Eye and Brain

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REVIEW

A Narrative Review of the Association Between Cataracts and Dementia

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Abstract: Dementia and cataract are two of the most prevalent conditions in older adults, together representing a substantial global health burden. Increasing evidence suggests a potential link between cataract and dementia, and this narrative review synthesizes current epidemiological and mechanistic evidence on their association. Recent cohort and case-control studies report a modestly increased risk of dementia in individuals with cataracts, though inconsistencies persist across populations. Mechanistic insights highlight roles for visual impairment and protein aggregation in this association. Importantly, cataract surgery shows a robust, protective effect against incident dementia, potentially via restoration of sensory input and enhanced cognitive engagement. Future studies may examine longitudinal, multi-ethnic cohorts that integrate genetic, imaging, and molecular data to investigate causality and the underlying biological mechanisms. In summary, our narrative review shows that cataract and dementia may be linked through multifactorial pathways, and maintaining visual health, particularly through timely cataract surgery, represents a potentially modifiable factor in dementia prevention strategies.

Keywords: cataract, dementia, cataract surgery

Introduction

Dementia is a major public health challenge in older adults, affecting an estimated 57 million people worldwide, with Alzheimer's disease (AD) being the most common cause.[1,2] Cataract, the leading cause of reversible visual impairment worldwide, imposes a substantial health burden on aging populations. In 2020, cataracts were responsible for approximately 15 million cases of blindness and 79 million cases of moderate to severe visual impairment worldwide among adults aged 50 and older.[3] Emerging evidence increasingly suggests a potential link between cataracts and the risk of developing AD and other dementias.[4,5] Studies not only explored the epidemiological association between cataracts and dementia but also examined the impact of cataract surgery on dementia risk and the pathways that may connect these conditions.[4,6] Understanding these relationships could provide critical insights for developing prevention strategies and interventions aimed at reducing cognitive decline and improving the quality of life among older adults. This review aims to synthesize current evidence on the association between cataracts and dementia and discuss potential underlying mechanisms.

Evidence Suggesting Potential Links Between Cataracts and Increased Dementia Risk

Several epidemiological studies indicate that cataracts are associated with a modestly increased risk of dementia. Notably, an analysis of the Taiwan National Health Insurance Program reported an adjusted hazard ratio (HR) of 1.43 (95% CI: 1.13–1.82) for AD in individuals with cataracts compared to those without cataracts, after accounting for confounders including sex, age, diabetes mellitus, head injury, and hypertension.[7] In another large Taiwanese cohort, senile cataracts were linked to increased odds of dementia after adjustment for demographic factors (OR = 1.67, 95% CI: 1.01–1.89).[8]

Received: 22 August 2025
Accepted: 6 December 2025
Published: 10 December 2025

Studies in Western populations echo these findings. Among 12,364 UK Biobank participants aged 55–73 years, the multivariable-adjusted HR for incident dementia in those with cataracts at baseline was 1.11 (95% CI: 1.00–1.24).[9] The Cardiovascular Health Study of 3,375 participants also found cataracts were significantly associated with increased risk in AD (HR = 1.34, 95% CI: 1.01–1.80) and vascular or mixed dementia (HR = 1.41, 95% CI: 1.02–1.95).[10] Moreover, a prospective analysis of over 300,000 UK Biobank participants reported that compared to health controls, nonsurgical cataracts were associated with a higher risk of all-cause dementia (HR = 1.21, 95% CI: 1.01–1.46) and AD (HR = 1.48, 95% CI: 1.11–1.98).[5] Finally, Medicare claims analyses on 126,650 participants detected a slightly increased risk of AD associated with cataracts diagnosis (HR = 1.06, 95% CI: 1.02–1.09).[11] Indeed, a recent meta-analysis of nine cohort or case-control studies with total of 448,140 participants reported increased risk of all-cause dementia (risk ratio [RR] = 1.24, 95% CI, 1.14–1.35), Alzheimer's disease (RR = 1.22, 95% CI, 1.10–1.35) and vascular dementia (RR = 1.29, 95% CI, 1.01–1.66).[4]

However, not all epidemiological studies reported significant associations between cataracts and dementia. The Shanghai Aging Study followed 1,659 non-demented residents aged 60 years and older for a mean of 5.2 years and found no significant association between cataracts and incident dementia (HR = 1.23, 95% CI: 0.85–1.79) or AD (HR = 1.14, 95% CI: 0.73–1.77).[12] Similarly, the Adult Changes in Thought study in the US, which included 3,877 participants and over 31,000 person-years of follow-up, observed no increased risk of AD associated with cataracts.[13] In addition, large cohort studies conducted in Hong Kong of 15,576 community-living older adults without baseline dementia found no significant association (HR = 0.95, 95% CI: 0.85–1.07).[14] These mixed results may reflect differences in study design, cohort characteristics, study duration, or the method for determining dementia. For instance, the positive associations observed in larger cohorts with longer follow-up times, such as cohorts in the UK Biobank and the Cardiovascular Health Study, may have increased statistical power and better ability to capture incident dementia cases over time. In contrast, smaller sample sizes and shorter follow-up durations, such as those seen in the Shanghai Aging Study, may limit the ability to detect modest relationships. Dementia ascertainment methods also varied. Some studies, such as the Shanghai Aging Study and the cohort study in Hong Kong, utilized detailed clinical evaluations and neuropsychological testing to more accurately diagnose dementia and AD, whereas larger cohort studies, such as those utilizing the UK Biobank, relied on administrative data, including hospital inpatient records, that may have introduced misclassification bias and over-estimated dementia or AD rates. Taken together, much, but not all, of the epidemiological evidence suggests that cataracts may modestly increase the risk of dementia and AD, while inconsistencies across studies highlight the need for further longitudinal research to clarify these associations.

In addition to the epidemiological studies, other studies have suggested a potential genetic link between cataracts and dementia.[6,15] Ferguson et al used an observational and Mendelian randomization (MR) approach in the UK Biobank data and found that cataracts were associated with dementia risk, and higher genetic risk for cataracts was associated with increased risk of vascular dementia (VaD); analysis using 2-sample MR estimators showed cataracts increased VaD risk 2-fold.[15] The study also found that the genetic risk for AD was not associated with cataracts, suggesting that the observational associations are not due to reverse causation.[15] Moreover, the study found that cataracts were associated with increased brain atrophy, and given that cataracts were found to be associated with VaD and white matter hyperintensity volumes (a marker of small-vessel ischemic disease),[16] they suggested vascular mechanisms linking cataracts and dementia, although further studies are required. In contrast, Man et al conducted a bidirectional two-sample study and found no genetic evidence on association between cataracts and the risk of AD (inverse variance weighted [IVW] OR =1.04, 95% CI: 0.98–1.10).[6] As such, further studies are required to elucidate the underlying genetic evidence on whether cataracts are associated with dementia risk through a genetic mechanism.

Mechanistic Insights

There are several possible mechanisms underlying the observed increased risk of dementia and AD in cataract patients, including visual acuity deficits and protein aggregation.

Visual Acuity Deficits as a Modifiable Factor for Dementia

Cataracts may lead to visual impairment, which has been associated with an increased risk of dementia, as evidenced by large-scale epidemiological studies and systematic reviews.[17,18] A meta-analysis by Kuzma et al found that among

patients with visual impairment, there was an increased risk of all-cause dementia (37,705 total participants; 3,415 total dementia cases; RR = 1.38; 95% CI: 1.19–1.59).[17] Similarly, a meta-analysis by Shang et al of 14 cohort studies with 6,204,827 participants and 171,888 dementia patients reported a pooled RR of 1.47 (95% CI: 1.36–1.60) for dementia associated with vision impairment.[18]

Visual impairment may mediate the relationship between cataracts and dementia through psychosocial and physiological mechanisms. Individuals with vision loss may experience reduced participation in social and physical activities, both recognized as risk factors for cognitive impairment and dementia.[19,20] Social engagement is considered a key contributor to maintaining cognitive function, as people with vision impairment may be reluctant to go outdoors due to fear of falling, leading them to avoid social gatherings.[21] This decline in social and physical activities may, in turn, be linked to diminished cognitive abilities.[21]

Additionally, the *sensory deprivation hypothesis* suggests that a persistent lack of adequate sensory input may lead to neuronal atrophy, ultimately contributing to cognitive decline.[22,23] The *cognitive compensation hypothesis* further proposes that cognitive resources may be diverted to compensate for visual deficits, thereby monopolizing these resources and contributing to cognitive decline.[24] These mechanisms may impair cognitive performance, reduce cognitive reserve, and increase executive dysfunction.[25]

Amyloid and Protein Aggregation in Cataracts Lenses

Protein insolubility and aggregation are fundamental pathogenic factors underlying a broad class of protein condensation diseases, including many neurodegenerative disorders such as AD.[26,27] Cataracts are increasingly recognized as a protein condensation disease of the eye lens, where clear crystallin proteins aggregate into insoluble, opaque deposits that scatter light and impair vision.[28] This protein condensation frequently involves the nucleation and growth of amyloid fibrils, a toxic aggregated state implicated in both cataracts and neurodegenerative diseases.[26]

Direct evidence for overlapping molecular pathology between cataracts and Alzheimer's disease comes from studies on postmortem lenses and brains.[29,30] Goldstein et al analyzed eyes and brains from individuals with AD and controls.[29] They found that amyloid-β (Aβ), the hallmark peptide in AD neuropathology, is present in the cytosol of lens fiber cells from AD patients. This finding suggests that lens Aβ may promote localized protein aggregation and extracellular amyloid formation in the lens, potentially contributing to the development of region-specific lens opacities known as supranuclear cataracts. These results raise the intriguing possibility that lens amyloid pathology might parallel or reflect cerebral amyloid deposition.

Further mechanistic insights have been gained from proteomic studies examining crystallin aggregates in normal aged versus cataractous lenses. Wang et al demonstrated that protein truncation, deamidation, and isomerization facilitate the binding of crystallin aggregates to lens cell membranes.[30] Notably, cataracts lenses exhibit distinct modifications to γ-crystallins—key structural proteins in the lens—possibly due to enhanced protein degradation, further destabilizing their native folding and fostering aggregation.[30] Complementing these findings, spectroscopic analysis by Alperstein and colleagues using two-dimensional infrared (2DIR) spectroscopy identified amyloid β-sheet secondary structure within cataractous lens tissue, a feature absent in juvenile lenses.[31] These chemical modifications, such as deamidation of specific amino acid residues, destabilize crystallins, increasing their tendency to misfold and self-assemble into amyloid-like fibrils.[32]

Despite shared features of protein aggregation, there are biochemical and pathological distinctions between cataracts and AD. For example, cataract-associated amyloid primarily consists of crystallin proteins localized within the lens, whereas AD pathology involves extracellular Aβ peptides accumulating in the brain.[33] Still, the human eye lens also contains Aβ, and a study by Schimansky and Yadav has shown interaction between Aβ and crystallin proteins, suggesting a possible interaction between AD and cataracts pathologies.[33] As such, there may be convergent molecular mechanisms of protein misfolding and amyloid fibril formation, suggesting potential overlapping principles linking cataractogenesis with neurodegenerative proteinopathies.[34] Importantly, AD-associated Aβ neuropathology begins decades before cognitive symptoms appear and evolves slowly, with emerging evidence of amyloid pathology in tissues beyond the brain.[34,35] The presence of lens Aβ offers a potentially optically accessible biomarker for early AD detection and longitudinal disease monitoring, bridging ocular pathology and systemic neurodegeneration.[36]

Treating Visual Impairment to Prevent Dementia or Reverse Changes

The therapeutic implication of the above-reviewed evidence for the association between cataracts and dementia is to treat cataracts and visual loss to avoid these adverse outcomes. Epidemiological evidence supports that cataracts surgery is strongly associated with a significant reduction in the risk of all-cause dementia and AD. A recent meta-analysis reported that cataract surgery reduces the risk of AD by approximately 25–30%, with a pooled RR of 0.74 (95% CI, 0.67–0.81).[4] This protective effect has been confirmed across diverse populations and large cohort studies.[5,37,38] For example, a large population-based cohort study from Taiwan, utilizing data from the National Health Insurance Research Database, followed 491,226 subjects aged 70 years and older. This study found that individuals who underwent cataract surgery had a significantly lower incidence of dementia compared with those diagnosed with cataracts but not undergoing surgery, with an HR of 0.77 (95% CI, 0.75–0.79).[37] Similarly, analysis from the Adult Changes in Thought study, a longitudinal cohort of cognitively normal participants, demonstrated that cataract extraction was associated with a 29% reduction in dementia risk (HR, 0.71; 95% CI, 0.62–0.83) after adjusting for education, race, smoking history, and stratifying by apolipoprotein E genotype, sex, and age at diagnosis.[38] Supporting these findings, prospective data from over 300,000 UK Biobank participants revealed that cataract surgery was linked to a substantially decreased risk of all-cause dementia (HR, 0.63; 95% CI, 0.42–0.95) and AD (HR, 0.40; 95% CI, 0.20–0.81) when compared to individuals with cataract who did not undergo cataract surgery.[5] Taken together, these studies underscore a consistent and clinically meaningful association between cataract surgery and lower dementia risk.

The mechanisms underlying this protective effect are not fully elucidated but are thought to involve restoration of sensory input, which may enhance cognitive engagement, reduce social isolation, and improve quality of life. Furthermore, improved retinal light exposure following cataract removal may benefit circadian rhythm regulation and brain health.[39] The strong and persistent reduction in dementia risk following surgery emphasizes the potential of cataract treatment as an important modifiable factor in dementia prevention strategies for aging populations.

Conclusion

The evidence reviewed suggests that there may be a significant association between cataracts and dementia. While the majority of studies indicate a modestly increased risk of dementia among individuals with cataracts, findings are not entirely consistent across various studies, likely due to their differences in study design, cohort characteristics, follow-up length, or the method for determining dementia. Insights into potential mechanisms, such as visual deprivation, genetic predisposition, and protein aggregation, support a plausible connection. Notably, cataract surgery was shown to be associated with a reduced risk of dementia, suggesting that maintaining good vision health may be an important and potentially modifiable factor in reducing cognitive decline. Nevertheless, important gaps remain in our understanding of causality, underlying biological mechanisms, and the variability of associations across populations. Future research may investigate their associations in large, longitudinal, and ethnically diverse cohorts. Mechanistic studies that integrate genetic, molecular, and neuroimaging data will be essential for disentangling potential causal pathways. Exploration of lens biomarkers, such as amyloid deposition, may also open new avenues for early detection of neurodegenerative disease. Given the global burden of cataract and dementia, fostering collaboration between ophthalmology, neurology, geriatrics, and public health will be critical to developing effective, accessible interventions for aging populations.

Institutional Review Board

IRB approval was not required for this manuscript as the investigation only involved publicly available data.

Funding

NIH Vision Core grant P30-EY01583–26 and Research to Prevent Blindness (New York, New York).

Disclosure

The authors have no conflict of interest in any part of the submitted work.

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