Nutrition 32 (2016) 601–608

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Basic nutritional investigation

Effects of different amounts and types of dietary fatty acids on the body weight, fat accumulation, and lipid metabolism in hamsters

Ji-Hua Yang M.S. [a], Jung-Su Chang Ph.D. [a], Chi-Long Chen M.D., Ph.D. [b], Chiu-Li Yeh Ph.D. [c], Yi-Wen Chien Ph.D. [a,*]

[a] School of Nutrition and Health Sciences, Taipei Medical University, Taipei, Taiwan, Republic of China
[b] Department of Pathology, Taipei Medical University and Wan Fang Hospital, Taipei, Taiwan, Republic of China
[c] Department of Food and Nutrition, Chinese Culture University, Taipei, Taiwan, Republic of China

ARTICLE INFO

Article history:
Received 23 October 2014
Accepted 19 November 2015

Keywords:
Total fat consumption
Saturated fatty acids
Monounsaturated fatty acids
Polyunsaturated fatty acids
Body fat accumulation

ABSTRACT

Objectives: The aim of this study was to explore the effects of different amounts of dietary fatty acids on body weight, fat accumulation, and lipid metabolism of hamsters.
Methods: Sixty male golden Syrian hamsters were randomly divided into six groups. Three of the groups (the S groups) were fed experimental diets containing 5%, 15%, and 20% (w/w) fat of soybean oil (S_5, S_{15}, and S_{20}, respectively), and the other three groups (the M groups) were fed the same proportions of an experimental oil mixture (M_5, M_{15}, and M_{20}, respectively). The experimental oil mixture consisted of 60% monounsaturated fatty acids (MUFAs) and a polyunsaturated-to-saturated fatty acid ratio of 5 with a mixture of soybean and canola oils. Food consumption was measured daily, and body weights were measured weekly. Serum insulin and leptin concentrations were measured and hepatic fatty acid metabolic enzymes and adipose differentiation markers were determined using an enzyme activity analysis and quantitative polymerase chain reaction.
Results: Results showed that the weight and weight gain of the S_{20} group were significantly greater than those of the other five groups. When the total fat consumption increased, the body weight, weight gain, and adipose tissue weight of the S groups significantly increased, but there were no significant differences in these parameters among the M groups. Serum low-density lipoprotein cholesterol concentrations were significantly lower in the M_{15} and S_{15} groups. The S_{20} group had significantly higher leptin and insulin concentrations and lipoprotein lipase was promoted, but the acetyl-coenzyme A oxidase and carnitine palmitoyltransferase-1, were significantly lower.
Conclusions: The study demonstrated that a special experimental oil mixture (with 60% MUFAs and a ratio of 5) with high fat can prevent body weight gain and body fat accumulation by lowering insulin concentrations and increasing hepatic lipolytic enzyme activities.

Introduction

Obesity is a condition of fat overaccumulation and is recognized as a major cause of metabolic syndrome. Obesity has been linked to the development of hypertension, hyperlipidemia, stroke, fatty liver, and diabetes mellitus [1]. It is generally believed that an imbalance between energy intake and energy expenditure is the main reason for overweight and obesity outcomes. Dietary and lifestyle patterns, as well

This project was supported by grant NSC99- 2320-B-038- 012-MY3. None of the results, data, or figures in this study has been published elsewhere, nor are they under consideration (from any of the contributing authors) by another publisher. J-HY was responsible for data collection and analysis. J-HY and Y-WC were responsible for the study design. All of the authors have read and approved the final submitted manuscript. None of the authors have any conflicts of interest to declare and they declare all sources of funding for research in this manuscript. The authors acknowledge Dr. Shih-Yi Huang for assistance with the Stabilwax-DA capillary column and G-3000 chromatography and Dr. Sung-Ling Yeh for assistance with the Veriti 96-well Thermal Cycler.
* Corresponding author. Tel.: +886 2 273 61661; fax: +886 2 273 73112.
E-mail address: ychien@tmu.edu.tw (Y.-W. Chien).

http://dx.doi.org/10.1016/j.nut.2015.11.010
0899-9007/

as several genes, are closely related to this development [2,3]. Dietary programs that reduce obesity through decreased energy intake from fat may be an effective method for improving complications caused by obesity [2]. High-fat diets (HFDs) are known to lead to a positive fat balance and consequently to adipose mass accumulation [4,5] because excess fat in the everyday diet is stored and contributes to increases in the adipose tissue mass [6]. Due to different effects of fat intake on health, the following recommendations were established. Intake of total fat is recommended to be 15% to 35% of energy, saturated fatty acids (SFAs) <10% of energy, trans-fatty acids <1% of energy, monounsaturated fatty acids (MUFAs) 10% to 15% of energy, and polyunsaturated fatty acids (PUFAs) 6% to 10% of energy [6]. Although several studies suggested that energy from fat rather than fat per se leads to weight increases, this remains controversial. For example, results from one study demonstrated that HFDs are highly energy dense and therefore contribute to weight gain [7], whereas another study indicated there is little association between fat intake and changes in weight [8], a conclusion reported in an 8-y follow-up investigation of the Nurses' Health Study [9].

Some studies proposed that the dietary fat composition, in addition to the amount of fat or energy taken in, can affect the development of obesity. Different types of fatty acids have different metabolic behaviors such as differences in oxidation and deposition rates that may contribute to weight change [10]. When comparing dietary MUFA intake with that of SFAs in humans, results showed a decrease in fat deposition with a diet rich in MUFAs [11]. Human studies showed that a diet with a high ratio of PUFAs to SFAs (P/S) could increase postprandial fat oxidation [12,13]. A previous study showed that oil with a high P/S ratio was important in lowering body fat accumulation, and high-MUFA oil with a high P/S ratio (HMHR; consisting of 60% MUFAs of the total fatty acids with a ratio of 5) may prevent HFD-induced increases in body weight and body fat [14]. Thus, not only the quantity of ingested fats, but also the composition of fatty acids is of pivotal importance for human health. Most European countries report high fat intakes (>35% energy); the intake of SFAs is especially high (>10% energy) [15]. Thus, it is important to define the effects of different nutritional patterns of dietary lipids (quantity and quality) on body weight, fat accumulation, and lipid metabolism.

Adipose tissue is a complex metabolic organ that is involved in regulating lipid and carbohydrate metabolism, and is an endocrine organ involved in the secretion of leptin and adiponectin [16]. Moreover, peroxisome proliferator-activated receptors (PPARs) are ligand-dependent transcription factors belonging to the nuclear receptor superfamily, and there are three PPAR isoforms: PPARα, PPARγ, and PPARβ/δ [17]. PPARγ is highly expressed in white adipose tissue, and its activation plays a key role in adipocyte differentiation and development [18]. PPARγ activation mediates expressions of several target genes implicated in adipose tissue accumulation, such as lipoprotein lipase (LPL), hormone-sensitive lipase (HSL), leptin, and adiponectin [19]. LPL is located on the surface of endothelial cells and is an enzyme responsible for the hydrolysis of blood triacylglycerol (TG)-derived lipoproteins, including chylomicrons and very low-density lipoproteins [20], hydrolyzed fatty acids that directly enter peripheral tissues and white adipose tissue. Animal experiments showed that an elevation of adipose tissue LPL activity is accompanied by increasing adipose tissue TG uptake [21]. In contrast, HSL is a key enzyme catalyzing the lipolysis of TG in adipose tissue, and its high activity attenuates TG accumulation in adipocytes [22]. Additionally, the use of a hamster model provides a distinct advantage over other rodent models because the hamster lipid metabolism more closely resembles that of humans [23]. Hamsters also exhibit dietary obesity because of decreases in energy expenditure (diet-induced thermogenesis), not overeating, that resembles human obesity. In this study, we compared the effects of two different qualities (soybean oil and an oil mixture) at three different quantities (5%, 15%, and 20% w/w) of dietary fat intake on the mechanism of fat accumulation by measuring enzyme activities and gene expressions.

Materials and methods

Animals and experimental design

In all, 60 male 7-wk-old golden Syrian hamsters were purchased from the National Laboratory Animal Breeding and Research Center (Taipei, Taiwan). Hamsters were communally housed in plastic cages (five per cage) and placed in an air-conditioned room (at 22°C ± 2°C with a 65% ± 5% relative humidity) with a 12-h light/dark cycle. For a 1-wk adaptation period, hamsters were fed water and a basic diet (Rodent Laboratory Chow 5001; PMI Nutrition International, St Louis, MO, USA).

After the 1-wk acclimation period, hamsters were randomly divided into six groups (each group n = 10). Three of the groups (the S groups) were fed experimental diets containing 5%, 15%, and 20% (w/w) fat from soybean oil (S_5, S_{15}, and S_{20} groups, respectively), and the other three groups (the M groups) were fed the same proportions of an experimental oil mixture (M_5, M_{15}, and M_{20} groups, respectively). The diet consisted of the AIN-93 M formulation with modifications [24], and the experimental oil mixture consisted of 60% MUFAs and a P/S ratio of 5 of a mixture of soybean and canola oils. All animals had free access to distilled water and the isocaloric diets for 12 wk. Food intake was measured daily, and body weights were measured weekly. After 12 wk of the experimental period, hamsters were starved for 12 h, and under inhalation anesthesia (diethyl ether), blood samples were collected by cardiac puncture and transferred into tubes. After immediate centrifugation (3500*g* for 20 min at 4°C), serum was obtained. The liver, spleen, kidneys, and white adipose tissue from epididymal and retroperitoneal locations were dissected and weighed. Liver and white adipose tissues from epididymal and retroperitoneal areas were quickly frozen in liquid nitrogen. All samples were stored at −80°C until analyzed. All animal experimental procedures followed published guidelines approved by the institutional animal care and use committee of Taipei Medical University (Taipei, Taiwan).

Fatty acid composition analysis

High-oleic canola oil and soybean oil were obtained from local supermarkets and analyzed using 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 a previously described method [25], and then separated and quantified using a Stabilwax-DA capillary column (30 m × 0.53-mm inner diameter, film thickness of 0.5 mm; RESTEK, Bellefonte, PA, USA) and a flame ionization detector on a G-3000 chromatograph (Hitachi, Tokyo, Japan). Fatty acid compositions of the experimental oils, were analyzed by gas chromatography. Each fatty acid level was expressed as a percentage of the total fatty acids.

Serum measurements

Using a spectrophotometer, serum total cholesterol (TC), TG, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), nonesterified fatty acids, glutamate oxaloacetate transaminase (GOT), glutamate pyruvate transaminase (GPT), and glucose levels were determined by enzymatic colorimetric assays using commercial enzyme kits (Randox Laboratory, Crumlin, Northland, UK). Serum insulin concentrations were determined using commercially available enzyme immunoassay enzyme-linked immunosorbent assay (ELISA) kits (Mercodia AB, Uppsala, Sweden); serum leptin concentrations were measured using a hamster leptin immunoassay (Blue Gene Biotech, Shanghai, China); and serum adiponectin concentrations were measured with a hamster adiponectin ELISA kit (Blue Gene Biotech) using a VERSAmax microplate reader (Molecular Devices, Sunnyvale, CA,