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EFFECTS OF MATERNEL DIETARY FIBER SUPPLEMENTATION ON BIOCHEMICAL PARAMETERS AND OXIDATIVE STRESS MARKERS IN OBESE PREGNANT RATS AND THEIR OFFSPRING

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Abstract

Description of the subject: Maternal nutrition before and during pregnancy, is an easily modifiable environmental factor that can affect fetal growth and development with potential long-term consequences. The concept of «fetal programming» implies fetal life alterations expose to further developpment of pathologies. The research for new treatments based on dietary fiber therapies has turned to finds their beneficial effects on the pathogenesis of maternal obesity. Dietary fiber has multiple properties with potential biological activity.

Objective : The current study investigates the role of a highly pure enriched cellulose diet in the modulation of biochemical parameters and oxidant/ antioxidant markers in cafeteria-induced obese rats and their offspring.

Methods: Female rats receive during two months before and during pregnancy control diet or cafeteria supplemented or not with cellulose. Pregnant rats and their offspring were also fed on similar diet. At the end of the experiment, biochemical parameters were analyzed, the liver is used to determine the oxidative stress markers in mitochondrial liver homogenates.

Results: The results show that the cafeteria diet induced obesity associated with various plasma metabolic disorders and increased intra-mitochondrial hepatic oxidative stress in both mothers and their offspring. Cellulose diet supplementation induced a modulation of the oxidative stress, improving metabolic status

Conclusion: Maternal dietary fiber supplementation enriched with cellulose displayed remarkable health benefits and can be a strategy against obesity and its complications.

Keywords: Cafeteria; dietary fiber (Cellulose); maternal obesity; pregnant rat; offspring; biochemical parameters; oxidative stress.

EFFETS DE LA SUPPLÉMENTATION MATERNELLE EN FIBRES ALIMENTAIRES SUR LES PARAMÈTRES BIOCHIMIQUES ET LES MARQUEURS DU STRESS OXYDATIF CHEZ LES RATES GESTANTES OBÈSES ET LEUR PROGÉNITURE.

Résumé

Description du sujet : La nutrition maternelle est un facteur environnemental facilement modifiable qui peut affecter le développement fœtal avec des conséquences potentielles à long terme. La notion de «programmation fœtale» implique qu'une altération durant la vie fœtale exposerait au développement ultérieur de pathologies. Les recherches de nouveaux traitements à base des fibres alimentaires se sont tournées vers leurs effets bénéfiques sur la pathogenèse de l'obésité maternelle. Les fibres alimentaires possèdent de multiples propriétés à activité biologique potentielle.

Objectifs : La présente étude étudie le rôle d'un régime enrichi en cellulose hautement purifiée dans la modulation des paramètres biochimiques et des marqueurs oxydants / antioxydants chez les rats obèses et leur progéniture induits par le régime cafeteria.

Méthodes : Les rats femelles reçoivent pendant deux mois avant et pendant la gestation le régime control témoin ou cafeteria supplémenté ou non en cellulose. Les progénitures issues de rates gestantes consomment le même régime. A la fin de l'expérimentation, les paramètres biochimiques sont analysés, le foie est utilisé pour déterminer les marqueurs du stress oxydatif sur des homogénats hépatiques mitochondriaux.

Résultats : Les résultats montrent que le régime cafeteria induit une obésité associée à divers désordres métaboliques plasmatiques et à l'augmentation du stress oxydatif intra mitochondriale hépatique chez les mères obèses et leurs progénitures. La supplémentation du régime en cellulose induit une modulation du stress oxydatif, améliorant l'état métabolique.

Conclusion : Une supplémentation maternelle en fibres alimentaires enrichie en cellulose présente des avantages remarquables pour la santé et peut constituer une stratégie de lutte contre l'obésité et ses complications.

Mots clés : Cafeteria ; fibres alimentaires (Cellulose) ; obésité maternelle ; rate gestante ; progéniture ; paramètres biochimiques ; stress oxydatif.

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INTRODUCTION

Obesity is a risk factor that is linked to chronic diseases such as type 2 diabetes mellitus (T2DM) and cardiovascular diseases (CVD) [1], it is also associated with a higher risk of premature death [2]. Nowadays, obesity and associated comorbidities represent a major challenge to the health system; they are a serious and important public health problem to people not only in Algeria but also throughout the entire worldwide. For the World Health Organization (WHO), obesity is an epidemic health problem with a higher prevalence in females in reproductive age [3]. Several studies have reported that obesity is associated with oxidative stress which is in turn related to inadequate antioxidant defenses and increased rates of free radical formation [4]. The HFD (high-fat diet) feeding is believed to be a reliable model for studying dietary obesity in humans. In animal models, the high-fat diet was effective in inducing obesity, as this has previously been demonstrated by Akyol *and coll.* [5]. Pregnancy is a state of oxidative stress [6]. Maternal obesity is considered as a chronic inflammatory state which has been shown to induce increased levels of free fatty acids (FFAs), reactive oxygen species (ROSs) and inflammatory cells. Recent evidence has revealed increased levels of lipid peroxidation products in the plasma of obese women during pregnancy [7]. Oxidative stress may be related to delivery or to a pre-existing fetal oxidative status [8]. The aim of the present work is to identify markers of oxidative stress in liver mitochondrial suspension in obese rats, and especially in pregnant female rats and their offspring, as a result of their high fat diet feeding. Mitochondria isolated from liver cells can be used to determine the SOD, catalase, GSH activities and to measure the carbonyl proteins and MDA concentrations. The liver is an organ that is attacked mainly by reactive oxygen species (ROS) [9]. Mitochondria are the major site where reactive oxygen species are produced [10]. Indeed, mitochondria are essential sources of reactive oxygen species (ROS) via their respiratory chain. In addition, mitochondria would produce 90% of cellular ROS in that way [11]. Today, dietary fibers are known to have an efficient protective effect against certain gastrointestinal diseases, such as constipation, colon cancer, gastroesophageal reflux disease, obesity, diabetes, hypertension and cardiovascular diseases [12]. Dietary fiber nutritional supplements have always been regarded as an effective way to treat and prevent chronic diseases caused by long-term high-fat diet intake in nowadays' society [13].

A fiber is generally regarded as potentially capable of diluting food energy, altering intestinal transit time and promoting satiety [14]. It is worth mentioning that the administration of fibers improves the lipid metabolism disorderly situation of hyperlipidemia rats as compared with the normal group. Moreover, supplemental total dietary fiber can induce the lowest body weight gain in rats, and decrease total cholesterol, triglyceride and low density lipoprotein cholesterol, while it has the capacity to increase high density lipoprotein cholesterol [15]. To the best of our knowledge there are no reports in the literature on the effect of dietary fiber on metabolic status during maternal obesity and their repercussions on the offspring. Because maternal obesity has profound effects on neonate metabolism in humans and also in animals, our aim was to evaluate the consequences of highly pure enriched cellulose (HPEC) supplementation in the diet before and during gestation on maternal and their offspring induced by cafeteria.

MATERIAL AND METHODES

1. Animals and experimental protocol

Female wistar rats (aged 1 month, n=72), weighing 90 to 100g, were obtained from Animal Resource Centre (Algeria). Animals were housed at 20±2°C with 2-3 in each cage, and maintained on a 12:12 h light/dark cycle. Rats were assigned to each diet group during 8 weeks of experimental period: The control group (control, C, n=18) was fed standard laboratory chow (ONAB, Algeria) before and during pregnancy. The second group (control cellulose, CC, n=18) was fed a control commercial diet enriched with highly-pure-enriched-cellulose at 10% before and during pregnancy. In group 3 (high fat diet group, HFD, n=18) was fed a cafeteria diet before and during pregnancy. In group four (high fat diet cellulose, HFDC, n=18), rats were fed on cafeteria diet supplemented with highly- pure- cellulose at 10% before and during gestation. The control diet (386 kcal/100g) was composed of 25% of energy as protein, 10% of energy as lipids, 5% of cellulose, and 65% of energy as carbohydrates (ONAB; Algeria). The components of the cafeteria diet were grinded cheese, bacon, potato chips, biscuits and chocolate (in a proportion of 2:2:2:1:1:1, by weight) mixed with standard chow (w/w) [16]. The cafeteria diet (523 kcal/100 g) was composed of 23% of energy as protein, 42% of energy as lipids, 5% of cellulose, and 35% of energy as carbohydrates. We have previously used this cafeteria diet and shown that it induced hyperphagia and obesity in rats [17].

Highly- pure- cellulose was obtained from Biochem Chemopharma in Montreal (Quebec), was added to the control or cafeteria diet at 10% (w/w). Fresh food was given daily and body weights were recorded. After mating, the first day of gestation was estimated by presence of spermatozoids in vaginal smears. Pregnant dams of each group were maintained on their respective diets throughout pregnancy and lactation. After weaning, the offspring continue to follow the same diet as their mothers. The study was conducted in accordance with the national guidelines for the care and use of laboratory animals. All the experimental protocols were approved by the Regional Ethical Committee.

2. Blood samples

After delivery, six mother rats from each groups were weighted and scarified, thus mothers were anaesthetized with intraperitoneal injection of sodium pentobarbital (60 mg/Kg of body weight). After overnight fasting, at days 90 for pups, six rats from each group were anesthetized with intraperitoneal injection of sodium pentobarbital (60 mg/kg of body weight). The female rats and of their offspring were not all sacrificed on the same day. The blood was taken from the abdominal aorta into heparinized tubes and plasma was used for biochemical determinations. The liver were removed, washed with ice-cold saline, quickly blotted, weighed and immersed in TSE (Tris–sucrose-EGTA) isolation at 4°C for extraction of hepatic mitochondria and redox marker determinations.

3. Determination of biochemical parameters

Plasma glucose, creatinine, total proteins, urea, uric acid, triglyceride and cholesterol were measured using colorimetric enzymatic kits (Spinreact, Ctra. Santa Coloma, Spain). Plasma alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) and lactate dehydrogenase (LDH) activities were determined using colorimetric enzymatic kits (Spinreact, Ctra. Santa Coloma, Spain).

4. Extraction of mitochondria from rat liver

The procedure employed for the isolation of hepatic mitochondria is based on the technique of subcellular fractionation (differential centrifugation) for cell fractionation, described by Frezza [18]; 10g from liver were crushed in a potter containing 30 ml of TSE (Tris–sucrose-EGTA) (250 Mm of sucrose, 50Mm of Tris, 5mM of EGTA; pH 7.2), allowing the liberation of mitochondria. The homogenate was subjected to 1770 rpm centrifugation for 10 minutes. The supernatant was collected and subjected to 9600 rpm centrifugation for 10 minutes. The mitochondrial nerve resulting is resuspended in 13ml of TSE. This suspension is again subjected to 9600 rpm centrifugation for 10 minutes. The nerve containing mitochondria is then taken in 15ml of TS buffer (Tris-sucrose) (250 Mm of sucrose, 50mM Tris; pH 7) then suffers a last centrifugation at 9600 rpm for 10 minutes. The final pellet is divided into two fractions: the first is taken in 200µl of TS buffer, for mitochondrial suspension and the second in the hypotonic solution (25mM of KH_2PO_4, 5Mm $Mgcl_2$; pH 7.2) used to study mitochondrial antioxidant enzymes.

5. Determination of liver mitochondrial oxidant/antioxidant status

-*Catalase activity :* The catalase (EC 1.11.1.6) activity was measured by spectrophotometric analysis of the rate of H_2O_2 decomposition at 240 nm, according to Aebi's method [19].

-*Superoxide dismutase (SOD) activity :* The superoxide dismutase activity was measured by means of the NADPH oxidation procedure and was expressed in units per gram of total proteins (U/g) [20].

-*Levels of reduced gluthaione (GSH) in liver:* Liver reduced glutathione (GSH) levels were assayed using a colorimetric method that is based on the reduction of 5,5-dithiobis-(2-nitrobenzoic) acid (DTNB) by GSH in order to generate 2-nitro-5-thiobenzoic acid, based on Sigma Aldrich Kit (Saint Louis, MO, USA).

-*Malondialdehyde (MDA) levels:* Levels of MDA (markers of lipid peroxidation) were estimated by the method of Draper and Hadley *and coll.* [21].

-*Protein carbonyl levels:*Protein carbonyls, used as markers of protein oxidation, were determined through the identification of protein carbonyl groups using 2,4-dinitrophenylhydrazine (DNPH) leading to the formation of stable dinitrophenyl (DNP) hydrazone adducts [22].

6. Statistical analysis of data

Results are expressed as means ± standard deviation (SD) and were tested for normal distribution using the Shapiro– Wilk test. Data not normally distributed were logarithmically transformed. Significant differences among the groups were analyzed statistically by a one-way analysis of variance (ANOVA). When significant changes were observed in ANOVA tests, Fisher least significant difference tests were applied to locate the source of significant difference.

The individual effects of the diets and cellulose supplementations were distinguished by two-way ANOVA. The significance level was set at $P < 0.05$. These calculations were performed using STATISTICA version 4.1 (STATSOFT, Tulsa, OK).

RESULTS

1. Effects of cellulose diet on body weight, food and energy intakes, and liver tissue weight

It was found that rat fed a high-fat diet (HFD) became obese; they exhibited increased body weight gain as compared to those having a control diet, regardless of cellulose supplementation, for both non-pregnant and pregnant rats with their offspring (Table 1). As expected, the group fed HFD showed higher food and energy intakes compared with control animals. Obese rats, fed HFD, had a significantly higher liver weight than that of rats in the control group (Table 1). On the other hand, no significant differences were observed in body weight, liver weight, food and energy intakes between control animals and control rat fed dietary cellulose.

Tableau 1: Body and liver weights, food and energy intakes in rats

Parameters	Control rats		Cafeteria diet	
	C	CC	HFD	HFDC
Before pregnancy				
Body weight	178.66±3.14^{c}	182.60±3.83^{c}	238.33±6.80^{a}	201.00±2.82^{b}
Food intake	37.75±5.45^{c}	38.43±4.80^{c}	50.16±10.37^{a}	40.68±3.45^{b}
Energy intake	118.13±2.91^{b}	110.91±2.46^{b}	185. 61±9.33^{a}	130.27±1.85^{b}
Relative liver tissue weight	8.83±0.10^{c}	8.38±0.37^{c}	11.04±0.51^{a}	9.35±0.34^{b}
During pregnancy				
Body weight	184.16±10.18^{c}	182.20±14.85^{c}	264.66±13.17^{a}	209.16±16.99^{b}
Food intake	35.05±0.79^{c}	37.26±2.40^{c}	49.16±4.21^{a}	40.60±4.43^{b}
Energy intake	147.11±7.47^{c}	144.87±5.73^{c}	243.50±4.87^{a}	170.03±2.58^{b}
Relative liver tissue weight	9.06±0.65^{c}	8.64±0.98^{c}	12.16±0.84^{a}	10.62±0.39^{b}
Offspring rats (90days)				
Body weight	182.83±15.10^{b}	180.40±8.17^{b}	235.00±47.41^{a}	208.00±23.38^{b}
Food intake	36.08±3.05^{c}	35.68±2.18^{c}	49.45±6.56^{a}	42.77±2.18^{b}
Energy intake	149.38±0.94^{c}	151.50±1.29^{c}	200.92±3.34^{a}	169.48±4.54^{b}
Relative liver tissue weight	8.04±0.55^{c}	7.94±0.74^{c}	10.69±0.83^{a}	9.21±0.80^{b}

Values are presented as means ± standard deviations (SD). C: control diet; CC: control diet enriched with cellulose at 10 %; HFD: high fat diet; HFDC: high fat diet enriched with cellulose at 10 %. Data were tested by one-way ANOVA and Tukey post hoc tests. Values with different superscript letters (a, b, c, d…) are significantly different at $p < 0.05$

2. Effects of cellulose diet on plasma biochemical parameters

Plasma glucose, cholesterol and triglycerides levels were significantly higher in obese, pregnant or non-pregnant rats as compared to those belonging to the control group (C). It is worth noting that cellulose supplementation induced a significant reduction in glycemia and lipidemia in obese rats (HFDC), as illustrated in Table 2**.** Obese rats showed a significant increase in plasma contents of urea, creatinine and uric acid compared with control rats. Table 2 indicates that the cellulose diet significantly decreased the plasma concentrations of urea, creatinine and uric acid in obese rats to the levels observed in obese rats, before and during pregnancy. However, no significant differences were noted in plasma albumin and protein levels between control and obese rats consuming the diet enriched with cellulose. AST, ALT activities turned out to be significantly higher in obese rats as compared to control rats. Moreover, cellulose supplementation induced a reduction in ALT and AST activities in obese rats, as displayed in Table 3. The LDH and ALP activities were significantly higher in all obese rats (HFD) as compared to those in control rats (C). These differences were remarkable in pregnant rats and their offspring as compared to HFDC group (Table 3).

Tableau 2: Plasma biochemical parameters in rats

Parameters	Control rats		Cafeteria diet	
	C	CC	HFD	HFDC
Before pregnancy				
Glucose(mmol/l)	5.60±0.38^{c}	5.55±0.89^{c}	11.45±0.43^{a}	7.68±1.22^{b}
Creatinine(µmol/l)	47.47±4.56^{c}	45.45±10.67^{c}	91.91±7.08^{a}	60.60±8.57^{c}
Urea(mmol/l)	4.60±0.33^{c}	4.59±0.18^{c}	8.36 ±1.69^{a}	6.31±0.43^{b}
Uric acid(mmol/l)	243.95±11.29^{c}	246.01±20.79^{c}	351.30±22.44^{a}	274.28±13.79^{b}
Albumin (g/l)	32. 00±1.56	34. 84±4.08	30.17±4. 16	29.75±2.53
Total proteins(g/l)	10.27±0.65^{b}	8.80±0.42^{c}	11.62±0.37^{a}	8.66±0.52^{c}
Total cholesterol (mmol/l)	1.04±0.05^{c}	1.09±0.08^{c}	1.84±0.20^{a}	1.45±0.33^{b}
Triglycerides (mmol/l)	0.48±0.05^{c}	0.43±0.06^{c}	0.94±0.08^{a}	0.57±0.07^{b}
During pregnancy				
Glucose(mmol/l)	7.22±1.24^{c}	7.64±1.32^{c}	13.15±0.76^{a}	9.17±2.69^{b}
Creatinine(µmol/l)	77.95±4.62^{c}	75.10±7.95^{c}	160.60±38.09^{a}	108.57±8.68^{b}
Urea(mmol/l)	4.73±0.08^{c}	5.04±0.26^{c}	9.76±1.05^{a}	7.70±1.90^{b}
Uric acid(mmol/l)	263.09±10.33^{b}	261.02±17.65^{b}	378±39.35^{a}	268.78±8.91^{b}
Albumin (g/l)	27.76±8.19	29.77±4.01	28.23±5.7	26.61±3.74
Total proteins(g/l)	9.12±0.36^{a}	7.98±0.92^{b}	10.05±0.43^{a}	8.18±0.82^{b}
Total cholesterol (mmol/l)	1.30±0.31^{c}	1.13±0.08^{c}	2.00±0.15^{a}	1.49±0.21^{b}
Triglycerides (mmol/l)	0.56±0.097^{c}	0.50±0.043^{c}	1.14±0.20^{a}	0.76±0.17^{b}
Offspring rats (90days)				
Glucose(mmol/l)	5.62±0.46^{c}	5.59±0.57^{c}	12.50±1.04^{a}	7.75±1.35^{b}
Creatinine(µmol/l)	72.76±8.28 ^{c}	70.75±6.35 ^{c}	120.20±10.43 ^{a}	80.80±8.28 ^{b}
Urea(mmol/l)	4.99±0.69^{c}	4.88±0.65^{c}	8.14±0.75^{a}	6.46±0.78^{b}
Uric acid(mmol/l)	254.29±17.35^{c}	266.71±25.51^{c}	379.76±22.55^{a}	301.33±9.95^{b}
Albumin (g/l)	32.21±4.63	29.75±3.29	31.52±2.97	30.28±3.29
Total proteins(g/l)	11.11±0.59^{b}	9.75±0.50^{b}	12.78±0.58^{a}	10.90±1.41^{b}
Total cholesterol (mmol/l)	1.18±0.11^{b}	1.03±0.54^{b}	2.02±0.13^{a}	1.24±0.17^{b}
Triglycerides (mmol/l)	0.47±0.08^{c}	0.49±0.09^{c}	0.99±0.22^{a}	0.67±0.09^{b}

Values are presented as means ± standard deviations (SD). C: control diet; CC: control diet enriched with cellulose at 10 %; HFD: high fat diet; HFDC: high fat diet enriched with cellulose at 10 %. Data were tested by one-way ANOVA and Tukey post hoc tests. Values with different superscript letters (a, b, c, d…) are significantly different at $p < 0.05$.

Tableau 3: Enzyme activities of the rats

Parameters	Control rats		Cafeteria diet	
	C	CC	HFD	HFDC
Before pregnancy				
AST(U/l)	79.04±8.31^{c}	74.08±6.32^{c}	151.95±12.14^{a}	101.91±3.26^{b}
ALT(U/l)	65.91±7.22^{c}	66.79±8.23^{c}	150.20±14.98^{a}	85.45±9.28^{b}
Alkaline phosphatase (U/l)	119.90±15.71^{c}	116.05±25.76^{c}	252.45±6.51^{a}	160±6.14^{b}
LDH(U/l)	804.41±43.79^{b}	823.29±49.19^{b}	994.85±79.22^{a}	827.40±125.09^{b}
During pregnancy				
AST(U/l)	70.58±10.65^{c}	69.41±3.44^{c}	140.87±18.07^{a}	107.20±14.32^{b}
ALT(U/l)	64.66±7.65^{c}	61.62±10.09^{c}	147.87±10.83^{a}	90.91±8.93^{b}
Alkaline phosphatase (U/l)	127±18.62^{c}	129.25±15.95^{c}	268±43.41^{a}	177.1±31.35^{b}
LDH(U/l)	864.33±33.07^{b}	879.25±45.82^{b}	999.77±27.86 ^{a}	869.26±18.36^{b}
Offspring rats 90days				
AST(U/l)	100.04±11.99^{b}	109.37±8.76^{b}	151.08±25.42^{a}	111.12±5.93^{b}
ALT(U/l)	70.58±0.90^{c}	69.41±11.64^{c}	150.50±8.35^{a}	105.75±14.90^{b}
Alkaline phosphatase (U/l)	122.10±29.29^{c}	125±16.62^{c}	254.10±17.83^{a}	157±9.88^{b}

LDH(U/l)	839.71 ± 35.75^{b}	827.40 ± 53.58^{b}	999.77 ± 30.35^{a}	840.53 ± 79.20^{b}

Values are presented as means ± standard deviations (SD). C: control diet; CC: control diet enriched with cellulose at 10 %; HFD: high fat diet; HFDC: high fat diet enriched with cellulose at 10 %. Data were tested by one-way ANOVA and Tukey post hoc tests. Values with different superscript letters (a, b, c, d…) are significantly different at $p < 0.05$.

3. Effects of cellulose diet on oxidant/ antioxidant markers in rats

Furthermore, a significant MDA level increase was observed in the liver mitochondrial suspension of obese rats as compared to that of control rats (Figure 1) and obese rats fed a diet supplemented with 10% cellulose. In obese pregnant rats and their offspring, at day 90, the oxidant/antioxidant status alterations were marked by a significant decrease in malondialdehyde (MDA) levels in the hepatic mitochondrial suspension as compared to that of the control group. Figure1 shows explicitly that the protein carbonyl levels in liver mitochondrial suspension were significantly higher in HFD-fed rats as compared to control rats and groups fed with diets supplemented with cellulose fiber. Our results suggest that pregnant rats and their offspring are most likely to have an increase in protein carbonyl levels as compared to non-pregnant rats. On the other hand, activities of antioxidant enzymes were found lower in obese rats than in control rats. Therefore, administering cellulose to obese rats leads to enhancing the activities of catalase and reduced glutathione (Figure 1). Moreover, SOD activity was significantly influenced by the diet given to rats. This activity was lower in obese groups as compared to that found in groups fed diets supplemented with cellulose fiber.

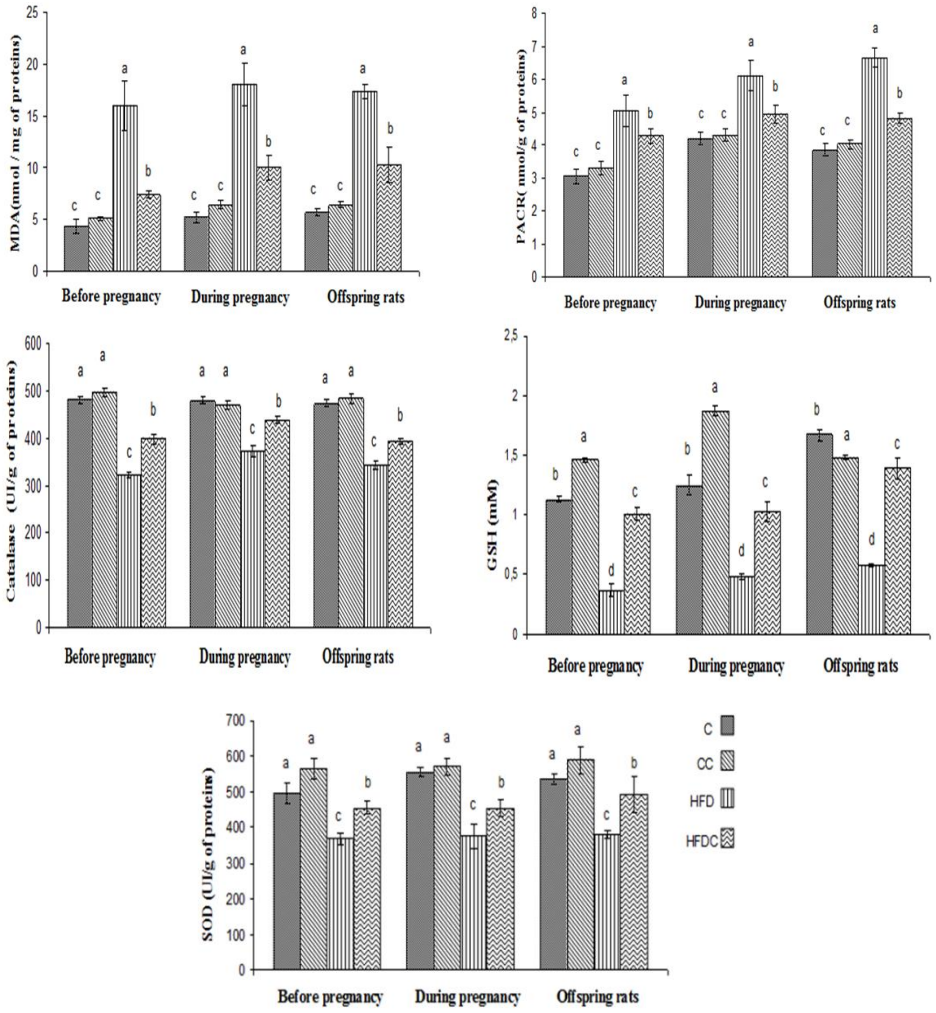

Figure 1 : Oxidant / antioxidant status in hepatic mitochondria in rats

Values are presented as means ± standard deviations (SD). C: control diet; CC: control diet enriched with cellulose at 10 %; HFD: high fat diet; HFDC: high fat diet enriched with cellulose at 10 %, Data were tested by one-way ANOVA and Tukey post hoc tests. Values with different superscript letters (a, b, c, d…) are significantly different at $p < 0.05$.

DISCUSSION

In this study, the authors made an attempt to demonstrate that high-fat diet is associated with abnormal oxidant/antioxidant status. The results of the present investigation provided evidence that high-fiber diet may reduce risk factors for obesity. These data corroborate the findings revealed by Papathanasopoulos *and coll.* [23]; these researchers also suggested that dietary fiber intake is inversely related to the risk of developing coronary heart disease. The present work allowed showing that high-fat diet (HFD) induces a significant increase in the liver and body weights, and in food and energy intakes as compared to C.

In addition, the results obtained indicate that high-fiber diets may reduce these parameters. Our findings are found to be in good agreement with those reported in other studies which revealed that fiber intake is inversely associated with body weight and body fat [24]. The bulking and viscosity properties of dietary fibers are preponderantly responsible for influencing satiety and satiation [25]. The present study revealed that HFD-fed rats had increased plasma glucose concentrations compared with those found in C pregnant rats and their offspring, which is consistent with previous studies [26]. Obese rats showed altered lipid concentrations, such as high plasma cholesterol and triglycerides levels, in comparison with control rats. The results of this study indicated that cellulose supplementation induces important lower in-vitro cholesterol, triglycerides and glycemia levels in HFD-fed pregnant or non-pregnant rats. These findings are in good agreement with those reported in other studies which suggest that high-fiber intake is associated with lower serum cholesterol concentration, reduced blood pressure, enhanced weight loss, better glycemic control, and improved gastrointestinal function [27]. Moreover, it is interesting to note that obese rats showed significantly higher plasma levels of urea, creatinine, uric acid and total proteins; these findings are similar to those encountered in several other studies [28]. Because obesity is typically associated with insulin resistance, and due to the fact that insulin regulates protein dynamics, it is reasonable to suspect that obesity can alter protein synthesis [29]. Previous studies have reported that dietary fiber contributes to additional benefits to chronic renal diseases [30]. In addition, it was found that significantly lower activities of AST, ALT, LDH and ALP were observed in rats whose diets were supplemented with cellulose fiber in comparison with obese rats. Moreover, many findings suggest that elevated ALT and AST activities are markers of liver damage and hepatic steatosis [31] but a hypocaloric diet rich in fibers causes reduced body weight and improved ALT and AST serum levels [32]. The present study was undertaken to ascertain the beneficial effects of cellulose on the oxidant /antioxidant status in liver mitochondria in obese rats before and during pregnancy as well as on their offspring (at day 90). Compared to controls, macrosomic rats presented higher MDA content accompanied by higher carbonyl protein levels, suggesting a decrease in those levels in groups fed the cellulose diet. Furthermore, research showed that a diet high in fat and carbohydrates induces a significant increase in oxidative stress (OS*)* and inflammation in people with obesity [33]. Obesity is attributed to disorders that affect the mitochondrial metabolism, which favors the generation of reactive oxygen species (ROSs*)* and the development of oxidative stress [34]. On the other hand, liver mitochondria catalase, SOD and GSH activities were found lower in HFD and HFDC groups in comparison with control rats fed the same diet. However, the antioxidant defense markers were lower, as this depends on the amount of body fat and central obesity [35]. When obesity persists for a long time, antioxidant sources can be depleted, thus decreasing the activity of enzymes such as superoxide dismutase (SOD) and catalase (CAT) [36]. These findings confirm that rats before pregnancy, rats at 90 days as well as their mothers show an altered oxidant and antioxidant status; similar results have been reported in obese children [37]. On day 90, the oxidant and antioxidant status in rats was found related to that of their mothers. It is well known that the oxidative stress (OS) is high during pregnancy. This alteration was found related to obesity; it would be worse when pregnancy and obesity are combined. Pregnancy and obesity are usually associated with higher oxidation levels [38].This study provides evidence that the oxidant/ antioxidant status is altered in obese subjects. It also indicates that cellulose fibers attenuate lipid and protein oxidation and preserve the anti-oxidation capacity.

CONCLUSION

The results of this study suggest that a cellulose fiber diet has beneficial effects on obesity and redox status. Therefore, dietary fiber interventions could present an opportunity for developing new strategies to treat obesity in woman before and during pregnancy and in their newborns.

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