The Effects of a Polynutrient Dietary Supplement on Physiological Measures and Mood State in Resistance Trained Men

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THE EFFECTS OF A POLYNUTRIENT DIETARY SUPPLEMENT ON PHYSIOLOGICAL MEASURES AND MOOD STATE IN RESISTANCE TRAINED MEN

By

Thomas Incledon

A DISSERTATION

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THE EFFECTS OF A POLYNUTRIENT DIETARY SUPPLEMENT ON PHYSIOLOGICAL MEASURES AND MOOD STATE IN RESISTANCE TRAINED MEN

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The purpose of the present study was to test the acute effects of a dietary supplement, having as its major ingredient an extract of ginseng, on grip strength, lower body power output, cardiovascular markers, metabolic markers, hormones, and mood state. Twelve experienced resistance-trained men (28.3 ± 5.7 yrs) were randomly administered placebo (P), single dose (SD) and double dose (DD) of the supplement on separate days. Diet and activity levels were kept constant across testing days. On each day, subjects began with the Profile of Mood States (POMSpre1), blood draws (BDpre1), blood pressure (BPpre1), and heart rate (HRpre1) assessments, then ingested the drink and sat quietly for 30 minutes. BDpre2, BPpre2, and HR pre1 were then taken. Subjects performed the grip strength and cycle ergometer tests followed immediately by BDpost, HRpost, and BPpost and POMSpost. The testing session ended with blood draws, heart rates, and blood pressures being taken 30 (post30), 60 (post60), 120 (post120) and 180 (post180) minutes post exercise. Grip strength did not differ between P, SD, or DD treatments. Cycle ergometry peak power (PP), average power (AP) and total work (TW) were significantly higher for the SD and DD than P; however, no significant difference existed between SD and DD treatments. For LH and T significant differences were found among all treatment conditions. There were no significant treatment effects for HR, BP, glucose, insulin,
lactate, GH or PRL or for the POMS. There was a significant treatment*time interaction for ACTH (p < .05). Post hoc analysis indicated that at Tpost ACTH was significantly lower for D treatment vs P or S treatments (p < .05) and at Tpost60 ACTH was significantly lower for S and D treatments vs P treatment (p < .05). There was significant differences in C between the D treatment (260.45 ± 15.58 nmol•L⁻¹) and the P (336.08 ± 27.59 nmol•L⁻¹) and S (311.14 ± 21.01 nmol•L⁻¹) treatments (p < .001). There was a significant difference for T:C ratio values among P (0.0810 ± 0.0090), S (0.0960 ± 0.0130) and D (0.1410 ± 0.0190) treatments (p < .001). Acute ingestion of a polynutrient supplement containing a standardized ginseng tract, was able to increase PP, AP, TW LH, and testosterone and decrease ACTH and cortisol. No significant effects were found for GH, PRL, insulin, glucose, lactate, HR, BP or POMS scores. Acute ingestion of a polynutrient supplement was able to increase performance and the anabolic environment in resistance trained men.
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Chapter 1: Introduction

The use of dietary supplements which contain a variety of nutrients is a common practice among athletes and individuals concerned with maintaining a healthy lifestyle [1, 2]; however; the number of studies that have examined the effects of polynutrient dietary supplements are limited. One rationale for consuming these products is the belief that taking a number of dietary supplements simultaneously can increase the likelihood that a given nutrient will be available in sufficient amounts to meet an individual’s needs during physical or mental stress [3]. Another possible advantage is that ingesting multiple supplements at the same time may provide greater benefits than if each were taken independently.

One popular polynutrient dietary supplement, ProEndorphin (Nutraceutics Corp., Saint Louis, MO), contains a Panax ginseng (P. ginseng) extract and a number of other ingredients including: thiamin hydrochloride, riboflavin 5-phosphate, niacin, pyridoxine hydrochloride, cyanocobalamin, biotin, calcium-D-pantothenate, sodium, potassium, DL-phenylalanine, 2-dimethylaminoethanol, kola nut extract, inositol, and taurine. These ingredients have the potential to enhance performance through a number of mechanisms. Caffeine has been shown to improve performance in both endurance and power-based sports [4]. Additionally, DL-phenylalanine has been shown to improve mood [5]. And finally, the combination of sodium citrate, taurine, glucuronolactone, caffeine, inositol, niacinamide, calcium-D-pantothenate, pyridoxine hydrochloride, and cyanocobalamin has been shown to improve physical performance and mood [6, 7].

Studies investigating P. ginseng extracts administered without other dietary supplements have yielded mixed results. Acute treatment studies indicated no effect of P.
ginseng on endurance or strength performance in healthy young adults [8], and limited
effect on the immune response to an acute exercise in healthy sedentary men [9].
Additionally, ginseng extract does not appear to improve psychomotor performance
during exercise without negatively affecting exercise capacity [10]. Similarly, short-term
treatment of ginseng had no effect on endurance exercise performance in healthy young

Chronic treatment studies also indicate that ginseng has no effect on endurance
exercise performance [12, 13], lactate threshold [13] or rate of recovery from exhaustive
exercise [14] in healthy men. Additionally, supplementation with P. ginseng has been
shown to be ineffective at improving pulmonary function or endurance exercise capacity
in patients with COPD [15].

In contrast, short-term research involving a P. ginseng extract combined with
vitamins, minerals, and other ingredients was shown to increase the physical working
capacity of healthy male sports teachers during treadmill testing [16].

Taking into account the popularity of polynutrient supplement use by adults
engaged in strength training [1] and power sports [17], and the lack of pertinent studies
with these populations, the aim of the present study was to test the effects of a
polynutrient dietary supplement containing P. ginseng on neuromuscular performance
and mood state in a sample of men with substantial weight-training histories.
Chapter 2: Methods

Subjects

Twelve resistance-trained men (mean ± SD age 28.3 ± 5.7 years, height 178.22 ± 6.84 cm, mass 90.41 ± 15.54 kg) with 11.4 ± 6.0 years of resistance training experience participated in the study. Subjects averaged 4.7 ± 0.7 and 3.0 ± 1.3 days per week for resistance and endurance training, respectively. Subjects’ ethnic backgrounds were Caucasian (5), Hispanic (5), African-American (1), and Bahamian (1). Sample size calculations using PASS software (Number Cruncher Statistical Systems, Kaysville, Utah) at \( \alpha = .05 \) indicated that a repeated measures design employing three treatment conditions requires sample sizes (n) 3 to 8 to yield power (P) values from 0.867 to >0.999.

All subjects completed a Physical Activity Readiness Questionnaire (rPAR-Q) [18, 19], and were prescreened for musculoskeletal, neurological, cardiovascular or other conditions for which resistance or power testing would be contraindicated. Prior to entrance into the study subjects completed a written informed consent approved by the University of Miami Subcommittee for the Use and Protection of Human Subjects.

Research Design

The study examined differences in the response variables due to ingestion of a single (SD) or double (DD) dose of ProEndorphin, or a placebo (P) consisting of a lime-flavored effervescent solution that was identical in color and taste to the supplement being tested, yet contained no active ingredients. The study used a double blind, placebo-controlled repeated measures protocol with a one-week washout between treatments. In
order to maintain the double blind conditions, all treatments involved ingesting drinks of the same taste, color, viscosity, and appearance. A research assistant randomly selected one of three coded powdered drink mixes (coded A, B, or C), prepared the appropriate drink (powder mixed with 180 mL of room temperature water in a colored container) and left the room to prevent contact with the subject or tester. Another assistant then administered the coded drink to the subject. For each treatment, the same protocol was always followed. The researchers and assistants involved in the study did not discuss test results or drink codes. The codes for the treatments were not broken until the data for all subjects were collected and analyzed.

**Timeline, Order of Testing and Blood Draws**

The timeline for the study is presented in Figure 1. The study protocol began with a two-day familiarization period. During the familiarization period the subjects were thoroughly informed concerning study goals and design. Additionally, subjects were taught the proper use of the grip strength dynamometer and the stationary cycle ergometer; and were allowed to practice the testing protocols that were to be used during each test. Previous research indicates that two consecutive familiarization sessions identical to the testing session provide sufficient practice to ensure test-retest reliability [20]. Equipment settings, including seat height and grip span were recorded so that they could be standardized for each subject across the testing conditions. At the conclusion of the second familiarization day, the order in which the supplements were to be administered was randomly assigned for each subject and samples were prepared and coded.
Subjects were then asked to return on days 8, 15 and 22 of the study so they might be tested under each treatment condition. They were contacted the day before each testing session and instructed not to eat after 10:00 PM. The testing protocol across the three testing days was standardized. Upon entering the laboratory, the subject was interviewed to confirm that he had not changed his diet nor ingested any supplements, caffeine or alcohol. We also confirmed that he was fasted, had been inactive at least 24 hours, and followed his typical sleep schedule (i.e. at least 6 hours per day). Finally he confirmed that he had not been exposed to any circumstances that would preclude him from testing (i.e. illness, injury, medications, etc). The subject then completed the Profile of Mood States (POMS_pre) in private room to reduce possible distractions. Next, a catheter was inserted in the subject’s antecubital vein, and the first blood draw (BD_pre1), blood pressure (BP_pre1), and heart rate (HR_pre1) were taken. The subject ingested the drink randomly assigned for that day and sat quietly for 30 minutes. A second blood draw (BD_pre2), blood pressure (BP_pre2), and heart rate (HR_pre1) was then taken. Next the subject performed the grip strength test followed by the cycle ergometer test. This was immediately followed by BD_post, HR_post, and BP_post and a post-test POMS (POMS_post). The testing session ended with blood draws, heart rates, and blood pressures being taken 30 (BD_post30, HR_post30, BP_post30), 60 (BD_post60, HR_post60, BP_post60), 120 (BD_post120, HR_post120, BP_post120) and 180 (BD_post180, HR_post180, BP_post180) minutes post exercise. Following testing the subject was provided food, and he was verified to be symptom free prior to being allowed to leave the laboratory.
Dietary Control

Subjects completed a 7-day food log while following their typical dietary patterns. In order to minimize the impact that dietary changes may have had on the treatment conditions, each subject was instructed to maintain his dietary pattern across the 21-day study period.

Activity Control

Subjects completed a previously validated 7-day activity log using their typical activity patterns. They were then asked to maintain the same weekly activity level throughout the duration of the study. Additionally, subjects were told to avoid exercise the 2 days before each treatment condition.

Performance Testing

To reduce the impact of diurnal variation across treatments subjects started testing between 8-10 AM. Additionally, for both performance tests consistent instructions were provided for all subjects and the same tone and volume were used each time to motivate subjects in order to minimize variability due to verbal cues.

Grip Strength Test Protocol (GST). Subjects performed the grip strength test using a handheld JAMAR hydraulic grip dynamometer (Asimow Engineering, Santa Fe Springs, CA, USA) using a standardized testing protocol. Prior to testing the grip span was adjusted to the distance that been determined during the familiarization days. Briefly, subjects were positioned in a straight-backed chair with both feet flat on the floor. The shoulder was adducted and neutrally rotated, the elbow was flexed at 90° with the
forearm in a neutral position and the wrist between 0 and 30° extension and between 0 and 15° ulnar deviation. The arm was not supported and the dynamometer was presented vertically and in line with the forearm to maintain standard forearm and wrist positions. Prior to testing, subjects warmed up using three contractions performed at 50%, 75%, and 90% of their maximum grip strength values established during the familiarization period. After the warm-up, 3 trials lasting three seconds each were performed separated by 60s passive recovery periods. During each trial, maximal grip values were read by the tester with the scale facing away from subject. The mean of the three trials was used for statistical analysis, as this method is preferred due to its higher test-retest reliability [21].

Cycle Ergometry Test Protocol. A Monark 883E (Monark Exercise AB, Vansbro, Sweden) cycle ergometer fitted with toe clips and an OptoSensor 2000 reading system interfaced with the laboratory computer (Sports Medicine Industries, Inc., St. Cloud, MN) was used to assess mechanical power. The OptoSensor 2000 measured flywheel velocity using reflective markers attached to the pedal sprockets and an optical sensor tachometer attached to the frame. Pedaling initiated data collection. The raw data were stored on the computer and analyzed using the POWER software program (Sports Medicine Industries, Inc., St. Cloud, MN). The software program calculated peak power, average power, total work, and fatigue index for each trial. Subjects began the test seated so that the heels of the feet touch the pedal with the knee joints extended. The seat height had been established during the familiarization days. Their feet were secured by toe clips. In the starting position the left foot was forward with both feet parallel to the ground. The angle of the seat and angle of the handlebars were preset and did not vary between subjects,
trials, or conditions. All rides were preceded by a warm-up during which the subject using a flywheel load equal to ~1% of his body mass for five minutes at a metronome cadence of 30 rpm. No subject showed signs of fatigue (i.e. inability to maintain cadence or standing up while pedaling), during the warm-up period. During the test subjects performed eight 6s maximal effort sprints separated by 30s of active recovery at 60 rpm with 1 kg resistance [22]. Peak power (PP), average power (AP), fatigue index (FI), and total work (TW) were calculated for each individual trial. In addition, averages for these variables were calculated for all trials combined.

Cardiovascular Measures

One-minute heart rate (HR) and noninvasive real-time blood pressure values were measured using the NIBP100 (Biopac Systems, Inc., Santa Barbara, CA). The system employs a tonometric technique, which records heart rate and intra-arterial pulse pressure that has been previously validated [23]. The tonometric sensor is placed just distal of the styloid process and held in place with a wrist brace. The NIBP100 also incorporates a built-in oscillometric cuff measurement system to calibrate the relative intra-arterial pressure readings to absolute values. The system outputs a continuous analog waveform representative of the blood pressure. Oscillometric measurement is performed at user-defined intervals to insure the accuracy of the blood pressure waveform generated by the NIBP100. The waveform is collected by computer (MP100 System, Biopac Systems, Inc., Santa Barbara, CA). Software (AcqKnowledge Biopac Systems, Inc., Santa Barbara, CA) extracts the systolic, diastolic, and mean blood pressure values on a beat-to-beat basis.
Glucose and Lactate Analysis

Glucose was analyzed using the Glucometer Elite Diabetes Care System (Bayer Corporation, Pittsburgh, PA). The device has been previously validated [24]. Lactate analysis was performed with the Lactate Pro (Fact Canada Consulting Ltd, British Columbia, Canada). The accuracy of the Lactate Pro has also been demonstrated in the literature [25, 26].

Blood Hormone Analysis

Blood samples (5 ml) were drawn from the right arm by a certified phlebotomist using a Jelco™ (Critikon, Tampa, FL) catheter to reduce the potential risks to subjects and minimize the discomfort of repetitive needle sticks. Blood was collected in tubes containing either EDTA or a serum separator (SST). Each tube was centrifuged. The centrifuged EDTA treated tubes provided plasma for adrenocorticotropic hormone (ACTH) analysis, while the SST tubes provided serum for analyses cortisol (C), growth hormone (GH), insulin (I), luteinizing hormone (LH), prolactin (Prl), and testosterone (T) levels. The samples were stored at –20°C to –84°C. The hormones were analyzed using the Immulite Immunoassay System (Diagnostic Products Corporation, Los Angeles, CA), an automated chemiluminescent analyzer. For analysis, subjects’ serum/plasma samples were thawed, shaken, and 400 uL of the sample was pipetted into sample cups. The sample cups were placed into bar code labeled cup holders resting on a carousel. The treatment information for each label was entered manually via a keyboard interfaced with the analyzer. Hormone test kits were placed behind each cup holder. The cup holder and test kit were advanced forward on the carousel in a timed sequence. The analyzer
automatically pipetted the necessary sample size from the sample cup into the appropriate hormone test kit. After a 70-minute (two carousel revolutions and centrifugal wash) incubation period that involved a two-site chemiluminescent enzyme immunometric assay (CIA), the hormone concentration was printed out. The Immulite Immunoassay System [27] and hormone test kits are all approved by the FDA for in vitro diagnostic testing as moderately complex tests and were previously validated [28].

All assays were performed according to the manufacturers' recommendations by skilled technicians. Each hormone was measured in duplicate and the mean value was used in statistical analyses. The intra and inter-assay coefficients of variation of the assays were: ACTH 3.1% and 8.8%; C 6.8% and 9.9%; GH 5.3% and 5.7%; I 4.3% and 4.8%; LH 4.8% and 7.2%; Prl 6.8% and 9.6%; and T 5.8% and 7.9%.

**Profile of Mood States (POMS)**

Mood states were measured using the Profile of Mood States (POMS). This 65-item questionnaire has been validated for assessing acute changes in mood before and after exercise [29]. For analysis, scores (on a 5-point scale of 0–4) were grouped into six subscales: tension–anxiety (T–A), depression–dejection (D), anger–hostility (A–H), vigor (V), fatigue (F), and confusion (C). Subscale scores were converted to T-scores for statistical analysis, and overall mood disturbance was also calculated.

**Statistical Procedures**

For grip strength a three-way ANOVA was used to detect differences among the three supplement conditions. For cycle ergometry each dependent variable (PP, AP, TW
and FR) was evaluated using a 3 (conditions) x 8 (trials) repeated measures ANOVA. Each bloodborne measure was analyzed using a 3 (condition) x 7 (time) repeated measures ANOVA. When significant main effects or interactions were detected, LSD post hoc tests were used to determine the sources. Statistical significance was set a priori at $p<.05$. All statistical procedures were performed using IBM ® PASW Statistics 18.0 for Windows (SPSS Inc., Chicago, Illinois).
Chapter 3: Results

Grip Strength.

The results of the grip strength tests are displayed in Table 1. There were no significant effects for treatment, trial or treatment-trial interaction.

Cycle Ergometer Test.

There were significant differences in peak power (PP), average power (AP) and total work (TW) for treatment (p<.001). For PP, the P treatment (845.208 ± 41.288) was significantly lower than the SD (883.292 ± 41.245) and DD (895.427 ± 42.433) treatments; however, there was no significant difference between SD and DD treatments (see Figure 2). For AP, the P treatment (632.437 ± 35.302) produced significantly lower values than the SD (656.948 ± 35.615) and DD (683.417 ± 36.546) treatments; and once again, there was no significant difference between SD and DD treatments.

For TW, the SD (3941.688 ± 213.687) and DD (4100.500 ± 219.277) treatments produced significantly higher values than the P treatment (3794.625 ± 211.810) (p<.001) (see Figure 3). No significant difference was detected between the SD and DD treatments. There was no significant difference in fatigue index (FI) for treatment (p=.088).

There were significant differences in PP (p<.05) and FI (p<.005) across trials (see Figures 4 & 5); however no significant differences were seen for either AP or TW.

Cardiovascular.

The systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate
(HR) results are displayed in Table 2. There was no significant effects of treatment nor treatment by time interaction on HR, SBP, or DBP (p<.05). There was a significant difference between HR_{pre1} and HR_{post120} and HR_{post180} (p<.005). There was also a significant difference between HR_{post1}, HR_{post30}, HR_{post60} and all other heart rate time points (p<.004). There was also a significant difference between SBP_{post} and all other time points (p<.001).

Metabolic Measures.

The Metabolic Measures results are reported in Table 3. There was no significant treatment effect for glucose, insulin or lactate; however, there were significant changes in each variable across time (p<.001). For glucose, time points T_{pre1} and T_{post30} were found to be significantly different than T_{pre2}, T_{post} and T_{post60}. There was also a significant difference in glucose between time point T_{pre2} and all post-test values.

For insulin, time points T_{pre1}, T_{post} and T_{post60} were significantly different than T_{pre2}, T_{post30}, T_{post120} and T_{post180}. Additionally, insulin levels at time points T_{pre2} and T_{post30} were significantly different than T_{post120} and T_{post180}.

For lactate time points T_{pre1}, T_{post}, T_{post30} and T_{post60} were significantly different than all other time points. There was also a significant difference between time points T_{pre2} and T_{post120}.

There were no significant differences in LH, SHBG or T across time points nor were there significant treatment by time interactions. For LH (see Figure 6) significant differences were found among all treatment conditions (P=3.49 ± 0.59 mlU·mL⁻¹; S=3.85 ± 0.60 mlU·mL⁻¹; D=4.39 ± 0.61 mlU·mL⁻¹; p < .001). There were also significant
differences for T values (see Figure 6) among all conditions P (23.15 ± 1.71 nmol·L⁻¹), S (25.95 ± 2.82 nmol·L⁻¹) and D (30.28 ± 3.09 nmol·L⁻¹) treatments (p < .001).

There was a significant treatment*time interaction on ACTH (p < .05) (see Table 4). Post hoc analysis indicated that at T_post ACTH was significantly lower for D treatment vs P or S treatments (p < .05) and at T_post60 ACTH was significantly lower for S and D treatments vs P treatment (p < .05).

There was significant differences in C between the D treatment (260.45 ± 15.58 nmol·L⁻¹) and the P (336.08 ± 27.59 nmol·L⁻¹) and S (311.14 ± 21.01 nmol·L⁻¹) treatments (p < .001) (see Figure 7). There were significant differences for C values between time point T_pre1 and T_pre2, T_post, T_post60, T_post120 and T_post180 (p < .002). There were also significant differences between time point T_pre2 and time points T_post, T_post120 and T_post180 (p < .002). Significant differences in C values were also detected between the T_post value and T_post120 and T_post180 (p < .002). There were significant differences for C values between time points T_post30 and T_post60 time points T_post120 and T_post180 (p < .002). Finally, significant differences in C levels were detected between time points T_post120 vs. T_post180 (see Table 5).

There was a significant difference for T:C ratio values among P (0.0810 ± 0.0090), S (0.0960 ± 0.0130) and D (0.1410 ± 0.0190) treatments (p < .001) (see Figure 7). There was also a significant time effect for T:C Ratio (p < .001). There were significant differences between time point T_pre1 and T_post, T_post60, T_post120 and T_post180. There were also significant differences in T/C ratio between time point T_pre2 vs. T_post, T_post120 and T_post180. There were also significant differences between the T_post value and T_post120 and T_post180. Finally, there were significant differences for T/C ratio between time
points $T_{\text{post}30}$ and $T_{\text{post}60}$ and time point $T_{\text{post}120}$ (see Table 5).

There was no significant effect of treatment on GH or PRL ($p < .05$). There was a significant time effect for GH ($p < .05$). For GH values significant differences were seen between time point $T_{\text{pre}2}$ and time points $T_{\text{post}}, T_{\text{post}30}$ and $T_{\text{post}60}$. There were significant differences for GH values between time point $T_{\text{post}}$ and time points $T_{\text{post}30}$ and $T_{\text{post}180}$. Finally, there were significant differences for GH values between time point $T_{\text{post}30}$ and $T_{\text{post}180}$ (see Table 6).

There was also a significant time effect for PRL ($p < .001$). There were significant differences between time point $T_{\text{pre}1}$ and $T_{\text{pre}2}, T_{\text{post}30}, T_{\text{post}60}, T_{\text{post}120}$ and $T_{\text{post}180}$. There were also significant differences for PRL values between time point $T_{\text{pre}2}$ and $T_{\text{post}60}, T_{\text{post}120}$ and $T_{\text{post}180}$. Significant differences were detected between time point $T_{\text{post}}$ and $T_{\text{post}60}, T_{\text{post}120}$ and $T_{\text{post}180}$. There were significant differences for PRL values between time point $T_{\text{post}30}$ and time points $T_{\text{post}60}, T_{\text{post}120}$ and $T_{\text{post}180}$. Finally, there were significant differences between time point $T_{\text{post}60}$ and time points $T_{\text{post}120}$ and $T_{\text{post}180}$ (see Table 6).

Differences between pre- and post- treatment POMS scores are shown in Table 7. There were no significant differences in the POMS subscales due to treatment. There were, however, significant differences seen across time points for the fatigue subscale ($p < .05$), indicating subjects were more fatigued after the exercise trials. There were no significant differences between Total Mood Disturbance Scores (TMDS) due to treatments or time.
Chapter 4: Discussion

The present study examined the effects of a popular polynutrient dietary supplement containing thiamin hydrochloride, riboflavin 5-phosphate, niacin, pyridoxine hydrochloride, cyanocobalamin, biotin, calcium-D-pantothenate, sodium, potassium, DL-phenylalanine, P. ginseng extract, 2-dimethylaminoethanol, kola nut extract, inositol, and taurine on physical and biochemical performance markers in a physically active population. We compared the acute effects of a single dose and a double dose of the supplement against placebo. At the doses tested, the supplement had no effect on grip strength. This is in contrast to previous research with athletes using a similar polynutrient supplement, which demonstrated an increase in grip strength after five weeks of ingestion [30]. The differences in results may be due to differences in the ginseng extract itself, the type of subject or the duration of treatment prior to testing.

At the doses tested, however, the supplement did significantly increased PP, AP and TW. There was no significant difference between SD and DD treatments on PP, AP or TW performances. These findings are in agreement with previous research demonstrating an increase in workload during treadmill exercise, using a similar polynutrient supplement containing a ginseng extract [16]. The cycle ergometry exercise protocol used in this study design was very challenging for subjects which would explain the significant effect of trial on PP and FI.

The acute safety concerns regarding ingestion of the polynutrient supplement were addressed in this study. SBP, DBP and HR results were similar for all treatment groups. The ability to perform more work [16] and improve quality of life [31] while maintaining healthy cardiovascular responses has been reported previously for
polynutrient supplements. The possible mechanisms to explain these findings include increased nitric oxide levels [32], improved oxygen delivery to tissues [16] and increased ability to handle physical and mental stress [30].

There were no significant effects of polynutrient supplementation on glucose, insulin or lactate responses to repeated sprint cycle ergometry in this study. Given that the supplement increased TW and similar lactate responses were found, our findings agree with earlier work indicating lower lactate levels at the same work load [16].

While previous studies have investigated the effects of polynutrient, ginseng preparations on testosterone and/or cortisol levels, there are limited data regarding their effects on other hormones in humans. Our study demonstrated an increase in LH and T, and a decrease in ACTH and C levels after polynutrient, ginseng treatment. These combined results produced a much higher T:C Ratio, indicating that polynutrient supplement ingestion may increase the anabolic environment. There were no significant effects on SHBG, GH or PRL levels. In contrast to our findings, previous research showed no significant effects of ginseng ingestion on testosterone, cortisol, growth hormone, and insulin-like growth factor-1 responses to acute resistance exercise [33]. Our study design used a specialized ginseng preparation in conjunction with multiple vitamins, minerals and other agents, which could easily explain the differences in research results. In addition, it was previously demonstrated that ginsenoside-Rb1 acts on the anterior pituitary directly and stimulates LH levels in male rats [34]. By increasing LH secretion from the anterior pituitary, levels of testosterone could also be stimulated. Testosterone modulates the nitric oxide-cGMP signaling pathway and androgenesis in Leydig cells [35]. Given that ginseng-extract preparations and ginsenoside-Rb1 have also
been shown to increase nitric oxide levels [32, 36] there may be direct and indirect effects (i.e. via testosterone) of ginseng to increase nitric oxide.

In the present study there was a significant increase in ACTH at \( T_{\text{post}} \) for all treatments, however it was significantly lower for the D treatments vs the P and S treatments. There was also a significant reduction in C after the D treatment vs the P and S treatments indicating that at higher dose this supplement can reduce the acute stress response seen with intense exercises.

There were no significant effects of polynutrient treatment on POMS subscale Tscores or Total Mood Disturbance Scores (TMDS). Previous studies have demonstrated improvements in mood after ginseng preparation ingestion [37]. In the present study subjects were highly motivated and very positive, so there could be little if any improvement in mood. A reduction in fatigue was expected but the results were not statistically significant. A larger samples size may be needed to detect a significant reduction in fatigue.
Chapter 5: Conclusions

Acute ingestion of a polynutrient dietary supplement containing a ginseng extract increased cycle ergometry performance and improved the anabolic environment (higher testosterone, lower cortisol, increased T:C Ratio). Future work should examine if the benefits of supplement ingestion improve with chronic administration, identify the specific ginsenosides involved, and possible impact of gut bacteria on nutrient metabolism and hence effects of polynutrient supplement.
REFERENCES


27. Babson, A.L., et al., The IMMULITE(TM) assay tube: A new approach to


Figure 1. Study timeline.

<table>
<thead>
<tr>
<th>Day 1 and Day 2</th>
<th>Day 8</th>
<th>Day 15</th>
<th>Day 22</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

Familiarizations  Condition 1  Condition 2  Condition 3
Figure 2. The effects of treatment on cycle ergometry peak and average power.

*Significantly higher than placebo (p<.001).
Figure 3. The effect of treatment on total work during cycle ergometry.

*Significantly higher than placebo (p<.001).
**Figure 4. Differences in cycle ergometer peak power across trials.**

*Significantly different than trials 6, 7, and 8 (p<.05).
† Significantly different than trials 5, 6, 7, and 8 (p<.05).
‡ Significantly different than trials 3, 7, and 8 (p<.05).
Figure 5. Differences in cycle ergometer fatigue index across trials.

*Significantly different than trials 4, 5, 7, and 8 (p<.005).
† Significantly different than trial 7 (p<.005).
Figure 6. The effects of treatment on lutenizing hormone and testosterone levels.

*Significant differences among all treatment conditions (p<.001).
Figure 7. The effects of treatment on cortisol and testosterone/cortisol ratio.

*Significant differences among all treatment conditions (p<.001).
**Significant differences between the double dose and all other treatment conditions (p<.001).
Table 1. Results for Grip Strength Testing

<table>
<thead>
<tr>
<th>Grip strength (kg)</th>
<th>Placebo</th>
<th>Single Dose</th>
<th>Double Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
<td>56.1 ± 7.7</td>
<td>53.9 ± 8.5</td>
<td>54.1 ± 7.4</td>
</tr>
<tr>
<td>Trial 2</td>
<td>55.6 ± 8.5</td>
<td>52.7 ± 8.0</td>
<td>55.2 ± 8.2</td>
</tr>
<tr>
<td>Trial 3</td>
<td>54.8 ± 8.0</td>
<td>52.6 ± 8.2</td>
<td>55.1 ± 7.1</td>
</tr>
<tr>
<td>Average of trials</td>
<td>55.5 ± 7.9</td>
<td>53.1 ± 8.1</td>
<td>54.8 ± 7.5</td>
</tr>
</tbody>
</table>
### Table 2. Cardiovascular responses across time.

<table>
<thead>
<tr>
<th>Time Point</th>
<th>Heart Rate (BPM)</th>
<th>Systolic Blood Pressure (mmHg)</th>
<th>Diastolic Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tₚｒₑ₁</td>
<td>58.3 ± 9.1*</td>
<td>122.9 ± 9.6</td>
<td>69.0 ± 5.7</td>
</tr>
<tr>
<td>Tₚｒₑ₂</td>
<td>56.7 ± 12.9</td>
<td>123.9 ± 12.2</td>
<td>70.5 ± 6.6</td>
</tr>
<tr>
<td>Tₚₒṣᵗ</td>
<td>97.9 ± 23.0†</td>
<td>150.2 ± 22.6‡</td>
<td>71.6 ± 11.4</td>
</tr>
<tr>
<td>Tₚₒṣᵗ₃₀</td>
<td>70.7 ± 12.6†</td>
<td>124.9 ± 10.1</td>
<td>68.9 ± 5.7</td>
</tr>
<tr>
<td>Tₚₒṣᵗ₆₀</td>
<td>65.5 ± 11.0†</td>
<td>122.9 ± 10.2</td>
<td>69.1 ± 5.8</td>
</tr>
<tr>
<td>Tₚₒṣᵗ₁₂₀</td>
<td>58.3 ± 9.7</td>
<td>123.2 ± 11.0</td>
<td>69.7 ± 4.7</td>
</tr>
<tr>
<td>Tₚₒṣᵗ₁₈₀</td>
<td>55.6 ± 9.0</td>
<td>124.3 ± 7.4</td>
<td>70.3 ± 7.7</td>
</tr>
</tbody>
</table>

* Significantly different than HRₚₒṣᵗ₁₂₀ and HRₚₒṣᵗ₁₈₀ (p<.005)
† Significantly different than all other time points (p<.004)
‡ Significantly different than all other time points (p<.001)
Table 3. Metabolic response across time.

<table>
<thead>
<tr>
<th>Time Point</th>
<th>Glucose (mmol/L)</th>
<th>Insulin (pmol/L)</th>
<th>Lactate (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>T&lt;sub&gt;pre1&lt;/sub&gt;</td>
<td>5.16 ± 0.55&lt;sup&gt;a&lt;/sup&gt;</td>
<td>38.10 ± 13.90&lt;sup&gt;c&lt;/sup&gt;</td>
<td>1.40 ± 0.50&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;pre2&lt;/sub&gt;</td>
<td>5.56 ± 0.83&lt;sup&gt;b&lt;/sup&gt;</td>
<td>48.80 ± 19.50&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.80 ± 1.10&lt;sup&gt;f&lt;/sup&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;post&lt;/sub&gt;</td>
<td>4.85 ± 0.58</td>
<td>34.50 ± 10.10&lt;sup&gt;e&lt;/sup&gt;</td>
<td>11.10 ± 4.40&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;post 30&lt;/sub&gt;</td>
<td>5.18 ± 0.77&lt;sup&gt;a&lt;/sup&gt;</td>
<td>50.70 ± 23.60&lt;sup&gt;d&lt;/sup&gt;</td>
<td>5.40 ± 2.00&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;post 60&lt;/sub&gt;</td>
<td>4.90 ± 0.55</td>
<td>35.50 ± 14.20&lt;sup&gt;c&lt;/sup&gt;</td>
<td>3.90 ± 2.10&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;post 120&lt;/sub&gt;</td>
<td>4.99 ± 0.69</td>
<td>28.40 ± 11.60</td>
<td>2.80 ± 1.60</td>
</tr>
<tr>
<td>T&lt;sub&gt;post 180&lt;/sub&gt;</td>
<td>4.90 ± 0.84</td>
<td>26.50 ± 13.00</td>
<td>2.30 ± 1.40</td>
</tr>
<tr>
<td>Mean</td>
<td>5.08 ± 0.13</td>
<td>37.50 ± 4.74</td>
<td>4.09 ± 1.24</td>
</tr>
</tbody>
</table>

<sup>a</sup>Significantly different than T<sub>pre2</sub>, T<sub>post</sub>, and T<sub>post 60</sub> (p < .001)
<sup>b</sup>Significantly different than all post-test values (p < .001)
<sup>c</sup>Significantly different than T<sub>pre2</sub>, T<sub>post30</sub>, T<sub>post120</sub> and T<sub>post 180</sub> (p < .001).
<sup>d</sup>Significantly different than T<sub>post120</sub> and T<sub>post 180</sub> (p < .001).
<sup>e</sup>Significantly different than all other time points (p < .001).
<sup>f</sup>Significantly different than T<sub>post120</sub> (p < .001).
Table 4. Changes in ACTH across time.

<table>
<thead>
<tr>
<th>ACTH (pmol/L)</th>
<th>Placebo (P)</th>
<th>Single Dose (S)</th>
<th>Double Dose (D)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>T pre1</td>
<td>5.39 ± 3.30</td>
<td>4.79 ± 2.13</td>
<td>4.15 ± 2.00</td>
</tr>
<tr>
<td>T pre2</td>
<td>3.71 ± 2.08</td>
<td>3.53 ± 1.58</td>
<td>3.22 ± 1.20</td>
</tr>
<tr>
<td>T post</td>
<td>23.02 ± 25.72</td>
<td>13.14 ± 10.52</td>
<td>6.99 ± 5.21 a</td>
</tr>
<tr>
<td>T post 30</td>
<td>6.26 ± 5.14</td>
<td>4.87 ± 3.20</td>
<td>3.59 ± 3.09</td>
</tr>
<tr>
<td>T post 60</td>
<td>3.77 ± 1.61</td>
<td>3.00 ± 1.26 b</td>
<td>2.85 ± 1.40 b</td>
</tr>
<tr>
<td>T post 120</td>
<td>3.07 ± 1.61</td>
<td>2.87 ± 1.10</td>
<td>2.62 ± 0.83</td>
</tr>
<tr>
<td>T post 180</td>
<td>2.84 ± 1.45</td>
<td>2.81 ± 0.73</td>
<td>2.79 ± 0.75</td>
</tr>
<tr>
<td>Mean</td>
<td>6.87 ± 1.30 a</td>
<td>5.00 ± 0.67 a</td>
<td>3.75 ± 0.43 a</td>
</tr>
</tbody>
</table>

aD was significantly different than P or S at T post (p<.05)

bS and D significantly different than P at T post 60 (p<.05)
Table 5. Changes in cortisol and testosterone/cortisol ratio across time points.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Cortisol (nmol/L)</th>
<th>T:C Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time Point</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>T\text{pre1}</td>
<td>384.42 ± 112.72\textsuperscript{a}</td>
<td>0.0765 ± 0.0362\textsuperscript{d}</td>
</tr>
<tr>
<td>T\text{pre2}</td>
<td>328.32 ± 110.70\textsuperscript{b}</td>
<td>0.0843 ± 0.0442\textsuperscript{b}</td>
</tr>
<tr>
<td>T\text{post}</td>
<td>294.60 ± 99.57\textsuperscript{c}</td>
<td>0.1042 ± 0.0506\textsuperscript{c}</td>
</tr>
<tr>
<td>T\text{post 30}</td>
<td>337.46 ± 128.19\textsuperscript{c}</td>
<td>0.0967 ± 0.0785\textsuperscript{c}</td>
</tr>
<tr>
<td>T\text{post 60}</td>
<td>313.76 ± 195.29\textsuperscript{c}</td>
<td>0.1100 ± 0.0779\textsuperscript{c}</td>
</tr>
<tr>
<td>T\text{post 120}</td>
<td>252.28 ± 152.66</td>
<td>0.1356 ± 0.1027</td>
</tr>
<tr>
<td>T\text{post 180}</td>
<td>207.06 ± 58.36</td>
<td>0.1338 ± 0.0652</td>
</tr>
<tr>
<td>Mean</td>
<td>305.56 ± 43.04</td>
<td>0.1059 ± 0.0232</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Significantly different than T\text{pre2}, T\text{post}, T\text{post 60}, T\text{post120}, and T\text{post 180} (p<.002)

\textsuperscript{b}Significantly different than T\text{post}, T\text{post120}, and T\text{post 180} (p<.002)

\textsuperscript{c}Significantly different than T\text{post120}, and T\text{post 180} (p<.002)

\textsuperscript{d}Significantly different than T\text{post}, T\text{post 60}, T\text{post120}, and T\text{post 180} (p<.001)

\textsuperscript{e}Significantly different than T\text{post 120} (p<.001)
Table 6. Changes in growth hormone and prolactin across time points.

<table>
<thead>
<tr>
<th>Variable</th>
<th>GH (ug/L)</th>
<th>Prolactin (pmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time Point</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>$T_{\text{pre1}}$</td>
<td>0.18 ± 0.32</td>
<td>429.22 ± 168.91$^d$</td>
</tr>
<tr>
<td>$T_{\text{pre2}}$</td>
<td>0.39 ± 1.59$^a$</td>
<td>340.34 ± 105.54$^c$</td>
</tr>
<tr>
<td>$T_{\text{post}}$</td>
<td>0.49 ± 1.04$^b$</td>
<td>355.55 ± 113.25$^e$</td>
</tr>
<tr>
<td>$T_{\text{post 30}}$</td>
<td>1.31 ± 1.84$^c$</td>
<td>326.71 ± 120.40$^e$</td>
</tr>
<tr>
<td>$T_{\text{post 60}}$</td>
<td>1.04 ± 1.64</td>
<td>285.63 ± 98.16$^f$</td>
</tr>
<tr>
<td>$T_{\text{post 120}}$</td>
<td>0.56 ± 1.85</td>
<td>254.35 ± 83.41</td>
</tr>
<tr>
<td>$T_{\text{post 180}}$</td>
<td>0.23 ± 0.57</td>
<td>235.45 ± 92.13</td>
</tr>
<tr>
<td>Mean</td>
<td>0.60 ± 0.63</td>
<td>318.18 ± 28.15</td>
</tr>
</tbody>
</table>

$^a$Significantly different than $T_{\text{post}}, T_{\text{post 30}}$ and $T_{\text{post 60}}$ (p<.05)
$^b$Significantly different than $T_{\text{post 30}}$ and $T_{\text{post 180}}$ (p<.05)
$^c$Significantly different than $T_{\text{post 180}}$ (p<.05)
$^d$Significantly different than $T_{\text{pre2}}, T_{\text{post}}, T_{\text{post 60}}, T_{\text{post 120}}$, and $T_{\text{post 180}}$ (p<.001)
$^e$Significantly different than $T_{\text{post}60}, T_{\text{post} 120}, T_{\text{post} 180}$ (p<.001)
$^f$Significantly different than $T_{\text{post} 120}$ and $T_{\text{post} 180}$ (p<.001)
Table 7. T scores and TMDS for Profile of Mood States.

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tension/anxiety</td>
<td>34.00 ± 4.92</td>
<td>35.00 ± 5.52</td>
</tr>
<tr>
<td>Depression</td>
<td>40.06 ± 5.88</td>
<td>40.31 ± 7.73</td>
</tr>
<tr>
<td>Anger/hostility</td>
<td>41.00 ± 5.86</td>
<td>41.00 ± 5.93</td>
</tr>
<tr>
<td>Vigor</td>
<td>45.00 ± 13.50</td>
<td>44.00 ± 13.06</td>
</tr>
<tr>
<td>Fatigue</td>
<td>42.00 ± 6.85</td>
<td>47.00 ± 9.37</td>
</tr>
<tr>
<td>Confusion</td>
<td>33.00 ± 4.53</td>
<td>33.00 ± 5.47</td>
</tr>
<tr>
<td>TMDS</td>
<td>144.00 ± 28.26</td>
<td>153.00 ± 33.37</td>
</tr>
</tbody>
</table>