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PROGNOSTIC FACTORS FOR PROGRESSION OF AGE-RELATED MACULAR DEGENERATION: A REVIEW ARTICLE

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ABSTRACT

Age-related macular degeneration is a leading cause of irreversible vision loss in older adults. Most of the resulting disability arises when early or intermediate disease progresses to late stages; geographic atrophy or neovascular disease. Identifying who is likely to progress is therefore a clinical priority. This narrative review summarizes published evidence on prognostic factors for progression, drawing mainly on recent systematic reviews and meta-analyses, landmark cohort studies, and randomised supplementation trials. Retinal phenotype is the strongest and most consistent predictor: large drusen burden, many small drusen, pigmentary change, and optical coherence tomography features such as hyperreflective foci and subretinal drusenoid deposits. Older age and risk variants at the CFH and ARMS2/HTRA1 loci add risk, although once early disease is present the phenotype appears more informative than genotype. Smoking is the best-supported modifiable factor, with a markedly stronger association with late disease, and the antioxidant-based AREDS formulation lowers progression risk in people with intermediate disease. Evidence for alcohol, lipids, body mass index, hypertension and diabetes is inconsistent. Important gaps remain, including social determinants, ethnic diversity, gene-environment interaction, and validated imaging-based or artificial-intelligence risk models. Many pooled estimates also combine disease onset with progression, which limits interpretation. In practice, retinal phenotype should be combined with counselling on modifiable risk when estimating individual prognosis.

KEYWORDS: Age-related macular degeneration; Prognostic factors; Disease progression; Drusen; Smoking; Genetics.

INTRODUCTION

Age-related macular degeneration (AMD) is a degenerative disease of the macula and a leading cause of severe, irreversible vision loss in older people. Global estimates put the number of affected individuals at around 200 million, with a projected rise to about 288 million by 2040.[1,2] Comprehensive clinical reviews describe its epidemiology, pathogenesis, and management.[3,4,5] Clinically, the disease is divided into early AMD (small drusen or pigment epithelium mottling), intermediate AMD (large drusen or pigmentary depigmentation or hyperpigmentation), and late AMD, which takes the form of geographic atrophy or neovascular disease.[6] Vision is lost mainly when disease reaches the late stages, so slowing or anticipating that transition is the central clinical aim.

The risk of progression varies enormously between individuals. The five-year probability of moving from early or intermediate to late disease has been reported to range from 0.4% to 53%, depending on disease severity.[7] Traditionally, drusen size, pigmentary abnormalities, and the status of the fellow eye have been the main clinical predictors, but multimodal imaging is now adding new biomarkers.[7] Understanding which characteristics predict progression allows clinicians to stratify follow-up, counsel patients on modifiable risks, and enrich clinical trials with people likely to progress.

Several recent syntheses have addressed this question. An overview of systematic reviews identified 17 relevant reviews and grouped the reported prognostic factors into 20 types across eight categories.[8] A meta-analysis of 18 cohort studies pooled risk estimates for demographic, lifestyle, metabolic, and retinal factors.[9] A systematic

review of prognostic factors for progression from intermediate to late AMD concluded that study heterogeneity precluded meta-analysis.[10] Earlier syntheses focused largely on incident rather than progressive disease.[9,11] The aim of the present narrative review is to bring these sources together with landmark cohort and trial evidence, to organize prognostic factors by type and modifiability, and to highlight the limitations and gaps that most affect interpretation. It is intended to complement, not replace, the systematic overview of Holt et al.

LITERATURE SOURCES AND APPROACH

This is a narrative review. Sources were identified through targeted searches in October 2026 for systematic reviews, population-based cohort studies, and randomised trials on prognostic factors for AMD progression, supplemented by examining the reference lists of recent reviews. Priority was given to recent systematic reviews and meta-analyses, large population-based cohorts, and randomised trials. No formal systematic search, dual screening, or risk-of-bias appraisal was performed, and effect estimates are quoted as reported by the original authors. Statements about the strength of evidence are therefore qualitative.

RESULTS AND DISCUSSION

Overview of the evidence base

The overview by Holt et al. included 17 systematic reviews and meta-analyses, most of which studied functional or structural ocular factors (10 of 17), lifestyle factors (7 of 17), or intervention-related factors (7 of 17). Across the reviews, 218 prognostic factors were reported; 79 were extracted and grouped into 20 types.[8] Their main conclusions were that the modifiable factors with the most evidence were antioxidant and multivitamin supplementation and reduced smoking, that most factors were non-modifiable, and that no review had examined social determinants.[8] In contrast, the systematic review by the Pinnacle study group could not pool its data because of heterogeneity; the most commonly reported factors were baseline drusen volume or size, linked to neovascular AMD, and outer retinal thinning, linked to geographic atrophy.[10] The meta-analysis by Tian and Zhang included 18 cohort studies with 38,697 participants followed for 2 to 15 years.[9] Its main pooled estimates are shown in Table 1, and the categories of evidence are summarized in Table 2.

Table 1: Pooled risk estimates from a meta-analysis of 18 cohort studies.[9]

Factor	Pooled OR (95% CI)	Early AMD, OR (95% CI)	Late AMD, OR (95% CI)
Age, per 5-year increment	1.14 (1.09–1.19)	1.11 (1.06–1.16)	1.18 (1.14–1.23)
Male vs female sex	0.84 (0.72–0.98)	0.96 (0.73–1.25)	0.81 (0.54–1.23)
Current smoking	1.28 (1.07–1.52)	1.16 (0.98–1.38)	2.61 (1.66–4.10)
Alcohol intake	1.30 (1.00–1.67)	1.38 (1.03–1.85)	1.13 (0.49–2.60)
HDL cholesterol, per 1 mmol/L	1.21 (1.08–1.36)	1.14 (1.04–1.26)	1.95 (1.16–3.28)†
LDL cholesterol, per 1 mmol/L	0.98 (0.84–1.13)	0.95 (0.85–1.07)	2.14 (1.21–3.78)†
Hypertension	1.03 (0.98–1.09)	0.99 (0.91–1.08)	1.03 (0.99–1.07)
Diabetes mellitus	1.04 (0.97–1.13)	0.98 (0.82–1.16)	1.23 (0.66–2.30)
Body mass index, per kg/m²	1.01 (0.98–1.04)	0.98 (0.92–1.05)	1.06 (0.97–1.17)
Total drusen >10% of grid	7.85 (4.66–13.23)	–	–
>10 small drusen	7.21 (2.10–24.72)	–	–
Retinal depigmentation	6.39 (2.48–16.44)	–	–
Retinal hyperpigmentation	6.03 (1.94–18.73)	–	–

OR, odds ratio; CI, confidence interval; HDL, high-density lipoprotein; LDL, low-density lipoprotein. †Based on a single study. Estimates for retinal features are for any AMD and were not stratified by stage. Reproduced as reported by the original authors.

Cohort evidence on progression

The population-based cohorts that underpin much of this evidence differ in size, region, and follow-up. In the meta-analysis of Tian and Zhang, twelve cohorts were from Europe, North America, Oceania, and Africa and six were from Asia.[9] The figures below are as listed in that meta-analysis. The Rotterdam Study followed 4,953 participants aged 55 years or older for 2 years,[12] the Beaver Dam Eye Study followed 2,119 participants for 15 years,[13] and the Blue Mountains Eye Study followed 2,474 participants aged 49 years or older for 15 years.[14] In Asia, the Handan Eye Study in rural China followed 5,048 participants for 6 years,[15] the Singapore Malay Eye Study followed 1,809 participants,[16] and the Singapore Indian Eye Study followed 2,105 participants, each for 6 years.[17] The Nakuru Eye Disease Cohort Study in Kenya followed 1,282 participants aged 50 years or older for 6 years and is one of few sources of African data,[18] and three jointly designed German cohorts with follow-up of up to 18 years have also contributed.[19] These studies used different AMD grading systems and adjusted for different sets of confounders, which is one reason pooled estimates should be interpreted cautiously.[9]

Retinal phenotype and imaging biomarkers

Retinal findings are the strongest predictors identified. In the pooled cohort analysis, total drusen covering more than 10% of the grid, more than 10 small drusen, depigmentation, and hyperpigmentation were each

associated with odds ratios between about 6 and 8.[9] These estimates came from few studies and have wide confidence intervals, so they indicate direction and rough magnitude rather than precise risk.[9] Small drusen have been examined as an early feature in the Beaver Dam Eye Study,[20] and structure-function work relates drusen and hyperreflective foci volumes to local retinal sensitivity in non-exudative disease.[21]

Optical coherence tomography has extended this picture. The main imaging biomarkers related to progression to advanced AMD include drusen volume, hyperreflective foci, reticular pseudodrusen or subretinal drusenoid deposits, incomplete retinal pigment epithelium and outer retinal atrophy, hypertransmission defects, and reflective drusen substructures.[7] Eyes with higher baseline drusen volume are at greater risk of progression to either neovascular AMD or geographic atrophy.[7] The Pinnacle review similarly found baseline drusen volume or size and outer retinal thinning to be the most frequently reported factors.[10] A practical limitation is that drusen measurements depend on the imaging platform and segmentation method, which complicates comparison between studies.[7]

Age and sex

Age is the most consistent demographic factor. In the pooled analysis, each 5-year increase in age was associated with higher odds of AMD, and the association was stronger for late disease (odds ratio 1.18) than for early disease (1.11).[9] Male sex appeared protective (odds ratio 0.84), but this finding was unstable in sensitivity analyses, was affected by publication bias, and lost significance after trim-and-fill adjustment, so it should be interpreted cautiously.[9] Hormonal differences have been proposed as one explanation for sex differences in age-related eye disease.[22]

Genetic factors

Variants in *CFH* and *ARMS2* are the best-characterized genetic risk factors. In the 20-year Beaver Dam Eye Study, participants were classified as low, intermediate, or high genetic risk on the basis of risk alleles at both loci. Among people aged 45 years without AMD, the estimated proportion developing late AMD by age 80 was 1.4%, 5.2%, and 15.3% in the three groups, respectively.[23] Importantly, the authors concluded that when early AMD is already present, knowing the phenotype contributes more to risk assessment than knowing the genotype at these two genes.[23] A later study of 502 individuals, adjusted for age and baseline severity, found that the time to late-stage disease was shortest in carriers of two risk alleles at each of the *ARMS2*/*HTRA1* and *CFH-CFHR5* loci, followed by those with two risk alleles at *ARMS2*/*HTRA1* alone, compared with *CFH-CFHR5* alone; this suggests the two loci act through distinct mechanisms.[24] Genetic factors were outside the scope of the cohort meta-analysis because most of the evidence comes from cross-sectional or case-control designs.[9]

Smoking

Smoking is the best-supported modifiable factor. Current smoking was associated with higher odds of AMD overall (odds ratio 1.28), and the association was much stronger for late disease (2.61) than for early disease (1.16, not statistically significant), with a significant difference between the stages.[9] This pattern suggests that smoking may act chiefly by accelerating progression toward late disease. Oxidative stress is the most commonly proposed mechanism,[25] and oxidative stress and inflammation contribute more broadly to degeneration of the retinal pigment epithelium.[26] In the Study of Osteoporotic Fractures, a cohort of 1,958 women aged 65 years or older followed for 5 years, smoking and alcohol use were examined in relation to AMD in the oldest old.[27] The overview of systematic reviews likewise named reduced smoking among the modifiable factors with the most evidence for slowing progression.[8]

Nutrition and supplementation

The original Age-Related Eye Disease Study (AREDS) reported that a formulation of antioxidant vitamins and zinc reduced progression to advanced AMD by about 25% in high-risk individuals.[28] At 10 years, participants in AREDS categories 3 and 4 who had been assigned to the formulation had lower odds of advanced AMD than those assigned to placebo (odds ratio 0.66, 95% CI 0.53-0.83), and the benefit persisted for neovascular AMD but not for geographic atrophy.[29] The AREDS2 trial then tested whether adding lutein and zeaxanthin, omega-3 fatty acids, or both could improve this formulation. It enrolled 4,203 participants aged 50 to 85 years with bilateral large drusen or with advanced AMD in one eye. Five-year progression to advanced AMD was 31% with placebo, 29% with lutein and zeaxanthin, 31% with omega-3 fatty acids, and 30% with both, and none of the comparisons reached statistical significance in the primary analyses.[30] Removing beta-carotene or lowering the zinc dose also had no apparent effect on progression.[30] Supplementation is an intervention rather than a risk factor in the strict sense, but it has been counted among prognostic factors in the overview of systematic reviews.[8] Evidence on dietary patterns remains limited, because much of it derives from case-control designs rather than prospective cohorts with standardized dietary assessment.[9]

Alcohol, lipids, and cardiometabolic factors

The evidence for these factors is inconsistent. Alcohol intake was modestly associated with AMD overall (odds ratio 1.30), but the result was borderline and changed in sensitivity analysis.[9] Higher HDL cholesterol was paradoxically associated with higher risk (odds ratio 1.21 per mmol/L), and the authors suggested that dysfunctional or oxidized HDL might be responsible; this remains a hypothesis. The association of LDL cholesterol with late AMD rested on a single study.[9] Total cholesterol, triglycerides, body mass index, hypertension, diabetes, cardiovascular disease, stroke,

and C-reactive protein showed no significant pooled associations.[9] Differences in how these exposures were measured and adjusted for across cohorts may partly explain the variation, and these findings need replication. Lipid and cardiovascular phenotypes have long been studied in relation to AMD,[31] and Mendelian randomization, metabolomic, and complement-focused studies are now probing whether lipid pathways play a causal role.[32,33,34]

Evidence gaps

Several gaps limit current prognostic models. First, the overview of systematic reviews found no review that examined social determinants, and it called for future work on social, systemic, and artificial-intelligence-identified biomarkers.[8] Second, most cohort data come from predominantly Caucasian populations, leaving risk profiles in Asian, African, and Hispanic populations less well defined, and few cohorts have studied gene-environment interactions or validated emerging biomarkers such as inflammatory cytokines.[9] Third, AMD definitions and grading systems differ between studies, and the adjustment factors used vary widely.[9,10] Finally, the eligibility criteria of the cohort meta-analysis required participants to be free of AMD at baseline, so its pooled estimates blend factors for disease onset with factors for progression of established disease.[9] Prognostic research restricted to people who already have intermediate disease is needed to answer the progression question directly.

Table 2: Summary of prognostic factors for AMD progression by category.

Category	Factors	Summary of evidence
Retinal phenotype	Large drusen burden, many small drusen, pigmentary change	Strongest associations in pooled cohort data, but based on few studies with wide confidence intervals [9]
Imaging biomarkers	Drusen volume, hyperreflective foci, subretinal drusenoid deposits, incomplete retinal pigment epithelium and outer retinal atrophy	Repeatedly linked to progression in imaging studies; heterogeneity has prevented pooling [7,10]
Demographic	Age; sex	Age is consistent and stronger for late disease; the sex association is unstable [9]
Genetic	*CFH*, *ARMS2*/*HTRA1* risk alleles	Raise the risk of late disease; phenotype more informative once early disease is present [23,24]
Lifestyle	Smoking; alcohol	Smoking is the best-supported modifiable factor; alcohol is inconsistent [8,9]
Nutritional supplementation	AREDS formulation; lutein, zeaxanthin, omega-3 fatty acids	AREDS formulation reduced progression in high-risk eyes; AREDS2 additions gave no further reduction in primary analyses [28,30]
Cardiometabolic	Body mass index, hypertension, diabetes, total cholesterol, triglycerides, HDL, LDL	Mostly no pooled association; HDL and LDL findings rest on few studies [9]
Social determinants	Socioeconomic status, education	Not examined in the systematic reviews of the overview [8]; sparse cohort data show no significant pooled association [9]

AMD, age-related macular degeneration; AREDS, Age-Related Eye Disease Study; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Clinical implications

Taken together, the evidence supports an approach that places retinal phenotype at the center of risk assessment: drusen burden, pigmentary change, imaging biomarkers, and the status of the fellow eye, interpreted alongside age. Smoking cessation is the clearest modifiable target, and AREDS-type supplementation has trial evidence in people with intermediate disease or advanced disease in one eye, and the investigators of the long-term follow-up consider that these patients should consider taking the formulation.[29] Genetic testing adds limited information once the phenotype is known.[23] Future models that combine phenotype, genotype, and systemic and social variables, ideally validated across ethnic groups, may allow individualized follow-up intervals.

Strengths and limitations of this review

This review brings together recent systematic evidence with landmark cohort and trial data and organizes it by factor type and modifiability. Its limitations are that it is narrative rather than systematic, that it relies partly on secondary summaries in systematic reviews that may overlap in the primary studies they include, and that it did not appraise the risk of bias of the sources. Conclusions should therefore be read as a structured synthesis of published findings, not a new quantitative estimate.

CONCLUSION

Retinal phenotype, particularly drusen burden, pigmentary change, and optical coherence tomography biomarkers, is the strongest predictor of progression of AMD, with age and genetic risk adding further

information. Smoking is the most clearly modifiable factor, and the AREDS formulation reduces progression in high-risk eyes. Evidence for alcohol, lipids, and cardiometabolic factors is inconsistent, and social determinants remain unstudied. Progress will depend on studies restricted to people with established early or intermediate disease, greater ethnic diversity, and validated multivariable prognostic models.

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