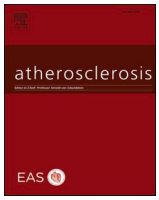
Atherosclerosis 391 (2024) 117482

Contents lists available at ScienceDirect

Atherosclerosis

journal homepage: www.elsevier.com/locate/atherosclerosis



Tracking of serum lipid levels from childhood to adulthood: Systematic review and meta-analysis

Oliver Stanesby [a,b], Matthew K. Armstrong [c], Petr Otahal [a], James P. Goode [a], Brooklyn J. Fraser [a,d], Kazuaki Negishi [e,f], Tetsuhiro Kidokoro [d,g], Tania Winzenberg [a], Markus Juonala [h,i], Feitong Wu [b,j], Rebecca K. Kelly [k], Bo Xi [l], Jorma S.A. Viikari [h,i], Olli T. Raitakari [m,n,o], Stephen R. Daniels [p], Grant R. Tomkinson [d], Costan G. Magnussen [b,d,m,n,*]

[a] *Menzies Institute for Medical Research, University of Tasmania, Hobart, Australia*
[b] *Baker Heart and Diabetes Institute, Melbourne, Australia*
[c] *Department of Health and Human Physiology, University of Iowa, USA*
[d] *Alliance for Research in Exercise, Nutrition and Activity (ARENA), Allied Health and Human Performance, University of South Australia, Adelaide, Australia*
[e] *Sydney Medical School Nepean, Faculty of Medicine and Health, Charles Perkins Centre Nepean, The University of Sydney, Sydney, Australia*
[f] *Nepean Hospital, Sydney, Australia*
[g] *Faculty of Sport Science, Nippon Sport Science University, Tokyo, Japan*
[h] *Department of Medicine, University of Turku, Turku, Finland*
[i] *Division of Medicine, Turku University Hospital, Turku, Finland*
[j] *Baker Department of Cardiometabolic Health, Faculty of Medicine, Dentistry and Health Sciences, University of Melbourne, Melbourne, Australia*
[k] *School of Medicine, University of Tasmania, Hobart, Australia*
[l] *Department of Epidemiology, School of Public Health/Qilu Hospital, Cheeloo College of Medicine, Shandong University, Jinan, China*
[m] *Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, Turku, Finland*
[n] *Centre for Population Health Research, University of Turku and Turku University Hospital, Turku, Finland*
[o] *Department of Clinical Physiology and Nuclear Medicine, Turku University Hospital, Turku, Finland*
[p] *Department of Pediatrics, University of Colorado School of Medicine, Aurora, CO, USA*

ARTICLE INFO

Keywords:
Lipids
Cholesterol
Triglycerides
Child
Adolescent
Systematic review
Meta-analysis
Cohort studies

ABSTRACT

Background and aims: The utility of lipid screening in pediatric settings for preventing adult atherosclerotic cardiovascular diseases partly depends on the lifelong tracking of lipid levels. This systematic review aimed to quantify the tracking of lipid levels from childhood and adolescence to adulthood.
Methods: We systematically searched MEDLINE, Embase, Web of Science, and Google Scholar in March 2022. The protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO; ID: CRD42020208859). We included cohort studies that measured tracking of lipids from childhood or adolescence (<18 years) to adulthood (≥18) with correlation or tracking coefficients. We estimated pooled correlation and tracking coefficients using random-effects meta-analysis. Risk of bias was assessed with a review-specific tool.
Results: Thirty-three studies of 19 cohorts (11,020 participants) were included. The degree of tracking from childhood and adolescence to adulthood differed among lipids. Tracking was observed for low-density lipoprotein cholesterol (pooled r = 0.55–0.65), total cholesterol (pooled r = 0.51–0.65), high-density lipoprotein cholesterol (pooled r = 0.46–0.57), and triglycerides (pooled r = 0.32–0.40). Only one study included tracking of non-high-density lipoprotein cholesterol (r = 0.42–0.59). Substantial heterogeneity was observed. Study risk of bias was moderate, mostly due to insufficient reporting and singular measurements at baseline and follow-up.
Conclusions: Early-life lipid measurements are important for predicting adult levels. However, further research is needed to understand the tracking of non-high-density lipoprotein cholesterol and the stability of risk classification over time, which may further inform pediatric lipid screening and assessment strategies.

* Corresponding author. Baker Heart and Diabetes Institute, Melbourne, Australia.
E-mail addresses: costan.magnussen@baker.edu.au, cosmag@utu.fi (C.G. Magnussen).

https://doi.org/10.1016/j.atherosclerosis.2024.117482
Received 12 December 2023; Received in revised form 15 February 2024; Accepted 15 February 2024
Available online 27 February 2024

1. Introduction

Dyslipidemia refers to a group of disorders characterized by abnormalities in the blood levels of lipids including elevated total cholesterol, low-density lipoprotein (LDL) cholesterol, non-high-density lipoprotein (non-HDL) cholesterol or triglycerides, or depressed high-density lipoprotein (HDL) cholesterol [1–3]. Dyslipidemia is a major risk factor for atherosclerotic cardiovascular disease (ASCVD) [4–9]. Though symptoms of ASCVD often do not appear until middle or late adulthood, the disease process can begin in childhood or earlier [10–17]. This early onset underscores the importance of primordial prevention and early intervention to slow or potentially reverse disease progression [18–23]. Therefore, screening of lipid levels in childhood and adolescence has been recommended to help curtail ASCVD, although recommendations are inconsistent [18,20,21,24–39] and controversial [40,41].

Over the past 40 years, longitudinal studies have assessed the degree to which lipid levels *track*—a term used to describe how measurements of the same variable in the same individuals change, or are stable, over time [13,18]. A recent analysis showed that most of the effect of child risk factors, including total cholesterol and triglycerides, on adult ASCVD events was mediated by the adult risk factors, suggesting that tracking of risk factors accounts for a large portion of the association between child risk factor levels and subsequent ASCVD outcomes [8]. Thus, the degree of tracking from childhood to adulthood is a key consideration in the evidence review process that informs pediatric lipid screening recommendations [20,21,25,28]. However, the one existing systematic review, a commissioned report for the United States Preventive Services Task Force up until September 2005, is out of date and did not include a meta-analysis [42]. Given the need for an up-to-date and comprehensive analysis of lipid tracking evidence, we conducted a systematic review and meta-analysis to determine how conventional lipid levels track from childhood and adolescence into adulthood.

2. Materials and methods

The review protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO; registration number: CRD42020208859). It was designed, conducted, and reported in alignment with systematic review best practices, including the PRISMA Statement 2020 (Supplemental Tables 1–2), Cochrane Handbook, Embase Guide, and University of Tasmania library guides [43–47]. This review analysed publicly available (published) aggregate data; therefore, consent for its reuse was not deemed necessary.

2.1. Eligibility criteria

Eligible studies were cohort studies that measured the degree of tracking of conventional lipid levels (total cholesterol, HDL cholesterol, LDL cholesterol, non-HDL cholesterol, or triglycerides) from childhood or adolescence (<18 years; baseline) to adulthood (≥18 years; follow-up) with a correlation coefficient or a tracking coefficient (which is comparable but not equivalent to a correlation coefficient [48]). The full eligibility criteria are described in Table 1.

2.2. Information sources

We identified studies by searching electronic databases, reference lists, topical reviews, and authors' personal databases. MEDLINE (via Ovid), Embase (via Ovid), Web of Science (Core Collection), and Google Scholar (first 200 results sorted by relevance) databases were searched for all records published until March 30, 2022 by a single researcher (Stanesby). We applied Bramer and colleagues' recommended optimal combination of electronic databases [49] and designed the search strategy in consultation with University of Tasmania library staff experienced in electronic literature searching.

2.3. Search strategy

The search strategy aimed to capture cohort studies that mentioned lipids, childhood or adolescence, adulthood, and tracking. Subject headings, keywords, titles, and abstracts (full text for Google Scholar) were searched. The full search strategy applied to each database is described in Supplemental Table 3.

2.4. Selection

Studies retrieved by our search were imported into Covidence online systematic review software (Veritas Health Innovation, Melbourne, VIC, Australia) and de-duplicated. Two researchers (Stanesby and Armstrong) independently screened the titles and abstracts against the eligibility criteria, then screened the full texts that passed title/abstract screening. Conflicts were resolved by an adjudicating vote from a third researcher (Magnussen).

2.5. Data extraction

Relevant data were extracted by a single researcher (Stanesby). Unreported information (missing data) was sought in other published material about the studies. A second researcher (Goode) verified the data extracted from 20% of the full texts (with 99% agreement and all disagreements being minor). Discrepancies were reviewed by three researchers (Stanesby, Goode, and Magnussen) until a consensus was reached, and the data were corrected if necessary. Extracted data were recorded and stored in Microsoft Excel (Microsoft, Redmond, WA, USA).

2.6. Data items and effect measures

The following information was extracted about each study: study

Table 1
Eligibility criteria.

Inclusion criteria
- Studies in humans.
- Cohort study (follow-up, longitudinal, prospective, or retrospective).
- Cohort/sample aged <18 years at baseline and ≥18 years at follow-up.[a]
- Lipid/lipoprotein levels (specifically HDL cholesterol, LDL cholesterol, non-HDL cholesterol, total cholesterol, or triglycerides) measured at baseline and follow-up via laboratory measurement of concentrations in fasted or non-fasted samples of serum, plasma or blood, or extraction from medical record, or calculation of LDL cholesterol or non-HDL cholesterol from such measurements.
- Quantified the degree of tracking of serum lipid/lipoproteins from baseline to follow-up with a correlation coefficient (i.e., Pearson, Spearman, Kendall, partial) or a tracking coefficient (via generalized estimating equations or a multivariate analysis of a banded model [48]).
- Full text empirical research article.

Exclusion criteria
- Studies that specifically focus on populations with known disease or special interest groups (e.g., obese children, people with familial hypercholesterolemia, and cancer patients).
- All study types other than cohort (e.g., randomized controlled trial, case-control, cross-sectional, review, qualitative, non-empirical).

HDL, High-density lipoprotein. LDL, Low-density lipoprotein.
[a] Entire sample, or at least one eligible estimate from a subgroup, all <18 years at baseline and all ≥18 years at follow-up.

name, study design, and country where data were collected. The following information was extracted about each eligible tracking estimate: type of lipid, effect estimate, tracking effect measure, variables used for adjustment (if any), sample size (total and by sex), sex, ethnicity (if stratified by ethnicity), age at baseline, length of follow-up, calendar years at baseline and follow-up, fasting status at baseline and follow-up as defined by the authors, number of measurements used to derive levels at baseline and follow-up, and measurement methods (e.g., assay, calculations).

2.7. Risk of bias assessment

A review-specific tool was developed to assess the risk of bias in studies (Supplemental Methods 1). The tool, modified from the Newcastle-Ottawa Scale for assessing the quality of cohort studies [50], comprised eight items and five domains: one item on selection bias, one item on attrition bias, three items on measurement bias, two items on confounding bias, and one item on conflict of interest bias. Studies were awarded a maximum of one point for each item. Two researchers (Stanesby and Fraser) independently assessed the risk of bias in the studies, with other researchers (Kidokoro and Negishi) assessing one non-English study. Discrepancies were reviewed and resolved by three researchers (Stanesby, Fraser, and Magnussen).

2.8. Synthesis methods

Analyses were stratified by type of lipid, type of coefficient, and whether the estimate was adjusted for covariates. Parametric and non-parametric analyses of triglycerides were separated because the typical distribution of triglyceride levels is right-skewed [51–53].

Descriptive statistics were used to summarize and compare the characteristics of the included studies and cohorts. DerSimonian-Laird random-effects meta-analysis was used to estimate the degree of tracking of lipids from childhood/adolescence to adulthood, quantified as pooled correlation or tracking coefficients, weighted by the inverse of the standard error of the effect estimate. Estimates were Z-transformed for meta-analysis to allow normal distributional approximation methods during pooling in meta-analysis and meta-regression, and back-transformed for interpretation. Analyses were conducted at the cohort-level, meaning that each unique cohort contributed up to one observation per meta-analysis. When available, the overall effect estimate for the cohort was used in meta-analysis. If no overall effect estimate was reported, the weighted mean of the subgroup estimates was used (the sample size pertaining to the calculated weighted means was the number of unique participants in the subgroups) [54]. Forest plots visually displayed the cohort-level and pooled effect estimates and their 95% confidence intervals (CIs).

Statistical (between-cohort) heterogeneity was quantified by the I^2 statistic and interpreted according to the Cochrane Handbook guide [55]. Univariable random-effects meta-regression analyses explored potential sources of between-cohort heterogeneity (pre-specified in the protocol, PROSPERO ID: CRD42020208859): sex (% male), baseline age, follow-up age, length of follow-up, calendar year of follow-up, region (insufficient variation for inclusion), fasting measurement, assay change from baseline to follow-up, number of measurements (blood draws) at each timepoint (insufficient variation for inclusion), and risk of bias assessment score.

All analyses were performed with Stata version 17 (StataCorp, College Station, TX, USA).

2.9. Reporting bias assessment

Publication bias was visually examined by funnel plots and statistically examined by Egger's test for funnel plot asymmetry (if study $n \geq 10$) [56,57]. If evidence of publication bias was found, sensitivity analyses examined results obtained after imputation of potentially missing studies due to publication bias with the non-parametric trim-and-fill method (rightmost-run and linear estimators).

2.10. Certainty assessment

Certainty in the body of evidence was assessed using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach [58,59]. This widely-endorsed approach grades the certainty of the evidence (high, moderate, low, and very low; defined in Supplemental Table 4) by considering the risk of bias in the studies, the precision of the estimate (confidence interval), the consistency of the study estimates (heterogeneity), the risk of publication or reporting biases, and how directly the evidence answers the question of interest.

3. Results

3.1. Study selection

The flow of studies through each phase of screening is shown in Fig. 1. After the removal of 1467 duplicates, 6105 unique studies were screened for eligibility, of which 6016 were excluded during title and abstract screening. The full texts of the remaining 89 studies were assessed during which another 56 studies were determined to be ineligible. The most frequent reasons for exclusion were: did not use required effect measures (n = 16); baseline measurement was not in childhood/adolescence (n = 10); follow-up measurement was not in adulthood (n = 9). Thirty-three studies were included (Supplemental Table 5) [48,60–91].

3.2. Study characteristics

This review extracted 483 lipid tracking estimates from 33 studies of 19 unique cohorts of 25–2446 participants, totaling 11,020 unique participants who provided data between 1967 and 2018 (Supplemental Table 6). The studies and cohorts are described in Table 2. The following cohorts were represented in multiple studies: Bogalusa Heart Study (6/33 studies), Cardiovascular Risk in Young Finns Study (4/33), Amsterdam Growth and Health Longitudinal Study (3/33), Beaver County Lipid Study (3/33), and Muscatine Study (3/33) (Supplemental Table 5). Of the 19 cohorts, six were from the United States, six from Europe, five from Asia, and two from Australia. Tracking of total cholesterol was assessed in all 19 cohorts, HDL cholesterol in 11, triglycerides in 10, LDL cholesterol in seven, and non-HDL cholesterol in one (Table 2). Three studies of non-HDL cholesterol were excluded in full text screening because the baseline sample included adults or tracking was not assessed by correlation or tracking coefficients [92–94]. Age at baseline varied but only one cohort had a mean age below 5 years at baseline. Length of follow-up ranged from 2 to 27 years. Tracking was measured with a correlation coefficient in all except one cohort (Pearson: eight cohorts, Spearman: seven cohorts, type unclear: six cohorts), and with a tracking coefficient derived using generalized estimating equations in three cohorts [48,73,85]. Most cohorts were fasted at both baseline and follow-up (14/19). Lipid levels were almost always derived from a single blood draw (17/19 cohorts), and only one cohort derived levels from multiple blood draws (at least a day apart) that were averaged.

3.3. Risk of bias in studies

Risk of bias scores are summarized in Table 2 and described in detail in Supplemental Table 7. All studies were assessed as having a moderate or high overall risk of bias (mean score = 4.9 out of 8), with a high risk of bias due to sample selection, attrition, measurement and potential conflicts of interest, and a low risk of bias due to confounding. Insufficient reporting contributed to the overall risk of bias, with studies providing insufficient detail for 34% of the risk of bias items (58% for representativeness of sample, 33% for adequacy of follow-up, 52% for

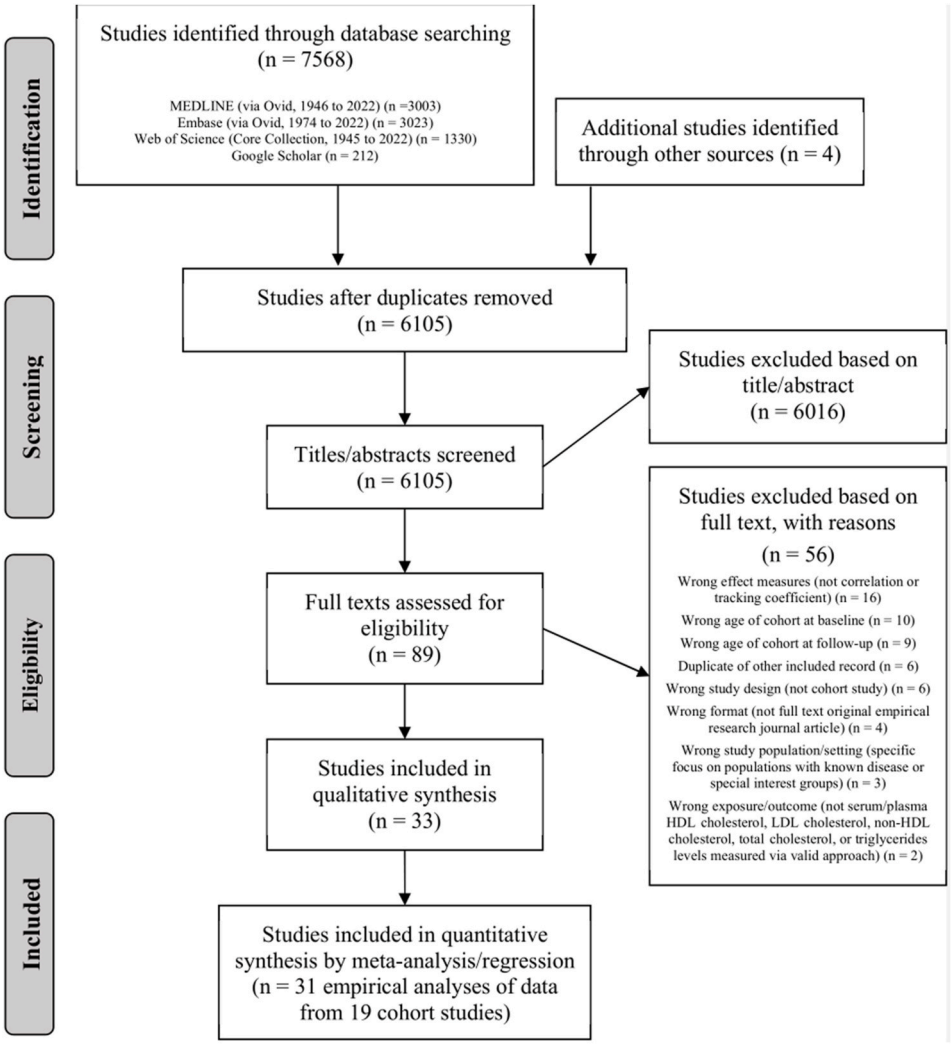


Fig. 1. Phases of study selection.
Databases searched: March 30, 2022; Figure adapted from the PRISMA statement [43]; Google scholar: Retrieved first 200 sorted by relevance on September 30, 2020 plus those published since 2020 in first 200 sorted by relevance on March 30, 2022; HDL: high-density lipoprotein; LDL: low-density lipoprotein.

lipid measurement protocols, 6% for number of measurements at timepoints, 24% for fasting status, 9% for confounding, 82% for conflicts of interest).

3.4. Results of syntheses

Positive correlations and tracking coefficients were observed for total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides, demonstrating tracking from childhood/adolescence to adulthood (Table 3 and Supplemental Fig. 1). The strongest tracking was observed for LDL cholesterol (range of the pooled unadjusted and adjusted correlation and tracking coefficients, r = 0.55–0.65) and total cholesterol (range of r = 0.51–0.65), with slightly weaker tracking of HDL cholesterol (range of r = 0.46–0.57), and substantially weaker tracking of triglycerides (range of r = 0.32–0.40). Meta-analysis of non-HDL cholesterol tracking was not possible because it was reported in a single study. That study reported non-HDL cholesterol correlation/tracking coefficients of r = 0.42 to 0.59 [73]. The pooled estimates varied by the type of coefficient and whether estimates were adjusted for covariates which is reflected in the ranges reported. There was evidence of substantial heterogeneity between cohorts for tracking of total cholesterol (range of I^2 for the pooled coefficients = 67–84%), and negligible to substantial heterogeneity for tracking of LDL cholesterol (range of I^2 = 20–87%), HDL cholesterol (range of I^2 = 1–88%) and triglycerides (range of I^2 = 21–85%).

Sensitivity analysis found no material change in the pooled results when excluding the estimates derived from follow-up periods that were shorter than 5 years (Supplemental Table 8). There were no statistically significant sex differences in lipid tracking (Supplemental Table 9).

Table 4 describes the results from meta-regression analyses investigating sources of between-cohort heterogeneity in reported tracking of total cholesterol and HDL cholesterol (data were insufficient for LDL cholesterol, non-HDL cholesterol, and triglycerides). The risk of bias score explained most of the between-cohort variation in tracking of total cholesterol (adjusted R^2 = 69%), and whether a different assay was used at follow-up than at baseline explained 40%; studies with lower risk of bias and studies that used the same assay at all timepoints tended to report higher tracking of total cholesterol. The length of follow-up, age at baseline and follow-up, calendar year, sex, and fasting status of the samples did not contribute to between-cohort variations in tracking of total cholesterol. The sex and baseline age of the samples each explained some of the between-cohort variation in tracking of HDL cholesterol (adjusted R^2 = 38% for sex and 34% for baseline age); studies of older cohorts at baseline and studies with a higher proportion of females tended to report higher tracking of HDL cholesterol. The age at follow-up, length of follow-up, calendar year, risk of bias score and whether a

Table 2
Characteristics of the 19 cohorts (from 33 studies).

	n (%)[a]
Lipids measured	
Total cholesterol	19 (100%)
HDL cholesterol	11 (58%)
LDL cholesterol	7 (37%)
Non-HDL cholesterol	1 (5%)
Triglycerides	10 (53%)
Tracking effect measure	
Pearson correlation coefficient	7 (37%)
Spearman correlation coefficient	7 (37%)
Partial Pearson correlation coefficient	1 (5%)
Partial Spearman correlation coefficient	1 (5%)
Partial correlation coefficient (type unclear)	4 (21%)
Tracking coefficient unadjusted[b]	3 (16%)
Tracking coefficient adjusted[b]	2 (11%)
Correlation coefficient, but type unclear	3 (16%)
Sex	
Males & females combined	13 (68%)
Males	10 (53%)
Females	10 (53%)
Baseline age[c]	
<5 years	1 (5%)
5–9 years	7 (37%)
10–14 years	15 (79%)
15–17 years	9 (47%)
Follow-up age[c]	
18–24 years	13 (68%)
25–29 years	10 (53%)
30–34 years	5 (26%)
≥35 years	6 (32%)
Length of follow-up[c]	
<5 years	3 (16%)
5–9 years	9 (47%)
10–14 years	11 (58%)
15–19 years	6 (32%)
≥20 years	7 (37%)
Calendar year of follow-up[c]	
1970s	5 (26%)
1980s	9 (47%)
1990s	5 (26%)
2000s	7 (37%)
≥2010s	3 (16%)
Region	
Asia	5 (26%)
Australia	2 (11%)
Europe	6 (32%)
United states	6 (32%)
Fasting measurement	
Fasted at baseline and follow-up	14 (74%)
Not fasted at baseline and follow-up	4 (21%)
Fasted at baseline but not follow-up	0 (0%)
Fasted at follow-up but not baseline	1 (5%)
Assay change from baseline to follow-up	
No	13 (68%)
Yes	8 (42%)
Number of measurements at each timepoint	
Single blood draw	17 (89%)
Multiple blood draws (≥1 day apart)	1 (5%)
	Mean (SD, min–max)
Sample size (at follow-up)	
N	580 (676, 25–2446)
Risk of bias (higher score = higher risk of bias)	
Selection bias [scored 0–1]	0.6 (0.5, 0–1)
Attrition bias [scored 0–1]	0.6 (0.4, 0–1)
Measurement bias [scored 0–3]	2.1 (0.6, 1–3)
Confounding bias [scored 0–2]	0.8 (0.7, 0–2)
Conflicts of interest bias [scored 0–1]	0.7 (0.4, 0–1)
Total risk of bias [scored 0–8]	4.9 (1.1, 3–8)

HDL, High-density lipoprotein. LDL, Low-density lipoprotein. SD, Standard deviation.

[a] Sum of rows for a variable may exceed n = 19 or 100% because cohorts with stratified analyses may apply to multiple categories.

[b] Via generalized estimated equations or multivariate analysis of a banded model [48].

[c] Sample/subsample mean (median if mean not reported).

different assay was used at follow-up than at baseline did not contribute to between-cohort variations in tracking of HDL cholesterol.

3.5. Reporting biases

There was no visual (Fig. 2) or statistical evidence of publication bias in the reporting of tracking of total cholesterol ($p = 0.49$) and HDL cholesterol ($p = 0.33$), and there were insufficient studies of LDL cholesterol (n = 6) and non-HDL cholesterol (n = 1) to test for it. There was minor visual and statistical evidence of publication bias in the reporting of the tracking of triglycerides ($p = 0.03$), with a deficit of small studies reporting a large effect size; although the change in results by imputation of potentially missing studies was negligible (linear estimator: identical; run estimator: <10% change in effect).

3.6. Certainty of evidence

Overall, there was moderate to high confidence in the pooled estimates and the true tracking of each lipid is likely to be close to the estimates, but there is a possibility that they are different. The GRADE rating was downgraded because there was moderate to high risk of bias in the studies, there was evidence of substantial variation between studies, and the evidence did not assess the stability of risk classification. However, the downgrade was partial because the risk of bias in studies was often due to insufficient detail in reporting, the level of heterogeneity could not be precisely estimated due to wide confidence intervals for I^2, the evidence directly answers the question of interest, and there was no evidence of publication biases found, except for negligible evidence related to triglycerides.

4. Discussion

This systematic review is the most comprehensive to date, including 25 studies and 12 additional cohorts that were not included in the previous review from 2005 [42]. It also represents the only meta-analysis to evaluate the extent of conventional lipid level tracking from childhood or adolescence to adulthood. Our findings suggest that child and adolescent total cholesterol, LDL cholesterol, and HDL cholesterol track strongly, and triglycerides track moderately, into adulthood when compared to other cardiovascular disease risk factors (F 3) [95–98]. We have moderate to high confidence in these estimates and the true tracking of each lipid is likely to be close to the estimates, but they may differ due to risk of bias, heterogeneity, and limited data for some lipids. Despite its relevance, non-HDL cholesterol could not be included in the meta-analysis since only one eligible study reported the necessary tracking data.

The included studies demonstrated a moderate to high overall risk of bias. This was mostly due to insufficient detail reported on sample selection, lipid measurement protocols, adequacy of follow-up, and conflicts of interest, as well as a single blood draw and measurement at baseline and follow-up. We found no evidence of publication biases, except for negligible evidence related to triglycerides. A common source of bias was attrition, with many studies not considering potential differential loss to follow-up or statistical approaches to mitigate it. When we examined the main meta-analysis, we found evidence of substantial variation between studies. However, due to wide confidence intervals that usually included the null, we could not precisely estimate the level of heterogeneity. Interestingly, our analysis of heterogeneity revealed that studies with lower risk of bias and those that used the same assay at all timepoints tended to report higher tracking of total cholesterol. This suggests that the degree of total cholesterol tracking may be higher than estimated by our meta-analysis. Lastly, it is worth noting that while screening guidelines recommend calculating levels from two lipid profile measurements to limit measurement error due to within-person variability [21,92,99,100], only one study adhered to this practice. This finding implies that our meta-analysis may underestimate the

Table 3
Random-effects meta-analysis of tracking of lipids from childhood/adolescence to adulthood.

	n cohorts (participants)	Pooled coefficient	CI		I^2 (CI)
			Lower	Upper	
Total cholesterol					
Correlation coefficient					
Unadjusted	15 (8969)	0.51	0.48	0.55	72% (3%, 87%)
Adjusted	6 (3210)	0.51	0.45	0.57	72% (0%, 91%)
Tracking coefficient					
Unadjusted	3 (709)	0.65	0.56	0.72	67% (0%, 91%)
Adjusted	2 (613)	0.65	0.50	0.76	84% (0%, 97%)
HDL cholesterol					
Correlation coefficient					
Unadjusted	8 (4093)	0.46	0.38	0.54	88% (15%, 96%)
Adjusted	3 (338)	0.57	0.48	0.66	14% (0%, 77%)
Tracking coefficient					
Unadjusted	3 (709)	0.54	0.49	0.59	1% (0%, 73%)
Adjusted	2 (613)	0.54	0.46	0.61	40% (0%, 88%)
LDL cholesterol					
Correlation coefficient					
Unadjusted	5 (3459)	0.55	0.47	0.62	87% (0%, 96%)
Adjusted	2 (290)	0.65	0.40	0.81	66% (0%, 93%)
Tracking coefficient					
Unadjusted	2 (528)	0.59	0.52	0.66	20% (0%, 84%)
Adjusted	1 (432)	Insufficient cohorts (r = 0.56, SE = 0.05)			
Non-HDL cholesterol					
Correlation coefficient					
Unadjusted	1 (432)	Insufficient cohorts (r = 0.42, SE = 0.05)			
Adjusted	0 (0)	Insufficient cohorts			
Tracking coefficient					
Unadjusted	1 (432)	Insufficient cohorts (r = 0.56, SE = 0.05)			
Adjusted	1 (432)	Insufficient cohorts (r = 0.56, SE = 0.05)			
Triglycerides					
Parametric					
Correlation coefficient					
Unadjusted	4 (3574)	0.40	0.30	0.49	85% (0%, 96%)
Adjusted	1 (265)	Insufficient cohorts (r = 0.29, SE = 0.06)			
Tracking coefficient					
Unadjusted	0 (0)	Insufficient cohorts			
Adjusted	0 (0)	Insufficient cohorts			
Non-parametric					
Correlation coefficient					
Unadjusted	5 (3469)	0.32	0.28	0.35	21% (0%, 72%)
Adjusted	1 (25)	Insufficient cohorts (r = 0.41, SE = 0.21)			
Tracking coefficient					
Unadjusted	0 (0)	Insufficient cohorts			
Adjusted	0 (0)	Insufficient cohorts			
Unclear					
Correlation coefficient					
Unadjusted	1 (1804)	Insufficient cohorts (r = 0.59, SE = 0.02)			
Adjusted	1 (48)	Insufficient cohorts (r = 0.54, SE = 0.15)			
Tracking coefficient					
Unadjusted	1 (432)	Insufficient cohorts (r = 0.40, SE = 0.05)			
Adjusted	1 (432)	Insufficient cohorts (r = 0.39, SE = 0.05)			

CI, 95% confidence interval. I^2, Percentage of total variability that is due to between-cohort heterogeneity. r, Correlation or tracking coefficient (weighted by sub-sample size if multiple estimates for cohort). SE, standard error. HDL, high-density lipoprotein. LDL, low-density lipoprotein. Unadjusted, Correlation coefficient or unadjusted tracking coefficient. Adjusted, Partial correlation or adjusted tracking coefficient. Parametric, Pearson. Non-parametric, Spearman rank-order correlation coefficient. Unclear, distribution assumptions of analysis unclear.
Pooled across cohorts using DerSimonian-Laird random-effects model (all pooled effects $p < 0.001$).

strength of tracking due to measurement error (high within-person variability) and that the true value may be higher in practice where current guidelines recommend multiple measurements.

Our review concentrated on the tracking of continuous lipid levels estimated from correlation and tracking coefficients. This approach allowed us to assess the consistency of individuals' relative positions (rank or level) over time [73,74,81]. However, we did not examine the tracking of categorical risk factor levels, which assesses the stability of risk classification over time—information that is more clinically useful [101]. Our focus on continuous levels was necessary to allow meta-analysis because cut-offs used to denote high risk vary widely across studies [42,60–63,65,67,69,72,74,76,78,80–82,84,85,89–93,101–118]. To define high risk, studies often arbitrarily use the upper quartile, quintile or other percentile of the sample distribution. Others occasionally apply pre-specified absolute thresholds for risk in adulthood, while a few use pre-specified absolute thresholds for risk in childhood or adolescence. This variability likely reflects a lack of consensus on cut-offs for lipid levels in childhood and adolescence, particularly before the 2011 National Heart, Lung, and Blood Institute-sponsored guidelines—when a high proportion of the papers identified through this review were published [20].

The primary lipid focus of ASCVD risk reduction in the pediatric setting has traditionally been on LDL cholesterol due, in part, to its strong predictive power for ASCVD risk in adults, its use to indicate familial hypercholesterolemia, and its superior tracking to other conventional lipids [20,21,25,28,42,119–125]—a notion supported by our

Table 4
Random-effects univariable linear meta-regression: Association between degree of tracking of lipids from childhood/adolescence to adulthood and study/sample characteristics.

	Total cholesterol (correlation coefficient, unadjusted)			HDL cholesterol (correlation coefficient, unadjusted)		
	n cohorts (participants)	B (CI)	Residual I^2 | Adjusted R^2	n cohorts (participants)	B (CI)	Residual I^2 | Adjusted R^2
Sex [per +10% male]	13 (8467)	−0.02 (−0.13, 0.10)	76% | −13%	6 (3591)	−0.17 (−0.35, 0.01) $^{p = 0.07}$	85% | 38%
Baseline age [per +1 year][a]	15 (8969)	0.00 (−0.02, 0.03)	72% | −8%	8 (4093)	0.04 (0.00, 0.08) $^{p = 0.05}$	88% | 34%
Follow-up age [per +10 year][a]	15 (8969)	−0.06 (−0.15, 0.03) $^{p = 0.18}$	68% | 8%	8 (4093)	0.11 (−0.09, 0.29)	86% | 14%
Length of follow-up [per +10 year][a]	15 (8969)	−0.06 (−0.13, 0.02) $^{p = 0.17}$	68% | 9%	8 (4093)	0.03 (−0.22, 0.27)	88% | −13%
Calendar year of follow-up [per +10 year][a]	15 (8969)	−0.01 (−0.05, 0.03)	73% | −10%	8 (4093)	−0.03 (−0.13, 0.08)	90% | −15%
Fasting status [unfasted at baseline or follow-up]	14 (8670)	0.06 (−0.06, 0.17)	73% | 1%		Insufficient data	
Assay change [from baseline to follow-up]	14 (8670)	−0.10 (−0.19, −0.02) $^{p = 0.02}$	64% | 40%	8 (4093)	0.00 (−0.24, 0.24)	89% | −21%
Total risk of bias [per +1 score]	15 (8969)	−0.05 (−0.07, −0.02) $^{p = 0.001}$	31% | 69%	8 (4093)	−0.02 (−0.12, 0.09)	89% | −13%

B, unstandardized beta coefficient from univariable meta-regression. CI, 95% confidence interval. HDL, high-density lipoprotein. Residual I^2, Residual variation due to heterogeneity. Adjusted R^2, Proportion of between-cohort variance explained by model. *p* values test the null hypothesis that the coefficient is equal to zero and are provided in superscript when <0.2.
Outcome variables are pooled correlation coefficients (unadjusted).
[a] Central tendency (mean, or median if mean not reported).

review. However, there is increasing emphasis on non-HDL cholesterol for ASCVD health and risk reduction in children [20,21,25,28] given its superior predictive utility to most conventional lipids (including LDL cholesterol) [20,122–126] and it is accurately measured without fasting or added cost [120,122–125,127–137]. Still, our review found limited data on the tracking of non-HDL cholesterol from childhood/adolescence to adulthood, with only one study providing a direct comparison to LDL cholesterol and none to apolipoprotein B [73]. Additionally, a related systematic review [138] found only two studies [139,140] with data on the tracking of apolipoprotein B from childhood/adolescence to adulthood (non-HDL cholesterol includes all apolipoprotein B-containing lipoproteins [141–143]). This shortfall in evidence emphasizes the need for further research to understand the tracking of non-HDL cholesterol and apolipoprotein B. The potential discordance between non-HDL cholesterol, LDL cholesterol, and apolipoprotein B has clinical relevance for adults [144–147], and may also apply to children and adolescents [126]. Filling these knowledge gaps could further refine considerations for lipid tracking and screening in pediatric populations.

Overall, these results suggest that more data are needed to fully evaluate the case for lipid screening based on tracking. Specifically, we lack comparable data for the stability of risk classification and have limited data on non-HDL cholesterol tracking. Given the very high heritability of HDL cholesterol levels [148,149], the somewhat lower tracking we observed for HDL cholesterol could be attributed to methodological differences in its measurement that have occurred over time [150], physiological changes during puberty, particularly in males [151], or the weakening of the genetic effect with age [152]. Furthermore, the valid estimation of non-HDL cholesterol from standard lipid measurements (non-HDL cholesterol = total cholesterol – HDL cholesterol) [3,120], the existence of standardized pediatric cut-offs to denote high risk [20,21,25,28], and data suitable for individual participant data meta-analysis offer promising avenues for future research. However, the potential reduction in risk of bias that could be achieved by more comprehensive reporting of key details such as sample selection, lipid measurement protocols, and conflicts of interest, along with consideration of differential loss to follow-up should not be overlooked. These needs could be readily addressed by existing cohort studies, implying that the necessary data may already be available but underutilized.

4.1. Strengths and limitations

The design, conduct, and reporting of this systematic review adhered to best practices [43–46] and was performed in consultation with leading experts in the field. Our study provides the most comprehensive synthesis of lipid tracking data to date and is the only one to offer a meta-analysis. We acknowledge several limitations. The available data were not sufficient to adequately evaluate small-study effects for LDL cholesterol and non-HDL cholesterol, or to perform meta-regression for LDL cholesterol, non-HDL cholesterol and triglycerides. Also, our meta-regression may have been underpowered to detect modest differences in tracking of total cholesterol and HDL cholesterol. These limitations highlight important gaps for future research to address. Our definition of childhood/adolescence as being under age 18 years may have resulted in the exclusion of some literature that used alternate age definitions, such as under age 21 years. Additionally, our review did not consider the stability of lipid risk classification over time. We did not a priori consider studies that examined the tracking of apolipoproteins or lipoprotein(a), as the potential clinical utility of these measures over conventional lipid measurements has emerged largely since the longitudinal studies in this review were initiated. However, our search captured a few studies that report tracking of apolipoproteins and lipoprotein(a) and some have emerged since [153]. Accordingly, we conducted a separate systematic review specifically focusing on apolipoprotein B tracking, which found apolipoprotein B levels track strongly during childhood, but to a similar degree as for LDL cholesterol [138].

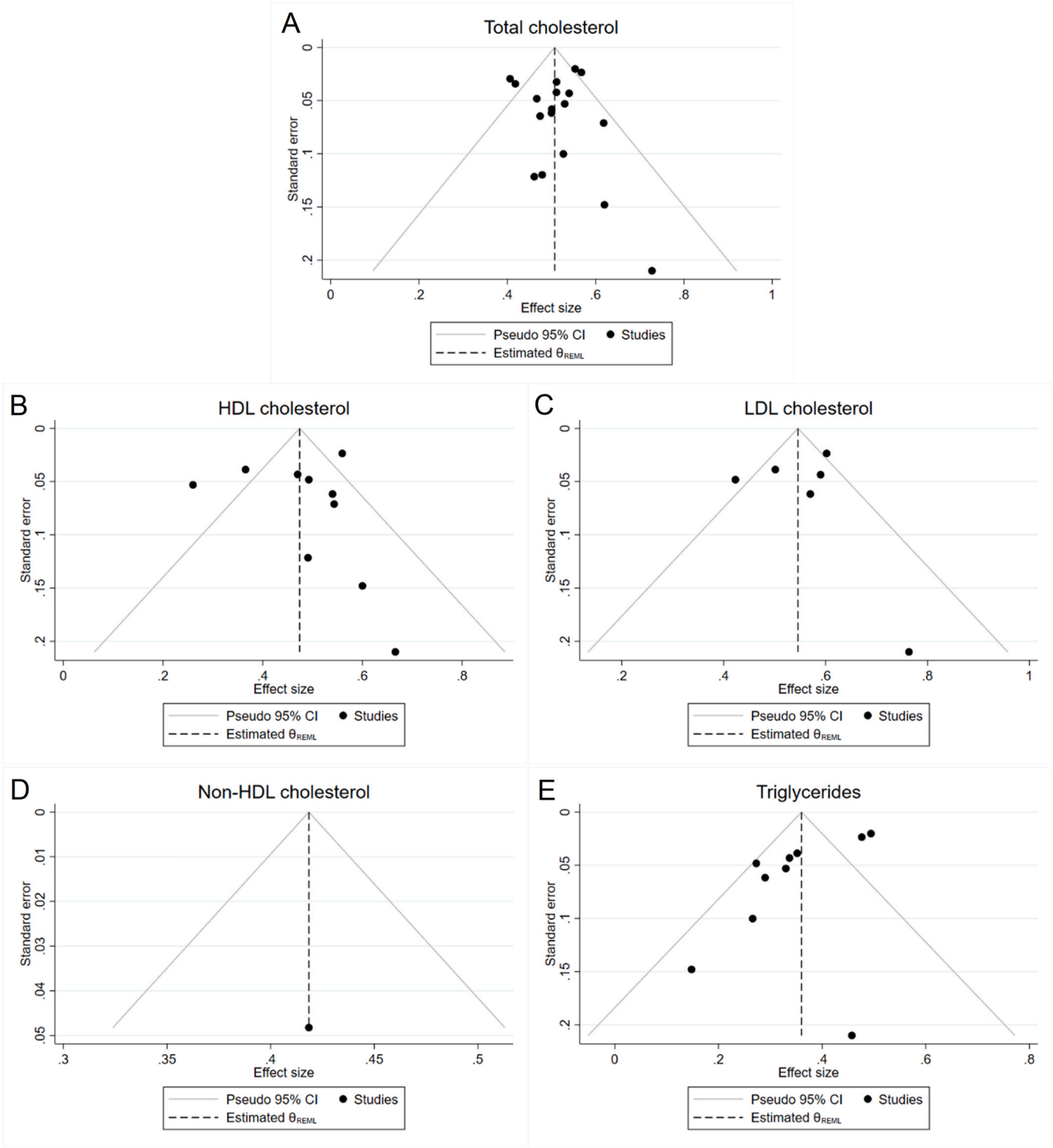


Fig. 2. Funnel plots of the effect size and standard error of tracking of lipids from childhood/adolescence to adulthood.
(A) Total cholesterol. (B) HDL cholesterol. (C) LDL cholesterol. (D) Non-HDL cholesterol. (E) Triglycerides. The circles represent the cohort-specific unadjusted correlation coefficients, the vertical line represents the average, and the dashed triangular region represents the boundary within which 95% of studies are expected to fall in the absence of both biases and heterogeneity. HDL: high-density lipoprotein; LDL: low-density lipoprotein.

However, there is an absence of data on apolipoprotein B tracking to adulthood [138].

4.2. Conclusions

Our systematic review and meta-analysis affirm that lipid levels track from childhood and adolescence into adulthood, with notable differences in the strength of tracking among the various lipids we examined. However, our study also identifies critical gaps in knowledge, particularly regarding the tracking of non-HDL cholesterol. Additionally, there is a comparative lack of data using current, standardized cut-offs for the stability of risk classification over time. Addressing these gaps through

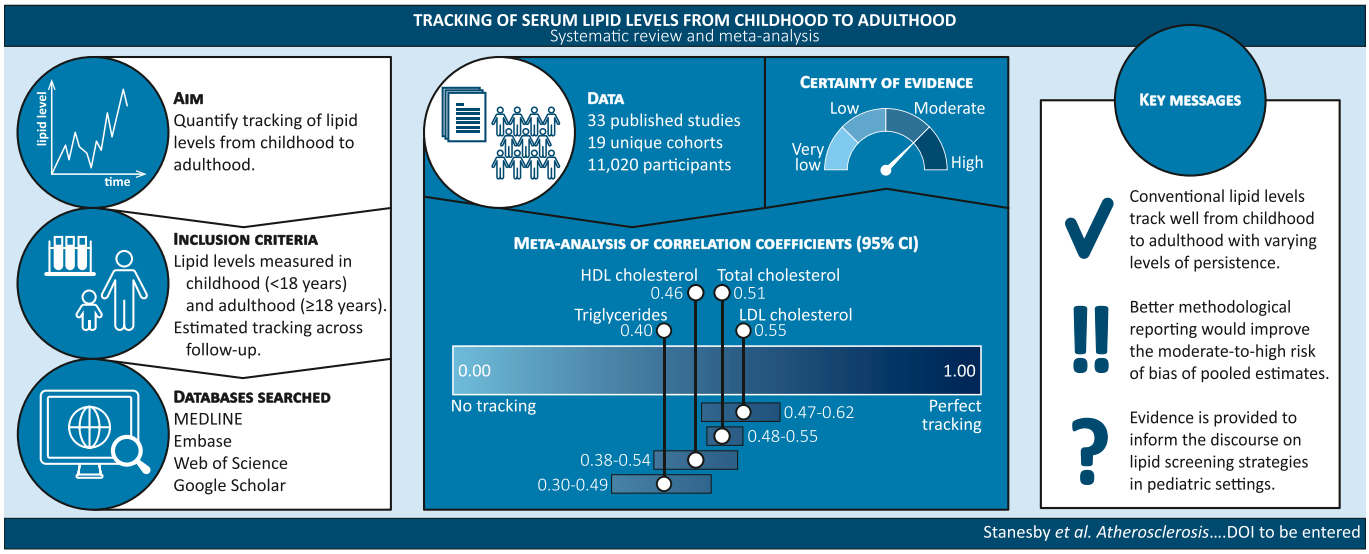


Fig. 3. Graphical abstract.

analysis of existing cohorts would be clinically relevant, potentially informing pediatric lipid screening guidelines and enhancing early ASCVD risk assessment strategies (Fig. 3).

Financial support

This work is supported by a National Health and Medical Research Council (NHMRC) Investigator Grant (APP1176494) for Dr Costan Magnussen. The contents of the published material are solely the responsibility of the individual authors and do not reflect the views of the NHMRC.

CRediT authorship contribution statement

Oliver Stanesby: Conceptualization, Methodology, Formal analysis, Data curation, Writing – original draft, Visualization, Project administration. **Matthew K. Armstrong:** Methodology, Writing – review & editing. **Petr Otahal:** Conceptualization, Methodology, Writing – review & editing, Visualization. **James P. Goode:** Methodology, Writing – review & editing, Visualization. **Brooklyn J. Fraser:** Methodology, Writing – review & editing. **Kazuaki Negishi:** Methodology, Writing – review & editing. **Tetsuhiro Kidokoro:** Methodology, Writing – review & editing. **Tania Winzenberg:** Methodology, Writing – review & editing. **Markus Juonala:** Writing – review & editing. **Feitong Wu:** Writing – review & editing. **Rebecca K. Kelly:** Writing – review & editing. **Bo Xi:** Writing – review & editing. **Jorma S.A. Viikari:** Writing – review & editing. **Olli T. Raitakari:** Writing – review & editing. **Stephen R. Daniels:** Writing – review & editing. **Grant R. Tomkinson:** Conceptualization, Methodology, Writing – review & editing. **Costan G. Magnussen:** Conceptualization, Methodology, Writing – original draft, Writing – review & editing, Visualization, Supervision, Project administration, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

We thank Michaela Venn, *GDip(IT), MSc(*AnimBehavEthol*),* from the University of Tasmania (Research Librarian) for contributing her expertise to the design of the systematic literature search strategy.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.atherosclerosis.2024.117482.

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