Journal of Cardiology and Cardiovascular Sciences

Mini Review Open Access

Non-fasting Remnant Cholesterol as an Independent Risk Factor for Atherosclerotic Vascular Disease

Reihane Taheri[1] and Spencer D Proctor[1]*

[1]Metabolic and Cardiovascular Diseases Laboratory, Division of Human Nutrition, University of Alberta, Edmonton, AB T6G 2E1, Canada.

Article Info

Article Notes
Received: May 18, 2026
Accepted: July 17, 2026

***Correspondence:**
Spencer D. Proctor, Metabolic and Cardiovascular Diseases Laboratory, Division of Human Nutrition, University of Alberta, 4-002E Li Ka Shing (LKS) Centre for Health Research Innovation, Edmonton, Alberta, Canada, T6G 2E1. Email: Spencer.proctor@ualberta.ca. Tel: +1 (780) 492-4672.

© 2026 Taheri R. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License.

Keywords:
Cardiovascular disease
Atherosclerosis
Non-fasting lipid
Remnant cholesterol
RC
Triglyceride rich lipoprotein
Cholesterol
Dyslipidemia
LDL

Abstract

Cardiovascular diseases (CVDs) remain the leading cause of mortality worldwide, with atherosclerosis as the primary underlying pathophysiology. Dyslipidemia is a well-established risk factor for atherosclerotic cardiovascular disease (ASCVD). While low-density lipoprotein cholesterol (LDL-C) has traditionally been considered as the primary atherogenic lipoprotein, growing evidence highlights the importance of non-fasting triglyceride-rich lipoproteins (TRLs) in cardiovascular risk. The premise for atherogenicity of non-fasting TRLs is associated with their relative composition of cholesterol as well as their proinflammatory profile within the arterial extracellular matrix. Non-fasting remnant cholesterol (RC) is representative of the cholesterol content of TRLs in post-prandial state and can include contributions from the very low-density lipoprotein (VLDL) and chylomicron synthesis pathways. Interestingly, while growing evidence has demonstrated that non-fasting RC is a significant independent risk factor for ASCVD (independent of LDL-C levels), clinical guidelines have not yet fully incorporated the adoption of RC assessment. Importantly, lifelong reductions in non-fasting RC exposure may meaningfully reduce ASCVD risk. Consequently, future guidelines may benefit from incorporating non-fasting RC screening and targeted interventions alongside traditional lipid measures.

Key Points

- Non-fasting remnant cholesterol (RC) represents the cholesterol content of very low-density lipoproteins and chylomicron remnant pathways.
- Studies support that non-fasting RC is a residual risk factor for atherosclerotic cardiovascular disease.
- Updated US Guidelines on the management of dyslipidemia (2026) include consideration of triglycerides and other atherogenic lipoproteins.
- Non-fasting measures of RC could improve lipid screening.

Introduction to Cardiovascular Diseases and the Role of Dyslipidemia

Cardiovascular diseases (CVDs) continue to be the primary cause of mortality worldwide (1). The main cause of CVD is atherosclerosis, a chronic inflammatory condition affecting arteries, marked by the accumulation of cholesterol, lipids, immune cells, and fibrous material within the intima (2). Fasting forms of dyslipidemia are well-established risk factors for atherosclerosis and are characterized by elevated low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and/or reduced high-density lipoprotein cholesterol (HDL-C) levels (3). Although clinical manifestations often emerge in midlife, it is important to appreciate that this process typically begins much

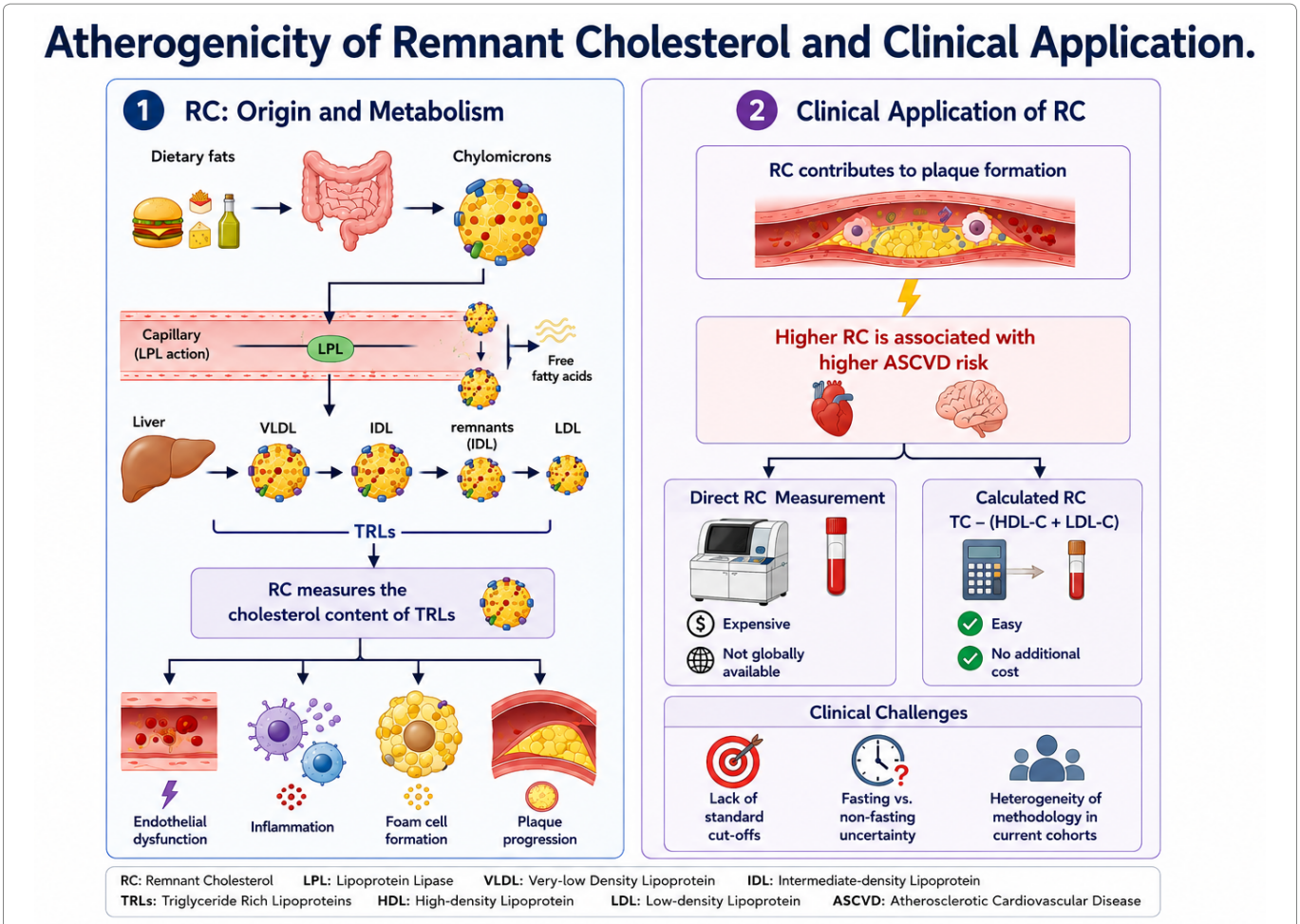

Figure 1. Atherogenicity of remnant cholesterol (RC) and its clinical application. (1) Origin and metabolism: dietary fats are processed into chylomicrons and hepatic VLDL, which undergo lipoprotein lipase (LPL) action to form IDL and LDL; RC reflects the cholesterol content of these triglyceride-rich lipoproteins (TRLs) and contributes to endothelial dysfunction, inflammation, foam cell formation, and plaque progression. (2) Clinical application: higher RC is associated with higher ASCVD risk, and can be assessed by direct measurement or calculated as TC − (HDL-C + LDL-C); clinical challenges include the lack of standardized cut-offs, fasting versus non-fasting uncertainty, and methodological heterogeneity across cohorts.

earlier, even during childhood, and offers an insightful perspective to better understand lifelong contributors of ASCVD risk (4).

Cholesterol from smaller, cholesterol-rich lipoproteins—such as chylomicron remnants, very low-density lipoprotein (VLDL) remnants, intermediate-density lipoproteins (IDL), and LDL—have been demonstrated to penetrate the arterial wall and accumulate (2). Persistent retention of these particles contributes to long term plaque development that can obstruct blood flow in smaller blood vessels, leading to conditions such as myocardial infarction (MI), transient ischemic attack and/or stroke (2, 5). Since LDL is the primary carrier of cholesterol, traditionally, fasting LDL-C has been viewed as the (sole) main contributor to cardiovascular risk (6). This has been underscored (particularly in North America) by the most recent revision of the Guidelines on the Management of Dyslipidemia by the American College of Cardiology/American Heart Association Joint Committee (ACC/AHA) in 2026 (7). However, with advances in understanding residual ASCVD risk, especially in Europe (8, 9), additional consideration to cholesterol carried by other small lipoproteins, particularly triglyceride-rich lipoproteins (TRLs), in the non-fasting state, became prominent.

Role of Triglyceride Rich Lipoproteins and Non-fasting Remnant Cholesterol in Atherosclerosis

TRLs are considered those derived during the non-fasting state from both the intestine and liver as chylomicrons/chylomicron-remnants and VLDL/VLDL-remnants respectively (10, 11). TRLs are inherently large when first secreted relative to other more dense lipoprotein particles, and in circulation become susceptible to lipolysis and become more cholesterol dense (10).

The role of TRLs in the development of ASCVD has a scientific history that can be dated to the 1970s when

Zilversmit proposed atherogenesis as a post prandial disease (12). At that time, we understood that lipoprotein particle size (>80 nm) (13) was the penultimate determining factor for atherogenesis (14). Advancements in our understanding of vascular permeability were made through the 1980–90s revealing that lipoprotein arterial permeability was inversely proportional to particle size (15, 16). However, it would not be until later that larger TRLs would be understood to also contribute to arterial cholesterol due to the formation of smaller remnant particles in vivo (17–19). Landmark imaging studies of arterial vessels enabled direct biochemical comparisons of remnant and LDL-C particles (20, 21). We now appreciate that non-fasting TRLs are sufficiently small to penetrate intact arterial endothelium (<80 nm) (10), and can accumulate in both pre-clinical models of atherosclerosis and human arterial vessels (15, 17, 19–23). Although TRLs have often been considered to be less cholesterol dense by % proportion, their larger size suggests that the absolute amount of their cholesterol mass remains significant for ASCVD (10). Therefore, TRLs play a significant role in atherogenesis, particularly in individuals with residual ASCVD risk (10, 11).

As a result, evaluating and managing TRLs has become increasingly important from a clinical perspective (24). Remnant cholesterol (RC) has been introduced as a measure of the cholesterol content within TRLs, which are VLDL and IDL in fasting state and CM remnants in non-fasting state (10). RC can be measured directly or calculated from standard lipid profiles using the formula: TC – (HDL-C + LDL-C) (4).

Association of Remnant Cholesterol and ASCVD Risk in Observational Studies

The clinical validity of non-fasting RC would not be fully appreciated without the Copenhagen City Heart Study (CCHS) (25, 26) and the Copenhagen General Population Study (CGPS) (27, 28). Evidence from these cohorts showed that non-fasting RC independently predicts cardiovascular risk, even when the LDL-C is in normal range in statin users (25, 29). The same association was also observed in 13 631 participants of the Canadian Alberta Tomorrow's Project (ATP) Cohort (30). Evidence from the UK Biobank study (31), Atherosclerosis Risk in Communities (ARIC) and Multi-Ethnic Study of Atherosclerosis (MESA) studies in USA (32), Kailuan (33) and ChinaHEART (34) studies in China, and National Health Insurance Service-Health Screening Cohort (NHIS-HEALS) in South Korea (35, 36) further supported the association of elevated RC and residual ASCVD risk across diverse populations. However, it should be acknowledged that the methodologies across these studies are not fully consistent. For instance, the CCHS, CGPS, and ATP cohorts used non-fasting serum samples to calculate RC, whereas the ARIC, MESA, Kailuan, ChinaHEART, and NHIS-HEALS cohorts used fasting serum samples.

A Mendelian Randomization (MR) study by Navarese EP, et al. (37) is one of the numerically powerful investigations to date, including 958 434 individuals from multiple international cohorts. The authors extracted 12 million single-nucleotide polymorphisms for RC and LDL-C from large-scale genome-wide association databases including the CARDIoGRAMplusC4D consortium (Coronary Artery Disease Genome-Wide Replication and Meta-Analysis Plus the Coronary Artery Disease Genetics), the Metastroke consortium, as well as the GLGC consortium (Global Lipids Genetics Consortium). By leveraging data from a combined sample of 958 434 participants, they reported a significant causal association between RC and the risk of CAD [odds ratio (OR): 1.51 per standard deviation (SD) unit increase in RC], MI (OR, 1.57 per SD unit increase in RC), as well as stroke (OR, 1.23 per SD unit increase in RC). The association remained consistent after accounting for the effects of RC associated genetic variants on LDL-C. This large-scale genetic study showed a robust causal association between RC and ASCVD and stroke across different populations and offers a substantial justification for the early screening of RC and the role of long-term therapeutic inhibition of high RC levels (37).

Beyond single RC measurements, cumulative RC, representing repeated measurements over time, has also emerged as a potentially stronger predictor of ASCVD (38, 39). This approach captures long-term exposure to TRLs and their cholesterol content, reflecting both dietary patterns and metabolic clearance efficiency (38, 40). These findings imply that long-term management of RC may be important for preventing ASCVD (38).

Association of Remnant Cholesterol and ASCVD Risk in Pediatrics

Early identification of either genetic or metabolic dyslipidemia in children and youth reduces future ASCVD risk. Our group previously showed that pediatrics at cardiometabolic risk may have normal fasting TC, non-HDL-C, and LDL-C but elevated non-fasting Apolipoprotein B-48 (ApoB48) and TG, reflecting TRLs metabolism abnormalities (41, 42). Pediatric guidelines increasingly support non-fasting non–high-density lipoprotein cholesterol (non-HDL-C) and Apolipoprotein B (ApoB) assessment to consider all atherogenic lipoproteins (4). Although studies on the association of RC and ASCVD in pediatric populations are limited, available evidence indicates that RC is associated with subclinical ASCVD risk and may be a stronger predictor than LDL-C (43–45).

Clinical Utility of Remnant Cholesterol

Several guidelines on adults and pediatrics, including the Canadian Cardiovascular Society (CCS) (46, 47), ACC/AHA (7, 48), and European Society of Cardiology/European Atherosclerosis Society (ESC/EAS) (8) incorporated

assessing non-fasting lipid profile for initial screening. In addition, they have recommended assessing either non-fasting non–HDL-C, or ApoB based on the clinician's preference (8, 46). European (49, 50), and American (51) epidemiological studies indicate that a significant proportion of individuals with dyslipidemia (from 8–33%) have discordant ApoB, non-HDL-C, or LDL-C. In these individuals, ApoB and RC are predictors of ASCVD and outperform both LDL-C and non-HDL-C (32, 50). These findings support the broader incorporation of ApoB and/or RC measurement into routine clinical practice. Since ApoB measurement is not globally feasible, calculating non-fasting RC might be a better approach for considering the residual risk of TRLs. However, most updated guidelines did not discuss assessing RC independently. It has been argued that, because non-HDL-C encompasses RC in addition to LDL-C, the separate assessment of RC may provide limited incremental value. However, a limitation of non–HDL-C is that a substantial proportion of its value is derived from LDL-C; therefore, changes in TRLs cholesterol may be less discernible, particularly in individuals with residual ASCVD risk characterized by discordance between RC and LDL-C (4, 30). Also, some evidence shows that RC is associated with ASCVD incidence even after adjusting for non-HDL-C (32). Therefore, RC may provide a more accurate reflection of TRLs cholesterol content relative to non-fasting non-HDL-C, without requiring additional tests or costs (25).

However, the clinical utility of RC is limited by several factors including variability in the definition of remnant lipoproteins in studies, the absence of standardized assays for RC measurement [Nuclear Magnetic Resonance (NMR) Spectroscopy, or Ultracentrifugation], high cost and limited availability of laboratory equipment globally for direct RC measurement, lack of harmonization for calculating RC, inconsistency in using fasting or non-fasting serum, and modest correlation of measured and calculated RC in patients with high TG (>4 mmol/L or 350 mg/dL) (52–55). In patients with high TG (>4 mmol/dL), the Friedewald equation and calculated RC are not accurate, and the Martin-Hopkins equation is used alternatively (53, 56). These limitations lead to non-equivalent values across studies, limited reproducibility, and poor comparability between investigations.

Another major challenge is the lack of globally standardized cut-off values. At present, most studies analyze RC as a continuous variable rather than applying uniform clinical thresholds. Although some cohorts have proposed categorical definitions or cut-offs—for example, ARIC and MESA (USA) using RC >0.6 mmol/L (24 mg/dL) (32), and the Kailuan study (China) (57) defining moderate risk as 0.5–1.0 mmol/L (19–30 mg/dL) and high risk as ≥1.0 mmol/L (~39 mg/dL), partly based on categorizations derived from the CGPS (58)—these thresholds were not established through standardized validation studies. Instead, they are largely based on study-specific approaches, including population percentiles (e.g., ≥95th percentile), LDL-C–derived distributions, and cohort-specific risk enrichment strategies. Consequently, what is considered a "high-risk" RC level appears to be context-dependent and varies across populations rather than reflecting a universally accepted global threshold. Future research should aim to establish standardized, validated cut-offs for RC across adult and pediatric populations, accounting for sex and ethnic differences. In addition, consensus on standardized measurement or calculation methods is needed to improve comparability across studies. Given that most existing evidence is based on calculated RC, harmonization of calculation approaches may further enhance consistency in research findings.

Interventions for Remnant Cholesterol Reduction

Currently, no clinical guidelines specifically recommend lifestyle interventions for the direct reduction of RC. However, guidelines advocate lifestyle modifications targeting TG and TRLs reduction, including weight management, limiting high-carbohydrate food intake, and regular physical activity (46, 59, 60). Evidence from clinical trials and epidemiological studies suggests that RC can be reduced by conventional lipid-lowering therapies, including statins, ezetimibe, and PCSK9 inhibitors (61, 62), as well as triglyceride-lowering agents such as omega-3 fatty acids and fibrates (63, 64). Notably, a portion of the reduction in ASCVD risk observed with statins, ezetimibe, and PCSK9 inhibitors has been attributed to decreases in RC in addition to LDL-C lowering (62). Furthermore, emerging therapies targeting TRLs metabolism, including ApoC-III and angiopoietin-like 3 protein (ANGPTL3) inhibitors, are currently under investigation for their potential to further reduce TRLs and RC levels (65).

Conclusion

In summary, a growing body of evidence supports a strong relationship between RC and ASCVD, independent of LDL-C and non-HDL-C. Lifelong reduction in RC exposure may meaningfully reduce ASCVD risk. Consequently, future guidelines may benefit from incorporating RC screening and targeted interventions alongside traditional lipid measures, particularly for identifying individuals with residual ASCVD risk or lipid discordance with LDL-C.

Declarations and Statements

Competing Interests

The authors declare that they have no competing interests.

Acknowledgements

The authors have no other acknowledgments to declare.

Funding

The concepts raised in this review have been supported in part by operating grants from the Health Outcomes Improvement Funds (Maternal, Newborn, Child and Youth Strategic Clinical Network (Alberta)), Women and Children's Health Research Institute (WCHRI) (Alberta), Heart and Stroke Foundation of Canada (HSFC), the Canadian Institute for Health Research (CIHR), and a scholarship from the Alberta Diabetes Institute (ADI).

Use of Artificial Intelligence

The schematic figure was generated using ChatGPT (GPT-5.5, reasoning mode) through an iterative and detailed prompt developed by the authors, and was subsequently reviewed and refined by the authors for scientific accuracy.

Disclosure

None.

References

1. Vaduganathan M, Mensah George A, Turco Justine V, Fuster V, Roth Gregory A. The Global Burden of Cardiovascular Diseases and Risk. Journal of the American College of Cardiology. 2022;80(25):2361–71.
2. Schaftenaar F, Frodermann V, Kuiper J, Lutgens E. Atherosclerosis: the interplay between lipids and immune cells. Current opinion in lipidology. 2016;27(3):209–15.
3. Perry W, Justin L, Amgad NM. Dyslipidemia and Its Role in the Pathogenesis of Atherosclerotic Cardiovascular Disease: Implications for Evaluation and Targets for Treatment of Dyslipidemia Based on Recent Guidelines. In: Samy IM, editor. Dyslipidemia. Rijeka: IntechOpen; 2019. p. Ch. 2.
4. Taheri R, Weaver O, Krysa JA, Vine DF, Khoury M, Proctor SD. Lipid Screening for Cardiovascular Risk in Youth: A Narrative Review of Current Guidelines and Potential Use of Nonfasting Measures. CJC Pediatric and Congenital Heart Disease. 2025.
5. Li Y, Zhu H, Zhang Q, Han X, Zhang Z, Shen L, et al. Smooth muscle-derived macrophage-like cells contribute to multiple cell lineages in the atherosclerotic plaque. Cell Discovery. 2021;7(1):111.
6. Borén J, Chapman MJ, Krauss RM, Packard CJ, Bentzon JF, Binder CJ, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease: pathophysiological, genetic, and therapeutic insights: a consensus statement from the European Atherosclerosis Society Consensus Panel. Eur Heart J. 2020;41(24):2313–30.
7. Blumenthal RS, Morris PB, Gaudino M, Johnson HM, Anderson TS, Bittner VA, et al. 2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Dyslipidemia: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol. 2026;87(19):2624–757.
8. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk: The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS). European Heart Journal. 2020;41(1):111–88.
9. Mach F, Koskinas KC, Roeters van Lennep JE, Tokgözoğlu L, Badimon L, Baigent C, et al. 2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias: Developed by the task force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). European Heart Journal. 2025;46(42):4359–78.
10. Heo JH, Jo SH. Triglyceride-Rich Lipoproteins and Remnant Cholesterol in Cardiovascular Disease. Journal of Korean medical science. 2023;38(38):e295.
11. Ginsberg HN, Packard CJ, Chapman MJ, Borén J, Aguilar-Salinas CA, Averna M, et al. Triglyceride-rich lipoproteins and their remnants: metabolic insights, role in atherosclerotic cardiovascular disease, and emerging therapeutic strategies—a consensus statement from the European Atherosclerosis Society. European Heart Journal. 2021;42(47):4791–806.
12. Zilversmit DB. Atherogenesis: a postprandial phenomenon. Circulation. 1979;60(3):473–85.
13. Simionescu N. Cellular aspects of transcapillary exchange. Physiol Rev. 1983;63(4):1536–79.
14. Ross R. George Lyman Duff Memorial Lecture. Atherosclerosis: a problem of the biology of arterial wall cells and their interactions with blood components. Arteriosclerosis. 1981;1(5):293–311.
15. Nordestgaard BG, Zilversmit DB. Large lipoproteins are excluded from the arterial wall in diabetic cholesterol-fed rabbits. J Lipid Res. 1988;29(11):1491–500.
16. Nordestgaard BG, Tybjaerg-Hansen A. IDL, VLDL, chylomicrons and atherosclerosis. Eur J Epidemiol. 1992;8 Suppl 1:92–8.
17. Mamo JC, Wheeler JR. Chylomicrons or their remnants penetrate rabbit thoracic aorta as efficiently as do smaller macromolecules, including low-density lipoprotein, high-density lipoprotein, and albumin. Coron Artery Dis. 1994;5(8):695–705.
18. Proctor SD, Mamo JC. Arterial fatty lesions have increased uptake of chylomicron remnants but not low-density lipoproteins. Coron Artery Dis. 1996;7(3):239–45.
19. Proctor SD, Mamo JC. Retention of fluorescent-labelled chylomicron remnants within the intima of the arterial wall—evidence that plaque cholesterol may be derived from post-prandial lipoproteins. Eur J Clin Invest. 1998;28(6):497–503.
20. Proctor SD, Mamo JCL. Intimal Retention of Cholesterol Derived From Apolipoprotein B100– and Apolipoprotein B48–Containing Lipoproteins in Carotid Arteries of Watanabe Heritable Hyperlipidemic Rabbits. Arteriosclerosis, Thrombosis, and Vascular Biology. 2003;23(9):1595–600.
21. Proctor SD, Vine DF, Mamo JC. Arterial permeability and efflux of apolipoprotein B-containing lipoproteins assessed by in situ perfusion and three-dimensional quantitative confocal microscopy. Arterioscler Thromb Vasc Biol. 2004;24(11):2162–7.
22. Rapp JH, Harris HW, Hamilton RL, Krupski WC, Reilly LM, Ehrenfeld WK, et al. Particle size distribution of lipoproteins from human atherosclerotic plaque: a preliminary report. J Vasc Surg. 1989;9(1):81–8.
23. Wadström BN, Wulff AB, Pedersen KM, Nordestgaard BG. Small Remnants versus Large Triglyceride-Rich Lipoproteins in Risk of Atherosclerotic Cardiovascular Disease. Clinical Chemistry. 2025;71(4):463–73.
24. Ginsberg HN, Packard CJ, Chapman MJ, Borén J, Aguilar-Salinas CA, Averna M, et al. Triglyceride-rich lipoproteins and their remnants: metabolic insights, role in atherosclerotic cardiovascular disease, and emerging therapeutic strategies-a consensus statement from the European Atherosclerosis Society. Eur Heart J. 2021;42(47):4791–806.
25. Varbo A, Benn M, Tybjærg-Hansen A, Jørgensen AB, Frikke-Schmidt R, Nordestgaard BG. Remnant cholesterol as a causal risk factor for ischemic heart disease. J Am Coll Cardiol. 2013;61(4):427–36.

26. Aguib Y, Al Suwaidi J. The Copenhagen City Heart Study (Østerbrundersøgelsen). Glob Cardiol Sci Pract. 2015;2015(3):33.

27. Varbo A, Nordestgaard BG. Remnant cholesterol and risk of ischemic stroke in 112,512 individuals from the general population. Annals of Neurology. 2019;85(4):550–9.

28. Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. Jama. 2007;298(3):299–308.

29. Varbo A, Benn M, Tybjærg-Hansen A, Nordestgaard BG. Elevated remnant cholesterol causes both low-grade inflammation and ischemic heart disease, whereas elevated low-density lipoprotein cholesterol causes ischemic heart disease without inflammation. Circulation. 2013;128(12):1298–309.

30. Weaver OR, Ye M, Vena JE, Eurich DT, Proctor SD. Non-fasting lipids and cardiovascular disease in those with and without diabetes in Alberta's Tomorrow Project: A prospective cohort study. Diabetic medicine: a journal of the British Diabetic Association. 2023:e15133.

31. Pilsgaard F, Emanuelsson F, Vedel-Krogh S, Nordestgaard BG, Kobylecki CJ. Elevated remnant cholesterol, but not LDL cholesterol, explains part of recurrent ASCVD events in individuals with and without impaired renal function: the Copenhagen General Population Study and the UK Biobank. Atherosclerosis. 2026;418:120798.

32. Quispe R, Martin SS, Michos ED, Lamba I, Blumenthal RS, Saeed A, et al. Remnant cholesterol predicts cardiovascular disease beyond LDL and ApoB: a primary prevention study. Eur Heart J. 2021;42(42):4324–32.

33. Zhang Y, Xu Q, Tian X, Xia X, Chen S, Liu F, et al. Longitudinal changes in remnant cholesterol and the risk of cardiovascular disease. Cardiovascular Diabetology. 2025;24(1):1.

34. Tian Y, Wu Y, Qi M, Song L, Chen B, Wang C, et al. Associations of remnant cholesterol with cardiovascular and cancer mortality in a nationwide cohort. Science Bulletin. 2024;69(4):526–34.

35. Huh JH, Han KD, Cho YK, Roh E, Kang JG, Lee SJ, et al. Remnant cholesterol and the risk of cardiovascular disease in type 2 diabetes: a nationwide longitudinal cohort study. Cardiovasc Diabetol. 2022;21(1):228.

36. Lee SJ, Kim S-E, Go T-H, Kang DR, Jeon H-S, Kim Y-I, et al. Remnant cholesterol, low-density lipoprotein cholesterol, and incident cardiovascular disease among Koreans: a national population-based study. European Journal of Preventive Cardiology. 2023;30(11):1142–50.

37. Navarese EP, Vine D, Proctor S, Grzelakowska K, Berti S, Kubica J, et al. Independent Causal Effect of Remnant Cholesterol on Atherosclerotic Cardiovascular Outcomes: A Mendelian Randomization Study. Arterioscler Thromb Vasc Biol. 2023;43(9):e373–e80.

38. Proctor SD, Wang M, Vine DF, Raggi P. Predictive utility of remnant cholesterol in atherosclerotic cardiovascular disease. Current opinion in cardiology. 2024.

39. Dai Y, Sheng Y, Zheng Z, Di W. Cumulative remnant cholesterol exposure during young adulthood and middle age and risk of cardiovascular events: A 30-year population-based cohort study. International Journal of Cardiology. 2024;414:132435.

40. Ference BA, Graham I, Tokgozoglu L, Catapano AL. Impact of lipids on cardiovascular health: JACC health promotion series. Journal of the American College of Cardiology. 2018;72(10):1141–56.

41. Krysa JA, Vine DF, Beilin LJ, Burrows S, Huang RC, Mori TA, et al. ApoB48-remnant lipoproteins are associated with increased cardiometabolic risk in adolescents. Atherosclerosis. 2020;302:20–6.

42. Wang Y, Pendlebury C, Dodd MM, Maximova K, Vine DF, Jetha MM, et al. Elevated remnant lipoproteins may increase subclinical CVD risk in pre-pubertal children with obesity: a case-control study. Pediatr Obes. 2013;8(5):376–84.

43. Di Costanzo A, Perla FM, D'Erasmo L, Arca M, Chiesa C, Pacifico L. Elevated Serum Concentrations of Remnant Cholesterol Associate with Increased Carotid Intima-Media Thickness in Children and Adolescents. The Journal of Pediatrics. 2021;232:133–9.e1.

44. Torres-Costoso A, Martínez-Vizcaíno V, Oliveira A, Sánchez-López M, Rodríguez-Gutiérrez E, de Arenas-Arroyo SN, et al. Beyond LDL cholesterol: remnant cholesterol is associated with cardiometabolic risk factors in children. BMC Med. 2025;23(1):28.

45. Wilke MS, Maximova K, Henderson M, Levy E, Paradis G, O'Loughlin J, et al. Adiposity in Children and CVD Risk: ApoB48 Has a Stronger Association With Central Fat Than Classic Lipid Markers. The Journal of Clinical Endocrinology & Metabolism. 2016;101(7):2915–22.

46. Pearson GJ, Thanassoulis G, Anderson TJ, Barry AR, Couture P, Dayan N, et al. 2021 Canadian Cardiovascular Society Guidelines for the Management of Dyslipidemia for the Prevention of Cardiovascular Disease in Adults. Canadian Journal of Cardiology. 2021;37(8):1129–50.

47. Khoury M, Bigras J-L, Cummings EA, Harris KC, Hegele RA, Henderson M, et al. The Detection, Evaluation, and Management of Dyslipidemia in Children and Adolescents: A Canadian Cardiovascular Society/Canadian Pediatric Cardiology Association Clinical Practice Update. Canadian Journal of Cardiology. 2022;38(8):1168–79.

48. Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019;139(25):e1082–e143.

49. Sniderman AD, Dufresne L, Pencina KM, Bilgic S, Thanassoulis G, Pencina MJ. Discordance among apoB, non-high-density lipoprotein cholesterol, and triglycerides: implications for cardiovascular prevention. Eur Heart J. 2024;45(27):2410–8.

50. De Oliveira-Gomes D, Joshi PH, Peterson ED, Rohatgi A, Khera A, Navar AM. Apolipoprotein B: Bridging the Gap Between Evidence and Clinical Practice. Circulation. 2024;150(1):62–79.

51. Degoma EM, Davis MD, Dunbar RL, Mohler ER 3rd, Greenland P, French B. Discordance between non-HDL-cholesterol and LDL-particle measurements: results from the Multi-Ethnic Study of Atherosclerosis. Atherosclerosis. 2013;229(2):517–23.

52. Chen J, Kuang J, Tang X, Mao L, Guo X, Luo Q, et al. Comparison of calculated remnant lipoprotein cholesterol levels with levels directly measured by nuclear magnetic resonance. Lipids Health Dis. 2020;19(1):132.

53. DuBroff R, Byrne P. Challenges in defining, measuring, and calculating remnant cholesterol. European Journal of Preventive Cardiology. 2025.

54. Stürzebecher PE, Katzmann JL, Laufs U. What is 'remnant cholesterol'? European Heart Journal. 2023;44(16):1446–8.

55. Wang L, Zhang Q, Wu Z, Huang X. A significant presence in atherosclerotic cardiovascular disease: Remnant cholesterol: A review. Medicine (Baltimore). 2024;103(27):e38754.

56. Wadström BN, Wulff AB, Pedersen KM, Jensen GB, Nordestgaard BG. Elevated remnant cholesterol increases the risk of peripheral artery disease, myocardial infarction, and ischaemic stroke: a cohort-based study. European Heart Journal. 2022;43(34):3258–69.

57. Zheng H, Chen G, Huo Z, Lan Y, Wang Y, Fu P, et al. Remnant Cholesterol and Atherosclerotic Cardiovascular Disease Risk in Populations With Different Low-Density Lipoprotein Cholesterol Elevations: A Prospective Cohort Study. J Am Heart Assoc. 2026;15(4):e045376.

58. Riis J, Nordestgaard BG, Afzal S. High remnant cholesterol and atherosclerotic cardiovascular disease in healthy women and men aged 70–100. European Journal of Preventive Cardiology. 2025;32(13):1169–77.

59. Visseren FLJ, Mach F, Smulders YM, Carballo D, Koskinas KC, Bäck M, et al. 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice. Eur J Prev Cardiol. 2022;29(1):5–115.

60. Writing Committee M, Blumenthal RS, Morris PB, Gaudino M, Johnson HM, Anderson TS, et al. 2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Dyslipidemia: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2026;153(17):e1154–e276.

61. Balling M, Roepstorff OG, Gerds TA, Varbo A, Mortensen MB, Langsted A, et al. Risk reduction of ASCVD attributed to lowering of remnant cholesterol from statins, fibrates, APOC3 inhibitors, and ANGPTL3 inhibitors: a cohort study. Atherosclerosis. 2025;409:120471.

62. Nordestgaard AT, Nordestgaard BG. Remnant cholesterol lowering in cardiovascular disease risk reduction in statin, ezetimibe, and PCSK9 inhibitor trials: meta-regression analyses. European Journal of Preventive Cardiology. 2025.

63. Ballantyne CM, Bays HE, Philip S, Doyle RT Jr, Braeckman RA, Stirtan WG, et al. Icosapent ethyl (eicosapentaenoic acid ethyl ester): Effects on remnant-like particle cholesterol from the MARINE and ANCHOR studies. Atherosclerosis. 2016;253:81–7.

64. Tsunoda F, Asztalos IB, Horvath KV, Steiner G, Schaefer EJ, Asztalos BF. Fenofibrate, HDL, and cardiovascular disease in Type-2 diabetes: The DAIS trial. Atherosclerosis. 2016;247:35–9.

65. Rosenson RS, Shaik A, Song W. New Therapies for Lowering Triglyceride-Rich Lipoproteins. JACC. 2021;78(18):1817–30.