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Effects of Astaxanthin on Human Sperm Morphology: A Systematic Review and Meta-Analysis

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Introduction: Oxidative stress plays a central role in the pathophysiology of male infertility, contributing to impaired sperm concentration, motility, morphology, and DNA integrity. Astaxanthin, a potent antioxidant carotenoid, has been proposed as a therapeutic agent for improving semen quality. However, clinical findings remain inconsistent. This systematic review and meta-analysis aimed to evaluate the effects of astaxanthin supplementation on conventional human semen morphology.

Methods: A systematic search of PubMed, Scopus, and Google Scholar was conducted up to December 2025 according to PRISMA guidelines. Randomized controlled trials evaluating the effects of astaxanthin supplementation on sperm morphology in adult men were included. Weighted mean differences (WMDs) with 95% confidence intervals (CIs) were calculated using a random-effects model based on changes from baseline. Between-study heterogeneity was assessed using the I^2 statistic.

Results: Four randomized controlled trials comprising 166 participants (86 interventions, 80 controls) met the inclusion criteria. Astaxanthin supplementation did not significantly affect sperm morphology (WMD: 0.02; 95% CI: -0.51, 0.55).

Conclusion: Current evidence does not support a significant beneficial effect of astaxanthin supplementation on conventional semen morphology in infertile men. Given the limited number of trials and substantial heterogeneity, larger well-designed randomized studies with standardized protocols and comprehensive reproductive endpoints are warranted.

Keywords: Astaxanthin; Male infertility; Sperm morphology; Antioxidants; Meta-analysis.

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Introduction

Infertility is a major global health concern, affecting approximately 15% of couples worldwide, with male factors contributing to nearly half of all cases [5, 6]. Impaired semen quality, including reduced sperm concentration, motility, morphology, and increased DNA fragmentation, remains a leading cause of male infertility [7]. Despite advances in assisted reproductive technologies, the identification of effective, non-invasive therapeutic strategies aimed at improving sperm quality continues to be a priority in reproductive medicine [8].

Accumulating evidence suggests that oxidative stress plays a central role in the pathophysiology of male infertility [9]. Reactive oxygen species (ROS), when produced in excess, can overwhelm the intrinsic antioxidant defense system of seminal plasma, leading to lipid peroxidation of sperm membranes, mitochondrial dysfunction, DNA damage, and apoptosis [10]. Given the high content of polyunsaturated fatty acids in the sperm plasma membrane and the limited cytoplasmic antioxidant capacity of spermatozoa, sperm cells are particularly vulnerable to oxidative injury [11, 12].

In recent years, antioxidant supplementation has emerged as a promising therapeutic approach for improving semen parameters. Among various antioxidants, astaxanthin, a xanthophyll carotenoid derived primarily from marine sources such as microalgae and seafood, has attracted considerable attention due to its potent antioxidant and anti-inflammatory properties [13]. Astaxanthin exhibits a unique molecular structure that enables it to span lipid bilayers and effectively neutralize free radicals both at the membrane surface and within the lipid core [14]. Experimental studies have demonstrated that astaxanthin may enhance mitochondrial function, reduce lipid peroxidation, modulate inflammatory pathways, and improve cellular redox balance [15, 16].

Preclinical investigations and clinical trials have suggested potential beneficial effects of astaxanthin supplementation on sperm motility, concentration, morphology, and DNA integrity [17-19]. However, findings across studies remain inconsistent, possibly due to differences in study design, dosage, treatment duration, baseline fertility status, and methodological quality. To date, no comprehensive quantitative synthesis has conclusively evaluated the magnitude and consistency of astaxanthin's effects on human sperm morphology. Therefore, the present systematic review and meta-analysis was conducted to comprehensively evaluate the effect of astaxanthin supplementation on human semen morphology. By synthesizing the available evidence, this study aims to provide a clearer understanding of the clinical utility of astaxanthin in the management of male infertility.

Methods

This meta-analysis was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement) [20]. Systematic search was done at the PubMed and Scopus for eligible studies published until December 2025.

Medical subject headings (MeSH) and non-MeSH keywords were used: ((“sperm DNA fragmentation”[tiab] OR SDF[tiab] OR DFI[tiab] OR fertility[tiab] OR “male infertility”[tiab] OR infertility[tiab] OR “sperm dysfunction”[tiab] OR “sperm DNA damage”[tiab] OR asthenozoospermia[tiab] OR oligozoospermia[tiab] OR oligoasthenozoospermia[tiab] OR oligoasthenoteratozoospermia[tiab] OR teratozoospermia[tiab] OR “normal sperm morphology”[tiab] OR “semen parameters”[tiab] OR “semen quality”[tiab] OR “sperm abnormal”[tiab] OR “sperm characteristics”[tiab] OR “human sperm”[tiab] OR “impaired spermatogenesis”[tiab] OR “Low Sperm Count”[tiab] OR Hypospermatogenesis[tiab] OR azoospermia[tiab]) AND (Astaxanthin[tiab] OR astaxanthine[tiab] OR ASTA[tiab] OR AstaCarox[tiab] OR ASX[tiab] OR E-astaxanthin[tiab])). The literature search was performed without restrictions on language or publication date. Duplicate studies were removed. References in all eligible papers were also searched for articles that were not found through the initial search.

Inclusion Criteria: Studies were eligible for inclusion if they met the following criteria: 1) randomized controlled trials (RCTs) employing either parallel or crossover designs; 2) enrolled participants aged ≥18 years; 3) evaluated effects of astaxanthin on semen morphology; 4) had appropriate control group; and 5) reported quantitative outcome data as means or medians with corresponding measures of variability, such as standard deviations (SDs), standard errors (SEs), 95% confidence intervals (CIs), interquartile ranges (IQRs), or ranges.

Exclusion Criteria: Observational studies, including cross-sectional and cohort designs, as well as animal model experiments, in vitro studies, narrative or systematic reviews, and studies lacking randomized allocation were excluded. Additionally, trials evaluating the effects of astaxanthin in combination with other interventions, studies with insufficient or incomplete outcome data, and those that did not assess key semen parameters, including sperm morphology, were excluded. Publications without an English full-text were also excluded from the analysis. Grey literature, book chapters, conference abstracts, interviews, comments, opinion pieces, methodological studies, editorials, and letters to the editor were also excluded.

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Data Extraction: Two reviewers independently extracted data from all eligible studies using a standardized data extraction form. The extracted information included: 1) first author's name; 2) year of publication; 3) study location; 4) study design; 5) participants' health status; 6) sample size in the astaxanthin and control groups; 7) mean age of participants; 8) semen parameters-namely sperm morphology measured at baseline and at the end of the intervention, as well as within-group changes; 9) type of astaxanthin administered; and 10) dosage of astaxanthin. When sperm parameters were reported using different measurement units, values were converted to the most commonly used units to ensure consistency across studies. In cases of missing, unclear, or ambiguous data, corresponding authors were contacted to obtain accurate and complete information.

Statistical Method: Pooled effect sizes were calculated using changes from baseline to the end of intervention and expressed as weighted mean differences (WMDs) with corresponding 95% CIs. A random-effects model was applied to all meta-analyses to account for potential between-study heterogeneity. When studies did not report mean changes and their corresponding SDs, these values were derived using the following formulas: mean change was calculated as the difference between final and baseline values, and the SD of the change was estimated by the following formula:

$$SD = \sqrt{SD_{Baseline}^2 + SD_{Final}^2 - (2 \times R \times SD_{Baseline} \times SD_{Final}}$$

Where, R represents the correlation coefficient between baseline and final measurements. When outcome data were reported as medians with IQRs, means and SDs were estimated using the method described by Hozo et al. [21]. SEs were converted to SDs using the formula:

$$SD = SE \times \sqrt{n}$$

Where, n denotes the sample size. Similarly, when results were presented as 95% CIs, SDs were derived using the formula:

$$SD = \frac{\sqrt{n} \times (Upper\ Limit - Lower\ Limit)}{3.92}$$

Between-study heterogeneity was evaluated using the I^2 statistic, with values greater than 50% indicating substantial heterogeneity. Subgroup analyses were conducted according to astaxanthin dosage, study sample size, type of control (placebo-controlled vs observational), study duration (2 to 4 months vs 4 to 6 months), and assessment method (computer-assisted sperm analysis [CASA] vs non-CASA). All statistical analyses were conducted using STATA software (version 12.0; StataCorp). A two-sided P-value< 0.05 was considered as statistically significant.

Results

Initially, 63 articles were found through a database search and reference lists. After removing duplicate studies, 59 publications remained. Screening by title and abstract resulted in 14 articles, of which 10 were excluded because they: had been conducted in perspective or retrospective design (n=2), did not report relevant data (n=1), had not performed randomized allocation (n=3), animal studies (n=1), used cell cultures (n=2) and were studies that evaluated the effects of carnitine in combination with other interventions (n=1). Finally, 4 articles remained for the final analysis [1-4]. A flow-diagram of the study's selection is displayed in Figure 1.

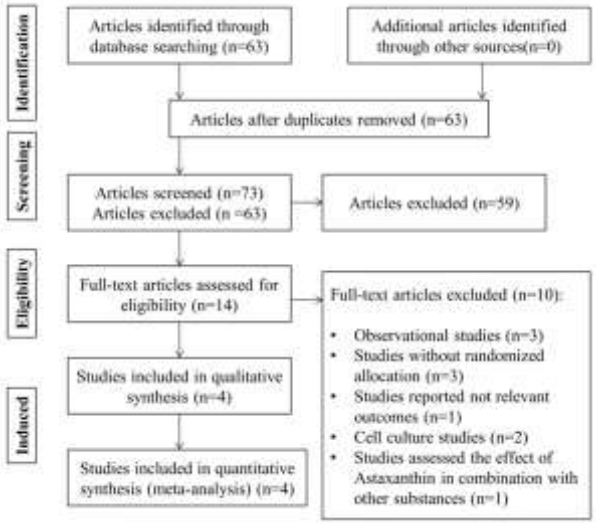

Figure 1: Flow diagram of study selection

Systematic Review: General characteristics of the included studies are summarized in Table 1. All studies were randomized clinical trials published in English, between 2005 and 2024. In total, 166 participants were included (86 in the intervention and 80 in the control group). Participants' age ranged between 25 and 45 yrs. All trials were conducted in infertile subjects, who had varicocele [3], primary or secondary infertility [2], oligoasthenoteratozoospermia [4], and infertility [1]. Studies were conducted in Belgium (n=1) [1], Indonesia (n=1) [22], Iran (n=1) [3], and Slovenia (n=1) [4]. The effect of astaxanthin supplementation was compared with a placebo in 4 studies [1-4]. Astaxanthin was administrated in doses varied between 6 and 16 mg/day, for intervention durations of 30 to 130 days. Astaxanthin type was

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ASX [3] or Astaxanthin in other three papers [1, 2, 4]. This drug supplements were given into either caplet [2] or capsule [1, 3, 4].

Meta-analysis

Effects of astaxanthin on sperm morphology

Among the three included studies evaluating the effect of astaxanthin supplementation on sperm morphology, one study reported a non-significant increase in sperm morphology compared with the control group [2]. Kumalic et al (2021) observed a non-significant decrease [4], while Salih et al. (2024) demonstrated a minimal and non-significant increase [3]. Pooling the effect sizes from these three studies using a random-effects model indicated that astaxanthin supplementation had no significant effect on sperm morphology compared with the control group (WMD: 0.02; 95% CI: -0.51, 0.55; P=0.447; I^2 = 0.0%) (Figure 2).

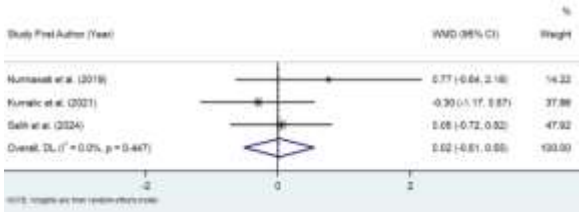

Figure 2. Forest plot showing the effects of astaxanthin supplementation on sperm morphology using the random effects model. Values are WMDs (95% CIs) comparing changes in sperm morphology over time between treatment and control groups. WMD, weighted mean difference

Discussion

The present systematic review and meta-analysis synthesized the available evidence from randomized controlled trials to evaluate the effects of astaxanthin supplementation on human semen morphology. Overall, pooled analyses demonstrated that astaxanthin supplementation did not significantly improve sperm morphology. These finding suggests that current evidence does not robustly support a clinically meaningful improvement in conventional semen morphology following astaxanthin supplementation.

From a biological perspective, the lack of consistent improvement in semen parameters is somewhat unexpected, given the well-established antioxidant properties of astaxanthin. Astaxanthin is known to exert potent free radical scavenging activity, stabilize lipid membranes, and improve mitochondrial function [16]. Since oxidative stress is a central contributor to sperm dysfunction [23], it is theoretically plausible that astaxanthin may enhance sperm quality by reducing lipid peroxidation and preserving DNA integrity [24, 25]. Experimental studies have demonstrated improvements in oxidative biomarkers and mitochondrial activity following astaxanthin exposure [26-28]. However, improvements in biochemical markers do not necessarily translate into measurable changes in standard semen parameters, particularly within short intervention durations.

Differences in baseline fertility status (e.g., varicocele, oligoasthenoteratozoospermia, and idiopathic infertility), dosage (6–16 mg/day), and duration (30–130 days) may have contributed to the inconsistent findings.

The substantial heterogeneity observed for sperm morphology further complicates interpretation. Variations in study design (parallel vs. double-blind), sample size, laboratory assessment methods (CASA vs. manual analysis), and baseline oxidative stress levels may explain the divergent results. In addition, semen analysis is inherently subject to intra-individual variability, which may attenuate the detection of intervention effects in small trials.

Comparison with previous literature reveals mixed findings. Individual randomized trials have reported improvements in specific semen morphology, while others have shown null or even negative effects [29, 30]. Preclinical studies and in vitro investigations suggest that astaxanthin may reduce reactive oxygen species and DNA fragmentation, yet these mechanistic benefits may not consistently translate into clinically measurable improvements in conventional semen analysis outcomes [17, 31]. Several strengths of this meta-analysis should be acknowledged. First, only randomized controlled trials were included, enhancing the internal validity of pooled estimates. Second, standardized statistical methods were applied using change-from-baseline data, reducing bias associated with baseline differences. Third, subgroup analyses were planned to explore potential sources of heterogeneity.

However, this study also has important limitations. The number of available trials was limited (n=4), with relatively small sample sizes, reducing statistical power. Considerable heterogeneity was present in most pooled analyses. Intervention duration varied and, in some studies, may have been shorter than a complete spermatogenic cycle (~74 days), potentially limiting observable effects. Furthermore, differences in astaxanthin formulation and bioavailability were not consistently reported. Finally, conventional semen parameters may not fully capture functional sperm competence, such as DNA fragmentation, oxidative stress markers, or fertilization outcomes.

Taken together, the current evidence does not provide strong support for routine astaxanthin supplementation as an effective monotherapy for improving conventional semen parameters in infertile men. Nevertheless, given its favorable safety profile and biological plausibility, astaxanthin may still have a role in specific subgroups characterized by elevated oxidative stress. Future large-scale, well-designed randomized controlled trials with standardized dosing

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regimens, adequate intervention duration, and comprehensive reproductive endpoints, including sperm DNA fragmentation, oxidative stress biomarkers, and pregnancy outcomes, are needed to clarify the therapeutic potential of astaxanthin in male infertility.

Limitations

Several limitations should be considered when interpreting the findings of this meta-analysis. First, only four randomized controlled trials with relatively small sample sizes were included, limiting statistical power and the precision of pooled estimates. Second, substantial heterogeneity was observed across most outcomes, likely due to differences in participant characteristics, baseline fertility status, astaxanthin dosage, intervention duration, and semen assessment methods.

Third, intervention periods in some trials may not have fully covered a complete spermatogenic cycle, potentially underestimating treatment effects. Fourth, variability in astaxanthin formulation and bioavailability was not consistently reported, which may have influenced clinical responses. Fifth, conventional semen morphology was the primary outcomes, while more sensitive functional markers such as sperm DNA fragmentation, oxidative stress biomarkers, and pregnancy outcomes were not consistently available for analysis. Finally, the limited number of included studies precluded formal assessment of publication bias.

Conclusion

In conclusion, this systematic review and meta-analysis indicates that astaxanthin supplementation does not significantly improve conventional semen morphology in infertile men. Given the limited number of available trials, small sample sizes, and substantial heterogeneity across studies, current evidence is insufficient to support the routine use of astaxanthin as a monotherapy for improving semen quality. Well-designed, large-scale randomized controlled trials with standardized dosing regimens and comprehensive reproductive outcomes are required to clarify the potential therapeutic role of astaxanthin in male infertility.

Ethics Declaration

Not applicable

Funding

Not applicable

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Table 1. Characteristics of randomized trials on the effects of Astaxanthin on sperm parameters included in the meta-analysis

N	Author's Name (Year)	Location	Subjects(n)	Health Condition	Age	Design	Duration	Control	Kind(s) of Astaxanthin (Dose)	Parameters	Change	
											Intervention	Control
1	Comhaire et al. (2005)[1]	Belgium	30: Astaxanthin (n=19) Palcebo (n=11)	Infertility	Astaxanthin (31.4±4.5) Placebo (33.2±5.6)	Randomized Trial	3 months	Placebo	Astaxanthin (16mg/day)	Sperm Morphology	1.8±1.42	1.4±7.33
2	Nurmawati et al. (2019)[2]	Indonesia	25: Astaxanthin (n=19) Palcebo (n=6)	Primary or secondary infertility	25.48± 6.66	Randomized Trial	1 month	Placebo	Astaxanthin (8mg/day)	Sperm Morphology	0.94±0.39	0.17±1.75
3	Salih et al. (2024)[3]	Iran	41: Astaxanthin (n=21) Palcebo (n=20)	Varicocele	Astaxanthin (35.8±4.61) Placebo (36.9±4.76)	Randomized Clinical Trial	3 months	Placebo	ASX (6mg/day)	Sperm Morphology	0.45±0.58	0.4±1.66
4	Kumalic et al. (2021)[4]	Slovenia	72: Astaxanthin (n=37) Palcebo (n=35)	Oligospermia with/without Astheno-or Teratozoospermia (O±A±T)	Astaxanthin (35±5.2) Placebo (36.4±5.5)	Randomized Double-Blind Trial	3 months	Placebo	Astaxanthin (16mg/day)	Sperm Morphology	-0.2±0.78	0.1±2.49

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