

Original article:

A Biochemical Study Of High-Density Lipoprotein Cholesterol (Hdl-C) Changes In Middle Aged Common People With Different Lifestyle

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Abstract:

Background and rationale: HDL cholesterol is one of the 5 major groups of lipoproteins cholesterol, which enable lipids like cholesterol and TG to be transported within the water based blood stream. In healthy persons, about thirty percent of blood cholesterol is carried by HDL cholesterol. HDL-C is a potent predictor of coronary heart disease. Genetic as well as environmental factors including lifestyle factors play a role as determinants of its level in the blood. To examine the effects of certain lifestyle factors on serum level of high density lipoprotein cholesterol in young adult people HDL cholesterol seems to protect against CVD which increases the risk for heart disease. **Subjects and methods:** Three hundred and twenty five young adult subjects of both sexes aged 18-45 years asymptomatic for cardiovascular diseases were interviewed according to special questionnaire including information on lifestyle habits. Physical examination was done, height, body weight, and blood pressure measurements were performed. Blood analysis to determine the blood level of high density lipoprotein cholesterol was done after 12 hours fasting. **Results and conclusion:** Smoking and obesity were the most significant risk factors associated with a decreased level of high density lipoprotein cholesterol. The level of HDL-C was 50.5±11.5 mg/dl in smokers compared with 57.7±12.5 mg/dl in non-smokers. Its level was 48.5 ±8.5 mg/dl in obese individuals compared to 57.5±11.7mg/dl in normal body weight subjects. Physical activity was not significantly associated with low level of HDL-C analysis, but it was found to be significantly associated with its level by the multiple regression analysis. High-density lipoprotein cholesterol level was a function of many factors, some of them were lifestyle related such as smoking, physical activity, and obesity. Therefore, efforts to encourage more physical activity, quitting smoking, consuming low fat diet, and keeping ideal body weight are recommended.

Keywords: HDL-C; Lipoprotein; CVD; CAD; Smoking.

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Introduction

Strong evidence from epidemiological and clinical trials had supported an inverse relationship between HDL-C and risk of coronary heart disease (CHD).^{1,2,7,8} For every 1mg/dl increase in serum HDL-C, there appears to be a corresponding 2% to 3% decrease in CHD risk and 4% to 5% decrease in cardiovascular diseases mortality.^{2,7} Approximately one quarter to one third of patients with pre-existing coronary disease and desirable total cholesterol [less

than 5.2 mmol/L] have low levels of HDL-C [less than 1 mmol/L] as the primary abnormality.^{2,3,9} The protective effects of HDL-C are multi factorial and many possibilities are suggested; HDL-C may prevent oxidation of LDL-C, so it protects against excess lipid accumulation in the blood vessel wall.^{3,10} It provides a reverse cholesterol transport from the tissues to the liver for metabolic conversion and excretion.^{3,11} It has also been hypothesized that cholesterol efflux from atherosclerotic lesion is promoted by HDL-C,

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possibly through a receptor related mechanism^{4,12}

In addition to constitutional determinants such as age, sex, ethnicity and genetic factors, other variables such as dietary fat, obesity and other lifestyle factors including physical activity, alcohol consumption and smoking habit are known to affect serum HDL- C level.^{5,13-17} There are many studies worldwide which studied the influence of lifestyle on serum lipids (including HDL-C),¹⁵⁻¹⁷ but studies which examined the relation between lifestyle factors and HDL-C serum levels in young adults asymptomatic for coronary heart diseases are scarce in Karaikal, India. after obtaining the approval Institutional research and ethical committee, this cross-sectional study was conducted in the department of Biochemistry, Vinayaka Missions Medical college and hospital, Karaikal, India between (January to march 2015) to examine the effect of certain lifestyle factors on serum HDL-C level.

Subjects And Methods:

Subjects:

The study population consisted of 325 young adult individuals of both sexes (aged 18-45 years) apparently healthy, with no history of cardiovascular diseases, who were selected randomly from three colleges nearby to the Vinayaka Missions medical college and hospital, Karaikal and from College Bus drivers.

Methods:

Interviewing was performed according to a special questionnaire form which covers the following aspects; Socio-demographic characteristics, medical history, aspects of dietary habits, alcohol consumption, physical activity, and smoking. Smoking habits: non- smokers defined by those who never smoke, Ex- smokers included those who stopped smoking before more than 3 months, while Current smokers were those who smoke regularly. Diet was classified on the type of food that individuals consumed, those who consumed a well known fatty diet such as butter, cream, cheese, solid fat etc, on most days of the week were considered to be on fatty diet. Physical activity was measured by using a combined index of leisure time physical activity which was calculated from the product of intensity, estimated duration of exercise and monthly frequency using the method reported by Raitakan et al[18,19]. Subjects with index of equal or higher than 80 were considered as constantly active, which nearly equals to an intensive physical activity for more than 2 hours/week. Subjects with an index value of less than or equals to 17 were considered as

constantly sedentary which nearly equals to 1 hour of light aerobic activity/ week. Those who have an index of 15-85 were considered moderately active.¹³ Height and weight were measured, and the body mass index (BMI) was calculated using the Quetlet index [BMI= weight in Kg/height in m²]²⁰ Non obese: BMI <30kg/ m², overweight: BMI 30- 34.2 kg/ m², and obese if BMI was >35 kg/ m².²¹ Clinical heart examination and pulse rate measurements were done to detect any heart abnormality. Biochemical measurements regarding serum HDL-C estimation was performed after 12 hours fasting. Serum HDL measurement was done by the Random Access Clinical chemistry analyzer (Model: XL-300) Low level of HDL-C was considered if the level was < 40 mg / dl^{22, 23}

Statistical Analysis Data was represented as mean and standard Deviation (SD) Analysis was done using SPSS (Version (16) software. The statistical analysis was made by the use of the Statistical Package for Social Science (SPSS) version 16. Analysis of variance and t- test were used when appropriate. Multiple regression analysis was used to determine whether observed differences in outcomes remained while controlling for potentially confounding variables. A P-value of <0.05 was considered significant.

Results

Table-1 demonstrates selected socio- demographic characteristics of the study population. The majority were in their early twenties. While those who were above 45 years of age formed only 3.38% of the total studied population. Regarding sex composition, the majority were men. 64.3% were with higher secondary school level of education, and 11.6% were with basic degree or more level of education.

Table 1. Socio-demographic characteristics of the study population

Character	No.	%
Age (years)		
18 – 24	195	60
25 – 29	65	20
30 – 34	32	9.8
35 – 39	22	6.7
40 – 45	11	3.38
Sex		
Men	265	81.5
Women	60	18.5

Character	No.	%
Residence		
Urban	224	68.9
Rural	101	31.1
Education		
Illiterate		
Primary & Intermediate	10	3.07
Higher Secondary	68	20.9
Basic Degree level and above	209	64.3
	38	11.6
Marital status		
Married	102	31.38
Single	223	68.61
Total	325	100

Table-2 shows the frequency of the lifestyle factors among the studied subjects. The fatty diet consumption was the most prevalent lifestyle among the study population. It was prevalent in 37.84% of them, followed by smoking which was prevalent in 27.69%. Obese and inactive subjects formed 7.6% and 15.38% of the total studied population respectively. Reliable information concerning alcohol intake was difficult to obtain from interviewees, therefore, the results were undependable. Only 3 individuals (1.53%) mentioned they were on alcohol-intake, so no further analysis was carried out regarding this variable.

Table 2. Frequency of the lifestyle determinants among the study population

Determinant	No.	%
Smoking		
Non-Smokers	225	69.23
Ex-smokers	10	3.07
Smokers	90	27.69
Physical activity		
Active	125	38.46
Moderately active	150	46.15
Inactive	50	15.38
Diet		
Non-fatty diet	202	62.15
Fatty diet	123	37.84
BMI		
Normal < 25	225	69.23
Overweight 25-29.9	75	23.07
Obese ≥ 30	25	7.69
Alcohol		
No Alcohol-Intake	320	98.46
Alcohol-Intake	5	1.53
Total	325	100

As shown in Table-3, HDL-C level was inversely associated with smoking and body mass index with a highly significant difference. HDL-C serum level was 50.5 ± 11.5 mg/dl in smokers in comparison with 57.5 ± 12.5 mg/dl in non-smokers, and its level was 55.7 ± 11.7 mg/dl in normal weight individuals compared 48.5 ± 8.5 mg/dl in obese persons. While HDL-C level was lower in fatty diet consumers (55.2 ± 12.5 mg/dl) and sedentary individuals (54.5 ± 12.3 mg/dl) in comparison with non-fatty diet consumers (56.03 ± 13.5 mg/dl) and active individuals (56.8 ± 11.9 mg/dl) respectively, but the differences were not significant.

Table 3. HDL-C level according to the studied lifestyle factors

Determinant	Mean \pm SD (mg/dl)	P-value
Smoking		
Non-smokers	57.5 ± 12.5	<0.001
Ex-smokers	55.3 ± 13.6	
Smokers	50.5 ± 11.5	
Physical activity		
Active	56.8 ± 11.9	NS
Moderately active	$56.2 \pm$	
Inactive	13.01 54.5 ± 12.3	
Diet		
Non-fatty diet	$56.03 \pm$	NS
Fatty diet	13.5 55.2 ± 12.5	
BMI (Kg/m²)		
Normal < 25	55.7 ± 11.7	<0.001
Overweight 25-29.9	53.4 ± 13.5	
Obese ≥ 30	48.5 ± 8.5	

To investigate the independent effects of selected risk factors on HDL-C level, a stepwise linear multiple regression analysis was performed (Table-4) the examined risk factors were the lifestyle determinants (i.e. smoking, physical activity, diet, and body mass index) in addition to age and sex. Body mass index, smoking and physical activity in addition to sex (females showed higher HDL-C level), and age were noticed to have highly significant effects on HDL-C concentration. BMI appears to be the strongest variable that explained 5.9% of the variability in HDL-C concentration, together with the other variables, they explained 17.9% of the variation in HDL-C concentration and about 82% of the variation is still left unaccounted for.

Table 4. Multiple regression analysis

Variable	Beta	R2	P-value
BMI	- 0.160	0.059	0.000
Smoking	- 0.181	0.104	0.000
Age	- 0.155	0.122	0.005
Sex	0.191	0.138	0.006
Physical activity	0.163	0.179	0.002

Discussion

This study has shown different contributions to the risk of low level of HDL-C from the major lifestyle factors. Smoking and HDL-C were inversely related, a result that had been reported in young people.^{24,25} This confirms the finding that smoking is associated with an unfavorable lipoprotein profile at whatever age; smoking is initiated during adolescence.²⁶ In accordance with the findings of other studies,^{27,28} BMI (an indicator of lifestyle habits such as physical inactivity and diet) was inversely associated with HDL-C, a result that was noted not only by univariate analysis, but also replicated by multiple regression analysis. The finding that physical activity was not significantly related to HDL-C which was noted in univariate analysis dropped out in the stepwise multiple regression analysis. This finding is consistent with that of Savig and Goldbourt¹⁷ Diet was the only lifestyle factor which was not found to contribute greatly to a significant HDL-C reduction. Although diet was suggested to be the major environmental factor that modulates lipid

and lipoprotein,²⁹ but the relation between fatty diet consumption and HDL-C level in this study was not found to be significant. However, its level was lower in fatty diet consumers in comparison with non-fatty diet consumers. Probably the relative proportion of carbohydrate to fat in the diet is more important factor than estimating the effect of dietary fat content alone (higher ratio is correlated with low HDL-C).³⁰ Also, favorable but not significant increase in HDL-C concentration was observed with modified lower carbohydrate diet.³¹ Dietary habit was not quantitatively measured, in this sense, inaccuracy was unavoidable, and thus may have failed to detect a real association between fatty diet and HDL-C level. As shown in the multiple regression analysis only 17.9% of the variation in HDL-C concentration was related to lifestyle factors, sex, and age. While about 82% of the variation in HDL-C left unaccounted for. This may explain the important effect of other environmental and genetic factors on HDL-C serum level. Genetic factors are known to play a major role as determinants of HDL-C, with estimate of heredity from about 45% to 65%.^{32,33} In conclusion, HDL-C level was primarily a function of many factors, some of them are lifestyle related such as smoking, physical activity and obesity. The results of this study clearly demonstrated the need to initiate preventive efforts early in life to encourage more physical activity, quitting smoking, keeping ideal body weight, and consuming healthy diet.

References:

1. C. Cui Y., Blumenthal R.S., Flaws J.A., Whiteman M.K., Langenberg P., Bachorik P.S., Bush T.L (2001). Non-high-density lipoprotein cholesterol level as a predictor of cardiovascular disease mortality. *Arch Intern Med.* **161**: 1413-9. <https://doi.org/10.1001/archinte.161.11.1413>
2. K. Sharett, A.B., Ballantyne, C.M. and Coady, S.A. (2001). Coronary heart disease prediction from lipoprotein cholesterol levels, triglycerides, lipoprotein (a), apolipoproteins A-1 and B, and HDL density subfractions. The Atherosclerosis Risk (ARIC) in Communities Study. *Circulation.* **104**: 1108-1113. <https://doi.org/10.1161/hc3501.095214>
3. E. Frost, P.H. and Havel, R.J. (1998). Rationale for use of non-high-density lipoprotein cholesterol rather than low-density lipoprotein cholesterol as a tool for lipoprotein cholesterol screening and assessment of risk and therapy. *Am J Cardiol.* **81**: 26B-31B. [https://doi.org/10.1016/S0002-9149\(98\)00034-4](https://doi.org/10.1016/S0002-9149(98)00034-4)
4. Ridker, P.M., Rifai, N., Cook, N.R., Bradwin, G. and Buring, J.E, (2005). Non-HDL cholesterol, apolipoproteins A-I and B100, standard lipid measures, lipid ratios, and CRP as risk factors for cardiovascular disease in women. *JAMA.* **294**: 326-333. <https://doi.org/10.1001/jama.294.3.326>
5. Gordon DJ, Witztum JL, Hunninghake D, Gates S, Glueck CJ. Habitual physical activity and HDL-C in men with primary hypercholesterolemia: The Lipid research Clinics Coronary Primary Prevention Trial. *Circulation* 1983; **67**(3): 512-520. <https://doi.org/10.1161/01.CIR.67.3.512>
6. Grundy, S.M. (2002). Low-Density Lipoprotein, Non-High-Density Lipoprotein, and Apolipoprotein B as Targets of Lipid-Lowering Therapy. *Circulation.* **106**: 2526-9. <https://doi.org/10.1161/01.CIR.0000038419.53000.D6>
7. Sacco RL, Benson RT, Kargman DE, Boden-Albala B, Tuck C, Lin IF, et al. HDL-C and Ischemic stroke in the elderly: The Northern Manhattan Stroke Study *JAMA* 2001; **285**: 2729-2735.
8. Aztalas BF, Schaefer EJ. HDL sub populations in pathological conditions. *Amer J Cardiology* 2003;**99**: 12E-17E.
9. Rubins HB, Robins SJ, Collins D, Iranmanesh A, Timothy J, Mann D, et al. Distribution of lipids in 8500 men with coronary artery disease. *Am J Cardiology* .1995; **75**: 1202-1205. [https://doi.org/10.1016/S0002-9149\(99\)80761-9](https://doi.org/10.1016/S0002-9149(99)80761-9)
10. Barter P. Is high-density lipoprotein the protector of the cardiovascular system? *European Heart Journal supplements* 2004; 6 (supplement A): A 19- A 22.
11. Miller M. Raising an isolated low HDL-C level: Why, how, and when? *Cleveland Clinic Journal of Medicine* 2003; **70**(6): 553-560. <https://doi.org/10.3949/ccjm.70.6.553>
12. Fuster V, Gotto AM, Libby P, Loscalzo J, McGill HC. Pathogenesis of coronary disease: The biologic role of risk factors. 27th Bethesda conference Task Force 1. *J Am Coll Cardiology* 1996; **27**: 964-976. [https://doi.org/10.1016/0735-1097\(96\)00014-9](https://doi.org/10.1016/0735-1097(96)00014-9)
13. Namekata T, Moore DE, Suzuki K, Mori M, Knopp RH, Marcovina SM, et al. Biological and lifestyle factors and lipid and lipoprotein levels among Japanese Americans in Seattle and Japanese men in Japan. *International Journal of Epidemiology* 1997; **26**(6): 1203-1213. <https://doi.org/10.1093/ije/26.6.1203>
14. De Oliveira Silva ER, Kong M, Han Z, Starr C, Kass EM, Juo SH, et al. Metabolic and genetic determinants of DL metabolism and hepatic lipase activity in normolipidemic females. *Journal of Lipid Research* 1999; **40**: 1211-1221.
15. Ellison RC, Zhang Y, Qureshi MM, Knox S, Arnett DK, Province MA. Lifestyle determinants of high-density lipoprotein cholesterol: The National Heart, Lung, and Blood Institute Family Heart Study. *Am Heart Journal* 2004; **147**(3): 529-535. <https://doi.org/10.1016/j.ahj.2003.10.033>
16. Wilsgaard T, Arnesen E. Change in serum lipids and body mass index by age, sex, and smoking status: The Thrombosis study 1986-1995. *Annals of Epidemiology* 2004; **14**(4): 265-273. <https://doi.org/10.1016/j.annepidem.2003.08.004>
17. Sagiv M, Goldbourt U. Influence of physical work on high-density lipoprotein cholesterol- Implications for the risk of coronary heart disease. *International Journal of Sports Medicine* 1994;**15**(5): 261-266 <https://doi.org/10.1055/s-2007-1021057>
18. Ilaan SK. Lipid profile and physical activity *J Bahrain Medical Society* 2004;**16**(4): 186-191.
19. Raitakan OT, Porkka KVK, Taimela S, Telama R, Rasanen L, Vilkari JS. Effects of persistent activity and inactivity on coronary risk factors in children and young adults: The Cardiovascular Risk in Young Finns Study. *Amer J Epidemiol* 1994;**140**(3):195-205. <https://doi.org/10.1093/oxfordjournals.aje.a117239>
20. Frier BM, Truswell AS, Shepherd J, et al. Nutritional factors in health and disease. In: Haslett C, Chillver ER, Hunter TA & Boon NA (Eds): Davidson's principles and practice of Medicine. ed. London. *Churchill living stone.* 2001.
21. WHO: Obesity, preventing and managing the global epidemic. Technical reports series 894, WHO, Geneva, Switzerland, 2000.
22. Burstein M, Scholnick HR, Morfin R. Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. *J Lipid Res* 1970; **11**:583-595.

23. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the third report on the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA* 2001; **285**(19): 2486-2497. <https://doi.org/10.1001/jama.285.19.2486>
 24. Miller ER, Appel LJ, Jiang L, Risby TH. Association between cigarette smoking and lipid peroxidation in a controlled feeding study. *Circulation* 1997; **96**: 1097-1101. <https://doi.org/10.1161/01.CIR.96.4.1097>
 25. Sharma SB, Dwivedi S, Prabhu KM, Singh G, Kumar N, Lal MK. Coronary risk variables in young asymptomatic smokers. *Indian J Med Res* 2005; **122**: 205-210. *MJBU*, VOL 26, No.1, 2008
 26. Clarke WR, Srinivasan SR, Shear CL, Hunter SM, Croft JB, Webber LS, Berenson GS. Cigarette smoking initiation and longitudinal changes in serum lipids and lipoproteins in early adulthood; The Bogalusa Heart study. *Amer J Epidemiol* 1986; **124**: 207-219. <https://doi.org/10.1093/oxfordjournals.aje.a114379>
 27. Plourde G. Impact of obesity on glucose and lipid profile in adolescents at different age groups in relation to adulthood. *BMC Family Practice* 200; **3**: 18-31. <https://doi.org/10.1186/1471-2296-3-18>
 28. Assmann G, Schulte H. Relation of HDL-C and triglycerides to incidence of atherosclerotic CAD (The PROCAM experience): A prospective cardiovascular Munster study. *Am J Cardiol* 1992; **70**(7): 733-737. [https://doi.org/10.1016/0002-9149\(92\)90550-I](https://doi.org/10.1016/0002-9149(92)90550-I)
 29. Schafer EJ. Lipoproteins, nutrition & heart disease. *Amer J Clin Nutr* 2002; **75**: 161-212.
 30. Gordon DJ, Witztum JL, Hunninghake D, Gates S, Glueck CJ. Habitual physical activity and HDL-C in men with primary hypercholesterolemia: The Lipid research Clinics Coronary Primary Prevention Trial. *Circulation* 1983; **67**(3): 512-520. <https://doi.org/10.1161/01.CIR.67.3.512>
 31. Aude YW, Agatston AS, Lopez-Jimenez F, Lieberman EH, Almon M, Hansen M, et al. The national cholesterol education program Diet vs a Diet Lower in Carbohydrate and Higher in Protein and Monosaturated Fat. *Arch Intern Med* 2004; **164**: 2141-2146. <https://doi.org/10.1001/archinte.164.19.2141>
 32. Cohen JC, Wang Z, Grundy SM, et al. Variation at the hepatic lipase and apolipoprotein A1/CIII/AIV loci is a major cause of genetically determined variation in plasma HDL-Cholesterol levels. *J Clin Invest* 1994; **94**: 2377-2384. <https://doi.org/10.1172/JCI117603>
 33. Inazu A, Nishimura Y, Terada Y, Mabuchi H. Effects of hepatic lipase gene promoter nucleotide variations on serum HDL-Cholesterol concentration in the general Japanese population. *J Hum Genet* 2001; **46**(4): 172-177. <https://doi.org/10.1007/s100380170084>
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