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Short-term effects of dietary fat on intramyocellular lipid in sprinters and endurance runners

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Abstract

The effect of short-term fat loading on intramyocellular lipid (IMCL) in different types of muscle in endurance runners and sprinters has not been fully elucidated yet. The purpose of this study was to investigate the effect of dietary lipid on IMCL in soleus muscle (SOL) and tibialis anterior muscle (TA) during training period in endurance runners and sprinters. Seven male endurance runners and 7 male sprinters were selected to participate in the study. We measured TA- and SOL-IMCL levels after 3-day course of isocaloric normal- (25%), high- (60%), and low-fat (10%) diet during training period by ^{1}H–magnetic resonance spectroscopy in each subject. In sprinters, TA- and SOL-IMCL levels were comparable after each diet protocol. However, in endurance runners, TA-IMCL levels after normal-fat and high-fat diets were 1.7 times and 3.0 times higher than that after low-fat diet, respectively. The SOL-IMCL values after normal-fat diet and high-fat diet were 1.5 times and 1.6 times higher than that after low-fat diet, respectively. In addition, the TA-IMCL level after high-fat diet, but not SOL-IMCL, was significantly higher compared with that after normal-fat diet. Our data suggested that short-term dietary fat challenge during training period significantly altered IMCL level in endurance runners, but not in sprinters. In addition, response to fat loading on IMCL was influenced by variation of muscle type in endurance runners. These phenotypic and regional differences might be explained by differences in type of exercise training and muscle fiber composition.

1. Introduction

Recent studies demonstrated that elevated levels of intramyocellular lipid (IMCL), consisting primarily of triglyceride, is associated with insulin resistance [1-4]. High IMCL content is linked to impaired mitochondrial bioenergic and oxidative capacity in skeletal muscle [5-7]. Intramyocellular lipid can be measured noninvasively by proton magnetic resonance spectroscopy (^{1}H-MRS) and is a useful marker of insulin resistance in the clinical setting. However, in endurance-trained athletes, who are profoundly insulin sensitive, IMCL level is paradoxically increased [8]. In addition, IMCL levels correlate with maximal oxygen uptake ($\dot{V}O_2$max) in lean healthy subjects [9]; and exercise training increases IMCL in older adults [10]. Thus, IMCL level is not invariably associated with insulin resistance. Whereas IMCL is well recognized as a physiological marker of skeletal muscle function, IMCL level seems to be affected by various factors that are not yet fully elucidated.

The muscular system consists of various types of muscles that contain different proportions of muscle fiber types. The soleus muscle (SOL) is a typical muscle containing a high proportion of type I muscle fibers (oxidative slow-twitch), whereas the tibialis anterior muscle (TA) is a typical muscle with a large fraction of type II fibers (glycolytic fast-twitch) [11]. It has been reported that sprinters have relatively low type I fiber composition and low aerobic capacity, whereas both of these parameters are high in endurance runners [12]. Previous histochemical studies showed that type I fiber contains 3-fold higher IMCL than type II fiber [13]. Reflecting this difference, the SOL contains ~3-fold greater IMCL than TA [14].

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Although the above-mentioned factors are determinants of IMCL, it has been reported also that IMCL is influenced by exercise and dietary fat. Previous studies suggested that endurance exercise decreased IMCL and that high dietary fat is associated with elevated IMCL contents [15-20]. In addition, it has been reported that a high-fat diet (55%-60% fat) for 3 days increased TA-IMCL by ~50%, whereas SOL-IMCL was not significantly affected in healthy subjects. The same study also reported that a low-fat diet (18%-23% fat) did not change IMCL level both in TA and SOL [1]. Similar short-term dietary challenges were applied to endurance runners during training period to enhance their performance because low IMCL may limit exercise performance [21]. However, the effect of fat loading on TA- and SOL-IMCL in endurance runners has not been fully elucidated yet. Accordingly, we hypothesized that a 3-day fat loading may affect IMCL level in endurance runners during training period and that response to fat loading on IMCL might be influenced by variation of muscle type seen in TA and SOL. In addition, the effect of those dietary challenges in sprinters has not been elucidated yet.

The present study was designed to examine the effect of 3-day fat loading on TA- and SOL-IMCL in endurance runners and sprinters during training period. For this purpose, we recruited 7 endurance runners and 7 sprinters. In each subject, we measured TA- and SOL-IMCL levels after a 3-day course of normal-, high-, and low-fat diet. We found that phenotype (endurance runners and sprinters) and muscle type (TA and SOL) influenced changes in IMCL induced by each diet.

2. Methods

2.1. Subjects and preliminary testing

Seven male endurance runners (mean time for running 5000 m = 15 minutes, 32 seconds) and 7 male sprinters (100- to 400-m runners) were selected to participate in the study. All subjects belonged to the track and field club of Juntendo University and were well trained, healthy, and not treated with medications for chronic diseases. All subjects gave written informed consent to the study, which was approved by the Ethics Committee of Juntendo University. The $\dot{V}O_2$max was determined by incremental exercise test with a calibrated mechanically braked cycle ergometer (818E; Monark, Vansbro, Sweden). In this test, the subject began pedaling at 1.5 kiloponds (kp); and the workload was increased by 1 kp every 4 minutes. Subjects were asked to keep a pedaling rate of 50 rpm. After 12 minutes of exercise, the workload was increased by 0.5 kp every 1 minute until exhaustion. Respiratory gas data were averaged every 30 seconds. Respiratory gases were analyzed using open-circuit auto O_2 and CO_2 analyzers with a hot-wire flow meter (AE300S; Minato Medical Science, Osaka, Japan). Total body fat content (% Fat) was measured by a specific analyzer (InBody; BIOSPACE, Tokyo, Japan).

2.2. Study design

The experimental protocol is outlined in Fig. 1. Each subject completed 3 trials in a randomized crossover design, with a 2- to 3-week washout period separating each trial. All subjects consumed low- or high-fat diet after 3 days of control normal-fat diet. The weekly training menu for endurance runners was ~20 km running per day at a self-selected pace (60%-70% of $\dot{V}O_2$max) during the intervention period, whereas that for sprinters was anaerobic running training (30- to 400-m sprint training). Both training menus were almost fixed in each protocol and managed by trainers. In all subjects, a training-free period was scheduled for about 20 hours before the measurement of IMCL. Intramyocellular lipid was measured by ^{1}H-MRS as described below. Afterward, blood samples were obtained from each subject for biochemical tests and respiratory gas analysis.

2.3. Dietary manipulation

During the intervention period, subjects were provided packed meal and prohibited to eat anything else. Every subject was provided isocaloric low-fat (10% fat, 70% carbohydrate, 20% protein), normal-fat (25% fat, 55% carbohydrate, 20% protein), or high-fat diet (60% fat, 20% carbohydrate, 20% protein) followed by 3-day normal-fat diet. The fat composition of high-fat diet was 32% saturated, 40% monounsaturated, and 28% polyunsaturated fatty acid. This fat composition is similar to the common diet of Japanese [22]. The energy and food content–controlled diets were prepared by a food company (Musashino Foods, Saitama, Japan).

2.4. Biochemical tests

Fasting blood glucose and free fatty acid (FFA) were measured using an autoanalyzer (SRL Laboratory, Tokyo, Japan). Plasma insulin and leptin concentrations were determined by radioimmunoassay (LINCO Research, St Charles, MO). Serum adiponectin concentrations were

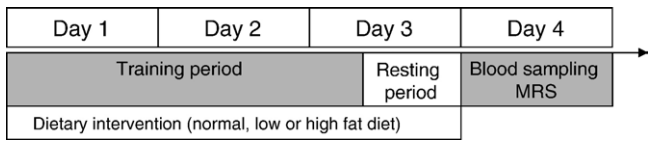

Fig. 1. Experimental protocol. All subjects consumed fat-adjusted foods for 3 days. Weekly training menu for endurance runners was ~20 km running per day at a self-selected pace (60%-70% of $\dot{V}O_2$max) during the intervention period and that for sprinters was anaerobic running training (30- to 400-m sprint training). Both training menus were almost fixed in each protocol and managed by trainers. All subjects had ~20-hour training-free period before the measurement of IMCL. The IMCL values of the right TA and SOL were measured by ^{1}H-MRS.

Table 1
Subjects' characteristics

	Sprinters	Endurance runners
n	7	7
Age (y)	20.7 ± 0.3	21.1 ± 0.4
Height (cm)	174.0 ± 2.2	170.0 ± 1.8 *
Weight (kg)	66.0 ± 2.5	58.4 ± 1.9 *
BMI (kg/m^2)	21.8 ± 0.4	20.2 ± 0.4 *
%Fat	11.5 ± 0.5	12.9 ± 1.0
$\dot{V}O_2$max (mL/[min kg])	49.7 ± 2.7	59.1 ± 6.1 *

Data are mean ± SEM. BMI indicates body mass index.
* $P < .05$ vs sprinters.

measured by an enzyme-linked immunosorbent assay (Otsuka Pharmaceuticals, Tokyo, Japan).

2.5. Proton magnetic resonance spectroscopy

Intramyocellular lipid was measured at fasting state as described previously [23-25]. Briefly, IMCL values of the right TA and SOL were measured by ^{1}H-MRS using a knee coil (VISART EX V4.40; Toshiba, Tokyo, Japan). Voxels ($1.2 \times 1.2 \times 1.2$ cm^3) were positioned in the muscle avoiding visible interfascial fat and blood vessels, and the voxel sites were matched carefully at each examination. Imaging parameters were set as follows: repetition time, 1500 milliseconds; echo time, 136 milliseconds; acquisition numbers, 192 and 1024 data points over a 1000-kHz spectral width. After examination, resonances were quantified by reference to the methylene signal intensity (S-fat), with peaks being observed at ~1.25 ppm. Intramyocellular lipid was quantified by the S-fat and using a creatine signal at 3.0 ppm (Cre) as the reference, and was expressed as a ratio relative to Cre (S-fat/Cre).

2.6. Statistical analysis

All data are expressed as mean ± SEM. Differences between 2 groups were tested by unpaired *t* tests. Repeated-measures analysis of variance and Student-Newman-Keuls test were applied to evaluate the effects of each dietary intervention on IMCL and other measured parameters.

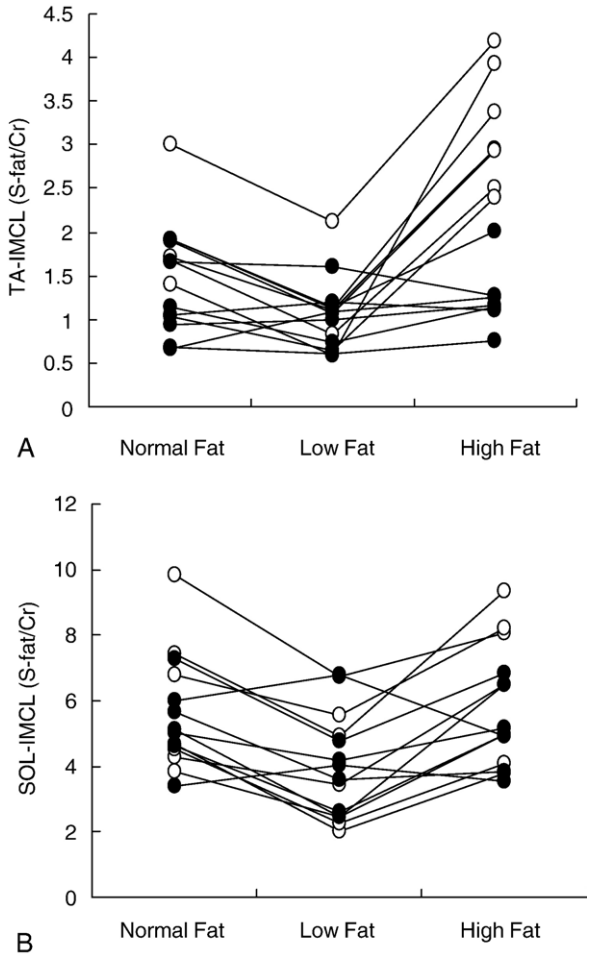

Fig. 2. Individual changes in IMCL values for endurance runners (open circles) and sprinters (closed circles) after dietary intervention in TA (A) and SOL (B). The mean IMCL values are listed in Table 3.

Simple linear regression analysis was performed to evaluate the correlation of metabolic parameters. Statistical significance was set at $P < .05$.

Table 2
Clinical parameters after each dietary intervention for sprinters and endurance runners

	Sprinters			Endurance runners		
	Low fat	Normal fat	High fat	Low fat	Normal fat	High fat
Weight (kg)	66.0 ± 2.4	66.0 ± 2.5	65.6 ± 2.4	58.3 ± 1.8	58.4 ± 1.9	58.0 ± 2.0
%Fat	11.8 ± 0.9	11.5 ± 0.5	11.4 ± 0.8	12.5 ± 1.1	12.9 ± 1.0	12.5 ± 0.9
Insulin (μU/mL)	4.4 ± 0.6	6.2 ± 0.7 *	3.9 ± 0.4 †	4.5 ± 0.4	5.6 ± 0.7	4.6 ± 0.7
Glucose (mg/dL)	90.0 ± 1.3	96.7 ± 1.1 *	87.7 ± 1.9 †	93.1 ± 2.2	93.9 ± 2.0	89.9 ± 2.1
FFA (mmol/L)	0.45 ± 0.03	0.31 ± 0.07	0.56 ± 0.07 †	0.41 ± 0.07	0.35 ± 0.06	0.47 ± 0.08
Leptin (ng/mL)	1.7 ± 0.2	1.3 ± 0.2	1.3 ± 0.2	1.5 ± 0.1	1.5 ± 0.1	1.3 ± 0.1 *
Adiponectin (μg/mL)	6.9 ± 0.4	8.4 ± 0.7 *	6.0 ± 0.5 †	9.7 ± 0.9 ‡	10.8 ± 1.4	8.9 ± 0.9 ‡

Data are mean ± SEM.
† $P < .05$ vs normal fat.
‡ $P < .05$ vs sprinters after same diet protocol.
* $P < .05$ vs low fat.

3. Results

3.1. Characteristics of subjects and high- and low-fat diet–induced changes in metabolic parameters

Table 1 lists the characteristics of participating subjects. The $\dot{V}O_2$max was measured before the study period, and the other data were obtained after the normal-fat–diet period. Endurance runners were significantly shorter, their body mass index was smaller, and $\dot{V}O_2$max was higher than sprinters. Table 2 shows the effects of dietary intervention on various metabolic parameters in the 2 groups of subjects. In both groups, dietary intervention did not change body weight or %Fat. However, fasting blood glucose and insulin concentrations were significantly decreased after low-fat and high-fat diet compared with normal-fat diet protocol in sprinters, but not in endurance runners. In sprinters, serum FFA concentration was significantly higher after high-fat diet than that after normal-fat diet. Serum adiponectin concentrations were significantly lower after low-fat and high-fat diet in sprinters, whereas no such changes were observed in endurance runners. Serum adiponectin concentrations were significantly higher in endurance runners after low-fat and high-fat diet compared with sprinters. Serum leptin concentrations were slightly but significantly decreased in endurance runners after high-fat diet (Table 2).

3.2. Changes in regional IMCL level in sprinters and endurance runners

In sprinters, TA- and SOL-IMCL levels were comparable after each diet protocol. However, in endurance runners, TA-IMCL levels after normal-fat and high-fat diets were 1.7 times and 3.0 times higher than that after low-fat diet, respectively (Fig. 2, Table 3). Furthermore, SOL-IMCL values after normal-fat diet and high-fat diet were 1.5 times and 1.6 times higher than that after low-fat diet, respectively. The TA-IMCL level after high-fat diet, but not SOL-IMCL, was significantly higher compared with that after normal-fat diet. Thus, dietary fat contents significantly altered IMCL level in endurance runners, but not in sprinters. In addition, response to fat loading on IMCL was influenced by variation of muscle type seen in TA and SOL in endurance runners.

The mean TA-IMCL levels of endurance runners after normal- and high-fat diet were significantly higher than those of sprinters, whereas SOL-IMCL was significantly higher only after high-fat diet (Table 3). Especially, the TA-IMCL levels after high-fat diet of all endurance runners were higher than those of sprinters (Fig. 2). On the other hand, TA-IMCL did not correlate with that of SOL after high-fat diet.

3.3. Correlation between IMCL and $\dot{V}O_2$max

In sprinters, TA- and SOL-IMCL levels did not correlate with $\dot{V}O_2$max after each dietary intervention (data not shown). On the other hand, in endurance runners, TA- and SOL-IMCL values after normal-fat and low-fat diet, but not high-fat diet, correlated significantly with $\dot{V}O_2$max (Fig. 3).

4. Discussion

In this study, we found that dietary fat contents influenced IMCL levels, especially TA-IMCL in endurance runners. These changes were only modest in sprinters in both muscle types. The TA-IMCL level after high-fat diet, but not SOL-IMCL, was significantly higher compared with that after normal-fat diet. In addition, TA-IMCL after normal- and low-fat diet correlated significantly with $\dot{V}O_2$max in endurance runners.

Previous reports suggested that endurance exercise reduced IMCL and that the recovery of IMCL was highly dependent on diet composition after exercise [18,19,23]. Larson-Meyer et al [19] demonstrated that 2-hour treadmill run (67% of $\dot{V}O_2$max) decreased SOL-IMCL by ~25%. Moderate-fat diet (35% of energy) allowed IMCL stores to return to baseline by 22 hours and overshoot by 70 hours after exercise [19]. However, low-fat diet (10% of energy) did not result in recovery of IMCL level to baseline level at 70 hours after exercise. Decombaz et al [18] showed that IMCL in TA decreased by 22% to 26% after 2-hour running at 50% $\dot{V}O_2$max. After 30-hour recovery with high-fat diet (55% of energy), the IMCL level was 30% to 45% higher than that pre-exercise, whereas low-fat diet (15% of energy) did not allow IMCL stores to return to baseline. In our protocol, endurance runners did daily training (running ~20 km/d at 60%-70% of $\dot{V}O_2$max); and we evaluated IMCL level after ~20-hour resting after last bout of exercise. In agreement with previous studies, our data suggest that the variation of IMCL level during training period is dependent on the amount of exercise and recovery time as well as dietary fat content in endurance runners.

Table 3
Intramyocellular lipid levels after each dietary intervention for sprinters and endurance runners

	Sprinters			Endurance runners		
	Low fat	Normal fat	High fat	Low fat	Normal fat	High fat
TA-IMCL (S-fat/Cr)	1.05 ± 0.12	1.15 ± 0.18	1.24 ± 0.14	1.07 ± 0.19	1.81 ± 0.23* †	3.18 ± 0.26* † ‡
SOL-IMCL (S-fat/Cr)	4.06 ± 0.55	5.10 ± 0.46	5.30 ± 0.46	3.92 ± 0.70	5.92 ± 0.83 *	6.43 ± 0.83* †

Data are mean ± SEM.
† $P < .05$ vs normal fat.
‡ $P < .05$ vs sprinters after same diet protocol.
* $P < .05$ vs low fat.

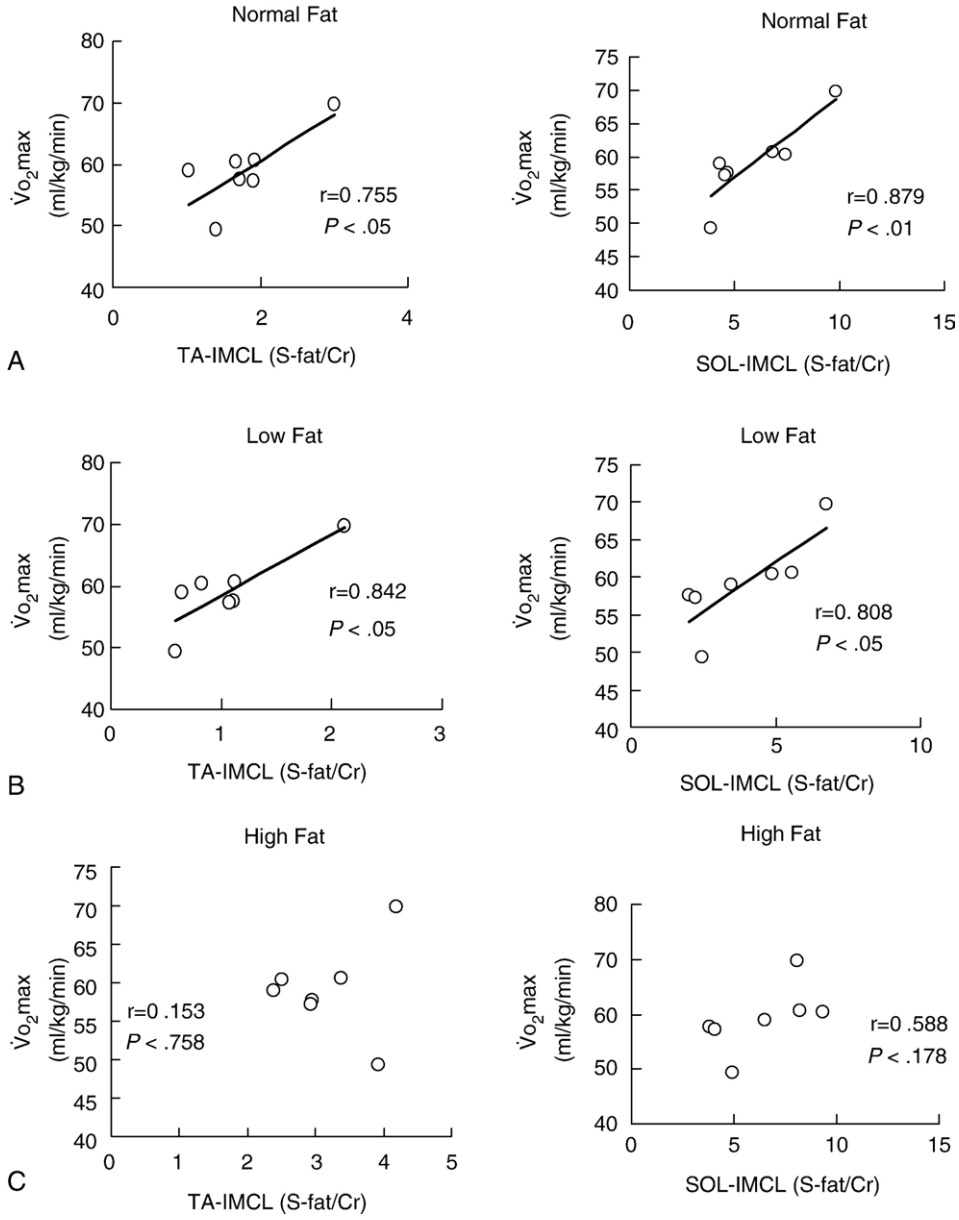

Fig. 3. Relationships between IMCL levels in TA (left) and SOL (right) and $\dot{V}O_2max$ after normal- (A), low- (B), and high-fat (C) dietary intervention in endurance runners.

In the present study, TA-IMCL level after high-fat diet, but not SOL-IMCL, was significantly higher compared with that after normal-fat diet, suggesting that response to fat loading on IMCL during training period was dependent on muscle type in endurance runners. As mentioned above, endurance exercise reduced IMCL and the recovery of IMCL was highly dependent on diet composition after exercise [18,19,23]. From these results, it is speculated that expenditure and recovery of IMCL differ between TA and SOL, thus influencing IMCL level during training period. Consistent with this hypothesis, it has been reported that the increase in TA-IMCL was more pronounced than that in SOL-IMCL after short-term FFA infusion and dietary fat loading, respectively [1]. On the other hand, one bout of prolonged moderate-intensity aerobic exercise reduced TA- and SOL-IMCL; and reduction rates of IMCL seemed to be similar [26,27]. Because SOL contains a high proportion of type I muscle fibers and TA contains a high proportion of type II fibers [11], these regional different response by fat loading might be explained, at least in part, by differences in muscle fiber composition. Further experiments are required to elucidate the exact mechanism of regional difference of IMCL response to exercise and fat loading.

In contrast to these results observed in endurance runners, dietary fat contents did not significantly alter IMCL level in sprinters. Bachmman et al [1] reported that there was high interindividual variation in the increase in IMCL contents by fat loading; and it was suggested that

this variation might reflect insufficient control of parameters, such as physical activity. We also reported previously that 2-week low calorie with low saturated fat diet plus exercise therapy in hospitalized type 2 diabetes mellitus patients resulted in 19% reduction of IMCL and 56% increase in peripheral insulin sensitivity and that these changes were not observed in patients on similar diet restriction but no exercise therapy [23]. In the study, physical activity correlated significantly with the percentage of changes in IMCL and glucose infusion rate [23]. In the present study, the weekly training menu for endurance runners was ~20 km running per day at a self-selected pace (60-70% of $\dot{V}O_2$max) during the intervention period, whereas that for sprinters was anaerobic running training (30- to 400-m sprint training). Thus, it is possible that the different response of IMCL level to dietary fat content between sprinters and endurance runners may be, at least partly, due to the type of exercise training.

In the present study, we observed that TA- and SOL-IMCL after normal-fat and low-fat diet significantly correlated with $\dot{V}O_2$max. In agreement with our data, previous data [8,9] demonstrated that the increase in both IMCL and oxidative capacity is observed in endurance-trained athletes with high $\dot{V}O_2$max and that TA-IMCL levels positively correlate with $\dot{V}O_2$max in lean healthy subjects. In addition, exercise training increases IMCL in older persons in parallel with an enhanced oxidative capacity [10]. Thus, in trained endurance runners, higher IMCL level might reflect enhanced oxidative capacity, which is in part assessed by our measurement of $\dot{V}O_2$max. On the other hand, in this study, IMCL does not correlate with $\dot{V}O_2$max after high-fat diet. It seems that high-fat diet might mask the relationship between IMCL and oxidative capacity. We cannot find the exact reason. Further experiments are required to elucidate the relationship between IMCL and $\dot{V}O_2$max.

Our results showed phenotypic differences in the response of serum adiponectin level to 3-day fat loading. Only in sprinters did serum adiponectin concentrations decrease significantly after low-fat and high-fat diet. Adiponectin is known to increase fat oxidation in muscle and liver, thus improving insulin resistance in rodents [28]. In humans, low adiponectin levels correlated with increased IMCL levels and insulin resistance in muscles [29-31]. Thus, adiponectin might be one of the factors balancing IMCL level after short-term fat change. Further experiments are required to elucidate the relationship between the response of IMCL to fat loading and adiponectin level.

In conclusion, our data suggested that short-term dietary fat challenge during training period significantly altered IMCL level in endurance runners, but not in sprinters. In addition, response to fat loading on IMCL was influenced by variation of muscle type in endurance runners. These phenotypic and regional differences might be explained by differences in type of exercise training and muscle fiber composition.

Acknowledgments

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