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Original Article

Comparative Effects of High Carbohydrate and Fat Diets on Brush Border Membrane Enzymes, Energy Metabolism and Antioxidant Defense Mechanisms in Rat Intestine and Liver

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ABSTRACT

Digestion, absorption and metabolism of foods are major functions of intestines as well as liver. Variations in major food components such as carbohydrates, fats and proteins in the diet has been shown to produce negative effects on human health, leading to several diseases. Effects of high carbohydrate diet and high fat diet on various serum parameters and enzymes of brush border membrane, carbohydrate metabolism and oxidative stress in small intestines and livers of rats were assessed. Rats were fed with high carbohydrate diets and/or high fat diets for 28 d. Serum glucose, cholesterol, inorganic phosphate (Pi) and serum alkaline phosphatase increased differentially by high carbohydrate diets and high fat diets. The activity of brush border membrane enzymes, alkaline phosphatase and sucrose, increased; however, γ-glutamyl transferase decreased by high carbohydrate diet whereas alkaline phosphatase and γ-glutamyl transferase increased; however, sucrase decreased by high fat diet. The activities of metabolic enzymes except lactate dehydrogenase, which was profoundly increased by high carbohydrate diet., significantly decreased by high carbohydrate diet and high fat diet in small intestine. The activity of lactate, malate, glucose-6-phosphate dehydrogenases and NADP-malic enzyme significantly increased; however, glucose-6-phosphatase and fructose-6-phosphatase decreased in the liver by high carbohydrate diets. However, these decreased by high fat diets. The two diets increased oxidative stress as apparent by increased lipid peroxidation with decreased activity of superoxide dismutase and catalase in mucosal and liver homogenates. In conclusion, consumption of high carbohydrate and fat diets caused extensive alterations in mucosal brush border membrane and liver, disrupted metabolic activity and antioxidant defence mechanism differentially as indicated by the changes in associated enzymes and other parameters.

Keywords: High carbohydrate diets, High fat diets, Intestine, Liver, Brush border membrane, Carbohydrate metabolism, Oxidative stress

Highlights

- Feeding of high carbohydrate diets and high fat diets differentially increased serum glucose, cholesterol, phospholipids and inorganic phosphate; however, serum creatinine was not affected by the two diets.
- High carbohydrate diets increased brush border membrane enzymes, alkaline phosphatase and sucrose, but decreased γ-glutamyl transferase activity. High fat diets increased alkaline phosphatase and γ-glutamyl transferase but decreased sucrase activity.
- High carbohydrate diets induced energy generation majorly by anaerobic glycolysis in the intestine but in the liver by oxidative metabolism as indicated by the enzymes involved.
- High fat diets decreased all the enzymes involved in glucose metabolism and its production in the intestine and liver.
- High carbohydrate diets and high fat diets caused oxidative stress in the intestine and liver as evident by increased lipid peroxidation and suppressed activities of superoxide dismutase and catalase.

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Introduction

Diet and nutrition play important roles in preservation of health; however, the relationship between diet and health is greatly controversal. Deficiencies, excesses and imbalances in the diet produce negative effects on human health that lead to several diseases such as obesity, diabetes, cardiovascular diseases (CVDs) and cancers. (1–4). Lack of awareness, socioeconomic status, cultural and religious restrictions and life style changes are responsible for dietary imbalances and health problems (5). In the current busy life, people are choosing fast street foods (e.g. pizzas and burgers) and fried Indian foods (e.g. puri-kachori, pakora, samosa), which include higher caloric densities but lesser nutrition values. Extreme dieters of urban societies, who wants to loose weight, are attracted to popular diets such as Atkins, Ornish and Mediterian diets with potential health risks (6–11).

The small intestine is the major primary site, where complex foods are digested to useful nutrients, absorbed and metabolized. Intestinal brush border membrane (BBM) that lines the epithelium contains certain hydrolytic enzymes [e.g. alkaline phosphatase (ALkPase), γ-glutamyl transferase (GGTase), maltase and sucrose] and transporters that are involved in end-stage digestion and absorption of nutrients (12–15). Liver is involved directly or indirectly in the intestine functions. This is the major site of oxidative metabolism of food components, including carbohydrates, fats and proteins. Nutritional stress (e.g. fasting, Ramadan fasting, restricted energy intake and dietary imbalances) and certain environmental factors have been shown to dramatically altered structures and functions of small intestine and liver (14–20).

High carbohydrate diets (HCD) containing high quantities of starch, sucrose, lactose and/or fructose increase the activity of disaccharidases such as sucrase, maltase and lactase as well as alkaline phosphatase in the intestinal BBM (21–24). The increase in the activities is due to increased synthesis of novel enzyme molecules (23, 25). In contrast, high fat diets (HFD) containing saturated fat such as lard, coconut oil and/or corn oil mixed with cholesterol cause damaging effects on intestine, liver and kidney (4, 26–28). The HFD decreases the activity of disaccharidases but increases ALkPase in small intestine (22, 28, 29) with hyperlipidaemia and deposition of triacylglycerol resulting in obesity (30). Moreover, HFD alone or in combination with ethanol causes severe liver injury and triggers oxidative stress (26, 31).

Much information on dietary regulation of small intestine disaccharidases, peptidases and hydrolases and metabolism have been reported (22). However, effects of HCD and HFD on the biochemical events and the mechanism involved in the cellular response to these diets are not completely revealed, neither those participating in inflammation and oxidative stress nor those involving in energy yielding metabolic activities in the small intestine and liver. Therefore, the aim of the present study was to assess the effects of HCD and HFD on various serum parameters and enzymes of carbohydrate metabolism (e.g. enzymes of glycolysis, TCA cycle, gluconeogenesis and HMP shunt pathways), BBM and oxidative stress parameters in rat intestine and liver.

Results indicated that HCD and HFD increased serum glucose, cholesterol, Pi and the activity of ALkPase. The BBM enzymes of ALkPase and sucrase increased; however, GGTase decreased by HCD. Furthermore, HFD increased ALkPase and GGTase while decreased sucrase activity. The HCD increased enzymes of glucose degradation but decreased enzymes of gluconeogenesis in intestine and liver. However, HFD decreased all the enzymes involved in glucose metabolism and production in the intestine and liver. Lipid peroxidation (LPO), indicator of oxidative stress increased; however, the activity of antioxidant enzymes, superoxide dismutase (SOD) and catalase, decreased by the two diets. Results of the present study suggest that HCD and HFD cause extensive alterations in intestinal BBM and liver, disrupt metabolic activity and antioxidant defence mechanism differentially as indicated by the changes in associated enzymes and parameters.

Materials and Methods

Chemicals

Sucrose, *p*-nitro phenyl phosphate, NADH and $NADP^+$ were purchased from Sigma, St Louis, MO, USA. All other chemicals included analytical grades and were purchased from Sigma, St Louis, MO, USA, or Sisco Research Laboratory, Mumbai, India.

Animals

Adult albino rats (Wistar strain) were purchased from All India Institute of Medical Sciences, New Delhi, India.

Diets

Normal Control Diets

The standard rat pellet diet was purchased from Amrut, Maharashtra, India

Preparation of High Carbohydrate Diets

The following ingredients were mixed with powdered normal control diets (NCD) to form high carbohydrate diets (HCD) as described by Wolffram and Scharrer (32): casein, 13%; carbohydrate, 77% (starch, 55% and sugar, 22%); corn oil, 4%; mineral mixture, 5%; and vitamin mixture, 2%.

Preparation of High Fat Diets

High fat diets (HFD) were prepared by mixing powdered rat NCD with 2% cholesterol dispersed in 6% heated corn oil (180 ^oC for 15 min) as described by Kritchevsky et al. (33) and modified by Yusufi et al. (4).

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Experimental Design

The experiments were carried out on male Wistar rats based on the guidelines approved by the institutional ethical committee and Ministry of Environment and Forests (CPCSEA), Government of India. The rats (ten rats per group), weighing 150–200 g were conditioned for 1 w in the animal facility and fed NCD and water *ad libitum*. Three groups of rats (16–20 rats per group in each experiment) were used in the study. Then, rats were fed with a diet either rich in carbohydrates (HCD) or fats (HFD) for 28 d. One group of rats received NCD and was used as control. The body weights of rats were recorded at the begining and completion of the experimens. Blood samples were collected and the intestine and liver extracted and processed for the preparation of homogenates and brush border membrane vesicles (BBMV) as described later. All the preparations and analyses of various parameters were carried out simultaneously under similar experimental conditions to avoid day-to-day variations.

Preparation of Homogenates and Brush Border Membranes Vesicles

After the completion of the experiment, rat intestines were extracted. The intestines were washed by flushing them with ice-cold buffered saline (1m M Tris-HCl and 9 g/l NaCl, pH 7.4). The livers were transferred into tris buffered saline (TBS) as described by Farooq et al. (16) and Khan et al. (34). The BBMV was prepared as described by Farooq et al. (14) using $CaCl_2$ precipitation and differential centrifugation technique. Mucosa scraped from 4–5 washed intestines was used for each BBMV preparation. Briefly, the mucosal scrapings were collected using beaker containing 50 mM mannitol and 5 mM tris-HCl, pH 7.5. The mucosal homogenate was diluted with the tris-mannitol buffer (15 ml/g tissue) and further homogenized using Ultra-Turrex T25 homogenizer (Janke & Kunkel GmbH & Co. KG, Staufen, Germany) with three pulses of 30 s each with 30-s intervals between pulses. Aliquots of mucosal homogenate were saved and quickly frozen for further analyses. Moreover, $CaCl_2$ was added to the filtrate to a final concentration of 10 mM and was set on ice for 20 min with intermittent stirring.

The homogenate was then centrifuged at 2000 g for 10 min using Beckman J2-M1 refrigerated centrifuge (Beckman Instruments, Palo Alto, CA, USA) and JA-17 rotor. The pellet was discarded and the supernatant was recentrifuged at 35000 g for 30 min. The pellet was resuspended in a small volume (1–2 ml) of 50 mM sodium maleate buffer, pH 6.8, with four complete passes using loose-fitting Dounce homogenizer, Wheaton, USA, and centrifuged at 35,000 g for 30 min using 15 ml of Corex glass tube and JA-20 rotor. The white outer fluffy portion of the pellet was resuspended carefully in a small volume of the highlighted buffer. The BBM suspension was quickly frozen in small aliquots and used for enzyme analysis. All the steps were strictly carried out at 0–4 ºC unless specified. A 10% liver homogenate was similarly prepared in 10 mM tris-HCl buffer, pH 7.5. The homogenates were centrifuged at 2000 g for 10 min at 4 ˚C to remove cell debris and the supernatant was aliquoted and stored at -20 ˚C for further assaying the enzymes of carbohydrate metabolism, free radical scavenging enzymes and estimation of total SH and LPO as previously described (16, 34).

Serum Chemistries

Serum samples were deproteinated with 3% trichloroacetic acid in a ratio of 1:3, set for 10 min and centrifuged at 2000 g for 10 min. The protein-free supernatant was used to assess inorganic phosphate and creatinine. Blood urea nitrogen (BUN) and cholesterol levels were assessed directly in serum samples. Glucose was estimated using o-toluidine method and commercial kits from Span Diagnostics, Mumbai, India. These parameters were assessed using standard procedures as stated in a previous study (35).

Enzyme assays

The activities of BBM biomarkers enzymes, ALkPase, GGTase and sucrase in the homogenates and BBM preparations were assessed as described previously (15). The enzymes of carbohydrate metabolism such as lactate (LDH), malate (MDH), glucose-6-phosphate (G6PDH) dehydrogenases and NADP-malic enzyme (ME) involved in oxidation of NADH or reduction of NADP were assessed by measuring the extinction changes at 340 nm using spectrophotometer (Cintra 5; GBC Scientific Equipment, Victoria Australia) as described elsewhere (14, 35). The other enzymes, glucose-6-phosphatase (G6Pase) and fructose-1,6-bisphospatase (FBPase) were assessed as described in previous studies (35). The activities of SOD, catalase and glutathione peroxidase (GSH-Px) were assessed as described by Priyamvada et al. (2010). The LPO and total SH-groups were estimated as described previously (36). Protein concentration was assessed using method of Lowry et al. (37) modified by Yusufi et al. (38).

Statistical analyses

All data were expressed as mean ±SE (standard error) for at least 4–5 various preparations. Independent sample t-test and one-way ANOVA test were used to analyze differences in means. Level of significance was set at 5%. Most of the changes between various groups were compared with control values for better understanding and clarity. All statistical analysis were carried out using SPSS v.20 software (IBM, USA).

Results

The present study was carried out to investigate the effects of HCD and HFD on body weights, serum parameters and enzymes of carbohydrate metabolism, intestinal BBM and oxidative stress parameters in rat intestine and liver. In general, rats were active and alert throughout the study.

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Body Weight and Weight of Mucosa

The effect of high carbohydrate (HCD) and high fat (HFD) diets was observed on the body and intestinal mucosa weight of rats. As shown in Table 1, feeding HCD for 28 days, caused marked increase (+24.5%) whereas that of HFD caused a significant decrease (-28%) in the body weighs of the rats compared to control (NCD-fed) rats. The weight of intestinal mucosa, however, did not change significantly.

Table 1. Effects of high carbohydrate diets and high fat diets on body weights and intestinal mucosa weights of the rats

Groups	Body weight (g)	Mucosa weight (g)
Control	233.39 ± 2.72	12.23 ± 0.07
HCD	300.00 ± 7.81* (+24.5%)	14.00 ± 1.15 (+14%)
HFD	168.35 ± 5.5* (-28%)	12.80 ± 0.62 (+5%)

Results are mean ± SEM of eight different preparations.
Values in parenthesis represent percent change from control.
*Significantly different from corresponding control values at $p<0.05$ or higher degree of significance by independent *t* test and ANOVA
[1]HCD, High carbohydrate diet; HFD, High fat diet

Effect of High Carbohydrate Diets and High Fat Diets on Serum Parameters

The effects of HCD and HFD were seen on various serum parameters and results are summarized in Table 2. Feeding of HCD and HFD included no significant effect on serum creatinine, while BUN slightly decreased by HCD (-16%) and HFD (-19%), indicating normal functioning of the kidneys, compared to control rats. Further, feeding of HCD resulted in significant increases in serum glucose (+92%), cholesterol (+44%) and ALkPase (+15%). In contrast, feeding of HFD significantly increased serum glucose (+115%), cholesterol (+77%) and ALkPase (+26%), compared to control rats. Although serum Pi (+18%) increased much lesser by HFD than HCD (+56%). In contrast, serum catalase significantly decreased by HCD (-24%) and HFD (-25%) (Table 2).

Effects of High Carbohydrate Diets and High Fat Diets on Brush Border Membrane Marker Enzymes in Mucosal Homogenate and Brush Border Membrane Vesicles

The effects of HCD and HFD were assessed on the activities of ALkPase, GGTase and sucrase in the homogenates and BBMV isolated from rat intestinal mucosa (Table 3). The activities of ALkPase (+59%) and sucrase (+38%) significantly increased whereas the activity of GGTase (-2%) was unaffected in the mucosal homogenate of HCD-fed rats. In contrast, the activity of sucrase decreased significantly (-71%) whereas the activities of ALkPase and GGTase were unaffected upon feeding HFD to the control rats (Table 3a).

The effects of HCD and HFD were further analyzed on the specific activities of BBM marker enzymes in BBMV isolated from intestinal mucosa. Results in Table 3b show that the activities of ALkPase (+101%) and sucrase (+68%) increased significantly while the activity of GGTase (-48%) significantly decreased in the isolated BBMV of HCD-fed rats (Table 3b). However, the activity of sucrase (-53%) decreased significantly and ALkPase (+44%) and GGTase (+21%) activities increased greatly by feeding HFD to rats, compared to control rats.

The kinetic parameters (Vmax and Km) were assessed by assaying the enzymes in BBMV isolated from intestinal mucosa. Results indicated that increases in ALkPase activity by HCD and HFD were due to increases in the Vmax and decreases in the Km values. The changes observed in GGTase activity in HCD-fed rats were due to decreases in Km. In HFD-fed rats, this was due to increased Vmax. In contrast, the increase in sucrase activity by HCD was due to increases in Km whereas the decrease in the activity was due to decreases in Vmax and Km values.

Table 2. Effects of high carbohydrate diets and high fat diets on various serum parameters of the rats

Parameters Groups	Creatinine (mg/dl)	BUN[1] (mg/dl)	Glucose (mg/dl)	Cholesterol (mg/dl)	Inorganic Pi (μmol/ml)	Catalase (Units/ml/min)	ALkPase[1] (KA units)
Control	13.02 ±0.48	43.55 ± 1.9	63.03 ± 4.6	58 ± 3.73	0.512 ± 0.033	122.03 ± 8.5	2.17 ± 0.05
HCD	12.72 ± 0.03 (-2%)	36.31 ± 2.6 (-16%)	127 ± 0.4* (+92%)	83.27 ± 4.39* (+44%)	0.801 ± 0.05* (+56%)	92.25 ± 2.33* (-24%)	2.49 ± 0.03 (+15%)
HFD	12.62 ± 0.21 (-3%)	35.28 ± 1.71 (-19%)	135.43 ± 7.12* (+115%)	109 ± 8.9* (+77%)	0.603 ± 0.042 (+18%)	91.44 ± 2.21* (-25%)	2.72 ± 0.08 (+26%)

Results are mean ± SEM of eight different preparations.
Values in parenthesis represent percent change from control.
*Significantly different from corresponding control values at $p<0.05$ or higher degree of significance by independent *t* test and ANOVA.
[1]BUN, Blood Urea Nitrogen; ALkPase, alkaline phosphatase; HCD, High carbohydrate diet; HFD, High fat diet.

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Table 3. Effects of high carbohydrate diets and high fat diets on the specific activities of alkaline phosphatase, γ-glutamyl transferase and sucrase in the (a) homogenate and (b) brush border membrane vesicles of the rat intestines

Enzymes Groups	ALkPase[1] (µmol/mg protein/h)	GGTase (µmol/mg protein/h)	Sucrase (µmol/mg protein/h)
(a) Homogenate			
Control	4.0 ± 0.09	1.73 ± 0.03	24.02 ± 0.47
HCD	6.37 ± 0.19* (+59%)	1.7 ± 0.07 (-2%)	33.25 ± 2.04 (+38%)
HFD	4.36 ± 0.08 (+9%)	1.8 ± 0.01 (+4%)	6.99 ± 1.04* (-71%)
(b) BBMV			
Control	39.17 ± 1.85	15.6 ± 1.3	205.0 ± 11.85
HCD	78.58 ± 5.49* (+101%)	8.0 ± 2.7* (-48%)	346.0 ± 21.48* (+68%)
HFD	56.38 ± 2.07* (+44%)	18.87 ± 0.56 (+21%)	96.23 ± 3.49* (-53%)

Results are mean ± SEM of eight different preparations.
Values in parenthesis represent percent change from control.
*Significantly different from corresponding control at $p<0.05$ or higher degree of significance by independent *t* test and ANOVA.
[1]AlkPase, alkaline phosphatase; GGTase, γ-glutamyl transferase; HCD, High carbohydrate diet; HFD, High fat diet

Table 4. Effects of high carbohydrate diets and high fat diets on the kinetic parameters of (a) alkaline phosphatase, (b) γ-glutamyl transferase and (c) sucrase in brush border membrane vesicles of the rat small intestines

Tissues	V_{max} (µmol/mg protein/h)	$K_m \times 10^{-3}$ M
ALkPase[1]		
Control	18.51	0.92
HCD	41.66 (+56%)	0.55 (-39%)
HFD	22.72 (+23%)	0.71 (-21%)
GGTase		
Control	5.78	1.33
HCD	6.25 (+8%)	0.54 (-59%)
HFD	8.33 (+44%)	1.33
Sucrase		
Control	250.00	41.66
HCD	250.00	62.50 (+50%)
HFD	40.00 (-84%)	3.57 (-91%)

Values in parenthesis represent percent change from control.
K_m (Michaelis Menton constant) and V_{max} (maximal velocity of enzyme reaction)
[1]AlkPase, alkaline phosphatase; GGTase, γ-glutamyl transferase; HCD, High carbohydrate diet; HFD, High fat diet

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Effects of High Carbohydrate Diets and High Fat Diets on Enzymes of Carbohydrate Metabolism in Intestinal and Liver Homogenates

To study the effects of nutritional stress on the metabolic activity of intestine using HCD and HFD, the activities of certain enzymes linked to carbohydrate metabolic pathways such as glycolysis, TCA cycle, HMP-shunt pathway and gluconeogenesis were assessed in the intestine and liver. The activity of LDH, a representative enzyme of glycolysis, greatly increased (+154%) ; however, MDH (-11%) and ICDH (-23%) activities decreased by feeding HCD to the control rats (Table 5). In contrast to HCD, the activities of LDH (-48%), MDH (-64%) and ICDH (-22%) significantly decreased by HFD. The activities of enzymes involved in glucose synthesis by gluconeogenesis of G6Pase and FBPase significantly decreased in HCD and HFD-fed rats, suggesting decreased synthesis of glucose in intestine by these diets (Table 5). The activities of G6PDH and ME that provide NADPH to various anabolic reactions and antioxidant mechanism were assessed in mucosal homogenates of HCD and HFD-fed rats.

The activities of G6PDH (an enzyme of HMP-shunt pathway) and ME were assessed under the two dietary conditions. Feeding of HCD and HFD to the rats significantly decreased the activities of G6PDH (HCD, -60% and HFD, -24%) and ME (HCD, -74% and HFD, -68%) in the intestine (Table 5). The metabolic activity of intestine was compared with that of liver under these dietary manipulations. The effects of HCD and HFD were differentially observed in the liver other than intestine. The HCD caused great increases in the activity of LDH (+41%), MDH (+30%), G6PDH (+68%) and ME (+40%) ; however, the activity of FBPase (-25%) and G6Pase (-20%) decreased by HCD in the liver. Like the intestine, the activity of LDH (18.5%), MDH (-13%), FBPase (-21%), G6Pase (-29%), G6PDH (-49%) and ME (-39%), significantly decreased by HFD in the liver (Table 5).

Effects of High Carbohydrate Diets and High Fat Diets on Enzymatic and Non-enzymatic Antioxidant Parameters in Intestinal Mucosa and Liver

It is well established that antioxidant status can be used as a biomarker to assess chronic disease risks and diets can modulate antioxidant defense system. The GSH and its redox cycle enzymes such as SOD, catalase and GSH-Px are important cellular defense systems against oxidative stress. The activities of SOD, catalase and GSH-Px and associated parameters of oxidative stress were assessed in control, HCD and HFD-fed rats (Table 6). The LPO assessed as the level of malondialdehyde (MDA) as a known indicator of tissue injury increased significantly in HCD (+21%) and HFD (+28%) fed rats. Total-SH levels of small intestine decreased in HCD (-23%) fed rats while increased significantly in HFD (+22%) fed rats, compared to control rats (Table 6). The activity of SOD (-41%) and catalase (-44%) was suppressed by HCD and HFD. The oxidative stress parameters were similarly affected by HCD and HFD in the liver. Moreover, LPO levels were enhanced by decreases in antioxidant enzyme (SOD and catalase) activities in the liver (Table 6).

Table 5. Effects of high carbohydrate diets and high fat diets on the specific activities of carbohydrate metabolic enzymes in the homogenates of rat intestines and livers

Enzymes Groups	LDH[1] (µmol/ mg protein/h)	MDH (µmol/ mg protein/h)	FBPase (µmol/ mg protein/h)	G6Pase (µmol/ mg protein/h)	G6PDH (nmol/ mg protein/h)	ME (nmol/ mg protein/h)	ICDH (nmol/ mg protein/h)
Intestine							
Control	52.0 ± 5.6	49.2 ± 9.3	1.46 ± 0.05	4.17 ± 1.0	213.0 ± 30.0	587.0 ± 30.0	203.0 ± 18.0
HCD	132.2 ± 11.4* (+154%)	43.6 ± 1.0 (-11%)	1.21 ± 0.04* (-17%)	2.96 ± 3.03* (-29%)	85.0 ± 10.0* (-60%)	152.0 ± 54.00* (-74%)	157.0 ± 14.0* (-23%)
HFD	26.8 ± 0.7* (-48%)	17.6 ± 1.3* (-64%)	1.11 ± 0.08* (-24%)	2.86 ± 3.8* (-31%)	162 ± 20.0* (-24%)	186.0 ± 57.0 (-68%)	158.0 ± 10.0* (-22%)
Liver							
Control	42.8 ± 5.46	98.65 ± 3.88	6.93 ± 0.26	4.12 ± 0.15	290.35 ± 7.56	379.50 ± 12.00	
HCD	60.24 ± 2.57* (+41%)	128.30 ± 7.99* (+30%)	5.20 ± 0.10* (-25%)	3.28 ± 0.12* (-20%)	487.20 ± 10.30* (+68%)	530.50 ±15.07* (+40%)	
HFD	34.88 ± 2.06 (-18.5%)	85.70 ± 3.59* (-13%)	5.50 ± 0.03* (-21%)	2.92 ± 0.10* (-29%)	148.25 ± 10.80* (-49%)	231.80 ±17.6* (-39%)	

Results are mean ± SEM of eight different preparations.
Values in parenthesis represent percent change from control.
*Significantly different from corresponding control at $p<0.05$ or higher degree of significance by independent *t* test and ANOVA.
[1] LDH, lactate dehydrogenase; MDH, malate dehydrogenase; FBPase, fructose 1,6-bisphosphatase; G6Pase, glucose 6-phosphatase; G6PDH, glucose 6-phosphate dehydrogenase; ME, NADP-malic enzyme; ICDH, isocitrate dehydrogenase; HCD, High carbohydrate diet; HFD, High fat diet

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Table 6. Effects of high carbohydrate diets and high fat diets on non-enzymatic and enzymatic antioxidant parameters in the homogenates of rat intestines and livers

Tissues	LPO[1] (nmoles/g tissue)	Total-SH (µmoles/g tissue)	SOD (Units/mg protein)	Catalase (µmoles/mg protein/min)	GPH-Px (µmoles/mg protein/min)
Intestine					
Control	68.92 ± 2.71	2.57 ± 0.08	18.38 ± 0.5	15.53 ± 0.9	0.056 ± 0.001
HCD	83.24 ± 4.28*	1.99 ± 0.12*	10.85 ± 1.10*	8.75 ± 0.5*	0.06 ± 0.004
	(+21%)	(-23%)	(-41%)	(-44%)	(+7%)
HFD	88.47 ± 1.20	3.00 ± 0.18*	15.84 ± 0.33	9.97 ± 0.53*	0.068 ± 0.005
	(+28%)	(+22%)	(-14%)	(-36%)	(+21%)
Liver					
Control	13.20 ± 0.78	11.96 ± 0.50*	33.75 ± 1.19	42.90 ± 4.24	0.034 ± 0.002
HCD	16.50 ± 1.77*	10.15 ± 0.40	31.15 ± 0.21	32.98 ± 0.79	0.036 ± 0.002
	(+25%)	(-15%)	(-7%)	(-23%)	(+6%)
HFD	16.10 ± 0.54*	24.41 ± 2.23*	22.27 ± 1.80	35.60 ± 1.25	0.031 ± 0.003
	(+21%)	(+104%)	(-33%)	(-20%)	(-10%)

Results are mean ± SEM of eight different preparations.
Values in parenthesis represent percent change from control values.
*Significantly different from corresponding controls at $p<0.05$ or higher degree of significance by independent *t* test and ANOVA.
[1] LPO, lipid peroxidation; SH, sulfhydryl; SOD, superoxide dismutase; GPH-Px, glutathione peroxidase; HCD, High carbohydrate diet; HFD, High fat diet

Discussion

The intestinal mucosa is a dynamic structure that undergoes biochemical, ultra-structural and morphological changes throughout the life and in response to the content of various nutrients in the diets, which can affect overall health (39, 40). The small intestine with liver play critical roles in the digestion and absorption of food components and recognition of food-derived signals (39, 40). Despite numerous studies, the effects of HCD and HFD on the structure, funtion and metabolic status of small intestine and liver have not thoughly been investigated. The effects of dietary manipuations on carbohydrate metabolic pathways such as glycolysis, TCA cycle, gluconeogenesis and HMP-shunt and oxidative stress paprameters in the intestine and liver are especially lacking. Thus, the present study was carried out to assess the comparative effects of HCD and HFD on various serum parameters and specific activities of various BBM enzymes, metabolic enzymes and antioxidant parameters in rat intestine and liver under similar experimental conditions.

As shown in the results, feeding of HCD resulted in significant body weight gains whereas significantly decreased by HFD. Serum creatinine was not affected by feeding HCD and HFD, indicating normal function of the kidneys. Serum glucose, cholesterol and Pi greatly increased by HCD and HFD. The great increases in serum glucose and cholesterol are not healthy indicators and may pose health risks. The diet-induced serum glucose might be due to glucose tolerance and hepatic isulin senstivity (3, 41). Increased serum cholesterol by HCD an HFD might alter membrane fluidity and hence the membrane organization and functions. The HFD causes inflammatory responses, deranges the homeostasis of cellular metabolism and is a key initiator of the metabolic syndrome (31).

Results further demonstrated that feeding of HCD and HFD to rats affected the activities of hydrolytic enzymes of ALkPase, GGTase and sucrase involved in end-stage digestion and absorption (Table 3a, 3b). The activity of ALkPase and sucrase greatly increased; however, activity of GGTase decreased by HCD in the intestine. Activity of ALkPase and GGTase increased by HFD but lesser than that by HCD. Moreover, activity of sucrase signifintly decreased by HFD. These effects of HCD and HFD were similar to those of previous studies (21, 24, 42). The differences in the effects could be attributed to the fact that these enzymes were located in various thicknesses of the BBM (38) and differentially regulated by dieatary status (14, 15, 22, 28). Sucrase activity increased significantly but decreased rapidly when HCD replaced by HFD (28). Serum and intestinal ALkPase activity increased by HFD; however, the mechanism of this increase is not fully understood (28). It is believed that increased serum ALkPase increases PI levels that phosphorylates metabolites of and increases ATP production. Kinetic analysis revealed that the linked increase or decrease in the activities of BBM enzymes in the intestine caused by HCD or HFD was due to increases in Vmax and decreases in Km values, except for sucrase in HCD-fed rats, where increases were due to increases in Km values. These partially indicated adaptive but specific alterations in the synthesis of enzyme molecules (12, 15, 24).

The macromolecular complex food components are generally digested to small molecules such as glucose, amino acids, fatty acids and Pi, which are efficiently absorbed by specific transporters across intestinal BBM. The absorptive functions of small intestine involve vital energy-dependent transport processes, which are supported by various energy yielding pathways of carbohydrate metabolism (e.g., glycolsis, TCA cycle, HMP-shunt

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pathway and gluconeogenesis). The present results showed that HCD and HFD caused specific alterations in enzyme activities involved in various metabolic pathways. The activity of LDH showed great increases by HCD but decreased significantly by HFD. The activities of TCA cycle enzymes of MDH and ICDH significantly decreased in the intestine by HCD and HFD. The activities of G6Pase and FBPase involved in gluconeogenesis significantly decreased HCD and HFD-fed rats (Table 4). The activities of two other enzymes of G6PDH and ME involved in generation of NADPH were significantly decreased by the two diets. However, the effect of these diets on liver metabolic activity was quite diifferent than that on intestine. The activity of LDH, MDH increased but the activity of G6Pase and FBPase decreased by HCD. The activity of lipogenic enzymes, including G6PDH and ME, increased by HCD in the liver. In contrast, all metabolic enzymes studied decreased by HFD.

Thus, it is clear that the enzyme of glycolysis, LDH, increased; however, those of TCA cycle gluconeogenesis and HMP-shunt pathway decreased in the intestine by HCD. In the liver, the enzymes involved in glycolysis, including TCA cycle and HMP-shunt, increased whereas the ezyme levels of gluconeogenesis were low (43). It is believed that increased serum ALkPase increases Pi levels that phosphorylates metabolites of glycolysis and increase ATP production. In contrast to HCD, all the enzmes of metabolisn decreased in the instine and liver by HFD. In HCD-fed rats, energy generation was focused majorly via anaerobic glycolysis, In the liver, oxidative metabolim was further seen. In small intestine, anaerobic metablism is further observed (14, 44). In HFD-fed rats, the metabolic activty decreased by anaerobic and aerobic pathways most likely due to lack of carbohydrates in the diet.

Oxidative stress is one of the causative factors of several diseases, ranging from cancers to CVDs (36). It is caused as a result of increased reactive oxygen species (ROS) generation, depressed antioxidant system or the two (17). There are only a few studies on oxidative stress and dietary imbalances. It has been reported that diets rich in fat cause liver injury and produce oxidative stress as indicated by enhanced LPO assessed as MDA concentrations (26, 31, 45). High sucrose diets have been reported to cause oxidative stress (46).

Feeding of HCD and HFD resulted in significant alterations in the enzymatic and non-enzymatic parameters of antioxidant defense mechanisms in the intestine and liver. The effects of HCD and HFD were assessed on LPO, total-SH and activities of antioxidant enzymes of SOD, catalase and GSH-Px. The LPO assessed as MDA levels significantly increased by HCD and HFD in the intestine and liver differentially. This was accompanied by significant decreases in the activity of SOD and catalase. (Table 5). The suppression of SOD and catalase activities might lead to significant increases in levels of LPO in the intestine and liver. The GSH-Px, a selenium-containing enzyme, was not affected by HCD but significantly increased by HFD in the intestine. Moreover, total SH content of the tissue decreased significantly by HCD but increased significantly in HFD-fed rats. Thus, prolonged feeding of HCD and HFD caused distresses in antioxidant defense mechanisms in rat intestine and liver as shown in preliminary reports (26, 31, 46).

In summary, the underlying mechanism; by which, HCD and HFD diets caused significant alterations in serum parameters, BBM enzyme activitties, oxidative stress parameters and overall metabolic health seemed complex. Serum glucose, cholesterol, Pi and ALkPase activity significantly but differentially increased by HCD and HFD. The BBM enzymes of ALkPase and sucrase increased; however, GGTase decreased by HCD. The HFD increased ALkPase and GGTase whereas significantly decreased sucrase activity. Kinetic analysis supported the fact that changes in enzyme activities were due to changes in Vmax or Km or the two parameters based on the substrate availability. The HCD induced increases in LDH activity and decreases in MDH. Moreover, ICDH activity indicated that energy generation occurred majorly by anaerobic glycolysis in intestine. In liver, energy production occurred by oxidative metabolism as indicated by HCD-induced increased activity of theses enzymes. In contrast, HFD decreased all the enzymes involved in glucose metabolism and its production. The HCD may not pose serious metabolic risks; HFD deranges the homeostasis of cellular metabolism and is a key initiator of the metabolic disorder. The HCD and HFD caused oxidative stress in intestine and liver as verified by increased LPO and suppressed activities of antioxidant enzymes of SOD and catalase. It is concluded that food enriched in excessive carbohydrates or fats include adverse effects on rat intestine and liver. Oxidative stress induced by HCD and HFD diets may be at least one of the causative factor of such adverse effects.

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Abbreviations

ALkPase, alkaline phosphatase

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BBM, brush border membrane
BBMV, BBM vesicles
FBPase, fructose 1,6-bisphosphatase
G6Pase, glucose 6-phosphatase
G6PDH, glucose 6-phosphate dehydrogenase
GGTase, γ-glutamyl transferase
SOD, superoxide dismutase
HFD, high fat diet
HCD, high carbohydrate diet
ICDH, isocitrate dehydrogenase
LDH, lactate dehydrogenase
LPO, lipid peroxidation
MDA, malondialdehyde
MDH, malate dehydrogenase
ME, NADP-malic enzyme
NAD, nicotinamide adenine dinucleotide
NADH, nicotinamide adenine dinucleotide reduced
NADPH, nicotinamide adenine dinucleotide phosphate reduced
NADP, nicotinamide adenine dinucleotide phosphate
ROS, reactive oxygen species
SH, sulfhydryl
SOD, superoxide dismutase
TCA, tricarboxylic acid

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