Comparative Studies on Adiponectin, Resistin, And Leptin in Both Obese and Lean Subjects in Imo State

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Abstract
This study was carried out to compare the levels of adinopectin, resistin, leptin in both obese and lean subjects in patients attending Federal Medical Center, Owerri. Eighty (80) subjects including male and female who were within the age range of 25-50 years attending the outpatient department at Federal Medical Center, Owerri were recruited for the study and they were divided into two groups of forty (40) obese and forty(40) lean subjects. Data obtained was analyzed using Statistical Package for Social Sciences (SPSS) version 21. Values was expressed as mean±standard deviation. The student t-test was used to compare the parameters (at level of significance 0.05). P< 0.05 was considered as statistically significant and P>0.05 was considered not statistically significant.The result of the present study showed that the mean values of adiponectin(1.45±0.67µg/ml) and resistin (5.60±1.61ng/ml) was significantly decreased in obese subjects while serum leptin (7.95±2.29ng/ml) was significantly increased in obese subjects when compared with the mean values of adiponectin (5.18+2.49 µg/ml), resistin (9.52±3.00 ng/ml) and leptin (2.29±1.13 ng/ml) in lean subjects at P<0.05 respectively. The mean values of adiponectin (1.49+0.59µg/ml, 2.70±0.36 µg/ml) and resistin (5.36+±1.20ng/ml, 7.29±1.21ng/ml) was significantly decreased in obese male and female subjects while the mean value of serum leptin (7.90±2.11 ng/ml, 9.28±0.19 ng/ml) was significantly increased in obese male and female subjects at P<0.05 respectively. When the mean values of adipokine was compared between obese male and female according to age, it was discovered that the mean values of adiponectin levels was increased with age in both obese male {25-35 years(1.50±0.76 µg/ml), 36-50years(3.90±1.58 µg/ml)} and female{25-35 years(3.0±0.68 µg/ml), 36-50years(5.42±3.38µg/ml)}. Leptin levels decreased with age in only women {25-35 years(8.51±3.48ng/ml), 36-50years(4.22±1.29ng/ml)} but increased with age in men{25-35 years(8.03±2.59ng/ml), 36-50years(12.80±3.59ng/ml)}. There was no significant difference in the mean value of resistin in both male and female at p>0.05 respectively. In conclusion, adipokines may decrease the insulin sensitivity of tissues and induce inflammation and the development of atherosclerosis, diabetes and psoriasis, as well as diabetic foot.
Key Words: adiponectin; resistin; leptin obese; lean subjects; Imo State

Introduction
Obesity is a substantial public health crisis in the United States, and internationally, with the prevalence increasing rapidly in numerous industrialized nations. A report from the National Center for Health Statistics stated that in US individuals aged 20 years or older, the prevalence of obesity rose steadily from 19.4% in 1997 to 31.4% for the period January-September 2017 [1]. Additionally, approximately 1/3 of the population is overweight defined as a BMI between 25 and 30 kg/m. Moreover, the obesity epidemic is not localized to the United States as there has been a marked increase in the prevalence of obesity worldwide. The number of individuals with morbid obesity (BMI > 40) has also greatly increased [2]. It should be noted that very athletic individuals may have a high BMI without excess body fat (the increase in weight is due to muscle mass) and as a consequence not have metabolic abnormalities. Of great concern is that the prevalence of obesity has also markedly increased in children. Obesity is associated with insulin resistance, alterations in lipid metabolism, and the metabolic syndrome, particularly when the excess adipose tissue is located in an intra-abdominal location or in the upper chest. Obesity is a risk factor for the development of cardiovascular disease, but it appears that much of this effect is accounted for by obesity inducing dyslipidemia, diabetes, hypertension, inflammation, and a procoagulant state. The majority of deaths related to high BMI are due to cardiovascular disease [3] Obesity simply means excess of body-fat. It is defined as having a body mass index (BMI) of greater than 30 kg/m^2. Healthy weight is defined as a BMI between 19 and 25 kg/m^2. Overweight is defined as a BMI between 25 and 30 kg/m^2. It is due to greater energy intake compared with energy expenditure [4]. It is difficult to study obesity because the abnormality is not a single disease and because the result of long-term follow up in large scale is not available in the existing literature. The knowledge that is available today is only a cross-sectional survey in the population. Obesity currently threatens the health, well- being and economic welfare of virtually every country in the world [5]. Over 300 million people are estimated to be obese. Obesity is considered a

chronic (long-term) disease, like high blood pressure or diabetes. It has many serious long-term consequences for health, and it is the second leading cause of preventable deaths in many countries [6] Although several classifications and definitions for degrees of obesity are accepted, the most widely accepted classifications are those from the World Health Organization (WHO), based on body mass index (BMI). Adipose tissue is a key endocrine organ that communicates with brain, muscle, liver, and pancreas, thereby maintaining energy homeostasis. Adipose tissue stores excess energy in the form of lipids and are thus able to dramatically change in size in accordance with changing metabolic needs [7]. Moreover, studies have shown that fat tissue exerts important endocrine functions which are mediated by a complex network of various soluble factors derived from adipocytes called adipocytokines including tumor necrosis factor a (TNF-a), Interleukin (IL) 6, leptin, adiponectin and resistin [8]. Some adipokines play a major role in insulin resistance and cardiovascular complications associated with obesity, especially central or visceral obesity [4]. At the cellular level, obesity is not solely pathology of adipocytes as there are other cell types within adipose tissue that participate as well. In fact, the presence of infiltrating macrophages in adipose tissue makes obesity comparable to a low-grade chronic inflammation with links between adipose cells and the immune system. At present comprehension of these concepts is essential for a better understanding of the pathophysiological mechanisms of insulin resistance and type 2 diabetes [9].Leptin, resistin, and adiponectin are important adipocytokines that influence both insulin sensitivity and inflammation, which are closely involved in the development of T2DM [10] Leptin is a pro-inflammatory molecule that plays a key role in the regulation of glucose and energy homeostasis. Leptin was one of the first adipocytokines identified, and immediately has drawn substantial research attention. The discovery of leptin in 1994 has provided a major new piece in the puzzle of obesity, as it has been found that its level was directly related to the quantity of body fat. Leptin is a 16 kDa non-glycosylated protein secreted in direct proportion to adipose tissue mass as well as nutritional status. Plasma leptin concentrations positively correlate with subcutaneous rather than intra-abdominal fat tissue mass [11] Leptin exerts an inhibitory effect on food intake and increases energy expenditure through thermogenesis and physical activity. The idea of leptin as an insulin sensitizing hormone and leptin deficiency or resistance as a potential link between obesity and diabetes has been reviewed recently [12] The discovery of adiponectin (ApN) occurred at about the same time as the discovery of leptin (1995/1996), but it did not receive major attention in the scientific community for the next few years until its markedly protective role in the pathogenesis of obesity-related disorders was acknowledged. Adiponectin is a 30-kDa adipocyte complement-related protein . Prospective studies in humans have shown that increased plasma concentrations of ApN were strongly and independently associated with reduced risk of type 2 diabetes [13] In contrast to most other adipokines, circulating ApN was negatively correlated with body mass index (BMI) and was markedly decreased in obese subjects and in patients with type 2 diabetes [14] Resistin is a 12.5-kDa cysteine-rich peptide that belongs to a family of resistin-like molecules with distinct expression patterns and biological effects. Resistin is primarily secreted from mature adipocytes in rodents whereas in humans it is expressed primarily from adipose infiltrating macrophages. It was suggested that resistin might link obesity with insulin resistance and diabetes. In humans, the role of resistin in insulin resistance remains controversial. Increased circulating levels of resistin are associated with incidence of obesity, insulin resistance, and inflammation (Takeishi, 2013). Most mouse studies, but not all, support the notion that resistin is an adipokine regulator of insulin action. However, most human studies show an entirely different picture [15] As regard to leptin, its mean serum levels were significantly higher in the obese groups compared with lean group which served as control, while the mean serum levels of resistin were increased significantly in obese subjects as compared to lean subjects. Other studies have it that Resistin levels were similar in lean and obese subjects and no significant correlation was observed between resistin levels and BMI. On the other hand, the mean serum levels of adiponectin were decreased significantly in obese groups when compared to lean subjects. On comparing the effect of weight loss on the serum levels of the three hormones, a significant decrease was found in the mean levels of both resistin and leptin and a significant increase in the mean level of adiponectin on obese subjects [16]. However, the notion of leptin as an anti-obesity hormone was called into question because the “common” form of obesity is typically associated with high leptin levels (that reflect high energy stores) and leptin resistance[17]. These findings could be explained on bases that although most obese humans have an elevated circulating level of leptin, they do not respond to this increased endogenous leptin level by reducing their food intake due to leptin resistance [18] Obesity is increasingly becoming an important public health concern among all age groups in most of the developed and underdeveloped world. Greater than 30% of the United States population is obese and in Nigeria, about 8.1%-22.2% are obese and at risk to develop insulin resistance and associated metabolic disorders, including hypertension, hyperlipidemia, fatty liver disease, atherosclerosis, and Type 2 diabetes mellitus [19] Fat tissue exerts important endocrine functions, which are mediated by a complex network of various soluble factors, derived from adipocytes, called adipocytokines including tumor necrosis factor a (TNF-a), Interleukin (IL) 6, leptin, adiponectin and resistin. Some adipokines play a major role in insulin resistance and cardiovascular complications associated with obesity, especially central or visceral obesity. Many authors have it that leptin and resistin which are adipocytes play1 a role in the development of obesity. However, few argue that resistin is same in both obese and lean subjects that there is no significant difference between the two. There is paucity of information in evaluating the relationship between adiponectin, resistin, and leptin in both obese and lean subjects so therefore this work is aimed at evaluating and comparing the levels of leptin, adiponectin, and

resistin in both obese and lean subject and also provide knowledge on the disparity associated between the resistin hormone in both the obese and lean subjects in Owerri.

Materials And Methods

Study area

The study was carried out in the medical out-patient department Federal Medical Center, Owerri, Imo State.

Ethical approval

Ethical approval was obtained from Federal Teaching Hospital Owerri

Study population

A total of 80 subjects including male and female subjects attending the outpatient department at Federal Medical Center, Owerri was recruited for the study. The 80 subjects was within the age range of 25-50 years. The 80 subjects was divided into two groups:
Group 1(Test) consists of 40 obese patients
Group 2(Control) consists of 40 lean subjects
Anthropometric measurements, including height, weight, waist circumference (WC) and hip circumference (HC) wasperformed on the subjects. Body Mass Index (BMI) was calculated as weight in (kg) divided by height in meters squared (m^2). Waist-to-hip ratio (WHR) was also calculated as waist circumference (WC) divided by hip circumference (HC). BMI was used to reflect the total body fat while waist circumference (WC) and Waist-to-hip ratio (WHR) was indirect measurements of body fat centralization

Selection criteria

Inclusion

The participants were those that met the enrollment criteria. The criteria are as follows:

(i) Subjects with low levels of physical activity
(ii) Lean subjects with a body mass index between 19 and 25 kg/m^2
(iii) Obese subjects with a BMI of over 30 kg/m^2
(iv) Subjects between the age range of 25-50 years

Exclusion

The following was excluded from the study they are:

I. Insulin dependent type 1 diabetes, concomitant disturbances of liver and thyroid, renal insufficiency and chronic inflammatory diseases.
II. Those with mental disorder or any intellectual disability
III. A current eating disorder or any psychiatric disorder (psychotic disorder, bipolar disorder, substance dependence or anxiety and depressive disorders)
IV. Pregnant women or those currently breast feeding
V. Elderly patients who are above the age of 50 years

Sample collection

About 10ml of venous blood was collected from each subject using the standard clean veni-puncture technique and dispensed into a labeled plain container. The blood samples was spun at 3000rpm for 5minutes and serum was separated into a new labeled plain container. The serum samples was then be taken to the Laboratory Complex, Federal Medical Center, Owerri where the following parameters was estimated: Adiponectin, Resistin, Leptin and Lipid profile.

Laboratory Procedures

All reagents used were commercially procured and the manufacturer's standard operating procedures was strictly followed.
Adiponectin, Resistin and Leptin were determined by Enzyme Linked immunosorbent Assay

Statistical Analysis

Data obtained wasanalysed using Statistical Package for Social Sciences (SPSS) version 21.Values was expressed as mean±standard deviation. The student t-test was used to compare the parameters (at level of significance 0.05). P< 0.05 was considered as statistically significant and P>0.05 was considered not statistically significant.

Results

Parameters	**Obese n=40**	**Lean n=40**	**T-Value**	**P-Value**
Adiponectin (µg/ml)	1.45±0.67	5.18+2.49	-9.486	0.0001
Resistin (ng/ml)	5.60±1.61	9.52±3.00	-7.080	0.0001
Leptin (ng/ml)	7.95±2.29	2.29±1.13	12.851	0.0001

Table 1: Mean ±SD values of Serum Adiponectin, Resistin and Leptin in Obese Subjects and Lean Subjects of study population.

Table 1 Shows that the mean values of serum adiponectin (1.45±0.67µg/ml),Resistin(5.6±1.61ng/ml) and leptin(7.95±2.29ng/ml) of obese subjects was significantly different (P=0.0001, P=0.0001 and P=0.001) when compared with the mean value of serum adiponectin (5.18±2.49µg/ml), resistin (9.52±3.00ng/ml) and leptin(2.29±1.13 ng/ml) of lean subjects. When the mean values of the adipocytokines variables in obese subjects was compared with the mean values of the adipocytokinesvariables in lean subjects it was found that the mean values of adiponectin and resistinwas increased in lean subjects while serum leptin was decreased in lean.

Parameters	**Obese Male n=20**	**Lean Male n=20**	**T-Value**	**P-Value**
Adiponectin µg/ml	1.49+0.59	4.60+1.23	4.321	0.001
Resistin ng/ml	5.36+±1.20	9.14+3.42	3.876	0.001
Leptin ng/ml	7.90±2.11	2.11±0.35	5.487	0.001

Table 2: Mean ±SD values of Serum Adiponectin, Resistin and Lectin in Obese Male and Lean Male Subjects.

Table 2 Shows that the mean values of serum adiponectin (1.49±0.59 µg/ml), Resistin(5.36±1.20ng/ml) and leptin(7.90±2.11ng/ml) of obese male subjects was significantly different (P=0.001, P=0.001 and P=0.001) when compared with the mean value of serum adiponectin (4.60±1.23µg/ml), resistin (9.14±3.42 ng/ml) and leptin (2.11±0.35 ng/ml) of lean male subjects. When the mean values of the adipocytokines variables in obese male subjects was compared with the mean values of the adipocytokines variables in lean male subjects it was found that the mean values of adiponectin and resistin was increased in lean male subjects while serum leptin was decreased in lean male subjects.

Parameters	**Obese Female n=20**	**Lean Female n=20**	**T-Value**	**P-Value**
Adiponectin µg/ml	2.70±0.36	7.14±3.28	3.478	0.001
Resistin ng/ml	7.29±1.21	12.13±0.26	6.457	0.001
Leptin ng/ml	9.28±0.19	6.85±1.89	4.352	0.003

Table 3: Mean ±SD values of Serum Adiponectin, Resistin and Leptin in Obese Female and Lean Female Subjects.

Table 3 Shows that the mean values of serum adiponectin (2.70±0.36µg/ml), Resistin(7.29±1.21ng/ml) and leptin(9.28±0.19ng/ml) of obese female subjects was significantly different (P=0.001, P=0.001 and P=0.003) when compared with the mean value of serum adiponectin (7.14±3.28µg/ml), resistin (12.13±0.26 ng/ml) and leptin (6.85±1.89 ng/ml) of lean female subjects. When the mean values of the adipocytokines variables in obese female subjects was compared with the mean values of the adipocytokines variables in lean female subjects it was found that the mean values of adiponectin and resistin was increased in lean female subjects while serum leptin was decreased in lean female subjects.

Parameters	**Obese Male n=20**	**Obese Female n=20**	**T-Value**	**P-Value**
Adiponectin µg/ml	1.49±0.76	2.70±0.28	3.444	0.104
Resistin ng/ml	5.36±1.21	7.29±1.66	7.932	0.243
Leptin ng/ml	7.90±1.19	9.28±2.89	4.278	0.345

Table 4: Mean ±SD values of Serum Adiponectin, Resistin and Lectin in Obese Male and Female Subjects.

Table 4 Shows that the mean values of serum adiponectin (1.49±0.76 µg/ml), Resistin(5.36±1.21ng/ml) and leptin(7.90±1.19ng/ml) of obese male subjects was not significantly different (P=0.104, P=0.243 and P=0.345) when compared with the mean value of serum adiponectin (2.70±0.28µg/ml), resistin (7.29±1.66 ng/ml) and leptin (9.28±2.89 ng/ml) of obese female subjects. When the mean values of the adipokinesin obese male subjects was compared with the mean values of the adipokines in obese female subjects

it was found that the mean values of adiponectin, resistin and leptin was decreased in obesemale subjects when compared with obese female subjects though not significantly at p>0.05.

Age Group (years)	**25-35 (years)**	**36-50 (years)**	**T-Value**	**P-Value**
Adiponectin µg/ml	**1.50**±0.76	**3.90**±1.58	**8.662**	**0.01**
Resistin ng/ml	**6.00**±1.45	**7.24**±2.66	**4.342**	**0.01**
Leptin ng/ml	**8.03**±2.59	**12.80**±3.59	**9.554**	**0.001**

Table 5: Mean ±SD values of Serum Adiponectin, Resistin and Lectin in Obese Male Subjects according to age.

Table 5 Shows that the mean values of serum adiponectin (1.50±0.76µg/ml), Resistin(6.00±1.45ng/ml) and leptin(8.03±2.59ng/ml) of obese male subjects within 25-35 years was significantly different (P=0.01, P=0.01 and P=0.001) when compared with the mean value of serum adiponectin (3.90±1.58µg/ml), resistin (7.24±2.66 ng/ml) and leptin (12.80±3.59 ng/ml) of obese male subjects within 36-50 years. When the mean values of the adipokines in obese male subjects within 25-35 years was compared with the mean values of the adipokines in obese male subjectswithin 36-50 years, it was found that the mean values of adiponectin and leptin was significantly increased in obese male subjects within 36-50 years when compared with obese male subjects within 25-35 years at P<0.05. However, there was no significant difference in the mean value of resistin in obese male within the age of 25-35 years when compared with the mean value of resistin in obese male within the age of 36-50 years at P>0.05.

Age Group (years)	**25-35 (years)**	**36-50 (years)**	**T-Value**	**P-Value**
Adiponectin µg/ml	**3.0**±0.68	**5.42**±3.38	**10.565**	0.03
Resistin ng/ml	**6.24**±2.59	**7.13**±2.45	7.756	0.245
Leptin ng/ml	**8.51**±3.48	**4.22**±1.29	3.224	0.001

Table 6: Mean ±SD values of Serum Adiponectin, Resistin and Lectin in Obese Female Subjects according to age.

Table 6 Shows that the mean values of serum adiponectin (3.00±0.68µg/ml), Resistin(6.24±2.59ng/ml) and leptin(8.51±3.48ng/ml) of obese female subjects within 25-35 years was significantly different (P=0.03, P=0.04 and P=0.001) when compared with the mean value of serum adiponectin (5.42±3.38µg/ml), resistin (7.13±2.45 ng/ml) and leptin (4.22±1.29 ng/ml) of obese female subjects within 36-50 years. When the mean values of the adipokines in obese female subjects within 25-35 years was compared with the mean values of the adipokines in obese female subjects within 36-50 years, it was found that the mean values of adiponectin was significantly increased in obese female subjects within 36-50 years when compared with obese female subjects within 25-35 years at P<0.05. There was a significant decrease in the mean value of serum leptin in obese female subjects within the age of 36-50 years when compared with the mean value of serum leptin in obese female subjects within the age of 25-35 years. However, there was no significant difference in the mean value of resistin in obese female within the age of 25-35 years when compared with the mean value of resistin in obese female within the age of 36-50 years at P>0.05.

Discussion

Obesity is increasingly becoming an important public health concern among all age groups in most of the developed and underdeveloped world. Greater than 30% of the United States population is obese and in Nigeria, about 8.1%-22.2% are obese and at risk to develop insulin resistance and associated metabolic disorders, including hypertension, hyperlipidemia, fatty liver disease, atherosclerosis, and Type 2 diabetes mellitus (Lana et al.,2004). This study shows that the mean values of serum adipokines of obese subjects was significantly different (P=0.0001, P=0.0001 and P=0.0001) when compared with the mean value of serum adipokines of lean subjects at p<0.05. When the mean values of the adipokines in obese subjects was compared with the mean values of the adipokines in lean subjects, it was found from the study that the mean values of adiponectin and resistin was significantly decreased in obese subjects while serum leptin was significantly increased in obese subjects at P<0.05 respectively. The result of the present study was in agreement with [20] who showed that adipokines were significantly altered in subjects with overweight and obesity in comparison with lean individuals, suggesting that adipokines might be early markers of changing from lean to overweight/obesity status, even before the occurrence of metabolic alterations. In contrast, other studies have shown that serum levels of adiponectin and resistin were significantly higher in obese subjects than in lean subjects [20] Leptin as an adipose tissue-specific adipokine is well known as a key molecule that regulates appetite, energy expenditure, behavior and glucose metabolism. Leptin plasma concentration increases in proportion to body fat mass [21] It is important to highlight that exogenous leptin is efficient in promoting weight loss in obese humans and mice genetic deficient in leptin but not in diet-induced obesity

[22]. There is a feedback loop where insulin stimulates leptin secretion from adipose tissue and leptin is decreased in low insulin states [23] Furthermore, strong positive associations exist between plasma leptin levels and body fat percentage. Other studies point towards leptin resistance. For example, plasma leptin levels and mRNA content decrease in individuals with obesity at the initial time of weight loss but increases as they continue to lose weight. Also, despite the expectation, leptin therapy's termination does not result in weight gain and hyperleptinemia. There is also evidence that hyperleptinemia does not mimic the CNS consequences of chronic weight gain in diet-induced obese (DIO) mice [24] A study found markedly higher fasting and 24-h leptin levels in obese male and female subjects compared with lean controls. The pathophysiological implications of the blunted diurnal variation in leptin levels in obesity is unclear but could play a role in the leptin resistance, thereby contributing to the development and maintenance of obesity [25] Leptin resistance occurs due to the leptin's inability to reach the target cells, reduced LEP-R expression, or disturbed LEP-R signaling. The concentration of leptin in cerebrospinal fluid does not increase further. Furthermore, it appears that excessive plasma leptin levels can result in decreased BBB permeability [26] Leptin has been shown to enhance insulin sensitivity in peripheral tissues and increase glucose uptake and oxidation in skeletal muscles. Moreover, leptin affects thermogenesis through regulation of brown adipose tissue-specific mitochondrial proteins. It is involved not only in lipid and glucose metabolism and immune body response, but also in blood pressure control, blood coagulation and fertility. Leptin is considered a potential marker of obesity-related complications. Elevated leptin levels correspond to atherosclerosis and neuropathy but not diabetic retino- and nephropathy [27] In several studies adiponectin levels measured in serum of obese individuals were significantly lower compared to the normal-weight subjects and correlated negatively with the presence of obesity-related complications [28] Waist circumference, a good predictor of visceral adiposity, was correlated with adiponectin in the whole sample only. The same correlation between abdominal obesity and metabolic disorders has been widely reported. Several explanations for this association between visceral adiposity and adiponectin levels have been proposed. Large visceral adipocytes with greater triglycerides stores may produce less adiponectin than small sub-cutaneous adipocytes, at least under culture conditions [29] and visceral adipocytes may produce factors that reduce adiponectin synthesis. Adiponectin may therefore have the potential to provide an important therapeutic tool to reduce the burden associated with obesity and related chronic diseases including diabetes and cardiovascular disease (CVD). Preliminary evidence points to increased adiponectin levels as a result of different dietary intervention strategies and associated weight loss [30] Since the initial investigation of resistin in numerous rodent models of obesity and insulin resistance, ongoing experimental data has generated further inconsistency. The result of the present study was not in agreement with [31] who showed that there is increased resistin expression in adipose tissue, particularly abdominal depots; furthermore, positive correlations between serum resistin and body fat content have also been reported. On the contrary, several studies have failed to demonstrate such correlations in rodents, with groups also reporting either reduced or no alteration of resistin levels in various models of obesity. Although it is difficult to address such diverse findings using similar, and in some instances the same, rodent models, inconsistencies may depend upon methodological differences [31,32] Resistin is increased in subjects who are obese and the levels of resistin directly correlate with plasma triglyceride levels. Moreover, resistin has been shown to stimulate hepatic VLDL production and secretion due to an increase in the synthesis of Apo B, triglycerides, and cholesterol. Finally, resistin is associated with a decrease in HDL-C and Apo A-I levels [33, 34] Obesity in humans was found to be associated with high resistin serum levels, this view however is not unanimous. High serum resistin level, due to its pro-inflammatory properties, was linked to the development of insulin resistance and type 2 diabetes (T2DM), to atherosclerosis and cardiovascular diseases in rodents and in some human studies. A study by [35] found a positive correlation between its protein level in VAT and low density lipoprotein (LDL) serum level in obese subjects [36] Notably, in isolated human adipocytes, resistin expression is very low, and its content in adipose tissue is proportional to the intensity of macrophages infiltration, which are the main source of this adipokine. Obesity in humans was found to be associated with high resistin serum levels, this view however is not unanimous. High serum resistin level, due to its pro-inflammatory properties, was linked to the development of insulin resistance and type2 diabetes (T2DM), to atherosclerosis and cardiovascular diseases in rodents and in some human studies [37] When the mean values of the adipokines in obese male subjects was compared with the mean values of the adipokines in lean male subjects it was also found that the mean values of adiponectin and resistin was significantly decreased in obese male subjects while the mean value of serum leptin was significantly increased in obese male subjects at $P<0.05$ respectively. When the mean values of the adipokines in obese female subjects was compared with the mean values of the adipokines in lean female subjects it was also found that the mean values of adiponectin and resistin was significantly decreased in obese female subjects while the mean value of serum leptin was significantly increased in lean female subjects at $P<0.05$ respectively. When the mean values of the adipokines in obese male subjects was compared with the mean values of the adipokines in obese female subjects it was found that the mean values of adiponectin, resistin and leptin was decreased in obese male subjects when compared with obese female subjects though not significantly at $p>0.05$. The reason for the difference between male and female is that they are more expressed in omental and abdominal subcutaneous white fat than in adipose tissue from the thigh and the breast [38, 39] When the mean values of the adipokines in obese male subjects within 25-35 years was compared with the mean values of the adipokines in obese male subjects within 36-50 years, it was found that the mean values of adiponectin and leptin was significantly increased in obese male subjects within 36-50 years

when compared with obese male subjects within 25-35 years at P<0.05 [40]. However, there was no significant difference in the mean value of resistin in obese male within the age of 25-35 years when compared with the mean value of resistin in obese male within the age of 36-50 years at P>0.05. When the mean values of the adipokines in obese female subjects within 25-35 years was compared with the mean values of the adipokines in obese female subjects within 36-50 years, it was found that the mean values of adiponectin was significantly increased in obese female subjects within 36-50 years when compared with obese female subjects within 25-35 years at P<0.05. There was a significant decrease in the mean value of serum leptin in obese female subjects within the age of 36-50 years when compared with the mean value of serum leptin in obese female subjects within the age of 25-35 years. However, there was no significant difference in the mean value of resistin in obese female within the age of 25-35 years when compared with the mean value of resistin in obese female within the age of 36-50 years at P>0.05.

Conclusion

In conclusion, adipokines may decrease the insulin sensitivity of tissues and induce inflammation and the development of atherosclerosis, diabetes and psoriasis, as well as diabetic foot.

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