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Basic nutritional investigation

Effects of different ratios of monounsaturated and polyunsaturated fatty acids to saturated fatty acids on regulating body fat deposition in hamsters

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ABSTRACT

Objective: Effects of monounsaturated fatty acid (MUFA) and polyunsaturated fatty acid consumption on regulating body fat accumulation and body weight gain are controversial between animal and human studies.
Methods: We designed a 2 × 2 factorial study, with two levels of MUFAs (60% and 30%) and two levels of polyunsaturated-to-saturated fatty acid (P/S) ratio (5 and 3) to prepare four kinds of experimental oils consisting of 60% MUFAs with a high or low P/S ratio (HMHR or HMLR, respectively) or 30% MUFAs with a high or low P/S ratio (LMHR or LMLR, respectively). Thirty-two male golden Syrian hamsters were randomly divided into four groups and fed the experimental diets containing 15% (w/w) fat for 12 wk.
Results: No difference was observed in the mean daily food intake. Hamsters fed the LMLR diet had increased weight gain, epididymal and retroperitoneal white adipose tissues, plasma non-esterified fatty acids, insulin, hepatic acetyl coenzyme A carboxylase and malic enzyme activities, and mRNA expressions of peroxisome proliferator-activated receptor-α and sterol regulatory element-binding protein-1c among all groups ($P < 0.05$). Hamsters fed the HMHR diet had lower plasma insulin levels and hepatic acetyl coenzyme A carboxylase activities among groups ($P < 0.05$) and elevated hepatic acyl coenzyme A oxidase and carnitine palmitoyltransferase-I activities compared with those fed the LMLR diet ($P < 0.05$).
Conclusion: Hamsters fed the LMLR diet had increased weight gain and body fat accumulation, whereas the HMHR diet appeared to be beneficial in preventing white adipose tissue accumulation by decreasing plasma insulin levels and increasing hepatic lipolytic enzyme activities involved in β-oxidation.

Introduction

Because a high-fat diet provides a higher dietary energy density, it is associated with obesity and related disorders including metabolic syndrome and chronic diseases [1,2]. Previous studies have shown that several dietary oils affect body weight gains and their use remains controversial [3–5]. Those results found that the increase in body weight was higher in animals fed soybean oil than those fed safflower and fish oils [4]. Rats fed high-fat fish and olive oils had greater body fat deposition than those fed safflower oil [5]. Some bioactive lipids, such as medium-chain fatty acids, diacylglycerols, conjugated fatty acids, and ω-3 polyunsaturated fatty acids (PUFAs), are involved in lipid storage and physiologic functions of lipid metabolism [6], whereas cooking oil contains only small amounts of these bioactive lipids. However, diets with a high polyunsaturated-to-saturated fatty acid (P/S) ratio could increase postprandial fat oxidation [7,8]. Thus, it is important to define the roles of the P/S ratio and monounsaturated fatty acids (MUFAs) on weight gain and fat accumulation using a 2 × 2 experimental design.

Several studies have also shown that the influences of MUFAs on regulating body weight remain controversial in human trials and animal models. Rats fed olive oil diets had higher body weight gain and abdominal fat deposition than those fed

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safflower or lard oil diets [5,9]. Olive oil consisting of a large amount of MUFAs and a small amount of PUFAs increased hepatic lipogenic enzyme activities, whereas sunflower and linseed oils containing a large amount of PUFAs decreased lipogenic enzyme activities [10]. Opposite effects of MUFAs on weight gain have also been reported; in a prospective cohort study, a high consumption of olive oil was not related to a greater weight gain [11]. Clifton et al. [12] reported that the effects of high-fat diets rich in MUFAs (canola and high-oleic sunflower oils) on weight loss were similar to those of low-fat diets. According to the controversies of these studies, the difference in fatty acid composition between olive oil and canola oil is the different amount of PUFA, which may be one of reasons for the effects of fats on body weight. Moreover, at the same fatty acid carbon chain length, fatty acid oxidation rates of MUFAs are fastest and followed by those of PUFAs and saturated fatty acids (SFAs) [13].

In contrast, fatty acids regulate lipogenesis through various transcription factors and nuclear receptors, including peroxisome proliferator-activated receptors (PPARs), hepatocyte nuclear factor-4α, liver X receptor, and sterol response element-binding proteins (SREBPs) [14]. SREBP-1c regulates hepatic lipogenic gene transcription and insulin-induced lipogenesis, and its target genes include fatty acid synthase (FAS), acetyl coenzyme A carboxylase (ACC), and the low-density lipoprotein receptor [15]. In the liver, a PUFA-enriched diet inhibits hepatic fatty acid synthesis by suppressing transcription of the SREBP-1c gene compared with a MUFA-enriched diet (using olive oil) [16–18]. In contrast, PPAR-α regulates genes that are involved in mitochondrial and peroxisomal fatty acid oxidation and transport [19]. Fatty acids are PPAR-α ligands and bind directly to the PPAR-α/9-cis-retinoic acid receptor dimer, and this complex in turn binds to PPAR response elements and activates a cascade of expressions of PPAR-α downstream genes, such as acyl coenzyme A oxidase (ACO) and carnitine palmitoyltransferase-1 (CPT-I). In addition, PUFAs have a higher affinity for PPAR response elements than SFAs or MUFAs [20].

Previous research has focused on the effects of various P/S ratios, ranging from 0.25 to 6.08, of dietary oils on lipid metabolism, especially on blood lipid profiles, in rats [21–24]. These ratios mostly lower than 3.0 were commonly discussed [22–24]. In addition, the use of a hamster model provides a distinct advantage over other rodent models because its lipid metabolism more closely resembles that of humans [25]. Hamsters also exhibit dietary obesity because of decreases in energy expenditure (diet-induced thermogenesis), not overeating, that resembles human obesity [26]. Therefore, it is important to determine whether changes in weight gain and fat accumulation after administration of cooking oil containing different P/S ratios and MUFA percentages is related to modifications in the expressions of SREBP-1c and PPAR-α mRNA. In this study, we compared the effects of two different P/S ratios (5 and 3) and two different MUFA percentages (60% and 30%) on the mechanism of fat accumulation by measuring hepatic enzyme activities and gene expressions.

Materials and methods

Animals and diets

In total, 32 male 7-wk-old golden Syrian hamsters, weighing 90 ~ 110 g, were purchased from the National Laboratory Animal Breeding and Research Center (Nan-Kang, Taipei, Taiwan). Hamsters were individually housed in wire-bottomed stainless steel cages and placed in an air-conditioned room (22°C, 65 ± 5% relative humidity) with a 12-h light, 12-h dark cycle, and free access to a basic diet (Rodent Laboratory Chow 5001, PMI, St. Louis, MO, USA) and water. After a 1-wk adaptation period, hamsters were randomly divided into four groups and each group was fed a different isocaloric high-fat diet for 12 wk. The experimental diets consisted of 150 g of experimental oil, 140 g of casein, 510.7 g of cornstarch, 100 g of sucrose, 50 g of cellulose as fiber, 10 g of the AIN-93 vitamin mixture, 35 g of the AIN-93 mineral mixture, 1.8 g of L-cystine, 2.5 g of choline-bitartrate, and 0.01 g/kg of tert-butyl-hydroquinone as an antioxidant according to the AIN-93 M formulation [27] and modification. All animals had free access to food and water. Food intake was measured daily, and body weight was measured weekly.

We designed a 2 × 2 factorial study, with two levels of MUFAs (60% and 30%) and two levels of the P/S ratio (5 and 3). The four kinds of experimental oils were abbreviated as HMHR, HMLR, LMHR, and LMLR. High-MUFA oils (HM) contained 60% MUFAs of total fatty acids with a high or low P/S ratio (HMHR and HMLR, respectively), and low-MUFA oils (LM) contained 30% MUFAs of total fatty acids with a high or low P/S ratio (LMHR and LMLR, respectively). In preparation of the experimental oils, cooking oils including high-oleic canola, olive, safflower, soybean, sunflower, and coconut were obtained from local supermarkets and analyzed by gas chromatography. Each fatty acid fraction was calculated three times with heptadecanoic acid (C17:0) as the internal standard. Fatty acids were converted to methyl esters with 14% boron trifluoride in methanol at 95°C for 30 min according to the method of Morisson and Smith [28] and then separated and quantified using a Stabilwax-DA capillary column (30 m × 0.53 mm inner diameter, film thickness 0.5 μm; RESTEK, Bellefonte, PA, USA) and a flame ionization detector on a G-3000 chromatograph (Hitachi, Tokyo, Japan). The HMHR oil was a combination of the high-oleic canola and olive oils; the HMLR oil was made up of safflower, soybean, and olive oils; and the LMHR and LMLR oils were composed of sunflower and coconut oils in different percentages. Fatty acid compositions of the experimental oils, analyzed by gas chromatography, are listed in Table 1. Each fatty acid level was expressed as a percentage of the total fatty acids (Table 1).

Experimental design

After 12 wk of the experimental period, hamsters were starved, and under inhalation anesthesia (diethyl ether), blood samples were collected by cardiac puncture and transferred to tubes containing ethylenediaminetetra-acetic acid. Plasma was immediately obtained after centrifugation (3500 rpm for 15 min at 4°C). Liver and white adipose tissues from epididymal and retroperitoneal locations were dissected, weighed, and quickly frozen in liquid nitrogen. All samples were stored at −80°C until being analyzed. All animal experimental procedures followed published guidelines [29] and were approved by the institutional animal care and use committee of Taipei Medical University (Taipei, Taiwan).

Plasma and hepatic lipid measurements

Plasma total cholesterol, triacylglycerol, high-density lipoprotein cholesterol, non-esterified fatty acid (NEFA), and glucose levels were determined by enzymatic colorimetric assays with commercial enzyme kits (Randox Laboratory,

Table 1
Fatty acid compositions (percentages) of experimental oil

	HMHR	LMHR	HMLR	LMLR
8:0	—	0.24 ± 0.02	—	0.56 ± 0.05
10:0	—	0.32 ± 0.01	—	0.72 ± 0.03
12:0	—	2.65 ± 0.03	—	6.03 ± 0.08
14:0	0.07 ± 0.02	1.14 ± 0.01	0.08 ± 0.00	2.54 ± 0.05
16:0	5.84 ± 0.02	7.42 ± 0.09	8.43 ± 0.15	7.76 ± 0.14
18:0	—	—	—	—
20:0	—	—	0.52 ± 0.06	—
ΣSFAs	5.90 ± 0.03	11.76 ± 0.11	9.03 ± 0.20	17.62 ± 0.17
16:1	0.15 ± 0.01	—	—	—
18:1	63.77 ± 0.22	28.53 ± 0.05	61.81 ± 0.19	27.18 ± 0.08
20:1	0.49 ± 0.42	0.57 ± 0.04	—	0.41 ± 0.01
ΣMUFAs	64.41 ± 0.22	29.10 ± 0.05	61.81 ± 0.19	27.60 ± 0.34
18:2	20.50 ± 0.20	58.45 ± 0.12	27.29 ± 0.16	54.10 ± 0.36
18:3	9.18 ± 0.07	0.69 ± 0.11	1.87 ± 0.06	0.68 ± 0.16
ΣPUFAs	29.68 ± 0.21	59.14 ± 0.08	29.16 ± 0.18	54.78 ± 0.31
P/S ratio	5.0	5.0	3.2	3.1

—, undetected; HMHR, high in monounsaturated fatty acids and a high ratio of polyunsaturated to saturated fatty acids; HMLR, high in monounsaturated fatty acids and a low ratio of polyunsaturated to saturated fatty acids; LMHR, low in monounsaturated fatty acids and a high ratio of polyunsaturated to saturated fatty acids; LMLR, low in monounsaturated fatty acids and a low ratio of polyunsaturated to saturated fatty acids; MUFAs, monounsaturated fatty acids; P/S ratio, ratio of polyunsaturated to saturated fatty acids; PUFA, polyunsaturated fatty acids; SFAs, saturated fatty acids
Data are presented as mean ± SD ($n = 3$).