Effects of Ginseng on Secretory IgA, Performance, and Recovery from Interval Exercise

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ABSTRACT

ENGELS, H.-J., M. M. FAHLMAN, and J. C. WIRTH. Effects of Ginseng on Secretory IgA, Performance, and Recovery from Interval Exercise. *Med. Sci. Sports Exerc.*, Vol. 35, No. 4, pp. 690–696, 2003. **Purpose:** This study examined the efficacy of ginseng to modulate secretory immunoglobulin A (SIgA), exercise performance, and recovery from repeated bouts of strenuous physical exertion. **Methods:** Using a double-blind, placebo-controlled, randomized design, 38 active healthy adults supplemented their diets with a standardized ginseng concentrate (400 $mg \cdot d^{-1}$ of G115; equivalent to 2 g of *Panax ginseng* C.A. Meyer root material) or placebo (lactose) for 8 wk. Before and after the intervention, each subject performed three consecutive 30-s Wingate tests interspersed with 3-min recovery periods under controlled laboratory conditions. SIgA secretion rate (S-SIgA) and the relation of SIgA to total protein were calculated from measures of saliva flow rate (SFR), and absolute SIgA and salivary protein concentrations in timed, whole unstimulated saliva samples collected before and after exercise testing. Peak and mean mechanical power output ($W \cdot kg^{-1}$) was measured with an infrared-beam optical-sensor array, and exercise recovery heart rate (HRR) was determined electrocardiographically. **Results:** Twenty-seven subjects (12 placebo, 15 ginseng) completed the study. Compared with rest, S-SIgA, SIgA:protein ratio, and SFR were lower after exercise at baseline ($P < 0.05$). Similarly, both peak and mean mechanical power output declined ($P < 0.01$) across consecutive Wingate tests. Postintervention minus preintervention change scores for salivary parameters, exercise performance, and HRR were similar between ginseng- and placebo-treated groups ($P > 0.05$). **Conclusion:** These findings do not support the hypothesis that ginseng may affect mucosal immunity as indicated by changes in secretory IgA at rest and after an exercise induced state of homeostatic disturbance. Supplementation with ginseng fails to improve physical performance and heart rate recovery of individuals undergoing repeated bouts of exhausting exercise. **Key Words:** *PANAX GINSENG*, INTENSE EXERCISE, IMMUNE FUNCTION, ERGOGENIC AID

Extracts of *Panax ginseng* C.A. Meyer, more commonly known as Chinese or Korean Ginseng, have a long and revered history of use in traditional medicine in Asian countries (27). They also have become readily accepted in many Western nations where people often consume these preparations, expecting that they may offer relief and protection in the fight against stress, disease, and fatigue (5,27). The increasingly widespread use of ginseng has led to renewed calls for research to assess its effectiveness as a therapeutic and ergogenic nutritional agent (4,28).

One particularly intriguing area of ginseng research concerns its ability to affect immune responses. Clinical trials of selected cellular immune parameters in healthy adults and people with suppressed immunity so far have shown equivocal results (12,20–22,26). Although some researchers have proposed that ginseng enhances the immune system (23) and should be considered a nutritional aid in the prevention and treatment of respiratory illnesses (20), further empirical tests are warranted to study the immunomodulatory efficacy of this popular herb.

The secretory immune system of the mucosal tissues of the upper respiratory tract provides the first barrier to colonization by pathogenic microorganisms that can cause upper respiratory tract infections (URTI) (17,18). Secretory immunoglobulin A (SIgA) is the primary immunoglobulin contained in the secretions of the mucosal immune system, and its levels in salivary fluids correlate more closely with resistance to respiratory infection caused by certain viruses than do serum antibodies or other immune parameters (17,18). Changes in SIgA are now widely recognized as an important physiological biomarker regarding the integrity of the human mucosal immune system (13,24).

It is generally acknowledged that strenuous exercise results in immune suppression and that immunosuppressed individuals may be at a greater risk for URTI (19). However, although the efficacy of nutritional agents as countermeasures to exercise immunosuppression and URTI is receiving notable research attention (19), the value of ginseng for that purpose, and specifically its immunomodulatory ability at the level of the respiratory mucosa, is largely unknown. Moreover, considering that the benefits of this herb are said

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to be more readily observed in times of fatigue and when conditions are especially imposing (5,6), research is needed to determine whether supplementation with ginseng can improve performance and alleviate fatigue when functional abilities are diminished such as through repetitive bouts of strenuous physical work (8). Therefore, the specific aims of this study were to examine the effects of prolonged dietary ginseng (*Panax ginseng* C.A. Meyer) supplementation on a) secretory immunoglobulin A, b) physical performance, and c) recovery responses of individuals undergoing exhausting interval exercise.

METHODS

Subjects. Thirty-eight university students took part in this study. Before experimental testing, subjects were cleared for research participation based on a physician's review of their medical history, present health status, and supine 12-lead resting ECG. In addition, each subject was queried about the present use of medications and dietary supplements, recent signs and symptoms of URTI, and habitual physical activity level (3). Only healthy, habitually active persons who were not taking any medications or dietary supplements, were free of signs and symptoms of URTI for the previous week, and exhibited no contraindications to strenuous exercise testing were included in the study. After receiving an explanation of the purpose, possible risks, and benefits associated with participating in the research, students provided their written informed consent. All study procedures were approved by the Institutional Human Investigation Committee.

Study design and general procedures. The study was conducted using a randomized, double-blind, placebo-controlled research design. Experimental testing was performed throughout the day, but each subject was always laboratory tested at the same time of day before and after an 8-wk (56–58 d) dietary supplementation period with *Panax ginseng* C.A. Meyer or placebo. Subjects were instructed to avoid strenuous physical exertion on the day before and leading up to the test, and to abstain from food and caffeine consumption for 3 h before a scheduled research appointment. The pre- and posttrial laboratory sessions were the same except that at the posttrial subjects also were required to answer specific questions to check compliance with the basic requirements for regular daily intake of the prescribed study capsules, avoidance of other dietary supplements, and the occurrence of any adverse events. Moreover, throughout the intervention period, subjects kept weekly research records to document the type, frequency, duration, and severity of any symptoms of URTI that they experienced unrelated to allergies.

Interval exercise and recovery protocol. Exercise testing was performed on a calibrated, friction-loaded leg cycle ergometer (Monark™, Varberg, Sweden) and consisted of three consecutive 30-s Wingate tests (Wingate I, II, and III) that were separated by 3-min recovery periods (11). Each of the exercise tests was performed against a braking force of 4.41 J·pedal revolution^{-1}·kg body weight^{-1}. Subjects were instructed to avoid pacing and maintain a maximal effort throughout each 30-s exercise bout. For safety and to improve exercise tolerance, timed recovery periods for each subject were always divided into an initial active recovery phase (pedaling at a controlled 50 W work rate for 90 s) followed by a passive recovery phase (quiet sitting). The duration of the passive recovery was 90 s after Wingate I and II but was extended to the 15-min recovery time point after Wingate III. Before exercise testing, each subject performed a standard warm-up protocol (11), and the cycle ergometer seat height was adjusted for leg length.

Power output measurement. Mechanical power output during exercise was measured using an infrared-beam optical-sensor array and computer interface (SMI OptoSensor 2000™ and SMI Power™ software, Sport Medicine Industries, St. Cloud, MN). Individual peak power values (PP; W·kg^{-1}) were determined from the highest power output produced during a single 5-s period of each 30-s test. Mean power values (MP; W·kg^{-1}) were calculated as the average of the total power output generated during each 30-s test.

Recovery heart rates. Exercise recovery heart rate (HRR) was assessed by telemetry (Model G-2400T, EatonCare Telemetry, Ann Arbor, MI) using a bipolar V_5 lead set and continuous ECG chart-paper recordings. Before statistical analysis, each subject's ECG record was transposed into consecutive 30-s heart rate interval values (beats·min^{-1}) for both Wingate I and II exercise recovery periods (i.e., 3 HRR scores each for active and passive recovery). After completion of Wingate III, heart rate was recorded continuously throughout the initial 90-s active recovery and again at min 3, 5, 10, and 15 during the extended passive recovery.

Saliva collection and analytical assays. Unstimulated whole saliva was collected continuously for 4 min each before the warm-up and again 5 min after completion of the interval exercise protocol. Samples were measured for volume to the nearest 0.1 mL, stored at −70°C until the end of the study, and then analyzed in one batch using previously established procedures (11,18) involving an enzyme-linked immunosorbent assay (ELISA) for the determination of absolute SIgA (μg·mL^{-1}) and a diagnostic kit (Bradford; Bio-Rad Total Protein Assay Kit, Bio-Rad Laboratories, Hercules, CA) based on Bradford's standard assay to measure total salivary protein (TSP; mg·mL^{-1}). SIgA secretion rate (S-SIgA; μg·min^{-1}) was computed from saliva flow rate (SFR; μl·min^{-1}) and absolute SIgA concentrations as measured directly in the ELISA. Because local drying of the oral surfaces after exercise affects salivary protein content, the absolute concentration of SIgA was also expressed relative to total protein (μg SIgA·mg protein^{-1}) (11,18). The coefficient of variation for SIgA and protein was 10.9% and 2.9%, respectively. For the ELISA used in this study, the minimum concentration of SIgA that can be distinguished from 0 is 2.5 μg·mL^{-1}. The minimum sensitivity of the protein assay kit is 0.35 μg·mL^{-1}.

Ginseng and placebo treatment. The ginseng treatment consisted of G115 (Pharmaton, Lugano, Switzerland),

a qualitatively and quantitatively standardized aqueous extract of *Panax ginseng* C.A. Meyer in capsular format. Independent research has repeatedly shown that this preparation is manufactured using high quality-control standards (10). A dosage level of 400 $mg \cdot d^{-1}$, equivalent to 2 g *Panax ginseng* C.A. Meyer root material (25), and an 8-wk trial intervention were chosen to extend the limited available experimental evidence relative to the immunological and performance altering potential of this standardized ginseng concentrate in humans (8–10,20–22). The placebo treatment consisted of gelatin capsules (Capsugel, Greenwood, SC) containing lactose (Baxter Scientific, Chicago, IL) and had the same size, weight, shape, and color as the ginseng capsules. Study participants were given a 60-d supply of either the ginseng or placebo capsules to take home after the preintervention laboratory session, and they were instructed to consume four 100 mg capsules per day, two in the morning (e.g., with breakfast) and two in the evening (e.g., with dinner), throughout the trial period.

Statistical analysis. One-way ANOVAs were initially performed to compare the basic physical (age, weight, and height), resting and postexercise salivary (SIgA, S-SIgA, SIgA:protein, SFR, and TSP), exercise performance (PP and MP), and recovery (HRR) characteristics of the ginseng (G) and placebo (P) study groups at baseline (preintervention). Independent of assignment to a specific treatment group, the effect of the experimental exercise protocol on saliva based parameters at the pretrial was then evaluated using a paired *t*-test (rest vs postexercise) and on physical performance characteristics (PP, MP) using repeated measures ANOVA with planned contrasts between Wingate I and II, and Wingate II and III. In the main analysis, the same salivary and physical performance parameters, as well as HRR, were then examined by ANOVA using postintervention minus preintervention change scores. Finally, between- and within-group differences in habitual physical activity levels (work, leisure, sport, and total activity) were analyzed using Wilcoxon rank sum tests. Statistical analyses were performed using the Statistical Analysis System (SAS, release 8, 1999, SAS Institute, Cary, NC). For all statistical tests, the significance level was set at $P < 0.05$. Values are reported as mean ± SEM.

RESULTS

Of the 38 subjects initially enrolled in this trial, 11 failed to complete one or more basic study requirements. Final data analyses were performed on complete exercise testing and HRR measurements obtained from 27 subjects (17 females, 10 males) of which 15 were in the ginseng and 12 in the placebo group. Analyses for saliva derived data was confined to 25 subjects (17 females, 8 males) of which 15 were in the ginseng and 10 in the placebo group.

An examination of pretrial values revealed no between group differences in physical characteristics (Table 1), interval exercise performance (PP and MP for Wingate I, II, and III), and HRR responses at baseline ($P > 0.05$). Similarly, resting and postexercise measures for absolute SIgA, SIgA:protein ratio, TSP, and SFR were similar between groups at the start of the trial ($P > 0.05$). Although within the normal range of expected values from this laboratory (11) and likely due to sampling error, both resting and postexercise S-SIgA levels at baseline were significantly lower in P (rest: 38.8 ± 6.8 $\mu g \cdot min^{-1}$; postexercise: 25.1 ± 5.1 $\mu g \cdot min^{-1}$) compared with G (rest: 62.1 ± 5.7 $\mu g \cdot min^{-1}$; postexercise: 42.8 ± 4.2 $\mu g \cdot min^{-1}$) ($P < 0.05$).

TABLE 1. Basic subject characteristics.

Variable	Placebo ($N = 12$)	Ginseng ($N = 15$)
Age (yr)	26.3 ± 1.9	26.1 ± 1.7
Body mass (kg)	67.7 ± 3.5	67.6 ± 3.4
Height (cm)	169.3 ± 2.1	170.2 ± 1.9
THPA[a]	8.99 ± 0.74	9.03 ± 0.48

Data are mean ± SEM.
[a] THPA, total habitual physical activity level; score is the sum of work, sport, and leisure activity indices according to Baecke et al. (3).

Compared with rest, and independent of assignment to a specific treatment group, postexercise values at the pretrial were lower for S-SIgA (35.7 ± 3.6 $\mu g \cdot min^{-1}$), SIgA:protein ratio (16.8 ± 1.8 μg SIgA·mg $protein^{-1}$), and SFR (377.6 ± 50.9 $\mu L \cdot min^{-1}$) ($P < 0.05$) but were not different for absolute SIgA (128.6 ± 16.4 $\mu g \cdot mL^{-1}$) and TSP (10.9 ± 2.7 $mg \cdot mL^{-1}$) ($P > 0.05$). Moreover, planned comparisons between Wingate I and II, and Wingate II and III showed that both peak and mean power output ($W \cdot kg^{-1}$) declined across consecutive Wingate tests ($P < 0.05$).

Analysis of variance on change scores (postintervention minus preintervention) revealed that the treatment (ginseng vs placebo) had no effect on SIgA (G: −29.9 ± 21.4 $\mu g \cdot mL^{-1}$; P: −39.0 ± 48.9 $\mu g \cdot mL^{-1}$), S-SIgA (G: +1.5 ± 12.5 $\mu g \cdot min^{-1}$; P: +14.9 ± 9.9 $\mu g \cdot min^{-1}$), SIgA:protein ratio (G: −4.3 ± 3.9 μg SIgA·mg $protein^{-1}$; P: +2.8 ± 3.1 μg SIgA·mg $protein^{-1}$), TSP (G: −1.2 ± 0.7 $mg \cdot mL^{-1}$; P: −4.1 ± 4.3 $mg \cdot mL^{-1}$), and SFR (G: +166.0 ± 54.7 $\mu L \cdot min^{-1}$; P: +89.0 ± 69.1 $\mu L \cdot min^{-1}$) at rest ($P > 0.05$). Moreover, it did not affect how any of the same parameters changed after the exhausting interval exercise protocol ($P > 0.05$), i.e., SIgA: G: −36.5 ± 29.3 $\mu g \cdot mL^{-1}$, P: +63.7 ± 53.1 $\mu g \cdot mL^{-1}$; S-SIgA: G: −15.2 ± 11.6 $\mu g \cdot min^{-1}$, P: −7.9 ± 9.9 $\mu g \cdot min^{-1}$; SIgA:protein ratio: G: +2.3 ± 3.5 μg SIgA·mg $protein^{-1}$, P: −2.2 ± 4.5 μg SIgA·mg $protein^{-1}$; TSP: G: +3.3 ± 6.9 $mg \cdot mL^{-1}$, P: +10.8 ± 6.5 $mg \cdot mL^{-1}$; and SFR: G: −48.7 ± 70.0 $\mu L \cdot min^{-1}$, P: −113.0 ± 57.3 $\mu L \cdot min^{-1}$ ($P > 0.05$). Study-group–specific findings (mean ± SEM) for pre- and postintervention salivary measures at rest and postexercise are illustrated in Figure 1 (SIgA, S-SIgA, and SIgA:protein) and Figure 2 (TSP and SFR).

Change scores for physical performance characteristics (PP and MP) for Wingate I, II, and III were observed to be similar between ginseng- and placebo-treated study groups ($P > 0.05$) (Fig. 3). Specifically, postintervention minus preintervention changes in PP were +0.04 ± 0.17 $W \cdot kg^{-1}$ for G and +0.31 ± 0.17 $W \cdot kg^{-1}$ for P for Wingate I ($P > 0.05$); +0.01 ± 0.13 $W \cdot kg^{-1}$ for G and +0.32 ± 0.14 $W \cdot kg^{-1}$ for P for Wingate II ($P > 0.05$); and −0.08 ± 0.15

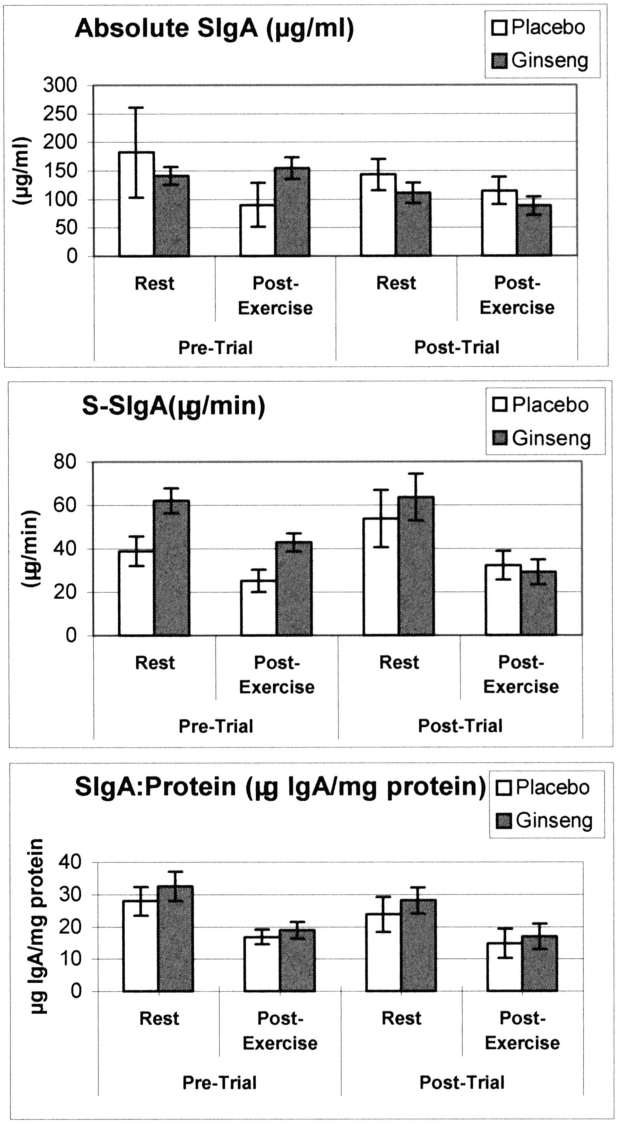

FIGURE 1—Concentrations of absolute SIgA, S-SIgA, and SIgA: protein at rest and after exhausting interval exercise before and after an 8-wk dietary supplementation period with ginseng or placebo. Values are mean ± SEM. See text for explanation of significant effects.

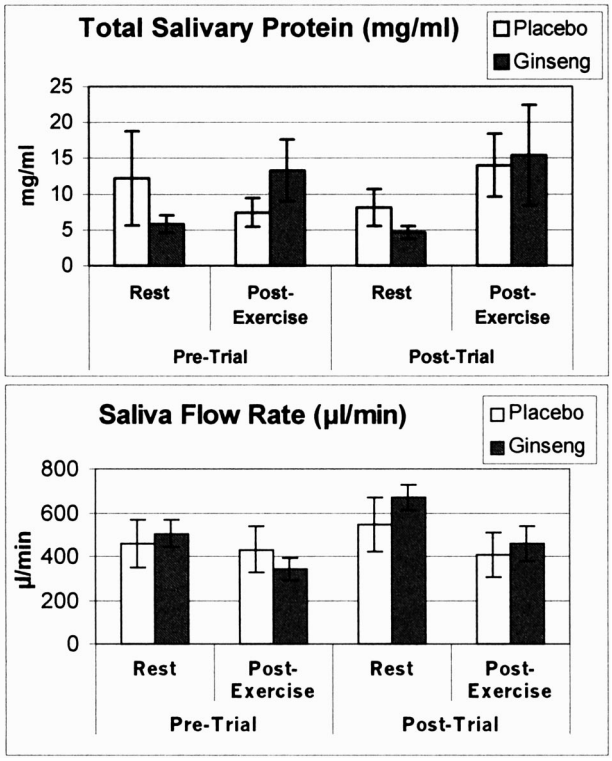

FIGURE 2—Total salivary protein concentration and saliva flow rate at rest and after exhausting interval exercise before and after an 8-wk dietary supplementation period with ginseng or placebo. Values are mean ± SEM. See text for explanation of significant effects.

$W \cdot kg^{-1}$ for G and $+0.25 \pm 0.20$ $W \cdot kg^{-1}$ for P for Wingate III ($P > 0.05$). The corresponding changes in MP were $+0.03 \pm 0.09$ $W \cdot kg^{-1}$ for G and -0.03 ± 0.14 $W \cdot kg^{-1}$ for P for Wingate I ($P > 0.05$); -0.09 ± 0.07 $W \cdot kg^{-1}$ for G and -0.06 ± 0.08 $W \cdot kg^{-1}$ for P for Wingate II ($P > 0.05$); and -0.13 ± 0.07 $W \cdot kg^{-1}$ for G and -0.08 ± 0.08 $W \cdot kg^{-1}$ for P for Wingate III ($P > 0.05$).

There was no significant difference between treatment groups in postintervention minus preintervention change scores for any of the 30 s HRR interval recordings, both active and passive, after Wingate I, II, and III ($P > 0.05$) (Fig. 4). Sequential active HRR change scores after Wingate I were $+1.5 \pm 1.3$ beats·min^{-1}, $+0.7 \pm 1.8$ beats·min^{-1}, and -0.4 ± 1.8 beats·min^{-1} for G, and $+2.6 \pm 2.4$ beats·min^{-1}, $+3.0 \pm 2.8$ beats·min^{-1}, and $+1.3 \pm 2.8$ beats·min^{-1} for P. Subsequent passive HRR change scores after Wingate I were -2.3 ± 2.1 beats·min^{-1}, -2.0 ± 3.1 beats·min^{-1}, and -2.1 ± 2.7 beats·min^{-1} for G, and $+2.9 \pm 3.6$ beats·min^{-1}, $+1.5 \pm 3.6$ beats·min^{-1}, and $+2.3 \pm 3.4$ beats·min^{-1} for P. Active HRR change scores after Wingate II were $+1.0 \pm 1.8$ beats·min^{-1}, -1.4 ± 2.5 beats·min^{-1}, and -1.2 ± 1.7 beats·min for G, and $+3.0 \pm 2.0$ beats·min^{-1}, $+2.8 \pm 2.3$ beats·min^{-1}, and $+2.8 \pm 2.7$ beats·min^{-1} for P, whereas passive HRR change scores after Wingate II were -1.7 ± 1.7 beats·min^{-1}, -1.6 ± 2.0 beats·min^{-1}, and -1.0 ± 2.0 beats·min^{-1} for G, and $+2.1 \pm 3.3$ beats·min^{-1}, $+0.6 \pm 3.4$ beats·min^{-1}, and $+3.1 \pm 3.3$ beats·min^{-1} for P. After Wingate III, active HRR change scores were $+0.6 \pm 1.8$ beats·min^{-1}, -1.2 ± 1.7 beats·min^{-1}, and -0.4 ± 2.4 beats·min for G, and $+1.1 \pm 1.3$ beats·min^{-1}, -0.4 ± 1.8 beats·min^{-1}, and -0.2 ± 2.4 beats·min^{-1} for P. Finally, extended HRR change scores for minute 3, 5, 10, and 15 after Wingate III were -0.1 ± 3.6 beats·min^{-1}, $+1.6 \pm 2.2$ beats·min^{-1}, $+2.3 \pm 2.5$ beats·min^{-1}, and $+0.4 \pm 3.0$ beats·min^{-1} for G, whereas they were $+1.7 \pm 2.3$ beats·min^{-1}, $+1.3 \pm 3.5$ beats·min^{-1}, $+1.6 \pm 3.5$ beats·min^{-1}, and $-0.8 \pm 0.4.2$ beats·min^{-1} for P.

Weekly research logs indicated a total of nine incidences of URTI signs and symptoms in 6 of the 27 subjects (3 ginseng, 3 placebo) during the intervention period. Episodes lasted from between 2 and 8 d, and ranged from mild (isolated stuffy nose and sore throat) to severe (multi-symptom sinus cold/flu requiring elimination of one or more daily activities). Planned double-blinded evaluations at the post-

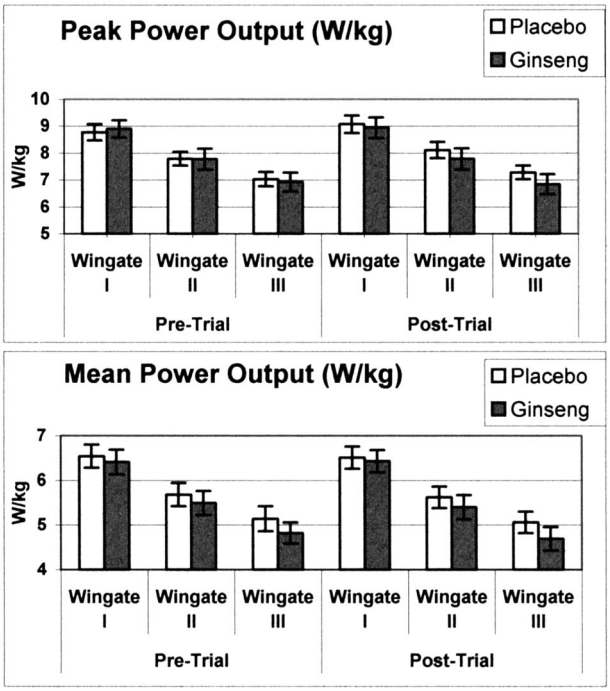

FIGURE 3—Peak and mean power output during Wingate I, II, and III before and after an 8-wk dietary supplementation period with ginseng or placebo. Values are mean ± SEM. See text for explanation of significant effects.

trial session revealed no incidences of adverse events likely attributable to the intake of treatment capsules. Moreover, an analysis of habitual physical activity indices (3) showed that there were no differences in the group total activity scores and its three components (work, sport, and leisure) between ginseng and placebo groups and that these scores were similar at the beginning and end of the trial ($P > 0.05$). Relative to established norms (3), the mean total habitual physical activity level for the participants of this study was above average for young adults.

DISCUSSION

The first major finding of this study was that ginseng (400 $mg \cdot d^{-1}$ of G115 for 8 wk) had no effects on mucosal immunity as measured by both resting and immediate post-exercise changes of SIgA. This failure to demonstrate an effect of ginseng at the level of the respiratory mucosa parallels findings by Srisurapanon et al. (26), who noted no beneficial immunomodulatory action of a standardized *Panax ginseng* extract (300 $mg \cdot d^{-1}$ for 8 wk) on peripheral blood leukocytes and lymphocyte subpopulations (CD3, CD4, CD8, CD4/CD8 ratio, CD19, and CD 25) in young healthy males. Similarly, Gaffney et al. (12) recently was unable to show an effect of another *Panax ginseng* preparation (equivalent to 2 $g \cdot d^{-1}$ of dried root) on total T-cells, CD4, CD8, CD4/CD8, B-lymphocytes, and natural killer cells of endurance athletes after a 6-wk intervention period.

On the other hand, after from 4 to 12 wk of dietary G115 intake at a dosage of 200 $mg \cdot d^{-1}$, Scaglione and colleagues (20–22) reported increases in natural killer cell activity, phagocytosis, chemotaxis, CD3, CD4, and CD4/CD8 ratio. Their studies involved healthy adults (22) and patients who suffered from chronic bronchitis (21) or were recommended for anti-

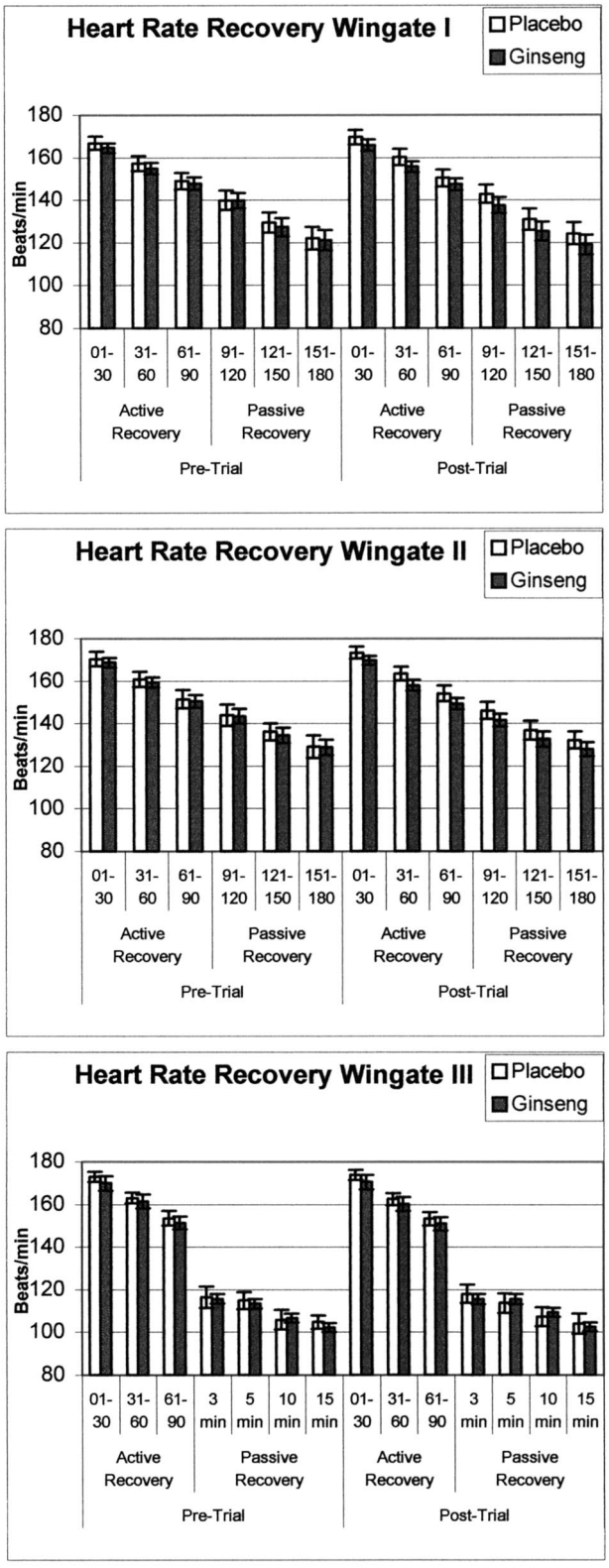

FIGURE 4—Heart rate recovery (mean ± SEM) after exhausting interval exercise before and after an 8-wk dietary supplementation period with ginseng or placebo. Data are shown as 30-s interval values obtained during active (01–90 s) and passive recovery (91–180 s, 3, 5, 10, and 15 min).

influenza vaccination (20). Although both animal (14,25) and human (7,16,23) *in vitro* research offers repeated support for the hypothesis that whole ginseng extracts or constituents thereof have immunomodulatory activity, conflicting data from the clinical human *in vivo* studies so far preclude definite conclusions about its efficacy at common levels used. Research is especially needed to help better identify the precise effector arms of the immune system that may or may not serve as the primary target of ginseng action.

Although ginseng failed to change the SIgA response, the observed exercise induced reductions in S-SIgA, SIgA:protein ratio, and SFR corroborate earlier findings of the acute effects of exhausting interval exercise on the mucosal immune system (11,18). Reasons for the suppression of SIgA are not completely understood but may involve a reduction in the migration of plasma cells, produced from B-cells, synthesizing and secreting SIgA due to sympathetically evoked vasoconstriction in the oral submucosa or changes in the transport of SIgA molecules across the mucosal epithelium (17). Moreover, it appears that a reduction in SFR accounts in part for this response (11,18).

Over the 8-wk trial duration, a total of nine occurrences of URTI symptoms were recorded by six (3 ginseng, 3 placebo) of the 27 subjects, and they were equally distributed between ginseng (5/15) and placebo (4/12) groups. These findings are not in accord with earlier clinical therapeutic trials that indicate an important role of ginseng (G115) in the prevention or treatment of respiratory illnesses (20,21). Moreover, although it has been proposed that a decrease of mucosal immunity caused by intense interval exercise in itself may be related to an increased risk of URTI (18), only three incidences were reported in the initial three weeks of the study. This substantiates recent data showing that completion of a single strenuous interval exercise session seems not to be associated with a heightened susceptibility to URTI in the weeks immediately after the test (11).

As summarized by Bahrke and Morgan (4), perpetual claims that the intake of ginseng is associated with an improved ability to adapt to many kinds of stresses, including those imposed by physical work, has over the last decade stimulated a number of randomized, placebo-controlled, double-blind investigations to more closely examine its value as a performance enhancing aid for exercise. The majority of these studies found that prolonged dietary intake (3–8 wk) of standardized *Panax ginseng* preparations had no significant effects on selected physiological parameters (e.g., $\dot{V}O_2$, blood lactate, and heart rate), performance, or HRR in rested, healthy adults undergoing single bouts of progressive (1,9,10) or supramaximal exercise (8). To extend this research, and specifically to test the proposition that the effects of ginseng are more readily apparent when conditions are especially taxing and the body is fatigued (5,6), this study employed an established, fatiguing interval exercise protocol (11). Findings suggest no ergogenic benefits of ginseng compared to placebo for PP, MP, and HRR using this form of repetitive, exhaustive physical work.

The desire to provide a precise scientific validation of the potential mechanisms of action and effects of ginseng is continuing to drive a considerable amount of basic research (27). However, although these studies often indicate that ginseng can produce effects via a variety of different physiological pathways and that multiple constituents of the plant are involved (2), it is equally important to acknowledge that recent controlled clinical studies in humans mostly provide no compelling evidence in support of the efficacy of this complex herb (4,28). Therefore, it is prudent to remain skeptical about claims which often attribute a variety of health and human performance related benefits to its intake at current levels used (8).

The G115 ginseng treatment utilized in this study is at the upper end of the clinically recommended daily dose for *Panax ginseng* (1–2 g of root material or equivalent preparations; based on ref. 5) but is substantially lower than the effective treatment levels typically employed with *in vitro* and animal model studies (12). Also particularly relevant to this study, although ginsenosides (a group of triterpenoid saponins) are the main active principles of ginseng, some research indicates a possibly important role of parts of the polysaccharide fraction of the ginseng plant in the modulation of the immune system (15,29). These findings may point out the necessity to standardize whole ginseng preparations for human clinical research not solely on the basis of their ginsenosides (G115 is titrated at a 4% ginsenosides level) but also based on other constituents with established biological activity.

In summary, this study has shown that prolonged dietary intake of a qualitatively and quantitatively standardized extract of *Panax ginseng* C.A. Meyer is not associated with important changes in secretory IgA at rest and after an exercise induced state of homeostatic disturbance. In addition, ginseng does not affect physical performance and recovery responses with repeated bouts of exhausting exercise in young, healthy adults.

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