Influence of a Diet Regimen on Glucose Homeostasis and Serum Lipid Levels in Male Elite Athletes

Ragnar Tegelman, Torwald Åberg, Roland Eklöf, Åke Pousette, Kjell Carlström, and Lars Berglund

Physical training affects carbohydrate metabolism and results in an increased insulin-stimulated glucose disposal. To investigate if carbohydrate and lipid metabolism would be affected by nutritional factors in optimally trained elite athletes, during a 1-year period we studied elite ice-hockey players on two Swedish top-performance teams. Players on one team were subjected to extensive dietary monitoring and intervention, whereas players on the second team continued their ordinary diet. Blood levels of insulin, C-peptide, glucose, hemoglobin A_{1C} (HbA_{1C}), lipids, and lipoproteins were measured repeatedly. Basal insulin levels and insulin resistance (IR) were significantly lower among athletes on both teams compared with a sedentary group, and muscle weight and body mass index were significantly higher. During the course of the study in the intervention group, insulin levels decreased (3.6 ± 0.3 *v* 6.2 ± 0.6 [mean ± SEM], $P < .05$) in conjunction with a decreased relative fat energy content, but returned toward baseline levels when relative fat energy content increased. IR decreased in parallel (0.59 ± 0.05 *v* 1.12 ± 0.12, $P < .05$) and followed a similar pattern, reverting toward baseline levels. Also, levels of HbA_{1C} changed during dietary manipulation. No changes in these parameters were observed among the elite players from the team not participating in the diet regimen. In contrast to the parameters for glucose homeostasis, no significant changes were found in serum lipid or lipoprotein levels in either team during the course of the study. The results verify the presence of an improved carbohydrate metabolism in elite athletes. The observed changes in glycemic control and glucose homeostasis as a consequence of dietary modification demonstrate further that nutritional factors may affect carbohydrate metabolism also in well-trained athletes.

IT IS WELL ESTABLISHED that physical training results in changes in carbohydrate metabolism with an increased insulin-stimulated glucose disposal that correlates with the improvement in physical fitness.[1,2] This might be due to several factors, among them a change in body composition with a reduction of adipose tissue and an increased muscular mass, which would enhance whole-body glucose disposal.[3,4] However, as shown in recent studies, several other mechanisms might also contribute, such as changes in blood flow and muscle glycogen metabolism.[5-9] Thus, healthy athletes were shown to have an increased muscular blood flow and glucose uptake, as well as an increased cellular metabolism of glucose.[9] In addition to changes in insulin sensitivity, physical stress also results in profound changes in the balance of other endocrine hormones and, interestingly, these changes are modulated by nutritional factors.[10-12] Recently, a connection between low fat intake and changes in endocrine parameters was found in a long-term study in athletes.[12] Since insulin resistance (IR) is linked to obesity and hypertriglyceridemia,[13-15] this finding prompted us to investigate whether nutritional factors may affect long-term lipid and glucose homeostasis also in well-trained athletes, where mechanisms for increased glucose disposal already are present. In the present long-term study in elite ice-hockey players from two top-performance Swedish ice-hockey teams, we report the effect of dietary variation on glucose homeostasis and on serum lipid and lipoprotein levels during a 1-year period. Players from one of the teams participated in a dietary program, while the other team, serving as a control, continued their ordinary diet.

SUBJECTS AND METHODS

Subjects

Twenty-two healthy elite ice-hockey players from the team of Djurgårdens Idrottsförening (DIF) in Stockholm and 21 elite ice-hockey players from the team of Södertälje Sportklubb (SSK) in Södertälje (35 km south of Stockholm) participated in the study. Both teams have been champions of the Swedish National Hockey League (NHL) and were competing among the top teams. All participants were conditioned ice-hockey players, professionally trained, and accustomed to hard competition for several years. Informed consent was obtained from each individual after a careful explanation of the purpose and nature of the study, which was approved by the Ethics Committee at Huddinge University Hospital.

Basal data and physical characteristics for the two groups are shown in Table 1. For comparison, data for age-matched sedentary males are also presented in the table. A physical examination and full standard laboratory biochemical test battery of blood and urine evaluating hepatic, biliary, renal, intestinal, and endocrinological functions were performed initially in all subjects and were normal in all cases. None of the players were given any medications during the course of the study. Although no routine banned-drug tests (analysis of gonadotropins, testosterone, sex hormone–binding globulin, and 17-hydroxyprogesterone) were performed as part of the study, previous tests of the participants have repeatedly proven negative. Further, some of the players who participated in international ice-hockey tournaments during the study were subjected to

From the Departments of Clinical Chemistry and Obstetrics and Gynecology, Karolinska Institute, Huddinge University Hospital, Huddinge; Nutricare Limited, Huddinge; and Kostcentrum, Sabbatsberg Hospital, Stockholm, Sweden.

Submitted February 7, 1995; accepted September 5, 1995.

Supported by grants from The Health Food Council, National Centre for Sports Research, The Swedish Sports Research Council, The Swedish Society of Medicine, The Swedish Nutrition Foundation, and The Swedish Medical Research Council (6804, 7137, 8722, and 10349). L.B. is a Florence Irving Associate Professor and an Established Scientist of The American Heart Association, New York City Affiliate.

Current address: R.T., Medilab, Täby, Sweden.

Address reprint requests to Lars Berglund, MD, Division of Preventive Medicine and Nutrition, Department of Medicine, College of Physicians and Surgeons of Columbia University, 630 W 168th St, New York, NY 10032.

0026-0495/96/4504-0005$03.00/0

Table 1. Basal Clinical and Anthropometric Data (mean ± SEM) of the Two Ice-Hockey Teams Compared With Sedentary Controls

Variable	DIF Team (n = 22)	SSK Team (n = 21)	Sedentary Controls (n = 34)
Age (yr)	24.3 ± 0.8	25.0 ± 0.9	25.3 ± 0.6
Height (cm)	180 ± 1	182 ± 1	183 ± 1
Weight (kg)	81.4 ± 1.3*	82.4 ± 1.7*	73.7 ± 1.5
Broca's index	101 ± 1	101 ± 2	89 ± 2
Body mass index	24.9 ± 0.2*	24.8 ± 0.4*	22.0 ± 0.4
Muscle weight (kg)	37.7 ± 0.6*	38.5 ± 1.0*	31.0 ± 0.9
Fat weight (kg)	11.8 ± 0.6	11.5 ± 0.7	11.5 ± 0.7
Body fat (%)	14.4 ± 0.6	13.9 ± 0.7	15.5 ± 0.7
HbA_{1c} (%)	4.0 ± 0.1	4.2 ± 0.1	4.1 ± 0.1
Glucose (mmol/L)	4.0 ± 0.1	3.7 ± 0.1	4.2 ± 0.1
C-peptide (nmol/L)	0.46 ± 0.02	0.44 ± 0.04	0.52 ± 0.03
Insulin (mU/L)	6.2 ± 0.6*	5.5 ± 0.8*	8.1 ± 0.7
IR	1.12 ± 0.12*	0.93 ± 0.14*	1.52 ± 0.16
Cholesterol (mmol/L)	4.1 ± 0.2	4.2 ± 0.2	4.4 ± 0.1
HDL cholesterol (mmol/L)	1.3 ± 0.1	1.1 ± 0.1	1.3 ± 0.1
LDL cholesterol (mmol/L)	2.5 ± 0.1	2.8 ± 0.2	2.7 ± 0.1
Triglycerides (mmol/L)	0.9 ± 0.1	0.8 ± 0.1	1.0 ± 0.1

NOTE. Broca's index = weight/height [(kg/cm-100) × 100].
*$P < .05$ *v* sedentary control group.

drug testing in connection with these events, and were always found to be negative.

Study Design

The present study covered a period of 12 months from May to May, with a break in the training program for vacation in April. The study period was divided into a preseason dry-land training period and a competition period (total, 5 months) of the Swedish NHL. During the study, DIF players participated in a diet program detailed below. SSK players maintained their ordinary diet. Five blood sampling periods were performed during the study. The first test period was the preseason in May (build-up period), which included both regular group training and individual summer exercise without any ice-hockey competition games. The second blood sample (ice-training period) was obtained in August during ice-training and preseason competition just before the start of the Swedish NHL. After another 3-month period, the third sample (competition period) was obtained in December after slightly more than half the Swedish NHL games had been completed. The fourth test (only the DIF team participated) was conducted in March during the initial part of the playoff period after more than 40 games had been played by each team. Both DIF and SSK teams were eliminated in the quarterfinal rounds of the playoff series in March to April, and at that time the players went on vacation. The fifth and final sampling period was in May during the following preseason period. Tests performed at the various occasions included blood samples as detailed below, analysis of body composition, and for the DIF team, a dietary record. The blood sampling schedule was standardized using identical staff and equipment at each sampling occasion.

Body composition was analyzed by the skinfold method of Durnin and Womersley,[16] and skeleton size was determined according to the method of von Döbeln.[17] Besides total body weight and height, skeleton, fat, and muscle masses were measured by a trained observer (T.Å.) using a Harpenden skinfold caliper and a condylometer suitable for field studies of non-obese subjects.[18-21] At every test occasion, biceps, triceps, subscapular, and suprailiac skinfold thicknesses were measured in triplicate in every subject by carefully standardized procedures (to the nearest .10 mm). Muscle weight was estimated by assuming nonmuscular fat-free soft tissue to be 1.5-fold skeleton weight.

Dietary Design

The diet design for the DIF team has been described in detail elsewhere.[12] The diet was introduced to the players in May and was followed during the entire ice-hockey season from May to the next March. Briefly, it contained approximately 52 to 58 energy percent (E%) carbohydrate, 17 to 19 E% protein, 25 to 30 E% fat, 36% (9 E%) saturated fatty acids, 46% (12 E%) monounsaturated fatty acids, and 18% (5 E%) polyunsaturated fatty acids. These levels differed markedly from those of the precourse diet (Table 2), which was similar to the standard Swedish diet as detailed below. The menu for the DIF team was formulated with the aid of a computer-based nutrition and recipe program (NutriTest; NutriCare, Huddinge, Sweden), and all meals were made from fresh and natural ingredients.[22] The diet was managed by the computerized NutriTest nutrition program, which contains approximately 1,000 items.[22] The program calculates the intake of macronutrients, as well as vitamins, trace elements, and minerals. The use of this program has been validated elsewhere.[12,22] Briefly, precourse data on energy intake (E%) for each individual on the DIF team were obtained using a 7-day dietary record and personal interviews by a dietitian before entrance to the study, and energy intake was calculated using the computerized nutrition program. Together with recommendations from the American Recommended Dietary Allowances, the American Dietetic Association Statement, the World Health Organization Food and Agriculture Organization, and the Swedish Medical Board/Swedish National Food Administration for different groups of subjects according to sex, age, physical fitness, body composition, and energy need or output, calculation of the precourse data provided the basis for individual food recommendations during the study.

The DIF team was told to avoid "cafeteria food" and clearly instructed to avoid "white food" such as fat, salt, and sugar. The reduced fat intake of the diets during the study period as compared with a standard Swedish diet (35 to 40 E%) was compensated for by an increase in compounds rich in slow-acting carbohydrates. The diets were adhered to by all subjects at home; at the hockey arena restaurant where the DIF team regularly had breakfast, lunch, and dinner, and during training camp. Special care was

Table 2. Diet Content (mean ± SEM) During Precourse Conditions and Different Diet Periods for the DIF Team (n = 22)

Period	Energy (kcal)	Carbohydrate		Fat		Protein		Fiber (g)
		g	E%	g	E%	g	E%	
Precourse	2,545 ± 104	280 ± 15	44.9 ± 1.1	114 ± 5	41.6 ± 1.0	94.5 ± 3.4	15.4 ± 0.4	13.9 ± 0.8
May	2,742 ± 203	357 ± 30	53.2 ± 1.6	88.8 ± 8.0	30.2 ± 1.4	117 ± 9	17.7 ± 0.6	24.1 ± 1.8
August	3,299 ± 109	466 ± 19	57.9 ± 1.1	87.4 ± 4.5	24.7 ± 1.0	153 ± 6	19.0 ± 0.5	30.0 ± 1.6
December	2,742 ± 124	349 ± 20	51.7 ± 1.2	88.8 ± 3.7	30.6 ± 1.0	129 ± 6	19.3 ± 0.5	23.3 ± 2.0
February	3,574 ± 268	463 ± 42	52.8 ± 1.5	114 ± 10	29.8 ± 1.2	148 ± 9	17.4 ± 0.7	22.3 ± 1.7
March	3,160 ± 117	394 ± 17	51.3 ± 1.3	106.9 ± 5.9	31.3 ± 1.1	143 ± 6	18.7 ± 0.5	23.7 ± 2.0

taken to instruct the restaurant staff to prepare the meals in accordance with the diets.

Each player maintained a dietary record five times during the study period, and all food consumption during these monitoring periods was recorded for 3 or 7 consecutive days (the latter was the most frequently used monitoring period). All food consumed during the individual record periods was weighed by each subject using portable electronic scales, and consumed food was also recorded in a standardized food diary (Matkontrollen; NutriCare), which transforms consumed food and beverages into standard portions. This registration simplified the long-term dietary records (as described above, repeated recording was performed during several 7-day periods). Every dish and food item occurs only once on the forms, and the participant need only indicate the number of times a particular food item was consumed during the study period. In addition to a facilitated monitoring, the repeated weighing procedures, thoroughly explained at preseason training camps, and the standardized food diary also facilitated the dietary compliance of each subject. Adherence to the diet was further verified during personal interviews with a clinical dietitian, and the consecutive dietary record of each player was coded, quantified, and analyzed using the computerized nutrition program. To ensure accurate record-keeping, all entries of food data were performed by experienced nutritionists. Results from the dietary monitoring periods (Table 2) were instantly calculated and presented to the subject. In addition, the pedagogics of the interactive software for self-reporting consumption for nutritional counseling reinforces acceptance and compliance. During the course of the study, the composition of the diet for the DIF team was changed at several occasions, mostly by varying the relative content of fat and carbohydrate (Table 2). These changes were made to balance variations in energy output during different phases of the ice-hockey season. In contrast to the DIF team, players on the SSK team were not informed about the food program or its possible effects on physical fitness.

Baseline energy intake for the DIF team was 15 E% protein, 42 E% fat, and 45 E% carbohydrate (Table 2). To avoid interference, no registration of the SSK team was made during the study period. However, after completion of the 1-year study, several dietary intake analyses were performed for the SSK team, and their mean energy intake was 16 E% protein, 37 E% fat, and 47 E% carbohydrate. Fat intake was 54% (20 E%) saturated fatty acids, 35% (13 E%) monounsaturated fatty acids, and 11% (4 E%) polyunsaturated fatty acids. This energy intake profile was thus similar to that of the DIF team during the precourse period. Also, an analysis of food intake was conducted for the sedentary controls, which was similar to the postcourse data of the SSK team. Indeed, this energy distribution corresponds to the average Swedish dietary intake.[12]

Biochemical Analysis

At each blood sampling session, approximately 50 mL blood was obtained by venipuncture of the antecubital vein in the morning (7 to 8 AM) following a 12-hour overnight fast. Serum was obtained by centrifugation at 3,000 rpm for 20 minutes at 4°C and was immediately frozen in aliquots, which were stored at −20°C until analyzed. Routine laboratory tests were performed at the Department of Clinical Chemistry of Huddinge University Hospital. Serum total cholesterol and triglyceride levels were analyzed by standard enzymatic procedures (Boehringer, Mannheim, Germany). High-density lipoprotein (HDL) cholesterol was determined after precipitation of apolipoprotein B–containing particles using phosphotungstic acid,[23] and low-density lipoprotein (LDL) cholesterol levels were calculated using the Friedewald formula.[24] Free fatty acids were assayed using nephelometric techniques in a Transcon 102FN automatic analyzer (Orion, Espoo, Finland) with a coefficient of variation of 1.3%.[25] Serum insulin and C-peptide levels were determined by radioimmunoassay using commercial kits obtained from Pharmacia Diagnostics (Uppsala, Sweden) and Behringwerke (Marburg, Germany). IR was calculated from fasting blood glucose and serum insulin concentrations using the homeostasis model assessment of Matthews et al[26]: IR = insulin/$22.5(e^{-\ln glucose})$. Hemoglobin A_{1C} (HbA_{1C}) was determined using ion-exchange chromatography in a fast-protein liquid chromatography (FPLC) system (Pharmacia, Uppsala, Sweden).[27] Detection limits and within- or between-assay variations were as follows: insulin, <2.5 mU/L, 5.9% and 6%; C-peptide, 0.2 nmol/L, 6% and 7%; and HbA_{1C}, 0.1%, 5%, and 5%. Phenotyping of apolipoprotein E was performed as described previously.[28] Briefly, serum was delipidated, separated by isoelectric focusing, blotted onto a nitrocellulose filter, and incubated with antibodies to apolipoprotein E and then with biotinylated secondary antibodies. Individual apolipoprotein E bands were detected using 9-aminocarbazol.

Statistical Methods

All relevant variables were approximately normally distributed. Statistical analysis was performed using Student's *t* test. A paired *t* test was used for differences within teams and unpaired *t* test for differences between teams. In some cases, ANOVA and analysis of covariance with rank statistics (Wilcoxon) was used. Values are presented as the mean ± SEM.

RESULTS

At baseline conditions, players on both teams had a higher body mass index as compared with the sedentary males, and, as demonstrated by body composition data, this was largely due to an increase in muscle weight (Table 1). Notably, fat weight did not differ between the three groups. However, as a result of the increased muscle mass in the athletes, relative amounts of body fat tended to be lower than in the control group, although this difference did not reach significance. Interestingly, although no significant difference was seen for baseline blood glucose levels, differences in other parameters of baseline carbohydrate metabolism were found between the two athlete groups on one hand and the sedentary group on the other. Both the DIF and the SSK groups had significantly lower baseline insulin levels, and baseline C-peptide levels also tended to be lower in the athlete groups compared with the sedentary group. These changes were reflected in a significantly lower IR in both athlete groups compared with the sedentary group (Table 1).

The carefully designed dietary changes, instituted primarily in fat and carbohydrate content for the DIF group (Table 2), were associated with changes in glucose homeostasis. The decrease in relative fat content and the simultaneous increase in carbohydrate content were associated with a decrease of both insulin and C-peptide levels (Fig 1). Changes in serum insulin levels were analyzed by an ANOVA two-tailed test using dietary fat intake and time of year as independent variables. Due to the fact that the calculations were based on 22 independent observations and the degree of freedom was 90, an alternative approach was used, ie, analysis of covariance, where the slope in simple regression on insulin as a function of fat percent was calculated. The slopes were then used in a Wilcoxon test.

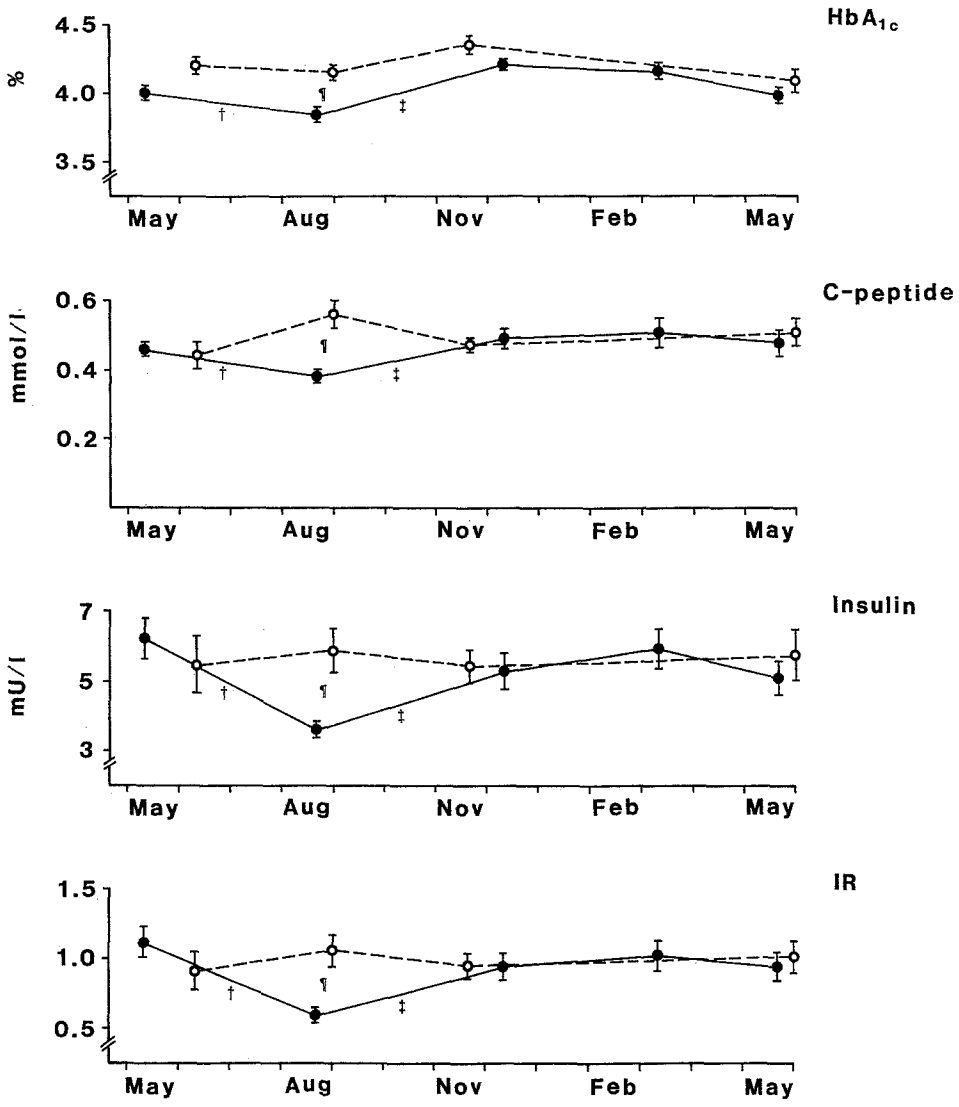

Fig 1. HbA_{1C}, C-peptide, insulin, and calculated IR in DIF (●) and SSK (○) teams during the study period (mean ± SEM). ¶P < .05, DIF *v* SSK; †P < .05, May *v* August sampling session; ‡P < .05 August *v* November sampling session.

There was a significant relation between serum insulin levels and relative fat consumption using both procedures (P = .003 by ANOVA; P = .0235 by Wilcoxon one-tailed test). Fasting glucose levels remained stable throughout the study, but the insulin to glucose ratio decreased significantly in connection with the decrease in fat E% (0.98 ± 0.08 in August *v* 1.55 ± 0.16 at the start of the study, P < .05). No significant changes were observed for the C-peptide to glucose ratio (0.10 ± 0.01 in August *v* 0.11 ± 0.01). In keeping with the changes in insulin levels, there was an even more pronounced decrease of IR in the DIF team (0.59 ± 0.05 in August *v* 1.12 ± 0.12 at the start of the study, P < .05). Further, HbA_{1C} levels also decreased in the DIF team at this time point, indicating an improvement in glycemic control. In contrast, no significant changes in the above parameters occurred in the SSK team (Fig 1). With the gradual relative increase in dietary fat E% for the DIF team, the observed changes in insulin levels, HbA_{1C}, IR, C-peptide levels, and insulin to glucose ratio reverted toward baseline conditions during November/December. Interestingly, IR did not fully return to the level observed before entrance to the food program.

The DIF and SSK teams were well matched with regard to baseline serum lipid levels (Table 1), and they did not differ significantly from the sedentary group in this respect. During the study, only minor changes in serum lipid levels were noted in the DIF team in response to dietary changes. There was a tendency for a decrease in serum triglycerides from sampling period 1 to sampling period 2, although this difference did not reach statistical significance (P < .07; Fig 2). This was accompanied by a decrease in dietary fat E% from 30 to 25 (P < .05). After sampling period 2, there was a gradual increase in serum triglycerides, which returned to baseline levels during the later part of the study. Serum cholesterol levels were largely unchanged in the DIF team during the study (Fig 2), and HDL and LDL cholesterol levels also remained stable during the study (data not shown). Also, for the SSK team, who did not participate in the dietary program, only minor changes were observed in serum lipid levels. Serum free fatty acid levels were ana-

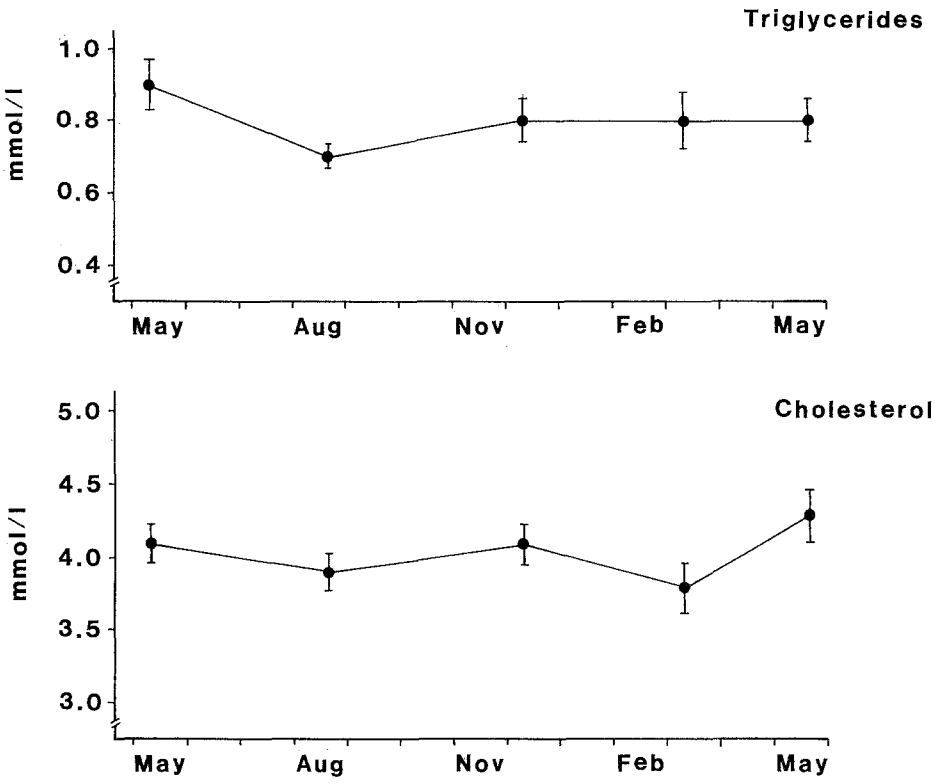

Fig 2. Serum levels of cholesterol and triglycerides in the DIF team during the study period (mean ± SEM).

lyzed in samples drawn at the beginning and end of the study, and there was no significant change between the two teams or the two sampling periods (baseline, 0.48 ± 0.03 *v* 0.47 ± 0.07 mmol/L for DIF and SSK, respectively; final, 0.44 ± 0.04 *v* 0.49 ± 0.04 mmol/L for DIF and SSK, respectively). The apolipoprotein E phenotype pattern in the elite athletes (ε2 0.03; ε3 0.78; ε4 0.19) was similar to the apolipoprotein E allele distribution in the general Swedish population.[29] In particular, the frequency of the apolipoprotein E4 allele, which has been demonstrated to affect response to diet, was not different (0.20 in a healthy Swedish population).

DISCUSSION

In the present study over a 1-year period, dietary changes in fat and carbohydrate intake were found to significantly affect variables of carbohydrate metabolism in professional ice-hockey players. So far, few long-term studies addressing these issues have been performed in athletes even though, in short-term experiments in normal volunteers, a relation between level of physical training and insulin-stimulated glucose utilization has been demonstrated.[15,30,31] Recently, IR, reduced sensitivity of glucose elimination to insulin, has been suggested to be important as a basic factor for disturbances of lipid and carbohydrate metabolism associated with the development of atherosclerosis.[15,32,33] Also in healthy nondiabetic subjects, interactions between IR, insulinemia, and lipids have been found.[34,35] It is therefore of interest that at baseline conditions both athlete groups had lower insulin levels and IR when compared with an age-matched group of healthy sedentary males (Table 1), in agreement with previous findings.[36,37] In preliminary studies, we have also found a lower postprandial increase in insulin and C-peptide in athletes as compared with sedentary controls (Tegelman, Hultman, Carlström, unpublished observations, January 1994). The lower insulin levels and IR occurred despite a higher body mass index in the athlete groups, which in previous studies has been primarily associated with an increased IR.[15,34] However, this has in most cases been due to obesity, whereas in our study fat weight did not differ between the athletes and the sedentary group. Clearly, the increased muscular mass in the athletes contributed significantly to the improved IR. This is compatible with the changes in blood flow and muscle glucose metabolism underlying an enhanced insulin sensitivity recently demonstrated in athletes.[9]

During the course of the study, the decrease in dietary relative fat intake and the increase in slow-acting carbohydrates and fiber intake in the DIF team occurred in parallel with a further decrease in serum insulin and C-peptide levels (Fig 1). During these conditions, HbA_{1C} levels also decreased. These findings clearly suggest a better glycemic control in the euglycemic athletes. It was not possible in the present study to perform repeated studies using the insulin clamp technique in participating athletes during the professional ice-hockey season, and it has to be underscored that the use of the current formula to estimate IR is associated with a higher degree of variation.[26] Nevertheless, it is notable that an improvement in glycemic control occurred in these well-trained individuals despite the fact that at baseline they showed signs of lower IR when compared with sedentary controls (Table 1). Consequently, at the

Table 3. Body Composition (mean ± SEM) in DIF and SSK Teams During the Study Period

Sampling Session	Body Weight (kg)		Muscle Weight (kg)		Fat Weight (kg)		Body Fat (%)	
	DIF	SSK	DIF	SSK	DIF	SSK	DIF	SSK
May	81.4 ± 1.3	82.4 ± 1.7	37.7 ± 0.6	38.5 ± 1.0	11.8 ± 0.6	11.5 ± 0.7	14.4 ± 0.6	13.9 ± 0.7
August	80.3 ± 1.4	83.9 ± 1.6	37.5 ± 0.8	39.5 ± 1.0	10.8 ± 0.5	11.7 ± 0.7	13.4 ± 0.4	13.8 ± 0.7
December	79.7 ± 1.2	83.2 ± 1.6	37.6 ± 0.7	37.9 ± 1.0	10.4 ± 0.5†	13.0 ± 0.7	12.9 ± 0.5†	15.5 ± 0.7
March	80.3 ± 1.3	NA	38.1 ± 0.7	NA	10.3 ± 0.4	NA	12.8 ± 0.4	NA
May	80.4 ± 1.2	82.6 ± 1.8	37.1 ± 0.7	38.1 ± 1.0	11.0 ± 0.5	12.4 ± 0.7	13.6 ± 0.5	14.9 ± 0.6

Abbreviation: NA, not available.
†$P < .005$, DIF *v* SSK.

start of the study, the athletes should already be in a more optimal carbohydrate homeostasis. However, most importantly, improvements in glycemic control were seen only in the team that participated in the diet management program (DIF), and not in the SSK team, which maintained their ordinary diet. Indeed, there was a statistically significant relation between relative fat intake and serum insulin levels in the DIF team over the entire study period. These findings clearly argue against an impact of seasonal variation and strongly indicate a direct relation between relative fat intake and glucose homeostasis. Since both teams were subjected to similar physical strain, these results further indicate that dietary changes contributed to the observed changes in carbohydrate homeostasis. Thus, dietary changes may also affect insulin sensitivity in well-trained athletes. This was further demonstrated by the gradual return to baseline insulin levels, IR, C-peptide levels, and HbA_{1c} levels in the DIF team in parallel with an increase in the relative dietary fat content. The underlying mechanisms behind such dietary modulation of insulin-stimulated glucose disposal in individuals in whom this is already enhanced remain to be determined. However, it is of interest that the DIF team demonstrated a decrease in fat weight during the study, but no such decrease occurred in the SSK team (Table 3). In previous studies, intraabdominal fat has correlated with decreased insulin clearance and increases in C-peptide levels, as well as development of type II diabetes.[38-40] Thus, the present results suggest that a close relation between dietary changes, reduced fat weight, and improved carbohydrate metabolism might also be present in well-trained athletes.

In contrast to the findings for carbohydrate metabolism, only relatively minor changes in blood lipids were noted in response to changes in dietary fat and carbohydrate intake in the DIF team. The most prominent finding, although not statistically significant, was the decrease in serum triglyceride levels that occurred in parallel with the decrease in C-peptide and insulin levels. In several previous studies, serum triglyceride levels have correlated positively with IR in both diabetics and nondiabetics.[15] However, the lack of effect in the athlete group could be due to the low baseline triglyceride levels.

The present study is one of few that have monitored long-term metabolic changes in athletes in response to dietary changes. In conclusion, our results confirm earlier findings that well-trained athletes, with a large muscle weight but not necessarily a lower fat weight, have an improved carbohydrate homeostasis and a reduced IR. We also extend these results with our finding that glycemic control and IR improved even further in this group during dietary manipulation. This underscores the impact of nutritional factors on carbohydrate and perhaps lipid metabolism in optimally trained athletes. Further studies are needed to explore the mechanisms behind these effects.

ACKNOWLEDGMENT

Part of this study was performed at The Clinical Research Centre, Huddinge University Hospital. We are indebted to Gunvor Nilsson and Gunnel Zeisig for excellent technical assistance, to all members of the two ice-hockey teams, DIF and SSK, and to our control subjects for excellent cooperation. We are also grateful to Professor Herman Adlercreutz and his staff at the Department of Clinical Chemistry, University of Helsinki at Mejlans Hospital, for performing the free fatty acid analyses, to Iréne Engström for performing the insulin analyses, and to Björn Jonsson and Anders Lindeberg, Department of Biostatistics, Kabi-Pharmacia, Stockholm, Sweden, for statistical advice.

REFERENCES

1. Soman VR, Koivisto VA, Deibert D, et al: Increased insulin sensitivity and insulin binding to monocytes after physical training. N Engl J Med 301:1200-1204, 1979

2. Mikines KJ, Sonne B, Farrel PA, et al: Effect of physical exercise on sensitivity and responsiveness to insulin in humans. Am J Physiol 254:E248-E259, 1988

3. Yki-Järvinen H, Koivisto VA: Effect of body composition on insulin sensitivity. Diabetes 32:965-969, 1983

4. DeFronzo RA, Jacot E, Jequier E, et al: The effect of insulin on the disposal of intravenous glucose. Results from indirect calorimetry and hepatic and femoral venous catheterization. Diabetes 30:1000-1007, 1981

5. Laakso M, Edelman SV, Brechtel G, et al: Decreased effect of insulin to stimulate skeletal muscle blood flow in obese man. A novel mechanism for insulin resistance. J Clin Invest 85:1844-1852, 1990

6. Laakso M, Edelman SV, Olefsky JM, et al: Kinetics of in vivo muscle insulin-mediated glucose uptake in human obesity. Diabetes 39:965-974, 1990

7. Edelman SV, Laakso M, Wallace P, et al: Kinetics of insulin-mediated and non-mediated glucose uptake in humans. Diabetes 39:955-964, 1990

8. Baron AD, Laakso M, Brechtel G, et al: Reduced capacity and affinity of skeletal muscle for insulin-mediated glucose uptake in noninsulin-dependent diabetic subjects. J Clin Invest 87:1186-1194, 1991

9. Ebeling P, Bourey R, Koranyi L, et al: Mechanism of enhanced insulin sensitivity in athletes. Increased blood flow, muscle glucose transport protein (GLUT-4) concentration, and glycogen synthase activity. J Clin Invest 92:1623-1631, 1993

10. Opstad PK, Aakvaag A: Decreased serum levels of oestradiol, testosterone and prolactin during prolonged physical strain and sleep deprivation, and the influence of a high calory diet. Eur J Appl Physiol 49:343-348, 1982

11. Adlercreutz H, Härkönen M, Kouppasalmi K, et al: Effect of training on plasma anabolic and catabolic steroid hormones and their response during physical exercise. Int J Sports Med 7:27-28, 1986

12. Tegelman R, Åberg T, Pousette Å, et al: Effects of a diet regimen on pituitary and steroid hormones in male ice hockey players. Int J Sports Med 13:424-430, 1992

13. Olefsky JM: The insulin receptor: Its role in insulin resistance in obesity and diabetes. Diabetes 25:1154-1166, 1976

14. Abbott WGH, Lillioja S, Young AA, et al: Relationships between plasma lipoprotein concentrations and insulin action in an obese hyperinsulinemic population. Diabetes 36:897-904, 1987

15. Reaven GM: The role of insulin resistance in human disease. Diabetes 37:1595-1607, 1988

16. Durnin JVGA, Womersley J: Body fat assessed from total body density and its estimation from skinfold thickness: Measurements on 481 men and women aged from 16 to 72 years. Br J Nutr 32:77-97, 1974

17. von Döbeln W: Human standard and maximal metabolic rate in relation to fat-free body mass. Acta Physiol Scand 37:1-79, 1956

18. Edwards DAW, Hammond WH, Heally MJR, et al: Design and accuracy of calipers for measuring subcutaneous tissue thickness. Br J Nutr 9:133-143, 1955

19. Heymsfield S, Clifford B, Seitz SB, et al: Anthropometric assessment of adult protein-energy malnutrition, in Wright RA, Heymsfield S (eds): Nutritional Assessment. Boston, MA, Blackwell Scientific, 1984, pp 27-82

20. Davidson S, Passmore R, Brock JF, et al: Obesity, in Human Nutrition and Dietetics. New York, NY, Churchill Livingstone, 1979, pp 244-254

21. Garrow JS: New approaches to body composition. Am J Clin Nutr 35:1152-1158, 1982

22. Åberg T, Åberg N, Lundberg E, et al: The computor—A necessary device during dietary treatment and information. Naringsforskning 35:116-122, 1991

23. Lopes-Virella MF, Stone P, Ellis S, et al: Cholesterol determination in high-density lipoproteins separated by three different methods. Clin Chem 23:882-884, 1977

24. Friedewald WT, Levy RI, Fredrickson DS: Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem 18:499-502, 1972

25. Härkönen M, Adlercrutz H: Fluorometric analysis in routine clinical chemistry. Trends Anal Chem 2:176-180, 1983

26. Matthews DR, Hosker JP, Rudenski AS, et al: Homeostasis model assessment: Insulin resistance and β-cell function from fasting glucose and insulin concentrations in man. Diabetologia 28:412-419, 1985

27. Jeppson JO, Jentorp P, Sundqvist C, et al: Measurements of hemoglobin A_{1C} by a new liquid chromatographic method: Clinical utility and relation to glucose tolerance evaluated. Clin Chem 32:1867-1872, 1986

28. Berglund L, Wiklund O, Eggertsen G, et al: Apolipoprotein E phenotypes in familial hypercholesterolaemia: Importance for expression of disease and response to therapy. J Intern Med 233:173-178, 1993

29. Eggertsen G, Tegelman R, Ericsson S, et al: Apolipoprotein E polymorphism in a healthy Swedish population: Variation of allele frequency with age and relation to serum lipid concentrations. Clin Chem 39:2125-2129, 1993

30. Rosenthal M, Haskell WL, Solomon R, et al: Demonstration of a relationship between level of physical training and insulin-stimulated glucose utilization in normal humans. Diabetes 32:408-411, 1983

31. Nuutila P, Knuuti MJ, Heinonen OJ, et al: Different alterations in the insulin-stimulated glucose uptake in the athlete's heart and skeletal muscle. J Clin Invest 93:2267-2274, 1994

32. Sowers JR: Insulin resistance, hyperinsulinemia, dyslipidemia, hypertension and accelerated atherosclerosis. J Clin Pharmacol 32:529-535, 1992

33. Garcia-Webb P, Bonser AM, Whiting D, et al: Insulin resistance—A risk factor for coronary heart disease? Scand J Clin Lab Invest 43:677-685, 1983

34. Winocour PH, Kaluvya S, Ramaiya K, et al: Relation between insulinemia, body mass index, and lipoprotein composition in healthy, non-diabetic men and women. Arterioscler Thromb 12:393-402, 1992

35. Godsland IF, Crook D, Walton C, et al: Influence of insulin resistance, secretion and clearance on serum cholesterol, triglycerides, lipoprotein cholesterol and blood pressure in healthy men. Arterioscler Thromb 12:1030-1035, 1992

36. Mikines KJ, Sonne B, Tronier B, et al: Effects of training and detraining on dose-response relationship between glucose and insulin secretion. Am J Physiol 256:E588-E596, 1989

37. Dela F, Mikines KJ, von Linstow M, et al: Does training spare insulin secretion and diminish glucose levels in real life? Diabetes Care 15:1712-1715, 1992 (suppl 4)

38. Peiris A, Mueller RA, Smith GA, et al: Splanchnic insulin metabolism in obesity. Influence of body fat distribution. J Clin Invest 78:1648-1657, 1986

39. Bergstrom RW, Newell-Morris LL, Leonetti DL, et al: Association of elevated fasting C-peptide level and increased intra-abdominal fat distribution with development of NIDDM in Japanese-American men. Diabetes 39:104-111, 1990

40. Bergstrom RW, Leonetti DL, Newell-Morris LL, et al: Association of plasma triglyceride and C-peptide with coronary heart disease in Japanese-American men with a high prevalence of glucose intolerance. Diabetologia 33:489-496, 1990