THE EFFECTS OF PLYOMETRICS OR RESISTANCE-TRAINING ON MARKERS OF BONE TURNOVER AND HORMONES IN MEN

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ROBERT S. ROGERS

Dr. Pamela S. Hinton, Thesis Supervisor

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Presented by Robert S. Rogers,
a candidate for the degree of Master of Arts in Exercise Physiology
and hereby certify that, in their opinion, it is worthy of acceptance.

Thesis Supervisor: Dr. Pamela S. Hinton

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Committee Members:

_____
Dr. John P. Thyfault

_____
Dr. Stephen P. Sayers

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"And his fame went throughout all Syria: and they brought unto him all sick people that were taken with divers diseases and torments, and those which were possessed with devils, and those which were lunatick, and those that had the paulsy; and he healed them." Matthew 4:24.

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ABSTRACT

ACUTE EFFECTS OF A SINGLE-BOUT OF RESISTANCE-TRAINING OR PLYOMETRICS ON MARKERS OF BONE TURNOVER AND HORMONES IN MEN

Department of Nutrition and Exercise Physiology, University of Missouri, Columbia, Missouri 65211

Weight-bearing exercise positively affects bone mineral density (BMD) and bone strength, presumably by altering the balance between bone formation and resorption. **PURPOSE:** The objective of the study was three-fold: 1) to determine the acute response of serum markers of bone turnover and hormones to a single-bout of resistance-training (RT) or plyometrics (PLY), 2) to compare the effects of fasting versus feeding on the hormonal and bone turnover response to RT or PLY; and 3) to determine the relationship of exercise mode (PLY/RT) and energy status (fed/fasted) on both the activity of the osteoblasts and osteoclasts and on circulating hormone concentration. **METHODS:** Twelve recreationally active males, aged 24-63 years, participated in this partially randomized cross-over study, which included five trials: PLY (fed/fasted), RT (fed/fasted), and a no-exercise, fasted control trial (CON) (n=6). Subjects completed all trials between 06:00 and 10:30 am. Subjects underwent a 10-hr overnight fast or were fed a liquid meal replacement containing 500 kcal, 12 g fat, 80 g carbohydrate, 18 g protein, 500 mg calcium, and 240 IU vitamin D two hours prior to exercise. RT consisted of three sets of ten repetitions of eight exercises performed at 60% (first set) and 80% (second and third sets) of one-repetition (1-RM) or ten-repetition maximum (10-RM). PLY consisted of 10 repetitions of 12 different jumps. Blood was drawn immediately prior to exercise (PRE), immediately following exercise (POST), and 15, 30, 60, 120 min, and 24 hr following PRE. Total testosterone (T), intact parathyroid hormone (PTH), and cortisol (COR) concentrations in serum were determined using chemiluminescent immunoassay. Serum bone-specific alkaline phosphatase (BAP) (bone formation marker) and tartrate-resistant acid phosphatase, isoform 5b (TRAP5b) (bone resorption marker) were measured using ELISA. A one-factor (time), repeated measures ANOVA was used to detect changes in dependent variables over time for each exercise trial (n=12) and CON (n=6); post hoc pairwise comparisons (LSD) were performed when apprioriate. A 2 x 2 repeated measures ANOVA was used to compare the interaction of exercise mode (PLY/RT), feeding (fed/fasted), and time. An independent t-test was performed when a signficiant interaction was determined. To compare the effects of exercise mode and energy status on both the activity of osteoblasts and osteoclasts and on circulating hormone concentration an area under the curve (AUC) was quantified during the 2-hr post exercise. Bivariate relationships between the AUC of the bone turnover markers and hormones were evaluated using Pearsons correlations. **RESULTS:** During the two-hours following all exercise trials, TRAP5b, COR, and PTH significantly decreased (main effect for time; $p<0.05$) (n=12). TEST significantly decreased following PLY-, PLY+, and RT+ (main effect for time; $p<0.05$) (n=12). BAP, COR, and PTH significantly decreased during the two-hours following the CON (main effect for time; $p<0.05$); however, TRAP5b did not change. A significant interaction between exercise mode and time was detected for COR and the concentration of COR at POST, 15MIN,

and 30MIN was significantly greater following the RT than PLY ($p<0.05$). The AUC of TRAP5b was positively correlated with the AUC of PTH ($r=0.452$, $p=0.001$). **CONCLUSIONS:** The results of the present study suggest favorable changes in bone formation and bone resorption as assessed by bone turnover markers (BAP and TRAP5b) following a single-bout of RT or PLY. The decrease in PTH following plyometrics and resistance-training positively correlated with a decrease in TRAP5b, suggesting that PTH may mediate the exercise-induced changes in osteoclast activity.

INTRODUCTION

Health burden of osteoporosis

Osteoporosis literally means "porous bone" (54), and it is the manifestation of a reduction in bone mineral density (BMD) and increased porosity. Osteoporosis is the leading cause of fractures in the elderly. The lifetime prevalence of suffering an osteoporosis-related fracture after the age of 50 years is between 40% and 50% in women and between 13% and 22% in men (61). Hip fractures are the most costly osteoporosis-related fracture. Hip fractures are estimated to occur in 16% of all Caucasian women over the age of 50 years and in 6% of all Caucasian men over the age of 50 years (31). This is of concern because the U.S. population over 50 years of age is predicted to grow by 60% from 2000 to 2025, reaching a total of 121 million by 2025 (20). Once an osteoporosis-related fracture occurs the risk of death is greater among men than women (17, 64, 70). Kiebzak *et al.* (70) reported that the one-year mortality rate in men following a hip fracture is 32% compared to 17% in women (70). Bruger *et al.* (17) reported that the one year mortality rate in men following a hip fracture to be 20% compared to 8.8% in women (17). Similarly, mortality following a vertebral fracture is also greater in men than women (22). The cost and prevalence of osteoporosis, along with an aging population, makes the disease a growing concern in the U.S.

Prevalence & treatment of osteoporosis in men

The prevalence of osteoporosis in men is under-appreciated. Data from the National Health and Nutrition Examination Survey (NHANES III, 1988-1994) indicate that between one and two million men in the U.S. have femoral osteoporosis and between eight and 13 million men have femoral osteopenia (87). The data for men was compared

to both men and women reference ranges and the prevalence rates for both comparisons were reported (87). In addition, available osteoporosis treatments are under utilized in men. The likelihood that a man suffering from osteoporosis will be prescribed a pharmacological anti-resorptive treatment is less than in women (36, 70), despite the approved use of the bisphosphonate alendronate to treat osteoporosis in men in late 2000 (96). Men are more likely to be prescribed supplemental calcium and vitamin D (70). Feldstein *et al*. (35) reported that only 7.1% of men aged 65 years and older were prescribed a pharmacological intervention for osteoporosis during the 18-month period following their initial osteoporosis-related fracture (35). Thus, effective treatments and preventative measures for males with osteopenia and osteoporosis must be developed, communicated to physicians, and disseminated to the general public.

Bone remodeling

Bone is a dynamic tissue that is continuously remodeled in adults via a process known as bone turnover. Bone remodeling is the process by which bones are re-shaped, maintained, and repaired by the coordinated actions of bone resorption cells (osteoclasts) and bone formation cells (osteoblasts) (32). The collaborative efforts of the osteoclasts and osteoblasts occur in a region of bone known as the basic mulitcellular unit (BMU) (97). Within the BMU, bone is broken down by the osteoclasts and then subsequently replaced by the osteoblasts. The lifespan of each active BMU is approximately six to nine months (89). Bone resorption occurs in a shorter amount of time (i.e., several weeks) compared to bone formation that occurs during several months (32).

Bone formation and bone resorption are both coupled and balanced so that the amount of bone removed during resorption is replaced by an equivalent amount of bone

during formation. Bone turnover is considered balanced when formation matches resorption. Bone turnover is coupled when the activation of the bone resorption process is matched by a subsequent activation of equal bone formation. During states of high rates of bone loss, such as during menopause, bone resorption is greater than bone formation. Thus, bone turnover is imbalanced and bone loss occurs. An increased rate of bone turnover is also detrimental to bone, independent of the balance between bone formation and resorption, because more BMUs are formed and weaken the overall bone structure (97, 101).

Mechanical strain

In addition to genetic and dietary factors, bone is responsive to strains applied to the skeleton. Mechanical strains above a minimal threshold must be applied to the skeleton to provide the stimulus necessary to maintain balance between resorption and formation. This response to loading is known as the "mechanostat" theory proposed by Harold Frost (38-40). The "mechanostat" theory states that strains that are of sufficient magnitude to reach the minimal threshold will induce bone formation. Strains that do not meet the minimum threshold have no effect (38-40). Dynamic, weight-bearing exercise that induces mechanical strains above the minimal threshold is important for the maintenance of bone strength (81, 123).

Physical activity and exercise

Due to the positive response of bone to mechanical strain, weight-bearing exercise has been promoted for the preservation of bone strength and mass throughout adulthood. The National Osteoporosis Foundation (NOF) and American College of Sports Medicine (ACSM) currently recommend weight-bearing and resistance-based exercise for the

preservation of bone mass (3). To support this contention, sports that require dynamic, weight-bearing movements effectively for maintain or increase BMD throughout adulthood.

Cross-sectional studies of athletic populations report that athletes who participate in sports that are both weight-bearing and high-impact (e.g., volleyball, basketball) have greater BMD than sedentary individuals, non-athlete controls, or athletes in non-weight-bearing or low-impact sports (e.g., swimming, cycling). These results have been confirmed in both men (11, 19) and women (11, 28, 33, 34, 82). For instance, Lee et al. (82) reported that female athletes performing high-impact sports, such as volleyball and basketball, had significantly greater BMD at the lumbar spine, femoral neck, trochanter, and Ward's triangle than swimmers, moderately active individuals, and sedentary controls ($p<0.05$) (82). Similarly, our laboratory group recently reported that male cyclists, whose training requires hours of weekly exercise with minimal impact loading upon the skeleton, have lower BMD of the spine than male runners (100). In agreement, Nichols *et al.* (95) reported that masters-aged cyclists have lower BMD of the hip and spine compared to younger cyclists and age-matched controls (95). Similar reductions in BMD among cyclists have been reported elsewhere (113, 118).

The ability of resistance-training to improve or maintain bone mass in pre- and postmenopausal women has been well documented (68, 69, 85, 90, 131). Similarly, resistance-training interventions have been reported to improve bone mass in both middle-aged (92, 106) and elderly men (92, 131, 139). In other studies, resistance-training intervention have not been successful in preserving BMD men (91) and women (10, 91, 112) due to insufficient length of intervention, insufficient loading, or an

extremely elderly population participated. The volume of training, length of training intervention, and loading characteristics differ among the various studies of resistance-training and BMD leading to conflicting results. Because each bone remodeling cycle is six to nine months in length and multiple bone remodeling cycles are necessary to achieve measurable changes in BMD by DXA, of the duration of the intervention must be adequate. Results of resistance-training interventions that are less than 12 months in length must be approached with some caution. The magnitude of strain applied to the skeleton is also important. Low-intensity resistance-training programs have not been successful at preserving bone loss compared with high-intensity resistance-training programs (131).

A growing body of evidence supports the positive influence of high-impact jump-training on bone strength. Jump-training, sometimes referred to as plyometrics, reportedly maintained BMD at weight-bearing skeletal sites in pre-menopausal women (9, 55, 66, 119, 128, 138) and postmenopausal women (44, 126). The effects of an exclusive jump-training program on bone have not been thoroughly studied in a male population of any age. A single study investigated the effect of a combined program of resistance-training and plyometrics on BMD in men and women (45). The combination of training modes in this study makes comparisons difficult.

Plyometrics induce the rapid development of strain in load-bearing skeletal sites both during takeoff and landing. Both muscle contraction and impact on the ground induce strain on the skeleton. Muscle contractions are thought to induce the greatest strain upon the skeleton and induce increases in BMD. Training that induces impact forces on upon landing has also been reported to increase BMD (63). To illustrate this,

college-aged females were randomized to three separate groups of exercise: weight lifting (predominately muscle loading), running (predominately gravitational loading), or remained sedentary. Despite large increases in muscular strength following weight lifting, runners and weight lifters had nearly identical increases in BMD of the lumbar spine (116).

Typically, plyometrics are utilized to increase power, a function of force and velocity, rather than maximal strength. Landing during plyometrics elicits ground reaction forces on the skeleton that are greater than body weight alone (133). Weeks *et al.* (133) reported that a squat jump elicits force 3.8-times that of bodyweight. More complicated plyometric movements, such as the tuck jump, depth jump, and drop jump elicit forces of 4.8-, 5.2-, and 5.5-times bodyweight, respectively (133). These forces induce a fluid shift through the bone cells, specifically the osteocytes, which act as important signaling cells (123). The communication between osteoblasts, osteocytes, and osteoclasts is hypothesized to behave as a neuronal network (123) that senses the fluid shift of the cytosol through the canaliculae from one osteocyte to another as bones are strained by mechanical loading (71, 123). The osteocytes then signal the bone lining cells to initiate the bone remodeling process.

Rest inserted between loading cycles enhances the osteogenic stimulus (102, 103, 117). Robling *et al.* (102) reported that partitioning bouts of external loading into four sessions throughout the day improved bone strength to a greater extent than exposing the skeleton to a single session of stress per day (102). Srinivasan *et al.* (117) reported that a ten second rest period inserted between the applications of external loads increased the

periosteal bone formation rate to greater extent than loading applied with no rest between applications (117).

In any training program, volume is an important variable to control; therefore, the number of jumps performed per session is an important variable during plyometric training. In animal models, ten jumps per day increase bone mass and strength (57). As few as five jumps per day have been reported to be as effective as 100 jumps per day for increasing bone mass and strength (125). These results have been confirmed in humans. Kato *et al.* (66) reported that ten maximal vertical jumps per day, three days per week, for six months increased BMD of the femoral neck and lumbar spine in young women compared to sedentary young women (66). Turner and colleagues (123) have hypothesized that bone becomes 'deaf' to loading after adequate strain has been applied to the skeleton (123). Volume and rest-period are important variables to control when designing plyometric training programs to improve bone mass in humans.

Hormonal mediators of exercise on bone: Acute hormonal responses to exercise

A single-bout of resistance-training increases the rate of skeletal muscle protein turnover (13) and increases serum concentrations of several hormones important to bone mass including testosterone (TEST), cortisol (COR), and growth hormone (29, 73). Single-bouts of resistance-training elicit an acute hormonal response that may influence bone remodeling. In men, single-bouts of resistance-training have been reported to increase serum TEST concentrations (30, 48, 73-75, 114). In humans and animals, androgens stimulate bone formation and inhibit bone resorption (130). Similar to TEST, serum concentration of COR have also been reported to increase following a single-bout of resistance-training (29, 73, 114). Long-term exposure to glucocorticoids increase bone

resorption, decrease bone formation, and is a well documented secondary cause of osteoporosis (21). Serum concentration of parathyroid hormone (PTH) has been reported to increase following a single-bout of resistance-training (5, 104). PTH is released in response to decreases in plasma calcium concentration. PTH induces calcium release from the skeleton by activating bone resorption to maintain plasma calcium concentration. The effects of PTH on the skeleton appear paradoxical. Abnormal increases in endogenous PTH production, as occurs in certain diseases including hyperparathyroidism, result in reductions in skeletal mass (12, 104). On the other hand, when exogenous PTH is administered intermittently, PTH may have an anabolic effect on the skeleton (78) and appears to be a viable treatment for osteoporosis (12).

Acute bone turnover response to exercise

Biochemical markers of bone turnover are often used to track short term changes in bone turnover that lead to long term changes in BMD (110). These markers are for demonstrating the potential effectiveness of pharmacological treatments and short-term exercise programs lasting less than one year (110). Bone-specific alkaline phosphatase (BAP) is a serum marker of bone formation that is specifically secreted by the osteoblasts during mineralization (110). Alkaline phosphatase is associated with the plasma membrane of many cells (132). The exact function of alkaline phosphate is unknown (110, 132). The bone-specific isoform, BAP, is created by post-translational carbohydrate modifications (80).

Acid phosphatase is a lysosomal tissue found in many cell types, including bone (132). Tartrate-resistant acid phosphatase (TRAP), specifically the 5b isoform (TRAP5b), is specific to the osteoclasts (110). The origins of TRAP, isoform 5a (TRAP5a), are not

completely known but are believed to originate from macrophages (110). TRAP5b is produced by the osteoclasts beginning early in cell development (50). TRAP5b is a marker of osteoclast number and activity and has become a reliable serum marker of bone resorption (50, 60). TRAP5b may aid in bone resorption itself by reacting with hydrogen, and it produces reactive oxygen species (51). TRAP5b has been reported to correlate with other markers of bone turnover and BMD (50) and to predict fracture risk (110).

Bone turnover markers have been reported to predict changes in BMD during hormone replacement therapy (HRT) (25, 105) and bisphosphonate treatment (94); however, the ability of these markers to predict fracture risk is controversial (86, 110). For instance, Chestnut *et al.* (25) reported that postmenopausal women receiving HRT with the greatest decreases in urinary NTX concentration during six months of treatment had the greatest gains in BMD following one year of treatment (25). In contrast, Marcus *et al.* (90) concluded that markers of bone turnover were of little value for predicting changes in BMD in postmenopausal women receiving HRT (90). BAP and TRAP5b respond to bisphosphonate treatment that results in increased BMD. Nenonen *et al.* (94) reported that following 12 months of alendronate treatment in healthy postmenopausal women increased lumbar spine BMD and decreased serum concentrations of BAP and TRAP5b. Changes in lumbar spine BMD after 12 months correlated significantly with changes in serum TRAP5b concentration ($r=-0.32$, $p=0.005$) (94).

Changes in bone turnover markers have been reported following exercise interventions. Bemben *et al.* (10) reported in middle-aged women that OC concentration tended to increase ($p>0.05$) following six months of resistance-training and that changes in OC concentration correlated with changes in total hip BMD ($r=0.42$, $p=0.04$). The

authors reported that no significant increases in hip BMD were observed following six months of resistance-training (10). In men, Guadalupe-Grau *et al.* (45) reported that strength training combined with plyometrics for nine weeks increased BMD of the whole body and lumbar spine by 0.8% and 2.0%, respectively. Serum OC concentration increased by 45%; however, a correlation between changes in OC and changes in BMD was not observed (45). The ability of bone turnover markers to predict changes in BMD has not been fully described.

A single-bout of resistance-training has been reported to decrease serum and urinary markers of bone resorption. Whipple *et al.* (136) reported serum concentrations of type I collagen N-telopeptide (NTX, a marker of bone resorption) to decrease one and eight hours following a single-bout of resistance-training compared with baseline concentration. The concentration of NTX had returned to baseline concentrations by 24 hours following resistance-training. In addition BAP increased significantly one hour following resistance-training and returned to baseline concentration by 24 hours following resistance-training (136).

Ashizawa *et al.* (4) reported reductions in both bone resorption and bone formation markers following a single-bout of resistance-training in male subjects. Plasma concentration of procollagen type I carboxy-terminal peptide (PICP, a marker of bone formation) and serum concentration of TRAP significantly decreased compared with baseline concentration one day following a single-bout of resistance training ($p<0.05$ and $p<0.01$, respectively). Ashizawa and colleagues reported that serum concentration of BAP significantly decreased compared to baseline concentration two and three days following a single-bout of resistance training ($p<0.01$) (4). The usage of different bone

turnover markers and the time frame of sampling make comparisons among these studies difficult.

Besides resistance-training, other modes of exercise including walking, running, and cycling have been reported to acutely alter concentrations of bone turnover markers. Welsh *et al.* (134) reported that serum concentrations of BAP and OC did not change in response to 30 minutes of intense treadmill walking in young men (134). Urinary deoxypyridinoline and pyridinoline concentrations significantly increased the day of the exercise and were 42.3% and 38% greater, respectively, the day after exercise (134). In postmenopausal women, 90 minutes of treadmill walking resulted in significant increases in PICP concentration 24 and 72 hours after exercise and a significant decrease in serum concentration of C-terminal pyridinoline cross-linked telopeptide of type I collagen (ICTP, a marker of bone resorption) one hour after exercise (121). After running a marathon, men have been reported to have significantly lower serum aminoterminal propeptide of type I collagen (PINP) concentration and significantly greater ICTP concentration immediately following the race compared with prior to the race (76). Interestingly, PINP concentration was significantly lower three days following the race compared with prior to the race (79).

Maimoun *et al.* (88) reported in men (average age 24.4 years) that following 50 minutes of cycling above ventilatory threshold (VT), serum concentration of OC had significantly increased at the end of exercise compared with before exercise. Serum concentration of BAP increased 30 minutes into exercise, regardless of whether intensity was above or below VT. Serum concentration of type I collagen C-telopeptide (CTX, a

marker of bone resorption) decreased 30 minutes into exercise and immediately after exercise compared with before exercise (88).

The response of bone turnover markers to plyometrics has not been fully elucidated. Some, but not all, reports of impact loading or jump training programs have been reported to alter concentration of bone turnover markers. In postmenopausal women, intense physical training that included strength training and jump training improved BMD of the lumbar spine; however, the training program did not illicit changes in serum concentrations of OC and CTX (67). In agreement, Vainionpaa *et al.* (127) reported that following a twelve month plyometric training program, serum PINP and TRAP5b concentration did not significantly change in pre-menopausal women (127). Elsewhere, Shibata *et al.* (111) reported that BAP concentration significantly increased by 12.3% ($p<0.05$) in pre-menopausal, Japanese women following six months of walking and jump training compared with a walking program alone (111). The authors reported that serum OC and NTX concentrations did not significantly change as a result of jump training (111).

A cross-sectional report of female athletes reported that women participating in high-impact sports (i.e., volleyball and basketball) and medium-impact sports (i.e., soccer and track) had significantly greater serum concentration of OC compared with low-impact sports (i.e., swimming) (28). A second cross-sectional study of male and female runners reported that serum concentrations of PICP and ICTP were 18% and 22.2% lower than compared with controls; however, no differences between OC and BAP were reported (17). Lima *et al.* (83) reported that children, ages 12-18 years, participating in impact loading (i.e., gymnastics, tennis, and basketball) had significantly greater BMD,

but significantly lower concentrations of BAP compared with children participating in active loading (i.e., swimming and water polo) (83). Consistent changes in the concentration of bone turnover markers induced by jump training interventions in women have not been consistently reported.

The influence of feeding and energy balance on markers of bone turnover

Bone turnover markers display large biological variability. Circadian variation, seasonal changes, and menstrual cycle are some of the factors that contribute to the biological variability (26, 86). Bone turnover markers have a circadian rhythm (56, 107) and diurnal rhythm (42, 62, 108) with peak concentrations achieved in the early morning before reaching a nadir in the afternoon. Typically serum measures of bone turnover have less variability than urinary markers (86). To further minimize the variability, protocols for assessing serum bone turnover markers typically include measurement following an overnight fast and no exercise for 24 to 48 hours prior to measurement. Clowes *et al.* (27) reported that the bone formation markers procollagen type I N-terminal propeptide (PINP) and OC, as well as several bone resorption markers (urinary NTX, serum NTX, urinary CTX, serum CTX, and urinary free deoxypyridinoline) were suppressed in the fed state compared with an eight-hour fast. PINP was 3.8% lower and serum CTX was 17.8% lower in the fed state compared with the fasted state ($p<0.05$). The results of this study indicate that markers of bone turnover are suppressed following feeding (27).

Energy balance also affects bone turnover. Ihle & Loucks (59) reported that concentrations of bone resorption and bone formation makers are altered in response to short-term (5 days) of low energy availability, i.e., energy intake – exercise energy

expenditure. Serum OC and PICP were suppressed at all restricted energy availabilities of 10, 20, and 30 kilocalories per kg of lean body mass per day (kcal/kgLBM/day). Urinary NTX concentration and indices of bone resorption/formation uncoupling significantly increased only during the energy availability treatment of 10 kcal/kgLBM/day (59). Likewise, energy balance alters the response of bone formation and resorption markers to exercise. For example, Zanker and Swaine (141) demonstrated that three consecutive days of a 60-minute run at 75% of VO_2max, suppressed PINP, a marker of bone formation, but only when participants were in negative energy balance (141). Thus, studies of the effects of exercise on bone turnover should control for energy status.

Influence of feeding on the hormonal response to resistance-training

As mentioned previously, a single-bout of resistance-training has been reported to increase the rate of skeletal muscle protein turnover (13) and increase the serum concentrations of several hormones important in bone remodeling (29, 73). Feeding prior to or immediately following a single-bout of resistance-training reportedly alters the hormonal response to resistance-training (17, 23, 122, 137).

Testosterone, growth hormone, and insulin are important anabolic hormones, the response of which may be altered by feeding either before or after a bout of resistance-training. Chandler *et al.* (23) reported that TEST concentration increased following resistance-training regardless of the composition of a meal consumed prior to exercise, although the TEST response was attenuated one hour following resistance-training after consumption of CHO and CHO/PRO compared with water (23).

Cortisol is a known catabolic hormone increases in response to a single-bout of resistance-training (29, 73). Thyfault *et al.* (122) reported that consumption of CHO prior to resistance-training did not alter the predicted increase in COR concentration following resistance-training (122). In contrast, Bird *et al.* (13) reported that consumption of a liquid CHO or CHO/EAA beverage suppressed the response of COR to a single-bout of resistance-training following a four hour fast and placebo beverage consumption (13).

RATIONALE

Bone turnover markers have been reported to predict changes in BMD during hormone replacement therapy (25, 105) and bisphosphonate treatment (94); however, the ability of these markers to predict fracture risk is controversial (86, 110). The relationship between changes in BMD and changes in bone turnover markers during exercise interventions has not been fully described. As mentioned previously, changes in bone turnover markers do not necessarily result in increases in BMD (10). Changes in bone turnover markers and changes in BMD are not necessarily correlated following exercise interventions (45). Changes in bone turnover are related to changes in fracture risk independent of changes in BMD (15).

A single-bout of resistance-training has been described to decrease bone resorption markers and alter bone formation markers (4, 136). The short term changes in bone turnover markers following a single-bout of exercise and this relationship to long term changes in BMD has not been fully described. The present study is part of a one year exercise intervention to describe changes in BMD in men following a resistance or plyometric training program. In addition, the study will measure the response of bone

turnover markers to a single-bout of resistance-training or plyometrics before and after long term training. The study will be able to describe the relationship between changes in bone turnover markers and changes in BMD. It is important to establish the relationship between changes in BMD and changes in bone turnover markers following exercise interventions to strengthen current recommendations regarding exercise to prevent osteoporosis and slow bone loss during aging.

The hormonal response to a single-bout of resistance-training has been reported to not be different between young and middle-aged men. Different responses have been reported between elderly men and middle aged or young men. The hormonal response following a single-bout of resistance-training still occurs but appears to be less responsive in elderly men (7, 48, 114). Markers of bone turnover increase during aging with a rapid increase observed in women following menopause. The increase in bone turnover with aging in men is more gradual (132). Because differences in the hormonal response to resistance-training may not appear until greater than the age of 65 years, a wide age range was included in the present study of between ages 24 and 65 years. This age range was also selected to reduce the chance of injury due to the intensity of the exercise bouts. In addition, due to the time constraints involved in the study, we anticipate difficulty recruiting participants and a large age range will allow more participants to qualify for participation.

Physically active men were selected for inclusion in the present study. Men participating in a resistance-training program or a plyometrics program have become accustomed to this form of training. Because of the training effect, the response of bone turnover markers and hormones in strength trained men will be different than the

response in endurance trained or sedentary men. A completely inactive population of men would not likely be able to complete the training protocols without an increased chance of injury. Men that do not participate in weight-bearing activities often have reduced BMD of the lumbar spine (100). Thus, an active population was chosen to decrease the chance of injury, allow for a wide range of inclusion, and to mimic the population that may benefit the most from recommendations to prevent bone loss and osteoporosis.

Bone turnover markers display a large amount of biological variability. To minimize the variability, protocols for measuring bone turnover markers typically include an overnight fast and no exercise for 24 to 48 hours prior to measurement. Markers of bone turnover are suppressed in response to feeding (27). Bone turnover markers have been reported to change in response to a single-bout of resistance-training when subjects are fed and in energy balance (4, 136). The response of bone turnover markers to a single-bout of resistance-training or plyometrics may be different in the fed state compared with the fasted state. Resting for 24 to 48 hours prior to measurement may also alter the response of bone turnover markers to a single-bout of exercise. Positive changes in bone remodeling may be missed by measuring bone turnover markers in the fasted and rested state.

The resistance-training and plyometric bouts in the present study were designed to maximally load the skeleton at the hip and spine. This design may differ from training programs designed to improve maximal strength, muscular hypertrophy, or power. Bone remodeling may be activated by a fluid shift through the bone that is detected by the osteocytes (71, 123). The shift of fluid through the bone that activates the bone

remodeling process may need a short amount of time to rest before being disrupted again (102, 117). In support of this, rest periods inserted between load exposures have been reported to enhance the osteogenic effect of applied strain (102, 117). Full skeletal recovery from a strenuous loading bout likely takes between four and eight hours (102). In the present study, three-minute rest periods between sets of resistance-training and two-minute rest periods between sets of jumps were included to allow the skeleton to rest following the application of strain. In addition, to allow for the skeleton further rest between individual jumps, a ten-second rest period was inserted between jumps. These rest periods are greater than what might be expected to induce a hormonal response following a bout of resistance-training (29, 73), but have still been reported to induce a change in bone turnover markers following a single-bout of resistance-training (4, 136). Shorter rest periods between sets may generate a greater hormonal response, but the purpose of the present study is to induce alterations in bone turnover markers which may not occur in combination with a change in circulating hormone concentrations.

PURPOSES & HYPOTHESES:

The purpose of the following study was three-fold:

PURPOSE #1: The first purpose of the present study was to describe the acute effects of a single-bout of resistance-training or plyometrics in the fed or fasted state on the concentrations of markers of bone formation, bone resorption, and hormones in serum. The acute response of these markers and hormones to exercise might help plan future exercise interventions for the maintenance of skeletal health. An additional purpose was to examine the changes in markers of bone turnover and hormones which occur

throughout the morning in the absence of exercise by examining the differences between exercise trials and a no-exercise, fasted control trial.

PURPOSE #2: The second purpose of the present study was to compare the effects of exercise mode (i.e., plyometrics vs. resistance-training) and energy status (i.e., fed vs. fasted) on serum concentrations of bone turnover markers and hormones.

PURPOSE #3: The final purpose of the present study was to compare the effects of exercise mode (i.e., plyometrics vs. resistance-training) and energy status (i.e., fed vs. fasted) on both the activity of the osteoblasts and osteoclasts and on circulating hormone concentration during the two hours post-exercise, as quantified by area under the curve (AUC). Furthermore, the purpose of the present study is to determine whether the osteoblast and osteoclast activity in response to exercise is different from that which occurs throughout the morning in the absence of exercise. In addition, the purpose of the present study is to determine the relationships between potential osteoblast and osteoclast exposure to hormones during the two hour post-exercise interval and bone cell activity by examining the correlations between bone turnover markers and hormones.

To assess bone formation, serum concentrations of BAP was measured. To assess bone resorption, serum concentrations of TRAP5b was measured. Serum hormone concentrations of COR, PTH, and TEST were also measured because these hormones might mediate exercise-induced changes in osteoblast and osteoclast activity.

HYPOTHESIS #1: We hypothesized that a single-bout of resistance-training or plyometrics would increase bone formation and decrease bone resorption. We also hypothesized that hormone concentration would increase following a single-bout of resistance-training or plyometrics regardless of feeding status. We hypothesized that

bone formation, bone resorption, and hormones would decrease following a no-exercise, fasted control trial due to the previously reported diurnal rhythm of these bone markers and hormones.

HYPOTHESIS #2: We hypothesized that feeding would both decrease bone formation and hormones compared with a ten-hour fast and increase bone resorption. We hypothesized that resistance-training would induce greater concentrations of TEST and COR compared with plyometrics, but that there would be no difference in PTH concentration between exercise modes (i.e., PLY vs. RT). Furthermore, we hypothesized that plyometrics would induce greater changes in bone formation and lesser changes in bone resorption compared with resistance-training.

HYPOTHESIS #3: Finally, we hypothesized that resistance-training or plyometrics would increase the response of osteoblasts and decrease the response of osteoclasts as assessed by the AUC analysis. The hormone concentration following a single-bout of resistance-training or plyometrics would be increased but this increase would be attenuated by consumption of a liquid meal replacement prior to exercise compared with a ten-hour fast. We hypothesized that bone resorption markers would be positively correlated with PTH and COR. We hypothesized that bone formation markers would be positively correlated with TEST. We also hypothesized that bone resorption would not be significantly correlated with TEST.

METHODS

Experimental Design. The following study was a partially randomized, cross-over design. Each subject (n=12) performed four exercise trials with a minimum of five days between trials: resistance-training without feeding (RT-); plyometrics without feeding (PLY-); resistance-training with feeding (RT+); and, plyometric trial with feeding (PLY+). A subset of the participants (n=6) also performed a no-exercise, fasted control trial (CON).

Participants performed the fasted exercise trials first, in random order. After completing the plyometrics and resistance-training trials in the fasted condition, subjects performed the trials in the fed condition in the same order. For the six subjects a control (CON) trial in the fasted state was performed during the week between the fasted and fed exercise trials. **Figure One** is a flow diagram displaying the order of the exercise trials.

For the fasted exercise trials, subjects consumed only water after their evening meal; participants remained fasted until completion of the blood collection two hours post-exercise. For the fed exercise trials, subjects consumed a liquid meal replacement two hours prior to exercise sessions. The nutrient composition of the liquid meal replacement was as follows: 500 kilocalories, 12 grams of fat, 80 grams of carbohydrate, 18 grams of protein, 500 mg of calcium, and 240 IU of vitamin D (Wal-Mart Stores, Inc., Bentonville, AR).

Prior to each exercise and control trial, a baseline blood sample (15 mL) was drawn in the morning between 6:00 AM and 10:30 AM via the antecubital vein by a trained phlebotomist. Blood was drawn in the morning to control for the circadian variation in serum hormones (52) and bone turnover makers (56, 107). Blood was drawn

immediately prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following the PRE blood sample. **Figure Two** is a flow chart of the blood draws performed during each exercise and control trial.

Participants. Twelve apparently healthy, physically active men between the ages of 24 and 65 years (n = 12; age 42.8 ± 4.2 y, height 181.1 ± 1.3 cm, weight 79.1 ± 2.9 kg) were recruited from the University of Missouri and Columbia, Missouri community by flyers posted on campus, at local bicycle and sporting goods stores, on the web sites of local cycling and running clubs, and by campus email. Subjects were determined to be physically active if participated in a minimum of five hours per week of purposeful exercise. Subjects were instructed to maintain their regular exercise program throughout the study, but to refrain from exercise 24 hours prior to each trial and 24 hours following each trial. Prior to participation, all participants were informed of the risks associated with the study, read a consent form, and provided written consent. The present study was conducted in accordance with the guidelines in the Declaration of Helsinki and was approved by the University of Missouri Health Sciences Institutional Review Board.

Inclusion criteria were as follows: male, aged 25 to 65 years, apparently healthy, and current participation in approximately five hours per week of purposeful exercise, excluding participation in consistent resistance-training or plyometric training. Exclusion criteria included current or previous medical condition affecting bone health, use of medications affecting bone health, implanted metal that would interfere with determination of BMD, recent fracture, cigarette smoking, excessive alcohol consumption, irregular sleep/wake cycle, or BMD of the hip or lumbar spine that would be classified as osteoporotic as defined by the World Health Organization (WHO). The

WHO definitions of osteoporosis and osteopenia were used to categorize participants as having normal BMD of the spine or hip (T-score > -1.0), osteopenia (T-score ≤ -1.0 and > -2.5), or osteoporosis (T-score ≤ -2.5) as established by the manufacturer's means for a young, adult population (65).

Anthropometric data. Participant body mass was determined to the nearest 0.05 kg. Height was determined to the nearest 0.5 cm. These results were used to calculate body mass index (BMI) (expressed in kilograms per square meter, kg/m^2).

Training and Diet Records. Current physical activity was quantified using a seven-day written training log listing activity type, duration, and intensity. The Compendium of Physical Activities was used to estimate daily energy expenditure during purposeful exercise only (2). Nutrient intake was assessed using a seven-day written diet record. The diet record, not including multivitamin-mineral supplements, was analyzed for energy, macronutrient, and micronutrient content (Food Processor 10.2; ESHA, Salem, OR).

Bone loading history. The Historical Leisure Activity Questionnaire (HLAQ) was used to collect information regarding training type, frequency, and duration throughout the lifespan. The HLAQ was developed to measure historical leisure time physical activity across the life span and relate the lifetime physical activity to bone density in postmenopausal women (77). The original HLAQ has been successfully modified for self-administration with good reliability for lifetime vigorous-intensity activities (24) and used successfully in this laboratory (99).

Bone-loading scores were quantified for adolescence (13-18 years of age), young adulthood (19-29 years of age), and adulthood (>30 years of age). Biomechanical ground

reaction forces (GRF) were used to calculate the bone-loading scores using methods established by Groothausen *et al.* (43). Based on the GRF, activities were scored into four categories: 0 (GRF < 1 times body weight; such as cycling, swimming, etc.), 1 (GRF between 1 x and 2 x body weight; such as rowing, etc.), 2 (GRF between 2 x and 4 x body weight; such as jogging), 3 (GRF > 4 x body weight; such as volleyball, basketball, gymnastics, etc.). A bone-loading exposure score (GRF–LV) was then calculated by the products of the GRF activity score (between 0 and 3), frequency, and duration for each period of life: adolescence, young adulthood, and adulthood. The GRF-LV scores for each period of life were summated to produce the lifetime GRF–LV score. The lifetime GRF–LV score for each individual was annualized (ANN GRF-LV) by dividing the lifetime GRF-LV score by the number of years > 13 years of age.

In addition to the GRF, Effective Load Stimulus (ELS) scores were used to calculate bone-loading scores by methods established by Weeks *et al.* (133). A summary of the ELS scores in available in **Table One.** Based on the ELS, activities were scored and a second set of bone-loading exposure scores (ELS-LV) was then calculated by the products of the ELS, frequency, and duration for each period of life: adolescence, young adulthood, and adulthood. The ELS-LV scores for each period of life were summated to produce the lifetime ELS-LV score. The lifetime ELS-LV score for each individual was annualized (ANN ELS-LV) by dividing the lifetime ELS-LV score by the number of years > 13 years of age.

Bone mineral content, density & body composition testing. Dual energy X-ray absorptiometry (DXA) (Hologic, Waltham, MA) was used to measure bone mineral content (BMC) and areal BMD of the whole body, lumbar spine, and total left hip by

performing three separate scans. The whole body scan was also used to determine body composition. Areal BMD (in grams per square centimeter, g/cm^2) is calculated by dividing BMC (in rams, g) by bone area (in centimeters squared, cm^2) by the software associated with the DXA scanner (Hologic QDR 4500A). The CVs for BMC and BMD of the lumbar spine and hip were < 1%.

Dietary Controls. Participants were instructed to record the meal consumed the evening prior to the first exercise trial and the meal consumed the evening after the trial (prior to the 24-hr blood draw). The subjects consumed these same meals prior to the subsequent trials.

Plyometrics trials. Each PLY session consisted of one set of ten repetitions for each of twelve different jumps with a ten-second rest period between jumps and a two-minute rest period between exercises. The following jumps were performed with a ten-second rest between jumps: squat jump, forward hop, lateral jump, split squat, lateral box push off, lateral hurdle, single-leg lateral, depth jump (10 cm height), and a jump off the box (10 cm height). Another series of jumps was completed in continuous fashion of two sets of five jumps, separated by a 20 second rest between sets and two-minute rest between exercises. Bounding, lateral bounding, box drill, and zigzags were completed in continuous fashion. The entire PLY session was performed in the following order: squat jump, forward hop, lateral jump, split squat, lateral box push off, bounding, box drill, lateral hurdle, zigzag, single-leg lateral hurdle, depth jump, and jump off the box. Subjects were instructed and encouraged by study personnel to perform each jump with maximal vertical effort.

Resistance-training trials. Each RT session consisted of three sets of ten repetitions for each exercise. The first set was performed at 60% of 1-RM (or 60% of 10-RM where appropriate). The second and third sets were performed at 80% of 1-RM (or 80% of 10-RM where appropriate). The exercises were performed in the following order: squat, dead lift, military press, lunge, bent over row, and calf raise. Following these exercises, participants performed two sets of ten repetitions of abdominal crunches and two sets of ten repetitions of low back extensions on a stability ball. All sets and exercises were separated by three-minute rest periods. Subjects were instructed and encouraged by study personnel to complete the prescribed number of repetitions for each lift.

Baseline familiarization and testing. Prior to participation in the trials, subjects visited the McKee Fitness Center for familiarization with the plyometrics and resistance-training. All exercise trials and familiarization were preceded by a ten-minute aerobic warm-up and followed by both a five-minute aerobic cool-down and a five-minute period of static stretching. Familiarization with the plyometrics consisted of successful completion of two to three, low-intensity jumps for each of the exercises. After the plyometric familiarization, participants were familiarized with the resistance-training exercises. Familiarization with the resistance-training exercises consisted of successful completion of low-resistance lifts. Participants were instructed how to perform each jump and lift safely, using proper technique, by qualified study personnel. Following resistance-training familiarization, participants underwent one-repetition maximum testing (1-RM) for squat, military press, dead lift and bent-over row. Following 1-RM testing, a ten-repetition maximum (10-RM) was determined for the lunge and calf-raise.

All exercises, where appropriate, were performed using standardized barbell and weight plates. Maximal strength testing was performed using the guidelines established by the National Strength and Conditioning Association (NSCA) (6).

Serum measures of hormones and bone turnover markers. Blood was dispensed into plasma separator tubes containing EDTA. The blood was centrifuged at 2000*g* for 15 minutes and the plasma removed and immediately frozen at -80 ºC. The concentration of total TEST, intact PTH, and COR were determined using a commercially available chemiluminescent immunoassay (Immulite 1000; Diagnostic Products, Los Angeles, CA). BAP was measured by ELISA (Quidel Corporation, San Diego, CA). Cross-reactivity of the anti-human BAP antibody is 3% to 8% with liver alkaline phosphatase and 0.4% with intestinal BAP. TRAP isoform 5b was measured by ELISA (Quidel Corporation, San Diego, CA). All CVs for BAP and TRAP5b were less than 10%. All samples for each subject were assayed on the same day to control for day-to-day variability. Hematocrit was analyzed in duplicate using a standard microcapillary technique. Hematocrit was used to determine changes in plasma volume using the method established by van Beaumont (129).

$$\%\Delta PV = [100/(100-H_1) * [100(H_1-H_2)]/H_2$$

Where H_1 = original hematocrit and H_2 = final hematocrit.

Statistical Analysis. Descriptive statistics (mean ± SEE) were performed on demographic, anthropometric, nutrient intake, serum hormones, physical activity, sports history, and bone loading variables. To meet the assumptions of ANOVA of normal distribution and equal variances, data were log transformed prior to analysis when necessary.

We hypothesized that concentrations of BAP, COR, PTH, and TEST would increase and TRAP5b concentration would decrease following RT or PLY, regardless of feeding status. To test this hypothesis, a one-factor (time), RMANOVA (n=12) was performed for each exercise trial to determine the main effect of time for each trial individually. In addition, to determine if the bone turnover markers and hormones exhibit changes following RT or PLY different than what occurs throughout the morning in the absence of exercise, a one-factor RMANOVA was performed for a no-exercise, fasted control (CON) trial (n=6). Post hoc pairwise comparisons were performed when there was a significant time effect. After the one-factor (time) RMANOVAs were completed the analyses was performed again including age as a covariate.

We hypothesized that concentrations of BAP, COR, PTH, and TEST would be reduced by feeding following RT or PLY, and that concentrations of TRAP5b would be increased by feeding following RT or PLY. We hypothesized that plyometrics would induce greater concentrations of BAP and lesser concentrations of TRAP5b compared with resistance-training. Furthermore, we hypothesized that resistance-training would induce greater concentrations of TEST and COR compared with plyometrics, but that there would be no difference in PTH concentration between exercise modes (i.e., PLY vs. RT). To test this hypothesis, a 2 x 2 x 7 RMANOVA was performed to test for significant main effects and interaction between exercise mode, energy status, and time (n=12). In the case of a significant interaction between exercise mode or energy status and time, an independent t-test was performed to test for differences between factors at each time point. Additionally, a one-factor RMANOVA was performed to determine significant differences between the exercise and control trials at each time point (n=6).

We hypothesized that RT or PLY would increase the response of osteoblasts and decrease the response of osteoclasts as assessed by an AUC analysis. Additionally, we hypothesized that TEST, COR, and PTH following RT or PLY would increase, but that increase would be attenuated by consumption of a liquid meal replacement prior to exercise compared with a ten hour fast. An area under the curve (AUC) analysis was performed to quantify the response of the bone turnover markers and hormones during the two hour time period following each trial. The time points between POST and 120 MIN were used to determine the AUC by finding the area above or below the concentration of each variable at POST. This method has been established for determining the AUC of glucose following a glucose tolerance test (120). In this fashion, the activity of the osteoblasts and osteoclasts can be quantified. In addition, the exposure of the bone cells to TEST, COR, and PTH may also be quantified. To test this hypothesis, a 2 x 2 ANOVA was performed to compare the differences of the AUC between exercise mode (i.e., PLY vs. RT) and feeding (i.e., feeding vs. fasted) (n=12). Additionally, a one-factor RMANOVA was performed to determine significant differences between the exercise and control trials (n=6). Post hoc pairwaise comparisons were performed when there was a significant effect of trial.

We hypothesized that the AUC of TRAP5b would be positively correlated with the AUC of PTH and COR. We also hypothesized that AUC of BAP would be positively correlated with AUC of TEST. To test this hypothesis, bivariate relationships between the AUC of TRAP5b and the AUC of PTH and COR were evaluated using Pearson's correlations. All statistical analyses were performed using PASW/SPSS, version 18.0. Statistical significance was established at an alpha of $p<0.05$ for all analyses.

RESULTS

Participant demographic, anthropometric, nutrient intake, serum hormones, physical activity, sports history, and bone loading descriptive statistics are displayed in **Table 2.** Mean calcium intake (1235 ± 219 mg/d) was greater than the current Adequate Intake (AI) for calcium recommended by the U.S. Dietary Reference Intakes (DRIs) (37). In addition, participants consumed 6.0 ± 2.0 μg/d of vitamin D. The AI for men above the age of 50 years is 10 μg/d of vitamin D. In the present study, men over the age of 50 consumed 6.1 ± 3.0 μg/d. The AI for men 19-50 years is 5 μg/d of vitamin D. In the present study, men under the age of 50 consumed 5.9 ± 3.0 μg/d. Serum measures of 25-dihydroxyvitamin D (25(OH)D) would be necessary to verify subject vitamin D status and are beyond the scope of the present report.

To test the hypothesis that either RT or PLY would increase serum concentrations of COR, PTH, TEST and BAP and decrease serum concentration of TRAP5b, one-factor (time) RMANOVAs were performed for each exercise trial (n=12). Serum BAP concentration did not significantly change during the two hours following any exercise trial (PLY-, RT-, PLY+, and RT+) (n=12, main effect for time, $p>0.05$) (**Table 3, Fig. 3**). Serum TRAP5b, COR, and PTH concentrations significantly decreased during the two hours following each exercise trial (PLY-, RT-, PLY+, and RT+) (main effect for time, $p<0.05$) (**Table 3, Fig. 4**, **5**, and **6**, respectively). Serum TEST concentration significantly decreased during the two hours following PLY-, PLY+, and RT+ (main effect for time, $p<0.05$), but did not significantly change during the two hours following RT- (main effect for time, $p>0.05$) (**Fig. 7**). Following PLY+, COR, PTH, and TEST concentrations were significantly greater at 24HR than compared with PRE ($p<0.05$)

(**Table 3**). Following RT+, COR and TEST concentrations were significantly greater at 24HR than compared with PRE ($p<0.05$) (**Table 3**). No significant interactions between time and age were observed except for PTH during RT+ ($n=12$) ($p<0.05$).

Diurnal variation was assessed by determination of a significant main effect for time following the control trial. To test whether the significant changes observed following the exercise trials were the result of the absence of exercise throughout the morning, one-factor (time) RMANOVAs were performed for the no-exercise control trial and each exercise trial ($n=6$; **Table 4**). Serum BAP concentration significantly decreased during the two hours following CON ($n=6$, main effect for time, $p<0.05$) (**Fig. 8**), and remained significantly less than PRE at 24HR ($p<0.05$). Likewise, serum COR concentration significantly decreased during the two hours following CON. By contrast, TRAP5b, PTH, and TEST did not exhibit significant diurnal changes during the early to late morning. Serum BAP concentration did not change during the two hours following RT and PLY, regardless of feeding ($n=6$, main effect for time, $p>0.05$) (**Fig. 8**). Serum TRAP5b significantly decreased during the two hours following RT+ ($n=6$, main effect for time, $p<0.05$) (**Fig. 9**). Serum COR concentration did not significantly change during the two hours following RT+ ($n=6$, main effect for time, $p<0.05$) (**Fig. 10**). Serum PTH concentration significantly decreased during the two hours following PLY-, PLY+, or RT+ ($n=6$, main effect for time, $p<0.05$) (**Fig. 11**). RT and PLY prevented reductions in BAP concentration and decreased TRAP5b concentration. RT+ prevented reductions in COR concentration and reduced TRAP5b concentration ($n=6$ only).

To test the hypothesis that feeding would decrease the concentrations of BAP, COR, PTH, and TEST and increase the concentration of TRAP5b, a 2 x 2 x 7

RMANOVA (n=12) was performed to compare the main effects of exercise mode (i.e., RT vs. PLY) and feeding (i.e., fed vs. fasted) and to test for significant interactions between exercise mode, energy status, and time. A significant main effect for time was detected for BAP, TRAP5b, COR, PTH, and TEST (**Fig. 3**, **4**, **5**, **6**, and **7**, respectively). A significant interaction between exercise mode and time was detected for COR only. To determine the differences between RT and PLY in COR concentration, an independent t-test was performed at each time point. The concentration of COR at POST, 15MIN, and 30MIN was significantly greater following the RT than PLY ($p<0.05$) (**Fig. 13**).

Additionally, to test the hypothesis that feeding would decrease the concentrations of BAP, COR, PTH, and TEST and increase the concentration of TRAP5b, and that these changes were different than the response in the absence of exercise throughout the morning, a one-factor (trial), RMANOVA (n=6) was performed to compare the means values at each time point among trials (RT+/-, PLY+/-, CON). No significant differences in serum BAP, TRAP5b, or TEST concentrations were detected among trials at any time point. Significant differences in serum COR at 15MIN and 60MIN were detected among trials (**Table 5**). COR concentration at 15MIN and 60MIN was significantly greater during RT+ than CON ($p<0.05$). A significant difference in serum concentration of PTH at PRE, 15MIN, 30MIN, 60MIN, and 120MIN was detected among trials (**Table 5**). As depicted in **Fig. 11** and reported in **Table 5**, PTH following PLY- increases to increase to a greater extent than RT- or any of the trials following feeding.

A 2 x 2 ANOVA (n=12) was performed to test for main effects and interactions of exercise mode (RT vs. PLY) and feeding (fed vs. fasted). The AUC of COR was

significantly greater following PLY compared with RT (main effect of exercise mode, $p<0.05$) (**Fig. 14**). There were no exercise mode or energy status main effects for BAP, TRAP5b, PTH, and TEST (**Table 6**). A one-factor (trial), RMANOVA was performed to compare exercise trials to CON to determine if the response of bone turnover markers and hormones to exercise is different than the response in the absence of exercise throughout the morning. No significant differences between trials were detected (n=6) (**Table 7**).

We also hypothesized that AUC of BAP would be positively correlated with AUC of TEST, but not correlated with the AUC of COR or PTH. We hypothesized that the AUC of TRAP5b would be positively correlated with the AUC of PTH and COR. We hypothesized that the AUC of TRAP5b would not be significantly correlated with the AUC of TEST. To test whether significant correlations existed between the bone turnover markers and the hormones a Pearson Coefficient Correlation Matrix was determined. AUC of TRAP5b was positively correlated with the AUC of PTH ($r=0.452$, $p=0.001$) (**Table 8**).

DISCUSSION

The purpose of this study was to investigate the effects of a single-bout of resistance-training or plyometrics on markers of bone turnover and hormones. An additional purpose was to investigate the influence of feeding prior to a single-bout of resistance-training or plyometrics on markers of bone turnover and hormones. We showed that during the two hours following a single-bout of resistance training or plyometrics BAP did not significantly change; however, following a resting control trial BAP significantly decreased. Thus, resistance-training or plyometrics may maintain BAP concentration following exercise as opposed to the decrease observed during the morning in the absence of exercise. This suggests a favorable change in bone formation following resistance-training or plyometrics. TRAP significantly decreased during the two hours following a single-bout of resistance-training or plyometrics but did not change following a resting control trial. This also suggests a favorable change in bone resorption following resistance-training or plyometrics. The decrease in PTH following plyometrics and resistance-training positively correlated with a decrease in TRAP5b, reflecting the potential influence of PTH on osteoclast activity.

Bone Turnover Response to a Single Bout of Plyometrics or Resistance-Training

We hypothesized that BAP concentration would increase and TRAP5b concentration would decrease following a bout of RT or PLY. In the present study, BAP did not significantly change following PLY or RT, regardless of feeding status. BAP concentration decreased following the resting control period. The decrease in BAP following the resting control period may reflect the diurnal variation of this marker of bone formation. Bone turnover markers have a circadian rhythm (56, 107) and diurnal

rhythm (42, 62, 108) with peak concentrations achieved in the early morning before reaching a nadir in the afternoon. These results suggest that bone formation is positively influenced by resistance-training or plyometrics. TRAP5b significantly decreased following PLY or RT but not following a resting control trial. These results suggest that bone resorption may be positively influenced by resistance-training or plyometrics.

The reported response of bone turnover markers to a single-bout of resistance-training is somewhat inconsistent (4, 104, 136). Ashizawa *et al.* (4) reported no significant changes in serum BAP or OC concentrations one day following a single-bout of resistance-training in untrained men (average age 24.5 years). The authors reported a significant decrease of 12% in PICP concentration one day following a single-bout of resistance-training. Bone resorption, assessed by TRAP concentration, decreased by 15% one day following a single-bout of resistance-training. Interestingly, the authors reported that BAP concentration significantly decreased compared with baseline concentration two and three days following a single-bout of resistance-training by 13% and 9%, respectively (4).

Rong *et al.* (104) reported that in young, healthy males (average age 23 years) a significant decrease in serum OC concentration was observed four hours following a single-bout of leg press (five sets of eight-repetitions at 85% of three-repetition maximum). The decrease observed in OC concentration was not different than the decrease observed during a resting control condition (97). Bone resorption, assessed by serum ICTP concentration, significantly decreased four hours following exercise. The authors also reported that following a resting control trial ICTP concentration did not

change (104). All exercise bouts in this trial were performed following a nine to ten hour fast.

Whipple *et al.* (136) reported no significant differences in BAP concentration before or immediately following a single-bout of resistance training in healthy, untrained men (average age 21.9 years) (15.0 U/L versus 16.1 U/L, $p>0.05$) (136). The authors reported that immediately following a single-bout of resistance-training the concentration of BAP was significantly greater than the same time point observed during a resting control trial (16.1 U/L versus 13.5 U/L, $p<0.05$). A second marker of bone formation PICP did not significantly change from baseline following resistance-training, nor was it significantly different than during a resting control trial (136). The authors reported a significant decrease in the concentration of serum NTX one and eight hours following a single-bout of resistance-training compared with baseline concentration (26.3 U/L at baseline, 20.3 U/L at 1-hr, and 18.1 U/L at 8-hr, $p<0.05$). In addition, the authors reported a significant decrease in NTX concentration immediately after, one, and eight hours following a resting control trial (24.8 U/L at baseline, 22.4 U/L immediately following, 21.9 U/L at 1-hr, and 21.3 at 8-hr, $p<0.05$). In this study, daily energy requirements were calculated using the Harris-Benedict equation and meals were prepared for subjects by study personnel (136).

The acute response of a single-bout of plyometrics on bone turnover markers has not been described in the literature. Interventions that include resistance-training and jump training may not result in changes in bone turnover markers (127) but still report increase in BMD (67). Some interventions have reported increases in BAP concentration,

but no changes in OC or NTX concentration, following jump training combined with walking in pre-menopausal women but no changes in BMD were observed (111).

The goal of any treatment for osteoporosis is to increase bone mass and reduce fracture risk. In the short-term, this can be investigated by changes in bone formation and bone resorption (110). An increase in bone formation and a decrease in bone resorption can be inferred to result in positive bone remodeling that may lead to increased BMD in the future. An increase or decrease of both bone formation and resorption simultaneously results in no positive skeletal changes. Bone turnover markers have been reported to predict changes in BMD during hormone replacement therapy (25, 105) and bisphosphonate treatment (94); however, the ability of these markers to predict changes in BMD and fracture risk is controversial (86, 110). Changes in bone turnover are related to changes in fracture risk independent of changes in BMD (15). The relationship between changes in BMD and changes in bone turnover markers during exercise interventions has not been fully described. Bemben *et al.* (10) reported in women that OC concentration tended to increase following six months of resistance-training and that changes in OC concentration correlated with changes in total hip BMD ($r=0.42$, $p=0.04$). The authors reported that no significant changes in BMD were detected in response to the resistance-training program (10). In men, Guadalupe-Grau *et al.* (45) reported that strength training combined with plyometrics for nine weeks increased BMD of the whole body and lumbar spine by 0.8% and 2.0%, respectively. Serum OC concentration increased by 45%; however, a correlation between changes in OC and changes in BMD was not observed (45). Thus, it has been hypothesized that short term changes in bone turnover markers may predict exercise-induced changes in BMD. The results of the

present study suggest that a single-bout of resistance-training or plyometrics may positively influence bone formation (BAP) and bone resorption (TRAP5b).

Hormonal Response to a Single-Bout of Plyometrics or Resistance-Training

Testosterone. We hypothesized that TEST concentration would increase following RT and PLY. In the present study, changes in serum concentration of TEST either decreased or did not change during exercise trials. In general, TEST has been reported to increase immediately following a single-bout of resistance-training (for reviews, see 29, 73).

The TEST response to a single-bout of resistance-training appears to be dependent upon many factors including intensity, volume, rest periods, and possibly dietary factors. Strength-training programs specifically designed to promote muscle hypertrophy appear to induce a greater increase in TEST concentration than those designed to promote strength and/or power gains (29, 49). Programs designed to promote muscle hypertrophy typically utilize lighter loads, greater volume, and shorter rest periods than those designed to increase strength and/or power.

In a study by Kraemer *et al.*, (74) a single-bout of resistance-training using lighter loads and shorter rest periods (hypertrophy bout) was compared with a separate bout using greater loads and longer rest periods (strength bout) in young men with recreational resistance-training experience. The strength bout included five repetition maximum loading and three minute rest periods. The hypertrophy bout included 10-RM loading and one minute rest periods. The authors reported that both programs produced significant increases in TEST concentration mid-exercise, immediately after, five minutes,

and 15 minutes following exercise compared with baseline ($p<0.05$). No difference between the separate bouts was detected (74).

In contrast, Hakkinen *et al.* (48) reported that performing 20 sets at 1-RM did not significantly increase TEST concentration in male strength athletes. In contrast, the authors reported that 10 sets of 10 repetitions performed at 70% 1-RM significantly increased TEST concentration (48). The latter protocol is representative of a program designed to induce muscle hypertrophy. All sets were separated by three minutes rest periods. Similarly, Crewther *et al.* (30) compared the TEST response of three different single-bouts of resistance-training in recreational male weight lifters. The protocols included a power bout (eight sets of six repetitions, 45% of 1-RM, three minute rest periods using ballistic power movements), a hypertrophy bout (10 sets of 10 repetitions, 75% 1-RM, two minute rest periods), or a maximal strength bout (six sets of four repetitions, 88% 1-RM, four minute rest periods using explosive intentions). Following the hypertrophy bout, salivary TEST concentration significantly increased by 26% mid-exercise and by 89% 60 minutes post-exercise when compared with pre-exercise concentration ($p<0.05$). TEST concentration did not significantly change in comparison following the power and maximal bouts ($p>0.05$) (30).

In contrast, Smilios *et al.* (115) reported that TEST concentration did not increase in response to a single-bout of resistance-training in young men with recreational resistance-training experience, regardless of protocol used. Three different bouts of resistance-training were designed to induce maximal strength, muscular hypertrophy, or strength endurance. Maximal strength included five repetitions at 88% 1-RM using three minute rest periods performed for two, four, or six sets of each exercise. Muscular

hypertrophy included 10 repetitions at 75% 1-RM using two minute rest periods performed for two, four, or six sets of each exercise. Strength endurance included 15 repetitions at 60% 1-RM using one minute rest periods performed for two or four sets for each exercise (115).

In the present study, the resistance-training and plyometric protocols were designed to produce a maximal osteogenic response in the spine and hip of men who were physically active but inexperienced weight-lifters and not currently performing resistance-training. This is in contrast to many studies which employed programs to promote muscle hypertrophy, muscular strength, power, or endurance in young men with recreational experience resistance-training (30, 48, 49, 74, 75, 115). Following a single-bout of resistance-training TEST concentration has been reported to increase in similarly in untrained men compared to strength-trained men (1).

The present study included several middle-aged men but no men considered to be elderly (greater than 65 years of age). Following a single-bout of resistance-training, TEST concentration has been reported to increase similarly in young men and in middle-aged men (48). Conflicting reports have been generated in elderly men. Hakkinen *et al.* (48) reported that TEST concentration significantly increased in young men and middle-aged men following a single-bout of resistance-training, but not in elderly men (48). Baker *et al.* (7) reported that although elderly men had significantly lower free and total TEST concentration compared to young or middle-aged men, free and total TEST concentration increased following a single-bout of resistance-training in all age groups (7). In contrast, Smilios *et al.* (114) reported that TEST concentration increased similarly between young and elderly men following a single-bout of resistance-training (114).

Energy consumption and energy balance prior to a single-bout of resistance-training may attenuate the TEST response as will be discussed shortly. Several of the reports discussed above were performed following an overnight fast (7, 48, 49, 114, 140). Yet other studies did not include fasting prior to baseline measurements or exercise (30, 74, 75). Short-term energy restriction, such as less than a 48-hour fast, has been reported to not alter concentrations of TEST (20).

Plyometrics induce the rapid development of strain in load-bearing skeletal sites both during takeoff and landing. The plyometrics program, despite inducing ground reaction forces on the skeleton of greater than three times bodyweight (133), does not induce muscular contractions with force characteristics similar to those induced resistance-training. Typically, plyometrics are utilized to increase power, a function of velocity and distance, rather than maximal strength. Crewther *et al.*, (30) mentioned previously, reported that explosive resistance-training did not increase TEST concentration (30). Likewise, Linnamo *et al.* reported that TEST concentration does not increase following a single-bout of explosive style weight-lifting using loads of 40% 1-RM (84). Although these training modes are not the same as plyometrics, it is likely a more fair comparison that a bout of resistance-training designed to promote hypertrophy or strength.

In the present study, TEST concentration decreased following a bout of resistance-training or plyometrics. In addition, the results of the present study suggest an increase in bone formation markers and a decrease in bone resorption markers occurred in response to a single-bout of resistance-training and plyometrics. In humans and animals, androgens and estrogens stimulate bone formation and inhibit bone resorption

(130). Androgens have been found to inhibit osteoclastogenesis, inhibit osteoclast activity, and stimulate osteoclast apoptosis (130). An increase in bone formation and decrease in bone resorption is expected following an increase in circulating TEST concentration. In men, serum concentration of OC has been correlated to free TEST concentration ($r=0.41$, $p<0.001$) prior to training. Following nine weeks of a combination of strength training and plyometrics, OC increased by 45% compared with baseline ($p<0.001$) and BMD of the whole body and lumbar spine significantly increased by 0.8% and 2.7% ($p<0.05$), respectively. Free TEST did not change as a result of training (45). In the present study, changes in bone turnover were observed following resistance-training or plyometrics despite a decrease in circulating TEST concentration. Thus, circulating TEST may not influence bone formation or bone resorption following a single-bout of resistance-training or plyometrics.

Cortisol. We hypothesized that COR concentration would increase following RT or PLY. In the present study, COR concentration significantly decreased following plyometrics or resistance-training. COR concentration also significantly decreased during the resting control trial. COR concentration was significantly greater following resistance-training compared with plyometrics. Similarly, the AUC of COR was significantly greater following resistance-training compared with plyometrics.

Generally, an increase in serum COR concentration following a single-bout of resistance-training has been reported (for a review, see 73). The intensity, volume, and rest periods utilized have all been implicated as mediators of the COR response to resistance-training (73). As with TEST, it is believed that programs designed to promote

muscle hypertrophy induce a greater increase in COR concentration than programs designed to promote strength (49, 72, 121, 140).

Zafeirdis *et al.* (140) explored the different hormonal responses to three different resistance-training sessions in lean young men with recreational resistance-training experience. The bouts were designed to stimulate muscle hypertrophy (four sets of 10 repetitions at 75% 1-RM using two minute rest periods), muscular strength (four sets of five repetitions at 88% 1-RM using three minute rest periods), or strength-endurance (four sets of 15 repetitions at 60% 1-RM using one minute rest periods). The study reported an increase in COR concentration immediately following and 30 minutes after the muscle hypertrophy and strength-endurance programs, but not following a muscular strength program (140).

As mentioned previously, Smilios *et al.* (114) reported that COR concentration increased following bouts designed to promote strength-endurance or muscle hypertrophy, but not following the maximal strength bout. COR concentration only increased following these bouts when four and six sets were performed, but not when only two sets were performed (114). Crewther *et al*. (30) reported that salivary COR concentration increased following a hypertrophy bout but not following a power or maximal strength bout (30). Hakkinen *et al.* (49) reported that 10 sets of 10 repetitions at 70% 1-RM increased serum concentration of COR following resistance-training, but 20 sets of one repetition at 100% 1-RM did not (49).

Unlike the present study, many of these reports used only young, recreationally trained men (49, 114, 140). The present study included several middle-aged men. Hakkinen *et al.* (49) reported that COR concentration increased in middle-aged men only

following a single-bout of resistance training but did not increase in young and elderly men (49). In contrast, Smilios *et al.* (114) reported that COR concentration increased in both young and elderly men following a single-bout of resistance-training (114). The response of COR to a single-bout of resistance-training may be modulated by age. In addition, the present study included men not participating in resistance-training. Following a single-bout of resistance-training COR concentration has been reported to increase in similarly in untrained men compared to strength-trained men (1).

In the present study, it is possible that the volume, intensity, and rest periods in the present study were insufficient to elicit an acute COR response. The decrease in COR following the resting control trial is likely a reflection of the natural diurnal rhythm of COR secretion. The concentration of COR appears to naturally peak between 0500-h and 0900-h in normal men before reaching nadir between 2000-h and 0300-h (41). Therefore, the decrease observed following RT and PLY, which also occurred following CON, may simply be the natural decrease of COR observed throughout the late-morning into the afternoon.

The present study found a decrease in cortisol concentration following a single-bout of resistance-training or plyometrics. In addition, the results of the present study suggest an increase in bone formation and a decrease in bone resorption occurred in response to a single-bout of resistance-training and plyometrics. Cortisol has been reported to directly influence bone mass (21). Glucocorticoid treatment is a well documented secondary cause of osteoporosis. Glucocorticoids increase bone resorption and decrease bone formation leading to reductions in BMD and increased fracture risk. Glucocorticoids directly decrease the replication of preosteoblasts and decrease the

function of mature osteoblasts. Thus, bone formation is suppressed and bone resorption is enhanced by COR. In men, serum concentration of OC has been negatively correlated to COR concentration ($r = -0.39$, $p<0.001$) prior to training. Following nine weeks of a combination of strength training and plyometrics, OC increased by 45% compared with baseline ($p<0.001$) and BMD of the whole body and lumbar spine significantly increased by 0.8% and 2.7% ($p<0.05$). COR concentration was not altered by nine weeks a combination of strength training and plyometrics (45). In the present study, an increase in bone formation and a decrease in bone resorption occurred following resistance-training or plyometrics concurrently with a decrease in COR concentration. The increase in bone formation and the decrease in bone resorption following a single-bout of resistance-training or plyometrics do not appear to be directly related to the post-exercise changes in COR.

Parathyroid Hormone. In the present study, PTH concentration significantly decreased in the period following RT or PLY, but did not significantly change following CON. A single-bout of resistance-training has been reported to alter the serum concentration of PTH. Ashizawa *et al.* (5) reported that PTH significantly decreased three hours after resistance-training compared with baseline (14.17 ± 2.91 pg/ml versus 8.72 ± 2.17, $p<0.05$) (5). By contrast, Rong *et al.* (104) reported that serum PTH concentration increased immediately following a single-bout of resistance training ($p<0.05$); however, the concentration returned to baseline by one hour after exercise cessation (104).

Different modes of exercise have also been reported to alter PTH concentration. Guillemant *et al.* (46) reported that in male triathletes, PTH concentration increased

during a bout of cycling (46). Likewise, Barry *et al.* (8) reported that PTH concentration increased following a bout of moderate-intensity cycling in competitive male cyclists (8). In pre-menopausal women, a combination of resistance-training and jump training for 12 months decreased serum PTH concentrations to a greater extent than compared with sedentary controls (-11.2 pg/ml vs. -2.2 pg/ml, $p<0.05$) (127).

In the present study, feeding reduced PTH concentration prior to exercise. PTH concentration decreased following a single-bout of resistance-training or plyometrics. In addition, the results of the present study suggest an increase in bone formation markers and a decrease in bone resorption markers occurred in response to a single-bout of resistance-training and plyometrics. PTH induces calcium release from the skeleton by activating bone resorption to maintain plasma calcium concentration. PTH directly acts on the osteoclasts to stimulate bone resorption and disrupts the osteoblasts' suppressive action on the osteoclasts. Abnormal increases in endogenous PTH production, as occurs in certain diseases including hyperparathyroidism, result in reductions in skeletal mass (12, 104). These results suggest that a decrease in the concentration of circulating PTH following a single-bout of resistance-training or plyometrics might mediate the decrease in a bone resorption marker observed following exercise.

Response of Bone Turnover Markers to Feeding versus Fasting

We hypothesized that feeding would decrease the concentration of BAP and increase the concentration of TRAP5b compared with fasting. The results of the present study demonstrate that energy status had no effect on BAP or TRAP5b during the 24 hours after a single-bout of RT or PLY. The results also demonstrate that exercise mode

had no effect on BAP or TRAP5b during the 24 hours following a single bout of RT or PLY.

Energy balance is important for the maintenance of bone turnover. Ihle & Loucks (59) reported that concentrations of bone resorption and bone formation makers are altered in response to short-term reduced dietary intake. In this cross-over designed study, subjects exercised daily for five days at 70% of VO_2max expending 15 kcal per kg of lean body mass (LBM) per day (kcal/kgLBM/day). The following energy availability treatments were adhered to during the five day experimental period: 10 kcal/kgLBM/day, 20 kcal/kgLBM/day, or 30 kcal/kgLBM/day. All experimental trials were followed by a two month wash-out period before being repeated in cross-over fashion by all subjects. The authors reported that serum OC concentration decreased by 28% during the 10 kcal/kgLBM/d energy availability treatment ($p=0.0001$), 32% during the 20 kcal/kgLBM/d energy availability treatment ($p=0.002$), and by 11% during the 30 kcal/kgLBM/d energy availability treatment ($p=0.02$). Serum concentration of PICP decreased by 26% during the 10 kcal/kgLBM/day energy availability treatment ($p=0.001$), by 19% during the 20 kcal/LBM/day energy availability treatment ($p=0.01$), and by 12% during the 30 kcal/kgLBM/day energy availability treatment ($p=0.03$). Urinary NTX concentration increased 34% only during the 10 kcal/kgLBM/day energy availability treatment ($p<0.001$) (59).

Likewise, energy balance alters the response of bone formation and resorption markers to exercise. For example, Zanker and Swaine (141) demonstrated that three consecutive days of a 60-minute run at 75% of VO_2max suppressed PINP, a marker of bone formation, but only when participants were in negative energy balance. The male

runners (average age 25 years) on one occasion consumed a diet of 50% of estimated energy requirements. On a second occasion, runners consumed a diet to maintain energy balance. Following energy restriction, serum concentrations of PINP decreased by 15% ($p=0.008$). Concentrations of PINP did not change following a diet to maintain energy balance (141). Thus, the importance of adequate energy intake to match energy expenditure is an important factor in the maintenance of bone turnover. The results of these studies indicate that energy restriction results in a suppression of bone formation markers and an increase in bone resorption markers. This is in conflict with the results of the present study where a 10-hr fast did not induce changes in bone formation and bone resorption, nor did fasting affect the response of the bone turnover markers to exercise. The ten-hour fast likely did not induce an energy imbalance of sufficient proportion to result in changes in concentration of bone turnover markers.

Although we were primarily interested in the effects of fasting/feeding as they related to energy balance, it is possible that calcium content of the liquid meal replacement used in the fed trial might have altered the bone turnover markers and PTH response. A single oral dose calcium (1000 mg) increased plasma concentration of calcium, reduced plasma concentration of PTH, and suppressed markers of bone resorption in healthy men (58). Guillemant *et al.* (46) reported that a 1,000-mg oral dose of calcium attenuated the serum increase in CTX that occurred during and following cycling. Male triathletes (23-37 years in age) cycled for 60 minutes at 80% of VO_2max after consuming water enriched with 1,000-mg of calcium on one occasion and un-enriched water on another occasion. Following consumption of water alone, serum CTX concentration significantly increased 30 minutes into the cycling bout. CTX

concentration remained elevated by 45-50% two hours after cycling ended. Consumption of the calcium-enriched water attenuated the CTX response to cycling. BAP concentrations did not change with exercise, regardless of calcium consumption (46). Calcium consumption alone may suppress bone resorption markers during rest and attenuate the increase in bone resorption markers during exercise.

Response of Bone Turnover Markers to Resistance-Training or Plyometrics

We hypothesized that a single-bout of plyometrics will lead to greater increases in BAP concentration and greater decreases in TRAP5b compared with a single-bout of resistance-training. Forces that act on the skeleton include gravitational loading and muscle loading (63). Resistance-training has been reported to improve or maintain bone mass in women and men (68, 69, 85, 90, 92, 106, 131, 139). The predominant source of force applied to the skeleton during resistance-training is by muscle loading (63). Jump-training has also been reported to improve or maintain bone mass in women (9, 44, 55, 66, 119, 128, 138). In men, the combination of strength training and jump-training improves BMD (45). Gravitational loading and muscle loading act in concert to apply force to the skeleton during jump-training (63).

Athletes that participate in sports that involve high-load, high-impact exercise (i.e., volleyball and basketball) that also apply a great amount of gravitational force to the skeleton have greater BMD of the hip and spine that sports that induce large loads while not including a great amount of gravitational forces (i.e., swimming and cycling) (34, 82, 100). Thus, our hypotheses was based on that a single-bout of plyometrics will include both the application of gravitational forces and muscle forces to the skeleton and induce greater increases in bone formation and greater decreases in bone resorption. A single-

bout of resistance-training will only include muscle force applied to the skeleton. The results of the present study indicate that a single-bout of plyometrics may not be superior to a single-bout of resistance-training at disrupting bone turnover.

Hormonal Response to Feeding Following a Single-Bout of Resistance-Training or Plyometrics

We hypothesized that the expected increases in COR, PTH, and TEST following RT and PLY would be decreased by feeding. We hypothesize that resistance-training will induce greater concentrations of TEST and COR compared with plyometrics, but there will be no difference in PTH between exercise mode (i.e., PLY vs. RT). In the present study, no significant differences between fed and fasted were detected. PTH was suppressed by feeding compared to fasting at PRE only. COR concentration increased to a greater extent following RT compared with PLY, but no differences in PTH or TEST concentration were detected between exercise modes.

As mentioned previously, a single-bout of resistance training reportedly increases the rate of skeletal muscle protein turnover (14) and increases the serum concentrations of several important hormones (29, 73). Feeding prior to or immediately following a single-bout of resistance-training has been reported to alter the hormonal response after a single-bout of resistance-training (16, 23, 122).

Chandler *et al.* (23) fed male weight lifters either water, an isocaloric CHO only, PRO only, or a CHO/PRO liquid supplement following a single-bout of resistance-training. The authors reported that in response to a single-bout of resistance-training, TEST concentration was significantly increased one hour following exercise compared to baseline. The consumption of the CHO or CHO/PRO supplement partially attenuated the

TEST response one hour following exercise (23). Thyfault *et al.* (122) reported that in response to a single-bout of resistance-training, serum concentration of COR significantly increased compared to baseline concentration in resistance-trained men. Consumption of CHO resulted in significantly increased COR immediately after and 1.5-hours following exercise ($p<0.05$). No differences between the treatment and placebo were reported (122). It has been reported elsewhere that COR concentration increases following a single-bout of resistance-training, regardless of CHO feeding (72) or CHO/PRO feeding (137). The authors of these studies concluded that the anabolic environment can be modulated by the composition of the caloric supplement (16, 23, 122). A single-bout of resistance-training appears to increase anabolic hormone concentration; however, this may be of minimal importance because protein synthesis is increased regardless of the magnitude of the hormonal response (135).

Relationship Between Bone Turnover Markers and Hormones

We observed a significant positive correlation between AUC of TRAP5b and AUC of PTH. PTH is released in response to decreases in plasma calcium concentration. PTH induces calcium release from the skeleton by activating bone resorption to maintain plasma calcium concentration. Abnormal increases in endogenous PTH production, as occurs in certain diseases including hyperparathyroidism, results in a reduction of skeletal mass and increased bone resorption (12, 104). Communication between the osteoblasts and osteoclasts is disrupted by PTH leading to an uncoupling bone turnover in favor of resorption. Osteoblasts secrete receptor activator for nuclear factor-κB ligand (RANKL) which interacts with the osteoclast receptor RANK. Thus, osteoblasts appear to have an inhibitory influence on osteoclasts (18). This interaction is partially controlled by

osteoprotegerin (OPG), a soluble "decoy" receptor for RANKL. PTH inhibits the expression of OPG by osteoblasts and osteoclasts, thereby reducing the osteoblastic inhibitory influence on osteoclastogenesis (18). TRAP5b is a marker of osteoclast number and activity (50, 60) and a decrease in TRAP5b is representative of a decrease in the activity and number of osteoclasts. Thus, the significant positive correlation between TRAP5b and PTH represents the pro-resorptive effects of PTH.

UNEXPECTED RESULTS

We expected bone resorption to be correlated with COR and PTH and bone formation to be correlated with TEST. No relationships between COR and bone resorption, or between TEST and bone formation were observed in the present study. However, a relationship between PTH and bone resorption was observed. Bone turnover markers have been reported to predict changes in BMD (25, 94, 105) and predict fracture risk (15); however, the ability of these markers to predict changes in BMD and fracture risk is controversial at this point (86, 110). As mentioned previously, positive long term changes in BMD and changes in bone turnover markers may appear regardless of changes in hormone concentration (10, 45). The results of the present study agree that acute changes in bone turnover post-exercise may not be related to changes in circulating COR and TEST; however, it appears that reductions in PTH after exercise might be responsible, at least in part, for the acute reductions in bone resorption markers.

We expected TEST, COR, and PTH concentration to increase following a single-bout of resistance-training or plyometrics. As mentioned previously, increases in TEST and COR following resistance-training occur following some single-bout of resistance-training, but not all (30, 73). Because the exercise bouts were designed to maximally

load the hip and the spine and included rest periods of sufficient length to allow for skeletal recovery the expected hormonal response may not have been elicited. We expected feeding to attenuate the hormonal and bone turnover response but the design of the resistance-training bout may not have been of sufficient intensity to illicit a response to allow for feeding to attenuate the response.

LIMITATIONS

The present study had several limitations. Only six participants performed the resting control trial. This made comparisons of the exercise trials to CON difficult. A single-factor (time) repeated measures ANOVA was performed for each exercise trial (n=12). This analysis was repeated a second time including only the participants in CON (n=6). A 2x2 repeated measures ANOVA (time) was performed for the four exercise trials (n=12). A one-factor (trial) repeated measures ANOVA was performed for the four exercise trials and CON (n=6).

Subject retention was relatively poor during the study. A total of 24 participants provided written consent and began the study; however, only 12 participants completed the study. Two subjects performed the familiarization and the first exercise trial; however, dropped out following the first trial. The remaining ten subjects who withdrew did so either following familiarization or immediately after providing written consent.

Changes in plasma volume have been reported to influence biochemical measures of bone turnover and hormones following a single-bout of resistance-training. Following a single-bout of resistance-training the decrease in plasma volume is typically 10% or less (74-76). Changes in plasma volume have been reported to diminish statistically significant responses of TEST and other hormones following single-bouts of exercise

(99), while other report that the significance remains (121). In some reports, the changes in plasma volume were not significant and, thus, a correction for plasma volume was not undertaken (75). In the present study, changes in plasma volume following resistance-training or plyometrics were not calculated and may lead to erroneous interpretations of the results.

Individuals unfamiliar with resistance-training and sedentary individuals are unable to achieve true maximal lifts. Strength gains typically seen during the first several weeks of a resistance-training program are due to improvements in the neuromuscular system, as opposed to increases in muscle fiber cross-sectional area (6). Individuals that do not have experience resistance-training do not have the proper neuromuscular development to perform maximal lifts, nor do the individuals have the technique necessary to generate maximal force during complicated lifts such as the squat or dead lift. In the present study, subjects had not recently been participating in a resistance-training program. Because of this, the subjects would have been unable to achieve a true maximal lift after only one familiarization session. Thus, the percentages of one-repetition maximum used to design each individual resistance-training bout would have been of less intensity than actually prescribed. Had the prescribed intensities been adequately met by trained subjects, the hormonal or bone turnover response may have been different.

The probability that a statistical test will make a type II error is considered the power of the test. Type II error is also known as a false negative result and is represented by beta (β). Power is 1 - β and typically power is set to 0.80 to minimize the chance of making a type II error. In this present study, several variables lack a statistical power to

minimize the chances of a false negative result. For the one-factor (time) repeated measures ANOVA of each exercise trial separately (n=12), several variables lacked the statistical power necessary to meet the minimum threshold of 0.80 (for a summary see **Appendix C**). For the 2 x 2 repeated measures ANOVA comparing the four exercise trials (n=12), the observed power to detected a main effect for time of each variable (BAP, TRAP5b, COR, PTH, and TEST) was greater than 0.80. Unfortunately, the power to detect differences between PLY and RT and to detect differences between fed and fasted was less than 0.80. For the one-factor (time) repeated measures ANOVA of each exercise trial and CON trial separately (n=6), several variables lacked the statistical power to meet a power of 0.80. These results weaken the overall strength of the present study's analyses.

FUTURE DIRECTIONS

In the future, more research is needed to describe the response of bone turnover markers and hormones to plyometrics. The shift of fluid through the bone matrix that is detected by the osteocytes that occurs during weight bearing exercise and jumping may be of greater importance than applying bending strain to the skeleton. Jump training has proven quite effective at improving bone mass in women and experimental animals, but more research is needed to describe its effects in men over the long term. The relationship between changes in bone turnover markers and future changes in BMD needs to be described in greater detail and is controversial at this point. Exercise interventions should include baseline, intermediate, and final measures of bone turnover markers for correlation to changes in BMD.

The relationship between exercise, changes in hormone concentration, and changes in bone turnover markers needs to be described further. A positive relationship between the response of anabolic hormones to exercise and the bone formation response to exercise may not be needed to promote positive change in BMD. In addition, a relationship between response of bone resorption and the response of catabolic hormones to exercise may not need to exist to allow for positive changes in BMD.

The influence of energy balance or energy restriction on the response of bone turnover markers to a single-bout of resistance-training or plyometrics requires more study. The present study could have been changed by inducing a significant negative energy balance during the 24 to 48 hours prior to resistance-training or plyometrics rather than simply following overnight fast. In addition, the hormonal response to resistance-training or plyometrics following short and long term energy restriction or a lack of energy balance could be examined.

Fluid shear stress is hypothesized to activate bone cells and induce the bone turnover process. *In vitro* techniques would allow for the interaction between fluid shear stress, the bone cells (osteoblasts, osteoclasts, and osteocytes), and hormones to be explored directly. Each subject displayed different body mass, ability, and motivation during the exercise trials. The *in vitro* techniques would eliminate concerns such as subject compliance, effort, body mass, etc. *In vitro* techniques do not take into account multiple systems that may influence the bone cells, limiting their usefulness in certain circumstances. Bone cells are responsive to not only hormones, but to various growth factors, pharmacological treatments, and inflammatory markers. It would be impossible to design *in vitro* experiments to include all of the possible influences on bone cells. *In*

vitro techniques do not take into account the variability between individuals in stress levels during exercise modes. Differences in training status, for instance, induce individually different hormonal and bone turnover responses that cannot be mimicked *in vitro.* Finally, genetic influence plays a large role in the variability of bone mass between individuals and cannot be included in cell culture techniques.

The present study could have been improved in several areas. First, the control trial could have included all subjects, allowing for a greater power to detect differences between the exercise trials, the control trial, and comparison to changes that occur throughout the day in the absence of exercise. Second, more subjects could have been included in the analysis. This would have allowed for greater strength during the individual trial main effect of time analyses. This could have been accomplished by either reducing the number of trials to reduce drop out or to increase the number of subjects that completed the four exercise trials and control trial. A heterogeneous population could have been included. Differences in the response of hormones following a single-bout of resistance-training with age could also occur following plyometrics. Age could also influence the response of bone turnover markers to a single-bout of resistance-training or plyometrics. Finally, a greater number of bone turnover markers and hormones could have been measured. Different markers of bone turnover have been reported to respond differently to exercise and short or long term fasting. Serum concentrations of OC could have been measured to describe bone formation. OC is a specific marker of osteoblast function and is thought be involved in osteoid mineralization (110). OC has a short-half life and may be a better marker of short-term changes in bone formation (132). Measuring serum crosslinked collagen telopeptides,

CTX or NTX, could have been measured to describe bone resorption. TRAP5b is a marker of osteoclast function and activity; however, CTX and NTX are markers of the breakdown of collagen (110). These differences in measures could have added to the explanation of the alterations in bone remodeling following exercise. Additional hormones that could have been measured include growth hormone and IGF-I, both of which have been reported to have anabolic effects on the skeleton.

CONCLUSIONS

We showed that during the two hours following a single-bout of resistance training or plyometrics BAP did not significantly change; however, following a resting control trial BAP significantly decreased. Thus, resistance-training or plyometrics may maintain BAP concentration following exercise as opposed to the decrease observed in the morning hours in the absence of exercise. This suggests a favorable change in bone formation following resistance-training or plyometrics. TRAP significantly decreased during the two hours following a single-bout of resistance-training or plyometrics but did not change following a resting control trial. This suggests a favorable change in bone resorption following resistance-training or plyometrics. The decrease in PTH following plyometrics and resistance-training positively correlated with a decrease in TRAP5b, suggesting that PTH might mediate the exercise-induced changes in osteoclast activity.

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APPENDIX A
Diagrams & Tables

Table 1: Effective Load Stimulus and Ground Reaction Force Values

Sport/Activity	ELS	GRF
Swimming	0.07	0
Cycling	0.12	0
Stairmaster	0.31	1
Walking/Hiking	0.40	1
Golf	0.40	1
Resistance Training	0.51	1
Ice Hockey	1.68	1
Running/Jogging	4.88	2
Triathalon	4.88	2
Cross-Country	6.52	2
Tennis	7.84	3
Racquetball	12.20	3
Squash	12.20	3
Kung Fu	12.65	3
Wrestling	12.65	1
Basketball	12.70	3
Baseball/Softball	13.60	2
Soccer	13.60	3
Volleyball	31.37	3

Modified from Weeks *et al.* (2008)

Table 2: Subject Descriptive Statistics

Age and anthropometric measures	
Age	42.8 ± 4.2
Height (cm)	181.1 ± 1.3
Weight (kg)	79.1 ± 2.9
BMI (kg/m^2)	24.1 ± 0.73
Exercise measures	
Exercise (hrs/wk)	5.3 ± 0.84
Exercise (kcal/wk)	3977.9 ± 644.7
Site specific bone measurements by DXA	
Whole Body BMC (g)	2961.5 ± 132.7
Whole Body (BMD (g/cm^2)	1.26 ± 0.03
Hip BMC (g)	46.3 ± 2.5
Hip BMD (g/cm^2)	1.08 ± 0.05
Hip T-score	0.3 ± 0.3
Ward's Triangle BMC (g)	0.91 ± 0.10
Ward's Triangle BMD (g/cm^2)	0.79 ± 0.09
Ward's T-score	0.1 ± 0.6
Lumbar Spine BMC (g)	84.6 ± 4.3
Lumbar Spine BMD (g/cm^2)	1.12 ± 0.04
Lumbar Spine T-score	0.2 ± 0.4
Bone loading history	
Total Annualized GRF Score	551 ± 129
Total Annualized ELS Score	1660 ± 409
Macronutrient & selected micronutrients	
Energy intake (kcal/day)	2755 ± 162
Protein intake (g/day)	107 ± 9
Carbohydrate intake (g/day)	357 ± 27
Total fat intake (g/day)	96 ± 11
Calcium intake (mg/day)	1235 ± 219
Vitamin D intake (IU/day)	241 ± 80
Vitamin D intake (μg/day)	6 ± 2
Strength assessment measures	
1-RM Squat (kg)	94.6 ± 8.8
1-RM Dead lift (kg)	86.7 ± 9.8
1-RM Bent over row (kg)	66.5 ± 4.3
1-RM Military press (kg)	45.5 ± 4.1
10-RM Lunge (kg)	42.0 ± 5.6
10-RM Calf raise (kg)	91.3 ± 6.4

Data are means ± SEM, n=12

Table 3: Changes in bone turnover markers and hormones over time for each exercise trial (n=12).

Bone-Specific Alkaline Phosphatase (BAP) U/L	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	17.1 ± 2.7	14.0 ± 2.8	16.0 ± 2.6	14.4 ± 2.3
POST	17.2 ± 2.6	13.9 ± 2.9	16.1 ± 2.7	14.4 ± 2.5
15MIN	15.9 ± 2.6	13.3 ± 2.5	14.5 ± 2.6	13.5 ± 2.2
30MIN	16.0 ± 2.4	12.9 ± 2.7	14.5 ± 2.4	13.8 ± 2.2
60MIN	14.1 ± 2.3	13.2 ± 2.9	14.4 ± 2.4	13.0 ± 2.3
120MIN	17.7 ± 2.3	13.0 ± 2.7	14.9 ± 2.6	13.4 ± 2.6
24HR	15.3 ± 1.9	13.8 ± 2.4	16.1 ± 2.6	13.7 ± 2.6
Main effect for time	*p=0.121*	*p=0.464*	*p=0.213*	*p=0.546*
Tartrate-Resistant Acid Phosphatase, 5b (TRAP5b) U/L	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	2.7 ± 0.4	2.9 ± 0.5	2.8 ± 0.5	2.8 ± 0.6
POST	2.7 ± 0.4	2.8 ± 0.5	2.7 ± 0.5	2.7 ± 0.5
15MIN	2.5 ± 0.4*†	2.6 ± 0.4*†	2.6 ± 0.4	2.6 ± 0.5*†
30MIN	2.6 ± 0.4	2.6 ± 0.4*†	2.6 ± 0.5*†	2.5 ± 0.4*
60MIN	2.7 ± 0.4	2.7 ± 0.5	2.7 ± 0.5	2.5 ± 0.4
120MIN	2.7 ± 0.4	2.8 ± 0.5	2.8 ± 0.6	2.7 ± 0.5
24HR	2.7 ± 0.5	2.8 ± 0.4	3.0 ± 0.6	2.8 ± 0.5†
Main effect for time	*p=0.038*	*p=0.014*	*p=0.044*	*p=0.001*
Cortisol (COR) µg/dL	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	17.9 ± 1.4†	15.5 ± 0.9	17.7 ± 1.1	14.3 ± 1.0
POST	13.2 ± 0.8*	13.3 ± 1.0	17.3 ± 1.3	15.0 ± 1.4
15MIN	11.6 ± 0.8*†	12.3 ± 0.9*†	15.2 ± 1.1†	13.1 ± 1.2†
30MIN	10.1 ± 0.8*†	10.2 ± 1.1*	14.5 ± 1.4†	11.8 ± 1.1†
60MIN	9.7 ± 0.8*†	9.5 ± 0.9*†	10.9 ± 0.8*†	11.4 ± 1.5*†
120MIN	9.7 ± 0.9*†	10.9 ± 0.9*†	9.6 ± 0.7*†	10.7 ± 1.1*†
24HR	16.9 ± 1.1†	18.4 ± 1.3*†	17.3 ± 1.6	18.1 ± 1.4*

Main effect for time	*p<0.001*	*p<0.001*	*p<0.001*	*p<0.001*
Intact Parathyroid Hormone (PTH) pg/dL	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	64.7 ± 11.1	41.7 ± 6.6	56.6 ± 10.7	50.7 ± 11.8
POST	64.5 ± 14.9	58.4 ± 14.0	56.1 ± 14.2	46.6 ± 12.3
15MIN	50.7 ± 10.6*†	43.0 ± 9.8†	38.8 ± 9.7†	34.2 ± 9.3*†
30min	43.5 ± 9.8*†	34.2 ± 8.1†	34.4 ± 9.0†	30.7 ± 8.3*†
60MIN	47.0 ± 9.7*†	30.6 ± 3.6*†	36.6 ± 8.7†	33.1 ± 7.4*†
120MIN	41.9 ± 8.4*†	40.9 ± 9.5†	39.5 ± 8.2†	37.5 ± 9.9*†
24HR	57.9 ± 9.0	57.4 ± 9.5*	67.1 ± 11.5	59.8 ± 11.1†
Main effect for time	*p=0.002*	*p<0.001*	*p=0.007*	*p<0.001*
Testosterone (TEST) ng/dL	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	917 ± 67	844 ± 81	984 ± 189	756 ± 71
POST	862 ± 63	924 ± 103	950 ± 163	867 ± 94
15MIN	805 ± 63*	960 ± 144	907 ± 188	824 ± 93
30MIN	792 ± 60	767 ± 97†	917 ± 149	784 ± 83
60MIN	803 ± 84	777 ± 93	818 ± 146	729 ± 80†
120MIN	710 ± 66†	834 ± 96	999 ± 219	849 ± 126
24HR	965 ± 77	929 ± 89*	845 ± 98	1010 ± 162*
Main effect for time	*p=0.004*	*p=0.005*	*p=0.089*	*p=0.039*

Data are means ± SEE. Significant main effect for time was followed up with post hoc pairwise comparisons (LSD). Only differences from PRE and POST are shown (See Tables 1-15, Appendix C for a complete pairwise comparison).

* denotes a significant difference from PRE

† denotes a significant difference from POST

Table 4: Comparisons between exercise and controls trials at each time point (n=6).

Bone-Specific Alkaline Phosphatase (BAP) U/L	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	19.1 ± 1.7†	18.4 ± 2.3	11.7 ± 2.8	15.1 ± 2.8	12.2 ± 2.3
POST	18.1 ± 1.4*	18.5 ± 1.7	10.8 ± 2.9	15.4 ± 2.8	11.9 ± 2.5
15MIN	16.9 ± 1.6*	16.9 ± 1.7	10.8 ± 2.7	13.9 ± 2.6	10.5 ± 2.4
30MIN	16.4 ± 1.4*	16.7 ± 1.8	10.9 ± 2.9	14.2 ± 2.3	12.2 ± 2.4
60MIN	16.8 ± 1.3*†	13.7 ± 2.1	10.3 ± 2.7	14.0 ± 2.4	10.9 ± 1.8
120MIN	17.9 ± 1.2	18.9 ± 2.2	10.9 ± 2.5	13.6 ± 2.5	11.4 ± 2.5
24HR	17.1 ± 1.5*†	15.0 ± 1.5	12.0 ± 2.4	16.6 ± 1.6	11.8 ± 2.0
Main effect for time	*p=0.002*	*p=0.128*	*p=0.134*	*p=0.298*	*p=0.480*
Tartrate-Resistant Acid Phosphatase, 5b (TRAP5b) U/L	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	2.7 ± 0.4	2.2 ± 0.2	2.4 ± 0.3	2.3 ± 0.4	2.3 ± 0.3
POST	2.5 ± 0.4	2.2 ± 0.3	2.4 ± 0.3	2.2 ± 0.4	2.3 ± 0.3
15MIN	2.3 ± 0.4	2.1 ± 0.3	2.2 ± 0.3	2.2 ± 0.3	2.1 ± 0.3†
30MIN	2.3 ± 0.3	2.2 ± 0.3	2.2 ± 0.3	2.1 ± 0.4	2.2 ± 0.3*
60MIN	2.3 ± 0.4	2.2 ± 0.3	2.3 ± 0.3	2.2 ± 0.4	2.3 ± 0.3
120MIN	2.4 ± 0.3	2.2 ± 0.3	2.3 ± 0.3	2.5 ± 0.5	2.4 ± 0.3†
24HR	2.3 ± 0.3	2.1 ± 0.3	2.3 ± 0.3	2.3 ± 0.5	2.4 ± 0.3
Main effect for time	*p=0.07*	*p=0.372*	*p=0.430*	*p=0.192*	*p=0.001*
Cortisol (COR) µg/dL	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	15.8 ± 1.6†	16.8 ± 2.1	15.0 ± 1.8	18.1 ± 2.1	14.4 ± 2.0
POST	11.8 ± 1.4*	13.5 ± 1.1	14.4 ± 1.3	18.0 ± 2.6	17.6 ± 1.6
15MIN	11.1 ± 1.4*†	11.5 ± 0.8*†	13.6 ± 1.3	15.8 ± 1.9	15.4 ± 1.8
30MIN	10.2 ± 1.4*	10.3 ± 0.9*†	10.9 ± 2.1	15.5 ± 2.6†	13.4 ± 1.7
60MIN	9.4 ± 1.2*†	10.2 ± 1.1*†	10.8 ± 1.1*†	11.2 ± 1.5†	14.5 ± 2.1
120MIN	11.3 ± 2.9	10.6 ± 1.1*	11.2 ± 1.6	10.8 ± 0.9†	11.9 ± 2.1
24HR	16.4 ± 2.0†	16.9 ± 2.1	19.5 ± 1.9*†	18.5 ± 3.0	18.4 ± 2.3

Main effect for time	*p<0.001*	*p<0.001*	*p=0.005*	*p=0.021*	*p=0.069*
Intact Parathyroid Hormone (PTH) pg/dL	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	45.3 ± 4.9	59.4 ± 7.2†	29.6 ± 6.8	46.6 ± 12.4	33.2 ± 10.3
POST	40.2 ± 7.4	49.1 ± 7.0*	43.8 ± 10.7	44.4 ± 16.4	31.1 ± 9.5
15MIN	35.7 ± 2.1	43.8 ± 8.4*	31.9 ± 6.1†	29.5 ± 8.1	21.0 ± 5.7†
30min	40.1 ± 7.1	35.1 ± 9.5*†	24.3 ± 4.6†	24.7 ± 5.5	19.7 ± 5.0*†
60MIN	39.5 ± 6.3	48.2 ± 13.3	24.3 ± 3.6†	29.8 ± 7.2	24.2 ± 5.1
120MIN	38.2 ± 6.9	35.6 ± 9.5*†	29.8 ± 6.8	29.8 ± 5.5	25.2 ± 8.6
24HR	46.6 ± 7.0	55.1 ± 11.9	49.4 ± 9.3*	58.4 ± 13.2	52.7 ± 12.5*†
Main effect for time	*p=0.125*	*p=0.007*	*p<0.001*	*p=0.198*	*p<0.001*
Testosterone (TEST) ng/dL	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	883 ± 97	874 ± 97	987 ± 127	1276 ± 326	887 ± 114
POST	918 ± 111	972 ± 87	910 ± 150	1129 ± 302	1099 ± 122
15MIN	917 ± 130	834 ± 83	1262 ± 229	1193 ± 342	1021 ± 132
30MIN	866 ± 120	856 ± 70	992 ± 159	1205 ± 236	940 ± 124
60MIN	889 ± 104	940 ± 130	997 ± 117	1006 ± 263	919 ± 95
120MIN	882 ± 153	881 ± 61	1035 ± 141	1375 ± 330	1121 ± 187
24HR	1165 ± 380	1094 ± 98	1043 ± 168	965 ± 138	1254 ± 292
Main effect for time	*p=0.974*	*p=0.145*	*p=0.057*	*p=0.494*	*p=0.309*

Data are means ± SEE. Significant main effect for time was followed up with post hoc pairwise comparisons (LSD). Only differences from PRE and POST are shown (See Tables 16-31, Appendix C for a complete pairwise comparison).

* denotes a significant difference from PRE

† denotes a significant difference from POST

Table 5: Comparisons between exercise and control trials at individual time points (n=6).

Bone-Specific Alkaline Phosphatase (BAP) U/L	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	19.1 ± 1.7	18.4 ± 2.3	11.7 ± 2.8	15.1 ± 2.8	12.2 ± 2.3
POST	18.1 ± 1.4	18.5 ± 1.7	10.8 ± 2.9	15.4 ± 2.8	11.9 ± 2.5
15MIN	16.9 ± 1.6	16.9 ± 1.7	10.8 ± 2.7	13.9 ± 2.6	10.5 ± 2.4
30MIN	16.4 ± 1.4	16.7 ± 1.8	10.9 ± 2.9	14.2 ± 2.3	12.2 ± 2.4
60MIN	16.8 ± 1.3	13.7 ± 2.1	10.3 ± 2.7	14.0 ± 2.4	10.9 ± 1.8
120MIN	17.9 ± 1.2	18.9 ± 2.2	10.9 ± 2.5	13.6 ± 2.5	11.4 ± 2.5
24HR	17.1 ± 1.5	15.0 ± 1.5	12.0 ± 2.4	16.6 ± 1.6	11.8 ± 2.0
Tartrate-Resistant Acid Phosphatase, 5b (TRAP5b) U/L	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	2.7 ± 0.4	2.2 ± 0.2	2.4 ± 0.3	2.3 ± 0.4	2.3 ± 0.3
POST	2.5 ± 0.4	2.2 ± 0.3	2.4 ± 0.3	2.2 ± 0.4	2.3 ± 0.3
15MIN	2.3 ± 0.4	2.1 ± 0.3	2.2 ± 0.3	2.2 ± 0.3	2.1 ± 0.3
30MIN	2.3 ± 0.3	2.2 ± 0.3	2.2 ± 0.3	2.1 ± 0.4	2.2 ± 0.3
60MIN	2.3 ± 0.4	2.2 ± 0.3	2.3 ± 0.3	2.2 ± 0.4	2.3 ± 0.3
120MIN	2.4 ± 0.3	2.2 ± 0.3	2.3 ± 0.3	2.5 ± 0.5	2.4 ± 0.3
24HR	2.3 ± 0.3	2.1 ± 0.3	2.3 ± 0.3	2.3 ± 0.5	2.4 ± 0.3
Cortisol (COR) µg/dL	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	15.8 ± 1.6	16.8 ± 2.1	15.0 ± 1.8	18.1 ± 2.1	14.4 ± 2.0
POST	11.8 ± 1.4	13.5 ± 1.1	14.4 ± 1.3	18.0 ± 2.6	17.6 ± 1.6
15MIN	11.1 ± 1.4*	11.5 ± 0.8*	13.6 ± 1.3	15.8 ± 1.9	15.4 ± 1.8
30MIN	10.2 ± 1.4	10.3 ± 0.9	10.9 ± 2.1	15.5 ± 2.6	13.4 ± 1.7
60MIN	9.4 ± 1.2*	10.2 ± 1.1	10.8 ± 1.1*	11.2 ± 1.5	14.5 ± 2.1
120MIN	11.3 ± 2.9	10.6 ± 1.1	11.2 ± 1.6	10.8 ± 0.9	11.9 ± 2.1
24HR	16.4 ± 2.0	16.9 ± 2.1	19.5 ± 1.9	18.5 ± 3.0	18.4 ± 2.3
Intact Parathyroid Hormone (PTH) pg/dL	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**

PRE	45.3 ± 4.9†	59.4 ± 7.2	29.6 ± 6.8†¶	46.6 ± 12.4	33.2 ± 10.3†¶
POST	40.2 ± 7.4	49.1 ± 7.0	43.8 ± 10.7	44.4 ± 16.4	31.1 ± 9.5
15MIN	35.7 ± 2.1	43.8 ± 8.4	31.9 ± 6.1	29.5 ± 8.1*$	21.0 ± 5.7*$
30min	40.1 ± 7.1	35.1 ± 9.5	24.3 ± 4.6$	24.7 ± 5.5$	19.7 ± 5.0*$
60MIN	39.5 ± 6.3	48.2 ± 13.3	24.3 ± 3.6$	29.8 ± 7.2*	24.2 ± 5.1$
120MIN	38.2 ± 6.9	35.6 ± 9.5	29.8 ± 6.8$	29.8 ± 5.5$	25.2 ± 8.6$†
24HR	46.6 ± 7.0	55.1 ± 11.9	49.4 ± 9.3	58.4 ± 13.2	52.7 ± 12.5
Testosterone (TEST) ng/dL	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
PRE	883 ± 97	874 ± 97	987 ± 127	1276 ± 326	887 ± 114
POST	918 ± 111	972 ± 87	910 ± 150	1129 ± 302	1099 ± 122
15MIN	917 ± 130	834 ± 83	1262 ± 229	1193 ± 342	1021 ± 132
30MIN	866 ± 120	856 ± 70	992 ± 159	1205 ± 236	940 ± 124
60MIN	889 ± 104	940 ± 130	997 ± 117	1006 ± 263	919 ± 95
120MIN	882 ± 153	881 ± 61	1035 ± 141	1375 ± 330	1121 ± 187
24HR	1165 ± 380	1094 ± 98	1043 ± 168	965 ± 138	1254 ± 292

Data are means ± SEE. Results of one-factor (trial) repeated measures ANOVA. Significant differences between trials were followed up by post hoc pairwise comparisons (LSD) of the trials at individual time points (n=12).
* denotes significantly different than RT+ ($p<0.05$)
† denotes significantly different than PLY- ($p<0.05$)
¶ denotes significantly different than RT- ($p<0.05$)
$ denotes significantly different than CON ($p<0.05$)

Table 6: Area under the curve (AUC) comparison between exercise mode and feeding (n=12).

Area Under the Curve	PLY FASTED	PLY FED	RT FASTED	RT FED
Bone-Specific Alkaline Phosphatase (BAP) (U min/L)	-175.9 ± 86.1	-209.0 ± 85.1	-86.7 ± 65.1	-114.9 ± 79.9
Tartrate-Resistant Acid Phosphatase 5b (TRAP5b) (U min/L)	-5.1 ± 4.2	-4.0 ± 6.7	-4.8 ± 9.8	-10.8 ± 11.5
Cortisol (COR) (μg min/dl)	-359.1 ± 71.7	-608.0 ± 73.5	-318.9 ± 67.1	-393.4 ± 101.4
Parathyroid Hormone (PTH) (pg min/dl)	-2162.9 ± 934.3	-2069.9 ± 840.2	-2556.6 ± 872.3	-1429.2 ± 484.6
Testosterone (TEST) (ng min/dl)	-9717.8 ± 2642.4	-1352.8 8225.1	-9529.1 ± 4776.0	11549.5 ± 5072.2

Data are means ± SEE. The AUC of COR was significantly greater following RT than following PLY (main effect in the 2x2 ANOVA).

Table 7: Area under the curve (AUC) comparisons between exercise and control trials (n=6).

Area Under the Curve	CONTROL	PLY FASTED	PLY FED	RT FASTED	RT FED
Bone-Specific Alkaline Phosphatase (BAP) (U min/L)	-124.1 ± 63.0	-262.4 ± 116.0	-3.6 ± 41.3	-157.1 ± 77.0	-58.9 ± 36.5
Tartrate-Resistant Acid Phosphatase 5b (TRAP5b) (U min/L)	-24.2 ± 7.3	2.0 ± 3.6	6.5 ± 11.7	7.1 ± 6.8	0.5 ± 6.3
Cortisol (COR) (µg min/dl)	-181.9 ± 75.4	-351.7 ± 110.7	-337.0 ± 115.1	-619.9 ± 128.4	-454.7 ± 194.3
Parathyroid Hormone (PTH) (pg min/dl)	-145.2 ± 354.0	-879.4 ± 756.3	-1903.2 ± 794.4	-1743.5 ± 1292.1	-895.0 ± 436.2
Testosterone (TEST) (ng min/dl)	-3664.9 ± 4126.9	-8817.6 ± 4802.9	-8194.2 ± 5841.6	4580.6 ± 15068.5	-16653.8 ± 8640.8

Data are means ± SEE. No significant differences among trials were detected by one-factor (trial) ANOVA.

Table 8: Pearson correlation coefficients for area under the curve analysis (n=12).

	BAP AUC	TRAP AUC	COR AUC	PTH AUC	TEST AUC
BAP AUC	1	0.145	-0.14	0.11	-0.073
TRAP AUC	0.145	1	-0.01	0.452*	0.143
COR AUC	-0.14	-0.01	1	0.209	0.159
PTH AUC	0.11	0.452*	0.209	1	0.308*
TEST AUC	-0.073	0.143	0.159	0.308*	1

Values are Pearson Correlation Coefficients (r)
* denotes a significant correlation ($p<0.05$)

Table 9: Percent Changes at 30 MIN and 60 MIN Compared with PRE (n=12).

Bone-Specific Alkaline Phosphatase (BAP)	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	-3.7% ± 4.6%	-8.7% ± 7.1%	-8.1% ± 6.6%	-1.9% ± 7.2%
60MIN	-11.7% ± 8.1%	-7.2% ± 6.5%	-11.3% ± 5.7%	-10.0% ± 5.6%
Tartrate-Resistant Acid Phosphatase, 5b (TRAP5b)	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	-2.0% ± 3.0%	-6.7% ± 2.7%	-8.2% ± 2.1%	-8.2% ± 2.3%
60MIN	-1.9% ± 2.9%	-4.6% ± 2.6%	-3.4% ± 2.9%	-6.4% ± 4.1%
Cortisol (COR)	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	-41.5% ± 5.1%	-34.9% ± 6.8%	-8.8% ± 18.0%	-15.2% ± 7.6%
60MIN	-43.9% ± 4.9%	-36.9% ± 6.0%	-34.6% ± 8.9%	-21.2% ± 9.5%
Intact Parathyroid Hormone (PTH)	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	-30.9% ± 8.8%	-15.6% ± 11.7%	-5.6% ± 40.5%	-37.9% ± 6.2%
60MIN	-25.5% ± 11.2%	-16.7% ± 10.2%	28.0% ± 73.7%	-28.0% ± 6.9%
Testosterone (TEST)	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	-11.2% ± 6.0%	-9.7% ± 6.8%	-2.5% ± 6.2%	-7.0% ± 9.4%
60MIN	-9.8% ± 9.5%	-7.8% ± 6.6%	-13.3% ± 5.9%	-2.7% ± 7.3%

Data are means ± SEE. No statistical analyses were performed on the percent change compared to PRE.

Table 10: Percent Changes at 30 MIN and 60 MIN Compared with PRE (n=6).

Bone-Specific Alkaline Phosphatase (BAP)	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	-13.1% ± 4.4%	-7.8% ± 3.1%	-10.2% ± 13.1%	-1.6% ± 5.6%	1.3% ± 13.1%
60MIN	-11.4% ± 3.3%	-19.4% ± 12.0%	-15.9% ± 8.8%	-6.1% ± 3.8%	-9.3% ± 6.6%
Tartrate-Resistant Acid Phosphatase, 5b (TRAP5b)	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	-12.6% ± 3.6%	-2.3% ± 5.4%	-7.7% ± 4.9%	-8.0% ± 2.9%	-6.4% ± 1.4%
60MIN	-11.3% ± 3.3%	-0.2% ± 3.6%	-3.3% ± 4.5%	-1.9% ± 4.6%	0.8% ± 2.5%
Cortisol (COR)	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	-34.4% ± 6.7%	-35.8% ± 7.4%	-30.2% ± 10.2%	3.7% ± 36.6%	-2.8% ± 1.3%
60MIN	-39.4% ± 6.4%	-37.0% ± 6.5%	-26.8% ± 5.1%	-30.0% ± 18.2%	1.3% ± 12.3%
Intact Parathyroid Hormone (PTH)	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	-13.1% ± 9.8%	-43.7% ± 9.5%	-7.5% ± 20.1%	27.4% ± 81.7%	-33.9% ± 10.0%
60MIN	-14.3% ± 7.0%	-20.7% ± 21.0%	-5.1% ± 18.5%	94.7% ± 148.4%	-17.7% ± 10.0%
Testosterone (TEST)	**CONTROL**	**PLY FASTED**	**PLY FED**	**RT FASTED**	**RT FED**
30MIN	0.2% ± 11.3%	0.7% ± 5.8%	-7.3% ± 11.5%	1.3% ± 10.5%	11.4% ± 13.9%
60MIN	2.4% ± 9.1%	9.3% ± 13.3%	3.1% ± 7.8%	-21.1% ± 7.0%	7.5% ± 8.5%

Data are means ± SEE. No statistical analyses were performed on the percent change compared to PRE.

Figure 1: Study Flow Diagram

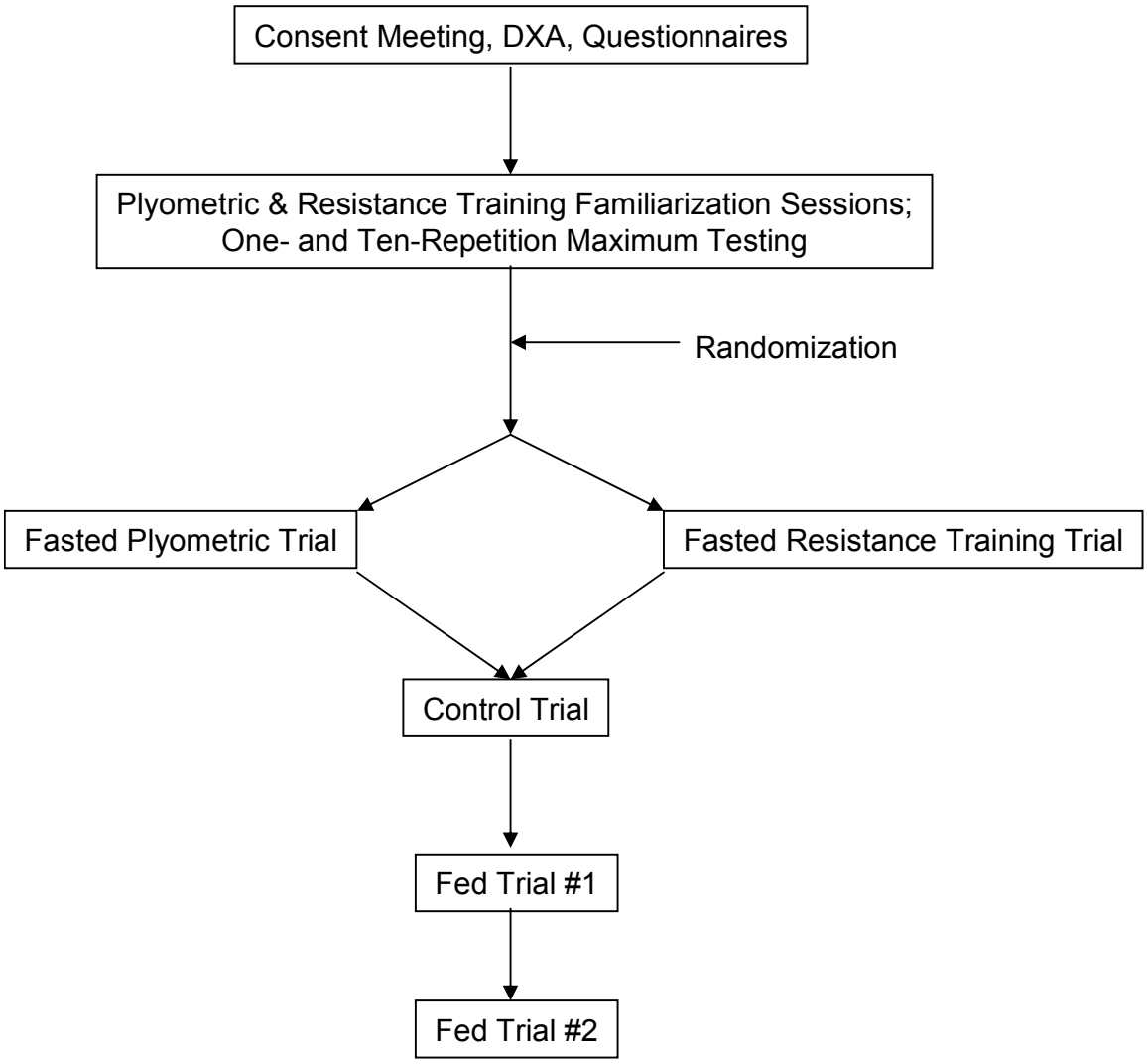
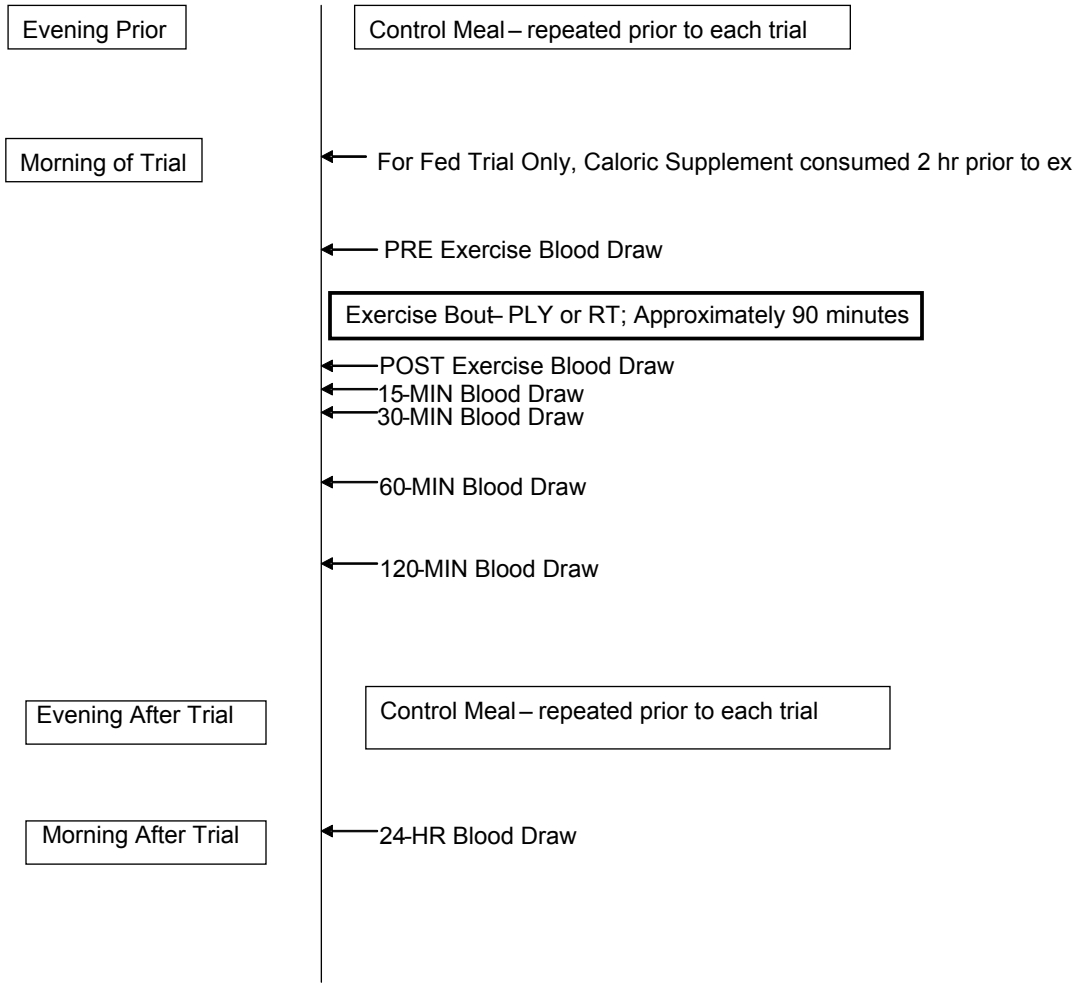


Figure 2: Blood Draw Timing



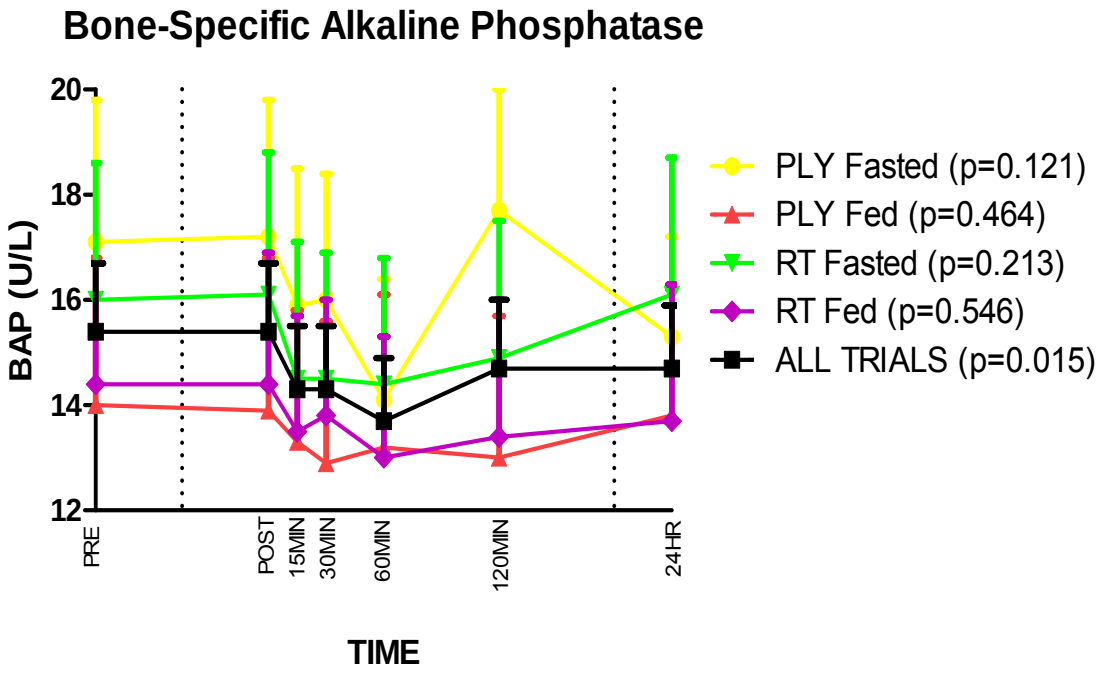


Figure 3: Bone-Specific Alkaline Phosphatase (BAP) response to different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw for the exercise trials: PLY-, RT-, PLY+, and RT+. "All trials" is the mean for all exercise trials combined; there was a significant main effect for time in the 2 x 2 x 7 repeated measures ANOVA. Data are means ± SEE. n=12

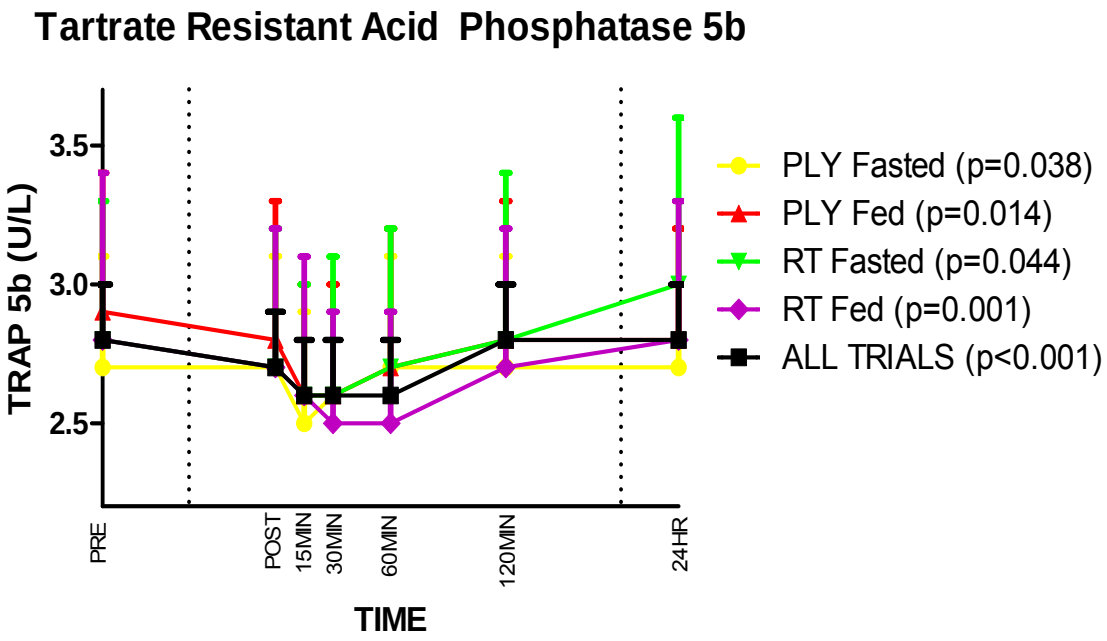


Figure 4: Tartrate-Resistant Acid Phsophatase, isoform 5b (TRAP5b), response to different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw for the exercise trials: PLY-, RT-, PLY+, and RT+ "All trials" is the mean for all exercise trials combined; there was a signficiant main effect for time in the 2 x 2 x 7 repeated measures ANOVA. Data are means ± SEE. n=12

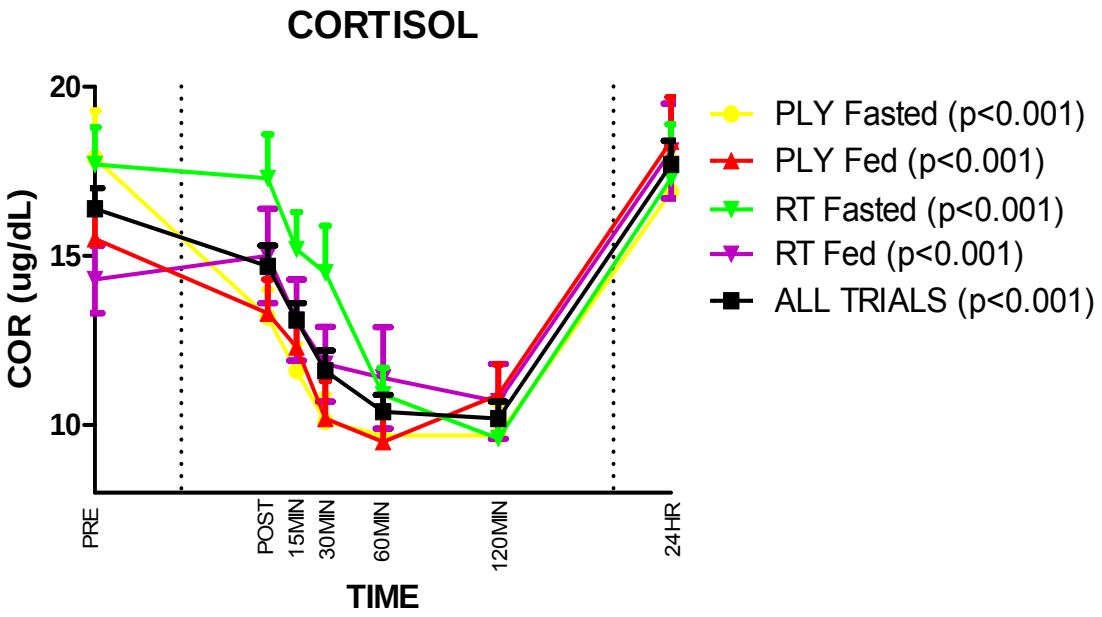


Figure 5: Cortisol (COR) response to different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw for the exercise trials: PLY-, RT-, PLY+, and RT+. "All trials" is the mean for all exercise trials combined; there was a significant main effect for time in the 2 x 2 x 7repeated measures ANOVA. Data are means ± SEE. n=12

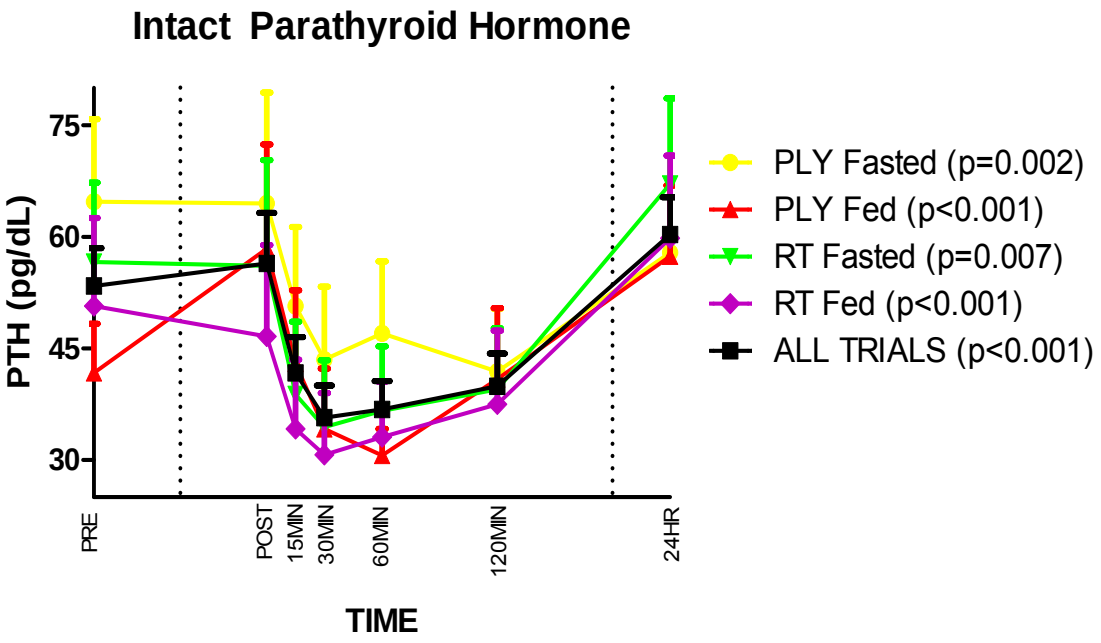


Figure 6: Intact Parathyroid Hormone (PTH) response to different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw for the exercise trials: PLY-, RT-, PLY+, and RT+. "All trials" is the mean for all exercise trials combined; there was a significant main effect for time in the 2 x 2 x 7 repeated measures ANOVA. Data are means ± SEE. n=12

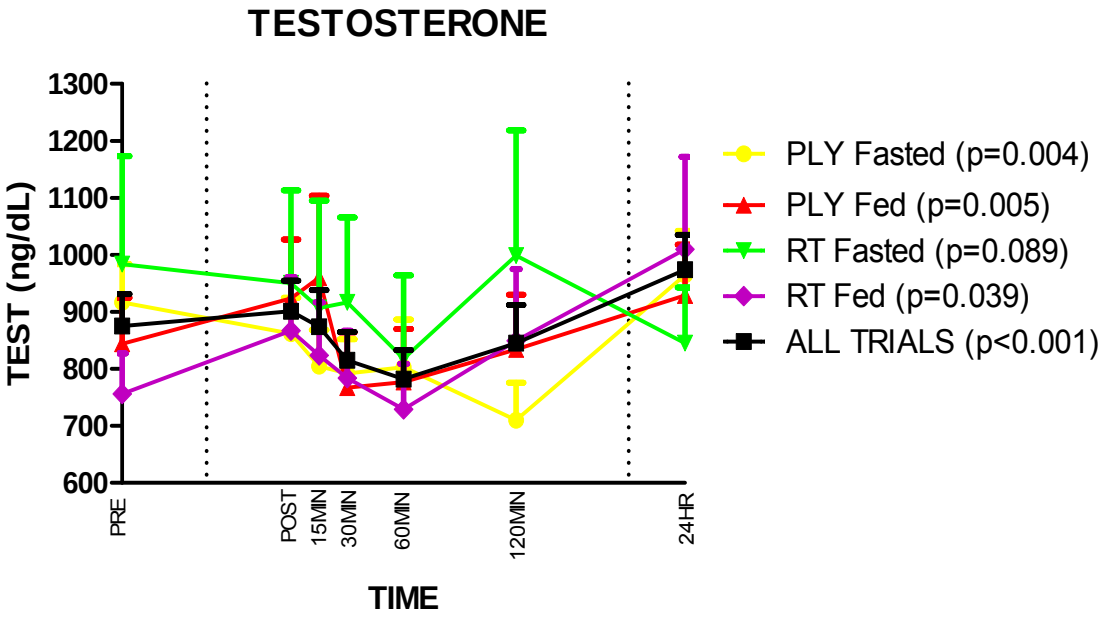


Figure 7: Testosterone (TEST) response to different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw for the exercise trials: PLY-, RT-, PLY+, and RT+. "All trials" is the mean for all exercise trials combined; there was a signficiant main effect for time in the 2 x 2 x 7 repeated measures ANOVA. Data are means ± SEE. n=12

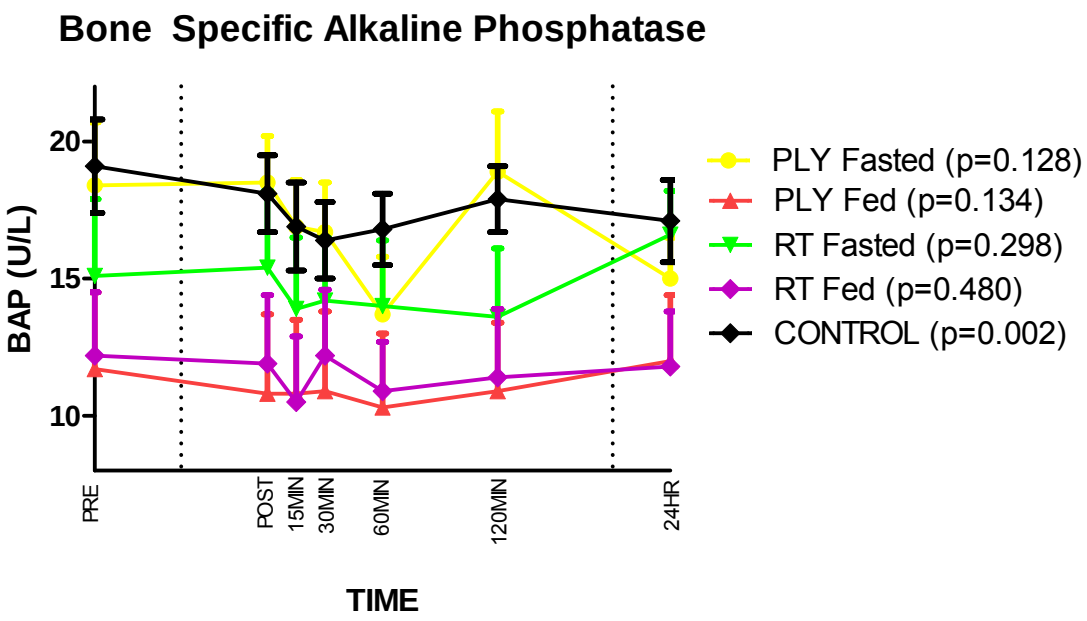


Figure 8: Bone-Specific Alkaline Phosphatase (BAP) response to CON and different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw. Data are means ± SEE, n=6

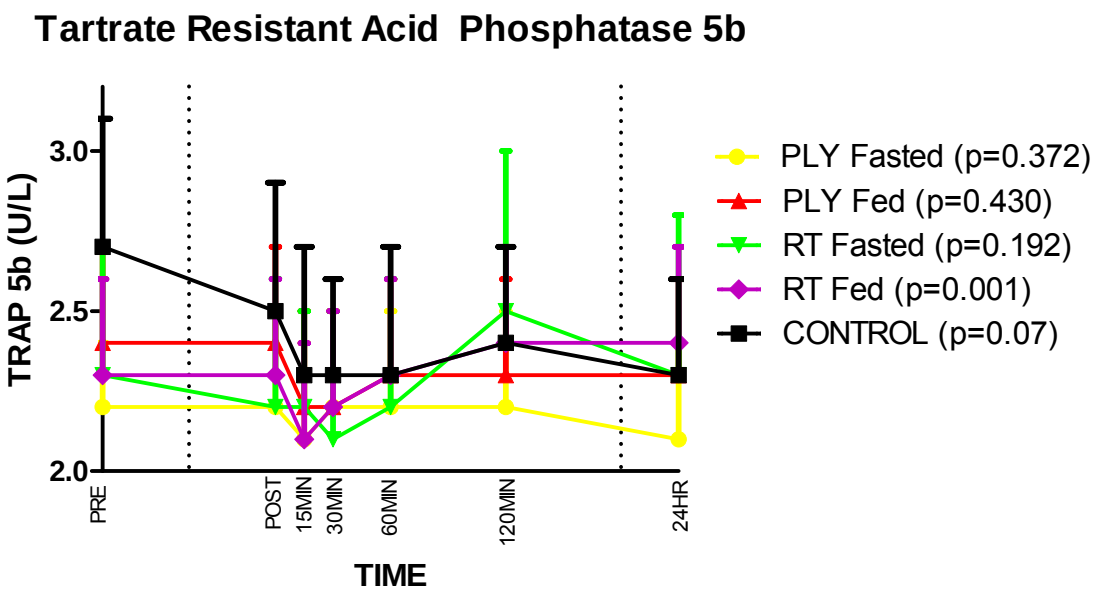


Figure 9: Tartrate-Resistant Acid Phosphatase, isoform 5b (TRAP5b) response to CON and different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw. Data are means ± SEE, n=6

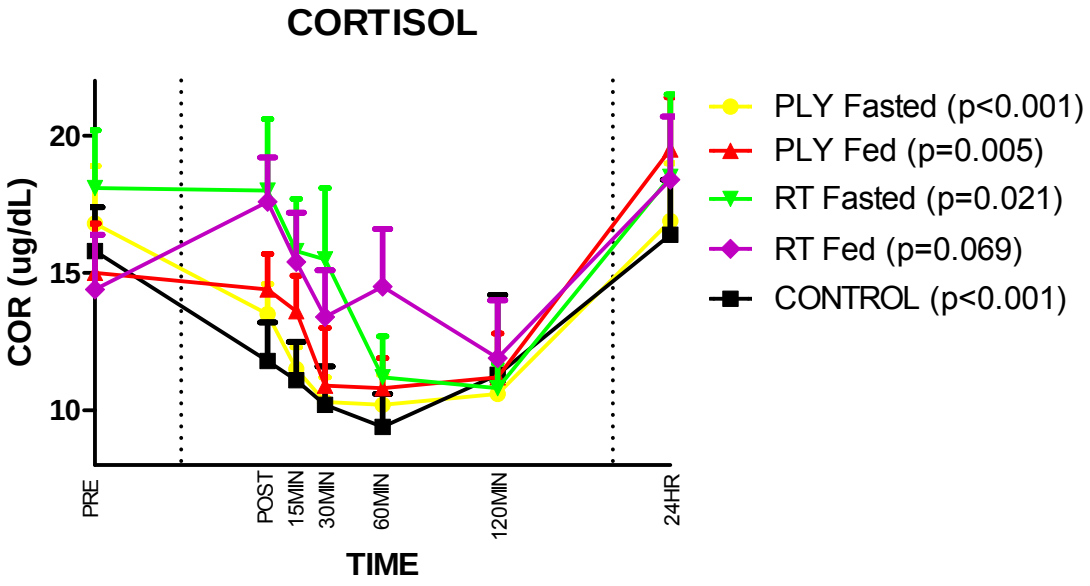


Figure 10: Cortisol (COR) response to CON and different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw. Data are means ± SEE, n=6

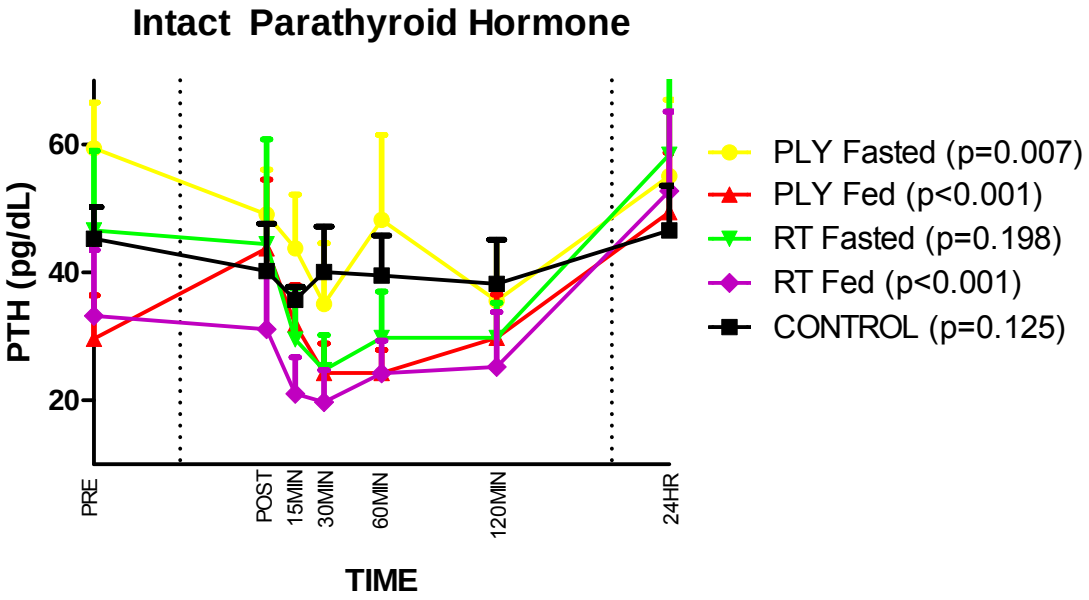


Figure 11: Intact Parathyroid Hormone (PTH) response to CON and different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw. Data are means ± SEE, n=6

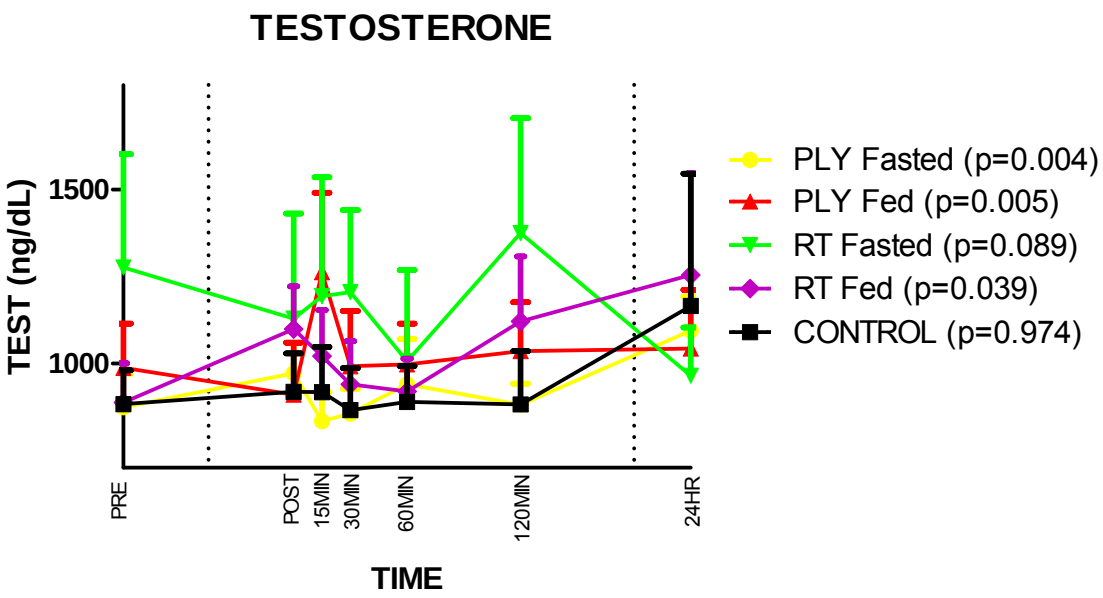


Figure 12: Testosterone (TEST) response to CON and different trials prior to exercise (PRE), immediately following exercise (POST), 15-min, 30-min, 60-min, 120-min, and 24-hr following PRE blood draw. Data are means ± SEE, n=6.

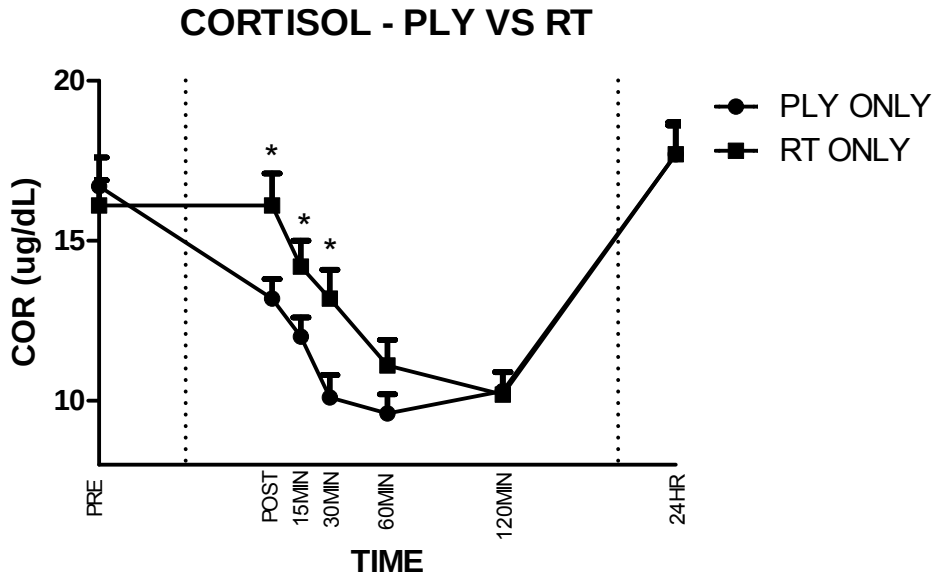


Figure 13: Different COR response to plyometrics or resistance-training. Results of 2 x 2 ANOVA with individual time point differences determined by an independent t-test. Data are means ± SEE, n=12
* denotes significant difference between trials at individual time points ($p<0.05$).

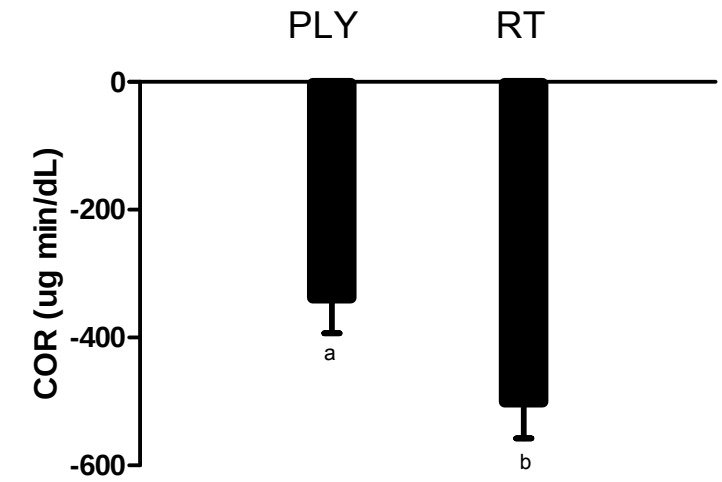


Figure 14: Exercise mode main effect on COR AUC. Data are means ± SEE, n=12. Bars with different letter superscripts are significantly different ($p<0.05$).

APPENDIX B

EXTENDED LITERATURE REVIEW

OSTEOPOROSIS – FROM THE MALE PERSPECTIVE

Literally, osteoporosis means "porous bone" (86). In elderly men and women, osteoporosis is the leading cause of fractures. Aging is associated with changes in bone mass and architecture that include decreased bone mineral density (BMD). In general, osteoporosis is viewed as a disease that predominately affects women. Recently, additional research has shown that approximately 20% of all osteoporosis-related fractures occur in men (174). Therefore, men are also at risk of developing osteoporosis and experiencing fractures as they age. Osteopenia is a precursor to osteoporosis and is also characterized by low BMD; however, the loss of bone mass is not as great as that of osteoporosis.

An expert panel of the World Health Organization (WHO) has set forth diagnostic criteria for the screening of osteopenia and osteoporosis based on the results of dual energy x-ray absorptiometry (DXA). Using the average BMD of the hip and lumbar spine of individuals aged 20 to 29 years as the standard for comparison, the WHO classifies individuals with a standard deviation (T-score) less than -1.0 but greater than -2.5 as having osteopenia. Those individuals with a T-score less than -2.5 are classified as having osteoporosis (107).

Two forms of osteoporosis develop in men: idiopathic and secondary osteoporosis. Idiopathic osteoporosis has no known cause although genetics likely play a large role (162). One-half to two-thirds of all cases of osteoporosis in men are secondary in nature. The cause of secondary osteoporosis can be traced directly to specific pathologies or detrimental life style choices. Secondary osteoporosis in men is commonly caused by weight loss and low body mass (84, 106), excessive alcohol

consumption (106, 196), cigarette smoking (196), low levels of recreational activity (106), glucocorticoid excess or treatment (162), and hypogonadism (162).

Body mass, fat mass, lean body mass. There is a large contribution of body mass on bone mass and BMD in both men and women (63, 140). Low body mass has been correlated with low BMD in men and postmenopausal women (106, 173). The separate contributions of lean body mass or fat mass on BMD are controversial (176). Reid et al. (180) reported fat mass to be positively correlated with total body BMD in postmenopausal women ($r=0.60$, $p<0.0001$) while lean body mass did not significantly correlate with total body BMD (180). A review by Reid and colleagues (176) concluded that in postmenopausal women, the correlation between fat mass and total body BMD ranges between $r = 0.34$ and $r = 0.55$. The correlation between lean body mass and total body BMD is less than that observed between fat mass and total body BMD ($0.18 < r < 0.20$) (176). In contrast, Wang et al. (234) reported that lean body mass had a greater influence than fat mass on BMD. Wang and colleagues (234) reported that although lean body mass and fat mass both correlated with BMD in women, the correlation between lean body mass and total body BMD was greater than the correlation between fat mass and total body BMD (234). It has been speculated that genetic factors account for between 60% and 80% of the variance in femoral neck BMD and lean body mass, but that environmental factors cannot be ignored (200).

Prevalence and Economic Burden. In the United States, approximately 1.5-million osteoporosis-related fractures occur annually (183). In 1995, $13.8 billion of national health care costs in the U.S. could be attributed to osteoporosis-related fractures. Of that total, $2.5 billion (18.4%) could be attributed to Caucasian males (174). By 2005,

it was predicted that $19 billion of U.S. health care costs will be attributed to osteoporosis-related fractures (23). Of this, 29% of fractures and 25% of the costs will be associated with fractures in men, but these estimates have not been verified beyond this prediction (23). The risk of suffering any osteoporosis-related fracture in an individual's lifetime is between 40% and 50% in women and between 13% and 22% in men (103). Fractures of the spine, hip, and distal forearm are the most clinically relevant osteoporosis-related fracture sites.

Hip Fractures. Using the WHO diagnostic criteria, the National Health and Nutrition Examination Survey (NHANES III, 1988-1994) reported the prevalence of osteoporosis of the hip in men over the age of 50 years to be between 3% and 6% while the prevalence of osteopenia of the hip to be between 24% and 47% (135). The discrepancy in prevalence rates was attributed to differences in the female or male reference data used in the comparison (135). Hip fractures are estimated to occur in 16% of Caucasian women over the age of 50 years and in 6% of Caucasian men over the age of 50 years (48). This is of concern because the U.S. population over 50 years of age is predicted to grow by 60% from 2000 to 2025 reaching a total of 121-million Americans by 2025 (27). The one-year mortality rate following a hip fracture in men is nearly double that in women. Kiebzak et al. (117) reported that the one-year mortality rate in men following a hip fracture is 32% compared to 17% in women (117). This nearly doubling of mortality in the first year following a hip fracture has been verified elsewhere (105, 220).

Vertebral Fractures. Only between 15% and 30% of vertebral fractures are brought to the attention of medical professionals (199). Nevertheless, these undiagnosed

vertebral fractures are associated with back pain, loss of height, respiratory dysfunction, social isolation, and reduced quality of life (199). The European Vertebral Study, which used vertebral deformities as a marker of vertebral osteoporosis, found that men and women between the ages 50 and 79 years have virtually identical prevalence rates of vertebral fractures (160). Similar to hip fractures, mortality following a vertebral fracture is greater in men that women (33).

Treatment in Men. Treatments available for osteoporosis are under utilized in men. The likelihood that men who suffer an osteoporosis-related fracture will be prescribed a pharmacological anti-resorptive treatment is less than in women (61, 117) despite approval of the use of the bisphosphonate alendronate to treat osteoporosis in men in late 2000 (163). Men are more likely to be prescribed a treatment of dietary calcium and vitamin D supplementation as opposed to a pharmacological intervention (117). Feldstein et al. (62) reported that only 7.1% of men aged 65 years and older were prescribed a medication for osteoporosis during the 18-month follow-up period after an initial osteoporosis-related fracture (62). Thus, effective treatments, screening, and preventative measures for osteopenia and osteoporosis in men must be developed, communicated to physicians, and disseminated to the general public.

GENETICS OF OSTEOPOROSIS

Twin and family studies have shown that heredity and genetics play a strong role in bone density and fracture risk. In twin studies, dizygotic twins have significantly greater variation in bone mass than monozygotic twins (207). Pocock et al. (169) reported greater correlations between monozygotic twins, than dizygotic twins, and BMD of the lumbar spine ($r = 0.92$ versus $r = 0.36$), femoral neck ($r = 0.73$ versus $r = 0.33$),

Ward's triangle (r = 0.85 versus r = 0.36), trochanter (r = 0.75 versus r =0 .47), and BMC of the forearm (r = 0.71 versus r = 0.50) (169). Environmental factors may play a greater role in BMD of the forearm and hip than at the spine where the correlation between monozygotic twins was the strongest (169).

Family history predisposes an individual to osteoporosis and increased fracture risk. Environmental factors within a family that include low amounts of physical activity, low dietary calcium intake, and cigarette smoking may be detrimental to bone accruement during adolescence and may contribute to greater bone loss during adulthood. Daughters of women with postmenopausal osteoporosis have been reported to have reduced BMD of the lumbar spine compared with daughters of women without osteoporosis (201). A meta-analysis by Kanis et al. (104) reported that a parental history of fractures, independent of BMD, increases the risk of children suffering a fracture of any site (RR = 1.17, 95% CI = 1.07-1.28) or the hip (RR = 1.49; 95% CI = 1.17-1.89) (104). Unfortunately, family studies cannot entirely separate environment from genetics.

As science has progressed and the human genome has been mapped, several candidate genes that may influence bone mass, fracture risk, and ultimately the development of osteoporosis, have been identified. In 1992, Morrison et al. (150) first described that polymorphisms of the gene encoding the vitamin D receptor (VDR) predict reduced serum osteocalcin concentration (a marker of bone formation) (150). Morrison and colleagues (149) went on to postulate that 75% of the genetic variation in bone density can be predicted by polymorphisms of the VDR gene (149). Heterozygotic and homozygotic alleles of the VDR gene have been associated with increased fracture risk independent of BMD in postmenopausal women (71). In contrast, other reports have

found no association between VDR polymorphisms and bone turnover or BMD (73). While polymorphisms of the VDR gene may influence BMD and fracture risk, the influence is likely small or insignificant (44, 58, 71).

As this line of research has continued, other genes that play a role in bone mass, fracture risk, and the development of osteoporosis have also been identified. Strong candidate genes include the genes that encode the estrogen receptor, collagen type Iα1, and collagen Iα2. Other candidate genes include genes for: transforming growth factor-β (TGF-β), methylentetrahydrofolate reductase, low-density lipoprotein receptor related protein 5 (LRP5), runt-related gene 2 (RUNX2), core binding factor A1 (CBFA1), interleukin-1 receptor antagonist, apolipoprotein E, and osteoprotegrin (for a review see ref 127). The influence of genetics on osteoporosis and fracture risk likely involved the interaction of multiple gene, in combination with environmental factors, and the influence of specific genes is controversial at this point (224).

BIOLOGY OF BONE

Function of the Skeleton. The major purpose of the skeleton is to protect vital organs, provide levers for skeletal muscle to induce movement, store minerals, and to produce red and white blood cells. Often thought of as an inert material, the skeleton is a dynamic tissue that is under constant renewal and regulation by a variety of mechanisms.

There are two main types of bone in the skeleton: cortical bone and cancellous bone. The cortical bone is hard and protective, while the cancellous bone (also known as trabecular bone) is 'spongy' and provides for metabolic activity and mineral exchange. The adult skeleton is composed of approximately 80% cortical bone and the remaining 20% is composed of trabecular bone. Bones are covered by a sheath called the

periosteum that contains blood vessels, nerve endings, and bone remodeling cells. The endosteum lines the interior cavity of long bones where the bone marrow is housed (56).

Bone is a composite material that allows it to resist the forces of compression, tension, and shear to prevent fracture. The hydroxyapatite crystals of the matrix allow bone to resist compressive forces. These crystals do not protect against tension or shear forces. Collagen fibrils are interlaced into the matrix to provide resistance against tensile and shear forces (191). Deformation of the skeleton, known as strain (ε), is represented as the change in length divided by the original length ($\varepsilon = \Delta L/L$) (191). During vigorous activity, peak strains can reach 3500 με in compression, 1000 με in tension, and 1500 με in shear (191). Bone remodeling is sensitive to changes in the magnitude of strain, the number of loading cycles, the distribution of the loading, and the rate of strain applied (191). A minimal strain threshold must be applied to signal remodeling; however, strain that is too great or applied too quickly may result in fracture.

Bone Remodeling. Bone modeling is the construction of new bone that occurs during development to shape the skeleton and to achieve of peak bone mass during adolescence and young adulthood. Peak bone mass is typically achieved during the third decade of life. Cooke in 1955 described the process of bone remodeling as the "ceaseless activity" that is ongoing throughout life to renew the skeleton (43). Bone remodeling is the process by which bone is re-shaped, maintained, and damage repaired by the coordinated actions of bone formation cells (osteoblasts) and bone resorption cells (osteoclasts). Remodeling can be viewed as preventative maintenance; it is an extensive process by which fatigued bone is replaced with new bone (56). It has been estimated that the skeleton is completely replaced every ten years (139).

Remodeling occurs by the collaborative effort of a number of cells that form a basic multi-cellular unit (BMU) (165). The BMU coordinates the efforts of osteoclasts, osteoblasts, a central vascular capillary, a nerve supply, and connective tissue with the coordinated goal of breaking down an area of bone and rebuilding new bone in its place (165). In a healthy adult, three to four million BMUs are initiated per year. The lifespan of each BMU is approximately six to nine months long (165). The BMU functions differently in cortical and trabecular bone. In cortical bone, the BMU burrows through the bone in a tunnel-like fashion. In trabecular bone, the BMU travels along the trabecular surface like a trench (165).

Remodeling is a sequential process consisting of four phases: activation, resorption, reversal, followed by formation (56). Activation begins with the recruitment of osteoclast precursor cells that attach themselves to the bone surface and develop into mature osteoclasts. The osteoclasts begin resorption by secreting acid, enzymes, and proteases to breakdown the bone matrix into its constituents (56). Bone resorption lasts approximately four to eight weeks at a single BMU (165). During the reversal phase, the osteoclasts secrete growth factors that attract osteoblast precursor cells and stimulate the proliferation and differentiation of these cells into mature osteoblasts (56). During bone formation the osteoblasts synthesize organic matrix that replaces the matrix that was previously removed by the osteoclasts. Osteoblasts that do not undergo apoptosis at the end of the bone remodeling cycle become entombed in the matrix and become osteocytes (56). **Figure 1** is an illustration of the bone remodeling cycle.

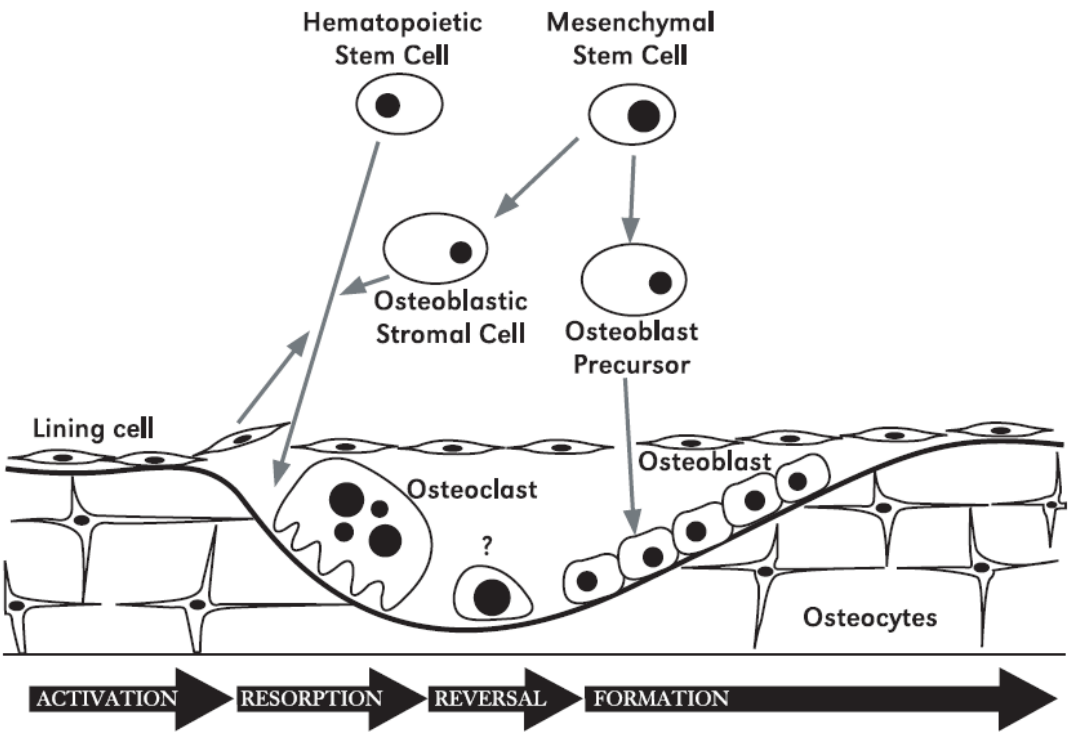


Figure 1: The Bone Remodeling Process. Figure from the Office of the Surgeon General's report regarding bone health. 2004.

Bone formation and bone resorption are both coupled and balanced so that the amount of bone removed during resorption is replaced by an equivalent amount of bone during formation. The combined process of bone formation and bone resorption is referred to as bone turnover. Bone turnover is considered balanced when formation matches resorption. The bone turnover process, and thus coupling, is regulated by electrical forces, mechanical forces, hormones, growth factors, and cytokines (235). During states of high rates of bone loss, such as during menopause, bone resorption is greater than bone formation. Thus bone turnover is imbalanced and bone loss occurs. An increased rate of bone turnover is also detrimental to bone, independent of the balance between bone formation and resorption, because more BMUs are formed and weaken the overall bone structure (164, 182).

BONE CELLS

The bone turnover process is the coordinated effort of specific bone cells: osteoclasts, osteoblasts, and osteocytes. Osteoclasts are responsible for bone removal and osteoblasts are responsible for replacing the removed bone. Osteocytes are now recognized as an important signaling cell.

Osteoclasts. Osteoclasts develop from hematopoietic cells of the monocyte-macrophage lineage (187). Once differentiated, these large (100- to 500-μm in diameter) (139), multinucleated cells attach to the bone surface by αvβ3 integrins (188). Utilizing the osteoclast's cytoskeleton, a sealing zone is formed between the osteoclast and the bone matrix called the resorption lacuna. Within the resorption lacuna specific ATP-driven proton pumps (vacuolar H^+-ATPases) secrete hydrochloric acid and enzymes into the sealing zone lowering the pH (139). The resulting acidic environment breaks down the hydroxyapatite crystals of the bone matrix (56). In addition to the hydrochloric acid secreted by the osteoclasts, other lysosomal enzymes (such as cathepsin K) (219), acid phosphatases, tartrate resistant acid phosphatase, and matrix metalloproteases-9 and -14 (MMP-9 and -14) (6) are secreted to aid bone resorption (56). The products of breakdown are endocytosed into the osteoclasts from the resorption lacuna and released from the cytosol through the plasma membrane opposite the resorption lacuna (154). The average lifespan of an osteoclast is approximately two weeks (139).

Osteoblasts. Osteoblasts are responsible for replacement of bone matrix that is removed by osteoclasts during remodeling. Osteoblasts develop from multipotent mesenchymal stem cells that also give rise to bone marrow stromal cells, chondrocytes, myocytes, and adipocytes (56). The growth and progression to mature osteoblasts, and

eventual apoptosis, is governed by steroids, polypeptide hormones, growth factors, and cytokines (7). The average lifespan of an osteoblast is three months, which is longer than the lifespan of osteoclasts (139). Osteoblasts are responsible for production of the osteoid matrix and the synthesis of many of the other proteins associated with the bone matrix, including osteocalcin and osteonectin (139).

Osteocytes. After the osteoblasts have synthesized the organic matrix, osteoblasts that do not undergo apoptosis are entombed in the bone matrix and become osteocytes. The osteocytes are connected to one another by gap junctions that extend through tubes in the bone matrix called canaliculae (56). It has been hypothesized that osteocytes sense changes in the mechanical properties of bone and communicate these changes with the bone-lining cells that initiate the bone remodeling process (24, 56). The communication between osteoblasts, osteocytes, and osteoclasts is hypothesized to behave like a neuronal network (221) that senses the fluid shift of the cytosol through the canaliculae from one osteocyte to another as bones are strained by mechanical loading (119, 223).

NUTRITION

Nutrition plays an important role in bone health throughout the lifecycle, including the accruement of bone mass during youth and aged-associated bone loss. Calcium, vitamin D, and protein play a primary role; however, phosphorus, magnesium, copper, zinc, vitamin C, and vitamin K also affect bone health (88). The Dietary Reference Intakes (DRIs) in the United States have been established by the Food and Nutrition Board of the National Academy of Sciences. An Adequate Intake (AI), established as the level of intake recommended to meet the individual needs of 97% to 98% of the population, has been set for calcium at 1,000 mg per day in adults under 50

years of age. After the age of 50 years, the AI increases to 1,200 mg per day of calcium to prevent age-associated bone loss. The AI for vitamin D in adults under 50 years of age is 5 μg per day, 10 μg per day for adults between 50 and 70 years of age, and 15 μg per day for adults over the age of 70 (65).

Calcium. Calcium is the most abundant mineral in the body and 99% of calcium is located in the skeleton. In addition to calcium's function in bone, it is also important for cellular structure, metabolic function, signal transmission, skeletal and heart muscle contractions, nerve function, blood clotting, and the activities of several enzymes (122). The skeleton acts as a reservoir for calcium such that when calcium intake is low, calcium can be brought from the skeleton to the blood stream to fulfill the functions mentioned above. The importance of calcium intake starts early in childhood when bones are being modeled and rapid linear growth occurs. Children that avoid dairy products have greater bone fragility during childhood and adolescence, which can be assumed to carry into adulthood (76). In a double-blind, placebo-controlled, calcium-supplementation trial, 100 adolescent girls with calcium intakes of less than 800-mg per day consumed a supplement containing either 1,000 mg per day of calcium or placebo. After one year, the girls that received the calcium supplement had significantly greater increases in total body BMD (3.80 ± 0.3% compared with 3.07 ± 0.29%, $p<0.05$) and lumbar spine BMD (3.66 ± 0.35% compared with 3.00 ± 0.43%, $p<0.05$), but not greater femoral neck BMD or BMC of any site (190). Similar results have been reported elsewhere (39). The benefits of calcium supplementation during adolescence were scrutinized in a recent Chochrane Database Review. The authors of the review report that the effect of calcium supplementation is small and limited to the upper body (237).

Later in life, when remodeling predominates and approximately 200 mg of calcium are removed and replaced from the skeleton each day, calcium intake plays an important role in preservation of bone mass. Calcium supplementation alone has been reported to slow bone loss in postmenopausal women (53, 179). Calcium plays an important role in skeletal health through all stages of life.

Vitamin D. Vitamin D is consumed in the diet and produced by the skin upon exposure to ultraviolet (UV) rays. The two major forms of vitamin D are ergocalciferol (vitamin D_2) and cholecalciferol (vitamin D_3). Between 800 and 1,000 IU of vitamin D per day are necessary to achieve a serum 25(OH)D concentration of $\geq$ 75 nM, which is associated with improved calcium absorption (89) and reduced risk of fractures (15, 52). Vitamin D insufficiency is quite prevalent among Americans. NHANES III data suggest that between 25% and 57% of Americans are vitamin D insufficient based on serum 25(OH)D concentrations less than 62.5 nM (134). The wide variance in prevalence estimates is likely due to the seasonal variations in the measurement of serum 25(OH)D. The reduced zenith angle of the sun during the winter months increases the atmospheric filtration of UVB rays (122). At northern latitudes of the U.S., sun exposure is inadequate to facilitate vitamin D formation except for a few summer months per year.

During adolescence, low serum concentrations of vitamin D are associated with low cortical BMD (35). Daily supplementation with 5 or 10 μg of vitamin D_3 in adolescent girls improved hip bone mass by 14.3% and 17.2%, respectively, compared with controls receiving placebo (232). The greater daily dose of vitamin D_3 (10 μg per day) was required to improve bone mass of the lumbar spine (232). A meta-analysis by Bischoff-Ferrari and colleagues (16) reported supplementation of between 600 and 700

IU per day (between 17.5 and 20 μg per day) of vitamin D is necessary to reduce the risk of non-vertebral fractures in the elderly (16). Supplementation with the combination of vitamin D and calcium reduces the risk of non-vertebral fractures (34) and reduces bone loss in elderly women (52). Similar benefits of supplemental calcium and vitamin D have also been reported in men (52). The combination of dietary or supplemental consumption of calcium and vitamin D is a hallmark of osteoporosis prevention and treatment.

ENDOCRINOLOGY

Parathyroid Hormone. Parathyroid hormone (PTH) has a major role in calcium homeostasis (22). PTH is secreted from the chief cells of the parathyroid gland in response to small decreases in plasma calcium concentration. Circulating PTH stimulates the release of calcium and phosphate from bone tissue, increases reabsorption of calcium in the kidney, decreases reabsorption of phosphate at the kidney, and stimulates the activity of renal 1-α-hydroxylase, the enzyme responsible for the synthesis of the active form of vitamin D, $1,25(OH)_2D_3$ (22).

Parathyroid hormone is commonly thought to induce bone loss because it stimulates the release of calcium from the skeleton. Chronic exposure to PTH increases osteoclast number and activity (22). Hyperparathyroidism results in bone loss (168) and increased incidence of fractures (112). Individuals with primary hyperparathyroidism have slight reductions in cancellous bone mass (i.e., spine), large loss of bone mass in the primary cortical skeleton (i.e., forearm), and intermediate bone loss of the hip (for a review see ref 192).

Communication between the osteoblasts and osteoclasts is disrupted by PTH leading to an uncoupling bone turnover in favor of resorption. Osteoblasts secrete receptor activator for nuclear factor-κB ligand (RANKL) which interacts with the osteoclast receptor RANK. Thus, osteoblasts appear to have an inhibitory influence on osteoclasts (22). This interaction is partially controlled by osteoprotegerin (OPG), a soluble "decoy" receptor for RANKL. PTH inhibits the expression of OPG by osteoblasts and osteoclasts, thereby reducing the osteoblastic inhibitory influence on osteoclastogenesis (22). Paradoxically, although chronic exposure to excess PTH reduces BMD, intermittent PTH treatment might increase bone mass and will be discussed later (120).

Sex Steroids. The loss of estrogen is commonly thought to be responsible for the loss of bone mass post-menopause. Estrogens and androgens play a substantial role in bone health. Both androgen and estrogen receptors are expressed in near equal numbers in the plasma membranes of bone cells. Bone cells also express 5α-reductase and aromatase activity, which are responsible for the conversion of testosterone to dihydroxytestosterone (DHT) and estradiol, respectively (198). The reduced circulating concentration of estrogen that accompanies menopause is largely responsible for the rapid bone mineral loss that occurs post-menopause. Estrogen replacement therapy is widely used during the early time course of menopause to slow bone loss. Estrogens have been shown to decrease bone resorption, osteoclastogenesis, and inhibit osteoclast function (198). Although estrogen is primarily a female sex steroid, its importance is often overlooked in men.

Testosterone has an anabolic influence on many tissues. Classically, it is believed that testosterone helped regulate bone turnover in men. This thought process has been challenged by population-based cross-sectional studies and individual genetic aberrations (to be discussed later) (59). Associations between low circulating concentrations of androgens and low BMD have not been consistently reported in men (5, 116, 172, 205). In contrast, strong associations between reduced estradiol concentrations and low BMD in men have been reported (5, 116, 205). An association between reduced free estradiol concentrations and low BMD in men has also been reported (215). The concentration of both estrogens and androgens decrease with age; however, the bioavailable free estrogens and androgens decrease to a greater extent, as sex-hormone binding globulin (SHBG), which is the predominant plasma sex hormone binding protein, increases with age (116).

Slemenda et al. (205) first reported a positive association between serum concentrations of estradiol and BMD in men (205). In men over the age of 65 years, Slemenda and colleagues reported that serum estradiol concentration was positively correlated with BMD of the radius ($r = 0.28$, $p<0.001$), lumbar spine ($r = 0.21$, $p<0.01$), femoral neck ($r = 0.33$, $p<0.001$), and trochanter ($r = 0.35$, $p<0.001$) (205). Serum testosterone concentrations were negatively correlated with BMD of the lumbar spine, femoral neck, and trochanter ($-0.28 < r < -0.20$, $p<0.05$) (205). Khosla et al. (116) confirmed these results by reporting that serum concentration of estradiol was the only independent predictor of BMD in men and women between the ages of 21 and 94 years (116).

The strongest evidence that estradiol plays a prominent role in bone density is from case study reports of individuals suffering from genetic defects or “experiments of

nature" (59). These reports have been generated in response to individuals presenting clinically with mutations of the estrogen receptor gene or a lack of the aromatase enzyme responsible for the conversion of testosterone to estrogen. Mutations of the estrogen receptor, result in tall stature, failed epiphyseal closure, and low BMD, despite normal concentrations of androgens and estrogens (209). A lack of the aromatase enzyme results in reduced concentrations of estradiol and severe loss of BMD due to an inability to convert testosterone to estradiol (30). Treatment of an individual lacking the aromatase enzyme with testosterone yielded no positive results. When the individual was treated with estradiol, bone mass improved and epiphyseal closure occurred (30). These results have been verified in other individuals with deficiencies of the aromatase enzyme (14). Regulation of skeletal mass by estradiol is important, but the androgens do play a role as well.

Growth Hormone & Insulin-Like Growth Factor I. The growth hormone (GH) – insulin-like growth factor-1 (IGF-1) axis plays an important role in bone development, longitudinal bone growth, skeletal maturation, acquisition of bone mass during childhood, and the bone remodeling process (75). GH is secreted from the anterior pituitary gland and is under the control of central and peripheral signals. The GH receptor (GHR) is expressed in a variety of tissues including bone (75). GH secretion decreases during aging (240). GH is important in coupling bone turnover because GH directly stimulates the osteoblasts and interacts both in a stimulatory and inhibitory fashion on osteoclasts (161).

GH deficiency (GHD) is an early model to study the interaction between GH and bone mass. GHD in animal models induces reductions in bone formation, reduced bone

turnover, and induces PTH resistance (75). Children that develop idiopathic GHD have reduced BMC (110) and BMD (54) as adults. Adult-onset GHD is associated with reduced BMD, but not to the extent of childhood onset GHD (97).

The primary source of circulating IGF-I is the liver. IGF-I is also secreted in both paracrine and autocrine fashions as IGF-I is secreted by tissues and cells that feedback on these same tissues and cells. An example of this occurs in osteoblasts which secrete IGF-I. This signal feeds back on these same osteoblasts to induce bone formation. The actions of IGF-I are affected by coupled to its binding proteins, specifically IGFBP-3, -4, and -5 (84). Serum concentrations of IGF-I and IGFBP-3 have been reported to be lower in individuals suffering an osteoporosis-related fracture than healthy controls (238). Small, early studies in middle-aged men with idiopathic osteoporosis reported lower plasma concentrations of IGF-I than men with average BMD (132). Larger, population-based studies have successfully detected associations between bone density and IGF-I in women (8, 121), but not all studies have reported this association (72). The Ranchero Bernadro Study reported reduced serum IGF-I concentrations to be correlated with reduced BMD of the spine ($\beta = 0.0009$, $p=0.0001$) and hip ($\beta = 0.0003$, $p=0.02$) in women only (8). No association was detected in men (8). In contrast, Garnero et al. (72) reported no correlation between reduced concentrations of IGF-I and BMD. The authors did note an association between reduced IGF-I and increased fracture risk, independent of BMD (72).

Osteoporosis treatments involving GH are being explored. In humans suffering from GHD, short-term administration of GH (less than one year) has not been successful at improving BMC or BMD. In some cases, reductions in BMD (96) or BMC (231) have

been reported. GH administration for longer periods of time results in improvements in BMD (10, 102, 159, 231) and markers of bone turnover (102). Johansson et al. (102) administered GH for two-years to 44 patients with GHD and osteopenia (102). The authors reported increases in BMD of the lumbar spine, femoral neck, femoral trochanter, and Ward's triangle of 3.8%, 4.1%, 5.6%, and 4.9%, respectively, after treatment with GHD (102).

Administration of GH to men with idiopathic osteoporosis has resulted in positive results when trials are carried out for greater than one-year (74). Gillberg and colleagues (74) randomized men to either a treatment of 0.4 mg of GH per day or intermittent injections of 0.8 mg of GH per day for two years (74). The authors reported that the 0.4 mg dose of GH per day increased BMD of the lumbar spine by 4.1% and total body BMD by 2.6%. The intermittent injections did not result in changes in MD (74). Unfortunately, GH treatment has adverse side effects, usually fluid retention, and it is no occur with GH treatment, usually associated with fluid retention, and it is not at a treatment onption at this time.

Leptin. In 1992, Reid et al. (180) reported that BMD in postmenopausal women is positively associated with fat mass, but not lean body mass (180). The discovery of leptin in 1994 led to speculation that adipose tissue, which secretes leptin, may signal bone tissue to increase bone mass in overweight and obese individuals (81, 176).

Leptin is one the body's signal of energy status. Serum concentrations of leptin increase as food intake increases and signal the hypothalamus to reduce appetite and increase energy expenditure as the body attempts to remain in homeostasis (81). Obesity is associated with elevated serum leptin concentrations increased bone size, and enhanced

cortical bone density (125). Pasco et al. (166) was the first to report a relationship between leptin and bone mass, independent of body weight and fat mass (166). Since this study, these results have been verified elsewhere (18, 217). However, a gender difference may exist in the relationship between leptin and BMD (18).

In young men, there appears to be a negative association between serum concentrations of leptin and BMD that is not evident in older men. Lorentzon et al. (136) reported in young, Swedish men that plasma leptin concentration is a negative predictor of total body, lumbar spine, and trochanter areal BMD (β = -0.08 to -0.13; p=0.01) (136). Negative associations between leptin and BMD in young obese and non-obese men have also been reported (148). The influence of leptin may be dependent upon age and gender.

Transgenic animal models and in vitro techniques have confirmed that leptin influences bone mass and interacts with bone cells. In vitro, leptin stimulates the differentiation and proliferation of osteoblasts (46) and inhibits osteoclastogenesis (95). Leptin deficient (*ob/ob*) mice, despite the development of obesity, have reduced femoral length, BMC, BMD, cortical thickness, and trabecular bone volume compared to wild-type mice (83). In contrast, the *ob/ob* mice have increased vertebral length, BMC, BMD, and trabecular bone volume compared to wild-type mice (83). Treatment of the *ob*/ob mice with leptin induces a 30% increase in total body BMC (82). In addition to leptin, other adipose-tissue-secreted signals that influence bone have been identified, including cytokines, estrogens, adiponectin, and resistin (81, 176).

Glucocorticoids. A common secondary form of osteoporosis is glucocorticoid-induced osteoporosis (GIO). Corticosteroids are commonly administered in the treatment of chronic disease such as rheumatoid arthritis, chronic obstructive pulmonary disease

(COPD), and chronic inflammatory bowel disease (29). Glucocorticoid treatment of these conditions induces a rapid decrease in BMD (29). Many of these diseases have an underlying pathology that enhances bone resorption in addition to the bone loss that results from the pharmacological treatment with corticosteroids themselves (29).

Bone formation decreases as a result of glucocorticoid treatment due to reduced osteoblast activity (50), inhibition of the secretion of type I collagen (29), and apoptosis of mature osteoblasts (167) and osteocytes (131). Glucocorticoid treatment induces pre-osteoblasts to differentiate into adipocytes as opposed to mature osteoblasts (28). In vitro, glucocorticoids induce osteoclastogenesis resulting in an increase in bone resorption (94). Overall, glucocorticoids uncouple bone turnover by initially increasing bone resorption but progressing to a state of both inhibited bone formation and stimulated bone resorption (29). These effects appear to have the greatest impact on trabecular bone (178). The detrimental influence of glucocorticoid treatment on bone mass has led researchers to employ pharmacological treatments that prevent bone loss in coordination with glucocorticoid treatment.

BIOCHEMICAL MARKERS OF BONE TURNOVER

Bone mineral density is a static measure of bone status. Techniques that are dynamic measures of bone cellular activity are expensive, time consuming, and often invasive. Dynamic measurements of bone turnover have been developed to track bone remodeling. The advancement of biochemical markers of bone turnover has been a major methodological advancement and these markers are an important research tool (26). Biochemical markers of bone metabolism have been useful in describing the physiology and patho-physiology of bone diseases (202). Currently, biochemical markers of bone

turnover are used to: monitor the effectiveness of therapies in the short term; monitor pharmacological interventions; predict future bone loss; select patients for therapy; and possibly predict of fracture risk (26). Unfortunately, markers of bone turnover are not entirely specific to bone tissue in some cases and are influenced by bone-specific diseases, menstrual cycle, bed rest, exercise, dietary extremes, malignancy, recent fractures, diurnal rhythms, and seasonal variations (202, 235).

Serum bone formation markers include bone-specific alkaline phosphatase (BAP), osteocalcin (OC), carboxyterminal propeptide of type I collagen (PICP), and aminoterminal propeptide of type I collagen (PINP). Markers of bone resorption may be measured in either the urine or serum. Urinary markers of bone resorption include total and free pyridinolines (Pyd), total and free deoxypyridinolines (Dpd), N-telopeptide of collagen crosslinks (NTx), and C-telopeptide of collagen crosslinks (CTx). Serum markers of bone resorption include NTx, CTx, cross-linked C-telopeptide of type I collagen (ICTP), and tartrate-resistant acid phosphatase isoform 5b (TRAP5b) (26, 80, 235). **Table 1** lists the biochemical markers of bone turnover.

Table 1: Markers of Bone Formation and Bone Resorption

Bone Formation	Bone Resorption
Total alkaline phosphatase (serum)	C-terminal pryidinoline cross-linked telopeptide of type I collagen (ICTP) (urinary or serum)
Bone specific alkaline phosphatase (BAP) (serum)	Free γ-carboxy glutamic acid (serum)
Osteocalcium (OC) (serum)	Tartrate resistant acid phosphatase (TRAP) (serum)
Carboxyterminal propeptide of type I collagen (PICP) (serum)	Calcium (urinary)
Aminoterminal propeptide of type I collagen (PINP) (serum)	Hydroxyproline (total, free) (urinary)
	Pyridinoline (Pyr) (total, free) (urinary)
	Deoxypyridinoline (Dpd) (total, free) (urinary)
	N-telopeptides (NTx) (urinary or serum)
	C-telopeptides (CTx) (urinary or serum)
	Hydroxylysine glycosides (urinary)

Table modified from reference Seibel MJ, 2003 (202).

Bone Formation Markers. Alkaline phosphatase is associated with the plasma membrane of many cells (235). Hypophosphatasia, in which individuals lack alkaline phosphatase and suffer from osteomalacia, confirms that the enzyme is important in the mineralization of the skeleton and teeth (80). Due to the many tissue sources of alkaline phosphatase it has become an almost obsolete marker of bone formation. The bone-specific isoform, BAP, is specific to bone tissue and has largely replaced alkaline phosphatase as a marker of bone formation (235).

Osteocalcin is a small protein, rich in glutamic acid, and is referred to as a Gla-protein. OC is widely accepted as a serum marker of bone formation, although its fragments are also released from the bone matrix during resorption (26). OC is developing a reputation as being a bone-secreted hormone that is involved in energy metabolism (64, 125). In transgenic animal models, OC knock-out mice (*Osteocalcin*$^{-/-}$) are glucose intolerant and overweight compared to wild-type mice (64). In the *Osteocalcin*$^{-/-}$ model, replacement of OC significantly reduces obesity and glucose intolerance (64). In addition to BAP and OC, procollagen extension peptides are byproducts of the synthesis of type I collagen and are useful serum measures of bone formation. The amino- and carboxy-terminal procollogen 1 extension peptides (PINP and PICP) are serum markers of bone formation (197).

Bone Resorption Markers. Acid phosphatase is a lysosomal enzyme found in a variety of tissues including the bone, prostate, platelets, erythrocytes, and spleen (235). Tartrate-resistant acid phosphatase has been used as a serum marker of bone resorption. The 5b-isoform has been shown to be more specific to bone, and is an indicator of osteoclast number (202). Pyr and Dpd are cross-links between type I collagen fibers and

are released during breakdown of type I collagen of which bone is the predominant source (235). Hydroxyproline is the posttranslational modification of the amino acid proline that is common in type I collagen. Bone releases most of the hydroxylproline that is metabolized by the liver and excreted in the urine (235). Cross-linked telopeptides are the amino- (NTx) and carboxy-terminal (CTx) fragments of type I collagen released during the breakdown of bone. Although NTx and CTx are excreted in the urine (235), serum assays have also been developed for CTx (19).

Higher rates of bone turnover are associated with greater and more rapid bone loss (57). Unfortunately, screening individuals for vertebral osteoporosis or osteopenia by biochemical markers of bone turnover alone has been of little success (202). Results from the Rotterdam Study indicated that women with increased urinary concentrations of bone resorption, assessed by Dpd, had an increased risk of suffering a hip fracture (230). Furthermore, Dresner-Pollak et al. (57) reported that in elderly women, bone loss of the hip was significantly negatively correlated with markers of bone turnover including urinary NTx, free Pyr, total Pyr, total Dpd, hydroxyproline, serum OC and BAP (57). Keen *et al.* reported no correlation between bone turnover markers (serum OC, serum total alkaline phosphatase, urinary pyridinoline and deoxypyridinoline) and future bone loss (111). At this point in time, biochemical markers of bone turnover provide an easy, non-invasive means to track treatment of osteoporosis in the short term, but the ability to predict bone loss and fracture risk is controversial.

WHY EXERCISE IS GOOD FOR BONE

Physical activity and exercise are often promoted as a means of osteoporosis prevention. Greater amounts of daily physical activity have been associated with greater

BMD (1, 20, 100, 208, 228); however not all studies have reported this association (143). Physical activity in the form of purposeful dynamic, weight-bearing exercise may be necessary to elicit true improvements in bone mass.

Mechanical Loading. The mechanical loading applied or strain placed upon the skeleton is responsible for the positive remodeling induced by dynamic, weight-bearing activities. In the 1960s, Harold Frost introduced the 'mechanostat' hypothesis. This was a redefining of Wolff's law which stated that bone tissue remodels itself in proportion to the loads applied to the skeleton (68). Charles Turner (222) has proposed three means by which bone strength is enhanced by mechanical loading. First, bone adapts in response to dynamic, not static loads. In 1984, Lanyon and Rubin (123) elegantly demonstrated this in the ulna of birds by externally loading a limb by static and dynamic means and showed that bone was not strengthened by static loads alone (123). Second, bone adapts when only short duration loads are applied. Longer duration application of force can actually be detrimental. Finally, bone adapts when the loading applied is greater than what the skeleton has already experienced (222). Athletes are a simple model to examine mechanical loading on the skeleton.

Human Cross-Sectional Studies. For many years it has been known that athletes have greater bone mass than active and sedentary populations (157). The type of loading experienced during different training and exercise regiments has important influence on bone strength and mass. Athletes who are involved in dynamic, high-impact sports, such as gymnastics (118, 156, 216), running (21, 137, 213), soccer (3), basketball (124), and volleyball (2, 60, 124) have greater BMD of the hip and spine compared with sedentary controls. Low-impact sports, such as swimming and cycling, do not elicit changes in

BMD at these sites. For instance, Lee et al. (124) reported that female athletes performing high-impact sports, such as volleyball and basketball, had significantly greater BMD at the lumbar spine, femoral neck, trochanter, and Ward's triangle than swimmers, moderately active individuals, and sedentary controls (124). Swimming is a non-weight bearing activity that generates forceful muscular contractions but does not appear to improve bone mass above that of a sedentary population from these cross-sectional studies (60, 124).

Another model of examining mechanical loading is in tennis players. Tennis induces large amounts of strain on both arms during play, especially the dominant arm. Haapasalo et al. (79) compared the dominant and non-dominant arms of age- and sex-matched non-playing controls and the dominant and non-dominant arms of the male and female players themselves. The results of this study concluded that tennis increased humeral BMC, BMD, and cortical wall thickness in the dominant arm of the players to a greater extent than that of the players' non-dominant arm and controls' dominant and non-dominant arms (79).

In general, running has a positive influence on bone mass (137). Running may have detrimental effects on the skeleton due to the high volume undertaken by some competitive individuals inducing an negative energy imbalance (137). Lumbar spine BMD has been reported to be reduced in female (194) and male (13) runners. Bilanin et al. (13) reported that men running an average of 92.2 km per week had significantly lower vertebral BMD compared with non-runners ($p<0.05$) despite calcium intakes above the recommended dietary intake (13). Bone appears to respond positively to dynamic,

weight-bearing activity unless this activity is above the threshold of volume where the bone can no longer adapt or energy intake is insufficient to match expenditure.

Strength training also provides an osteogenic stimulus. Conroy et al. (42) reported that junior weightlifters (average age of 17 years) had greater BMD at all sites measured (lumbar spine and proximal femur) than age-matched controls (45). These young weightlifters also had greater femoral neck and lumbar spine BMD compared with adults who did not compete in weight lifting (ages 20-39 years old) (45). Karlsson et al. (108) reported that male weightlifters had greater BMD of the whole body, trochanter, and lumbar spine than age-matched controls (10%, 2%, and 13%, respectively) (108). Karlsson and colleagues (108) also reported that BMD of the total body and spine of former weightlifters that had retired from competition remained greater than age-matched controls (108).

It is important to note that a genetic predisposition may cause athletes to compete in their respective sports. An individual with genetically low body mass and low BMD may not be suited for high-impact sports and an individual selection bias towards other activities may occur. Individuals with greater body mass and greater BMD may gravitate towards high-impact sports. Because of this limitation, cross-sectional studies cannot fully elucidate the influence of exercise training on BMD.

Animal Exercise Intervention Studies

A number of different animal models have been employed to investigate the interaction between dynamic, high-impact exercise and bone. These models include running (12, 101, 146, 171), swimming (85, 158, 214), resistance-training (236), and jumping (98, 99, 225, 226). Raab et al. (171) trained Fischer 344 female rats of different

ages (2.5- and 25-months old), to run for one-hour per day at 15% grade at a speed of 15-m/min (old rats) or 36-m/min (young rats), five-days per week for ten weeks. Running increased the fat-free dry weight of the femur and breaking force of the femur and humerus regardless of age (171). Similar results have been reported elsewhere (12, 146).

Jump-training that is dynamic and weight-bearing, and elicits large impact forces upon landing increased bone strength parameters in a number of rat models (98, 99, 225, 226). Umemura et al. (226) compared the osteogenic stimulus of jump-training to running in female Fischer 344 rats. Jump-training consisted of 100 jumps to a height of 40 cm per session, five days per week, for eight weeks. Treadmill running consisted of running at 30 mile/min, 60 minutes per session, five days per week for eight weeks. Rats who performed jump-training significantly increased fat-free dry weight of the femur and tibia compared with sedentary and running rats. In addition, in older rats (27 mo), running did not increase fat-free dry weight of the femur compared with sedentary rats, while jump-training successfully increased the fat-free dry weight of the femur (226). A large number of jumps per training session is not necessary to stimulate bone formation.

In another study, Umemura et al. (225) compared the osteogenic response of different volumes of jump-training. Immature female Fischer 344 rats were trained to jump (40 cm in height) five, ten, 20, 40, or 100 jumps per session, five days per week for eight weeks. The rats jumping only five times per session had significantly greater fat-free dry weight and maximal breaking stress of the femur and tibia compared with sedentary rats, controlling for body mass (225). A greater number of jumps per session produced only non-significant increases in fat-free dry weight of the tibia compared with five jumps per session (225). These results have been verified in ovariectomized rats, in

which 10 jumps per day significantly increased fat-free dry weight, ash weight, and ultimate breaking force of the tibia compared to non-jumping ovariectomized rats (99). Jumping has not only been reported to be an effective osteogenic stimulus, but also requires a minimal volume of training to induce positive remodeling.

Animal Mechanical Loading

Some of the most conclusive reports that have tested Frost's 'mechanostat' hypothesis are based on experiments in which loads were applied directly to the skeleton by ex vivo means. Turner and colleagues (221) reported that the tibia of adult female rats exposed to external bending for two weeks increased bone formation rate when external bending forces were applied at frequencies of between 0.5-Hz and 2.0-Hz (221). The amount of loading that can be applied to a limb is saturable, similar to the results of jump-training.

In a classical study, Rubin & Lanyon (194) provided evidence that 36 cycles of external loading applied per day were as effective at inducing bone formation as 1,800 cycles of external loading applied per day at the same strain magnitude (193). These results mirror the results of Umemura and colleagues (225) mentioned previously in that five jumps per session were effective as 100 jumps per session (225). Turner and colleagues have hypothesized that bone cells become 'deaf' to a greater number of strains applied above a particular threshold (221).

A number of studies have reported that insertion of rest periods between bouts of loading enhances the osteogenic stimulus (185, 186, 211). Robling et al. (185) reported that partitioning bouts of external loading throughout the day improved bone strength. Rat limbs were externally loaded 90-times per session, four times per day, or externally

loaded 360-times in a single-bout per day. Rat limbs subjected to four loading bouts per day had greater BMC, BMD, and midshaft cross-sectional area of the loaded limb than the loaded limb exposed to a single-bout of loading each day (185). Insertion of rest periods between individual applications of load has also proven effective. Srinivasan et al. (211) reported that a ten-second rest period inserted between the application of external loads resulted in greater periosteal bone formation rate compared with repetitive loading cycles (211). Mechanical loading applied externally to the skeleton of animals improves of bone strength in vivo.

Human Exercise Interventions

Cross-sectional studies of athletes have led to the conclusion that dynamic, weight-bearing exercise is required to maintain or increase bone mass. This conclusion has been verified by animal mechanical loading and exercise interventions. Controlled, preferable randomized, human exercise interventions are able to verify the causality reported by cross-sectional studies. The most successful and well-researched modes of exercise in humans include resistance-training, walking, and jump-training.

Resistance-training. Resistance-training has been reported to increase BMD in adolescents (155) and pre- and postmenopausal women (113, 114, 133, 152, 233). Resistance-training interventions have also been successful at increasing BMD in young men (70), middle-aged men (147, 195), and elderly men (233, 239). Not all resistance training interventions have been successful in women (11, 144, 170, 204), as will be discussed later.

Menkes et al. (147) reported that 16-weeks of resistance-training in middle-aged men, 50 to 70 years of age, improved BMD of the lumbar spine by 2.0% and of the

femoral neck by 3.8% ($p<0.05$). Age-matched controls, not performing resistance-training, did not improve BMD (147). A marker of bone formation, OC, increased by 19% after 12-wk and remained significantly greater than baseline at 16-wk ($p<0.05$). BAP also significantly increased by 26% after 16-wk compared to baseline ($p<0.05$). No changes in bone resorption assessed by serum TRAP concentration were reported (147).

In agreement, Ryan et al. (195) reported that resistance-training in middle-aged men improved BMD of the femoral neck by 2.8%, which was significantly greater than that of sedentary controls ($p<0.001$) (195). OC and BAP did not significantly change compared to baseline in either group; however, TRAP increased significantly in the training group only ($p<0.05$) (195). Both of these reports used Keiser K-300 resistance-training equipment and started with a loading equal to a five-repetition maximum and progressively decreased the weight until 15 repetitions were completed for a variety of exercises (147, 195). Unfortunately, the subjects in these studies were not randomized which weakens the results (147, 195).

Vincent and Braith (233) trained a population of elderly men and women (average age 68 years) for six months and reported increases of 1.96% in femoral neck BMD in the group using high loads (i.e., 80% one-repetition maximum). The group that performed exercises using low loads (i.e., 50% one-repetition maximum) did not improve BMD (233). Serum concentration of OC in both the light-load and high-load groups increased by 25.1% and 39.0%, respectively, relative to baseline ($p<0.05$). Serum BAP concentrations increased by 7.1% compared with baseline ($p<0.05$) in the high-load group only (233). Performing resistance-training with adequate intensity and loading is important for inducing bone formation.

In agreement with Vincent and Braith's comparison of different loading programs, Kerr et al. (114) compared two resistance-training programs in postmenopausal women: three sets performed at eight-repetition maximum and three sets performed at a twenty-repetition maximum, which the authors referred to as endurance resistance-training. In the group training with greater loads, BMD of the trochanteric hip ($p<0.01$), intertrochanteric hip ($p<0.05$), Ward's triangle ($p<0.05$), and the ultra distal radius ($p<0.01$) improved compared with both controls and the endurance resistance-training group (114). Pruitt et al. (170) reported no differences in hip or lumbar spine BMD in healthy, older women performing a high- or light-loading resistance-training program for one year (170). The length of the resistance-training intervention may also be important.

Vincent and Braith reported no significant increases in BMD when low loads were used as part of a 16-week resistance-training program (233). The short time period of the study confounds the results because the bone remodeling cycle lasts four to six months (139). It can be inferred that more than a single bone remodeling cycle would need to be completed before improvements in bone mass can be detected by DXA (142). Thus, it is important that Vincent and Braith were able to show increased concentrations of serum markers of bone formation. The length of any exercise intervention needs to be greater than six months to allow for multiple remodeling cycles to occur and achieve detectable change in BMD. Shorter studies have often not reported positive results regardless of the loading utilized (11, 70).

Resistance-training is widely accepted as a means of maintaining and possibly improving bone mass. Numerous medical associations and research bodies, including the National Osteoporosis Foundation, National Institutes of Health, and American College

of Sports Medicine (ACSM), support resistance training for osteoporosis treatment in elderly individuals to maintain bone mass and reduce the risk of falling (4).

Walking and Running. Cross-sectional and animal exercise interventions have conclusively reported that running can improve bone mass, but only to a point at which the volume of running becomes detrimental. This inflection point is variable per individual and a set volume of training above which bone loss may occur cannot be established. Running is also unlikely to be undertaken and sustained in elderly populations.

Walking interventions in men and women have been somewhat successful (37, 153, 181). Chein et al. (40) reported that six months of walking and stair stepping using a 20 cm bench in postmenopausal, osteopenic women, 48 to 65 years of age, improved BMD of the lumbar spine by 2.0% ($p<0.05$) and femoral neck by 6.8% ($p<0.05$) compared with baseline. In the sedentary group, lumbar spine BMD significantly decreased by -2.3% ($p<0.05$) from baseline (40). Exercise consisted of walking at 70% of VO_{2max} for 30-min per day, three days per week for 24-weeks, followed by ten minutes of stepping exercise using a 20-centimeter high step (40). Unfortunately, the study participants were not randomized and chose the intervention based on likely compliance. The women also participated in exercise other than walking further confounding the results.

Few true running interventions have been reported in the literature. A great deal of osteoporosis research is directed at elderly populations who would likely not be able to sustain a running program. In addition, many aerobic exercise interventions progress from a walking intensity to a slow running speed thus confounding the results. The

results of walking or running cannot be differentiated from one another by these study designs.

Jump-Training. Jump-training has been investigated in children (69, 91, 138), pre-menopausal women (9, 92, 109, 210, 229) and postmenopausal women (9, 36, 77, 210, 213, 217). In pre-menopausal women, a high-impact training program that included jumping exercises was reported to increase femoral neck BMD by 1.6% from baseline. This increase was significantly greater than the 0.6% increase reported in the sedentary controls ($p=0.006$) (92). Vainionpaa et al. (229) investigated the effect of a jump-training program consisting of: step patterns, stamping, jumping, running, and walking in pre-menopausal women (age 35 to 45 years). The jump-training group demonstrated a gain in femoral neck BMD of 1.1% that was significantly greater than the control group (0.4%, $p<0.05$) (229). In agreement with animal studies, a minimal number of jumps may be completed to induce positive bone remodeling. Kato et al. (109) reported that only ten maximal vertical jumps per day, three days per week, for six months increased BMD of the femoral neck and lumbar spine in young women (average age 20.7 years) compared to sedentary young women (109).

Postmenopausal women may not respond in the same fashion as pre-menopausal women to jump-training. Bassey et al. (9) compared the affect of 50 jumps per day, six days per week, of mean height 8.5-centimeters on both pre- and postmenopausal women. The results revealed a significant increase of 2.85% in femoral neck BMD of the pre-menopausal women that was significantly greater than the change in BMD at the femoral neck of the pre-menopausal controls (9). Unfortunately, no changes in BMD in postmenopausal women were reported (9). The authors speculated that postmenopausal

women may not be responsive to this mode of training (9). Similar investigations have confirmed these age specific results (213).

Jump-training appears to be a viable means of maintaining or increasing bone mass in young women and possibly postmenopausal women. Research needs to be expanded to include males. A single study compared young men and women combined both resistance-training and plyometric jump-training. Following nine weeks of training, serum OC concentration increased 45% and 27% in men and women compared to baseline, respectively. The authors reported significant increases in BMC, but not BMD (78). The separate effects of resistance-training and jump-training can not be elucidated from this study design.

TREATMENT OPTIONS

In general, men are not prescribed pharmacological interventions for osteoporosis both due to a lack of screening for osteoporosis and a lack of adequate treatment following an osteoporotic fracture (62, 117). The most common prevention and treatment strategy in men is dietary supplementation with calcium and vitamin D (117). Testosterone has proven to be ineffective and has serious side effects such as increased risk of prostate cancer and cardiovascular disease (66). Estrogen replacement therapy is commonly used in postmenopausal women to prevent bone loss and has been proven effective since the 1970s (31, 130). Unfortunately, this treatment is not viable in men and in women carries an increased risk of venous thrombosis, pulmonary embolism, stroke, myocardial infarction, and breast cancer (129). Many new pharmacological treatments are available for those suffering from osteopenia and osteoporosis including calcitonin, bisphosphonates, and PTH.

Calcitonin. Calcitonin is a peptide secreted by the thyroid gland that is effective in the treatment for osteoporosis when administered as a pharmacological agent (35). Salmon calcitonin has become the most widely used form due to its high potency and is available in both injectable and nasal spray forms (37). Calcitonin binds to the osteoclast membrane receptors and initiates the process of osteoclast withdrawal from the local bone remodeling sites (37). In addition, calcitonin may stimulate the osteoblasts and inhibit the signaling function of the osteocytes (55).

In a large cohort of postmenopausal women enrolled in the Prevent Recurrence of Osteoporotic Fractures (PROOF) Study, Chesnut et al. (38) reported that treatment with 200 IU per day of salmon calcitonin nasal spray reduced the risk of new vertebral fractures by 33%, although BMD only improved by one to two percent (38). Toth et al. reported that men suffering from idiopathic osteoporosis treated with nasal spray salmon calcitonin improved lumbar spine BMD by 3.5% and femoral neck BMD by 3.2%, both significantly greater than placebo treated controls (218). Calcitonin is effective at treating osteoporosis in both men and women and is commonly used to treat osteoporosis associated with Paget's disease.

Bisphosphonates. Bisphosphonates have been reported to improve BMD and reduce fracture risk in posmenopausal women (49, 128, 145) and men (163, 184). Bisphosphonates reduce overall bone turnover allowing more time for the BMU to induce greater mineralization (17). Many bisophosphonates are currently available including etidronate, clodronate, tiludronate, pamidronate, neridronate, olpadronate, alendronate, ibandronate, risedronate, and zoledronate. Currently, only alendronate, risedronate, and

ibandronate are approved by the U.S. Food and Drug Administration (FDA) for the prevention and treatment of osteoporosis (17, 47).

In men, alendronate has been reported to increase BMD and reduce fracture risk (163, 184). Bisphosphonates have been reported to protect against bone loss induce by glucocorticoid treatment in men (41, 175). Orwoll et al. (163) reported a 7.1% increase in BMD of the lumbar spine, 2.5% increase in BMD of the femoral neck, and a 2.0% increase in total body BMD from baseline in men, all of which were significantly greater than the placebo group ($p<0.001$) (163). Of ultimate importance regarding the osteoporosis-treatment drugs, these authors reported significantly fewer vertebral fractures in the alendronate treatment group than placebo group ($p=0.02$) (163). Alendronate has been generally well tolerated by the men in these studies (184). Alendronate appears to be a beneficial treatment option in males. As with many pharmacological treatments, side effects and patient tolerance are extremely important and varied. Osteonecrosis of the jaw and atrial fibrillation occurring in patients being treated with bisphosphonates have been reported but are highly disputed (90, 203).

Parathyroid Hormone. Use of parathyroid hormone (PTH) as a treatment for osteoporosis is paradoxical because one function of PTH is to enhance bone resorption. Individuals with primary hyperparathyroidism present clinically with slight reductions in cancellous bone mass (i.e., spine), large loss of bone mass in the primary cortical skeleton (i.e., forearm), and intermediate bone loss of the hip, likely due to the hip's composition of both cancellous and cortical bone (for a review see ref 192). Most treatments for osteoporosis, such as estrogen, bisphosphonates, and calcitonin, are anti-resorptive and prevent osteoclast-induced bone loss. In contrast, PTH may have a true anabolic affect

by activating bone formation through the osteoblasts while concurrently inhibiting osteoclast activity (191).

Daily, intermittent administration of PTH peptide 1-34 (145) and PTH(1-84) can reduce vertebral fracture risk, and possibly non-vertebral fracture risk, in postmenopausal women (42). In men, an early study by Slovik et al. (206) in osteopenic men found that injections of human PTH fragment and oral ingestion of 1,25-dihydroxyvitamin D significantly improved spinal BMD assessed by quantitative computed tomography (QCT) (206). Larger clinical trials have verified the treatment efficacy of PTH. In a randomized, double-blind, placebo-controlled trial of 23 osteoporotic men aged 30 to 68 years, Kurland et al. (120) treated the men with PTH(1-34). After 18 months, lumbar spine BMD increased 13.5% ($p<0.001$) and femoral neck BMD increased 2.9% ($p<0.05$) from baseline compared to placebo administered controls (120). Because of the high cost and required daily subcutaneous injections, PTH treatment is often reserved for the most severe cases of osteoporosis (115).

CONCLUSION

Osteoporosis is a disease of bone loss induced by secondary means or of idiopathic nature. Twenty-percent of all osteoporosis-related hip fractures occur in men. Despite this, pharmacological treatments in men are underutilized. Bone remodeling is under controlled by a variety of factors with hormones garnering the greatest attention. A few of the hormones that are known to influence bone mass include sex steroids, glucocorticoids, calcitonin, and PTH. Exercise has been touted as a means to slow bone loss during aging or therapy. Dynamic, weight-bearing exercise of an intensity great enough to induce bone formation is necessary. Modes of exercise that are appropriate

include running, resistance training, and jump training. Non-weight bearing exercise (i.e., cycling, swimming, and walking) likely will not induce bone formation. The first line of treatment for osteoporosis includes vitamin D and calcium supplementation in addition to a pharmacological intervention. Appropriate prevention strategies include calcium and vitamin D supplementation along with a long-term exercise program that is dynamic and weight-bearing in nature.

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APPENDIX C

PAIRWISE COMPARISONS

Pairwise Comparisons

Measure:TRAP

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.009	.010	.411	-.031	.014
	3	.027*	.011	.032	.003	.052
	4	.011	.013	.436	-.019	.041
	5	-.006	.012	.596	-.032	.020
	6	-.008	.014	.570	-.038	.022
	7	.002	.015	.886	-.030	.034
2	1	.009	.010	.411	-.014	.031
	3	.036*	.012	.010	.010	.062
	4	.020	.010	.073	-.002	.041
	5	.002	.009	.810	-.017	.022
	6	.001	.010	.946	-.021	.023
	7	.011	.011	.345	-.013	.035
3	1	-.027*	.011	.032	-.052	-.003
	2	-.036*	.012	.010	-.062	-.010
	4	-.016	.011	.179	-.042	.009
	5	-.034*	.011	.012	-.059	-.009
	6	-.035	.016	.053	-.071	.001
	7	-.025	.018	.178	-.064	.013
4	1	-.011	.013	.436	-.041	.019
	2	-.020	.010	.073	-.041	.002
	3	.016	.011	.179	-.009	.042
	5	-.017	.010	.095	-.038	.004
	6	-.019	.013	.182	-.048	.010
	7	-.009	.013	.515	-.037	.020
5	1	.006	.012	.596	-.020	.032
	2	-.002	.009	.810	-.022	.017
	3	.034*	.011	.012	.009	.059
	4	.017	.010	.095	-.004	.038
	6	-.002	.008	.862	-.020	.017
	7	.009	.011	.440	-.015	.032
6	1	.008	.014	.570	-.022	.038
	2	-.001	.010	.946	-.023	.021
	3	.035	.016	.053	-.001	.071
	4	.019	.013	.182	-.010	.048
	5	.002	.008	.862	-.017	.020
	7	.010	.006	.102	-.002	.023
7	1	-.002	.015	.886	-.034	.030
	2	-.011	.011	.345	-.035	.013
	3	.025	.018	.178	-.013	.064
	4	.009	.013	.515	-.020	.037
	5	-.009	.011	.440	-.032	.015
	6	-.010	.006	.102	-.023	.002

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 1: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in tartrate-resistant acid phosphatase 5b during PLY Fasted (n=12). Main effect for time p=0.038.

Pairwise Comparisons

Measure:CORTISOL

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.128*	.031	.002	.059	.197
	3	.183*	.031	.000	.114	.251
	4	.250*	.036	.000	.171	.329
	5	.270*	.039	.000	.184	.356
	6	.271*	.053	.000	.155	.387
	7	.021	.021	.349	-.026	.067
2	1	-.128*	.031	.002	-.197	-.059
	3	.055*	.011	.000	.030	.080
	4	.122*	.018	.000	.083	.161
	5	.142*	.025	.000	.088	.196
	6	.143*	.050	.015	.034	.253
	7	-.107*	.032	.007	-.179	-.036
3	1	-.183*	.031	.000	-.251	-.114
	2	-.055*	.011	.000	-.080	-.030
	4	.067*	.015	.001	.033	.101
	5	.087*	.021	.002	.041	.134
	6	.089	.050	.106	-.022	.199
	7	-.162*	.034	.001	-.238	-.086
4	1	-.250*	.036	.000	-.329	-.171
	2	-.122*	.018	.000	-.161	-.083
	3	-.067*	.015	.001	-.101	-.033
	5	.020	.015	.203	-.013	.053
	6	.021	.048	.665	-.085	.128
	7	-.229*	.037	.000	-.310	-.148
5	1	-.270*	.039	.000	-.356	-.184
	2	-.142*	.025	.000	-.196	-.088
	3	-.087*	.021	.002	-.134	-.041
	4	-.020	.015	.203	-.053	.013
	6	.001	.040	.979	-.087	.089
	7	-.249*	.040	.000	-.338	-.161
6	1	-.271*	.053	.000	-.387	-.155
	2	-.143*	.050	.015	-.253	-.034
	3	-.089	.050	.106	-.199	.022
	4	-.021	.048	.665	-.128	.085
	5	-.001	.040	.979	-.089	.087
	7	-.251*	.051	.000	-.363	-.138
7	1	-.021	.021	.349	-.067	.026
	2	.107*	.032	.007	.036	.179
	3	.162*	.034	.001	.086	.238
	4	.229*	.037	.000	.148	.310
	5	.249*	.040	.000	.161	.338
	6	.251*	.051	.000	.138	.363

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 2: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in cortisol during PLY Fasted (n=12). Main effect for time p<0.001.

Pairwise Comparisons

Measure:PTH

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.013	.043	.762	-.081	.108
	3	.114*	.033	.006	.041	.187
	4	.198*	.056	.004	.076	.321
	5	.175*	.060	.015	.042	.308
	6	.202*	.049	.002	.095	.309
	7	.041	.038	.306	-.043	.124
2	1	-.013	.043	.762	-.108	.081
	3	.101*	.027	.003	.042	.160
	4	.185*	.041	.001	.094	.276
	5	.162*	.072	.047	.003	.320
	6	.189*	.046	.002	.088	.289
	7	.027	.030	.389	-.040	.094
3	1	-.114*	.033	.006	-.187	-.041
	2	-.101*	.027	.003	-.160	-.042
	4	.084*	.030	.017	.018	.150
	5	.061	.054	.281	-.057	.179
	6	.088*	.036	.033	.009	.167
	7	-.073	.037	.073	-.155	.008
4	1	-.198*	.056	.004	-.321	-.076
	2	-.185*	.041	.001	-.276	-.094
	3	-.084*	.030	.017	-.150	-.018
	5	-.023	.069	.739	-.174	.127
	6	.004	.034	.919	-.072	.079
	7	-.158*	.053	.013	-.275	-.040
5	1	-.175*	.060	.015	-.308	-.042
	2	-.162*	.072	.047	-.320	-.003
	3	-.061	.054	.281	-.179	.057
	4	.023	.069	.739	-.127	.174
	6	.027	.054	.629	-.092	.146
	7	-.134	.072	.090	-.293	.025
6	1	-.202*	.049	.002	-.309	-.095
	2	-.189*	.046	.002	-.289	-.088
	3	-.088*	.036	.033	-.167	-.009
	4	-.004	.034	.919	-.079	.072
	5	-.027	.054	.629	-.146	.092
	7	-.161*	.046	.005	-.262	-.061
7	1	-.041	.038	.306	-.124	.043
	2	-.027	.030	.389	-.094	.040
	3	.073	.037	.073	-.008	.155
	4	.158*	.053	.013	.040	.275
	5	.134	.072	.090	-.025	.293
	6	.161*	.046	.005	.061	.262

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 3: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in parathyroid hormone during PLY Fasted (n=12). Main effect for time $p<0.001$.

Pairwise Comparisons

Measure:TEST

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.026	.033	.448	-.047	.099
	3	.058*	.024	.036	.005	.111
	4	.064	.033	.077	-.008	.136
	5	.072	.047	.156	-.032	.175
	6	.119	.057	.061	-.006	.245
	7	-.020	.045	.666	-.118	.078
2	1	-.026	.033	.448	-.099	.047
	3	.032	.020	.147	-.013	.076
	4	.038	.020	.087	-.006	.082
	5	.045	.025	.093	-.009	.100
	6	.093*	.032	.015	.022	.164
	7	-.046	.033	.192	-.119	.027
3	1	-.058*	.024	.036	-.111	-.005
	2	-.032	.020	.147	-.076	.013
	4	.006	.017	.721	-.031	.044
	5	.014	.030	.658	-.053	.080
	6	.062	.043	.179	-.033	.156
	7	-.078*	.033	.039	-.151	-.004
4	1	-.064	.033	.077	-.136	.008
	2	-.038	.020	.087	-.082	.006
	3	-.006	.017	.721	-.044	.031
	5	.008	.031	.813	-.061	.076
	6	.055	.036	.153	-.024	.134
	7	-.084*	.033	.028	-.157	-.011
5	1	-.072	.047	.156	-.175	.032
	2	-.045	.025	.093	-.100	.009
	3	-.014	.030	.658	-.080	.053
	4	-.008	.031	.813	-.076	.061
	6	.048	.046	.323	-.054	.149
	7	-.091*	.030	.011	-.157	-.026
6	1	-.119	.057	.061	-.245	.006
	2	-.093*	.032	.015	-.164	-.022
	3	-.062	.043	.179	-.156	.033
	4	-.055	.036	.153	-.134	.024
	5	-.048	.046	.323	-.149	.054
	7	-.139*	.048	.014	-.244	-.035
7	1	.020	.045	.666	-.078	.118
	2	.046	.033	.192	-.027	.119
	3	.078*	.033	.039	.004	.151
	4	.084*	.033	.028	.011	.157
	5	.091*	.030	.011	.026	.157
	6	.139*	.048	.014	.035	.244

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 4: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in testosterone during PLY Fasted (n=12). Main effect for time p<0.001.

Pairwise Comparisons

Measure:TRAP

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.011	.010	.315	-.013	.035
	3	.024	.011	.060	-.001	.049
	4	.031*	.009	.010	.010	.052
	5	.010	.014	.513	-.023	.043
	6	-.014	.017	.420	-.053	.024
	7	-.002	.010	.862	-.026	.022
2	1	-.011	.010	.315	-.035	.013
	3	.013	.013	.347	-.016	.042
	4	.020*	.009	.049	.000	.040
	5	-.001	.011	.901	-.027	.024
	6	-.025	.017	.176	-.065	.014
	7	-.013	.019	.517	-.057	.031
3	1	-.024	.011	.060	-.049	.001
	2	-.013	.013	.347	-.042	.016
	4	.007	.007	.351	-.010	.024
	5	-.014	.011	.254	-.040	.012
	6	-.038*	.016	.047	-.075	-.001
	7	-.026	.017	.182	-.066	.015
4	1	-.031*	.009	.010	-.052	-.010
	2	-.020*	.009	.049	-.040	.000
	3	-.007	.007	.351	-.024	.010
	5	-.021*	.008	.022	-.039	-.004
	6	-.045*	.014	.011	-.077	-.014
	7	-.033	.018	.099	-.074	.008
5	1	-.010	.014	.513	-.043	.023
	2	.001	.011	.901	-.024	.027
	3	.014	.011	.254	-.012	.040
	4	.021*	.008	.022	.004	.039
	6	-.024	.011	.067	-.050	.002
	7	-.012	.021	.600	-.061	.037
6	1	.014	.017	.420	-.024	.053
	2	.025	.017	.176	-.014	.065
	3	.038*	.016	.047	.001	.075
	4	.045*	.014	.011	.014	.077
	5	.024	.011	.067	-.002	.050
	7	.012	.022	.582	-.037	.062
7	1	.002	.010	.862	-.022	.026
	2	.013	.019	.517	-.031	.057
	3	.026	.017	.182	-.015	.066
	4	.033	.018	.099	-.008	.074
	5	.012	.021	.600	-.037	.061
	6	-.012	.022	.582	-.062	.037

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 5: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in tartrate-resistant acid phosphatase during RT Fasted (n=12). Main effect for time p=0.044.

Pairwise Comparisons

Measure:CORTISOL

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.024	.066	.724	-.127	.175
	3	.083	.062	.218	-.060	.225
	4	.110	.073	.169	-.058	.279
	5	.240*	.058	.003	.106	.374
	6	.257*	.056	.002	.127	.387
	7	-.011	.021	.624	-.059	.038
2	1	-.024	.066	.724	-.175	.127
	3	.059*	.013	.002	.028	.089
	4	.086*	.015	.000	.051	.122
	5	.216*	.017	.000	.175	.256
	6	.233*	.035	.000	.152	.314
	7	-.035	.057	.563	-.167	.098
3	1	-.083	.062	.218	-.225	.060
	2	-.059*	.013	.002	-.089	-.028
	4	.028	.019	.174	-.015	.071
	5	.157*	.019	.000	.114	.201
	6	.175*	.036	.001	.092	.258
	7	-.093	.055	.128	-.220	.033
4	1	-.110	.073	.169	-.279	.058
	2	-.086*	.015	.000	-.122	-.051
	3	-.028	.019	.174	-.071	.015
	5	.129*	.025	.001	.072	.186
	6	.147*	.038	.005	.059	.234
	7	-.121	.064	.093	-.268	.025
5	1	-.240*	.058	.003	-.374	-.106
	2	-.216*	.017	.000	-.256	-.175
	3	-.157*	.019	.000	-.201	-.114
	4	-.129*	.025	.001	-.186	-.072
	6	.017	.038	.660	-.071	.105
	7	-.250*	.047	.001	-.359	-.142
6	1	-.257*	.056	.002	-.387	-.127
	2	-.233*	.035	.000	-.314	-.152
	3	-.175*	.036	.001	-.258	-.092
	4	-.147*	.038	.005	-.234	-.059
	5	-.017	.038	.660	-.105	.071
	7	-.268*	.053	.001	-.391	-.145
7	1	.011	.021	.624	-.038	.059
	2	.035	.057	.563	-.098	.167
	3	.093	.055	.128	-.033	.220
	4	.121	.064	.093	-.025	.268
	5	.250*	.047	.001	.142	.359
	6	.268*	.053	.001	.145	.391

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 6: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in cortisol during RT Fasted (n=12). Main effect for time $p<0.001$.

Pairwise Comparisons

Measure:PTH

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.045	.108	.690	-.293	.204
	3	.116	.141	.436	-.210	.441
	4	.157	.132	.268	-.147	.462
	5	.110	.145	.468	-.224	.445
	6	.066	.105	.547	-.176	.309
	7	-.126	.098	.233	-.351	.099
2	1	.045	.108	.690	-.204	.293
	3	.160*	.048	.010	.050	.271
	4	.202*	.043	.002	.103	.301
	5	.155*	.055	.022	.029	.281
	6	.111*	.042	.030	.014	.208
	7	-.081	.042	.087	-.178	.015
3	1	-.116	.141	.436	-.441	.210
	2	-.160*	.048	.010	-.271	-.050
	4	.041*	.018	.049	.000	.082
	5	-.005	.045	.908	-.109	.098
	6	-.050	.051	.359	-.167	.068
	7	-.242*	.069	.008	-.401	-.083
4	1	-.157	.132	.268	-.462	.147
	2	-.202*	.043	.002	-.301	-.103
	3	-.041*	.018	.049	-.082	.000
	5	-.047	.038	.254	-.134	.041
	6	-.091	.040	.050	-.182	.000
	7	-.283*	.062	.002	-.426	-.140
5	1	-.110	.145	.468	-.445	.224
	2	-.155*	.055	.022	-.281	-.029
	3	.005	.045	.908	-.098	.109
	4	.047	.038	.254	-.041	.134
	6	-.044	.046	.360	-.149	.061
	7	-.236*	.062	.005	-.380	-.092
6	1	-.066	.105	.547	-.309	.176
	2	-.111*	.042	.030	-.208	-.014
	3	.050	.051	.359	-.068	.167
	4	.091	.040	.050	.000	.182
	5	.044	.046	.360	-.061	.149
	7	-.192*	.035	.001	-.272	-.112
7	1	.126	.098	.233	-.099	.351
	2	.081	.042	.087	-.015	.178
	3	.242*	.069	.008	.083	.401
	4	.283*	.062	.002	.140	.426
	5	.236*	.062	.005	.092	.380
	6	.192*	.035	.001	.112	.272

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 7: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in parathyroid hormone during RT Fasted (n=12). Main effect for time p=0.007.

Pairwise Comparisons

Measure:TRAP

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.003	.011	.759	-.020	.027
	3	.031*	.010	.009	.010	.053
	4	.032*	.013	.029	.004	.060
	5	.022	.012	.090	-.004	.048
	6	.003	.011	.793	-.021	.027
	7	.001	.015	.942	-.032	.034
2	1	-.003	.011	.759	-.027	.020
	3	.028*	.010	.014	.007	.049
	4	.029*	.012	.034	.003	.055
	5	.019	.015	.233	-.014	.051
	6	.000	.011	.971	-.025	.024
	7	-.002	.012	.857	-.029	.025
3	1	-.031*	.010	.009	-.053	-.010
	2	-.028*	.010	.014	-.049	-.007
	4	.001	.008	.926	-.017	.018
	5	-.009	.010	.392	-.032	.014
	6	-.029*	.008	.005	-.047	-.010
	7	-.030	.014	.055	-.062	.001
4	1	-.032*	.013	.029	-.060	-.004
	2	-.029*	.012	.034	-.055	-.003
	3	-.001	.008	.926	-.018	.017
	5	-.010	.008	.216	-.027	.007
	6	-.029*	.009	.006	-.048	-.010
	7	-.031	.015	.056	-.063	.001
5	1	-.022	.012	.090	-.048	.004
	2	-.019	.015	.233	-.051	.014
	3	.009	.010	.392	-.014	.032
	4	.010	.008	.216	-.007	.027
	6	-.019	.010	.086	-.042	.003
	7	-.021	.019	.303	-.064	.022
6	1	-.003	.011	.793	-.027	.021
	2	.000	.011	.971	-.024	.025
	3	.029*	.008	.005	.010	.047
	4	.029*	.009	.006	.010	.048
	5	.019	.010	.086	-.003	.042
	7	-.002	.016	.909	-.037	.033
7	1	-.001	.015	.942	-.034	.032
	2	.002	.012	.857	-.025	.029
	3	.030	.014	.055	-.001	.062
	4	.031	.015	.056	-.001	.063
	5	.021	.019	.303	-.022	.064
	6	.002	.016	.909	-.033	.037

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 8: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in tartrate-resistant acid phosphatase during PLY Fed (n=12). Main effect for time p=0.014.

Pairwise Comparisons

Measure:CORTISOL

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.072	.042	.112	-.020	.164
	3	.103*	.040	.024	.016	.191
	4	.220*	.056	.002	.097	.342
	5	.224*	.045	.000	.124	.323
	6	.163*	.030	.000	.097	.229
	7	-.073*	.028	.026	-.136	-.010
2	1	-.072	.042	.112	-.164	.020
	3	.031*	.010	.012	.008	.054
	4	.147	.070	.057	-.006	.301
	5	.152*	.022	.000	.103	.200
	6	.091*	.039	.040	.005	.177
	7	-.145*	.036	.002	-.225	-.065
3	1	-.103*	.040	.024	-.191	-.016
	2	-.031*	.010	.012	-.054	-.008
	4	.116	.065	.100	-.026	.259
	5	.120*	.018	.000	.080	.161
	6	.060	.041	.170	-.030	.149
	7	-.176*	.038	.001	-.260	-.093
4	1	-.220*	.056	.002	-.342	-.097
	2	-.147	.070	.057	-.301	.006
	3	-.116	.065	.100	-.259	.026
	5	.004	.062	.949	-.133	.141
	6	-.057	.072	.448	-.215	.102
	7	-.293*	.068	.001	-.442	-.143
5	1	-.224*	.045	.000	-.323	-.124
	2	-.152*	.022	.000	-.200	-.103
	3	-.120*	.018	.000	-.161	-.080
	4	-.004	.062	.949	-.141	.133
	6	-.061	.043	.186	-.156	.034
	7	-.297*	.040	.000	-.386	-.208
6	1	-.163*	.030	.000	-.229	-.097
	2	-.091*	.039	.040	-.177	-.005
	3	-.060	.041	.170	-.149	.030
	4	.057	.072	.448	-.102	.215
	5	.061	.043	.186	-.034	.156
	7	-.236*	.033	.000	-.309	-.163
7	1	.073*	.028	.026	.010	.136
	2	.145*	.036	.002	.065	.225
	3	.176*	.038	.001	.093	.260
	4	.293*	.068	.001	.143	.442
	5	.297*	.040	.000	.208	.386
	6	.236*	.033	.000	.163	.309

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 9: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in cortisol during PLY Fed (n=12). Main effect for time p<0.001.

Pairwise Comparisons

Measure:PTH

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.105	.064	.126	-.245	.035
	3	.009	.053	.869	-.108	.126
	4	.110	.051	.054	-.002	.222
	5	.109*	.046	.039	.006	.211
	6	.029	.042	.502	-.064	.123
	7	-.142*	.048	.013	-.248	-.036
2	1	.105	.064	.126	-.035	.245
	3	.114*	.020	.000	.071	.158
	4	.215*	.033	.000	.144	.287
	5	.214*	.052	.002	.100	.328
	6	.135*	.042	.008	.043	.227
	7	-.037	.040	.378	-.125	.051
3	1	-.009	.053	.869	-.126	.108
	2	-.114*	.020	.000	-.158	-.071
	4	.101*	.016	.000	.065	.137
	5	.100*	.038	.024	.016	.183
	6	.020	.030	.506	-.045	.086
	7	-.151*	.035	.001	-.228	-.075
4	1	-.110	.051	.054	-.222	.002
	2	-.215*	.033	.000	-.287	-.144
	3	-.101*	.016	.000	-.137	-.065
	5	-.001	.033	.969	-.074	.072
	6	-.080*	.020	.002	-.124	-.036
	7	-.252*	.039	.000	-.338	-.166
5	1	-.109*	.046	.039	-.211	-.006
	2	-.214*	.052	.002	-.328	-.100
	3	-.100*	.038	.024	-.183	-.016
	4	.001	.033	.969	-.072	.074
	6	-.079	.038	.063	-.163	.005
	7	-.251*	.042	.000	-.343	-.159
6	1	-.029	.042	.502	-.123	.064
	2	-.135*	.042	.008	-.227	-.043
	3	-.020	.030	.506	-.086	.045
	4	.080*	.020	.002	.036	.124
	5	.079	.038	.063	-.005	.163
	7	-.172*	.042	.002	-.264	-.079
7	1	.142*	.048	.013	.036	.248
	2	.037	.040	.378	-.051	.125
	3	.151*	.035	.001	.075	.228
	4	.252*	.039	.000	.166	.338
	5	.251*	.042	.000	.159	.343
	6	.172*	.042	.002	.079	.264

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 10: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in parathyroid hormone during PLY Fed (n=12). Main effect for time $p<0.001$.

Pairwise Comparisons

Measure:TEST

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.028	.027	.324	-.087	.031
	3	-.031	.033	.366	-.105	.042
	4	.057	.031	.094	-.012	.126
	5	.049	.034	.179	-.026	.124
	6	.014	.035	.691	-.063	.092
	7	-.060*	.018	.007	-.101	-.020
2	1	.028	.027	.324	-.031	.087
	3	-.004	.029	.896	-.067	.059
	4	.085*	.017	.001	.046	.123
	5	.076	.037	.060	-.004	.157
	6	.042	.038	.290	-.041	.125
	7	-.033	.033	.350	-.106	.041
3	1	.031	.033	.366	-.042	.105
	2	.004	.029	.896	-.059	.067
	4	.089*	.029	.010	.025	.152
	5	.080*	.031	.025	.012	.148
	6	.046	.036	.223	-.032	.124
	7	-.029	.037	.451	-.110	.052
4	1	-.057	.031	.094	-.126	.012
	2	-.085*	.017	.001	-.123	-.046
	3	-.089*	.029	.010	-.152	-.025
	5	-.008	.035	.817	-.085	.068
	6	-.043	.037	.277	-.125	.040
	7	-.117*	.037	.009	-.199	-.036
5	1	-.049	.034	.179	-.124	.026
	2	-.076	.037	.060	-.157	.004
	3	-.080*	.031	.025	-.148	-.012
	4	.008	.035	.817	-.068	.085
	6	-.034	.022	.146	-.083	.014
	7	-.109*	.043	.028	-.204	-.014
6	1	-.014	.035	.691	-.092	.063
	2	-.042	.038	.290	-.125	.041
	3	-.046	.036	.223	-.124	.032
	4	.043	.037	.277	-.040	.125
	5	.034	.022	.146	-.014	.083
	7	-.075	.037	.070	-.156	.007
7	1	.060*	.018	.007	.020	.101
	2	.033	.033	.350	-.041	.106
	3	.029	.037	.451	-.052	.110
	4	.117*	.037	.009	.036	.199
	5	.109*	.043	.028	.014	.204
	6	.075	.037	.070	-.007	.156

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 11: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in testosterone during PLY Fed (n=12). Main effect for time p=0.005.

Pairwise Comparisons

Measure:TRAP

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.021	.011	.073	-.002	.044
	3	.044*	.014	.010	.013	.076
	4	.039*	.011	.005	.014	.063
	5	.034	.021	.137	-.013	.081
	6	.007	.015	.643	-.025	.039
	7	-.016	.016	.363	-.052	.021
2	1	-.021	.011	.073	-.044	.002
	3	.023*	.008	.011	.007	.040
	4	.018	.011	.133	-.006	.042
	5	.013	.021	.534	-.032	.058
	6	-.014	.009	.158	-.034	.006
	7	-.036*	.014	.023	-.067	-.006
3	1	-.044*	.014	.010	-.076	-.013
	2	-.023*	.008	.011	-.040	-.007
	4	-.006	.010	.593	-.028	.017
	5	-.010	.021	.632	-.056	.036
	6	-.037*	.011	.005	-.061	-.014
	7	-.060*	.014	.001	-.090	-.030
4	1	-.039*	.011	.005	-.063	-.014
	2	-.018	.011	.133	-.042	.006
	3	.006	.010	.593	-.017	.028
	5	-.005	.014	.744	-.035	.026
	6	-.032*	.012	.020	-.057	-.006
	7	-.054*	.015	.004	-.088	-.021
5	1	-.034	.021	.137	-.081	.013
	2	-.013	.021	.534	-.058	.032
	3	.010	.021	.632	-.036	.056
	4	.005	.014	.744	-.026	.035
	6	-.027	.020	.201	-.071	.017
	7	-.050	.026	.079	-.106	.007
6	1	-.007	.015	.643	-.039	.025
	2	.014	.009	.158	-.006	.034
	3	.037*	.011	.005	.014	.061
	4	.032*	.012	.020	.006	.057
	5	.027	.020	.201	-.017	.071
	7	-.023*	.009	.025	-.042	-.003
7	1	.016	.016	.363	-.021	.052
	2	.036*	.014	.023	.006	.067
	3	.060*	.014	.001	.030	.090
	4	.054*	.015	.004	.021	.088
	5	.050	.026	.079	-.007	.106
	6	.023*	.009	.025	.003	.042

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 12: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in tartrate-resistant acid phosphatase during RT Fed (n=12). Main effect for time p=0.001.

Pairwise Comparisons

Measure:CORTISOL

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.007	.057	.902	-.134	.119
	3	.054	.047	.284	-.052	.160
	4	.095	.049	.081	-.014	.204
	5	.140*	.060	.041	.007	.273
	6	.131*	.039	.007	.044	.218
	7	-.095*	.019	.001	-.138	-.053
2	1	.007	.057	.902	-.119	.134
	3	.061*	.016	.004	.024	.098
	4	.102*	.022	.001	.053	.151
	5	.147*	.034	.001	.072	.222
	6	.138*	.055	.031	.015	.260
	7	-.088	.068	.225	-.240	.064
3	1	-.054	.047	.284	-.160	.052
	2	-.061*	.016	.004	-.098	-.024
	4	.041*	.011	.003	.017	.065
	5	.086*	.028	.013	.023	.149
	6	.077	.048	.138	-.029	.183
	7	-.149*	.058	.028	-.278	-.020
4	1	-.095	.049	.081	-.204	.014
	2	-.102*	.022	.001	-.151	-.053
	3	-.041*	.011	.003	-.065	-.017
	5	.045	.031	.174	-.024	.114
	6	.036	.053	.514	-.082	.154
	7	-.190*	.059	.009	-.322	-.058
5	1	-.140*	.060	.041	-.273	-.007
	2	-.147*	.034	.001	-.222	-.072
	3	-.086*	.028	.013	-.149	-.023
	4	-.045	.031	.174	-.114	.024
	6	-.009	.049	.855	-.118	.100
	7	-.235*	.070	.007	-.390	-.080
6	1	-.131*	.039	.007	-.218	-.044
	2	-.138*	.055	.031	-.260	-.015
	3	-.077	.048	.138	-.183	.029
	4	-.036	.053	.514	-.154	.082
	5	.009	.049	.855	-.100	.118
	7	-.226*	.041	.000	-.318	-.134
7	1	.095*	.019	.001	.053	.138
	2	.088	.068	.225	-.064	.240
	3	.149*	.058	.028	.020	.278
	4	.190*	.059	.009	.058	.322
	5	.235*	.070	.007	.080	.390
	6	.226*	.041	.000	.134	.318

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 13: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in cortisol during RT Fed (n=12). Main effect for time p<0.001.

Pairwise Comparisons

Measure:PTH

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.044	.026	.117	-.013	.100
	3	.187*	.042	.001	.094	.280
	4	.227*	.039	.000	.141	.314
	5	.162*	.039	.002	.077	.247
	6	.167*	.058	.015	.039	.294
	7	-.100	.052	.083	-.215	.016
2	1	-.044	.026	.117	-.100	.013
	3	.143*	.026	.000	.086	.200
	4	.183*	.024	.000	.130	.237
	5	.118*	.027	.001	.058	.178
	6	.123*	.046	.021	.022	.223
	7	-.143*	.052	.018	-.257	-.030
3	1	-.187*	.042	.001	-.280	-.094
	2	-.143*	.026	.000	-.200	-.086
	4	.040	.027	.169	-.020	.100
	5	-.025	.035	.497	-.103	.053
	6	-.021	.037	.593	-.102	.061
	7	-.287*	.059	.000	-.416	-.157
4	1	-.227*	.039	.000	-.314	-.141
	2	-.183*	.024	.000	-.237	-.130
	3	-.040	.027	.169	-.100	.020
	5	-.065*	.019	.005	-.106	-.024
	6	-.061	.032	.087	-.132	.010
	7	-.327*	.053	.000	-.443	-.211
5	1	-.162*	.039	.002	-.247	-.077
	2	-.118*	.027	.001	-.178	-.058
	3	.025	.035	.497	-.053	.103
	4	.065*	.019	.005	.024	.106
	6	.004	.044	.923	-.092	.101
	7	-.262*	.042	.000	-.353	-.170
6	1	-.167*	.058	.015	-.294	-.039
	2	-.123*	.046	.021	-.223	-.022
	3	.021	.037	.593	-.061	.102
	4	.061	.032	.087	-.010	.132
	5	-.004	.044	.923	-.101	.092
	7	-.266*	.061	.001	-.400	-.133
7	1	.100	.052	.083	-.016	.215
	2	.143*	.052	.018	.030	.257
	3	.287*	.059	.000	.157	.416
	4	.327*	.053	.000	.211	.443
	5	.262*	.042	.000	.170	.353
	6	.266*	.061	.001	.133	.400

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 14: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in parathyroid hormone during RT Fed (n=12). Main effect for time $p<0.001$.

Pairwise Comparisons

Measure:TEST

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.052	.037	.186	-.133	.029
	3	-.028	.032	.401	-.099	.043
	4	-.010	.040	.802	-.097	.077
	5	.028	.037	.465	-.053	.108
	6	-.023	.038	.549	-.107	.060
	7	-.106*	.043	.032	-.201	-.011
2	1	.052	.037	.186	-.029	.133
	3	.024	.034	.493	-.051	.099
	4	.042	.032	.214	-.028	.112
	5	.080*	.035	.041	.004	.156
	6	.029	.034	.419	-.046	.104
	7	-.054	.038	.181	-.137	.029
3	1	.028	.032	.401	-.043	.099
	2	-.024	.034	.493	-.099	.051
	4	.018	.029	.547	-.045	.081
	5	.056	.038	.170	-.028	.139
	6	.005	.033	.891	-.068	.077
	7	-.078	.056	.189	-.200	.044
4	1	.010	.040	.802	-.077	.097
	2	-.042	.032	.214	-.112	.028
	3	-.018	.029	.547	-.081	.045
	5	.038	.025	.165	-.018	.094
	6	-.013	.037	.731	-.096	.069
	7	-.096	.054	.101	-.214	.022
5	1	-.028	.037	.465	-.108	.053
	2	-.080*	.035	.041	-.156	-.004
	3	-.056	.038	.170	-.139	.028
	4	-.038	.025	.165	-.094	.018
	6	-.051	.040	.230	-.139	.037
	7	-.134*	.046	.015	-.235	-.032
6	1	.023	.038	.549	-.060	.107
	2	-.029	.034	.419	-.104	.046
	3	-.005	.033	.891	-.077	.068
	4	.013	.037	.731	-.069	.096
	5	.051	.040	.230	-.037	.139
	7	-.082	.049	.122	-.191	.026
7	1	.106*	.043	.032	.011	.201
	2	.054	.038	.181	-.029	.137
	3	.078	.056	.189	-.044	.200
	4	.096	.054	.101	-.022	.214
	5	.134*	.046	.015	.032	.235
	6	.082	.049	.122	-.026	.191

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 15: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in testosterone during RT Fed (n=12). Main effect for time p=0.039.

Pairwise Comparisons

Measure:CORT

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.083	.044	.116	-.029	.196
	3	.154*	.044	.017	.041	.266
	4	.205*	.045	.006	.089	.321
	5	.211*	.041	.003	.107	.315
	6	.192*	.050	.012	.064	.320
	7	-.005	.009	.593	-.029	.018
2	1	-.083	.044	.116	-.196	.029
	3	.070*	.017	.009	.026	.114
	4	.122*	.031	.011	.042	.201
	5	.128*	.036	.017	.035	.220
	6	.109	.065	.156	-.059	.276
	7	-.089	.044	.098	-.201	.023
3	1	-.154*	.044	.017	-.266	-.041
	2	-.070*	.017	.009	-.114	-.026
	4	.051	.025	.100	-.014	.117
	5	.057	.027	.086	-.012	.126
	6	.038	.061	.557	-.118	.195
	7	-.159*	.044	.015	-.272	-.046
4	1	-.205*	.045	.006	-.321	-.089
	2	-.122*	.031	.011	-.201	-.042
	3	-.051	.025	.100	-.117	.014
	5	.006	.014	.678	-.029	.041
	6	-.013	.054	.819	-.151	.125
	7	-.210*	.041	.004	-.315	-.106
5	1	-.211*	.041	.003	-.315	-.107
	2	-.128*	.036	.017	-.220	-.035
	3	-.057	.027	.086	-.126	.012
	4	-.006	.014	.678	-.041	.029
	6	-.019	.055	.742	-.160	.121
	7	-.216*	.037	.002	-.312	-.120
6	1	-.192*	.050	.012	-.320	-.064
	2	-.109	.065	.156	-.276	.059
	3	-.038	.061	.557	-.195	.118
	4	.013	.054	.819	-.125	.151
	5	.019	.055	.742	-.121	.160
	7	-.197*	.045	.007	-.313	-.082
7	1	.005	.009	.593	-.018	.029
	2	.089	.044	.098	-.023	.201
	3	.159*	.044	.015	.046	.272
	4	.210*	.041	.004	.106	.315
	5	.216*	.037	.002	.120	.312
	6	.197*	.045	.007	.082	.313

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 16: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in cortisol during PLY Fasted (n=6). Main effect for time p<0.001.

Pairwise Comparisons

Measure:PTH

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.091*	.026	.017	.025	.157
	3	.154*	.044	.018	.040	.268
	4	.286*	.082	.018	.074	.497
	5	.162	.099	.162	-.092	.416
	6	.266*	.056	.005	.122	.409
	7	.069	.049	.219	-.057	.194
2	1	-.091*	.026	.017	-.157	-.025
	3	.063	.041	.186	-.043	.169
	4	.195*	.076	.050	.000	.389
	5	.071	.109	.541	-.208	.350
	6	.175*	.063	.040	.012	.338
	7	-.022	.040	.607	-.126	.082
3	1	-.154*	.044	.018	-.268	-.040
	2	-.063	.041	.186	-.169	.043
	4	.132*	.050	.045	.004	.260
	5	.008	.082	.924	-.204	.220
	6	.112	.047	.065	-.010	.234
	7	-.085	.068	.265	-.259	.089
4	1	-.286*	.082	.018	-.497	-.074
	2	-.195*	.076	.050	-.389	.000
	3	-.132*	.050	.045	-.260	-.004
	5	-.124	.117	.340	-.425	.178
	6	-.020	.058	.742	-.168	.128
	7	-.217	.095	.070	-.460	.026
5	1	-.162	.099	.162	-.416	.092
	2	-.071	.109	.541	-.350	.208
	3	-.008	.082	.924	-.220	.204
	4	.124	.117	.340	-.178	.425
	6	.104	.099	.345	-.152	.359
	7	-.093	.123	.484	-.411	.224
6	1	-.266*	.056	.005	-.409	-.122
	2	-.175*	.063	.040	-.338	-.012
	3	-.112	.047	.065	-.234	.010
	4	.020	.058	.742	-.128	.168
	5	-.104	.099	.345	-.359	.152
	7	-.197*	.068	.034	-.371	-.023
7	1	-.069	.049	.219	-.194	.057
	2	.022	.040	.607	-.082	.126
	3	.085	.068	.265	-.089	.259
	4	.217	.095	.070	-.026	.460
	5	.093	.123	.484	-.224	.411
	6	.197*	.068	.034	.023	.371

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 17: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in parathyroid hormone during PLY Fasted (n=6). Main effect for time p=0.007.

Pairwise Comparisons

Measure:CORTISOL

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.032	.124	.808	-.313	.377
	3	.075	.117	.555	-.249	.399
	4	.094	.137	.530	-.286	.474
	5	.243	.110	.092	-.063	.549
	6	.224	.098	.084	-.047	.495
	7	-.007	.038	.855	-.113	.098
2	1	-.032	.124	.808	-.377	.313
	3	.043	.021	.115	-.016	.102
	4	.062*	.013	.009	.025	.098
	5	.211*	.032	.003	.123	.299
	6	.192*	.049	.017	.057	.326
	7	-.040	.108	.733	-.340	.261
3	1	-.075	.117	.555	-.399	.249
	2	-.043	.021	.115	-.102	.016
	4	.019	.030	.562	-.064	.102
	5	.168*	.032	.006	.079	.257
	6	.149	.058	.063	-.013	.311
	7	-.082	.103	.467	-.368	.203
4	1	-.094	.137	.530	-.474	.286
	2	-.062*	.013	.009	-.098	-.025
	3	-.019	.030	.562	-.102	.064
	5	.149*	.039	.018	.042	.256
	6	.130	.059	.090	-.032	.293
	7	-.101	.119	.444	-.433	.230
5	1	-.243	.110	.092	-.549	.063
	2	-.211*	.032	.003	-.299	-.123
	3	-.168*	.032	.006	-.257	-.079
	4	-.149*	.039	.018	-.256	-.042
	6	-.019	.055	.745	-.172	.133
	7	-.250*	.087	.046	-.493	-.008
6	1	-.224	.098	.084	-.495	.047
	2	-.192*	.049	.017	-.326	-.057
	3	-.149	.058	.063	-.311	.013
	4	-.130	.059	.090	-.293	.032
	5	.019	.055	.745	-.133	.172
	7	-.231	.087	.057	-.474	.011
7	1	.007	.038	.855	-.098	.113
	2	.040	.108	.733	-.261	.340
	3	.082	.103	.467	-.203	.368
	4	.101	.119	.444	-.230	.433
	5	.250*	.087	.046	.008	.493
	6	.231	.087	.057	-.011	.474

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 18: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in cortisol during RT Fasted (n=6). Main effect for time p=0.021.

Pairwise Comparisons

Measure:CORTISOL

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.015	.052	.786	-.118	.148
	3	.036	.044	.448	-.076	.148
	4	.197	.097	.099	-.054	.447
	5	.140*	.028	.004	.067	.213
	6	.137	.054	.051	-.001	.275
	7	-.120*	.035	.018	-.210	-.031
2	1	-.015	.052	.786	-.148	.118
	3	.021	.017	.267	-.022	.065
	4	.182	.144	.262	-.188	.552
	5	.125*	.027	.006	.056	.195
	6	.122	.051	.062	-.009	.253
	7	-.135*	.046	.032	-.253	-.018
3	1	-.036	.044	.448	-.148	.076
	2	-.021	.017	.267	-.065	.022
	4	.161	.132	.277	-.178	.499
	5	.104*	.018	.002	.057	.151
	6	.101	.056	.132	-.043	.245
	7	-.156*	.047	.021	-.278	-.035
4	1	-.197	.097	.099	-.447	.054
	2	-.182	.144	.262	-.552	.188
	3	-.161	.132	.277	-.499	.178
	5	-.057	.121	.659	-.368	.255
	6	-.060	.138	.683	-.416	.296
	7	-.317	.128	.056	-.646	.011
5	1	-.140*	.028	.004	-.213	-.067
	2	-.125*	.027	.006	-.195	-.056
	3	-.104*	.018	.002	-.151	-.057
	4	.057	.121	.659	-.255	.368
	6	-.003	.047	.947	-.124	.118
	7	-.261*	.037	.001	-.355	-.166
6	1	-.137	.054	.051	-.275	.001
	2	-.122	.051	.062	-.253	.009
	3	-.101	.056	.132	-.245	.043
	4	.060	.138	.683	-.296	.416
	5	.003	.047	.947	-.118	.124
	7	-.257*	.049	.003	-.384	-.130
7	1	.120*	.035	.018	.031	.210
	2	.135*	.046	.032	.018	.253
	3	.156*	.047	.021	.035	.278
	4	.317	.128	.056	-.011	.646
	5	.261*	.037	.001	.166	.355
	6	.257*	.049	.003	.130	.384

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 19: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in cortisol during PLY Fed (n=6). Main effect for time p=0.005.

Pairwise Comparisons

Measure:PTH

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.147	.110	.241	-.431	.137
	3	-.039	.085	.665	-.256	.179
	4	.071	.074	.382	-.120	.262
	5	.060	.079	.482	-.143	.264
	6	-.008	.051	.886	-.140	.124
	7	-.236*	.069	.019	-.415	-.058
2	1	.147	.110	.241	-.137	.431
	3	.108*	.034	.025	.020	.196
	4	.218*	.055	.010	.077	.359
	5	.207*	.077	.044	.008	.405
	6	.139	.071	.107	-.043	.321
	7	-.090	.060	.194	-.243	.064
3	1	.039	.085	.665	-.179	.256
	2	-.108*	.034	.025	-.196	-.020
	4	.110*	.027	.010	.040	.180
	5	.099	.049	.097	-.026	.224
	6	.031	.051	.566	-.099	.162
	7	-.197*	.040	.004	-.301	-.094
4	1	-.071	.074	.382	-.262	.120
	2	-.218*	.055	.010	-.359	-.077
	3	-.110*	.027	.010	-.180	-.040
	5	-.011	.035	.760	-.100	.078
	6	-.079	.035	.075	-.170	.012
	7	-.308*	.026	.000	-.375	-.241
5	1	-.060	.079	.482	-.264	.143
	2	-.207*	.077	.044	-.405	-.008
	3	-.099	.049	.097	-.224	.026
	4	.011	.035	.760	-.078	.100
	6	-.068	.055	.274	-.210	.074
	7	-.296*	.038	.001	-.395	-.198
6	1	.008	.051	.886	-.124	.140
	2	-.139	.071	.107	-.321	.043
	3	-.031	.051	.566	-.162	.099
	4	.079	.035	.075	-.012	.170
	5	.068	.055	.274	-.074	.210
	7	-.229*	.027	.000	-.297	-.160
7	1	.236*	.069	.019	.058	.415
	2	.090	.060	.194	-.064	.243
	3	.197*	.040	.004	.094	.301
	4	.308*	.026	.000	.241	.375
	5	.296*	.038	.001	.198	.395
	6	.229*	.027	.000	.160	.297

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 20: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in parathyroid hormone during PLY Fed (n=6). Main effect for time $p<0.001$.

Pairwise Comparisons

Measure:TRAP

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.011	.015	.511	-.028	.049
	3	.039	.017	.067	-.004	.082
	4	.029*	.006	.006	.013	.045
	5	-.003	.011	.801	-.030	.024
	6	-.008	.012	.544	-.038	.023
	7	-.020	.017	.279	-.064	.023
2	1	-.011	.015	.511	-.049	.028
	3	.028*	.004	.001	.019	.038
	4	.018	.012	.175	-.011	.048
	5	-.013	.017	.463	-.057	.030
	6	-.018*	.006	.032	-.035	-.002
	7	-.031	.016	.108	-.072	.010
3	1	-.039	.017	.067	-.082	.004
	2	-.028*	.004	.001	-.038	-.019
	4	-.010	.013	.482	-.044	.024
	5	-.042	.020	.087	-.092	.009
	6	-.047*	.007	.001	-.066	-.028
	7	-.059*	.015	.010	-.098	-.021
4	1	-.029*	.006	.006	-.045	-.013
	2	-.018	.012	.175	-.048	.011
	3	.010	.013	.482	-.024	.044
	5	-.032*	.010	.022	-.057	-.007
	6	-.037*	.009	.008	-.059	-.015
	7	-.049*	.015	.024	-.089	-.010
5	1	.003	.011	.801	-.024	.030
	2	.013	.017	.463	-.030	.057
	3	.042	.020	.087	-.009	.092
	4	.032*	.010	.022	.007	.057
	6	-.005	.013	.726	-.039	.029
	7	-.018	.019	.401	-.067	.032
6	1	.008	.012	.544	-.023	.038
	2	.018*	.006	.032	.002	.035
	3	.047*	.007	.001	.028	.066
	4	.037*	.009	.008	.015	.059
	5	.005	.013	.726	-.029	.039
	7	-.013	.011	.288	-.040	.015
7	1	.020	.017	.279	-.023	.064
	2	.031	.016	.108	-.010	.072
	3	.059*	.015	.010	.021	.098
	4	.049*	.015	.024	.010	.089
	5	.018	.019	.401	-.032	.067
	6	.013	.011	.288	-.015	.040

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 21: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in tartrate-resistant acid phosphatase during RT Fed (n=6). Main effect for time p=0.001.

Pairwise Comparisons

Measure:PTH

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.022	.041	.617	-.084	.128
	3	.190	.078	.060	-.011	.391
	4	.202*	.061	.022	.045	.360
	5	.099	.049	.098	-.026	.224
	6	.138	.086	.168	-.083	.359
	7	-.223*	.039	.002	-.324	-.122
2	1	-.022	.041	.617	-.128	.084
	3	.168*	.049	.019	.041	.295
	4	.180*	.041	.007	.076	.285
	5	.077	.039	.107	-.024	.177
	6	.116	.067	.142	-.055	.288
	7	-.245*	.049	.004	-.370	-.120
3	1	-.190	.078	.060	-.391	.011
	2	-.168*	.049	.019	-.295	-.041
	4	.013	.051	.816	-.119	.144
	5	-.091	.057	.171	-.238	.056
	6	-.052	.050	.348	-.180	.076
	7	-.413*	.063	.001	-.574	-.252
4	1	-.202*	.061	.022	-.360	-.045
	2	-.180*	.041	.007	-.285	-.076
	3	-.013	.051	.816	-.144	.119
	5	-.104*	.024	.007	-.164	-.043
	6	-.064	.052	.274	-.198	.070
	7	-.426*	.068	.001	-.599	-.252
5	1	-.099	.049	.098	-.224	.026
	2	-.077	.039	.107	-.177	.024
	3	.091	.057	.171	-.056	.238
	4	.104*	.024	.007	.043	.164
	6	.039	.069	.591	-.137	.216
	7	-.322*	.051	.001	-.452	-.192
6	1	-.138	.086	.168	-.359	.083
	2	-.116	.067	.142	-.288	.055
	3	.052	.050	.348	-.076	.180
	4	.064	.052	.274	-.070	.198
	5	-.039	.069	.591	-.216	.137
	7	-.361*	.089	.010	-.589	-.133
7	1	.223*	.039	.002	.122	.324
	2	.245*	.049	.004	.120	.370
	3	.413*	.063	.001	.252	.574
	4	.426*	.068	.001	.252	.599
	5	.322*	.051	.001	.192	.452
	6	.361*	.089	.010	.133	.589

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 22: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in parathyroid hormone during RT Fed (n=6). Main effect for time $p<0.001$.

Pairwise Comparisons

Measure:BAP

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.021*	.007	.030	.003	.039
	3	.056*	.015	.014	.017	.095
	4	.064*	.024	.041	.004	.125
	5	.054*	.017	.026	.010	.099
	6	.024	.022	.314	-.031	.080
	7	.049*	.014	.015	.014	.084
2	1	-.021*	.007	.030	-.039	-.003
	3	.035	.016	.077	-.005	.075
	4	.043	.019	.070	-.005	.092
	5	.033*	.012	.037	.003	.064
	6	.003	.016	.844	-.037	.044
	7	.028*	.011	.043	.001	.055
3	1	-.056*	.015	.014	-.095	-.017
	2	-.035	.016	.077	-.075	.005
	4	.008	.020	.687	-.043	.060
	5	-.002	.017	.931	-.044	.041
	6	-.032	.019	.154	-.080	.017
	7	-.006	.013	.630	-.039	.026
4	1	-.064*	.024	.041	-.125	-.004
	2	-.043	.019	.070	-.092	.005
	3	-.008	.020	.687	-.060	.043
	5	-.010	.008	.274	-.031	.011
	6	-.040*	.013	.030	-.074	-.006
	7	-.015	.013	.311	-.049	.019
5	1	-.054*	.017	.026	-.099	-.010
	2	-.033*	.012	.037	-.064	-.003
	3	.002	.017	.931	-.041	.044
	4	.010	.008	.274	-.011	.031
	6	-.030*	.010	.035	-.057	-.003
	7	-.005	.008	.560	-.025	.015
6	1	-.024	.022	.314	-.080	.031
	2	-.003	.016	.844	-.044	.037
	3	.032	.019	.154	-.017	.080
	4	.040*	.013	.030	.006	.074
	5	.030*	.010	.035	.003	.057
	7	.025	.015	.152	-.013	.063
7	1	-.049*	.014	.015	-.084	-.014
	2	-.028*	.011	.043	-.055	-.001
	3	.006	.013	.630	-.026	.039
	4	.015	.013	.311	-.019	.049
	5	.005	.008	.560	-.015	.025
	6	-.025	.015	.152	-.063	.013

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 23: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in bone-specific alkaline phosphatase during CON (n=6). Main effect for time p=0.002.

Pairwise Comparisons

Measure:CORTISOL

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.130*	.026	.004	.065	.196
	3	.157*	.031	.004	.078	.237
	4	.196*	.049	.011	.069	.323
	5	.230*	.047	.004	.109	.351
	6	.184	.082	.075	-.027	.395
	7	-.011	.028	.715	-.084	.062
2	1	-.130*	.026	.004	-.196	-.065
	3	.027*	.010	.049	9.272E-5	.053
	4	.065	.033	.103	-.019	.150
	5	.100*	.032	.026	.017	.182
	6	.054	.063	.435	-.109	.216
	7	-.141*	.037	.012	-.235	-.048
3	1	-.157*	.031	.004	-.237	-.078
	2	-.027*	.010	.049	-.053	-9.272E-5
	4	.039	.038	.361	-.060	.137
	5	.073	.031	.066	-.007	.153
	6	.027	.064	.692	-.137	.190
	7	-.168*	.037	.006	-.262	-.074
4	1	-.196*	.049	.011	-.323	-.069
	2	-.065	.033	.103	-.150	.019
	3	-.039	.038	.361	-.137	.060
	5	.034	.027	.253	-.034	.102
	6	-.012	.048	.817	-.136	.113
	7	-.207*	.057	.016	-.354	-.059
5	1	-.230*	.047	.004	-.351	-.109
	2	-.100*	.032	.026	-.182	-.017
	3	-.073	.031	.066	-.153	.007
	4	-.034	.027	.253	-.102	.034
	6	-.046	.051	.411	-.178	.086
	7	-.241*	.050	.005	-.369	-.113
6	1	-.184	.082	.075	-.395	.027
	2	-.054	.063	.435	-.216	.109
	3	-.027	.064	.692	-.190	.137
	4	.012	.048	.817	-.113	.136
	5	.046	.051	.411	-.086	.178
	7	-.195	.095	.095	-.439	.049
7	1	.011	.028	.715	-.062	.084
	2	.141*	.037	.012	.048	.235
	3	.168*	.037	.006	.074	.262
	4	.207*	.057	.016	.059	.354
	5	.241*	.050	.005	.113	.369
	6	.195	.095	.095	-.049	.439

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 24: Pairwise results from one-factor (time) RMANOVA to detect main effect changes in cortisol during CON (n=6). Main effect for time p<0.001.

Pairwise Comparisons

Measure:COR15MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.131	.055	.064	-.273	.011
	3	-.071	.051	.224	-.203	.061
	4	-.120*	.036	.021	-.214	-.027
	5	.026	.054	.649	-.112	.165
2	1	.131	.055	.064	-.011	.273
	3	.060	.081	.491	-.148	.268
	4	.011	.051	.844	-.120	.142
	5	.157	.094	.157	-.086	.400
3	1	.071	.051	.224	-.061	.203
	2	-.060	.081	.491	-.268	.148
	4	-.049	.048	.350	-.173	.074
	5	.097	.041	.062	-.007	.201
4	1	.120*	.036	.021	.027	.214
	2	-.011	.051	.844	-.142	.120
	3	.049	.048	.350	-.074	.173
	5	.147*	.048	.029	.022	.271
5	1	-.026	.054	.649	-.165	.112
	2	-.157	.094	.157	-.400	.086
	3	-.097	.041	.062	-.201	.007
	4	-.147*	.048	.029	-.271	-.022

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 25: Pairwise results from 2 x 2 RMANOVA detect main effect differences between trials for cortisol at 15 min (n=6). Main effect between trials, p=0.048.

Pairwise Comparisons

Measure:COR60MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.032	.043	.481	-.142	.077
	3	-.024	.050	.645	-.152	.103
	4	-.144	.057	.052	-.289	.002
	5	.042	.032	.244	-.039	.123
2	1	.032	.043	.481	-.077	.142
	3	.008	.080	.923	-.198	.215
	4	-.111	.084	.245	-.328	.106
	5	.074	.069	.332	-.103	.252
3	1	.024	.050	.645	-.103	.152
	2	-.008	.080	.923	-.215	.198
	4	-.120*	.026	.005	-.185	-.054
	5	.066	.030	.079	-.011	.143
4	1	.144	.057	.052	-.002	.289
	2	.111	.084	.245	-.106	.328
	3	.120*	.026	.005	.054	.185
	5	.185*	.039	.005	.086	.284
5	1	-.042	.032	.244	-.123	.039
	2	-.074	.069	.332	-.252	.103
	3	-.066	.030	.079	-.143	.011
	4	-.185*	.039	.005	-.284	-.086

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 26: Pairwise results from 2 x 2 RMANOVA detect main effect differences between trials for cortisol at 60 min (n=6). Main effect between trials, p=0.036.

Pairwise Comparisons

Measure:PTHPRE

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.218	.160	.231	-.194	.630
	3	.334*	.080	.009	.127	.541
	4	.309*	.060	.004	.154	.465
	5	.112*	.033	.019	.028	.196
2	1	-.218	.160	.231	-.630	.194
	3	.116	.149	.473	-.268	.500
	4	.091	.149	.566	-.291	.473
	5	-.106	.160	.536	-.518	.305
3	1	-.334*	.080	.009	-.541	-.127
	2	-.116	.149	.473	-.500	.268
	4	-.024	.065	.723	-.192	.143
	5	-.222*	.071	.026	-.404	-.040
4	1	-.309*	.060	.004	-.465	-.154
	2	-.091	.149	.566	-.473	.291
	3	.024	.065	.723	-.143	.192
	5	-.198*	.070	.038	-.379	-.016
5	1	-.112*	.033	.019	-.196	-.028
	2	.106	.160	.536	-.305	.518
	3	.222*	.071	.026	.040	.404
	4	.198*	.070	.038	.016	.379

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 27: Pairwise results from 2 x 2 RMANOVA detect main effect differences between trials for parathyroid hormone at PRE (n=6). Main effect between trials, p=0.034.

Pairwise Comparisons

Measure:PTHPOST

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.206*	.074	.039	.016	.396
	3	.141	.075	.118	-.051	.334
	4	.346*	.098	.017	.093	.598
	5	.055	.065	.431	-.111	.221
2	1	-.206*	.074	.039	-.396	-.016
	3	-.065	.117	.605	-.366	.237
	4	.140	.104	.237	-.128	.407
	5	-.151	.106	.214	-.423	.122
3	1	-.141	.075	.118	-.334	.051
	2	.065	.117	.605	-.237	.366
	4	.204	.088	.067	-.021	.430
	5	-.086	.073	.293	-.274	.102
4	1	-.346*	.098	.017	-.598	-.093
	2	-.140	.104	.237	-.407	.128
	3	-.204	.088	.067	-.430	.021
	5	-.290*	.099	.032	-.545	-.036
5	1	-.055	.065	.431	-.221	.111
	2	.151	.106	.214	-.122	.423
	3	.086	.073	.293	-.102	.274
	4	.290*	.099	.032	.036	.545

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 28: Pairwise results from 2 x 2 RMANOVA detect main effect differences between trials for parathyroid hormone at 15 min (n=6). Main effect between trials, p=0.011.

Pairwise Comparisons

Measure:PTH30MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.127	.078	.163	-.073	.327
	3	.120	.068	.141	-.056	.295
	4	.226*	.080	.036	.022	.431
	5	-.100	.062	.169	-.260	.060
2	1	-.127	.078	.163	-.327	.073
	3	-.008	.075	.923	-.201	.185
	4	.099	.052	.113	-.034	.232
	5	-.227*	.082	.040	-.439	-.015
3	1	-.120	.068	.141	-.295	.056
	2	.008	.075	.923	-.185	.201
	4	.107	.050	.086	-.022	.235
	5	-.220*	.028	.001	-.292	-.148
4	1	-.226*	.080	.036	-.431	-.022
	2	-.099	.052	.113	-.232	.034
	3	-.107	.050	.086	-.235	.022
	5	-.326*	.072	.006	-.512	-.140
5	1	.100	.062	.169	-.060	.260
	2	.227*	.082	.040	.015	.439
	3	.220*	.028	.001	.148	.292
	4	.326*	.072	.006	.140	.512

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 29: Pairwise results from 2 x 2 RMANOVA detect main effect differences between trials for parathyroid hormone at 30 min (n=6). Main effect between trials, p=0.001.

Pairwise Comparisons

Measure:PTH60MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.185*	.038	.005	.087	.282
	3	.232	.099	.065	-.021	.485
	4	.246	.111	.077	-.039	.532
	5	.025	.102	.819	-.238	.287
2	1	-.185*	.038	.005	-.282	-.087
	3	.047	.076	.562	-.148	.243
	4	.062	.086	.504	-.158	.282
	5	-.160	.082	.107	-.370	.049
3	1	-.232	.099	.065	-.485	.021
	2	-.047	.076	.562	-.243	.148
	4	.014	.048	.776	-.109	.138
	5	-.208*	.019	.000	-.257	-.158
4	1	-.246	.111	.077	-.532	.039
	2	-.062	.086	.504	-.282	.158
	3	-.014	.048	.776	-.138	.109
	5	-.222*	.039	.002	-.323	-.121
5	1	-.025	.102	.819	-.287	.238
	2	.160	.082	.107	-.049	.370
	3	.208*	.019	.000	.158	.257
	4	.222*	.039	.002	.121	.323

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 30: Pairwise results from 2 x 2 RMANOVA detect main effect differences between trials for parathyroid hormone at 60 min (n=6). Main effect between trials, p=0.008.

Pairwise Comparisons

Measure:PTH120MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.050	.029	.143	-.024	.124
	3	.061	.045	.239	-.056	.177
	4	.182*	.068	.044	.008	.357
	5	-.061	.037	.162	-.157	.035
2	1	-.050	.029	.143	-.124	.024
	3	.011	.050	.837	-.117	.139
	4	.132	.086	.183	-.088	.353
	5	-.111*	.022	.004	-.167	-.054
3	1	-.061	.045	.239	-.177	.056
	2	-.011	.050	.837	-.139	.117
	4	.122	.054	.075	-.018	.261
	5	-.122*	.042	.035	-.230	-.013
4	1	-.182*	.068	.044	-.357	-.008
	2	-.132	.086	.183	-.353	.088
	3	-.122	.054	.075	-.261	.018
	5	-.243*	.086	.036	-.463	-.023
5	1	.061	.037	.162	-.035	.157
	2	.111*	.022	.004	.054	.167
	3	.122*	.042	.035	.013	.230
	4	.243*	.086	.036	.023	.463

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Table 31: Pairwise results from 2 x 2 RMANOVA detect main effect differences between trials for parathyroid hormone at 120 min (n=6). Main effect between trials, p=0.005.

	PLY Fasted	PLY Fed	RT Fasted	RT Fed
BAP	0.624	0.350	0.512	0.308
TRAP	0.778	0.868	0.758	0.973
COR	1.000	1.000	1.000	0.999
PTH	0.999	1.000	0.913	1.000
TEST	0.930	0.926	0.669	0.776

Table 32: Observed power during the one-factor (time) RMANOVAs performed to detect significant main effects of each trial (n=12).

	CONTROL	PLY Fasted	PLY Fed	RT Fasted	RT Fed
BAP	0.970	0.586	0.578	0.407	0.312
TRAP	0.685	0.372	0.338	0.498	0.976
COR	0.999	1.000	0.929	0.829	0.679
PTH	0.591	0.914	0.998	0.491	1.000
TEST	0.093	0.564	0.714	0.305	0.413

Table 33: Observed power during the one-factor (time) RMANOVAs performed to detect significant main effects for each trial (n=6).

	PRE	POST	15MIN	30MIN	60MIN	120MIN	24HR
BAP	0.477	0.568	0.490	0.423	0.423	0.628	0.401
TRAP5B	0.211	0.178	0.079	0.097	0.066	0.124	0.161
COR	0.239	0.654	0.674	0.452	0.720	0.066	0.260
PTH	0.725	0.592	0.861	0.980	0.889	0.918	0.094
TEST	0.319	0.141	0.362	0.349	0.080	0.315	0.075

Table 34: Observed power during 2 x 2 ANOVA performed to detect difference at each individual time points between trials (n=6).

Tests of Within-Subjects Effects

Measure:BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.120	6	.020	2.680	.015	.061	16.083	.861
	Greenhouse-Geisser	.120	4.147	.029	2.680	.032	.061	11.117	.748
	Huynh-Feldt	.120	5.010	.024	2.680	.023	.061	13.429	.808
	Lower-bound	.120	1.000	.120	2.680	.109	.061	2.680	.359
TIME * fastfed	Sphericity Assumed	.034	6	.006	.760	.602	.018	4.559	.299
	Greenhouse-Geisser	.034	4.147	.008	.760	.557	.018	3.151	.245
	Huynh-Feldt	.034	5.010	.007	.760	.580	.018	3.807	.271
	Lower-bound	.034	1.000	.034	.760	.388	.018	.760	.136
TIME * plyrt	Sphericity Assumed	.034	6	.006	.768	.595	.018	4.611	.302
	Greenhouse-Geisser	.034	4.147	.008	.768	.551	.018	3.187	.248
	Huynh-Feldt	.034	5.010	.007	.768	.574	.018	3.850	.274
	Lower-bound	.034	1.000	.034	.768	.386	.018	.768	.137
TIME * fastfed * plyrt	Sphericity Assumed	.034	6	.006	.769	.595	.018	4.611	.303
	Greenhouse-Geisser	.034	4.147	.008	.769	.551	.018	3.187	.248
	Huynh-Feldt	.034	5.010	.007	.769	.574	.018	3.850	.274
	Lower-bound	.034	1.000	.034	.769	.386	.018	.769	.137
Error(TIME)	Sphericity Assumed	1.831	246	.007					
	Greenhouse-Geisser	1.831	170.037	.011					
	Huynh-Feldt	1.831	205.404	.009					
	Lower-bound	1.831	41.000	.045					

a. Computed using alpha = .05

Table 35: Observed Power during 2 x 2 x 7 RMANOVA (time) to detect a main effect of time, to detect differences between fed vs. fasted, and to detect differences between PLY vs. RT in bone-specific alkaline phosphatase.

Tests of Within-Subjects Effects

Measure:TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.057	6	.010	9.418	.000	.187	56.511	1.000
	Greenhouse-Geisser	.057	4.022	.014	9.418	.000	.187	37.885	1.000
	Huynh-Feldt	.057	4.841	.012	9.418	.000	.187	45.594	1.000
	Lower-bound	.057	1.000	.057	9.418	.004	.187	9.418	.850
TIME * fastfed	Sphericity Assumed	.008	6	.001	1.344	.238	.032	8.063	.522
	Greenhouse-Geisser	.008	4.022	.002	1.344	.256	.032	5.406	.414
	Huynh-Feldt	.008	4.841	.002	1.344	.249	.032	6.506	.461
	Lower-bound	.008	1.000	.008	1.344	.253	.032	1.344	.205
TIME * plyrt	Sphericity Assumed	.007	6	.001	1.156	.331	.027	6.937	.453
	Greenhouse-Geisser	.007	4.022	.002	1.156	.332	.027	4.650	.359
	Huynh-Feldt	.007	4.841	.001	1.156	.332	.027	5.597	.399
	Lower-bound	.007	1.000	.007	1.156	.289	.027	1.156	.183
TIME * fastfed * plyrt	Sphericity Assumed	.002	6	.000	.340	.915	.008	2.043	.146
	Greenhouse-Geisser	.002	4.022	.001	.340	.851	.008	1.370	.126
	Huynh-Feldt	.002	4.841	.000	.340	.883	.008	1.648	.134
	Lower-bound	.002	1.000	.002	.340	.563	.008	.340	.088
Error(TIME)	Sphericity Assumed	.248	246	.001					
	Greenhouse-Geisser	.248	164.917	.002					
	Huynh-Feldt	.248	198.479	.001					
	Lower-bound	.248	41.000	.006					

a. Computed using alpha = .05

Table 36: Observed Power during 2 x 2 x 7 RMANOVA (time) to detect a main effect of time, to detect differences between fed vs. fasted, and to detect differences between PLY vs. RT in tartrate-resistant acid phosphatase.

Tests of Within-Subjects Effects

Measure:CORTISOL

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.667	6	.444	42.625	.000	.516	255.748	1.000
	Greenhouse-Geisser	2.667	3.068	.869	42.625	.000	.516	130.754	1.000
	Huynh-Feldt	2.667	3.600	.741	42.625	.000	.516	153.467	1.000
	Lower-bound	2.667	1.000	2.667	42.625	.000	.516	42.625	1.000
TIME * fastfed	Sphericity Assumed	.104	6	.017	1.655	.133	.040	9.930	.627
	Greenhouse-Geisser	.104	3.068	.034	1.655	.179	.040	5.077	.430
	Huynh-Feldt	.104	3.600	.029	1.655	.170	.040	5.959	.471
	Lower-bound	.104	1.000	.104	1.655	.206	.040	1.655	.241
TIME * plyrt	Sphericity Assumed	.135	6	.023	2.159	.048	.051	12.957	.763
	Greenhouse-Geisser	.135	3.068	.044	2.159	.095	.051	6.624	.545
	Huynh-Feldt	.135	3.600	.038	2.159	.084	.051	7.775	.594
	Lower-bound	.135	1.000	.135	2.159	.150	.051	2.159	.300
TIME * fastfed * plyrt	Sphericity Assumed	.018	6	.003	.283	.945	.007	1.699	.127
	Greenhouse-Geisser	.018	3.068	.006	.283	.842	.007	.869	.104
	Huynh-Feldt	.018	3.600	.005	.283	.871	.007	1.019	.108
	Lower-bound	.018	1.000	.018	.283	.598	.007	.283	.081
Error(TIME)	Sphericity Assumed	2.503	240	.010					
	Greenhouse-Geisser	2.503	122.703	.020					
	Huynh-Feldt	2.503	144.017	.017					
	Lower-bound	2.503	40.000	.063					

a. Computed using alpha = .05

Table 37: Observed Power during 2 x 2 x 7 RMANOVA (time) to detect a main effect of time, to detect differences between fed vs. fasted, and to detect differences between PLY vs. RT in cortisol.

Tests of Within-Subjects Effects

Measure:PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.396	6	.399	27.063	.000	.404	162.375	1.000
	Greenhouse-Geisser	2.396	3.448	.695	27.063	.000	.404	93.319	1.000
	Huynh-Feldt	2.396	4.096	.585	27.063	.000	.404	110.854	1.000
	Lower-bound	2.396	1.000	2.396	27.063	.000	.404	27.063	.999
TIME * fastfed	Sphericity Assumed	.054	6	.009	.607	.724	.015	3.643	.241
	Greenhouse-Geisser	.054	3.448	.016	.607	.634	.015	2.094	.184
	Huynh-Feldt	.054	4.096	.013	.607	.662	.015	2.487	.199
	Lower-bound	.054	1.000	.054	.607	.440	.015	.607	.118
TIME * plyrt	Sphericity Assumed	.164	6	.027	1.852	.090	.044	11.112	.685
	Greenhouse-Geisser	.164	3.448	.048	1.852	.132	.044	6.386	.509
	Huynh-Feldt	.164	4.096	.040	1.852	.120	.044	7.586	.560
	Lower-bound	.164	1.000	.164	1.852	.181	.044	1.852	.264
TIME * fastfed * plyrt	Sphericity Assumed	.120	6	.020	1.355	.234	.033	8.130	.526
	Greenhouse-Geisser	.120	3.448	.035	1.355	.257	.033	4.672	.382
	Huynh-Feldt	.120	4.096	.029	1.355	.251	.033	5.550	.422
	Lower-bound	.120	1.000	.120	1.355	.251	.033	1.355	.206
Error(TIME)	Sphericity Assumed	3.541	240	.015					
	Greenhouse-Geisser	3.541	137.931	.026					
	Huynh-Feldt	3.541	163.848	.022					
	Lower-bound	3.541	40.000	.089					

a. Computed using alpha = .05

Table 38: Observed Power during 2 x 2 x 7 RMANOVA (time) to detect a main effect of time, to detect differences between fed vs. fasted, and to detect differences between PLY vs. RT in parathyroid hormone.

Tests of Within-Subjects Effects

Measure:TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.427	6	.071	8.762	.000	.180	52.571	1.000
	Greenhouse-Geisser	.427	4.703	.091	8.762	.000	.180	41.210	1.000
	Huynh-Feldt	.427	5.806	.074	8.762	.000	.180	50.875	1.000
	Lower-bound	.427	1.000	.427	8.762	.005	.180	8.762	.823
TIME * fastfed	Sphericity Assumed	.052	6	.009	1.059	.388	.026	6.353	.415
	Greenhouse-Geisser	.052	4.703	.011	1.059	.383	.026	4.980	.360
	Huynh-Feldt	.052	5.806	.009	1.059	.387	.026	6.148	.407
	Lower-bound	.052	1.000	.052	1.059	.310	.026	1.059	.171
TIME * plyrt	Sphericity Assumed	.056	6	.009	1.155	.331	.028	6.932	.452
	Greenhouse-Geisser	.056	4.703	.012	1.155	.333	.028	5.434	.392
	Huynh-Feldt	.056	5.806	.010	1.155	.331	.028	6.709	.444
	Lower-bound	.056	1.000	.056	1.155	.289	.028	1.155	.183
TIME * fastfed * plyrt	Sphericity Assumed	.019	6	.003	.399	.879	.010	2.394	.165
	Greenhouse-Geisser	.019	4.703	.004	.399	.838	.010	1.877	.150
	Huynh-Feldt	.019	5.806	.003	.399	.874	.010	2.317	.163
	Lower-bound	.019	1.000	.019	.399	.531	.010	.399	.095
Error(TIME)	Sphericity Assumed	1.950	240	.008					
	Greenhouse-Geisser	1.950	188.134	.010					
	Huynh-Feldt	1.950	232.257	.008					
	Lower-bound	1.950	40.000	.049					

a. Computed using alpha = .05

Table 39: Observed Power during 2 x 2 x 7 RMANOVA (time) to detect a main effect of time, to detect differences between fed vs. fasted, and to detect differences between PLY vs. RT in testosterone.

Appendix D
Statistical Analyses

Note: This data is the result of log (base 10) transformation in order to meet the assumption of normality.

One-factor (Time) Repeated Measures ANOVA – Plyometrics Fasted (n=12)

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.108	6	1.802E-02	1.762	.121	.138	10.574	.624
	Greenhouse-Geisser	.108	2.143	5.044E-02	1.762	.192	.138	3.777	.342
	Huynh-Feldt	.108	2.678	4.037E-02	1.762	.180	.138	4.719	.389
	Lower-bound	.108	1.000	.108	1.762	.211	.138	1.762	.228
Error(TIME)	Sphericity Assumed	.675	66	1.022E-02					
	Greenhouse-Geisser	.675	23.575	2.862E-02					
	Huynh-Feldt	.675	29.458	2.291E-02					
	Lower-bound	.675	11.000	6.135E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.118	6	1.965E-02	1.973	.084	.165	11.837	.678
	Greenhouse-Geisser	.118	2.233	5.278E-02	1.973	.159	.165	4.406	.383
	Huynh-Feldt	.118	3.193	3.692E-02	1.973	.135	.165	6.299	.474
	Lower-bound	.118	1.000	.118	1.973	.190	.165	1.973	.246
TIME * AGE	Sphericity Assumed	7.734E-02	6	1.289E-02	1.294	.274	.115	7.767	.469
	Greenhouse-Geisser	7.734E-02	2.233	3.463E-02	1.294	.296	.115	2.891	.263
	Huynh-Feldt	7.734E-02	3.193	2.422E-02	1.294	.293	.115	4.133	.321
	Lower-bound	7.734E-02	1.000	7.734E-02	1.294	.282	.115	1.294	.178
Error(TIME)	Sphericity Assumed	.597	60	9.958E-03					
	Greenhouse-Geisser	.597	22.331	2.676E-02					
	Huynh-Feldt	.597	31.927	1.871E-02					
	Lower-bound	.597	10.000	5.975E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.209E-02	6	2.015E-03	2.381	.038	.178	14.285	.778
	Greenhouse-Geisser	1.209E-02	3.025	3.996E-03	2.381	.087	.178	7.203	.546
	Huynh-Feldt	1.209E-02	4.302	2.810E-03	2.381	.061	.178	10.242	.663
	Lower-bound	1.209E-02	1.000	1.209E-02	2.381	.151	.178	2.381	.291
Error(TIME)	Sphericity Assumed	5.586E-02	66	8.464E-04					
	Greenhouse-Geisser	5.586E-02	33.280	1.679E-03					
	Huynh-Feldt	5.586E-02	47.321	1.180E-03					
	Lower-bound	5.586E-02	11.000	5.078E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.833E-03	6	4.722E-04	.521	.790	.050	3.129	.195
	Greenhouse-Geisser	2.833E-03	2.996	9.455E-04	.521	.671	.050	1.563	.144
	Huynh-Feldt	2.833E-03	4.849	5.843E-04	.521	.754	.050	2.528	.176
	Lower-bound	2.833E-03	1.000	2.833E-03	.521	.487	.050	.521	.100
TIME * AGE	Sphericity Assumed	1.532E-03	6	2.553E-04	.282	.943	.027	1.691	.120
	Greenhouse-Geisser	1.532E-03	2.996	5.111E-04	.282	.838	.027	.845	.098
	Huynh-Feldt	1.532E-03	4.849	3.159E-04	.282	.917	.027	1.367	.112
	Lower-bound	1.532E-03	1.000	1.532E-03	.282	.607	.027	.282	.077
Error(TIME)	Sphericity Assumed	5.433E-02	60	9.055E-04					
	Greenhouse-Geisser	5.433E-02	29.965	1.813E-03					
	Huynh-Feldt	5.433E-02	48.487	1.121E-03					
	Lower-bound	5.433E-02	10.000	5.433E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.949	6	.158	20.724	.000	.653	124.345	1.000
	Greenhouse-Geisser	.949	2.603	.365	20.724	.000	.653	53.951	1.000
	Huynh-Feldt	.949	3.482	.273	20.724	.000	.653	72.166	1.000
	Lower-bound	.949	1.000	.949	20.724	.001	.653	20.724	.985
Error(TIME)	Sphericity Assumed	.504	66	7.636E-03					
	Greenhouse-Geisser	.504	28.636	1.760E-02					
	Huynh-Feldt	.504	38.304	1.316E-02					
	Lower-bound	.504	11.000	4.581E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.166	6	2.774E-02	3.584	.004	.264	21.506	.932
	Greenhouse-Geisser	.166	2.505	6.645E-02	3.584	.034	.264	8.978	.672
	Huynh-Feldt	.166	3.743	4.446E-02	3.584	.016	.264	13.418	.808
	Lower-bound	.166	1.000	.166	3.584	.088	.264	3.584	.402
TIME * AGE	Sphericity Assumed	3.963E-02	6	6.605E-03	.853	.534	.079	5.121	.311
	Greenhouse-Geisser	3.963E-02	2.505	1.582E-02	.853	.460	.079	2.138	.195
	Huynh-Feldt	3.963E-02	3.743	1.059E-02	.853	.495	.079	3.195	.239
	Lower-bound	3.963E-02	1.000	3.963E-02	.853	.377	.079	.853	.133
Error(TIME)	Sphericity Assumed	.464	60	7.739E-03					
	Greenhouse-Geisser	.464	25.048	1.854E-02					
	Huynh-Feldt	.464	37.434	1.240E-02					
	Lower-bound	.464	10.000	4.643E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.559	6	9.322E-02	6.583	.000	.374	39.496	.999
	Greenhouse-Geisser	.559	2.996	.187	6.583	.001	.374	19.721	.954
	Huynh-Feldt	.559	4.242	.132	6.583	.000	.374	27.921	.989
	Lower-bound	.559	1.000	.559	6.583	.026	.374	6.583	.647
Error(TIME)	Sphericity Assumed	.935	66	1.416E-02					
	Greenhouse-Geisser	.935	32.954	2.836E-02					
	Huynh-Feldt	.935	46.657	2.003E-02					
	Lower-bound	.935	11.000	8.497E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.256	6	4.267E-02	3.056	.011	.234	18.337	.882
	Greenhouse-Geisser	.256	2.780	9.209E-02	3.056	.048	.234	8.497	.631
	Huynh-Feldt	.256	4.344	5.894E-02	3.056	.023	.234	13.276	.784
	Lower-bound	.256	1.000	.256	3.056	.111	.234	3.056	.353
TIME * AGE	Sphericity Assumed	9.693E-02	6	1.615E-02	1.157	.341	.104	6.942	.421
	Greenhouse-Geisser	9.693E-02	2.780	3.486E-02	1.157	.341	.104	3.217	.268
	Huynh-Feldt	9.693E-02	4.344	2.231E-02	1.157	.344	.104	5.026	.346
	Lower-bound	9.693E-02	1.000	9.693E-02	1.157	.307	.104	1.157	.164
Error(TIME)	Sphericity Assumed	.838	60	1.396E-02					
	Greenhouse-Geisser	.838	27.803	3.013E-02					
	Huynh-Feldt	.838	43.441	1.928E-02					
	Lower-bound	.838	10.000	8.377E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.160	6	2.668E-02	3.517	.004	.242	21.101	.930
	Greenhouse-Geisser	.160	3.023	5.295E-02	3.517	.025	.242	10.632	.733
	Huynh-Feldt	.160	4.297	3.725E-02	3.517	.012	.242	15.113	.847
	Lower-bound	.160	1.000	.160	3.517	.088	.242	3.517	.402
Error(TIME)	Sphericity Assumed	.501	66	7.587E-03					
	Greenhouse-Geisser	.501	33.255	1.506E-02					
	Huynh-Feldt	.501	47.270	1.059E-02					
	Lower-bound	.501	11.000	4.552E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.107	6	1.775E-02	2.499	.032	.200	14.994	.797
	Greenhouse-Geisser	.107	2.766	3.851E-02	2.499	.084	.200	6.913	.535
	Huynh-Feldt	.107	4.312	2.470E-02	2.499	.052	.200	10.777	.683
	Lower-bound	.107	1.000	.107	2.499	.145	.200	2.499	.299
TIME * AGE	Sphericity Assumed	7.451E-02	6	1.242E-02	1.748	.125	.149	10.489	.615
	Greenhouse-Geisser	7.451E-02	2.766	2.694E-02	1.748	.183	.149	4.836	.390
	Huynh-Feldt	7.451E-02	4.312	1.728E-02	1.748	.153	.149	7.539	.508
	Lower-bound	7.451E-02	1.000	7.451E-02	1.748	.216	.149	1.748	.224
Error(TIME)	Sphericity Assumed	.426	60	7.104E-03					
	Greenhouse-Geisser	.426	27.662	1.541E-02					
	Huynh-Feldt	.426	43.122	9.884E-03					
	Lower-bound	.426	10.000	4.262E-02					

a. Computed using alpha = .05

One-factor (Time) Repeated Measures ANOVA – Plyometrics Fed (n=12)

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	3.807E-02	6	6.346E-03	.953	.464	.080	5.719	.350
	Greenhouse-Geisser	3.807E-02	2.883	1.321E-02	.953	.424	.080	2.748	.232
	Huynh-Feldt	3.807E-02	4.016	9.481E-03	.953	.443	.080	3.827	.277
	Lower-bound	3.807E-02	1.000	3.807E-02	.953	.350	.080	.953	.145
Error(TIME)	Sphericity Assumed	.439	66	6.658E-03					
	Greenhouse-Geisser	.439	31.712	1.386E-02					
	Huynh-Feldt	.439	44.172	9.948E-03					
	Lower-bound	.439	11.000	3.995E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	3.592E-02	6	5.987E-03	.867	.525	.080	5.201	.316
	Greenhouse-Geisser	3.592E-02	2.726	1.318E-02	.867	.461	.080	2.363	.206
	Huynh-Feldt	3.592E-02	4.223	8.507E-03	.867	.497	.080	3.660	.258
	Lower-bound	3.592E-02	1.000	3.592E-02	.867	.374	.080	.867	.135
TIME * AGE	Sphericity Assumed	2.501E-02	6	4.168E-03	.603	.726	.057	3.621	.223
	Greenhouse-Geisser	2.501E-02	2.726	9.173E-03	.603	.603	.057	1.645	.154
	Huynh-Feldt	2.501E-02	4.223	5.923E-03	.603	.671	.057	2.548	.187
	Lower-bound	2.501E-02	1.000	2.501E-02	.603	.455	.057	.603	.109
Error(TIME)	Sphericity Assumed	.414	60	6.907E-03					
	Greenhouse-Geisser	.414	27.262	1.520E-02					
	Huynh-Feldt	.414	42.227	9.814E-03					
	Lower-bound	.414	10.000	4.144E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.557E-02	6	2.595E-03	2.921	.014	.210	17.526	.868
	Greenhouse-Geisser	1.557E-02	3.110	5.006E-03	2.921	.046	.210	9.085	.653
	Huynh-Feldt	1.557E-02	4.477	3.477E-03	2.921	.026	.210	13.076	.778
	Lower-bound	1.557E-02	1.000	1.557E-02	2.921	.115	.210	2.921	.345
Error(TIME)	Sphericity Assumed	5.862E-02	66	8.882E-04					
	Greenhouse-Geisser	5.862E-02	34.210	1.714E-03					
	Huynh-Feldt	5.862E-02	49.243	1.190E-03					
	Lower-bound	5.862E-02	11.000	5.329E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.379E-02	6	2.298E-03	2.793	.018	.218	16.756	.846
	Greenhouse-Geisser	1.379E-02	3.344	4.123E-03	2.793	.050	.218	9.340	.651
	Huynh-Feldt	1.379E-02	5.730	2.407E-03	2.793	.020	.218	16.001	.832
	Lower-bound	1.379E-02	1.000	1.379E-02	2.793	.126	.218	2.793	.327
TIME * AGE	Sphericity Assumed	9.244E-03	6	1.541E-03	1.872	.100	.158	11.233	.651
	Greenhouse-Geisser	9.244E-03	3.344	2.764E-03	1.872	.148	.158	6.261	.465
	Huynh-Feldt	9.244E-03	5.730	1.613E-03	1.872	.104	.158	10.727	.635
	Lower-bound	9.244E-03	1.000	9.244E-03	1.872	.201	.158	1.872	.236
Error(TIME)	Sphericity Assumed	4.938E-02	60	8.230E-04					
	Greenhouse-Geisser	4.938E-02	33.445	1.476E-03					
	Huynh-Feldt	4.938E-02	57.297	8.618E-04					
	Lower-bound	4.938E-02	10.000	4.938E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.892	6	.149	11.740	.000	.516	70.439	1.000
	Greenhouse-Geisser	.892	2.524	.353	11.740	.000	.516	29.632	.996
	Huynh-Feldt	.892	3.338	.267	11.740	.000	.516	39.183	.999
	Lower-bound	.892	1.000	.892	11.740	.006	.516	11.740	.876
Error(TIME)	Sphericity Assumed	.836	66	1.266E-02					
	Greenhouse-Geisser	.836	27.765	3.010E-02					
	Huynh-Feldt	.836	36.713	2.276E-02					
	Lower-bound	.836	11.000	7.596E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.213	6	3.549E-02	2.862	.016	.223	17.171	.856
	Greenhouse-Geisser	.213	2.644	8.055E-02	2.862	.062	.223	7.566	.583
	Huynh-Feldt	.213	4.041	5.270E-02	2.862	.035	.223	11.563	.728
	Lower-bound	.213	1.000	.213	2.862	.122	.223	2.862	.334
TIME * AGE	Sphericity Assumed	9.147E-02	6	1.524E-02	1.229	.304	.109	7.375	.446
	Greenhouse-Geisser	9.147E-02	2.644	3.460E-02	1.229	.316	.109	3.250	.275
	Huynh-Feldt	9.147E-02	4.041	2.264E-02	1.229	.314	.109	4.967	.351
	Lower-bound	9.147E-02	1.000	9.147E-02	1.229	.294	.109	1.229	.171
Error(TIME)	Sphericity Assumed	.744	60	1.240E-02					
	Greenhouse-Geisser	.744	26.437	2.815E-02					
	Huynh-Feldt	.744	40.407	1.842E-02					
	Lower-bound	.744	10.000	7.441E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.673	6	.112	11.210	.000	.505	67.257	1.000
	Greenhouse-Geisser	.673	3.026	.222	11.210	.000	.505	33.915	.998
	Huynh-Feldt	.673	4.302	.156	11.210	.000	.505	48.223	1.000
	Lower-bound	.673	1.000	.673	11.210	.006	.505	11.210	.861
Error(TIME)	Sphericity Assumed	.661	66	1.001E-02					
	Greenhouse-Geisser	.661	33.281	1.985E-02					
	Huynh-Feldt	.661	47.322	1.396E-02					
	Lower-bound	.661	11.000	6.005E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.155	6	2.583E-02	2.594	.027	.206	15.567	.814
	Greenhouse-Geisser	.155	2.686	5.769E-02	2.594	.079	.206	6.969	.543
	Huynh-Feldt	.155	4.134	3.749E-02	2.594	.049	.206	10.724	.687
	Lower-bound	.155	1.000	.155	2.594	.138	.206	2.594	.308
TIME * AGE	Sphericity Assumed	6.332E-02	6	1.055E-02	1.060	.396	.096	6.361	.386
	Greenhouse-Geisser	6.332E-02	2.686	2.357E-02	1.060	.377	.096	2.848	.243
	Huynh-Feldt	6.332E-02	4.134	1.532E-02	1.060	.390	.096	4.382	.309
	Lower-bound	6.332E-02	1.000	6.332E-02	1.060	.327	.096	1.060	.154
Error(TIME)	Sphericity Assumed	.597	60	9.954E-03					
	Greenhouse-Geisser	.597	26.861	2.224E-02					
	Huynh-Feldt	.597	41.336	1.445E-02					
	Lower-bound	.597	10.000	5.972E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.135	6	2.249E-02	3.474	.005	.240	20.841	.926
	Greenhouse-Geisser	.135	3.463	3.897E-02	3.474	.020	.240	12.027	.773
	Huynh-Feldt	.135	5.247	2.571E-02	3.474	.007	.240	18.227	.896
	Lower-bound	.135	1.000	.135	3.474	.089	.240	3.474	.398
Error(TIME)	Sphericity Assumed	.427	66	6.474E-03					
	Greenhouse-Geisser	.427	38.089	1.122E-02					
	Huynh-Feldt	.427	57.721	7.402E-03					
	Lower-bound	.427	11.000	3.884E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.794E-02	6	4.657E-03	.701	.650	.066	4.206	.257
	Greenhouse-Geisser	2.794E-02	3.544	7.885E-03	.701	.580	.066	2.484	.195
	Huynh-Feldt	2.794E-02	6.000	4.657E-03	.701	.650	.066	4.206	.257
	Lower-bound	2.794E-02	1.000	2.794E-02	.701	.422	.066	.701	.118
TIME * AGE	Sphericity Assumed	2.861E-02	6	4.768E-03	.718	.637	.067	4.306	.263
	Greenhouse-Geisser	2.861E-02	3.544	8.072E-03	.718	.570	.067	2.543	.199
	Huynh-Feldt	2.861E-02	6.000	4.768E-03	.718	.637	.067	4.306	.263
	Lower-bound	2.861E-02	1.000	2.861E-02	.718	.417	.067	.718	.120
Error(TIME)	Sphericity Assumed	.399	60	6.644E-03					
	Greenhouse-Geisser	.399	35.441	1.125E-02					
	Huynh-Feldt	.399	60.000	6.644E-03					
	Lower-bound	.399	10.000	3.987E-02					

a. Computed using alpha = .05

One-factor (Time) Repeated Measures ANOVA – Resistance-Training Fasted (n=12)

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	4.381E-02	6	7.302E-03	1.456	.213	.154	8.737	.512
	Greenhouse-Geisser	4.381E-02	1.434	3.054E-02	1.456	.265	.154	2.089	.223
	Huynh-Feldt	4.381E-02	1.662	2.636E-02	1.456	.265	.154	2.420	.240
	Lower-bound	4.381E-02	1.000	4.381E-02	1.456	.262	.154	1.456	.187
Error(TIME)	Sphericity Assumed	.241	48	5.014E-03					
	Greenhouse-Geisser	.241	11.476	2.097E-02					
	Huynh-Feldt	.241	13.294	1.810E-02					
	Lower-bound	.241	8.000	3.009E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	5.737E-03	6	9.562E-04	.170	.983	.024	1.019	.088
	Greenhouse-Geisser	5.737E-03	1.397	4.107E-03	.170	.770	.024	.237	.068
	Huynh-Feldt	5.737E-03	1.887	3.041E-03	.170	.834	.024	.320	.071
	Lower-bound	5.737E-03	1.000	5.737E-03	.170	.693	.024	.170	.065
TIME * AGE	Sphericity Assumed	4.228E-03	6	7.047E-04	.125	.993	.018	.751	.077
	Greenhouse-Geisser	4.228E-03	1.397	3.027E-03	.125	.811	.018	.175	.063
	Huynh-Feldt	4.228E-03	1.887	2.241E-03	.125	.873	.018	.236	.065
	Lower-bound	4.228E-03	1.000	4.228E-03	.125	.734	.018	.125	.061
Error(TIME)	Sphericity Assumed	.236	42	5.630E-03					
	Greenhouse-Geisser	.236	9.778	2.418E-02					
	Huynh-Feldt	.236	13.207	1.790E-02					
	Lower-bound	.236	7.000	3.378E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.300E-02	6	2.167E-03	2.368	.044	.228	14.209	.758
	Greenhouse-Geisser	1.300E-02	2.678	4.855E-03	2.368	.105	.228	6.341	.487
	Huynh-Feldt	1.300E-02	4.152	3.131E-03	2.368	.071	.228	9.833	.629
	Lower-bound	1.300E-02	1.000	1.300E-02	2.368	.162	.228	2.368	.274
Error(TIME)	Sphericity Assumed	4.392E-02	48	9.149E-04					
	Greenhouse-Geisser	4.392E-02	21.422	2.050E-03					
	Huynh-Feldt	4.392E-02	33.219	1.322E-03					
	Lower-bound	4.392E-02	8.000	5.489E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.190E-02	6	1.984E-03	2.289	.053	.246	13.733	.732
	Greenhouse-Geisser	1.190E-02	2.228	5.342E-03	2.289	.130	.246	5.101	.414
	Huynh-Feldt	1.190E-02	3.784	3.146E-03	2.289	.089	.246	8.661	.571
	Lower-bound	1.190E-02	1.000	1.190E-02	2.289	.174	.246	2.289	.258
TIME * AGE	Sphericity Assumed	7.507E-03	6	1.251E-03	1.443	.221	.171	8.659	.499
	Greenhouse-Geisser	7.507E-03	2.228	3.369E-03	1.443	.267	.171	3.216	.274
	Huynh-Feldt	7.507E-03	3.784	1.984E-03	1.443	.249	.171	5.461	.375
	Lower-bound	7.507E-03	1.000	7.507E-03	1.443	.269	.171	1.443	.181
Error(TIME)	Sphericity Assumed	3.641E-02	42	8.669E-04					
	Greenhouse-Geisser	3.641E-02	15.599	2.334E-03					
	Huynh-Feldt	3.641E-02	26.489	1.374E-03					
	Lower-bound	3.641E-02	7.000	5.201E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.654	6	.109	11.739	.000	.595	70.433	1.000
	Greenhouse-Geisser	.654	1.601	.408	11.739	.002	.595	18.795	.956
	Huynh-Feldt	.654	1.939	.337	11.739	.001	.595	22.766	.979
	Lower-bound	.654	1.000	.654	11.739	.009	.595	11.739	.850
Error(TIME)	Sphericity Assumed	.446	48	9.285E-03					
	Greenhouse-Geisser	.446	12.809	3.480E-02					
	Huynh-Feldt	.446	15.515	2.873E-02					
	Lower-bound	.446	8.000	5.571E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.178	6	2.960E-02	3.330	.009	.322	19.978	.897
	Greenhouse-Geisser	.178	1.622	.109	3.330	.080	.322	5.402	.470
	Huynh-Feldt	.178	2.343	7.580E-02	3.330	.055	.322	7.802	.586
	Lower-bound	.178	1.000	.178	3.330	.111	.322	3.330	.351
TIME * AGE	Sphericity Assumed	7.231E-02	6	1.205E-02	1.356	.255	.162	8.134	.471
	Greenhouse-Geisser	7.231E-02	1.622	4.458E-02	1.356	.290	.162	2.199	.218
	Huynh-Feldt	7.231E-02	2.343	3.086E-02	1.356	.288	.162	3.177	.266
	Lower-bound	7.231E-02	1.000	7.231E-02	1.356	.282	.162	1.356	.173
Error(TIME)	Sphericity Assumed	.373	42	8.890E-03					
	Greenhouse-Geisser	.373	11.356	3.288E-02					
	Huynh-Feldt	.373	16.402	2.276E-02					
	Lower-bound	.373	7.000	5.334E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.552	6	9.204E-02	3.439	.007	.301	20.634	.913
	Greenhouse-Geisser	.552	1.480	.373	3.439	.077	.301	5.090	.469
	Huynh-Feldt	.552	1.736	.318	3.439	.066	.301	5.970	.516
	Lower-bound	.552	1.000	.552	3.439	.101	.301	3.439	.372
Error(TIME)	Sphericity Assumed	1.285	48	2.676E-02					
	Greenhouse-Geisser	1.285	11.840	.109					
	Huynh-Feldt	1.285	13.889	9.249E-02					
	Lower-bound	1.285	8.000	.161					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.231	6	3.843E-02	1.390	.241	.166	8.343	.482
	Greenhouse-Geisser	.231	1.266	.182	1.390	.281	.166	1.761	.197
	Huynh-Feldt	.231	1.639	.141	1.390	.282	.166	2.279	.224
	Lower-bound	.231	1.000	.231	1.390	.277	.166	1.390	.176
TIME * AGE	Sphericity Assumed	.124	6	2.064E-02	.747	.615	.096	4.482	.262
	Greenhouse-Geisser	.124	1.266	9.781E-02	.747	.442	.096	.946	.127
	Huynh-Feldt	.124	1.639	7.557E-02	.747	.470	.096	1.224	.140
	Lower-bound	.124	1.000	.124	.747	.416	.096	.747	.117
Error(TIME)	Sphericity Assumed	1.161	42	2.764E-02					
	Greenhouse-Geisser	1.161	8.865	.131					
	Huynh-Feldt	1.161	11.474	.101					
	Lower-bound	1.161	7.000	.166					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.125	6	2.079E-02	1.939	.089	.162	11.632	.669
	Greenhouse-Geisser	.125	3.055	4.082E-02	1.939	.143	.162	5.923	.454
	Huynh-Feldt	.125	4.552	2.740E-02	1.939	.112	.162	8.824	.575
	Lower-bound	.125	1.000	.125	1.939	.194	.162	1.939	.243
Error(TIME)	Sphericity Assumed	.643	60	1.072E-02					
	Greenhouse-Geisser	.643	30.555	2.105E-02					
	Huynh-Feldt	.643	45.518	1.413E-02					
	Lower-bound	.643	10.000	6.433E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.121	6	2.019E-02	1.962	.087	.179	11.774	.670
	Greenhouse-Geisser	.121	3.150	3.846E-02	1.962	.140	.179	6.182	.462
	Huynh-Feldt	.121	5.582	2.170E-02	1.962	.093	.179	10.953	.644
	Lower-bound	.121	1.000	.121	1.962	.195	.179	1.962	.241
TIME * AGE	Sphericity Assumed	8.766E-02	6	1.461E-02	1.420	.224	.136	8.519	.506
	Greenhouse-Geisser	8.766E-02	3.150	2.783E-02	1.420	.257	.136	4.473	.343
	Huynh-Feldt	8.766E-02	5.582	1.570E-02	1.420	.229	.136	7.925	.485
	Lower-bound	8.766E-02	1.000	8.766E-02	1.420	.264	.136	1.420	.187
Error(TIME)	Sphericity Assumed	.556	54	1.029E-02					
	Greenhouse-Geisser	.556	28.352	1.960E-02					
	Huynh-Feldt	.556	50.237	1.106E-02					
	Lower-bound	.556	9.000	6.174E-02					

a. Computed using alpha = .05

One-factor (Time) Repeated Measures ANOVA – Resistance-Training Fed (n=12)

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	3.616E-02	6	6.026E-03	.836	.546	.071	5.016	.308
	Greenhouse-Geisser	3.616E-02	3.216	1.124E-02	.836	.490	.071	2.689	.219
	Huynh-Feldt	3.616E-02	4.701	7.691E-03	.836	.524	.071	3.931	.268
	Lower-bound	3.616E-02	1.000	3.616E-02	.836	.380	.071	.836	.133
Error(TIME)	Sphericity Assumed	.476	66	7.207E-03					
	Greenhouse-Geisser	.476	35.377	1.345E-02					
	Huynh-Feldt	.476	51.713	9.199E-03					
	Lower-bound	.476	11.000	4.324E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	5.409E-02	6	9.015E-03	1.254	.292	.111	7.525	.455
	Greenhouse-Geisser	5.409E-02	3.062	1.766E-02	1.254	.308	.111	3.840	.304
	Huynh-Feldt	5.409E-02	5.008	1.080E-02	1.254	.298	.111	6.280	.407
	Lower-bound	5.409E-02	1.000	5.409E-02	1.254	.289	.111	1.254	.174
TIME * AGE	Sphericity Assumed	4.442E-02	6	7.403E-03	1.030	.415	.093	6.179	.375
	Greenhouse-Geisser	4.442E-02	3.062	1.451E-02	1.030	.394	.093	3.153	.254
	Huynh-Feldt	4.442E-02	5.008	8.870E-03	1.030	.410	.093	5.157	.336
	Lower-bound	4.442E-02	1.000	4.442E-02	1.030	.334	.093	1.030	.151
Error(TIME)	Sphericity Assumed	.431	60	7.188E-03					
	Greenhouse-Geisser	.431	30.619	1.408E-02					
	Huynh-Feldt	.431	50.076	8.612E-03					
	Lower-bound	.431	10.000	4.313E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	3.549E-02	6	5.915E-03	4.335	.001	.283	26.008	.973
	Greenhouse-Geisser	3.549E-02	2.563	1.385E-02	4.335	.016	.283	11.109	.774
	Huynh-Feldt	3.549E-02	3.408	1.041E-02	4.335	.008	.283	14.773	.862
	Lower-bound	3.549E-02	1.000	3.549E-02	4.335	.061	.283	4.335	.476
Error(TIME)	Sphericity Assumed	9.006E-02	66	1.365E-03					
	Greenhouse-Geisser	9.006E-02	28.192	3.195E-03					
	Huynh-Feldt	9.006E-02	37.489	2.402E-03					
	Lower-bound	9.006E-02	11.000	8.187E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	6.668E-03	6	1.111E-03	.769	.597	.071	4.615	.281
	Greenhouse-Geisser	6.668E-03	2.537	2.628E-03	.769	.502	.071	1.951	.180
	Huynh-Feldt	6.668E-03	3.811	1.750E-03	.769	.546	.071	2.931	.219
	Lower-bound	6.668E-03	1.000	6.668E-03	.769	.401	.071	.769	.125
TIME * AGE	Sphericity Assumed	3.361E-03	6	5.601E-04	.388	.884	.037	2.326	.152
	Greenhouse-Geisser	3.361E-03	2.537	1.325E-03	.388	.730	.037	.983	.111
	Huynh-Feldt	3.361E-03	3.811	8.818E-04	.388	.807	.037	1.477	.127
	Lower-bound	3.361E-03	1.000	3.361E-03	.388	.547	.037	.388	.087
Error(TIME)	Sphericity Assumed	8.670E-02	60	1.445E-03					
	Greenhouse-Geisser	8.670E-02	25.370	3.417E-03					
	Huynh-Feldt	8.670E-02	38.113	2.275E-03					
	Lower-bound	8.670E-02	10.000	8.670E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.477	6	7.944E-02	6.644	.000	.399	39.866	.999
	Greenhouse-Geisser	.477	1.833	.260	6.644	.008	.399	12.179	.840
	Huynh-Feldt	.477	2.224	.214	6.644	.004	.399	14.776	.893
	Lower-bound	.477	1.000	.477	6.644	.028	.399	6.644	.643
Error(TIME)	Sphericity Assumed	.717	60	1.196E-02					
	Greenhouse-Geisser	.717	18.329	3.914E-02					
	Huynh-Feldt	.717	22.239	3.226E-02					
	Lower-bound	.717	10.000	7.173E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	9.591E-02	6	1.598E-02	1.308	.269	.127	7.849	.469
	Greenhouse-Geisser	9.591E-02	1.739	5.514E-02	1.308	.294	.127	2.276	.229
	Huynh-Feldt	9.591E-02	2.360	4.064E-02	1.308	.295	.127	3.087	.269
	Lower-bound	9.591E-02	1.000	9.591E-02	1.308	.282	.127	1.308	.176
TIME * AGE	Sphericity Assumed	5.755E-02	6	9.592E-03	.785	.585	.080	4.711	.284
	Greenhouse-Geisser	5.755E-02	1.739	3.309E-02	.785	.457	.080	1.366	.154
	Huynh-Feldt	5.755E-02	2.360	2.439E-02	.785	.488	.080	1.853	.175
	Lower-bound	5.755E-02	1.000	5.755E-02	.785	.399	.080	.785	.125
Error(TIME)	Sphericity Assumed	.660	54	1.222E-02					
	Greenhouse-Geisser	.660	15.655	4.215E-02					
	Huynh-Feldt	.660	21.238	3.107E-02					
	Lower-bound	.660	9.000	7.331E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.020	6	.170	16.214	.000	.596	97.282	1.000
	Greenhouse-Geisser	1.020	3.061	.333	16.214	.000	.596	49.629	1.000
	Huynh-Feldt	1.020	4.375	.233	16.214	.000	.596	70.930	1.000
	Lower-bound	1.020	1.000	1.020	16.214	.002	.596	16.214	.955
Error(TIME)	Sphericity Assumed	.692	66	1.048E-02					
	Greenhouse-Geisser	.692	33.670	2.055E-02					
	Huynh-Feldt	.692	48.122	1.438E-02					
	Lower-bound	.692	11.000	6.291E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.450	6	7.499E-02	8.712	.000	.466	52.275	1.000
	Greenhouse-Geisser	.450	3.311	.136	8.712	.000	.466	28.843	.993
	Huynh-Feldt	.450	5.640	7.978E-02	8.712	.000	.466	49.135	1.000
	Lower-bound	.450	1.000	.450	8.712	.014	.466	8.712	.758
TIME * AGE	Sphericity Assumed	.176	6	2.926E-02	3.399	.006	.254	20.395	.917
	Greenhouse-Geisser	.176	3.311	5.303E-02	3.399	.026	.254	11.253	.743
	Huynh-Feldt	.176	5.640	3.113E-02	3.399	.007	.254	19.169	.903
	Lower-bound	.176	1.000	.176	3.399	.095	.254	3.399	.385
Error(TIME)	Sphericity Assumed	.516	60	8.607E-03					
	Greenhouse-Geisser	.516	33.105	1.560E-02					
	Huynh-Feldt	.516	56.396	9.158E-03					
	Lower-bound	.516	10.000	5.164E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.130	6	2.173E-02	2.371	.039	.177	14.226	.776
	Greenhouse-Geisser	.130	3.767	3.461E-02	2.371	.071	.177	8.931	.616
	Huynh-Feldt	.130	5.973	2.182E-02	2.371	.039	.177	14.162	.774
	Lower-bound	.130	1.000	.130	2.371	.152	.177	2.371	.290
Error(TIME)	Sphericity Assumed	.605	66	9.164E-03					
	Greenhouse-Geisser	.605	41.436	1.460E-02					
	Huynh-Feldt	.605	65.703	9.205E-03					
	Lower-bound	.605	11.000	5.498E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	5.276E-02	6	8.793E-03	.933	.478	.085	5.596	.340
	Greenhouse-Geisser	5.276E-02	3.495	1.509E-02	.933	.447	.085	3.260	.249
	Huynh-Feldt	5.276E-02	6.000	8.793E-03	.933	.478	.085	5.596	.340
	Lower-bound	5.276E-02	1.000	5.276E-02	.933	.357	.085	.933	.141
TIME * AGE	Sphericity Assumed	3.915E-02	6	6.524E-03	.692	.657	.065	4.152	.254
	Greenhouse-Geisser	3.915E-02	3.495	1.120E-02	.692	.584	.065	2.419	.192
	Huynh-Feldt	3.915E-02	6.000	6.524E-03	.692	.657	.065	4.152	.254
	Lower-bound	3.915E-02	1.000	3.915E-02	.692	.425	.065	.692	.117
Error(TIME)	Sphericity Assumed	.566	60	9.427E-03					
	Greenhouse-Geisser	.566	34.952	1.618E-02					
	Huynh-Feldt	.566	60.000	9.427E-03					
	Lower-bound	.566	10.000	5.656E-02					

a. Computed using alpha = .05

One-factor (Time) Repeated Measures ANOVA – Control (n=6)

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.988E-02	6	3.313E-03	4.761	.002	.488	28.564	.970
	Greenhouse-Geisser	1.988E-02	2.402	8.277E-03	4.761	.026	.488	11.434	.723
	Huynh-Feldt	1.988E-02	4.776	4.162E-03	4.761	.004	.488	22.739	.933
	Lower-bound	1.988E-02	1.000	1.988E-02	4.761	.081	.488	4.761	.423
Error(TIME)	Sphericity Assumed	2.088E-02	30	6.960E-04					
	Greenhouse-Geisser	2.088E-02	12.009	1.739E-03					
	Huynh-Feldt	2.088E-02	23.882	8.743E-04					
	Lower-bound	2.088E-02	5.000	4.176E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	9.973E-03	6	1.662E-03	2.877	.029	.418	17.264	.793
	Greenhouse-Geisser	9.973E-03	2.118	4.709E-03	2.877	.110	.418	6.093	.426
	Huynh-Feldt	9.973E-03	5.688	1.753E-03	2.877	.033	.418	16.366	.774
	Lower-bound	9.973E-03	1.000	9.973E-03	2.877	.165	.418	2.877	.258
TIME * AGE	Sphericity Assumed	7.015E-03	6	1.169E-03	2.024	.102	.336	12.143	.615
	Greenhouse-Geisser	7.015E-03	2.118	3.313E-03	2.024	.191	.336	4.286	.313
	Huynh-Feldt	7.015E-03	5.688	1.233E-03	2.024	.107	.336	11.512	.596
	Lower-bound	7.015E-03	1.000	7.015E-03	2.024	.228	.336	2.024	.197
Error(TIME)	Sphericity Assumed	1.386E-02	24	5.777E-04					
	Greenhouse-Geisser	1.386E-02	8.471	1.637E-03					
	Huynh-Feldt	1.386E-02	22.752	6.094E-04					
	Lower-bound	1.386E-02	4.000	3.466E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.980E-02	6	3.300E-03	2.208	.070	.306	13.248	.685
	Greenhouse-Geisser	1.980E-02	2.582	7.670E-03	2.208	.142	.306	5.700	.409
	Huynh-Feldt	1.980E-02	5.578	3.550E-03	2.208	.076	.306	12.316	.658
	Lower-bound	1.980E-02	1.000	1.980E-02	2.208	.197	.306	2.208	.228
Error(TIME)	Sphericity Assumed	4.484E-02	30	1.495E-03					
	Greenhouse-Geisser	4.484E-02	12.908	3.474E-03					
	Huynh-Feldt	4.484E-02	27.889	1.608E-03					
	Lower-bound	4.484E-02	5.000	8.967E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	5.295E-03	6	8.825E-04	.527	.782	.116	3.161	.174
	Greenhouse-Geisser	5.295E-03	2.232	2.373E-03	.527	.626	.116	1.176	.114
	Huynh-Feldt	5.295E-03	6.000	8.825E-04	.527	.782	.116	3.161	.174
	Lower-bound	5.295E-03	1.000	5.295E-03	.527	.508	.116	.527	.088
TIME * AGE	Sphericity Assumed	4.630E-03	6	7.716E-04	.461	.830	.103	2.764	.156
	Greenhouse-Geisser	4.630E-03	2.232	2.075E-03	.461	.665	.103	1.028	.105
	Huynh-Feldt	4.630E-03	6.000	7.716E-04	.461	.830	.103	2.764	.156
	Lower-bound	4.630E-03	1.000	4.630E-03	.461	.535	.103	.461	.083
Error(TIME)	Sphericity Assumed	4.021E-02	24	1.675E-03					
	Greenhouse-Geisser	4.021E-02	8.926	4.504E-03					
	Huynh-Feldt	4.021E-02	24.000	1.675E-03					
	Lower-bound	4.021E-02	4.000	1.005E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.328	6	5.470E-02	7.724	.000	.607	46.341	.999
	Greenhouse-Geisser	.328	1.564	.210	7.724	.017	.607	12.081	.777
	Huynh-Feldt	.328	2.150	.153	7.724	.008	.607	16.602	.882
	Lower-bound	.328	1.000	.328	7.724	.039	.607	7.724	.608
Error(TIME)	Sphericity Assumed	.212	30	7.082E-03					
	Greenhouse-Geisser	.212	7.821	2.717E-02					
	Huynh-Feldt	.212	10.748	1.977E-02					
	Lower-bound	.212	5.000	4.249E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.141	6	2.343E-02	3.236	.018	.447	19.418	.845
	Greenhouse-Geisser	.141	1.428	9.842E-02	3.236	.121	.447	4.622	.361
	Huynh-Feldt	.141	2.554	5.503E-02	3.236	.073	.447	8.267	.534
	Lower-bound	.141	1.000	.141	3.236	.146	.447	3.236	.284
TIME * AGE	Sphericity Assumed	3.874E-02	6	6.456E-03	.892	.516	.182	5.351	.283
	Greenhouse-Geisser	3.874E-02	1.428	2.712E-02	.892	.424	.182	1.274	.133
	Huynh-Feldt	3.874E-02	2.554	1.516E-02	.892	.463	.182	2.278	.174
	Lower-bound	3.874E-02	1.000	3.874E-02	.892	.398	.182	.892	.114
Error(TIME)	Sphericity Assumed	.174	24	7.239E-03					
	Greenhouse-Geisser	.174	5.713	3.041E-02					
	Huynh-Feldt	.174	10.218	1.700E-02					
	Lower-bound	.174	4.000	4.343E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	6.349E-02	6	1.058E-02	1.839	.125	.269	11.034	.591
	Greenhouse-Geisser	6.349E-02	2.965	2.142E-02	1.839	.184	.269	5.452	.379
	Huynh-Feldt	6.349E-02	6.000	1.058E-02	1.839	.125	.269	11.034	.591
	Lower-bound	6.349E-02	1.000	6.349E-02	1.839	.233	.269	1.839	.198
Error(TIME)	Sphericity Assumed	.173	30	5.754E-03					
	Greenhouse-Geisser	.173	14.823	1.165E-02					
	Huynh-Feldt	.173	30.000	5.754E-03					
	Lower-bound	.173	5.000	3.452E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.456E-02	6	4.093E-03	.773	.599	.162	4.637	.246
	Greenhouse-Geisser	2.456E-02	2.133	1.151E-02	.773	.499	.162	1.648	.144
	Huynh-Feldt	2.456E-02	5.782	4.247E-03	.773	.595	.162	4.468	.241
	Lower-bound	2.456E-02	1.000	2.456E-02	.773	.429	.162	.773	.106
TIME * AGE	Sphericity Assumed	4.550E-02	6	7.584E-03	1.432	.244	.264	8.591	.450
	Greenhouse-Geisser	4.550E-02	2.133	2.134E-02	1.432	.293	.264	3.054	.233
	Huynh-Feldt	4.550E-02	5.782	7.870E-03	1.432	.246	.264	8.279	.439
	Lower-bound	4.550E-02	1.000	4.550E-02	1.432	.298	.264	1.432	.154
Error(TIME)	Sphericity Assumed	.127	24	5.296E-03					
	Greenhouse-Geisser	.127	8.531	1.490E-02					
	Huynh-Feldt	.127	23.127	5.496E-03					
	Lower-bound	.127	4.000	3.178E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.638E-02	6	2.730E-03	.201	.974	.039	1.208	.093
	Greenhouse-Geisser	1.638E-02	1.713	9.562E-03	.201	.789	.039	.345	.072
	Huynh-Feldt	1.638E-02	2.519	6.503E-03	.201	.864	.039	.507	.077
	Lower-bound	1.638E-02	1.000	1.638E-02	.201	.672	.039	.201	.066
Error(TIME)	Sphericity Assumed	.407	30	1.356E-02					
	Greenhouse-Geisser	.407	8.566	4.748E-02					
	Huynh-Feldt	.407	12.595	3.229E-02					
	Lower-bound	.407	5.000	8.134E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.118	6	1.971E-02	1.607	.188	.287	9.644	.502
	Greenhouse-Geisser	.118	1.468	8.057E-02	1.607	.268	.287	2.359	.206
	Huynh-Feldt	.118	2.688	4.400E-02	1.607	.246	.287	4.320	.297
	Lower-bound	.118	1.000	.118	1.607	.274	.287	1.607	.167
TIME * AGE	Sphericity Assumed	.112	6	1.873E-02	1.528	.212	.276	9.166	.478
	Greenhouse-Geisser	.112	1.468	7.658E-02	1.528	.281	.276	2.242	.198
	Huynh-Feldt	.112	2.688	4.182E-02	1.528	.263	.276	4.106	.283
	Lower-bound	.112	1.000	.112	1.528	.284	.276	1.528	.161
Error(TIME)	Sphericity Assumed	.294	24	1.226E-02					
	Greenhouse-Geisser	.294	5.871	5.013E-02					
	Huynh-Feldt	.294	10.751	2.737E-02					
	Lower-bound	.294	4.000	7.357E-02					

a. Computed using alpha = .05

One-factor (Time) Repeated Measures ANOVA – Plyometrics Fasted (n=6)

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.116	6	1.929E-02	1.823	.128	.267	10.935	.586
	Greenhouse-Geisser	.116	1.135	.102	1.823	.232	.267	2.069	.211
	Huynh-Feldt	.116	1.245	9.299E-02	1.823	.230	.267	2.268	.222
	Lower-bound	.116	1.000	.116	1.823	.235	.267	1.823	.197
Error(TIME)	Sphericity Assumed	.317	30	1.058E-02					
	Greenhouse-Geisser	.317	5.675	5.594E-02					
	Huynh-Feldt	.317	6.223	5.102E-02					
	Lower-bound	.317	5.000	6.350E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	7.899E-02	6	1.317E-02	1.102	.390	.216	6.613	.348
	Greenhouse-Geisser	7.899E-02	1.114	7.092E-02	1.102	.358	.216	1.228	.136
	Huynh-Feldt	7.899E-02	1.622	4.869E-02	1.102	.372	.216	1.788	.163
	Lower-bound	7.899E-02	1.000	7.899E-02	1.102	.353	.216	1.102	.130
TIME * AGE	Sphericity Assumed	3.081E-02	6	5.135E-03	.430	.852	.097	2.579	.148
	Greenhouse-Geisser	3.081E-02	1.114	2.766E-02	.430	.565	.097	.479	.083
	Huynh-Feldt	3.081E-02	1.622	1.899E-02	.430	.629	.097	.697	.092
	Lower-bound	3.081E-02	1.000	3.081E-02	.430	.548	.097	.430	.081
Error(TIME)	Sphericity Assumed	.287	24	1.195E-02					
	Greenhouse-Geisser	.287	4.455	6.435E-02					
	Huynh-Feldt	.287	6.490	4.418E-02					
	Lower-bound	.287	4.000	7.167E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	5.190E-03	6	8.649E-04	1.124	.372	.184	6.745	.372
	Greenhouse-Geisser	5.190E-03	2.714	1.912E-03	1.124	.369	.184	3.051	.230
	Huynh-Feldt	5.190E-03	6.000	8.649E-04	1.124	.372	.184	6.745	.372
	Lower-bound	5.190E-03	1.000	5.190E-03	1.124	.338	.184	1.124	.140
Error(TIME)	Sphericity Assumed	2.308E-02	30	7.694E-04					
	Greenhouse-Geisser	2.308E-02	13.569	1.701E-03					
	Huynh-Feldt	2.308E-02	30.000	7.694E-04					
	Lower-bound	2.308E-02	5.000	4.616E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.233E-03	6	2.056E-04	.240	.959	.057	1.442	.100
	Greenhouse-Geisser	1.233E-03	2.360	5.226E-04	.240	.824	.057	.567	.079
	Huynh-Feldt	1.233E-03	6.000	2.056E-04	.240	.959	.057	1.442	.100
	Lower-bound	1.233E-03	1.000	1.233E-03	.240	.650	.057	.240	.067
TIME * AGE	Sphericity Assumed	2.552E-03	6	4.254E-04	.497	.804	.111	2.984	.166
	Greenhouse-Geisser	2.552E-03	2.360	1.082E-03	.497	.652	.111	1.174	.112
	Huynh-Feldt	2.552E-03	6.000	4.254E-04	.497	.804	.111	2.984	.166
	Lower-bound	2.552E-03	1.000	2.552E-03	.497	.520	.111	.497	.086
Error(TIME)	Sphericity Assumed	2.053E-02	24	8.554E-04					
	Greenhouse-Geisser	2.053E-02	9.440	2.175E-03					
	Huynh-Feldt	2.053E-02	24.000	8.554E-04					
	Lower-bound	2.053E-02	4.000	5.132E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.320	6	5.327E-02	10.101	.000	.669	60.606	1.000
	Greenhouse-Geisser	.320	2.438	.131	10.101	.002	.669	24.623	.970
	Huynh-Feldt	.320	4.928	6.486E-02	10.101	.000	.669	49.773	1.000
	Lower-bound	.320	1.000	.320	10.101	.025	.669	10.101	.720
Error(TIME)	Sphericity Assumed	.158	30	5.273E-03					
	Greenhouse-Geisser	.158	12.188	1.298E-02					
	Huynh-Feldt	.158	24.638	6.421E-03					
	Lower-bound	.158	5.000	3.164E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	7.833E-02	6	1.306E-02	2.184	.080	.353	13.103	.655
	Greenhouse-Geisser	7.833E-02	2.507	3.125E-02	2.184	.158	.353	5.475	.374
	Huynh-Feldt	7.833E-02	6.000	1.306E-02	2.184	.080	.353	13.103	.655
	Lower-bound	7.833E-02	1.000	7.833E-02	2.184	.214	.353	2.184	.209
TIME * AGE	Sphericity Assumed	1.472E-02	6	2.454E-03	.411	.865	.093	2.463	.143
	Greenhouse-Geisser	1.472E-02	2.507	5.874E-03	.411	.717	.093	1.029	.103
	Huynh-Feldt	1.472E-02	6.000	2.454E-03	.411	.865	.093	2.463	.143
	Lower-bound	1.472E-02	1.000	1.472E-02	.411	.557	.093	.411	.079
Error(TIME)	Sphericity Assumed	.143	24	5.978E-03					
	Greenhouse-Geisser	.143	10.027	1.431E-02					
	Huynh-Feldt	.143	24.000	5.978E-03					
	Lower-bound	.143	4.000	3.587E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.387	6	6.445E-02	3.717	.007	.426	22.301	.914
	Greenhouse-Geisser	.387	2.518	.154	3.717	.046	.426	9.361	.624
	Huynh-Feldt	.387	5.283	7.320E-02	3.717	.010	.426	19.637	.881
	Lower-bound	.387	1.000	.387	3.717	.112	.426	3.717	.347
Error(TIME)	Sphericity Assumed	.520	30	1.734E-02					
	Greenhouse-Geisser	.520	12.592	4.131E-02					
	Huynh-Feldt	.520	26.416	1.969E-02					
	Lower-bound	.520	5.000	.104					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.202	6	3.371E-02	1.779	.146	.308	10.675	.551
	Greenhouse-Geisser	.202	2.214	9.135E-02	1.779	.224	.308	3.939	.287
	Huynh-Feldt	.202	6.000	3.371E-02	1.779	.146	.308	10.675	.551
	Lower-bound	.202	1.000	.202	1.779	.253	.308	1.779	.179
TIME * AGE	Sphericity Assumed	6.548E-02	6	1.091E-02	.576	.746	.126	3.456	.188
	Greenhouse-Geisser	6.548E-02	2.214	2.958E-02	.576	.598	.126	1.275	.120
	Huynh-Feldt	6.548E-02	6.000	1.091E-02	.576	.746	.126	3.456	.188
	Lower-bound	6.548E-02	1.000	6.548E-02	.576	.490	.126	.576	.091
Error(TIME)	Sphericity Assumed	.455	24	1.895E-02					
	Greenhouse-Geisser	.455	8.856	5.135E-02					
	Huynh-Feldt	.455	24.000	1.895E-02					
	Lower-bound	.455	4.000	.114					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	5.932E-02	6	9.887E-03	1.744	.145	.259	10.462	.564
	Greenhouse-Geisser	5.932E-02	2.133	2.781E-02	1.744	.221	.259	3.720	.293
	Huynh-Feldt	5.932E-02	3.768	1.575E-02	1.744	.185	.259	6.569	.421
	Lower-bound	5.932E-02	1.000	5.932E-02	1.744	.244	.259	1.744	.191
Error(TIME)	Sphericity Assumed	.170	30	5.671E-03					
	Greenhouse-Geisser	.170	10.667	1.595E-02					
	Huynh-Feldt	.170	18.838	9.030E-03					
	Lower-bound	.170	5.000	3.402E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	7.558E-02	6	1.260E-02	2.843	.031	.415	17.058	.787
	Greenhouse-Geisser	7.558E-02	2.258	3.348E-02	2.843	.107	.415	6.418	.441
	Huynh-Feldt	7.558E-02	6.000	1.260E-02	2.843	.031	.415	17.058	.787
	Lower-bound	7.558E-02	1.000	7.558E-02	2.843	.167	.415	2.843	.256
TIME * AGE	Sphericity Assumed	6.378E-02	6	1.063E-02	2.399	.059	.375	14.396	.703
	Greenhouse-Geisser	6.378E-02	2.258	2.825E-02	2.399	.143	.375	5.417	.379
	Huynh-Feldt	6.378E-02	6.000	1.063E-02	2.399	.059	.375	14.396	.703
	Lower-bound	6.378E-02	1.000	6.378E-02	2.399	.196	.375	2.399	.224
Error(TIME)	Sphericity Assumed	.106	24	4.431E-03					
	Greenhouse-Geisser	.106	9.030	1.178E-02					
	Huynh-Feldt	.106	24.000	4.431E-03					
	Lower-bound	.106	4.000	2.658E-02					

a. Computed using alpha = .05

One-factor (Time) Repeated Measures ANOVA – Plyometrics Fed (n=6)

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	5.671E-02	6	9.452E-03	1.793	.134	.264	10.758	.578
	Greenhouse-Geisser	5.671E-02	2.439	2.325E-02	1.793	.205	.264	4.373	.327
	Huynh-Feldt	5.671E-02	4.933	1.150E-02	1.793	.152	.264	8.844	.512
	Lower-bound	5.671E-02	1.000	5.671E-02	1.793	.238	.264	1.793	.195
Error(TIME)	Sphericity Assumed	.158	30	5.272E-03					
	Greenhouse-Geisser	.158	12.194	1.297E-02					
	Huynh-Feldt	.158	24.664	6.412E-03					
	Lower-bound	.158	5.000	3.163E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	4.797E-02	6	7.995E-03	1.426	.246	.263	8.557	.448
	Greenhouse-Geisser	4.797E-02	2.184	2.197E-02	1.426	.293	.263	3.114	.235
	Huynh-Feldt	4.797E-02	6.000	7.995E-03	1.426	.246	.263	8.557	.448
	Lower-bound	4.797E-02	1.000	4.797E-02	1.426	.298	.263	1.426	.153
TIME * AGE	Sphericity Assumed	2.360E-02	6	3.933E-03	.702	.651	.149	4.209	.225
	Greenhouse-Geisser	2.360E-02	2.184	1.081E-02	.702	.534	.149	1.532	.136
	Huynh-Feldt	2.360E-02	6.000	3.933E-03	.702	.651	.149	4.209	.225
	Lower-bound	2.360E-02	1.000	2.360E-02	.702	.449	.149	.702	.100
Error(TIME)	Sphericity Assumed	.135	24	5.606E-03					
	Greenhouse-Geisser	.135	8.734	1.541E-02					
	Huynh-Feldt	.135	24.000	5.606E-03					
	Lower-bound	.135	4.000	3.364E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	6.346E-03	6	1.058E-03	1.022	.430	.170	6.129	.338
	Greenhouse-Geisser	6.346E-03	3.038	2.089E-03	1.022	.411	.170	3.104	.225
	Huynh-Feldt	6.346E-03	6.000	1.058E-03	1.022	.430	.170	6.129	.338
	Lower-bound	6.346E-03	1.000	6.346E-03	1.022	.359	.170	1.022	.132
Error(TIME)	Sphericity Assumed	3.106E-02	30	1.035E-03					
	Greenhouse-Geisser	3.106E-02	15.191	2.045E-03					
	Huynh-Feldt	3.106E-02	30.000	1.035E-03					
	Lower-bound	3.106E-02	5.000	6.212E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	8.992E-03	6	1.499E-03	1.508	.218	.274	9.050	.473
	Greenhouse-Geisser	8.992E-03	3.015	2.982E-03	1.508	.262	.274	4.548	.301
	Huynh-Feldt	8.992E-03	6.000	1.499E-03	1.508	.218	.274	9.050	.473
	Lower-bound	8.992E-03	1.000	8.992E-03	1.508	.287	.274	1.508	.159
TIME * AGE	Sphericity Assumed	7.214E-03	6	1.202E-03	1.210	.335	.232	7.260	.382
	Greenhouse-Geisser	7.214E-03	3.015	2.393E-03	1.210	.348	.232	3.648	.247
	Huynh-Feldt	7.214E-03	6.000	1.202E-03	1.210	.335	.232	7.260	.382
	Lower-bound	7.214E-03	1.000	7.214E-03	1.210	.333	.232	1.210	.138
Error(TIME)	Sphericity Assumed	2.385E-02	24	9.937E-04					
	Greenhouse-Geisser	2.385E-02	12.061	1.977E-03					
	Huynh-Feldt	2.385E-02	24.000	9.937E-04					
	Lower-bound	2.385E-02	4.000	5.962E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.419	6	6.976E-02	3.919	.005	.439	23.511	.929
	Greenhouse-Geisser	.419	1.460	.287	3.919	.078	.439	5.722	.463
	Huynh-Feldt	.419	1.910	.219	3.919	.059	.439	7.486	.550
	Lower-bound	.419	1.000	.419	3.919	.105	.439	3.919	.362
Error(TIME)	Sphericity Assumed	.534	30	1.780E-02					
	Greenhouse-Geisser	.534	7.302	7.314E-02					
	Huynh-Feldt	.534	9.552	5.591E-02					
	Lower-bound	.534	5.000	.107					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.192	6	3.193E-02	1.791	.144	.309	10.747	.554
	Greenhouse-Geisser	.192	1.401	.137	1.791	.243	.309	2.510	.218
	Huynh-Feldt	.192	2.465	7.772E-02	1.791	.216	.309	4.416	.310
	Lower-bound	.192	1.000	.192	1.791	.252	.309	1.791	.180
TIME * AGE	Sphericity Assumed	.106	6	1.770E-02	.993	.453	.199	5.956	.314
	Greenhouse-Geisser	.106	1.401	7.578E-02	.993	.394	.199	1.391	.141
	Huynh-Feldt	.106	2.465	4.307E-02	.993	.421	.199	2.447	.186
	Lower-bound	.106	1.000	.106	.993	.375	.199	.993	.122
Error(TIME)	Sphericity Assumed	.428	24	1.783E-02					
	Greenhouse-Geisser	.428	5.605	7.635E-02					
	Huynh-Feldt	.428	9.861	4.339E-02					
	Lower-bound	.428	4.000	.107					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.450	6	7.497E-02	7.193	.000	.590	43.157	.998
	Greenhouse-Geisser	.450	1.978	.227	7.193	.012	.590	14.230	.830
	Huynh-Feldt	.450	3.266	.138	7.193	.002	.590	23.494	.956
	Lower-bound	.450	1.000	.450	7.193	.044	.590	7.193	.579
Error(TIME)	Sphericity Assumed	.313	30	1.042E-02					
	Greenhouse-Geisser	.313	9.892	3.161E-02					
	Huynh-Feldt	.313	16.332	1.915E-02					
	Lower-bound	.313	5.000	6.253E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.137	6	2.291E-02	2.307	.067	.366	13.841	.683
	Greenhouse-Geisser	.137	1.824	7.536E-02	2.307	.169	.366	4.208	.318
	Huynh-Feldt	.137	4.110	3.344E-02	2.307	.100	.366	9.482	.545
	Lower-bound	.137	1.000	.137	2.307	.203	.366	2.307	.217
TIME * AGE	Sphericity Assumed	7.435E-02	6	1.239E-02	1.248	.318	.238	7.487	.393
	Greenhouse-Geisser	7.435E-02	1.824	4.076E-02	1.248	.337	.238	2.276	.190
	Huynh-Feldt	7.435E-02	4.110	1.809E-02	1.248	.330	.238	5.129	.308
	Lower-bound	7.435E-02	1.000	7.435E-02	1.248	.327	.238	1.248	.140
Error(TIME)	Sphericity Assumed	.238	24	9.930E-03					
	Greenhouse-Geisser	.238	7.296	3.267E-02					
	Huynh-Feldt	.238	16.441	1.450E-02					
	Lower-bound	.238	4.000	5.958E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	6.363E-02	6	1.060E-02	2.336	.057	.318	14.015	.714
	Greenhouse-Geisser	6.363E-02	2.780	2.289E-02	2.336	.121	.318	6.494	.451
	Huynh-Feldt	6.363E-02	6.000	1.060E-02	2.336	.057	.318	14.015	.714
	Lower-bound	6.363E-02	1.000	6.363E-02	2.336	.187	.318	2.336	.239
Error(TIME)	Sphericity Assumed	.136	30	4.540E-03					
	Greenhouse-Geisser	.136	13.900	9.798E-03					
	Huynh-Feldt	.136	30.000	4.540E-03					
	Lower-bound	.136	5.000	2.724E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.040E-02	6	1.733E-03	.326	.917	.075	1.957	.121
	Greenhouse-Geisser	1.040E-02	2.631	3.950E-03	.326	.783	.075	.858	.093
	Huynh-Feldt	1.040E-02	6.000	1.733E-03	.326	.917	.075	1.957	.121
	Lower-bound	1.040E-02	1.000	1.040E-02	.326	.599	.075	.326	.073
TIME * AGE	Sphericity Assumed	8.696E-03	6	1.449E-03	.273	.944	.064	1.637	.108
	Greenhouse-Geisser	8.696E-03	2.631	3.305E-03	.273	.820	.064	.718	.085
	Huynh-Feldt	8.696E-03	6.000	1.449E-03	.273	.944	.064	1.637	.108
	Lower-bound	8.696E-03	1.000	8.696E-03	.273	.629	.064	.273	.069
Error(TIME)	Sphericity Assumed	.127	24	5.312E-03					
	Greenhouse-Geisser	.127	10.525	1.211E-02					
	Huynh-Feldt	.127	24.000	5.312E-03					
	Lower-bound	.127	4.000	3.187E-02					

a. Computed using alpha = .05

One-factor (Time) Repeated Measures ANOVA – Resistance Training Fasted (n=6)

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	9.991E-03	6	1.665E-03	1.293	.298	.244	7.756	.407
	Greenhouse-Geisser	9.991E-03	1.609	6.211E-03	1.293	.326	.244	2.079	.183
	Huynh-Feldt	9.991E-03	2.527	3.954E-03	1.293	.324	.244	3.266	.236
	Lower-bound	9.991E-03	1.000	9.991E-03	1.293	.319	.244	1.293	.144
Error(TIME)	Sphericity Assumed	3.092E-02	24	1.288E-03					
	Greenhouse-Geisser	3.092E-02	6.434	4.805E-03					
	Huynh-Feldt	3.092E-02	10.107	3.059E-03					
	Lower-bound	3.092E-02	4.000	7.729E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	5.745E-03	6	9.575E-04	.634	.701	.175	3.805	.192
	Greenhouse-Geisser	5.745E-03	1.493	3.847E-03	.634	.529	.175	.947	.102
	Huynh-Feldt	5.745E-03	3.629	1.583E-03	.634	.635	.175	2.301	.148
	Lower-bound	5.745E-03	1.000	5.745E-03	.634	.484	.175	.634	.089
TIME * AGE	Sphericity Assumed	3.739E-03	6	6.232E-04	.413	.861	.121	2.477	.136
	Greenhouse-Geisser	3.739E-03	1.493	2.504E-03	.413	.631	.121	.616	.084
	Huynh-Feldt	3.739E-03	3.629	1.030E-03	.413	.780	.121	1.498	.111
	Lower-bound	3.739E-03	1.000	3.739E-03	.413	.566	.121	.413	.075
Error(TIME)	Sphericity Assumed	2.718E-02	18	1.510E-03					
	Greenhouse-Geisser	2.718E-02	4.480	6.066E-03					
	Huynh-Feldt	2.718E-02	10.887	2.496E-03					
	Lower-bound	2.718E-02	3.000	9.059E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.282E-02	6	2.137E-03	1.593	.192	.285	9.556	.498
	Greenhouse-Geisser	1.282E-02	1.933	6.635E-03	1.593	.263	.285	3.078	.240
	Huynh-Feldt	1.282E-02	3.707	3.459E-03	1.593	.230	.285	5.904	.363
	Lower-bound	1.282E-02	1.000	1.282E-02	1.593	.276	.285	1.593	.166
Error(TIME)	Sphericity Assumed	3.221E-02	24	1.342E-03					
	Greenhouse-Geisser	3.221E-02	7.731	4.166E-03					
	Huynh-Feldt	3.221E-02	14.829	2.172E-03					
	Lower-bound	3.221E-02	4.000	8.052E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.135E-02	6	1.892E-03	1.298	.308	.302	7.786	.380
	Greenhouse-Geisser	1.135E-02	1.408	8.062E-03	1.298	.341	.302	1.827	.154
	Huynh-Feldt	1.135E-02	3.165	3.586E-03	1.298	.332	.302	4.108	.248
	Lower-bound	1.135E-02	1.000	1.135E-02	1.298	.337	.302	1.298	.129
TIME * AGE	Sphericity Assumed	5.968E-03	6	9.947E-04	.682	.666	.185	4.094	.205
	Greenhouse-Geisser	5.968E-03	1.408	4.239E-03	.682	.504	.185	.961	.104
	Huynh-Feldt	5.968E-03	3.165	1.885E-03	.682	.591	.185	2.160	.147
	Lower-bound	5.968E-03	1.000	5.968E-03	.682	.469	.185	.682	.092
Error(TIME)	Sphericity Assumed	2.624E-02	18	1.458E-03					
	Greenhouse-Geisser	2.624E-02	4.224	6.212E-03					
	Huynh-Feldt	2.624E-02	9.496	2.763E-03					
	Lower-bound	2.624E-02	3.000	8.747E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.312	6	5.199E-02	3.120	.021	.438	18.720	.829
	Greenhouse-Geisser	.312	1.339	.233	3.120	.132	.438	4.177	.335
	Huynh-Feldt	.312	1.764	.177	3.120	.110	.438	5.504	.404
	Lower-bound	.312	1.000	.312	3.120	.152	.438	3.120	.275
Error(TIME)	Sphericity Assumed	.400	24	1.666E-02					
	Greenhouse-Geisser	.400	5.356	7.468E-02					
	Huynh-Feldt	.400	7.057	5.668E-02					
	Lower-bound	.400	4.000	9.999E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.234	6	3.894E-02	2.599	.054	.464	15.592	.703
	Greenhouse-Geisser	.234	1.328	.176	2.599	.187	.464	3.450	.252
	Huynh-Feldt	.234	2.773	8.425E-02	2.599	.124	.464	7.207	.425
	Lower-bound	.234	1.000	.234	2.599	.205	.464	2.599	.208
TIME * AGE	Sphericity Assumed	.130	6	2.170E-02	1.448	.251	.326	8.690	.423
	Greenhouse-Geisser	.130	1.328	9.809E-02	1.448	.314	.326	1.923	.162
	Huynh-Feldt	.130	2.773	4.696E-02	1.448	.296	.326	4.017	.252
	Lower-bound	.130	1.000	.130	1.448	.315	.326	1.448	.139
Error(TIME)	Sphericity Assumed	.270	18	1.498E-02					
	Greenhouse-Geisser	.270	3.983	6.772E-02					
	Huynh-Feldt	.270	8.320	3.242E-02					
	Lower-bound	.270	3.000	8.991E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.468	6	7.799E-02	1.572	.198	.282	9.432	.491
	Greenhouse-Geisser	.468	1.335	.350	1.572	.276	.282	2.099	.192
	Huynh-Feldt	.468	1.755	.267	1.572	.270	.282	2.758	.224
	Lower-bound	.468	1.000	.468	1.572	.278	.282	1.572	.164
Error(TIME)	Sphericity Assumed	1.191	24	4.961E-02					
	Greenhouse-Geisser	1.191	5.341	.223					
	Huynh-Feldt	1.191	7.019	.170					
	Lower-bound	1.191	4.000	.298					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.179	6	2.977E-02	.492	.806	.141	2.951	.155
	Greenhouse-Geisser	.179	1.155	.155	.492	.554	.141	.568	.084
	Huynh-Feldt	.179	2.047	8.725E-02	.492	.638	.141	1.007	.100
	Lower-bound	.179	1.000	.179	.492	.534	.141	.492	.080
TIME * AGE	Sphericity Assumed	.101	6	1.691E-02	.279	.939	.085	1.677	.105
	Greenhouse-Geisser	.101	1.155	8.782E-02	.279	.661	.085	.323	.069
	Huynh-Feldt	.101	2.047	4.956E-02	.279	.770	.085	.572	.078
	Lower-bound	.101	1.000	.101	.279	.634	.085	.279	.067
Error(TIME)	Sphericity Assumed	1.089	18	6.051E-02					
	Greenhouse-Geisser	1.089	3.466	.314					
	Huynh-Feldt	1.089	6.141	.177					
	Lower-bound	1.089	3.000	.363					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	7.423E-02	6	1.237E-02	.921	.494	.156	5.526	.305
	Greenhouse-Geisser	7.423E-02	2.645	2.806E-02	.921	.447	.156	2.436	.191
	Huynh-Feldt	7.423E-02	5.890	1.260E-02	.921	.493	.156	5.425	.302
	Lower-bound	7.423E-02	1.000	7.423E-02	.921	.381	.156	.921	.124
Error(TIME)	Sphericity Assumed	.403	30	1.343E-02					
	Greenhouse-Geisser	.403	13.225	3.047E-02					
	Huynh-Feldt	.403	29.448	1.368E-02					
	Lower-bound	.403	5.000	8.059E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	7.627E-02	6	1.271E-02	.870	.531	.179	5.217	.276
	Greenhouse-Geisser	7.627E-02	2.465	3.094E-02	.870	.470	.179	2.144	.168
	Huynh-Feldt	7.627E-02	6.000	1.271E-02	.870	.531	.179	5.217	.276
	Lower-bound	7.627E-02	1.000	7.627E-02	.870	.404	.179	.870	.113
TIME * AGE	Sphericity Assumed	5.206E-02	6	8.677E-03	.594	.732	.129	3.561	.193
	Greenhouse-Geisser	5.206E-02	2.465	2.112E-02	.594	.604	.129	1.463	.128
	Huynh-Feldt	5.206E-02	6.000	8.677E-03	.594	.732	.129	3.561	.193
	Lower-bound	5.206E-02	1.000	5.206E-02	.594	.484	.129	.594	.093
Error(TIME)	Sphericity Assumed	.351	24	1.462E-02					
	Greenhouse-Geisser	.351	9.862	3.558E-02					
	Huynh-Feldt	.351	24.000	1.462E-02					
	Lower-bound	.351	4.000	8.772E-02					

a. Computed using alpha = .05

One-factor (Time) Repeated Measures ANOVA – Resistance Training Fed (n=6)

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.799E-02	6	4.665E-03	.943	.480	.159	5.657	.312
	Greenhouse-Geisser	2.799E-02	2.323	1.205E-02	.943	.430	.159	2.190	.183
	Huynh-Feldt	2.799E-02	4.459	6.277E-03	.943	.465	.159	4.204	.261
	Lower-bound	2.799E-02	1.000	2.799E-02	.943	.376	.159	.943	.125
Error(TIME)	Sphericity Assumed	.148	30	4.949E-03					
	Greenhouse-Geisser	.148	11.615	1.278E-02					
	Huynh-Feldt	.148	22.296	6.659E-03					
	Lower-bound	.148	5.000	2.969E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.325E-02	6	3.874E-03	.695	.656	.148	4.170	.223
	Greenhouse-Geisser	2.325E-02	1.982	1.173E-02	.695	.526	.148	1.378	.129
	Huynh-Feldt	2.325E-02	4.903	4.741E-03	.695	.631	.148	3.407	.199
	Lower-bound	2.325E-02	1.000	2.325E-02	.695	.451	.148	.695	.100
TIME * AGE	Sphericity Assumed	1.467E-02	6	2.445E-03	.439	.846	.099	2.632	.150
	Greenhouse-Geisser	1.467E-02	1.982	7.401E-03	.439	.658	.099	.869	.099
	Huynh-Feldt	1.467E-02	4.903	2.992E-03	.439	.813	.099	2.150	.137
	Lower-bound	1.467E-02	1.000	1.467E-02	.439	.544	.099	.439	.081
Error(TIME)	Sphericity Assumed	.134	24	5.575E-03					
	Greenhouse-Geisser	.134	7.929	1.687E-02					
	Huynh-Feldt	.134	19.611	6.822E-03					
	Lower-bound	.134	4.000	3.345E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.580E-02	6	2.634E-03	4.956	.001	.498	29.735	.976
	Greenhouse-Geisser	1.580E-02	2.325	6.797E-03	4.956	.024	.498	11.523	.729
	Huynh-Feldt	1.580E-02	4.468	3.537E-03	4.956	.004	.498	22.143	.930
	Lower-bound	1.580E-02	1.000	1.580E-02	4.956	.077	.498	4.956	.437
Error(TIME)	Sphericity Assumed	1.595E-02	30	5.315E-04					
	Greenhouse-Geisser	1.595E-02	11.626	1.372E-03					
	Huynh-Feldt	1.595E-02	22.340	7.137E-04					
	Lower-bound	1.595E-02	5.000	3.189E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.749E-03	6	4.582E-04	.797	.581	.166	4.783	.254
	Greenhouse-Geisser	2.749E-03	2.147	1.280E-03	.797	.490	.166	1.712	.147
	Huynh-Feldt	2.749E-03	5.876	4.679E-04	.797	.580	.166	4.684	.250
	Lower-bound	2.749E-03	1.000	2.749E-03	.797	.422	.166	.797	.107
TIME * AGE	Sphericity Assumed	2.151E-03	6	3.585E-04	.624	.710	.135	3.742	.202
	Greenhouse-Geisser	2.151E-03	2.147	1.002E-03	.624	.569	.135	1.339	.125
	Huynh-Feldt	2.151E-03	5.876	3.661E-04	.624	.707	.135	3.665	.200
	Lower-bound	2.151E-03	1.000	2.151E-03	.624	.474	.135	.624	.095
Error(TIME)	Sphericity Assumed	1.379E-02	24	5.748E-04					
	Greenhouse-Geisser	1.379E-02	8.590	1.606E-03					
	Huynh-Feldt	1.379E-02	23.502	5.869E-04					
	Lower-bound	1.379E-02	4.000	3.449E-03					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.121	6	2.014E-02	2.289	.069	.364	13.737	.679
	Greenhouse-Geisser	.121	2.080	5.808E-02	2.289	.161	.364	4.763	.345
	Huynh-Feldt	.121	4.377	2.761E-02	2.289	.097	.364	10.021	.563
	Lower-bound	.121	1.000	.121	2.289	.205	.364	2.289	.216
Error(TIME)	Sphericity Assumed	.211	24	8.796E-03					
	Greenhouse-Geisser	.211	8.322	2.537E-02					
	Huynh-Feldt	.211	17.508	1.206E-02					
	Lower-bound	.211	4.000	5.278E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: COR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.183E-02	6	3.639E-03	.352	.900	.105	2.112	.121
	Greenhouse-Geisser	2.183E-02	1.889	1.156E-02	.352	.707	.105	.665	.083
	Huynh-Feldt	2.183E-02	6.000	3.639E-03	.352	.900	.105	2.112	.121
	Lower-bound	2.183E-02	1.000	2.183E-02	.352	.595	.105	.352	.072
TIME * AGE	Sphericity Assumed	2.501E-02	6	4.169E-03	.403	.867	.118	2.419	.133
	Greenhouse-Geisser	2.501E-02	1.889	1.324E-02	.403	.675	.118	.762	.089
	Huynh-Feldt	2.501E-02	6.000	4.169E-03	.403	.867	.118	2.419	.133
	Lower-bound	2.501E-02	1.000	2.501E-02	.403	.571	.118	.403	.075
Error(TIME)	Sphericity Assumed	.186	18	1.034E-02					
	Greenhouse-Geisser	.186	5.668	3.284E-02					
	Huynh-Feldt	.186	18.000	1.034E-02					
	Lower-bound	.186	3.000	6.203E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.780	6	.130	12.879	.000	.720	77.275	1.000
	Greenhouse-Geisser	.780	2.490	.313	12.879	.001	.720	32.067	.993
	Huynh-Feldt	.780	5.155	.151	12.879	.000	.720	66.390	1.000
	Lower-bound	.780	1.000	.780	12.879	.016	.720	12.879	.816
Error(TIME)	Sphericity Assumed	.303	30	1.009E-02					
	Greenhouse-Geisser	.303	12.449	2.431E-02					
	Huynh-Feldt	.303	25.774	1.174E-02					
	Lower-bound	.303	5.000	6.054E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.276	6	4.598E-02	4.654	.003	.538	27.926	.957
	Greenhouse-Geisser	.276	2.762	9.989E-02	4.654	.026	.538	12.854	.732
	Huynh-Feldt	.276	6.000	4.598E-02	4.654	.003	.538	27.926	.957
	Lower-bound	.276	1.000	.276	4.654	.097	.538	4.654	.379
TIME * AGE	Sphericity Assumed	6.562E-02	6	1.094E-02	1.107	.387	.217	6.643	.350
	Greenhouse-Geisser	6.562E-02	2.762	2.376E-02	1.107	.383	.217	3.058	.217
	Huynh-Feldt	6.562E-02	6.000	1.094E-02	1.107	.387	.217	6.643	.350
	Lower-bound	6.562E-02	1.000	6.562E-02	1.107	.352	.217	1.107	.130
Error(TIME)	Sphericity Assumed	.237	24	9.878E-03					
	Greenhouse-Geisser	.237	11.047	2.146E-02					
	Huynh-Feldt	.237	24.000	9.878E-03					
	Lower-bound	.237	4.000	5.927E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	8.079E-02	6	1.347E-02	1.252	.309	.200	7.512	.413
	Greenhouse-Geisser	8.079E-02	2.448	3.301E-02	1.252	.327	.200	3.065	.238
	Huynh-Feldt	8.079E-02	4.970	1.625E-02	1.252	.315	.200	6.223	.366
	Lower-bound	8.079E-02	1.000	8.079E-02	1.252	.314	.200	1.252	.150
Error(TIME)	Sphericity Assumed	.323	30	1.075E-02					
	Greenhouse-Geisser	.323	12.238	2.636E-02					
	Huynh-Feldt	.323	24.852	1.298E-02					
	Lower-bound	.323	5.000	6.453E-02					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure: TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	1.815E-02	6	3.026E-03	.246	.956	.058	1.479	.102
	Greenhouse-Geisser	1.815E-02	2.184	8.312E-03	.246	.804	.058	.538	.078
	Huynh-Feldt	1.815E-02	6.000	3.026E-03	.246	.956	.058	1.479	.102
	Lower-bound	1.815E-02	1.000	1.815E-02	.246	.646	.058	.246	.068
TIME * AGE	Sphericity Assumed	2.794E-02	6	4.656E-03	.379	.885	.087	2.275	.134
	Greenhouse-Geisser	2.794E-02	2.184	1.279E-02	.379	.712	.087	.828	.095
	Huynh-Feldt	2.794E-02	6.000	4.656E-03	.379	.885	.087	2.275	.134
	Lower-bound	2.794E-02	1.000	2.794E-02	.379	.571	.087	.379	.077
Error(TIME)	Sphericity Assumed	.295	24	1.228E-02					
	Greenhouse-Geisser	.295	8.737	3.373E-02					
	Huynh-Feldt	.295	24.000	1.228E-02					
	Lower-bound	.295	4.000	7.368E-02					

a. Computed using alpha = .05

2 x 2 Repeated Measures ANOVA comparing exercise modes (RT vs. PLY) and feeding (fed vs. fasted) (n=12)

Tests of Within-Subjects Effects

Measure:BAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.120	6	.020	2.680	.015	.061	16.083	.861
	Greenhouse-Geisser	.120	4.147	.029	2.680	.032	.061	11.117	.748
	Huynh-Feldt	.120	5.010	.024	2.680	.023	.061	13.429	.808
	Lower-bound	.120	1.000	.120	2.680	.109	.061	2.680	.359
TIME * fastfed	Sphericity Assumed	.034	6	.006	.760	.602	.018	4.559	.299
	Greenhouse-Geisser	.034	4.147	.008	.760	.557	.018	3.151	.245
	Huynh-Feldt	.034	5.010	.007	.760	.580	.018	3.807	.271
	Lower-bound	.034	1.000	.034	.760	.388	.018	.760	.136
TIME * plyrt	Sphericity Assumed	.034	6	.006	.768	.595	.018	4.611	.302
	Greenhouse-Geisser	.034	4.147	.008	.768	.551	.018	3.187	.248
	Huynh-Feldt	.034	5.010	.007	.768	.574	.018	3.850	.274
	Lower-bound	.034	1.000	.034	.768	.386	.018	.768	.137
TIME * fastfed * plyrt	Sphericity Assumed	.034	6	.006	.769	.595	.018	4.611	.303
	Greenhouse-Geisser	.034	4.147	.008	.769	.551	.018	3.187	.248
	Huynh-Feldt	.034	5.010	.007	.769	.574	.018	3.850	.274
	Lower-bound	.034	1.000	.034	.769	.386	.018	.769	.137
Error(TIME)	Sphericity Assumed	1.831	246	.007					
	Greenhouse-Geisser	1.831	170.037	.011					
	Huynh-Feldt	1.831	205.404	.009					
	Lower-bound	1.831	41.000	.045					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TRAP

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.057	6	.010	9.418	.000	.187	56.511	1.000
	Greenhouse-Geisser	.057	4.022	.014	9.418	.000	.187	37.885	1.000
	Huynh-Feldt	.057	4.841	.012	9.418	.000	.187	45.594	1.000
	Lower-bound	.057	1.000	.057	9.418	.004	.187	9.418	.850
TIME * fastfed	Sphericity Assumed	.008	6	.001	1.344	.238	.032	8.063	.522
	Greenhouse-Geisser	.008	4.022	.002	1.344	.256	.032	5.406	.414
	Huynh-Feldt	.008	4.841	.002	1.344	.249	.032	6.506	.461
	Lower-bound	.008	1.000	.008	1.344	.253	.032	1.344	.205
TIME * plyrt	Sphericity Assumed	.007	6	.001	1.156	.331	.027	6.937	.453
	Greenhouse-Geisser	.007	4.022	.002	1.156	.332	.027	4.650	.359
	Huynh-Feldt	.007	4.841	.001	1.156	.332	.027	5.597	.399
	Lower-bound	.007	1.000	.007	1.156	.289	.027	1.156	.183
TIME * fastfed * plyrt	Sphericity Assumed	.002	6	.000	.340	.915	.008	2.043	.146
	Greenhouse-Geisser	.002	4.022	.001	.340	.851	.008	1.370	.126
	Huynh-Feldt	.002	4.841	.000	.340	.883	.008	1.648	.134
	Lower-bound	.002	1.000	.002	.340	.563	.008	.340	.088
Error(TIME)	Sphericity Assumed	.248	246	.001					
	Greenhouse-Geisser	.248	164.917	.002					
	Huynh-Feldt	.248	198.479	.001					
	Lower-bound	.248	41.000	.006					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:CORTISOL

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.667	6	.444	42.625	.000	.516	255.748	1.000
	Greenhouse-Geisser	2.667	3.068	.869	42.625	.000	.516	130.754	1.000
	Huynh-Feldt	2.667	3.600	.741	42.625	.000	.516	153.467	1.000
	Lower-bound	2.667	1.000	2.667	42.625	.000	.516	42.625	1.000
TIME * fastfed	Sphericity Assumed	.104	6	.017	1.655	.133	.040	9.930	.627
	Greenhouse-Geisser	.104	3.068	.034	1.655	.179	.040	5.077	.430
	Huynh-Feldt	.104	3.600	.029	1.655	.170	.040	5.959	.471
	Lower-bound	.104	1.000	.104	1.655	.206	.040	1.655	.241
TIME * plyrt	Sphericity Assumed	.135	6	.023	2.159	.048	.051	12.957	.763
	Greenhouse-Geisser	.135	3.068	.044	2.159	.095	.051	6.624	.545
	Huynh-Feldt	.135	3.600	.038	2.159	.084	.051	7.775	.594
	Lower-bound	.135	1.000	.135	2.159	.150	.051	2.159	.300
TIME * fastfed * plyrt	Sphericity Assumed	.018	6	.003	.283	.945	.007	1.699	.127
	Greenhouse-Geisser	.018	3.068	.006	.283	.842	.007	.869	.104
	Huynh-Feldt	.018	3.600	.005	.283	.871	.007	1.019	.108
	Lower-bound	.018	1.000	.018	.283	.598	.007	.283	.081
Error(TIME)	Sphericity Assumed	2.503	240	.010					
	Greenhouse-Geisser	2.503	122.703	.020					
	Huynh-Feldt	2.503	144.017	.017					
	Lower-bound	2.503	40.000	.063					

a. Computed using alpha = .05

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
corpre	Equal variances assumed	.082	.776	.518	45	.607	.01688	.03258	-.04874	.08249
	Equal variances not assumed			.518	44.751	.607	.01688	.03260	-.04879	.08255
corpost	Equal variances assumed	.263	.611	-2.357	46	.023	-.08025	.03405	-.14880	-.01171
	Equal variances not assumed			-2.357	43.895	.023	-.08025	.03405	-.14889	-.01162
cor15min	Equal variances assumed	.036	.850	-2.058	46	.045	-.06788	.03299	-.13428	-.00147
	Equal variances not assumed			-2.058	45.069	.045	-.06788	.03299	-.13432	-.00144
cor30min	Equal variances assumed	1.335	.254	-2.670	46	.010	-.12401	.04644	-.21750	-.03052
	Equal variances not assumed			-2.670	42.333	.011	-.12401	.04644	-.21772	-.03030
cor60min	Equal variances assumed	.090	.765	-1.374	46	.176	-.05760	.04193	-.14199	.02680
	Equal variances not assumed			-1.374	45.205	.176	-.05760	.04193	-.14203	.02684
cor120min	Equal variances assumed	.177	.676	.115	45	.909	.00453	.03921	-.07444	.08350
	Equal variances not assumed			.116	44.990	.909	.00453	.03918	-.07439	.08345
cor24hr	Equal variances assumed	.564	.456	.086	44	.932	.00276	.03196	-.06165	.06716
	Equal variances not assumed			.086	41.175	.932	.00276	.03221	-.06228	.06779

Tests of Within-Subjects Effects

Measure:PTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	2.396	6	.399	27.063	.000	.404	162.375	1.000
	Greenhouse-Geisser	2.396	3.448	.695	27.063	.000	.404	93.319	1.000
	Huynh-Feldt	2.396	4.096	.585	27.063	.000	.404	110.854	1.000
	Lower-bound	2.396	1.000	2.396	27.063	.000	.404	27.063	.999
TIME * fastfed	Sphericity Assumed	.054	6	.009	.607	.724	.015	3.643	.241
	Greenhouse-Geisser	.054	3.448	.016	.607	.634	.015	2.094	.184
	Huynh-Feldt	.054	4.096	.013	.607	.662	.015	2.487	.199
	Lower-bound	.054	1.000	.054	.607	.440	.015	.607	.118
TIME * plyrt	Sphericity Assumed	.164	6	.027	1.852	.090	.044	11.112	.685
	Greenhouse-Geisser	.164	3.448	.048	1.852	.132	.044	6.386	.509
	Huynh-Feldt	.164	4.096	.040	1.852	.120	.044	7.586	.560
	Lower-bound	.164	1.000	.164	1.852	.181	.044	1.852	.264
TIME * fastfed * plyrt	Sphericity Assumed	.120	6	.020	1.355	.234	.033	8.130	.526
	Greenhouse-Geisser	.120	3.448	.035	1.355	.257	.033	4.672	.382
	Huynh-Feldt	.120	4.096	.029	1.355	.251	.033	5.550	.422
	Lower-bound	.120	1.000	.120	1.355	.251	.033	1.355	.206
Error(TIME)	Sphericity Assumed	3.541	240	.015					
	Greenhouse-Geisser	3.541	137.931	.026					
	Huynh-Feldt	3.541	163.848	.022					
	Lower-bound	3.541	40.000	.089					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TEST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TIME	Sphericity Assumed	.427	6	.071	8.762	.000	.180	52.571	1.000
	Greenhouse-Geisser	.427	4.703	.091	8.762	.000	.180	41.210	1.000
	Huynh-Feldt	.427	5.806	.074	8.762	.000	.180	50.875	1.000
	Lower-bound	.427	1.000	.427	8.762	.005	.180	8.762	.823
TIME * fastfed	Sphericity Assumed	.052	6	.009	1.059	.388	.026	6.353	.415
	Greenhouse-Geisser	.052	4.703	.011	1.059	.383	.026	4.980	.360
	Huynh-Feldt	.052	5.806	.009	1.059	.387	.026	6.148	.407
	Lower-bound	.052	1.000	.052	1.059	.310	.026	1.059	.171
TIME * plyrt	Sphericity Assumed	.056	6	.009	1.155	.331	.028	6.932	.452
	Greenhouse-Geisser	.056	4.703	.012	1.155	.333	.028	5.434	.392
	Huynh-Feldt	.056	5.806	.010	1.155	.331	.028	6.709	.444
	Lower-bound	.056	1.000	.056	1.155	.289	.028	1.155	.183
TIME * fastfed * plyrt	Sphericity Assumed	.019	6	.003	.399	.879	.010	2.394	.165
	Greenhouse-Geisser	.019	4.703	.004	.399	.838	.010	1.877	.150
	Huynh-Feldt	.019	5.806	.003	.399	.874	.010	2.317	.163
	Lower-bound	.019	1.000	.019	.399	.531	.010	.399	.095
Error(TIME)	Sphericity Assumed	1.950	240	.008					
	Greenhouse-Geisser	1.950	188.134	.010					
	Huynh-Feldt	1.950	232.257	.008					
	Lower-bound	1.950	40.000	.049					

a. Computed using alpha = .05

One-factor (trial) Repeated Measures ANOVA comparing exercise trials and control (n=6)

Tests of Within-Subjects Effects

Measure:BAPPRE

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.311	4	.078	1.916	.147	.277	7.665	.477
	Greenhouse-Geisser	.311	2.147	.145	1.916	.193	.277	4.114	.320
	Huynh-Feldt	.311	3.813	.082	1.916	.151	.277	7.307	.462
	Lower-bound	.311	1.000	.311	1.916	.225	.277	1.916	.205
Error(TRIAL)	Sphericity Assumed	.812	20	.041					
	Greenhouse-Geisser	.812	10.733	.076					
	Huynh-Feldt	.812	19.066	.043					
	Lower-bound	.812	5.000	.162					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:BAPPOST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.436	4	.109	2.335	.091	.318	9.342	.568
	Greenhouse-Geisser	.436	1.980	.220	2.335	.148	.318	4.625	.363
	Huynh-Feldt	.436	3.272	.133	2.335	.108	.318	7.642	.501
	Lower-bound	.436	1.000	.436	2.335	.187	.318	2.335	.239
Error(TRIAL)	Sphericity Assumed	.933	20	.047					
	Greenhouse-Geisser	.933	9.901	.094					
	Huynh-Feldt	.933	16.361	.057					
	Lower-bound	.933	5.000	.187					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:BAP15MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.375	4	.094	1.976	.137	.283	7.903	.490
	Greenhouse-Geisser	.375	2.024	.185	1.976	.188	.283	3.999	.317
	Huynh-Feldt	.375	3.408	.110	1.976	.151	.283	6.734	.443
	Lower-bound	.375	1.000	.375	1.976	.219	.283	1.976	.209
Error(TRIAL)	Sphericity Assumed	.948	20	.047					
	Greenhouse-Geisser	.948	10.119	.094					
	Huynh-Feldt	.948	17.041	.056					
	Lower-bound	.948	5.000	.190					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:BAP30MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.299	4	.075	1.683	.193	.252	6.732	.423
	Greenhouse-Geisser	.299	1.705	.176	1.683	.240	.252	2.869	.249
	Huynh-Feldt	.299	2.497	.120	1.683	.224	.252	4.202	.313
	Lower-bound	.299	1.000	.299	1.683	.251	.252	1.683	.186
Error(TRIAL)	Sphericity Assumed	.889	20	.044					
	Greenhouse-Geisser	.889	8.523	.104					
	Huynh-Feldt	.889	12.483	.071					
	Lower-bound	.889	5.000	.178					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:BAP60MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.284	4	.071	1.686	.193	.252	6.743	.423
	Greenhouse-Geisser	.284	1.758	.161	1.686	.239	.252	2.964	.254
	Huynh-Feldt	.284	2.638	.108	1.686	.220	.252	4.447	.325
	Lower-bound	.284	1.000	.284	1.686	.251	.252	1.686	.186
Error(TRIAL)	Sphericity Assumed	.842	20	.042					
	Greenhouse-Geisser	.842	8.792	.096					
	Huynh-Feldt	.842	13.189	.064					
	Lower-bound	.842	5.000	.168					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:BAP120MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.429	4	.107	2.640	.064	.346	10.560	.628
	Greenhouse-Geisser	.429	2.030	.211	2.640	.119	.346	5.361	.411
	Huynh-Feldt	.429	3.429	.125	2.640	.076	.346	9.053	.573
	Lower-bound	.429	1.000	.429	2.640	.165	.346	2.640	.263
Error(TRIAL)	Sphericity Assumed	.812	20	.041					
	Greenhouse-Geisser	.812	10.152	.080					
	Huynh-Feldt	.812	17.146	.047					
	Lower-bound	.812	5.000	.162					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:BAP24HR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.144	4	.036	1.682	.203	.296	6.727	.401
	Greenhouse-Geisser	.144	1.249	.115	1.682	.261	.296	2.101	.194
	Huynh-Feldt	.144	1.544	.093	1.682	.255	.296	2.596	.220
	Lower-bound	.144	1.000	.144	1.682	.264	.296	1.682	.172
Error(TRIAL)	Sphericity Assumed	.342	16	.021					
	Greenhouse-Geisser	.342	4.997	.068					
	Huynh-Feldt	.342	6.175	.055					
	Lower-bound	.342	4.000	.085					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TRAPPRE

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.019	4	.005	.801	.539	.138	3.202	.211
	Greenhouse-Geisser	.019	2.358	.008	.801	.491	.138	1.887	.162
	Huynh-Feldt	.019	4.000	.005	.801	.539	.138	3.202	.211
	Lower-bound	.019	1.000	.019	.801	.412	.138	.801	.114
Error(TRIAL)	Sphericity Assumed	.118	20	.006					
	Greenhouse-Geisser	.118	11.788	.010					
	Huynh-Feldt	.118	20.000	.006					
	Lower-bound	.118	5.000	.024					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TRAPPOST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.014	4	.004	.653	.632	.115	2.611	.178
	Greenhouse-Geisser	.014	2.248	.006	.653	.556	.115	1.467	.137
	Huynh-Feldt	.014	4.000	.004	.653	.632	.115	2.611	.178
	Lower-bound	.014	1.000	.014	.653	.456	.115	.653	.102
Error(TRIAL)	Sphericity Assumed	.110	20	.005					
	Greenhouse-Geisser	.110	11.239	.010					
	Huynh-Feldt	.110	20.000	.005					
	Lower-bound	.110	5.000	.022					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TRAP15MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.004	4	.001	.172	.950	.033	.688	.079
	Greenhouse-Geisser	.004	2.114	.002	.172	.855	.033	.363	.071
	Huynh-Feldt	.004	3.702	.001	.172	.942	.033	.637	.078
	Lower-bound	.004	1.000	.004	.172	.696	.033	.172	.063
Error(TRIAL)	Sphericity Assumed	.112	20	.006					
	Greenhouse-Geisser	.112	10.571	.011					
	Huynh-Feldt	.112	18.512	.006					
	Lower-bound	.112	5.000	.022					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TRAP30MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.006	4	.001	.268	.895	.051	1.072	.097
	Greenhouse-Geisser	.006	1.974	.003	.268	.768	.051	.529	.081
	Huynh-Feldt	.006	3.253	.002	.268	.862	.051	.872	.092
	Lower-bound	.006	1.000	.006	.268	.627	.051	.268	.071
Error(TRIAL)	Sphericity Assumed	.111	20	.006					
	Greenhouse-Geisser	.111	9.869	.011					
	Huynh-Feldt	.111	16.264	.007					
	Lower-bound	.111	5.000	.022					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TRAP60MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.003	4	.001	.099	.981	.019	.397	.066
	Greenhouse-Geisser	.003	2.464	.001	.099	.937	.019	.245	.063
	Huynh-Feldt	.003	4.000	.001	.099	.981	.019	.397	.066
	Lower-bound	.003	1.000	.003	.099	.765	.019	.099	.058
Error(TRIAL)	Sphericity Assumed	.148	20	.007					
	Greenhouse-Geisser	.148	12.321	.012					
	Huynh-Feldt	.148	20.000	.007					
	Lower-bound	.148	5.000	.030					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TRAP120MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.007	4	.002	.403	.804	.075	1.613	.124
	Greenhouse-Geisser	.007	1.683	.004	.403	.647	.075	.679	.094
	Huynh-Feldt	.007	2.442	.003	.403	.715	.075	.985	.104
	Lower-bound	.007	1.000	.007	.403	.553	.075	.403	.082
Error(TRIAL)	Sphericity Assumed	.090	20	.004					
	Greenhouse-Geisser	.090	8.416	.011					
	Huynh-Feldt	.090	12.210	.007					
	Lower-bound	.090	5.000	.018					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TRAP24HR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.013	4	.003	.608	.663	.132	2.431	.161
	Greenhouse-Geisser	.013	2.105	.006	.608	.575	.132	1.279	.122
	Huynh-Feldt	.013	4.000	.003	.608	.663	.132	2.431	.161
	Lower-bound	.013	1.000	.013	.608	.479	.132	.608	.094
Error(TRIAL)	Sphericity Assumed	.088	16	.006					
	Greenhouse-Geisser	.088	8.421	.010					
	Huynh-Feldt	.088	16.000	.006					
	Lower-bound	.088	4.000	.022					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:CORPRE

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.017	4	.004	.969	.452	.195	3.875	.239
	Greenhouse-Geisser	.017	2.501	.007	.969	.431	.195	2.423	.184
	Huynh-Feldt	.017	4.000	.004	.969	.452	.195	3.875	.239
	Lower-bound	.017	1.000	.017	.969	.381	.195	.969	.120
Error(TRIAL)	Sphericity Assumed	.070	16	.004					
	Greenhouse-Geisser	.070	10.003	.007					
	Huynh-Feldt	.070	16.000	.004					
	Lower-bound	.070	4.000	.017					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:CORPOST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.146	4	.037	2.784	.055	.358	11.137	.654
	Greenhouse-Geisser	.146	1.644	.089	2.784	.124	.358	4.577	.375
	Huynh-Feldt	.146	2.343	.062	2.784	.097	.358	6.524	.472
	Lower-bound	.146	1.000	.146	2.784	.156	.358	2.784	.274
Error(TRIAL)	Sphericity Assumed	.263	20	.013					
	Greenhouse-Geisser	.263	8.220	.032					
	Huynh-Feldt	.263	11.715	.022					
	Lower-bound	.263	5.000	.053					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:COR15MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.119	4	.030	2.899	.048	.367	11.594	.674
	Greenhouse-Geisser	.119	1.704	.070	2.899	.114	.367	4.939	.398
	Huynh-Feldt	.119	2.495	.048	2.899	.084	.367	7.231	.508
	Lower-bound	.119	1.000	.119	2.899	.149	.367	2.899	.283
Error(TRIAL)	Sphericity Assumed	.205	20	.010					
	Greenhouse-Geisser	.205	8.519	.024					
	Huynh-Feldt	.205	12.473	.016					
	Lower-bound	.205	5.000	.041					

a. Computed using alpha = .05

Pairwise Comparisons

Measure:COR15MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.131	.055	.064	-.273	.011
	3	-.071	.051	.224	-.203	.061
	4	-.120*	.036	.021	-.214	-.027
	5	.026	.054	.649	-.112	.165
2	1	.131	.055	.064	-.011	.273
	3	.060	.081	.491	-.148	.268
	4	.011	.051	.844	-.120	.142
	5	.157	.094	.157	-.086	.400
3	1	.071	.051	.224	-.061	.203
	2	-.060	.081	.491	-.268	.148
	4	-.049	.048	.350	-.173	.074
	5	.097	.041	.062	-.007	.201
4	1	.120*	.036	.021	.027	.214
	2	-.011	.051	.844	-.142	.120
	3	.049	.048	.350	-.074	.173
	5	.147*	.048	.029	.022	.271
5	1	-.026	.054	.649	-.165	.112
	2	-.157	.094	.157	-.400	.086
	3	-.097	.041	.062	-.201	.007
	4	-.147*	.048	.029	-.271	-.022

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Tests of Within-Subjects Effects

Measure:COR30MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.187	4	.047	1.809	.167	.266	7.237	.452
	Greenhouse-Geisser	.187	1.949	.096	1.809	.215	.266	3.526	.287
	Huynh-Feldt	.187	3.177	.059	1.809	.185	.266	5.747	.390
	Lower-bound	.187	1.000	.187	1.809	.236	.266	1.809	.196
Error(TRIAL)	Sphericity Assumed	.517	20	.026					
	Greenhouse-Geisser	.517	9.744	.053					
	Huynh-Feldt	.517	15.883	.033					
	Lower-bound	.517	5.000	.103					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:COR60MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.114	4	.029	3.180	.036	.389	12.720	.720
	Greenhouse-Geisser	.114	1.468	.078	3.180	.109	.389	4.668	.391
	Huynh-Feldt	.114	1.928	.059	3.180	.088	.389	6.129	.466
	Lower-bound	.114	1.000	.114	3.180	.135	.389	3.180	.306
Error(TRIAL)	Sphericity Assumed	.179	20	.009					
	Greenhouse-Geisser	.179	7.340	.024					
	Huynh-Feldt	.179	9.638	.019					
	Lower-bound	.179	5.000	.036					

a. Computed using alpha = .05

Pairwise Comparisons

Measure:COR60MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.032	.043	.481	-.142	.077
	3	-.024	.050	.645	-.152	.103
	4	-.144	.057	.052	-.289	.002
	5	.042	.032	.244	-.039	.123
2	1	.032	.043	.481	-.077	.142
	3	.008	.080	.923	-.198	.215
	4	-.111	.084	.245	-.328	.106
	5	.074	.069	.332	-.103	.252
3	1	.024	.050	.645	-.103	.152
	2	-.008	.080	.923	-.215	.198
	4	-.120*	.026	.005	-.185	-.054
	5	.066	.030	.079	-.011	.143
4	1	.144	.057	.052	-.002	.289
	2	.111	.084	.245	-.106	.328
	3	.120*	.026	.005	.054	.185
	5	.185*	.039	.005	.086	.284
5	1	-.042	.032	.244	-.123	.039
	2	-.074	.069	.332	-.252	.103
	3	-.066	.030	.079	-.143	.011
	4	-.185*	.039	.005	-.284	-.086

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Tests of Within-Subjects Effects

Measure:COR120MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.006	4	.001	.098	.982	.019	.391	.066
	Greenhouse-Geisser	.006	2.818	.002	.098	.954	.019	.275	.063
	Huynh-Feldt	.006	4.000	.001	.098	.982	.019	.391	.066
	Lower-bound	.006	1.000	.006	.098	.767	.019	.098	.058
Error(TRIAL)	Sphericity Assumed	.288	20	.014					
	Greenhouse-Geisser	.288	14.089	.020					
	Huynh-Feldt	.288	20.000	.014					
	Lower-bound	.288	5.000	.058					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:COR24HR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.019	4	.005	1.060	.408	.209	4.240	.260
	Greenhouse-Geisser	.019	1.825	.010	1.060	.387	.209	1.935	.168
	Huynh-Feldt	.019	3.278	.006	1.060	.404	.209	3.474	.231
	Lower-bound	.019	1.000	.019	1.060	.361	.209	1.060	.127
Error(TRIAL)	Sphericity Assumed	.072	16	.005					
	Greenhouse-Geisser	.072	7.302	.010					
	Huynh-Feldt	.072	13.110	.006					
	Lower-bound	.072	4.000	.018					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:PTHPRE

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.467	4	.117	3.214	.034	.391	12.857	.725
	Greenhouse-Geisser	.467	1.695	.276	3.214	.096	.391	5.447	.433
	Huynh-Feldt	.467	2.471	.189	3.214	.067	.391	7.943	.551
	Lower-bound	.467	1.000	.467	3.214	.133	.391	3.214	.308
Error(TRIAL)	Sphericity Assumed	.727	20	.036					
	Greenhouse-Geisser	.727	8.473	.086					
	Huynh-Feldt	.727	12.356	.059					
	Lower-bound	.727	5.000	.145					

a. Computed using alpha = .05

Pairwise Comparisons

Measure:PTHPRE

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.218	.160	.231	-.194	.630
	3	.334*	.080	.009	.127	.541
	4	.309*	.060	.004	.154	.465
	5	.112*	.033	.019	.028	.196
2	1	-.218	.160	.231	-.630	.194
	3	.116	.149	.473	-.268	.500
	4	.091	.149	.566	-.291	.473
	5	-.106	.160	.536	-.518	.305
3	1	-.334*	.080	.009	-.541	-.127
	2	-.116	.149	.473	-.500	.268
	4	-.024	.065	.723	-.192	.143
	5	-.222*	.071	.026	-.404	-.040
4	1	-.309*	.060	.004	-.465	-.154
	2	-.091	.149	.566	-.473	.291
	3	.024	.065	.723	-.143	.192
	5	-.198*	.070	.038	-.379	-.016
5	1	-.112*	.033	.019	-.196	-.028
	2	.106	.160	.536	-.305	.518
	3	.222*	.071	.026	.040	.404
	4	.198*	.070	.038	.016	.379

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Tests of Within-Subjects Effects

Measure:PTHPOST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.178	4	.045	2.455	.079	.329	9.819	.592
	Greenhouse-Geisser	.178	1.939	.092	2.455	.138	.329	4.760	.374
	Huynh-Feldt	.178	3.148	.057	2.455	.099	.329	7.726	.510
	Lower-bound	.178	1.000	.178	2.455	.178	.329	2.455	.248
Error(TRIAL)	Sphericity Assumed	.363	20	.018					
	Greenhouse-Geisser	.363	9.695	.037					
	Huynh-Feldt	.363	15.738	.023					
	Lower-bound	.363	5.000	.073					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:PTHPOST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.438	4	.109	4.367	.011	.466	17.469	.861
	Greenhouse-Geisser	.438	2.836	.154	4.367	.024	.466	12.387	.744
	Huynh-Feldt	.438	4.000	.109	4.367	.011	.466	17.469	.861
	Lower-bound	.438	1.000	.438	4.367	.091	.466	4.367	.395
Error(TRIAL)	Sphericity Assumed	.501	20	.025					
	Greenhouse-Geisser	.501	14.182	.035					
	Huynh-Feldt	.501	20.000	.025					
	Lower-bound	.501	5.000	.100					

a. Computed using alpha = .05

Pairwise Comparisons

Measure:PTHPOST

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.206*	.074	.039	.016	.396
	3	.141	.075	.118	-.051	.334
	4	.346*	.098	.017	.093	.598
	5	.055	.065	.431	-.111	.221
2	1	-.206*	.074	.039	-.396	-.016
	3	-.065	.117	.605	-.366	.237
	4	.140	.104	.237	-.128	.407
	5	-.151	.106	.214	-.423	.122
3	1	-.141	.075	.118	-.334	.051
	2	.065	.117	.605	-.237	.366
	4	.204	.088	.067	-.021	.430
	5	-.086	.073	.293	-.274	.102
4	1	-.346*	.098	.017	-.598	-.093
	2	-.140	.104	.237	-.407	.128
	3	-.204	.088	.067	-.430	.021
	5	-.290*	.099	.032	-.545	-.036
5	1	-.055	.065	.431	-.221	.111
	2	.151	.106	.214	-.122	.423
	3	.086	.073	.293	-.102	.274
	4	.290*	.099	.032	.036	.545

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Tests of Within-Subjects Effects

Measure:PTH30MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.383	4	.096	7.170	.001	.589	28.682	.980
	Greenhouse-Geisser	.383	2.410	.159	7.170	.007	.589	17.279	.889
	Huynh-Feldt	.383	4.000	.096	7.170	.001	.589	28.682	.980
	Lower-bound	.383	1.000	.383	7.170	.044	.589	7.170	.578
Error(TRIAL)	Sphericity Assumed	.267	20	.013					
	Greenhouse-Geisser	.267	12.048	.022					
	Huynh-Feldt	.267	20.000	.013					
	Lower-bound	.267	5.000	.053					

a. Computed using alpha = .05

Pairwise Comparisons

Measure:PTH30MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.127	.078	.163	-.073	.327
	3	.120	.068	.141	-.056	.295
	4	.226*	.080	.036	.022	.431
	5	-.100	.062	.169	-.260	.060
2	1	-.127	.078	.163	-.327	.073
	3	-.008	.075	.923	-.201	.185
	4	.099	.052	.113	-.034	.232
	5	-.227*	.082	.040	-.439	-.015
3	1	-.120	.068	.141	-.295	.056
	2	.008	.075	.923	-.185	.201
	4	.107	.050	.086	-.022	.235
	5	-.220*	.028	.001	-.292	-.148
4	1	-.226*	.080	.036	-.431	-.022
	2	-.099	.052	.113	-.232	.034
	3	-.107	.050	.086	-.235	.022
	5	-.326*	.072	.006	-.512	-.140
5	1	.100	.062	.169	-.060	.260
	2	.227*	.082	.040	.015	.439
	3	.220*	.028	.001	.148	.292
	4	.326*	.072	.006	.140	.512

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Tests of Within-Subjects Effects

Measure:PTH60MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.328	4	.082	4.726	.008	.486	18.906	.889
	Greenhouse-Geisser	.328	1.368	.240	4.726	.060	.486	6.466	.516
	Huynh-Feldt	.328	1.710	.192	4.726	.045	.486	8.080	.594
	Lower-bound	.328	1.000	.328	4.726	.082	.486	4.726	.421
Error(TRIAL)	Sphericity Assumed	.347	20	.017					
	Greenhouse-Geisser	.347	6.841	.051					
	Huynh-Feldt	.347	8.548	.041					
	Lower-bound	.347	5.000	.069					

a. Computed using alpha = .05

Pairwise Comparisons

Measure:PTH60MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.185*	.038	.005	.087	.282
	3	.232	.099	.065	-.021	.485
	4	.246	.111	.077	-.039	.532
	5	.025	.102	.819	-.238	.287
2	1	-.185*	.038	.005	-.282	-.087
	3	.047	.076	.562	-.148	.243
	4	.062	.086	.504	-.158	.282
	5	-.160	.082	.107	-.370	.049
3	1	-.232	.099	.065	-.485	.021
	2	-.047	.076	.562	-.243	.148
	4	.014	.048	.776	-.109	.138
	5	-.208*	.019	.000	-.257	-.158
4	1	-.246	.111	.077	-.532	.039
	2	-.062	.086	.504	-.282	.158
	3	-.014	.048	.776	-.138	.109
	5	-.222*	.039	.002	-.323	-.121
5	1	-.025	.102	.819	-.287	.238
	2	.160	.082	.107	-.049	.370
	3	.208*	.019	.000	.158	.257
	4	.222*	.039	.002	.121	.323

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Tests of Within-Subjects Effects

Measure:PTH120MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.194	4	.048	5.177	.005	.509	20.707	.918
	Greenhouse-Geisser	.194	1.522	.127	5.177	.044	.509	7.880	.592
	Huynh-Feldt	.194	2.051	.095	5.177	.027	.509	10.618	.702
	Lower-bound	.194	1.000	.194	5.177	.072	.509	5.177	.452
Error(TRIAL)	Sphericity Assumed	.187	20	.009					
	Greenhouse-Geisser	.187	7.611	.025					
	Huynh-Feldt	.187	10.255	.018					
	Lower-bound	.187	5.000	.037					

a. Computed using alpha = .05

Pairwise Comparisons

Measure:PTH120MIN

(I) TRIAL	(J) TRIAL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.050	.029	.143	-.024	.124
	3	.061	.045	.239	-.056	.177
	4	.182*	.068	.044	.008	.357
	5	-.061	.037	.162	-.157	.035
2	1	-.050	.029	.143	-.124	.024
	3	.011	.050	.837	-.117	.139
	4	.132	.086	.183	-.088	.353
	5	-.111*	.022	.004	-.167	-.054
3	1	-.061	.045	.239	-.177	.056
	2	-.011	.050	.837	-.139	.117
	4	.122	.054	.075	-.018	.261
	5	-.122*	.042	.035	-.230	-.013
4	1	-.182*	.068	.044	-.357	-.008
	2	-.132	.086	.183	-.353	.088
	3	-.122	.054	.075	-.261	.018
	5	-.243*	.086	.036	-.463	-.023
5	1	.061	.037	.162	-.035	.157
	2	.111*	.022	.004	.054	.167
	3	.122*	.042	.035	.013	.230
	4	.243*	.086	.036	.023	.463

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

*. The mean difference is significant at the .05 level.

Tests of Within-Subjects Effects

Measure:PTH24HR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.011	4	.003	.268	.894	.063	1.072	.094
	Greenhouse-Geisser	.011	2.011	.006	.268	.773	.063	.539	.079
	Huynh-Feldt	.011	4.000	.003	.268	.894	.063	1.072	.094
	Lower-bound	.011	1.000	.011	.268	.632	.063	.268	.069
Error(TRIAL)	Sphericity Assumed	.165	16	.010					
	Greenhouse-Geisser	.165	8.042	.021					
	Huynh-Feldt	.165	16.000	.010					
	Lower-bound	.165	4.000	.041					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TESTPRE

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.072	4	.018	1.250	.322	.200	5.000	.319
	Greenhouse-Geisser	.072	3.006	.024	1.250	.327	.200	3.758	.268
	Huynh-Feldt	.072	4.000	.018	1.250	.322	.200	5.000	.319
	Lower-bound	.072	1.000	.072	1.250	.314	.200	1.250	.150
Error(TRIAL)	Sphericity Assumed	.288	20	.014					
	Greenhouse-Geisser	.288	15.032	.019					
	Huynh-Feldt	.288	20.000	.014					
	Lower-bound	.288	5.000	.058					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TESTPOST

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.025	4	.006	.488	.745	.089	1.950	.141
	Greenhouse-Geisser	.025	2.279	.011	.488	.650	.089	1.111	.114
	Huynh-Feldt	.025	4.000	.006	.488	.745	.089	1.950	.141
	Lower-bound	.025	1.000	.025	.488	.516	.089	.488	.089
Error(TRIAL)	Sphericity Assumed	.257	20	.013					
	Greenhouse-Geisser	.257	11.396	.023					
	Huynh-Feldt	.257	20.000	.013					
	Lower-bound	.257	5.000	.051					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TEST15MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.086	4	.022	1.429	.261	.222	5.714	.362
	Greenhouse-Geisser	.086	2.206	.039	1.429	.283	.222	3.151	.251
	Huynh-Feldt	.086	4.000	.022	1.429	.261	.222	5.714	.362
	Lower-bound	.086	1.000	.086	1.429	.286	.222	1.429	.165
Error(TRIAL)	Sphericity Assumed	.301	20	.015					
	Greenhouse-Geisser	.301	11.030	.027					
	Huynh-Feldt	.301	20.000	.015					
	Lower-bound	.301	5.000	.060					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TEST30MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.069	4	.017	1.373	.279	.215	5.494	.349
	Greenhouse-Geisser	.069	1.907	.036	1.373	.298	.215	2.619	.224
	Huynh-Feldt	.069	3.053	.023	1.373	.288	.215	4.193	.295
	Lower-bound	.069	1.000	.069	1.373	.294	.215	1.373	.160
Error(TRIAL)	Sphericity Assumed	.250	20	.013					
	Greenhouse-Geisser	.250	9.535	.026					
	Huynh-Feldt	.250	15.265	.016					
	Lower-bound	.250	5.000	.050					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TEST60MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.009	4	.002	.178	.947	.034	.712	.080
	Greenhouse-Geisser	.009	2.172	.004	.178	.855	.034	.387	.072
	Huynh-Feldt	.009	3.900	.002	.178	.944	.034	.694	.080
	Lower-bound	.009	1.000	.009	.178	.691	.034	.178	.064
Error(TRIAL)	Sphericity Assumed	.257	20	.013					
	Greenhouse-Geisser	.257	10.859	.024					
	Huynh-Feldt	.257	19.502	.013					
	Lower-bound	.257	5.000	.051					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TEST120MIN

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.087	4	.022	1.235	.328	.198	4.941	.315
	Greenhouse-Geisser	.087	1.863	.047	1.235	.331	.198	2.302	.203
	Huynh-Feldt	.087	2.926	.030	1.235	.332	.198	3.615	.261
	Lower-bound	.087	1.000	.087	1.235	.317	.198	1.235	.149
Error(TRIAL)	Sphericity Assumed	.354	20	.018					
	Greenhouse-Geisser	.354	9.316	.038					
	Huynh-Feldt	.354	14.632	.024					
	Lower-bound	.354	5.000	.071					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:TEST24HR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
TRIAL	Sphericity Assumed	.020	4	.005	.160	.956	.038	.639	.075
	Greenhouse-Geisser	.020	1.588	.012	.160	.810	.038	.254	.065
	Huynh-Feldt	.020	2.464	.008	.160	.892	.038	.394	.069
	Lower-bound	.020	1.000	.020	.160	.710	.038	.160	.061
Error(TRIAL)	Sphericity Assumed	.488	16	.031					
	Greenhouse-Geisser	.488	6.354	.077					
	Huynh-Feldt	.488	9.856	.050					
	Lower-bound	.488	4.000	.122					

a. Computed using alpha = .05

Area Under the Curve (AUC) – 2 x 2 ANOVA (n=12)

Tests of Between-Subjects Effects

Dependent Variable:bapauc

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Corrected Model	108131.872[a]	3	36043.957	.488	.692	.033	1.464	.141
Intercept	1008934.316	1	1008934.316	13.660	.001	.241	13.660	.951
fastfed	98528.070	1	98528.070	1.334	.254	.030	1.334	.204
plyrt	11014.168	1	11014.168	.149	.701	.003	.149	.066
fastfed * plyrt	72.553	1	72.553	.001	.975	.000	.001	.050
Error	3176044.133	43	73861.491					
Total	4276333.810	47						
Corrected Total	3284176.005	46						

a. R Squared = .033 (Adjusted R Squared = -.035)

b. Computed using alpha = .05

Tests of Between-Subjects Effects

Dependent Variable:trapauc

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Corrected Model	346.772[a]	3	115.591	.134	.939	.009	.401	.072
Intercept	1795.970	1	1795.970	2.078	.157	.046	2.078	.291
fastfed	124.802	1	124.802	.144	.706	.003	.144	.066
plyrt	72.182	1	72.182	.084	.774	.002	.084	.059
fastfed * plyrt	146.736	1	146.736	.170	.682	.004	.170	.069
Error	37157.210	43	864.121					
Total	39329.310	47						
Corrected Total	37503.982	46						

a. R Squared = .009 (Adjusted R Squared = -.060)

b. Computed using alpha = .05

Tests of Between-Subjects Effects

Dependent Variable:corauc

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Corrected Model	565628.840[a]	3	188542.947	2.642	.062	.159	7.926	.605
Intercept	8083742.137	1	8083742.137	113.271	.000	.730	113.271	1.000
fastfed	187781.891	1	187781.891	2.631	.112	.059	2.631	.354
plyrt	302129.535	1	302129.535	4.233	.046	.092	4.233	.520
fastfed * plyrt	86288.213	1	86288.213	1.209	.278	.028	1.209	.189
Error	2997389.855	42	71366.425					
Total	1.153E7	46						
Corrected Total	3563018.695	45						

a. R Squared = .159 (Adjusted R Squared = .099)

b. Computed using alpha = .05

Tests of Between-Subjects Effects

Dependent Variable:pthauc

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Corrected Model	7.860E6	3	2619996.425	.347	.791	.024	1.042	.112
Intercept	1.981E8	1	1.981E8	26.270	.000	.379	26.270	.999
fastfed	178859.849	1	178859.849	.024	.878	.001	.024	.053
plyrt	4368495.031	1	4368495.031	.579	.451	.013	.579	.115
fastfed * plyrt	3138199.513	1	3138199.513	.416	.522	.010	.416	.097
Error	3.243E8	43	7542116.001					
Total	5.305E8	47						
Corrected Total	3.322E8	46						

a. R Squared = .024 (Adjusted R Squared = -.044)

b. Computed using alpha = .05

Tests of Between-Subjects Effects

Dependent Variable:testauc

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Corrected Model	6.992E8	3	2.331E8	.678	.570	.045	2.035	.181
Intercept	3.032E9	1	3.032E9	8.826	.005	.170	8.826	.827
fastfed	2.938E8	1	2.938E8	.855	.360	.020	.855	.148
plyrt	1.181E8	1	1.181E8	.344	.561	.008	.344	.088
fastfed * plyrt	3.164E8	1	3.164E8	.921	.343	.021	.921	.155
Error	1.477E10	43	3.435E8					
Total	1.861E10	47						
Corrected Total	1.547E10	46						

a. R Squared = .045 (Adjusted R Squared = -.021)

b. Computed using alpha = .05

Area Under the Curve (AUC) – One-Factor (Trial) RMANOVA (n=6)

Tests of Within-Subjects Effects

Measure:bap

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
trial	Sphericity Assumed	233648.269	4	58412.067	1.594	.215	.242	6.374	.402
	Greenhouse-Geisser	233648.269	2.254	103660.372	1.594	.246	.242	3.592	.280
	Huynh-Feldt	233648.269	4.000	58412.067	1.594	.215	.242	6.374	.402
	Lower-bound	233648.269	1.000	233648.269	1.594	.263	.242	1.594	.178
Error(trial)	Sphericity Assumed	733127.011	20	36656.351					
	Greenhouse-Geisser	733127.011	11.270	65051.814					
	Huynh-Feldt	733127.011	20.000	36656.351					
	Lower-bound	733127.011	5.000	146625.402					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:trap

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
trial	Sphericity Assumed	4008.143	4	1002.036	2.518	.074	.335	10.074	.604
	Greenhouse-Geisser	4008.143	2.246	1784.285	2.518	.121	.335	5.657	.421
	Huynh-Feldt	4008.143	4.000	1002.036	2.518	.074	.335	10.074	.604
	Lower-bound	4008.143	1.000	4008.143	2.518	.173	.335	2.518	.253
Error(trial)	Sphericity Assumed	7957.553	20	397.878					
	Greenhouse-Geisser	7957.553	11.232	708.485					
	Huynh-Feldt	7957.553	20.000	397.878					
	Lower-bound	7957.553	5.000	1591.511					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:cortisol

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
trial	Sphericity Assumed	728935.994	4	182233.998	1.815	.175	.312	7.258	.431
	Greenhouse-Geisser	728935.994	2.015	361834.298	1.815	.224	.312	3.656	.275
	Huynh-Feldt	728935.994	4.000	182233.998	1.815	.175	.312	7.258	.431
	Lower-bound	728935.994	1.000	728935.994	1.815	.249	.312	1.815	.182
Error(trial)	Sphericity Assumed	1606859.615	16	100428.726					
	Greenhouse-Geisser	1606859.615	8.058	199406.026					
	Huynh-Feldt	1606859.615	16.000	100428.726					
	Lower-bound	1606859.615	4.000	401714.904					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:pth

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
trial	Sphericity Assumed	1.236E7	4	3091024.240	.976	.443	.163	3.904	.253
	Greenhouse-Geisser	1.236E7	2.233	5536928.615	.976	.416	.163	2.179	.184
	Huynh-Feldt	1.236E7	4.000	3091024.240	.976	.443	.163	3.904	.253
	Lower-bound	1.236E7	1.000	1.236E7	.976	.369	.163	.976	.128
Error(trial)	Sphericity Assumed	6.334E7	20	3167044.660					
	Greenhouse-Geisser	6.334E7	11.165	5673103.424					
	Huynh-Feldt	6.334E7	20.000	3167044.660					
	Lower-bound	6.334E7	5.000	1.267E7					

a. Computed using alpha = .05

Tests of Within-Subjects Effects

Measure:test

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
trial	Sphericity Assumed	1.453E9	4	3.632E8	.783	.549	.135	3.134	.207
	Greenhouse-Geisser	1.453E9	1.714	8.475E8	.783	.468	.135	1.343	.139
	Huynh-Feldt	1.453E9	2.522	5.761E8	.783	.505	.135	1.976	.164
	Lower-bound	1.453E9	1.000	1.453E9	.783	.417	.135	.783	.112
Error(trial)	Sphericity Assumed	9.272E9	20	4.636E8					
	Greenhouse-Geisser	9.272E9	8.571	1.082E9					
	Huynh-Feldt	9.272E9	12.609	7.354E8					
	Lower-bound	9.272E9	5.000	1.854E9					

a. Computed using alpha = .05

Bivariate Relationships

Correlations

		bapauc	trapauc	corauc	pthauc	testauc
Pearson Correlation	bapauc	1.000	.145	-.140	.110	-.073
	trapauc	.145	1.000	-.010	.452	.143
	corauc	-.140	-.010	1.000	.209	.159
	pthauc	.110	.452	.209	1.000	.308
	testauc	-.073	.143	.159	.308	1.000
Sig. (1-tailed)	bapauc	.	.169	.177	.233	.314
	trapauc	.169	.	.474	.001	.171
	corauc	.177	.474	.	.082	.145
	pthauc	.233	.001	.082	.	.019
	testauc	.314	.171	.145	.019	.
N	bapauc	46	46	46	46	46
	trapauc	46	46	46	46	46
	corauc	46	46	46	46	46
	pthauc	46	46	46	46	46
	testauc	46	46	46	46	46

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	-185.190	75.495		-2.453	.019
	trapauc	.943	1.612	.100	.585	.562
	corauc	-.143	.151	-.149	-.948	.349
	pthauc	.013	.018	.128	.709	.482
	testauc	-.002	.002	-.104	-.647	.521

a. Dependent Variable: bapauc

Appendix E
Study Forms

CONSENT FORM TO PARTICIPATE IN A RESEARCH STUDY

INVESTIGATOR'S NAME: PAMELA S. HINTON
PROJECT # 1097239
DATE OF PROJECT APPROVAL: OCTOBER 31, 2007

FOR HS IRB USE ONLY
APPROVED
HS IRB Authorized Representative — Date: 5/15/09
EXPIRATION DATE: 10·31·2009

Study Title: Hormonal and Bone Turnover Marker Response to an Acute Bout of Resistance or Plyometric Exercise

INTRODUCTION

This consent may contain words that you do not understand. Please ask the investigator or the study staff to explain any words or information that you do not clearly understand.

This is a research study. Research studies include only people who choose to participate. As a study participant you have the right to know about the procedures that will be used in this research study so that you can make the decision whether or not to participate. The information presented here is simply an effort to make you better informed so that you may give or withhold your consent to participate in this research study.

Please take your time to make your decision and discuss it with your family and friends.

You are being asked to take part in this study because you have are a healthy, non-sedentary male. Long-term participation in high intensity training has been shown to slow the rate of bone mineral loss with aging and can improve bone mineral density. Certain markers of bone turnover have been shown in the long-term to parallel improvements in bone mineral density; however, the acute effects of high intensity exercise on bone turnover makers are not as well studied.

This study is being sponsored by the Department of Nutritional Sciences, University of Missouri-Columbia.

In order to participate in this study, it will be necessary to give your written consent.

WHY IS THIS STUDY BEING DONE?

The purpose of this study is to determine the effects of singles bouts of weight lifting (resistance exercise) and high-intensity jumping (plyometrics) as compared to a non-exercise control trial, on changes in bone turnover marker of the blood in males. This study is being done because the long term effects of high intensity, weight-bearing exercise have been shown to slow the rate of bone mineral loss with aging and improve bone mineral density. However, the short term effects of high intensity exercise on markers of bone turnover have not been fully characterized.

HOW MANY PEOPLE WILL TAKE PART IN THE STUDY?

A maximum of 30 people will take part in this study at the University of Missouri-Columbia.

WHAT IS INVOLVED IN THE STUDY?

The study will require 8 visits over approximately 6 weeks at the McKee Gymnasium Fitness Center, with the longest sessions lasting approximately 3 hours for the exercise familiarization session or to complete the exercise session and to collect the blood draws 2 hours after the completion of the exercise trial. During the study you will continue your normal exercise program throughout, except for the two days prior to each exercise session. Thus, you will maintain your normal life at home, work, or school. You are allowed to quit at any time without penalty or loss of any benefits. Additionally, you may be asked to discontinue the study if the research and medical staff determine it is in your best interest to do so. You will also be provided with the results of your diet analysis, body composition, and bone density tests.

Visit 1: Begin the informed consent process and describe the study purpose and the requirements.

All participants must: 1) be males between 25-65 years of age; 2) be apparently healthy and non-sedentary; 3) be willing to keep records of exercise training and food intake; 4) be willing and able to provide accurate information about your medical history; 5) be willing and able to participate in resistance exercise and high-intensity plyometric jumping; 6) be free of disease that affects bone; and, 7) not have used or currently use any medication that affects bone.

Visit 2: If you decide to participate in the study you will come back for Visit 2 and sign the consent form. You will fill out a physical activity readiness, health history, and physical activity history questionnaire. You must provide information about your medical history, including history of illness, injuries, and drug treatment that may affect your ability to safely and effectively participate in the study. If you are at moderate health risk for participating in this research study (45 years of age or old, or have 2 or more of the following risk factors: family history of heart disease in first degree relative, hypertension, hypercholesterolemia, or impaired fasting glucose), you will need to obtain a medical clearance from a doctor to participate in this exercise study.

You also must provide accurate information about your past exercise participation (i.e., training and competition in sports). Additionally, your height and weight will be taken and a bone mineral density scan will be performed to determine by DXA (dual X-ray absorptiometry).

If you meet the eligibility requirements of the study (i.e., age, activity level, no diseases or medications that affect bone), you will be instructed how to complete a diet record and physical training log to record your dietary intake and physical training while participating in this study.

Visit 3: You will return to the lab to become familiar with both the high-intensity jumping (plyometrics) and weight lifting (resistance) exercise sessions. You will also be given nutrient guidelines on the makeup of a meal to be consumed prior to each exercise session (i.e., carbohydrate, fat, and protein amount).

You will undergo an instructional and familiarization training for the plyometric jump training procedures and exercises. Qualified study personnel will guide you throughout the sessions so that all exercises can be safely completed using proper form and technique. Upon completing this instructional session, you will perform five jumps of each exercise to assess your mastery of the techniques.

You will also undergo an instructional and familiarization session on the procedures for the resistance exercise sessions. Qualified study personnel will guide you through all the exercises to be performed so that all exercises can be safely completed using the proper technique. Upon completing this practice session, you will undergo one-repetition maximum testing (1-RM) for the exercises to be used for the resistance exercise trial. The following exercises will be tested for 1-RM: dead lift, squat, military press, and bent over row. Resistance levels for all other exercises will be performed at an intensity over 3 sets in which a maximum of 8-10 repetitions can be performed (lunge and calf raise) or 15 repetitions (low back extensions and bent-knee abdominal crunches).

Visits 4-8: Five to seven days after your third visit to the Exercise Physiology Laboratory, you will return for your randomly assigned experimental sessions.

The night prior to a session you must consume a meal using the nutrient guidelines that will be given to you then remain fasted until the next day (skipping breakfast) when you will return to the lab and report between 7:00 AM and 10:00 AM to participate in your assigned session (weight lifting, high-intensity jumping, or non-exercise control). Prior to performing the sessions without the moderate calorie snack, your blood glucose concentration will be checked using a glucose monitor.

The order in which you participate in the experimental sessions will be randomly assigned. For two exercise sessions (once for resistance exercise and once for plyometrics exercise) the addition of a moderate calorie snack (~500 calories) will be provided 2 hours before the exercise. This will result in a total of 5 sessions, each lasting approximately 3 hours:

- 1 fasted control session
- 2 fasted exercise sessions (resistance and plyometrics)
- 2 fed exercise sessions (~500 calories two hours before exercise, resistance and plyometrics)

The control session will consist of participants sitting, relaxed, in a chair for 1 hour and 30 minutes.

The resistance exercise session will consist of a warm up by cycling for 10-min on a stationary bicycle, then 3 sets of 8-15 repetitions for seven different exercises (dead lift, squat, bent over row, military press, lunge, calf raise, low back extensions, and bent-knee abdominal crunches). After the session has been completed, you will cool down for ten minutes by completing a low intensity static stretching routine and

cycling for 5-min on a stationary bicycle. Qualified study personnel will assist you throughout the session so that all exercises can be safely completed using the proper form and technique.

The plyometric jump training session will consist of a warm up by cycling for 10-min on a stationary bicycle, then 10 repetitions of 12 different jumps (squat jumps, forward hops, split squat jumps, tuck jumps, lateral box push offs, bounding, bounding with rings (lateral), box drill with rings, lateral hurdle jumps, zigzag hops, single leg lateral hops, and 10 cm depth jumps). There will be a rest interval of 10 seconds between jumps and 1-2 minutes between different jumping exercises. Participants will cool down by completing a low intensity static stretching routine and cycling for 5-min on a stationary bicycle. Qualified study personnel will assist you through the exercises so that all exercises can be safely completed using proper form and technique.

You will have a small amount (5 ml) of blood drawn at a few time points during each experimental session visit. During the experimental sessions, blood will be drawn immediately before performing the exercise and control session, immediately after, and 15 minutes, 30 minutes, 1 hour, 2 hours, and 24 hours after. To minimize the number of times a needle will be inserted to collect a blood sample, a flexible teflon-catheter will be placed into a forearm vein, and used to collect all the blood samples for the 2 hours following the exercise and the 1 hour 30 minute control period. The blood samples will be taken from a vein in your forearm using the same procedure as would be followed at a health clinic. The amount of blood taken is very small and will not affect your health (~21 milliliters or about 1 ½ tablespoons). The blood will be used to measure hormones and markers of bone formation and breakdown. Your blood will be analyzed for factors that may affect your bone mass.

Your blood will be kept frozen for 5 years after the study is completed and the results may be published in a research journal. No additional tests will be performed on your blood sample.

HOW LONG WILL I BE IN THE STUDY?

The study will take approximately 6 weeks to complete.

You can stop participating at any time. Your decision to withdraw from the study will not affect in any way your medical care and/or benefits.

WHAT ARE THE RISKS OF THE STUDY?

While on the study, you are at risk for the side effects described below. You should discuss these with the investigator and/or your doctor. There may also be other side effects that we cannot predict.

Risks and side effects related to the study tests and procedures we are studying include:

There is a possibility of bruising and soreness at the site of blood draw. Sterile procedures will be used so the chance of getting an infection is very remote.

As a result of the overnight fast, there is the possibility that you will experience symptoms of low blood sugar after the overnight fast, including lightheadedness and dizziness.

There is a possibility of injury to the skeletal and muscular systems due to the high intensity nature of the resistance exercise training and the plyometric jump training. Muscle soreness will almost certainly occur.

There is a possibility of cardiovascular complications, such as arrhythmias and myocardial ischemia during exercise. If for any reason during participation in the study's exercise sessions you feel pain or discomfort in the chest, arms, back, neck or jaw, shortness of breath, nausea, or lightheadedness please inform study personnel immediately so that proper medical treatment may be provided.

To ensure individuals who are at moderate health risk (**45 years of age or older**, or have **2 or more of the following risk factors:** family history of heart disease in first degree relative, hypertension, hypercholesterolemia, or impaired fasting glucose) for vigorous exercise, you will be required to obtain medical clearance from a doctor before you are able to participate in this exercise study. All financial costs associated with obtaining medical clearance for study participation are the responsibility of you, the subject.

Reproductive risks: The effects of the DXA scan on the male reproductive system are unknown but could cause harm. If you have questions about the reproductive issues, please discuss them with the investigator or your doctor.

You will be exposed to a small amount of radiation. Radiation effects are cumulative. You should always inform future doctors of your participation in this study.

For the reasons stated above the investigator will observe you closely while giving the study described above and, if you have any worrisome symptoms, notify the investigator immediately. Dr. Pam Hinton's telephone number is (573) 882-4137. For more information about risks and side effects, ask the investigator or contact Dr. Hinton at (573) 882-4137.

ARE THERE BENEFITS TO TAKING PART IN THE STUDY?

If you agree to take part in this study, there may or may not be direct medical benefit to you. You may expect to benefit from taking part in this research to the extent that you are contributing to medical knowledge. We hope the information learned from this study will benefit those who have reduced bone mineral density.

Upon completion of the study, you will receive a dietary analysis, body composition analysis, and bone density scan.

WHAT OTHER OPTIONS ARE THERE?

You have the option to not participate in this study.

WHAT ABOUT CONFIDENTIALITY?

Information produced by this study will be stored in the investigator's file and identified by a code number only. The code key connecting your name to specific information about you will be kept in a separate, secure location. Information contained in your records may not be given to anyone unaffiliated

with the study in a form that could identify you without your written consent, except as required by law. If the investigator conducting this study is not your primary, or regular doctor, she must obtain your permission before contacting your regular doctor for information about your past medical history or to inform them that you are in this trial.

It is possible that your medical and/or research record, including sensitive information and/or identifying information, may be inspected and/or copied by the study sponsor (and/or its agent), the Food and Drug Administration (FDA), federal or state government agencies, University of Missouri Health Sciences Institutional Review Board or hospital accrediting agencies, in the course of carrying out their duties. If your record is inspected or copied by the study sponsor (and/or its agents), or by any of these agencies, the University of Missouri-Columbia will use reasonable efforts to protect your privacy and the confidentiality of your medical information.

The results of this study may be published in a medical book or journal or used for teaching purposes. However, your name or other identifying information will not be used in any publication or teaching materials without your specific permission.

WHAT ARE THE COSTS?

There is no cost to you for the study procedures. You will not be charged for blood tests that are part of this research study. You are responsible for the cost associated with obtaining medical clearance from a doctor if required.

WILL I BE PAID FOR PARTICIPATING IN THE STUDY?

You will be compensated $50 for completion of the duration of the study. You will also be provided a dietary analysis, body composition, and bone density scan.

WHAT IF I AM INJURED?

It is not the policy of the University of Missouri to compensate human subjects in the event the research results in injury. The University of Missouri, in fulfilling its public responsibility, has provided medical, professional and general liability insurance coverage for any injury in the event such injury is caused by the negligence of the University of Missouri, its faculty and staff. The University of Missouri also will provide, within the limitations of the laws of the State of Missouri, facilities and medical attention to subjects who suffer injuries while participating in the research projects of the University of Missouri. In the event you have suffered injury as the result of participation in this research program, you are to contact the Risk Management Officer, telephone number (573) 882-1181, at the Health Sciences Center, who can review the matter and provide further information. This statement is not to be construed as an admission of liability.

WHAT ARE MY RIGHTS AS A PARTICIPANT?

Participation in this study is voluntary. You do not have to participate in this study. Your present or future care will not be affected should you choose not to participate. If you decide to participate, you can change your mind and drop out of the study at any time without affecting your present or future care in the University of Missouri-Columbia. Leaving the study will not result in any penalty or loss of benefits to which you are entitled. In addition, the investigator of this study may decide to end your

participation in this study at any time after she has explained the reasons for doing so and has helped arrange for your continued care by your own doctor, if needed.

You will be informed of any significant new findings discovered during the course of this study that might influence your health, welfare, or willingness to continue participation in this study.

WHOM DO I CALL IF I HAVE QUESTIONS OR PROBLEMS?

If you have any questions regarding your rights as a participant in this research and/or concerns about the study, or if you feel under any pressure to enroll or to continue to participate in this study, you may contact the University of Missouri Health Sciences Institutional Review Board (which is a group of people who review the research studies to protect participants' rights) at (573) 882-3181

You may ask more questions about the study at any time. For questions about the study or a research-related injury, contact Dr. Pam Hinton at (573) 882-4137.
A copy of this consent form will be given to you to keep.

SIGNATURE

I confirm that the purpose of the research, the study procedures, the possible risks and discomforts as well as potential benefits that I may experience have been explained to me. Alternatives to my participation in the study also have been discussed. I have read this consent form and my questions have been answered. My signature below indicates my willingness to participate in this study.

Subject/Patient*	Date
Legal Guardian/Advocate/Witness (if required)**	Date
Physician Signature (required if physician approval needed for study participation as indicated)***	Date

*A minor's signature on this line indicates his/her assent to participate in this study. A minor's signature is not required if he/she is under 7 years old. Use the "Legal Guardian/Advocate/Witness" line for the parent's signature, and you may use the "Additional Signature" line for the second parent's signature, if required.

**The presence and signature of an impartial witness is required during the entire informed consent discussion if the patient or patient's legally authorized representative is unable to read.

***The "Additional Signature" line may be used for the second parent's signature, if required. This line may also be used for any other signature which is required as per federal, state, local, sponsor and/or any other entity requirements.

"If required" means that the signature line is signed only if it is required as per federal, state, local, sponsor and/or any other entity requirements.

SIGNATURE OF STUDY REPRESENTATIVE

I have explained the purpose of the research, the study procedures, identifying those that are investigational, the possible risks and discomforts as well as potential benefits and have answered questions regarding the study to the best of my ability.

Study Representative**** Date

****Study Representative is a person authorized to obtain consent. Per the policies of the University of Missouri Health Care, for any 'significant risk/treatment' study, the Study Representative must be a physician who is either the Principal or Co-Investigator. If the study is deemed either 'significant risk/non-treatment' or 'minimal risk,' the Study Representative may be a non-physician study investigator.

UNIVERSITY OF MISSOURI-COLUMBIA
Institutional Review Board

HIPAA AUTHORIZATION FORM

Authorization for the Use and Disclosure of Personal Health Information Resulting from Participation in a Research Study

FOR IRB USE ONLY
Acknowledged
Dan Vincent 3/2/09
IRB Authorized Representative Date

Principal Investigator's Name: Dr Hinton
Project # 1097239
Project Title: Hormonal and Bone Turnover Marker Response to an Acute Bout of Resistance or Plyometric Exercise

You have agreed to participate in the study mentioned above. This authorization form gives more detailed information about how your health information will be protected.

1. Description of the information
My authorization applies to the information described below. Only this information may be used and/or disclosed in accordance with this authorization:
Demographic and anthropomorphic information, results of bone density scan, blood tests, and medical history

2. Who may use and/or disclose the information
I authorize the following persons (or class of persons) to make the authorized use and disclosure of my PHI:
Principal investigator and graduate students research assistants.

3. Who may receive the information
I authorize the following persons (or class of persons) to receive my personal health information
University Missouri IRB, publications, research collaborators, and current and potential funding agencies

4. Purpose of the use or disclosure
My PHI will be used and/or disclosed upon request for the following purposes:
Publications and presentation that will not identify me, auditing, administrative and billing reviews, study outcomes including safety and efficacy

5. Expiration date or event
This authorization expires upon:
☐ The following date: _____
☐ End of research study
☒ No expiration date
☐ Other: _____

6. Right to revoke authorization
I understand that I have a right to revoke this authorization at any time. My revocation must be in writing in a letter sent to the Principal Investigator at 106 McKee Gym, University of Missouri, Columbia, MO 65211. I am

aware that my revocation is not effective to the extent that the persons I have authorized to use and/or disclose my PHI have already acted in reliance upon this authorization.

7. Statement that re-disclosures are no longer protected by the HIPAA Privacy Rule
I understand that my personal health information will only be used as described in this authorization in relation to the research study. I am also aware that if I choose to share the information defined in this authorization to anyone not directly related to this research project, the law would no longer protect this information. In addition, I understand that if my personal health information is disclosed to someone who is not required to comply with privacy protections under the law, then such information may be re-disclosed and would no longer be protected.

8. Right to refuse to sign authorization and ability to condition treatment, payment, enrollment or eligibility for benefits for research related treatment
I understand that I have a right not to authorize the use and/or disclosure of my personal health information. In such a case I would choose not to sign this authorization document I understand I will not be able to participate in a research study if I do not do so. I also understand that treatment that is part of the research project will be conditioned upon my authorization for the use and/or disclosure of my personal health information to and for use by the research team.

9. Suspension of right to access personal health information
I agree that I will not have a right to access my personal health information obtained or created in the course of the research project until the end of the study.

10. If I have not already received a copy of the University of Missouri Healthcare Privacy Notice, I may request one. If I have any questions or concerns about my privacy rights I should contact, the HS Privacy Officer at 573-882-9054 or the Campus Privacy Officer at 573-882-7254.

11. Individuals' signature and date
I certify that I have received a copy of the authorization.

_____ _____
Signature of Research Participant Date

_____ _____
Research Participant's Legally Authorized Representative Date

_____
Describe Representative Authority to Act for the Participant

Acute Exercise and Bone Turnover in Males

Subject number _____

These questions ask about your medical and sports history. Please fill in the blank or circle the appropriate response.

1. Current age _____

2. Race/Ethnicity:

   Caucasian/White
   African-American/Black
   Hispanic/Latino/Mexican-American
   Asian American/Pacific Islander
   Other: _____ (specify)

3. Do you regularly consume soy foods? yes no

4. Do you currently take a calcium supplement? yes no

   What dose? _____mg

5. Are you currently taking any medications?

   _____(specify)

6. Are you currently taking any anti-inflammatory steroids?

   _____(specify)

7. Have you ever been diagnosed with a disease that affects bone

   (Cushing's disease, hyperthyroidism, leukemia, Crohn's disease, chronic

   liver disease, rheumatoid arthritis, etc)? yes no

   What was the diagnosis? _____

   When was the diagnosis? _____

   What is your current treatment? _____

8. Have you had any sports related fractures or stress fractures in the past 5 years?

Fracture: yes no
Number _____
Location on

body_____

_____

Year_____

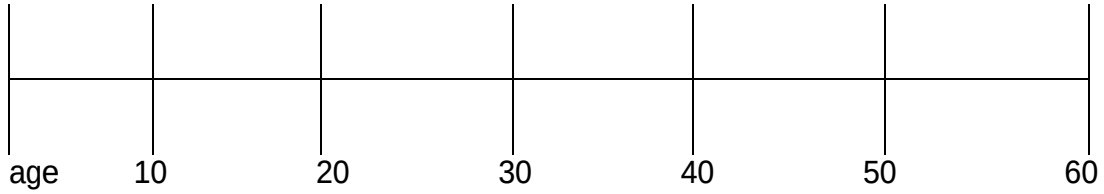
Stress Fracture: yes no
Number _____
Location on

body_____

_____

Year_____

9. Please use the timeline below to indicate what sports (include strength training) you trained for or played competitively during your lifetime.



For each sport listed please describe approximately how many hours per week you trained or competed in this sport. If you competed, please indicate the level of competition.

Sport	Ages	Hours per week	Weeks per year	Level of Competition

10. What sports do you compete in now (include strength training)? How many hours per week do you train for or compete in this sport? At what level do you compete?

Sport	Hours per week	Weeks per year	Level of Competition

Acute Exercise and Bone Study

Physical **A**ctivity **R**eadiness **Q**uestionnaire

Subject number _____

These questions ask about your readiness to participate in this research study's physical activity component. Please read each question carefully and answer each one honestly. Check **YES** or **NO**

YES	NO	
☐	☐	11. Has you doctor ever said that you have a heart condition and that you should only do physical actvitiy recommended by a doctor?
☐	☐	12. Do you feel pain in your chest when you do physical activity?
☐	☐	13. In the past month, have you had chest pain when you were not doing physical activity?
☐	☐	14. Do you lose your balance because of dizziness or do you ever lose consciousness?
☐	☐	15. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?
☐	☐	16. Do you have high blood pressure (systolic $\geq$ 140 mm Hg or diastolic $\geq$ 90 mm Hg)?
☐	☐	17. Is your doctor currently prescribing drugs (for example, water pills) for blood pressure or heart condition?
☐	☐	18. Do you know of any other reason why you should not do physical activity?
☐	☐	19. Do you have a family history of heart disease (for example, heart attack or sudden death) in first degree relative (male <55 years or female <65 years old)?
☐	☐	20. Currently a smoker or quit within previous 6 months?

☐ ☐ 21. Do you have high cholesterol? (Total cholesterol > 200 mg/dl, high-density lipoprotein cholesterol < 35 mg/dl, low-density lipoprotein > 130 mg/dl)

☐ ☐ 22. Do you have impaired fasting glucose? (for example ≥ 110 mg/dl)

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

_____

Name Date

_____

Participant Signature Study Representative

University of Missouri-Columbia
Exercise Physiology Lab

Acute Exercise and Bone Turnover in Males
Physical Activity Log

Subject # _____
Week_____ Dates __/__/__-__/__/__
Weight (lbs)_____

Day	Date	Exercise Mode	Total Time (hrs:min)	Distance (miles/yards)	Average Pace (min/mile, mph, yds/min)	Max HR	Avg HR	Intensity (L, M, H)
Mon								
Tues								
Wed								
Thur								
Fri								
Sat								
Sun								

University of Missouri-Columbia Exercise Physiology Lab

Acute Exercise and Bone Turnover in Males
7-Day Dietary Record

In order for us to assess your dietary habits, we need for you to log your diet for seven consecutive days.

- Don't choose days that are holidays or special occasions when you are eating in a way that is not representative of your normal dietary habits.
- Please record immediately after each meal or snack.
- Please record portion sizes as accurately as possible.

Please return the records to the lab at your earliest convenience.

University of Missouri-Columbia
Exercise Physiology Lab

Acute Exercise and Bone Turnover in Males
Dietary Record

Subject #:_____ Date:_____ Day:_____

Meal	Food	Amount (tsp, cup, oz)	Time of day
Breakfast:			
Lunch:			
Dinner:			
Snack:			

Comments: _____