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Commentary

Identify and Act on LDL: A New Proposed Algorithm Linking Cardiovascular Risk Assessment to LDL-C Goals

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Abstract

Traditional cardiovascular risk calculators estimate population-based 5–10-year cardiovascular event rates but do not fully capture the life-course nature of atherosclerotic cardiovascular disease (ASCVD). Risk may be underestimated in younger adults with substantial cumulative exposure to atherogenic lipoproteins, overestimated in some older adults, and sub-optimally estimated in patients with important risk-enhancing factors, including lipoprotein(a) [Lp(a)], sex-specific risk modifiers, inflammatory conditions, and chronic kidney disease. Advances in vascular imaging such as computed tomography coronary angiography (CTCA) further complement conventional risk assessment by enabling direct identification of subclinical atherosclerosis and underlying disease burden. Despite these advances, contemporary cardiovascular risk management remains fragmented. Risk calculators, risk-enhancing factors, vascular imaging, and lipid management recommendations are frequently applied in isolation, often creating an implementation gap between cardiovascular risk assessment and personalized, guideline-directed LDL-C goals. To address this challenge, we propose the Identify and Act algorithm: a pragmatic implementation framework that integrates established evidence-based approaches into a structured clinical pathway directly linking cardiovascular risk assessment to personalized LDL-C goals. Rather than replacing existing risk calculators or lipid guidelines, the Identify and Act algorithm provides a practical framework for translating cardiovascular risk into personalized LDL-C treatment goals. By integrating short-term and lifetime risk assessment with risk refinement strategies, the framework aims to reduce the fragmentation of cardiovascular risk management, minimize clinical inertia, and facilitate implementation of evidence-based lipid-lowering strategies. Prospective validation is required to determine whether this approach advances LDL-C goal attainment, is cost-effective, and ultimately improves cardiovascular outcomes.

Keywords: Cardiovascular risk assessment; atherosclerotic cardiovascular disease; LDL cholesterol; lifetime cardiovascular risk; cardiovascular risk stratification; lipid-lowering therapy; risk-enhancing factors; lipoprotein(a); coronary artery calcium; preventive cardiology

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1. Introduction

Despite advances in the identification and management of cardiovascular disease over the past three decades, atherosclerotic cardiovascular disease (ASCVD) remains the

leading cause of morbidity and mortality worldwide [1,2]. ASCVD is a chronic, progressive disease driven by cumulative lifetime exposure to ApoB-containing lipoproteins, of which LDL-C accounts for the majority, in conjunction with comorbidities that promote endothelial dysfunction, vascular inflammation, and plaque instability [2–6]. This life-course model of atherosclerosis highlights the importance of identifying at-risk individuals earlier, particularly within the primordial and primary prevention stages [7–9].

Contemporary cardiovascular risk assessment strategies primarily rely on validated short-term risk prediction tools such as PREVENT, SCORE2, QRISK3, and AusCVDRisk, which estimate the risk of cardiovascular events using traditional clinical variables [10–14]. These models typically incorporate risk factors such as age, sex, smoking status, blood pressure, and diabetes mellitus and are designed to improve population-level risk stratification and preventive care.

Important limitations nevertheless remain. Traditional cardiovascular risk factors account for only 50% of the attributable burden of cardiovascular disease in data from the Global Cardiovascular Risk Consortium, highlighting the contribution of additional biological, genetic, inflammatory, environmental, and sex-specific determinants of cardiovascular risk [4,15–26]. In addition, most contemporary risk prediction models remain heavily influenced by chronological age, frequently underestimating risk in younger individuals with substantial lifetime exposure to atherogenic lipoproteins while overestimating risk in some older adults. Although newer models such as PREVENT and QRISK Lifetime incorporate longer-term risk estimation, most contemporary approaches remain focused on short-term event prediction despite the prolonged subclinical evolution of atherosclerosis (Table 1) [1,2,7–9,15,27–30].

Table 1. Commonly used short-term and lifetime cardiovascular risk calculators.

Calculator	Region	Age (Years)	Time Horizon	Risk Categories/Thresholds
ASCVD Risk Estimator/Pooled Cohort Equations	United States	40–79	10-year	Low < 5%; Borderline 5–7.4%; Intermediate 7.5–19.9%; High ≥ 20%
PREVENT	United States	30–79	10-year and 30-year	10-year risk: Low < 3%; Borderline 3–<5%; Intermediate 5–<10%; High ≥ 10%
QRISK3	United Kingdom	25–84	10-year	Low < 10%; Moderate 10–20%; High ≥ 20%
QRISK Lifetime	United Kingdom	25–84	Lifetime risk	High lifetime risk defined as ~39% (>75th centile), ~50% > 90th centile)
SCORE2	Europe	40–69	10-year	<50 years: Low–Moderate < 2.5%, High 2.5–<7.5%, Very high ≥ 7.5%; 50–69 years: Low–Moderate < 5%, High 5–<10%, Very high ≥ 10%
SCORE2-OP	Europe	>70	10-year	Low < 7.5%; Moderate 7.5–15%; High > 15%
AusCVDRisk	Australia	45–79	5-year	Low < 5%; Intermediate 5–<10%; High ≥ 10%

Summary of commonly used cardiovascular risk calculators, including region of derivation, age inclusion, time horizon, and commonly used risk categories. ASCVD = atherosclerotic cardiovascular disease; PREVENT = Predicting Risk of Cardiovascular Disease events; SCORE2 = Systematic Coronary Risk Evaluation 2. Age-stratified SCORE2 thresholds reflect the 2021 ESC Prevention Guidelines, while the fixed thresholds in Table 3 reflect the 2025 ESC/EAS Dyslipidemia Guidelines. These reflect different approaches to risk categorization. The Identify and Act algorithm uses the 2025 thresholds to guide LDL-C goal assignment [10,12–14].

Beyond traditional risk-factor assessment, preventive cardiology has evolved substantially. Causal and risk-enhancing biomarkers such as lipoprotein(a) [Lp(a)], inflammatory markers, chronic kidney disease, metabolic dysfunction, adverse pregnancy outcomes, and premature menopause are increasingly recognized as important modifiers of cardiovascular risk [15–25]. In addition, advances in vascular imaging, including coronary artery calcium (CAC) scoring, carotid plaque assessment, and CTCA now permit direct identification of subclinical atherosclerosis and thus a more personalized assessment of underlying disease burden [9,26,31–33]. Collectively, these developments have shifted cardiovascular prevention from a probabilistic population-based estimation toward a more individualized and biologically informed model of care.

Despite these advances, cardiovascular risk management remains fragmented. Risk calculators, risk-enhancing factors, imaging modalities, and lipid management pathways are frequently applied in isolation, contributing to clinical inertia and the delayed implementation of evidence-based care [34–39]. The challenge in contemporary preventive cardiology is not just identifying cardiovascular risk, but personalizing LDL-C goals and moving the patient toward timely, guideline-directed, lipid-lowering strategies [3,11,27–29].

In this paper, we propose the Identify and Act algorithm, a contemporary framework for cardiovascular risk management that integrates traditional risk estimation tools with lifetime risk assessment, risk-enhancing factors, vascular imaging, and emerging biomarkers, such as Lp(a). The algorithm is designed to support earlier identification of at-risk individuals and facilitate more individualized preventive strategies across the life course. Importantly, the Identify and Act algorithm is not a new cardiovascular risk prediction tool; rather, it brings these established approaches together to guide clinicians from cardiovascular risk assessment to guideline-directed LDL-C goals.

2. The Identify and Act Algorithm

The framework comprises five sequential steps that guide clinicians from cardiovascular risk assessment to evidence-based LDL-C treatment goals (Figure 1).

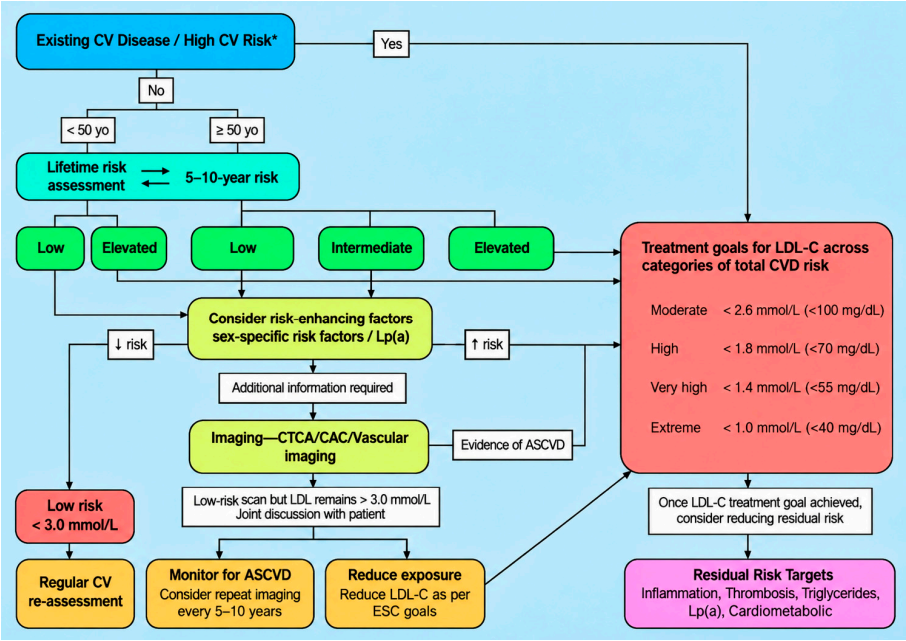


Figure 1. The proposed Identify and Act algorithm. * High CV risk: stage 3–5 CKD or familial hypercholesterolemia (FH); lifetime risk assessment: QRISK Lifetime, PREVENT; 5–10-year risk assessment: AusCVDrisk, QRISK3, SCORE2, PREVENT. Treatment goals. Low risk: Low lifetime

and low 5–10-year risk with no additional risk/risk-enhancing factors (RFs). Moderate risk: Elevated lifetime risk and low/intermediate 5–10-year risk, or low lifetime risk and intermediate 5–10-year risk, without additional RFs or evidence of ASCVD on imaging; young patients with T1DM (<35 years) or T2DM (<50 years) with diabetes duration < 10 years and no other major risk factors. High risk: elevated lifetime risk and low/intermediate 5–10-year risk + additional RFs, or elevated 5–10-year risk; markedly elevated single risk factors, particularly TC > 8 mmol/L (310 mg/dL), LDL-C > 4.9 mmol/L (190 mg/dL), or BP ≥ 180/110 mmHg, FH without other major risk factors, moderate CKD (eGFR 30–59 mL/min/1.73 m^2), or diabetes mellitus without target organ damage, with duration ≥10 years or another additional risk factor. Very high risk: clinical or imaging-confirmed ASCVD, SCORE2/SCORE2-OP ≥20%, FH with ASCVD or another major risk factor, severe CKD (eGFR < 30 mL/min/1.73 m^2), diabetes mellitus with target organ damage, ≥3 major risk factors, or early-onset T1DM of long duration (>20 years). Extreme Risk: ASCVD patients with recurrent vascular events despite maximally tolerated statin-based therapy, polyvascular disease (e.g., coronary and peripheral arterial disease). Arrows indicate the direction of clinical decision-making. Colours distinguish the key stages of the algorithm, including initial risk identification (blue), risk estimation (green), risk refinement (lime green), LDL-C treatment goals (red), ongoing management (orange), and residual risk assessment (purple). CAC = coronary artery calcium score. CTCA = CT coronary angiogram. CVD = cardiovascular disease. ASCVD = atherosclerotic cardiovascular disease. CV = cardiovascular.

3. Initial Classification

1. Identify patients with established ASCVD or very high baseline risk [e.g., familial hypercholesterolemia, stage 3–5 chronic kidney disease (CKD)] [10,11]. These patients proceed directly to guideline-directed LDL-C goals and LDL-C-lowering strategies [11,27–29].
2. For others, use both short-term (5–10-year) and lifetime risk calculators, with lifetime risk generally more informative in individuals aged <50 years, and short-term risk more useful in those aged ≥50 years (Table 1) [10,12–14].

4. Risk Interpretation

1. In this algorithm, short-term (5–10-year) risk is described as low, intermediate, or elevated to distinguish the assessment phase from the guideline-derived treatment-risk categories. The relationship between calculator-specific terminology (used in PREVENT, QRISK3, AusCVDRisk, and SCORE2) (Table 1), Identify and Act categories, and treatment-risk categories is summarized in Table 2 [10,12–14].
2. Elevated lifetime risk or elevated short-term risk: proceed to appropriate guideline-directed LDL-C goals and LDL-C-lowering strategies [27–29].
3. Low or intermediate risk—refine assessment.

Table 2. Mapping cardiovascular risk categories across calculators, the Identify and Act algorithm, and LDL-C treatment categories.

Identify and Act Assessment Category	Typical 5–10-Year Risk Calculator Categories	Interpretation in Algorithm	Potential Final LDL-C Treatment Category *
Low	Low/Low–Moderate	Reassuring short-term risk; assess lifetime risk and risk enhancers as appropriate	Usually Low
Intermediate	Borderline/Intermediate/Moderate	Risk is uncertain enough to warrant refinement with risk-enhancing factors +/− imaging	Moderate or High, depending on refinement

Table 2. *Cont.*

Identify and Act Assessment Category	Typical 5–10-Year Risk Calculator Categories	Interpretation in Algorithm	Potential Final LDL-C Treatment Category *
Elevated	High/Very high	Sufficient risk to proceed toward guideline-directed LDL-C treatment goals	Usually Moderate–Very high, depending on clinical context
Established ASCVD/predefined high-risk condition	Calculator not required	Proceed directly to treatment categorization	High/Very high/Extreme

* Final treatment category is determined by the total clinical assessment, not by the calculator category alone.

5. Risk Refinement

1. Consider risk-enhancing factors such as, but not limited to, Lp(a) ≥ 105 nmol/L, sex-specific RFs, major psychiatric disorders, and immune/inflammatory disease [27–29].
2. If these factors shift the overall assessment toward higher risk, progress to appropriate guideline-directed LDL-C goals and LDL-C-lowering strategies [27–29]
3. If uncertainty persists, consider vascular imaging (CAC, CTCA, or carotid ultrasound) to further refine risk [9,26,31–33].

6. Translation to LDL-C Goals

1. Translate overall cardiovascular risk into appropriate guideline-directed LDL-C goals [8,27–29]
2. Lifestyle optimization should underpin cardiovascular risk reduction at all levels of risk, alongside LDL-C-lowering strategies where indicated.
3. Representative risk categories and corresponding LDL-C, non-HDL-C, and ApoB treatment goals are summarized in Table 3 [8,27–29].

Table 3. Contemporary cardiovascular risk categories and corresponding lipid treatment goals integrated within the Identify and Act algorithm.

Risk Category	Risk Assessment	Characteristic's	LDL-C Goal (mmol/L)	LDL-C Goal mg/dL	Non HDL-C Goal (mmol/L)	ApoB Goal (mg/dL)
Low risk	Low lifetime and low 5–10-year risk with no additional RFs	• SCORE2/SCORE2-OP < 2%	<3.0	<116	<3.8	
Moderate risk	Elevated lifetime risk and low/intermediate 5–10-year risk, or low lifetime risk and intermediate 5–10-year risk, without additional RFs or evidence of ASCVD on imaging.	• SCORE2/SCORE2-OP ≥ 2% and <10% • Young patents with T1DM (<35 years) or T2DM (<50 years) with diabetes duration <10 years and no other major risk factors	<2.6	<100	<3.4	<100
High risk	Elevated lifetime and low/intermediate 5–10-year risk + RFs OR High 5–10-year risk	• SCORE2/SCORE2-OP ≥ 10% and <20% • Markedly elevated single risk factors, particularly TC > 8 mmol/L (310 mg/dL), LDL-C > 4.9 mmol/L (190 mg/dL), or BP ≥ 180/110 mmHg • Familial hypercholesterolemia (FH) without other major risk factors • Moderate CKD (eGFR 30–59 mL/min/1.73 m^2) • Diabetes mellitus without target organ damage, with duration ≥10 years or another additional risk factor	<1.8 and ≥50% reduction from baseline	<70	<2.6	<80

Table 3. *Cont.*

Risk Category	Risk Assessment	Characteristic's	LDL-C Goal (mmol/L)	LDL-C Goal mg/dL	Non HDL-C Goal (mmol/L)	ApoB Goal (mg/dL)
Very high risk	High CV risk	• Clinical or imaging-confirmed ASCVD • SCORE2/SCORE2-OP ≥ 20% • FH with ASCVD or another major risk factor • Severe CKD (eGFR < 30 mL/min/1.73 m^2) • Diabetes mellitus with target organ damage, ≥3 major risk factors, or early-onset T1DM of long duration (>20 years)	<1.4 and ≥50% reduction from baseline	<55	<2.2	<65
Extreme risk	Existing CV disease	• ASCVD patients with recurrent vascular events despite maximally tolerated statin-based therapy • Polyvascular disease (e.g., coronary and peripheral arterial disease)	<1.0 and ≥50% reduction from baseline	<40	<1.8	<55

Risk categories, representative clinical characteristics, and suggested LDL-C, non-HDL-C, and ApoB treatment goals adapted from contemporary ESC/EAS dyslipidemia and cardiovascular prevention guidelines, incorporating elements of SCORE2/SCORE2-OP risk stratification and lifetime cardiovascular risk assessment [28,29].

7. Follow-Up

1. For patients at low risk, repeat cardiovascular risk estimation every 3–5 years [8,27–29].
2. For patients at intermediate-to-high risk, reassess cardiovascular risk and LDL-C goals at least annually [8,27–29]
3. Review and refine LDL-C goals as cardiovascular risk changes over time [8,27–29].
4. Once LDL-C goals have been addressed, other contributors to residual cardiovascular risk, including elevated triglycerides, inflammation, and thrombosis, should be considered where clinically relevant (Figure 1).
5. For patients who do not achieve LDL-C goals and decline lifestyle or LDL-C-lowering therapy, repeat imaging may be considered after 5–10 years, with consideration of cumulative radiation exposure when CT-based imaging is used [8,27–29].

8. Discussion

Despite advances in preventive cardiology, ASCVD remains the leading cause of morbidity and mortality worldwide [1,2]. Although cardiovascular risk assessment has become increasingly sophisticated, translating this evidence into timely, personalized, preventive care remains a persistent challenge. Clinical inertia, delayed treatment intensification, and fragmented application of risk assessment tools continue to contribute to suboptimal implementation of evidence-based lipid management [34,36–39].

The Identify and Act algorithm was developed to address this implementation gap. It was developed by author consensus, drawing on practical experience in primary care and specialist lipid practice. It does not introduce a new cardiovascular risk prediction model; instead, it organizes established risk assessment tools within a structured pathway, linking cardiovascular risk assessment directly to guideline-directed LDL-C treatment goals. By combining short-term and lifetime risk estimation with risk-enhancing factors and vascular imaging, the framework aims to facilitate earlier, more consistent, identification of LDL-C goals and implementation of appropriate preventive lipid-lowering therapy.

Traditional cardiovascular risk calculators remain the cornerstone of primary prevention, providing robust estimates of future cardiovascular events across diverse populations. They should be applied from their minimum validated age, with earlier assessment considered in individuals at increased cardiovascular risk. However, important limitations persist. Most prediction tools rely heavily on chronological age, potentially underestimating risk

in younger individuals with substantial cumulative exposure to atherogenic lipoproteins while overestimating risk in some older adults.

In addition, differences in cohorts, calibration, and healthcare systems limit direct comparison between calculators. Risk calculators also vary in how they account for ethnicity and population-specific risk, including First Nations status, and may have limited validation in populations with CKD. Accordingly, the Identify and Act algorithm avoids reliance on rigid numerical thresholds during its initial risk assessment. Instead, individuals are categorized according to low, intermediate, or elevated short-term and lifetime cardiovascular risk, allowing the framework to remain adaptable across different risk calculators and healthcare settings (Table 2). Where short-term and lifetime risk categories are discordant, the higher risk category should guide management [10,12–14].

Lifetime cardiovascular risk assessment is a central component of the algorithm because it identifies younger individuals with substantial cumulative exposure to atherogenic lipoproteins despite apparently reassuring short-term risk estimates. Several validated tools are available for lifetime risk estimation, including QRISK Lifetime and PREVENT [13,15,40]. In regions where population-specific lifetime risk calculators are unavailable, these tools may be used cautiously to inform clinician–patient discussion, recognizing that they have not been validated in all populations. Importantly, thresholds for elevated lifetime risk differ between calculators and should therefore be interpreted within the context of the specific model being used. For example, a lifetime cardiovascular risk ≥39% has been used to identify elevated lifetime risk in younger adults using established US lifetime-risk models [15].

Risk-enhancing factors provide additional insight into the biological vulnerability of an individual beyond traditional risk-factor assessment, and are particularly valuable among individuals classified as at intermediate risk [27–29]. A persistently elevated high-sensitivity C-reactive protein (hsCRP $>$ 2 mg/L) level is associated with increased cardiovascular risk. Another important inherited cardiovascular RF is Lp(a) [16]. Lp(a) is an ApoB-containing lipoprotein with an attached apolipoprotein(a) moiety, is >90% genetically determined, and reaches adult concentrations by early childhood [20]. Elevated concentrations are causally associated with ASCVD and aortic valve stenosis and confer risk in a concentration-dependent manner [16–20]. Individuals with markedly elevated Lp(a) concentrations may experience cardiovascular risk approaching that observed in familial hypercholesterolemia [17]. While dedicated Lp(a)-lowering therapies are not yet widely available in clinical practice, current management strategies aim to reduce collective cardiovascular risk through aggressive lowering of other ApoB-containing lipoproteins [27,28]. Within this context, incorporation of Lp(a) into the algorithm serves not merely as an additional biomarker, but as a mechanism to identify individuals who may benefit from earlier intensification of preventive strategies despite apparently reassuring short-term risk estimates.

Similarly, sex-specific and female-predominant cardiovascular RFs are increasingly recognized as important determinants of lifetime cardiovascular risk yet remain incompletely represented in conventional risk calculators [23,24]. Adverse pregnancy outcomes, pre-eclampsia, gestational diabetes mellitus, premature menopause, and polycystic ovarian syndrome are associated with increased future cardiovascular risk and may identify women with underlying endothelial dysfunction and adverse cardiometabolic susceptibility years before overt ASCVD develops. In addition, several disorders that disproportionately affect women, including rheumatoid arthritis, systemic lupus erythematosus, migraine disorders, and depression, are independently associated with accelerated vascular disease [23,25]. Recognition of these modifiers within the algorithm is intended to refine interpretation of cardiovascular vulnerability and support earlier preventive intervention through per-

sonalized LDL-C goals in individuals whose risk may otherwise be underestimated by conventional calculators alone.

Beyond biomarker-based risk refinement, vascular imaging has increasingly transformed cardiovascular prevention from probabilistic estimation toward direct visualization of disease burden. Carotid ultrasound, CAC scoring, and CTCA provide incremental prognostic information beyond traditional risk calculators and permit direct assessment of subclinical atherosclerosis [9,26,31–33]. Carotid ultrasound enables identification of early vascular disease and assessment of carotid plaque burden, while CAC scoring has emerged as a relatively accessible and validated tool for risk reclassification, particularly among intermediate-risk populations [31–33]. A CAC score of zero is associated with low short-term cardiovascular event rates in selected populations, although the absence of coronary calcification does not exclude non-calcified plaque [31,32]. The significance of imaging findings should be interpreted in the context of age and baseline risk rather than according to rigid thresholds; for example, a small burden of calcified plaque in a younger individual may carry greater significance than the same absolute CAC score in an older individual. Increasing plaque or CAC burden should therefore progressively strengthen consideration of a higher-risk classification and more intensive LDL-C lowering. CTCA provides more detailed assessment of total plaque burden, stenosis severity, and plaque morphology, offering further biological insight into the underlying atherosclerotic process [31].

Importantly, imaging may also play a behavioral and implementation role within preventive cardiology. Visualization of subclinical atherosclerosis has been associated with improved patient adherence to lifestyle modification and lipid-lowering therapy and may help overcome therapeutic inertia by reinforcing the tangible presence of vascular disease [35]. In this regard, imaging serves not only as a diagnostic modality but also as a communication tool that may strengthen shared decision-making between clinician and patient.

A central principle of the Identify and Act algorithm is that risk refinement should directly translate into actionable therapeutic goals. Recognition of elevated cardiovascular risk alone does not alter the trajectory of ASCVD unless it results in a meaningful reduction in cumulative LDL-C exposure. Accordingly, the algorithm links risk stratification directly to ESC-defined LDL-C goals, which are supported across current international dyslipidemia guidelines [28,29]. This approach emphasizes that LDL-C goals should reflect not only the presence of symptomatic ASCVD, but also lifetime exposure to atherogenic lipoproteins together with comorbidities that promote endothelial dysfunction. By integrating these elements within a single pathway, the algorithm seeks to create a clearer conceptual relationship between risk burden and treatment intensity for both clinicians and patients [34,37,39]. Although discussion of pharmacological management is beyond the scope of this review, clinicians have access to a broad range of evidence-based lipid-lowering therapies, including statins, ezetimibe, bempedoic acid and PCSK9-targeted therapies, which can be used individually or in combination to achieve guideline-recommended LDL-C goals. Patients presenting with higher baseline LDL-C concentrations or greater absolute cardiovascular risk will frequently require combination therapy to achieve these goals.

The major strength of this algorithm lies in its simplicity and broad clinical applicability. Rather than introducing a novel risk calculator or biomarker, the model integrates established evidence-based tools into a unified and pragmatic structure capable of being used across both primary care and specialist lipid clinics (Box 1). The algorithm incorporates readily available investigations with robust evidence linking them to cardiovascular outcomes and allows adaptation across different healthcare systems and regional risk calculators. The visual nature of the pathway may also facilitate clinician–patient communication

and shared decision-making, particularly when discussing lifetime risk, imaging findings, and rationale for LDL-C-lowering therapy (Table 4).

Table 4. Clinical vignettes illustrating the application of the Identify and Act algorithm.

Clinical Vignette 1: Discordance Between Short-Term and Lifetime Risk A 42-year-old man presents for cardiovascular risk assessment. His estimated 5–10-year cardiovascular risk is low; however, lifetime risk is considered elevated given his BMI (34), LDL-C (4.1 mmol/L) level, and history of severe depression. Coronary artery calcium scoring was not performed. Applying the Identify and Act on LDL algorithm, discordance between short-term and lifetime risk supports classification as moderate cardiovascular risk, with an LDL-C goal of <2.6 mmol/L. Lifestyle modification and shared decision-making regarding lipid-lowering therapy are recommended.	Clinical Vignette 2: Risk Reclassification Following Identification of Risk-Enhancing Factors A 71-year-old woman with a history of smoking and treated hypertension presents for an age-related health assessment. Her systolic blood pressure is 135 mmHg, with a total cholesterol-to-HDL-C ratio of 4.3. Short-term cardiovascular risk assessment identifies intermediate risk, while lifetime risk is considered elevated. A history of premature menopause is identified as an additional risk-enhancing factor. Applying the Identify and Act on LDL algorithm, these findings support classification as high cardiovascular risk, with a corresponding LDL-C goal of <1.8 mmol/L. Lifestyle modification and shared decision-making regarding lipid-lowering therapy are recommended.
Clinical Vignette 3: Risk Reclassification Following Cardiovascular Imaging A 68-year-old man with rheumatoid arthritis presents for a health assessment. His estimated 5–10-year and lifetime cardiovascular risks are both considered low. Further risk refinement identifies an Lp(a) of 280 nmol/L. CTCA is subsequently performed, demonstrating widespread coronary atherosclerotic plaque and a CAC of 350. Applying the Identify and Act on LDL algorithm, these findings support reclassification as very high cardiovascular risk, with a corresponding LDL-C goal of < 1.4 mmol/L. Lifestyle modification and shared decision-making regarding lipid-lowering therapy are recommended.	

Box 1. Clinical implications box.

- Assess cardiovascular risk using both short-term (5–10-year) and lifetime risk estimation, with lifetime risk particularly informative in adults <50 years.
- In patients with low or intermediate estimated risk, refine assessment using risk-enhancing factors (e.g., Lp(a), sex-specific factors, inflammatory disorders) and vascular imaging where appropriate.
- Translate the overall cardiovascular risk assessment directly into guideline-directed LDL-C treatment goals to support timely lipid-lowering strategies.
- Reassess cardiovascular risk over time, recognizing that ASCVD is progressive and management should evolve as risk changes.

However, this simplicity also represents an inherent limitation. No single-page algorithm can fully capture the complexity of an advanced cardiovascular risk assessment or account for the precise magnitude of risk associated with individual biomarkers, imaging findings, or comorbidities. The algorithm is therefore intended as a practical clinical scaffold rather than a replacement for detailed guideline-based assessment or specialist judgment.

Further limitations relate to accessibility and cost. Several investigations incorporated within the algorithm, including Lp(a) measurement and vascular imaging, remain incompletely reimbursed in many healthcare systems and may therefore increase short-term healthcare expenditure [21,32]. Imaging modalities such as CTCA also carry small but

relevant risks associated with radiation exposure and contrast administration [31]. Moreover, while the algorithm aims to improve identification of at-risk individuals, broader application of imaging and biomarkers may increase the potential for over-classification or over-treatment in some populations. Prospective validation studies are therefore required to determine the balance between improved risk detection and false-positive classification.

Future research should evaluate the clinical utility, feasibility, and cost-effectiveness of integrated prevention algorithms such as this across diverse populations. Prospective studies comparing this model with traditional cardiovascular risk management are needed to determine whether integration of lifetime risk, risk-enhancing factors, and imaging translates into earlier treatment initiation, improved LDL-C goal attainment, reduced cumulative LDL-C exposure, and ultimately, lower cardiovascular event rates. Additional research is also required to better define optimal ages for initiation of cardiovascular risk assessment and biomarker testing, particularly in light of recent recommendations supporting earlier identification of lifetime cardiovascular risk [27]. As preventive cardiology continues to evolve toward more personalized and biology-driven approaches, integrated algorithms capable of translating complex evidence into routine clinical practice may become increasingly important.

9. Conclusions

The Identify and Act algorithm provides a pragmatic implementation framework that integrates contemporary cardiovascular risk assessment into a structured clinical pathway linking cardiovascular risk directly to personalized, guideline-directed LDL-C goals. By combining short-term and lifetime cardiovascular risk assessment, risk-enhancing factors, vascular imaging, and contemporary lipid targets, the framework is designed to facilitate earlier, more consistent translation of cardiovascular risk into personalized LDL-C goals.

Although the individual components of the framework are well established within contemporary preventive cardiology, their integration into a single implementation-focused pathway represents the principal innovation of the Identify and Act algorithm. By simplifying the transition from cardiovascular risk assessment to personalized LDL-C goals, the framework aims to reduce fragmentation of cardiovascular risk management, minimize clinical inertia, and facilitate the delivery of evidence-based lipid-lowering therapy. Prospective validation is now required to determine whether this implementation strategy improves LDL-C goal attainment, cost-effectiveness, and ultimately, cardiovascular outcomes.

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