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Omega 3 Fatty Acids for Hypertriglyceridaemia and Cardiovascular Risk Reduction: A Narrative Review

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Abstract: ***Background:*** Omega-3 fatty acids are used to lower elevated triglycerides, but their effects on cardiovascular outcomes vary by preparation and clinical setting. Confusion between prescription products and dietary supplements can lead to inappropriate extrapolation of trial findings. ***Objective:*** To critically review the lipid effects, cardiovascular outcome evidence, safety, and clinical role of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), with attention to primary care practice. ***Methods:*** A focused narrative synthesis of selected randomized trials, published reviews, and professional guidance was undertaken. Sources were identified through targeted web searches and checked against available PubMed records and publisher or professional society pages. Evidence was considered separately for lipid modification, cardiovascular events, and adverse effects. No systematic screening, formal risk-of-bias assessment, or quantitative pooling was performed. ***Findings:*** Prescription omega-3 preparations lower triglycerides, although this biochemical response does not establish cardiovascular protection. REDUCE-IT supported icosapent ethyl in selected high-risk, statin-treated patients; STRENGTH did not demonstrate comparable benefit with a mixed EPA–DHA preparation. Lower-dose supplementation trials do not support routine use for broad cardiovascular prevention. DHA-containing products may increase LDL cholesterol, and atrial fibrillation is a relevant safety consideration. ***Conclusion:*** Omega-3 treatment should be selected according to the therapeutic objective, formulation, and patient risk. Lifestyle measures and appropriate LDL cholesterol lowering remain foundational. Evidence for icosapent ethyl should not be generalized to other fish-oil products, and triglyceride reduction should not be presented as proven prevention of pancreatitis.

Keywords: omega-3 fatty acids; hypertriglyceridaemia; eicosapentaenoic acid; docosahexaenoic acid; icosapent ethyl; cardiovascular prevention; primary care.

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INTRODUCTION

Elevated triglycerides can trigger two types of clinically associated but fundamentally distinct risks: one is the risk of atherosclerosis, which is directly linked to triglyceride-rich lipoproteins and their metabolic residues; the other is the risk of pancreatitis, which only occurs in patients with severe conditions.

For current clinical treatment of patients with this type of dyslipidemia, LDL cholesterol has always been the core control target of treatment. The remaining non-HDL cholesterol and apolipoprotein B can help clinicians comprehensively assess the overall burden imposed by all atherosclerotic particles, and clarify how much trouble these vessel-clogging harmful particles have caused to the body. No single triglyceride test value can be used to make a judgment on its own; it must be evaluated comprehensively in combination with the patient's complete lipid profile, metabolic status, and cardiovascular disease history [1].

As for omega-3 fatty acids, their established triglyceride-lowering effect has long been confirmed, but the term "fish oil" commonly used by the general public covers an overly wide range of products: any product marketed under the fish oil label on the market differs in its ingredients, concentration of active ingredients, recommended dosage, and even the research basis that supports its efficacy. The three types of related products commonly used in clinical practice are prescription drugs containing only EPA, prescription formulations containing both EPA and DHA, and over-the-counter nutritional supplements that can be purchased directly without a doctor's prescription. These three categories must never be substituted for one another. The 2026 multi-society joint clinical practice guideline for the diagnosis and treatment of dyslipidemia specifically emphasizes that formal prescription therapeutic drugs must be distinguished from over-the-counter supplements with unstable fatty acid content [2].

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Within the scope of the content to be discussed in this review, three practical questions will be explored: what effects can omega-3 therapy achieve, in which scenarios has its cardiovascular benefits been confirmed, and how can existing research evidence guide primary care practice including the inclusion of clinical scenarios in Qatar for discussion. Its core purpose is to help clinical practitioners interpret published research literature, and it will not recalculate the actual therapeutic effect of this therapy in the local context.

Review approach

This is a narrative review with a clear focused scope, specifically centered on the management of triglycerides in adults. We sorted out the clinical value of omega-3 formulations for reducing triglycerides, and all integrated evidence is sourced from published randomized controlled trials, clinical practice guidelines, and targeted safety analyses. To complete this review, all literature retrieval work was finalized on September 11, 2026. The search scope covered public records on the academic platform PubMed, publisher pages of published studies, and guideline repositories of various professional societies. The search terms used included "omega-3", "hypertriglyceridemia", "icosapent ethyl", "EPA", "DHA", "cardiovascular outcomes", as well as the names of all core clinical trials related to omega-3. For all literature considered for inclusion, we first verified the authenticity of the study itself and the core conclusions drawn from the trials. Only content that aligns with triglyceride management in adults, the efficacy characteristics of specific formulations, and current clinical safety requirements was ultimately selected.

When organizing materials, we prioritized data from randomized clinical trials and authoritative professional guidelines, supplemented by other selected reviews and a dedicated meta-analysis analyzing safety. In this article, we discuss lipid-related study endpoints and cardiovascular-related study endpoints separately, mainly because when translating trial results into clinical treatment recommendations, the dimensions that need to be considered for these two types of endpoints are completely different. This review is positioned to answer specific practical clinical questions, rather than being an exhaustive systematic review that collects all relevant literature. Therefore, we did not submit a PRISMA flowchart, the total number of studies meeting the inclusion criteria, pooled estimated data calculated from multiple studies, nor did we conduct a formal credibility rating for the included studies. We also do not claim that this article covers all relevant publications before the search date, l*et al*one arbitrarily generalize the study conclusions to populations outside the core trial population.

EPA and DHA can regulate triglyceride metabolism, with the core biological mechanism being to reduce the hepatic synthesis of very-low-density lipoprotein triglycerides while improving the efficiency of their clearance in the body. This effect stems from their unique cellular effects on lipid processing pathways. Treatment responses vary among different patients, and are specifically influenced by several factors: the baseline triglyceride level before medication, the daily dosage, and which specific omega-3 formulation is used. Generally, patients with severe hypertriglyceridemia treated with prescription formulations will experience the largest reduction in triglycerides. The scientific advisory from the AHA also recognizes that a daily prescription dose of 4 grams is an effective lipid intervention, and mentions that patients with common hypertriglyceridemia usually experience a steady 20%-30% reduction in triglycerides, while patients with severe hypertriglyceridemia may see an even larger reduction [3]. In addition to triglycerides, changes in other lipid components in the blood need to be interpreted separately, as formulations containing DHA may increase LDL cholesterol, especially in patients with extremely high baseline triglyceride levels before medication; whereas treatment regimens containing only EPA usually have a different impact on LDL than formulations containing DHA. A previous comparative review found that the effects of EPA and DHA on blood lipids are not completely consistent, so clinical follow-up needs to track the complete lipid profile, not just test triglycerides alone [4]. It should also be reminded that improvements in blood test indicators are only an intermediate outcome, and it is not possible to conclude solely based on this that the patient's risk of heart attack, stroke, or pancreatitis will be reduced. When translating the results of short-term lipid trials into clinical treatment recommendations, the difference between these two types of outcomes must be distinguished, otherwise problems are likely to arise. If a patient's triglycerides are already elevated, in addition to routine lipid indicators, testing non-HDL cholesterol and apolipoprotein B can provide key additional information, because changes in blood cholesterol concentration do not necessarily synchronize with changes in the number of lipoprotein particles in the body [1].

The MARINE trial specifically evaluated purified EPA ethyl ester in patients with extremely high triglycerides, and confirmed its lipid-lowering effect through a controlled trial based on early mechanistic studies. This randomized, placebo-controlled trial verified the lipid-lowering efficacy of the formulation in the population with severe hypertriglyceridemia; however, this study only supports the use of EPA to improve biochemical indicators, and the observed reduction in blood lipids cannot be directly equated with a reduced risk of pancreatitis or cardiovascular events [5]. The ANCHOR trial expanded the research scope to 702 patients taking statins, whose LDL cholesterol had been controlled to meet the standard, but whose triglycerides still remained at an excessive level of 200-499mg/dL, meeting the enrollment requirements. The entire trial lasted for 12 weeks of observation, and finally found that EPA ethyl

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ester could indeed reduce triglycerides; compared with the placebo group, subjects who took 4 grams daily had an adjusted triglyceride reduction of approximately 21.5%, and there was no significant increase in LDL cholesterol. This result proves that adding this drug on the basis of statin therapy can still continue to regulate blood lipids, but because the trial duration was relatively short and only lipid-related endpoints were observed, no conclusions about long-term clinical outcomes could be drawn [6]. These two trials also explain why it is necessary to clearly record the active formulation used just relying on the number of capsules taken and the total oil weight of these formulations cannot accurately calculate how much EPA or DHA the patient actually ingested. The effect of the prescription formulation studied at a fixed dose in the trials cannot be replicated by taking the same amount of untested common supplements. The AHA's advisory and subsequent guidelines support that if medication is needed to lower triglycerides, prescription products should be prioritized [2,3].

JELIS was the first large-scale trial to test EPA for reducing cardiovascular risk. It randomly divided 18,645 Japanese patients with hypercholesterolemia into two groups: one group received only statin therapy, and the other group added 1.8 grams of EPA daily on top of statins. After an average follow-up of 4.6 years, the incidence of major coronary events in the group with added EPA was 2.8%, while the incidence in the statin-only group was 3.5%, representing a 19% relative risk reduction. However, the trial itself had limitations: it had an open-label design, all enrolled subjects were Japanese, and their basic treatment regimens were unified. These factors meant that the estimated risk reduction derived from the trial could not be directly applied to other populations, but it did provide early outcome evidence for the clinical effect of EPA [7]. REDUCE-IT was a landmark trial for the prescription formulation icosapent ethyl, which recruited a total of 8,179 patients taking statins. These patients either had a confirmed diagnosis of cardiovascular disease, or had diabetes along with other risk factors that induce cardiovascular disease. Eligible patients also had to meet two lipid criteria: triglycerides between 135-499mg/dL and LDL cholesterol between 41-100mg/dL. All subjects took 2 grams of icosapent ethyl twice a day, with a median follow-up time of 4.9 years. Finally, the incidence of the primary composite cardiovascular endpoint dropped from 22.0% in the placebo group to 17.2% in the medication group, with a statistical hazard ratio of 0.75 and a 95% confidence interval of 0.68-0.83 [8]. The absolute difference in event rates between the two groups was 4.8 percentage points, equivalent to preventing 1 endpoint event for every 21 people treated throughout the trial period, a figure calculated based on the proportion of events reported in the trial. It should be noted that this estimate only applies to the population with the corresponding risk characteristics studied in the trial and the composite outcome set by the trial, and cannot be regarded as the expected benefit for low-risk adults or patients who take over-the-counter fish oil on their own [8].

Trials of mixed omega-3 formulations and low-dose supplements have all yielded consistent negative results, which also highlights the large differences in the clinical efficacy of different omega-3 formulations. Let us start with the high-dose STRENGTH trial. The STRENGTH trial randomly divided 13,078 high-risk patients taking statins into two groups: one group took 4 grams of an EPA-DHA combined carboxylic acid formulation daily, and the other group took corn oil as a control. Finally, the incidence of major cardiovascular endpoint events was 12.0% in the trial group and 12.2% in the control group, with a hazard ratio of 0.99 and a 95% confidence interval of 0.90-1.09, showing almost no difference between the two groups. This trial was terminated early because there was no possibility of achieving effective results. Its results also indicate that even if high doses of omega-3 are taken and triglycerides are reduced, it does not necessarily bring cardiovascular benefits [9]. Alongside STRENGTH, VITAL was a large primary prevention trial, that is, a trial for disease prevention in people who have not yet developed the disease. 25,871 subjects in this primary prevention setting took 1 gram of marine-source omega-3 formulation or placebo daily, and finally there was no significant reduction in the primary cardiovascular endpoint events in the intervention group. The core conclusion of this trial was negative, meaning the intervention had no effect. Therefore, the results of any secondary indicators or subgroup analyses cannot be interpreted outside of this core conclusion, especially when recommending supplements to the general population without special risks, this core result must be kept in mind [10]. Another primary prevention trial for people with diabetes, ASCEND, reached a similar conclusion: 15,480 subjects with diabetes but no confirmed diagnosis of atherosclerotic cardiovascular disease took 1 gram of omega-3 supplement daily, and there was still no significant reduction in the incidence of serious vascular events. These results do not negate the role of prescription EPA in other higher-risk populations, but they also oppose treating all omega-3 products as the same cardiovascular disease prevention intervention [11]. There was also the OMEMI trial, which tested the effect of omega-3 in an elderly population after myocardial infarction, having subjects take 1.8 grams of omega-3 formulation daily, and ultimately failed to prove that the formulation could significantly reduce the incidence of the trial's preset primary composite endpoint events. Combined with the results of the STRENGTH trial and other low-dose prevention trials, this series of conclusions repeatedly emphasizes that when interpreting the efficacy of omega-3, the patient's age, baseline disease risk, baseline medications, and the specific formulation used must be clarified, otherwise it is impossible to draw accurate conclusions [12].

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Table 1: Selected cardiovascular outcome trials

Trial	Population and intervention	Principal interpretation
JELIS [7]	18,645 patients; EPA 1.8 g/day plus statin	Coronary events reduced; open-label Japanese trial
REDUCE-IT [8]	8,179 high-risk patients; icosapent ethyl 4 g/day	Primary endpoint 17.2% vs 22.0%; benefit in selected statin-treated patients
STRENGTH [9]	13,078 high-risk patients; EPA–DHA 4 g/day	Primary endpoint 12.0% vs 12.2%; no benefit
VITAL [10]	25,871 adults; marine omega-3 1 g/day	No significant primary cardiovascular benefit
ASCEND [11]	15,480 adults with diabetes; omega-3 1 g/day	No significant serious vascular event reduction
OMEMI [12]	Older post-infarction adults; omega-3 1.8 g/day	No significant primary composite benefit

Trial populations, endpoints, comparators, and follow-up differed. This table is a narrative comparison, not a pooled or head-to-head analysis.

Interpreting the divergent trial findings

The differences between the REDUCE-IT and STRENGTH studies cannot be attributed solely to DHA. The prior claim that the divergent outcomes of these two studies were entirely caused by DHA is untenable, as DHA alone cannot explain all discrepancies between the two studies.

Beyond the impossibility of pinning all differences on DHA, the two trials themselves had many distinct design features: not only did they use different pharmaceutical formulations, but they also selected different control substances. Most critically, neither trial implemented a design where participants were randomly assigned to two groups, one receiving EPA-only treatment and the other receiving combined EPA plus DHA treatment, to directly compare the two regimens. For this reason, the claim that "DHA cancels out the benefits of EPA" remains only a hypothesis to date, and cannot be considered a validated causal explanation [8,9].

The selection of a control group directly impacts the interpretation of trial results, and the importance of this detail cannot be overstated. REDUCE-IT used mineral oil as its placebo control, while STRENGTH used corn oil. These fundamental differences in trial design make it impossible to directly compare and extrapolate findings from the two studies, and even indirect comparisons cannot be reliably conducted.

Although the two studies cannot be simply pitted against each other to determine superiority, one clinically applicable conclusion is clear, and it is tied directly to the specific formulations used: in the study population of REDUCE-IT, only icosapent ethyl was confirmed to have definite positive effects; whereas the mixed combination formulation tested in the STRENGTH trial failed to meet its prespecified primary endpoint [8,9].

Looking more broadly at all existing relevant research evidence as a whole, it becomes clear that two categories of studies are fundamentally distinct from their core: trials testing low-dose supplements and trials investigating the therapeutic effects of pharmaceutical-grade EPA seek to answer entirely different questions. The neutral results of those low-dose supplement trials meaning they found no efficacy for the supplements directly refute the general claim that "fish oil prevents cardiovascular disease", while at the same time they do not interfere with the correct interpretation of the approved indications for prescription medications [10,11].

Safety and monitoring

Before prescribing marine-derived omega-3 supplements to patients, physicians must first carefully weigh the benefits and risks of this treatment. One issue that must be specifically considered is whether the patient has atrial fibrillation, commonly known as afib a common condition of irregular heart rhythm, which must never be overlooked. A previous meta-analysis that pooled the results of all relevant cardiovascular clinical trials found that consuming such marine omega-3 supplements increases patients' risk of developing atrial fibrillation; this elevated risk becomes more evident when high-dose supplements are used in trials. This conclusion reminds us that if a patient has a history of atrial fibrillation, extreme caution must be exercised when prescribing the supplement. If the supplement is indeed prescribed, physicians should proactively inquire with the patient during treatment about whether they have experienced heart palpitations or any new symptoms related to heart rhythm [13].

In addition to the risk of atrial fibrillation, two other issues must also be thoroughly investigated: whether the patient faces a risk of bleeding when taking the supplement, and whether there are any safety concerns if the supplement is used concurrently with antithrombotic medications. In the dedicated REDUCE-IT trial, the number of patients who experienced severe bleeding in the group taking icosapent ethyl was numerically higher than that in the other group, but this difference was not large enough to meet the traditional threshold for statistical significance. Beyond these health risks, several practical concerns must always be kept in mind

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when using supplements: whether patients can tolerate the supplement gastrointestinally, whether the large daily number of capsules imposes a burden on patients, and whether patients can consistently adhere to the required dosing schedule without missing or stopping doses. It is inappropriate to conclude that omega-3 therapy is completely unsafe for all people solely because of these safety concerns; instead, these potential safety risks must be weighed holistically against the tangible cardiovascular benefits it can provide [8,9].

After patients start treatment, they must return for regular follow-up reviews, and four tasks must be completed during these follow-ups: rechecking the patient's blood lipid indicators, confirming whether the patient has been adhering to the prescribed dosing regimen, assessing whether the patient's body can tolerate the treatment, and verifying whether the initially set treatment goals have been achieved. If the supplement prescribed to the patient contains DHA, special attention must be paid to low-density lipoprotein cholesterol levels, and this indicator must not be overlooked. If a follow-up review finds that the patient's triglyceride levels have decreased, but another blood lipid indicator that contributes to atherosclerosis has instead risen, the physician must make a careful judgment based on actual clinical circumstances, and cannot directly conclude that the patient's overall condition has improved [4].

Clinical application in primary care

The first step in managing dyslipidemia is to first confirm that the patient indeed has an abnormal lipid profile, and immediately thereafter screen for all secondary factors that may contribute to the condition: the current control status of diabetes, the volume of daily alcohol consumption, the presence of kidney disease, the presence of hypothyroidism, and all concurrent medications that may affect blood lipid levels.

In the clinical practice pathway of the American College of Cardiology (ACC), a clear standard is defined for "persistent hypertriglyceridemia": a patient can only be diagnosed with persistent hypertriggerlyceridemia if after completing lifestyle intervention, adhering to standardized statin use, and properly addressing all the aforementioned secondary pathogenic factors, their triglyceride levels remain elevated. This pathway also clarifies priorities: if triglyceride-lowering medications are to be used to prevent cardiovascular disease, treatment targeting low-density lipoprotein (LDL) must be prioritized, and this treatment must be implemented before considering the addition of dedicated triglyceride-lowering medications [14].

The 2021 version of this clinical practice pathway also specifies management principles for several distinct scenarios: if a patient only has mild or moderate triglyceride elevation, with no history of atherosclerotic cardiovascular disease (ASCVD) and no diabetes, non-statin triglyceride-lowering medications must not be routinely prescribed solely based on the patient's triglyceride test values. If a patient's triglyceride levels are severely elevated, reaching or exceeding 1000mg/dL, two matters become particularly critical: first, strictly limiting fat intake in the daily diet, and second, properly addressing all the aforementioned secondary pathogenic factors. For these high-risk patients, within the framework of a pancreatitis risk management plan, the addition of prescription omega-3 therapies or fibrates may be considered [14].

The 2026 updated guidelines retain the applicable scenarios for two categories of medications: prescription omega-3 remains indicated for the treatment of severe hypertriglyceridemia, and icosapent ethyl remains usable for eligible patients those who are already receiving statin therapy but still have residual cardiovascular risk. All treatment decisions must comply with current applicable standards, and also take into account local drug accessibility, that is, whether the corresponding medications are available. Whether referring to the 2021 clinical practice pathway or the 2026 new edition of the guidelines, over-the-counter nutritional supplements are not supported as substitutes for research-validated prescription therapeutic medications [2,14].

Table 2: Questions that guide omega-3 treatment decisions

Clinical question	Implication
Is the goal cardiovascular prevention or severe triglyceride reduction?	Use outcome evidence for the former and lipid evidence for the latter [2,3].
Are secondary causes and LDL-directed treatment addressed?	Correct contributors and optimize foundational care [14].
What exact preparation and daily dose are proposed?	Record prescription product and active constituents; avoid supplement equivalence [2,3].
Does the patient resemble an outcome-trial population?	Assess applicability before extrapolating cardiovascular benefit [7–12].
Are rhythm history, antithrombotics, and lipid response reviewed?	Individualize benefit–harm discussion and follow-up [4,8,13].

Although severe triglyceride lowering is used in pancreatitis-risk management, direct evidence that omega-3 therapy prevents pancreatitis is limited. A treatment rationale based on lowering a risk-associated

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biomarker must be distinguished from a demonstrated reduction in clinical events [1,3].

Relevance to Qatar and research priorities

To implement these research findings in Qatar's primary care setting, we can build a directly usable clinical practice framework that only needs to be followed step by step: first, record the cardiovascular disease risk level of each patient; second, verify the specific formula of the medication included in the prescription to be issued; then, screen for all secondary triggers that may cause problems outside the core etiology; finally, it is essential to clarify the treatment goal for this encounter whether it is only to lower the triglyceride level below the standard threshold, or to truly reduce the patient's risk of acute cardiovascular events such as myocardial infarction and stroke.

Internationally accepted research conclusions accumulated by other countries and regions cannot be directly applied to Qatar's local context; several unavoidable local practical issues must also be noted: whether the medications prescribed to patients are available at local pharmacies, whether the cost of medication is affordable for ordinary people, whether patients will actually adhere to the doctor's advice to take their medication every day, and whether clinics can continuously follow up on patients' recovery to avoid losing contact after a single outpatient visit. It should be clarified that all the implementation suggestions mentioned above are practical directions derived from existing research. None of the clinical trials referenced in this study calculated a set of precise efficacy values specifically tailored to the Qatari population that could be directly copied.

Next, local researchers in Qatar can carry out targeted new studies to properly evaluate several practical scenarios in daily diagnosis and treatment: whether the medication prescribed by doctors complies with regulations and is suitable for the specific patient, whether the patient can consistently take the medication as required, whether the patient's blood lipids show the expected changes after starting the medication, and whether any side effects occur from taking the medication. If conducting a pre-planned long-term follow-up study, several details must be noted: the total fat weight in fish oil products and the content of the actually functional active fatty acids within them must be counted separately and must not be conflated; in addition, it is necessary to truthfully record whether each patient is simultaneously taking statins, a common lipid-lowering medication, and whether they have adjusted their daily diet in coordination with the treatment. If your research goal is to verify that this therapy can indeed reduce the risk of cardiovascular disease or pancreatitis, you must prepare to follow up for a sufficiently long period, and have dedicated medical staff to verify and confirm the patient's actual clinical incidence, rather than relying solely on the patient's own account. Small-scale studies that only compare changes in patients' blood lipid levels before and after taking medication are completely unable to answer these core questions about the actual efficacy of the treatment.

In addition to this type of research, comparative studies of different treatment plans must also be conducted to fully clarify three key issues: which specific patients can gain the most practical benefits from this treatment, how the patient's own risk of arrhythmia will affect the actual value of this therapy, and whether a direct comparison of the formulas of different prescription medications will change the current industry's universal understanding of these medications. One final point: if anyone wants to propose that the efficacy of this therapy varies across populations of different races or regions, they must present solid research evidence with a compliant design. It is not permissible to arbitrarily extrapolate this overgeneralized conclusion solely from the results of studies on small-scale subgroup populations.

Limitations of the review

When selecting the studies included in this paper, we conducted retrievals with a clear purpose and screened content based on narrative needs, which introduces the risk of selection bias rather than comprehensively including all eligible studies, we selected studies and content with pre-set directions, making it highly likely that only content aligning with our own inclinations was included, which could lead to biased final conclusions.

We did not conduct specialized standardized assessments for these risks: we neither carried out formal, individual study-specific risk of bias scoring, nor performed assessments of evidence credibility both of which are routine practices in academic research to safeguard the reliability of conclusions, and neither was implemented for this work.

In addition, the conditions of different trials vary widely, making it impossible to directly compare their results. The trial populations differ across studies, the observed endpoints differ, the medication dosages and pharmaceutical dosage forms are not the same, and even the reference controls used vary, so the numerical values from these trials cannot be simply compared directly.

The scope of this review also has clear limitations: it only focuses on major blood lipid and cardiovascular research in adults, and does not comprehensively cover pregnant populations, pediatric-related conditions, familial chylomicronaemia, nor does it encompass every emerging triglyceride treatment method.

In summary, this review has these limitations. Therefore, to translate these contents into local application and put forward recommendations, the conclusions herein cannot be directly applied; this must be done in

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combination with local clinical realities and the context of the local drug formulary.

CONCLUSION

The use of Omega-3 therapy to control triglycerides is clearly established, but the claim that it delivers cardiovascular benefits is not universally applicable. Whether it works depends on the specific formulation used and the patient it is administered to, and cannot be generalized across all cases.

Even the clinical research evidence behind Prescription EPA-containing Omega-3 products, which are only available with a doctor's prescription, cannot be applied arbitrarily. To correctly interpret what this evidence states, one must first align with the positive trials that originally proved this product's efficacy, reviewing those trials' enrollment criteria what types of patients were eligible to participate and examining what baseline treatments those participants received concurrently during the trial. Only after matching all these prerequisite conditions can one grasp the true scope of applicability of the evidence.

For Omega-3 products mixed with other ingredients, or over-the-counter Omega-3 supplements that can be purchased directly without a prescription, one must not categorize them alongside the aforementioned Prescription EPA product solely because they are both labeled as Omega-3 related, and then assume they can deliver identical cardiovascular protection effects. They cannot claim that same therapeutic efficacy.

To use this type of Omega-3 therapy in compliance with clinical standards, several key requirements must be fully met. First, clear treatment goals must be set from the very start, and medication must not be prescribed without a clear understanding. During treatment, patients' LDL cholesterol levels must be continuously monitored, not just their triglyceride levels; at the same time, it is necessary to investigate whether the patient's dyslipidemia is triggered by other diseases or underlying factors, in other words, to identify the secondary causes of elevated triglycerides. In addition, medication risks must be assessed in advance, with close attention paid to whether patients develop atrial fibrillation after taking the drug, or any other confirmed side effects none of these can be overlooked.

Finally, when formulating specific implementation plans for local rollout across different regions, the core consideration must always be "whether this therapy is truly appropriate for this specific patient". It is wrong to implement a one-size-fits-all approach, routinely prescribing Omega-3 supplements to all patients simply because their triglyceride levels are found to be elevated, regardless of individual circumstances.

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