IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 24, Issue 10 Ser. 5 (October. 2025), PP 30-36
www.iosrjournals.org

Occurrence of Dyslipidaemia in Patients with Tuberculosis

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Abstract
Background: *Dyslipidaemia is an increasingly recognized comorbidity in individuals with tuberculosis (TB), potentially arising from infection-induced metabolic alterations and drug-related hepatotoxicity. In high-burden countries such as Nigeria, TB control strategies largely prioritize infectious disease management, with limited focus on coexisting metabolic disorders. This study evaluated the prevalence and pattern of dyslipidaemia among TB patients and explored its association with socio-demographic variables.*
Methods: *A hospital-based cross-sectional study was conducted among 171 newly diagnosed adult pulmonary TB patients attending the Directly Observed Treatment Short-course (DOTS) clinic at the University of Port Harcourt Teaching Hospital (UPTH), Rivers State, Nigeria. Data collection involved structured interviewer-administered questionnaires, and fasting venous blood samples were analysed for lipid profile parameters—total cholesterol, triglycerides, low-density lipoprotein (LDL), and high-density lipoprotein (HDL)—using enzymatic colorimetric assays. Dyslipidaemia was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) guidelines. Associations between lipid abnormalities and patient characteristics were assessed using chi-square tests, with statistical significance set at $p < 0.05$.*
Results: *Dyslipidaemia was present in 75.4% of the study population. Abnormalities included elevated total cholesterol in 44%, raised triglycerides in 48%, low HDL in 15.8%, and elevated LDL in 75.4% of participants. Age was significantly associated with dyslipidaemia ($p = 0.001$), with the highest prevalence observed in those aged 40–49 years (88.1%) and 50–59 years (85.7%). Educational level was also significant ($p = 0.0001$), with tertiary education holders showing the highest prevalence (90%). Occupational status demonstrated a significant relationship ($p = 0.011$), with unskilled workers (80.2%) and skilled workers (77.8%) more affected than participants in the "Others" category (50%). No statistically significant association was found with sex or marital status.*
Conclusion: *Dyslipidaemia is common among TB patients in this setting, particularly among older adults, those with higher educational attainment, and individuals in formal employment. These findings underscore the importance of incorporating lipid profile assessment and cardiovascular risk evaluation into TB management protocols, especially within tertiary healthcare facilities. Early detection and appropriate intervention may reduce long-term cardiovascular complications in TB survivors.*
Keywords: *Tuberculosis, Dyslipidaemia, LDL, HDL, Cardiovascular risk, Nigeria, Metabolic disorders*

Date of Submission: 13-10-2025 Date of Acceptance: 28-10-2025

I. INTRODUCTION

Tuberculosis (TB) remains one of the most widespread and deadly infectious diseases globally, with a disproportionate impact on low- and middle-income countries. In 2022, the World Health Organization (WHO) estimated that 10.6 million individuals developed TB worldwide, resulting in approximately 1.3 million deaths among HIV-negative individuals and an additional 167,000 deaths among people living with HIV[1,2]. The disease burden is particularly severe in regions marked by poverty, undernutrition, and weak healthcare systems.

Emerging evidence points to an increasing overlap between TB and non-communicable metabolic disorders such as dyslipidaemia[3,4]. Dyslipidaemia—defined by abnormalities in lipid metabolism, including elevated total cholesterol, low-density lipoprotein cholesterol (LDL-C), triglycerides, or reduced high-density lipoprotein cholesterol (HDL-C)—is a well-established risk factor for cardiovascular disease[5,6]. Its occurrence in TB patients is not fully understood but is believed to be influenced by factors such as chronic inflammation, nutritional deficits, drug-induced metabolic changes, and immune system activation[7,8]. Classically, it presents as elevated plasma triglycerides and/or cholesterol, often accompanied by reduced HDL-C and increased LDL-C, typically associated with high dietary fat intake and increased hepatic lipid metabolism[9].

The relationship between TB and lipid metabolism is complex. Mycobacterium tuberculosis has been shown to interact with host lipids, promoting the formation of lipid-rich "foam cells" that aid bacterial persistence and immune evasion[10,11]. Systemic inflammation and metabolic stress during active TB can further disrupt lipid homeostasis[5,8], while anti-TB medications, particularly rifampicin and isoniazid, have been implicated in altering lipid profiles[5,6]. Elevated cholesterol levels have been associated with increased TB risk, as the pathogen relies on host-derived lipids for survival, contributing to foam cell formation[12]. In TB–diabetes comorbidity, impaired lipid metabolism may result in high very low-density lipoprotein (VLDL), low HDL-C, elevated sphingomyelin, and increased remnant total cholesterol lipoproteins, with LDL-C elevation linked to reduced LDL lipolysis. Lawson et al. reported elevated LDL-C in 29% of their cohort, high total cholesterol in 11.7%, high triglycerides in 6.6%, and low HDL-C in 50.5%[13].

Despite these findings, there is limited data on the prevalence and pattern of dyslipidaemia in TB patients, particularly within sub-Saharan Africa. Characterizing dyslipidaemia in this population is essential for developing integrated TB care strategies and reducing long-term cardiovascular complications in TB survivors. This study therefore seeks to determine the prevalence of dyslipidaemia among TB patients at the University of Port Harcourt Teaching Hospital and to examine its socio-demographic and clinical correlations.

II. MATERIALS AND METHODS

2.1 Study Design and Setting

This hospital-based descriptive cross-sectional study was carried out at the Directly Observed Treatment, Short-course (DOTS) Clinic of the University of Port Harcourt Teaching Hospital (UPTH) in Rivers State, Nigeria. UPTH serves as a tertiary referral center, offering diagnostic and treatment services to patients from both urban and rural areas across southern Nigeria. Data collection spanned a 12-month period.

2.2 Study Population

The study population comprised adult patients aged 18 years and above who had a new diagnosis of pulmonary tuberculosis and had initiated anti-TB treatment within the study period. Participants were recruited consecutively. Eligibility criteria included a confirmed diagnosis of pulmonary TB through GeneXpert testing or sputum smear microscopy and a willingness to provide informed consent. Individuals were excluded if they had a prior diagnosis or history of treatment for dyslipidaemia, were on lipid-lowering therapy, had HIV co-infection, chronic liver disease, or were pregnant.

2.3 Sample Size Determination

The minimum sample size for this study was calculated using Kish's formula for sample size estimation in populations greater than 10,000:

$n = (Z^2 \times p \times q) / d^2$

Where:

- n = required sample size for large populations
- Z = standard normal deviate at 95% confidence level (1.96)
- p = estimated prevalence of diabetes mellitus among tuberculosis patients, set at 15.1% (0.151) based on findings from a similar study by Lawson et al. (Abuja, Nigeria)
- q = 1 − p = 0.849
- d = desired precision or margin of error = 0.05

Substituting the values:

$n = (1.96^2 \times 0.151 \times 0.849) / (0.05)^2$

$n = (3.8416 \times 0.128199) / 0.0025 = 0.4926 / 0.0025 = 197$

Since the total population of newly diagnosed TB patients at the University of Port Harcourt Teaching Hospital (UPTH) in the previous year was estimated at 707 (obtained from hospital records), a finite population correction was applied:

$Nf = n / [1 + (n/N)] = 197 / [1 + (197/707)] = 197 / 1.2786 \approx 154$

To account for an anticipated 10% non-response rate, the adjusted final sample size was:

$154 + (10\% \text{ of } 154) = 154 + 15.4 \approx 169$

The study therefore adopted a final sample size of 171 participants, allowing for adequate power and potential exclusions.

2.4 Data Collection

Data collection was conducted using a structured, interviewer-administered questionnaire that obtained information on socio-demographic characteristics (age, sex, education, marital status, occupation), lifestyle behaviours (smoking status, alcohol consumption), and clinical history (family history of cardiovascular disease, duration of TB illness). Anthropometric parameters, including weight and height, were measured using calibrated equipment, and body mass index (BMI) was subsequently calculated.

2.5 Laboratory Analysis

Following an overnight fast of 8–12 hours, venous blood samples were obtained from all participants. Lipid profile parameters—total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C)—were measured using enzymatic colorimetric assays on an automated biochemistry analyzer. Dyslipidaemia was classified based on the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) criteria as: TC ≥ 200 mg/dL, LDL-C ≥ 130 mg/dL, HDL-C < 40 mg/dL for men or < 50 mg/dL for women, and TG ≥ 150 mg/dL.

2.6 Data Management and Analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 25. Descriptive statistics were applied to summarize participant characteristics and lipid profile parameters. Associations between dyslipidaemia and categorical variables were evaluated using the chi-square test or Fisher's exact test, with statistical significance defined as $p < 0.05$.

2.7 Ethical Considerations

Ethical clearance was granted by the Research and Ethics Committee of the University of Port Harcourt Teaching Hospital. Written informed consent was obtained from each participant before data collection and sample acquisition. Participant confidentiality was maintained through data anonymization and secure storage of all records. Individuals identified with dyslipidaemia were referred for appropriate clinical evaluation and management.

III. RESULTS

Table 1 summarizes the baseline characteristics of the 171 TB patients included in the study. Males constituted 53.8% of the cohort, while females accounted for 46.2%. The largest age category was 30–39 years (39.8%), followed by 18–29 years (27.5%), with a mean age of 30.03 ± 8.89 years. Most participants had attained tertiary education (64.3%), while 27.5% had secondary-level education and 8.2% had only primary education. More than half were married (59.1%). In terms of occupation, half of the participants (50.3%) were engaged in unskilled work, while 36.8% held skilled or professional positions.

Table 1: Socio-demographic Characteristics of patients

Characteristics	Frequency (n = 171)	Percent (%)
Sex		
Male	92	53.8
Female	79	46.2
Age Bracket (in years)		
18 – 29	47	27.5
30 – 39	68	39.8
40 – 49	42	24.6
50 – 59	14	8.2
Mean age ± SD	30.03± 8.89	
Education level		
Primary	14	8.2
Secondary	47	27.5
Tertiary	110	64.3
Marital status		
Single	66	38.6
Married	101	59.1
Widow/widower	4	2.3
Occupation		
Skilled/professional	63	36.8
Unskilled	86	50.3
Others	22	12.9

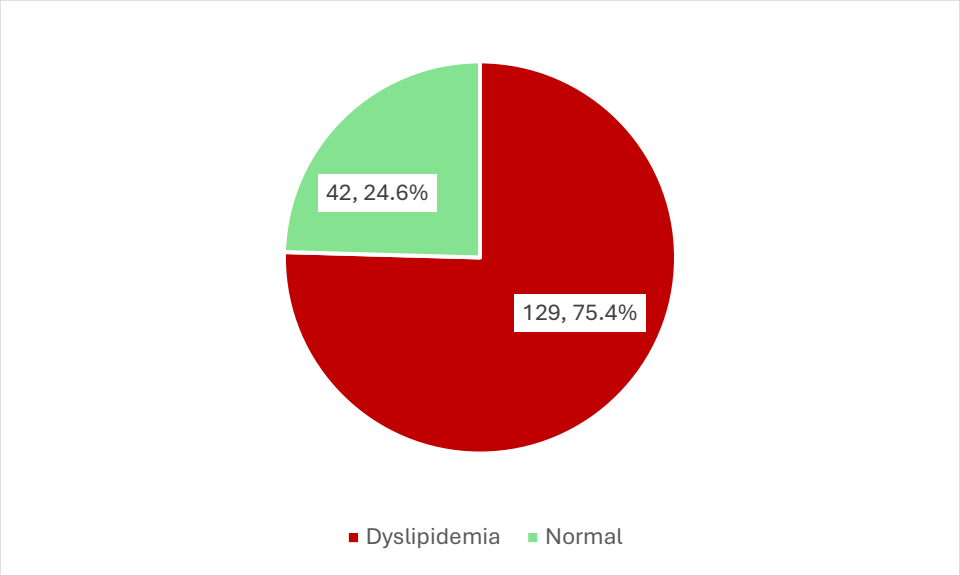

Figure 1: Prevalence of dyslipidaemia in TB Patients

Figure 1 shows that there was a 75.4% overall prevalence of dyslipidaemia among the study participants.

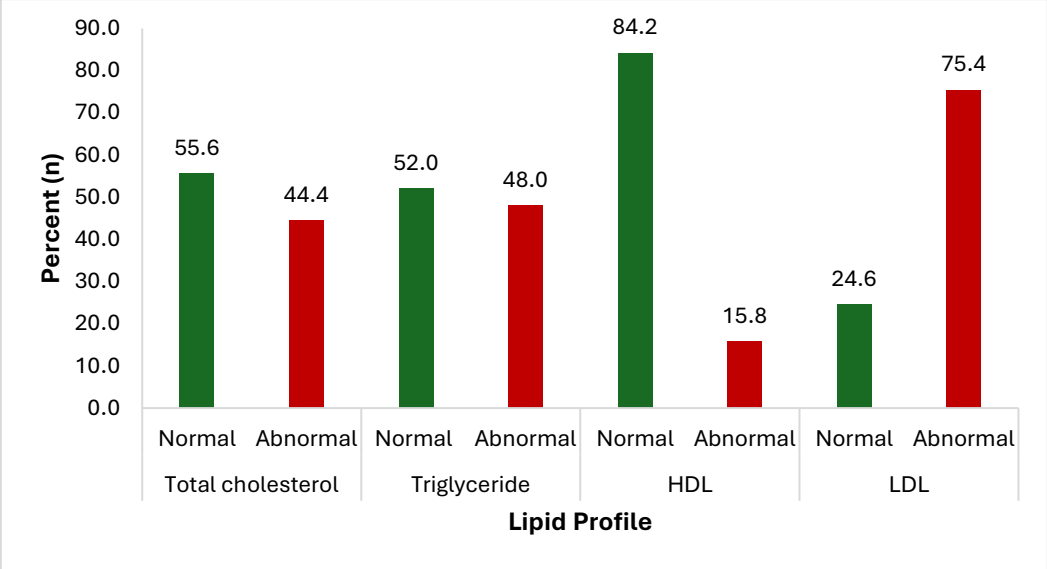

Figure 2: Distribution of Dyslipidaemia in TB Patients

Figure 2 shows the distribution of dyslipidaemia among the patients, 44% had abnormal cholesterol, 48% had abnormal triglyceride, 15.8% had abnormal HDL and 75.4% had abnormal LDL.

Table 2: Association of Demographic factors and dyslipidaemia in TB Patients

Characteristics	**Dyslipidaemia n, (%)**	**No Dyslipidaemia n, (%)**	**Total n, (%)**	**Chi-square (p-value)**
Sex				
Male	74(80.4)	18(19.6)	**92(100.0)**	2.68 (0.101)
Female	55(69.6)	24(30.4)	**79(100.0)**	
Age Bracket (in years)				
18 – 29	26(55.3)	21(44.7)	**47(100.0)**	15.28 (0.001)*
30 – 39	54(79.4)	14(20.6)	**68(100.0)**	
40 – 49	37(88.1)	5(11.9)	**42(100.0)**	
50 – 59	12(85.7)	2(14.3)	**14(100.0)**	
Education level				
Primary	7(50.0)	7(50.0)	**14(100.0)**	35.29 (0.0001)*
Secondary	23(48.9)	24(51.1)	**47(100.0)**	
Tertiary	99(90.0)	11(10.0)	**110(100.0)**	
Marital status				
Single	47(71.2)	19(28.8)	**66(100.0)**	2.80 (0.2456)
Married	80(79.2)	21(20.8)	**101(100.0)**	

Widow/widower	2(50.0)	2(50.0)	**4(100.0)**	
Occupation				
Skilled/professional	49(77.8)	14(22.2)	**63(100.0)**	8.93 (0.011)*
Unskilled	69(80.2)	17(19.8)	**86(100.0)**	
Others	11(50.0)	11(50.0)	**22(100.0)**	

**statistically significant (p<0.05)*

Table 2 shows the analysis of associations between socio-demographic factors and dyslipidaemia. The results revealed that although 80.4% of males and 69.6% of females had dyslipidaemia, the difference was not statistically significant ($p = 0.101$). A strong and statistically significant association was observed with age ($p = 0.001$), as the prevalence of dyslipidaemia was lowest among participants aged 18–29 years (55.3%) and highest among those aged 40–49 (88.1%) and 50–59 (85.7%). Educational attainment also showed a highly significant association with dyslipidaemia ($p = 0.0001$), with 90% prevalence among those with tertiary education compared to 50% and 48.9% among those with primary and secondary education, respectively. No significant association was found with marital status ($p = 0.2456$). In contrast, occupational status demonstrated a statistically significant relationship ($p = 0.011$), with the highest dyslipidaemia prevalence among unskilled workers (80.2%) and skilled/professional workers (77.8%), while participants classified under the others category had a considerably lower prevalence (50%).

IV. DISCUSSION

This study identified a markedly high prevalence of dyslipidaemia among tuberculosis (TB) patients, with 75.4% of participants affected, a figure substantially higher than the 25%–45% range reported in the general Nigerian population depending on geographic and demographic variations[1] and consistent with growing evidence that active TB perturbs lipid metabolism through inflammation-driven pathways and metabolic stress[2]. Older age (≥40 years) showed a strong association with dyslipidaemia, mirroring observations by Sun et al.[3] and Boillat-Blanco et al.[4] that lipid abnormalities intensify with declining metabolic flexibility and cumulative inflammatory load, and dovetailing with population data that tie advancing age to atherogenic shifts in lipoprotein profiles[20]. An unexpectedly high prevalence among participants with tertiary education likely reflects urban lifestyle transitions—reduced physical activity, energy-dense diets, and circadian disruption from knowledge-economy work—despite presumed health literacy, a pattern echoed among Nigerian urban professionals by Okafor et al.[5] and by multi-country analyses that link rapid urbanization to cardio-metabolic risk accumulation[21,22]. Occupational status also mattered: both skilled and unskilled workers showed elevated dyslipidaemia, consistent with Ekeke et al.[6] and with literature connecting job strain, shift work, and precarious employment to adverse lipid phenotypes via neuroendocrine stress axes and sleep fragmentation[23,24]. Mechanistically, chronic TB provokes a cytokine milieu (TNF-α, IL-6) that suppresses hepatic ApoA-I synthesis, impairs reverse cholesterol transport, and accelerates LDL oxidation[7], while Mycobacterium tuberculosis co-opts host lipids and drives foam-cell formation to enable persistence and immune evasion[8]; first-line agents such as rifampicin and isoniazid further remodel lipid homeostasis through hepatic enzyme induction and mitochondrial stress, compounding the atherogenic shift[8,9]. Beyond traditional lipids, functional changes matter: HDL becomes pro-inflammatory under oxidative stress, with altered paraoxonase-1 activity and compromised cholesterol efflux capacity that track worse outcomes in infectious and cardiometabolic settings[25,26], and TB-associated hypoalbuminaemia and micronutrient deficits can depress measured cholesterol while masking vascular risk[27]. Additional evidence strengthens the metabolic-infection nexus: in TB–diabetes comorbidity, patients exhibit highly atherogenic signatures with elevated VLDL triglycerides, sphingomyelins, and remnant cholesterol together with reduced HDL, as shown by Vrieling et al.[19]; Mendelian randomization by Du et al. suggests differential, fraction-specific links between lipids and TB susceptibility, with HDL-C, LDL, and triglycerides positively associated with TB risk but total cholesterol inversely related[8]; cohort work by Chidambaram et al. ties higher baseline HDL and total cholesterol to lower mortality and dampened inflammation[11]; and studies by Mani et al.[2] and Suresh et al.[9] show that low lipid levels accompany greater TB severity yet tend to normalize on therapy, arguing for bidirectional causality and potential use of lipids as dynamic biomarkers. Host genetics may shape these trajectories—polymorphisms in APOE, CETP, and LDLR influence lipoprotein handling and infectious susceptibility in diverse populations[28,29]—while gut dysbiosis during TB and antibiotic exposure likely perturbs bile-acid and short-chain-fatty-acid signaling that modulates hepatic lipid synthesis[30]. Therapeutically, adjunct strategies deserve attention: statins exhibit immunomodulatory and antimycobacterial-adjunct effects in observational and preclinical work, with signals for improved TB outcomes and reduced mortality independent of lipid lowering[13,31], metformin has been associated with enhanced host-directed control and better clinical endpoints in TB-diabetes cohorts[32], and omega-3 polyunsaturated fatty acids may temper TB-related inflammation and restore macrophage lipid handling in experimental models[33]. Together, these strands underscore a syndemic interface between infection and metabolism that TB programs cannot ignore; integrating fasting lipids or non-fasting lipid panels, BMI and waist-related

metrics, and simple dietary/physical-activity screening into DOTS visits would enable early risk stratification, while targeted counseling, timely referral for lipid management, and careful pharmacovigilance for drug-induced dyslipidaemia could mitigate downstream cardiovascular risk, particularly in tertiary centers serving urbanizing catchments where cardiometabolic risk converges with infectious burden[21,22,23]. Early identification and focused management of dyslipidaemia, coupled with prospective follow-up through intensive and continuation TB treatment phases, should clarify reversibility, disentangle causal pathways, and, critically, reduce the atherosclerotic complications that increasingly shape survivorship in high-burden settings.

V. CONCLUSION

This study not only confirms the high prevalence of dyslipidaemia among TB patients, but also identifies significant socio-demographic correlates, including age, educational status, and occupation. These findings reinforce the necessity of early lipid screening and comprehensive risk assessment in all TB patients, particularly those at heightened metabolic risk. Future studies should adopt a longitudinal design to determine whether dyslipidaemia resolves with TB treatment or persists as a chronic comorbidity. Moreover, prospective cohort data could help delineate whether metabolic disturbances influence TB outcomes such as treatment failure, relapse, or mortality.

REFERENCES

[1]. Global tuberculosis report 2023. 2023 [cited 2025 Jun 18]; Available from: https://iris.who.int/.

[2]. Parivakkam Mani A, K S, K DK, Yadav S. Assessment of Lipid Profile in Patients With Pulmonary Tuberculosis: An Observational Study. Cureus [Internet]. 2023 May 20 [cited 2025 Jun 19];15(5). Available from: https://pubmed.ncbi.nlm.nih.gov/37342750/

[3]. Boillat-Blanco N, Ramaiya KL, Mganga M, Minja LT, Bovet P, Schindler C, et al. Transient hyperglycemia in patients with tuberculosis in Tanzania: Implications for diabetes screening algorithms. Journal of Infectious Diseases [Internet]. 2016 Apr 1 [cited 2025 Jun 18];213(7):1163–72. Available from: https://pubmed.ncbi.nlm.nih.gov/26609005/

[4]. Viswanathan V, Kumpatla S, Aravindalochanan V, Rajan R, Chinnasamy C, Srinivasan R, et al. Prevalence of diabetes and pre-diabetes and associated risk factors among tuberculosis patients in India. PLoS One [Internet]. 2012 Jul 26 [cited 2025 Jun 18];7(7). Available from: https://pubmed.ncbi.nlm.nih.gov/22848473/

[5]. Gebremicael G, Amare Y, Challa F, Gebreegziabxier A, Medhin G, Wolde M, et al. Lipid Profile in Tuberculosis Patients with and without Human Immunodeficiency Virus Infection. Int J Chronic Dis [Internet]. 2017 Jan 1 [cited 2025 Jun 19];2017(1):3843291. Available from: /doi/pdf/10.1155/2017/3843291

[6]. Ahmed N, Amjad I, Malik ZA, Naseer A, Raza M, Imtiaz A, et al. Effects of Anti-Tuberculosis Drugs on Lipid Profile in Pulmonary Tuberculosis Patients. Journal of the Dow University of Health Sciences (JDUHS) [Internet]. 2022 Jan 27 [cited 2025 Jun 19];16(2):78–82. Available from: https://www.jduhs.com/index.php/jduhs/article/view/1235

[7]. Vrieling F, Ronacher K, Kleynhans L, van den Akker E, Walzl G, Ottenhoff THM, et al. Patients with Concurrent Tuberculosis and Diabetes Have a Pro-Atherogenic Plasma Lipid Profile. EBioMedicine [Internet]. 2018 Jun 1 [cited 2025 Jun 19];32:192–200. Available from: https://www.sciencedirect.com/science/article/pii/S2352396418301683

[8]. Baluku JB, Nalwanga R, Kazibwe A, Olum R, Nuwagira E, Mugenyi N, et al. Association between biomarkers of inflammation and dyslipidemia in drug resistant tuberculosis in Uganda. Lipids Health Dis [Internet]. 2024 Dec 1 [cited 2025 Jun 19];23(1). Available from: https://pubmed.ncbi.nlm.nih.gov/38429714/

[9]. Isago H. The Association between Dyslipidemia and Pulmonary Diseases. J Atheroscler Thromb [Internet]. 2024 Sep 1 [cited 2025 Jun 19];31(9):1249–59. Available from: http://doi.org/10.5551/jat.RV22021

[10]. Jo YS, Han K, Kim D, Yoo JE, Kim Y, Yang B, et al. Relationship between total cholesterol level and tuberculosis risk in a nationwide longitudinal cohort. Sci Rep [Internet]. 2021 Dec 1 [cited 2025 Jun 19];11(1):1–9. Available from: https://www.nature.com/articles/s41598-021-95704-1

[11]. Vrieling F, Ronacher K, Kleynhans L, van den Akker E, Walzl G, Ottenhoff THM, et al. Patients with Concurrent Tuberculosis and Diabetes Have a Pro-Atherogenic Plasma Lipid Profile. EBioMedicine [Internet]. 2018 Jun 1 [cited 2025 Jun 19];32:192–200. Available from: https://pubmed.ncbi.nlm.nih.gov/29779698/

[12]. Lawson L, Muc M, Oladimeji O, Iweha C, Opoola B, Abdurhaman ST, et al. Tuberculosis and diabetes in Nigerian patients with and without HIV. International Journal of Infectious Diseases [Internet]. 2017 Aug 1 [cited 2025 Jun 19];61:121–5. Available from: https://pubmed.ncbi.nlm.nih.gov/28652217/

[13]. Xing L, Jing L, Tian Y, Yan H, Zhang B, Sun Q, et al. Epidemiology of dyslipidemia and associated cardiovascular risk factors in northeast China: A cross-sectional study. Nutrition, Metabolism and Cardiovascular Diseases [Internet]. 2020 Nov 27 [cited 2025 Jun 19];30(12):2262–70. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0939475320303070

[14]. Zhang M, Deng Q, Wang L, Huang Z, Zhou M, Li Y, et al. Prevalence of dyslipidemia and achievement of low-density lipoprotein cholesterol targets in Chinese adults: A nationally representative survey of 163,641 adults. Int J Cardiol [Internet]. 2018 Jun 1 [cited 2025 Jun 19];260:196–203. Available from: https://pubmed.ncbi.nlm.nih.gov/29622441/

[15]. Sun GZ, Li Z, Guo L, Zhou Y, Yang HM, Sun YX. High prevalence of dyslipidemia and associated risk factors among rural Chinese adults. Lipids Health Dis [Internet]. 2014 Dec 12 [cited 2025 Jun 19];13(1):189. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC4292831/

[16]. Okafor AM, Ngwu EK, Ayogu RNB. Prevalence and associated factors of dyslipidaemia among university workers in Southeast Nigeria: a cross-sectional study. Archives of Public Health [Internet]. 2021 Dec 1 [cited 2025 Jun 19];79(1):1–9. Available from: https://archpublichealth.biomedcentral.com/articles/10.1186/s13690-021-00600-9

[17]. Ekeke N, Aniwada E, Chukwu J, Nwafor C, Meka A, Alphonsus C, et al. Screening diabetes mellitus patients for tuberculosis in southern nigeria: A pilot study. Adv Respir Med [Internet]. 2020 [cited 2025 Jun 19];88(1):6–12. Available from: https://pubmed.ncbi.nlm.nih.gov/32153002/

[18]. Emmanuel Chukwuemeka O, Rosita Chinechelum A, John Ekenedilichukwu O, Samuel Chukwuemeka M, Rosemary Adamma A, Christian Ejike O. Evaluation of dyslipidemia prevalence among undergraduate university students. J Clin Res Rep. 2022 Nov 21;12(1):01–7.

[19]. Baluku JB, Ronald O, Bagasha P, Okello E, Bongomin F. Prevalence of cardiovascular risk factors in active tuberculosis in Africa: a systematic review and meta-analysis. Sci Rep [Internet]. 2022 Dec 1 [cited 2025 Jun 19];12(1):1–14. Available from: https://www.nature.com/articles/s41598-022-20833-0
[20]. Kibirige D, Andia-Biraro I, Kyazze AP, Olum R, Bongomin F, Nakavuma RM, et al. Burden and associated phenotypic characteristics of tuberculosis infection in adult Africans with diabetes: a systematic review. Sci Rep. 2023 Dec 1;13(1).
[21]. Ferrara A, Barrett-Connor E, Shan J. Total, LDL, and HDL cholesterol decrease with age in older men and women. The Rancho Bernardo Study 1984–1994. Circulation. 1997;96(1):37–43.
[22]. Yusuf S, Reddy S, Ôunpuu S, Anand S. Global burden of cardiovascular diseases: part II: variations in cardiovascular disease by specific ethnic groups and geographic regions and prevention strategies. Circulation. 2001;104(23):2855–64.
[23]. BeLue R, Okoror TA, Iwelunmor J, Taylor KD, Degboe AN, Agyemang C, et al. An overview of cardiovascular risk factor burden in sub-Saharan African countries: a socio-cultural perspective. Global Health. 2009;5:10.
[24]. Kivimäki M, Steptoe A. Effects of stress on the development and progression of cardiovascular disease. Nat Rev Cardiol. 2018;15(4):215–29.
[25]. Vyas MV, Garg AX, Iansavichus AV, Costella J, Donner A, Laugsand LE, et al. Shift work and vascular events: systematic review and meta-analysis. BMJ. 2012;345:e4800.
[26]. Kontush A, Chapman MJ. Functionally defective high-density lipoprotein: a new therapeutic target at the crossroads of dyslipidemia, inflammation, and atherosclerosis. Pharmacol Rev. 2006;58(3):342–74.
[27]. Besler C, Luscher TF, Landmesser U. Molecular mechanisms of vascular effects of High-density lipoprotein: alterations in cardiovascular disease. EMBO Mol Med. 2012;4(4):251–68.
[28]. Chien YF, Chen YT, Hsueh PR, Yang PC, Yu CJ. High serum total cholesterol levels are associated with better outcomes in patients with bacteremia. Crit Care Med. 2005;33(8):1688–93.
[29]. Singh PP, Singh S. Hyperlipidemia and cardiovascular risk in South Asians. Can J Cardiol. 2015;31(11):1421–32.
[30]. Benn M, Nordestgaard BG, Grande P, Schnohr P, Tybjærg-Hansen A. PCSK9 R46L, low-density lipoprotein cholesterol levels, and risk of ischemic heart disease: 3 independent studies and meta-analyses. J Am Coll Cardiol. 2010;55(25):2833–42.
[31]. Wang Z, Klipfell E, Bennett BJ, Koeth R, Levison BS, Dugar B, et al. Gut flora metabolism of phosphatidylcholine promotes cardiovascular disease. Nature. 2011;472(7341):57–63.
[32]. Dutta NK, Bruiners N, Pinn ML, Zimmerman MD, Prideaux B, Dartois V, et al. Statin adjunctive therapy shortens the duration of TB treatment in mice. J Antimicrob Chemother. 2016;71(6):1570–7.
[33]. Lee MC, Lee CH, Lee MR, Shu CC, Wang JY, Lee LN, et al. The use of metformin is associated with a reduced risk of active tuberculosis: a systematic review with meta-analysis and dose–response analysis. Front Med. 2021;8:609466.
[34]. Yaqoob P, Pala HS, Cortina-Borja M, Newsholme EA, Calder PC. Encapsulated fish oil enriched in α-tocopherol alters plasma phospholipid and mononuclear cell fatty acid compositions but not mononuclear cell functions. Eur J Clin Invest. 2000;30(3):260–74