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Association between non-high density lipoprotein-cholesterol fractions and presence of allostatic load among industrial workers

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ABSTRACT

Objectives: Non-high density lipoprotein-cholesterol (non-HDL-C) fraction is the total cholesterol (TC) minus HDL-C. It is not a routinely reported component of lipid profile and is used in lipoprotein lowering therapy and prediction of coronary artery disease, target organ damage and atherosclerosis. Allostatic load (AL) is an imbalance between repetitive chronic exposure to stress and adaptive response. The present study investigates the association between non-HDL-C and its fractions (non-HDL-C/HDL-C, non-HDL-C/TC, non-HDL-C/triglyceride [TG] and non-HDL-C/low-density lipoprotein-cholesterol [LDL-C]) and the presence of AL to determine, which fractions of non-HDL-C predict the diagnostic accuracy and optimal cut points.

Materials and Methods: The study design is cross-sectional and data were collected from 169 male industrial workers. AL was measured using neuroendocrine (cortisol and dehydroepiandrosterone sulphate), cardiovascular (systolic blood pressure, diastolic blood pressure and heart rate), metabolic (TC, TG, HDL-C and LDL-C) and anthropometric (waist-hip ratio and body mass index) factors. The fractions of non-HDL-C/HDL-C, non-HDL-C/TC, non-HDL-C/TG and non-HDL-C/LDL-C were calculated using non-HDL-C, HDL-C, TC, TG and LDL-C values.

Results: About 43.2% and 56.8% of workers had low and high AL, respectively. The non-HDL-C and its fractions such as non-HDL-C/HDL-C, non-HDL-C/TC and non-HDL-C/LDL-C were significantly increased in the high AL group. Stepwise regression analysis was used to examine the association between non-HDL-C fractions and AL. The fractions of non-HDL-C ($\beta = 0.785$, $P = 0.001$), non-HDL-C/TC ($\beta = -0.336$, $P = 0.001$) and non-HDL-C/LDL-C ($\beta = 0.295$, $P = 0.001$) influenced AL by 38.6%. The AUC with 95% CI in the high AL group was as follows: non-HDL-C 0.766 (0.696–0.837, $P = 0.001$); non-HDL-C/HDL-C 0.638 (0.555–0.721, $P = 0.002$); non-HDL-C/TC 0.635 (0.552–0.712, $P = 0.003$) and non-HDL-C/LDL-C 0.520 (0.433–0.607, $P = 0.657$). Non-HDL-C and its fractions were more precisely predicted in the high AL category of workers than in the low AL category. Non-HDL-C predicted the most precisely, followed by non-HDL-C/HDL-C, non-HDL-C/TC, non-HDL-C/LDL-C and non-HDL-C/TG.

Conclusion: According to the present study, non-HDL-C and its fractions such as non-HDL-C/HDL-C, non-HDL-C/TC and non-HDL-C/LDL-C should be considered regular lipid profiles and could be used as biomarkers to reduce the risk of AL.

Keywords: Allostatic load, Non-high density lipoprotein-cholestrol, Non-high density lipoprotein-cholestrol/high-density lipoprotein-cholestrol, Non-high density lipoprotein-cholestrol/low-density lipoprotein-cholestrol, Non-high density lipoprotein-cholestrol/total cholesterol, Non-high density lipoprotein-cholestrol/triglyceride, Industrial workers

INTRODUCTION

The risk of cardiovascular disease (CVD) is determined by lipid stress reactivity.[1] All lipoproteins, such as low-density lipoprotein (LDL), very LDL (VLDL), intermediate lipoprotein and lipoprotein (a), have atherogenic qualities and are included in the non-high density lipoprotein-cholesterol (HDL-C) fraction. It is a simple matter of deducting HDL-C from total cholesterol (TC) and classifying it as bad cholesterol. Non-HDL-C levels have a stronger relationship with Apo-lipoprotein B and predict CVD risk better than LDL-cholesterol (LDL-C). The non-HDL-C fraction is found more stable in coronary artery disease (CAD) assessment as compared to LDL-C.[2] It is also used as a predictor for target organ damage[3] and lipoprotein lowering therapy.[4] The assessment of non-HDL-C is considered a good indicator for CAD,[5] carotid atherosclerosis[6] and CVD mortality.[7] When compared to LDL-C, a recent study found a substantial correlation between non-HDL-C and atherosclerotic disease.[8]

The fraction of non-HDL-C/TC is used to screening of dysbetalipoproteinemia, which is a genetic lipid disorder, caused by a mutation in the apolipoprotein E gene and characterised by cholesterol-enriched lipoproteins in plasma[9] Non-HDL-C/TC marker is also used to measure the severity of coronary artery lesions and cardiovascular outcomes.[10] The fraction of non-HDL-C/triglyceride (TG) concentrations predicts the CVD risk better than LDL-C.[11] The non-HDL-C/HDL-C is used for prediction of metabolic disorders such as metabolic syndrome and insulin resistance,[12] chronic kidney disease of unknown aetiology,[13] dyslipidemia-related CVD risk,[14] peritoneal dialysis mortality[15] and carotid plaques in stroke risk population.[16]

Allostatic load (AL) is an imbalance between repetitive exposure to chronic stress and adaptive response.[17] Allostasis is the process of achieving homeostasis through physiological changes in the hypothalamus, pituitary, adrenal glands and various signal molecules in the body.[18] Workers from industrial settings had higher levels of psychological, physical and occupational stress.[19] In underdeveloped countries, preventing work-related stress is a major challenge.[20] Work-related stress is the second-largest task in occupational safety.[21] Recent research reported that AL has positively associated with ageing,[22] anthropometrics[23] and poorer health outcomes.[24] In animal experiments, the inclusion of TC and TG in AL measurement better predicts the disease risk.[25] The regular or routine lipid profiles such as TC, LDL-C, HDL-C and TC/HDL-C are significantly associated with AL.[26] Obeng-Gyasi *et al*.[27] reported elevated levels of non-HDL-C fraction in high AL subjects. Given the literature review, the association between non-HDL-C and its fractions (non-HDL-C/HDL-C, non-HDL-C/TC, non-HDL-C/TG and non-HDL-C/LDL-C) and the presence of AL, as well as the diagnostic accuracy and optimal cut points of non-HDL-C and its fractions in workers with AL, needs to be explored to find out which fractions of non-HDL-C predict the risk of AL.

MATERIALS AND METHODS

This study adopted a cross-sectional design. We enrolled 169 male industrial workers engaged in the manufacturing processes of Pb-battery and phosphate fertilisers. The enrolled workers were working as operators. The demographic details such as age, height, weight, waist circumference (WC), hip circumference (HC) and personal habits such as type of diet, smoking, alcohol consumption and chewing of tobacco products were collected using a pre-tested questionnaire. The average age of the workers was 35.3 years (range 19–60 years) and their average height and weight were 165.1 cm and 63.6 kg, respectively. Worker's mean WC and HC were found to be 35.6 and 36.0 inches, respectively. The majority of the subjects were married (76.9%) and ate a non-vegetarian diet (83.4%). Smoking and alcohol consumption were reported by 18.9% and 34.3% of workers, respectively. The data were collected during the years 2020–21 in India. The study protocol was approved by the Regional Occupational Health Centre (Southern) Institutional Ethics Council. Informed written consent was obtained before the recruitment of all the participants. The study included subjects who had been willing to participate and did not have a history of high blood pressure, thyroid disease, genetically inherited or metabolic disease.

Anthropometrics

Body mass index (BMI): With a lightweight outfit and no footwear, the subjects height and weight were measured in meters and kilograms, respectively, using a non-extendable metallic measuring tape and a pre-calibrated weighing machine. The BMI was calculated as a measure of a persons weight (in kilograms) by height (in square meters), represented as kilogram/meter^2.

Waist-hip ratio (WHR) was calculated as the ratio of the WC to the HC. The WC measurement was taken mid-way between the ribs and the iliac crest, while the HC measurement was taken as the maximum value measured at the buttocks. The WHR was computed using WC and HC values, as described by Dobbelsteyn *et al*.[28]

Cardiovascular activity

Blood pressure and heart rate (systolic blood pressure, diastolic blood pressure and heart rate) of the subjects were monitored using a HEM-7112 digital monitor after they had rested for 5 min in a sitting position. The average of two blood pressure readings was used, as suggested by Nasothimiou *et al*.[29]

Blood collection: From each subject, 3 ml of fasting whole venous blood were taken and transferred to vacutainer tubes obtained from M/s Labtech disposables (India). Serum was separated by centrifugation at 4°C for 10 min at 4000 RPM. The serum was used to determine the neuroendocrine activity (Cortisol and DHEA-S) and metabolic (TC, TG and HDL-C) parameters.

Neuroendocrine parameters

Cortisol: The level of serum cortisol was determined using an ELISA kit (Catalogue. No. CO368S, Calbiotech, USA) as per the protocol of the manufacturer. The absorbance of samples and standards was measured using the Lisa Scan EM microplate reader, India, at 450 nm. The detection range of protocol was noted as 0–500 ng/mL and the sensitivity is 1.16 ng/mL.

DHEA-S: The level of serum DHEA-S was estimated using the competitive ELISA kit (Catalog. no. DH291S, Calbiotech, USA) as per the protocol of the manufacturer. The absorbance of samples and standards was measured using the Lisa Scan EM microplate reader, India, at 450 nm. The detection range of this procedure is 0–10 μg/mL and the sensitivity is 0.023 μg/mL.

Metabolic parameters

Serum TG level was determined using the Prietest diagnostic kit method developed by Fossati and Prencipe,[30] with Trinder reaction,[31] 1969 (Catalog no. TRIG 05 10; Robonik, India). The recorded absorbance of samples was correlated with TG concentration. The minimum detection limit of this method is 1 mg/dL.

Serum HDL-C levels were quantified by the Lopes-Virella *et al*.[32] method (Catalog no. HDC PPT 02 50; Robonik, India). In this technique, the high-density lipoproteins (HDLs) were separated from chylomicrons, VLDLs and LDLs using phosphotungstic acid and magnesium ions. The separated HDLs were used to measure cholesterol with the Prietest diagnostic kit established on the principle of Trinder reaction, 1969 (Catalogue No. CHO 02 50; Robonik, India). The minimum detection concentration of this approach is 4 mg/dL.

The serum TC levels were determined using the Allain *et al*.[33] technique. In this approach, cholesterol was measured using a Prietest diagnostic kit established on the principle of Trinder reaction[31] (Catalogue No. CHO 02 50; Robonik, India). The end product obtained from this reaction is quinonimine, which is generated through the interactions of 4-aminoantipyrine, phenol and H_2O_2. The absorbance of the sample was recorded using the Robonik Prietest Touch Biochemistry Analyzer, India. The minimum detection concentration of this approach is 4 mg/dL.

LDL-C: Using Friedewald *et al*.[34] equation, the level of LDL-C was estimated using the individual values of TG, cholesterol (TC) and HDL-C.

LDL-C (mg/dL) = (TC) − (HDL-C) − (TG/5).

Assessment of AL

The creation of AL was done using the physiological dysregulation of cardiovascular activity, metabolic, neuroendocrine and anthropometrics. The highest risk quartile method was used to determine the AL. The people count falling in the highest risk value is assigned as 1 and the people count falling in less than the high-risk value is allotted as 0. The AL score was calculated by summing the 11 dichotomous scores of each of the 11 markers. The value of the AL score ranged from 0 to 11 points.

Non-HDL-C and its fractions, such as non-HDL-C/HDL-C, non-HDL-C/TC, non-HDL-C/TG and non-HDL-C/LDL-C, were calculated based on subjects values of non-HDL-C, HDL-C, TC, TG and LDL-C.

Statistical analysis

The data were analysed using the Statistical Package for the Social Sciences version 20. The data were presented as mean ± SD. For the comparison of data between AL categories and non-HDL-C fractions among workers, a Student's *t*-test was utilised. To determine the association between non-HDL-C fractions and the occurrence of AL, a general linear regression model was used. The results of the model are reported as unstandardized coefficients (B), standard error, standardised coefficients (β) and P-value. The diagnostic accuracy and optimal cut points to existence of AL categories were assessed using a receiver operating characteristic curve-area under the curve (ROC-AUC) analysis. The probability value of notable findings is <0.05.

RESULTS

The cutoff values that are used to create an AL among workers are shown in [Table 1]. A total of 11 biomarkers such as neuroendocrine, cardiovascular activity, metabolic and anthropometrics were used to construct the AL. The different type of methods is adopted to create the AL score, which includes quartile distributions, logistic regression, factor analysis, z-scores and grade of membership.[35] In the present study, we constructed the AL score among workers using the quartile distribution method.[36] The AL score was constructed in adolescents in an Indian study[37] using eight parameters with the highest threshold distribution method and the study found that 59% of subjects had no AL (<3.0) and 41% of subjects had AL (>3). We created an AL score from 11 biomarker measurements. The average total score obtained was 2.9 (SD

Table 1: Cutoff values of AL parameters among industrial workers.

AL parameter (*n*=169)	Cutoff values	Absent *n* (%)	Present *n* (%)
Body mass index (kg/m^2)	>25.6	87 (51.5)	82 (48.5)
Waist to hip ratio	>1.0	100 (59.2)	69 (40.8)
Systolic blood pressure (mmHg)	>140.0	94 (55.6)	75 (44.4)
Diastolic blood pressure (mmHg)	>83.0	98 (58.0)	71 (42.0)
Heart rate (Beats/min)	>88.0	128 (76.0)	41 (24.0)
Triglyceride (mg/dL)	>245.8	102 (60.0)	67 (40.0)
Cholesterol (mg/dL)	>176.1	98 (58.0)	71 (42.0)
Low-density lipoprotein-cholesterol (mg/dL)	>107.0	91 (54.0)	78 (46.0)
High-density lipoprotein-cholesterol (mg/dL)	<40.6	73 (43.0)	96 (57.0)
Cortisol (ng/mL)	>113.5	116 (69.0)	53 (31.0)
Dehydroepiandrosterone sulfate (DHAE-S) (μg/mL)	<2.9	107 (63.0)	62 (37.0)

AL: Allostatic load

Table 2: Comparison of non-HDL-c fractions among industrial workers with low and high AL category.

Parameters	Low AL (<2.9) (*n*=73)	High AL (>2.9) (*n*=96)	*P*-value
Non-HDL-C	107±26	140±37*	0.001
Non-HDL-C/HDL-C	3.7±2.0	5.1±3.0*	0.001
Non-HDL-C/TC	0.75±0.1	0.80±0.1*	0.001
Non-HDL-C/TG	0.80±0.4	0.83±0.5	0.757
Non-HDL-C/LDL-C	1.50±0.4	2.00±1.9*	0.039

*P<0.05, AL: Allostatic load, LDL-C: Low-density lipoprotein-cholesterol, HDL-C: High-density lipoprotein-cholesterol, TC: Total cholesterol, TG: Triglyceride

= 1.97), with the highest component score being nine and the lowest being zero. The study participants were divided into two groups based on their total average score: low AL (<2.9) and high AL (>2.9).

The comparison of non-HDL-C and its fractions between low AL and high AL groups are presented in [Table 2]. The non-HDL-C (P = 0.001) and its fractions such as non-HDL-C/HDL-C (P = 0.001), non-HDL-C/TC (P = 0.001) and non-HDL-C/LDL-C (P = 0.039) were significantly increased in the high AL group (>2.9) as compared to low AL group (<2.9); however, there was no significant difference noted in the non-HDL-C/TG fraction (P = 0.757).

[Table 3] displays the results of correlation coefficients (r) between AL and non-HDL-C and its fractions among workers. The association between AL score and non-HDL-C (r = 0.495, P = 0.001) and its fraction such as non-HDL-C/HDL-C (r = 0.314, P = 0.001), non-HDL-C/TC (r = 0.219, P = 0.004) and non-HDL-C/LDL-C (r = 0.240, P = 0.002) found a positive and significant association. Similarly, the association between AL score and non-HDL-C/TG (r = −0.082, P = 0.287) found a negative and insignificant association. The association between non-HDL-C and its fractions such as non-HDL-C/HDL-C (r = 0.761, P = 0.001), non-HDL-C/TC (r=0.753, P = 0.001) and non-HDL-C/TG (r = 0.278, P = 0.001) found a positive and significant association. Similarly, the association between non-HDL-C and non-HDL-C/LDL-C (r = −0.122, P = 0.113) was found to be negative and insignificant.

The frequency distribution of allostatic component scores among workers is presented in [Figure 1]. Seventeen workers had zero scores and two workers had a score of more than nine. The majority of workers had the score of three components.

A stepwise multiple linear regression analysis of variables for non–HDL-C and its fractions and AL among workers are shown in [Table 4]. In this model, the AL score was used as a continuous dependent variable and non-HDL-C and its fractions such as non-HDL-C/HDL-C, non-HDL-C/TC, non-HDL-C/TG and non-HDL-C/LDL-C were used as independent variables (predictors). In model-1, the predictor of non-HDL-C alone has influence of 24.5% on the AL (F = 54.32, P = 0.001). The predictors such as Non-HDL-C and non-HDL-C/LDL-C have a 33.7% influence on the AL in Model-2 (F = 42.18, P = 0.001). The predictors such as non-HDL-C, non-HDL-C/TC and non-HDL-C/LDL-C have a 38.6% influence on the AL in Model-3 (F = 34.56, P = 0.001).

ROC-AUC of non-HDL-C and its fraction among low and high AL categories is presented in [Figures 2 and 3]. The results of the ROC-AUC analysis are shown in [Table 5]. In high AL category, the AUC for non-HDL-C and its fractions as non-HDL-C was 0.766 (95% CI: 0.696–0.837, P = 0.001), non-HDL-C/HDL-C 0.638 (95% CI: 0.555–0.721, P = 0.002), non-HDL-C/TC 0.635 (95% CI: 0.552–0.712, P = 0.003), non-HDL-C/TG 0.480 (95% CI: 0.393–0.567, P = 0.661) and non-HDL-C/LDL-C 0.520 (95% CI: 0.433–0.607, P = 0.657). The AUC and 95% CI of non-HDL-C and its fractions in low AL category workers were found to be a null hypothesis (AUC < 0.5).

Table 3: Pearson correlation coefficient (r) between allostatic load and Non-HDL-C fraction among workers.

Parameters	AL	Non-HDL-C	Non-HDL-C/HDL-C	Non-HDL-C/TC	Non-HDL-C/TG	Non-HDL-C/LDL-C
AL	1.000	-	-	-	-	-
Non-HDL-C	0.495**	1.000	-	-	-	-
Non-HDL-C/HDL-C	0.314**	0.761**	1.000	-	-	-
Non-HDL-C/TC	0.219**	0.753**	0.813**	1.000	-	-
Non-HDL-C/TG	−0.082	0.278**	0.345**	0.317**	1.000	-
Non-HDL-C/LDL-C	0.240**	−0.122	−0.148*	−0.120	−0.0.430**	1.000

**Correlation is significant at the 0.01 levels, *Correlation is significant at the 0.05 levels, AL: Allostatic load, LDL-C: Low-density lipoprotein-cholesterol, HDL-C: High-density lipoprotein-cholesterol, TC: Total cholesterol, TG: Triglyceride

Table 4: Step-wise regression analysis between non-HDL-C fractions and allostatic load among workers.

Model	Predictors	B	SE	β	P	R^2
Model-1	Non-HDL-C	0.026	0.004	0.495	0.001*	0.245
Model-2	Non-HDL-C	0.028	0.003	0.533	0.001*	
	Non-HDL-C/LDL-C	0.404	0.084	0.305	0.001*	0.337
Model-3	Non-HDL-C	0.041	0.005	0.785	0.001*	
	Non-HDL-C/LDL-C	0.392	0.082	0.295	0.001*	0.386
	Non-HDL-C/TC	−6.714	1.852	−0.336	0.001*	

*P<0.05. AL: Allostatic load, LDL-C: Low-density lipoprotein-cholesterol, HDL-C: High-density lipoprotein-cholesterol, TC: Total cholesterol, B: Unstandardized coefficient , SE: Standard error, β: Standardized coefficient, P: Probability, R^2: R Square

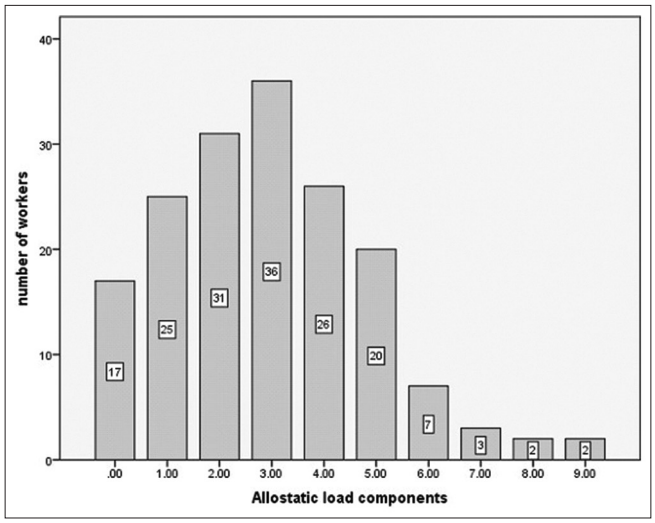

Figure 1: Frequency distribution of allostatic load component score among workers.

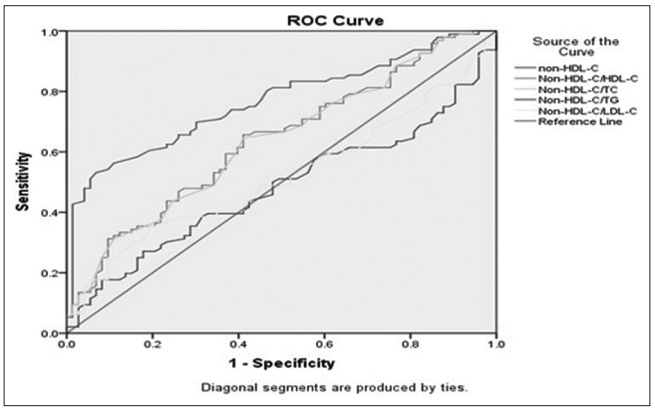

Figure 2: Receiver operating characteristic curves of non-high-density lipoprotein-cholesterol fraction for high allostic load (>2.9).

DISCUSSION

This study investigated the association between non-HDL-C and its fractions and presence of AL in industrial workers. AL is an integrated score of biological and clinimetric data.[24] It is used as an early marker of stress in healthy people.[38] Jung *et al.*[39] reported that the total average AL score was 2.6 (1.7 SD), with the highest being seven and the lowest being zero among office workers, from a total of 11 biomarker measurements. In the current study, we noted average total score is 2.9 (SD = 1.97), with the highest being nine and the lowest being zero. The average AL score noted in this study was close to the AL score observed in Indian adolescents.[37] AL significantly predicts all cause of mortality and future illness and disability.[40] Moore *et al.*[41] reported that 48.6% of the US male adult population had high AL. In the present study, we found that 56.8% of male workers had high AL. The AL identified in this study was 1.2% greater than in the adult male population of the US. Obesity, hypertension and CVD risk factors were found to be more prevalent in industrial employees in recent research.[42-44] These risk factors may contribute to the development AL among industrial workers.

Table 5: ROC-AUC analysis of non-HDL-C fraction among workers with low and high AL.

Type	Non-HDL-C fractions	AUC	95% CI	*P*	Optimal cutoff	Sensitivity (%)	Specficity (%)
AL (High)	Non-HDL-C	0.766	0.696–0.837	0.001	117.5	70.0	70.0
	Non-HDL-C/HDL-C	0.638	0.555–0.721	0.002	3.8	60.4	60.3
	Non-HDL-C/TC	0.635	0.552–0.712	0.003	0.79	60.4	60.3
	Non-HDL-C/TG	0.480	0.393–0.567	0.661	0.69	53.0	43.8
	Non-HDL-C/LDL-C	0.520	0.433–0.607	0.657	1.4	49.0	49.3
AL (Low)	Non-HDL-C	0.234	0.163–0.304	0.001	117.5	30.0	30.0
	Non-HDL-C/HDL-C	0.362	0.279–0.445	0.002	3.8	39.6	40.0
	Non-HDL-C/TC	0.365	0.281–0.448	0.003	0.79	39.7	39.6
	Non-HDL-C/TG	0.520	0.433–0.607	0.661	0.69	47.0	56.0
	Non-HDL-C/LDL-C	0.480	0.393–0.567	0.657	1.4	50.7	51.0

ROC-AUC: Receiver operating characteristic curve-area under the curve, AL: Allostatic load, LDL-C: Low-density lipoprotein-cholesterol, HDL-C: High-density lipoprotein-cholesterol, TC: Total cholesterol, TG: Triglyceride

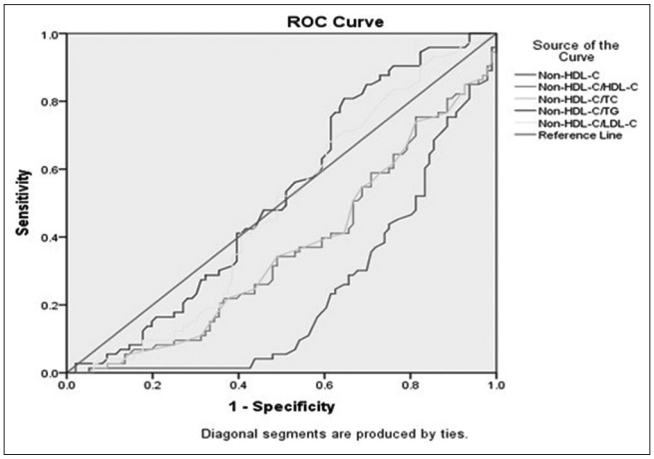

Figure 3: Receiver operating characteristic curves of non-high-density lipoprotein-cholesterol fraction for diagnosis of low allostatic load (<2.9).

The presence of excess energy substrates, such as lipids and causes metabolic stress, which leads to weight gain and mitochondrial stress.[45] Integrating TG and TC into the AL assessment increases the ability to predict the disease risk[25] In the present study, the AL score was computed using TC, TG, HDL-C and LDL-C. According to Memiah *et al*.,[46] HDL-C and TG are significant predictors of AL. In addition, non-HDL-C was employed to determine the CVD risk in high AL conditions.[47] Non-HDL-C fraction is the TC minus HDL-C. It is not a regular part of lipid profile and is used in lipoprotein lowering therapy[4] and prediction of CAD,[2] target organ damage[3] and atherosclerosis.[8] The diagnostic accuracy and optimal cut points of non-HDL-C and its fractions such as non-HDL-C/HDL-C, non-HDL-C/TC, non-HDL-C/TG and non-HDL-C/LDL-C in workers with AL need to be explored. In this study, we reported significantly increased levels of non-HDL-C, non-HDL-C/HDL-C, non-HDL-C/TC and non-HDL-C/LDL-C in the high AL (>2.9) group as compared to the low AL (<2.9) group.

During the present study, it was noted that there was a positive and significant association between non-HDL-C and its fraction (non-HDL-C/HDL-C, non-HDL-C/TC and non-HDL-C/TG) and AL among workers. A negative and insignificant association was noted between AL and non-HDL-C/TG. Zilioli *et al*.[48] also noted a similar association between the metabolic lipids (TG, HDL-C and LDL-C) and AL.

CONCLUSION

The present study examined the association between non-HDL-C and its fraction with AL among industrial workers. The findings of the model indicated that fractions such as non-HDL-C, non-HDL-C/TC and non-HDL-C/LDL-C were significantly associated with AL. Furthermore, we also used the ROC-AUC analysis for prediction of non-HDL-C fractions in low and high AL categories. Non-HDL-C and its fractions were more precisely predicted in the high AL category of workers than in the low AL category. Non-HDL-C predicted the most precisely, followed by non-HDL-C/HDL-C, non-HDL-C/TC, non-HDL-C/LDL-C and non-HDL-C/TG. The present study recommends that the non-HDL-C and its fractions were considered as regular lipid profile and could be used as biomarkers to minimise the risk of AL.

Limitation of study

The present study examined the association between non-HDL-C and its fraction with AL in a small sample size. The AL is influenced by factors such as socioeconomic status, ethnicity, household crowding and so on. In the forthcoming study, we recommend a large frame of sample size with consideration of the mentioned parameters.

Declaration of patient consent

Patient's consent not required as there are no patients in this study.

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ICMR-National Institute of Occupational Health.

Conflicts of interest

There are no conflicts of interest.

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