostasis may be more applicable instead to an acute stressor.

A couple of findings, however, are consistent with Model II, Glass and Singer's after-effects phenomenon. Food consumption most clearly follows this pattern. Regardless of intervention and sex, food consumption increased following stress, before decreasing again at the end of the experiment. The decrease that occurred at the end of the experiment indicates the increase was not solely time-related (i.e., rats were eating more because they were still growing in adulthood), but rather resembles an after-effects phenomenon.

Negative affect in females also resembles Model II, the after-effects phenomenon. Unlike with food consumption, negative affect does not uniformly follow this pattern. Rather, it appears only in females and this pattern is changed by exercise. Based on these results, it appears that the combined stressor used in this experiment produces some after-effects in a limited number of stress-related variables, with more robust effects in food consumption than in negative affect.

Some findings are consistent with Model III, resilience, primarily in the fecal corticosterone findings. Overall, the combined sleep disturbance and predator stress increased fecal corticosterone levels during the stressor and the fecal corticosterone levels rapidly returned to normal. This phenomenon occurred overall, and particularly in the males and social enrichment groups. In addition to fecal corticosterone, exercise in the female rats appeared to lead to resilience in negative affect. This effect was not as robust as that of fecal corticosterone, but it appears that exercise may increase stress resilience in females in at least this one stress-related variable.

None of the findings of this project support Model IV, post-traumatic growth/thriving. One dependent variable, positive affect, appeared to increase post-stress in the females that did not receive exercise training. There did not, however, exist a disruption during the stressor which is part of the profile of post-traumatic growth. The stressor may not have been intense enough to cause a disruption, but perhaps acted as a stress-inoculator for positive affect in this particular group.

To summarize, several findings of this project are consistent with Glass and Singer's after-effects phenomenon (Model II) and with resilience (Model III). It appears that the combined sleep disturbance and predator stressor was effective to produce a stress response and that some interventions were effective in reducing the stress response. **For females, exercise appeared to counteract the after-effects of stress** in terms of behavioral indicates suggestive of negative affect. **For males, social enrichment appeared to enhance stress resilience** in terms of fecal corticosterone. Based on the center time activity and fecal corticosterone findings, males appear to be more stress-resistant than do females. The center time activity findings indicate that the males are less anxious and the fecal corticosterone findings indicate that the males display more stress resilience than females.

Limitations

This doctoral dissertation project had two unanticipated limitations that are relevant to interpreting the results: (1) the use of a repeated-measures within-

subject design rather than a mixed design that also included stress as a between-subjects variable; and (2) the visualization difficulty of measuring movement in pigmented Long-Evans rats in the forced swim test.

Repeated-measures within-subject design. All animals in this experiment were exposed to stress. This element of the experimental design was important to determine if stress resilience changed over time. The definition of resilience is a process that occurs after exposure to a stressor by which an individual returns to their pre-stressor functioning (Luthar, 1991; Luthar & Cicchetti, 2000; Masten, 2001; Rutter, 1993). Resilience is conditional upon exposure to a stressor. Stress resilience, therefore, could not be determined without exposure to a stressor.

An ABA within-subject design would have been appropriate if hypotheses were confirmed as expected. If the stressor had interrupted the subjects' functioning followed by a gradual return to baseline and the two interventions increased the subjects' stress resilience indicated by a rapid return to baseline, then a no-stress between-subjects treatment group would not have been necessary to demonstrate stress resilience. Rather, it was more ethical and economical given these expectations not to include a no-stress control group to reduce the number of animals in this experiment to the minimum number required. Additionally, reducing the number of subjects also was more logistically feasible, given the cost of animals and their *per diem*, as well as in terms of planning and personnel. Given these findings, however, a no-stress control group would have aided in interpretation of how stress affected the treatment

groups over time. This particular type of control group would have allowed for a comparison of stressed and non-stressed animals to determine how their stress responses changed over time.

Forced swim test. The forced swim test was originally developed by Porsolt and was based on Seligman's model of learned helplessness. Immobility in the forced swim test is often understood to be an index of depression, with greater immobility interpreted as greater depression. Although the forced swim test has been used extensively in the literature, there are some indications that the test in itself is a stressor. Interpretations of the forced swim test results, therefore, must take into consideration whether or not the test is stressful. This procedure has been used successfully in the Grunberg laboratory to detect differences in stress responses with both Sprague Dawley and Long-Evans rats (Berger, 2009; Perry, 2009). In this experiment, however, the results indicated that there were no significant differences between the treatment groups. The cameras used during the forced swim test had difficulty detecting the Long Evans rats because of their black and white coloring (versus the all white coat of Sprague Dawley and other albino rats). This difficulty resulted in inaccurate recordings of the forced swim test and greater variance due to measurement error than would be expected. The variance was so great that no differences were detected between the treatment groups. In retrospect, the investigator realized that piloting was done with albino rats and that light adjustments should have been made to more accurately detect swim behavior in the black and white Long-Evans rats used in this experiment. Placing a light on the ground near the

forced swim cylinders provides illumination underneath the rats. This light provides enough of a contrast between the Long-Evans rats and the water for the cameras to detect movement (or immobility) of the Long-Evans rats. This step was not taken in this experiment and likely caused the great amount of variance that contributed to the lack of significant differences between treatment groups.

Clinical Implications

Human research on the effects of exercise training and social environment on stress resilience would be necessary for informing clinical practice in preventing and treating stress-related conditions. If, however, the present findings generalize to the human condition, the clinical implications of this research are that exercise and social interventions may hold value for increasing stress resilience. **Exercise training appeared to be of value for females to reduce their negative affect and to reduce anxiety.** Whereas females appeared to increase expression of negative affect overall post stress, females receiving exercise training returned to their pre-stress levels at the first measurement following stress exposure. Exercise overall appeared to reduce anxiety in females. **Social enrichment appeared to be of value for males in returning to pre-stress levels of fecal corticosterone and reducing anxiety, particularly post-stress.**

Military Implications

If the present findings generalize to the human condition, then these findings also may have implications for the military. The military currently requires all service members to pass a physical fitness test. Maintaining or even heightening requirements for physical fitness tests may lead to increased exercise training, which may be particularly beneficial for females. Allowing time for exercise during the workday and other increased opportunities to exercise when feasible also may serve the interests of the military in terms of its members' mental health. Perhaps increasing group exercise opportunities will tap into both exercise and social interventions for increasing stress resilience. In terms of the social environment, maintaining housing conditions where individuals are paired with a roommate (which often occurs in military barracks) may be beneficial for enhancing stress resilience in males.

Future Directions

Future directions may either address study limitations or build on the study findings. The study limitations may be addressed through several avenues. Adding a no-stress control group may help further understanding of how this particular stressor affected the stress responses of Long-Evans rats.

Although the forced swim test has been used extensively in the literature to address the learned helplessness aspect of depression, another test may avoid some of the methodological limitations (i.e., stressful experience, cameras picking up on animals with dark fur): for example the sucrose intake/preference

test. The sucrose intake/preference test appears to tap into another aspect of depression: anhedonia (Baker et al., 2006; Grippo, Moffitt, & Johnson, 2008; Konkle et al., 2003; McCormick et al., 2009; Rygula et al., 2008). The sucrose intake/intake preference test also has a considerable research basis and may be better suited as an index of depression, particularly with Long-Evans rats. This measure was not used in this doctoral dissertation for two reasons: (1) the equipment was not available for use within the laboratory, and (2) the researcher had no prior experience with conducting this measure.

Rat studies. Future directions within animal research include expanding subject characteristics and methodological aspects. In terms of subjects, considering other rat strains and different rat ages would enhance understanding of stress resilience in rats. In terms of methodology, in addition to the changes suggested above, additional biological measures may also improve understanding of stress resilience. Other biological measures that have been suggested as being related to stress resilience include DHEA, neuropeptide Y, testosterone, estradiol, oxytocin, and vasopressin. These measures are described in more detail below.

Certain biological substrates, neurosteroids, and peptides may help combat the negative effects of stress on physiological functioning. In particular, DHEA and neuropeptide Y may help the body to resume normal functioning (Charney, 2004). DHEA appears to counteract the negative effects of cortisol in the hippocampus, and also appears to increase positive affect. Neuropeptide Y is an anxiolytic and impairs fear memory. Greater levels of DHEA and

neuropeptide Y in response to stress are associated with decreased stress responsivity (Charney, 2004) and therefore may be important for stress resilience.

Estrogen is another biological substrate that influences the stress response and may help the body resume normal functioning. However, the relationship between estrogen and the stress response is unclear and is related to gender (Charney, 2004). Females consistently show greater physiological responses to both acute and chronic stressors, which many investigators have attributed to sex hormone differences (Charney, 2004). The amount of estradiol appears to be a key factor in how females respond physiologically to stressors, where relatively low doses of estradiol may be more beneficial than relatively high doses of estradiol (Charney, 2004). Measuring estradiol, therefore, may assist researchers in further understanding biological sex differences in stress responses and perhaps stress resilience.

Oxytocin and vasopressin are two neuropeptides that may indirectly influence physiological responses to stress through their actions on social behavior. Oxytocin and vasopressin are released from the posterior pituitary gland and circulate in the bloodstream (Guyton & Hall, 2000). In social animals, oxytocin and vasopressin increase social interaction and activate the dopamine reward circuit in the brain (Charney, 2004). To date, it appears that no researchers have experimentally examined this relationship. Several authors (Charney, 2004; Taylor et al., 2000), however, have suggested that this relationship is important for stress resilience. In addition, oxytocin appears to

have anxiolytic properties which are stronger in females than in males (Charney, 2004). Greater levels of oxytocin and vasopressin are associated with decreased stress responsiveness and may be important for stress resilience.

Although DHEA, neuropeptide Y, estrogen, oxytocin, and vasopressin have been linked to stress resilience, the exact nature of these linkages remains unclear (Bonne et al., 2004; Charney, 2004; Southwick et al., 2005). It is also unclear how these physiological components of the stress response are related to psychological concomitants of the stress response, such as anxiety and depression. Altering an individual's physiological stress response through one of these biochemicals may then better prepare that individual to cope with the stressor, which may then enhance resilience.

Another important future direction for stress resilience research is to examine how various stressors may be related to resilience. This experiment used a combined chronic and repeated acute stressor. Future experiments may vary the stressor type (i.e., acute, chronic, intermittent), intensity (i.e., mild, moderate, severe, extreme), frequency (i.e., hourly, daily), and duration (i.e., one day, one week, one month). As more research on stress resilience is conducted in animals, the impact of the temporal pattern of stress exposure will be better understood and knowledge regarding this element can be applied to humans.

Exploring more fully how exercise training and social enrichment may increase resilience also would add to understanding stress resilience. Varying the type, intensity, frequency, and duration of exercise training would allow researchers to have a better understanding of how much exercise is optimal for

increasing stress resilience. Varying the type and amount of social enrichment would allow researchers to have a better understanding of how much social interaction is optimal for increasing stress resilience

Human studies. The human research on stress resilience is limited in that it is primarily correlational. The use of experiments or quasi-experiments will help to elucidate our understanding of human stress resilience. If the animal findings generalize to humans, then one area in which human studies will be particularly helpful is using exercise as an intervention for increasing stress resilience in females. Exercise training appeared to increase stress resilience for females in terms of their negative affect. A human study in which females are assigned to either an exercise training group or a non-exercise control group and their stress responses (e.g., emotions) measured after exposure to a stressor would be valuable to explore how exercise training may increase stress resilience in human females. Social housing appeared to increase stress resilience for males in terms of their biological stress response, negative affect, and anxiety. A human study in which males were assigned to either live alone or with roommates would be valuable in exploring how social environments may increase stress resilience in human males. Conducting qualitative field research in a deployed setting on the processes of affiliation, cohesiveness, and social support in military members could also provide valuable insight into the role of the social environment on stress resilience.

Conclusions

The rat model used in this experiment was valuable to study stress resilience. One of the major findings of this project was the number of sex differences in response to stress and during the post-stress recovery period. Females and males responded differently to the same combined sleep disturbance and predator stressor. Additionally, females and males appeared to respond differently to the exercise training and social enrichment interventions. Based on the negative affect ultrasonic vocalization results, exercise appeared to increase stress resilience in females. Based on the fecal corticosterone and anxiety index (center time activity), social enrichment appeared to increase stress resilience in males. These findings suggest that it may be worthwhile to pursue exercise as a means to increase stress resilience in females. These findings also suggest that it may be worthwhile to try different types of social enrichment to increase stress resilience in males. If these findings generalize to humans, then it may be possible to increase stress resilience and prevent or limit the negative sequelae that result from exposure to stressful events. Stress, however, is a complex phenomenon that is affected by environmental, social, psychological, and biological factors. Therefore, the present findings are a beginning to identify optimal strategies to enhance stress resilience in males and females. Additional investigations need to consider the wide variety of factors that may be used in men and women to help cope with stress, to increase stress resistance, and to enhance stress resilience.

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Appendix A – Experiment Timeline

Phase	Exp. Day	Age	Procedure	DVs
Baseline	1	50	Arrival, group assignment, FC	FC
	2	51	Gentling, BW, FC, HCA	BW, FC, HCA
	3	52	Gentling	
	4	53	Gentling	
	5	54	Open field acclimation	
	6	55	Baseline open field, fCort	OF, fCort
	7	56	Exercise training, USV	USV
	8	57	Exercise training, FC	FC
	9	58	Exercise training, BW, FC, HCA	BW, FC, HCA
	10	59		
	11	60	Exercise training, FST	FST
	12	61	Exercise training, FST	FST
	13	62		
	14	63	Exercise training	
Stress	15	64	Stress, Exercise training, FC	FC
	16	65	Stress, Exercise training, BW, FC, HCA	BW, FC, HCA
	17	66	Stress	
	18	67	Stress, Exercise training	
	19	68	Stress, Exercise training	
	20	69	Stress, OF, fCort	OF, fCort
	21	70	Stress, Exercise training, USV	USV
	22	71	Stress, Exercise training, FC	FC
	23	72	Stress, Exercise training, BW, FC, HCA	BW, FC, HCA
	24	73	Stress	
	25	74	Stress, Exercise training	
	26	75	Stress, Exercise training, FST	FST
	27	76	Stress	
	28	77	Stress, Exercise training	
Post-Stress	29	78	Exercise training, FC	FC
	30	79	Exercise training, BW, FC, HCA	BW, FC, HCA
	31	80		
	32	81	Exercise training	
	33	82	Exercise training, FST	FST
	34	83	OF, fCort	OF, fCort
	35	84	Exercise training, USV	USV
	36	85	Exercise training, FC	FC
	37	86	Exercise training, BW, FC, HCA	BW, FC, HCA
	38	87		
	39	88	Exercise training	

Phase	Exp. Day	Age	Procedure	DVs
Post-Stress (continued)	40	89	Exercise training, FST	FST
	41	90	OF, fCort	OF, fCort
	42	91	Exercise training	USV
	43	92	Exercise training, FC	FC
	44	93	Exercise training, BW, FC, HCA	BW, FC, HCA
	45	94		
	46	95	Exercise training	
	47	96	Exercise training, FST	FST
	48	97	OF, fCort	OF, fCort
	49	98	Exercise training, USV	USV
	50	99	Exercise training, FC	FC
	51	100	Exercise training, BW, FC, HCA	BW, FC, HCA
	52	101		
	53	102	Exercise training	
	54	103	Exercise training	
	55	104		
	56	105	Sac	sCort

BW = Body weight

FC = Food consumption

fCort = Fecal corticosterone

FST = Forced swim test

HCA = Home cage activity

OF = Open field

sCort = Serum corticosterone

USV = Ultrasonic vocalization

Appendix B – Pictures

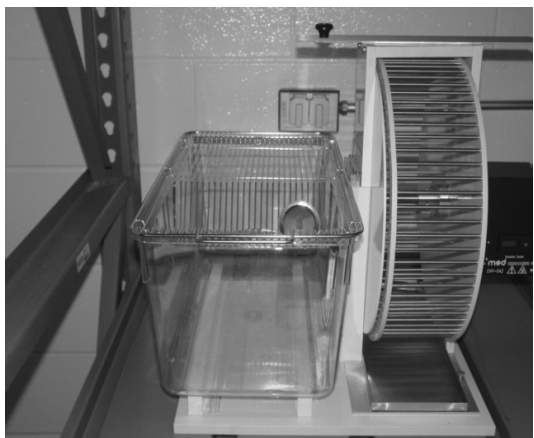


Figure 1. Exercise training wheel

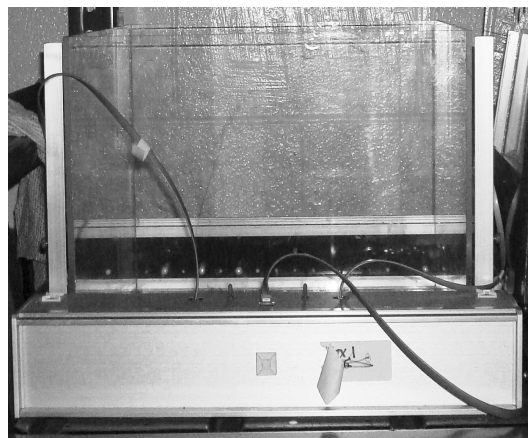


Figure 2. Open field locomotor chamber

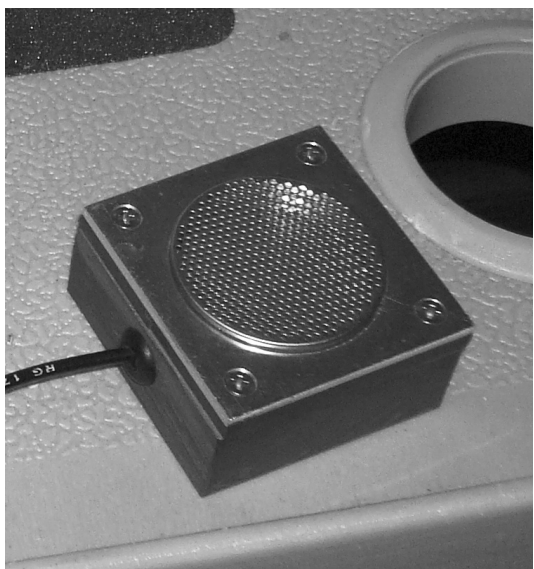


Figure 3. Ultrasonic vocalization detector



Figure 4. Forced swim test cylinders

Appendix C – Home Cage Activity and Social Interaction Rating Sheet

Directions: Complete Parts A and B for each condition TWO times.

Time 1 (first 30 sec interval)

A. Level of Activity

1 2 3 4 5 6 7

None Some low Cnst low Some mod Cnst mod Some high Cnst high

Enter subject # and activity rating for each subject in the group. Rating below should correspond to arrangement on the housing rack.

For example: (Subject) # 404: (Rating) 4.

# _____ :	# _____ :	# _____ :	# _____ :
# _____ :	# _____ :	# _____ :	# _____ :

B. Record the number of subjects **in this condition** that are engaged in the following behaviors at the end of the observation period.

Eating	Grooming	Awake/not moving	Moving HZ	Rearing	Sleeping

Time 2 (second 30 sec interval)

A. Level of Activity

# _____ :	# _____ :	# _____ :	# _____ :
# _____ :	# _____ :	# _____ :	# _____ :

B. Type of Activity

Eating	Grooming	Awake/not moving	Moving HZ	Rearing	Sleeping

C. Social Interaction

Rate the type of social interaction for the pair housed subjects:

Passive	Dominant/ Submissive	Aggressive/ Aggressive	Supportive/ Supportive

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Figure 40. The effects of exercise training and social housing on food consumption in young adult, male, Long-Evans rats.

Figures

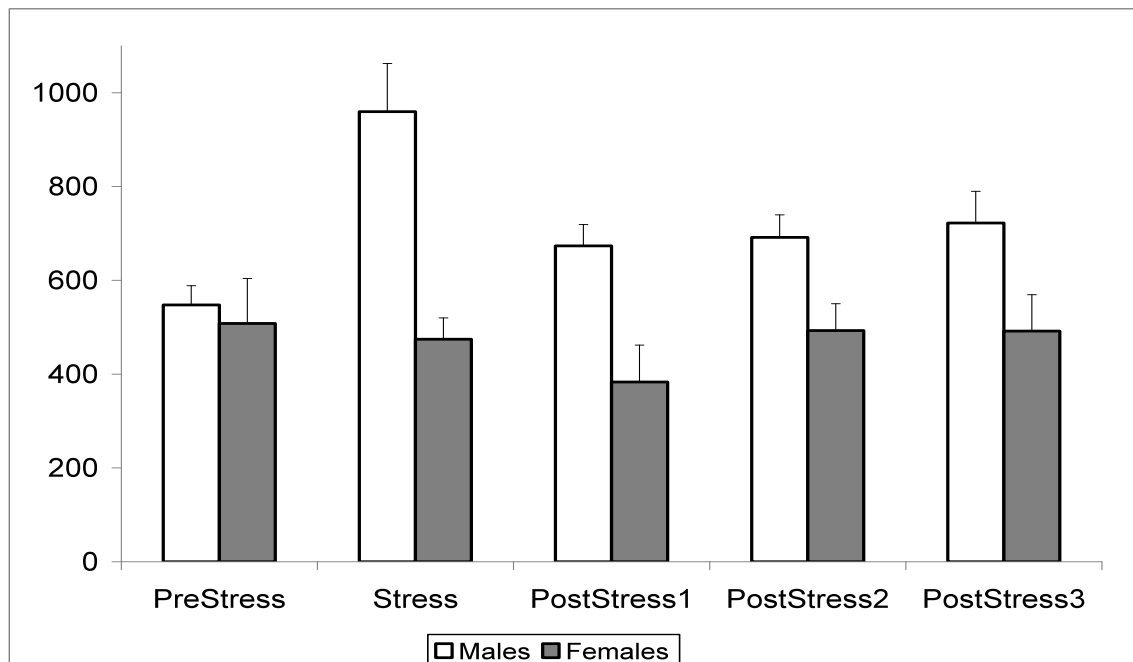


Figure 1. The effects of sex on fecal corticosterone over time in young adult, Long-Evans rats.

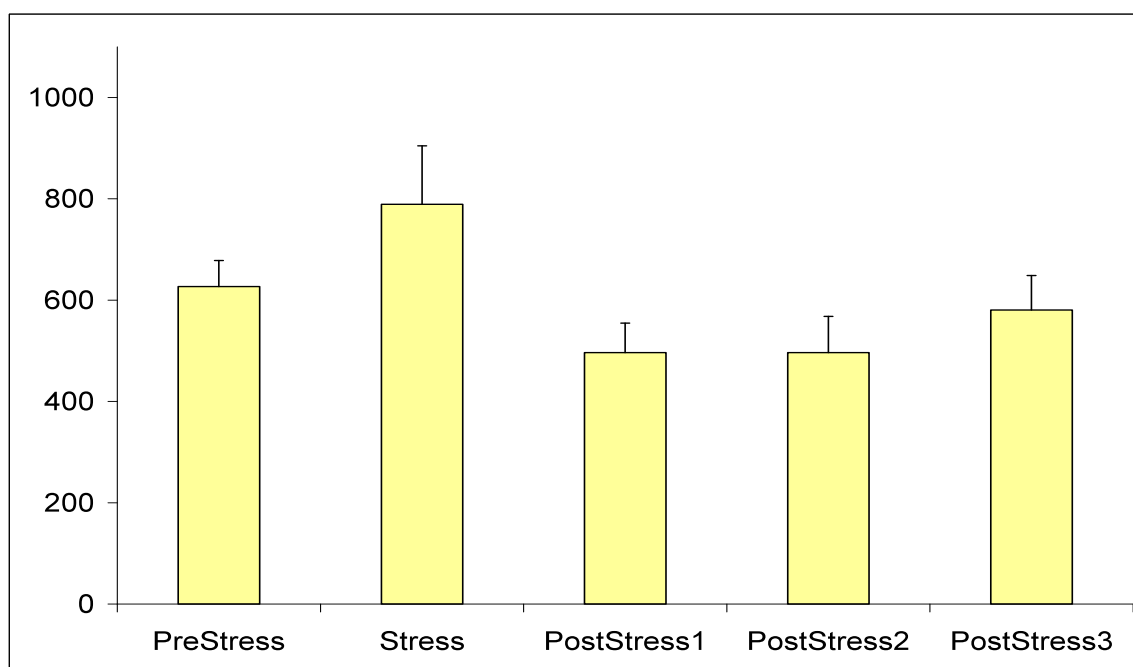


Figure 2. Fecal corticosterone values over time in young adult, Long-Evans rats.

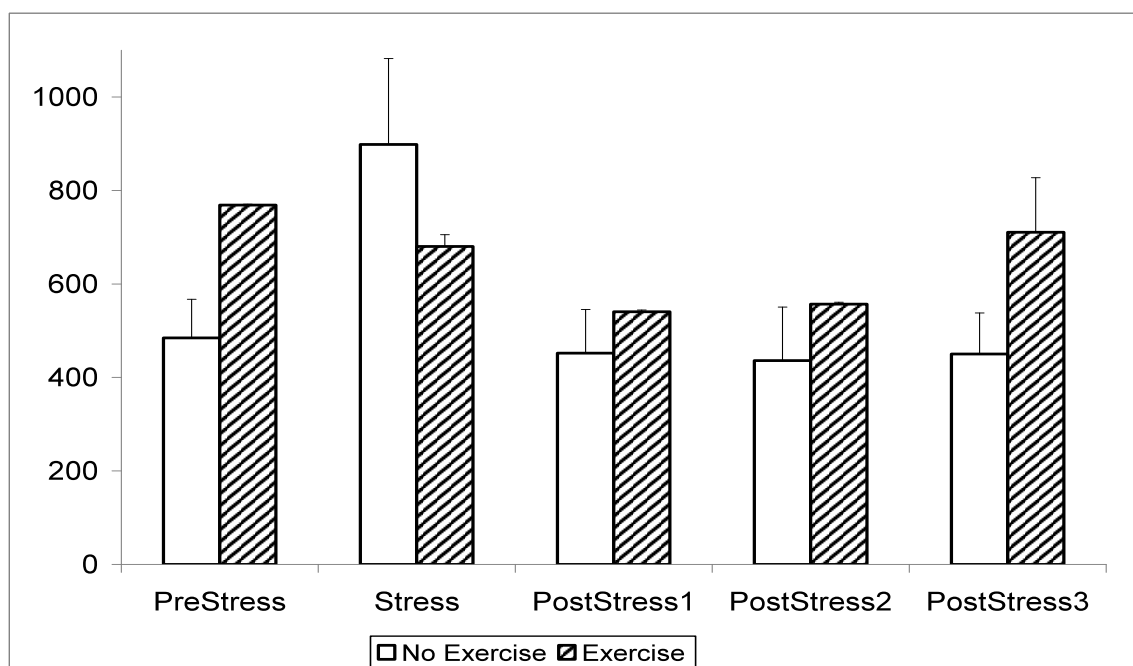


Figure 3. The effects of exercise on fecal corticosterone over time in young adult, Long-Evans rats.

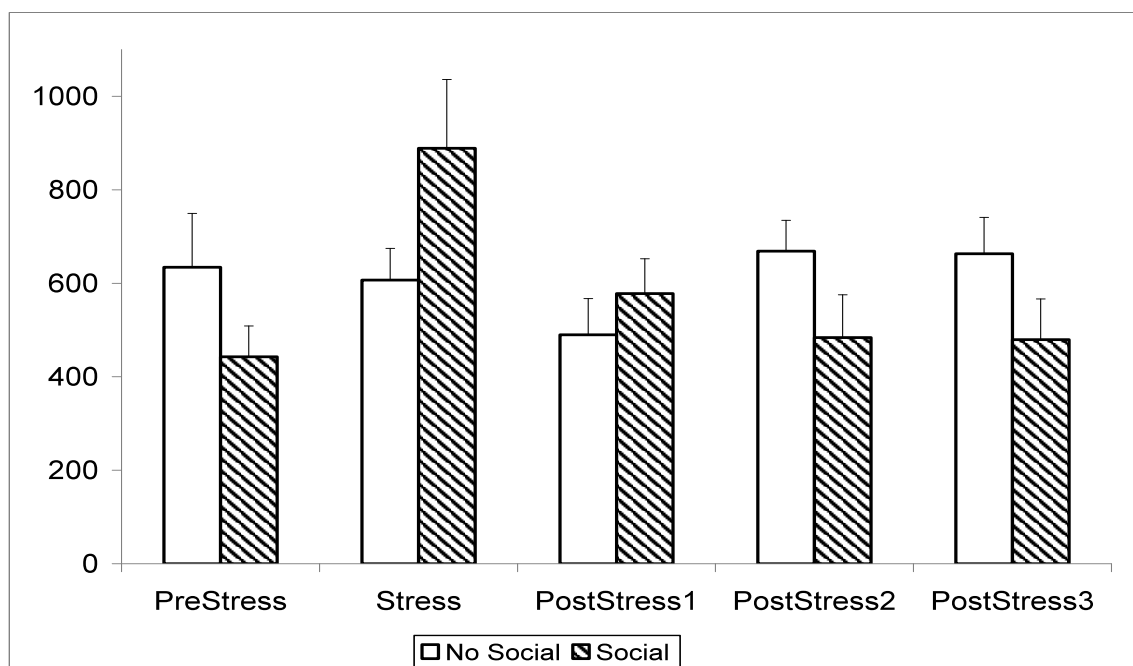


Figure 4. The effects of social housing on fecal corticosterone over time in young adult, Long-Evans rats.

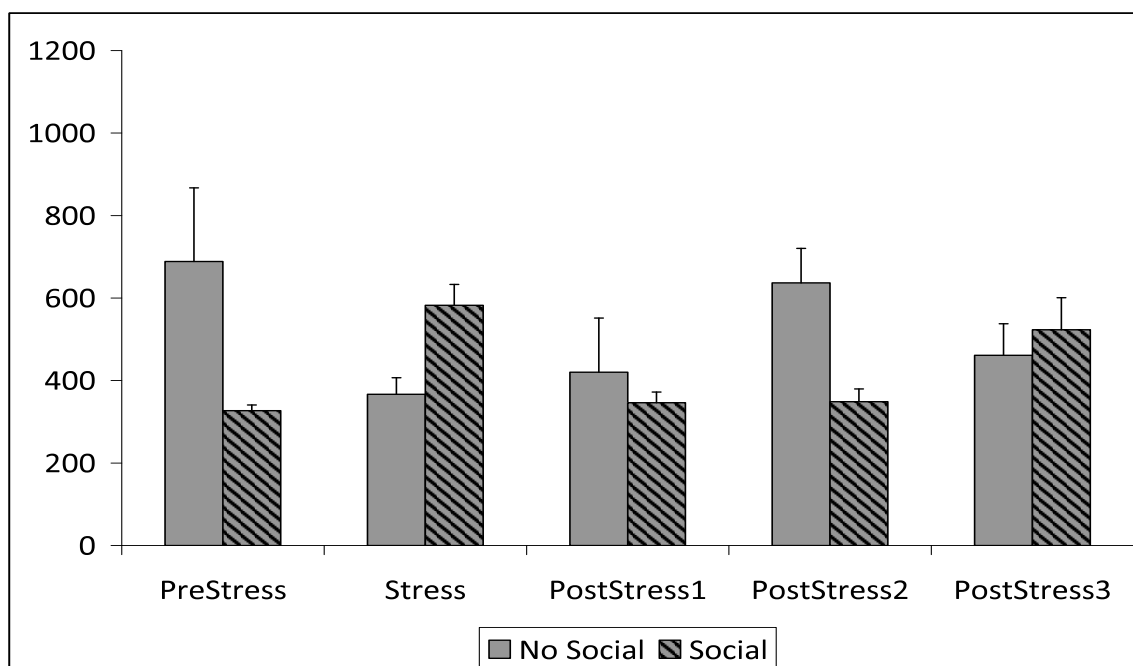


Figure 5. The effects of social housing on fecal corticosterone in young adult, female, Long-Evans rats

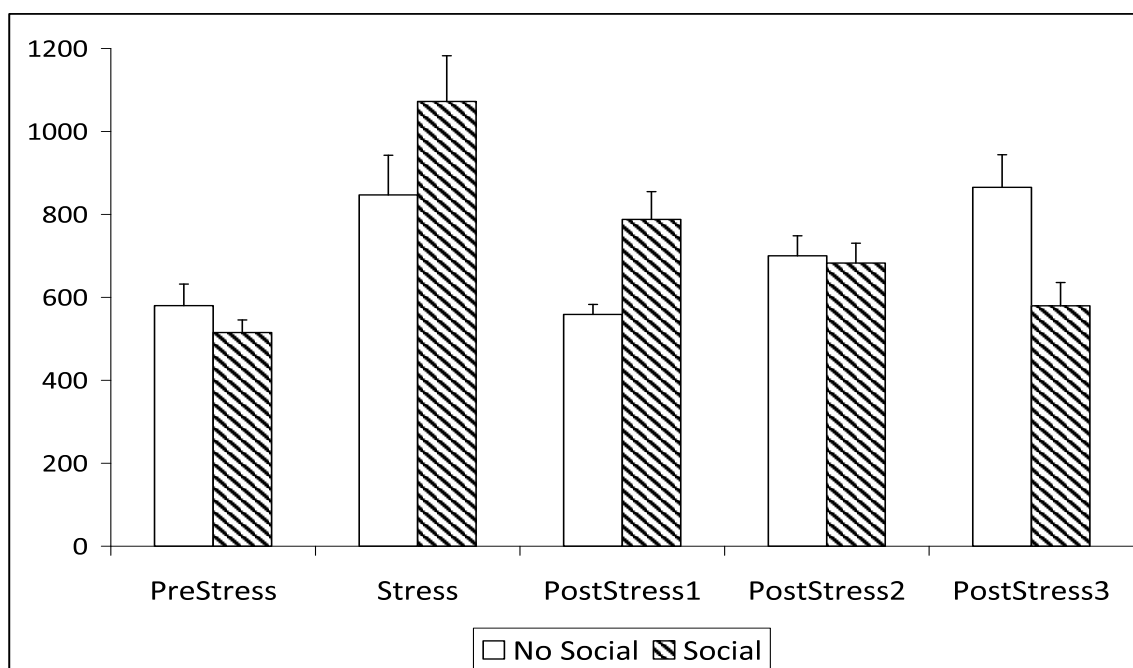


Figure 6. The effects of social housing on fecal corticosterone in young adult, male, Long-Evans rats.

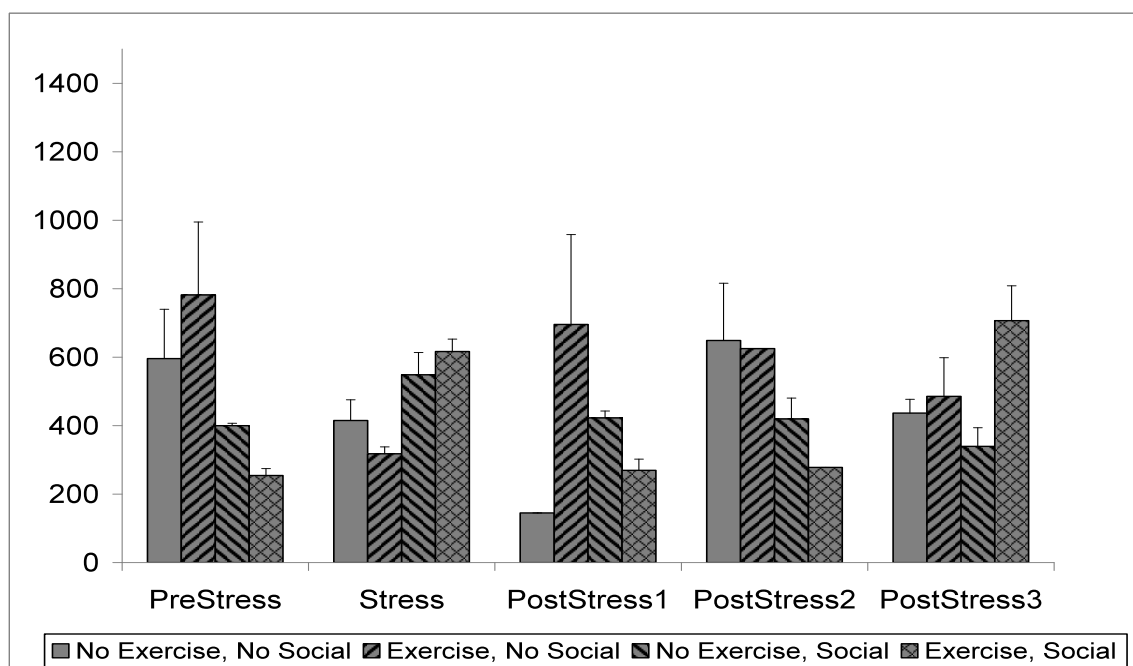


Figure 7. The effects of exercise training and social housing on fecal corticosterone in young adult, female, Long-Evans rats.

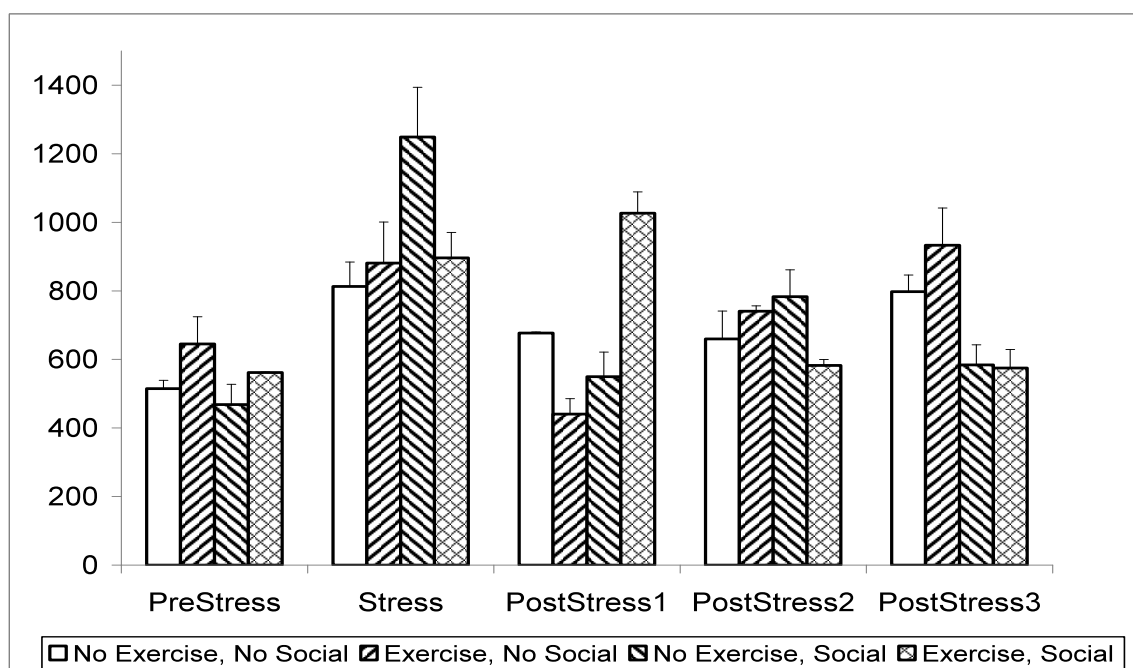


Figure 8. The effects of exercise training and social housing on fecal corticosterone in young adult, male, Long-Evans rats.

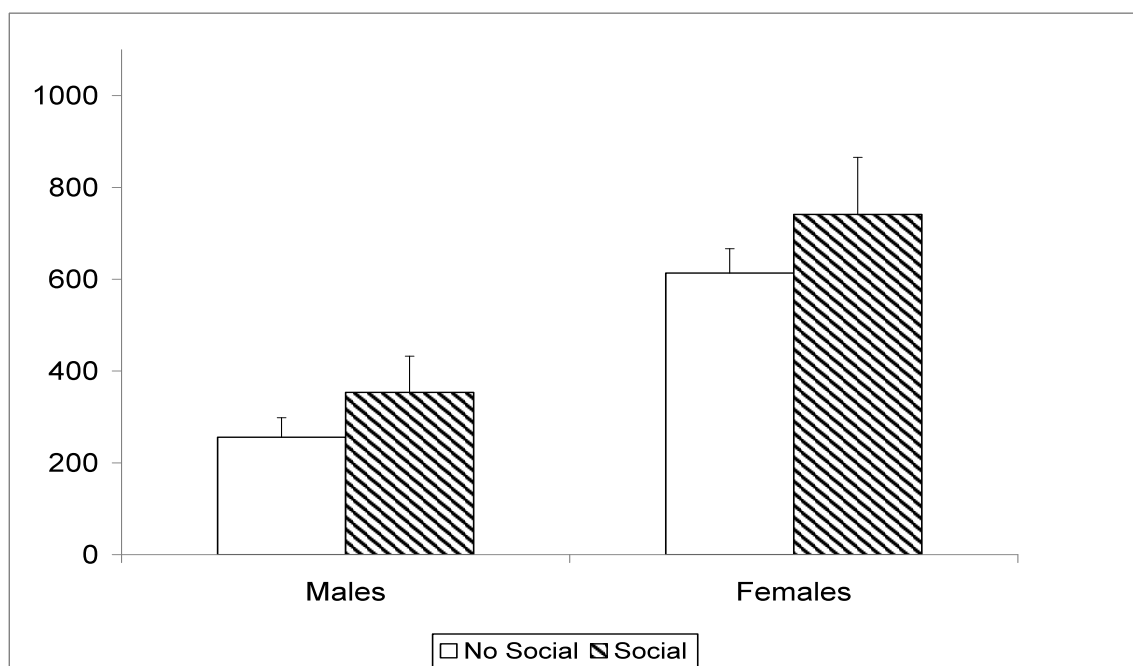


Figure 9. The effects of social housing on serum corticosterone in male and female, young adult, Long-Evans rats.

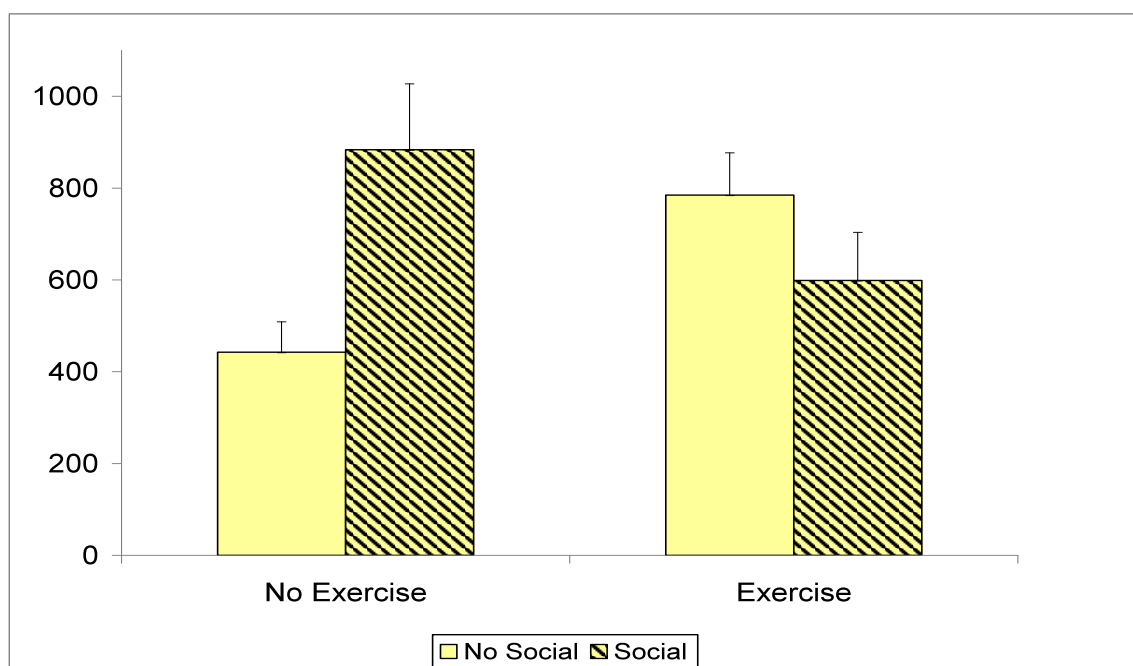


Figure 10. The effects of exercise training and social housing on serum corticosterone in young adult, female, Long-Evans rats.

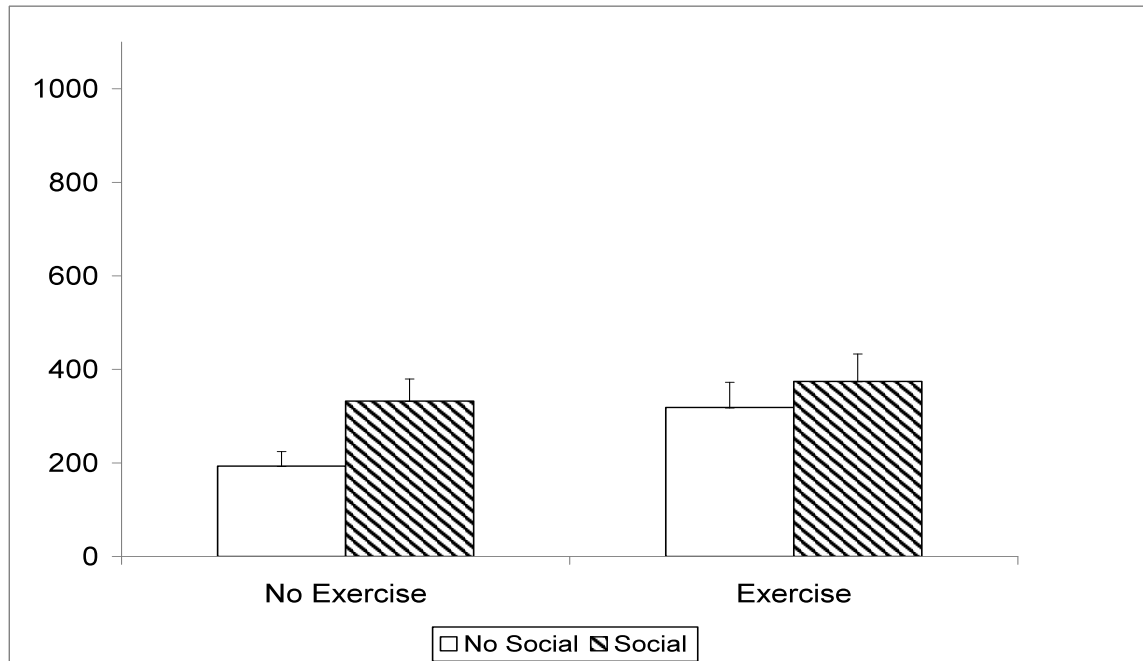


Figure 11. The effects of exercise training and social housing on serum corticosterone in young adult, male, Long-Evans rats.

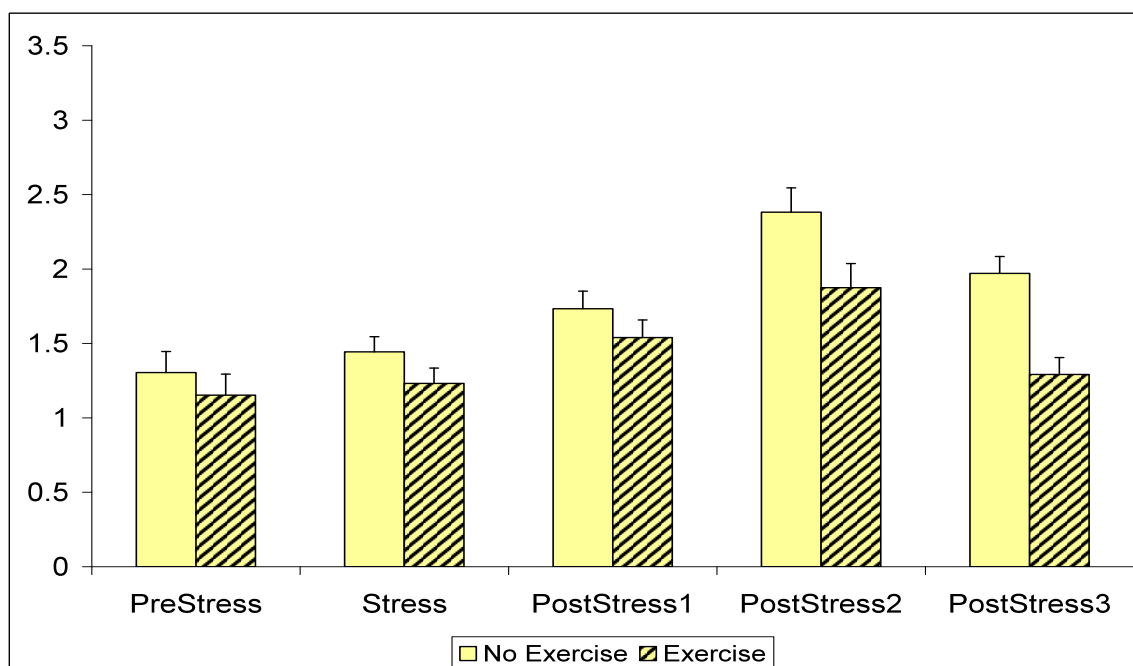


Figure 12. The effects of exercise on center time ratio values over time in young adult, Long-Evans rats.

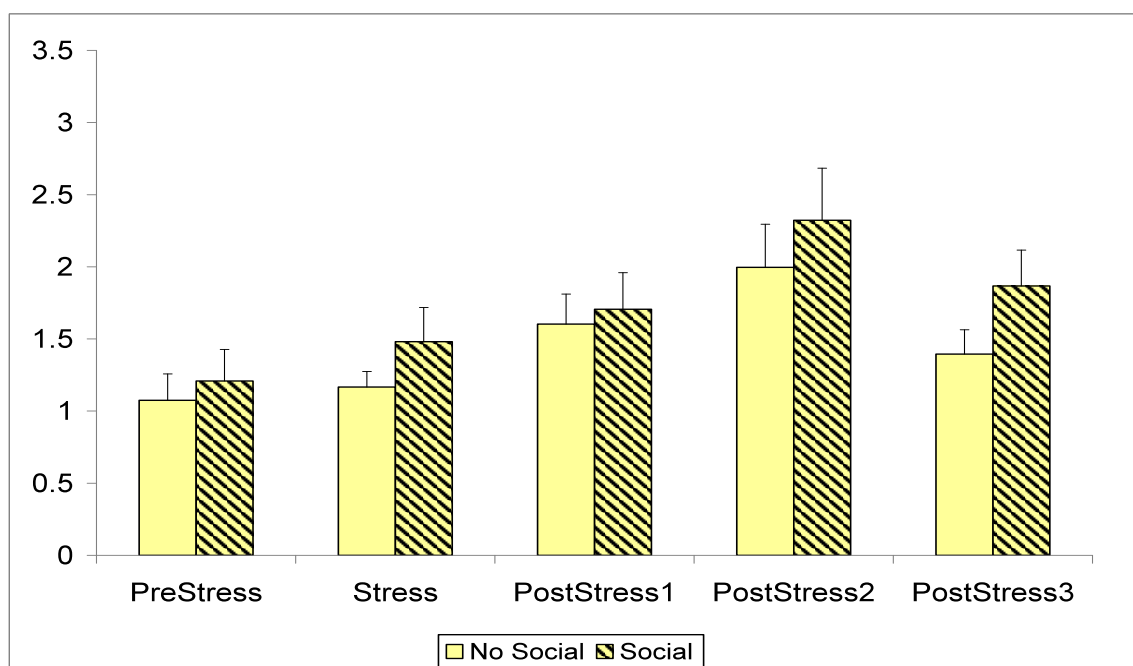


Figure 13. The effects of social housing on center time ratio values over time in young adult, Long-Evans rats.

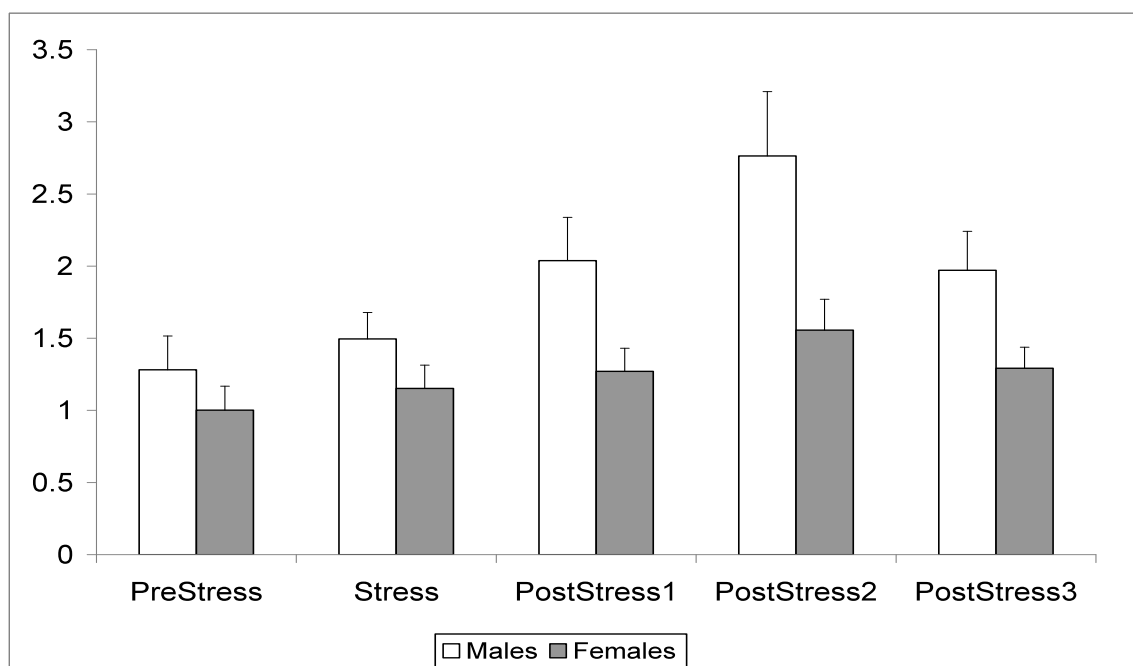


Figure 14. Center time ratio values over time in male and female, young adult, Long-Evans rats.

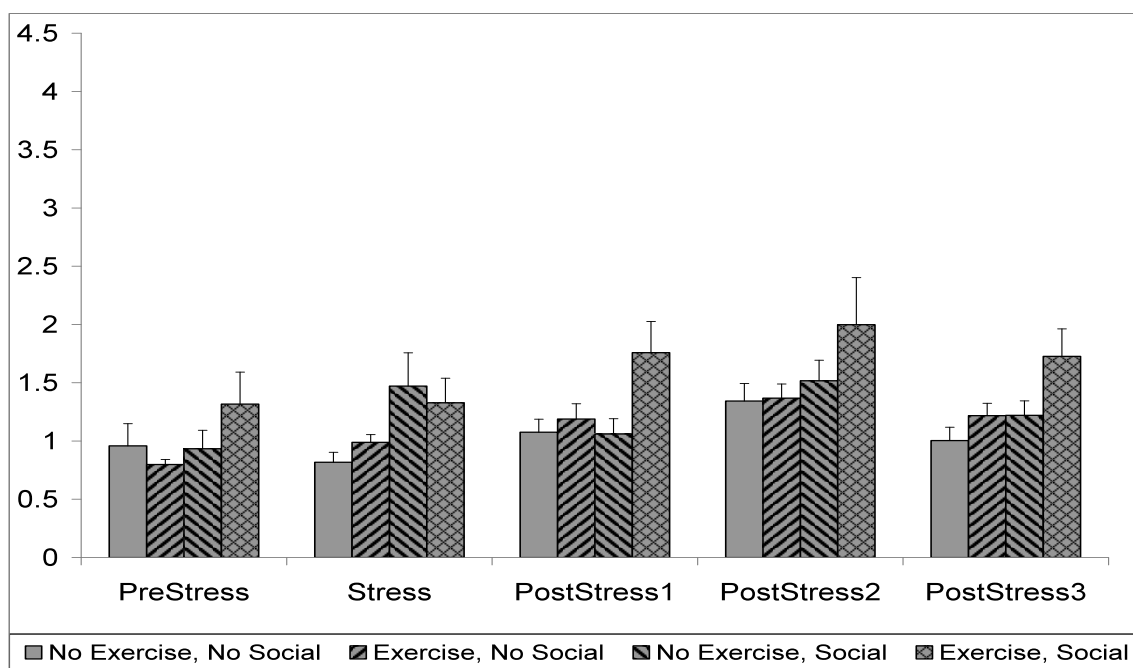


Figure 15. The effects of exercise training and social housing on center time activity in young adult, female, Long-Evans rats.

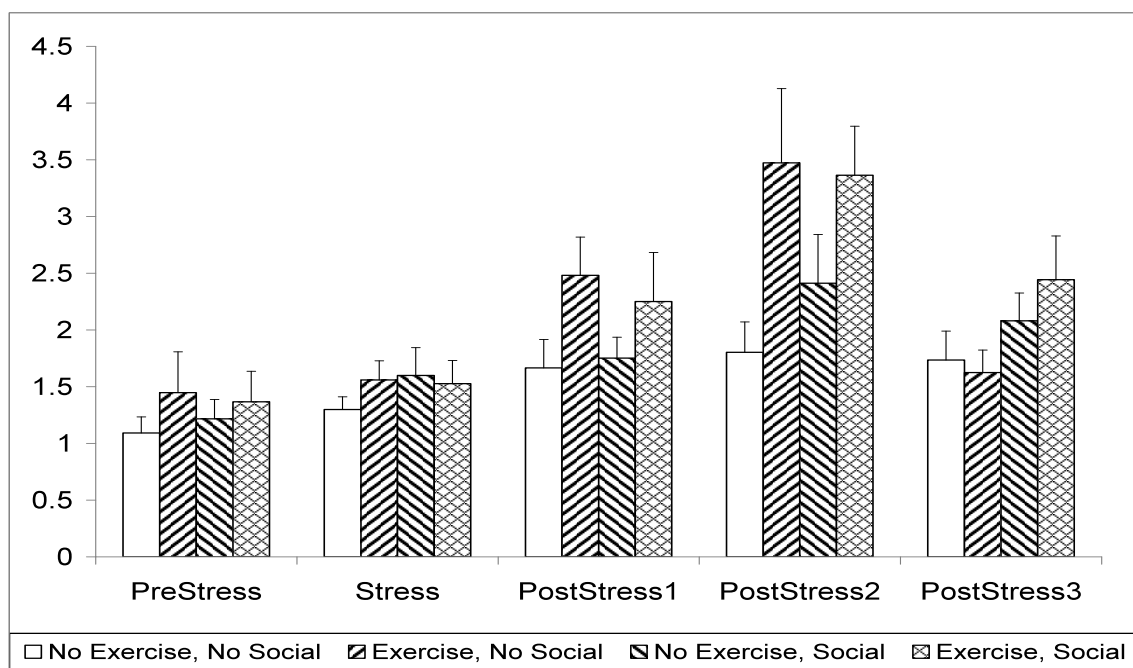


Figure 16. The effects of exercise training and social housing on center time activity in young adult, male, Long-Evans rats.

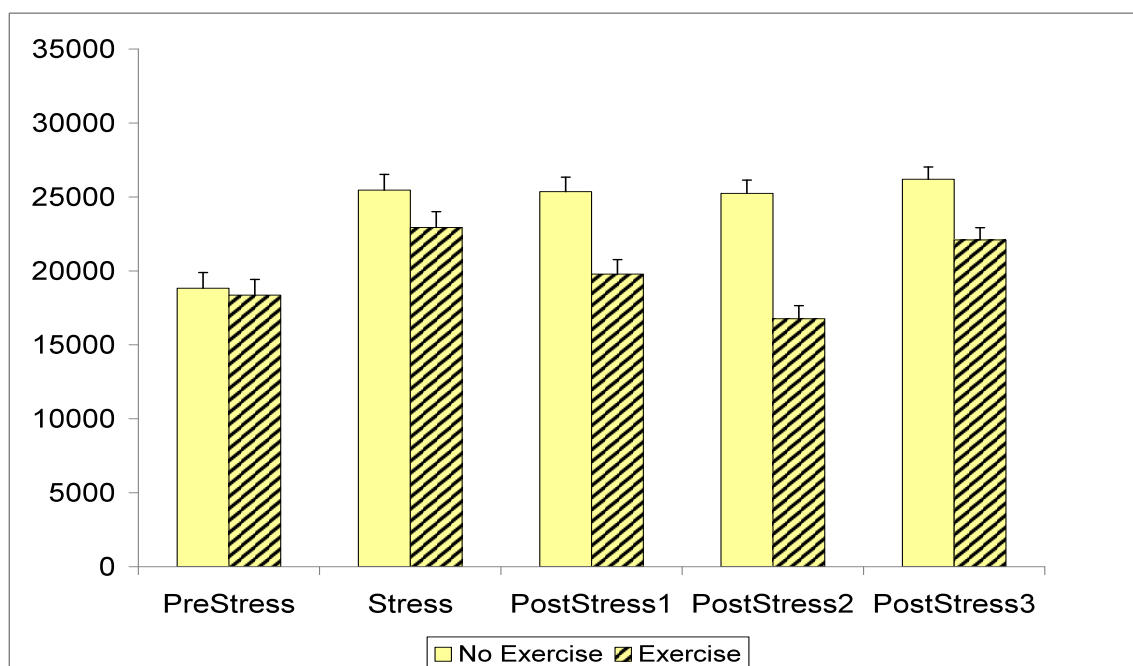


Figure 17. Effects of exercise training on horizontal activity in young adult, Long-Evans rats.

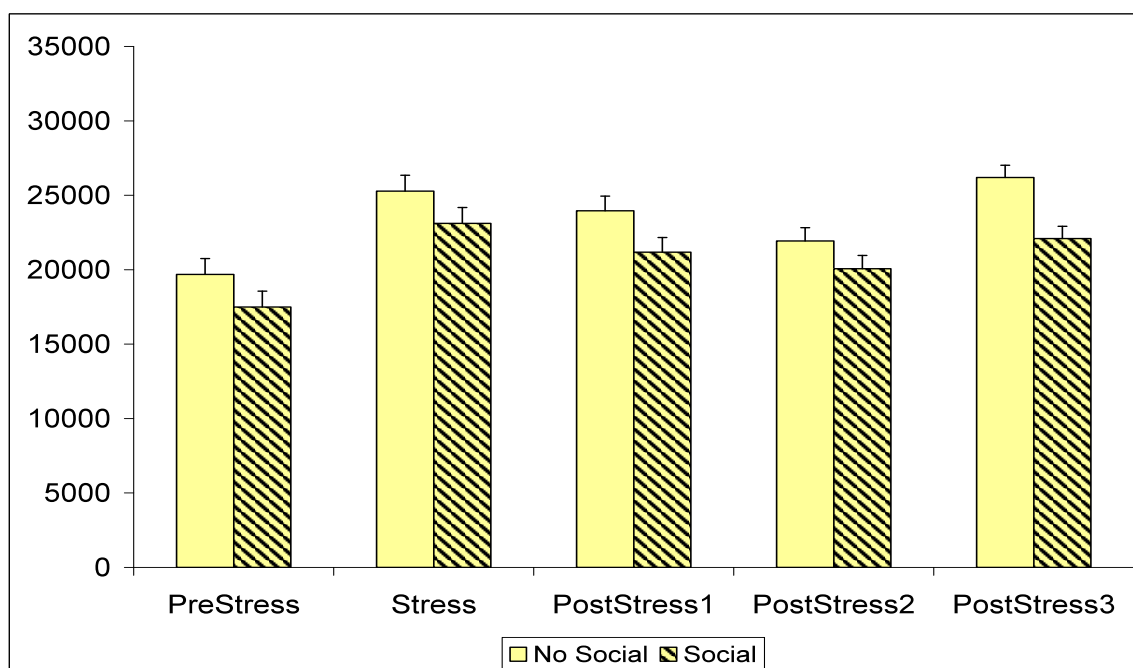


Figure 18. Effects of social housing on horizontal activity in young adult, Long-Evans rats.

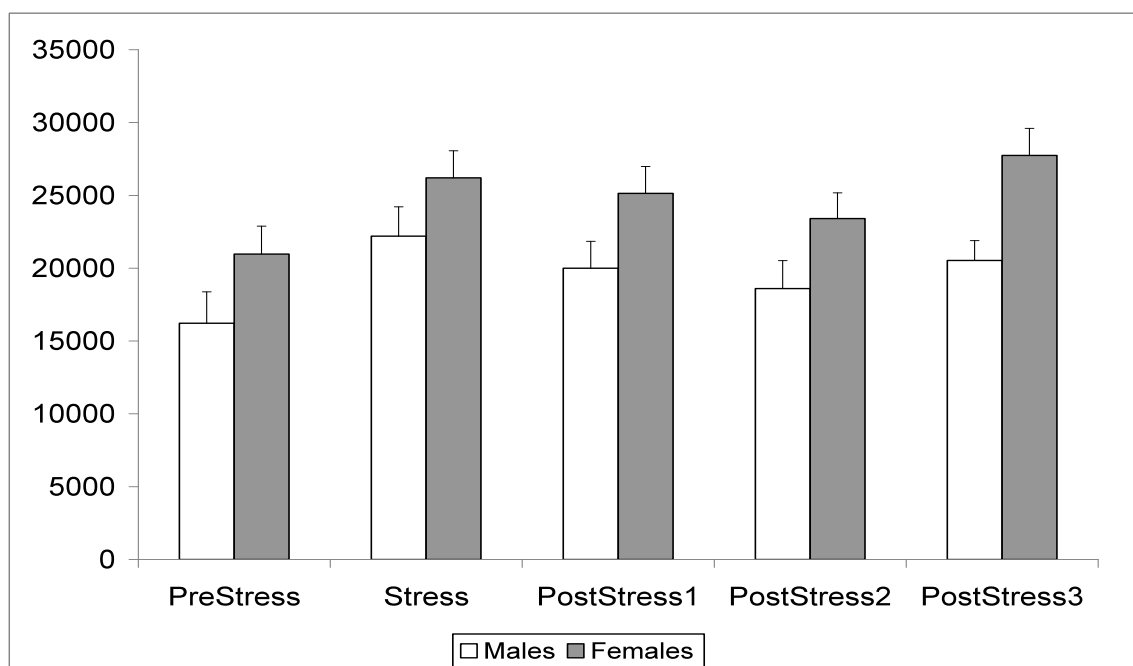


Figure 19. Horizontal activity over time in male and female, young adult, Long-Evans rats.

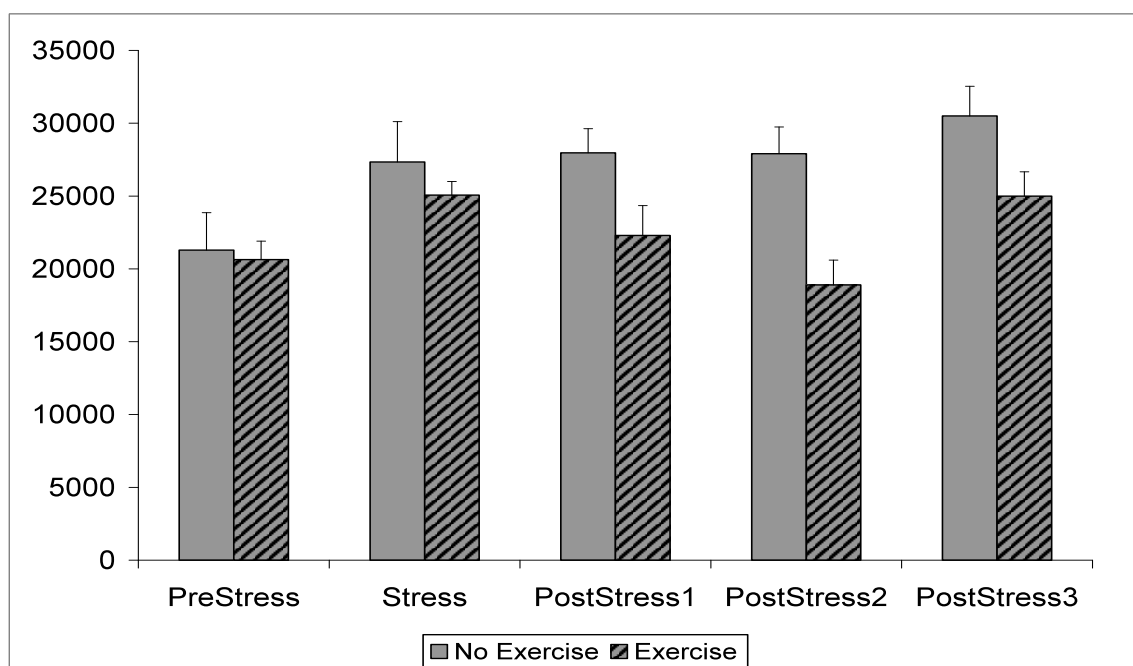


Figure 20. The effects of exercise training on horizontal activity over time in young adult, female, Long-Evans rats.

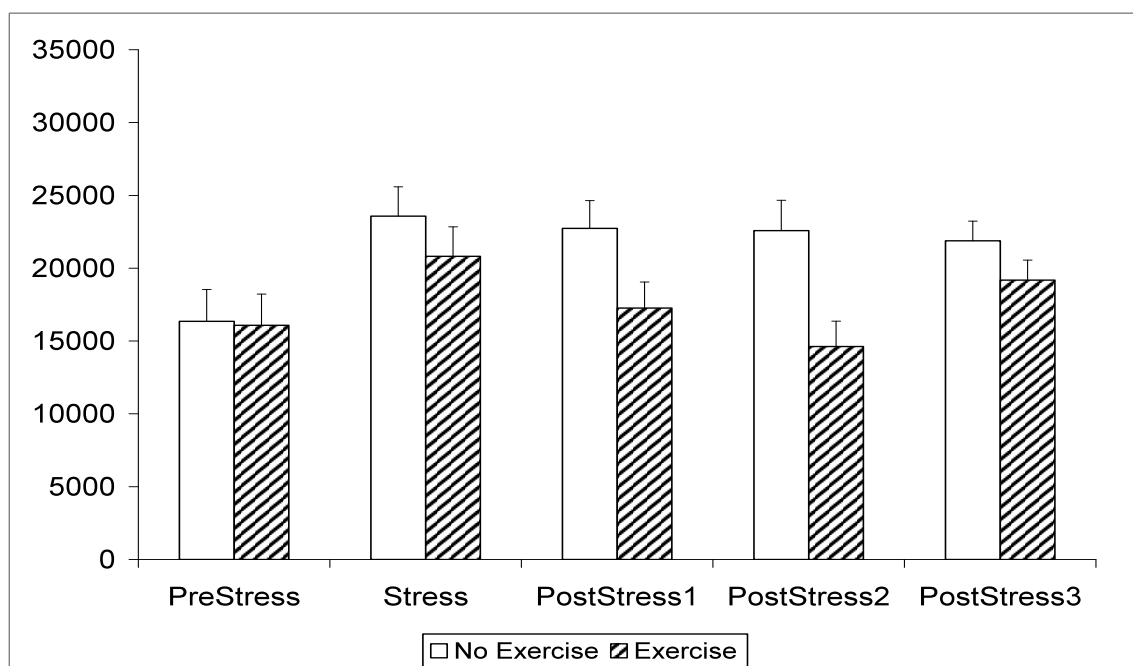


Figure 21. The effects of exercise training on horizontal activity over time in young adult, male, Long-Evans rats.

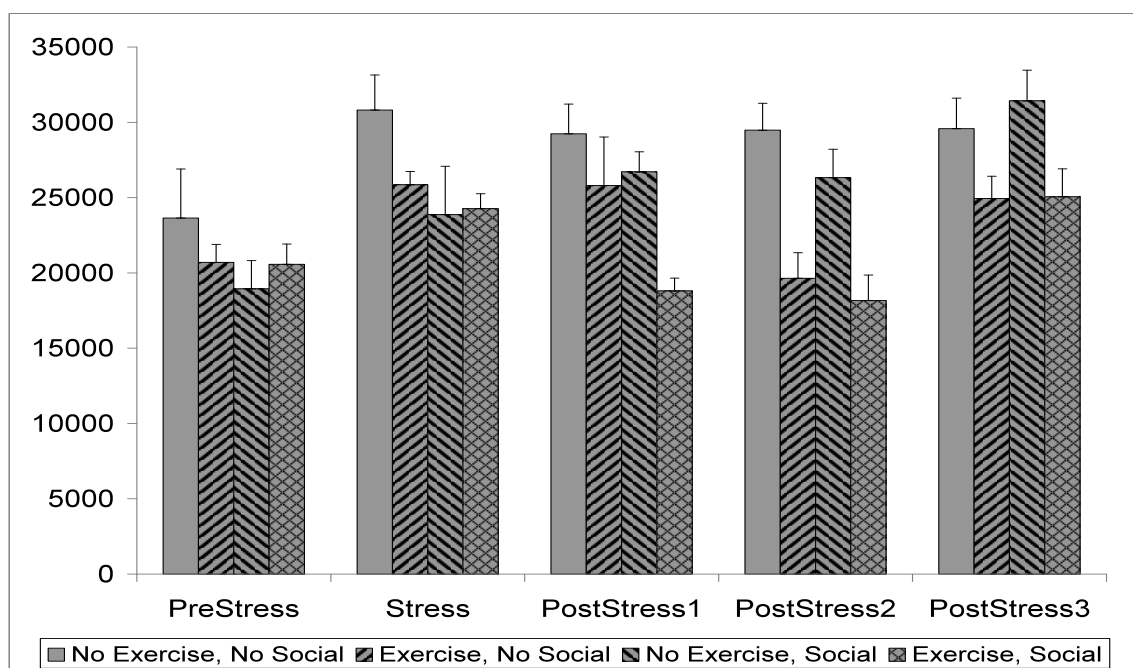


Figure 22. Effects of exercise training and social housing on horizontal activity in female, young adult, Long-Evans rats.

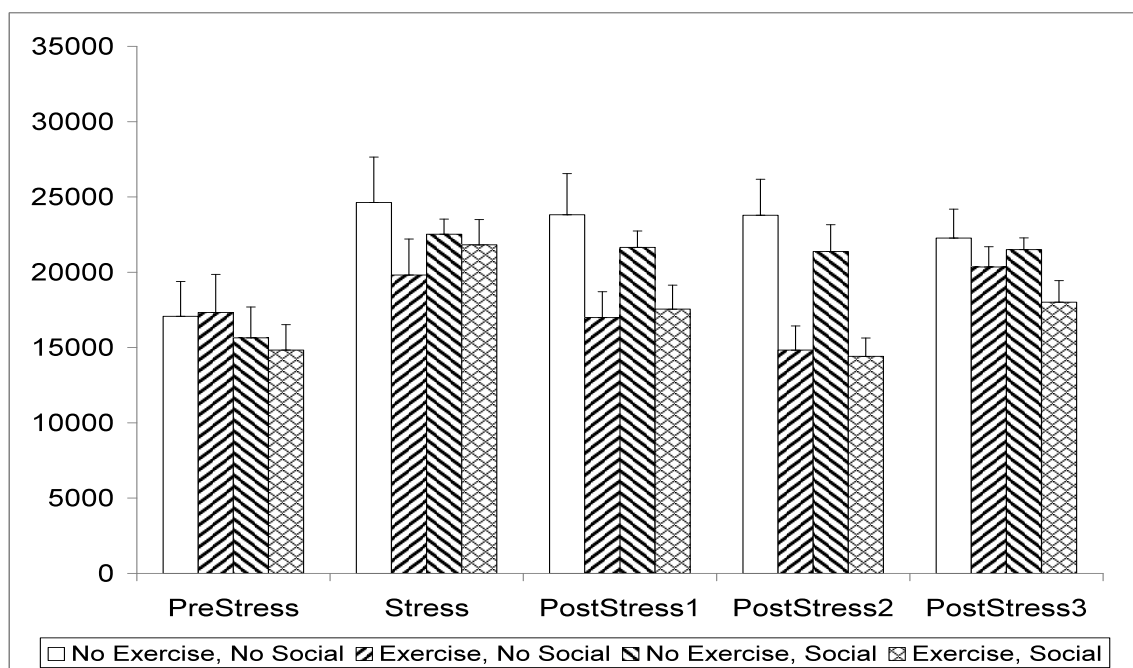


Figure 23. Effects of exercise training and social housing on horizontal activity in male, young adult, Long-Evans rats.

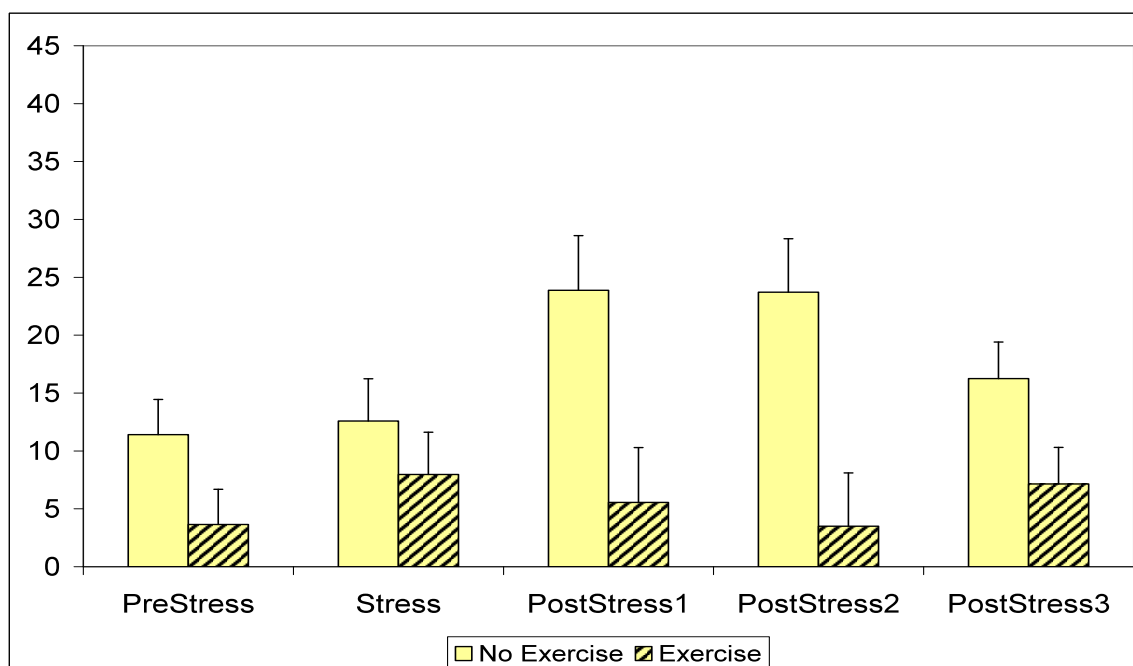


Figure 24. Effects of exercise training on negative affect in young adult, Long-Evans rats.

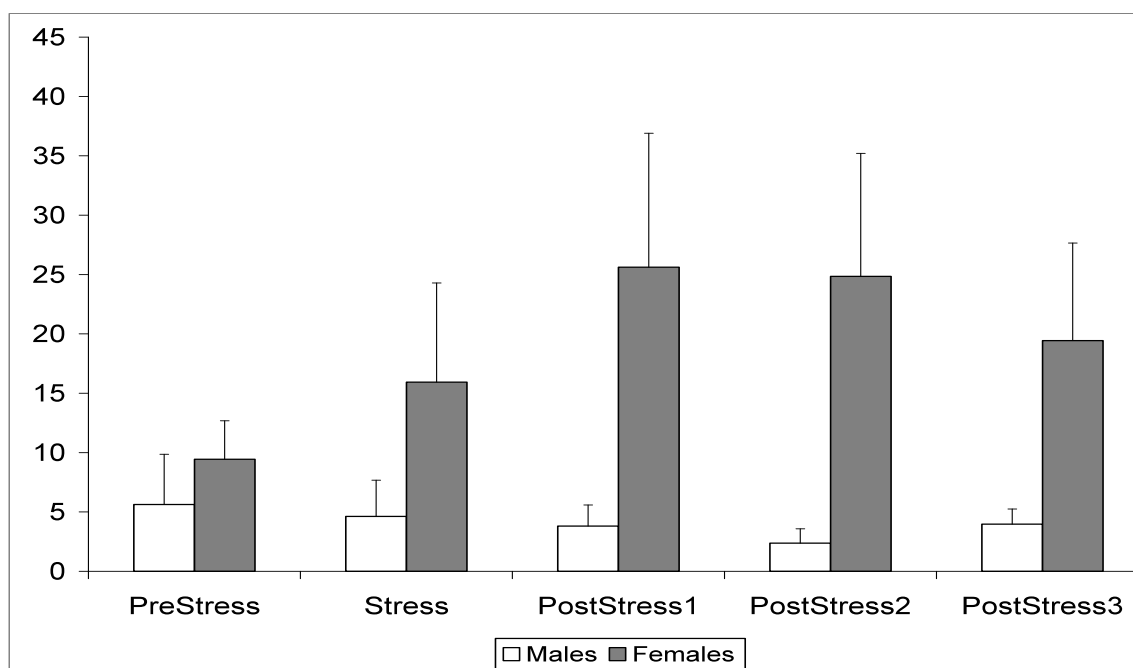


Figure 25. Negative affect over time in young adult, male and female, Long-Evans rats.

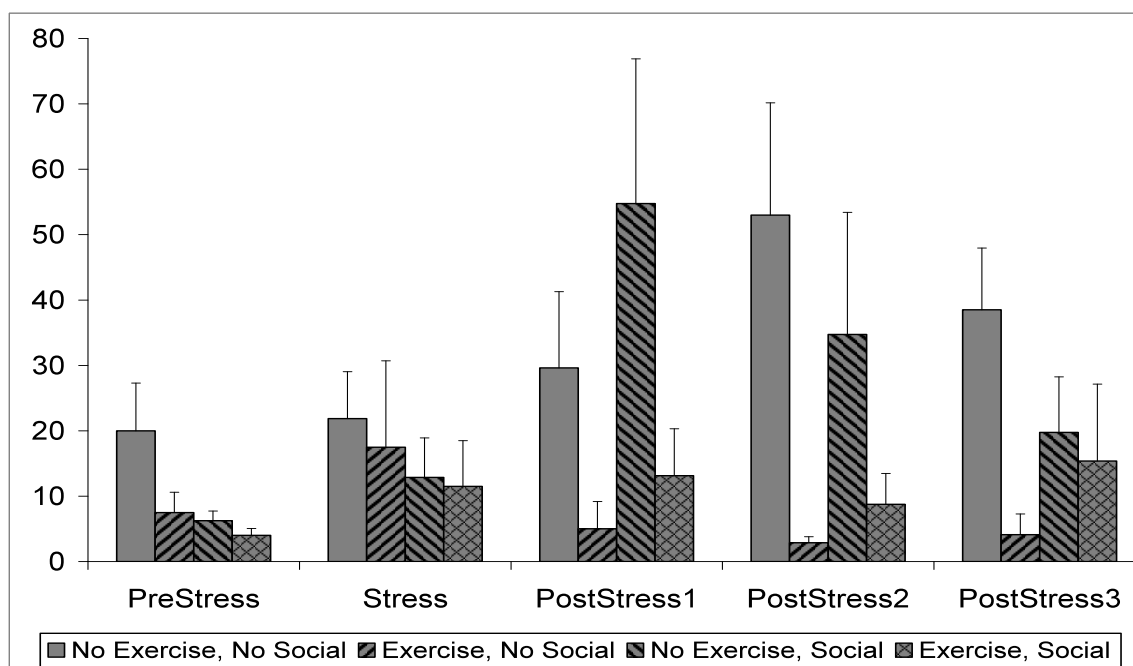


Figure 26. The effect of exercise training and social housing on negative affect in young adult, female, Long-Evans rats.

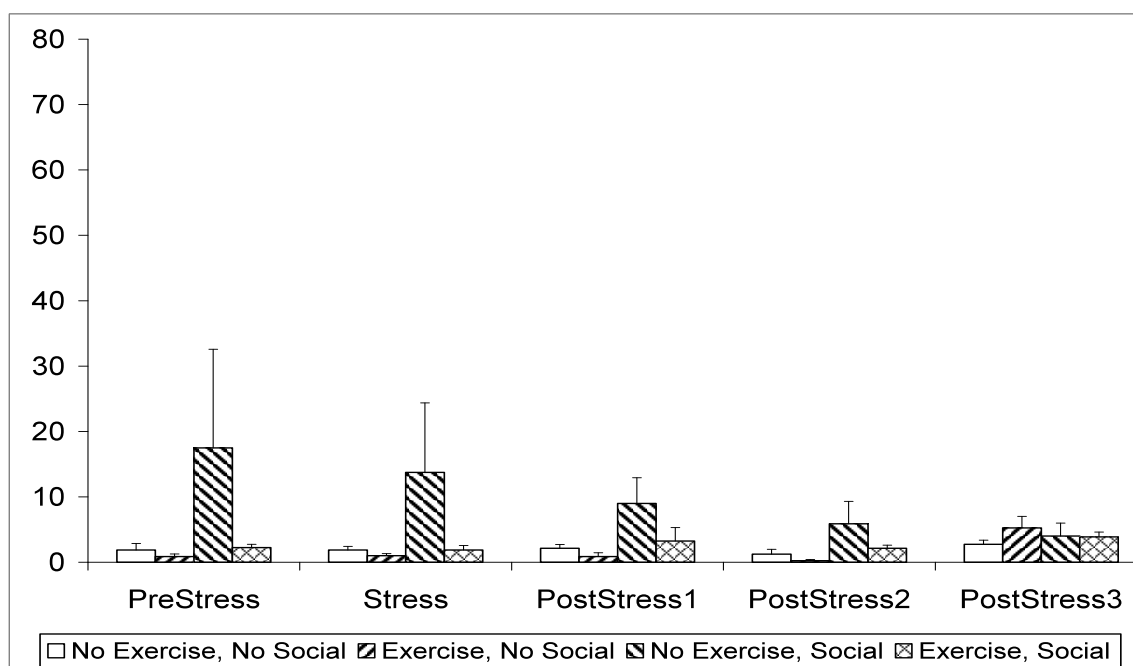


Figure 27. The effect of exercise training and social housing on negative affect in young adult, male, Long-Evans rats.

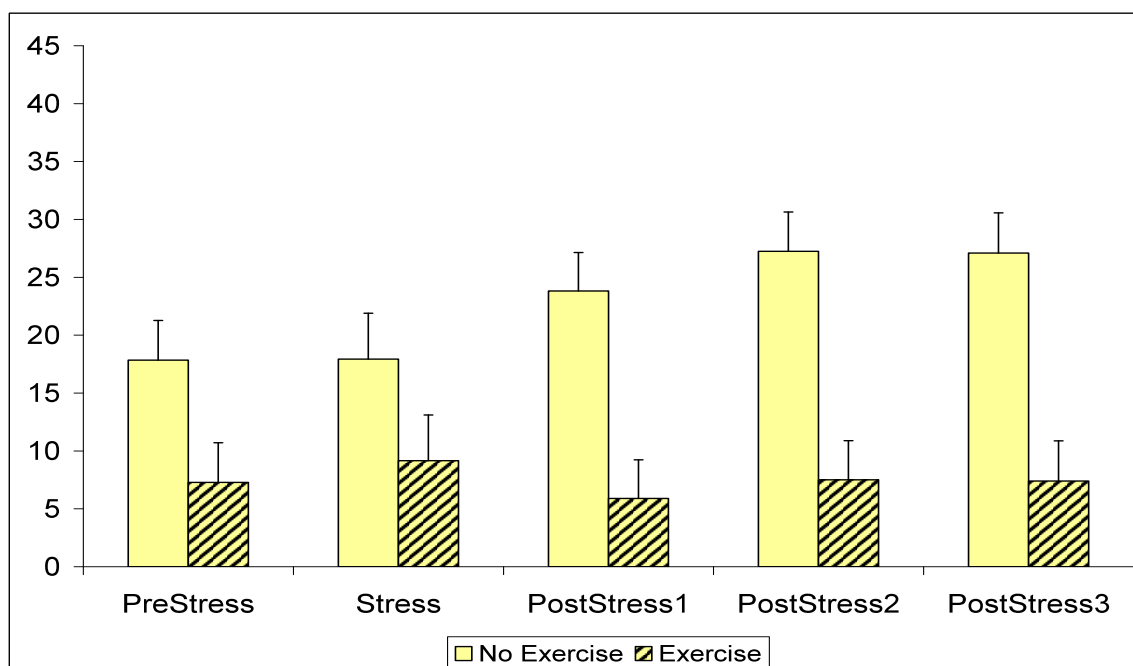


Figure 28. Effects of exercise training on positive affect in young adult, Long-Evans rats.

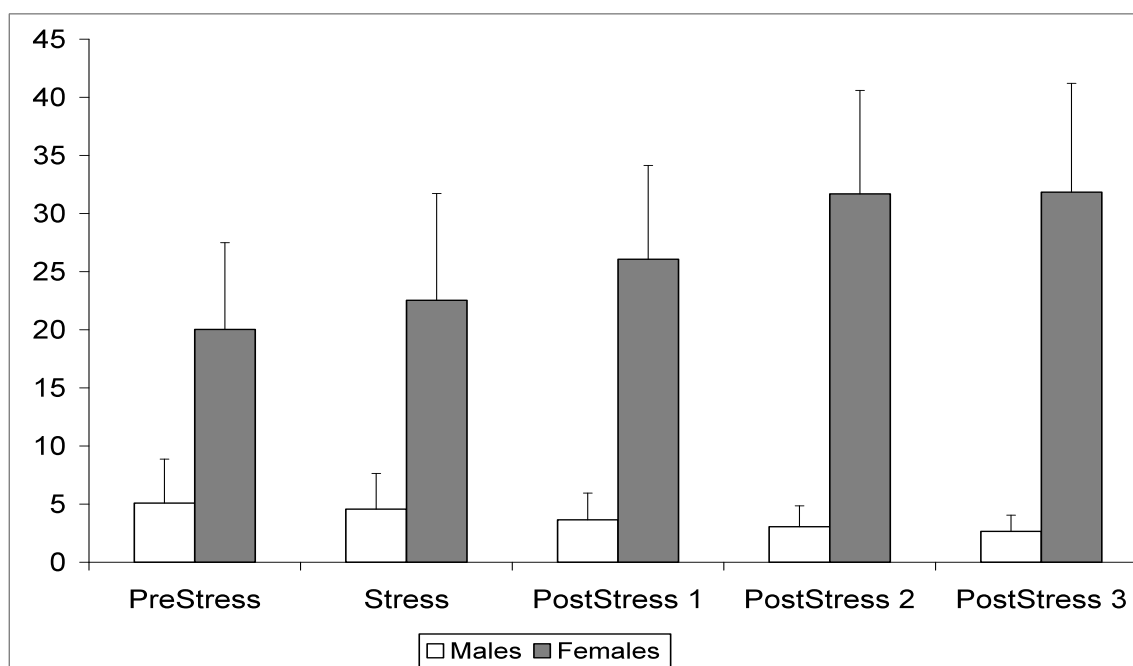


Figure 29. Positive affect over time in male and female, young adult, Long-Evans rats.

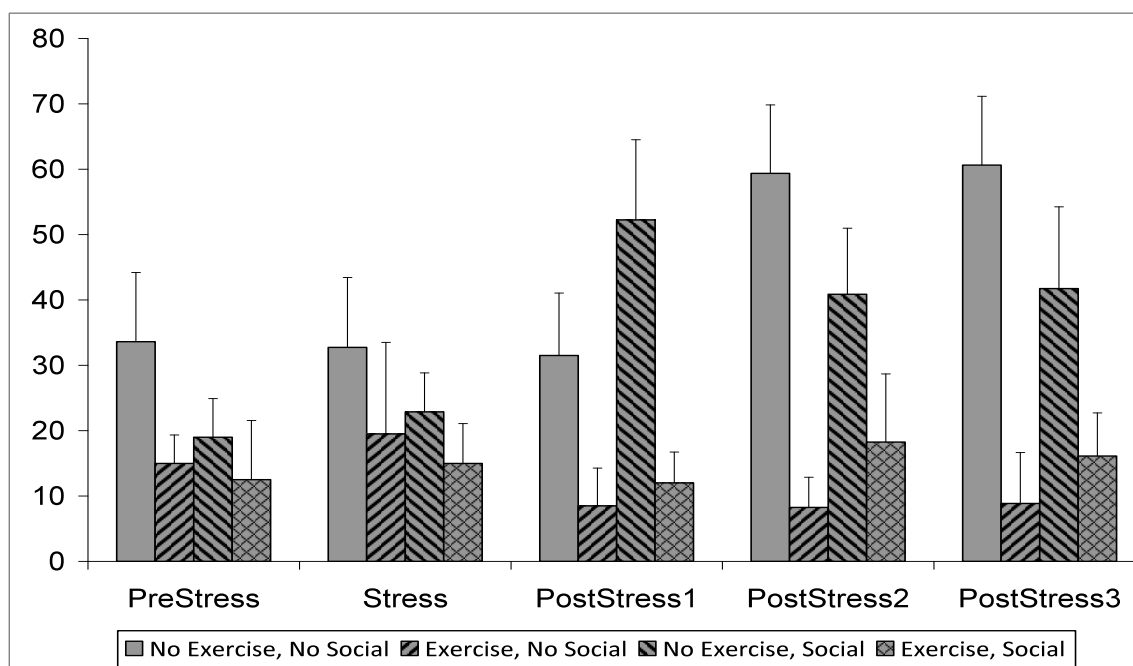


Figure 30. The effects of exercise training and social housing on positive affect over time in young adult, female, Long-Evans rats.

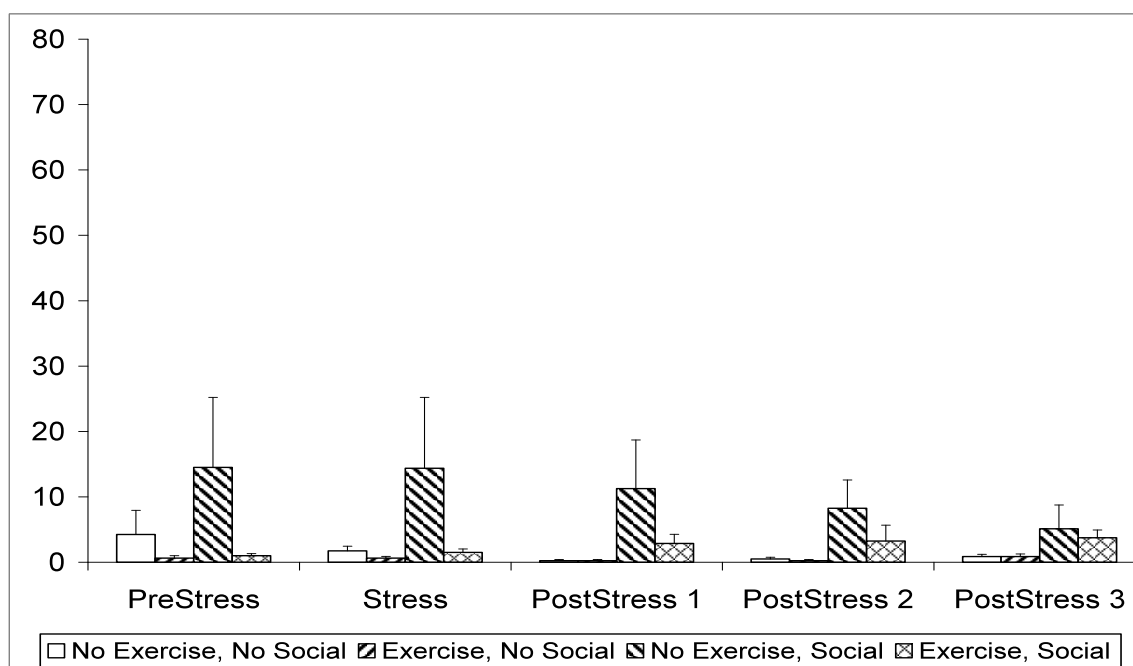


Figure 31. The effects of exercise training and social housing on positive affect over time in young adult, male, Long-Evans rats.

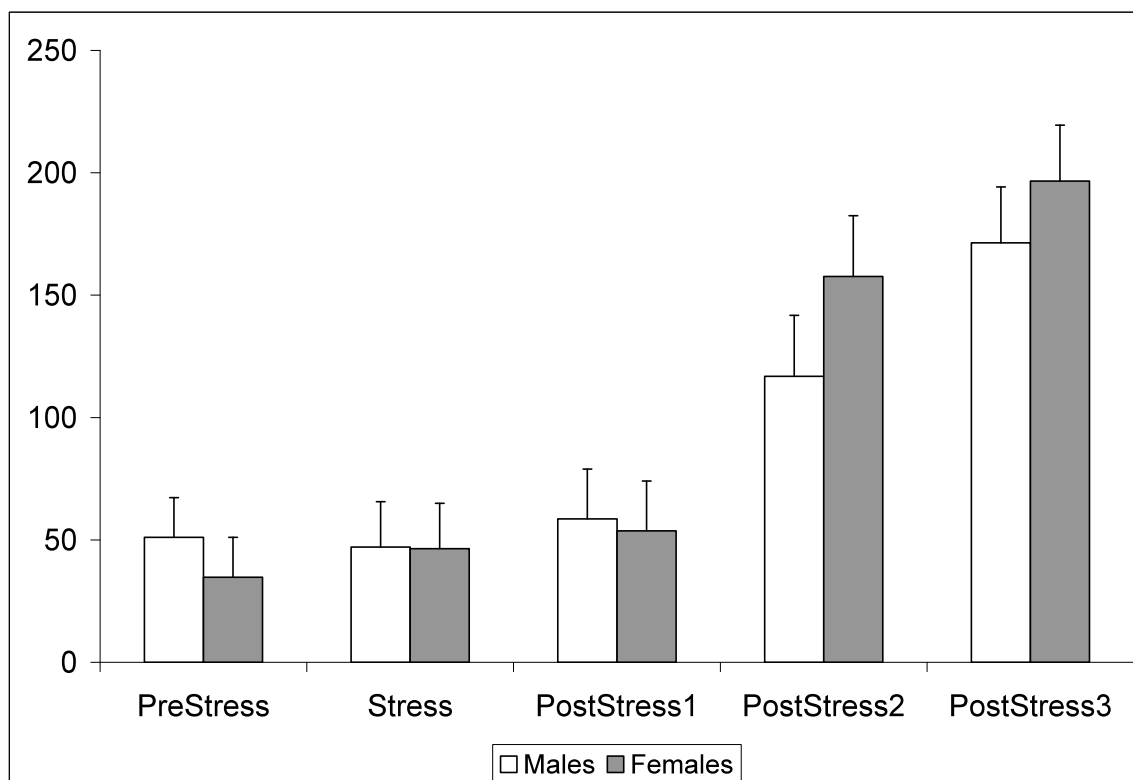


Figure 32. Forced swim immobility (s) over time in young adult, Long-Evans rats.

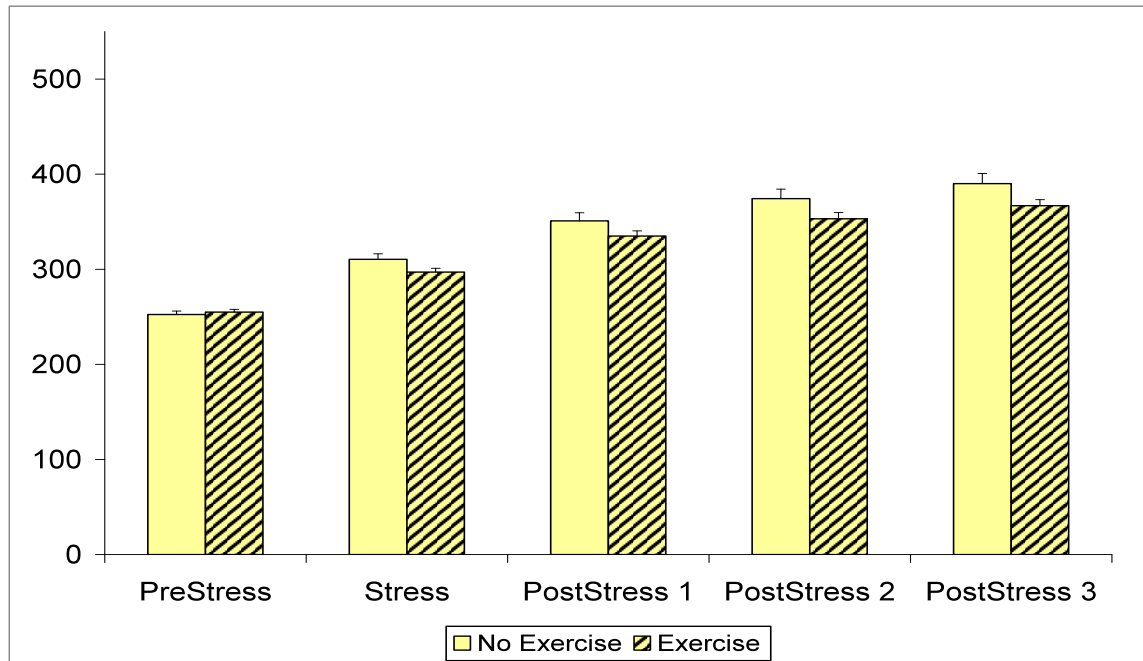


Figure 33. The effects of exercise training on body weight over time in young adult, Long-Evans rats.

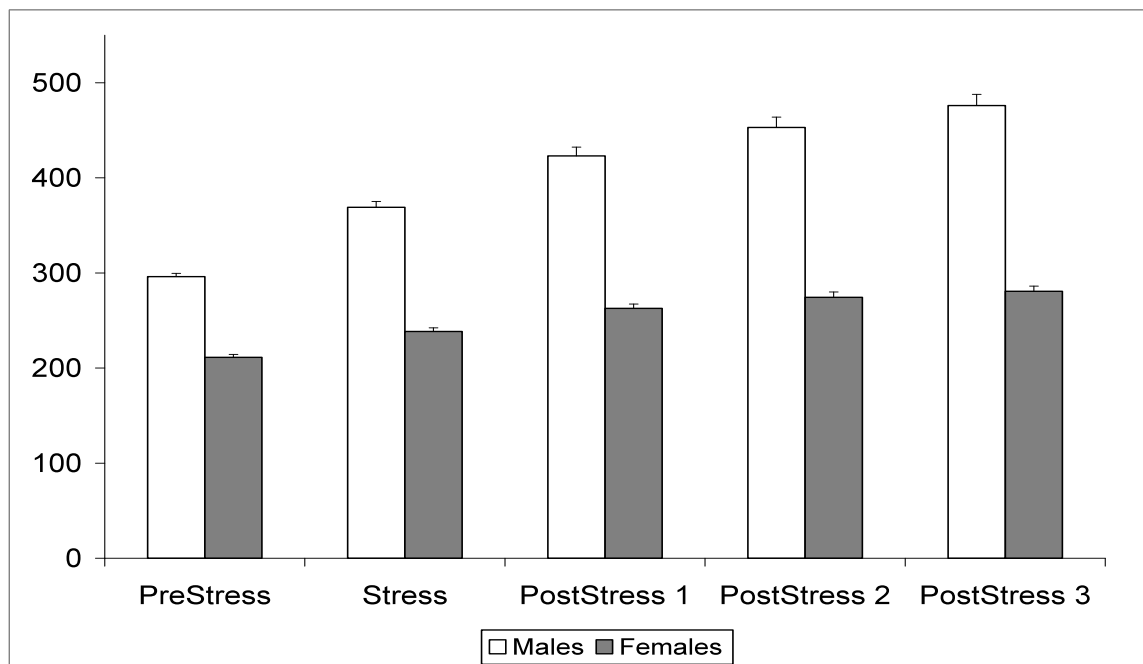


Figure 34. Body weight over time in male and female, young adult, Long-Evans rats.

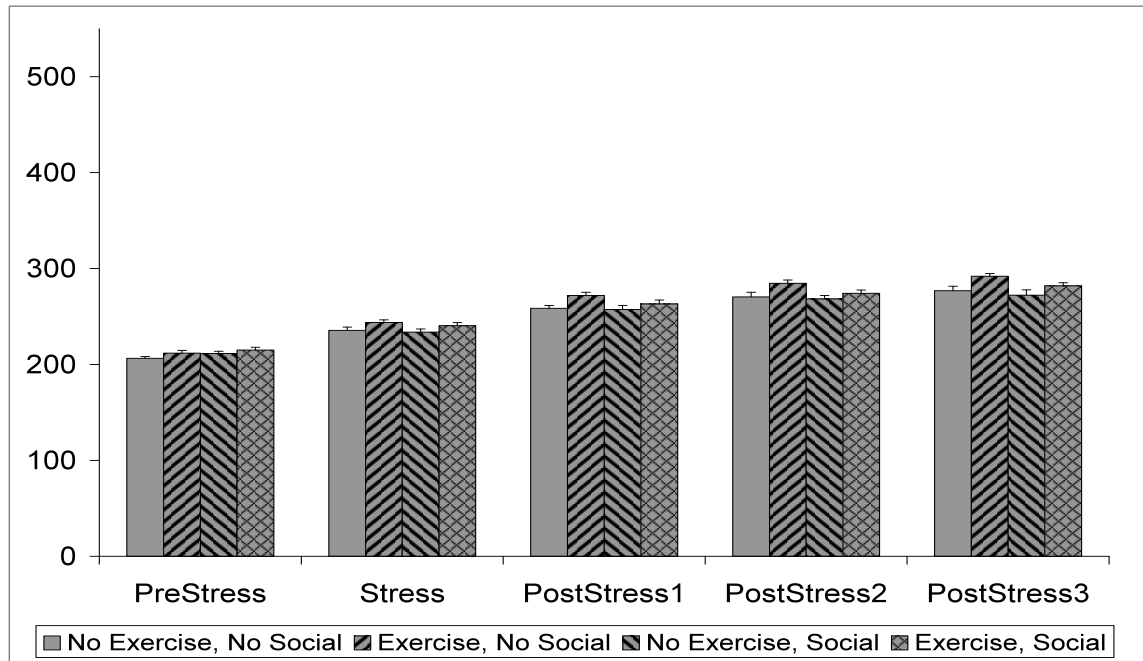


Figure 35. Effects of exercise training and social housing on body weight in young adult, female, Long-Evans rats.

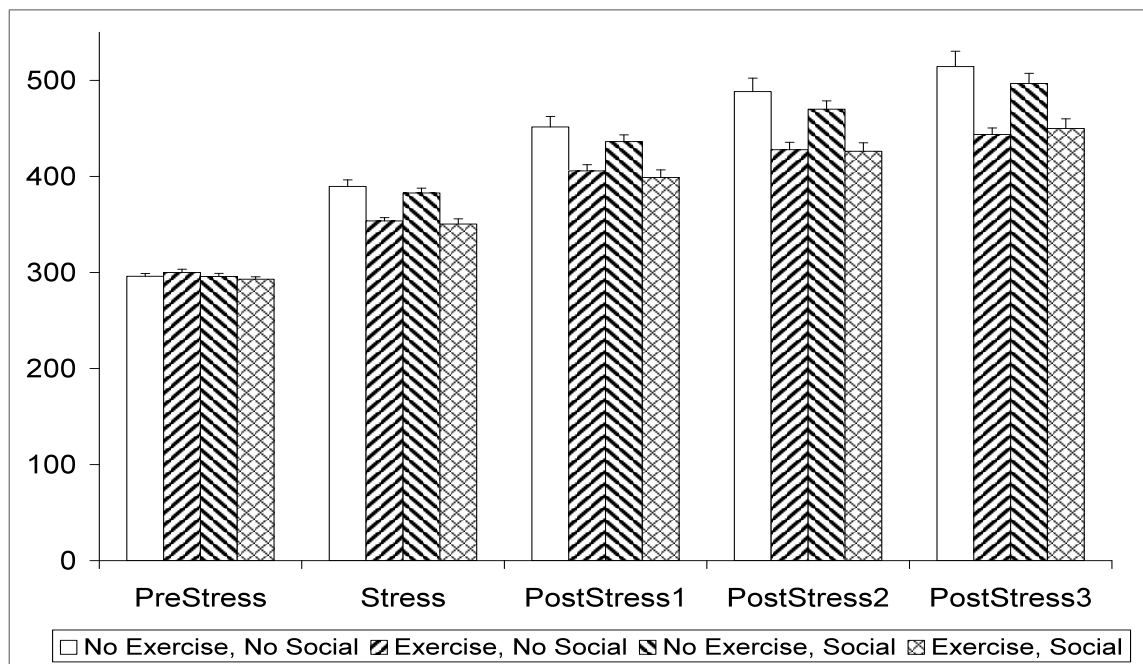


Figure 36. Effects of exercise training and social housing on body weight in young adult, male, Long-Evans rats.

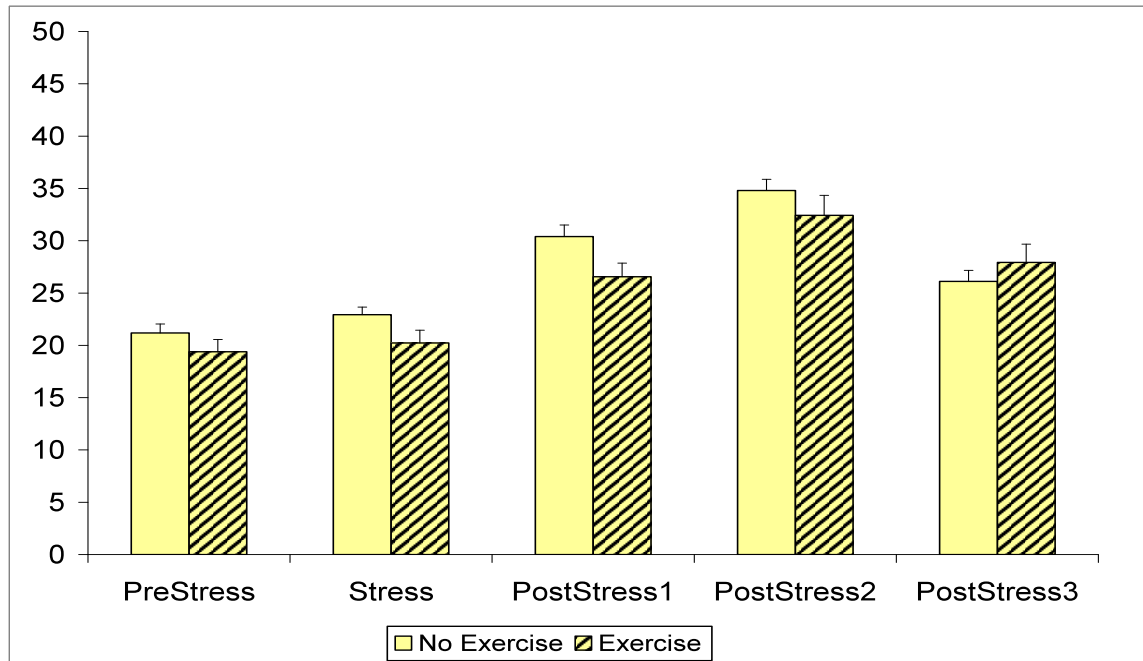


Figure 37. The effects of exercise training on food consumption over time in young adult, Long-Evans rats.

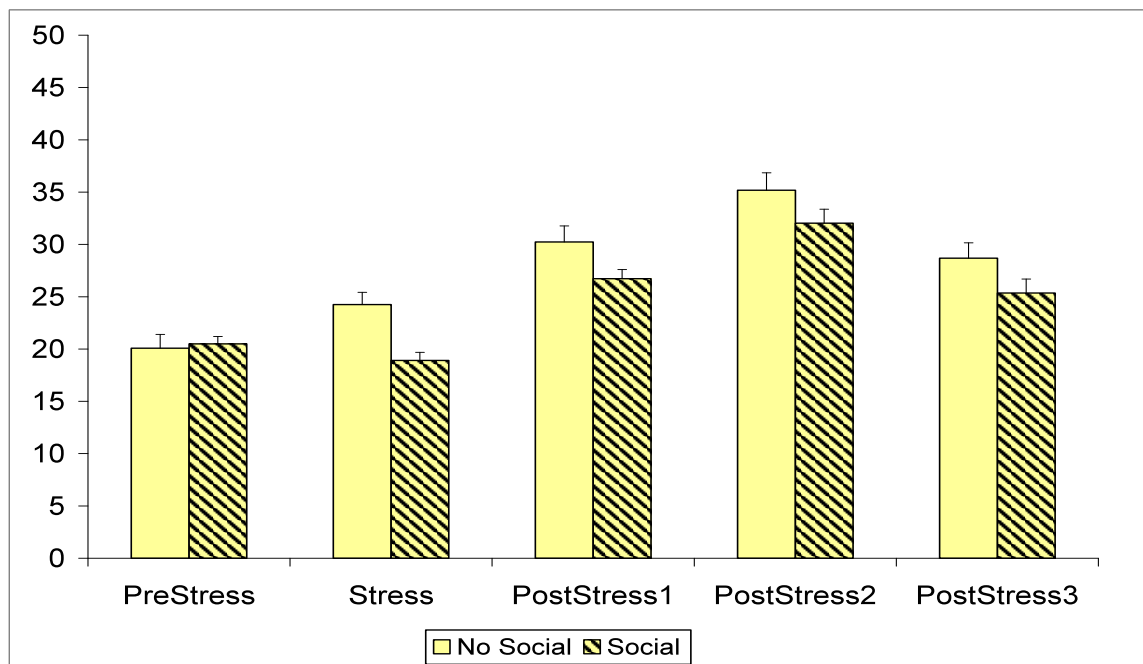


Figure 38. The effects of social housing on food consumption over time in young adult, Long Evans rats.

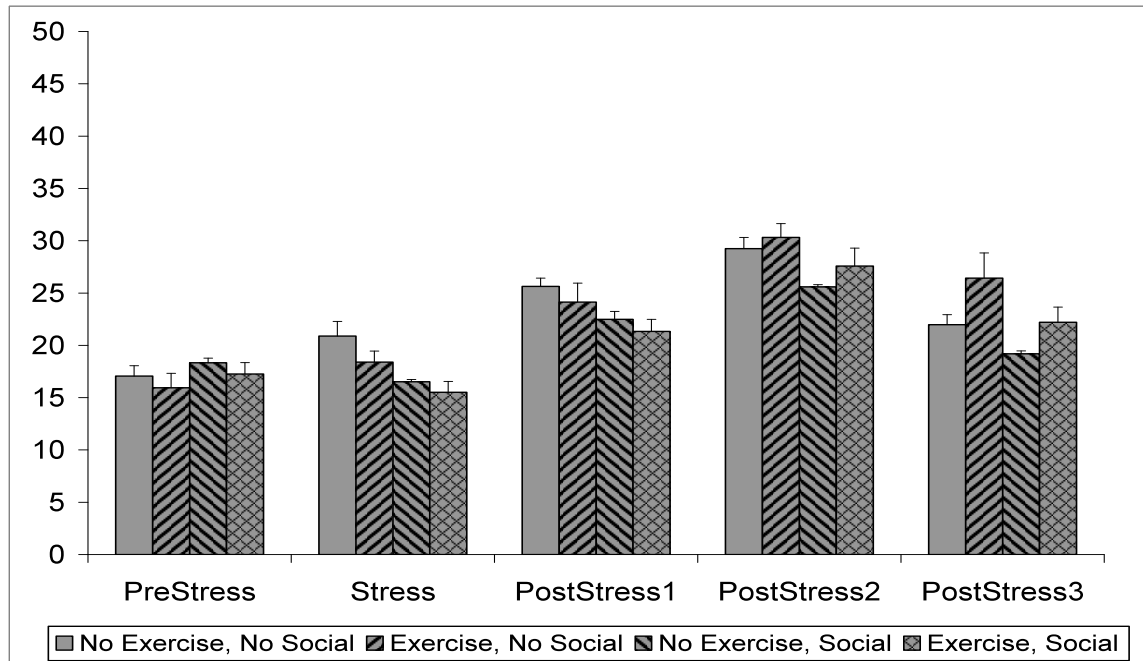


Figure 39. The effects of exercise training and social housing on food consumption in young adult, female, Long-Evans rats.

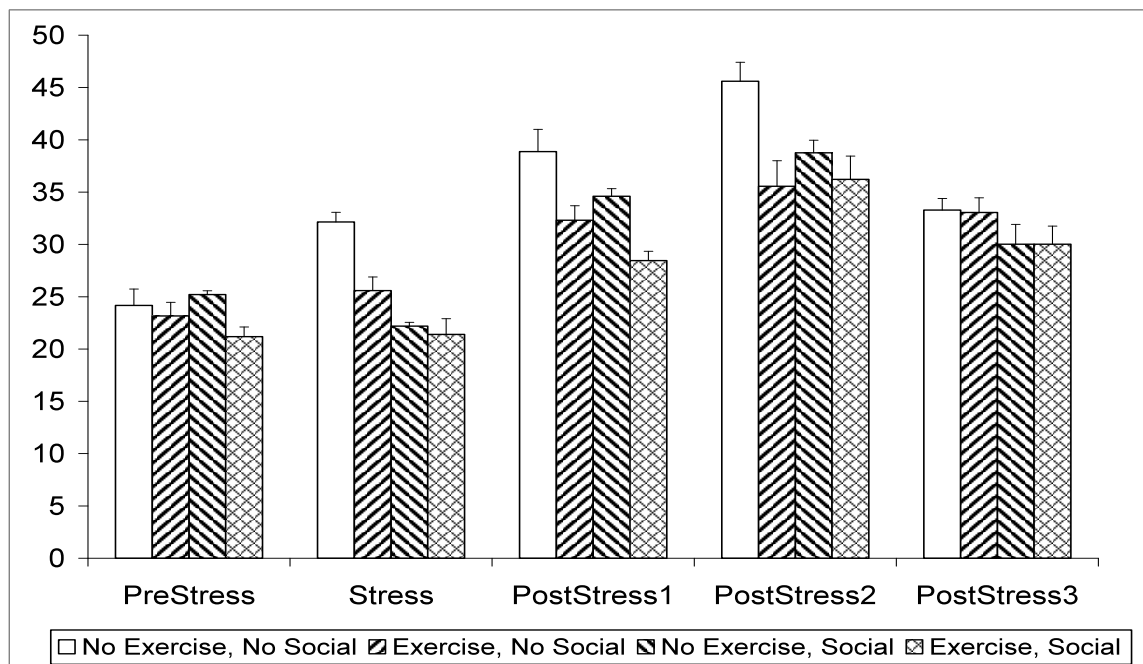


Figure 40. The effects of exercise training and social housing on food consumption in young adult, male, Long-Evans rats.

Appendix E – Statistical Tables

Table 1. Fecal Corticosterone repeated measures ANOVA, Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	1.694E7	1	1.694E7	103.851	.001
Sex	1076363.569	1	1076363.569	6.598	.062
Exercise	136381.707	1	136381.707	.836	.412
Social	25439.150	1	25439.150	.156	.713
Sex * Exercise	19185.052	1	19185.052	.118	.749
Sex * Social	38098.702	1	38098.702	.234	.654
Exercise * Social	258895.707	1	258895.707	1.587	.276
Sex * Exercise * Social	70039.613	1	70039.613	.429	.548
Error	652576.904	4	163144.226		

Table 2. Fecal Corticosterone repeated measures ANOVA, Within-Subject Effects

Tests of Within-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Time	553526.061	4	138381.515	4.910	.009
Time * Sex	289092.048	4	72273.012	2.564	.078
Time * Exercise	382403.688	4	95600.922	3.392	.034
Time * Social	550010.498	4	137502.624	4.879	.009
Time * Sex * Exercise	65548.893	4	16387.223	.581	.680
Time * Sex * Social	54947.750	4	13736.938	.487	.745
Time * Exercise * Social	246024.379	4	61506.095	2.182	.117
Time * Sex * Exercise * Social	351120.814	4	87780.203	3.115	.045
Error(Time)	450936.420	16	28183.526		

Table 3. Fecal Corticosterone repeated measures ANOVA with file split by exercise, Between-Subjects Effects

Tests of Between-Subjects Effects						
Exercise Source		Type III Sum of Squares	df	Mean Square	F	Sig.
No	Intercept	7289398.374	1	7289398.374	34.449	.010
	Sex	718070.923	1	718070.923	3.394	.163
	Social	63359.363	1	63359.363	.299	.622
	Sex * Social	2505.207	1	2505.207	.012	.920
	Error	634805.217	3	211601.739		
Yes	Intercept	9700323.980	1	9700323.980	545.830	.027
	Sex	389641.741	1	389641.741	21.925	.134
	Social	215346.347	1	215346.347	12.117	.178
	Sex * Social	101949.968	1	101949.968	5.737	.252
	Error	17771.687	1	17771.687		

Table 4. Fecal Corticosterone repeated measures ANOVA with file split by exercise, Within-Subject Effects

Tests of Within-Subjects Effects						
Exercise Source		Type III Sum of Squares	df	Mean Square	F	Sig.
No	Time	778145.319	4	194536.330	5.783	.008
	Time * Sex	129118.334	4	32279.583	.960	.464
	Time * Social	107223.542	4	26805.886	.797	.550
	Time * Sex * Social	103724.665	4	25931.166	.771	.565
	Error(Time)	403686.130	12	33640.511		
Yes	Time	179940.177	4	44985.044	3.808	.112
	Time * Sex	222079.598	4	55519.900	4.700	.082
	Time * Social	668040.342	4	167010.086	14.138	.013
	Time * Sex * Social	295250.354	4	73812.589	6.249	.052
	Error(Time)	47250.290	4	11812.573		

Table 5. Fecal Corticosterone Stress Period ANOVA (with pre-stress as a covariate) with pre-stress as a covariate

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	2.546E6	8	318253.227	4.387	.002
Intercept	3016437.696	1	3016437.696	41.577	.000
Prestress	3710.389	1	3710.389	.051	.823
Sex	1508142.490	1	1508142.490	20.787	.000
Exercise	36525.548	1	36525.548	.503	.485
Social	380765.814	1	380765.814	5.248	.031
Sex * Exercise	17176.474	1	17176.474	.237	.631
Sex * Social	40.846	1	40.846	.001	.981
Exercise * Social	4528.296	1	4528.296	.062	.805
Sex * Exercise * Social	29879.013	1	29879.013	.412	.527
Error	1741226.955	24	72551.123		
Total	2.271E7	33			
Corrected Total	4287252.771	32			

a. R Squared = .594 (Adjusted R Squared = .458)

Table 6. Fecal Corticosterone Stress Period ANOVA (with pre-stress as a covariate) with pre-stress as a covariate and file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Female	Corrected Model	195029.745 ^a	4	48757.436	1.984	.173
	Intercept	1233531.828	1	1233531.828	50.203	.000
	Prestress	3404.499	1	3404.499	.139	.717
	Exercise	3138.270	1	3138.270	.128	.728
	Social	136968.312	1	136968.312	5.574	.040
	Exercise * Social	3845.508	1	3845.508	.157	.701
	Error	245706.785	10	24570.679		

	Total	3981363.300	15			
	Corrected Total	440736.531	14			
Male	Corrected Model	526652.289 ^b	4	131663.072	1.188	.362
	Intercept	817598.009	1	817598.009	7.379	.018
	Prestress	55437.738	1	55437.738	.500	.492
	Exercise	79862.557	1	79862.557	.721	.411
	Social	238083.540	1	238083.540	2.149	.166
	Exercise * Social	23272.777	1	23272.777	.210	.654
	Error	1440388.321	13	110799.102		
	Total	1.873E7	18			
	Corrected Total	1967040.610	17			

a. R Squared = .443 (Adjusted R Squared = .220)

b. R Squared = .268 (Adjusted R Squared = .042)

Table 7. Fecal Corticosterone Resilience analysis (post-stress period repeated measures ANOVA with stress period as a covariate), Between-Subjects Effects

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	668006.375	1	668006.375	19.622	.002
Stress	82878.957	1	82878.957	2.435	.153
Sex	327815.228	1	327815.228	9.629	.013
Exercise	148238.289	1	148238.289	4.354	.067
Social	51723.774	1	51723.774	1.519	.249
Sex * Exercise	23296.447	1	23296.447	.684	.429
Sex * Social	11861.393	1	11861.393	.348	.570
Exercise * Social	40003.095	1	40003.095	1.175	.307
Sex * Exercise * Social	220885.088	1	220885.088	6.488	.031
Error	306388.471	9	34043.163		

Table 8. Fecal Corticosterone Resilience analysis (post-stress period repeated measures ANOVA with stress period as a covariate), Within-Subject Effects

Tests of Within-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Resilience	19414.957	2	9707.478	.368	.697
Resilience * Stress	14146.104	2	7073.052	.268	.768
Resilience * Sex	13010.866	2	6505.433	.247	.784
Resilience * Exercise	68562.820	2	34281.410	1.301	.297
Resilience * Social	178755.563	2	89377.781	3.392	.056
Resilience * Sex * Exercise	88856.949	2	44428.475	1.686	.213
Resilience * Sex * Social	65768.737	2	32884.369	1.248	.311
Resilience * Exercise * Social	149999.418	2	74999.709	2.846	.084
Resilience * Sex * Exercise * Social	210154.480	2	105077.240	3.987	.037
Error(Resilience)	474332.966	18	26351.831		

Table 9. Fecal Corticosterone Resilience analysis (post-stress period repeated measures ANOVA with stress period as a covariate) with file split by sex, Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Female	Intercept	210935.292	1	210935.292	83.041	.070
	Stress	5654.326	1	5654.326	2.226	.376
	Exercise	72848.763	1	72848.763	28.679	.118
	Social	1476.984	1	1476.984	.581	.585
	Exercise * Social	155209.691	1	155209.691	61.103	.081
	Error	2540.119	1	2540.119		
Male	Intercept	501680.416	1	501680.416	13.019	.009
	Stress	111323.780	1	111323.780	2.889	.133
	Exercise	44287.409	1	44287.409	1.149	.319
	Social	81315.118	1	81315.118	2.110	.190
	Exercise * Social	60217.705	1	60217.705	1.563	.251
	Error	269749.203	7	38535.600		

Table 10. Fecal Corticosterone Resilience analysis (post-stress period repeated measures ANOVA with stress period as a covariate) with file split by sex, Within-Subject Effects

Tests of Within-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Female	Resilience	48976.376	2	24488.188	1.023	.494
	Resilience * Stress	55095.435	2	27547.718	1.151	.465
	Resilience * Exercise	52457.754	2	26228.877	1.096	.477
	Resilience * Social	20334.857	2	10167.429	.425	.702
	Resilience * Exercise * Social	20336.363	2	10168.181	.425	.702
	Error(Resilience)	47871.510	2	23935.755		
Male	Resilience	4173.902	2	2086.951	.076	.927

Resilience * Stress	1797.910	2	898.955	.033	.968
Resilience * Exercise	115432.137	2	57716.068	2.106	.159
Resilience * Social	249597.364	2	124798.682	4.553	.030
Resilience * Exercise * Social	381559.397	2	190779.698	6.961	.008
Error(Resilience)	383714.215	14	27408.158		

Table 11. Fecal Corticosterone Pre-Stress ANOVA

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	904104.435 ^a	7	129157.776	1.620	.168
Intercept	9020062.458	1	9020062.458	113.163	.000
Sex	12695.747	1	12695.747	.159	.693
Exercise	35277.788	1	35277.788	.443	.511
Social	368173.842	1	368173.842	4.619	.040
Sex * Exercise	17097.449	1	17097.449	.214	.647
Sex * Social	178135.518	1	178135.518	2.235	.145
Exercise * Social	68723.261	1	68723.261	.862	.361
Sex * Exercise * Social	44285.514	1	44285.514	.556	.462
Error	2391260.015	30	79708.667		
Total	1.375E7	38			
Corrected Total	3295364.450	37			

a. R Squared = .274 (Adjusted R Squared = .105)

Table 12. Fecal Corticosterone Pre-Stress ANOVA with file split by sex

Tests of Between-Subjects Effects

Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Female	Corrected Model	764857.549 ^a	3	254952.516	1.977	.161
	Intercept	4503532.943	1	4503532.943	34.927	.000
	Exercise	1755.179	1	1755.179	.014	.909
	Social	570490.313	1	570490.313	4.424	.053

	Exercise * Social	120373.514	1	120373.514	.934	.349
	Error	1934131.407	15	128942.094		
	Total	7375946.954	19			
	Corrected Total	2698988.956	18			
Male	Corrected Model	108450.621 ^b	3	36150.207	1.186	.348
	Intercept	4527493.195	1	4527493.195	148.563	.000
	Exercise	47325.797	1	47325.797	1.553	.232
	Social	15909.245	1	15909.245	.522	.481
	Exercise * Social	1246.829	1	1246.829	.041	.842
	Error	457128.607	15	30475.240		
	Total	6377564.761	19			
	Corrected Total	565579.228	18			

a. R Squared = .283 (Adjusted R Squared = .140)

b. R Squared = .192 (Adjusted R Squared = .030)

Table 13. Fecal Corticosterone Stress ANOVA

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	3.900E6	7	557128.545	8.737	.000
Intercept	2.462E7	1	2.462E7	386.085	.000
Sex	2817499.765	1	2817499.765	44.185	.000
Exercise	73718.050	1	73718.050	1.156	.288
Social	583593.440	1	583593.440	9.152	.004
Sex * Exercise	48833.385	1	48833.385	.766	.386
Sex * Social	274.761	1	274.761	.004	.948
Exercise * Social	49078.908	1	49078.908	.770	.385
Sex * Exercise * Social	256574.677	1	256574.677	4.024	.051
Error	2678200.289	42	63766.674		
Total	3.457E7	50			
Corrected Total	6578100.106	49			

a. R Squared = .593 (Adjusted R Squared = .525)

Table 14. Fecal Corticosterone Stress ANOVA with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Female	Corrected Model	255868.813 ^a	3	85289.604	3.880	.027
	Intercept	4743580.507	1	4743580.507	215.815	.000
	Exercise	1123.471	1	1123.471	.051	.824
	Social	245784.929	1	245784.929	11.182	.004
	Exercise * Social	35741.417	1	35741.417	1.626	.218
	Error	395637.800	18	21979.878		
	Total	5711916.153	22			
	Corrected Total	651506.613	21			
Male	Corrected Model	810375.827 ^b	3	270125.276	2.840	.059
	Intercept	2.552E7	1	2.552E7	268.377	.000
	Exercise	140403.828	1	140403.828	1.476	.236
	Social	352641.671	1	352641.671	3.708	.066
	Exercise * Social	306848.378	1	306848.378	3.226	.085
	Error	2282562.490	24	95106.770		
	Total	2.885E7	28			
	Corrected Total	3092938.316	27			

a. R Squared = .393 (Adjusted R Squared = .292)

b. R Squared = .262 (Adjusted R Squared = .170)

Table 15. Fecal Corticosterone Post-Stress 1 ANOVA

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	1.638E6	7	233998.947	2.517	.046
Intercept	6233276.680	1	6233276.680	67.046	.000
Sex	470375.998	1	470375.998	5.059	.035
Exercise	141784.281	1	141784.281	1.525	.230
Social	33458.656	1	33458.656	.360	.555
Sex * Exercise	8523.540	1	8523.540	.092	.765

Sex * Social	128478.637	1	128478.637	1.382	.252
Exercise * Social	34.699	1	34.699	.000	.985
Sex * Exercise * Social	700441.325	1	700441.325	7.534	.012
Error	2045340.932	22	92970.042		
Total	1.456E7	30			
Corrected Total	3683333.564	29			

a. R Squared = .445 (Adjusted R Squared = .268)

Table 16. Fecal Corticosterone Post-Stress 1 ANOVA with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Female	Corrected Model	398208.421 ^a	3	132736.140	.557	.660
	Intercept	1174991.271	1	1174991.271	4.926	.062
	Exercise	78774.177	1	78774.177	.330	.583
	Social	11039.535	1	11039.535	.046	.836
	Exercise * Social	247470.781	1	247470.781	1.038	.342
	Error	1669570.795	7	238510.114		
	Total	4485604.809	11			
	Corrected Total	2067779.217	10			
Male	Corrected Model	931823.035 ^b	3	310607.678	12.399	.000
	Intercept	8375291.880	1	8375291.880	334.325	.000
	Exercise	66799.452	1	66799.452	2.667	.123
	Social	242343.519	1	242343.519	9.674	.007
	Exercise * Social	587393.158	1	587393.158	23.448	.000
	Error	375770.136	15	25051.342		
	Total	1.007E7	19			
	Corrected Total	1307593.172	18			

a. R Squared = .193 (Adjusted R Squared = -.153)

b. R Squared = .713 (Adjusted R Squared = .655)

Table 17. Fecal Corticosterone Post-Stress 2 ANOVA

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	419456.982 ^a	7	59922.426	1.162	.367
Intercept	6105856.116	1	6105856.116	118.435	.000
Sex	171792.454	1	171792.454	3.332	.083
Exercise	22099.437	1	22099.437	.429	.520
Social	101538.650	1	101538.650	1.970	.176
Sex * Exercise	556.940	1	556.940	.011	.918
Sex * Social	79904.514	1	79904.514	1.550	.228
Exercise * Social	43157.422	1	43157.422	.837	.371
Sex * Exercise * Social	7319.598	1	7319.598	.142	.710
Error	1031092.157	20	51554.608		
Total	1.254E7	28			
Corrected Total	1450549.140	27			

a. R Squared = .289 (Adjusted R Squared = .040)

Table 18. Fecal Corticosterone Post-Stress 2 ANOVA with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Female	Corrected Model	149483.783 ^a	3	49827.928	.393	.766
	Intercept	1457595.667	1	1457595.667	11.493	.028
	Exercise	10226.570	1	10226.570	.081	.791
	Social	124620.084	1	124620.084	.983	.378
	Exercise * Social	5145.570	1	5145.570	.041	.850
	Error	507288.667	4	126822.167		
	Total	2766430.149	8			
	Corrected Total	656772.450	7			
Male	Corrected Model	119867.310 ^b	3	39955.770	1.220	.335
	Intercept	7579805.540	1	7579805.540	231.531	.000
	Exercise	14238.125	1	14238.125	.435	.519

Social	1178.370	1	1178.370	.036	.852
Exercise * Social	78314.201	1	78314.201	2.392	.141
Error	523803.490	16	32737.718		
Total	9772378.000	20			
Corrected Total	643670.800	19			

a. R Squared = .228 (Adjusted R Squared = -.352)

b. R Squared = .186 (Adjusted R Squared = .034)

Table 19. Fecal Corticosterone Post-Stress 3 ANOVA

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	1.366E6	7	195135.237	4.748	.001
Intercept	1.341E7	1	1.341E7	326.352	.000
Sex	482731.337	1	482731.337	11.747	.002
Exercise	167706.936	1	167706.936	4.081	.051
Social	113872.105	1	113872.105	2.771	.105
Sex * Exercise	47758.573	1	47758.573	1.162	.288
Sex * Social	275086.747	1	275086.747	6.694	.014
Exercise * Social	17183.862	1	17183.862	.418	.522
Sex * Exercise * Social	121947.586	1	121947.586	2.967	.094
Error	1479397.883	36	41094.386		
Total	2.103E7	44			
Corrected Total	2845344.545	43			

a. R Squared = .480 (Adjusted R Squared = .379)

Table 20. Fecal Corticosterone Post-Stress 3 ANOVA with file split by sex

Tests of Between-Subjects Effects

Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Female	Corrected Model	332965.350 ^a	3	110988.450	2.252	.139
	Intercept	3227684.002	1	3227684.002	65.498	.000
	Exercise	144594.919	1	144594.919	2.934	.115

	Social	12823.669	1	12823.669	.260	.620
	Exercise * Social	84561.752	1	84561.752	1.716	.217
	Error	542067.050	11	49278.823		
	Total	4733810.000	15			
	Corrected Total	875032.400	14			
Male	Corrected Model	614398.132 ^b	3	204799.377	5.462	.005
	Intercept	1.492E7	1	1.492E7	398.038	.000
	Exercise	28675.177	1	28675.177	.765	.390
	Social	584073.050	1	584073.050	15.578	.001
	Exercise * Social	37404.028	1	37404.028	.998	.327
	Error	937330.833	25	37493.233		
	Total	1.629E7	29			
	Corrected Total	1551728.966	28			

a. R Squared = .381 (Adjusted R Squared = .212)

b. R Squared = .396 (Adjusted R Squared = .323)

Table 21. Serum Corticosterone ANOVA

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	3.289E6	7	469879.548	8.653	.000
Intercept	1.516E7	1	1.516E7	279.222	.000
Sex	2184017.167	1	2184017.167	40.222	.000
Exercise	49554.223	1	49554.223	.913	.344
Social	198965.764	1	198965.764	3.664	.061
Sex * Exercise	11815.196	1	11815.196	.218	.643
Sex * Social	3562.685	1	3562.685	.066	.799
Exercise * Social	495370.619	1	495370.619	9.123	.004
Sex * Exercise * Social	290514.961	1	290514.961	5.350	.024
Error	2986479.334	55	54299.624		
Total	2.151E7	63			
Corrected Total	6275636.170	62			

a. R Squared = .524 (Adjusted R Squared = .464)

Table 22. Serum Corticosterone ANOVA with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Corrected Model	145661.761 ^a	3	48553.920	2.560	.075
	Intercept	2970530.065	1	2970530.065	156.633	.000
	Exercise	55861.698	1	55861.698	2.946	.097
	Social	75972.800	1	75972.800	4.006	.055
	Exercise * Social	13827.263	1	13827.263	.729	.400
	Error	531017.550	28	18964.913		
	Total	3647209.376	32			
	Corrected Total	676679.311	31			
Female	Corrected Model	865920.189 ^b	3	288640.063	3.174	.040
	Intercept	1.418E7	1	1.418E7	155.905	.000
	Exercise	6375.892	1	6375.892	.070	.793

Social	125683.531	1	125683.531	1.382	.250
Exercise * Social	758985.368	1	758985.368	8.346	.008
Error	2455461.784	27	90943.029		
Total	1.787E7	31			
Corrected Total	3321381.973	30			

a. R Squared = .215 (Adjusted R Squared = .131)

b. R Squared = .261 (Adjusted R Squared = .179)

Table 23. Center Time Ratio repeated measures ANOVA Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	811.598	1	811.598	675.905	.000
Sex	14.589	1	14.589	12.150	.001
Exercise	9.716	1	9.716	8.091	.006
Social	12.297	1	12.297	10.241	.002
Sex * Exercise	.628	1	.628	.523	.473
Sex * Social	.017	1	.017	.015	.905
Exercise * Social	.018	1	.018	.015	.904
Sex * Exercise * Social	.022	1	.022	.018	.893
Error	67.242	56	1.201		

Table 24. Center Time Ratio repeated measures ANOVA Within-Subject Effects

Tests of Within-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Time	31.203	4	7.801	20.919	.000
Time * Sex	3.933	4	.983	2.637	.035
Time * Exercise	3.460	4	.865	2.320	.058
Time * Social	10.527	4	2.632	7.057	.000
Time * Sex * Exercise	1.269	4	.317	.851	.494
Time * Sex * Social	1.397	4	.349	.936	.444
Time * Exercise * Social	1.458	4	.365	.978	.421
Time * Sex * Exercise * Social	1.226	4	.307	.822	.512
Error(Time)	83.528	224	.373		

Table 25. Center Time Ratio repeated measures ANOVA with file split by sex,
Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	521.905	1	521.905	394.976	.000
	Exercise	2.703	1	2.703	2.045	.164
	Social	6.620	1	6.620	5.010	.033
	Exercise * Social	.040	1	.040	.030	.864
	Error	36.998	28	1.321		
Female	Intercept	304.281	1	304.281	281.702	.000
	Exercise	7.641	1	7.641	7.074	.013
	Social	5.694	1	5.694	5.272	.029
	Exercise * Social	.000	1	.000	.000	.992
	Error	30.244	28	1.080		

Table 26. Center Time Ratio repeated measures ANOVA with file split by sex,
Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Time	.875	3.514	9	.941	.934	1.000	.250
Female	Time	.446	21.330	9	.011	.748	.938	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 27. Center Time Ratio repeated measures ANOVA with file split by sex,
Within-Subject Effects

Tests of Within-Subjects Effects							
Sex	Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Time	Sphericity Assumed	28.026	4	7.006	17.846	.000
		Greenhouse- Geisser	28.026	3.735	7.504	17.846	.000
	Time * Exercise	Sphericity Assumed	2.914	4	.728	1.856	.123
		Greenhouse- Geisser	2.914	3.735	.780	1.856	.128
	Time * Social	Sphericity Assumed	6.957	4	1.739	4.430	.002
		Greenhouse- Geisser	6.957	3.735	1.863	4.430	.003
	Time * Exercise * Social	Sphericity Assumed	.356	4	.089	.227	.923
		Greenhouse- Geisser	.356	3.735	.095	.227	.913
	Error(Time)	Sphericity Assumed	43.972	112	.393		
		Greenhouse- Geisser	43.972	104.577	.420		
Female	Time	Sphericity Assumed	7.110	4	1.778	5.033	.001
		Greenhouse- Geisser	7.110	2.993	2.376	5.033	.003
	Time * Exercise	Sphericity Assumed	1.815	4	.454	1.285	.280
		Greenhouse- Geisser	1.815	2.993	.606	1.285	.285
	Time * Social	Sphericity Assumed	4.967	4	1.242	3.516	.010

Time * Exercise * Social	Greenhouse- Geisser	4.967	2.993	1.659	3.516	.019
	Sphericity Assumed	2.329	4	.582	1.648	.167
	Greenhouse- Geisser	2.329	2.993	.778	1.648	.185
Error(Time)	Sphericity Assumed	39.556	112	.353		
	Greenhouse- Geisser	39.556	83.805	.472		

Table 28. Center Time Ratio Stress Period ANOVA (with pre-stress as a covariate)

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	4.726 ^a	8	.591	1.700	.119
Intercept	30.106	1	30.106	86.612	.000
CtrRationW1	.008	1	.008	.024	.878
Sex	.847	1	.847	2.437	.124
Exercise	.687	1	.687	1.978	.165
Social	1.807	1	1.807	5.197	.027
Sex * Exercise	.311	1	.311	.894	.349
Sex * Social	.394	1	.394	1.133	.292
Exercise * Social	.551	1	.551	1.585	.213
Sex * Exercise * Social	.086	1	.086	.247	.621
Error	19.118	55	.348		
Total	138.294	64			
Corrected Total	23.844	63			

a. R Squared = .198 (Adjusted R Squared = .082)

Table 29. Center Time Ratio Stress Period ANOVA (with pre-stress as a covariate) with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Corrected Model	.403 ^a	4	.101	.299	.876
	Intercept	16.218	1	16.218	48.226	.000
	CtrRationW1	.011	1	.011	.032	.859
	Exercise	.042	1	.042	.126	.725
	Social	.232	1	.232	.690	.413
	Exercise * Social	.108	1	.108	.322	.575
	Error	9.080	27	.336		
	Total	77.096	32			
	Corrected Total	9.483	31			
Female	Corrected Model	3.506 ^b	4	.876	2.369	.078
	Intercept	13.658	1	13.658	36.915	.000
	CtrRationW1	.046	1	.046	.124	.728
	Exercise	.915	1	.915	2.473	.127
	Social	1.936	1	1.936	5.233	.030
	Exercise * Social	.564	1	.564	1.523	.228
	Error	9.990	27	.370		
	Total	61.198	32			
	Corrected Total	13.495	31			

a. R Squared = .042 (Adjusted R Squared = -.099)

b. R Squared = .260 (Adjusted R Squared = .150)

Table 30. Center Time Ratio Resilience analysis (post-stress period repeated measures ANOVA with stress period as a covariate), Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	40.917	1	40.917	49.578	.000
Stress Period	10.750	1	10.750	13.025	.001
Sex	11.169	1	11.169	13.533	.001
Exercise	6.288	1	6.288	7.619	.008
Social	7.677	1	7.677	9.302	.004
Sex * Exercise	.023	1	.023	.028	.868
Sex * Social	1.589	1	1.589	1.925	.171
Exercise * Social	.250	1	.250	.303	.584
Sex * Exercise * Social	2.848E-5	1	2.848E-5	.000	.995
Error	45.392	55	.825		

Table 31. Center Time Ratio Resilience analysis (post-stress period repeated measures ANOVA with stress period as a covariate), Within-Subject Effects

Tests of Within-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Resilience	.342	2	.171	.485	.617
Resilience * Stress Period	.855	2	.427	1.212	.301
Resilience * Sex	.351	2	.175	.497	.609
Resilience * Exercise	1.634	2	.817	2.316	.103
Resilience * Social	4.300	2	2.150	6.096	.003
Resilience * Sex * Exercise	1.051	2	.525	1.490	.230
Resilience * Sex * Social	.131	2	.066	.186	.831
Resilience * Exercise * Social	.488	2	.244	.692	.503
Resilience * Sex * Exercise * Social	.140	2	.070	.198	.821
Error(Resilience)	38.790	110	.353		

Table 32. Center Time Ratio Resilience analysis (post-stress period repeated measures ANOVA with stress period as a covariate) with file split by sex, Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	23.522	1	23.522	21.995	.000
	Stress Period	5.586	1	5.586	5.224	.030
	Exercise	2.847	1	2.847	2.662	.114
	Social	8.373	1	8.373	7.829	.009
	Exercise * Social	.138	1	.138	.129	.722
	Error	28.875	27	1.069		
Female	Intercept	16.527	1	16.527	27.049	.000
	Stress Period	5.184	1	5.184	8.484	.007
	Exercise	3.443	1	3.443	5.635	.025
	Social	1.189	1	1.189	1.946	.174
	Exercise * Social	.105	1	.105	.172	.682
	Error	16.497	27	.611		

Table 33. Center Time Ratio Resilience analysis (post-stress period repeated measures ANOVA with stress period as a covariate) with file split by sex, Within-Subject Effects

Tests of Within-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Resilience	.475	2	.238	.609	.548
	Resilience * Stress Period	.515	2	.257	.659	.522
	Resilience * Exercise	2.033	2	1.016	2.602	.083
	Resilience * Social	1.768	2	.884	2.264	.114
	Resilience * Exercise * Social	.120	2	.060	.153	.858
	Error(Resilience)	21.091	54	.391		
Female	Resilience	.033	2	.017	.051	.950
	Resilience * Stress Period	.424	2	.212	.650	.526

Resilience * Exercise	.666	2	.333	1.021	.367
Resilience * Social	2.427	2	1.213	3.720	.031
Resilience * Exercise * Social	.451	2	.226	.692	.505
Error(Resilience)	17.615	54	.326		

Table 34. Horizontal Activity repeated measures ANOVA, Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	1.563E11	1	1.563E11	2824.792	.000
Sex	1.152E9	1	1.152E9	20.816	.000
Exercise	3.089E8	1	3.089E8	5.582	.022
Social	5.493E8	1	5.493E8	9.928	.003
Sex * Exercise	1.083E7	1	1.083E7	.196	.660
Sex * Social	4.992E7	1	4.992E7	.902	.346
Exercise * Social	1.723E7	1	1.723E7	.311	.579
Sex * Exercise * Social	38019.200	1	38019.200	.001	.979
Error	3.098E9	56	5.533E7		

Table 35. Horizontal Activity repeated measures ANOVA, Within-Subject Effects

Tests of Within-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Time	1.428E9	4	3.570E8	14.943	.000
Time * Sex	2.570E8	4	6.425E7	2.689	.032
Time * Exercise	2.276E9	4	5.690E8	23.818	.000
Time * Social	5.122E7	4	1.281E7	.536	.709
Time * Sex * Exercise	2.090E7	4	5225122.558	.219	.928
Time * Sex * Social	9.916E7	4	2.479E7	1.038	.389
Time * Exercise * Social	1.325E8	4	3.313E7	1.387	.239
Time * Sex * Exercise * Social	8.538E7	4	2.134E7	.894	.469
Error(Time)	5.351E9	224	2.389E7		

Table 36. Horizontal Activity repeated measures ANOVA with file split by sex,
Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	6.530E10	1	6.530E10	989.673	.000
	Exercise	1.020E8	1	1.020E8	1.546	.224
	Social	1.340E8	1	1.340E8	2.031	.165
	Exercise * Social	9442008.900	1	9442008.900	.143	.708
	Error	1.848E9	28	6.599E7		
Female	Intercept	9.214E10	1	9.214E10	2062.617	.000
	Exercise	2.177E8	1	2.177E8	4.873	.036
	Social	4.652E8	1	4.652E8	10.414	.003
	Exercise * Social	7823402.500	1	7823402.500	.175	.679
	Error	1.251E9	28	4.467E7		

Table 37. Horizontal Activity repeated measures ANOVA with file split by sex,
Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Time	.344	28.195	9	.001	.642	.788	.250
Female	Time	.807	5.653	9	.775	.913	1.000	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 38. Horizontal Activity repeated measures ANOVA with file split by sex,
Within-Subject Effects

Tests of Within-Subjects Effects							
Sex	Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Time	Sphericity Assumed	1.180E9	4	2.949E8	14.105	.000
		Greenhouse- Geisser	1.180E9	2.568	4.593E8	14.105	.000
	Time * Exercise	Sphericity Assumed	1.281E9	4	3.203E8	15.321	.000
		Greenhouse- Geisser	1.281E9	2.568	4.989E8	15.321	.000
	Time * Social	Sphericity Assumed	1.121E8	4	2.803E7	1.341	.259
		Greenhouse- Geisser	1.121E8	2.568	4.366E7	1.341	.269
	Time * Exercise * Social	Sphericity Assumed	6.597E7	4	1.649E7	.789	.535
		Greenhouse- Geisser	6.597E7	2.568	2.569E7	.789	.487
	Error(Time)	Sphericity Assumed	2.342E9	112	2.091E7		
		Greenhouse- Geisser	2.342E9	71.911	3.256E7		
Female	Time	Sphericity Assumed	5.053E8	4	1.263E8	4.701	.002
		Greenhouse- Geisser	5.053E8	3.650	1.384E8	4.701	.002
	Time * Exercise	Sphericity Assumed	1.016E9	4	2.539E8	9.449	.000
		Greenhouse- Geisser	1.016E9	3.650	2.782E8	9.449	.000
	Time * Social	Sphericity Assumed	3.826E7	4	9565568.819	.356	.839

	Greenhouse-Geisser	3.826E7	3.650	1.048E7	.356	.823
Time * Exercise * Social	Sphericity Assumed	1.519E8	4	3.798E7	1.413	.234
	Greenhouse-Geisser	1.519E8	3.650	4.162E7	1.413	.238
Error(Time)	Sphericity Assumed	3.010E9	112	2.687E7		
	Greenhouse-Geisser	3.010E9	102.208	2.945E7		

Table 39. Horizontal Activity Stress Period ANOVA (with pre-stress as a covariate)

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	8.630E8	8	1.079E8	3.367	.003
Intercept	1.637E9	1	1.637E9	51.096	.000
PreStress Period	2.666E8	1	2.666E8	8.321	.006
Sex	7.090E7	1	7.090E7	2.213	.143
Exercise	8.834E7	1	8.834E7	2.757	.103
Social	2.888E7	1	2.888E7	.901	.347
Sex * Exercise	1506953.231	1	1506953.231	.047	.829
Sex * Social	6.584E7	1	6.584E7	2.055	.157
Exercise * Social	6.661E7	1	6.661E7	2.079	.155
Sex * Exercise * Social	620197.006	1	620197.006	.019	.890
Error	1.762E9	55	3.204E7		
Total	4.010E10	64			
Corrected Total	2.625E9	63			

a. R Squared = .329 (Adjusted R Squared = .231)

Table 40. Horizontal Activity Stress period ANOVA (with pre-stress as a covariate) with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Corrected Model	5.119E8	4	1.280E8	5.489	.002
	Intercept	5.113E8	1	5.113E8	21.930	.000
	PreStress Period	4.173E8	1	4.173E8	17.900	.000
	Exercise	5.340E7	1	5.340E7	2.290	.142
	Social	1.088E7	1	1.088E7	.467	.500
	Exercise * Social	4.551E7	1	4.551E7	1.952	.174
	Error	6.295E8	27	2.331E7		
	Total	1.691E10	32			
	Corrected Total	1.141E9	31			
Female	Corrected Model	2.505E8	4	6.262E7	1.731	.172
	Intercept	1.259E9	1	1.259E9	34.807	.000
	PreStress Period	5351063.165	1	5351063.165	.148	.704
	Exercise	3.976E7	1	3.976E7	1.099	.304
	Social	1.279E8	1	1.279E8	3.537	.071
	Exercise * Social	4.830E7	1	4.830E7	1.335	.258
	Error	9.767E8	27	3.617E7		
	Total	2.320E10	32			
	Corrected Total	1.227E9	31			

a. R Squared = .449 (Adjusted R Squared = .367)

b. R Squared = .204 (Adjusted R Squared = .086)

Table 41. Horizontal Activity Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	3.214E9	1	3.214E9	99.900	.000
Stress Period	2.120E8	1	2.120E8	6.592	.013
Sex	3.013E8	1	3.013E8	9.366	.003
Exercise	1.496E8	1	1.496E8	4.649	.035
Social	2.928E8	1	2.928E8	9.102	.004
Sex * Exercise	2.186E7	1	2.186E7	.680	.413
Sex * Social	954572.369	1	954572.369	.030	.864
Exercise * Social	2.584E7	1	2.584E7	.803	.374
Sex * Exercise * Social	2.159E7	1	2.159E7	.671	.416
Error	1.769E9	55	3.217E7		

Table 42. Horizontal Activity Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Mauchly's Test of Sphericity

Mauchly's Test of Sphericity							
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Resilience	.892	6.182	2	.045	.902	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 43. Horizontal Activity Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Within-Subject Effects

Tests of Within-Subjects Effects						
Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Resilience	Sphericity Assumed	1.897E8	2	9.483E7	4.547	.013
	Greenhouse-Geisser	1.897E8	1.805	1.051E8	4.547	.016
Resilience * Stress Period	Sphericity Assumed	1.179E8	2	5.897E7	2.827	.063
	Greenhouse-Geisser	1.179E8	1.805	6.535E7	2.827	.069
Resilience * Sex	Sphericity Assumed	1.181E8	2	5.906E7	2.832	.063
	Greenhouse-Geisser	1.181E8	1.805	6.545E7	2.832	.069
Resilience * Exercise	Sphericity Assumed	1.929E9	2	9.647E8	46.250	.000
	Greenhouse-Geisser	1.929E9	1.805	1.069E9	46.250	.000
Resilience * Social	Sphericity Assumed	6.354E7	2	3.177E7	1.523	.223
	Greenhouse-Geisser	6.354E7	1.805	3.521E7	1.523	.224
Resilience * Sex * Exercise	Sphericity Assumed	9694695.367	2	4847347.683	.232	.793
	Greenhouse-Geisser	9694695.367	1.805	5371656.369	.232	.770
Resilience * Sex * Social	Sphericity Assumed	3.545E7	2	1.772E7	.850	.430
	Greenhouse-Geisser	3.545E7	1.805	1.964E7	.850	.420
Resilience * Exercise * Social	Sphericity Assumed	2.764E7	2	1.382E7	.663	.518
	Greenhouse-Geisser	2.764E7	1.805	1.531E7	.663	.503
Resilience * Sex * Exercise * Social	Sphericity Assumed	3.653E7	2	1.827E7	.876	.419
	Greenhouse-Geisser	3.653E7	1.805	2.024E7	.876	.410
Error(Resilience)	Sphericity Assumed	2.294E9	110	2.086E7		
	Greenhouse-Geisser	2.294E9	99.263	2.311E7		

Table 44. Horizontal Activity Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	1.352E9	1	1.352E9	46.376	.000
	Stress Period	2.163E8	1	2.163E8	7.415	.011
	Exercise	1.940E7	1	1.940E7	.665	.422
	Social	1.345E8	1	1.345E8	4.613	.041
	Exercise * Social	1275685.383	1	1275685.383	.044	.836
	Error	7.874E8	27	2.916E7		
Female	Intercept	1.899E9	1	1.899E9	54.267	.000
	Stress Period	3.304E7	1	3.304E7	.944	.340
	Exercise	1.639E8	1	1.639E8	4.685	.039
	Social	1.885E8	1	1.885E8	5.387	.028
	Exercise * Social	3.288E7	1	3.288E7	.940	.341
	Error	9.446E8	27	3.499E7		

Table 45. Horizontal Activity Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Resilience	.734	8.029	2	.018	.790	.955	.500
Female	Resilience	.958	1.114	2	.573	.960	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 46. Horizontal Activity Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Within-Subject Effects

Tests of Within-Subjects Effects			Type III Sum of Squares	df	Mean Square	F	Sig.
Sex	Source						
Male	Resilience	Sphericity Assumed	3.977E8	2	1.989E8	12.660	.000
		Greenhouse- Geisser	3.977E8	1.580	2.517E8	12.660	.000
	Resilience * Stress Period	Sphericity Assumed	2.375E8	2	1.187E8	7.560	.001
		Greenhouse- Geisser	2.375E8	1.580	1.503E8	7.560	.003
	Resilience * Exercise	Sphericity Assumed	9.730E8	2	4.865E8	30.976	.000
		Greenhouse- Geisser	9.730E8	1.580	6.158E8	30.976	.000
	Resilience * Social	Sphericity Assumed	8.056E7	2	4.028E7	2.565	.086
		Greenhouse- Geisser	8.056E7	1.580	5.098E7	2.565	.100
	Resilience * Exercise * Social	Sphericity Assumed	1.122E7	2	5608043.908	.357	.701
		Greenhouse- Geisser	1.122E7	1.580	7097953.240	.357	.652
	Error(Resilience)	Sphericity Assumed	8.482E8	54	1.571E7		
		Greenhouse- Geisser	8.482E8	42.665	1.988E7		
Female	Resilience	Sphericity Assumed	4421579.172	2	2210789.586	.090	.914
		Greenhouse- Geisser	4421579.172	1.920	2303472.125	.090	.907

Resilience * Stress Period	Sphericity Assumed	481399.781	2	240699.891	.010	.990
	Greenhouse- Geisser	481399.781	1.920	250790.709	.010	.989
Resilience * Exercise	Sphericity Assumed	9.447E8	2	4.724E8	19.233	.000
	Greenhouse- Geisser	9.447E8	1.920	4.922E8	19.233	.000
Resilience * Social	Sphericity Assumed	2.350E7	2	1.175E7	.478	.622
	Greenhouse- Geisser	2.350E7	1.920	1.224E7	.478	.615
Resilience * Exercise * Social	Sphericity Assumed	3.591E7	2	1.795E7	.731	.486
	Greenhouse- Geisser	3.591E7	1.920	1.871E7	.731	.481
Error(Resilience)	Sphericity Assumed	1.326E9	54	2.456E7		
	Greenhouse- Geisser	1.326E9	51.827	2.559E7		

Table 47. Negative Affect repeated measures ANOVA, Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	42827.513	1	42827.513	38.369	.000
Sex	17940.050	1	17940.050	16.072	.000
Exercise	11520.000	1	11520.000	10.321	.002
Social	140.450	1	140.450	.126	.724
Sex * Exercise	5330.112	1	5330.112	4.775	.033
Sex * Social	825.612	1	825.612	.740	.393
Exercise * Social	46.512	1	46.512	.042	.839
Sex * Exercise * Social	1462.050	1	1462.050	1.310	.257
Error	62507.700	56	1116.209		

Table 48. Negative Affect repeated measures ANOVA, Mauchly's Test of Sphericity

Mauchly's Test of Sphericity							
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Time	.481	39.834	9	.000	.719	.857	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 49. Negative Affect repeated measures ANOVA, Within-Subject Effects

Tests of Within-Subjects Effects						
Time	Sphericity Assumed	2052.081	4	513.020	1.560	.186
	Greenhouse-Geisser	2052.081	2.876	713.462	1.560	.203
Time * Sex	Sphericity Assumed	3858.669	4	964.667	2.933	.022
	Greenhouse-Geisser	3858.669	2.876	1341.572	2.933	.037
Time * Exercise	Sphericity Assumed	3012.719	4	753.180	2.290	.061
	Greenhouse-Geisser	3012.719	2.876	1047.454	2.290	.083
Time * Social	Sphericity Assumed	1763.581	4	440.895	1.341	.256
	Greenhouse-Geisser	1763.581	2.876	613.157	1.341	.264
Time * Sex * Exercise	Sphericity Assumed	5017.356	4	1254.339	3.814	.005
	Greenhouse-Geisser	5017.356	2.876	1744.421	3.814	.012
Time * Sex * Social	Sphericity Assumed	2104.169	4	526.042	1.600	.175
	Greenhouse-Geisser	2104.169	2.876	731.572	1.600	.193
Time * Exercise * Social	Sphericity Assumed	1702.019	4	425.505	1.294	.273
	Greenhouse-Geisser	1702.019	2.876	591.753	1.294	.279
Time * Sex * Exercise * Social	Sphericity Assumed	1277.106	4	319.277	.971	.424
	Greenhouse-Geisser	1277.106	2.876	444.021	.971	.405
Error(Time)	Sphericity Assumed	73666.300	224	328.867		
	Greenhouse-Geisser	73666.300	161.069	457.359		

Table 50. Negative Affect repeated measures ANOVA with file split by sex,
Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	2665.056	1	2665.056	6.344	.018
	Exercise	589.056	1	589.056	1.402	.246
	Social	823.556	1	823.556	1.960	.172
	Exercise * Social	493.506	1	493.506	1.175	.288
	Error	11762.225	28	420.079		
Female	Intercept	58102.506	1	58102.506	32.059	.000
	Exercise	16261.056	1	16261.056	8.972	.006
	Social	142.506	1	142.506	.079	.781
	Exercise * Social	1015.056	1	1015.056	.560	.460
	Error	50745.475	28	1812.338		

Table 51. Negative Affect repeated measures ANOVA with file split by sex,
Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Time	.001	193.797	9	.000	.280	.314	.250
Female	Time	.460	20.511	9	.015	.690	.855	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 52. Negative Affect repeated measures ANOVA with file split by sex,
Within-Subject Effects

Tests of Within-Subjects Effects							
Sex	Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Time	Sphericity Assumed	181.600	4	45.400	.524	.718
		Greenhouse- Geisser	181.600	1.119	162.352	.524	.494
	Time * Exercise	Sphericity Assumed	418.600	4	104.650	1.208	.312
		Greenhouse- Geisser	418.600	1.119	374.232	1.208	.287
	Time * Social	Sphericity Assumed	335.225	4	83.806	.967	.428
		Greenhouse- Geisser	335.225	1.119	299.694	.967	.343
	Time * Exercise * Social	Sphericity Assumed	224.025	4	56.006	.646	.631
		Greenhouse- Geisser	224.025	1.119	200.280	.646	.444
	Error(Time)	Sphericity Assumed	9704.150	112	86.644		
		Greenhouse- Geisser	9704.150	31.320	309.843		
Female	Time	Sphericity Assumed	5729.150	4	1432.287	2.508	.046
		Greenhouse- Geisser	5729.150	2.761	2074.972	2.508	.070
	Time * Exercise	Sphericity Assumed	7611.475	4	1902.869	3.332	.013
		Greenhouse- Geisser	7611.475	2.761	2756.709	3.332	.027
	Time * Social	Sphericity Assumed	3532.525	4	883.131	1.546	.194

Time * Exercise * Social	Greenhouse- Geisser	3532.525	2.761	1279.403	1.546	.212
	Sphericity Assumed	2755.100	4	688.775	1.206	.312
	Greenhouse- Geisser	2755.100	2.761	997.837	1.206	.312
Error(Time)	Sphericity Assumed	63962.150	112	571.091		
	Greenhouse- Geisser	63962.150	77.310	827.346		

Table 53. Negative Affect Stress period ANOVA (with pre-stress as a covariate)

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	10966.903 ^a	8	1370.863	4.620	.000
Intercept	1423.066	1	1423.066	4.796	.033
PreStress Period	7492.966	1	7492.966	25.252	.000
Sex	1206.417	1	1206.417	4.066	.049
Exercise	5.363	1	5.363	.018	.894
Social	4.333	1	4.333	.015	.904
Sex * Exercise	35.870	1	35.870	.121	.729
Sex * Social	20.436	1	20.436	.069	.794
Exercise * Social	28.121	1	28.121	.095	.759
Sex * Exercise * Social	6.049	1	6.049	.020	.887
Error	16320.034	55	296.728		
Total	34052.000	64			
Corrected Total	27286.937	63			

a. R Squared = .402 (Adjusted R Squared = .315)

Table 54. Negative Affect Stress period ANOVA (with pre-stress as a covariate)
with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Corrected Model	7168.853 ^a	4	1792.213	511.264	.000
	Intercept	13.991	1	13.991	3.991	.056
	PreStress Period	6276.603	1	6276.603	1790.523	.000
	Exercise	3.622	1	3.622	1.033	.318
	Social	1.376	1	1.376	.393	.536
	Exercise * Social	2.030	1	2.030	.579	.453
	Error	94.647	27	3.505		
	Total	7948.000	32			
	Corrected Total	7263.500	31			
Female	Corrected Model	1790.509 ^b	4	447.627	.747	.569
	Intercept	1973.278	1	1973.278	3.292	.081
	PreStress Period	1256.384	1	1256.384	2.096	.159
	Exercise	14.401	1	14.401	.024	.878
	Social	42.368	1	42.368	.071	.792
	Exercise * Social	16.660	1	16.660	.028	.869
	Error	16185.366	27	599.458		
	Total	26104.000	32			
	Corrected Total	17975.875	31			

a. R Squared = .987 (Adjusted R Squared = .985)

b. R Squared = .100 (Adjusted R Squared = -.034)

Table 55. Negative Affect Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	13810.072	1	13810.072	14.450	.000
Stress Period	9419.358	1	9419.358	9.856	.003
Sex	11046.453	1	11046.453	11.558	.001
Exercise	9535.669	1	9535.669	9.978	.003
Social	329.652	1	329.652	.345	.559
Sex * Exercise	10703.153	1	10703.153	11.199	.001
Sex * Social	252.772	1	252.772	.264	.609
Exercise * Social	429.998	1	429.998	.450	.505
Sex * Exercise * Social	333.261	1	333.261	.349	.557
Error	52564.308	55	955.715		

Table 56. Negative Affect Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Within-Subject Effects

Tests of Within-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Resilience	248.201	2	124.101	.405	.668
Resilience * Stress Period	142.017	2	71.008	.232	.794
Resilience * Sex	502.335	2	251.168	.819	.443
Resilience * Exercise	1159.441	2	579.721	1.891	.156
Resilience * Social	1627.611	2	813.805	2.655	.075
Resilience * Sex * Exercise	448.029	2	224.015	.731	.484
Resilience * Sex * Social	1069.984	2	534.992	1.745	.179
Resilience * Exercise * Social	1389.437	2	694.718	2.266	.109
Resilience * Sex * Exercise * Social	1242.750	2	621.375	2.027	.137
Error(Resilience)	33722.567	110	306.569		

Table 57. Negative Affect Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	529.387	1	529.387	31.653	.000
	Stress Period	746.804	1	746.804	44.652	.000
	Exercise	2.088	1	2.088	.125	.727
	Social	41.250	1	41.250	2.466	.128
	Exercise * Social	7.217	1	7.217	.431	.517
	Error	451.571	27	16.725		
Female	Intercept	17939.922	1	17939.922	9.424	.005
	Stress Period	9386.880	1	9386.880	4.931	.035
	Exercise	20065.900	1	20065.900	10.541	.003
	Social	683.703	1	683.703	.359	.554
	Exercise * Social	739.077	1	739.077	.388	.538
	Error	51398.411	27	1903.645		

Table 58. Negative Affect Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Within-Subject Effects

Tests of Within-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Resilience	136.068	2	68.034	6.667	.003
	Resilience * Stress Period	540.460	2	270.230	26.482	.000
	Resilience * Exercise	19.642	2	9.821	.962	.388
	Resilience * Social	18.716	2	9.358	.917	.406
	Resilience * Exercise * Social	11.133	2	5.567	.546	.583
	Error(Resilience)	551.040	54	10.204		
Female	Resilience	1040.576	2	520.288	.869	.425
	Resilience * Stress Period	425.041	2	212.521	.355	.703
	Resilience * Exercise	1590.447	2	795.224	1.328	.274
	Resilience * Social	2495.225	2	1247.612	2.083	.134
	Resilience * Exercise * Social	2614.312	2	1307.156	2.182	.123
	Error(Resilience)	32348.042	54	599.038		

Table 59. Positive Affect repeated measures ANOVA, Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	73144.513	1	73144.513	72.040	.000
Sex	40951.250	1	40951.250	40.333	.000
Exercise	18819.112	1	18819.112	18.535	.000
Social	159.613	1	159.613	.157	.693
Sex * Exercise	9202.050	1	9202.050	9.063	.004
Sex * Social	1377.800	1	1377.800	1.357	.249
Exercise * Social	70.312	1	70.312	.069	.793
Sex * Exercise * Social	1656.200	1	1656.200	1.631	.207
Error	56858.750	56	1015.335		

Table 60. Positive Affect repeated measures ANOVA, Mauchly's Test of Sphericity

Mauchly's Test of Sphericity							
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Time	.699	19.493	9	.021	.854	1.000	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 61. Positive Affect repeated measures ANOVA, Within-Subject Effects

Tests of Within-Subjects Effects						
Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Time	Sphericity Assumed	1197.144	4	299.286	1.236	.297
	Greenhouse-Geisser	1197.144	3.417	350.387	1.236	.298
Time * Sex	Sphericity Assumed	2558.281	4	639.570	2.641	.035
	Greenhouse-Geisser	2558.281	3.417	748.772	2.641	.043
Time * Exercise	Sphericity Assumed	1772.419	4	443.105	1.829	.124
	Greenhouse-Geisser	1772.419	3.417	518.762	1.829	.135
Time * Social	Sphericity Assumed	1343.231	4	335.808	1.386	.240
	Greenhouse-Geisser	1343.231	3.417	393.144	1.386	.245
Time * Sex * Exercise	Sphericity Assumed	4392.231	4	1098.058	4.534	.002
	Greenhouse-Geisser	4392.231	3.417	1285.543	4.534	.003
Time * Sex * Social	Sphericity Assumed	1004.294	4	251.073	1.037	.389
	Greenhouse-Geisser	1004.294	3.417	293.942	1.037	.383
Time * Exercise * Social	Sphericity Assumed	1808.656	4	452.164	1.867	.117
	Greenhouse-Geisser	1808.656	3.417	529.368	1.867	.128
Time * Sex * Exercise * Social	Sphericity Assumed	1061.644	4	265.411	1.096	.359
	Greenhouse-Geisser	1061.644	3.417	310.728	1.096	.356
Error(Time)	Sphericity Assumed	54254.500	224	242.208		
	Greenhouse-Geisser	54254.500	191.332	283.563		

Table 62. Positive Affect repeated measures ANOVA with file split by sex,
Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	2318.006	1	2318.006	5.244	.030
	Exercise	851.006	1	851.006	1.925	.176
	Social	1237.656	1	1237.656	2.800	.105
	Exercise * Social	522.006	1	522.006	1.181	.286
	Error	12376.725	28	442.026		
Female	Intercept	111777.756	1	111777.756	70.360	.000
	Exercise	27170.156	1	27170.156	17.103	.000
	Social	299.756	1	299.756	.189	.667
	Exercise * Social	1204.506	1	1204.506	.758	.391
	Error	44482.025	28	1588.644		

Table 63. Positive Affect repeated measures ANOVA with file split by sex,
Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Time	.018	106.752	9	.000	.353	.406	.250
Female	Time	.755	7.408	9	.595	.883	1.000	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 64. Positive Affect repeated measures ANOVA with file split by sex,
Within-Subject Effects

Tests of Within-Subjects Effects							
Sex	Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Time	Sphericity Assumed	132.087	4	33.022	.536	.709
		Greenhouse- Geisser	132.087	1.411	93.587	.536	.528
	Time * Exercise	Sphericity Assumed	326.712	4	81.678	1.327	.264
		Greenhouse- Geisser	326.712	1.411	231.484	1.327	.269
	Time * Social	Sphericity Assumed	56.563	4	14.141	.230	.921
		Greenhouse- Geisser	56.563	1.411	40.076	.230	.717
	Time * Exercise * Social	Sphericity Assumed	138.338	4	34.584	.562	.691
		Greenhouse- Geisser	138.338	1.411	98.016	.562	.516
	Error(Time)	Sphericity Assumed	6895.900	112	61.571		
		Greenhouse- Geisser	6895.900	39.519	174.497		
Female	Time	Sphericity Assumed	3623.337	4	905.834	2.142	.080
		Greenhouse- Geisser	3623.337	3.531	1026.007	2.142	.089
	Time * Exercise	Sphericity Assumed	5837.938	4	1459.484	3.452	.011
		Greenhouse- Geisser	5837.938	3.531	1653.107	3.452	.014
	Time * Social	Sphericity Assumed	2290.962	4	572.741	1.354	.254

Time * Exercise * Social	Greenhouse-Geisser	2290.962	3.531	648.723	1.354	.258
	Sphericity Assumed	2731.962	4	682.991	1.615	.175
	Greenhouse-Geisser	2731.962	3.531	773.600	1.615	.183
Error(Time)	Sphericity Assumed	47358.600	112	422.845		
	Greenhouse-Geisser	47358.600	98.882	478.941		

Table 65. Positive Affect Stress period ANOVA (with pre-stress as a covariate)

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	14872.501 ^a	8	1859.063	4.929	.000
Intercept	1630.915	1	1630.915	4.324	.042
PreStress Period	7310.266	1	7310.266	19.381	.000
Sex	1148.554	1	1148.554	3.045	.087
Exercise	96.266	1	96.266	.255	.615
Social	8.724	1	8.724	.023	.880
Sex * Exercise	5.786	1	5.786	.015	.902
Sex * Social	127.953	1	127.953	.339	.563
Exercise * Social	59.296	1	59.296	.157	.693
Sex * Exercise * Social	16.887	1	16.887	.045	.833
Error	20745.359	55	377.188		
Total	47363.000	64			
Corrected Total	35617.859	63			

a. R Squared = .418 (Adjusted R Squared = .333)

Table 66. Positive Affect Stress period ANOVA (with pre-stress as a covariate)
with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Corrected Model	6586.139 ^a	4	1646.535	41.871	.000
	Intercept	.232	1	.232	.006	.939
	PreStress Period	5553.514	1	5553.514	141.226	.000
	Exercise	1.987	1	1.987	.051	.824
	Social	33.734	1	33.734	.858	.363
	Exercise * Social	18.465	1	18.465	.470	.499
	Error	1061.736	27	39.324		
	Total	8314.000	32			
	Corrected Total	7647.875	31			
Female	Corrected Model	4032.464 ^b	4	1008.116	1.450	.245
	Intercept	3129.922	1	3129.922	4.502	.043
	PreStress Period	2668.870	1	2668.870	3.839	.060
	Exercise	186.564	1	186.564	.268	.609
	Social	90.112	1	90.112	.130	.722
	Exercise * Social	.005	1	.005	.000	.998
	Error	18771.505	27	695.241		
	Total	39049.000	32			
	Corrected Total	22803.969	31			

a. R Squared = .861 (Adjusted R Squared = .841)

b. R Squared = .177 (Adjusted R Squared = .055)

Table 67. Positive Affect Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	18742.944	1	18742.944	39.619	.000
Stress Period	10246.819	1	10246.819	21.660	.000
Sex	16985.326	1	16985.326	35.903	.000
Exercise	11845.018	1	11845.018	25.038	.000
Social	445.067	1	445.067	.941	.336
Sex * Exercise	12255.186	1	12255.186	25.905	.000
Sex * Social	1.055	1	1.055	.002	.963
Exercise * Social	290.608	1	290.608	.614	.437
Sex * Exercise * Social	380.211	1	380.211	.804	.374
Error	26019.556	55	473.083		

Table 68. Positive Affect Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Within-Subject Effects

Tests of Within-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Resilience	213.480	2	106.740	.458	.634
Resilience * Stress Period	111.655	2	55.827	.239	.787
Resilience * Sex	418.577	2	209.288	.898	.410
Resilience * Exercise	38.525	2	19.262	.083	.921
Resilience * Social	1037.419	2	518.710	2.225	.113
Resilience * Sex * Exercise	234.422	2	117.211	.503	.606
Resilience * Sex * Social	574.463	2	287.231	1.232	.296
Resilience * Exercise * Social	1654.768	2	827.384	3.549	.032
Resilience * Sex * Exercise * Social	1027.906	2	513.953	2.205	.115
Error(Resilience)	25642.345	110	233.112		

Table 69. Positive Affect Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	210.216	1	210.216	7.615	.010
	Stress Period	2357.462	1	2357.462	85.396	.000
	Exercise	.173	1	.173	.006	.937
	Social	194.420	1	194.420	7.043	.013
	Exercise * Social	3.537	1	3.537	.128	.723
	Error	745.371	27	27.606		
Female	Intercept	26370.894	1	26370.894	28.172	.000
	Stress Period	7889.828	1	7889.828	8.429	.007
	Exercise	23637.834	1	23637.834	25.252	.000
	Social	241.836	1	241.836	.258	.615
	Exercise * Social	669.286	1	669.286	.715	.405
	Error	25273.713	27	936.063		

Table 70. Positive Affect Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Within-Subject Effects

Tests of Within-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Resilience	68.941	2	34.470	2.445	.096
	Resilience * Stress Period	1589.734	2	794.867	56.371	.000
	Resilience * Exercise	6.844	2	3.422	.243	.785
	Resilience * Social	7.673	2	3.836	.272	.763
	Resilience * Exercise * Social	1.610	2	.805	.057	.945
	Error(Resilience)	761.433	54	14.101		
Female	Resilience	64.803	2	32.402	.076	.927
	Resilience * Stress Period	324.578	2	162.289	.380	.686
	Resilience * Exercise	119.128	2	59.564	.139	.870
	Resilience * Social	1361.273	2	680.636	1.593	.213
	Resilience * Exercise * Social	2551.176	2	1275.588	2.985	.059
	Error(Resilience)	23078.255	54	427.375		

Table 71. Home Cage Activity and Center Time Ratio Correlations

		Correlations				
		Center Time PreStress	Center Time Stress	Center Time PostStress 1	Center Time PostStress 2	Center Time PostStress 3
HCA PreStress	Pearson Correlation	.152	.053	-.013	-.049	.046
	Sig. (2-tailed)	.232	.678	.919	.703	.718
	N	64	64	64	64	64
HCA Stress	Pearson Correlation	-.102	.199	.207	.379**	.145
	Sig. (2-tailed)	.424	.115	.101	.002	.251
	N	64	64	64	64	64
HCA PostStress1	Pearson Correlation	.112	-.137	-.118	-.138	-.149
	Sig. (2-tailed)	.378	.280	.355	.276	.239
	N	64	64	64	64	64
HCA PostStress2	Pearson Correlation	-.138	.159	.027	.150	-.034
	Sig. (2-tailed)	.277	.210	.830	.236	.790
	N	64	64	64	64	64
HCA PostStress3	Pearson Correlation	.006	.304*	.131	.421**	.248*
	Sig. (2-tailed)	.965	.015	.303	.001	.048
	N	64	64	64	64	64

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Table 72. Home Cage Activity and Horizontal Activity Correlations

		Correlations				
		Horiz. Act.PreStress	Horiz. Act. Stress	Horiz. Act. PostStress1	Horiz. Act. PostStress2	Horiz. Act. PostStress3
HCA PreStress	Pearson Correlation	.220	.012	.017	.005	-.161
	Sig. (2-tailed)	.081	.926	.895	.971	.204
	N	64	64	64	64	64
HCA Stress	Pearson Correlation	-.148	-.177	-.137	-.115	-.323**
	Sig. (2-tailed)	.243	.161	.280	.367	.009
	N	64	64	64	64	64
HCA PostStress1	Pearson Correlation	.339**	.335**	.476**	.565**	-.182
	Sig. (2-tailed)	.006	.007	.000	.000	.149
	N	64	64	64	64	64
HCA PostStress2	Pearson Correlation	.016	.041	-.167	-.110	-.116
	Sig. (2-tailed)	.900	.746	.188	.387	.363
	N	64	64	64	64	64
HCA PostStress3	Pearson Correlation	.077	.020	.358**	.376**	-.369**
	Sig. (2-tailed)	.547	.877	.004	.002	.003
	N	64	64	64	64	64
**. Correlation is significant at the 0.01 level (2-tailed).						

Table 73. Home Cage Activity and Forced Swim Test Correlations

		Correlations				
		FST PreStress	FST Stress	FST PostStress1	FST PostStress2	FST PostStress3
HCA PreStress	Pearson Correlation	.071	.193	.245	.255*	.167
	Sig. (2-tailed)	.578	.127	.051	.042	.186
	N	64	64	64	64	64
HCA Stress	Pearson Correlation	-.163	.033	.062	.089	.035
	Sig. (2-tailed)	.199	.796	.624	.485	.782
	N	64	64	64	64	64
HCA PostStress1	Pearson Correlation	-.155	.066	.044	.034	.130
	Sig. (2-tailed)	.221	.606	.729	.787	.305
	N	64	64	64	64	64
HCA PostStress2	Pearson Correlation	-.063	-.089	.204	.001	-.053
	Sig. (2-tailed)	.620	.483	.106	.995	.675
	N	64	64	64	64	64
HCA PostStress3	Pearson Correlation	-.165	-.034	.139	.190	-.014
	Sig. (2-tailed)	.194	.789	.273	.132	.914
	N	64	64	64	64	64

*. Correlation is significant at the 0.05 level (2-tailed).

Table 74. Social Interaction Observed and Expected Frequencies

		Frequencies				
		1	2	3	4	Total
PreStress	Category	Passive	Dominant/ Submissive	Aggressive	Supportive	
	Observed N	13	7	6	6	32
	Expected N	8.0	8.0	8.0	8.0	
	Residual	5.0	-1.0	-2.0	-2.0	
Stress	Category	Passive	Dominant/ Submissive	Aggressive	Supportive	
	Observed N	12	14	6	0	32
	Expected N	8.0	8.0	8.0	8.0	
	Residual	4.0	6.0	-2.0	-8.0	
PostStress1	Category	Passive	Dominant/ Submissive	Aggressive	Supportive	
	Observed N	24	5	1	2	32
	Expected N	8.0	8.0	8.0	8.0	
	Residual	16.0	-3.0	-7.0	-6.0	
PostStress2	Category	Passive	Dominant/ Submissive	Aggressive	Supportive	
	Observed N	13	11	8	0	32
	Expected N	8.0	8.0	8.0	8.0	
	Residual	5.0	3.0	.0	-8.0	
PostStress3	Category	Passive	Dominant/ Submissive	Aggressive	Supportive	
	Observed N	17	11	4	0	32
	Expected N	8.0	8.0	8.0	8.0	
	Residual	9.0	3.0	-4.0	-8.0	

Table 75. Social Interaction Chi-Square Test Statistics

Test Statistics					
	PreStress	Stress	PostStress1	PostStress2	PostStress3
Chi-Square	4.250 ^a	15.000 ^a	43.750 ^a	12.250 ^a	21.250 ^a
df	3	3	3	3	3
Asymp. Sig.	.236	.002	.000	.007	.000

a. 0 cells (.0%) have expected frequencies less than 5. The minimum expected cell frequency is 8.0.

Table 76. Social Interaction Pre-Stress period Observed and Expected Frequencies with file split by sex

Crosstab					
			Sex		
			Male	Female	Total
PreStress	Passive	Count	7	6	13
		Expected Count	6.5	6.5	13.0
	Dominant/Submissive	Count	5	2	7
		Expected Count	3.5	3.5	7.0
	Aggressive	Count	0	6	6
		Expected Count	3.0	3.0	6.0
	Supportive	Count	4	2	6
		Expected Count	3.0	3.0	6.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 77. Social Interaction Pre-Stress period Chi-Square Test Statistics with file split by sex

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.029 ^a	3	.045
Likelihood Ratio	10.403	3	.015
Linear-by-Linear Association	.207	1	.650
N of Valid Cases	32		

a. 6 cells (75.0%) have expected count less than 5. The minimum expected count is 3.00.

Table 78. Social Interaction Pre-Stress period Observed and Expected Frequencies with file split by exercise

Crosstab			Exercise		
			No Exercise	Exercise	Total
PreStress	None/Passive	Count	6	7	13
		Expected Count	6.5	6.5	13.0
	Dominant/Submissive	Count	5	2	7
		Expected Count	3.5	3.5	7.0
	Aggressive	Count	1	5	6
		Expected Count	3.0	3.0	6.0
	Supportive	Count	4	2	6
		Expected Count	3.0	3.0	6.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 79. Social Interaction Pre-Stress period Chi-Square Test Statistics with file split by exercise

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.696 ^a	3	.195
Likelihood Ratio	4.996	3	.172
Linear-by-Linear Association	.023	1	.880
N of Valid Cases	32		

a. 6 cells (75.0%) have expected count less than 5. The minimum expected count is 3.00.

Table 80. Social Interaction Stress period Observed and Expected Frequencies with file split by sex

Crosstab					
			Sex		
			Male	Female	Total
Stress	None/Passive	Count	10	2	12
		Expected Count	6.0	6.0	12.0
	Dominant/Submissive	Count	5	9	14
		Expected Count	7.0	7.0	14.0
	Aggressive	Count	1	5	6
		Expected Count	3.0	3.0	6.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 81. Social Interaction Stress period Chi-Square Test Statistics with file split by sex

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	9.143 ^a	2	.010
Likelihood Ratio	9.892	2	.007
Linear-by-Linear Association	8.267	1	.004
N of Valid Cases	32		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 3.00.

Table 82. Social Interaction Stress period Observed and Expected Frequencies with file split by exercise

Crosstab			Exercise		
			No Exercise	Exercise	Total
Stress	None/Passive	Count	6	6	12
		Expected Count	6.0	6.0	12.0
	Dominant/Submissive	Count	6	8	14
		Expected Count	7.0	7.0	14.0
	Aggressive	Count	4	2	6
		Expected Count	3.0	3.0	6.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 83. Social Interaction Stress period Chi-Square Test Statistics with file split by exercise

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.952 ^a	2	.621
Likelihood Ratio	.966	2	.617
Linear-by-Linear Association	.230	1	.632
N of Valid Cases	32		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 3.00.

Table 84. Social Interaction Post-Stress 1 period Observed and Expected Frequencies with file split by sex

Crosstab			Sex		
			Male	Female	Total
PostStress1	None/Passive	Count	10	14	24
		Expected Count	12.0	12.0	24.0
	Dominant/Submissive	Count	5	0	5
		Expected Count	2.5	2.5	5.0
	Aggressive	Count	1	0	1
		Expected Count	.5	.5	1.0
	Supportive	Count	0	2	2
		Expected Count	1.0	1.0	2.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 85. Social Interaction Post-Stress 1 period Chi-Square Test Statistics with file split by sex

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.667 ^a	3	.034
Likelihood Ratio	11.760	3	.008
Linear-by-Linear Association	.045	1	.833
N of Valid Cases	32		

a. 6 cells (75.0%) have expected count less than 5. The minimum expected count is .50.

Table 86. Social Interaction Post-Stress 1 period Observed and Expected Frequencies with file split by exercise

Crosstab			Exercise		
			No Exercise	Exercise	Total
PostStress1	None/Passive	Count	12	12	24
		Expected Count	12.0	12.0	24.0
	Dominant/Submissive	Count	3	2	5
		Expected Count	2.5	2.5	5.0
	Aggressive	Count	1	0	1
		Expected Count	.5	.5	1.0
	Supportive	Count	0	2	2
		Expected Count	1.0	1.0	2.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 87. Social Interaction Post-Stress 1 period Chi-Square Test Statistics with file split by exercise

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.200 ^a	3	.362
Likelihood Ratio	4.360	3	.225
Linear-by-Linear Association	.401	1	.526
N of Valid Cases	32		

a. 6 cells (75.0%) have expected count less than 5. The minimum expected count is .50.

Table 88. Social Interaction Post-Stress 2 period Observed and Expected Frequencies with file split by sex

Crosstab			Sex		
			Male	Female	Total
PostStress2	None/Passive	Count	8	5	13
		Expected Count	6.5	6.5	13.0
	Dominant/Submissive	Count	3	8	11
		Expected Count	5.5	5.5	11.0
	Aggressive	Count	5	3	8
		Expected Count	4.0	4.0	8.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 89. Social Interaction Post-Stress 2 period Chi-Square Test Statistics with file split by sex

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.465 ^a	2	.177
Likelihood Ratio	3.562	2	.168
Linear-by-Linear Association	.048	1	.827
N of Valid Cases	32		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 4.00.

Table 90. Social Interaction Post-Stress 2 period Observed and Expected Frequencies with file split by exercise

Crosstab					
			Exercise		
			No Exercise	Exercise	Total
PostStress2	None/Passive	Count	6	7	13
		Expected Count	6.5	6.5	13.0
	Dominant/Submissive	Count	4	7	11
		Expected Count	5.5	5.5	11.0
	Aggressive	Count	6	2	8
		Expected Count	4.0	4.0	8.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 91. Social Interaction Post-Stress 2 period Chi-Square Test Statistics with file split by exercise

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.895 ^a	2	.235
Likelihood Ratio	2.999	2	.223
Linear-by-Linear Association	1.198	1	.274
N of Valid Cases	32		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 4.00.

Table 92. Social Interaction Post-Stress 3 period Observed and Expected Frequencies with file split by sex

Crosstab			Sex		
			Male	Female	Total
PostStress3	None/Passive	Count	8	9	17
		Expected Count	8.5	8.5	17.0
	Dominant/Submissive	Count	7	4	11
		Expected Count	5.5	5.5	11.0
	Aggressive	Count	1	3	4
		Expected Count	2.0	2.0	4.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 93. Social Interaction Post-Stress 3 period Chi-Square Test Statistics with file split by sex

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.877 ^a	2	.391
Likelihood Ratio	1.934	2	.380
Linear-by-Linear Association	.062	1	.804
N of Valid Cases	32		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 2.00.

Table 94. Social Interaction Post-Stress 3 period Observed and Expected Frequencies with file split by exercise

Crosstab			Exercise		
			No Exercise	Exercise	Total
PostStress3	None/Passive	Count	9	8	17
		Expected Count	8.5	8.5	17.0
	Dominant/Submissive	Count	4	7	11
		Expected Count	5.5	5.5	11.0
	Aggressive	Count	3	1	4
		Expected Count	2.0	2.0	4.0
	Total	Count	16	16	32
		Expected Count	16.0	16.0	32.0

Table 95. Social Interaction Post-Stress 3 period Chi-Square Test Statistics with file split by exercise

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.877 ^a	2	.391
Likelihood Ratio	1.934	2	.380
Linear-by-Linear Association	.062	1	.804
N of Valid Cases	32		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 2.00.

Table 96. Forced Swim Test repeated measures ANOVA, Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	923902.227	1	923902.227	50.780	.000
FSTTraining	245795.678	1	245795.678	13.510	.001
Sex	6195.866	1	6195.866	.341	.562
Exercise	8313.955	1	8313.955	.457	.502
Social	13826.101	1	13826.101	.760	.387
Sex * Exercise	44481.002	1	44481.002	2.445	.124
Sex * Social	4937.690	1	4937.690	.271	.604
Exercise * Social	6737.951	1	6737.951	.370	.545
Sex * Exercise * Social	43179.212	1	43179.212	2.373	.129
Error	1000685.335	55	18194.279		

Table 97. Forced Swim Test repeated measures ANOVA, Within-Subject Effects

Tests of Within-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Time	367590.906	4	91897.726	7.268	.000
Time * FSTTraining	97250.439	4	24312.610	1.923	.108
Time * Sex	34717.121	4	8679.280	.686	.602
Time * Exercise	31250.996	4	7812.749	.618	.650
Time * Social	33785.961	4	8446.490	.668	.615
Time * Sex * Exercise	32885.703	4	8221.426	.650	.627
Time * Sex * Social	16054.247	4	4013.562	.317	.866
Time * Exercise * Social	31667.936	4	7916.984	.626	.644
Time * Sex * Exercise * Social	30855.337	4	7713.834	.610	.656
Error(Time)	2781578.273	220	12643.538		

Table 98. Forced Swim Test repeated measures ANOVA with file split by sex,
Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	389955.486	1	389955.486	25.377	.000
	FSTTraining	153921.219	1	153921.219	10.017	.004
	Exercise	46234.784	1	46234.784	3.009	.094
	Social	2915.922	1	2915.922	.190	.667
	Exercise * Social	43829.304	1	43829.304	2.852	.103
	Error	414894.699	27	15366.470		
Female	Intercept	539060.123	1	539060.123	25.114	.000
	FSTTraining	98125.117	1	98125.117	4.572	.042
	Exercise	7907.806	1	7907.806	.368	.549
	Social	15079.852	1	15079.852	.703	.409
	Exercise * Social	7664.381	1	7664.381	.357	.555
	Error	579539.979	27	21464.444		

Table 99. Forced Swim Test repeated measures ANOVA with file split by sex,
Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Time	.786	6.114	9	.729	.897	1.000	.250
Female	Time	.510	17.121	9	.047	.775	1.000	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 100. Forced Swim Test repeated measures ANOVA with file split by sex,
Within-Subject Effects

Tests of Within-Subjects Effects							
Sex	Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Time	Sphericity Assumed	84229.892	4	21057.473	1.571	.187
		Greenhouse- Geisser	84229.892	3.588	23472.505	1.571	.193
	Time * FSTTraining	Sphericity Assumed	94823.788	4	23705.947	1.769	.140
		Greenhouse- Geisser	94823.788	3.588	26424.726	1.769	.148
	Time * Exercise	Sphericity Assumed	29455.706	4	7363.926	.550	.700
		Greenhouse- Geisser	29455.706	3.588	8208.478	.550	.681
	Time * Social	Sphericity Assumed	18967.358	4	4741.840	.354	.841
		Greenhouse- Geisser	18967.358	3.588	5285.670	.354	.821
	Time * Exercise * Social	Sphericity Assumed	17544.791	4	4386.198	.327	.859
		Greenhouse- Geisser	17544.791	3.588	4889.240	.327	.840
	Error(Time)	Sphericity Assumed	1447296.501	108	13400.894		
		Greenhouse- Geisser	1447296.501	96.888	14937.810		
Female	Time	Sphericity Assumed	325737.347	4	81434.337	6.734	.000
		Greenhouse- Geisser	325737.347	3.099	105098.397	6.734	.000
	Time * FSTTraining	Sphericity Assumed	30636.106	4	7659.026	.633	.640

	Greenhouse-Geisser	30636.106	3.099	9884.668	.633	.601
Time * Exercise	Sphericity Assumed	34784.121	4	8696.030	.719	.581
	Greenhouse-Geisser	34784.121	3.099	11223.016	.719	.548
Time * Social	Sphericity Assumed	27268.315	4	6817.079	.564	.689
	Greenhouse-Geisser	27268.315	3.099	8798.058	.564	.646
Time * Exercise * Social	Sphericity Assumed	43219.133	4	10804.783	.893	.471
	Greenhouse-Geisser	43219.133	3.099	13944.553	.893	.451
Error(Time)	Sphericity Assumed	1306072.317	108	12093.262		
	Greenhouse-Geisser	1306072.317	83.683	15607.452		

Table 101. Forced Swim Test Stress period ANOVA (with pre-stress as a covariate)

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	76291.552 ^a	8	9536.444	.878	.541
Intercept	82044.324	1	82044.324	7.555	.008
FST1	11095.929	1	11095.929	1.022	.317
Sex	258.921	1	258.921	.024	.878
Exercise	494.529	1	494.529	.046	.832
Social	48.595	1	48.595	.004	.947
Sex * Exercise	37626.842	1	37626.842	3.465	.068
Sex * Social	1501.439	1	1501.439	.138	.711
Exercise * Social	1296.293	1	1296.293	.119	.731
Sex * Exercise * Social	12835.946	1	12835.946	1.182	.282
Error	597265.004	55	10859.364		

Total	813479.310	64			
Corrected Total	673556.556	63			

a. R Squared = .113 (Adjusted R Squared = -.016)

Table 102. Forced Swim Test Stress period ANOVA (with pre-stress as a covariate) with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Corrected Model	51130.670 ^a	4	12782.667	1.258	.311
	Intercept	77253.981	1	77253.981	7.601	.010
	FST1	11053.854	1	11053.854	1.088	.306
	Exercise	28213.101	1	28213.101	2.776	.107
	Social	1621.304	1	1621.304	.160	.693
	Exercise * Social	26490.188	1	26490.188	2.606	.118
	Error	274432.957	27	10164.184		
	Total	393009.090	32			
	Corrected Total	325563.627	31			
Female	Corrected Model	102270.705 ^b	4	25567.676	2.810	.045
	Intercept	17793.093	1	17793.093	1.955	.173
	FST1	77197.969	1	77197.969	8.484	.007
	Exercise	37687.075	1	37687.075	4.142	.052
	Social	332.433	1	332.433	.037	.850
	Exercise * Social	2389.569	1	2389.569	.263	.612
	Error	245676.154	27	9099.117		
	Total	420470.220	32			
	Corrected Total	347946.859	31			

a. R Squared = .157 (Adjusted R Squared = .032)

b. R Squared = .294 (Adjusted R Squared = .189)

Table 103. Forced Swim Test Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	2272452.981	1	2272452.981	90.483	.000
Stress Period	22052.291	1	22052.291	.878	.353
Sex	40897.162	1	40897.162	1.628	.207
Exercise	464.116	1	464.116	.018	.892
Social	3059.613	1	3059.613	.122	.728
Sex * Exercise	13555.200	1	13555.200	.540	.466
Sex * Social	534.020	1	534.020	.021	.885
Exercise * Social	362.900	1	362.900	.014	.905
Sex * Exercise * Social	2323.976	1	2323.976	.093	.762
Error	1381307.168	55	25114.676		

Table 104. Forced Swim Test Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Within-Subject Effects

Tests of Within-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Resilience	503623.986	2	251811.993	17.184	.000
Resilience * Stress Period	39332.349	2	19666.174	1.342	.266
Resilience * Sex	20006.965	2	10003.483	.683	.507
Resilience * Exercise	7299.577	2	3649.789	.249	.780
Resilience * Social	47868.373	2	23934.186	1.633	.200
Resilience * Sex * Exercise	33060.392	2	16530.196	1.128	.327
Resilience * Sex * Social	9214.299	2	4607.149	.314	.731
Resilience * Exercise * Social	18609.968	2	9304.984	.635	.532
Resilience * Sex * Exercise * Social	28602.960	2	14301.480	.976	.380
Error(Resilience)	1611947.622	110	14654.069		

Table 105. Forced Swim Test Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	1032408.126	1	1032408.126	35.452	.000
	Stress Period	7155.386	1	7155.386	.246	.624
	Exercise	13432.774	1	13432.774	.461	.503
	Social	4355.011	1	4355.011	.150	.702
	Exercise * Social	4388.745	1	4388.745	.151	.701
	Error	786267.363	27	29121.013		
Female	Intercept	1259941.795	1	1259941.795	64.230	.000
	Stress Period	80305.383	1	80305.383	4.094	.053
	Exercise	3123.216	1	3123.216	.159	.693
	Social	1444.183	1	1444.183	.074	.788
	Exercise * Social	899.448	1	899.448	.046	.832
	Error	529631.328	27	19615.975		

Table 106. Forced Swim Test Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Within-Subject Effects

Tests of Within-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Resilience	172449.941	2	86224.971	6.412	.003
	Resilience * Stress Period	46461.016	2	23230.508	1.727	.187
	Resilience * Exercise	29533.458	2	14766.729	1.098	.341
	Resilience * Social	32137.586	2	16068.793	1.195	.311
	Resilience * Exercise * Social	528.436	2	264.218	.020	.981
	Error(Resilience)	726198.306	54	13448.117		
Female	Resilience	371534.664	2	185767.332	11.773	.000
	Resilience * Stress Period	26558.780	2	13279.390	.842	.437

Resilience * Exercise	7907.151	2	3953.575	.251	.779
Resilience * Social	19757.008	2	9878.504	.626	.539
Resilience * Exercise * Social	49130.122	2	24565.061	1.557	.220
Error(Resilience)	852061.868	54	15778.923		

Table 107. Body Weight repeated measures ANOVA, Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	1521.270	1	1521.270	1.119	.295
Week1	15795.162	1	15795.162	11.614	.001
Sex	26839.160	1	26839.160	19.735	.000
Exercise	21112.320	1	21112.320	15.524	.000
Social	3183.768	1	3183.768	2.341	.132
Sex * Exercise	39355.960	1	39355.960	28.939	.000
Sex * Social	1046.900	1	1046.900	.770	.384
Exercise * Social	103.172	1	103.172	.076	.784
Sex * Exercise * Social	2129.374	1	2129.374	1.566	.216
Error	74797.582	55	1359.956		

Table 108. Body Weight repeated measures ANOVA, Mauchly's Test of Sphericity

Mauchly's Test of Sphericity							
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Time	.102	121.719	9	.000	.435	.513	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 109. Body Weight repeated measures ANOVA, Within-Subject Effects

Tests of Within-Subjects Effects						
Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Time	Sphericity Assumed	311.653	4	77.913	.602	.661
	Greenhouse-Geisser	311.653	1.740	179.160	.602	.527
Time * Week1	Sphericity Assumed	310.687	4	77.672	.600	.663
	Greenhouse-Geisser	310.687	1.740	178.605	.600	.528
Time * Sex	Sphericity Assumed	4285.970	4	1071.493	8.282	.000
	Greenhouse-Geisser	4285.970	1.740	2463.876	8.282	.001
Time * Exercise	Sphericity Assumed	6647.625	4	1661.906	12.845	.000
	Greenhouse-Geisser	6647.625	1.740	3821.521	12.845	.000
Time * Social	Sphericity Assumed	852.399	4	213.100	1.647	.163
	Greenhouse-Geisser	852.399	1.740	490.019	1.647	.201
Time * Sex * Exercise	Sphericity Assumed	10710.857	4	2677.714	20.697	.000
	Greenhouse-Geisser	10710.857	1.740	6157.352	20.697	.000
Time * Sex * Social	Sphericity Assumed	222.740	4	55.685	.430	.787
	Greenhouse-Geisser	222.740	1.740	128.047	.430	.624
Time * Exercise * Social	Sphericity Assumed	391.260	4	97.815	.756	.555
	Greenhouse-Geisser	391.260	1.740	224.924	.756	.455
Time * Sex * Exercise * Social	Sphericity Assumed	866.484	4	216.621	1.674	.157
	Greenhouse-Geisser	866.484	1.740	498.116	1.674	.196
Error(Time)	Sphericity Assumed	28463.097	220	129.378		
	Greenhouse-Geisser	28463.097	95.674	297.502		

Table 110. Body Weight repeated measures ANOVA with file split by sex,
Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	1013.569	1	1013.569	.544	.467
	Week1	23302.373	1	23302.373	12.513	.001
	Exercise	62720.203	1	62720.203	33.680	.000
	Social	63.116	1	63.116	.034	.855
	Exercise * Social	2378.824	1	2378.824	1.277	.268
	Error	50279.920	27	1862.219		
Female	Intercept	5895.998	1	5895.998	9.915	.004
	Week1	955.022	1	955.022	1.606	.216
	Exercise	2320.846	1	2320.846	3.903	.059
	Social	1075.562	1	1075.562	1.809	.190
	Exercise * Social	351.848	1	351.848	.592	.448
	Error	16055.430	27	594.646		

Table 111. Body Weight repeated measures ANOVA with file split by sex,
Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Time	.077	65.302	9	.000	.429	.523	.250
Female	Time	.150	48.212	9	.000	.469	.578	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 112. Body Weight repeated measures ANOVA with file split by sex,
Within-Subject Effects

Tests of Within-Subjects Effects							
Sex	Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Time	Sphericity Assumed	530.548	4	132.637	.764	.551
		Greenhouse- Geisser	530.548	1.714	309.449	.764	.453
	Time * Week1	Sphericity Assumed	1777.595	4	444.399	2.559	.043
		Greenhouse- Geisser	1777.595	1.714	1036.808	2.559	.096
	Time * Exercise	Sphericity Assumed	18103.592	4	4525.898	26.062	.000
		Greenhouse- Geisser	18103.592	1.714	10559.176	26.062	.000
	Time * Social	Sphericity Assumed	200.761	4	50.190	.289	.885
		Greenhouse- Geisser	200.761	1.714	117.097	.289	.716
	Time * Exercise * Social	Sphericity Assumed	1476.429	4	369.107	2.125	.083
		Greenhouse- Geisser	1476.429	1.714	861.148	2.125	.137
	Error(Time)	Sphericity Assumed	18755.464	108	173.662		
		Greenhouse- Geisser	18755.464	46.291	405.163		
Female	Time	Sphericity Assumed	1378.687	4	344.672	4.985	.001
		Greenhouse- Geisser	1378.687	1.877	734.512	4.985	.012
	Time * Week1	Sphericity Assumed	772.818	4	193.204	2.794	.030

	Greenhouse-Geisser	772.818	1.877	411.728	2.794	.074
Time * Exercise	Sphericity Assumed	466.690	4	116.673	1.687	.158
	Greenhouse-Geisser	466.690	1.877	248.635	1.687	.197
Time * Social	Sphericity Assumed	211.301	4	52.825	.764	.551
	Greenhouse-Geisser	211.301	1.877	112.573	.764	.463
Time * Exercise * Social	Sphericity Assumed	69.704	4	17.426	.252	.908
	Greenhouse-Geisser	69.704	1.877	37.136	.252	.764
Error(Time)	Sphericity Assumed	7467.907	108	69.147		
	Greenhouse-Geisser	7467.907	50.679	147.356		

Table 113. Body Weight Stress period ANOVA

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	287842.679 ^a	8	35980.335	256.830	.000
Intercept	238.034	1	238.034	1.699	.198
PreStress	4793.238	1	4793.238	34.214	.000
Sex	1495.866	1	1495.866	10.678	.002
Exercise	3908.153	1	3908.153	27.897	.000
Social	267.008	1	267.008	1.906	.173
Sex * Exercise	5616.433	1	5616.433	40.090	.000
Sex * Social	103.884	1	103.884	.742	.393
Exercise * Social	105.706	1	105.706	.755	.389
Sex * Exercise * Social	103.554	1	103.554	.739	.394
Error	7705.183	55	140.094		
Total	6198929.210	64			
Corrected Total	295547.862	63			

a. R Squared = .974 (Adjusted R Squared = .970)

Table 114. Body Weight Stress period ANOVA with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Corrected Model	15979.200 ^a	4	3994.800	40.756	.000
	Intercept	184.182	1	184.182	1.879	.182
	PreStress	6455.322	1	6455.322	65.858	.000
	Exercise	9752.749	1	9752.749	99.499	.000
	Social	1.339	1	1.339	.014	.908
	Exercise * Social	371.864	1	371.864	3.794	.062
	Error	2646.500	27	98.019		
	Total	4376368.120	32			
	Corrected Total	18625.700	31			

Female	Corrected Model	670.308 ^b	4	167.577	1.400	.261
	Intercept	1541.657	1	1541.657	12.877	.001
	PreStress	164.058	1	164.058	1.370	.252
	Exercise	286.921	1	286.921	2.397	.133
	Social	108.633	1	108.633	.907	.349
	Exercise * Social	2.483	1	2.483	.021	.887
	Error	3232.541	27	119.724		
	Total	1822561.090	32			
	Corrected Total	3902.850	31			

a. R Squared = .858 (Adjusted R Squared = .837)

b. R Squared = .172 (Adjusted R Squared = .049)

Table 115. Body Weight Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	1072.239	1	1072.239	2.353	.131
Stress	69359.104	1	69359.104	152.211	.000
Sex	.432	1	.432	.001	.976
Exercise	154.068	1	154.068	.338	.563
Social	261.592	1	261.592	.574	.452
Sex * Exercise	192.242	1	192.242	.422	.519
Sex * Social	3.778	1	3.778	.008	.928
Exercise * Social	125.803	1	125.803	.276	.601
Sex * Exercise * Social	792.962	1	792.962	1.740	.193
Error	25062.286	55	455.678		

Table 116. Body Weight Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Mauchly's Test of Sphericity

Mauchly's Test of Sphericity							
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Resilience	.758	14.959	2	.001	.805	.946	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 117. Body Weight Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Within-Subject Effects

Tests of Within-Subjects Effects						
Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Resilience	Sphericity Assumed	85.609	2	42.805	.656	.521
	Greenhouse-Geisser	85.609	1.610	53.162	.656	.489
Resilience * Stress	Sphericity Assumed	271.607	2	135.803	2.083	.129
	Greenhouse-Geisser	271.607	1.610	168.662	2.083	.140
Resilience * Sex	Sphericity Assumed	119.279	2	59.640	.915	.404
	Greenhouse-Geisser	119.279	1.610	74.070	.915	.386
Resilience * Exercise	Sphericity Assumed	151.446	2	75.723	1.161	.317
	Greenhouse-Geisser	151.446	1.610	94.045	1.161	.309
Resilience * Social	Sphericity Assumed	39.361	2	19.680	.302	.740
	Greenhouse-Geisser	39.361	1.610	24.442	.302	.692
Resilience * Sex * Exercise	Sphericity Assumed	226.548	2	113.274	1.737	.181
	Greenhouse-Geisser	226.548	1.610	140.682	1.737	.188

Resilience * Sex * Social	Sphericity Assumed	145.830	2	72.915	1.118	.331
	Greenhouse- Geisser	145.830	1.610	90.557	1.118	.321
Resilience * Exercise * Social	Sphericity Assumed	142.633	2	71.316	1.094	.339
	Greenhouse- Geisser	142.633	1.610	88.572	1.094	.328
Resilience * Sex * Exercise * Social	Sphericity Assumed	77.820	2	38.910	.597	.552
	Greenhouse- Geisser	77.820	1.610	48.325	.597	.518
Error(Resilience)	Sphericity Assumed	7173.082	110	65.210		
	Greenhouse- Geisser	7173.082	88.570	80.988		

Table 118. Body Weight Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Between-Subjects Effects

Tests of Between-Subjects Effects

Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	1572.914	1	1572.914	2.317	.140
	Stress	58334.241	1	58334.241	85.935	.000
	Exercise	12.264	1	12.264	.018	.894
	Social	57.435	1	57.435	.085	.773
	Exercise * Social	726.022	1	726.022	1.070	.310
	Error	18328.084	27	678.818		
Female	Intercept	31.341	1	31.341	.149	.703
	Stress	12061.034	1	12061.034	57.151	.000
	Exercise	136.691	1	136.691	.648	.428
	Social	266.542	1	266.542	1.263	.271
	Exercise * Social	170.278	1	170.278	.807	.377
	Error	5698.032	27	211.038		

Table 119. Body Weight Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Resilience	.710	8.918	2	.012	.775	.935	.500
Female	Resilience	.839	4.563	2	.102	.861	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 120. Body Weight Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Within-Subject Effects

Tests of Within-Subjects Effects							
Sex	Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Resilience	Sphericity Assumed	33.531	2	16.766	.172	.842
		Greenhouse-Geisser	33.531	1.550	21.634	.172	.787
	Resilience * Stress	Sphericity Assumed	221.555	2	110.778	1.138	.328
		Greenhouse-Geisser	221.555	1.550	142.943	1.138	.318
	Resilience * Exercise	Sphericity Assumed	196.466	2	98.233	1.009	.371
		Greenhouse-Geisser	196.466	1.550	126.756	1.009	.355
	Resilience * Social	Sphericity Assumed	177.802	2	88.901	.913	.407
		Greenhouse-Geisser	177.802	1.550	114.714	.913	.386

	Resilience * Exercise	Sphericity	207.210	2	103.605	1.064	.352
	* Social	Assumed					
		Greenhouse-Geisser	207.210	1.550	133.688	1.064	.339
	Error(Resilience)	Sphericity	5258.294	54	97.376		
		Assumed					
		Greenhouse-Geisser	5258.294	41.849	125.650		
Female	Resilience	Sphericity	91.712	2	45.856	1.337	.271
		Assumed					
		Greenhouse-Geisser	91.712	1.723	53.237	1.337	.270
	Resilience * Stress	Sphericity	112.510	2	56.255	1.640	.203
		Assumed					
		Greenhouse-Geisser	112.510	1.723	65.310	1.640	.207
	Resilience * Exercise	Sphericity	50.899	2	25.449	.742	.481
		Assumed					
		Greenhouse-Geisser	50.899	1.723	29.546	.742	.463
	Resilience * Social	Sphericity	21.928	2	10.964	.320	.728
		Assumed					
		Greenhouse-Geisser	21.928	1.723	12.729	.320	.696
	Resilience * Exercise * Social	Sphericity	7.702	2	3.851	.112	.894
		Assumed					
		Greenhouse-Geisser	7.702	1.723	4.471	.112	.866
	Error(Resilience)	Sphericity	1852.329	54	34.302		
		Assumed					
		Greenhouse-Geisser	1852.329	46.513	39.824		

Table 121. Food Consumption repeated measures ANOVA, Between-Subjects Effects

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	1187.462	1	1187.462	37.424	.000
Week1	54.068	1	54.068	1.704	.197
Sex	1485.652	1	1485.652	46.822	.000
Exercise	270.433	1	270.433	8.523	.005
Social	606.530	1	606.530	19.116	.000
Sex * Exercise	333.845	1	333.845	10.522	.002
Sex * Social	9.357	1	9.357	.295	.589
Exercise * Social	29.640	1	29.640	.934	.338
Sex * Exercise * Social	20.147	1	20.147	.635	.429
Error	1681.673	53	31.730		

Table 122. Food Consumption repeated measures ANOVA, Mauchly's Test of Sphericity

Mauchly's Test of Sphericity							
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Time	.485	37.220	9	.000	.776	.955	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 123. Food Consumption repeated measures ANOVA, Within-Subject Effects

Tests of Within-Subjects Effects						
Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Time	Sphericity Assumed	61.893	4	15.473	1.491	.206
	Greenhouse-Geisser	61.893	3.106	19.930	1.491	.218
Time * Week1	Sphericity Assumed	45.690	4	11.423	1.101	.357
	Greenhouse-Geisser	45.690	3.106	14.713	1.101	.352
Time * Sex	Sphericity Assumed	70.521	4	17.630	1.699	.151
	Greenhouse-Geisser	70.521	3.106	22.709	1.699	.167
Time * Exercise	Sphericity Assumed	200.714	4	50.179	4.835	.001
	Greenhouse-Geisser	200.714	3.106	64.632	4.835	.003
Time * Social	Sphericity Assumed	236.655	4	59.164	5.701	.000
	Greenhouse-Geisser	236.655	3.106	76.205	5.701	.001
Time * Sex * Exercise	Sphericity Assumed	97.561	4	24.390	2.350	.055
	Greenhouse-Geisser	97.561	3.106	31.415	2.350	.072
Time * Sex * Social	Sphericity Assumed	64.544	4	16.136	1.555	.188
	Greenhouse-Geisser	64.544	3.106	20.784	1.555	.201
Time * Exercise * Social	Sphericity Assumed	107.734	4	26.934	2.595	.037
	Greenhouse-Geisser	107.734	3.106	34.691	2.595	.052
Time * Sex * Exercise * Social	Sphericity Assumed	64.600	4	16.150	1.556	.187
	Greenhouse-Geisser	64.600	3.106	20.802	1.556	.201
Error(Time)	Sphericity Assumed	2200.171	212	10.378		
	Greenhouse-Geisser	2200.171	164.592	13.367		

Table 124. Food Consumption repeated measures ANOVA with file split by sex,
Between-Subjects Effects

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	638.579	1	638.579	16.846	.000
	Week1	6.276	1	6.276	.166	.687
	Exercise	554.623	1	554.623	14.631	.001
	Social	362.886	1	362.886	9.573	.005
	Exercise * Social	49.601	1	49.601	1.309	.263
	Error	1023.471	27	37.906		
Female	Intercept	533.998	1	533.998	20.542	.000
	Week1	56.101	1	56.101	2.158	.154
	Exercise	.189	1	.189	.007	.933
	Social	225.090	1	225.090	8.659	.007
	Exercise * Social	.100	1	.100	.004	.951
	Error	649.893	25	25.996		

Table 125. Food Consumption repeated measures ANOVA with file split by sex,
Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Time	.279	32.418	9	.000	.636	.812	.250
Female	Time	.471	17.642	9	.040	.806	1.000	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 126. Food Consumption repeated measures ANOVA with file split by sex, Within-Subject Effects

Tests of Within-Subjects Effects							
Sex	Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Time	Sphericity Assumed	101.330	4	25.332	2.128	.082
		Greenhouse-Geisser	101.330	2.545	39.822	2.128	.114
	Time * Week1	Sphericity Assumed	107.181	4	26.795	2.251	.068
		Greenhouse-Geisser	107.181	2.545	42.121	2.251	.100
	Time * Exercise	Sphericity Assumed	168.394	4	42.098	3.536	.009
		Greenhouse-Geisser	168.394	2.545	66.178	3.536	.025
	Time * Social	Sphericity Assumed	171.899	4	42.975	3.609	.008
		Greenhouse-Geisser	171.899	2.545	67.555	3.609	.023
	Time * Exercise * Social	Sphericity Assumed	156.432	4	39.108	3.285	.014
		Greenhouse-Geisser	156.432	2.545	61.477	3.285	.033
	Error(Time)	Sphericity Assumed	1285.875	108	11.906		
		Greenhouse-Geisser	1285.875	68.703	18.716		
Female	Time	Sphericity Assumed	27.174	4	6.793	.803	.526
		Greenhouse-Geisser	27.174	3.222	8.433	.803	.504
	Time * Week1	Sphericity Assumed	6.383	4	1.596	.189	.944

	Greenhouse-Geisser	6.383	3.222	1.981	.189	.915
Time * Exercise	Sphericity Assumed	120.496	4	30.124	3.559	.009
	Greenhouse-Geisser	120.496	3.222	37.395	3.559	.016
Time * Social	Sphericity Assumed	126.754	4	31.689	3.744	.007
	Greenhouse-Geisser	126.754	3.222	39.337	3.744	.012
Time * Exercise * Social	Sphericity Assumed	17.886	4	4.472	.528	.715
	Greenhouse-Geisser	17.886	3.222	5.551	.528	.677
Error(Time)	Sphericity Assumed	846.423	100	8.464		
	Greenhouse-Geisser	846.423	80.557	10.507		

Table 127. Food Consumption Stress period ANOVA (with pre-stress as a covariate)

Tests of Between-Subjects Effects					
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	1454.116 ^a	8	181.764	19.946	.000
Intercept	568.638	1	568.638	62.399	.000
PreStress	.007	1	.007	.001	.979
Sex	363.114	1	363.114	39.846	.000
Exercise	82.373	1	82.373	9.039	.004
Social	392.255	1	392.255	43.044	.000
Sex * Exercise	23.302	1	23.302	2.557	.116
Sex * Social	60.048	1	60.048	6.589	.013
Exercise * Social	66.086	1	66.086	7.252	.009
Sex * Exercise * Social	9.554	1	9.554	1.048	.311
Error	482.985	53	9.113		
Total	31627.386	62			
Corrected Total	1937.101	61			

a. R Squared = .751 (Adjusted R Squared = .713)

Table 128. Food Consumption Stress period ANOVA (with pre-stress as a covariate) with file split by sex

Tests of Between-Subjects Effects						
Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Corrected Model	577.816 ^a	4	144.454	14.220	.000
	Intercept	402.683	1	402.683	39.639	.000
	PreStress	3.550	1	3.550	.349	.559
	Exercise	106.497	1	106.497	10.483	.003
	Social	403.135	1	403.135	39.684	.000
	Exercise * Social	55.393	1	55.393	5.453	.027
	Error	274.283	27	10.159		
	Total	21380.291	32			
	Corrected Total	852.099	31			

Female	Corrected Model	100.249 ^b	4	25.062	3.133	.032
	Intercept	176.402	1	176.402	22.051	.000
	PreStress	5.161	1	5.161	.645	.429
	Exercise	7.430	1	7.430	.929	.344
	Social	74.305	1	74.305	9.288	.005
	Exercise * Social	12.299	1	12.299	1.537	.227
	Error	199.997	25	8.000		
	Total	10247.095	30			
	Corrected Total	300.246	29			

a. R Squared = .678 (Adjusted R Squared = .630)

b. R Squared = .334 (Adjusted R Squared = .227)

Table 129. Food Consumption Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Between-Subjects Effects

Tests of Between-Subjects Effects

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	2270.417	1	2270.417	60.912	.000
Stress Period	35.329	1	35.329	.948	.335
Sex	1386.714	1	1386.714	37.204	.000
Exercise	43.619	1	43.619	1.170	.284
Social	161.402	1	161.402	4.330	.042
Sex * Exercise	323.568	1	323.568	8.681	.005
Sex * Social	1.024	1	1.024	.027	.869
Exercise * Social	6.697	1	6.697	.180	.673
Sex * Exercise * Social	13.091	1	13.091	.351	.556
Error	2050.048	55	37.274		

Table 130. Food Consumption Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Mauchly's Test of Sphericity

Mauchly's Test of Sphericity							
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Resilience	.830	10.079	2	.006	.855	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 131. Food Consumption Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate), Within-Subject Effects

Tests of Within-Subjects Effects						
Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Resilience	Sphericity Assumed	98.078	2	49.039	5.996	.003
	Greenhouse-Geisser	98.078	1.709	57.389	5.996	.005
Resilience * Stress Period	Sphericity Assumed	28.874	2	14.437	1.765	.176
	Greenhouse-Geisser	28.874	1.709	16.895	1.765	.181
Resilience * Sex	Sphericity Assumed	39.501	2	19.750	2.415	.094
	Greenhouse-Geisser	39.501	1.709	23.113	2.415	.103
Resilience * Exercise	Sphericity Assumed	218.887	2	109.444	13.382	.000
	Greenhouse-Geisser	218.887	1.709	128.077	13.382	.000
Resilience * Social	Sphericity Assumed	8.540	2	4.270	.522	.595
	Greenhouse-Geisser	8.540	1.709	4.997	.522	.567
Resilience * Sex * Exercise	Sphericity Assumed	40.881	2	20.440	2.499	.087
	Greenhouse-Geisser	40.881	1.709	23.921	2.499	.096

Resilience * Sex * Social	Sphericity Assumed	2.206	2	1.103	.135	.874
	Greenhouse- Geisser	2.206	1.709	1.291	.135	.842
Resilience * Exercise * Social	Sphericity Assumed	67.897	2	33.949	4.151	.018
	Greenhouse- Geisser	67.897	1.709	39.729	4.151	.024
Resilience * Sex * Exercise * Social	Sphericity Assumed	32.739	2	16.369	2.002	.140
	Greenhouse- Geisser	32.739	1.709	19.156	2.002	.147
Error(Resilience)	Sphericity Assumed	899.594	110	8.178		
	Greenhouse- Geisser	899.594	93.996	9.571		

Table 132. Food Consumption Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Between-Subjects Effects

Tests of Between-Subjects Effects

Sex	Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Intercept	1452.664	1	1452.664	29.032	.000
	Stress Period	1.478	1	1.478	.030	.865
	Exercise	289.399	1	289.399	5.784	.023
	Social	97.163	1	97.163	1.942	.175
	Exercise * Social	29.727	1	29.727	.594	.448
	Error	1350.995	27	50.037		
Female	Intercept	839.439	1	839.439	33.464	.000
	Stress Period	55.603	1	55.603	2.217	.148
	Exercise	70.638	1	70.638	2.816	.105
	Social	80.793	1	80.793	3.221	.084
	Exercise * Social	1.296	1	1.296	.052	.822
	Error	677.301	27	25.085		

Table 133. Food Consumption Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Mauchly's Test of Sphericity

Mauchly's Test of Sphericity								
Sex	Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon ^a		
						Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Male	Resilience	.843	4.434	2	.109	.864	1.000	.500
Female	Resilience	.678	10.087	2	.006	.757	.911	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Table 134. Food Consumption Resilience analysis (post-stress repeated measures ANOVA with stress period as a covariate) with file split by sex, Within-Subject Effects

Tests of Within-Subjects Effects							
Sex	Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Male	Resilience	Sphericity Assumed	90.769	2	45.385	5.391	.007
		Greenhouse-Geisser	90.769	1.729	52.501	5.391	.011
	Resilience * Stress Period	Sphericity Assumed	49.847	2	24.923	2.961	.060
		Greenhouse-Geisser	49.847	1.729	28.831	2.961	.069
	Resilience * Exercise	Sphericity Assumed	145.249	2	72.625	8.627	.001
		Greenhouse-Geisser	145.249	1.729	84.011	8.627	.001
	Resilience * Social	Sphericity Assumed	16.859	2	8.430	1.001	.374
		Greenhouse-Geisser	16.859	1.729	9.751	1.001	.365

	Resilience * Exercise * Social	Sphericity Assumed	100.069	2	50.034	5.944	.005
		Greenhouse- Geisser	100.069	1.729	57.879	5.944	.007
	Error(Resilience)	Sphericity Assumed	454.575	54	8.418		
		Greenhouse- Geisser	454.575	46.681	9.738		
Female	Resilience	Sphericity Assumed	30.021	2	15.011	1.933	.155
		Greenhouse- Geisser	30.021	1.513	19.838	1.933	.166
	Resilience * Stress Period	Sphericity Assumed	4.742	2	2.371	.305	.738
		Greenhouse- Geisser	4.742	1.513	3.134	.305	.677
	Resilience * Exercise	Sphericity Assumed	103.108	2	51.554	6.639	.003
		Greenhouse- Geisser	103.108	1.513	68.132	6.639	.006
	Resilience * Social	Sphericity Assumed	.458	2	.229	.029	.971
		Greenhouse- Geisser	.458	1.513	.303	.029	.940
	Resilience * Exercise * Social	Sphericity Assumed	7.394	2	3.697	.476	.624
		Greenhouse- Geisser	7.394	1.513	4.886	.476	.572
	Error(Resilience)	Sphericity Assumed	419.304	54	7.765		
		Greenhouse- Geisser	419.304	40.861	10.262		