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Original Article

Sulforaphane ameliorated oxidative stress, inflammatory release and obesity hormones abnormalities induced by high fructose and or high fat diet in rats

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Abstract

The undesired complications and side effects of drugs used in obesity management is the main challenge worldwide. For that, complementary or alternative therapy depending on natural source for obesity management is the target of nutritionist for its safety. The rational of this study to investigate the role of sulforaphane (SR) in amelioration biochemical markers related to oxidative stress, inflammation and obesity hormones high fructose and or high fat diet fed rats. Eight groups of male rats (6 rats/group). Group 1: Rats fed normal diet. GP II: normal rats given SR (10 mg/kg bw). Animals in groups (III-V) were fed either high fructose (HF) or high fat (HFA) or (HF/HFA) diet. However, rats in Groups (VI-VIII): are similar to previous groups but treated orally 10 mg/kg bw SR. Data obtained showed that, SR protected from hepatic abnormalities by normalizing ALT and AST levels. A significant improvement in insulin sensitivity, restoring changes in lipid profile (TC, TG, LDL-c and HDL-c), anti-inflammatory by reducing levels of IL-6 and TNF-α, antioxidant and keeps obesity hormones from alterations compared with untreated rats. Conclusion: supplementation of SR ameliorated biochemical abnormalities induced by HF and or HFA and related hormonal, oxidative stress and inflammations. For that, it is promising to be used in the treatment of obesity-related metabolic syndrome.

Keywords: Sulforaphane, Obesity, High fructose/ high fat, Ghrelin, Leptin, Adiponectin, Rats

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Introduction

The prevalence of obesity in the world population has been rising rapidly, emerging as a major health problem. Obesity contributes to the increased risk of other metabolic disease as diabetes, CVD, and certain types of cancer, thereby increasing global mortality (Morgen and Sorensen 2014; Shah et al. 2014). Ghrelin is known as a peptide hunger hormone that play a role in the regulation of body weight and energy balance, it increases food intake and stimulate accumulation of fats (Santiago-Fernández et al., 2017). Adipocytes produce endocrine signals that modulate cytokine secretion and signaling to the brain (Guilherme et al., 2019). Adiponectin is a peptide hormone produced by adipose tissue, showed anti-diabetic, anti-inflammatory, and enhance insulin sensitivity and causes weight loss (Achari and Jain, 2017). Leptin is a hormone that enhances energy utilization and satiety control (Mitrou et al., 2011). The obesity is distributed in all ages young, adolescents and old (Mirmiran et al., 2010). Due to low energetics source, cruciferous can be used in weight management. The isothiocyanates including (sulforaphane and Phenyl Ethyl Isothiocyanates), are the mani active components of cruciferous that impact on metabolism. In addition, sulforaphane protect against diabetes induced in rats (Song et al., 2009; de Souza et al., 2012). Not only sulforaphane but also other isothiocyanates which have common structures "-N=C=S", play a crucial role in weight control. It was reported that SR exhibited multifunction against many chronic diseases such as neurodegenerative abnormalities and cancer (Hua et al., 2022). In addition, it exerts hepatoprotective and cardioprotective activities (Guan et al., 2021; Song et al., 2020). The action of SR was mediated through activation of Nrf-2 pathway which controls antioxidant activity (Uddin et al., 2020). Identification of natural products is attention of many scientist for development safe anti- obese agent (Thounaojam et al., 2013; Torres-Fuentes et al., 2014). The rational of this study the role of SR in amelioration obesity related biochemical markers (inflammatory mediators, antioxidant, hormonal changes) in HF and or HFA diet fed rats.

Material and Methods

Animals and experimental design

Forty-eight male Wistar albino rats weighting (110-140 g) were used in this study. They were housed under standard condition and allowed water and standard pellet diet *ad libitum*. The experiment was carried out according to guidelines of committee for experiment animal care and handling at King Abdulaziz University. The animals were randomly divided into eight groups of six animals each. Group 1: Rats fed normal diet as control. Animals in groups (II-IV) were fed either high fructose (HF) or high fat (HFA) or (HF/HFA). However, rats in Groups (V-VIII): are similar to previous groups but treated orally 10 mg/kg bw sulforaphane (SR) (dissolved in saline). The dose of SR was given according to (Okulicz and Hertig, 2016). The feeding and treatment were done for 12 weeks. Normal diet (22% protein, 43% carb, and 4% fats). HFA (15% protein, 24% carb, and 57% fats). At the end of the experiment. Blood was collected and serum was separated for biochemical assays. Glycated hemoglobin (HA1c) was determined in fresh blood samples, fasting blood sugar, insulin, HOMA-IR, liver function tests (ALT, AST), lipid profile (total cholesterol, triglycerides, LDL-c, HDL-c), inflammatory markers (IL-6 and TNF-α), were evaluated in serum samples. Plasma levels of Ghrelin, adiponectin and leptin were also evaluated by ELISA kits. The liver was excised immediately and homogenized in ice-cold 0.1M Tris-HCl buffer. The homogenate was used for estimating superoxide dismutase (SOD), reduced glutathione (GSH), and glutathione reductase (GRase), malondialdehyde (MDA) by colorimetric methods using commercial kits.

Histological examination

Samples of liver tissue were stored in 10% formalin. Slices of tissue were obtained by microtome, stained with hematoxylin and eosin and examined with light microscope using 400X magnification.

Statistical analysis

All values were expressed as the mean ± SE using SPSS version 20. The Tukey post hoc test was performed to examine whether there is a significant difference between different treatment groups or all analyses, the value of $P < 0.05$ was considered significant.

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Results

The body weight changes presented in table (1) revealed the weight gain in rats fed normal diet, HF, HFA, HF/HFA untreated and treated with SR. A significant increase in body weight gain in HF, HFA or HF/HFA diet compared with normal diet. Treatment with SR caused a significant reduction ($P<0.001$) versus untreated but not returned to normal. It was found that the levels of FBG, HA1c, insulin and HOMA-IR (figures1-4) were statistically increased in rats fed HF/HFA diet versus control ($p < 0.001$).

Table-1. Changes of body weight during experiment (Mean ±SD).

Parameters	Control	Control +SR	HF	HFA	HF/HFA	HF+SR	HFA+SR	HF/HFA+SR
Initial body weight (gm)	110±8.7	122±8.5	125±11	130±10.5	140±9.9	136±12	138±13	127±14
Final body weight (gm)	230±19[a]	215±22[a]	280±26[a]	305±28[a]	310±28[a]	220±25[a,b]	231±19[a,b]	250±21[a,b]
Weight gain %	109%	76%	124%	134%	121%	61%	67%	96%

(a) significant versus control, (b) significant versus SR.

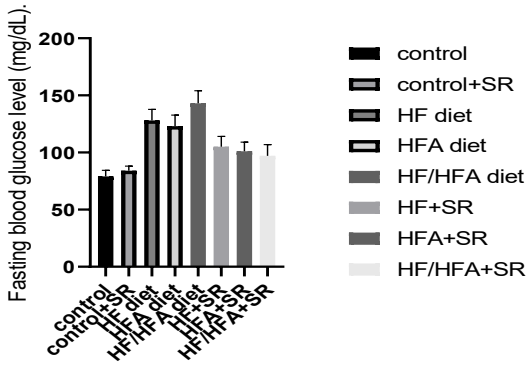

Figure-1. Fasting blood glucose level (Mean ±SD).

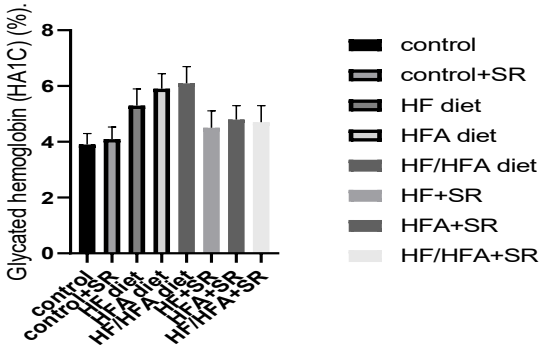

Figure-2. Glycated hemoglobin level in studied groups (Mean ±SD).

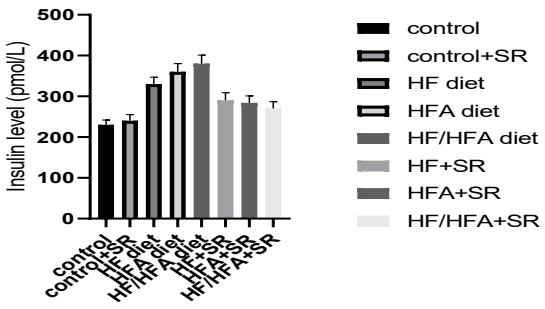

Figure-3. Insulin level in all studied groups (Mean ±SD).

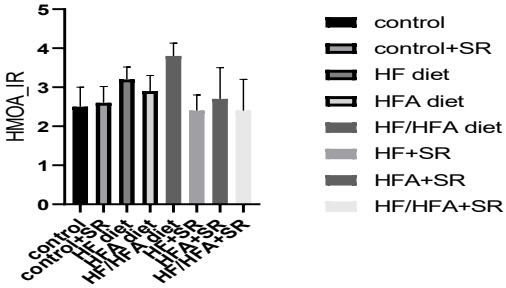

Figure-4. HOMA-IR-changes in all studied groups.

However, treated rats with SR caused a significant reduction (p< 0.001) compared with untreated and HOMA-IR was recovered but not reached to normal range. Liver enzymes (ALT and AST) were significantly elevated in HF, HFA or HF/HFA diet versus control. Treatment with SR prevented this elevation (figure 5,6). Data obtained in table 2 indicated a statistical increase in lipid profile (T. cholesterol, TG, LDL-c) and reduction in HDL-c in HF/HFA fed diet rats compared with normal rats (p<0.001). Rats treated with SR revealed lowered cholesterol, LDL-c and increased HDL-c compared with untreated. Results obtained found that HF/HFA fed rats results in a statistical elevation in MDA level and decreased in activities of glutathione reductase and glutathione peroxidase (p < 0.001) in comparison with control (table 3).

Table-2. Serum lipid profile in all studied groups (Mean ±SD).

Parameters	**Control**	**Control +SR**	**HF**	**HFA**	**HF/HFA**	**HF+SR**	**HFA+SR**	**HF/HFA+SR**
T. cholesterol(mg/dL)	144±5.7	137±6.7	190±18.1a	250±17 [a]	260±19.1 [a,b]	171±13.8[a,b]	210±19 [a,b]	190±20 [a,b]
Triglyceride (mg/dL)	130±11	128±10	185±19	196±16	198±16[a,b]	156±12.4,[b]	167±17 [a,b]	172±14.5 [a,b]
LDL-c(mg/dL)	60± 2.4	64±3.1	99±8.4 [a,b]	115±12.1[b]	130±7.2[a,b]	115±6.2 [a,b]	98±9.1[b]	87±7.2[a,b]
HDL-c (mg/dL)	39± 3.2	44±4	29±1.1 b	32±2.2 [b]	30±2.1 [b]	33±2.8 [b]	35±1.3 [b]	38±2.9 [b]

(a) significant versus control, (b) significant versus untreated with SR.

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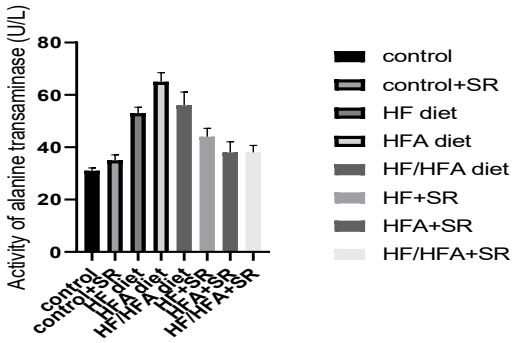

Figure-5. Activity of alanine transaminase (Mean ±SD).

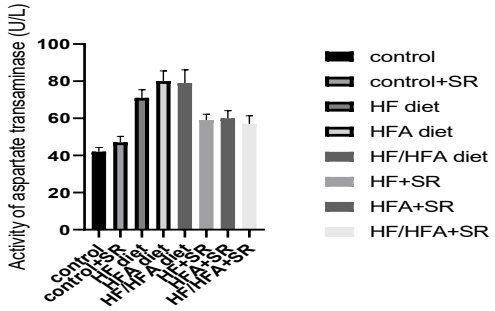

Figure-6. Activity of aspartate transaminase (Mean ± SD).

Table-3. Oxidative stress markers in the liver tissue of studied groups (Mean ± SD).

	MDA (µmol/mg protein)	**GSH (µmol /mg protein)**	**SOD (U/mg protein)**	**GSRase (U/mg protein)**
Control	32 ± 2.3	22 ± 1.1	780 + 65	1112 + 154
Control +SR	36 ± 2.9	20 ± 1.1	821 ± 64	1200 ± 172
HF	65 ± 4.5 a	11 ±1 a,b	510 ± 54 a,b	870 ± 46 a,b
HFA	71 ± 6.1 a,b	8 ± 0.4 a,b	449 ± 47 a,b	790 ± 61 a,b
HF/HFA	78 ± 6.7 a,b	10 ± 1.2 a,b	470 ± 41 a,b	672 ± 45 a,b
HF+SR	52 ± 5.1 a,b	15 ± 3.1 a,b	621 ± 60 a,b	930 ± 89 a,b
HFA+ SR	55 ± 4.7 a,b	13 ± 2a,b	750 ± 71 a,b	880 ± 76 a,b
HF/HFA+ SR	65 ± 5.5 ^{a,b}	16 ± 1.6 ^{a,b}	800 ± 71 ^{a,b}	944 ± 60 ^{a,b}

(a) significant versus control, (b) significant versus SR. SR: Sulforaphane, MDA: Malondialdehyde, GHS: reduced glutathione, SOD: superoxide dismutase, Grase: glutathione reductase.

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However, treatment with SR enhances activities of glutathione reductase and glutathione peroxidase and lowers MDA level. Data in (figure 7,8) showed that rats fed HF or HFA or HF/HFA a significant elevation in the levels of IL-1β, TNF-α levels in liver tissue versus control (p <0.001). Treatment with SR reduced their (p <0.001) versus untreated rats. Table (4) showed changes in serum obesity hormones (Ghrelin, leptin and adiponectin). It was found that, Ghrelin and adiponectin levels were significantly decreased while leptin level increased (p<0.001) in HF, HFA and HF/HFA died versus normal diet. Treatment with SR resulted in a significant elevation in Ghrelin and adiponectin levels and reduction in leptin level versus untreated (p<0.001).

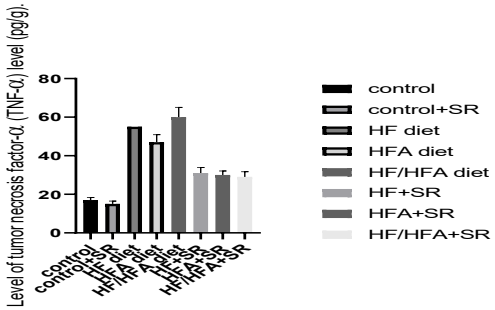

Figure-7. The level of TNF-α in all studied groups (Mean ±SD).

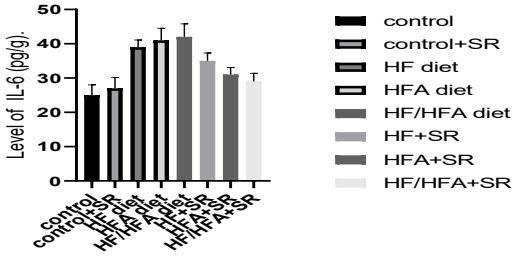

Figure-8. The level of IL-1β in all studied groups (Mean ±SD).

Table-4. Levels of Ghrelin, leptin and adiponectin of studied groups (Mean ± S.D).

	Ghrelin (ng/mL)	**Leptin (pg/mL)**	**Adiponectin (ng/mL)**
Control	3.2 ± 0.4	336 ±34	110 +9.8
Control +SR	3.3 ± 0.33	327±44	112 ±9.9
HF	2.8 ± 0.8 [a]	345 ±31 [a,b]	89 ±8.1 [a,b]
HFA	2.6± 0.7 [a,b]	350±28 [a,b]	86 ±6.8 [a,b]
HF/HFA	2.2± 0.4 [a,b]	355± 23 [a,b]	80 ±7.1 [a,b]
HF+SR	2.9± 0.5 [a,b]	331 ± 35 [a,b]	93 ±8.1 [a,b]
HFA+ SR	2.7± 0.7[a,b]	333 ±41[a,b]	90 ±9 [a,b]
HF/HFA+ SR	2.8± 0.3 [a,b]	329 ± 31 [a,b]	93 ±9.2 [a,b]

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Discussion

Natural products are expected to improve abnormality as lifestyle-related diseases. This is supported by the development of functional foods targeting metabolic syndrome. Obesity is produced from high fat cell number and size of adipose tissue that led to predisposing of many diseases as metabolic syndrome (Silva et al., 2024). The isothiocyanate compounds including SR are the main active ingredients in cruciferous vegetables and exert different biological activity as anti-cancer (Luo et al., 2024). This study investigated the anti-obesity activity of SR and its mechanism of action, to combat obesity and related metabolic disease

In current study, rats fed on HF or HFA or HF/HFA diet showed a significant weight gain throughout the experimental period compared to rats fed normal diet (comparison initial and final weight). The increase in body weight may be due to hyperphagia caused by increased energetic diet. High energetic diet causes hyperphagia that leads to obesity (Sjostrom et al., 1998). Treatment with SR caused a significant decrease in body weight versus untreated. This may be due to suppressing appetite (Ioannides-Demos et al., 2005). Previous work found that, intraperitoneal injection of SR (10 mg/kg) for 30 days reduced body weight in female mice fed HFD (Liu et al., 2021). Suggesting that, SR may stimulate weight loss via fatty acid oxidation and cellular energy expenditure. In the current study, rats fed on HF or HFA or HF/HFA led to elevated serum lipid profile (TG, TC and LDL-c) and reduction of HDL-c level versus normal diet fed rats. This is in agreement with our data, the effects of green tea and citrus derived polyphenols in humans were investigated (Yoshitomi et al., 2021). Elevated cholesterol levels can cause several diseases such as atherosclerosis and stroke. However, there was a significant reduction in TC, TG and LDL-c levels in rats treated with SR compared with HFD untreated. The SR may cause inhibition of lipase enzyme activity that affects intestinal fat absorption and proved as useful medication for the management of hyperlipidemia and promise used as anti-obese. Also, previous study, HFA diet was used to stimulate insulin resistance, hyperinsulinemia, and hyperlipidemia (Bastard et al., 2006). SR showed its ability in glycemic control as indicated by lowering FBG, insulin and HOMA-IR. Also, SR treated rats significantly improved glucose and insulin sensitivity (Wu et al., 2022). It was reported that, supplementation of dried broccoli rich with SR reduced blood pressure and improved glucose level in (Noble et al., 2022). Rats fed on HF or HFA of HF/HFA diet showed a significant elevation in level of MDA and reduction of GSH, SOD and GRase versus normal rats. However, treatment with SR improved antioxidant capacity and decreased MDA level. Similar data obtained, pretreatment with SR (5mg/kg) decreased oxidative stress and mitochondrial dysfunction markers in rats (Folbergrová et al., 2023). Previous study showed that, increase in the level of SOD activity after SR treatment compared with the control. In addition, SR may exert direct antioxidant effect and, in this way, protects the antioxidant enzymes (Shoenberg and Beger, 1993). Oxidative stress is associated with metabolic and physiological abnormalities and accompanied with increased lipid peroxidation and release free radicals. In addition, reduced antioxidant potential enzymes (SOD, GRAse and GSH) (Kaynar et al., 2005). The data obtained deduced the association of HF/HFA diet with lipid peroxidation as indicated by elevated MDA. On the other hand, SR exerts its antioxidant potential by decreasing MDA and elevating antioxidant enzymes.

SR significantly enhanced the translocation of GLUT4 (Xu et al., 2018), which stimulate glucose uptake in skeletal muscle. As the adipose tissue produces adiponectin, leptin, and TNF-α (Pogodzi´nski et al., 2022), it is considered as an endocrine organ. Adipose tissue contains macrophages that cause inflammations in obesity (Berger et al., 2017). In this study, rats fed on HF or HFA of HF/HFA diet showed a significant elevation in levels of TNF-α and IL-6 versus normal. Treatment with SR reduced their levels significantly compared with untreated. It was found that, SR treated animals fed HF or HFA or HF/HFA significantly reduced the level of TNF-α and IL-6. This indicated the inflammatory response was improved in treated versus untreated. In addition, adiponectin improves metabolic syndrome via normalizing the lipid profile, glucose level and HOMA-IR sensitivity (Ntzouvani et al., 2016). In current study, rats fed on HF or HFA of HF/HFA diet showed a significant reduction in gherlin and adiponectin levels versus normal diet. However, SR normalize significantly these abnormalities. Ghrelin, a hormone, elevated in fasting and is decreased in fed state. In obesity, level of ghrelin is reduced and not changed in fed (Inui et al., 2004). Adiponectin is a hypoglycemic hormone that enhances insulin sensitivity. In obesity, adiponectin level lowered

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significantly results in abnormalities in metabolism (Nigro et al., 2014). It was reported that, the adiponectin was reduced in insulin-resistant and elevated in insulin sensitivity after loss weight (Sethi et al., 2018). However, leptin counteracts effect of ghrelin. The leptin was found to enhance insulin sensitivity (Munzberg and Myers, 2005). Serum leptin reduced in fasting, dieting, or uncontrolled T1DM (Feng et al., 2013).

The energy balance hormones in the body that control food intake and metabolism include (Leptin and ghrelin). Leptin inhibits intake of foods and weight control (Haslam and James, 2005). However, the ghrelin helps with food intake. Abnormal ghrelin secretion led to obesity (Hamann and Matthaei, 1996). Our data in parallel with that reported a significant decrease in ghrelin levels and elevation in leptin levels in response to HF/HFD versus normal. The decreased level of leptin in rats given SR spot in reduction fat content and obesity (Faggioni et al., 2000).

Conclusion

Supplementation of SR ameliorated HF/HFD-induced obesity related abnormalities such as insulin resistance, dyslipidemia, anti-inflammatory, antioxidant and obesity hormones. For that, it is promising that SR is used in the treatment of obesity-related metabolic disease.

Study Limitations

No arability for immunohistochemistry of target tissue as adipose tissue and liver.

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Disclaimer: None.

Conflicts of Interest: None.

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Data Availability Statement

All data generated or analyzed during this study are included in this published article [and its supplementary information files].

Informed Consent Statement

Not applicable.

Contribution of Authors

Kumosani TA, Barbour EK & Moselhy SS: Conceptualized the idea for the study, designed research methodology and edited the manuscript.

Yaghmoor SS: Conducted experiments, collected data and edited the manuscript.

Moselhy SS: Analyzed and interpreted data and wrote the first draft of manuscript.

All authors read and approved the final draft of the manuscript.

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