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# Leptin Concentrations in African Blacks with Metabolic Syndrome and Type 2 Diabetes Mellitus

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**Abstract: Background:** Mortality rate from metabolic/cardiometabolic syndromes (MS/CMS) and type 2 diabetes mellitus (DM2) are highly prevalent in African blacks known with higher mortality from cardiovascular diseases than caucasians. Leptin, a satiety-regulating hormone increases in obesity and is associated with cardiovascular risk and prediction of MS. This study is designed to evaluate leptin in Nigerians with MS and DM2 to assist in the early diagnosis and prevention of metabolic diseases. **Methods:** 136 participants (45 with MS, 47 with DM2 and 44 apparently healthy individuals (controls)) aged 18-80 years were included in a cohort study at the University College Hospital, Ibadan. Measures of adiposity-%body fat, body mass index (BMI), waist and hip circumferences (WC and HC respectively), waist to hip ratio (WHR), and blood pressure were obtained by standard methods. 10 ml of blood were obtained from each participant after an overnight fast (10-14 h) and analysed for leptin, total cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL), and glucose by standard methods while low density lipoprotein (LDL) was calculated. Data obtained were analysed statistically with SPSS software version 16.0. **Results:** Weight, BMI, WC, HC, WHR, %body fat, blood pressure, TG, LDL-C, and glucose were significantly higher while HDL-C was significantly lower in individuals with MS and DM2 compared with controls ($p<0.039$). Leptin levels were significantly higher in MS group and not in DM2 group when compared with controls ($p=0.000$). Leptin did not correlate with any of the biochemical indices ($p>0.05$) tested but correlated significantly with different measures of adiposity in all groups. Leptin correlated negatively but significantly with blood pressure in MS group only. **Conclusion:** Increases in leptin levels in both MS and DM2 groups might reflect adiposity. Observed high leptin levels in MS group might be a compensatory mechanism for maintenance of weight/fat loss and blood pressure. Its routine analysis may assist in assessing adiposity associated with MS and DM2 for probable prevention of metabolic diseases.

**Key words:** Leptin, type2 diabetes mellitus, metabolic syndrome, dyslipideamia, adiposity, African blacks.

## 1. Introduction

The prevalence of diabetes mellitus is very high worldwide [1]. Metabolic and cardiometabolic syndromes (MS/CMS) are related syndromes of global concern [2] that identify individuals at greatest risk for developing cardiovascular diseases (CVD) and predispose to type2 diabetes mellitus (DM2) worldwide [1-7]. MS, a concurrence of disturbed glucose and insulin metabolism, overweight and

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abdominal fat distribution, mild dyslipideamia, and hypertension, is associated with subsequent development of DM2 and CVD(8). This syndrome, formerly known as Syndrome X and Insulin Resistance Syndrome was recently known as cardiometabolic syndrome [9, 10]. CMS is a constellation of metabolic, renal, and cardiovascular risk factors including central or visceral obesity, hypertension, insulin resistance/ hyperinsulinemia, dyslipideamia, microalbuminuria, oxidative stress, increased inflammation, and hypercoagulability [10].

Individuals from black heritage have higher prevalence and mortality rate from CVD than caucasians [7]. Nigeria is Africa's most populous country with over 140 million people [11] of black heritage where communicable diseases such as malaria and human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS) still cause significant morbidity and mortality [12]. The increased prevalence of non-communicable diseases is well documented in Nigeria [12-16]. The prevalence of type 2 diabetes (DM2) has risen from less than 3% [14] to 20.5% [15] and MS is thought to affect 12.1-30.8% of Nigerians [12, 15, 16, 19]. A study of 1458 adults in South Eastern Nigeria showed prevalence of 18.0% and 10% of CMS in a semi-urban and rural community respectively. This prevalence of CMS increased to 34.7% and 24.7% respectively when a population with hypertension was studied [20]. These observations may have implications for the health-care sector [15]. Identification of those at risk for early diagnosis and prevention might be beneficial to individuals particularly of African heritage [7] and reduce the strain on the already insufficient health resources in Africa [12].

Leptin is an adipocyte-derived, satiety-regulating hormone that acts within the hypothalamus and other brain sites [21], associated with cardiovascular risk and prediction of metabolic syndrome in both males and females [22]. It has been shown to be proliferative, proinflammatory, prothrombotic, and pro-oxidative [23]. The routine analysis of leptin has therefore been suggested to be beneficial in the early prediction of MS [22].

Hyperleptinaemia is common in obesity and reflects increased adiposity and leptin resistance [21, 23]. Impaired leptin causes ectopic accumulation of triglycerides in non-adipose organs and tissues such as skeletal muscles and pancreas. Accumulation of triglycerides and longchain free fatty acids in these organs triggers ceramide synthesis that causes apoptosis through stimulation of inducible nitric oxide synthase. Lipotoxicity developing in skeletal muscle and pancreas causes insulin resistance and β cell dysfunction, respectively, and could be responsible for the development of DM2 [23]. Chronic overexpression of central leptin induces a leptin resistance that mimics many of the characteristics associated with diet-induced or adult-onset obesity including reduced leptin receptors, diminished signaling, and impaired responsiveness to exogenous leptin. Exaggerated diet-induced obesity due to blockade of leptin receptors by leptin antagonist has been demonstrated [21]. Leptin levels were reported to be higher in pre-diabetic and diabetic than in normoglycaemic men [24]. This study is therefore designed to evaluate leptin and its relationship with other metabolic and cardiometabolic risk factors in Nigerians with MS and DM2 to assist in the early diagnosis and prevention of metabolic diseases.

## 2. Subjects and Methods

### *2.1 Study Design and Duration*

The study was a cross-sectional survey conducted over a period of 6 months after ethical approval was obtained from the Joint Ethical Committee of University of Ibadan/University College Hospital, Ibadan, Nigeria (UI/UCH).

### *2.2 Subjects*

A total of 136 participants (87 females and 49 males)

of age range (18-80 years) were recruited for this study after informed consent. These were 47 patients with DM2, 45 individuals with MS and 44 apparently healthy individuals. Those on medications (antihypertensive, lipid lowering, and hormonal medications), cardiovascular diseases like stroke and individuals who did not give consent were exempted. Participants were part of cohort study on Risk Assessment for type 2 Diabetes Mellitus and Dementia in Individuals with Metabolic Syndrome at the University College Hospital, Ibadan.

*2.3 Individuals with Type 2 Diabetes Mellitus*

These were participants diagnosed with type 2 diabetes mellitus without renal diseases by consultant physicians. They were recruited while attending the diabetic clinic at the Medical out Patient department of the UCH, Ibadan. Their mean (s.e) microalbuminuria to creatinine ratio on spot urine of 2.98 mg/g (1.71) was within normal reference range [10].

*2.4 Individuals with Metabolic Syndrome*

These participants were randomly recruited within Ibadan using International Diabetic Federation (IDF) criteria (abdominal obesity: waist circumference (WC) >94 cm and at least two of the following: hypertriglyceridemia (plasma triglycerides (TG) > 150 mg/dl), low HDL-C (plasma HDL-C < 40 mg/dl), high blood pressure (blood pressure >130/85 mmHg) and high fasting glucose (plasma glucose > 100 mg/dl) [25].

*2.5 Controls*

These were apparently healthy, non-diabetic participants with normal body mass index (BMI) without MS using the IDF criteria, randomly recruited within Ibadan. Fasting plasma glucose was determined to exclude DM2.

*2.6 Sample Collection*

10 ml of venous blood sample was asceptically obtained by venopuncture from the participants after an overnight fast (10-14 h). 4 ml was dispensed into potassium ethylene diamine tetra acid ($K_3$EDTA) tube for the determination of lipid profile (total cholesterol (TC), TG, and HDL-C)). 2 ml was dispensed into fluoride oxalate tube for plasma glucose estimation while 4 ml was dispensed into plain serum tubes and kept for 1-2 hours to clot to obtain serum for the estimation of leptin. All samples were centrifuged at 500 g for 5 min after which plasma and serum were aspirated in small aliquots into clean vials and stored at -20$^{o}$C until analysis was done.

*2.7 Anthropometric Indices and Blood Pressure Measurements*

Weight, height, BMI, WC and hip circumference (HC ) and waist /hip ratio (WHR), percentage body fat (%body fat) and blood pressure (systolic and diastolic) were obtained from the participants by standard methods as described elsewhere [19]. Body fat was measured using Omron BF400 (Omron Healthcare. Co. Ltd, Ukyo-ku Kyoto, Japan).

*2.8 Biochemical Indices in Blood*

Serum leptin was estimated by enzyme immunoassay (Diagnostic Automation, Inc., CA). Plasma TG, TC, HDL and glucose were estimated by enzymatic methods using commercial kits (Dialab Produktion, Austria) while LDL-C was calculated using Friedwald's formula [26].

*2.9 Statistical Analysis*

Data were analyzed using the Statistical Package for Social Sciences (SPSS) software 17.0 version. Analysis of Variance (ANOVA) and Posthoc tests were used for comparison of variables, while Pearson's correlation coefficient was used to find relationships between quantitative variables. Two-tailed independent t-test of significance, at 95% confidence limit with $p<0.05$ were considered significant for the variables.

## 3. Results

### *3.1 Comparisons of Blood Pressure, Anthropometric and Biochemical Indices in MS, DM2 and Control Groups*

Table 1 compares the mean ± s.e of height, weight, BMI, WC, HC, WHR, % body fat, systolic and diastolic blood pressure, TG, TC, LDL-C, HDL-C, glucose and leptin of individuals with MS, DM2 and controls. All indices tested except height and TC showed significant differences ($p<0.010$) among MS, DM2 and control groups.

PostHoc test showed significantly higher differences in weight, BMI, WC, HC, WHR, %body fat, systolic and diastolic blood pressure, TG, LDL-C and glucose but significantly lower difference in HDL-C in individuals with MS and DM2 compared with controls ($p<0.039$). Leptin levels were significantly higher in MSgroup and not DM2 group when compared with controls ($p=0.000$). BMI, HC, %body fat, systolic and diastolic blood pressure, TG and leptin were significantly higher while glucose was significantly lower in MS than DM2 groups ($p<0.021$) (Table 2).

**Table 1 Comparison of blood pressure, anthropometric and biochemical indices in individuals with metabolic syndrome, type 2 diabetes mellitus and controls using ANOVA**

| Index | Control n=44 | Metabolic Syndrome n=45 | Type 2 Diabetes Mellitus n=47 | F | p |
|---|---|---|---|---|---|
| Height (m) | 164.84±1.243 | 156.35±3.619 | 162.67±1.360 | 3.530 | 0.032 |
| Weight (kg) | 58.97±1.143 | 75.44±2.456 | 71.47±2.257 | 17.172 | 0.000* |
| BMI (kg/m$^2$) | 21.65±0.353 | 29.29±0.843 | 27.01±0.755 | 31.601 | 0.000* |
| Waist circumference (cm) | 78.82±0.859 | 103.13±1.912 | 99.57±1.712 | 68.216 | 0.000* |
| Hip circumference (cm) | 91.34±0.883 | 106.51±1.836 | 100.26±1.670 | 24.237 | 0.000* |
| WHR | 0.86±0.007 | 0.97±0.014 | 0.99±0.008 | 46.938 | 0.000* |
| Systolic BP (mmHg) | 116.36±1.263 | 147.78±3.451 | 131.49±3.261 | 29.464 | 0.000* |
| Diastolic BP (mmHg) | 73.41±0.793 | 86.44±1.774 | 77.55±1.417 | 22.411 | 0.000* |
| Percentage body fat | 20.80±1.212 | 41.07±1.092 | 33.78±1.377 | 66.981 | 0.000* |
| Triglyceride (mg/dl) | 57.20±3.73 | 90.76±5.73 | 75.17±4.34 | 12.619 | 0.000* |
| Total Cholesterol (mg/dl) | 148.75±4.01 | 161.07±7.12 | 161.94±6.89 | 1.386 | 0.254 |
| LDL-C (mg/dl) | 84.66±4.30 | 105.51±6.61 | 108.68±6.43 | 4.812 | 0.010* |
| HDL-C (mg/dl) | 53.50±1.52 | 36.91±1.69 | 38.21±1.78 | 29.950 | 0.000* |
| Glucose (mg/dl) | 78.30±1.28 | 99.33±6.62 | 136.04±6.72 | 27.722 | 0.000* |
| Leptin (ng/ml) | 9.73±1.38 | 28.11±3.37 | 16.19±2.68 | 12.421 | 0.000* |

values are mean ± s.e, *= significant, n= number of subjects, F=analysis of variance, p=probability, BMI=body mass index, WHR= waist to hip ratio, BP = blood pressure, LDL-C=low density lipoprotein cholesterol, HDL-C = high density lipoprotein cholesterol, ANOVA=analysis of variance.

### *3.2 Relationship of Leptin with Blood Pressure, Anthropometric and Biochemical Indices in MS, DM2 and Control Groups*

Table 3 shows correlation of leptin with blood pressure, anthropometric biochemical indices tested in individuals with MS, DM2 and controls. Correlations of leptin with all biochemical indices tested -TG, TC, LDL-C, HDL-C and glucose were not significant ($p<0.05$). However, leptin correlated positively and significantly with BMI, HC and %body fat ($p<0.05$) in all groups (MS, DM2 and controls) tested. Leptin correlated negatively but significantly with blood pressure (systolic and diastolic) in MS group only. Leptin also correlated significantly and positively with WC in diabetic group only and WHR in controls only ($p<0.05$). The correlations of leptin with height in both diabetic and control groups were negative but significant ($p<0.05$).

**Table 2 Comparison of blood pressure, anthropometric and biochemical indices in individuals with metabolic syndrome, type2 diabetes mellitus, and controls using post-hoc test.**

| Index | Groups | Mean difference | p-value |
|---|---|---|---|
| Weight (kg) | MS vs Control | 16.463 | 0.000* |
| | DM2 vs Control | 12.500 | 0.000* |
| | MS vs DM2 | 3.963 | 0.172 |
| Body Mass Index (kg/m$^2$) | MS vs Control | 7.639 | 0.000* |
| | DM2 vs Control | 5.360 | 0.000* |
| | MS vs DM2 | 2.279 | 0.020* |
| Waist circumference (cm) | MS vs Control | 24.315 | 0.000* |
| | DM2 vs Control | 20.756 | 0.000* |
| | MS vs DM2 | 3.559 | 0.110 |
| Hip circumference (cm) | MS vs Control | 15.170 | 0.000* |
| | DM2 vs Control | 8.914 | 0.000* |
| | MS vs DM2 | 6.256 | 0.004* |
| Waist-Hip Ratio | MS vs Control | 0.109 | 0.000* |
| | DM2 vs Control | 0.130 | 0.000* |
| | MS vs DM2 | 0.021 | 0.138 |
| Percentage Body Fat | MS vs Control | 20.271 | 0.000* |
| | DM2 vs Control | 12.981 | 0.000* |
| | MS vs DM2 | 7.290 | 0.000* |
| Systolic blood pressure (mmHg) | MS vs Control | 31.414 | 0.000* |
| | DM2 vs Control | 15.126 | 0.000* |
| | MS vs DM2 | 16.288 | 0.000* |
| Diastolic blood pressure (mmHg) | MS vs Control | 13.035 | 0.000* |
| | DM2 vs Control | 4.114 | 0.038* |
| | MS vs DM2 | 8.891 | 0.000* |
| Triglyceride(mg/dl) | MS vs Control | 33.551 | 0.000* |
| | DM2 vs Control | 17.966 | 0.007* |
| | MS vs DM2 | 15.585 | 0.019* |
| High density lipoprotein cholesterol (mg/dl) | MS vs Control | -16.589 | 0.000* |
| | DM2 vs Control | -15.287 | 0.000* |
| | MS vs DM2 | -1.302 | 0.580 |
| Low- density lipoprotein cholesterol (mg/dl) | MS vs Control | 20.852 | 0.015* |
| | DM2 vs Control | 24.022 | 0.005* |
| | MS vs DM2 | -3.170 | 0.703 |
| Glucose (mg/dl) | MS vs Control | 21.038 | 0.009* |
| | DM2 vs Control | 57.747 | 0.000* |
| | MS vs DM2 | -36.709 | 0.000* |
| Leptin (ng/ml) | MS vs Control | 18.384 | 0.000* |
| | DM2 vs Control | 6.464 | 0.084 |
| | MS vs DM2 | 11.920 | 0.002* |

vs=versus, *= significant, p=probability, MS=metabolic syndrome, DM2=type2 diabetes mellitus.

## 4. Discussion

Overweight and obesity are epidemic across the globe. In our study, known cardiometabolic risk factors showed significantly higher differences in weight, BMI, WC, HC, WHR, %body fat, systolic and diastolic blood pressure, TG, LDL-C and glucose but significantly lower differences in HDL-C in individuals with MS and DM2 compared with controls ($p<0.039$). These observations are consistent with earlier reports [12, 13, 15, 18-20] and confirm findings that no single adiposity measure directly identifies MS despite significant correlations of measures of adiposity with cardiovascular risk [27]. However, significantly higher serum leptin level was demonstrated in MS group than both DM2 and control groups in our study ($p=0.000$). MS has been shown as prediabetic phase [19]. Al-Daghri et. al. [24] reported higher leptin levels in both pre-diabetic and diabetic than normoglycaemic men. Our observations partly

agree with theirs as comparison of leptin levels between DM2 and control groups showed no significant difference in our study (p=0.084). We also did not find significant correlations of leptin with glucose and lipid concentrations in MS, DM2 and controls (p>0.075). Lean diabetics are known to demonstrate reduced levels of leptin [28] and dyslipidaemias do not appear to be strongly associated with leptin levels in humans [29].

**Table 3 Correlation of leptin with anthropometric, blood pressure and biochemical indices in individuals with metabolic syndrome, type2 diabetes mellitus and controls using pearson's correlation coefficient.**

| Indices | Leptin | | |
|---|---|---|---|
| | Metabolic Syndrome n = 45 (r, p-value) | Type2 Diabetes Mellitus n = 47 (r, p-value) | Controls n = 44 (r, p-value) |
| Height (cm) | 0.010, 0.947 | -0.361, 0 .013* | -0.328, 0.030* |
| Body Mass Index (kg/m$^2$) | 0.326, 0 .029* | 0.434, 0.002* | 0.527, 0.000* |
| Waist Circumference (cm) | 0.231, 0.127 | 0.373, 0.010* | 0.203, 0.187 |
| Hip Circumference (cm) | 0.430, 0.003* | 0.345, 0.018* | 0.569,0.000* |
| Waist Hip Ratio | -0.228, 0.133 | 0.074, 0.623 | -0.372, 0.013* |
| Systolic B.P (mmHg) | -0.412, 0.005* | -0.045, 0.765 | 0.066, 0.673 |
| Diastolic B.P (mmHg) | -0.370, 0.012* | -0.019, 0.901 | -0.072, 0.643 |
| Percentage body fat (%)<br>Triglyceride (mg/dl) | 0.392, 0.008*<br>0.027, 0.858 | 0.476, 0 .001*<br>0.207, 0.164 | 0.734, 0 .000*<br>-0.204, 0.184 |
| Total cholesterol (mg/dl) | -0.205, 0.178 | 0.262, 0.075 | -0.118, 0.447 |
| Low Density Lipoprotein (mg/dl) | -0.219, 0.149 | 0.188, 0.206 | -0.161, 0.295 |
| High Density Lipoprotein (mg/dl) | -0.014, 0.929 | 0.237, 0.109 | 0.196, 0.203 |
| Glucose (mg/dl) | -0.113, 0.45 | -0.056, 0.707 | 0.147, 0.341 |

*= significant; r = pearson correlation; p = probability; BP = blood pressure.

Leptin behaves as a potent anorexic and energy-enhancing hormone in most young or lean animals [30]. The significantly positive correlations between leptin and different measures of adiposity-BMI, HC and %body fat (p<0.05) in all groups (MS, DM2 and controls) in our study, suggest that plasma leptin reflects adipose tissue mass and is greatly increased in obesity [23]. BMI is a measure of general adiposity and HC is a measure of subcutaneous adiposity [31]. Subcutaneous fat tissue is the major source of leptin [32]. We also observed significant correlations of leptin with measures of adiposity in specific groups studied. Leptin correlated positively and significantly with WHR in controls only (p<0.05) and WC in diabetic group only (p<0.05). WC is a measure of abdominal adiposity [31]. Visceral adipose tissue secretes leptin, and obesity especially visceral adipose tissue accumulation, increases the risk of developing DM2 [32-34]. The preference of WC over other measures of central adiposity in studies of obesity and cardiovascular disease risk factors has been reported [35]. Leptin also correlated negatively but significantly with height in both diabetic and control groups (p<0.05) in our study. WC and waist-height ratio have been shown as two among the best predictors for individual MS components [27]. Our findings show that selective dysregulation of different body fat depots probably plays an important role in the metabolic complications of obesity [33].

It appears that the observed increases in leptin levels in our study reflect adiposity associated with MS and DM2 and may be a compensatory mechanism for the maintenance of weight/fat loss. Luke et al. [36] showed exponential response of leptin to increase in body fat stores in blacks. Association of elevated plasma leptin with obesity and not necessarily with the type 2

diabetic state has been reported [34]. A dose response relationship with weight and fat loss was observed with subcutaneous recombinant leptin injections in both lean and obese subjects. Thus, administration of exogenous leptin appears to induce weight loss in some obese subjects with elevated endogenous serum leptin [37]. Moreover, we observed negative but significant correlation of leptin with blood pressure (systolic and diastolic) in MS group only ($p<0.05$) suggesting leptin's enhancement of body steady state or homeostasis. Leptin has been shown in previous studies to promote nitric oxide release by the vascular endothelium that could potentially decrease blood pressure [23, 38, 39].

In conclusion, we observed cardiometabolic factors in individuals with MS and DM2 with none specifically identifying MS. Increases in leptin levels in groups particularly MS in this study probably reflect adiposity associated with MS and DM2 and might be a compensatory mechanism for maintenance of weight/fat loss and blood pressure. The routine analysis of serum leptin may therefore assist in the assessment of adiposity associated with MS and DM2 for probable prevention of metabolic diseases.

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