

Chromium and exercise training: effect on obese women

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ABSTRACT

GRANT, K. E., R. M. CHANDLER, A. L. CASTLE, and J. L. IVY. Chromium and exercise training: effect on obese women. *Med. Sci. Sports Exerc.*, Vol. 29, No. 8, pp. 992-998, 1997. Chromium supplementation may affect various risk factors for coronary artery disease (CAD) and non-insulin-dependent diabetes mellitus (NIDDM), including body weight and composition, basal plasma hormone and substrate levels, and response to an oral glucose load. This study examined the effects of chromium supplementation ($400 \mu\text{g}\cdot\text{d}^{-1}$), with or without exercise training, on these risk factors in young, obese women. Chromium picolinate supplementation resulted in significant weight gain in this population, while exercise training combined with chromium nicotinate supplementation resulted in significant weight loss and lowered the insulin response to an oral glucose load. We conclude that high levels of chromium picolinate supplementation are contraindicated for weight loss in young, obese women. Moreover, our results suggest that exercise training combined with chromium nicotinate supplementation may be more beneficial than exercise training alone for modification of certain CAD and NIDDM risk factors.

CHROMIUM SUPPLEMENTATION, BODY WEIGHT, BODY COMPOSITION, GLUCOSE, INSULIN, LIPIDS

A "clustering" of risk factors has been associated with the development and progression of coronary artery disease (CAD) and non-insulin-dependent diabetes mellitus (NIDDM). These factors include body weight and composition (7,26,35), plasma glucose and insulin levels (20,26), and basal plasma lipid values (triglycerides, total cholesterol, low density lipoprotein-cholesterol (LDL-C) and high-density-lipoprotein cholesterol (HDL-C)) (5,26).

Trivalent chromium is a mineral essential for normal insulin function (18,30). Some but not all previous research (1,2,9,13,19,22,25,27,29) suggests that chromium supplementation may favorably alter the above noted risk factors noted above. Chromium is thought to cause these changes via its potentiating effect on insulin (24). Both endurance and resistance exercise training have also been

shown to alter some of these variables in a similar manner (1,10,17,23,31,32,34).

Although chromium supplementation shows promise as an intervention for altering the risk factors involved in the development of CAD and NIDDM, results published to date are contradictory and inconclusive. Additionally, as the effects of chromium supplementation seem to be similar to those seen with exercise training, interactions between these treatments should be investigated. Previous work observing concurrent chromium supplementation and exercise training has been restricted to effects on body weight and composition, with conflicting results (6,11,12,14,16).

Chromium picolinate is the most heavily used, studied, and promoted chromium compound, but *in vitro* work suggests that chromium nicotinate may be also viable in modifying the aforementioned risk factors. In this study, chromium picolinate was of primary interest, but it was thought that preliminary data on chromium nicotinate, tested under the best conditions (combined with exercise training), would be valuable as well. The present study of the effects of chromium supplementation on young, obese women had two objectives: first, to determine if chromium picolinate supplementation alone favorably alters body weight and composition, glucose tolerance, and plasma lipids, and whether these effects could be augmented with exercise training; and second, to provide data on the effectiveness of chromium nicotinate supplementation combined with exercise training.

METHODS

Subjects. Subjects were 43 healthy, sedentary, obese females. Various statistical cut-off definitions are used for obesity, but for the purpose of this investigation a subject was considered "obese" if she had higher than the recommended body fat percentage for young women (the recommended percentage is 20-25%) (4). We recognize that a portion of our population was only mildly obese. Prior to acceptance, questionnaires were used to determine the health status and activity patterns of the subjects. None of the subjects documented any health prob-

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lems, nor medication for such conditions. Age ranged from 18 to 35 yr with a mean of 24.4 ± 0.70 yr. Initial weight ranged from 50.8 to 96.1 kg with a mean of 71.3 ± 1.9 kg. Percent body fat as determined by hydrostatic weighing ranged from 25.0 to 45.0% with a mean of $33.0\% \pm 0.91$. Initial $\dot{V}O_{2\max}$ values ranged from 1.53 to $3.30 \text{ L}\cdot\text{min}^{-1}$, with a mean value of $2.38 \pm 0.13 \text{ L}\cdot\text{min}^{-1}$. Each subject was informed of the potential risks and benefits associated with participation before signing an informed consent document. The study was approved by the Institutional Review Board of The University of Texas.

Experimental design. Subjects were randomly assigned to one of four treatment groups: chromium picolinate supplementation without exercise training (CP), exercise training with placebo (E/P), exercise training with chromium picolinate supplementation (E/CP), and exercise training with chromium nicotinate supplementation (E/CN). The treatment period was 9 wk. The effects of stage of the menstrual cycle were not controlled. Although pre- and post-testing were conducted at different stages of the presumed 28-d menstrual cycle, the effects of this, if any, were randomized among all study participants and thus should not have influenced the data.

Chromium supplements and placebo tablets were prepared by Shaklee, Inc., USA (San Francisco, CA) and shipped to the investigator in coded bottles of 100 tablets each. Subjects were given 16 tablets each week containing chromium picolinate (200 μg), chromium nicotinate (200 μg) or placebo (inert ingredients). Subjects were given verbal instructions to take one tablet each morning and evening throughout the treatment and post-testing period, resulting in a dosage of 400 μg daily for those receiving chromium supplements. Subjects were asked to return unused tablets each week. Compliance was measured by counting returned tablets. There were no problems with compliance.

Exercise training consisted of a cross-training program with the following components:

1) Step aerobics—a type of aerobic dancing using upbeat music, a bench of 12–24 inches in height, and an instructor to guide participants through a series of moves designed to provide a full-body aerobic workout. These 1-h classes were attended twice a week by each exercising subject and were taught by certified instructors. Subjects were allowed to pick their own bench height. Instructors gradually increased exercise intensity as the study progressed.

2) Cycling—cycling exercise was conducted twice a week for 30 min at a target heart rate of 75–80% maximal heart rate (determined during the initial $\dot{V}O_{2\max}$ test). Universal Aerobicycles (Universal Gym Equipment, Inc., Cedar Rapids, IO) were used. Target heart rate was programmed into the ergometer at the beginning of each session and monitored during exercise with an on-

board computer adjusting resistance to maintain target heart rate.

3) Resistance training—resistance training was conducted twice a week with The Powercise Fitness System (TruTrac Therapy Products, Inc., Temecula, CA). Five separate machines in which resistance is set by an electronic braking device were used. This was a “double positive” system eliciting concentric-only contractions from agonist/antagonist muscle groups. The five machines exercised all major muscle groups of the upper and lower body. Each subject’s maximum strength was determined initially by lifting progressively heavier weights with each repetition (10-lb increments) until the load could no longer be lifted. Once maximal strength had been determined, workout weights were automatically set at 50% maximal strength, and the subject was guided through a work-out consisting of three sets of 15 repetitions on each machine. Subjects followed a pacing light which allowed approximately 1.5 s per concentric contraction. Rest time between sets was 25–28 s. Time between machines was not tightly controlled. Subjects were encouraged throughout the study to increase the weight lifted while still completing three sets of 15 repetitions, and these increases were documented. Subjects were also instructed to do three sets of 20 “abdominal crunches” (sit-ups).

All training sessions were supervised by at least one investigator. Attendance, heart rates during cycling exercise, and resistance training parameters (weight lifted and completion of repetitions and sets) were recorded. Subjects were asked not to alter their diet during the course of the study.

Experimental protocol. During the week prior to initiation of treatment, subjects were weighed on two separate occasions using a Health-o-Meter scale (Continental Scale Corporation, Bridgeview, IL) with a sensitivity of ± 200 g and subjected to body composition analysis via hydrostatic weighing (3). $\dot{V}O_{2\max}$ was determined on a treadmill using a modified Burke protocol, with inspired volumes and expired gasses measured as previously described for our laboratory (37). Fasting blood samples were drawn on two separate days, and an oral glucose tolerance test (OGTT) was conducted as subsequently described.

During the 9-wk treatment period, supplementation and exercise training were administered as detailed above. In the 9th wk of treatment, all pre-test measurements were repeated. Tests were administered in the same order both pre- and post-treatment. Supplementation and exercise training continued through the post-testing period. All subjects involved in exercise training participated in a step aerobics session two days prior to the post-OGTT and rested on the day prior to the test, resulting in approximately 40 h between the last bout of exercise and the OGTT.

TABLE 1. Anthropometric data.

	CP	E/P	E/CP	E/CN
Body weight (kg)				
Pre	66.30(±3.79)	76.40(±3.06)	69.80(±3.39)	70.60(±4.16)
Post	68.20(±4.21)*	77.10(±2.88)	69.70(±3.53)	69.50(±3.94)*
Body fat (%)				
Pre	33.37(±1.94)	32.18(±1.87)	33.45(±1.82)	32.84(±1.92)
Post	33.80(±1.92)	31.45(±1.62)	32.83(±2.18)	31.81(±1.89)
Fat mass (kg)				
Pre	22.70(±2.52)	25.20(±2.27)	23.90(±2.43)	24.00(±2.81)
Post	23.70(±2.67)	24.50(±1.88)	23.50(±2.69)	22.80(±2.55)
Fat free mass (kg)				
Pre	43.50(±1.78)	51.20(±1.60)	45.80(±1.51)	46.50(±1.68)
Post	44.50(±1.84)	52.60(±1.64)	46.20(±1.38)	46.90(±1.77)

Values are mean ± SE. CP, chromium picolinate supplementation; E/P, exercise with placebo; E/CP, exercise with chromium picolinate supplementation; E/CN, exercise with chromium nicotinate supplementation. * = significantly different from pre.

Sample collection and analyses. Plasma for hormone and substrate analysis was obtained between 7 and 9 a.m. after a 12-h fast which included no caffeine or nicotine. Blood (10 mL) was collected via venipuncture of an antecubital vein. Determination of glucose and insulin response to an oral glucose load was conducted on the same day as one of the pre-treatment blood samples. Subjects ingested a room-temperature beverage containing 100 g of dextrose (Tru-Glu 100 orange/carbonated, 10 oz. Fischer Scientific, Pittsburgh, PA), and blood samples (3 mL) were obtained from a 21-gauge indwelling venous catheter (Baxter Healthcare, Deerfield, IL) in an antecubital vein prior to ingestion and 15, 30, 60, 90, 120, and 180 min post-ingestion.

All blood samples were anti-coagulated with 250 mL ethylenediaminetetraacetic acid (EDTA) (Sigma Chemical Company, St. Louis, MO). Aliquots of whole blood (200 mL) from basal samples were used for glycosylated hemoglobin determination via an affinity resin column, colorimetric, endpoint procedure (Sigma Diagnostics, St. Louis, MO). The remaining samples were centrifuged at $1,000 \times g$ for 15 min.; plasma was then removed and frozen at -20°C for subsequent analysis. Insulin concentration was determined by radioimmunoassay (ICN Biomedicals, Inc., Costa Mesa, CA). Plasma samples were analyzed by enzymatic assay for glucose, triglycerides and total cholesterol (Sigma Diagnostics). HDL-C was determined enzymatically following the precipitation of LDL-C and VLDL-C (Sigma Diagnostics). LDL-C was calculated with the equation: $\text{LDL-C} = (\text{Total cholesterol}) - (\text{HDL-C}) - (\text{Triglycerides}/5)$ (Sigma Diagnostics). Standards were run with each assay to verify consistency. All samples were run in duplicate, with each subject's samples run sequentially.

Statistical analyses. A multivariate ANOVA was run for each variable on pre- and post-values across all treatment groups. Pre- and post-values, as well as time points in the OGTT, were treated as repeated measures. If a significant F value was observed ($P < 0.05$), further analysis was done to determine where these changes occurred. An *a priori* significance ($P < 0.05$) was used as a criteria for further tests. *Post-hoc* tests were repeated

measure ANOVAs or Fishers PSLD ($P < 0.05$), depending on the nature of the data. These low stringency were used to protect against the chance of committing a type II error because of the small sample size and therefore limited power of the statistical design. Analyses were conducted using Statistic Package for Social Sciences (SPSS) software, version 6.0 for Macintosh.

RESULTS

Subjects participating in exercise training completed 90.0% (± 2.2) of the prescribed training sessions. There were no differences in compliance between treatment groups. Heart rates during cycling were consistently between 75 and 80% of HR_{max} . Heart rates during step aerobics were very high, ranging from 175 to 190 $\text{beats}\cdot\text{min}^{-1}$ (85–95% of HR_{max}). $\text{VO}_{2\text{max}}$ was not significantly altered in any treatment group.

After treatment, there was a significant increase in body weight in the CP group and a significant decrease in body weight in the E/CN group (Table 1). There were no significant changes in body fat percentage, fat mass, or fat-free mass (Table 1). Fat-free mass in the E/P group was significantly higher than in all other groups both pre- and post-treatment. It is noteworthy that the nonsignificant higher initial weight in the E/P group can be attributed almost entirely to greater fat-free mass.

There were no significant differences in pre- and post-basal plasma glucose or insulin levels. Glycosylated hemoglobin was also unchanged following treatment.

Pre- and post-treatment glucose tolerance and insulin response curves for each treatment group are illustrated in Figure 1. Comparisons of glucose tolerance curves or area under the glucose curves (Fig. 2) indicated no significant treatment effect for any group. Likewise, there were no significant improvements in the insulin response curves following the CP ($P = 0.433$), E/P ($P = 0.087$), or E/CP ($P = 0.110$) treatments (Fig. 3). However, after the E/CN treatment there was a significant decline in the insulin response at 60, 90, and 120 min after the oral glucose load which resulted in a significant decline in the overall insulin response curve ($P = 0.041$) (Fig. 1).

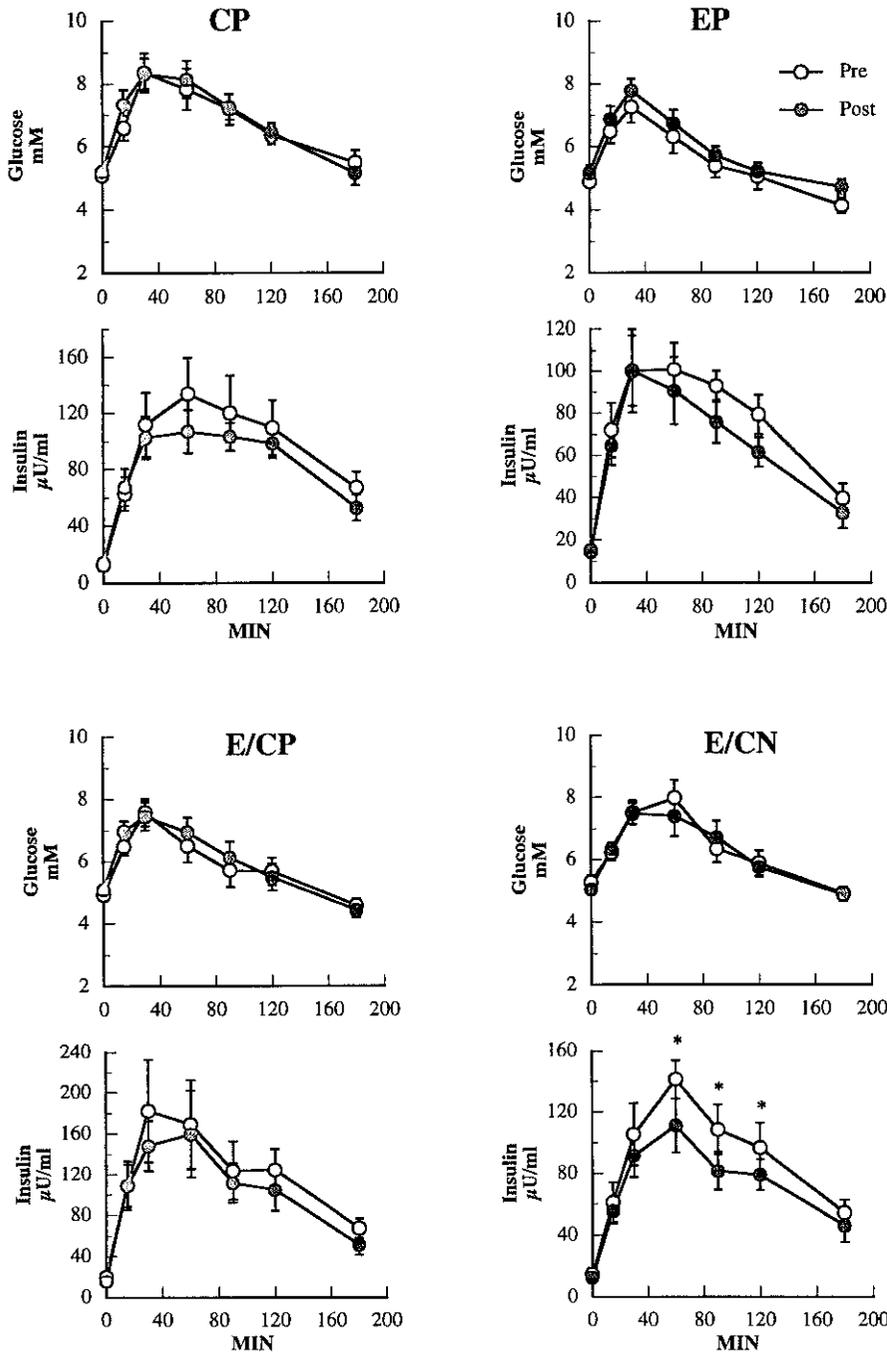


Fig. 1. Glucose tolerance and insulin response curves derived from a 3-h oral glucose tolerance test. Curves represent pre- (○) and post- (●) 9-wk treatment period in subjects receiving chromium supplementation (CP), exercise training with placebo (E/P), chromium picolinate (E/CP), or exercise training with chromium nicotinate (E/CN). * = significantly different from corresponding pre-value.

The area under the insulin response curve for the E/CN treatment was also found to be significantly reduced post training.

No significant differences were found for triglycerides, total cholesterol, LDL-C, and HDL-C between pre- and post-treatment samples.

DISCUSSION

Previous research has shown that chromium supplementation influences body weight and composition (19,25), basal plasma hormone, and substrate levels in-

cluding glucose, insulin, triglycerides, total cholesterol, LDL-C and HDL-C (1,2,9,27-29), and glucose and insulin response to an oral glucose load (13,22). These findings have most frequently been observed in specific population subsets such as non-insulin dependent diabetics and groups deficient in chromium intake. Exercise training has also been shown to alter these variables in a similar manner (10,15,32,33). As abnormalities in these parameters are risk factors for the development of CAD and NIDDM, interventions such as chromium supplementation and exercise training, if successful, could have an impact on public health by decreasing the incidence of

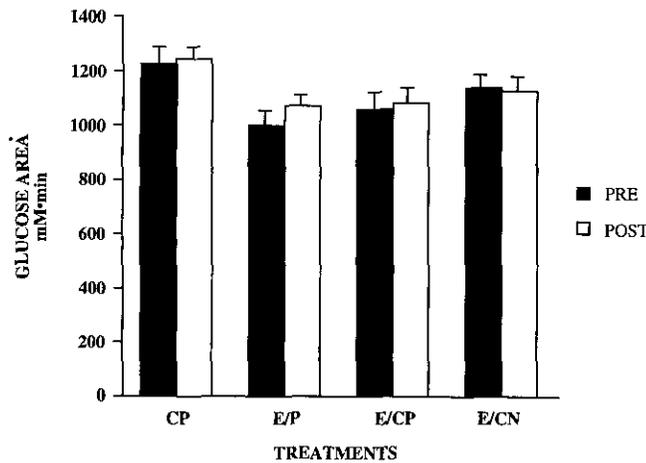


Figure 2—Area under the curve derived from glucose data from a 3-h oral glucose tolerance test. Values are area pre and post 9-wk treatment period in subjects receiving chromium supplementation (CP), exercise training with placebo (E/P), exercise training with chromium picolinate (E/CP), or exercise training with chromium nicotinate (E/CN).

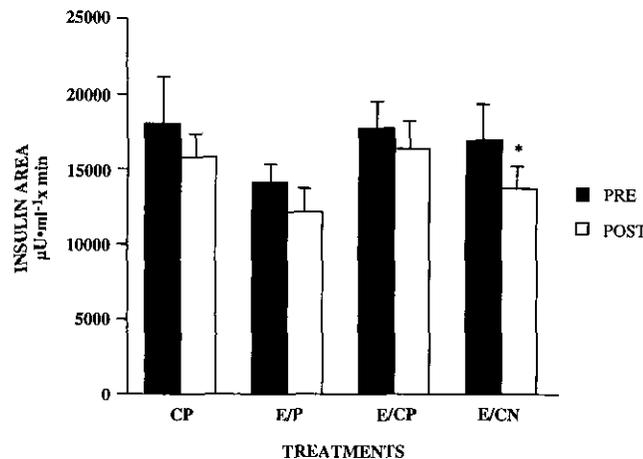


Figure 3—Area under the curve derived from insulin data from a 3-h oral glucose tolerance test. Values are area pre and post 9-wk treatment period in subjects receiving chromium supplementation (CP), exercise training with placebo (E/P), exercise training with chromium picolinate (E/CP), or exercise training with chromium nicotinate (E/CN). * = significantly different from pre-value.

these common diseases (7,26,35). To this end, our study compared the effects of chromium picolinate supplementation, exercise training, or both in young, obese women. Data were also gathered on the effects of chromium nicotinate supplementation combined with exercise training, as *in vitro* work suggests chromium nicotinate may be effective in altering these risk factors as well.

Body weight increased significantly in the CP group following treatment. This is an important finding, as chromium picolinate is often promoted as an aid to weight reduction. Our results indicate that without exercise, not only may chromium picolinate supplementation be ineffective in causing weight loss, but may result in weight gain. Weight gain has not been seen with previous studies of chromium supplementation (19,25,28), possi-

bly because of uncontrolled diet and activity patterns. Also, our subject population (young, obese females) had not been studied; this may account for the difference in findings. Similarly, the amount of chromium administered in the present study ($400 \mu\text{g}\cdot\text{d}^{-1}$) was twice the amount previously studied with females. It is possible that at this concentration chromium has a facilitating effect on weight gain, whereas at lower concentrations it may have an inhibitory effect.

No significant changes in body weight were seen in the E/P or E/CP groups, confirming previous research indicating weight loss is not often seen with 9 wk of exercise training (32). There was, however, a significant decrease in body weight in the E/CN group. To our knowledge, this is the first study to suggest that chromium nicotinate supplementation combined with exercise training may be an effective means of weight loss.

There were no significant changes in relative body fat, fat mass, or fat-free mass following treatment. Previous research has shown chromium picolinate supplementation decreasing fat mass and increasing fat-free mass (19,25). Previous studies of exercise training have shown increases in fat free mass as well (32). Although studies with young men (11) and women (16) suggest that combining exercise training with chromium picolinate supplementation increases the body composition changes that occur with exercise training, this finding has not been confirmed (6,14).

As with body weight, discrepancies between the results of this study and previous research may be a result of differences in study populations, uncontrolled diet and activity patterns, and/or the amount of chromium administered.

$\dot{V}O_{2\text{max}}$ levels did not increase significantly following the training period. This may have been a result of lack of specificity of testing: pre- and post-max tests were conducted on a treadmill and training consisted of cycling and aerobics. Additionally, the resistance component of the training program may have masked or diminished aerobic changes.

Basal glucose levels were not significantly altered in any treatment group. This result is consistent with other studies of chromium supplementation (1,36) and exercise training (34) in a normo-glycemic population. Studies of chromium supplementation in hyperglycemic populations have produced conflicting results, showing both decreases (2,27) and no change (21) in basal glucose levels after exercise training.

The response of plasma glucose levels to an oral glucose load was significantly higher in the CP group both pre- and post-treatment when compared with the E/P group. Group means imply an inverse relationship between fat-free mass and glucose area under the curve, suggesting that a greater fat-free mass allows for more rapid disposal of an absolute amount of glucose. Individ-

ual data, however, indicated a poor correlation between these two factors ($r = -0.26$).

No treatment effect was seen for glucose response to an oral glucose load. As with basal glucose levels, subjects initially exhibited normal glucose levels following an oral glucose load. Previous studies showing improvements in this parameter with chromium supplementation (2,8,13,22) or exercise training (34) observed elderly or hyperglycemic populations; even in these populations, chromium supplementation has not always been shown to be effective (33).

Glycosylated hemoglobin, an indication of plasma glucose levels over a 6-wk period, was unchanged after treatment. Previous work has shown that exercise training has a minimal effect on glycosylated hemoglobin levels (34). Chromium picolinate supplementation has been shown to decrease glycosylated hemoglobin in elderly and diabetic populations (12,27), although this has not always been observed (21).

Basal insulin levels, initially within a normal range, were not significantly altered in any group following treatment. This supports the findings of another study observing chromium supplementation in a young, healthy population (36). Past results with chromium supplementation in hyperglycemic subjects have been mixed, with both decreases (2) and no effect (31) on basal insulin levels being reported. Decreases in basal insulin levels with exercise training have been most readily observed in hyperglycemic and elderly subjects (34).

Insulin response to an oral glucose load was significantly reduced in the E/CN group after treatment. An improvement in insulin response has been shown with hyperglycemic subjects (8). No significant change in insulin response was observed in the other exercise-trained groups; this was unexpected as decreases have previously been documented with exercise training (17,23,31,34). However, this decrease has been found to be very short lived (17,23); thus our inability to detect a decreased insulin response to a glucose challenge in these subjects may have been a result of the rapid decay of

improved insulin action. Whatever the cause for the lack of an exercise training effect on insulin action in the E/P and E/CP groups, the results do suggest that the combination of exercise training and chromium nicotinate may be beneficial.

Basal plasma lipids did not change with treatment. Previous studies have shown varying results: many have shown exercise training to have no effect (10,15), and reports of the effects of chromium picolinate supplementation have been inconclusive, although chromium supplementation has been previously shown to have no effect on triglyceride (1,29,36), total cholesterol (1,29,36), HDL-C (21,26,36), and LDL-C (1,21,36) levels. Studies that have shown alterations in lipid levels with chromium supplementation employed hyperlipidemic subjects (1). It was interesting to note that the few subjects with abnormal initial lipid levels moved toward normalization following treatment, although these changes were not significant (data not shown).

In summary, high levels of chromium picolinate supplementation without concurrent exercise training caused significant weight gain in young, obese females, while exercise training combined with chromium nicotinate supplementation resulted in several potentially beneficial changes, including significant weight loss and a lower insulin response to an oral glucose load. We conclude that high ($400 \mu\text{g}\cdot\text{d}^{-1}$) supplemental amounts of chromium picolinate are contraindicated for weight loss in young, obese women, while exercise training combined with chromium nicotinate supplementation may be more beneficial than exercise training alone in providing some protection against CAD and NIDDM through risk factor modification. We suggest that further study of the efficacy of chromium nicotinate supplementation be done.

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