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CALORIC RESTRICTION OR PURSLANE EXTRACT ALLEVIATE HIGH FAT DIET EFFECTS ON THE RELEASE OF PRO-AND ANTI INFLAMMATORY CYTO- OR ADIPOKINES IN OBESE RAT

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Obesity, Cytokines, Adipokines, Systemic inflammation.

Abstract

Obesity is a medical condition characterized by excessive body fat with high body mass index (BMI), which leads to serious impairment of health. The pathological conditions associated with excess weight may be related to adiposity-induced inflammation as obesity is characterized by a state of chronic low grade inflammation accompanied with altered circulatory levels of inflammatory mediators (Inflam Med) such as cytokines and adipokines .Therefore ,the present study was designed to investigate the effects of caloric restricted diet (Rest) or purslane (portulaca oleracea L) extract (purs ext) on high fat diet (HFD)induced obesity and changes in the levels of some Inflam Med including the pro-inflammatory IL-1β, IL-6, IL- 12 , TNF-α and resistin,and the anti-inflammatory IL-2 and adiponectin in rats. Fifty mature male rats were divided into 5 groups (10 animals each). 1st fed on normal diet, 2nd fed on HFD for 18 weeks, 3rd fed on HFD for 18 weeks then caloric restricted diet for 4 weeks (Rest-4), 4th fed on HFD for 18 weeks then caloric restricted diet for 8 weeks (Rest-8) , 5th fed on HFD and gavaged daily 1g/kg BW of purs ext (HFD + purs ext) for 18 weeks. Induced obesity caused an increase in the anthropometric measures [final BW,BMI and abdominal circumference(AC)], serum levels of IL-1β, IL-2, IL-6, IL-12 , TNF-α and resistin, and in contrast , a decrease in serum adiponectin level. These HFD induced disturbances were for some extent alleviated in rats fed on caloric restricted diet or orally supplemented with purslane extract, indicating similar positive efficacy of both to protect the body against the inflammatory response to obesity.

Introduction

Obesity is a medical condition characterized by excessive body fat with high body mass index (BMI), which leads to serious impairment of health (**WHO, 2014**). Excess body fat increases the risk of cardiovascular disease, hypertension, hepatic disease related to obesity, namely the nonalcoholic steatohepatitis (NASH) and the nonalcoholic fatty liver disease (NAFLD), type 2 diabetes, and may contribute to a decline in cognitive abilities, and dementia (**Clark, 2006 ; Ikeoka *et al*., 2010; Rizvi, 2010**). All of the pathological conditions associated with excess weight may be related to adiposity-induced inflammation as obesity is characterized by a state of chronic low grade inflammation accompanied with altered circulatory levels of inflammatory mediators (Inflam Med) such as cytokines and adipokines in obese subject(**Zeyda *et al*.,2007, Vachharajani and Granger, 2009, Leon-Cabrera *et al*., 2013, Duburcq *et al*., 2014; Kornblith *et al*., 2015 ; Ohkura *et al*., 2015**).

Cytokines are a broad category of small proteins that are important in cell signaling and regulation of host responses to infection, immune responses and inflammation.They are produced by various types of cells, including immune cells like macrophages, B lymphocytes, T lymphocytes and mast cells, as well as endothelial cells, fibroblasts, and various stromal cells; a given cytokine may be produced by more than one type of cell (**Stedman, 2006 ; Lackie, 2010**).These cytokines include interleukines and inflammation-related adipokines such as

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resistin ,adiponectin and TNF-*α* **(Wang and Nakayama, 2010).** Cytokine production is one of the first steps of the immune response and can provide important information regarding the nature of any immunotoxic responses **(Ai *et al*., 2013 , Ibrahim *et al*., 2015)**. Some cytokines are pro-inflammatory , that tend to make the disease itself or its symptoms worse by causing fever, inflammation, tissue destruction, and in some cases, even shock and death.They are produced predominantly by activated macrophages and are involved in the up-regulation of inflammatory response There is abundant evidence that certain pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α are involved in the process of pathological pain (***Zhang and An, 2007)*** . Other cytokines are anti-inflammatory, serve to reduce inflammation and promote healing (Dinarello 2000).They represent a series of immune-regulatory molecules that control the pro-inflammatory cytokine response (**Opal and De Palo , 2000**) . A balance between pro-inflammatory and anti-inflammatory cytokines is necessary to maintain health (**Dinarello,1997 ; Cavaillon 2001**).Major pro-inflammatory cytokines include IL-1β, IL-6,IL-12 and TNF-α while IL-2 is considered important anti-inflammatory cytokines. IL-6 is primarily considered to be pro-inflammatory although it also possesses anti-inflammatory capacities **(Murphy *et al*., 2008 ,Stolevik, 2012)**. Its production is increased by IL-1 and TNF-α, and is associated with thrombotic cardiovascular events **(Ziccardi *et al*., 2002, Shirai, 2004 , Lukic *et al*., 2014)**.TNF-α is a prototypical inflammatory cytokine, originally characterized as an endotoxin-induced serum factor that causes necrosis of certain tumor cell types. So, it has been found to be a key regulator of the immune response, capable of diverse cellular effects, including apoptosis, necrosis, inflammatory effects, proliferative or growth-promoting effects, and hematopoetic effects **(Sethi *et al*., 2002)**.

Adipokines are bioactive polypeptides produced by and released from white adipose tissue . They play an important role in the development of health problems associated with obesity including inflammation (**Leal and Mafra, 2013**) .Obesity leads to increased expression of pro-inflammatory adipokines and diminished expression of anti-inflammatory adipokines, resulting in the development of a chronic, low-grade inflammatory state (**Nakamura et al., 2014**).Resistin is a cysteine-rich pro-inflammatory adipokine produced by macrophages as well as adipocytes in humansand has higher levels in obese subjects as compared to lean subjects. These higher levels were positively correlated with changes in the BMI and the visceral fat area (**Yang *et al*., 2007; Haseeb *et al*., 2009 ; Asano *et al*., 2010**). Resistin is associated with an increased production of pro-inflammatory cytokines and a decreased production of anti-inflammatory cytokines (**Silswal *et al*., 2005 ; Vachharajani and Granger, 2009 ; Stofkova, 2010**). Plasma resistin has been proposed as a marker of ischemic injury **(Chu *et al*., 2008)** , and may represent a link between metabolic signals, inflammation and atherosclerosis **(Daniel *et al*., 2010)**. Adiponectin an anti-inflammatory adipokine produced by adipocytes. It plays an important role in modulating the inflammatory network involved in the pathophysiology of cardiovascular disease, it promotes an anti-atherogenic and anti-inflammatory program of gene expression **(Deng *al et*., 2010 and Ran *et al*., 2010)**. Since obesity is generally associated with decreased plasma levels of adiponectin, this adipokine has been implicated in the pro-inflammatory state associated with obesity (**Vachharajani and Granger, 2009**). It was additionally claimed to regulate lipid and glucose metabolism, food intake and body weight (Liu and Liu, 2009). High-fat diet has been found in experimental animal models to induce an inflammatory response in the hypothalamic areas that control feeding behavior and energy homeostasis by regulating downstream neurons (**Velloso, 2009**).

Numerous investigations have proved that medicinal herbs contain diverse classes of compounds such as polyphenols, flavonoids, carotenoids and alkaloids **(Yoshida *et al*., 1989, Velioglu *et al*., 1998,Carr *et al*., 2000 and Ibrahim et al., 2015).** The flavonoids comprise the most common group of plant polyphenols and are reported to exhibit a wide variety of biological effects, including hepato-protective and anti-inflammatory effects, as well as antioxidant and free radical scavenging activities **(Middleton *et al*., 1992, Nijveldt *et al*., 2001and Mannaa *et al*., 2015).**

Purslane (*Portulacaoleracea*) is an annual green herb belong to the family Portulacaceae. It grows in almost any unshaded area as flower beds corn fields. It is used sometimes, as a nutritional food **(Boulos and El-Hadidi, 1984).** It contain β-carotene , a wide variety of fatty acids**,** omega 3 and high amounts of vitamins E and C **(Abd El-Latif, 2008 and Oliveira *et al*., 2009).** This plant has several biologically active chemicals including flavonoids, alkaloids and other phenolic compounds which increase its antioxidant capacity **(Xiang *et al*., 2005, Xu *et al*., 2006 and Lim & Qual, 2007)**. Flavonoids isolated from purslane, such as apigenin and luteolin, have been shown to possess anti-inflammatory, anticarcinogenic, carminative, antispasmodic, and mildsedative properties **(Xu *et al*., 2006,Abas *et***

***al*., 2006, Abd El-Latif, 2008**).This study aimed at evaluating the possible protective role of caloric restriction or purslane extract against the inflammatory response of experimentally high fat diet-induced obesity in male rats.

Materials & Methods

1. Experimental Animals

Fifty adult male albino rats weighing 155-185 g. were used in this study. They were obtained from the animal house of Faculty of Veterinary Medicine, Zagazig University and acclimatized for two weeks prior to the experiment .Animals were housed in stainless wire cages at room temperature (22-25 °C) and a photoperiod of 12 h. light-dark cycle. Animals were permitted for free standard laboratory diet and drinking tap water *ad libitum*. The European Committee directive and national rules on animal care were followed in this experiment.

2. Preparation of Purslane Extract

Purslane was collected from corn fields of farmers around Zagazig city, dried in shade under laboratory conditions for 15 days, transferred to oven at 60 C for 48 h., then grinded into a finely powdered material. The powder was extracted with distilled water in water bath at 80 C, then extracted by maceration in room temperature 5 times, each time one week by 70% ethanol. The total extract was concentrated under reduced pressure. 200 g. of purslane extract were dissolved in 2 liter of distilled water to get extract solution (1ml equivalent to 100 mg of purslane).

3. Experimental Design

The initial and final body weight of rats were determined. Rats were divided into five groups (n=10). **1st group (control):** Rats were fed on normal chow diet (5% diet derived fat, 18% proteins, and 77% carbohydrates) for 18weeks **(Svegliati-Baroni *et al*., 2006)**. **2nd group (HFD):** Rats were fed on high fat diet (58% fat, 18% protein, and24% carbohydrates) **(Svegliati-Baroni et al., 2006)** for 18 weeks. **3rd group (HFD + Rest 4):** Rats were fed on highfat diet for 18 weeks then followed by diet restriction by feeding on normal chow diet for four weeks. **4th group (HFD + Rest 8):** Rats were fed on high fat diet for 18 weeks then followed by diet restriction by feeding on normalchow diet for eight weeks. **5th group (HFD + purs. ext.):** Rats were fed on high fat diet and gavaged every day with 1 g/ kg BW of purslane extract (**El-Newary, 2012)** for 18 weeks.

4. Anthropometric Measures

At the end of the experimental period, weight and lengthof rats were recorded **(Novelli *et al*, 2007)** and body mass index (BMI) was calculated as weight (g)/length^2 (cm^2) **(Novelli et al, 2007)**. In addition abdominal circumference (AC) was recorded **(Gerbaix *et al*., 2010)**.

5. Blood collection and serum samples preparation:

At the end of experimental period, animals were slightly anesthetized with diethylether and blood samples were withdrawn from retro-orbital venous plexus **(Yang *et al.*, 2006)**, then collected in a plastic centrifuge tube and allowed to clot andthen centrifuged at 3000 rpm for 15 minutes at 4^{0}C where the clear serum was separated (Nishizawa *et al*, 2002) for the determination of adiponectin, resistin, TNF-α and interleukins levels.

6. Biochemical assay

Serum levels ofresistin, adiponectin, IL-1β, IL-2, IL-6, IL- 12 and TNF-α, were determined by enzyme linked immunosorbent assay (ELISA) technique using kits manufactured by Assay Pro. Co., USA **(Engvall & Perlmann, 1971)**.

7. Statistical Analysis

Data were presented as mean ± SE. The statistical analysis is done by using SPSS program (version 14) (SPSS Inc. Chicago, IL, USA). The obtained data were subjected to one way analysis of variance (ANOVA) to compare the treated groups together **(Kirkwood 1989)**. All statements of significance were based on probability of $P<0.05$.

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Results

Data presented in table (1) demonstrate that feeding of rats on HFD for 18 weeks caused significant increases in the anthropometric measures (BW, BMI, AC) and in serum levels of resistin , IL-1β, IL-2, IL-6, IL- 12 and TNF-α and a significant decrease in serum level of adiponectin as compared to the control group. It can also be observed in table (1) that caloric diet restriction for 4 or 8 weeks , or oral administration of purslane extract were found to alleviate these effects on all parameters measured . In addition, data demonstrated graphically in Fig 1 (A-G) revealed a significant positive correlation between BMI and serum resistin, TNF-α, IL-1β, IL-2, IL-6 & IL-12 levels in obese group ($r=0.640$; $P < 0.05$ and $r =0.882$; $P < 0.01$, $r =0.794$; $P < 0.01$, $r=0.666$; $P < 0.05$, $r =0.696$; $P < 0.05$ and $r =0.670$; $P < 0.05$) respectively while these data explained a significant negative correlation between BMI and serum adiponectin level ($r =0.731$; $P < 0.05$).

Table 1: Effects of caloric restricted diet for 4 (Rest-4) or 8 (Rest-8) weeks , or oral administration of purslane extract (purs ext) for 18 weeks on high fat diet (HFD) induced changes in the anthropometric measures [final bod weight (Final BW) , body mass index (BMI) and abdominal circumference (AC)] , and in some inflammatory mediators , the adipokines resistin and adiponectin and the cytokines IL-1β , IL-2 , IL-6 , IL-12 and TNF-α in male rats . Each value is expressed as a mean ± SE of 10 separate animals .

Groups / Parameters	Cont	HFD	HFD + Rest-4	HFD + Rest-8	HFD + pursext
Initial BW (g)	**164.40 ± 1.89**	**168.70 ± 2.72**	**164.00 ± 1.20**	**164.00 ± 1.20**	**167.50 ± 0.83**
Final BW (g)	**271.00 ± 6.48**	**416.30 ± 9.72** $^{a+}$	**349.60 ± 5.79** $^{a+,b+}$	**313.40 ± 6.27** $^{a+,b+}$	**336.40 ± 5.13**$^{a+,b+}$
BMI (g / cm^2)	**0.55 ± 0.01**	**0.77 ± 0.03** $^{a+}$	**0.69 ± 0.02** $^{a+,b+}$	**0.63 ± 0.03** $^{a+,b+}$	**0.68 ± 0.02** $^{a+,b+}$
AC(cm)	**17.4 ± 0.27**	**21.2 ± 0.37** $^{a+}$	**19.8 ± 0.47**$^{a+,b-}$	**18.65 ± 0.46**$^{a-,b+}$	**18.75 ± 0.31**$^{a-,b+}$
IL-1β (pg/ml)	**5.61 ± 0.19**	**22.17 ± 1.30** $^{a+}$	**17.46 ± 0.82** $^{a+,b+}$	**11.77 ± 1.02** $^{a+,b+}$	**14.09 ± 0.83**$^{a+,b+}$
IL-2 (pg/ml)	**6.85 ± 0.30**	**22.82 ± 1.15** $^{a+}$	**16.53 ± 0.91** $^{a+,b+}$	**9.88 ± 0.56** $^{a+,b+}$	**12.20 ± 0.97**$^{a+,b+}$
IL-6 (pg/ml)	**8.75 ± 0.66**	**23.22 ± 1.23** $^{a+}$	**18.41 ± 0.74** $^{a+,b+}$	**12.72 ± 0.69** $^{a+,b+}$	**14.63 ± 0.81**$^{a+,b+}$
IL-12 (pg/ml)	**6.85 ± 0.30**	**22.82 ± 1.15** $^{a+}$	**16.53 ± 0.91** $^{a+,b+}$	**9.88 ± 0.56** $^{a+,b+}$	**12.20 ± 0.97**$^{a+,b+}$
TNF-α (pg/ml)	**27.85 ± 2.51**	**63.50 ± 4.94** $^{a+}$	**52.98 ± 3.33** $^{a+,b+}$	**37.68 ± 1.97** $^{a+,b+}$	**44.44 ± 3.51**$^{a+,b+}$
Resistin (ng/ml)	**7.86 ± 0.38**	**23.71 ± 1.40** $^{a+}$	**17.28 ± 0.96** $^{a+,b+}$	**10.91 ± 0.51** $^{a+,b+}$	**13.78 ± 0.91**$^{a+,b+}$
Adiponectin (mg/L)	**6.11± 0.11**	**3.2 ± 0.17**$^{a+}$	**4.67 ± 0.16** $^{a+,b+}$	**5.27 ± 0.16**$^{a+,b+}$	**5.21 ± 0.19** $^{a+,b+}$

a+: Significant when compared with the control value (P 0.05).
a- : Non significant when compared with the control value (P 0.05).
b+: Significant when compared with HFD-group (P 0.05).
b- : Non significant when compared with HFD-group (P 0.05).

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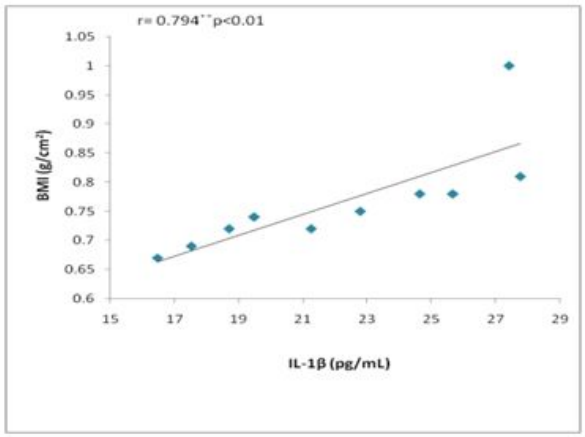

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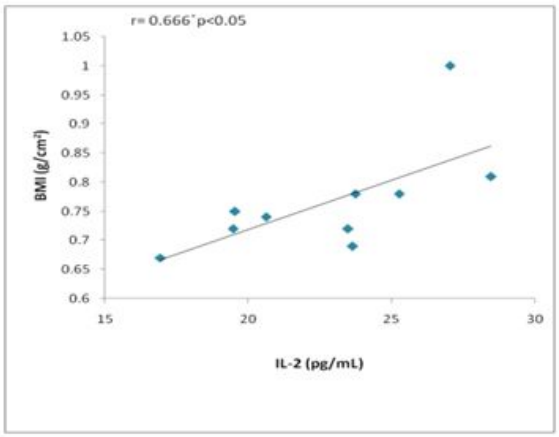

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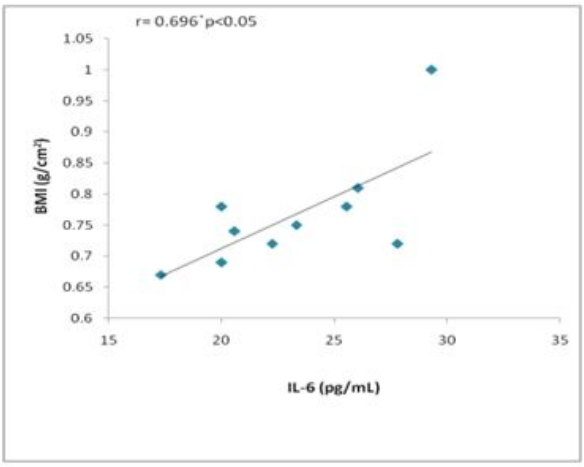

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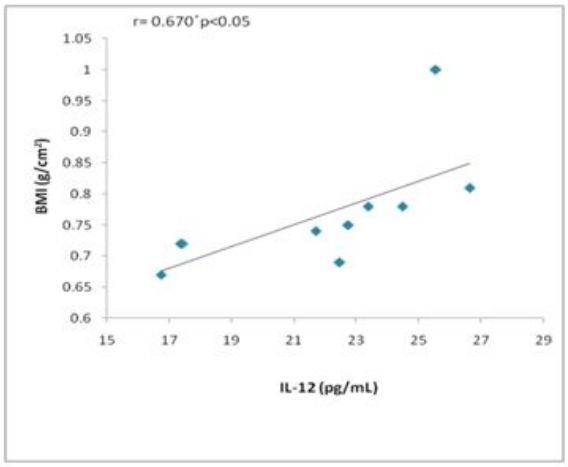

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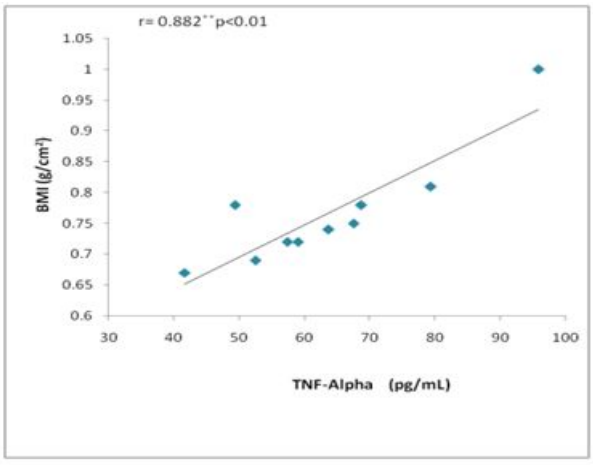

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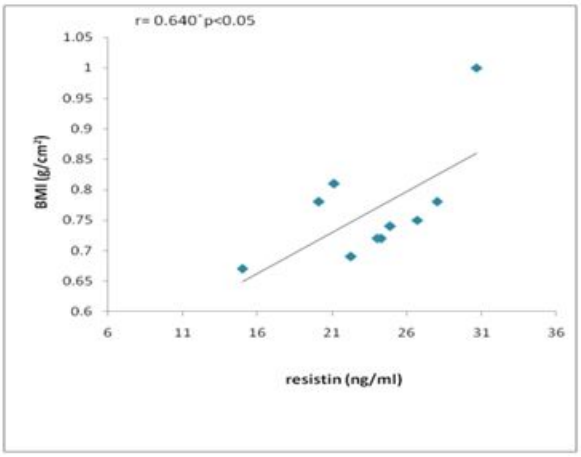

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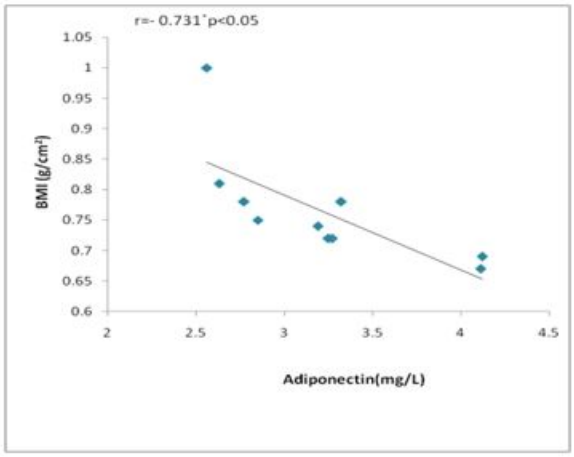

G

Figure 1 (A - G) : Correlations between BMI and serum IL-1β, IL-2, IL-6,IL-12 TNF-α , resistin and adiponectin levels .

Discussion

The present data revealed that feeding rats on HFD for 18 weeks induced a significant increase in BW, BMI, AC, serum IL-1β, IL-2, IL-6, IL- 12 TNF-α and resistin,levels, while, caused a significant decrease in the level of serum adiponectin as compared to control group. Whereas, the diet restriction for 4 or 8 weeks after 18 weeks of HFD or daily gavaged purslane extract during HFD feeding for 18 weeks showed a significant decrease in the levels of BW, BMI, AC, resistin, IL-1β, IL-2, IL-6, IL- 12 and TNF-α but showed a significant increase in the level of adiponectin as compared to HFD group.Thereby, it has been substaintiated that there was a significant positive correlation between BMI (obesity marker) and serum resistin, TNF-α and interleukins levels in obese group, while, there was a significant negative correlation between BMI and adiponectin levels, these results are coincident with **Morgan & Edrees (2013)**, **Baltaci *et al*., (2016)** and **Ibrahim *et al*., (2017)**, they demonstrated that the obesity was positively correlated with key metabolic syndrome markers such as elevated levels of BW, BMI, AC, triglycerides, total cholesterol.The hypertriglyceridemia as a result of obesity induces risk factor of metabolic disturbance and promote oxidative stress state,which could trigger the progression of abnormal lipid metabolism generating a reactive oxygen species (ROS) including superoxide anions, hydrogen peroxide and hydroxyl radicals that react with unsaturated fatty acid chains in cell membrane lipids causing lipid peroxidation, which promotes many health problems including disturbancein thelevels of resistin and adiponectin **(Kai *et al*., 2015).** Moreover, plasma resistin level were reported to be associated with many inflammatory markers such as TNF-α and interleukins (**Hossen *et al*., 2010 and Giordano *et al*., 2011**). In addition, several investigators have hypothesized that obesity is recognized as a key player inthe pathogenesis of several inflammatory disorders andleads to a state of chronic low-grade systemic inflammation accompanied with profound release of pro-inflammatory cytokines ,TNF-α and interleukins **(Brightling *et al*., 2008, Shore, 2008 and kim *et al*., 2015)**. Also, increased oxidative stress as a result of obesity activate macrophages that secrete these inflammatory cytokines which in turn decrease insulin action on adipocytes and determine hypoadiponectin and increase leptin **(Xu *et al*., 2003 ,Suganami *et al*., 2005 and Tesauro *et al*., 2011)** they propose a novel mechanism by which TNF-α activation might be involved via increased oxidative stress in the pathophysiology of vascular dysfunction in patients with obesity-related metabolic syndromes. In the same context, resistin is also produced by immune cells and, therefore, is related to the activation of inflammatory processes. The pro-inflammatory properties of resistin include the secretion of TNF-α and interleukins and impairment of the anti-inflammatory effect of adiponectin **(Zimmermann *et al*., 2013)**.Thereby, adiponectin has a negative correlation with measures of obesity, resistin, TNF-α and interleukins levels and increases with weight loss **(Hansen *et al*., 2009, Anfossi *et al*., 2010 and kim *et al*., 2015).** Adiponectin modulates the inflammatory response by down-regulating the expression of pro-inflammatory mediators, such as TNF-α, IL-6 and IL-12 and by up regulating anti- inflammatory molecules, such as the antagonist receptor of IL-1β and IL-2 **(Tilg and Hotamisligil, 2006 ,Cirillo *et al*., 2012).** Moreover, adiponectin inhibits the oxygen free radical production and ameliorates the

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endothelial function in mice genetically modified to develop hyperlipidemia and atherosclerosis **(Chen *et al*., 2010).** Adiponectin suppresses lipid accumulation and class A scavenger receptor expression in human monocyte- derived macrophages **(Cai *et al*., 2010 ,Ohashi *et al*., 2010)**.

The present study revealed that, the pro-inflammatory cytokines; TNF-α and the interleukins IL-1β, IL-2, IL-6 & IL-12 levels are positively correlated with obesity measures and reduce with weight loss, this may be due to that the adipose tissues have been identified as one of the main sources of these cytokines and its level is elevated in adipose tissue and plasma of obese patients. Interestingly, the plasma levels of these cytokines are closely related to the extent of visceral adipose tissue, while, weight loss and life style changes reduce these levels **(Zhang *et al*., 2010, and Matsui, *et al*., 2014).**

Data of this investigation revealed that in rats fed on caloric restricted diet or supplemented orally with purslane extract caused improvement of obesity measures and reduced the inflammatory responses induced by HFD. This may be due to reduction of lipid peroxidation and ROS generation, thereby restoring normal body weight is an appropriate strategy for reducing endothelial dysfunction and controlling inflammatory mediators, such as TNF-α , interleukins, resistin and adiponectin **(Phelan and kerins, 2014, Kim *et al*., (2015)**.

In the present study oral supplementation of HFD fed rats with purslane extract was effective in alleviating the increase in BW gain and showed amazing modulation in serum levels of resistin, adiponectin, TNF-α and interleukins as compared to the obese rats, These protective effects of purslane extract could be possibly due to its ability to inhibit the accumulation of fats and its wide biological effects including anti-inflammatory, antioxidant and free radical scavenging activities **(Nijveldt *et al.*, 2001).** The presence of phytochemicals including glycosides, flavonoids, alkaloids, terpenoids, omega-3 fatty acids, phenolic and other antioxidant compounds in purslane extract could possess lipid lowering properties that help in preventing or slowing the progress of obesity associated diseases **(Kamal Uddin *et al*., 2014 ,Sadeghi *et al*., 2016)**. The flavonoids comprise the most common group of plant polyphenols and are reported to exhibit a wide variety of biological effects, including antioxidant, anti-inflammatory and free radical scavenging activities **(Chan *et al.*, 2000, Xu *et al.*, 2006 , Ibrahim *et al.*, 2015).** Polyphenol has been associated with lowering intestinal absorption of triglycerides by inhibition of pancreatic lipase and increased lipoprotein lipase activity. This could result in modified lipid profile levels **(Ikeda *et al*., 2005 ,Bursill *et al.*, 2007)**. Also, high omega-3 fatty acids levels in purslane may be responsible for modulating the lipid profile in hypercholesterolemic subjects and considered as an anti-inflammatory agent may be used to reduce the disturbance of adipocytokines level in obese subjects **(Tabatabaie and Shahbaz, 2015)**. Moreover flavonols as quercetin and catechin were found to inhibit the production of TNF-α by lipopolysaccharide-activated macrophages **(Saraf *et al*., 2007, Lee *et al*., 2008, Ali *et al*., 2011)**. Furthermore, terpenoids that are the main constituents of purslane can modulate the activity of peroxisome proliferator- activated receptors (PPARs) which are ligand – dependent transcription factors, belonging to the nuclear receptors **(Goto *et al*., 2010 and De Sousa, 2012).** PPAR- α have the potential to decrease lipid levels and are commonly used to treat hyper-triglyceridemia and other dyslipidemic state **(Goldwasser *et al*., 2010).** Additionally, purslane extract contains linoleic acid, phytosterols and ascorbic acid which play their role in modulating obese parameters by increase cholesterol excretion from the body, and inhibition of cholesterol absorption**.** Therefore, the modulating effect of purslane extract on progress of obesity associated-problems could be explained due to the presence of these bioactive compounds **(Khandelwal *et al*., 2013 and Kai *et al*., 2015).**Moreover purslane extract is a rich source of antioxidant vitamins as vitamin E in the form of alpha-tocopherol which is a chain-breaking antioxidants that prevent the propagation of free radicals activities. It has chromanol ring with hydroxyl group which can donate hydrogen atom to reduce free radicals and hydrophobic side chain which allow for penetration into the biological membrane. It protects the poly unsaturated fatty acids within the membrane phospholipids, plasma lipoprotein and low density lipoproteins against oxidation **(Rosenau *et al*., 2007)**. In addition, purslane extract is a good source of coenzyme Q10 that has been demonstrated to have potent antioxidant effects against lipid peroxidation and may offer protection against obesity-associated problems by preventing lipid peroxide formation and modulate oxidative stress state **(Ross, 2007).** In conclusion, the results described herein, provide clear evidence that oral supplementation of rats with purslane extract matches the effects of caloric diet restriction in alleviating the disturbances in **anthropometric measures** and pro-and anti-inflammatory cytokines or adipokines induced by high fat diet in obese rats

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