Assessment of Serum Nesfatin-1 Level, Cardiovascular Parameters, and Metabolic Risk Factors among Obese and Non-Obese Adults

ABSTRACT

Aim: To assess the association between serum Nesfatin-1 level and cardio metabolic risk factors among obese and non-obese adults in order to identify cardiovascular abnormalities associated with obesity and explore potential correlations with Nesfatin-1 levels..

Study design: The study utilized a comparative descriptive design and purposive sampling technique to recruit participants.

Place and duration of study: The study was carried out at the Department of Family Medicine, Ekiti State University Teaching Hospital, Ado-Ekiti, Ekiti State. This study was carried out for the duration of (3) Months

Types of clinical study: This study is a case-control study under observational study

Methodology: The study involved 120 participants, divided into two groups based on their body mass index. The chosen participants were divided into two; Group A (BMI $\geq$ 30.0 kg/m^2) and Group B (BMI 18.5-24.9kg/m^2). Clinical and demographic parameters were obtained through a structured questionnaire, and anthropometric measurements were taken. Serum nesfatin-1, leptin, and tumour necrosis factor alpha were quantitatively assayed. Fasting blood glucose, total cholesterol, and low density lipoprotein were determined.

Results: The mean serum nesfatin-1 (ng/mL), leptin (ng/mL) high density lipoprotein (mmol/L) and tissue necrosis factor–alpha (pg/mL) levels were significantly lower (t = -4.256; p = 0.001 and t = -5.106; p = 0.054, t = -5.282; p = 0.001) in the obese participants (4.25 ± 0.45, 6.22 ± 0.92, 0.854 ± 0.57, 49.76 ± 2.54 respectively) when compared with the non-obese participants (4.64 ± 0.55, 6.56 ± 0.50, 0.942 ± 0.077, 55.67 ± 4.81 respectively). A strong positive correlation existed between total cholesterol (r = 0.290; p = 0.025), low density lipoprotein (r = 0.205; p = 0.116), triglyceride (r = -0.157; p = 0.231) and systolic blood pressure (r = 0.075; p = 0.05) with serum nesfatin-1 among obese participants. A strong positive correlation was also observed between serum nesfatin-1 with fasting blood glucose and tumor necrosis factor-alpha (r = 0.041, p = 0.756; r = 0.070, p = 0.592 respectively) in obese participants.

Conclusion: Nesfatin-1 plays a role in the development of cardiovascular and metabolic diseases and hence could be regarded as a potential biomarker for metabolic and cardiovascular risk in obesity.

KEYWORDS: Nesfatin-1, cardiometabolic risks, tumour necrosis factor-alpha, obesity.

1.0 INTRODUCTION

The excess growth of body fat tissue is one of the characteristics found in obese individuals and is sporadically becoming a serious global epidemic health challenge that plagues millions of children and adults across the globe (1). Research has confirmed that the major regulator of energy homeostasis is the adipose tissue (2). However, adipose tissue will grow and expand excessively when intake and expenditure of energy are out of balance (3). Obesity is a disorder that is brought about by interplay of numerous genetic, nutritional, lifestyle, and environmental variables (4). The primary pathogenesis of obesity involves controlling cellular processes and physical inactivity to either an increase in appetite or a decrease in calorie usage. This dysregulation causes excessive adiposities formation to occur, which elevates cytokine release and results in the manifestation of the circulatory complications. Hyperlipidemia, atherosclerosis, and anomalies of the cardiovascular system are linked to these complications. Adipose tissue increases significantly as a result of obesity, and this increase in adipose tissue necessitates an increased metabolism rate. Effectively, there is a corresponding increase in the size or number of blood vessels (5). Being overweight negatively affects blood flow as well as the structure and function of cardiac arteries due to increases in total blood volume and cardiac output (6). Furthermore, cardiac output, cardiac stroke volume, and heart rate all have a linear connection. As a result, those who have a high BMI are

prone to have an increase in heart workload than people who are of normal weight (6).Therefore, controlling obesity is crucial for preventing and treating these associated conditions. Other conditions attributed with over -weight include diabetes mellitus, cardiovascular diseases with motion diseases. Likewise, it possesses a consequential impact on such person's social, financial and psychological status which tends to cause depression (7).

Emerging cardiovascular risk factors have been identified, one of which is a 82-equino acid peptide called nesfatin-1, discovered by Oh *et al* in 2006. The hormone circulates extensively in the nervous system both the central and the peripheral (8-9). It had been proven to play a major role modulating food intake with lipid metabolism, preventing fat buildup, promoting lipid breakdown, and generally inhibiting the onset of lipid-related disorders like obesity and metabolic syndrome (10). It significantly impacts the relationship between dietary intake and a body mass index. It contributes crucially in controlling appetite and storage of fat. Increased nesfastin-1 in the hypothalamus relates with a decrease in hunger, "a sense of fullness," and an elevated probability of a loss in weight and body fat (11). Nesfatin-1 concentration in circulation had been found to be abnormal in heart diseases (12). Additionally, higher blood lipids and lipoproteins are associated to obesity (13). Heightened levels of triglycerides, VLDL, Apo B, and non-HDL-C are among the lipid imbalances frequently observed in people suffering from obesity (14).Generally, HDL-C and Apo A-I levels are low (15). Most of the time, LDL-C levels are within the normal range, but there is an elevation in the small dense LDL level, which leads to a rise in the number of LDL particles (16).The small dense LDL particles are assumed to be more pro-atherogenic compared to the large LDL particles for a variety of reasons (17). A rise in abnormal lipid levels which is related to an increased BMI can results in cardiovascular and metabolic diseases

Morbidity and mortality are significantly impacted by obesity. nesfatin-1, a regulator of body mass and appetite, is one of the pathophysiological elements of obesity. The purpose of this research is to examine the correlation between serum nesfatin-1 level with the anthropometric indices, cardiovascular parameters and other metabolic risk factors in obese and non-obese adults in Nigeria because there have been very few studies that have looked at the relationship between serum nesfatin-1 levels and cardio-metabolic risk factors among obese with non-obese individuals.

2.0 MATERIALS AND METHODOLOGY

2.1 Materials

Height and weight of all participants were measured to the nearest 0.1cm and 0.1kg respectively using WunderRE300digital scale. Waist, neck and hip circumferences were assessed with a tape measure. The subject stood with feet closed together, the tape was placed mid-point between sub-coastal and supra-iliac landmark for the measurement of waist circumference while for the hip circumference. The tape was placed on the greater trochanter with the tape parallel to the floor. The neck circumference measurement was done between the mid-cervical spine and mid anterior neck just below the laryngeal prominence with head in the Frankfurt plane. The body mass index was calculated using Quetelet Formula and classified using WHO classification method

At a sitting position after 5 minutes of rest, the systolic blood pressure, diastolic blood pressure and pulse rate were measured using a validated digital blood pressure monitor (OMRON). Measurement of resting blood pressure and pulse rate was done three times, two minutes apart. Mean arterial blood pressure, pulse pressure and rate pressure product were derived from the resting blood pressure figures.

2.2 Study Area

Case control observational study method was used. The participants included the patients that attended the outpatient unit of Department of Family Medicine, Ekiti State University Teaching Hospital, Ado-Ekiti, Ekiti State for follow-up. The study included participants who met the study inclusion criteria and gave their consent. Clinical and demographic characteristics such as age, gender, marital status, and occupation were gathered using a pre-tested structured interview questionnaire. Ethical clearance was sought and granted by The Ethics and Research Committee of Ekiti State University Teaching Hospital Ado-Ekiti, Ekiti state.

2.3 Study Population and Period of Study

A total of 120 participants were chosen based on their Body Mass Index (BMI).The chosen participants were divided into two groups: Group A consists of (60) sixty participants with BMI ≥ 30.0 kg/m² and Group B consists of (60) sixty participants with BMI between 18.5 and 24.9kg/m². This study was carried out for duration of three (3) Months (February-April 2023). At least 6 samples and filled questionnaires were collected per day centrifuged and serum separated for analysis

2.4 Inclusion Criteria

The Inclusion criteria for group A is a BMI ≥ 30.0kg/m² and age range of 18-65 years while the inclusion criteria for group B is a BMI of 18.5-24.9kg/m² and age range of 18-65 years.

2.5 Exclusion Criteria

Exclusion criteria for both groups include Age < 18 years or > 65 years, patients that are critically ill, pregnant or lactating mothers.

2.6 Data Collection

A structured questionnaire was designed and administered to the participants to obtain relevant demographic information and clinical characteristics

2.7 Administration of Questionnaires

Well structured and approved questionnaires were given to consented participants to provide information on their socio-demography and health status. Confidentiality was maintained as no details related to participant's identity were used.

2.8 Anthropometry measurements and laboratory evaluations

All participants underwent physical examinations and laboratory evaluations. Height was measured with a standard stadiometer. (OMRI height meter) Weight and height were recorded to the nearest 0.1 kg and 0.1 cm, respectively. Blood samples for glucose, lipid profile Tumour necrosis factor-alpha leptin and nesfatin-1 were taken after 10–12 h overnight fasting. Cut-off points for abnormal lipid levels (total cholesterol [TC] ≥200mg/dL, low-density lipoprotein cholesterol [LDL-C] ≥ 130mg/dL, high-density lipoprotein cholesterol [HDL-C] ≤ 35mg/dL, and triglycerides [TG]) ≥150 mg/dL) were from the Third Report of the National Cholesterol Education Program (17) and the American Diabetes Association.(18) Dyslipidemia was defined as presence of one or more abnormal serum lipid levels.

2.9 Sample Collection

Overnight fasted participants were allowed to sit comfortably, the arm was gently palpated to identify suitable vein and also to assess the suitability such as being bouncy, soft, straight and capable of refilling when compressed with a large lumen and well supported. A tourniquet was

applied to the upper arm approximately 7-10cm away from the chosen site, the radial pulse must still be palpable. The site was then cleaned with methylated spirit swab and allowed to dry before the insertion of the needle. A total of 10ml of blood sample was withdrawn into plain sample bottles allowed to clot for one hour at room temperature, after full clot retraction the sample is then centrifuged at 4500rpm for 5 minutes at 4ºC. the serum is then extracted and stored at -20ºC (19) until analyzed according to the manufacturer protocols.

For plasma sample, the blood was collected in an EDTA bottle, allowed to clot for one hour and then centrifuged at 3000rpm for 10 minutes. Plasma concentrations of nesfatin-1 were measured in duplicate and 10 replicates per enzyme-linked immunosorbent assay (ELISA) plate were used as internal quality controls. Human plasma nesfatin-1 levels were measured using a commercially available ELISA kit (Innovative Research.USA) according to the manufacturer's instructions. The assay has a detection sensitivity of 7.8 pg/ml and Optical density value for each well were determined at once with a micro-plate reader set at 450nm.

For lipid analysis, the blood sample (4ml) was collected into lithium heparin bottle. Plasma total cholesterol, high- and low-density lipoprotein cholesterol and triglyceride concentrations were determined using Abell-Kendall protocol. For glucose analysis, the blood sample (3ml) was collected into fluoride oxalate bottle while the plasma glucose were determined using glucose oxidase method.

Data obtained was analyzed using descriptive and inferential statistics A P-value of ≤ 0.05 was taken as statistically significant.

3.0 RESULTS AND DISCUSSION

3.1 Results

Table 1: Serum Biochemical Parameters Between Obese and Non-Obese Participants

	Group A (n=60)	Group B (n=60)	t	p-value
Nesfatin-1(ng/mL)	4.25±0.45	4.64±0.55	-4.256	< 0.001*
FBG (mmol/L)	5.23 ± 0.65	4.45 ± 0.62	-1.181	<0.001*
Leptin(ng/mL)	6.22 ± 0.92	6.56 ± 0.50	-5.106	<0.001*
TNF-α(ng/mL)	49.76±2.54	55.67±4.81	-5333	<0.001*
Cholesterol(mmol/L)	3.55±0.42	3.68±1.86	8.670	<0.001
TAG (mmol/L)	8.16±0.48	8.15± 0.76	0.000	1.000
VLDL (mmol/L)	1.73±0.09	1.74±.0.45	5.067	<0.001
HDL(mmol/L)	0.854±0.57	0.942±0.77	-5.282	<0.001*
LDL(mmol/L)	2.52±0.70	2.57±1.98	11.151	<0.001

Significant p-value < 0.05

Table 2: Relationship of Nesfatin-1 With Indices of Lipid Profile Among Obese and Non Obese Participants

Parameters	Group A(n=60)		Group B (n = 60)	
	r	p –value	r	p - value
Cholesterol(mmol/L)	0.290	0.025*	0.093	0.001*
TG (mmol/L)	-0.157	0.231	0.041	0.001*
VLDL(mmol/L)	0.065	0.621	0.041	0.001*
HDL(mmol/L)	0.154	-0.239	-0.106	0.01
LDL(mmol/L)	0.205	0.116	0.144	0.001*

Lipid Profile Increases or Decreases in Response to Changes in Serum Nesfatin-1 (Except For LDL In Obese and TAG

In Non-Obese), p-value < 0.05.

Table 3: Relationship of Nesfatin-1 with Anthropometric Indices among Obese and Non-Obese Participants

Anthropometric Parameters	**Group A (n=60)**		**Group B (n=60)**	
	r	**p-value**	**r**	**p-value**

Body Mass Index (kg/m2)	-0.067	0.600	0.106	0.420
Hip Circumference (cm)	0.112	0.392	0.151	0.251
Waist Circumference(cm)	-0.082	0.532	-0.067	0.611
Waist/Hip Ratio	0.062	0.001	-0.032	0.012
Neck Circumference (cm)	-0.173	0.186	-0.133	0.312

A Positive Correlation (r-0.112, p=0.392) in Obese and (r = 0.151; p = 0.251) in Non-Obese, p-value < 0.05.

Table 4: Relationship of Nesfatin-1 With Indices of Blood Pressure Factors Among Obese and Non- Obese Participants

Indices of Blood Pressure	**Group A (n=60)**		**Group B (n=60)**	
	r	**p-value**	**r**	**p-value**

SBP (mmHg)	0.075	0.05*	0.050	0.702
PR (beats/minute)	0.020	0.881	-0.059	0.653
DBP (mmHg)	0.012	0.928	0.157	0.232
MAP (mmHg)	0.067	0.608	0.241	0.064
PP (mmHg)	-0.084	0.050	-0.093	0.479

A weak Positive Significant Correlation with Nesfatinin Non-Obese SBP ($r = 0.075$; $p \geq 0.05$)

And A Negative Correlation with Pulse Pressure($p = - 0.084$; $p < 0.05$), p-value < 0.05.

Table 5: Relation of Nesfatin-1 With Biochemical Parameters Among Obese and Non-Obese Participants

Biochemical Parameters	**Group A (n=60)**		**Group B (n=60)**	
	R	***P*-value**	**r**	***P*-value**
FBG (mmol/L)	0.041	0.756	0.110	0.420

Leptin (ng/mL)	-0.102	0.437	-0.209	0.109
TNF (ng/mL)	-0.070	0.592	0.091	0.491

Significant *P*-.05

Table 6: Correlation of Serum Nesfatin-1 Level with Cardiometabolic Risk Factors Among the Study Participants

Indices of Blood Pressure	**Participants (n=120)**	
	R	***P*-value**
Body Mass Index (kg/m^2)	-0.347**	0.000
Fasting Blood Glucose (mmol/L)	-0.131	0.152
Hip circumference (cm)	-0.132	0.151
Waist circumference (cm)	-0.300*	0.001
Waist/Hip Circumference	0.024	0.001

Neck circumference (cm)	-0.326**	<0.001
Systolic Blood Pressure (mmHg)	-0.083	0.369
Pulse Pressure (mmHg)	-0.173	0.059
Diastolic Blood Pressure (mmHg)	0.005	0.953
Mean Arterial Pressure (mmHg)	0.043	0.641
Tumour Necrosis Factor-α(ng/mL)	0.473□□	0.001
Leptin (ng/mL)	0.431□□	0.054
Triglyceride (mmol/L)	0.030	0.001
Total Cholesterol(mmol/L)	0.554	0.259
High Density Lipoprotein (mmol/L)	0.429	0.329
Low Density Lipoprotein (mmol/L)	0.652	0.002

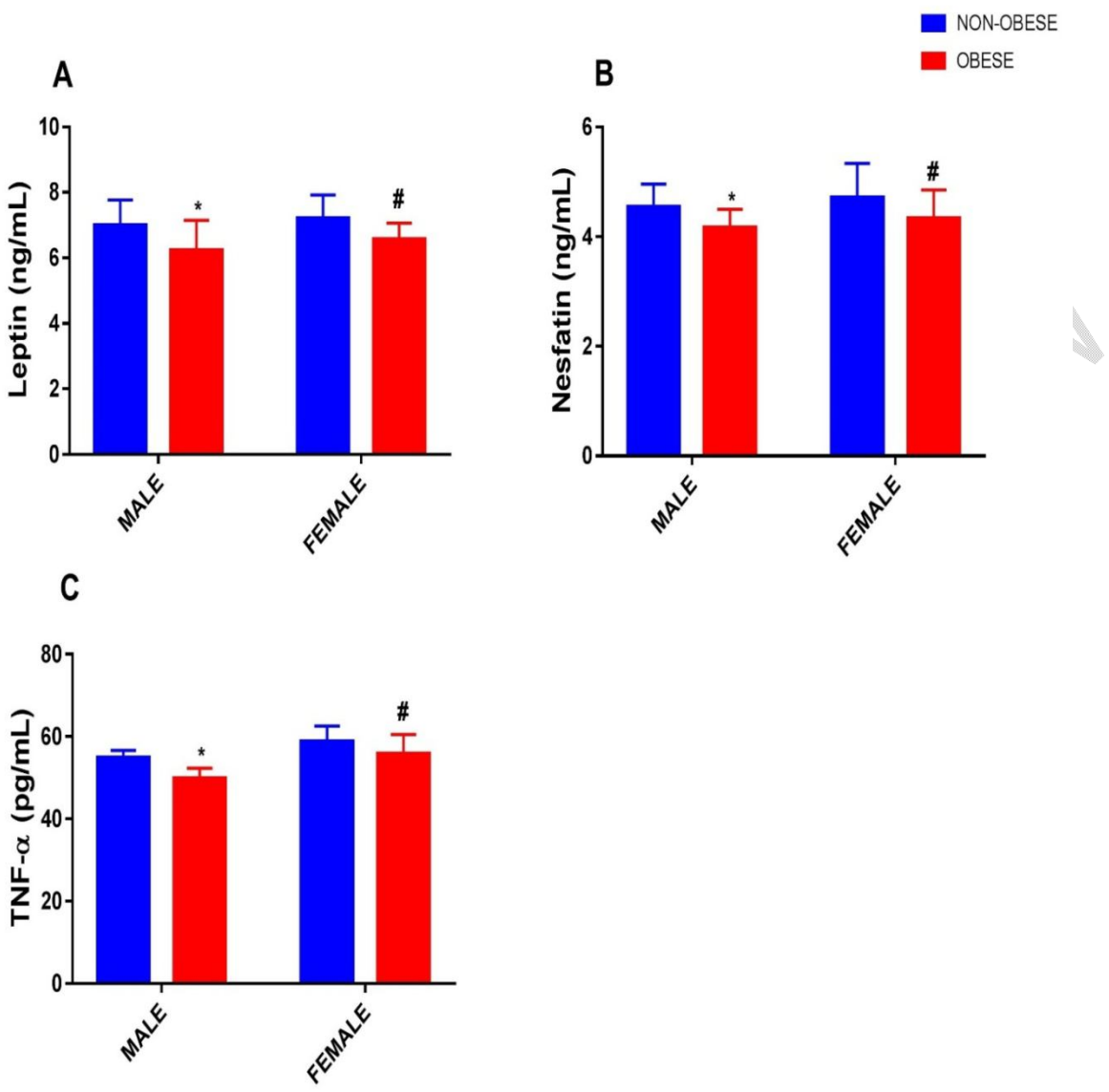

Figure 1: Comparison of (A) Leptin, (B) Nesfatin, and (C) TNF-α between non-obese and obese study participants. $^{*}P < 0.05$ against obese male and $^{\#}P < 0.05$ against obese female. Where TNF-α is tumor necrotic factor alpha. This implies that serum leptin, nesfatin and TNF-α decrease in obese category than non-obese category of this study.

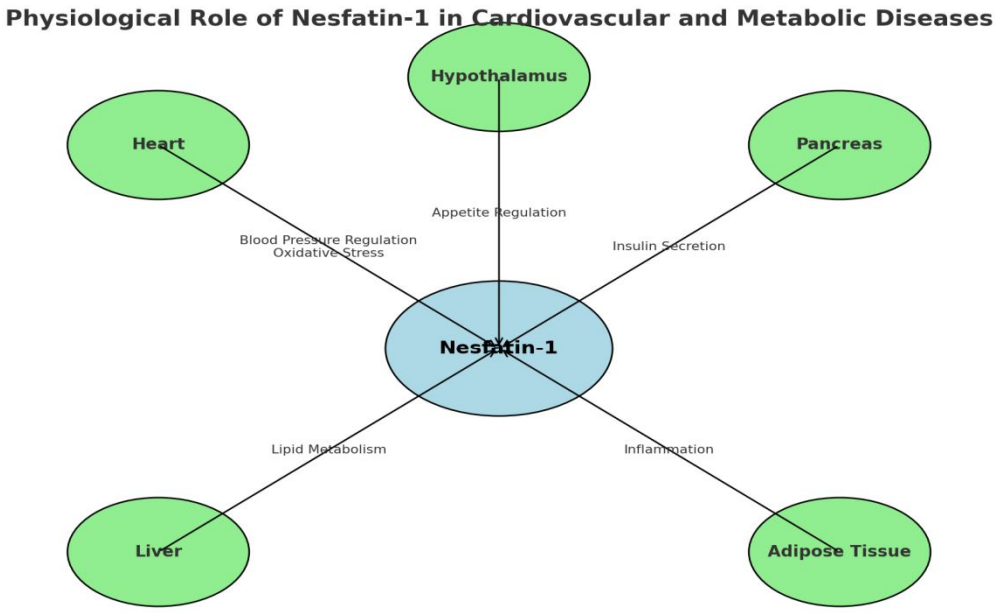

Figure 2: Schematic illustration of the possible way by which nesfatin-1 exerts its physiological role in the development of cardiovascular and metabolic diseases. Culled from goggle image.

3.2 Discussion

The purpose of this study was to compare the relationship between serum nesfatin-1 level to cardiovascular parameters and metabolic risk factors in obese and non-obese adults. Obesity was shown to be associated with many diseases such as cardiovascular diseases, T2 DM, sleep apnea, hypertension and cancer or can even trigger these diseases, for this reasons it is essential sto develop new treatment strategies to reduce its prevalence. nesfatin-1 is one of the emerging cardiovascular risk factors (20). Currently, it is being considered as a potential new anti-obesity treatment (21).

The findings of this study revealed that there was a significant decrease in serum nesfatin-1 level when compared with the parameters of obesity such as body mass index (r = -0.067; p = 0.600), waist circumference (r = - 082; p = 0.532) and neck circumference (r =- 0.173; p = -0.186) which depicted a strong negative but non-significance correlation with serum nesfatin-1 among the obese participants while the hip circumference shows a positive correlation (r = 0.112, p = 0.392) in obese and (r = 0.151; P 0.251) when compared with non-obese participants. This was also the pattern of earlier reported studies among obese children (22). The same pattern was also reported by (23-24) who found a negative correlation between serum Nesfatin-1 levels and Body Mass Index in healthy individuals. These findings may have confirmed the role of Nesfatin-1as an anorexigenic peptide which had been linked to appetite regulation, weight loss, and/or malnutrition, it helps the body to regulate and balance food intake and energy expenditure through the regulatory center for energy balance in the hypothalamus (25).

This is one of the few studies that have been conducted to assess the association between serum nesfatin-1 levels and cardio-metabolic risk factors among obese and non-obese adults, according to a review of published publications. Previous research found a negative correlation between serum nesfatin-1 levels and BMI (26). According to this data, adults with obesity who have low nesfatin-1 levels may have uncontrollably high food intake. nesfatin-1 may play a role in energy balance, according to animal research. According to Oh *et al*., obesity and overweight could result from a lack of nesfatin-1 function in vivo (11).

In contrast, some research in the adult population discovered a link between Body Mass Index and serum Nesfatin-1 levels in children who are obese. Also some study in the adult population discovered a link between BMI and serum nesfatin-1 levels (27-28). In children who are obese, Anwar *et al*. discovered a positive relationship between serum Nesfatin-1 and BMI SDS (29).These inconsistencies could be caused by variations in evaluation techniques (such as sandwich-type ELISA, which only recognizes nesfatin-1, vs ELISA for NUCB2 and Nesfatin-1 experimental conditions, commercial kits, and populations might contribute to these discrepancies. Also when Nesfatin-1 was chronically infused into the third ventricle of rats, it consistently decreased body weight growth. Meanwhile, persistent intra-cerebroventricular treatment of antibodies directed against the gene producing nesfatin/NUCB2 causes rats to gain weight (11). Nesfatin-1 may affect hunger through a central mechanism since it can cross the blood-brain barrier through an unsaturable mechanism (30-31). According to a study, nesfatin-1 decreases food intake in all mice, including obese ones with leptin gene knockdowns. This discovery demonstrates Nesfatin-1's effectiveness in suppressing appetite since it operates outside of the leptin pathway (11). These data indicated that nesfatin-1 plays a role in body weight regulation as well as the physiological control of feeding behavior in rats. (32).

Contrary to the conclusions of this study, it was claimed that there was no significant relationship between body mass index and Nesfatin-1.The lifestyle of the subjects and their race may be the reason why this study contradicts prior investigations in which low levels of nesfatin-1 were found in obese individuals and those of the non-obese

In this study of obese participants, Total Cholesterol (TC), Very Low Density Lipoprotein (VLDL) and High Density Lipoprotein (HDL) revealed a very strong positive and significant correlation with serum Nesfatin-1, except for Low Density Lipoprotein (LDL) and Triglyceride (TG) which shows a strong negative association in obese participants showing that this group is more susceptible to cardiovascular illnesses, (33)

Previous research has demonstrated the role of nesfatin-1 hormone in regulating lipid metabolism and food intake, inhibiting fat build up and decomposition, and generally preventing the onset of lipid-related diseases such as obesity and metabolic syndrome.

The TG/HDL-C (surrogate marker of cardio-metabolic dyslipidaemia: small-dense LDL) and TC/HDL-C indexes have been suggested as potential markers to determine atherogenic risk (34-35). Yin, *et al*. demonstrated that nesfatin-1 regulates peripheral lipid accumulation and hepatic lipid metabolism in mice in addition to its involvement in insulin and glucose metabolism (36) and Tekin, *et al*. associated the metabolic syndrome's elements and nesfatin-1 to them (37). Animal studies reported that Nesfatin-1 may regulate lipid metabolism. Nesfatin-1 stimulates fatty-acid oxidation by activating AMP-activated protein kinase in diabetic rats, chronic subcutaneous infusion of nesfatin-1 reduced plasma cholesterol and triglyceride and elevate HDL-c levels in other animal model (38).

With regard to triglyceride concentrations, this study revealed significant differences between the two study groups, whereas HDL-C only revealed significant differences between the non-obese group. In the obese group, there was a positive correlation between Nesfatin-1 and triglycerides as well as the TG/HDL-C and TC/HDL-C indexes, which may indicate a role for Nesfatin-1 as a cardiovascular risk factor.

These results differ from what Abaci *et al*. (22) and by Kim *et al*. (39) reported in teenagers who did not find a connection between Nesfatin-1 and triglycerides. Since this study group consisted of adults and the other groups included children as young as 5, the discrepancies in the outcomes can be attributed to age disparities.

This study showed a strong positive correlation between Nesfatin-1 and Mean arterial pressure and pulse pressure among the obese subjects. Additionally, there was a strong positive correlation between pulse pressure and mean arterial pressure. Other factors did not significantly correlate. This may be due to the distribution of Nesfatin-1 mRNA protein in the central nervous system, where the neurons that produce vasopressin, corticotropin releasing hormone, POMC, Oxytocin, melanin concentrating hormone, and many other substances are also located. These results suggests that Nesfatin-1 may also play a role in cardiovascular control and other processes (40). These findings imply that Nesfatin-1 may also be involved in the regulation of the heart and other systems (41). According to prior research, administering Nesfatin-1 intravenously increased blood pressure and interfered with the smooth muscle cells' ability to relax after being exposed to sodium nitroprusside (42).

This study's findings demonstrate a positive and significant relationship between serum Nesfatin-1 and serum TNF-α in obese participants demonstrating that TNF-α levels are significantly correlated in these people. TNF-α is substantially linked with serum Nesfatin-1

and leptin. This is in keeping with their findings (35). However, there was no discernible relationship between the TNF-α and the anorexigenic hormones in the non-obese participants. This may be due to the link between adipose tissue and inflammation that is chronic and releases cytokines such as TNF-α. This pro-inflammatory condition can lead to disorders like metabolic syndrome, which is an accumulation of cardio-metabolic risk factors including obesity. Studies have shown that obesity causes an increase in pro-inflammatory cytokines and macrophage infiltration of the expanded adipose tissue (43). Disorders like metabolic syndrome, which is an accumulation of cardio-metabolic risk factors including obesity, can be caused by this pro-inflammatory state. Increased levels of pro-inflammatory cytokines and macrophage infiltration of the enlarged adipose tissue are brought on by obesity (44-45). TNF level in the plasma has been discovered to be greater in obese people, proving a link between raised level of TNF and obesity, according to studies (46). TNF-α levels were shown to be high in the adipose tissue of obese mice in a study, demonstrating that there is an increase in cytokine release in settings where obesity is present. TNF is frequently elevated in pathophysiological conditions in adipose tissues (47).

The outcome of this investigation also revealed that none of the metabolic hormones and TNF-α were significantly correlated in the subjects. This result differs with one by Çatll*et al*; (2015), which found a link between the inflammatory markers and metabolic hormones (Nesfatin-1 and Leptin).

4.0 CONCLUSION AND RECOMMENDATION

4.1 Conclusion

This study showed that Serum level of Nesfatin-1 was negatively correlated with cardiometabolic risk factors such as body mass index, neck circumference and waist circumference with a strong positive correlation with mean arterial pressure and pulse pressure among the obese subjects. Additionally, there was a positive correlation between Nesfatin-1 and triglycerides as well as the TG/HDL-C and TC/HDL-C indexes, which may indicate a role for Nesfatin-1 as a cardiovascular risk factor. These finding suggests that Nesfatin-1 could play a role in the development of cardiovascular and metabolic diseases and hence can be regarded as a potential biomarker for cardiovascular and metabolic risk especially in the obese individual.

4.2 RECOMMENDATION

This study could pave ways for further exploration into the clinical significance of Nesfatin-1 in the treatment of obesity and also as a potential biomarker of cardiometabolic disorders.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

We declared that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT etc) and text-to-image generators have been used during writing or editing of manuscript

CONSENT

Participants consents were collected and preserved

ETHICAL APPROVAL

Ethical clearance was obtained from Ethics and Research Committee of the Ekiti State University Teaching Hospital, Ado-Ekiti.

Disclaimer (Artificial intelligence)

Option 1:

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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