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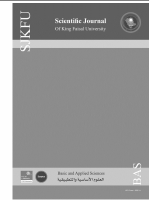

Effects of Brassica Rapa on Dyslipidaemia and Oxidative Damage in Rats Consuming a Hyperlipidic Diet

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Abstract

This research investigated the influence of a hyperlipidic diet in Wistar rats and the therapeutic effect of Brassica rapa powder (BrP). A hyperlipidic diet was administered followed by oral supplementation of BrP (5%) for 28 days. The BrP supplementation caused a decrease in weight (- 41%), glycaemia (-12%), creatinemia (-51%), aspartate aminotransferase (AST) level (-80%), and cholesterolaemia (-25%) associated with an increase in HDLc level (+59%). However, the hepatic lipid profile showed a decrease in phospholipids (PL) (-59%) and total cholesterol (TC) (-67%). In addition, oxidative stress assessment showed a decrease in the serum (-22%), renal (-30%) and adipose (-8%) levels of TBARS. Furthermore, BrP supplementation boosted glutathione (GSH) protective activity in adipose tissue with a maximum of +49%. In conclusion, Brassica rapa enhances the serum and tissue balance of metabolic and antioxidant status.

KEYWORDS
Antioxidant, biomarkers, fat, in vivo, redox status, turnip

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1. Introduction

Hypercholesterolaemia is a disorder characterized by abnormal cholesterol secretion and absorption into the blood, which can lead to a variety of diseases, such as inflammation, atherosclerosis, renal failure, non-insulin-dependent diabetes, and aging (Kim *et al.*, 2014). Biomolecules with hypocholesterolaemic and phytotherapeutic potential are of great interest to the health community who can prevent and treat dyslipidaemic disorders, because the medical prescription of certain hypolipidaemic medicines, such as statins and fibrates, can cause many side effects or intolerances (Jung *et al.*, 2008). The enzymatic antioxidant defence is also compromised by hyperlipidaemia, namely the activities of glutathione peroxidase (GSH-Px), catalase (CAT), and superoxide dismutase (SOD). Analogously, hyperlipidaemia may have a cause in the redox imbalance. Similarly, antioxidants may be able to prevent the development of hyperlipidaemia by correcting the early events that lead to redox imbalance. The species *Brassica rapa* (Br) or "turnip" is an excellent source of antioxidants and secondary metabolites (Yerou *et al.*, 2022). This plant species has many therapeutic properties, including inhibition of oxidative stress, induction of detoxification enzymes, reduction of cancer cell proliferation, inhibition of carcinogenic mutations, protection of the human body against free radicals, treatment of hepatitis, jaundice, furuncle and sore throat, and it also has immunostimulant and anti-diabetic effects (Kapusta-Duch *et al.*, 2012). These biological functions are due to the abundance of various bioactive substances, including phenylpropanoid derivatives, sterol glucosides (glucosinolates), flavonoids (glycosides of isorhamnetin, kaempferol, and quercetin), and indole alkaloids. Also, due to the synergistic action of glucosinolates, polyphenols, and triterpenes, considered the main complexes in cruciferous, and the most studied regarding its structure, mechanism of action and the effects in human cells (Šamec and Salopek-Sondi, 2019). Several researchers have highlighted the impact of Brassicaceae on cardiovascular pathologies (including hypertension and stroke) and dyslipidaemia (Raiola *et al.*, 2018), obesity (López- Chillón *et al.*, 2019), metabolic syndrome (Al-Snafi, 2015), type 2 diabetes (Chen *et al.*, 2018), osteoporosis (Dias, 2019), cancer (Thomas-Charles and Fennell, 2019), and antimicrobial (reduced microbial colonization), anti-inflammatory, antifungal, anti-parasitic, and antiviral activities (Ramirez *et al.*, 2020). In light of this data, this research aims to study the effects of a hyperlipidic (atherogenic) diet induced in vivo and the action of Brassica powder (BrP) supplementation by estimating blood metabolic and redox status.

2. Materials and Methods

2.1. Plant Matter:

The edible portion of *Brassica rapa* (Br), also known as turnip and frequently consumed by Algerians, was considered when selecting the plant. The fresh Br rhizome was obtained in November 2022 from the Ghriss local market, which is 19 km from the Mascara province in Western Algeria (latitude: 35°23'47.90"N, longitude: 0° 08' 24.97" E). Br rhizomes were prepared in the laboratory and then dried for an entire day at 38°C. Following drying, the plant material was ground into a powder to produce *Brassica rapa* powder (BrP), which was then stored for later use in hermetic glass containers.

2.2. Experimental Design:

The general recommendations for using live animals in scientific research by the Pasteur Institute, Algiers, were followed; likewise, Mustapha Stambouli University approved all the experimental procedures used in this study. The experiment was conducted on 30 male *Wistar* rats, weighing approximately 200±10g at two months of age. Table 1 shows the distribution of animal groups (n = 10) and the constituents of the standard and experimental diets. *Ad libitum* access to food and water was provided, and the protocols for the care and handling of laboratory animals were followed. In animal husbandry, the following standards were maintained: ambient temperature of 24°C, relative humidity of 60%, and 12 light: 12 dark hours. The body weight (Bw) of the animals was recorded once a

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week for 28 days, and their food intake was recorded every day.

Table 1: Composition of the standard and experimental diets (g/kg)

Composition	Standard diet	Experimental diet	
	CG	HFD	HFD-BrP (5%)
CAS	200	200	200
Sucrose	50	50	50
Corn starch	590	340	340
Cellulose	50	50	50
Vitamin mix	20	20	20
Mineral mix	40	40	40
Lipids (olive, nut, sunflower)	50	-	-
Lamb fat	-	300	300
BrP	-	-	50

CG: control group; HFD: High Fat Diet; HFD-BrP: High-Fat Diet supplemented with 5% of BrP

After a 12-hour fast on the 28th day of the experiment, the rats in each group were weighed and anesthetized via an intraperitoneal injection of a chloral solution (10%) at a rate of 3 ml/kg of Bw. An aortic puncture was conducted to obtain blood samples, which were then centrifuged at 1000 rpm for 20 min at 4°C (Sigma, 4K10 Bioblock Scientific, Germany). The serums were preserved in EDTA-Na2 (0.1%) (Merck, Germany). After twice successively washing and centrifuging the remaining pellet containing erythrocytes at 4000 rpm for 20 minutes at 4°C using NaCl (0.9%) (Merck, Germany), the cells were lysed by adding ice-cold water, incubated for 15 minutes in ice, and then recentrifuged. Kidneys, heart, liver, and adipose tissue were obtained, washed with an ice-cold NaCl (0.9%) solution, dried, and weighed; each organ was portioned and preserved at -80°C. All samples were stored until analysis.

2.3. Serum and Hepatic Biochemical Assays:

At sacrifice, a glucometer was used to measure the fasting blood glucose. Transaminase serums were estimated by the method of Reitman and Frankel (1957) using the Spinreact kit (Spain), serum total protein (Burtis and Ashwood, 1994) and serum albumin (Tietz et al., 1990) using the Chronolab kit (Switzerland). For the lipid balance, the Spinreact kit (Spain) was used: serum triglycerides (TG) (Burtis and Ashwood, 1994), serum total cholesterol (TC) (Richmond, 1973), and serum high-density lipoprotein (HDLc) (Lopes-Virella et al., 1977). The Friedwal formula was used to determine the serum low-density lipoprotein (LDLc): LDLc = TC- HDLc-(TG/5), serum creatinine (Schirmeister, 1964) and serum urea (Fawcett and Scott, 1960) using the Human kit (Germany), and serum uric acid (Friedewald et al., 1972) using the Biocon kit (Germany). All analyses were performed following the instructions of the kit supplier. A cold extraction of lipids on 1 g of sample was conducted in the presence of a chloroform/methanol mixture (Biochem chemopharma, UK), (4/1, V/V). The lipid extract was removed from the solvents under vacuum evaporation at 48°C (Büchi rotary evaporator, Germany). The total lipids were taken up in 10 ml of isopropanol for the analysis of various lipid fractions (Delsal, 1944). According to an enzymatic colorimetric method, total cholesterol (TC), triglycerides (TG) (Kit Spinreact, Girona Spain) and phospholipids (PL) (Kit Cypress, Belgium) were determined.

2.4. Oxidative Stress Status:

The method of Quintanilha et al. (1982) was used to determine the serum TBARS levels. First, 100 µl of the sample was diluted in 0.9 ml of NaCl (0.9%). After that, 20 µl of 2% butyl-hydroxy toluene (BHT) (Sigma-Aldrich Chemie, Germany) and 1 ml of TBA (15% TCA and 0.375% TBA in 0.5N HCl) were incorporated into this solution. Following 30 minutes of incubation at 100°C and ice cooling, the samples were centrifuged at 4000 x g for 10 minutes at 4°C. The measurement was made at 532 nm using spectrophotometry, and the results were presented as mmol/ml of serum. The protocol of Okawa et al. (1979) was followed to estimate the lipoperoxidation of the liver, heart, kidneys, and adipose tissue. We crushed 100 mg of tissue in 0.9 ml of 1.15% KCL. Likewise, 1.5 ml of acetic acid (20%, pH 3.5), 1.5 ml of tissue homogenate, 0.2 ml of a solution containing SDS (8.1%), and 1.5 ml of TBA (0.8%) constitute the reaction medium. After adding 4 ml of distilled water to adjust the final volume, the mixture was vortexed for 30 seconds and heated to 95°C for 1 hour. After adding 1 ml of distilled water and 5 ml of butanol, the tubes were agitated and centrifuged at 1000 x g for 10 minutes. Expressed as mmol/g tissue, the reading was obtained at 532 nm. The procedure described by Sedlak and Lindsay (1968) was used to analyse the reduced glutathione (GSH) in the tissue and erythrocyte samples. After preparing the homogenate in 0.1 M phosphate buffer (pH 6), 400 µl of cold distilled water, 100 µl of TCA 50%, and 500 µl of the sample (tissue homogenate, erythrocyte lysate) were mixed. After shaking, each sample was centrifuged at 1200 x g for 15 minutes. After centrifugation, 500 µl of supernatant was mixed with 100 µl of 0.01 M DTNB (5,5'-dithiobis (2-nitrobenzoic acid) and 400 µl of 0.4 M Tris buffer (pH 8.9). The absorbance at 412 nm was measured after 5 minutes of incubation. For erythrocytes and tissues, the results were expressed in µmol/ml and µmol/g, respectively.

2.5. Statistical Analysis:

Mean ± Standard Error (M±SE) with (n = 6) was used to express the results. In order to compare the means, students' tests were used (Statistica software, version 4.1, Statsoft, Tulsa, Oklahoma, USA), ^{a}CG vs HFD; ^{b}CG vs. HFD+BrP; ^{c} HFD vs HFD+BrP.

3. Results

3.1. Body and Organ Weight:

During the 28 days of experimentation, the two groups (CG and HFD) showed weight gain of 65.25 gr and 75.25 gr, respectively; however, after 28 days, we noted a stability in the weight of the HFD+BrP group with weight decreases of -41% and -27% comparatively with HFD ($p < 0.001$) and CG ($p < 0.01$), respectively (see Table 2).

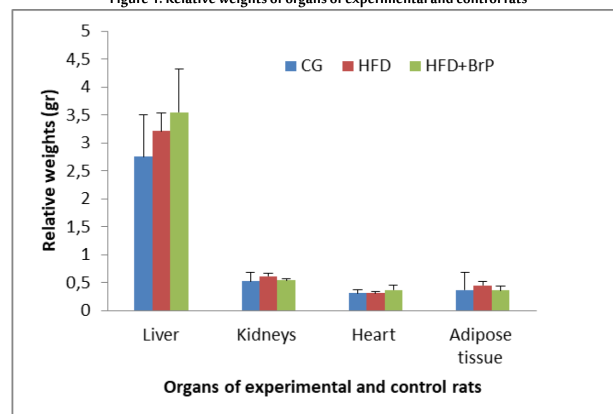
Table 2: Weight evolution of experimental and control rats

Group	Day 0	Day 7	Day 14	Day 21	Day 28
CG	160.25±27	188.375±29	202.06±25^{a**}	215.5±20^{a**}	225.5±21^{a**,b**}
HFD	200.25±20	228.38±25	252.06±32 ^{a**,c**}	265.50±19 ^{a**,c**}	275.50±23 ^{a**,c***}
HFD +BrP	199.67±29	198.67±24	192.83±15^{c**}	198.67±21^{c**}	164.00±24^{c***,b**}

CG: Control group; HFD: High-fat diet; HFD-Br: High-fat diet supplemented with 5% of BrP; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

The relative weight of the target organs is similar in all groups of rats (see Figure 1); the difference between the groups is statistically insignificant ($p > 0.05$).

Figure 1: Relative weights of organs of experimental and control rats



CG: Control group; HFD: High-fat diet; HFD-Br: High-fat diet supplemented with 5% of BrP

3.2. Serum and Hepatic Biochemistry:

The hyperlipidic diet caused hyperglycaemia, hyperuraemia, and hypercreatinemia with an augmentation in AST serum. In addition, a

disorder in the lipid profile was detected. BrP supplementation showed the following corrective actions: hypoglycaemia (-12%), hypocreatinemia (-51%), a strong decrease in AST serum level (-80%), hypochlesterolaemia (-25%), all of which are associated with an elevation in the HDLc serum level (+59%) (see Table 3).

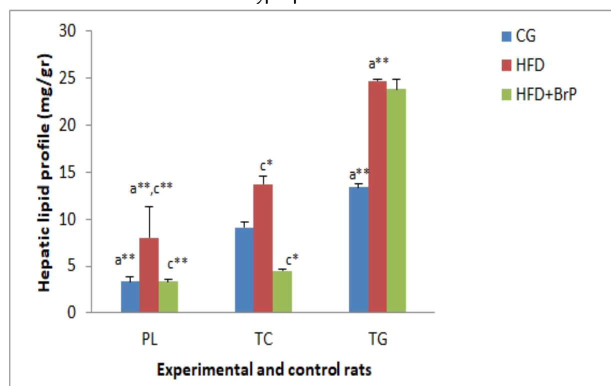
Table 3: Effect of BrP on serum glucose, kidney/liver biomarkers and lipid profile following 28 days of oral exposure of hyperlipidaemic rats

Parameters	CG	HFD	HFD+ BrP
Blood glucose (mg/dl)	258.69±31^{a**}	345.48±17.5 ^{a**,c*}	305.40±11.8^{c*}
Kidney biomarkers			
Urea (mg/dl)	7.21±0.91^{b*}	6.82±0,81^{c*}	5.31±1.29 ^{b*,c*}
Creatinine (mg/dl)	0.64±0.31^{a**}	1.24±0.76 ^{a*,c*}	0.60±0.27^{c*}
Uric acid (mg/dl)	4.58±1.4	6.67±2.5	6.10±1.8
Liver biomarkers			
Total proteins (g/dl)	7.23±2	7.66±0.81	8.55±1.44
Albumin (g/dl)	2.35±0.96	2.92±0,36	2.94±0.72
AST (UI/l)	106.8±29 ^{a**,b**}	256.7±11.9 ^{a**,c***}	50.8±12 ^{b**,c***}
ALT (UI/l)	67.81±29	83.42±39	70.00±31
Lipid profile			
TG (mmol /L)	1.66 ±0.07^{b}	1.14 ± 0.04 ^{c}	0.65 ± 0.02^{b*,c*}
TC (mmol /L)	1.41 ± 0.41^{a}	2.54 ± 0.64^{a**,c*}	1.89 ±0.53^{c}
HDLc (mmol /L)	0.78±0.33^{a*}	0.35 ±0.13^{a*,c**}	0.84 ± 0.33^{c**}
LDLc (mmol /L)	1.22 ±0.60	1.64 ±0.13	1.47 ±0.63

CG: Control group; HFD: High-fat diet; HFD-Br: High-fat diet supplemented with 5% of BrP; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; TC: Total cholesterol; TG: Triglycerides; HDLc: High-density lipoprotein cholesterol; LDLc: Low-density lipoprotein cholesterol; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.01$

The hepatic concentrations of PL, TC, and TG increased significantly in the HFD group, according to our findings. However, we also noticed that PL (-59%) and TC (-67%) levels decreased significantly in the BrP supplementation group compared to the HFD group (see Figure 2).

Figure 2: Effect of BrP on the hepatic lipid profile following 28 days of oral exposure in hyperlipidaemic rats



CG: Control group; HFD: High-fat diet; HFD-Br: High-fat diet supplemented with 5% of BrP; TC: Total cholesterol; TG: Triglycerides; PL: Phospholipids; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

3.3. Serum, Erythrocyte and Tissue Oxidative Stress Biomarkers:

According to the results in Table 4, the hyperlipidic diet caused a significant increase of TBARS in serum (+29%, $p < 0.05$) and renal tissue (+43%, $p < 0.01$) compared to HFD+BrP. Also, in adipocytes compared to CG (+32%, $p < 0.01$) and HFD+BrP (+9%, $p < 0.05$), the protective action of BrP decreased the amount of TBARS in serum (-22%, $p < 0.05$), renal (-30%, $p < 0.01$) and adipose tissue (-8%, $p < 0.05$) compared to HFD. In addition, BrP supplementation enhanced GSH protective activity in adipose, with an increase of +35% ($p < 0.05$) and +49% ($p < 0.05$) compared with HFD and CG, respectively.

Table 4: Effect of BrP on serum, erythrocyte and tissue oxidative stress biomarkers following 28 days of oral exposure in hyperlipidaemic rats

Parameters	CG	HFD	HFD+ BrP
Serum TBARS (µmol/l)	**5.7±0.9**	**6.7±1.9 ^{c*}**	**5.2± 0.6c***
Tissue TBARS (µmol/gr)			
Liver	115.38±24.43	131.05±18.5	114.67±24
Heart	86.16±10.06	90.33±4.9	95.07±28
Kidneys	116.08±37.1	121±22.3^{c**}	84.75±32.1^{c**}
Adipose tissue	113.30±9.89^{a**}	149.54±29.7 ^{a**,c*}	137.53±42.4^{c*}
Erythrocytic GSH (µmol/l)	0.58±0.12	0.66±0.35	0.70±0.29
Tissue GSH (µmol/l)			
Liver	49.72±9.87	50.15±6.51	48.04±1.82
Heart	51.22±1.18	60.88±3.04	66.56±2.50
Kidneys	53.38±2.45	59.63±1.53	59.21±1.77
Adipose tissue	52,88±7.77^{b*}	58.28±2.58^{c*}	78.71±1.340 $^{b*, c*}$

CG: Control group; HFD: High-fat diet; HFD-Br: High-fat diet supplemented with 5% of BrP; TBARS: thiobarbituric acid reactive substances; GSH: Reduced glutathione; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

4. Discussion

The hyperlipidic diet in animals decreases the rate of satiety, induces increased weight gain, stimulates lipogenesis in visceral adipose tissue, and causes insulin resistance and lipid abnormalities. In this work, BrP supplementation appeared to decrease and stabilize the body weight of the animals. This indicates that BrP has a satiety effect that may boost the β3-adrenergic receptor in β3-AR-dependent lipolysis and limit the formation of lipids in fatty tissue cells (An *et al.*, 2010). In addition, a significant weight reduction was detected in the rats with fructose-induced metabolic syndrome supplemented with a hydroalcoholic extract of Br (400 mg/kg/day), with an elevation in hepatic glycogen concentration, compared to the reference group (metformin, 10 mg/kg/day) (Abo-youssef and Mohammed, 2013). The hyperglycaemia observed in our study following a hyperlipidic diet was due to a decrease in insulin excretion by the pancreas accompanied by polyuria and glycosuria (Ghasemi *et al.*, 2014), whose pathogenesis is characterized by insulin resistance, pancreatic β-cell dysfunction and apoptosis. In this context, the quantity and quality of dietary fat can modify glucose tolerance and insulin sensitivity. A high fat content in the diet leads to the deterioration of glucose tolerance through a number of mechanisms, including reduced insulin receptor binding, impaired glucose transport through reduced expression of transporter 4 (GLUT4), reduced glycogen synthase levels, and accumulation of triglycerides in skeletal muscle. The fatty acid composition of the diet influences the composition of tissue phospholipids, which can be linked to insulin action by altering membrane fluidity and insulin signalling. In addition, saturated fatty acids increase hepatic triglycerides, insulin resistance and harmful ceramides. Thus, it has been reported that the consumption of saturated fatty acids (SFAs) leads to a risk of developing type 2 diabetes (Micha and Mozaffarian, 2010). This increase of glycaemia was corrected by the administration of 5% BrP, which is in agreement with numerous studies confirming the antihyperglycemic effect of BrP in diabetic rats, as Br can boost insulin secretion and block intestinal glucose absorption (Amrita-Bhowmik *et al.*, 2009). Our data supports the pathogenic role of dyslipidaemia in the apparition and development of renal disease, which is explained by an increase in creatinine serum. According to Hattori *et al.* (1999), a high-fat diet causes an infiltration of macrophages in the kidneys, which leads to glomerulosclerosis. Administration of BrP normalized the creatinine level in hyperlipidic rats. This is consequent of the richness of Brassicaceae by anthocyanins, antioxidants and free radical scavengers.

A perturbation in liver enzyme activity is possibly an indication of liver injury or damage. However, triglyceride accumulation inside hepatocytes caused lipotoxicity due to oxidative stress inside the hepatocytes (Lim *et al.*, 2012). This correlates with our study in which an augmentation in AST was noted. BrP treatment caused a reduction in liver enzyme activity, which could be attributed to its ability to balance lipoperoxidation and cure lysed cells.

This investigation confirms the development of a lipid profile perturbation characterized by elevated triglyceride serum levels and total cholesterol and decreased HDLc. However, HDL prevents LDL from oxidation and prevents the development of atherosclerosis. Nevertheless, the hyperglycaemia previously detected in rats can causes HDL glycation and damages its protective functions, which can lead to atherosclerosis genesis. BrP administration improves metabolic disorder by inducing HDLc synthesis and blocking key enzymes of cholesterol and triglyceride synthesis because the high presence of flavonoids (quercetin), glucosinolates and coumarins actively participate to reduce hepatic fat and triglyceride levels (Soliman *et al.*, 2016).

Lipoperoxidation mainly attacks polyunsaturated fatty acids (PUFAs) of the biological membrane and perturbs the biological function of membrane proteins via receptor inactivation and protease enzyme activation, leading to an alteration in membrane permeability with cellular damage. Likewise, hyperglycaemia causes lipoperoxidation by stimulating the glycation and polyol pathways, which leads to the strong generation of free radicals. Our findings support previous research in rats with metabolic syndrome and diabetics treated with BrP, which shows that HFD elevates serum and tissue (renal and adipose) levels of TBARS, while treatment with BrP diminishes TBAR levels (Feillet-Coudray *et al.*, 2009). Moreover, Kim et al. (2006) reported that Br hydroalcoholic extract minimizes the damage caused by oxidative stress. The mechanism for these corrective actions can be attributed to quercetin, which is known to reduce TBAR levels via free radical-scavenging, inhibiting oxidative degradation, and metal-chelating. Furthermore, Brassica's antiradical activity is associated with the presence of a high concentration of secondary metabolites that can scavenge singlet and triplet oxygen or decomposing peroxides, which neutralize reactive oxygen species (ROS) (Fresco *et al.*, 2010). The natural non-enzymatic antioxidant system relies largely on glutathione, which protects cells from oxidative damage. The enzyme glutathione peroxidase functions primarily as a reducing agent and helps remove hydrogen peroxide. Our research confirms an enhancement of GSH activity in adipose tissue in the BrP-supplemented group, confirming the antiradical activity of BrP. Our results are consistent with those of Abo-youssef and Mohammed (2013), who confirmed an augmentation of GSH in rats with metabolic syndrome and treated with 70% ethanolic extract of Br.

Furthermore, this research has classic limitations. Due to the short duration of the study, it would be desirable to extend the duration of the investigation (subchronic exposure, more than 3 months). The route of administration by gavage for each animal would give greater exactitude and more reliability. The size of the sample was small; therefore, increasing the number of groups and doses will permit reproducibility. Finally, for a better understanding of the therapeutic effect of BrP, it would be preferable to study other parameters such as target organ histophysiology, rat neurobehavior and other biomarkers of oxidative stress.

5. Conclusion

Our results show that the hyperlipidic diet caused obesity and metabolic abnormalities associated with renal and adipose lipoperoxidation in the hyperlipidic rats. Moreover, these perturbations were corrected by *Brassica rapa* powder supplementation (significant antihyperglycaemic, antihyperlipidaemic, renoprotective, hepatoprotective and defensive effects). *Brassica rapa* contains promising molecules for the prevention and potentialisation of treatments for metabolic pathologies (obesity, diabetes, dyslipidaemia). In conclusion, these favourable biological actions associated with *Brassica rapa* in attenuating metabolic and oxidative disorders induced by a hyperlipidic diet may represent an interesting advance in the search for new therapeutic agents against this dyslipidaemia. We strongly suggest introducing *Brassica rapa* into the diet of patients with metabolic disorders as a food or dietary complement. Finally, we would like to point out that these results can only be extrapolated to humans after long-term studies on several animal species.

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