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ASSOCIATION BETWEEN EGG CONSUMPTION AND NON-FAMILIAL HYPERCHOLESTEROLEMIA OR ADVERSE LIPID PROFILE IN ADULTS: A SYSTEMATIC REVIEW

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ABSTRACT

Background/Objective: Hypercholesterolemia is a major risk factor for cardiovascular disease, with elevated LDL-C playing a central role in the development of atherosclerosis. The increase in LDL-C concentrations has been directly associated with dietary cholesterol and saturated fat intake. Because eggs are one of the richest dietary sources of cholesterol, their role in cardiovascular health has been widely debated. However, findings regarding the effects of egg consumption on lipid profile markers remain inconsistent. Therefore, the aim of this study was to synthesize the overall evidence from randomized controlled trials on the effect of egg consumption or dietary cholesterol derived from eggs on LDL-C concentrations. **Methods:** A systematic review of RCTs was conducted to evaluate the effects of egg consumption on lipid profile markers following the rules of the PRISMA Statement. The literature search was performed in PubMed for studies published between 2020 and 2026, eligible studies included adult subjects, assessing egg consumption as the main exposure or intervention, and reporting outcomes related to hypercholesterolemia or lipid profile. **Results:** A total of 133 articles were identified with the initial search. Finally, a total of 9 studies were included in the review, and 124 studies were excluded as they did not meet the predefined inclusion criteria. Overall, the lipid-related findings were heterogeneous across the included RCTs, although most studies did not report statistically significant between-group differences in conventional lipid markers, including total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). **Discussion/Conclusion:** Recent RCTs do not show a consistent adverse effect of egg consumption on LDL-C or other lipid markers in adults. However, current evidence does not support definitive conclusions, as responses appear to be context-dependent and influenced by overall dietary patterns and comparator diets. Further well-designed trials are needed to clarify these effects.

Keywords: Egg consumption, Dietary cholesterol, Low-density lipoprotein cholesterol (LDL-C), Lipid profile, Hypercholesterolemia.

ABSTRACT

Context/Objectiu: La hipercolesterolèmia és un factor de risc establert per la malaltia cardiovascular, on l'LDL-C elevat juga un rol central en el desenvolupament de l'arterioesclerosi. L'augment en les concentracions d'LDL-C ha estat directament associat amb la ingesta de colesterol dietari i de greixos saturats. Donat que els ous són una de les fonts alimentàries més riques en colesterol, el seu rol en la salut cardiovascular ha estat àmpliament debatut. Malgrat tot això, les evidències sobre els efectes del consum d'ou sobre marcadors del perfil lipídic continuen sent inconsistents. D'aquesta manera, l'objectiu d'aquest estudi ha estat el de sintetitzar el global de les evidències d'assajos clínics aleatoritzats sobre l'efecte del consum d'ous o colesterol dietari derivat d'ous sobre les concentracions d'LDL-C. **Mètodes**: Una revisió sistemàtica d'ECAs va ser realitzada per tal d'avaluar els efectes del consum d'ous sobre marcadors de perfil lipídic seguint les normatives de PRISMA Statement. La recerca literària va ser realitzada a PubMed per estudis publicats entre el 2020 i el 2026, els estudis aptes van incloure subjectes adults, analitzant com a principal exposició o intervenció el consum d'ous, i com a resultats reportats aquells relacionats a la hipercolesterolèmia o el perfil lipídic. **Resultats**: Un total de 133 articles van ser identificats en la primera cerca inicial. Finalment, un total de 9 estudis van ser inclosos en la revisió, i 124 estudis van ser exclosos, ja que no van complir els criteris d'inclusió predefinits. En total, les evidències trobades en relació amb canvis relacionats als lípids van ser heterogenis en els diferents ECAs, tot i que la majoria dels estudis no van reportar diferències estadísticament significatives entre grups en marcadors convencionals de lípids, incloent-hi el colesterol total (CT), colesterol de lipoproteïnes de baixa densitat (LDL-C), colesterol de lipoproteïnes d'alta densitat (HDL-C), i triglicèrids (TG). **Discussió/Conclusions**: Els ECAs recents no mostren un efecte advers consistentment entre el consum d'ou sobre l'LDL-C o altres marcadors lipídics en adults. Tot i això, l'evidència actual no sosté conclusions definitives, ja que les respostes semblen ser dependents del context i influenciades per patrons dietaris globals i comparadors dietètics. Es necessiten més assaigs ben dissenyats per poder aportar llum sobre aquests efectes.

Paraules clau: Consum d'ous, Colesterol dietètic, Colesterol de lipoproteïnes de baixa densitat (LDL-C), Perfil lipídic, Hipercolesterolèmia.

1 | INTRODUCTION

Hypercholesterolemia is a major contributor to the development of atherosclerosis. While its etiology is multifactorial, it functions as a key permissive factor that allows other cardiovascular risk factors to exert their pathological effects. Notably, elevated cholesterol levels are considered the only necessary condition for plaque formation and thus play a central role in the development of cardiovascular disease (CVD) [1, 2]. According to the most recent Global Burden of Disease analysis, CVDs accounted for 437 million disability-adjusted life years (DALYs) and 19.2 million deaths globally in 2023, with high LDL-C among the leading modifiable contributors to cardiovascular burden [3]. This condition is closely linked to CVD, which remains one of the leading causes of morbidity and mortality worldwide, accounting for approximately one third of all deaths according to the World Health Organization (WHO) [4]. Because these lipid abnormalities are strongly influenced by modifiable exposures, diet is one of the main pathways through which cardiovascular risk may be shaped over time [5].

At the mechanistic level, hypercholesterolemia contributes to the development of atherosclerosis through mechanisms involving lipid accumulation, oxidative stress, and chronic vascular inflammation [6]. LDL particles can infiltrate the arterial wall, where they undergo modification and promote endothelial dysfunction, favoring the recruitment of inflammatory cells. This process results in the formation of lipid-laden foam cells and marks the early stages of plaque development. In addition, hypercholesterolemia is associated with increased production of reactive oxygen species and pro-inflammatory mediators, which further amplify vascular damage and contribute to plaque progression and instability. Altogether, these mechanisms drive vascular injury and the development of atherosclerotic cardiovascular disease [6].

Given this well-established relationship, dietary factors influencing total cholesterol levels have been a central focus of nutritional research and public health recommendations. The European Food Safety Authority (EFSA) published a scientific opinion in 2010 on dietary reference values for fats, concluding that there is a positive dose–response relationship between dietary cholesterol intake and circulating LDL-C concentrations, while also emphasizing the more significant impact of saturated fatty acids on blood LDL-c levels [7].

National dietary guidelines have also addressed recommendations regarding cholesterol intake. For example, the United States Department of Agriculture Dietary Guidelines for Americans 2025–2030 do not provide a specific quantitative limit for dietary cholesterol [8]. However, earlier editions (2020–2025) recommended that cholesterol intake should be kept as low as possible while maintaining nutritional adequacy [9]. Similarly, the American Heart Association, in its latest recommendations

published in 2026, advises limiting the consumption of cholesterol-rich foods as part of a heart-healthy dietary pattern [10]. In this same line, The National Lipid Association reports that dietary cholesterol intake is associated with increases in circulating LDL-C levels and supports the adoption of dietary patterns that are inherently low in cholesterol [11].

Eggs are among the most cholesterol-rich foods and have therefore been the subject of ongoing debate in relation to cardiovascular health. With regard to egg consumption recommendations across European countries, dietary guidelines vary considerably. Countries such as Bulgaria, Ireland, and Finland allow for the consumption of up to one egg per day. In contrast, nations including Belgium, Denmark, Greece, Spain, Italy, Cyprus, Malta, Netherlands, Austria, and Romania recommend a more moderate intake of approximately 3–4 eggs per week. Other European countries, such as France, Sweden, Slovenia, United Kingdom, Switzerland, Iceland, and Norway do not provide specific quantitative recommendations for egg consumption, although they indicate that eggs can be included as part of a healthy and balanced diet. Germany, on the other hand, provides the most restrictive guidance, recommending an intake of approximately one egg per week [12]. This heterogeneity in recommendations reflects ongoing uncertainty and inconsistency in the evidence regarding the health effects of egg consumption. Current literature suggests that egg intake can increase LDL cholesterol in a dose-dependent manner, although individual responses vary. Importantly, these effects are influenced by the overall dietary context and the foods eggs replace, with more favorable lipid outcomes observed when eggs are substituted with plant-based alternatives [13–16].

In 2020, a comprehensive systematic review and meta-analysis synthesized evidence from randomized controlled trials to evaluate the effects of egg intake on circulating lipid parameters. This review included 66 trials involving 3,185 participants and assessed a wide range of outcomes, including total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides, very low-density lipoprotein (VLDL), lipid ratios, and apolipoproteins. The findings indicated that egg consumption was associated with significant increases in total cholesterol, LDL-C, HDL-C, and apolipoproteins, while no significant effects were observed for triglycerides, VLDL, or the LDL-C/HDL-C ratio. Furthermore, dose–response analyses suggested both linear and non-linear relationships between egg intake and specific lipid markers [17].

Therefore, the aim of the present study was to provide an updated systematic review of the literature, expanding upon the previous review by Masoumeh Khalighi Sikaroudi et al. (2020). This update incorporates additional randomized controlled trials published since the original review, enabling a more comprehensive and current synthesis of the available evidence. Additionally, this review aims to

refine the research question by considering methodological differences across studies, particularly in relation to dietary background and baseline cholesterol intake, which may influence the observed effects of egg consumption on lipid profiles and, consequently, on the development of hypercholesterolemia.

2 | HYPOTHESIS AND AIMS

2.1. Hypothesis

Consumption of 1 egg per day has a negative impact on LDL-C concentrations and a less favorable lipid profile, potentially contributing to the development of non-familial hypercholesterolemia in adults. Furthermore, baseline cholesterol levels and habitual dietary cholesterol intake prior to the intervention influence the magnitude and direction of these effects.

2.2. Objectives

2.2.1. Primary objective:

To synthesize the overall evidence from randomized controlled trials on the effect of egg consumption or dietary cholesterol derived from eggs on LDL-C concentrations.

2.2.2. Secondary objectives:

- To assess the effect of egg consumption on other lipid profile parameters associated with hypercholesterolemia, including total cholesterol, non-HDL-C, Apolipoprotein B (Apo B), triglycerides, and HDL-C.
- To evaluate the impact of egg consumption on lipid ratios, including LDL-C/HDL-C and total cholesterol to HDL-C (TC/HDL-C).
- To explore potential sources of heterogeneity across studies, including differences in population characteristics and baseline lipid status.
- To highlight the crucial role of baseline dietary cholesterol intake in modulating the effects of egg consumption on blood lipid levels.

3 | METHODS

3.1. Study Design.

This study was realized by following the rules of the *PRISMA Statement* (Preferred Reporting Items for Systematic Reviews and Meta-Analyses), ensuring a transparent and reproducible approach to the

selection and analysis of relevant literature. Patient consent was not required as the study relied on previously published data, and therefore ethical approval was not necessary.

3.2. Research Question.

The research question was formulated according to the *PICO framework* **(Table 1)**, which allows for a structured approach to identifying relevant evidence: The population included adults (≥18 years old) from the general population or clinical populations without familial hypercholesterolemia. The intervention or exposure consisted of egg consumption, higher egg consumption, or dietary cholesterol derived from eggs, while comparators included lower egg consumption, no egg consumption, egg substitutes, or alternative breakfasts and dietary patterns.

Table 1. PICOS criteria for inclusion and exclusion of studies.

Element (PICO)	**Description**
P (Population)	Adults (≥18 years old) from the general population or clinical populations without familial hypercholesterolemia
I (Intervention / Exposure)	Egg consumption, higher egg intake, or dietary cholesterol derived from eggs.
C (Comparator)	Lower egg consumption, no egg consumption, egg substitutes, or alternative breakfasts and dietary patterns
O (Outcome)	**Primary outcomes:** LDL-C. **Secondary outcomes:** Additional lipid parameters such as total cholesterol, LDL-C/HDL-C ratio, non-HDL-C, Apolipoprotein B (ApoB), HDL-C, triglycerides, and the total cholesterol to HDL-C ratio (TC/HDL).

**LDL-C: Low Density Lipoprotein Cholesterol; HDL-C: High Density Lipoprotein Cholesterol; TC: Total Cholesterol*

These outcomes were selected to evaluate the impact of egg consumption and dietary cholesterol on lipid-related cardiovascular risk in adults without familial hypercholesterolemia.

3.3. Eligibility Criteria.

3.3.1. Inclusion criteria.

Only randomized clinical trials (RCTs) were included. Eligible trials only investigated adults (≥18 years old) subjects, assessing egg consumption as the main exposure or intervention, and reporting

outcomes related to hypercholesterolemia or lipid profile. Additionally, only studies conducted in populations without diagnosed familial hypercholesterolemia and published in English or Spanish were considered. Studies published between 2020 and 2026 were included.

3.3.2. Exclusion criteria.

Observational studies were excluded, as well as trials that included only patients with hypercholesterolemia. Studies focused on genetic dyslipidemias, including familial hypercholesterolemia, were also excluded. Also, studies were excluded if were conducted in populations with diagnosed chronic diseases not aligned with the study objective (e.g., type 1 diabetes, or severe metabolic disorders). Furthermore, studies conducted in children or adolescents (<18 years old), as well as animal or in vitro research, were not considered.

3.4. Search Strategy.

An updated systematic review search was conducted to expand the evidence provided by the systematic review of M.K. Sikaroudi et al. (2020). The search aimed to identify studies evaluating the association between egg consumption and lipid profile. We screened the PubMed database up to May 2026. The search strategy combines keywords and Medical Subject Headings (MeSH) related to “egg consumption”, “LDL”, “HDL”, and other “lipid profile parameters”, using Boolean operators (AND, OR).

Applying that, we established our search strategy:

("Adult"[Mesh] OR adult*[tiab] OR "healthy adult*"[tiab] OR "general population"[tiab]) AND ("Eggs"[Mesh] OR egg[tiab] OR eggs[tiab] OR "egg consumption"[tiab] OR "high egg intake"[tiab] OR "egg-based diet"[tiab]) AND ("Cholesterol, LDL"[Mesh] OR "Cholesterol, HDL"[Mesh] OR "Cholesterol"[Mesh] OR "Dyslipidemias"[Mesh] OR "Apolipoproteins B"[Mesh] OR LDL[tiab] OR "LDL-C"[tiab] OR HDL[tiab] OR "HDL-C"[tiab] OR "total cholesterol"[tiab] OR "lipid profile"[tiab] OR dyslipid*[tiab] OR hypercholesterolemia[tiab] OR "apolipoprotein B"[tiab])

3.5. Study Selection Process.

Our systematic literature search was conducted using a predefined search strategy. All retrieved articles were independently screened by two reviewers (DLB, ALG). An initial screening was performed based between 2020 and 2026, title and abstract to assess potential eligibility. Then, a second screening was performed based on the full text to decide which articles finally include in the review.

3.6. Data Extraction.

Data extraction was performed using a structured data extraction form designed for this systematic review. The following information was collected from each included study: first author, country, year of publication, study design, sample size, participant characteristics, intervention and comparator details, duration of the intervention, lipid-related outcomes assessed, and main findings.

When available, information on dietary dose, background diet, and comparator quality was also recorded to facilitate interpretation of the findings considering the broader nutritional context.

3.7. Risk of Bias Assessment.

The risk of bias of the included studies was assessed using the Cochrane Risk of Bias 2 (RoB 2) tool [18]. This tool evaluates five domains: randomization process, deviations from intended interventions, missing outcome data, measurement of the outcome, and selection of the reported results. Each domain was judged as “low risk”, “some concerns”, or “high risk of bias”, following the RoB 2 algorithm. An overall risk of bias of judgement was then derived for each study.

3.8. Data synthesis.

A descriptive synthesis (including text and tables) was conducted to summarize the findings of the included studies. Due to the heterogeneity in study design, populations, and interventions, a meta-analysis was not performed. The studies were synthesized according to the main lipid outcome parameters analyzed, including low-density lipoprotein cholesterol (LDL-C), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), also including the diagnostic of dyslipidemia and hypercholesterolemia, and triglycerides (TG) and ApoB. When available and appropriately reported, studies were also considered according to participants' baseline dietary cholesterol intake.

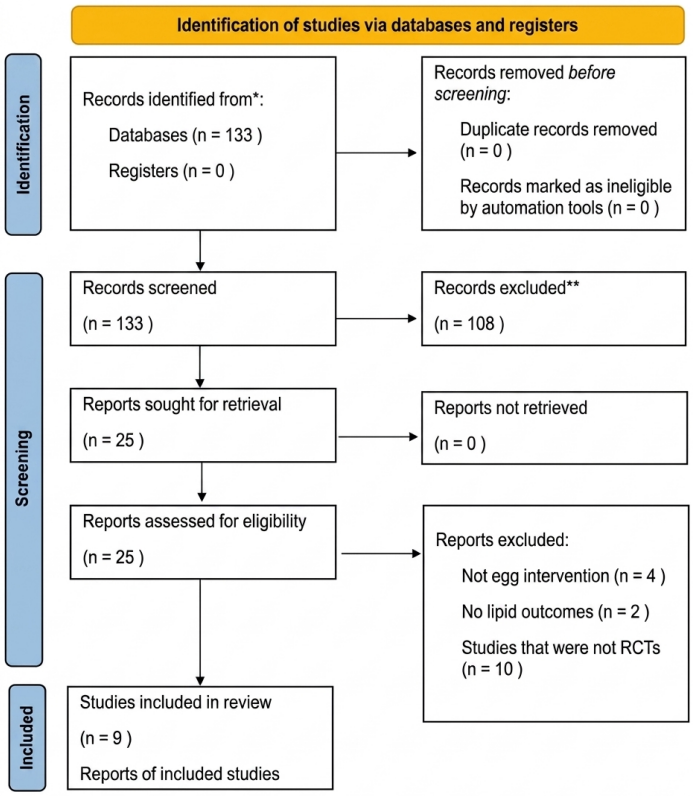
4 | RESULTS

4.1. Study Characteristics.

Figure 1 depicts the study selection process. A total of 133 articles were identified with the initial search. Following the initial screening, a total of 108 articles were excluded based on title and abstract, as they did not meet the predefined inclusion criteria. The remaining 25 reports were assessed for eligibility. After a full-text review, 16 studies were further excluded for the following reasons: not

assessing egg consumption as the primary intervention (n = 4), lacking relevant lipid-related outcomes (n = 2), or studies that were not RCTs (n = 10). Finally, a total of 9 studies were included in the review.

Figure 1. PRISMA flow diagram.



The 9 RCTs included in the qualitative synthesis were published between 2020 and 2026. **Table 2 depicts the characteristics of the included studies**. Most of the studies were performed in the United States (n = 6) [20, 21, 23, 24, 25, 27] with others carried out in China [22] or Australia [19, 26]. Both parallel [19, 21, 23, 25] and crossover [20, 22, 24, 26, 27] designs were represented.

Table 2. Study characteristics.

Study & Country	Design	Sample & Population	Intervention	Comparator	Duration	Outcomes Assessed	Main Findings
Keogh et al., 2020 (Australia) [19]	Parallel RCT	N=76 overweight adults	Weight-loss diet + 2 eggs/day (breakfast)	Cereal-based breakfast diet	6 months	Body weight, lipid profile, metabolic markers	Similar weight loss (~6–7 kg) between groups under energy restriction. No significant differences in lipid profiles or metabolic markers. Egg intake controlled only at breakfast; no restriction at other meals.

Maki et al., 2020 (USA) [20]	Crossover RCT	N=30 adults with prediabetes/metabolic syndrome	2 eggs/day (breakfast)	High-carbohydrate breakfast	2 × 4 weeks	Lipid profile, cardiometabolic markers	No significant differences in cardiometabolic outcomes. Greater LDL-C reduction was observed in high-carbohydrate conditions.
Njike et al., 2021 (USA) [21]	Crossover RCT	N=33 adults at risk of T2D	Plant-based diet + 2 eggs/day	Plant-based diet without eggs	2 × 6 weeks (+ 4-week washout)	Lipid profile, cardiometabolic risk markers	No significant differences between conditions in cardiometabolic outcomes.
Liu et al., 2022 (China) [22]	Crossover RCT	N=70 healthy young adults	0–2 eggs/day	Egg-free diet	11 weeks	Lipid profile, satiety	No adverse effects on lipid profiles. Increased satiety during egg consumption periods. Other dietary cholesterol sources are allowed in control condition.
Galvis et al., 2023 (USA) [23]	Parallel RCT	N=105 healthy adults	2 eggs/day or annatto-enriched eggs	Egg white-based diet	8 weeks	Lipid profile, cardiovascular risk markers	No significant differences in lipid profile or cardiovascular risk markers between groups. No additional effect of annatto enrichment.
Andersen et al., 2023 (USA) [24]	Crossover RCT	N=28 healthy adults	3 whole eggs/day	Egg white-based diet	16 weeks	Lipid profile, choline levels	Increased HDL-C and plasma choline with egg consumption. No adverse cardiometabolic effects compared with egg white conditions.
Nouhravesh et al., 2025 (USA) [25]	Parallel RCT	N=140 older adults with cardiovascular risk	Fortified eggs	<2 eggs/week diet	4 months	Lipid profile	No significant differences in LDL-C or HDL-C between groups.
Carter et al., 2025 (Australia) [26]	Crossover RCT	N=61 healthy adults	2 eggs/day	Low-egg or egg-free diet	5 weeks per phase	Lipid profile	No significant differences in LDL-C. Lipid changes appeared more related to saturated fat intake than dietary cholesterol intake.
Njike et al., 2026 (USA) [27]	Crossover RCT	N=39 adults with hyperlipidemia	DASH diet + 2 eggs/day	DASH diet without eggs	8 weeks per phase	Lipid profile, vascular function	No significant differences in LDL-C or vascular function between conditions.

**LDL-C: Low Density Lipoprotein Cholesterol; HDL-C: High Density Lipoprotein Cholesterol; TC: Total Cholesterol; RCT: Randomized Control Trial*

Sample size ranged from small quantities to 30 to 140 individuals [19-27]. The populations studied were heterogeneous and included a general population from healthy adults [22-24, 26] to participants at elevated cardiometabolic risk or with hyperlipidemia [25, 27] , participants at risk of type 2 diabetes (T2DM) [20, 21]. There was a wide range of participants ages, from young adults to older populations [19, 22, 23, 25] and all studies included both genders [19-27].

All intervention protocols specified the consumption of eggs at doses ranging from 1 to 2 eggs per day or 12 eggs per week in some studies. In several studies, eggs were incorporated into specific dietary

patterns, such as plant-based diets or DASH diets [21, 27], whereas others evaluated fortified [25] or combined interventions such as washout periods and time-restricted eating [21, 22]. Comparator groups typically include egg-free diets, higher-carbohydrate breakfast alternatives [20], or restricted egg intake in their usual dietary patterns [19, 26].

The duration of the interventions went from 4 to 16 weeks [20-27], with crossover studies that included washout periods between interventions periods [21, 22]. The primary outcomes assessed across studies focusing on lipid-related parameters, including total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, and apolipoproteins like ApoB [19-27].

Overall, included studies were methodologically diverse with respect to population characteristics: The study populations ranged from healthy young adults and healthy adults [22-25] to overweight individuals [19], adults with prediabetes or metabolic syndrome, participants at risk of type 2 diabetes [20, 21, 26, 27], older adults with previous cardiovascular events or multiple cardiometabolic risk factors, and adults with hyperlipidemia. There was also variability among the types of background diets studied ranging from dietary patterns like plant based [21] to high-carb breakfast control [19, 20]. In addition, intervention designs were not uniform, with variations in egg dosage, where most trials evaluate the intake up to two eggs per day [19-23, 26, 27]. Methodological approaches differed between the substitution of alternative foods [19, 20, 26] and egg addition versus exclusion protocols [21-23, 27]. Finally, intervention duration (from 4 weeks to several months), and study design split between parallel [19, 22-25] and crossover trials [20, 21, 26, 27]. These heterogeneities should be considered when interpreting the findings of this review.

4.2. Risk of Bias in Studies

Overall, most studies were classified as having a low risk of bias, although four studies presented some concerns [20, 21, 24, 26] mainly related to missing outcome data and deviations from intended interventions. None of the studies were classified as having a high risk of bias (Table 3).

Table 3. Risk of bias assessment of included studies.

Study	Randomization process	Deviations from intended interventions	Missing outcome data	Measurement of outcome	Selection of reported results	Overall
Keogh et al., 2020 [19]	Low	Low	Low	Low	Low	Low
Maki et al., 2020 [20]	Low	Low	Some concerns	Low	Low	Some concerns

Njike et al., 2021 [21]	Low	Some concerns	Low	Low	Low	Some concerns
Qin et al., 2022 [22]	Low	Low	Low	Low	Low	Low
DiMarco et al., 2023 [23]	Low	Low	Low	Low	Low	Low
Andersen et al., 2023 [24]	Low	Some concerns	Low	Low	Some concerns	Some concerns
Nouhravesh et al., 2025 [25]	Low	Some concerns	Some concerns	Low	Low	Some concerns
Carter et al., 2025 [26]	Low	Low	Low	Low	Low	Low
Njike et al., 2026 [27]	Low	Low	Low	Low	Low	Low

Most of the included studies were generally considered to have a low risk of bias, mainly because they used solid randomization methods, measured outcomes consistently, and reported their findings in a clear and complete way. That said, a few studies raised “some concerns”, mostly due to missing outcome data and some deviations from the planned interventions, which may have introduced attrition or performance-related bias. Importantly, none of the studies were judged to be at high risk of bias.

4.3. Synthesis of Results.

Overall, the lipid-related findings were heterogeneous across the included randomized controlled trials, although most studies did not report statistically significant between-group differences in conventional lipid markers, including total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). A detailed description of the characteristics and results of each included study is presented in **Annex 1.**

4.3.1. Egg consumption and LDL-cholesterol.

Across the 9 included RCTs, the overall pattern of results indicates that egg consumption does not consistently lead to statically significant changes in LDL-C levels. The majority of studies [19, 21-25, 27] reported no significant between-group differences in LDL-C when comparing egg-containing diets with control conditions.

Only two studies [20, 26] reported statistically significant differences between egg intervention and control group. In the study by Maki et al. [20], the reduction in LDL-C was significantly smaller following an egg-based breakfast compared with a higher-carbohydrate non-egg breakfast (-2.9% vs –6.0%, respectively; p-value = 0.023), suggesting a relatively less favorable LDL-C response. In contrast, the study by Carter et al. [26] found that an egg-containing diet (2 eggs daily) combined with lower saturated fat intake resulted in significantly lower LDL-C levels compared with control diet that was characterized by high cholesterol intake (600mg/day), high saturated fat content (12% of energy), and included the consumption of 1 egg per week (103.6 vs 109.3 mg/dL; p = 0.02).

Although methodological differences existed across studies, most RCTs consistently reported no significant effect of egg consumption on LDL-C levels.

4.3.2. Egg consumption and total cholesterol.

In the 3 included RCTs, findings consistently suggest that egg consumption has little to no effect on total cholesterol (TC) levels. Most studies [19, 21-23, 25, 27] did not report statistically significant differences in TC when comparing egg-containing diets with their respective control conditions.

In a few cases (n = 2), modest changes were observed, although these did not reach statistical significance. For example, in the plant-based trial by Njike et al. [21], the egg-free phase resulted in a greater reduction in TC compared to the egg-inclusive phase (−7.2 vs 0.7 mg/dL; p-value = 0.058). Similarly, Maki et al. [20] reported slight decreases in TC in both intervention arms, with no meaningful differences between the egg-based and non-egg breakfast conditions (−0.8% vs −2.4%; p-value = 0.353).

Altogether, the evidence from our selected RCTs demonstrates a lack of effect of egg consumption on total cholesterol. Any small variations observed across these studies appear inconsistent and are unlikely to be clinically relevant.

4.3.3. Egg consumption and HDL-cholesterol.

Among 8 of the 9 included RCTs, in general, HDL-C concentrations were not significantly affected by egg consumption compared to control conditions. Most studies [19-21, 23, 25-27] did not report statically significant between group differences in HDL-C levels, with some of them noting non-significant increases, for instance, in participants aged 65 and older [25]. Although the RCT by Ma et al, did report a statistically significant increase ($p < 0.05$) in HDL-C levels across all groups (group with

no eggs, group 1 egg/day and group 2 eggs/day) as the study progressed from from autumn to winter, nevertheless the authors discussed that may be due to the increased energy intake during that period and the reduced exercise activity [22]. As well as the RCT by Andersen et al, where the consumption of three whole eggs per day increased a subset of large HDL particles (H6P, 10.8 nm) compared to the egg-free diet and egg-white periods ($p < 0.05$) [24].

The actual evidence suggests that egg consumption does not consistently affect total HDL-C concentrations in adults. Nevertheless, findings from Andersen et al. [24] suggest that whole egg intake may influence HDL particle size distribution. These effects, however, were observed in a small subgroup and should be interpreted with caution.

4.3.4. Egg consumption and triglycerides.

Across the 9 included RCTs, triglyceride concentrations did not differ significantly between egg-containing interventions and their respective comparators. This pattern was consistent across different dietary contexts, including energy-restricted diets, higher-carbohydrate breakfast comparators, plant-based dietary patterns, egg-white conditions, restricted egg-intake comparators, and broader heart-healthy dietary interventions [19-21, 23, 25-27].

In the study by Liu L et al. [22], triglyceride concentrations shown an increase during the 2-eggs/day phase compared with the lower-intake phases; however, this change was not statistically significant. In Nouhravesh et al. [25], triglycerides were included among the secondary lipid outcomes, but not statistically significant between-group differences in triglycerides was specifically reported in the study summary.

Taken together, the available RCTs show a consistent pattern for this outcome: egg consumption was not associated with statistically significant changes in triglyceride concentrations across the populations, and dietary contexts studied.

4.3.5. Egg consumption and other lipid outcomes: ApoB, non-HDL-C, and lipid ratios.

Only a limited number of studies reported data on ApoB [25, 26], non-HDL-C [24] or other lipid ratios [23, 24].

Related to ApoB, the study by Carter et al. [26] shown that ApoB levels were lower when participants followed a dietary pattern that included eating eggs compared to control diets. In addition, the

researchers noted that shift toward smaller LDL particles that are considered more atherogenic among participants who began eating eggs compared to control groups. In contrast, the intervention by Nouhravesh et al. [25] reported only a non-statistically significant low levels of ApoB in the fortified egg group.

When comparing lipid ratios, both reports by Andersen et al. [24] and Galvis et al. [23] evaluated the LDL-C/HDL-C and TC/HDL-C ratios, and both studies found no statistically significant differences between egg intervention and control conditions. Further, as previously mentioned the study by Andersen et al. [24] demonstrated that women using combined oral contraceptives (n = 11) had a statistically significant lower levels in TC/HDL-C across the time of consumption of whole eggs, however this finding should be replicated in larger samples. Non-HDL-C was reported in the study by Andersen et al. [24], with no significant differences observed between the two dietary conditions.

To summarize, the limited evidence available for these outcomes does not show conclusive negative effects of egg consumption on ApoB, non-HDL-C, or other lipid ratio. The results published by Carter et al. [26] shown reductions in ApoB levels should be considered in the context of the specific dietary context of that trial since saturated fat was also reduced in that trial.

5 | DISCUSSION

5.1. Summary of main findings.

This systematic review synthesizes evidence on the effect of egg consumption on lipid profiles and cardiovascular risk markers in the adult population. In general, the RCTs included in this review show that egg consumption does not adversely affect LDL-C levels or other conventional lipid measures in adult populations. Although the effects of egg consumption varied across dietary context, most included trials did not report statistically significant differences in LDL-C, total cholesterol, HDL-C, or triglycerides between egg-containing interventions and their comparators.

Across the studies, there was heterogeneity, particularly in the comparison of groups involving higher-carbohydrate or lower-saturated fat diets. However, these varying effects did not consistently translate into an overall less favorable lipid profile. In addition, there were no adverse effects on lipid-related cardiovascular risk markers when eggs were incorporated into established healthy dietary patterns such as the DASH diet.

Taken together, the current body of research does not show a consistent adverse effect of egg consumption on conventional lipid markers in adult populations. Instead, the findings suggest that lipid responses may differ according to the study context, particularly the nature of the comparator diet, background saturated fat intake, and the broader dietary pattern in which egg consumption is assessed.

5.2. Interpretation.

Interestingly, the results of our research do not support our initial hypothesis, that the consumption of 1 egg would negatively affect on LDL-C concentrations and a less favorable lipid profile. Instead, the included RCTs generally show neutral or non-significant effects on lipid parameters. Several factors may help explain these findings, including inter-individual variability, differences in study design, and the dietary context in which eggs are consumed.

5.2.1. Role of dietary context and baseline cholesterol intake in modulating lipid responses to egg consumption.

A key observation from this review is that the impact of egg consumption on lipid profiles seems to depend, in part, on the overall diet and participants' usual cholesterol intake, rather than by egg intake alone.

The variation in LDL-C levels between studies that report statistically significant differences can, in part, be attributed to the dietary backgrounds of the study participants and how they may have impacted the direction and magnitude of cardiometabolic outcomes. In the study by Maki et al. [20], eggs replaced a higher-carbohydrate breakfast containing the same caloric content and especially lower in saturated fats and dietary cholesterol. That explains the relatively less favorable LDL-C outcome in the egg group. However, egg intake was not controlled at other meals, which could also explain the lack of major differences. In contrast, in the intervention by Carter et al. [26] it was shown that when egg consumption was combined with a reduction of saturated fat intake, LDL-C levels were significantly lower than in the control group, which included a higher amount of saturated fat. Furthermore, in this study, saturated fat intake was positively associated with an increase in LDL-C, whereas dietary cholesterol provided from eggs showed no independent association.

Also, this important role of dietary context and baseline cholesterol intake can be seen in the RCT by Njike et al. 2026 [27], where baseline dietary cholesterol intake in the DASH diet without eggs group was 445.8 ± 200.1 mg/d whereas in the DASH diet with egg group it was 273.5 ± 177.9 mg/d.

Subsequently, regarding baseline dietary cholesterol intake, this review identified a notable gap in reporting across included studies. Among the studies included in the review, four of them didn't report the baseline dietary cholesterol intake of participants [19, 23, 26, 27]; four did report baseline dietary cholesterol intake [20, 22, 24, 25]; and one study did not report baseline cholesterol intake; however, because it used a randomized crossover design with a previous 4-week plant-based period and washout period, baseline dietary cholesterol exposure during that phase was likely very low, although not directly reported [21]. This variability limits the ability to extract conclusions about whether baseline cholesterol intake modulates the lipid response to egg consumption from this RCTs. However, it is justifiable that participants with higher habitual cholesterol intake at baseline may present a blunted response to additional dietary cholesterol from eggs, a phenomenon consistent with the concept of hypo-responsiveness described in the broader literature [15].

Also, studies that included egg consumption in structured dietary patterns, such as DASH diet [32] or plant-based diets [21], found no adverse lipids effects, suggesting that the overall dietary pattern may attenuate any potential cholesterol raising effect of eggs.

Overall, this observation suggests that isolating the effect of egg consumption on lipid profiles is methodologically challenging when the quality and composition of the background diet are not adequately controlled or reported. Future RCTs should assess and report on the baseline dietary cholesterol intake and ensure comparator diets are matched for saturated fat content, to allow more precise conclusions.

5.2.2. Inter-individual variability.

In this systematic review, the population studied were heterogeneous and some studies included a healthy population [22-24, 26] while the rest of studies included from participants with overweight or obesity [19, 20], or at risk of type 2 diabetes (T2D) [21], with prior CV event or two CV risk factors [25] or with hyperlipidemia [27]. However, in the report by Galvis, et al. [23], the authors mention different limitations like not considering the individual characteristics of the volunteers that play a key role in shaping their metabolic and physiological responses. Also, other studies did not mention their limitations like Ma, et al. [22] or Andersen, et al. [24], but both studies only focused on egg consumptions and ignored other factors like other dietary and lifestyle factors that could affect their lipid profile and did not report inter-individual variability of their sample. It is important to emphasize that in all studies the dietary adherence was self-reported, although some of them provided the eggs

to participants to improve compliance or looked for specific micronutrients to assess compliance [21, 25-27], and one of them also used blood analyses as an objective verification [24].

The meta-analysis by Hopkings, et al. [15] highlighted that baseline dietary cholesterol was a statistically stronger predictor of change in plasma cholesterol than was added dietary cholesterol, thus the effects of added dietary cholesterol in individuals will be quite different depending on how much was consumed at baseline. This important nuance was not taken into account in many of the RCTs included in the present systematic review when selecting participants [19, 23, 26, 27].

This variability in baseline dietary cholesterol intake modulates the response in serum cholesterol mainly when dietary cholesterol intake is low (within the span of 0 mg/d to 100 mg/d), because many populations already consume high dietary cholesterol diets observing a correlation between its intake and serum concentration is difficult, such as was observed in epidemiological studies in american population which already consumed high amounts of cholesterol (350 mg/d - 900 mg/d) [28].

While when investigating the effects of dietary cholesterol on serum cholesterol in populations with very low dietary cholesterol intake, such as the Tarahumara Indians, whose intake ranges from 17 mg/d to 144 mg/d on average, a statistically significant positive linear correlation between dietary cholesterol intake and total plasma cholesterol levels was found ($r = 0.90$, $P < 0.0\ 1$) [29].

Also it has been shown that in some animal studies there were differences in cholesterol absorption efficiency and conversion of cholesterol to bile acids as determinants of hypo- and hyperresponsiveness to dietary cholesterol. Similarly, it has been reported that in humans, increased intestinal sterol absorption efficiency may play an important role in determining higher serum cholesterol concentrations [15].

Furthermore, it is essential to consider that the substantial inter-individual variability in response to dietary cholesterol may be impacted heavily by genetic factors. Research has shown that both genetic variants and mutations within the APOE gene are linked to changes in lipid and lipoprotein profiles by altering hepatic receptor regulation [30-32]. Also, variations in the Niemann-Pick C1-Like 1 (NPC1L1) protein, which is involved in regulation of cholesterol intestinal and biliary absorption [33], could further explain why some individuals present an exaggerated or atypical hypercholesterolemic response compared to the general population. Importantly, these underlying genetic profiles were not measured or controlled for in the RCTs included in this review; therefore, this confounding variable will prevent us from isolating the effects of egg intake alone.

Overall, this evidence supports the idea that inter-individual variability is a key determinant of the lipid response to dietary cholesterol, provided from eggs, affects blood lipid levels and the reason for some of the differing results found in studies on egg consumption.

5.2.3. Influence of study design and dietary substitutions.

An important point when interpreting the lipid effects of egg consumption is that eggs are not consumed in isolation from the rest of the diet. In RCTs, increasing egg intake necessarily implies some form of dietary substitution, or, at least, the incorporation of eggs into a specific dietary context. Across the trials included in the present review, eggs were not evaluated against a single standardized comparator. Depending on the study design, egg-containing interventions were compared with cereal-based breakfasts [19], higher-carbohydrate breakfasts [20], egg-free diets [21, 24, 27], egg-white conditions [22, 23], or were tested within broader dietary patterns such as plant-based or DASH-type diets [21, 26, 27]. Therefore, the relevant methodological point is not that eggs replaced an entire dietary pattern, but that the dietary context and comparator condition differed substantially between interventions.

This is not a minor methodological issue. Under an isocaloric framework, the effect of increasing the intake of one food cannot be interpreted independently from the food, meal, or dietary pattern that is reduced, removed, or used as the comparator. In this sense, the comparator diet is not simply a background feature of the trial, but part of the dietary intervention itself [13, 34].

This helps explain part of the heterogeneity observed across the included RCTs. In some studies, eggs were compared with dietary alternatives lower in saturated fat or dietary cholesterol [20, 26], whereas in others they were introduced within broader structured dietary patterns that may have attenuated, compensated for, or modified the lipid response [21,27]. Therefore, differences between trials should not be interpreted as evidence of a contradictory body of literature. Rather, they reflect the fact that egg intake was tested under different dietary contrasts and background conditions. In many cases, they are better understood as the result of different dietary contrasts [34].

This point is particularly clear when comparing two of the most informative trials included in the present review. In Maki et al. (2020) [20], an egg-based breakfast was compared with a higher-carbohydrate non-egg breakfast, and the egg group shown a significantly smaller reduction in LDL-C than the comparator diet [20]. By contrast, in Carter et al. (2025) [26], the egg-containing diet was

tested within a dietary pattern that simultaneously reduced saturated fat intake and was associated with lower LDL-C and ApoB than the control diet, although it was also accompanied by a shift toward smaller LDL particles [26]. Considered together, these studies suggest that the lipid response observed in recent trials cannot be attributed to egg intake alone. It must be interpreted in relation to the broader nutritional context in which eggs were consumed and, especially, to the dietary alternative against which they were compared [20, 26].

The same reasoning applies to trials in which eggs were evaluated within trials that compared entire dietary patterns (e.g., plant-based or DASH-type interventions), in which egg inclusion formed part of a broader dietary contrast; eggs were incorporated into broader dietary patterns. Studies that included eggs within plant-based or DASH-type dietary approaches generally did not report statistically significant worsening of conventional lipid outcomes [21,27]. In the context of the present review, this supports the main pattern observed across the recent RCTs: egg consumption did not show a consistent adverse effect on conventional lipid markers. However, this does not necessarily demonstrate that eggs are uniformly lipid-neutral. A more coherent interpretation is that the overall dietary matrix could attenuate, offset, or modify the lipid effects of dietary cholesterol from eggs. From this perspective, the lack of a uniform pattern across recent RCTs is not unexpected. When the comparator changes, the estimated effect of egg consumption changes with it [17, 34].

5.3. Comparison with existing literature.

The findings of the present review should be interpreted in the context of the previous systematic review and meta-analysis by Khalighi Sikaroudi et al. (2020), which included 66 randomized controlled trials with 3,185 participants. That meta-analysis reported that egg consumption was associated with significant increases in total cholesterol, LDL-C, HDL-C, the TC/HDL-C ratio, and apolipoproteins, while no significant effects were observed for several other lipid outcomes [17].

At first glance, these results may seem to differ from those of the present review, in which the nine more recent RCTs predominantly did not show statistically significant adverse changes in LDL-C or other conventional lipid markers after egg consumption. However, this apparent discrepancy should be interpreted considering the methodological characteristics of the studies included in each synthesis. A few factors could explain these differences. First, the previous meta-analysis included a broader and more heterogeneous body of evidence, with studies differing in population characteristics, intervention duration, egg dose, background diet, dietary cholesterol exposure, and comparator conditions [17]. In contrast, the present review focused only on RCTs published from 2020 onwards. Many of these recent trials evaluated egg consumption within more structured dietary

contexts or against dietary comparators, rather than as a simple addition of eggs to an otherwise habitual diet which some of the oldest studies included in the previous review were like [19-27]. Therefore, both reviews do not necessarily summarize the same dietary contrasts, even though they address the same general research question.

Second, the nature of the comparator diet is likely to be highly relevant. In the trials included in the present review, egg-containing interventions were compared with cereal-based breakfasts [19], higher-carbohydrate breakfasts [20], egg-free diets [21, 24, 27], egg-white conditions [22, 23], restricted egg intake [25, 26], or were incorporated within broader dietary patterns such as plant-based or DASH-type diets [21, 27]. This distinction is important because the lipid response attributed to egg consumption does not depend only on the egg itself, but also on the food, meal, or dietary pattern against which it is compared. In nutritional interventions, the comparator is therefore not a neutral background element, but part of the dietary contrast being tested.

Third, differences in saturated fat intake and overall dietary quality may have contributed to the observed lipid responses. In Maki et al. [20], the egg-based breakfast produced a smaller reduction in LDL-C than the higher-carbohydrate non-egg breakfast. In contrast, in Carter et al. [26], the egg-containing condition was combined with lower saturated fat intake and was associated with lower LDL-C and ApoB than the control diet, although it also shifted LDL particles toward a smaller profile. These findings suggest that the effect observed in recent trials may be shaped more by the global dietary contrast than by egg intake alone. In this sense, egg consumption should not be interpreted independently from the saturated fat content, dietary quality, and replacement foods present in each intervention.

Fourth, baseline dietary cholesterol intake may be another relevant but insufficiently reported factor. Hopkins previously described that baseline dietary cholesterol intake may influence the serum cholesterol response to additional dietary cholesterol [15]. However, the meta-analysis by Khalighi Sikaroudi et al. did not explicitly evaluate baseline dietary cholesterol intake as a methodological characteristic across included trials [17]. Similarly, in the present review, this variable was inconsistently reported, which limited the ability to formally assess whether habitual dietary cholesterol intake modifies the lipid response to egg consumption.

Previous systematic reviews and meta-analyses remain relevant because they support the biological plausibility that dietary cholesterol from eggs can influence circulating lipid markers under certain

conditions [15, 17, 28, 33-36]. Nevertheless, these findings should not be interpreted as directly equivalent to the pattern observed in the recent RCTs included in the present review. In our synthesis, egg consumption predominantly did not produce statistically significant adverse changes in conventional lipid markers when tested within specific dietary contexts and against active comparators. This does not demonstrate that eggs are universally lipid-neutral, but rather suggests that their lipid effect may depend on the broader nutritional context in which they are consumed.

Lastly another important factor to take into account is that the systematic review from Khalighi Sikaroudi et al. (2020) [17] included some studies that in the intervention group the amount of added dietary cholesterol was much higher (ranging from 800 mg/d to 1470 mg/d) [37-41], allowing serum cholesterol differences to be more visible despite of higher background dietary intakes [15]. In contrast, from our 9 RCTs , the medium dietary cholesterol intake from the intervention groups, where egg diet was, was on average 509 mg/d, which could in part explain the differences in results from the previous systematic review [17] where egg consumption was postulated to increase LDL-C as well as TC among others, while the results from our included studies show no correlation.

Overall, the present results should be understood as a refinement of the existing literature rather than as a contradiction of it. Current evidence suggests that the direction and magnitude of the lipid response to egg consumption may depend on multiple interacting factors, including comparator diet, saturated fat intake, overall dietary pattern, intervention dose and duration, population characteristics, and baseline dietary cholesterol exposure [15, 17, 38].

5.4. Limitations.

This review has several limitations. First, the number of included trials was relatively small, and sample sizes were modest in several studies. Second, the included RCTs were heterogeneous in terms of population characteristics, intervention dose, comparator diet, background dietary pattern, study duration, and lipid outcomes assessed. This heterogeneity limited the ability to draw a solid conclusion across studies.

Third, many included trials were not specifically powered to detect differences in LDL-C concentrations as their main predefined outcome. Therefore, the absence of statistically significant changes in some studies should be interpreted with caution, particularly when lipid outcomes were secondary or

exploratory endpoints. Fourth, baseline dietary cholesterol intake, one of the methodological factors of greatest interest in this review, was not consistently reported across studies. This limited the ability to formally assess whether habitual cholesterol intake modifies the lipid response to egg consumption. This is relevant because participants with different baseline cholesterol exposures may not respond in the same way to additional dietary cholesterol from eggs.

Fifth, because egg interventions were frequently embedded within broader dietary patterns, it is difficult to isolate the independent effect of eggs from the effect of the overall dietary intervention. This is particularly relevant in studies where saturated fat intake, carbohydrate intake, or overall diet quality differed between intervention arms.

Despite these limitations, this review has several strengths. It focuses specifically on RCTs, updates the evidence published after the 2020 systematic review and meta-analysis, and interprets recent findings in relation to dietary context, comparator quality, and baseline dietary cholesterol intake. These aspects are essential for understanding why lipid responses to egg consumption may differ across studies and why conclusions based solely on egg intake, without considering the broader dietary matrix, may be incomplete.

5.5. Future research directions

Future randomized controlled trials are warranted to clarify the lipid response to egg consumption under different dietary contexts. Although recent RCTs provide useful evidence, several methodological aspects still limit the ability to determine whether the effect of eggs on blood lipids depends on the egg itself, the comparator diet, or the broader nutritional background in which the intervention is conducted.

First, future studies should provide a more detailed characterization of the baseline diet. In particular, habitual dietary cholesterol intake, saturated fat intake, fiber intake, and overall dietary quality should be systematically assessed and reported. This information is essential to determine whether individuals with different nutritional backgrounds respond differently to additional dietary cholesterol from eggs.

Second, comparator diets should be more clearly defined and, when possible, matched for key dietary variables such as energy intake, saturated fat, fiber, and overall diet quality. Since egg-containing interventions are often compared with different foods, meals, or dietary patterns, future trials should

be designed to better distinguish the specific effect of egg consumption from the effect of the broader dietary contrast.

Third, longer interventions and larger sample sizes are needed to improve the detection of clinically meaningful changes in LDL-C and other lipid-related outcomes. Many available trials are relatively short and are not specifically powered to evaluate LDL-C as the main endpoint. Future studies should therefore consider LDL-C, non-HDL-C, ApoB, and lipid ratios as predefined outcomes, rather than only secondary or exploratory variables.

In addition, future research should include a more comprehensive lipid profile. Conventional lipid markers may not fully capture changes in lipoprotein quality or atherogenic particle burden. Therefore, outcomes such as ApoB, LDL particle number, LDL particle size, and non-HDL-C should be more consistently assessed. This is particularly relevant considering findings such as those reported by Carter et al. [26], where LDL-C and ApoB decreased, but LDL particles shifted toward a smaller profile.

5.6. Overall conclusion

In conclusion, recent RCTs predominantly do not show significant adverse effects of egg consumption on LDL-C or other conventional lipid markers in adult populations. However, the current evidence should not be interpreted as demonstrating that eggs are universally lipid-neutral across all dietary contexts. Rather, the findings suggest that lipid response to egg consumption might be context-dependent and may be influenced by comparator diet, saturated fat intake, background dietary pattern, and baseline dietary cholesterol exposure. Therefore, egg consumption should not be interpreted in isolation, but within the dietary pattern in which eggs are included and the foods they replace.

Future trials with better characterization of baseline diet, more standardized comparator conditions, longer follow-up, larger sample sizes, and broader lipid profiling are needed. Future research should clarify whether lipid responses differ among individuals with low versus high habitual dietary cholesterol intake, different baseline LDL-C concentrations, metabolic risk profiles, or potential hyper- and hypo-responsiveness to dietary cholesterol. Likewise, it should determine whether the effect of eggs differs when they replace lower-cholesterol, lower-saturated-fat foods, or when they are incorporated into dietary patterns with lower saturated fat intake and higher overall diet quality.

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7 | ANNEX

Annex 1. Summary of included studies evaluating egg consumption and lipid profile outcomes

The study from **B.Keogh et al.** (n=76) investigated the effect of two energy-restricted weight loss diets, one including an egg-based breakfast and other a cereal-based breakfast on weight loss in overweight adults for a 6-month period. Regarding blood lipids, the authors noted there was no change in total cholesterol, HDL-cholesterol, LDL cholesterol and triglyceride levels.

The study from **Njike et al.** (n=33) investigated the effect of a plant-based diet plus two eggs per day versus an exclusively plant-based diet without eggs on endothelial function as a primary outcome in

adults at risk of type 2 diabetes. They also measured as secondary outcomes the lipid profile (TC, LDL-C, HDL-C, TG, TC/HDL-C), where they did not found significant differences between-condition, although the egg-free plant-based phase diet shown numerically greater reductions in TC and LDL-C (TC: −7.2 vs 0.7 mg/dL; p=0.0580; LDL-C: −6.2 vs 0.2 mg/dL; p=0.1096), although these between-condition differences were not statistically significant.

The study from **Nouhravesh et al.** (n=140) investigated the effects of the inclusion of fortified eggs ≥12 Eggland's Best fortified eggs/week (~1.7/day) for 4 months versus a non-egg supplemented diet (<2 eggs/week) for the egg comparison, in older adults with prior CV event or 2 or more CV risk factors (diabetes, BMI ≥30, hypertension, dyslipidemia, CKD). Their co-primary outcomes were: LDL-c, HDL-c at 4 months; as secondary outcomes, they measured total cholesterol, triglycerides, LDL particle number and size. The authors found no significant difference in LDL or HDL at 4 months between Fortified egg group (FE) versus non-egg group. Other notable secondary/exploratory signals: FE group: numerical decrease in ApoB was found.

The study from **Maki et al.** (n=30) investigated the effects of substituting egg-based breakfast foods (providing 12 eggs per week) with higher-carbohydrate, non-egg breakfast foods (consisting of a variety of foods such as cereals with milk, waffles with syrup, granola bar, nuts, fruits or cheese) in a randomized crossover trial in men and women at risk of type 2 diabetes over two 4-week intervention periods. Their primary outcome was insulin sensitivity. They also assessed blood lipids, including total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides. No significant differences were observed in total cholesterol between conditions (−0.8% vs −2.4%; p=0.353). However, LDL cholesterol decreased significantly less following the egg-based breakfast compared to the non-egg condition (−2.9% vs −6.0%; p=0.023). No significant differences were observed in HDL cholesterol between conditions, and neither intervention significantly affected other lipid parameters.

The study from **Andersen et al.** (n=26) investigated the effect of consuming an egg-free diet, three egg whites per day, and three whole eggs per day on comprehensive clinical metabolic, immune, and hematologic profiles in young, healthy adults (18–35 years) over a 16-week randomized crossover intervention trial. Each intervention period lasted for 4 weeks. Regarding blood lipids, the authors found no significant differences in fasting total cholesterol, LDL-cholesterol (LDL-C), HDL-cholesterol (HDL-C), non-HDL-C, triglycerides, or the total cholesterol: HDL-C ratio across the diet periods. However, whole egg intake specifically increased a subset of large HDL particles (H6P, 10.8 nm) compared to the egg-free and egg-white diet periods ($p < 0.05$). In a subgroup analysis of female

participants using combined oral contraceptives (COC, n=11), whole egg intake significantly decreased the total cholesterol:HDL-C ratio compared to non-users ($p < 0.05$).

The study from **Carter et al.** (n=61) investigated the independent effects of egg-derived cholesterol versus saturated fat (SFA) on LDL cholesterol in healthy adults over three 5-week crossover phases. The authors found that the EGG diet (2 eggs/day, 6% SFA) significantly reduced LDL-C compared to the control diet (1 egg/week, 12% SFA) (103.6 vs. 109.3 mg/dL; p=0.02). Additionally, while the EGG diet lowered ApoB levels (p=0.006), it resulted in a shift toward smaller, more atherogenic LDL particles (p=0.004). Notably, SFA intake was positively correlated with LDL-C (beta=0.35, p=0.002), whereas dietary cholesterol showed no such correlation.

The study from **Qin Ma et al.** (n=70) investigated the effect of a 3-phase intervention (0, 1, and 2 eggs/day) over 11 weeks on cholesterol homeostasis in healthy young Chinese adults. Regarding blood lipids, the authors found no significant differences in total cholesterol (TC), LDL-C, or HDL-C between the three phases ($p > 0.05$). While triglyceride (TG) levels shown a slight numerical increase with 2 eggs/day, the change was not statistically significant (1.16 vs 1.31 mmol/L; $p > 0.05$). Additionally, the researchers found that egg consumption did not significantly alter markers of cholesterol synthesis (lathosterol) or absorption (campesterol and sitosterol), suggesting that an intake of up to 2 eggs per day does not disrupt cholesterol balance in this population.

The study from **Galvis et al.** (n=105) investigated the effect of consuming two eggs per day (either standard eggs or annatto-enriched eggs) compared to an egg-white control diet in healthy adults over an 18-week crossover trial (three 6-week periods). Regarding blood lipids, the authors found no significant differences in total cholesterol, LDL-C, HDL-C, or triglycerides between the egg-consuming phases and the egg-free phase ($p > 0.05$). Specifically, for the standard egg phase versus the egg-free phase, TC was 179.6 vs. 176.6 mg/dL, and LDL-C was 107.0 vs. 104.9 mg/dL. The study also measured the LDL/HDL and TC/HDL ratios, finding no significant changes, which led the authors to conclude that daily egg consumption does not increase cardiovascular risk in healthy individuals.

The study from **Njike et al.** (n=51) investigated the effect of daily egg consumption (2 eggs/day) compared to egg exclusion within a heart-healthy diet in adults with hyperlipidemia over two 8-week crossover periods. Regarding blood lipids, the authors found no significant differences between the egg-inclusion and egg-exclusion phases for total cholesterol (192.3 vs. 191.0 mg/dL), LDL-C (116.3 vs. 115.3 mg/dL), HDL-C (51.0 vs. 50.8 mg/dL), or triglycerides (124.7 vs. 124.6 mg/dL) ($p > 0.05$ for all).

Additionally, there were no significant differences in endothelial function (the primary outcome), blood pressure, or body weight. The authors concluded that incorporating eggs into a heart-healthy diet does not adversely affect the cardio-metabolic profile of individuals with hyperlipidemia.

TUTOR EVALUATION FORM/FITXA AVALUACIÓ TUTOR

ENSENYAMENT: Nutrició Humana i Dietètica

NOM DE L'ALUMNE: David Lozano Baena, Alejandro Ledesma Gonzalez i David Rovira Pallàs

TÍTOL DEL TREBALL: Association between Egg Consumption and Non-Familial Hypercholesterolemia or Adverse Lipid Profile in Adults: A Systematic Review.

SEGUIMENT I AVALUACIÓ DEL TREBALL PER PART DEL TUTOR DEL TREBALL PRÀCTIC (0-10)	
Ha mostrat capacitats d'anàlisi, síntesi i raonament al llarg del treball	8
El seu grau d'implicació durant el desenvolupament del treball ha estat elevat	10
El procés d'elaboració del treball ha estat continuat	9
Ha mostrat habilitat de cerca i gestió de la informació	10
Ha mostrat capacitat d'organització i planificació	10
Ha seguit la normativa pròpia del Centre en quan a la presentació escrita del treball	10
El treball és ordenat i redactat amb cura, expressant-se correctament amb la llengua escollida	9
Els resultats del treball són originals	10
El treball presentat supera les expectatives del tutor	9
Comentaris del tutor	
MITJANA DE LA NOTA DEL TUTOR (0-10)	9,44

AVALUACIÓ: FAVORABLE X

NO FAVORABLE ☐

NOM I SIGNATURA DEL TUTOR*:

Nerea Becerra Tomas

NEREA BECERRA TOMÁS - DNI 39929690B

Firmado digitalmente por NEREA BECERRA TOMÁS - DNI 39929690B
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Andrés Díaz López

DIAZ LOPEZ, ANDRES (FIRMA)

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Reus, a 25 de maig de 2026