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ARTICLE

Frequency of low-density-lipoprotein-cholesterol measurement and risk of major adverse cardiovascular outcomes: a 5-million-person nationwide cohort study

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Abstract

Cardiovascular disease (CVD) is a leading cause of mortality globally and in the UK, with significant efforts focused on early risk identification and prevention. Elevated low-density lipoprotein cholesterol (LDL-C) is a key modifiable risk factor for cardiovascular disease (CVD), yet the impact of LDL-C monitoring frequency on major adverse cardiovascular events (MACE) remains unclear. This study evaluated the relationship between LDL-C measurement frequency and the risk of MACE in a large, nationwide cohort. A retrospective cohort study using anonymised electronic health records from the Clinical Practice Research Datalink (CPRD) Aurum, linked to hospitalisation, social deprivation, and mortality data. The cohort included individuals registered for at least one year with at least one recorded LDL-C measurement between 1 January 2000 and 31 December 2022. The primary outcome was MACE, defined as a composite of non-fatal coronary heart disease, non-fatal stroke, or cardiovascular death. Multivariable Cox proportional hazards models and Kaplan-Meier survival plot were used to estimate the incidence and hazard ratios (HRs) by LDL-C monitoring frequency. The study cohort comprised 5,133,574 individuals, with 2,733,144 (53.2%) being women. The median follow-up duration was 3.31 years (IQR: 7.39-12.11). Among the 5,133,574 individuals, the incidence of MACE declined with more frequent LDL-C monitoring, from 1937.0 (95% CI: 1928.2-1945.8) events per 100,000 person-years (one measurement) to 1615.4 (95% CI: 1605.8-1625.0), 1484.6 (95% CI: 1473.7-1495.6), and 1204.9 (95% CI: 1200.1-1209.6) for those with two, three, and four or more measurements, respectively. Compared to individuals with one LDL-C measurement, the adjusted HRs for MACE were 0.703 (95% CI: 0.698-0.709), for two measurements, 0.570 (95% CI: 0.565-0.575) for three, and 0.312 (95% CI: 0.310-0.314) for four or more. The Kaplan-Meier curve demonstrated improved event-free survival with increased LDL-C monitoring (log-rank p<0.0001). More frequent LDL-C monitoring was associated with a lower risk of MACE. These findings highlight the potential benefits of regular LDL-C monitoring as a potentially impactful strategy for CVD prevention and long-term risk management.

Key words: low-density lipoprotein cholesterol; MACE; cardiovascular disease; cohort data.

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Introduction

Globally, cardiovascular disease (CVD) is one of the leading causes of mortality, accounting for 32% of all deaths.[1] In the United Kingdom (UK) CVD is the second most common cause of mortality, accounting for 27% of all deaths.[2] Guidelines on the prevention and management of CVD continue to evolve, with early identification and monitoring of modifiable risk factors playing a critical role in reducing long-term cardiovascular risk.[3] Elevated levels of low-density lipoprotein cholesterol (LDL-C) are a well-established cause for atherosclerotic CVD,[4,5] and evidence from multiple randomised controlled trials (RCTs) have

shown that lowering LDL-C levels with lipid-lowering therapies significantly reduces the risk of cardiovascular events.[5,6]

The UK National Institute for Health and Care Excellence (NICE) guidelines recommend an initial full lipid profile to estimate an individual's risk of CVD, followed by reassessment at three months and annually thereafter for individuals at high risk and initiated on lipid-lowering medication.[7] LDL-C has become a key biomarker for CVD risk stratification and treatment decisions,[7–9] including the initiation and intensification of established lipid-lowering therapies such as statins.

While the role of LDL-C levels in CVD risk is well established, and the frequency of lipid monitoring has been linked to treatment changes,[10-13] less is known about how the frequency of LDL-C measurement may influence long-term cardiovascular outcomes, including major adverse cardiovascular events (MACE).[14] An understanding of the relationship with MACE risk can inform strategies to optimise prevention and treatment pathways for CVD.

This study aimed to evaluate the relationship between the frequency of LDL-C measurements over time and the risk of MACE in a large, nationwide cohort using routinely collected electronic health records in the UK.

Methods

Study design and data

This retrospective cohort study used anonymised electronic health records from the Clinical Practice Research Datalink (CPRD) AURUM datasets. CPRD contains information on about 20% of the UK population and is broadly representative of age, sex, ethnicity, geographical spread, and socioeconomic deprivation. It is one of the largest databases of longitudinal medical records from primary care in the world and has been validated for epidemiological research for a wide range of conditions.[15] We used the subset of CPRD records that linked information from primary care, secondary care from Hospital Episodes Statistics (HES admitted patient care) data, social deprivation data, and death records from the Office for National Statistics (ONS) (Figure 1).

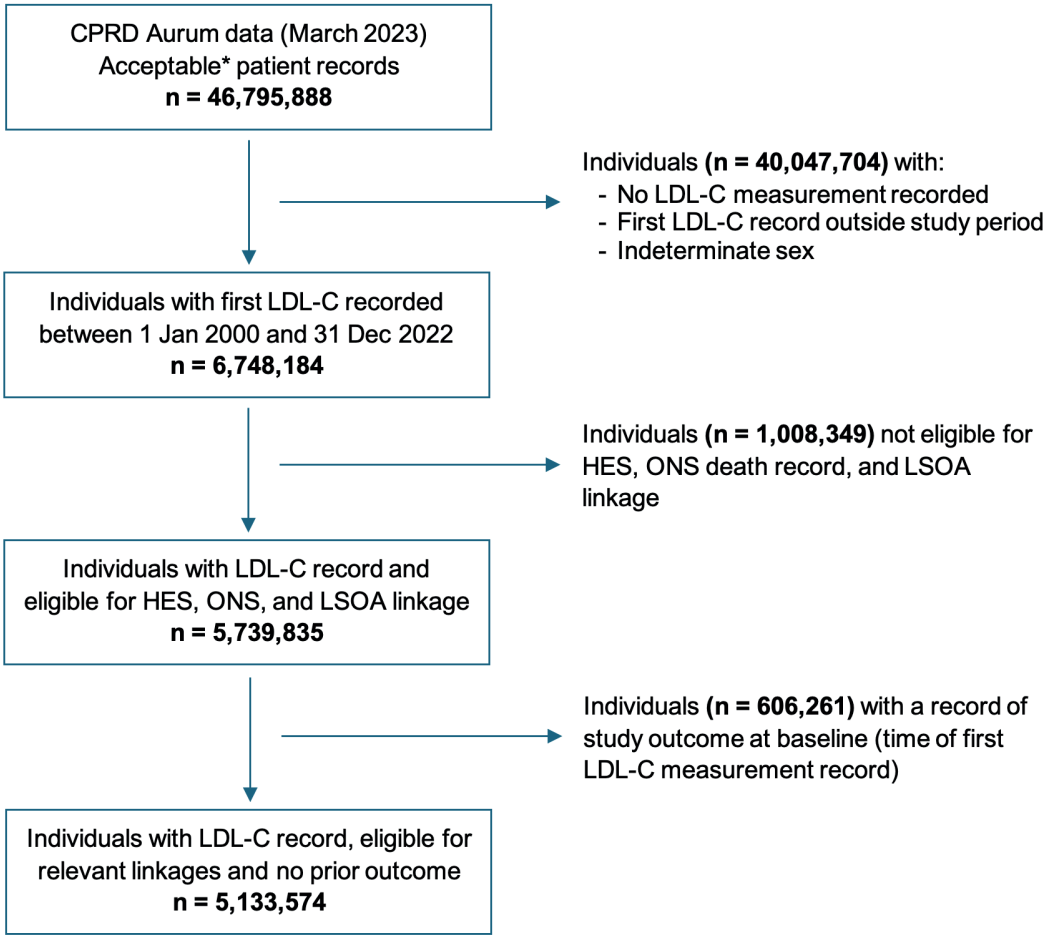

Figure 1. Study flow diagram. CPRD, Clinical Practice Research Datalink; HES, hospital episode statistics; LDL-C, low-density lipoprotein cholesterol; LSOA, layer super output area; ONS, Office of National Statistics.

Study population

The study included individuals registered with a general practice for at least one year, with at least one low-density lipoprotein cholesterol measurement recorded during the study period (1 January 2000 to 31 December 2022) and whose records were classified by CPRD as acceptable for use in research and eligible for HES and ONS linkage.

Exposure

The primary exposure variable was the frequency of low-density lipoprotein cholesterol (LDL-C) measurements recorded for each individual during their follow-up period. This was categorised into groups: one, two, three, and four or more LDL-C measurements.

Outcome

Major adverse cardiovascular event (MACE) was defined as a composite of non-fatal coronary heart disease, non-fatal stroke, or cardiovascular disease-related death.

Statistical analyses

Descriptive statistics were reported as absolute values (counts) and relative frequencies (percentages). For non-normally distributed data, medians and interquartile ranges (IQRs) were reported, while means and 95% confidence intervals (CIs) were used for normally distributed data. Multivariable Cox proportional hazards models were used, with Kaplan-Meier curves to determine the incidence rates and hazard ratios. All analyses were performed using Stata version 18.5.

Results

There was a total of 5,133,574 individuals with at least one LDL-C measurement recorded that met the study eligibility criteria. Of these, 39.3% had only one LDL-C measurement, 17.7% had two, 10.4% had three, and 32.5% had four or more recordings. Females made up a slightly higher proportion of the cohort (53.2%) compared to males (46.8%). The median age at the time of the first LDL-C record increased with the number of measurements, from 36 years (IQR: 46-58) among those with one record to 47 years (IQR: 56-65) among those with four or more. Similarly, median follow-up time rose with the number of LDL-C measurements, ranging from 1.33 years (IQR: 3.53-7.12) for individuals with one record to 8.70 years (IQR: 12.37-15.80) for those with four or more. Table 1 and Table 2 summarise the characteristics and prevalence of comorbid conditions and prescribed medications of individuals included in the study, respectively. As shown in Table 3, an inverse relationship was observed between the frequency of LDL-C (low-density lipoprotein cholesterol) recordings and the incidence of major adverse cardiovascular events (MACE). Individuals with only one LDL-C record had the highest incidence rate at 1937.0 events per 100,000 person-years. In contrast, those with two, three, and four or more LDL-C measurements experienced progressively lower incidence rates of 1615.4, 1484.6, and 1204.9, respectively. This pattern was also reflected in both unadjusted and adjusted hazard ratios. Compared to individuals with just one LDL-C measurement (the reference group), those with two LDL-C records had an adjusted hazard ratio (HR) of 0.703 (95% CI: 0.698-0.709), indicating a 30% lower risk of MACE. The adjusted HR decreased further to 0.570 (95% CI: 0.565-0.575) for those with three LDL-C recordings, representing a 43% reduction in risk of MACE. The most substantial risk reduction was observed among individuals with four or more LDL-C measurements, who had an adjusted HR of 0.312 (95% CI: 0.310-0.314), equating to a 69% lower risk of a major adverse cardiovascular event compared to the reference group. The Kaplan-Meier survival analysis Figure 2 further supported the observed relationship between the frequency of LDL-C measurements and the risk of major adverse cardiovascular events (MACE). Survival curves diverged early and remained consistently separated over the 20-year follow-up period, with individuals who had only one LDL-C measurement demonstrating the lowest cumulative survival. In contrast, those with four or more LDL-C recordings had the highest survival probabilities throughout the follow-up. The log-rank test confirmed these differences were statistically significant ($p<0.0001$). These findings align with the incidence and hazard ratio analyses, showing that more frequent LDL-C monitoring is associated with substantially lower long-term cardiovascular events risk.

Discussion

In this large nationwide cohort study, we found an association between the frequency of LDL-C measurements and the risk of MACE. Individuals with more frequent LDL-C testing had significantly lower incidence rates and risk of MACE. Individuals with four or more LDL-C measurements had a 69% lower risk of MACE outcome compared to those with only one measurement, highlighting the potential importance of regular lipid monitoring in CVD reduction.

These findings are consistent with existing evidence that has shown the increased frequency of lipid measurements is associated with CVD risk-lowering strategies, such as therapy initiation and intensification. Patients with two or more lipid measurements were significantly more likely to undergo medication intensification (adjusted ORs: 4.37[16] and 1.51[17]), while those with at least one lipid measurement were more likely to initiate statin therapy compared to individuals with no lipid testing.[16] Frequent lipid testing has also been linked with better adherence to treatment[17] and improved clinical outcomes in individuals with established CVD.[18] Additionally, the Patient and Provider Assessment of Lipid Management (PALM) registry supports these associations, showing that lipid monitor-

ing facilitates goal-directed therapy through personalised treatment adjustments.[19] In our study, lipid-lowering therapy use increased with LDL-C monitoring frequency, from 3.6% among those with one LDL-C to 9.7% among those with four or more.

Furthermore, studies from both US and European cohorts have shown that structured lipid testing programmes are associated with lower cardiovascular event rates and greater statin adherence.[16,20] The findings of this study highlight the potential clinical importance of regular LDL-C monitoring as part of cardiovascular disease prevention strategies. Regular lipid measurements may facilitate the timely initiation and intensification of lipid-lowering therapies, thereby reducing the risk of adverse cardiovascular outcomes. The observed graded reduction in MACE risk with increasing frequency of LDL-C testing suggests that more frequent lipid assessments could serve as a marker of proactive cardiovascular care and may be a modifiable component of risk reduction pathways. These findings support the integration of routine LDL-C monitoring into clinical guidelines and highlight the need for healthcare systems to promote consistent follow-up and monitoring practices.

To our knowledge, this is the first large-scale study to directly evaluate the association between LDL-C monitoring frequency and the risk of MACE. Strengths of this study include the use of a high-quality, nationally representative dataset, linkage to hospital and mortality data, and robust statistical adjustment for key confounders.[15] However, several limitations should be

Table 1. Characteristics of the study cohort with low-density lipoprotein cholesterol (LDL-C) measurement recorded (n=5,133,574).

	Categories for the total number of LDL-C measurements recorded over the follow-up period				
	1	2	3	4 or more	Total
	2,020,018 (39.3%)	910,847 (17.7%)	534,760 (10.4%)	1,667,949 (32.5%)	5,133,574 (100.0%)
Age at time of first LDL-C record	36 (46 - 58)	41 (50 - 62)	43 (52 - 63)	47 (56 - 65)	41 (51 - 62)
Follow-up in years (median, IQR)	1.33 (3.53 - 7.12)	3.51 (6.64 - 10.31)	5.08 (8.46 - 12.08)	8.70 (12.37 - 15.80)	3.31 (7.39 - 12.11)
Ethnicity					
White	1,584,555 (78.4%)	738,941 (81.1%)	439,043 (82.1%)	1,376,997 (82.6%)	4,139,536 (80.6%)
Asian	141,325 (7.0%)	61,105 (6.7%)	35,588 (6.7%)	122,287 (7.3%)	360,305 (7.0%)
Black	77,985 (3.9%)	33,888 (3.7%)	20,058 (3.8%)	66,262 (4.0%)	198,193 (3.9%)
Mixed	25,538 (1.3%)	9,745 (1.1%)	5,179 (1.0%)	14,947 (0.9%)	55,409 (1.1%)
Other	27,923 (1.4%)	10,248 (1.1%)	5,509 (1.0%)	14,368 (0.9%)	58,048 (1.1%)
Unknown	162,692 (8.1%)	56,920 (6.2%)	29,383 (5.5%)	73,088 (4.4%)	322,083 (6.3%)
2019 index of multiple deprivation					
1-Least deprived	381,742 (18.9%)	175,987 (19.3%)	101,186 (18.9%)	283,240 (17.0%)	942,155 (18.4%)
2	375,989 (18.6%)	170,280 (18.7%)	98,621 (18.4%)	286,662 (17.2%)	931,552 (18.1%)
3	358,436 (17.7%)	155,628 (17.1%)	89,057 (16.7%)	257,899 (15.5%)	861,020 (16.8%)
4	373,679 (18.5%)	158,425 (17.4%)	90,963 (17.0%)	273,041 (16.4%)	896,108 (17.5%)
5-Most deprived	336,178 (16.6%)	144,232 (15.8%)	83,833 (15.7%)	254,537 (15.3%)	818,780 (15.9%)
Unknown	193,994 (9.6%)	106,295 (11.7%)	71,100 (13.3%)	312,570 (18.7%)	683,959 (13.3%)
Sex					
Male	957,126 (47.4%)	424,471 (46.6%)	247,325 (46.2%)	771,508 (46.3%)	2,400,430 (46.8%)
Female	1,062,892 (52.6%)	486,376 (53.4%)	287,435 (53.8%)	896,441 (53.7%)	2,733,144 (53.2%)
Current smoker status	260,658 (14.0%)	117,890 (13.6%)	67,865 (13.2%)	186,322 (11.5%)	632,735 (13.0%)
Biomarkers at baseline (median with IQR)					
Diastolic blood pressure, mmHg	70 (78 - 82)	70 (80 - 84)	70 (80 - 85)	74 (80 - 88)	70 (80 - 85)
Systolic blood pressure, mmHg	113 (124 - 136)	115 (127 - 140)	118 (130 - 140)	120 (132 - 146)	118 (130 - 140)
Body mass index, kg/m2	23.3 (26.6 - 30.8)	23.8 (27.1 - 31.2)	24.2 (27.4 - 31.4)	24.9 (28.0 - 32.0)	24.0 (27.3 - 31.4)
Glycated haemoglobin level, mmol/mol	34.00 (38.00 - 43.00)	35.00 (39.89 - 49.73)	36.00 (42.00 - 54.10)	39.89 (49.73 - 61.75)	35.00 (40.98 - 53.01)
Total cholesterol level, mmol/L	4.40 (5.10 - 5.80)	4.60 (5.30 - 6.00)	4.70 (5.40 - 6.10)	4.80 (5.60 - 6.34)	4.60 (5.30 - 6.10)
HDL cholesterol level, mmol/L	1.16 (1.40 - 1.70)	1.16 (1.40 - 1.70)	1.16 (1.40 - 1.70)	1.13 (1.40 - 1.69)	1.15 (1.40 - 1.70)
Non-HDL cholesterol level, mmol/L	2.80 (3.48 - 4.20)	2.90 (3.61 - 4.40)	3.02 (3.80 - 4.60)	3.20 (3.92 - 4.80)	2.89 (3.50 - 4.30)
Triglyceride level, mmol/L	0.80 (1.20 - 1.70)	0.88 (1.20 - 1.80)	0.90 (1.28 - 1.81)	1.00 (1.40 - 2.00)	0.90 (1.27 - 1.82)
Missing values (n, %)					
Diastolic blood pressure	992,023 (49.1%)	446,990 (49.1%)	266,348 (49.8%)	854,897 (51.3%)	2,560,258 (49.9%)
Systolic blood pressure	991,266 (49.1%)	446,523 (49.0%)	266,009 (49.7%)	853,242 (51.2%)	2,557,040 (49.8%)
Body mass index	1,201,429 (59.5%)	522,122 (57.3%)	296,082 (55.4%)	867,623 (52.0%)	2,887,256 (56.2%)
Glycated haemoglobin level	1,836,462 (90.9%)	839,203 (92.1%)	496,258 (92.8%)	1,527,178 (91.6%)	4,699,101 (91.5%)
Total cholesterol level	4,344 (0.2%)	2,472 (0.3%)	1,799 (0.3%)	9,387 (0.6%)	18,002 (0.4%)
HDL cholesterol level	6,792 (0.3%)	3,636 (0.4%)	2,392 (0.4%)	13,175 (0.8%)	25,995 (0.5%)
Non-HDL cholesterol level	1,452,404 (71.9%)	759,523 (83.4%)	479,322 (89.6%)	1,617,915 (97.0%)	4,309,164 (83.9%)
Triglyceride level	22,676 (1.1%)	10,478 (1.2%)	6,448 (1.2%)	27,486 (1.6%)	67,088 (1.3%)

acknowledged. As with all observational studies using routine clinical data, residual confounding, incomplete data, and potential misclassification cannot be fully ruled out. LDL-C testing and MACE events may not have been uniformly captured, introducing possible ascertainment and information bias. Additionally, reverse causality is a potential limitation of the study. Patients at higher cardiovascular risk may have undergone more frequent LDL-C testing, for example due to rising lipid

Table 2. Baseline prevalence of comorbidities and medication prescriptions in the study cohort (n=5,133,574).

	Categories for the total number of LDL-C measurements recorded over the follow-up period				
	1	2	3	4 or more	Total
Presence of comorbid condition at baseline (n, %)					
Alcohol misuse	73,861 (3.7%)	28,383 (3.1%)	14,970 (2.8%)	33,443 (2.0%)	150,657 (2.9%)
Any cancer	188,043 (9.3%)	92,855 (10.2%)	56,016 (10.5%)	169,456 (10.2%)	506,370 (9.9%)
Arrhythmia	86,915 (4.3%)	40,950 (4.5%)	24,714 (4.6%)	77,625 (4.7%)	230,204 (4.5%)
Atrial fibrillation	27,278 (1.4%)	13,659 (1.5%)	8,301 (1.6%)	25,383 (1.5%)	74,621 (1.5%)
Bipolar disorder	8,906 (0.4%)	4,556 (0.5%)	2,879 (0.5%)	9,942 (0.6%)	26,283 (0.5%)
Cardiomyopathy	2,790 (0.1%)	1,395 (0.2%)	832 (0.2%)	2,659 (0.2%)	7,676 (0.1%)
Chronic liver disease	6,171 (0.3%)	2,699 (0.3%)	1,559 (0.3%)	4,295 (0.3%)	14,724 (0.3%)
Chronic kidney disease	29,559 (1.5%)	14,875 (1.6%)	9,198 (1.7%)	25,019 (1.5%)	78,651 (1.5%)
Chronic obstructive pulmonary disease	44,565 (2.2%)	21,905 (2.4%)	13,380 (2.5%)	36,297 (2.2%)	116,147 (2.3%)
Chronic pancreatitis	1,336 (0.1%)	721 (0.1%)	389 (0.1%)	1,453 (0.1%)	3,899 (0.1%)
Depression	347,285 (17.2%)	158,615 (17.4%)	92,657 (17.3%)	258,132 (15.5%)	856,689 (16.7%)
Epilepsy	29,208 (1.4%)	13,786 (1.5%)	8,375 (1.6%)	23,415 (1.4%)	74,784 (1.5%)
Erectile dysfunction	63,318 (3.1%)	30,695 (3.4%)	18,191 (3.4%)	56,840 (3.4%)	169,044 (3.3%)
Family history of CHD	361,918 (17.9%)	177,462 (19.5%)	108,598 (20.3%)	356,510 (21.4%)	1,004,488 (19.6%)
Heart failure	14,033 (0.7%)	6,320 (0.7%)	3,704 (0.7%)	10,200 (0.6%)	34,257 (0.7%)
Hypercholesterolaemia	41,645 (2.1%)	26,400 (2.9%)	19,111 (3.6%)	108,438 (6.5%)	195,594 (3.8%)
Hypertension	239,154 (11.8%)	145,374 (16.0%)	103,654 (19.4%)	519,962 (31.2%)	1,008,144 (19.6%)
Hyperthyroidism	17,411 (0.9%)	9,285 (1.0%)	5,980 (1.1%)	21,704 (1.3%)	54,380 (1.1%)
Hypothyroidism	65,190 (3.2%)	35,966 (3.9%)	23,644 (4.4%)	92,411 (5.5%)	217,211 (4.2%)
Migraine	146,681 (7.3%)	64,631 (7.1%)	37,258 (7.0%)	101,724 (6.1%)	350,294 (6.8%)
Non-alcoholic fatty liver disease	5,966 (0.3%)	2,524 (0.3%)	1,534 (0.3%)	4,227 (0.3%)	14,251 (0.3%)
Parkinson's disease	3,931 (0.2%)	1,641 (0.2%)	848 (0.2%)	1,850 (0.1%)	8,270 (0.2%)
Psychosis	8,161 (0.4%)	3,669 (0.4%)	2,122 (0.4%)	6,773 (0.4%)	20,725 (0.4%)
Schizophrenia	10,955 (0.5%)	6,014 (0.7%)	3,954 (0.7%)	15,273 (0.9%)	36,196 (0.7%)
Systemic lupus erythematosus	2,102 (0.1%)	1,119 (0.1%)	704 (0.1%)	2,069 (0.1%)	5,994 (0.1%)
Sleep apnoea	11,352 (0.6%)	5,232 (0.6%)	3,117 (0.6%)	9,420 (0.6%)	29,121 (0.6%)
Transient ischaemic attack	13,132 (0.7%)	7,212 (0.8%)	4,875 (0.9%)	20,609 (1.2%)	45,828 (0.9%)
Type 1 diabetes mellitus	10,028 (0.5%)	5,946 (0.7%)	4,046 (0.8%)	21,918 (1.3%)	41,938 (0.8%)
Type 2 diabetes mellitus	48,336 (2.4%)	30,078 (3.3%)	21,887 (4.1%)	146,967 (8.8%)	247,268 (4.8%)
Venous thromboembolism	19,727 (1.0%)	9,610 (1.1%)	5,984 (1.1%)	18,626 (1.1%)	53,947 (1.1%)
Prescribed medication at baseline (n, %)					
Lipid-lowering therapy	72,456 (3.6%)	44,555 (4.9%)	31,160 (5.8%)	161,213 (9.7%)	309,384 (6.0%)
Antiplatelet medication	52,188 (2.6%)	29,679 (3.3%)	20,152 (3.8%)	91,999 (5.5%)	194,018 (3.8%)
Antihypertensive medication	249,957 (12.4%)	146,135 (16.0%)	101,575 (19.0%)	485,072 (29.1%)	982,739 (19.1%)
Antidiabetic medication	43,896 (2.2%)	26,489 (2.9%)	18,452 (3.5%)	110,716 (6.6%)	199,553 (3.9%)

Table 3. Incidence rate and risk of major adverse cardiovascular event (n=5,133,574).

	Number of MACE events	Follow-up (person-years)	Incidence rate* (95% CI)	Unadjusted hazard ratio (95% CI)	Adjusted hazard ratio (95% CI)
One (1) LDL-C recorded	186,191	96.123	1937.0 (1928.2 - 1945.8)	Reference	Reference
Two (2) LDL-C recorded	107,795	66.731	1615.4 (1605.8 - 1625.0)	0.786 (0.780 - 0.792)	0.703 (0.698 - 0.709)
Three (3) LDL-C recorded	70,574	47.538	1484.6 (1473.7 - 1495.6)	0.690 (0.684 - 0.696)	0.570 (0.565 - 0.575)
Four (4) or more LDL-C recorded	246,101	204.259	1204.9 (1200.1 - 1209.6)	0.492 (0.489 - 0.495)	0.312 (0.310 - 0.314)

MACE, major adverse cardiovascular event (defined as a composite of non-fatal coronary heart disease, non-fatal stroke, and cardiovascular disease-related death); *per 100,000 person-years; CI, confidence interval; LDL-C, low-density lipoprotein cholesterol; adjusted for age at time of first LDL-C measurement record, sex, ethnicity, social deprivation (index of multiple deprivation, quintiles), current smoker status, first LDL-C level, diagnosis of arrhythmia, cancer, chronic kidney disease, heart failure, hypercholesterolaemia, hypothyroidism, non-alcoholic fatty liver disease, systemic lupus erythematosus, transient ischaemic attack, type-2 diabetes mellitus, family history of coronary heart disease, prescription of lipid-lowering therapy, antiplatelet, antihypertensive and antidiabetic therapy.

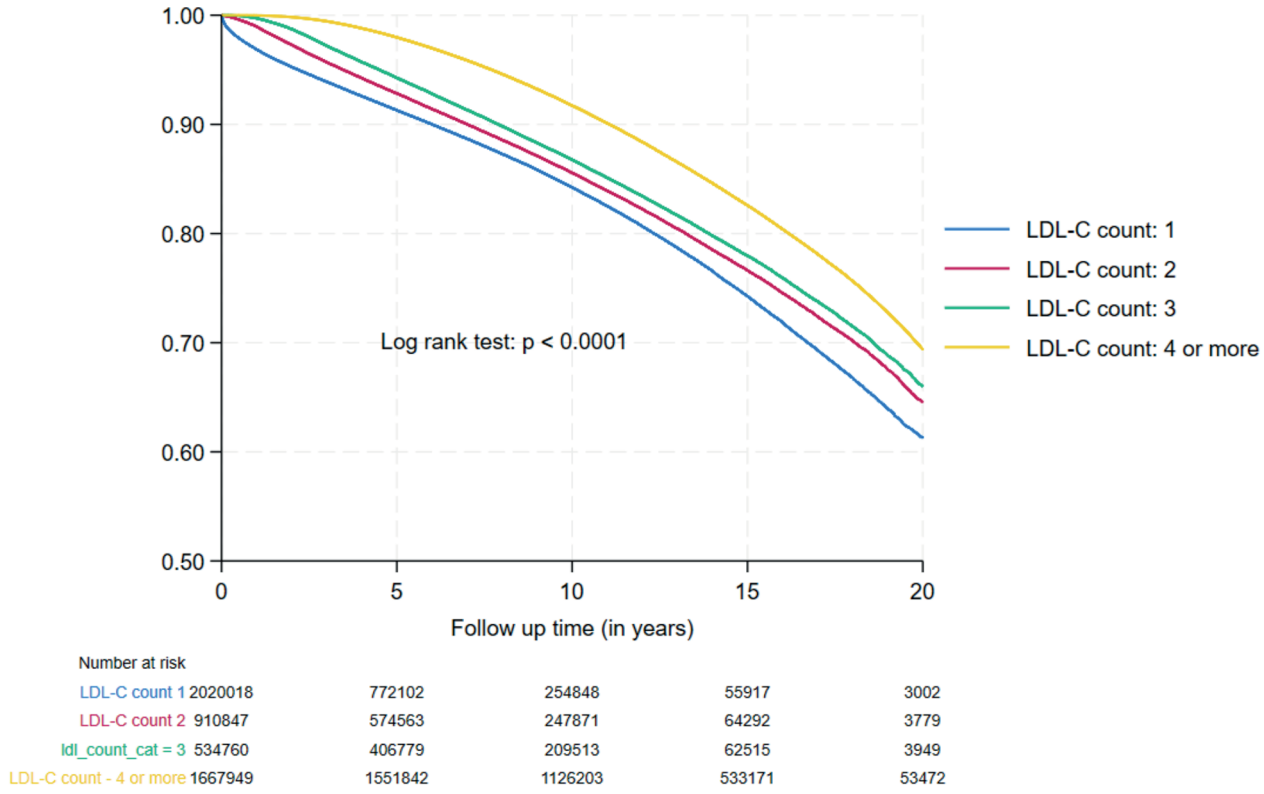

Figure 2. Kaplan-Meier plot.

levels or early signs of disease. While our multivariable adjustment accounted for measured risk factors, we cannot exclude the possibility that increased testing was partly a response to higher baseline risk rather than a cause of reduced MACE. This may have influenced the observed associations. Despite these limitations, the study provides important insights into real-world lipid monitoring practices and outcomes.[21,22]

This study contributes evidence to the growing body of literature on cardiovascular risk management by demonstrating a clear association between the frequency of LDL-C testing and reduced MACE risk. While previous studies have highlighted the role of lipid testing informs treatment decisions, our findings extend this by linking monitoring frequency to meaningful clinical outcomes. Given the simplicity, affordability, and availability of lipid testing, promoting regular monitoring could be a practical and impactful strategy in routine CVD prevention. Further research should explore causal mechanisms and evaluate the potential benefits of embedding structured lipid monitoring protocols into routine care to improving long-term cardiovascular outcomes.

Contributions

RKA, conceived and performed the data analysis; RKA, DMN, took the lead in writing the manuscript. All authors provided critical review, feedback, and interpretation of findings, read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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Conflict of interest

WD reports consulting fees and speaker honoraria from Aimediq, Astra Zeneca, Bayer, Boehringer Ingelheim, Boston Scientific, Medtronic, Vifor Pharma, travel support from Pharmacosmos, and research support to the Institute from EU (Horizon2020), German Ministry of Education and Research, German Center for Cardiovascular Research, German Pension Insurance (regional Mitteldeutschland), Boehringer Ingelheim, Vifor Pharma. The remaining authors have no competing interests.

Ethics approval

This study has been approved through the CPRD Research Data Governance for MHRA database research (reference number: 22_ 002319).

Availability of data and materials

This study is based in part on data from the CPRD obtained under licence from the UK Medicines and Healthcare products Regulatory Agency (MHRA). Requests to access CPRD data are reviewed via the CPRD RDG process to ensure that the proposed research is of benefit to patients and public health. Additional information is available on the CPRD website: https://www.cprd.com/safeguarding-patient-data

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