Effects of a Pre-Exercise Meal on Plasma Growth Hormone Response and Fat Oxidation during Walking

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ABSTRACT: The purpose of this study was to determine the effects of a pre-exercise meal on the plasma human growth hormone (hGH) response and fat oxidation during walking. Subjects (n=8) were randomly provided with either 1 g/kg body weight of glucose in 200 mL water (CHO) or 200 mL water alone (CON) 30 min prior to exercise and subsequently walked on a treadmill at 50% of VO_2max for 60 min. Plasma hGH concentrations were significantly higher in subjects who received CHO compared to those who received CON at 15 and 30 min. The fat oxidation rate in the CHO was significantly lower than the CON while walking for 5~15, 25~35 and 45~55 min. Plasma FFA levels were also significantly lower in the CHO compared to the CON at 30, 45 and 60 min. Plasma glucose levels in the CHO were significantly lower while plasma insulin levels were significantly higher than in the CON at 15 and 30 min. Therefore, the results of this study suggest that the elevation of plasma hGH levels due to the intake of a pre-exercise meal may not be strongly related to fat oxidation and plasma free fatty acid (FFA) levels during low-intensity exercise.

Keywords: fat oxidation, human growth hormone, free fatty acid, walking, cabohydrate

INTRODUCTION

Exercise is the most potent physiologic stimulus for GH release (1,2) and the magnitude of the GH response to exercise is influenced by various factors such as age, gender, body composition, substrate intake, physical fitness, and intensity, nature, and duration of the exercise (1). One of the physiological effects of hGH is the stimulation of lipolysis in adipocytes, which results in the release of non-esterified fatty acids (NEFAs) and glycerol (3-5). To our knowledge, no study has sought to determine the relationship between plasma hGH concentration and fat oxidation during exercise by determination of substrate utilization during exercise after intake of a pre-exercise meal. Therefore, the primary purpose of the present study was to address this relationship.

The ingestion of carbohydrates as a pre-exercise meal is a common strategy to delay fatigue that is frequently utilized by both professional and recreational athletes (6). Carbohydrate ingestion before and during exercise reduces fat oxidation during a subsequent exercise bout (7,8). Thus, fat oxidation in active people is often under the influence of insulin response to normal dietary carbohydrates. However, differences in metabolic and hormonal responses due to the ingestion of carbohydrates are still not completely understood.

The increase in blood hGH that occurs during prolonged exercise has been theorized to stimulate the hormone sensitive lipase of adipocytes and increase plasma levels of fatty acids. The strongest support for this hypothesis comes from a study by Wee et al. (9) which reported that one of the roles of the GH response to exercise may be to provide NEFAs and glycerol as an energy resource during recovery from exercise. These findings are supported by other studies showing that the administration of GH stimulates lipolysis in humans and rats (3,10-12). Although the well-established notion that hGH promotes lipolysis in adipose tissue and that an elevation of hGH is associated with increased plasma free fatty acid (FFA) levels (4,13), the physiological relationship between plasma hGH, plasma FFA, and fat oxidation has not been carefully investigated. One study reported that the administration of growth hormone increased lipolytic parameters substantially more than ex-

ercise alone, but did not further augment whole body fat oxidation (14). Although the secretion of hGH can be affected by various factors such as sleep, exercise stress, and energy availability (5,15), the role of elevated hGH levels during low-intensity exercise needs to be investigated with respect to lipolytic parameters and whole body fat oxidation. The purpose of this study was therefore to determine the relationship between elevated hGH levels due to the intake of a pre-exercise meal and fat oxidation during low-intensity exercise.

SUBJECTS AND METHODS

Subjects
Eight healthy male college students volunteered to serve as study subjects. The subjects were an average of 23.3 ± 0.9 years of age, 66.4 ± 1.2 kg in body weight, and 173.5 ± 0.9 cm in height. Their maximum O_2 consumption (VO_2max) averaged 56.6 ± 2.4 mL/kg/min. The subjects had no history of chronic disease, were following a normal dietary regimen, and were not taking any medications. This study was approved by the Kyungpook National University Institutional Review Board.

Experimental and control trials
Subjects were given a cardio-respiratory fitness test to determine their VO_2max prior to experimental trials. Subjects were asked to maintain their normal eating regimen throughout the duration of the study and to refrain from exercise or alcohol consumption 48 hr before testing. Subjects reported for the trials at 06:00 AM following a 12 hr overnight fast. An intravenous catheter was inserted into an antecubital vein and resting blood samples were collected from the catheter. The subjects consumed either a mixture of 1.0 g glucose per 1.0 kg body weight in 200 mL water as an experimental treatment (CHO) or 200 mL water as a control treatment (CON). Thirty minutes following treatment, the subjects walked for 60 min on a treadmill at 50% of VO_2max. All subjects randomly underwent one of the two trials (experimental trial or control trial). Blood samples (5 mL) were collected at 30 and 15 minutes before each trial (i.e., ～30 min and ～15 min), and at 15 min intervals during exercise.

Blood analyses
The 5 mL blood samples were transferred into chilled heparin-coated tubes and centrifuged (3,000 rpm) at $4^{o}C$ for 20 min. The plasma was then removed and stored at $-20^{o}C$ until use. Plasma glucose was measured using a glucose analyzer (YSI 1500 Stat Plus, Yellow Springs Instruments, Yellow Springs, OH, USA). Plasma insulin and hGH levels were assayed by radioimmunoassay us-

ing a kit (Linco Research Inc., St. Louis, MO, USA). The level of plasma FFAs was determined according to the method developed by Noma et al. (16).

Substrate oxidation rate
In order to calculate carbohydrate and fat oxidation rates, oxygen uptake (VO_2) and carbon dioxide output (VCO_2) were measured over three 10-minute periods (5～15 min, 25～35 min, and 45～55 min) while the subjects walked on the treadmill. The following formulas were used (17): fat oxidation rate (g/min)$=1.67\times VO_2$ (L/min) $-1.67\times VCO_2$ (L/min) $-0.307\times$protein oxidation rate; and carbohydrate oxidation rate (g/min)$=4.55\times VCO_2$ (L/min) $-3.21\times VO_2$ (L/min) $-0.459\times$protein oxidation rate. The protein oxidation rate was estimated as follows: protein oxidation rate (g/min)$=$[energy expenditure (kJ/min)$\times0.12$ (16.74 kJ)]. We assumed that protein oxidation encompassed approximately 12% of the resting energy expenditure of each study subject. Energy expenditure (kJ/min) during walking was calculated as the sum of each macronutrient oxidation rate (g/min) multiplied by a conversion factor (carbohydrate and protein$=16.74$; fat$=37.66$).

Statistical analysis
Values are expressed as the mean$\pm$SE. Two-way (treatment$\times$time) repeated measures analysis of variance (ANOVA) was used to test the mean differences between the treatments (CON vs. CHO) and interaction factors at various times. The sphericity assumption was justified by using the t-test. All statistical procedures were performed using SPSS 18.0 (SPSS In., Chicago, IL, USA) at a significance level at 0.05.

RESULTS

Plasma hGH concentration
Plasma hGH levels increased significantly in the CON treatment after 45 min of exercise compared to pre-exercise levels (1.8 ± 0.1 μg/L). hGH levels increased after 30 min of exercise in the CHO treatment compared with levels 30 min prior to exercise (1.9 ± 0.3 μg/L) and were significantly higher than those of the CON treatment at 15 min (3.6 ± 1.02 vs. 1.7 ± 0.09 μg/L, $P<0.05$) and 30 min (6.2 ± 1.4 vs. 2.7 ± 0.4 μg/L, $P<0.05$) (Fig. 1).

Fat oxidation rate
The fat oxidation rate of the CHO treatment was significantly lower compared to the CON treatment after 5～15 min (10.8 ± 2.1 vs. $20.2\pm3.2\%$, $P<0.05$), 25～35 min (17.3 ± 3.2 vs. $28.4\pm3.1\%$, $P<0.05$), and 45～55 min (20.6 ± 3.2 vs. 31.7 ± 3.0 %, $P<0.05$) of walking (Fig. 2).
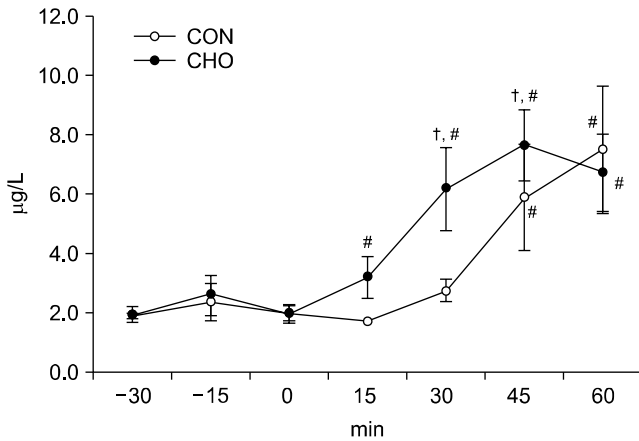
Fig. 1. Plasma hGH concentrations. Control treatment, CON; Experimental treatment, CHO. Values are means±SE, n=8. [†]P<0.05 vs CON; [#]P<0.05 vs −30 min.

Fig. 3. Plasma glucose concentrations. Control treatment, CON; Experimental treatment, CHO. Values are means±SE, n=8. [†]P<0.05 vs CON; [#]P<0.05 vs −30 min.

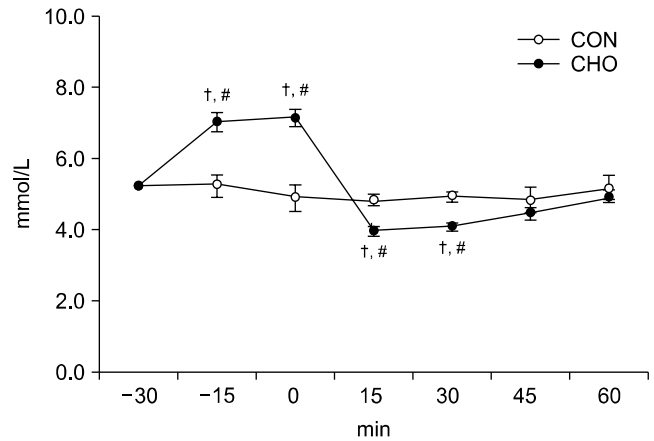Fig. 2. Fat oxidation rate. Control treatment, CON; Experimental treatment, CHO. Values are means±SE, n=8. [†]P<0.05 vs CON.

Fig. 4. Plasma insulin concentrations. Control treatment, CON; Experimental treatment, CHO. Values are means±SE, n=8. [†]P<0.05 vs CON; [#]P<0.05 vs −30 min.

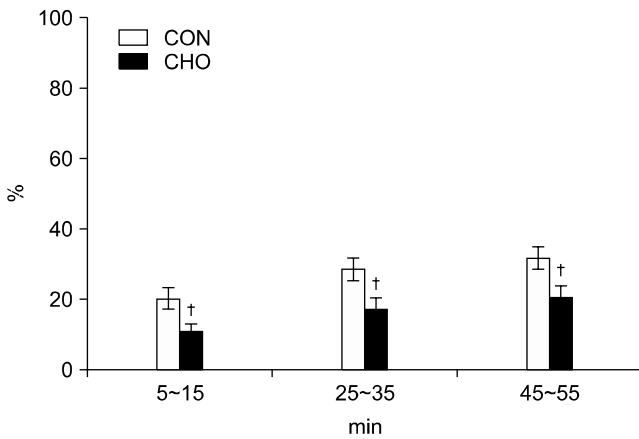## Plasma glucose concentration

The concentration of plasma glucose in the CHO treatment increased compared to the levels measured prior to CHO consumption (pre-level; 5.2±0.1 mmol/L) at 30 min before exercise due to substrate consumption. During exercise, the glucose levels of the CHO treatment dropped significantly to 3.9±0.1 mmol/L and 4.1±0.1 mmol/L after 15 and 30 min of exercise, respectively. Glucose levels increased to pre-treatment levels during the last 30 min of exercise. Glucose concentrations in the CON treatment were maintained throughout the entire trial. Glucose levels at 15 and 30 min were significantly lower in the CHO treatment than in the CON treatment at the same time points (CON: 4.8±0.2 and 4.9±0.1 mmol/L, respectively) (Fig. 3).

Plasma insulin concentration

Plasma levels of insulin were unchanged in the CON treatment but were significantly elevated as a result of the substrate load in the CHO treatment prior to exercise. Insulin levels in the CHO treatment were sig-
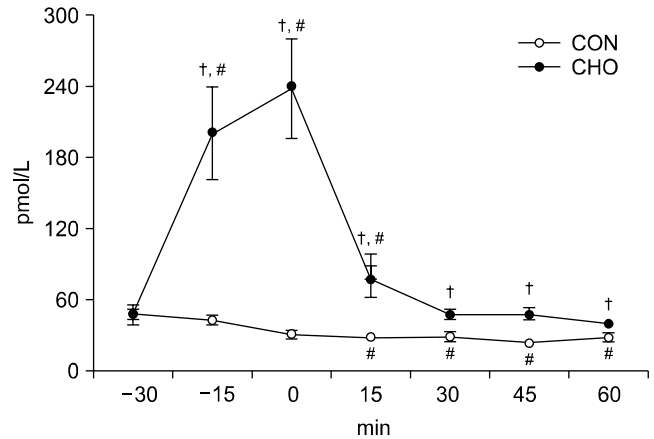
nificantly higher than those in the CON treatment at the onset of exercise, but decreased to the levels observed in the CON treatment after 30 min of exercise (Fig. 4).

Plasma FFA concentration

While FFA levels in the CON treatment increased slightly during exercise, the change was not statistically significant. FFA levels in the CHO treatment significantly decreased during exercise. FFA levels were significantly different between the CHO and CON at 30 min (0.13±0.02 vs. 0.26±0.03 mmol/L, P<0.05), 45 min (0.13±0.03 vs. 0.30±0.03 mmol/L, P<0.05), and 60 min (0.16±0.02 vs. 0.36±0.04 mmol/L, P<0.05) (Fig. 5).

DISCUSSION

To the best of our knowledge, the present study is the first to investigate the relationship between the elevation of plasma hGH levels and fat oxidation during
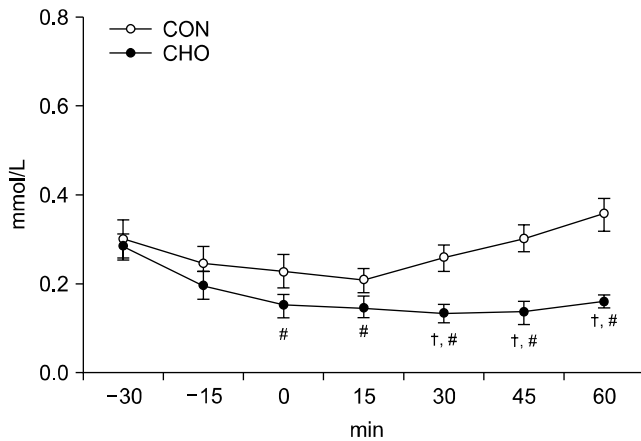
Fig. 5. Plasma FFA concentrations. Control treatment, CON; Experimental treatment, CHO. Values are means±SE, n=8. [†]P<0.05 vs CON; [#]P<0.05 vs −30 min.

mild-intensity exercise after the consumption of carbohydrates as a pre-exercise meal. The results of this study demonstrate that plasma hGH levels may not be strongly related to fat oxidation rates. Plasma FFA levels, as a marker of lipolytic rate, were not elevated even with a significant increase in plasma hGH levels following the intake of a pre-exercise meal. Thus, the results of the current study suggest that an increase in hGH concentration may not significantly affect fat oxidation and lipolytic activity.

The elevation of plasma hGH during exercise has been shown to be affected by the intensity and duration of exercise, work output during exercise, muscle mass used during exercise, and carbohydrate intake (5,15,18-23). The exercise stimulus used in this study was mild, at only 50% of the subjects' VO2max, but was long enough to promote a significant increase in plasma hGH levels (Fig. 1). Dore et al. (15) demonstrated that when subjects consume carbohydrates during aerobic exercise, hGH declines in comparison to those subjects who do not consume carbohydrates. However, in the present study, hGH levels were found to be significantly elevated in the CHO treatment during walking. According to the results of previous studies, this elevation in the CHO treatment was a result of a reduction in plasma glucose levels induced by the intake of carbohydrates as a pre-exercise meal (24,25).

Increases in plasma hGH typically occur in concert with an increase in fat oxidation during prolonged aerobic exercise. Previous studies postulate that an increase in plasma hGH during aerobic exercise may be partly responsible for stimulation of fat oxidation (4,26-31). The findings of previous studies provide evidence in support of this hypothesis, indicating that both administration of hGH and elevation of plasma hGH during exercise stimulate an increase in the rate of lipolysis in tissues (3,10,12). Furthermore, this increase in lipolysis ele-

vates plasma FFA levels, thereby increasing the rate of fat oxidation during prolonged exercise. However, according to the results of a study by Hansen et al. (14), human growth hormone administration combined with aerobic exercise significantly increased lipolytic parameters more than exercise alone, but did not increase whole body fat oxidation. The results of our study were similar to the results of Hansen et al. (14) in that an increase in plasma hGH levels occurred at a lower rate of fat oxidation and a lower concentration of plasma FFA in subjects in the CHO treatment compared to those who received the control treatment; however, this finding is contrary to those reported in previous studies (3,10,12,14). Thus, we suggest that the role of the plasma hGH response during aerobic exercise might not be strongly related to lipolytic stimulus or the fat oxidation rate.

If hGH could stimulate lipolysis during exercise, this effect might be easily suppressed by other factors such as plasma insulin (5,23). This conclusion is contradictory with the results of previous studies which suggested that elevation of plasma hGH might be related to lipolysis (3,5,9,18). Exercise-induced lipolysis is affected by various factors, including substrate status, the adrenergic system, and insulin status (5). A possible explanation for the lower lipolysis and fat oxidation rates seen in the CHO group in the present study is that an increase in plasma insulin concentration (Fig. 4) following the consumption of carbohydrates as a pre-exercise meal eliminated or attenuated the effect of hGH on lipolysis. The reduction in lipolysis and fat oxidation following carbohydrate ingestion necessitates a compensatory increase in carbohydrate oxidation in order to maintain energy production during exercise. As has been previously reported, carbohydrate ingestion and the resultant insulin response increases blood glucose uptake and, presumably, oxidation during exercise (7,32). The increase in plasma insulin seen in this investigation may have significantly suppressed or attenuated the effects of hGH on fat oxidation and lipolysis. Since the elevation of plasma insulin during low-intensity exercise can supply CHO to active muscles, we can infer that the effects of hGH on lipolysis and fat oxidation may have been eliminated or suppressed in the current study.

Generally, plasma cortisol levels were elevated during prolonged aerobic exercise (33). This elevation of cortisol levels could stimulate the rate of lipolysis and gluconeogenesis processes to supply active muscles with substrates (34,35). However, when the stress of aerobic exercise was mild as in the current study, the changes of plasma cortisol concentrations were not observed in the previous studies (35-37). Thus, because the exercise stress of our experimental protocol in this study was relatively mild in the comparison to the previous studies

(35-37), we did not determine the plasma cortisol levels. But, because we could not eliminate the possibility of plasma cortisol changes, we suggest that the changes of plasma cortisol should be determined in the next investigation.

While plasma glucose does not normally decrease in a 1 hour walking period, a significant decrease in plasma glucose can occur when carbohydrates are consumed as a pre-exercise meal (7,38,39). This phenomenon reflects an imbalance between the rate of carbohydrate oxidation and the rate of carbohydrate supplementation in muscle during exercise. In the present study, the reduction in plasma glucose levels during the first 30 min of exercise following carbohydrate intake was due to a significant increase in muscle glucose uptake without a compensatory increase in glucose supplementation into blood. We concluded that the effect of the CHO meal on plasma glucose was due to the additive effect of insulin and exercise (40,41). This effect has been explained in previous studies and reviews (42,43). Therefore, the mechanism responsible for the higher hGH response in the CHO group might be low blood glucose levels, as insulin-induced hypoglycemia can stimulate the release of hGH (44).

In summary, this study provides evidence that an increase in plasma hGH levels due to lower blood glucose levels resulting from the consumption of a pre-exercise meal does not elevate plasma FFA levels or the fat oxidation rate during low intensity aerobic exercise. Therefore, we conclude that elevated hGH concentrations may not be strongly related to the fat oxidation rate or plasma FFA levels during low-intensity exercise.

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AUTHOR DISCLOSURE STATEMENT

The authors declare no conflict of interest.

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