Effect of a Two-month Detraining on Glucose Tolerance and Insulin Sensitivity in Athletes-link to Adrenal Steroid Hormones

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Abstract

Reduction in physical activity has been demonstrated to associate with the increased risk in insulin resistance and type 2 diabetes. To determine whether alteration in insulinemia, due to abstention from regular exercise training, is associated with changes in serum dehydroepiandrosterone sulfate (DHEA-S) and cortisol, 18 highly trained badminton players (21.2 ± 0.3 years) were enrolled into a 2-month detraining study. Fasting serum insulin, glucose, DHEA-S, and cortisol were determined at trained state and at day 60 of detraining. Glucose tolerance and insulin sensitivity were assessed by an oral glucose tolerance test (OGTT). The 2-month detraining increased fasting glucose and insulin concentrations and body weight slightly, but did not significantly affect glucose tolerance and insulin response curve, in which 10 subjects had increased and 8 subjects had slightly decreased in the area under curve for insulin (IAUC). In the subjects with increased IAUC, serum cortisol was also elevated (from 0.44 ± 0.07 to 0.83 ± 0.26 U/l, $P < 0.05$) in parallel, and serum creatine kinase (CK) was unaltered during detraining. Whereas in the subjects with decreased IAUC, serum cortisol (from 0.51 ± 0.19 to 0.54 ± 0.14 U/l, no significance) was not changed and serum creatine kinase (from 461 ± 179 to 151 ± 21 U/l) was decreased during detraining. Two groups of detrained subjects exhibited a similar reduction in serum DHEA-S levels and slight elevation in body weight. The novel finding of the study is that the changes in serum cortisol, but not DHEA-S, were associated with the change in insulin sensitivity during early phase of lifestyle change from physically active to sedentary, and this response appears to be varied individually among athletes.

Key Words: physical inactivity, obesity, insulin resistance, hyperinsulinemia, cortisol, DHEA-S

Introduction

Environmental change in modern society that decreasing levels of physical activities to maintain daily living has been thought as a major cause in the development of type 2 diabetes (15). The early sign of this disease is the reduction in insulin sensitivity, characterized by a compensatory higher insulin response under an oral glucose challenge (20), which typically requires a long period of time to progress into clinical stage (9).

Two adrenal endocrine changes have been proposed to involve with the development of insulin resistance: [1] reduction in the formation of DHEA-S (7, 23, 24); [2] abnormal cortisol metabolism (21, 22). This hypothesis is partly supported by the evidence in a longitudinal study that reduction in DHEA-S with aging occurred in paralleled with the increase in insulin concentration (23) and patients with high circulating cortisol displayed a decrease in insulin sensitivity (21). Furthermore, increasing serum DHEA-S with oral DHEA supplementation was found to improve glucose tolerance and insulin sensitivity in middle-aged women (28), while elevating morning

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cortisol levels caused an opposing effect (19). Very few studies have investigated the role of these adrenal steroid hormones in the early developmental stage toward insulin resistance due to lifestyle shift from physically active to sedentary.

Serum DHEA-S was previously found to be modulated by an individual's physical activity status, in which a single bout of exercise or exercise training raises basal serum DHEA levels (1, 3). This response is also varied by the exercise training regimen (25). Serum cortisol levels can be increased by an acute bout of exercise (10), which demonstrates that cortisol metabolism can also be modulated by physical activity. Yet, studies investigating the effect of physical activity levels on basal cortisol levels have reported inconsistent results. It was shown that abstention from regular training leads to a decreased (12) or unchanged (16) plasma cortisol levels. The purpose of the study was to investigate the role of DHEA-S and cortisol on insulin sensitivity during lifestyle change from physically active to sedentary in highly trained athletes. This information would improve the understanding on the interrelationships between carbohydrate metabolism and the adrenal endocrine changes during the early developmental stages of insulin resistance due to reduced physical activity.

Materials and Methods

Human Subjects

Eighteen elite college badminton players (competed in national levels, age 21.2 ± 0.3 years) voluntarily undertook a 2-month detraining. All subjects were trained 3 h daily and 6 days a week before detraining. During the 2-month detraining period, all subjects were restricted to any form of exercise training or vigorous physical activity. Their dietary intake during the detraining period was not significantly different from trained state, according to a one-week dietary recall record. BMI (body mass index) was used as an indicator of being overweight (calculated as kilogram per square meter). Aims and methods were explained to all subjects, who then gave formal consent. Ethical approval for the study was obtained from the Human Subject Committee of Taipei Physical Education College. Blood glucose and insulin curves were plotted and total areas under the curve (AUCs) were calculated geometrically. The trapezoidal rule was used for calculation of areas under the curve (AUC) of glucose and insulin responses. All AUC are incremental (*i.e*. change from baseline values). After detraining, the subjects with the insulin AUC increased above the *Pre* value are designated as "IAUC-increased" group (N = 10); whereas the counterpart subjects are designated as "IAUC-decreased" group (N = 8).

Oral Glucose Tolerance Test (OGTT)

An oral glucose tolerance test (OGTT) was performed one-week before and at day 60 of detraining. The test procedure was according to the method previously described by Chou *et al*. (6). A 75-gram of glucose (Roquette Italia S.p.A., Cassano Spinola, Alessandria, Italy) was orally delivered with 500 ml of pure water. Blood samples were collected from the fingertips at 0 (fasting value), 30, 60, and 90 min. A Lifescan glucose analyzer (Los Angeles, CA, USA) was utilized for glucose concentration determination.

Insulin, Cortisol, and DHEA-S

Plasma sample was collected from 200 µl of fingertip blood. The insulin, cortisol, and DHEA-S were determined on an ELISA analyzer (Tecan Genios, Salzburg, Australia) with the use of commercially available ELISA kits (Diagnostic Systems Laboratories, Inc. Webster, TX, USA), by a manner in accordance of the manufacture's instruction.

Creatine Kinase and Cholesterol

Total plasma cholesterol was measured on a Beckman spectrophotometer analyzer with Sigma Trinder's reaction (Sigma, St. Louis, MO, USA), according to the manufacturer's procedure. CK was directly determined on a Reflotron Plus Analyzer (Roche Diagnostic, Basel, Switzerland) according to its standard procedure provided by the manufacture.

Statistical Analysis

A paired *t*-test was used to compare the mean differences between the Pre (trained state) and the Post (detrained state) values for all subjects. For comparison of variables between the subjects with increased and decreased IAUCs, independent *t* test was used. A level of $P < 0.05$ was set as significant on all tests, and all values are expressed as means ± standard error.

Results

Table 1 shows the physical characteristics and metabolic measures of the subjects in the trained (Pre) and detrained (Post) states. Two-month detraining significantly increased the body weight and BMI ($P < 0.05$), while GAUC (area under curve for glucose) and IAUC (area under curve for insulin) were not significantly changed due to great individual variations with detraining. The blood level of muscle CK was significantly reduced with detraining ($P < 0.05$), indicating that the muscle damage was significantly

Table 1. Physical characteristics and metabolic measures of the subjects during training (Pre) and at day 60 of detraining (Post). BMI: body mass index; GAUC: glucose area under curve; IAUC: insulin area under curve; CK: creatine kinase; DHEA-S: dehydroepiandrosterone sulfate; C/D: cortisol-to-DHEA-S ratio. *Significant difference from Pre, $P < 0.05$.

	Pre	Post
Weight (kg)	65.0 ± 1.6	68.1 ± 1.9*
BMI	21.9 ± 0.5	22.9 ± 0.6*
GAUC	2888 ± 243	3118 ± 276
IAUC	3338 ± 639	3802 ± 661
CK (U/l)	359 ± 105	174 ± 22*
Cholesterol (mg/dl)	149.9 ± 6.6	143.0 ± 6.7
Cortisol (U/l)	0.48 ± 0.10	0.67 ± 0.14
DHEA-S (ng/ml)	16.9 ± 1.5	3.3 ± 1.1*
C/D	0.029 ± 0.005	2.036 ± 0.736*

reduced by detraining. The total cholesterol level was not different between trained and detrained states. The level of cortisol was increased ($P < 0.05$) and DHEA-S ($P < 0.001$) was reduced with detraining.

Figure 1. shows glucose (A) and insulin (B) levels under fasted and glucose-challenged conditions. Both fasted glucose and insulin levels were significantly elevated with detraining ($P < 0.05$), while glucose tolerance and insulin response were not altered, in which 10 subjects exhibited increases but 8 subjects exhibited slightly decreases in the area under curve (AUC) for insulin.

To determine the association between changes in insulinemia and adrenal endocrines with detraining, all subjects were further divided into two groups: subjects with increased IAUC and decreased IAUC. The data of detraining effect on physical characteristics and metabolic measures in both groups is shown in Table 2. For the subjects with increased IAUC, the blood level of CK was not changed; whereas for the subjects with decreased IAUC, the blood CK level was significantly reduced ($P < 0.01$). For both groups, body weight and BMI were increased with detraining, but total cholesterol levels were not altered in both groups. Serum cortisol levels were significantly elevated by detraining in the subjects with increased IAUC ($P < 0.05$), but remained unchanged in the subjects with decreased IAUC. Under trained state, DHEA-S levels in the subjects with increased IAUC were greater than those of the subjects with decreased IAUC during detraining ($P < 0.05$). With detraining, both groups demonstrated significant reductions in DHEA-S ($P < 0.001$). Under trained state, the IAUC, GAUC,

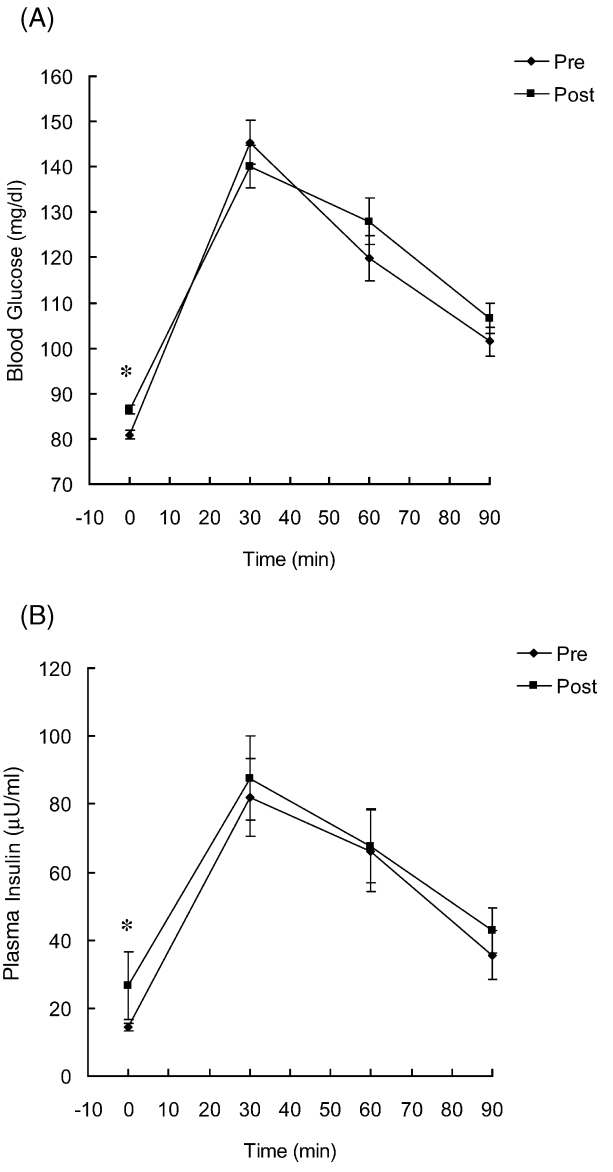

Fig. 1. Blood glucose (A) and plasma insulin (B) concentrations under oral glucose tolerance test during training (Pre) and after 2-month detraining (Post). *Significant difference from Pre, $P < 0.05$.

cholesterol, creatine kinase levels in the subjects with decreased IAUC was greater than those with increased IAUC during detraining.

Discussion

Reduction in physical activity due to lifestyle transition in modern society has been believed to associate with development of insulin resistance (15). Epidemiological and interventional studies of exercise training strongly support the efficacy of increasing physical activity for diabetes prevention and treatment (13, 15). In the current study, despites fasted glucose

Table 2. Physical characteristics and metabolic measures during training (Pre) and at day 60 of detraining (Post) for the IAUC-increased and IAUC-decreased groups. *Significant difference from Pre; #Signficant difference from the subjects with IAUC-increased group, $P < 0.05$.

	IAUC-increased		IAUC-decreased	
	Pre	Post	Pre	Post
Age (years)	20.8 ± 0.56	–	21.6 ± 0.34	–
Height (cm)	168.7 ± 0.9	–	74.8 ± 1.7#	–
Weight (kg)	63.8 ± 3.4	66.8 ± 4.3*	65.9 ± 1.4	69.0 ± 0.8*
BMI	22.4 ± 1.0	23.3 ± 1.4*	21.5 ± 0.4	22.5 ± 0.3*
GAUC	2226 ± 289	2950 ± 183*	3418 ± 281#	3252 ± 483
IAUC	1444 ± 264	3773 ± 1127*	4852 ± 879#	3826 ± 835
CK (U/l)	232 ± 68	204 ± 43	461 ± 79#	151 ± 20*
Cholesterol (mg/dl)	134 ± 1.0	134 ± 7.1	162.6 ± 10.4#	150.6 ± 10.4
Cortisol (U/l)	0.44 ± 0.07	0.83 ± 0.26*	0.51 ± 0.19	0.54 ± 0.14
DHEA-S (ng/ml)	19.6 ± 2.6	2.0 ± 1.0*	14.7 ± 1.7#	4.4 ± 1.8*
C/D	0.03 ± 0.01	1.83 ± 0.73*	0.03 ± 0.01	2.20 ± 1.23*

and insulin levels were slightly elevated, the whole-body glucose tolerance and insulin response were unchanged with 2-month detraining in the elite badminton players. Due to a great individual difference, 10 subjects showed an increase in IAUC, but not for the rest of 8 subjects. Subjects were thus divided into 2 groups, subjects with increased IAUC and subjects with decreased IAUC, to determine the changes of DHEA-S and cortisol from trained to detrained states. Intriguingly, we found that the first group showed a paralleled elevation in the morning cortisol concentration with detraining, but not for the second group. This result suggests that the early change in insulin sensitivity due to reduced physical activity was involved with the alteration in cortisol metabolism, which is reasonable as the cortisol is generally known to antagonize insulin action (21).

Although DHEA-S decline has been reported to associated with the development of insulin resistance, the current study does provide strong support for the possibility that decline in DHEA-S contributes to the detraining-associated reduction in insulin sensitivity. This is based on the result that the subjects with decreased IAUC had also shown a substantial decline in DHEA-S.

The underlying mechanism accounting for the increase in basal cortisol levels with detraining is unknown. Cortisol concentration usually rises followed a CK increase with trauma or stress condition (14, 27). Serum levels of the myocellular proteins creatine kinase, a muscle damage marker and a biochemical marker of training stress, is usually elevated during high-level of athletic training. However, the subjects with paralleled increases in IAUC and cortisol showed no change in blood CK, whereas the counterpart group displayed a decline in blood CK in this study. Apparently, the possibility that increased cortisol levels were associated with muscle tissue damage can be precluded.

The group variation in cortisol alteration during reduced physical activity may explain the inconsistent results observed in previous detraining studies. For instance, Hortobagyi *et al*. (12) reported a decrease in plasma cortisol with 2 weeks of detraining in power athletes, while Kraemer *et al*. (16) showed no change in basal cortisol levels with 6 weeks of detraining in recreationally strength-trained men. Alen *et al*. (2) investigated in 21 male subjects and found that cortisol levels decreased with strength training, suggesting that cortisol levels would be greater in sedentary state. Difference in cortisol levels has recently been found to relate with glucocorticoid receptor gene polymorphism, which implicates the individual variation in insulin sensitivity and susceptibility to type 2 diabetes (22, 26). The result of the current study provides a new knowledge that the detraining-associated reduction in insulin sensitivity is linked to the change of cortisol metabolism with individual variation. Whether difference in cortisol response corresponding to the change in physical activity status is also related to the glucocorticoid gene polymorphism requires further investigation.

Increasing BMI, secondary to reduced physical activity, is generally known as an important factor underlying insulin resistance (13, 15). However, body weight and BMI for the subjects with and without IAUC increased were both elevated. This result indicates that the effect of slight body weight increase may not completely reflect on their insulin sensitivity during the early stage of reduced physical activity. Furthermore, the 2-month detraining period on the magnitude of body weight increase appears to be related to the previous exercise training load. The

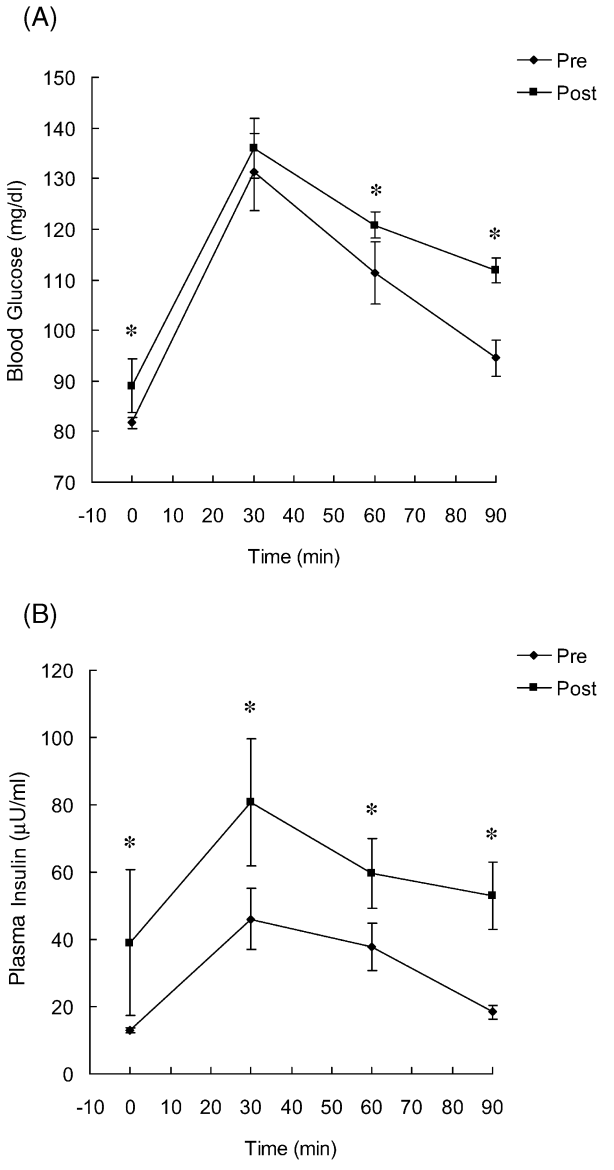

Fig. 2. Blood glucose (A) and plasma insulin (B) concentrations under oral glucose tolerance test during training (Pre) and after 2-month detraining (Post) for the subjects with increased IAUC. *Significant difference from Pre, $P < 0.05$.

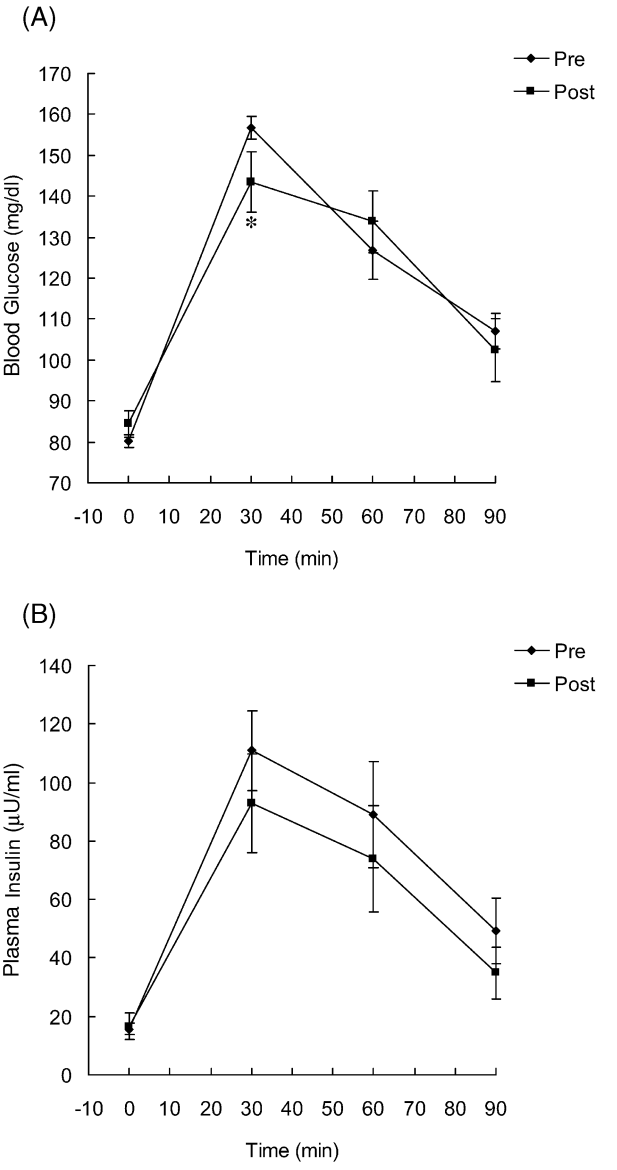

Fig. 3. Blood glucose (A) and plasma insulin (B) concentrations under oral glucose tolerance test during training (Pre) and after 2-month detraining (Post) for the subjects with decreased IAUC. *Significant difference from Pre, $P < 0.05$.

current result is different from previous study by Chen *et al*. (5) that young dancers with a weekly 9-h dance practice did not cause a significant increase in BMI with 2 months of abstention from dance activity. The subjects in the current study were elite badminton players competing in national level with regular training of 3 h a day, 6 days a week, which can not be considered as the same degree of detraining gradient as in the dancer study.

Another interesting finding of the study is that the subjects with increased IAUC during detraining had greater initial DHEA-S and lower IAUC in trained state than their counterpart group. It seems that the subjects with lower insulin sensitivity and DHEA-S at beginning did not change much in insulin sensitivity with detraining. DHEA-S may be essential for the expression of exercise training adaptation in a reversible fashion. We speculate that only the individuals with initially high DHEA-S exhibited improvement in insulin sensitivity but not for the low DHEA-S individual, and the training effect reversed as the exercise training stopped. It was previously reported that serum DHEA-S levels generally decreases under major stresses (8, 11) and during the recovery (17, 18), implicating its

role in stress-related adaptation. Boudou *et al*. (4) has recently found that exercise training-induced blood triglyceride-lowering effect is negatively correlated with DHEA-S levels in diabetes patients. Since triglyceride levels are generally increased during development of insulin resistance, Boudou's result suggests that high levels of DHEA-S is essential for exercise training adaptation. For those subjects with initially high DHEA-S in trained state may have better capability for training-induced adaptation, and thus reverse quickly as regular training is removed, but this reversible phenomenon would not occur in the subjects with initially low DHEA-S which exhibits higher IAUC.

In conclusion, the current study found that detraining response in insulinemia appears to be varied individually in elite badminton players, in which the subjects showing an increase in IAUC also displayed an increase in serum cortisol levels. This result suggests that the early developmental stage of insulin resistance due to reduced physical activity is linked to the change in cortisol metabolism. In addition, the small increase in body weight during the 2 months of detraining was not sufficient to produce a noticeable effect on glucose tolerance and insulin sensitivity in these athletes.

Acknowledgments

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