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Levels Of Nesfatin-1 At Various Glycemic Status In Type 2 Diabetes Mellitus

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Abstract

Introduction: Nesfatin-1(NES-1) is an 82-amino acid-length anorexigenic polypeptide linked to the regulation of glucose metabolism. Type 2 Diabetes is associated with alterations in glucose metabolism. Some studies revealed high levels of nesfatin-1 in newly diagnosed patients of type 2 diabetes, but others reported lower levels in diabetic patients. Due to the contradictory results of NES-1 in the literature and no studies being carried out in Manipur and the North-Eastern states of India, it was planned to study the serum NES-1 level among patients with Type 2 diabetes mellitus at different glycemic statuses.

Methodology: This cross-sectional study was conducted in the Department of Biochemistry, in collaboration with the Medicine department and the Multidisciplinary Research Unit, RIMS, Imphal, which included 40 newly diagnosed individuals, 40 diagnosed individuals on treatment, and 40 normal healthy individuals. The serum nesfatin-1 was estimated with a Sandwich ELISA kit.

Results: Mean age and sex showed no significant differences among the cases and controls. Mean serum Nesfatin-1 levels were significantly elevated in newly diagnosed cases (1.03 ± 0.60 ng/mL) compared with diagnosed cases (0.09 ± 0.05 ng/mL) and controls (0.62 ± 0.28 ng/mL). In the study, diagnosed cases exhibited significantly lower levels of Nesfatin -1 than controls**.** Diagnosed cases demonstrated higher levels of insulin resistance, HbA1c, and fasting blood glucose compared to newly diagnosed and controls, indicating greater metabolic disturbance.

Conclusion: The altered levels and correlations observed in the present study further suggest that nesfatin-1 may serve as a potential biomarker for metabolic dysregulation and insulin resistance. However, variations in nesfatin-1 levels across different studies indicate that its role may depend on the progression of the disease, treatment status, and metabolic environment.

Keywords: biomarker, Diabetes Mellitus, insulin resistance, Nesfatin-1, polypeptide

Introduction

Nesfatin-1 (NES-1) is an 82-amino acid anorexigenic peptide derived from nucleobindin-2 (NUCB2) that is involved in the regulation of glucose homeostasis. Experimental studies have demonstrated that nesfatin-1 improves insulin sensitivity and reduces insulin resistance. Furthermore, the co-localization of nesfatin-1 with insulin in pancreatic β-cells suggests a role in insulin secretion. Insulin resistance was assessed using the homeostatic model assessment of insulin resistance (HOMA-IR).[1-2]

Latest clinical observations proposed NES-1 to play a possible role in diabetic hyperphagia.[3] It was discovered by Oh-I and colleagues in 2006 ***having*** an approximate weight of 9.8 kilo Dalton and a half-life of 23.5 minutes. It has three parts; the middle part is described as an active part of nesfatin-1 having the key role for the anorexic effect of nesfatin-1.[4]

The hypothalamus secretes the polypeptide and is responsible for the regulation of feeding behaviour which decreases during fasting and is involved in the regulation of food intake suggesting its involvement in the regulation of energy homeostasis.[5] NES-1 can inhibit food intake via melatonin system activation, independent of the leptin pathway which explains the mechanism of inhibition of food intake with the direct inhibition of an orexigenic substance.[6] A recent study suggests that NES-1 levels are increased by dehydration and found it positively correlated with plasma sodium concentration and plasma osmolality.[7]

Molecular and animal studies suggested its involvement in enhancement of insulin sensitivity by promoting peripheral glucose uptake and decreasing gluconeogenesis via different mechanistic pathways.[8] The major function of NES-1 is inhibition of food intake in time-dependent, dose-dependent, and insulin-dependent manner.[9] NES-1 is stable in blood for 20 minutes after intravenous injection and can pass through the blood brain barrier in both directions.[10] It has been found that its expression level is 20-fold higher in the gastric oxyntic mucosa than in the brain.[11]

Diabetes Mellitus (DM) is a condition characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism caused by defects in insulin secretion, insulin action, or both. The characteristic symptoms include thirst, polyuria, blurring of vision, weight loss, and complications like diabetic ketoacidosis, retinopathy, neuropathy, nephropathy, coronary artery disease and cerebrovascular diseases.[12] Non-vascular complications include infections, cataract, uropathy and sexual dysfunction.[13]

The prevalence of Type 2 Diabetes Mellitus has been increasing steadily all over the world. In 2024, according to IDF- 106.9 million people are from the South East-Asia and 89.8 million people belong to India. The prevalence of diabetes in adults in India is 10.5%.[14] In the state of Manipur, the prevalence of Diabetes is 5.1% as of 2017 in the ICMR-INDIAB population-based cross-sectional study done by Anjan RM et al.[15]

Prevention of diabetes is through lifestyle modification. However, no cure is available despite new insights and drugs. Management should be tailored to improve the quality of life of individuals with T2DM.[16] Type 2 Diabetes is associated with alteration in glucose metabolism, insulin resistance, abnormality in fasting serum glucose (FSG), and also impaired glucose tolerance.[17] Studies evaluating the relationship of circulating nesfatin-1 in patients of type 2 diabetes have produced conflicting results. Some studies revealed high levels of nesfatin-1 in newly diagnosed patients of type 2 diabetes[18] but others reported lower nesfatin-1 levels in diabetic patients.

According to the IDF criteria

Test Criteria	Diabetes Mellitus
1. Fasting plasma glucose	≥7 mmol/l or ≥126 mg/dl
2. 2 hrs plasma glucose after OGTT*	≥ 11.1 mmol/l or ≥200 mg/dl
3. Random plasma glucose	≥11.1 mmol/l or ≥ 200mg/dl
4. Glycosylated haemoglobin	≥ 6.5%

*OGTT= Oral glucose tolerance test

It is important to understand whether nesfatin-1 can be used as a supportive tool in the diagnosis and treatment of glucose metabolic impairments. Many studies have explored the role of nesfatin-1 in insulin sensitivity and insulin resistance in T2DM among various population across the world. However, no studies have

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been carried out in Manipur and North-Eastern states of India. Due to the contradictory results of nesfatin-1 in the literature, it was planned to study the serum nesfatin-1 level among patients with Type 2 diabetes mellitus.

Study Objectives: To estimate and compare the level of Nesfatin-1 in normal healthy individuals, newly diagnosed (IDF criteria) cases without treatment and diagnosed (more than one year under treatment) cases of type 2 Diabetes Mellitus with treatment.

Materials And Methods:

Study Design: Cross-sectional study

Study setting: The study was carried out in the department of Biochemistry in collaboration with the Department of Medicine and the Multidisciplinary Research Unit, Regional Institute of Medical Sciences, Imphal, a tertiary care hospital in Manipur, that receives about 2000 cases of T2DM annually.

Duration of Study: 24 months (01/02/2024-01/01/2026)

Study Population: The sample size was calculated by taking values of S.D = 1 of high-density lipoprotein in patients with T2DM and d = 0.196 from the study conducted by Algul S et al[20], by applying the formula $n = \frac{(Z)^2 \text{ x } (S.D)2}{(d)^2}$. Forty participants were taken in each group, consisting of 40 newly diagnosed (IDF criteria) cases of Type 2 Diabetes Mellitus who are not on treatment, 40 diagnosed (more than one year) cases of diabetes mellitus on treatment were recruited from the Endocrinology/Medicine OPD and wards in consultation with the Endocrinologist and also in conjunction with patients admitted in the Medicine ward. Another 40 age (Range of ±4 years) and sex matched normal healthy individuals without any physical or mental ailments were recruited from the patient party or OPD attendees as controls. Altogether 120 people were included in the study. Individuals whose age is 18 years and above, who gave written consent to participate in the study voluntarily, were included in the study.

Patients of Type 1 diabetes, chronic kidney disease, anorexia nervosa, coronary artery diseases, lung diseases, adrenal disorders, history of vascular diseases, chronic liver disease, history of recent trauma or surgery, patients on steroid therapy, thyroid dysfunction, cancer patients, epilepsy patients, hormone replacement therapy and pregnant women were excluded from the study. Approval from the institutional Research Ethics Board, RIMS vide REB no. A/REB/Prop(FP)214/141/20/2023.

Data collection

Detailed history regarding the duration of disease, age of onset of disease, associated symptoms of complications were taken, and a written informed consent was taken from all the subjects. Standard protocols were used to measure body weight and height with appropriate validation and quality control procedures. Body mass index (BMI) was calculated as weight (kg) divided by the square of the height (m^2). After acquiring informed consent, and a minimum of 8-hour fasting, 6ml of venous blood sample was drawn from the individuals included in the study. About 2 ml of blood was collected in a fluoride vial for the estimation of fasting blood glucose, and 2 ml of blood was collected in an EDTA vial for the estimation of HbA1c. The remaining 2ml of blood was collected in a plain vial and centrifuged at 3000 rpm for 10-15 minutes, and serum was separated. The fasting blood glucose of the subjects was estimated immediately. Two ml serum was aliquoted and preserved at -20°C for the insulin and Nesfatin-1 assay. Serum NES-1 and insulin were estimated by ELISA using Human NES1(Nesfatin 1) and Human INS (Insulin) ELISA Kit. Estimation of HbA1c was done with Human HbA1c ELISA kit. The Beckman Coulter DxC AU 700 autoanalyzer was used for the quantitative determination of the biochemical parameters. Insulin resistance was calculated by using the equation:

HOMA-IR = [Glucose (mg/dl) × fasting insulin (µU/ml)]/405

Statistical analysis: The data were analyzed using IBM SPSS version 26.0 for Windows. Descriptive statistics like mean, standard deviation (SD) and proportion were used to summarize the findings. To compare the means of serum nesfatin-1 levels and other parameters between newly diagnosed, diagnosed diabetics on treatment and controls, ANOVA test was used.

Results

The mean age and height of the participants were found not to differ significantly among the three groups ($p > 0.05$). However, significant differences

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were observed in weight, BMI, systolic blood pressure, diastolic blood pressure, and waist circumference among the groups (p value < 0.05). Table 1 shows a comparison of the mean (±SD) levels of metabolic parameters among controls, newly diagnosed and diagnosed cases using one-way ANOVA test.

Newly diagnosed and diagnosed cases demonstrated higher body weight, BMI, waist circumference, and blood pressure levels compared to controls, suggesting the presence of metabolic risk factors among the cases.

The distribution of participants according to sex among the cases is shown in Table 2. Among the newly diagnosed cases, females (27) outnumbered males (13). In contrast, among the diagnosed cases, males (23) were more than females (17). However, the difference in sex distribution between the groups was not statistically significant ($\chi^2 = 5.614$, $p > 0.05$).

Table 3 presents the comparison of biochemical parameters among controls, newly diagnosed cases, and diagnosed cases. The mean serum Nesfatin-1 levels were significantly higher in newly diagnosed cases compared to diagnosed cases, with Nesfatin-1 levels of 1.03 ± 0.60 ng/ml, 0.09 ± 0.05 ng/ml in diagnosed cases, and 0.62 ± 0.28 ng/ml in controls.

Similarly, fasting insulin, fasting blood glucose (FBG), HOMA-IR, and glycated haemoglobin (HbA1c) levels showed statistically significant differences among the groups ($p < 0.05$). The diagnosed cases demonstrated the highest levels of insulin resistance markers, including insulin, FBG, and HOMA-IR, indicating greater metabolic disturbance as the disease progresses.

Fig. 1 shows a comparison of the number of participants based on their daily physical exercise activity. No significant difference was observed between the control and the newly diagnosed groups. In the diagnosed cases, the number of participants with regular physical exercise was only 15 (37%), whereas participants with no regular physical exercise were 25 (63%), which was statistically significant ($p < 0.05$), indicating a higher prevalence of sedentary lifestyle among diagnosed cases.

Table 4 shows a comparison of liver function tests among the study groups using one way ANOVA test. The mean (±SD) levels of Total bilirubin, Direct bilirubin, Globulin, Aspartate aminotransferase (AST), Alanine transaminase (ALT), Alkaline phosphatase (ALP), and Gamma-glutamyl transferase (GGT) were not statistically significant ($p > 0.05$), whereas total protein and albumin levels were significantly lower in newly diagnosed and diagnosed cases compared to controls ($p < 0.05$).

Table 5 shows the comparison of lipid profile and kidney function parameters among the groups.

The mean levels of urea and creatinine were significantly higher in newly diagnosed and diagnosed cases compared to controls ($p < 0.05$). Similarly, triglycerides (TG) and LDL levels were significantly elevated, while HDL levels were significantly reduced in the cases compared to controls ($p < 0.05$). However, total cholesterol did not show a statistically significant difference among the groups. These findings suggest the presence of dyslipidemia and possible early renal involvement in the cases.

Discussion

Diabetes mellitus is a multifactorial metabolic disorder characterized by persistent hyperglycemia, particularly elevated fasting blood glucose levels, resulting from a combination of insulin resistance, genetic predisposition, and environmental and dietary influences. The present study aimed to evaluate and compare the levels and interrelationships of various clinical, metabolic, and biochemical parameters among three groups: newly diagnosed patients with type 2 diabetes mellitus who have not yet initiated treatment, patients with type 2 diabetes mellitus who have been on treatment for more than one year, and healthy control subjects.

In the current study, serum Nesfatin-1 level was found to be significantly lower in diagnosed cases on treatment and controls compared with the newly diagnosed cases. This finding is in accordance with studies by Algul S et al[20], Zhai T et al[21], Mohammad NJ et al[22], and Samani SM et al[23], which reported increased levels of nesfatin-1 in newly diagnosed patients. Likewise, Matta RA et al[24], Huang K et al[25] have shown decreased levels of nesfatin-1 in patients diagnosed with type 2 Diabetes mellitus compared to newly diagnosed, drug-naive patients and controls. Nesfatin-1 has been shown to alter glucose metabolism by enhancing insulin sensitivity and decreasing insulin resistance. Lower nesfatin-1 levels

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could explain the considerably higher insulin resistance in the diagnosed patients[11].

Zhang Z et al[18], showed that newly diagnosed and IGT patients had higher NS-1 levels compared to the controls, in contrast to the study by AbuHanoud A et al[26], where higher nesfatin-1 levels were found in pre diabetic and diabetic patients when compared to non-diabetics.

The age and height distribution did not differ significantly among the groups, indicating that the study populations were comparable with respect to age and height. However, body weight, BMI, waist circumference, and blood pressure were significantly higher in newly diagnosed and diagnosed cases compared to controls. In a study by Tsimihodimos V et al.[27] the incidence of hypertension increases significantly in the presence of diabetes mellitus, and weight gain may be one factor that contributes to the development of both hypertension and diabetes mellitus.

The newly diagnosed patients had significantly higher weights than the diagnosed cases. Higher systolic blood pressure and waist circumference in the diagnosed cases were found. These findings indicate the presence of central obesity and metabolic risk factors in the disease groups. Central obesity, reflected by increased waist circumference and BMI, has been strongly linked with metabolic abnormalities and type 2 diabetes. Adipose tissue dysfunction leads to altered secretion of metabolic hormones and peptides, including adiponectin, resistin, and nesfatin-1, which influence insulin sensitivity and glucose homeostasis.[28]

Experimental studies have demonstrated that nesfatin-1 can enhance glucose-stimulated insulin secretion and reduce blood glucose levels, suggesting that it may function as an important regulator of pancreatic β-cell activity. These mechanisms support the hypothesis that alterations in nesfatin-1 levels may contribute to the development of insulin resistance and impaired glucose metabolism.[29]

The current study also observed significantly elevated fasting blood glucose, fasting insulin, HbA1c, and HOMA-IR values in newly diagnosed and diagnosed cases compared to controls, indicating the presence of insulin resistance and poor glycaemic control. Insulin resistance is a central feature in the pathogenesis of type 2 diabetes, where peripheral tissues fail to respond adequately to insulin, resulting in hyperglycemia and compensatory hyperinsulinemia. This is in line with the study by Mierzyński R et al., where decreased nesfatin-1 levels have been observed in individuals with diabetes and were negatively correlated with metabolic parameters such as fasting glucose, HbA1c, and HOMA-IR. Similarly, studies in gestational diabetes have demonstrated lower nesfatin-1 concentrations accompanied by increased insulin resistance and glucose levels.[30]

Mohammad NJ et al[22] reported significantly lower serum nesfatin-1 levels in patients with type 2 diabetes mellitus and demonstrated negative correlations with BMI, fasting blood glucose, fasting insulin, HbA1c, and HOMA-IR, suggesting that decreased nesfatin-1 is associated with worsening insulin resistance and glycemic control. Similarly, Samani SM et al[23] observed reduced NES-1 concentrations in newly diagnosed diabetic and obese subjects compared with normal-weight controls, although no significant association with age, sex, or BMI was noted. Their findings suggested that obesity and T2DM may share a common pathophysiological mechanism involving impaired nesfatin-1-mediated regulation of energy and glucose homeostasis.

Zhang Z et al[18], demonstrated that plasma nesfatin-1 levels were elevated in newly diagnosed T2DM and positively correlated with fasting plasma glucose, HbA1c, and HOMA-IR, suggesting a compensatory response to hyperglycemia and insulin resistance. Similarly, the meta-analysis by Zhai T et al[21], confirmed higher nesfatin-1 concentrations in newly diagnosed patients but lower levels in treated or long-standing T2DM, indicating that circulating nesfatin-1 may vary with disease duration and therapeutic status.

The conflicting results among studies suggest that circulating nesfatin-1 levels may vary according to disease stage and metabolic status, exhibiting an initial compensatory increase in newly diagnosed T2DM, followed by a decline with chronic disease progression and worsening insulin resistance.

Limitations

The present study has several limitations. The relatively small sample size and single-center setting may limit the generalizability of the findings. Moreover, the absence of longitudinal follow-up

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prevented assessment of temporal changes in nesfatin-1 with disease progression and treatment. Potential confounding factors, including differences in treatment regimens, lifestyle factors, and inflammatory markers, were not evaluated. Therefore, larger prospective multicentric studies are required to further elucidate the role of nesfatin-1 in type 2 diabetes mellitus.

Conclusion

The findings of the present study suggest that nesfatin-1 plays a significant role in glucose homeostasis, insulin secretion, and energy balance.

The altered levels of NES-1 observed in the present study further suggest that nesfatin-1 may serve as a potential biomarker for metabolic dysregulation and insulin resistance. However, variations in nesfatin-1 levels across different studies indicate that its role may depend on disease stage, treatment status, and metabolic environment.

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Table 1: Distribution of controls, diagnosed cases and newly diagnosed cases by clinical and metabolic parameters

Parameters	Control (n=40) Mean ± SD	Newly diagnosed cases(n=40) Mean ± SD	Diagnosed cases(n=40) Mean ± SD	p-value
Age (years)	51.20 ± 13.58	54.08 ± 16.46	56.23 ± 11.06	0.271
Height (cm)	163.40±9.34	159.85±8.96	161.98±7.82	0.192
Weight (kg)	60.00±8.51	69.23±9.70	67.70±9.42	0.000
BMI (kg/m^2)	22.35±1.54	26.60±3.78	26.38±2.31	0.000
Systolic BP (mmHg)	116.40±8.52	129.40±12.99	131.50±11.16	0.000
Diastolic BP (mmHg)	76.30±8.19	85.45±8.22	86.45±7.32	0.000
Waist circumference (cm)	79.50±3.66	97.00±10.20	88.98±7.31	0.000

Table 2: Sex-wise distribution of participants among cases:

	Number of newly diagnosed cases	Number of diagnosed cases	Chi-squared	p value
Male	13	23	5.614	0.06
Female	27	17		

Table 3: Distribution of biochemical parameters among controls, diagnosed cases and newly diagnosed cases:

Parameters	Control (n=40) Mean ± SD	Newly diagnosed cases(n=40) Mean ± SD	Diagnosed cases (n=40) Mean ± SD	p value
Nesfatin-1 (ng/ml)	0.62 ± 0.28	1.03 ± 0.60	0.09 ± 0.05	0.000
Insulin (mIU/L)	4.41 ± 1.08	4.86 ± 2.73	10.34 ± 2.51	0.000
FBG (mg%)	90.88 ± 9.68	170.63 ± 37.39	207.85 ± 37.16	0.000
HOMA-IR	1.02 ± 0.43	2.09 ± 1.49	5.38 ± 2.07	0.000
HbA1c (%)	4.42 ± 1.11	7.77 ± 1.41	6.39 ± 1.76	0.000

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Fig 1: Distribution of participants by physical activity among controls, newly diagnosed and diagnosed cases

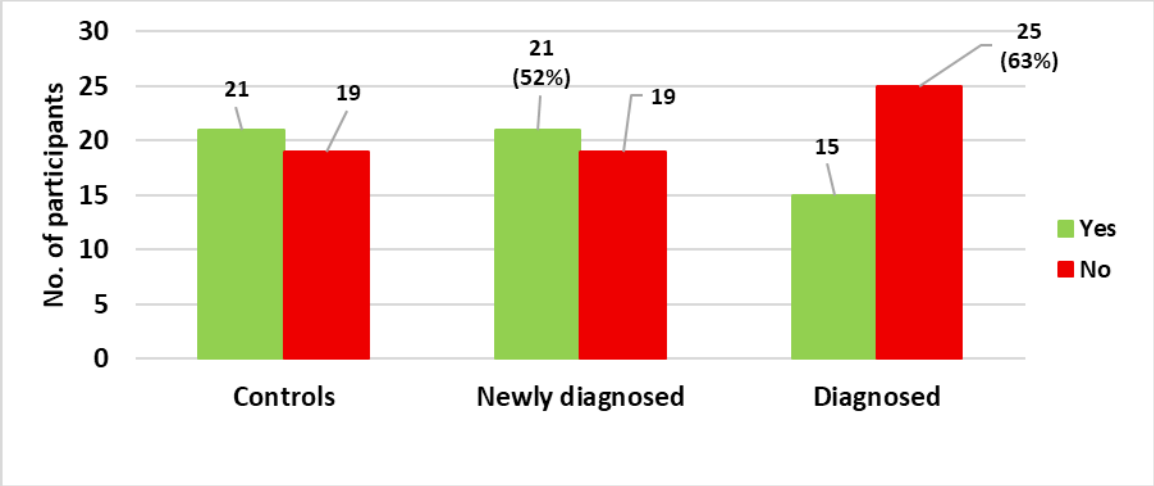

Table 4: Distribution of participants by liver function test among controls, diagnosed cases and newly diagnosed cases:

Parameters	Control (n=40) Mean ± SD	Newly diagnosed cases(n=40) Mean ± SD	Diagnosed cases (n=40) Mean ± SD	p value
Total bilirubin (mg%)	0.57 ± 0.18	0.64 ± 0.23	0.63 ± 0.17	0.204
Direct bilirubin (mg%)	0.16 ± 0.74	0.22 ± 0.16	0.20 ± 0.09	0.077
Total protein (gm%)	7.61 ± 0.39	7.05 ± 0.44	6.68 ± 0.60	0.000
Albumin (gm%)	4.13 ± 0.39	3.59 ± 0.37	3.29 ± 0.49	0.000
Globulin (gm%)	3.52 ± 0.39	3.46 ± 0.31	3.39 ± 0.51	0.364
AST (IU)	30.95± 12.47	34.73 ± 22.38	26.88 ± 13.64	0.116
ALT (IU)	28.68 ± 18.13	32.90 ± 18.76	29.40 ± 14.52	0.505
ALP (IU)	80.53± 26.02	87.88 ± 26.99	93.70 ± 20.26	0.06
GGT (IU)	35.40 ± 17.16	38.73± 13.15	31.33 ± 9.19	0.054

Table 5: Distribution of participants by lipid profile and kidney function tests among the controls, newly diagnosed and diagnosed cases:

Parameters	Control (n=40) Mean ± SD	Newly diagnosed cases (n=40) Mean ± SD	Diagnosed cases (n=40) Mean ± SD	p-value
Urea (mg%)	17.95 ± 4.96	23.53 ± 4.95	22.38 ± 5.50	0.000
Creatinine (mg%)	0.64 ± 0.12	0.95 ± 0.31	1.03 ± 0.18	0.000

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Total cholesterol (gm%)	150.90 ± 23.02	164.35 ± 29.02	158.78 ± 21.09	0.053
TG (gm%)	109.23 ± 38.49	149.55 ± 29.00	154.08 ± 47.03	0.000
LDL (gm%)	94 ± 20.17	108.13 ± 29.12	119.48 ± 36.55	0.001
HDL (gm%)	50.95 ± 10.16	44.98 ± 7.79	40.38 ± 8.76	0.000

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