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EFFECTS OF THREE-DAY BED REST ON METABOLIC, HORMONAL AND CIRCULATORY RESPONSES TO AN ORAL GLUCOSE LOAD IN ENDURANCE OR STRENGTH TRAINED ATHLETES AND UNTRAINED SUBJECTS

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The study was designed to find out (1) whether the effect of 3-day bed rest on blood glucose (BG) and plasma insulin (IRI) responses to glucose ingestion depends on preceding physical activity and (2) whether plasma adrenaline (A), noradrenaline (NA) and cardiovascular changes following a glucose load are modified by bed rest. Eleven sedentary students (22.5 ± 0.3 yrs), 8 long distance runners (18.6 ± 0.3 yrs) and 10 strength trained athletes (21.2 ± 2.1 yrs) were examined before and after bed rest. Plasma IRI, BG, NA, A, heart rate (HR), and blood pressure (BP) were measured during 2 hrs following glucose (75 g) ingestion. The responses of BG and IRI to glucose load were calculated as incremental areas under the curves (auc). Both in athletes and untrained subjects bed rest markedly increased IRIauc, while BGauc was elevated only in sedentary subjects ($p < 0.05$). The greatest increases in IRIauc and IRI/BG ratios were found in the endurance athletes. The data from all subjects ($n = 29$) revealed that the initial plasma NA and glucose-induced increases in NA and A were lowered after bed rest ($p < 0.01$). These effects were most pronounced in the endurance athletes. Bed rest did not influence HR or BP in any group. It is concluded that (1) the athletes have more adequate compensation for the bed-rest-induced decrement in insulin sensitivity than sedentary men; (2) three-day bed rest diminishes basal sympathetic activity and attenuates sympathoadrenal response to oral glucose; (3) endurance athletes have greater sympathetic inhibition than strength athletes or sedentary men.

K e y - w o r d s: *oral glucose tolerance test, bed rest, ·insulin, catecholamines, physical training*

INTRODUCTION

Both endurance and strength training increase whole body glucose disposal and insulin sensitivity, resulting in improved glucose tolerance and reduced insulin response to a glucose load (for rev. see 1). These adaptive changes are attributed to enhancement of the muscle glucose transporter (GLUT-4) content

and glycogen synthase activity, to increased number of capillaries in skeletal muscles, increased muscle mass, and to decreased plasma triacylglycerol concentration. Contrary to these training effects, during restriction of physical activity (bed rest deconditioning) increased plasma insulin response to glucose administration with or without deterioration of glucose tolerance was found (2—7). Lipman et al. (3) reported significant glucose intolerance occurring within the first three days of bed rest in men and concluded that the disturbances in carbohydrate metabolism were not caused by hypogravia since monkeys immobilized in the vertical body position, also exhibited abnormal blood glucose curves following glucose ingestion. Dolkas and Greenleaf (2) investigated effects of isotonic and isometric leg exercise during 14-day bed rest in healthy subjects and showed a significant inverse relationship ($r = -0.99$) between the plasma insulin response to a glucose load and mean total daily energy expenditure. More recent data have indicated that a bed-rest-induced decrease in whole body insulin action is caused by reduced sensitivity of inactive muscles to this hormone (8,9). Moreover, Vukovich et al. (10) showed that reduced insulin action, caused by a 6-day inactivity in endurance athletes, was associated with diminished content of GLUT-4 in the gastrocnemius muscle. Some of these previous studies concerning the effects of inactivity on carbohydrate tolerance and insulin action were performed with athletes and some with sedentary subjects. We are aware of only one study (6) comparing the effect of bed rest on plasma insulin response during an oral glucose tolerance test (OGTT) in sedentary subjects and highly trained athletes. It was shown that elevation of plasma insulin, induced by 7 days of bed rest, was greater in untrained than in trained subjects, suggesting that exercise training preceding bed rest attenuates loss of insulin sensitivity. However, this study was performed with only four sedentary and four trained subjects. Therefore, it seemed worthwhile to further explore the relationship between the level or type of habitual physical activity and the effect of bed rest on glucose regulation. The present study was designed to compare an influence of three-day bed rest on blood glucose and plasma insulin responses to an oral glucose load in endurance- and strength-trained athletes and in sedentary subjects.

Activation of the sympathetic nervous system after carbohydrate ingestion is considered as a mechanism contributing to the dissipation of excess energy (11) and prevention of postprandial fall in blood pressure (for rev. see 12). Recent study by Ritz et al. (13) did not show significant changes in dietary induced thermogenesis after 8 and 42 days of bed rest. However, the responses of plasma catecholamines to a meal or glucose ingestion after bed rest have not been examined. Thus, the second purpose of the present investigation was to determine whether a short-term bed rest modifies plasma catecholamines, heart rate and blood pressure following glucose ingestion, and whether this effect depends on the preceding activity of the subjects.

<center>MATERIALS AND METHODS</center>

Subjects

Eleven sedentary students, eight long distance runners and 10 strength-trained athletes (body builders and wrestlers) participated in this study after giving informed consent. The protocol was approved by the local Ethical Committee. All subjects were clinically healthy and had negative family history of diabetes. The athletes trained regularly 7 days per week for at least 3 years. Characteristics of the subjects is given in *Table 1*.

Table 1. Anthropometric characteristics of subjects (mean $\pm$ SD)

Group	Age (years)	Body mass (kg)	Height (cm)	Body mass index $(kg \cdot m^{-2})$
Sedentary subjects (n = 11)	22.5 ± 1.1	75.8 ± 6.9	179.6 ± 4.6	23.4 ± 1.4
Endurance athletes (n = 8)	18.6 ± 0.7	72.0 ± 5.8	178.8 ± 6.7	22.6 ± 2.0
Strength athletes (n = 10)	21.2 ± 2.1	73.5 ± 11.3	174.6 ± 6.4	24.0 ± 2.7

Experimental protocol

This study was conducted in the students' hospital where the subjects reported in the evening 2—3 days after the last training session (day 0). Next morning, following an overnight fast, an intravenous cannula was inserted into the antecubital vein 30 min prior to base-line cardiovascular measurements, and the first blood sample was taken. Following this, the supine subjects were submitted to the 120 min oral glucose tolerance test (OGTT). For this purpose they drank 75 g of glucose dissolved in 150 ml of luke-warm water.

Venous blood samples were analyzed for plasma insulin (IRI) and blood glucose (BG) immediately prior to, and at 30, 60, 90, and 120 min following glucose ingestion; and for plasma catecholamines before glucose ingestion, and at the 60 and 120 min. Heart rate (HR) and blood pressure (BP) measurements were made before and every 30 min after the glucose load. The second similar OGTT test was performed following three days of horizontal bed rest. During bed rest the subjects were allowed to get up to shower once daily, and go to the toilet, read books, listen to the radio and watch TV in the supine position. The subjects' diet consisted of three freshly made meals per day with a total energy intake 12,000 kJ (50 % carbohydrates, 35% fat, 15% protein)

Analytical methods

Blood glucose (BG) was determined enzymatically using commercial kits (Boehringer Diagnostica Mannheim, Germany). Plasma insulin (IRI) level was measured by radioimmunoassay using reagent kits from the Institute of Atomic Energy (Świerk, Poland). Plasma adrenaline (A) and noradrenaline (NA) levels were determined by the radioenzymatic method of Da Prada and Zurcher (14) using the Catechola sets produced by the Institute for Research, Production and Application of Radioisotopes (Prague, Czechs). The plasma samples for catecholamine determination were stored at $-70°C$ until assayed. The analytical error for this method was 10.8% for [A] and 8.7% for [NA].

Calculations.

The integrated responses of BG and plasma IRI to the glucose load were expressed as incremental areas under their respective time curves (auc), and the ratios of IRIauc to BGauc were calculated.

Statistics

Nonparametric Wilcoxon and Whitney-Mann tests were used for comparisons between pre- and post-bed-rest data and between groups, respectively. The null hypothesis was rejected when $P < 0.05$. The data are presented as means ($\pm$ SE) unless otherwise stated.

RESULTS

Blood glucose and plasma insulin responses to the glucose load

Prior to bed rest there were no significant differences between athletes and sedentary subjects in the responses of BG, and IRI to oral glucose load (*Figs. 1—2*). However, in the sedentary subjects the fasting insulin level was significantly higher than in the endurance athletes and strength trained athletes ($p < 0.01$) and IRIauc tended to be greater ($p = 0.071$ and $p = 0.052$, respectively).
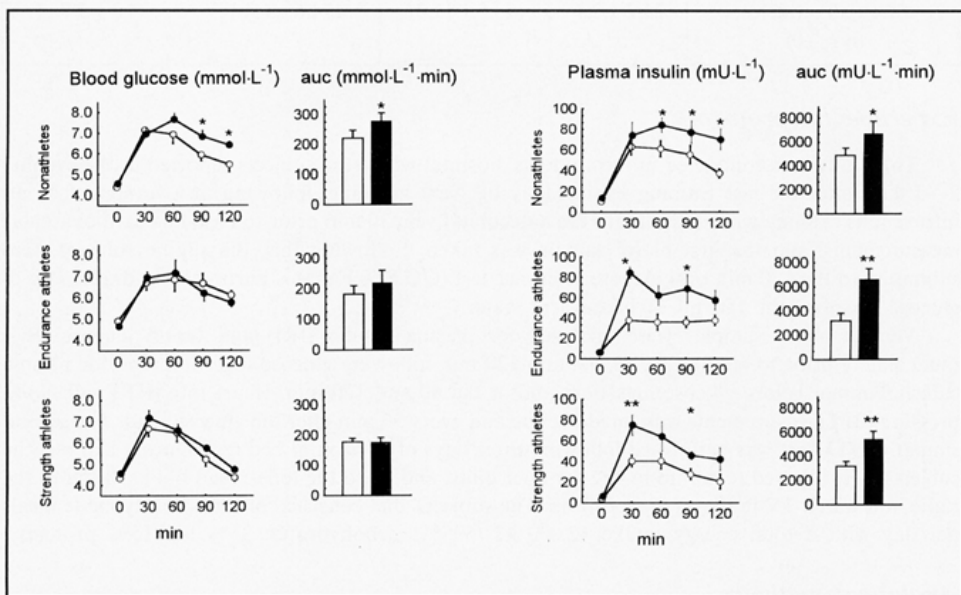

Fig. 1. Mean ($\pm$ SE) blood glucose concentrations and areas under blood glucose curves during the oral glucose tolerance test before (open circles and bars) and after (filled circles and bars) bed rest in athletes and sedentary (nonathletes) subjects. Asterisks denote significant differences between pre- and post-bed-rest data;*p < 0.05.

Fig. 2. Mean ($\pm$ SE) plasma insulin concentrations and areas under plasma insulin curves during the oral glucose tolerance test before (open circles and bars) and after (filled circles and bars) bed rest in athletes and sedentary (nonathletes) subjects. Asterisks denote significant differences between pre- and post-bed-rest data;*p < 0.05, ** p < 0.01.

Following bed rest, the BG levels at 90 and 120 min of the OGTT and BGauc were significantly higher (by approx. 30%) than before bed rest in the sedentary subjects, whereas the BG response in the athletes was not significantly affected by bed rest (*Fig. 1*). In both athletes and sedentary subjects plasma IRI levels during the OGTT were enhanced after bed rest (*Fig. 2*). However, in the sedentary subjects significant IRI differences between pre- and post-bed-rest values were found at 60, 90 and 120 min, whereas in both endurance- and strength-trained athletes the greatest elevation occurred at 30 min of OGTT (*Fig. 2*). As a result, the ratio of plasma IRI to BG was significantly greater after than before bed rest only at 120 min in the sedentary subjects, while the differences in this ratio occurred in the earlier phases of the OGTT in the athletes (*Fig.3*). The areas under the insulin curves were increased significantly after bed rest in all three groups with the most pronounced enhancement in the endurance-trained athletes (by approx. 100%) and the least effect (by approx. 40%) in the sedentary students (*Fig. 2*). During bed rest, the ratio of IRIauc to BGauc was increased significantly ($p < 0.01$) only in the endurance athletes (*Fig. 3*).
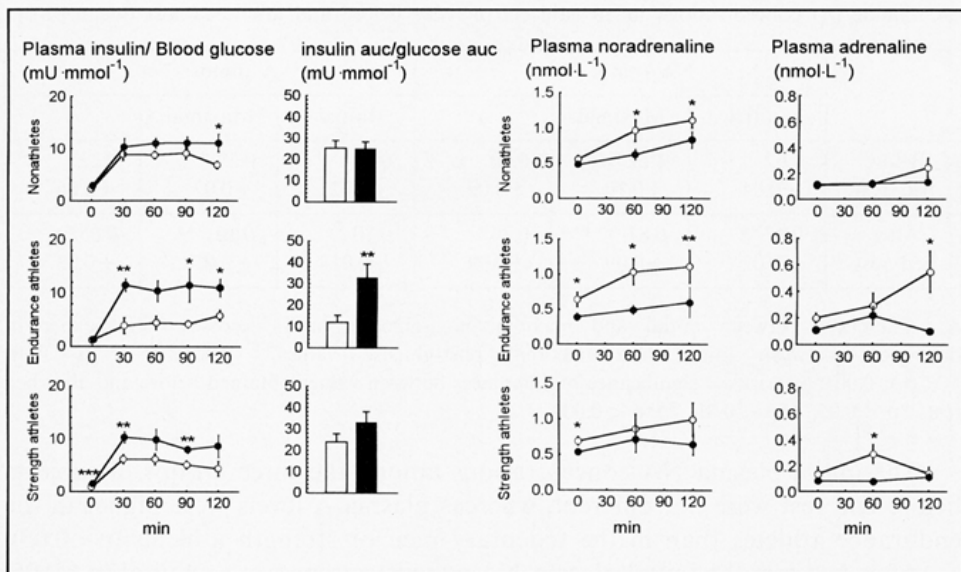

Fig. 3. Mean ($\pm$ SE) plasma insulin to blood glucose ratios and the ratios between areas under the curves of plasma insulin and bood glucose during the oral glucose tolerance test before (open circles and bars) and after (filled circles and bars) bed rest in athletes and sedentary (nonathletes) subjects. Asterisks denote significant differences between pre- and post-bed-rest data;*$p < 0.05$, ** $p < 0.01$.

Fig. 4. Mean ($\pm$ SE) plasma catecholamine concentrations during oral glucose tolerance test before (open circles) and after (filled circles) bed rest in athletes and untrained subjects. Asterisks denote significant differences between pre- and post-bed-rest data; *$p < 0.05$, **$p < 0.01$.

Comparison of BGauc and IRIauc values obtained after bed rest between the three groups did not reveal significant differences except for BGauc in the strength-trained athletes which was smaller than in the sedenty subjects ($p < 0.01$).

Plasma catecholamine, heart rate and blood pressure responses to the glucose load

Data from all subjects ($n = 29$) revealed that the initial values of plasma NA were significantly lowered after bed rest, whereas the decrement in plasma A did not reach statistical significance ($p = 0.083$). Glucose ingestion caused significant increases in both catecholamines with maximal values occurring at either 60 or 120 min of OGTT. Both the maximal post-glucose values and the glucose-induced increases in plasma catecholamines were reduced significantly by bed rest (*Table 2*).

Table 2. Comparison of the initial and maximal post-glucose plasma noradrenaline (NA) and adrenaline (A) concentrations in all subjects ($n = 29$) before and after bed rest (mean $\pm$ SE).

	NA ($nmol \cdot l^{-1}$)			A ($nmol \cdot l^{-1}$)		
	Initial	Maximal	Δ	Initial	Maximal	Δ
Before bed rest	0.62 ± 0.04	1.22 [+++] ± 0.10	0.60 ± 0.09	0.14 ± 0.02	0.37 [+++] ± 0.07	0.23 ± 0.06
After bed rest	0.47 [**] ± 0.02	0.81 [++***] ± 0.09	0.34 [*] ± 0.09	0.10 ± 0.01	0.16 [+**] ± 0.02	0.05 [**] ± 0.02

Δ = differences between initial and maximal post-glucose values; crosses = significance of differences between initial and maximal post-glucose values, [+]$p < 0.05$, [++]$p < 0.01$, [+++]$p < 0.001$; asterisks = significance of differences between values obtained before and after bed rest, [*]$p < 0.05$, [**]$p < 0.01$, [***]$p < 0.001$.

The initial plasma NA concentrations among the three groups of subjects before bed rest were not different, whereas plasma A levels were higher in the endurance athletes than in the sedentary men or strength athletes ($p < 0.05$). After bed rest the initial plasma NA concentrations were reduced ($p < 0.05$) in both groups of athletes but not in the sedentary subjects, while the initial plasma A was not affected significantly in any group (*Fig. 4*). The maximal post-glucose plasma NA levels were reduced in the endurance-trained subjects ($p < 0.01$) and sedentary subjects ($p < 0.05$), and almost reached significance ($p = 0.055$) in the strength-trained athletes. The glucose-induced increments (Δ) in plasma NA were significantly diminished ($p < 0.01$) only in the endurance athletes. The maximal post-glucose plasma A concentration was significantly lowered ($p < 0.01$) after bed rest only in the endurance athletes, and the

glucose-induced increments in the level of this hormone were not significantly reduced in any group.

Bed rest did not modify HR or BP during the OGTT in any group (*Table 3*).

Table 3. Heart rate (HR) and mean blood pressure (MBP) during the OGTT before and after bed rest.

	SEDENTARY SUBJECTS					ENDURANCE ATHLETES					STRENGTH ATHLETES				
Time (min)	0	30	60	90	120	0	30	60	90	120	0	30	60	90	120
HR (min^{-1})															
before	60±2	66±4	64±3	64±4	64±4	63±4	63±3	63±2	63±3	65±3	54±3	61±3	61±3	59±3	62±3
after	63±4	63±2	65±3	65±3	67±3	61±4	63±3	63±4	60±3	61±3	56±2	60±4	57±2	63±4	65±4
MBP (mmHg)															
before	88±2	86±2	85±2	89±2	88±2	84±3	85±4	85±2	88±4	86±4	80±3	77±3	78±2	77±2	79±2
after	83±2	84±2	84±2	86±2	85±2	83±3	85±4	85±4	89±3	88±4	84±2	82±2	84±2	86±3	85±2

DISCUSSION

The increase in the plasma insulin response to glucose ingestion after a short-term bed rest confirmed previous findings (2—7). The new findings of the present study are that this hyperinsulinemic response was more pronounced in the athletes, (especially endurance-trained) than in the sedentary subjects, whilst the significantly enhanced glucose response occurred only in the latter group. Although in none of the subjects blood glucose levels during OGTT met the criteria of impaired glucose tolerance (15), it appears that athletes benefit from their regular physical activity preceding bed rest deconditioning. This finding is in agreement with the suggestion of Wegmann *et al.* (6) that previous physical conditioning can attenuate some adverse metabolic consequences of bed rest. However, the above authors reported that their physically trained subjects exhibited a markedly smaller change in insulin secretion during OGTT than nonathletes and concluded that physical training ameliorates the insulin resistance induced by bed rest. This conclusion was not confirmed by our data because both endurance- and strength-trained athletes showed even greater enhancement in the plasma insulin response to the glucose challenge following bed rest than sedentary men.

It should be emphasized that the pattern of plasma insulin changes during OGTT after three days of bed rest in athletes differed from that in sedentary subjects. In the sedentary subjects, within the first 30 min of the OGTT, the plasma insulin response was similar to that before bed rest and the differences

started to be significant beyond 60 min; whereas in both groups of athletes the differences between the pre- and post-bed-rest insulin levels were significant already at 30 min of the OGTT, and the values were similar at 120 min.

It seems unlikely that the differences in blood glucose and insulin responses to the glucose load between athletes and sedentary subjects after bed rest were related to the residual effect of the last exercise training session in the former. Apart from the three days of controlled rest in the supine position, the athletes abstained from vigorous exercise for at least 2 days before bed rest. Because the insulin to glucose ratios in the early phase of the OGTT were higher in the athletes, it is possible that the pancreatic β-cells in trained subjects are more sensitive to the glycemic stimulus, or they have greater promptly releasable insulin reserves than the sedentary men. However, it is also possible that the inactivity-induced decrease in insulin action in skeletal muscles in trained subjects is greater than in the sedentary men. The mechanism underlying differences in the plasma insulin response to a glucose load between these groups cannot be explained on the basis of the present experiments. Nevertheless, by taking into account differences in the degree of glucose tolerance deterioration after bed rest between the sedentary and physically active men, it can be concluded that the latter exhibit more efficient control of their blood glucose level.

The present data concerning the effect of bed rest on basal plasma catecholamines obtained in a relatively large group of subjects ($n = 29$) confirmed an inhibition of the sympathetic nervous system (SNS) activity without changes in adrenomedullary secretion as reported previously by Goldstein et al. (16) Maass et al. (17) Sigaudo et al. (18) Sigaudo et al. (19). In all of these investigations a reduction in urinary NA excretion after bed rest was a consistent finding, while plasma NA concentration only tended to decrease most probably due to the small number of subjects, and substantial interindividual variability. Moreover, our data suggest that the effect of bed rest on SNS activity depends on the level of preceding physical training, because the significant decline in the basal plasma NA concentration after bed rest occurred in the athletes but not in the sedentary men.

In accordance with previous reports demonstrating a rise in plasma catecholamines after carbohydrate intake (20—24), the present data also showed an elevation in plasma noradrenaline and adrenaline concentrations following glucose ingestion. These increases can be partly attributed to enhanced sympathetic input to the skeletal muscles, as demonstrated by muscle the sympathetic microneurography (25, 26). It has been hypothesized that the increase of sympathetic activity, which follows glucose loading, is caused by baroreflex activation because insulin and possibly gut hormones released after nutrient ingestion result in muscle and splanchnic vasodilation (12), There are also data indicating that hyperinsulinemia per se stimulates sympathetic activity (27, 28).

A new finding of our study is that bed rest deconditioning attenuates the increase in plasma catecholamines after a glucose load. The mechanism of this effect cannot be explained based on the present data; however, it may be speculated that reduction in glucose-stimulated catecholamine release is caused by decreased baroreflex sensitivity after bed rest. Deterioration of baroreflex function or resetting of baroreceptors have been considered as possible causes of orthostatic intolerance after prolonged bed rest (for rev. see 29). Another possibility is that the attenuated plasma catecholamine response to the glucose load after bed rest is a consequence of decreased insulin sensitivity. In the present study, the effect of bed rest on the glucose-induced increase in plasma catecholamine concentration appeared to depend on the level and kind of the previous physical training. The most pronounced effect was found in the endurance-trained athletes and the least effect in the strength-trained subjects.

In accordance with data from other studies performed in young men (see for rev. 12), glucose ingestion in the present investigation did not influence blood pressure and caused only slight increases in heart rate. In spite of the diminished plasma catecholamine response to the glucose load, the short-term bed rest did not affect either HR or blood pressure during the OGTT in the supine position.

Summary: the present data demonstrated that only three days of bed rest increase the plasma insulin response to an oral glucose load; more so in athletes (especially endurance-trained) than in sedentary men. Only in the latter bed rest enhanced the blood glucose response. These data suggest that the benefit of physical training preceding bed rest can be attributed to more rapid and adequate compensation for the decrement in peripheral insulin sensitivity, by enhancement of the hormone secretion. It was also shown that a short-term bed rest decreases the basal plasma noradrenaline levels and attenuates activation of the sympathoadrenal system induced by glucose ingestion.

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