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Effects of Submaximal Exercise on Glucose Tolerance in Obese Subjects

Chikashi YAMAMOTO*, Sayoko HAYAMIZU**,
Tetsuo OHKUWA***, Yuzo SATO****

The effect of one period of submaximal exercise on glucose tolerance was examined in 5 obese subjects (obesity index: 138.3±14.4%). After submaximal exercise on the treadmill for 20 min., subjects had a glucose tolerance test (submaximal ex. GTT), and metabolic changes were compared with the case in O-GTT.

Submaximal exercise showed a tendency to improve glucose tolerance and further stimulate lipolysis. It is, therefore, suggested that repetition of submaximal exercise may improve in insulin sensitivity with reduction of bodyweight in obese subjects.

In practice, obesity remains one of the most difficult diseases to treat in spite of the fact that both the cause and treatment of it are theoretically known. A calorie-controlled diet and exercise designed to meet nutritional requirements, yet produce a daily calorific deficit, is the best form of therapy.[2. 3. 23)] Unfortunately, dietary treatment has met with little success and we still have an obese population.

Physical exercise has been shown to play a beneficial role in the treatment of diabetes and obesity since ancient times.[2. 3. 12. 13)] But an effective physical exercise prescription for obese subjects and diabetic patients has yet to be determined. Therefore, an appropriate prescription, which specifies the kind of exercise, duration, frequency and intensity, should be developed.

In previous studies[22. 30)] we reported that glucose tolerance was aggravated after exhaustive exercise in obese subjects, though normal subjects were shown to have improved glucose tolerance. The exhaustive exercise might be a stressor to obese subjects and cause the suppression of insulin effects, thus we pointed out that exhaustive exercise is not appropriate for obese persons as physical therapy. Previous studies[10. 28)] showed that the suitable exercise intensity for obesity was 60% of $\dot{V}O_2$max., or exercise of about 120 heart beats per minute. Consequently this study was undertaken to examine the effects of submaximal exercise on metabolic changes in a glucose tolerance test.

Subjects and methods

Five obese subjects, aged 19-28yrs, were studied at the Research Center of Health, Physical Fitness and Sports, Nagoya University. Characteristics of the subjects are shown in Table 1. None of the subjects participated in competitive athletics on regular basis. The nature, purpose and possible risks involved in the study were carefully explained to all subjects before their voluntary consent to participate was obtained. Subjects performed two kinds of tests;

1) Oral glucose tolerance test (O-GTT)

100g glucose was administered orally and

*Nagoya Gakuin University **Aichi Prefectural University
***Nagoya Institute of Technology ****Nagoya University

Table 1. Characteristics of the subjects

subject	Height (cm)	Weight (kg)	Obese Index (%)
Y. M	178	85	121.1
M. Y	173	101.5	154.4
S. H	175	90	133.3
F. T	173	100	152.2
K. S	180	94	130.5
Mean± S.D	175.8± 2.8	94.1±6.8	138.3± 14.4

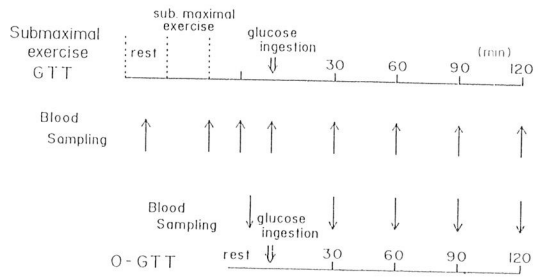

Figure 1. Experimental procedure

Table 2. Intensities of exercise

Oxygen Uptake during Exercise

$\dot{V}O_2$Max. (l/min)	$\dot{V}O_2$ (l/min) 4'-5'	9'-10'	14'-15'	19'-20'
3.56 ±0.55	2.32±040	2.22±0.36	2.21±0.37	2.32±0.29
	% $\dot{V}O_2$ Max.			
	66.3±10.9	63.2±7.9	63.2±13.5	66.7±11.9

Values are means ±SE.

blood samples were drawn before, as well as 30, 60, 90, and 120 min. after glucose ingestion.

2) Submaximal exercise glucose tolerance test (submax. ex. GTT)

Oral glucose tolerance test was performed 30 minutes after one 20 minute period of submaximal exercise. Blood samples were collected from the antecubital vein, before, immediately following exercise and at two intervals of 15 minutes followed by four intervals of 30 minutes there after, up to 120 minutes after glucose ingestion. 100g glucose was ingested orally over 2-3 minutes, begining 30 min. after the end of exercise. These studies were performed as above after overnight fasting of 12-14 hours. Each test was carried out at one week intervals. The experimental procedure of the study is depicted in Fig. 1. The subjects exercised for 20 min. on a treadmill to attain a work load culculated to result in a pulmonary oxygen uptake of approximately 60% of the previously determined maximal value. Lactate, β-hydroxybutyrate, acetacetate and glycerol were determined enzymatically in whole blood. Glucose was analyzed in whole blood and plasma FFA level was determined by the autoanalyzer method. Plasma insulin and growth hormone levels were analyzed by a radio-immunoassay.

Results

1) Intensity of exercise

Intensities of submaximal running are shown in Table 2. Intensity changed slightly from 63.2± 13.5% at 14−15 min. to 66.7±11.9% of $\dot{V}O_2$max. at 19−20 min. of exercise and were moderately higher than the expected levels.

2) Blood glucose (Fig. 2)

After the administration of glucose a rapid increase in blood glucose concentration was

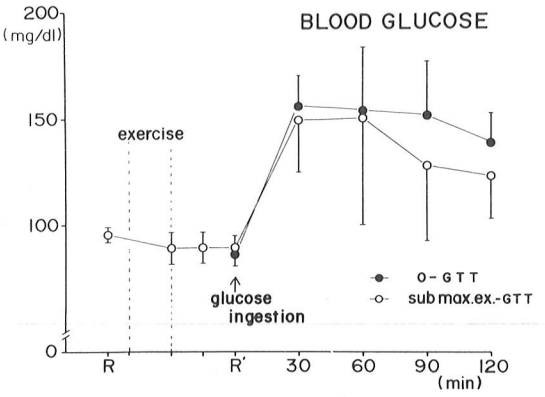

Figure 2. Changes in blood glucose concentrations during O-GTT and submaximal ex. GTT. Points are mean±SE.

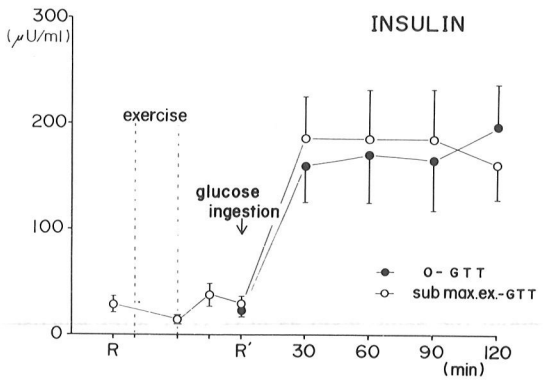

Figure 3. Changes in insulin concentrations during O-GTT and submaximal ex. GTT. Points are mean ± SE.

observed reaching a plateau value of 150−160 mg/dl at 60−120 min. following glucose ingestion. Blood gluose levels slightly decreased after submaximal exercise, but increased to 149.4 and 151.6 mg/dl at 30 and 60 min. after glucose ingestion, respectively. In the submaximal exercise GTT, lower levels were reflected at 90 and 120 min. after glucose ingestion than those of O-GTT, but weren't statistically significant. Significant differences weren't observed between O-GTT and submaximal exercise GTT.

3) Insulin (I R I, Fig 3)

Resting levels of insulin showed hyperinsulinamia from 30 to 120 min. after glucose ingestion. Insulin concentration after overnight fasting was 23.1±5.3 μU/ml, and increased to 159.6±36.5 μU/ml 30 min. after glucose ingestion, and maintained higher levels to 120 min. after ingestion. In submaximal exercise GTT insulin concentration, 29.5 μ U/ml at rest, decreased slightly to 14.5 μ U/ml at the end of exercise, and returned to the fasting level 30 min. after exercise. After glucose ingestion insulin concentration increased to 184.6 ± 43.7 μ U/ml, and a hyperinsulinamic condition persisted through out the test.

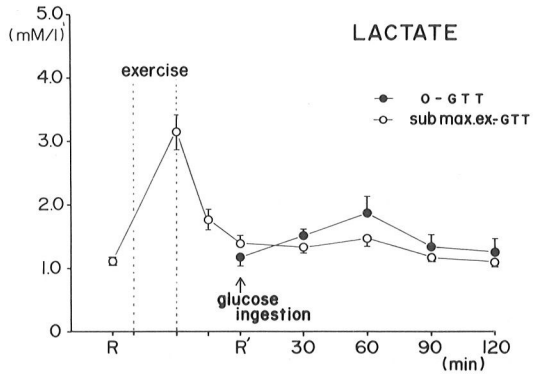

Figure 4. Changes in lactate concentrations during O-GTT and submaximal ex. GTT. Points are mean ± SE.

4) Lactate (Fig. 4)

In O-GTT blood lactate showed slight increase to 1.52±0.09, 1.88±0.30 mM/l, at 30 and 60 min. after glucose ingestion, respectively. While in submaximal ex. GTT, blood lactate concentration increased 1.12±0.05 to 3.15± 0.38 mM/l, immediately after exercise, and began decreasing, but showed a slight increase 60 min. after glucose ingestion.

5) Free fatty acid (F F A, Fig. 5)

Fasting plasma FFA concentrations were 0.68±0.01 mEq/*l* and 0.59±0.10 mEq/*l* in O-GTT and submaximal ex. GTT, respectively. FFA levels decreased gradually after glucose ingestion in O-GTT. After submaximal exercise FFA concentration increased to 0.95, 0.87 mEq/*l* 15 and 30 min. after exercise, then, decreased to about the same level as in O-GTT after glucose ingestion.

6) Glycerol (Fig. 6)

Fasting serum glycerol concentrations were 0.161±0.027, 0.138±0.014 mM/*l* in O-GTT and submaximal ex. GTT, respectively. Glycerol concentrations decreased after orally ingested glucose as FFA concentration, which means the suppression of lipolysis by glucose ingestion. Glycerol concentrations significantly increased to 0.254±0.051 mM/*l* after exercise.

7) Growth hormone (H G H, Fig. 7)

Growth hormone level didn't reflect any change after oral glucose administration in O-GTT other than a small decrease in the first 30 min. Submaximal exercise made growth hormone concentration significantly increase from 1.88± 0.35 to 6.82±2.34 ng/ml. However, growth hormone concentration decreased to approximately resting level 30 min. after glucose ingestion.

8) Ketone body (Fig. 8)

Fasting ketone levels were 0.123±0.014 mM/*l*, which changed slightly after glucose ingestion. While in the submaximal exercise GTT fasting ketone body concentration of 0.104 increased during exercise and showed its peak of 0.265 mM/l 30 min. after the end of exercise.

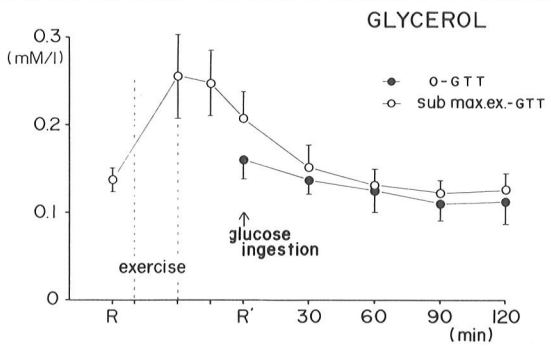

Figure 6. Changes in glycerol concentrations during O-GTT and submaximal ex. GTT. Points are mean ± SE.

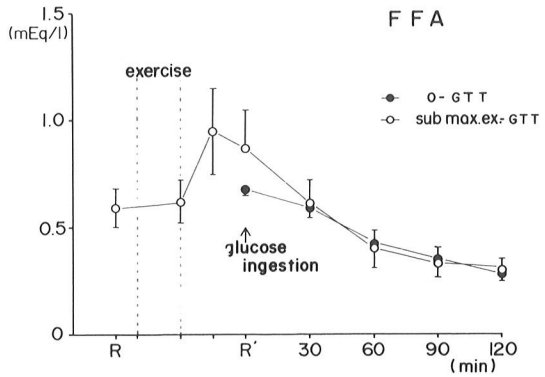

Figure 5. Changes in FFA concentrations during O-GTT and submaximal ex. GTT. Points are mean ± SE.

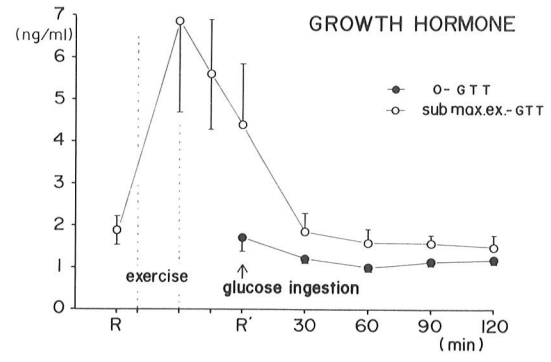

Figure 7. Changes in growth hormone concentrations during O-GTT and submaximal ex. GTT. Points are mean ± SE.

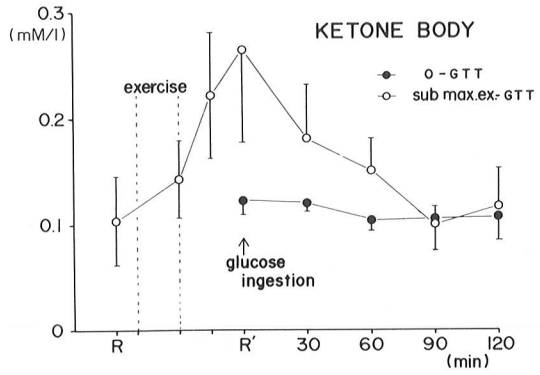

Figure 8. Changes in ketone body concentrations during O-GTT and submaximal ex. GTT. Points are mean ± SE.

After glucose ingestion ketone body levels began decreasing and returned to the fasting level.

Discussion

The beneficial effects of exercise are well documented,[2.3.10.19.21] but a definitive exercise prescription for obesity has never been made other than the general acceptance that submaximal exercise is a suitable way to reduce the body weight and to improve the metabolic disturbances in obese subjects. As peripheral insulin resistance is the main cause of the metabolic syndrome of obesity, an increase in insulin sensitivity would be most desirable in obese subjects. In fact, any treatment, such as physical trainig or calorie restriction that increases the peripheral insulin sensitivity would be the most appropriate from a pathophysiological point of view.[2.3.23]

Lipolysis is presumable from increases in plasma FFA and serum glycerol. Plasma FFA rises not only during exercise,[24.30.31] but also after exercise.[5.24.27] In the present study plasma FFA and serum glycerol markedly increased during and after exercise. The peak of FFA was higher than that after exhaustive exercise[29] despite that the peaks of glycerol were almost the same, which probably reflects the suppression of FFA mobilization as an energy source. FFA release was augmented by submaximal exercise,[24.28] but plasma FFA levels fell during and after exhaustive exercise or exercise approaching maximal intensity.[24.27.28] Issekutz[9] found the suppressive effect of lactate on FFA mobilization as an energy source, and Rodahl[27] reported that FFA increased markedly during recovery when blood lactate returned to resting levels. Therefore, exercise intensity should not cross the anaerobic threshold. Submaximal exercise performed in this study was appropriate for obese subjects because the lactate level was lower than the anaerobic threshold and lipolysis was stimulated effectively. Hyperketonemia, probably caused by increases in FFA, was detected after submaximal exercise. However, this phenomenum is not pathological but physiological in nature.

Growth hormone level showed a marked increase during exercise, as Hartley[6] reported that growth hormone concentration increased with work and reached greater values at moderate than during heavy loads.

The relations between glucose tolerance, tissue sensitivity to insulin and training are well discussed,[22.25] but there is little study[7.17] of the effects of exercise on glucose tolerance. Physical training improves glucose tolerance with increased tissue sensitivity to insulin in obese subjects, while the lack of exercise and hyperphargia lower it.[17.18.20] Then, Beck-Nielsen,[1] Koivisto[15] and others[16] reported that glucose tolerance and insulin binding to monocytes and adipocytes were diminished in obese patients, and that they were improved by carbohydrate restriction, reduction of body weight, or training.[19.25] In athletes insulin binding to mono-

cytes is higher than in normal subjects.[14] In recent reports, Heath[7] provided evidence that the last period of exercise produced a improved glucose tolerance on GTT. Ivy[11] showed a similar result. LeBlanc[17] also presented a result in that insulin response to glucose was decreased after an inactive period of 3 days on IV-GTT, but this improved 18h after 1 h of physical activity at 70% of $\dot{V}o_2$max. These results suggest that the effects of exercise on glucose tolerance and glucose uptake in tissue and liver persisted for many hours. Heath[7] postulated three reasons as follows: (1) an increase in tissue sensitivity to insulin[26] which may be mediated by increased binding of insulin to its receptors on the cell membrane,[14.25] (2) depression of mucle and liver glycogen, which makes "glucose storage space"[4] available, (3) an increase in muscle cell permeability to glucose, which is brought about by the insulinlike effect of muscle contractile activity and can persist for a long time after contractile activity stops.[4.8]

In the present study, changes in blood glucose concentrations after submaximal exercise were lower at 90 and 120 th. minutes than those on O-GTT, but these differences weren't statistically significant. Insulin concentrations in response to glucose were approximately the same levels in O-GTT and submaximal exercise GTT. In our previous study concerning the effect of exhaustive exercise on GTT, glucose tolerance was improved after exercise in normal subjects, while it was aggravated in obese patients. Why the effect of exercise wasn't detected in this study is presumed to be that one period of submaximal exercise at 60% of $\dot{V}o_2$max. for 20 minutes was not enough work and intensity to bring about the depression of muscle and liver glycogen. Provided that this submaximal exercise is repeated 3 or 4 times, glucose tolerance would be improved, but care must be taken to avoid allowing the intensity to cross the anaerobic threshold.

In conclusion, submaximal exercise at about 60% of $\dot{V}o_2$ max. stimulated lipolysis and repetition of this exercise seems to improve the metabolism and glucose tolerance in obese patients with the effective reduction of body weight. Therefore, this exercise is appropriate for obese patients as physical exercise treatment.

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