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A highly saturated fat-rich diet is more obesogenic than diets with lower saturated fat content

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Abstract

The present study tested the hypothesis that a saturated fatty acid (SFA)–rich diet is more obesogenic than diets with lower SFA content. In 8 female Sprague-Dawley rats fed a low-SFA canola or a moderate-SFA lard-rich diets at 67% of energy for 26 days, body weight gain, final body weight, obesity index, and food and energy intake were comparable. Twenty-nine rats were fed canola or high-SFA butter-rich diets (67% of energy) or chow for 50 days; then high-fat feeding was followed by ad libitum low-fat feeding (27% of energy) for 28 days and by a food-restricted low-fat diet for 32 days. High-fat feeding resulted in a greater body weight gain ($P < .04$), final body weight ($P < .04$), and energy intake ($P < .008$) in butter-fed rats than in canola- and chow-fed controls, after 26 or 50 days. Ad libitum canola and butter low-fat diets or chow feeding resulted in similar weight change, whereas food-restricted low-fat diets led to comparable weight loss and final weight. Canola-fed animals adjusted their intake based on diet energy density, whereas lard and butter-fed animals failed to do so. Abdominal fat ($P = .012$) and plasma leptin ($P = .005$) were higher in chow-fed controls than in canola-fed rats, but comparable with those of butter-fed rats. Prone and resistant phenotypes were detected with high-fat feeding. In conclusion, only feeding the high-SFA butter-rich diet led to obesity development and failure to adjust intake based on the energy density and preserving body fat even after weight loss. The high availability of SFA-rich foods in today's obesogenic environment could contribute to develop and maintain obesity.

Keywords: Obesity; Dietary fats; Fatty acids; Weight loss diet; Abdominal fat; Leptin; Rat
Abbreviations: ANCOVA, analysis of covariance; ANOVA, analysis of variance; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids.

1. Introduction

Obesity is a major health problem with an increasing prevalence throughout the world [1]. Excessive energy intake and reduced physical activity are important variables in the development of obesity. Dietary patterns, mainly those favoring fat intake, often have been blamed for the increase in adiposity [2,3]. High-fat diets have been used to induce obesity in animals in a model first called nutritional obesity, but later renamed as dietary obesity (reviewed in Hariri and Thibault [4]). Animal models of genetic obesity have identified single-gene mutations involved in the control of energy homeostasis, although inherited human genetic predisposition involves multiple genes [5,6]. The paradigm of dietary obesity in animal is an appropriate model for studying human obesity in the context of an environment where energy-dense foods and diets are highly available.

Fatty acid composition of dietary fat may play an important role in body weight regulation. Studies in animals and in humans have shown that polyunsaturated fatty acids (PUFAs) and monounsaturated fatty acids (MUFAs) are

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more readily used for fuel, whereas saturated fatty acids (SFAs) are more likely to be accumulated in adipose tissue [7-9]. Experimental demonstration of this principle remains inconclusive. No constant pattern of outcome was found in rats fed high-fat diets with different saturation of fatty acids [10-18]. However, most of the available data used male animals fed diets containing moderate (beef tallow, lard, or palm oil) or low (olive oil, safflower oil, or corn oil) concentrations of SFA (reviewed in Hariri and Thibault [4]).

Dietary obesity was reversed by using diets low in fat fed ad libitum [19-23], whereas in other studies, feeding low-fat diets resulted in a plateau in body weight [24-26]. Besides, it was shown that feeding responses differ when animals were fed diets of higher or lower energy density. For example, animals adjusted intake when switched to a diet of higher energy density by decreasing food intake and maintaining energy intake [25], or failed to adjust and reduced energy intake when switched to a lower energy-dense diet [20,21].

Leptin and ghrelin are important hormones in the control of food intake and body weight. Leptin is an obese gene product produced by adipose tissue, and rises in plasma levels result in a decrease in food intake and increase in energy expenditure [27]. Ghrelin is a peptide released by cells in the fundus of the stomach, which rises before and falls after each ad libitum meal and increases food intake [28].

Although there has been extensive research on dietary obesity, more work is needed to prove that fatty acid profile in high-fat diets can regulate body weight. Thus, we conducted the present study to test the hypothesis that an SFA-rich diet is more obesogenic than diets with lower SFA content. The main objectives were to (1) examine the effect of high-fat diets containing high-, moderate-, and low-SFA content on food intake and body weight gain; (2) test reversal of obesity with low-fat diets; (3) analyze the feeding response when animals are switched to diets of higher or lower energy density; and (4) measure abdominal fat, plasma leptin, and active and total ghrelin levels after weight loss. We used genetically similar adult female Sprague-Dawley rats fed low-SFA canola oil (7%), moderate-SFA lard (43%), and high-SFA butter (68%) high-fat diets at 67% of energy to test obesity development, and then canola and butter in low-fat diets at 27% of energy to test obesity reversal. Female rats' body weight becomes stable in early adulthood, whereas male rats continue to gain weight throughout their lives [29]. Human subjects of both sexes have a growth pattern more like that of female rats. The estrous cycle of female animals synchronizes when they live together [30]. Total body weight gain was also used to categorize animals as obesity prone, resistant, and intermediate. Lee obesity index [31] was measured, as reliable correlations were found between this index and body fat [32,33].

By using the animal model of dietary obesity that shares many features with human obesity [34], these experiments will provide much important data on how dietary fatty acid profile affects the development and treatment of obesity.

2. Methods and materials

2.1. Animals and diets

Adult female Sprague-Dawley rats weighing 250 to 310 g (13 weeks of age) obtained from Charles River Laboratories (St-Constant, Quebec) were used for these experiments. They were housed in individual cages, under controlled temperature (22-25°C) and humidity (70%) and with a 12:12

Table 1
Diet composition (g/100 g)

	Purina chow [a]	High-fat diets			Low-fat diets	
		Canola [b]	Lard [c]	Butter [d]	Canola [b]	Butter [d]
Total protein	18.1	10.6	10.6	10.6	16	16
Total carbohydrate	57.3	33.7	33.7	33.7	51	51
Total fat:	4.5 [e]	39.8	39.8	39.8	11	11
from chow	4.5	2.6	2.6	2.6	4	4
from other sources	–	37.2	37.2	37.2	7	7
SFA	–	2.6	16	25.3	0.5	4.8
MUFA	–	22.7	17.5	10.4	4.3	2
PUFA	–	11.9	3.7	1.5	2.3	0.3
Fiber	3.4	2	2	2	3	3
Vitamins and minerals	3.7	2.2	2.2	2.2	3.3	3.3
Pectin [f]	–	4	4	4	4	4
Percent of energy from fat	12	67	67	67	27	27
Energy (kJ/g)	14.2	22.2	22.2	22.2	15.1	15.1

[a] Charles River rodent chow 5075, St-Constant, Quebec.
[b] President's Choice, Canada.
[c] Tenderflake, Maple Leaf, Canada.
[d] My Country, Lactantia, Canada.
[e] Provided by fish meal, beef tallow, soybean meal, corn, and wheat.
[f] MP Biomedicals, Inc, USA.