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NUTRITIONAL AND LIFESTYLE FACTORS IN AGE-RELATED MACULAR DEGENERATION: A NARRATIVE REVIEW FROM A LIFESTYLE MEDICINE PERSPECTIVE

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Abstract:
Background: Age-related macular degeneration (AMD) is a leading cause of irreversible vision loss among older adults worldwide, affecting approximately 196 million people globally in 2020, with projections reaching 288 million by 2040. While genetic predisposition plays a significant role, modifiable nutritional and lifestyle factors have emerged as critical determinants in the development and progression of AMD. This narrative review aims to synthesize the current evidence on nutritional and lifestyle interventions relevant to AMD within the framework of lifestyle medicine. **Methods:** A comprehensive literature search was conducted across PubMed/MEDLINE, Scopus, Cochrane Library, and Google Scholar for English-language articles published from January 2000 through March 2025. Search terms combined "age-related macular degeneration" OR "AMD" with specific lifestyle and nutritional terms. Study types included were randomized controlled trials (RCTs), prospective and retrospective cohort studies, case-control studies, cross-sectional studies, systematic reviews, meta-analyses, and clinical practice guidelines. Exclusion criteria included non-English publications, conference abstracts without full-text availability, case reports, editorials, and studies focused exclusively on pharmacological or surgical interventions without a lifestyle component. The Scale for the Assessment of Narrative Review Articles (SANRA) guidelines were followed. **Results:** Strong evidence supports the role of AREDS2 supplementation (vitamins C, E, lutein, zeaxanthin, zinc, and copper) in reducing AMD progression by up to 25%. Mediterranean diet adherence is associated with a 41% reduced risk of incident advanced AMD. Smoking is the most significant modifiable risk factor, with current smokers having a 2- to 4-fold increased risk. Physical activity, weight management, and ultraviolet light protection demonstrate additional protective effects. Emerging evidence also links sleep quality, gut microbiome composition, and stress management to retinal health. **Conclusion:** Nutritional and lifestyle factors play a substantial role in AMD prevention and management. A lifestyle medicine approach

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integrating dietary optimization, smoking cessation, physical activity, weight management, UV protection, and adequate sleep represents a comprehensive strategy for reducing AMD burden. Clinicians should incorporate these modifiable factors into routine ophthalmic practice.

Keywords: age-related macular degeneration, lifestyle medicine, nutrition, Mediterranean diet, AREDS2, carotenoids, smoking cessation, physical activity, retinal health

1. Introduction

Age-related macular degeneration (AMD) is a progressive degenerative disease of the central retina that represents the leading cause of irreversible vision loss among older adults in developed countries. Globally, AMD accounts for approximately 8.7% of all blindness, and the number of affected individuals was estimated at 196 million in 2020, with projections suggesting an increase to 288 million by 2040 [1]. The disease is characterized by the accumulation of extracellular deposits (drusen) beneath the retinal pigment epithelium, leading to chronic oxidative stress, inflammation, and eventual destruction of photoreceptors in the macular region [2].

AMD is classified into two principal forms: dry (atrophic) AMD, which accounts for approximately 85–90% of cases and progresses through geographic atrophy; and wet (neovascular or exudative) AMD, characterized by aberrant choroidal neovascularization that can lead to rapid and severe vision loss [2]. While anti-vascular endothelial growth factor (anti-VEGF) therapies have revolutionized the treatment of neovascular AMD, there remains no curative treatment for either form. Consequently, prevention and risk reduction through modifiable factors have become a focal point of clinical and public health strategies.

The pathogenesis of AMD is multifactorial, involving complex interactions between genetic susceptibility, age-related changes, oxidative stress, chronic inflammation, and environmental exposures [20]. Among environmental factors, nutritional intake and lifestyle behaviors have emerged as significant modulators of disease risk and progression. This aligns closely with the principles of lifestyle medicine, which emphasizes evidence-based therapeutic interventions in nutrition, physical activity, stress management, sleep optimization, avoidance of risky substances, and social connectedness as foundational pillars of health [22].

This narrative review aims to comprehensively synthesize the current evidence on nutritional and lifestyle factors influencing AMD development and progression, organized within a lifestyle medicine framework. By integrating findings from landmark clinical trials, systematic reviews, meta-analyses, and observational studies, this review provides clinicians with actionable insights for incorporating lifestyle-based strategies into ophthalmic care.

2. Methods

A comprehensive literature search was performed across four electronic databases: PubMed/MEDLINE, Scopus, Cochrane Library, and Google Scholar, covering articles published from January 2000 through March 2025. The search strategy employed Boolean combinations of MeSH terms and free-text keywords structured around two core concepts. The first concept targeted the disease ("age-related macular degeneration" OR "AMD" OR "macular degeneration" OR "retinal degeneration"). The second concept targeted modifiable factors ("nutrition" OR "diet" OR "Mediterranean diet" OR "AREDS" OR "AREDS2" OR "lutein" OR "zeaxanthin" OR "carotenoids" OR "omega-3 fatty acids" OR "antioxidants" OR "smoking" OR "tobacco" OR "physical activity" OR "exercise" OR "obesity" OR "body mass index" OR "ultraviolet" OR "sunlight exposure" OR "sleep" OR "gut microbiome" OR "lifestyle medicine"). These two concepts were combined using the AND operator.

Eligible study designs included randomized controlled trials (RCTs), prospective and retrospective cohort studies, case-control studies, cross-sectional studies, systematic reviews, meta-analyses, and clinical practice guidelines published by recognized professional organizations. Exclusion criteria comprised non-English language publications, conference abstracts without full-text availability, case reports, letters to the editor, editorials, animal studies without direct clinical translation, and studies focused exclusively on pharmacological (e.g., anti-VEGF therapy) or surgical interventions without a lifestyle or nutritional component.

Titles and abstracts were screened for relevance, and full texts of potentially eligible articles were reviewed. Reference lists of included systematic reviews and key original articles were manually searched (backward citation tracking) to identify additional relevant publications. Priority was given to large-scale RCTs (particularly the AREDS and AREDS2 studies), population-based prospective cohorts, and recent systematic reviews and meta-analyses published within the past five years (2020–2025) to ensure currency of evidence. Earlier landmark studies were retained when they provided foundational evidence not superseded by more recent work. This narrative review was conducted and reported in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA) guidelines.

3. Nutritional Factors in AMD

3.1 AREDS and AREDS2 Supplementation

The Age-Related Eye Disease Study (AREDS) and its successor AREDS2 represent the most influential clinical trials examining nutritional supplementation for AMD. The original AREDS trial (2001) demonstrated that a formulation containing high-dose vitamin C (500 mg), vitamin E (400 IU), beta-carotene (15 mg), and zinc (80 mg with 2 mg copper) reduced the 5-year risk of progression from intermediate to advanced AMD by approximately 25% [25].

AREDS2 (2013) evaluated the addition of lutein (10 mg) and zeaxanthin (2 mg), docosahexaenoic acid (DHA, 350 mg) and eicosapentaenoic acid (EPA, 650 mg), and modifications to the original formula. While the primary analysis showed no statistically significant additional benefit of lutein/zeaxanthin or omega-3 fatty acids when added to the original AREDS formula, the study revealed that lutein/zeaxanthin could safely and effectively replace beta-carotene [3]. This was clinically significant because beta-carotene was associated with an increased risk of lung cancer in smokers.

The 10-year follow-up of AREDS2 (Report 28, 2022) provided compelling long-term evidence. Participants assigned to lutein/zeaxanthin had an associated 9% reduction in progression to late AMD (HR 0.91, 95% CI 0.84–0.99) compared with those not receiving these carotenoids. When directly compared with beta-carotene, lutein/zeaxanthin demonstrated a 15% risk reduction (HR 0.85, 95% CI 0.73–0.98). Furthermore, beta-carotene was associated with a nearly doubled risk of lung cancer (OR 1.82, 95% CI 1.06–3.12), whereas lutein/zeaxanthin showed no such association [4]. These findings firmly established the AREDS2 formulation with lutein/zeaxanthin as the standard of care for patients with intermediate AMD or advanced AMD in one eye.

3.2 Carotenoids: Lutein, Zeaxanthin, and Meso-Zeaxanthin

Lutein and zeaxanthin are xanthophyll carotenoids that constitute the macular pigment, a yellow-colored layer concentrated in the fovea. These pigments serve two critical protective functions: filtering phototoxic blue light before it reaches the photoreceptors, and acting as potent antioxidants that neutralize reactive oxygen species in the retinal environment [23,24,27]. Meso-zeaxanthin, a stereoisomer of zeaxanthin found predominantly at the epicenter of the fovea, is derived from the enzymatic conversion of lutein within the retina.

Unlike beta-carotene, humans cannot synthesize lutein and zeaxanthin endogenously; these must be obtained through dietary intake. Rich dietary sources include dark green leafy vegetables (kale, spinach, collard greens), egg yolks, corn, orange peppers, and various fruits [27]. Epidemiological studies consistently demonstrate an inverse relationship between dietary carotenoid intake and AMD risk. Higher macular pigment optical density (MPOD), which reflects retinal carotenoid concentration, is associated with reduced AMD risk and improved visual function [23].

3.3 Omega-3 Fatty Acids

The retina, particularly the photoreceptor outer segments, has the highest concentration of docosahexaenoic acid (DHA) of any tissue in the human body. DHA is essential for maintaining the structural integrity and fluidity of photoreceptor cell membranes and possesses anti-inflammatory and neuroprotective properties [19]. Epidemiological evidence from the AREDS cohort demonstrated that participants with the highest dietary intake of omega-3 fatty acids had a 30% reduced likelihood of developing advanced AMD compared with those with the lowest intake (OR 0.61, 95% CI 0.41–0.90) [19].

However, the AREDS2 trial did not demonstrate a statistically significant benefit of DHA/EPA supplementation (350 mg DHA + 650 mg EPA) when added to the AREDS formula [3]. This apparent contradiction may be explained by the relatively high baseline omega-3 intake in the study population, the modest supplementation dose used, or the potential that dietary omega-3 intake through whole food sources may provide different bioavailability and synergistic effects compared with supplements. Current evidence supports encouraging omega-3 rich food intake (fatty fish such as salmon, mackerel, sardines consumed at least twice weekly) rather than routine supplementation for AMD prevention [22].

3.4 Mediterranean Diet

The Mediterranean dietary pattern, characterized by high consumption of fruits, vegetables, legumes, whole grains, nuts, olive oil, and fish, with moderate wine intake and low consumption of red meat and processed foods, has emerged as a particularly promising dietary approach for AMD prevention. This dietary pattern provides a comprehensive array of antioxidants, anti-inflammatory compounds, carotenoids, and omega-3 fatty acids that collectively support retinal health [5,6,7].

The EYE-RISK Consortium study, pooling data from two large European prospective cohorts (the Rotterdam Study and the Alienor Study), demonstrated that higher adherence to the Mediterranean diet was associated with a 41% reduced risk of incident advanced AMD (HR 0.59, 95% CI 0.37–0.95) [5]. The Coimbra Eye Study similarly showed that high Mediterranean diet adherence (mediSCORE ≥6) was protective against AMD (OR 0.62, 95% CI 0.38–0.97), with the protective effect driven largely by fruit consumption and antioxidant micronutrient intake [6].

A systematic review and meta-analysis published in 2025 further consolidated this evidence, confirming that greater Mediterranean diet adherence was consistently associated with lower odds of AMD development and reduced progression to advanced stages. The protective mechanism is multifactorial, involving reduced systemic inflammation, decreased oxidative damage to retinal tissues, improved vascular endothelial function, and enhanced delivery of macular pigment precursors [8]. These findings provide strong justification for recommending the Mediterranean diet as a cornerstone of lifestyle-based AMD prevention strategies.

3.5 Dietary Glycemic Index and Carbohydrate Quality

Emerging evidence suggests that dietary glycemic index (GI) and glycemic load may influence AMD risk through mechanisms related to advanced glycation end-products (AGEs), oxidative stress, and microvascular dysfunction. High-GI diets promote the formation of AGEs, which accumulate in Bruch's membrane and the retinal pigment epithelium, contributing to drusen formation and complement activation [21]. Observational data indicate that participants consuming diets with lower GI had a reduced risk of large drusen and advanced AMD. While the evidence is still evolving, advising patients to favor whole grains, legumes, and minimally processed carbohydrates

over refined sugars and starches aligns with both AMD risk reduction and broader cardiometabolic health goals [21].

4. Lifestyle Factors in AMD

4.1 Smoking

Smoking is unequivocally the most significant modifiable risk factor for AMD. The association between smoking and AMD has been consistently demonstrated across multiple study designs, populations, and geographic regions. A comprehensive review of epidemiological evidence found that current smokers have a 2- to 4-fold increased risk of AMD compared with never-smokers [9,10]. The dose-response relationship is well established: the risk increases with the number of pack-years of smoking, and heavy smokers (≥40 pack-years) face the greatest risk for both geographic atrophy and choroidal neovascularization [11].

The biological mechanisms through which smoking damages the retina are multifold. Cigarette smoke contains numerous oxidants and pro-inflammatory compounds that deplete macular carotenoid pigments, reduce retinal antioxidant defenses, impair choroidal blood flow, activate the complement cascade in retinal cells, and promote endothelial dysfunction in retinal vasculature [10,32]. Importantly, smoking also diminishes the therapeutic efficacy of anti-VEGF treatments in neovascular AMD and reduces the protective benefits of AREDS supplementation.

The encouraging finding is that smoking cessation reduces AMD risk. Former smokers have a significantly lower risk than current smokers, and after 20 or more years of cessation, the risk approaches that of never-smokers [9,10]. This provides a powerful motivational message for patients and underscores the role of smoking cessation counseling as a critical component of AMD prevention within the lifestyle medicine framework.

4.2 Physical Activity

A growing body of evidence supports a protective association between regular physical activity and AMD. The Blue Mountains Eye Study, a large Australian prospective cohort, found that participants who engaged in regular walking had a reduced 15-year incidence of AMD [12]. A systematic review and meta-analysis by McGuinness et al. (2017) reported that physically active individuals had approximately 8–10% lower odds of any AMD compared with sedentary counterparts [13].

The protective mechanisms are likely mediated through improved cardiovascular fitness, reduced systemic inflammation, enhanced antioxidant enzyme activity, better regulation of blood pressure, and improved lipid profiles – all of which contribute to maintaining adequate choroidal perfusion and reducing oxidative stress in the retina [12,30]. The Tromsø Study demonstrated that regular physical activity (30 minutes or more per session) was significantly associated with reduced late AMD in women (OR 0.41, 95% CI 0.18–0.96) [30]. Current evidence supports recommending at least 150

minutes of moderate-intensity aerobic activity per week as part of a comprehensive AMD prevention strategy, consistent with general physical activity guidelines [15].

4.3 Body Weight and Obesity

Elevated body mass index (BMI) and central obesity have been associated with increased AMD risk and progression. Seddon et al. (2003) demonstrated that higher BMI, larger waist circumference, and higher waist-hip ratio were associated with progression of AMD, with obese individuals (BMI ≥30) having a significantly increased risk of progression to advanced AMD compared with those of normal weight [16]. Adams et al. (2011) confirmed that abdominal obesity, measured by waist circumference, was independently associated with early AMD [17].

Obesity promotes AMD through multiple pathways, including increased systemic inflammation (elevated C-reactive protein and pro-inflammatory cytokines), dyslipidemia, insulin resistance, and enhanced oxidative stress. Adipose tissue-derived inflammation can compromise the retinal pigment epithelium and impair choroidal vascular function. Furthermore, obesity reduces the bioavailability and retinal delivery of protective carotenoid pigments, as these lipophilic compounds may become sequestered in adipose tissue [29]. Weight management through dietary modification and regular physical activity therefore represents a dual-benefit strategy that addresses both systemic metabolic risk and retinal health.

4.4 Sunlight and Ultraviolet Exposure

Cumulative lifetime exposure to ultraviolet (UV) and high-energy blue light has been implicated as a risk factor for AMD, though the evidence is more heterogeneous compared with smoking. Chronic light exposure contributes to photochemical damage and oxidative stress in the retina, particularly in individuals with low macular pigment density who lack adequate blue light filtration [14]. Schick et al. (2016) reported that a history of high sunlight exposure was significantly associated with AMD, particularly in populations with lighter iris color [14].

Protective recommendations include wearing UV-blocking sunglasses and wide-brimmed hats during prolonged outdoor exposure, avoiding direct sunlight during peak hours, and maintaining adequate macular carotenoid levels through dietary intake to enhance endogenous blue light filtration [22,26]. These measures are particularly relevant in tropical and subtropical regions with high ambient UV radiation levels.

4.5 Sleep Quality and Circadian Rhythm

Emerging evidence suggests a relationship between sleep disturbances and AMD, although this area requires further investigation. The retina is highly metabolically active and undergoes critical repair processes during sleep, including photoreceptor outer segment renewal and autophagy-mediated clearance of cellular debris. Disrupted sleep and circadian misalignment may impair these restorative processes and increase oxidative stress in the retinal environment [26,28].

Preliminary studies have reported associations between obstructive sleep apnea (OSA), which causes intermittent hypoxia, and increased AMD risk. The gut-retina axis hypothesis also implicates circadian disruption through its effects on gut microbiome composition, systemic inflammation, and immune dysregulation [28]. While the evidence is still emerging, optimizing sleep hygiene and addressing sleep disorders aligns with the broader lifestyle medicine framework and may confer additional retinal protective benefits.

4.6 Gut Microbiome and the Gut-Retina Axis

A novel and rapidly evolving area of research concerns the gut-retina axis, whereby the composition and function of the gut microbiome influences systemic inflammation, immune regulation, and retinal health. Dysbiosis, characterized by reduced microbial diversity and increased abundance of pro-inflammatory bacteria, has been associated with enhanced complement activation and elevated systemic inflammatory markers implicated in AMD pathogenesis [28].

Dietary patterns rich in fiber, polyphenols, and fermented foods – such as the Mediterranean diet – support a diverse and anti-inflammatory gut microbiome, potentially providing an additional mechanism through which diet protects against AMD. Although direct interventional evidence in AMD is limited, the conceptual framework linking diet, gut microbiome health, systemic inflammation, and retinal disease represents a promising avenue for future research and reinforces the value of dietary optimization as a cornerstone of AMD prevention [26,28,29].

5. Integrated Lifestyle Medicine Approach to AMD Prevention

The six pillars of lifestyle medicine – whole-food, predominantly plant-based nutrition; regular physical activity; stress management; restorative sleep; avoidance of risky substances; and positive social connections – each have relevance to AMD prevention and management. The evidence reviewed demonstrates that AMD is not solely a genetic destiny but a condition significantly influenced by modifiable behaviors [15,22].

A practical, integrated approach for clinicians includes: (1) recommending the AREDS2 formula for patients with intermediate AMD or advanced AMD in one eye; (2) encouraging a Mediterranean-style dietary pattern rich in dark leafy greens, colorful fruits and vegetables, fatty fish, nuts, and olive oil; (3) implementing tobacco cessation interventions using evidence-based counseling and pharmacotherapy; (4) prescribing regular moderate-intensity physical activity (150 minutes per week); (5) promoting weight management through dietary and activity modifications; (6) advising UV protection through sunglasses and hats; and (7) optimizing sleep quality and duration [15,22,26].

Importantly, these lifestyle interventions provide benefits beyond ocular health, simultaneously reducing the risk of cardiovascular disease, diabetes, cognitive decline, and other chronic conditions that frequently co-occur with AMD in aging populations.

This holistic approach positions ophthalmology within the broader context of preventive medicine and person-centered care.

6. Limitations and Future Directions

Several limitations of the existing evidence base should be acknowledged. The majority of nutritional epidemiology studies are observational, limiting causal inference. Dietary assessment methods (food frequency questionnaires) are subject to recall bias. The heterogeneity of AMD classification systems across studies complicates cross-study comparisons. Additionally, gene-environment interactions remain incompletely characterized; individuals with specific complement factor H (CFH) or ARMS2 polymorphisms may respond differently to nutritional interventions [18].

Future research priorities include well-designed randomized controlled trials of comprehensive lifestyle interventions in AMD, investigation of the gut-retina axis as a therapeutic target, precision nutrition approaches guided by genetic profiles, exploration of circadian rhythm optimization for retinal health, and development of validated lifestyle assessment tools for integration into ophthalmic practice. The identification of interactions among diet, gut microbiota, supplementation, physical activity, genotype, BMI, and sleep patterns represents a new frontier in personalized AMD prevention and management [26].

7. Conclusion

This narrative review demonstrates that nutritional and lifestyle factors play a substantial and clinically meaningful role in the prevention and management of age-related macular degeneration. Strong evidence supports the benefits of AREDS2 supplementation for at-risk individuals, Mediterranean diet adherence for AMD prevention, smoking cessation as the highest-priority modifiable intervention, and regular physical activity and weight management as complementary protective measures. Emerging evidence further implicates sleep quality, UV protection, and gut microbiome health as relevant factors within a holistic prevention framework.

A lifestyle medicine approach offers an evidence-based, patient-centered paradigm for AMD risk reduction that simultaneously addresses the multimorbidity frequently encountered in aging populations. Ophthalmologists and primary care physicians alike should incorporate assessment and counseling on modifiable lifestyle factors as a standard component of care for patients at risk of or living with AMD. By embracing this integrative approach, clinicians can empower patients to actively participate in preserving their vision and overall health.

Conflict of Interest Statement

The author declares no conflicts of interest.

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