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Fatty diets retarded the propulsive function of and attenuated motility in the gastrointestinal tract of rats

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ARTICLE INFO

Article history:
Received 24 July 2012
Revised 17 December 2012
Accepted 28 December 2012

Keywords:
Fatty diet
Propulsive function
Gastroduodenal transit
Dyspepsia
Constipation
Rat

ABSTRACT

Digestive functions are considered to be alterable by the ingestion of fatty diets. This study aimed to investigate the hypothesis that dietary fats may exert site-specific effects on the propulsive functions of the gastrointestinal (GI) tract. After male Wistar rats were fed either low-fat diet or high-fat diet (HFD) for 8 weeks, the propulsive function of the luminal contents of the entire GI tract was simultaneously examined in vivo. In comparison with a low-fat diet, an HFD significantly increased the body weight gains but significantly decreased the diet and caloric intakes, fecal weights, and fecal pellet numbers. Gastric emptying in the HFD-fed rats tended to be delayed, but this was not significant. High-fat diet feeding significantly slowed the small bowel transit times, and the luminal residuals emptied from the gastric antrum were largely accumulated in the proximal parts of the small intestine. An HFD also significantly prolonged the colonic transit times. In conclusion, fatty diets retarded the propulsive function of the entire GI tract, and the delayed gastroduodenal transit of fatty diets may act as a primary causal factor for producing the attenuated motile function of the GI tract in rats.

1. Introduction

The gastrointestinal (GI) tract routinely encounters macronutrients such as carbohydrates, lipids, and proteins, and the nutrient sensing of GI tracts is highly sensitive to changes in the macronutrient composition of meals. Termination of meal ingestion by satiation is mediated by feedback signals arising from the GI tract, and these mechanisms are likely modulated by the composition of the macronutrients contained in meals [1-3]. Accumulating evidence has shown that the ability of the GI tract to sense dietary fats affects gut functions, including motility and secretion [4,5], and chronic ingestion of fatty diets changes the sensitivity of satiation signals and luminal transit rates of GI contents [2,6,7]. Therefore, dietary fats are strongly considered to be potential factors that contribute to dysregulation of appetite through modulation of the motility of GI tracts.

It has been suggested that, in rats, ingestion of fatty diets leads to hyperphagia and obesity, which are associated with impairment of fat-induced activation of vagal afferent pathways [8,9], and also slows the gastric emptying rates [5]. However, these suggestions are not necessarily consistent

Abbreviations: GI, gastrointestinal; LFD, low-fat diet; HFD, high-fat diet; PBS, phosphate-buffered saline; i.p., intra-peritoneum; T-C, total cholesterol; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; TG, triglyceride; ANOVA, analysis of variance; FFAs, free fatty acids.

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http://dx.doi.org/10.1016/j.nutres.2012.12.008

with current knowledge, which indicates that delayed gastric emptying produces a reduction of appetite. Moreover, different phenomena have been reported in human studies, suggesting consumption of fatty meals causes an increase of gastric emptying [2,10] or no change of upper gut motility [11] in response to dietary fats. Obese individuals have demonstrated more complex phenomena with different studies, suggesting that their gastric emptying rates are similar [12], faster [13], or slower [14] compared with lean individuals. In addition, several studies have reported the mouth-to-cecum transit times of fatty diets. Normal subjects have similar rates of gut transit times with low-fat diets (LFDs) [15], whereas obese patients are found to have shorter transit times following either low- or high- fat meals [16]. These findings may indicate that fatty diets modulate GI motility differently, dependent on the physical status of the subjects and the region of the GI tract under study.

Although previous research has dramatically expanded our understanding of the participation of dietary fats in modulation of GI motility, there are still numerous questions to be answered. Moreover, previous results have demonstrated that it is difficult to completely understand the motile dysfunction occurring in the entire GI tract, the effects of dietary fats on specific regions of the GI tract, in response to ingestion of fatty diets. Based on these considerations, we hypothesized that dietary fats may exert site-specific effects on the propulsive functions of GI tracts, and a specific part of the gut may act as a primary causal factor in producing retarded propulsive function of the entire GI tract. To test our hypothesis, we simultaneously examined gastric emptying, small bowel transit, and defecatory function in rats fed high-fat diets.

2. Methods and materials

2.1. Animals

Male Wistar rats (7 weeks old) were purchased from the SamTako BioKorea (Kyungki, Korea) and acclimatized under standard conditions for laboratory animals: a temperature of 23°C ± 2°C, a relative humidity of 50% to 60%, and a 12-hour dark-light cycle. Rats were housed in polypropylene cages and allowed free access to a solid rodent diet (Samyang Co, Kyungki, Korea) and drinking water. All experimental protocols were approved by the Institutional Animal Care and Use Committee at Wonkwang University.

2.2. Experimental designs and diets

After acclimatization for 1 week, rats were randomly divided into 2 groups (24 animals per group) and fed either an LFD (pellet type, Research Diets 12450B, New Brunswick, NJ, USA) or high-fat diet (HFD) (pellet type, Research Diets 12451, New Brunswick, NJ, USA) for 8 weeks. The diets provided 3.85 kcal/g of energy for the LFD (70% carbohydrate, 20% protein, and 10% fat) and 4.73 kcal/g of energy for the HFD (35% carbohydrate, 20% protein, and 45% fat). The ingredient composition of the diets is shown in Table. Food intake, body weight, and fecal output were measured weekly for the duration of the experiment.

Table – Ingredient composition of the diets fed to rats for 8 weeks

Ingredient (g/kg diet)	Diets [a,b]	
	Low fat	High fat
Casein, 80 mesh	200.0	200.0
L-Cystine	3.0	3.0
Cornstarch	315.0	72.8
Maltodextrin 10	35.0	100.0
Sucrose	350.0	172.8
Cellulose, BW200	50.0	50.0
Soybean oil	25.0	25.0
Lard	20.0	177.5
Mineral mix, S10026	10.0	10.0
Dicalcium phosphate	13.0	13.0
Calcium carbonate	5.5	5.5
Potassium citrate	16.5	16.5
Vitamin mix, V10001	10.0	10.0
Choline bitartrate	2.0	2.0

[a] Semipurified diets were supplied by Research Diets, Inc.
[b] Protein (% energy): low fat, 19.2; high fat, 24.0; carbohydrate (% energy): low fat, 67.3; high fat, 41.0; Fat (% energy): low fat, 4.3; high fat, 24.0; Total kcal/g: low fat, 3.85; high fat, 4.73.

2.3. Measurement of gastric emptying

Gastric emptying was measured according to a method described previously with a slight modification [5]. In brief, at 8 weeks after feeding with either an LFD or HFD, rats were starved overnight but received water ad libitum and were orally administered 1 mL of a test meal that contained 0.05% (wt/vol) phenol red in aqueous carboxymethyl cellulose (4.5%, wt/vol). The animals were euthanized by CO_2 asphyxiation and cervical dislocation 10 minutes after administration of the test meals, and the abdomen was quickly opened, followed by clamping of both the gastroesophageal and gastroduodenal junctions. The stomachs were removed from the abdominal cavity and rinsed with phosphate-buffered saline (pH 7.4). To elute the phenol red, the stomachs were incised in 20 mL of 0.1 N NaOH and vigorously shaken for 30 minutes, and the supernatants were recovered by centrifugation at 3000*g* for 10 minutes. To precipitate the proteins, the supernatants (0.5 mL) were combined with 50 μL of 20% trichloroacetic acid and vigorously mixed. The samples were centrifuged at 10000*g* for 30 minutes, and 0.5 N NaOH (0.4 mL) was added into 0.3 mL of supernatant to develop the color. The absorbance of samples at 558-nm wavelength was measured with a spectrophotometer (U-2000; Hitachi, Tokyo, Japan). Gastric emptying was estimated using the following formulation: (amount of residual phenol red/phenol red present in stomach immediately after administration) × 100.

2.4. Measurement of small bowel transit

The small bowel transit was measured according to a method described previously with a slight modification [5]. In brief, at 8 weeks after feeding with either an LFD or HFD, rats were starved overnight but allowed drinking water ad libitum and orally administered 1 mL of a 0.05% (wt/vol) phenol red-containing test meal. The animals were euthanized by CO_2