Visual Impairment, Eye Conditions, and Diagnoses of Neurodegeneration and Dementia

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Abstract

IMPORTANCE Vision and eye conditions are associated with increased risk for Alzheimer disease and related dementias (ADRDs), but the nature of the association and the underlying biological pathways remain unclear. If causal, vision would be an important modifiable risk factor with viable population-level interventions.

OBJECTIVE To evaluate potentially causal associations between visual acuity, eye conditions (specifically cataracts and myopia), neuroimaging outcomes, and ADRDs.

DESIGN, SETTING, AND PARTICIPANTS A cohort and 2-sample bidirectional mendelian randomization (MR) study was conducted using UK Biobank participants and summary statistics from previously published genome-wide association studies on cataract, myopia, and AD. The participants included in the analysis were aged 55 to 70 years without dementia at baseline (calendar years 2006 to 2010), underwent genotyping, and reported on eye conditions; a subset completed visual acuity examinations (n = 69 852-71 429) or brain imaging (n = 36 591-36 855). Data were analyzed from August 15, 2022, through November 28, 2023.

EXPOSURE Self-reported cataracts, visual acuity, and myopia measured by refraction error.

MAIN OUTCOMES AND MEASURES ADRD, AD, and vascular dementia were identified from electronic medical records. Total and regional brain volumes were determined using magnetic resonance imaging.

RESULTS The sample included 304 953 participants (mean [SD] age, 62.1 [4.1] years; 163 825 women [53.72%]); 14 295 (4.69%) had cataracts and 2754 (3.86%) had worse than 20/40 vision. Cataracts (hazard ratio [HR], 1.18; 95% CI, 1.07-1.29) and myopia (HR, 1.35; 95% CI, 1.06-1.70) were associated with a higher hazard of ADRD. In MR analyses to estimate potential causal effects, cataracts were associated with increased risk of vascular dementia (inverse variance-weighted odds ratio [OR], 1.92; 95% CI, 1.26-2.92) but were not associated with increased dementia (OR, 1.21; 95% CI, 0.98-1.50). There were no associations between myopia and dementia. In MR for potential reverse causality, AD was not associated with cataracts (inverse variance-weighted OR, 0.99; 95% CI, 0.96-1.01). Genetic risk for cataracts was associated with smaller total brain (β = −597.43 mm³; 95% CI, −1 077.87 to −177.00 mm³) and gray matter (β = −375.17 mm³; 95% CI, −680.10 to −70.24 mm³) volumes, but not other brain regions.

CONCLUSIONS AND RELEVANCE In this cohort and MR study of UK Biobank participants, cataracts were associated with increased risk of dementia, especially vascular dementia, and reduced total

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Abstract (continued)

brain volumes. These findings lend further support to the hypothesis that cataract extraction may reduce the risk for dementia.

Introduction

Visual impairment and ocular disease are emerging as potential modifiable risk factors for Alzheimer disease (AD) and related dementias (ADRDs). 1 Self-reported vision impairment, 2,3 eye conditions such as cataracts, 4,5 and poor visual acuity 6-8 are associated with an increased risk of dementia. If causal, this association would be clinically meaningful; some vision impairments can be reversed or corrected. For example, observational studies have reported that cataract surgery is associated with a decreased risk of dementia. 4,9

Establishing the causality of this association is methodologically difficult. Both vision impairment and ADRDs can develop slowly over decades, they share risk factors, 10,11 and early ADRDs could affect visual processing (reverse causation). 12 Additionally, previous studies are observational and may be affected by unmeasured confounding by social or other health factors 13-15 or confounding by indication. 16 Alternative approaches are needed to inform whether vision care could delay the onset of ADRD.

Genetic variants associated with eye conditions or AD 17-19 can be leveraged to estimate causal associations, because genetic variants are likely independent of key confounders. Mendelian randomization (MR) uses genetic variants, single-nucleotide variants (SNVs), as instruments, allowing for an unbiased estimate of causal effect, provided certain assumptions are met. 20,21 Few studies have used MR methods to evaluate associations between vision impairments and dementia. One study reported null findings for the association between cataracts and AD; however, this study did not examine vision impairments or dementia more broadly. 22 Furthermore, previous studies have not investigated related indicators of ADRD, such as neuroimaging markers, which would provide convergent evidence and elucidate biological pathways.

We evaluated the possible associations between visual impairment, cataracts, and ADRD in the UK Biobank (UKB). With recent interest in visual acuity 23 and cataracts 9 as dementia risk factors, we focused our genetic analyses on cataracts and myopia. With MR, we first used genetic risk for AD to evaluate whether shared genetics or incipient AD increases the risk of later vision impairment or eye conditions (Figure 1A). Second, we used genetic risk for eye conditions (cataracts and myopia) to evaluate whether incipient eye conditions increase the risk of ADRD, vascular dementia (VaD), or AD (Figure 1B). Third, to explore biological pathways, we evaluated eye conditions and brain magnetic resonance imaging (MRI) outcomes.

Methods

Study Population

The UKB is an ongoing biobank study that enrolled more than 500 000 individuals aged 40 to 69 years from 2006 to 2010 across the UK. 24 Participants provided blood samples, survey responses, and their electronic health records are linked for follow-up through November 2021; a subset completed visual acuity tests (calendar years 2006-2010) and brain MRI (calendar years 2014-2010). This cohort study followed the Strengthening the Reporting of Observational Studies in Epidemiology using Mendelian Randomization (STROBE-MR) guidelines (eAppendix 1). 25 Ethics approval for data collection was obtained from the National Health Service National Research Ethics Service, and all participants provided written informed consent. Participants receive compensation for travel.
We included participants aged 55 years or older in this analysis (age 55-70 years at baseline). We excluded individuals with dementia at baseline or without self-reported visual history. Analyses using genetic risk score (GRS) or MR were further restricted to those with European genetic ancestry due to concerns that variants may not generalize to those with non-European ancestry.

**Eye Disorders and Visual Acuity**

Participants self-reported their history of eye problems (cataracts, glaucoma, age-related macular degeneration, and diabetic retinopathy) during their baseline visit. Participants also self-reported their history of cataract surgery. For analyses in which cataracts were the outcome, we used additional diagnoses of senile cataracts that were obtained from linked electronic health record data through the end of 2021. In a subset, the presence of myopia was determined from refraction error, measured by noncycloplegic autorefraction, and visual acuity was measured in both eyes as the logMAR chart. The measure from the better eye was used. Binary 20/40 vision was calculated using a logMAR cutoff of 0.3.

**Dementia**

Incident dementia cases, including AD, VaD, and nonspecific dementia, were ascertained based on dementia diagnoses from linked primary care and inpatient electronic health records through 2021 using *International Statistical Classification of Diseases and Related Health Problems, 10th Revision*, codes (eTable 1 in Supplement 1). In sensitivity analyses, we restricted the outcome to dementia subtypes (AD or VaD).

**Brain Volumes**

We selected 5 neuroimaging regions of interest as secondary outcomes. Based on research showing their association with cognitive decline, we included measures of total brain volume, total gray matter volume, hippocampal volume (both hemispheres), white matter hyperintensity volume, and mean cortical thickness in an AD signature region. The AD signature region is a surface area–weighted average of cortical thicknesses across 6 regions. We also included the volume of 1 visual region (ie, lateral occipital cortex) under the hypothesis that visual impairment may cause atrophy in visual tracts and regions.

![Figure 1. Hypothesized Associations Between Cataracts or Other Eye Conditions and Brain Regions, Leading to Dementia](jamanetworkopen.fig1)
Magnetic resonance imaging data were obtained from the first imaging visit and were acquired using identical scanners and the same protocol across sites. Volumetric, cortical thickness, and surface area measures were obtained using FreeSurfer. All volumetric measures were adjusted for intracranial volume.

**Genetic Instruments for Cataracts, Myopia, and AD**

Samples were genotyped in batches using 2 closely related arrays. We selected genetic instruments based on previously published genome-wide association studies (GWAS) for cataracts, myopia, and AD. We created GRS based on genome-wide significant SNVs identified in each GWAS (P < 1 × 10^{-8} unless otherwise specified). Genetic risk scores were calculated by multiplying each individual’s risk allele count for each locus by the effect estimates for that SNV from the respective GWAS and summing scores to create a weighted sum. SNPs in linkage disequilibrium were removed (r^2 > 0.3). A primary GRS for cataracts (cataracts-GRS) was calculated from 43 SNVs from a 23andMe replication GWAS (347,209 cases and 2,887,246 controls) on cataracts (eTable 2 in Supplement 1). We examined a secondary GRS for cataracts based on variants independent from those identified in the UKB to minimize any weak instrument bias. The secondary cataract-GRS was based on 7 SNVs (P < 1 × 10^{-7}) identified from participants with European ancestry from the Genetic Epidemiology Research in Adult Health and Aging (GERA) study (28,092 cases and 50,487 controls). Final GRS were transformed into a z score. The primary GRS for myopia (myopia-GRS) was limited to 55 SNVs that replicated in 23andMe and were statistically significant (P < 1 × 10^{-8}) from the 23andMe replication study (eTable 3 in Supplement 1). Secondary analyses used a myopia-GRS (106 SNVs) created from the study’s GERA subcohort. In addition, a GRS for AD (AD-GRS) was created using effect estimates from a large GWAS on dementia that did not include the UKB. We focused on genetic risk for AD since genetic risk for AD is well described. The final AD-GRS was a weighted sum of 27 SNVs, including 2 apolipoprotein E (APOE) ε4 alleles (eTable 4 in Supplement 1). We replicated our analyses using a version of the AD-GRS without the APOE alleles.

**Covariates**

Age at visit (baseline or imaging), self-reported sex (male and female), self-reported racial and ethnic identity (Asian, Black, Chinese, White, multiple races, and other), quintiles of index of multiple deprivation by country of origin (highest, above average, average, below average, and lowest), and 6 indicators for a history of relevant comorbidities (falls, broken bones, cardiovascular disease, stroke, diabetes, and problems hearing) were reported at baseline. We adjusted for racial and ethnic identity because there are racial disparities in visual impairment and dementia that we do not believe are explained by other covariates included in UKB data. Genetic models (including GRS and MR models) were adjusted for age, sex, and the first 10 principal components to adjust for confounding by population stratification. Imaging models included adjustment for imaging center. Models including visual acuity and myopia were additionally adjusted for use of corrective lenses.

**Statistical Analysis**

**Observational Analyses**

We assessed the association between eye problems (cataracts, myopia, binary 20/40 vision, glaucoma, diabetic retinopathy, and age-related macular degeneration) and all-cause dementia, AD, and VaD using Cox proportional hazards regression models censored for death, loss to follow-up, or end of study period. In a secondary model focused on cataracts, we evaluated whether a history of cataract surgery modified associations with dementia risk. We also investigated cross-sectional differences in imaging markers using linear regressions.

**Mendelian Randomization**

To better investigate possible causality, we used genetic risk to evaluate the associations between cataracts, myopia, and dementia. There are 4 main assumptions that need to be met for MR. First,
the relevance condition requires that SNVs are associated with the risk factor or exposure (eg, SNVs in the AD-GRS must be associated with AD). We confirmed that each GRS (cataracts-GRS, myopia-GRS, and AD-GRS) was associated with its corresponding exposure (eTable 5 in Supplement 1). Second, the independence assumption requires that there are no unmeasured confounders of the SNVs and outcome. Third, the exclusion restriction assumption states that the only effect of the SNVs on the outcome operates through the exposure. Fourth, we must assume monotonicity, meaning that our defined GRS does not have opposite effects on individuals within the sample.

We first modeled the association of 2 cataracts-GRS and myopia-GRS with all-cause dementia using logistic regressions. To evaluate potential reverse causation, we additionally modeled the association between AD-GRS and 3 vision outcomes (cataracts, myopia, and binary 20/40 vision) using logistic regressions. These GRS allow us to leverage individual-level outcome data to examine potential associations. Next, we used 2-sample MR estimators and conducted sensitivity analyses to evaluate MR assumptions. Using the SNVs from each GRS as instruments, we conducted 2-sample MR assessing 5 associations: from (1) cataracts to all-cause dementia, (2) cataracts to VaD, (3) AD to cataracts, (4) AD to myopia, and (5) AD to 20/40 vision. Single-nucleotide variant associations with outcomes were estimated in the UKB using the same covariate adjustments (age, sex, and principal components) as the external GWAS for exposure data, and reference alleles were harmonized. We report estimates from 4 MR approaches, MR Egger, weighted median, inverse variance weighted, and weighted mode. Additionally, we report the MR-Egger intercept and the MR pleiotropy residual sum and outlier (MR-PRESSO) global test as tests for horizontal pleiotropy. There were too few SNVs to use the secondary cataracts-GRS from GERA in 2-sample MR analyses. All analyses were conducted in R, version 1.4.17 (R Project for Statistical Computing) and used the TwoSampleMR package, version 0.5.6. Data were analyzed from August 15, 2022, through November 28, 2023. Statistical tests were performed with an α = .05 and were 2-tailed and unpaired.

**Results**

We included 308 272 participants aged 55 years or older in this analysis. We excluded individuals with dementia at baseline (n = 192 [0.06%]) or without self-reported visual history (n = 3127 [1.01%]). The full analytic sample included 304 953 participants (mean [SD] age, 62.06 [4.08] years; 163 825 women [53.72%]; 141 128 men [46.28%]). Smaller subsets of participants had available brain MRI data (ranging from 36 591 for total brain volume to 36 855 for lateral occipital volume) and/or had completed visual acuity examinations at initial assessment (69 852 for myopia and 71 429 for worse than 20/40 vision). During a mean (SD) follow-up of 12.71 (2.09) years, 7676 incident dementia cases were identified. When restricted to individuals with available data, 14 295 participants (4.69%) reported a history of cataracts, and 19 670 had myopia (28.16%), and 2754 (3.86%) had worse than 20/40 vision (Table 1). Those who reported cataracts were older at baseline (mean [SD], 63.94 [3.84] vs 61.96 [4.07] years) and more likely to be female (8505 [59.50%] vs 155320 [53.44%]) compared with those without cataracts (n = 290 658).

**Associations With Dementia Risk**

Cataracts were associated with all-cause dementia (hazard ratio [HR], 1.18; 95% CI, 1.07-1.29), AD (HR, 1.25; 95% CI, 1.07-1.47), and VaD (HR, 1.25; 95% CI, 1.01-1.55) (Table 2). When adjusted for cataract surgery (n = 2581 [0.5%]), cataracts were still associated with dementia (HR, 1.19; 95% CI, 1.08-1.32). Cataract surgery was not associated with dementia (odds ratio [OR], 0.99; 95% CI, 0.57-1.70), although 95% CIs were wide. There was no interaction between cataracts and cataract surgery (P = .72). Poor visual acuity was also associated with all-cause dementia (HR, 1.35; 95% CI, 1.06-1.70). However, myopia was not associated with dementia (HR, 0.92; 95% CI, 0.81-1.05) (Table 2). Glaucoma and age-related macular degeneration were not associated with dementia, although CIs were consistent with moderate increased risk. Diabetic retinopathy was associated with...
an increased hazard of all-cause dementia (HR, 1.63; 95% CI, 1.40-1.91) (eTable 6 in Supplement 1) and subtypes.

**Associations With Brain MRI Volumes**
Cataracts were associated with smaller total gray matter volume (β = −2483.27 mm³; 95% CI, −4225.21 to −741.34 mm³) (Figure 2; eTable 7 in Supplement 1), smaller lateral occipital volume (β = −243.47 mm³; 95% CI, −416.81 to −70.12 mm³), and greater white matter hyperintensity volume (β = 531.00 mm³; 95% CI, 79.87-982.13 mm³). Cataracts-GRS was associated with smaller total gray matter volume (β = −375.17 mm³; 95% CI, −680.10 to −70.24 mm³) and total brain volume (β = −597.43 mm³; 95% CI, −1077.87 to −117.00 mm³). There was no evidence that genetic risk for cataracts was associated with AD-related cortical thinning (eTable 7 in Supplement 1).

**Mendelian Randomization**
Genetic risk for cataracts (primary cataracts-GRS) was not associated with all-cause dementia (OR, 1.02; 95% CI, 0.99-1.05) or AD (OR, 1.00; 95% CI, 0.96-1.05), but it was associated with VaD (OR, 1.09; 95% CI, 1.03-1.16) (eTable 8 in Supplement 1). The secondary cataracts-GRS was associated with all-cause dementia (OR, 1.23; 95% CI, 1.20-1.26), AD (OR, 1.30; 95% CI, 1.25-1.35), and VaD (OR, 1.20; 95% CI, 1.14-1.26) (eTable 9 in Supplement 1). The myopia-GRS (primary and secondary) was not associated with any dementia outcome. In the reverse direction, AD-GRS was associated with lower...
odds of myopia (OR, 0.97; 95% CI, 0.95-0.99), but not with cataracts (OR, 0.99; 95% CI, 0.97-1.01) (eTable 10 in Supplement 1).

Using MR on our primary cataract-GRS, estimates for the possible causal association of cataracts with dementia were all greater than 1 (estimates ranging from 1.21 to 1.37) (Table 3), although no estimates were statistically significant. There was no evidence of horizontal pleiotropy, using the MR-Egger method ($\beta = -0.01; P = .59$). Similar estimates were noted with MR-PRESSO analysis (OR, 1.21; 95% CI, 0.98-1.50; global test for horizontal pleiotropy, $P = .37$). The association between individual cataract SNVs and all-cause dementia is shown in eFigure 1 in Supplement 1, with no evidence for large outliers. Mendelian randomization indicated greater odds of VaD with cataract (inverse variance weighted (OR, 1.92; 95% CI, 1.26-2.92, with all MR estimates ranging from 1.92 to 2.33) (Table 3), and all estimates were statistically significant except for MR-Egger (OR, 2.33; 95% CI, 0.90-6.02), which also provided no evidence of horizontal pleiotropy (intercept = −0.01; $P = .66$) (eFigure 2 in Supplement 1).

We checked for possible reverse causation and found that AD SNVs were not associated with cataracts (IVW OR, 0.99; 95% CI, 0.96-1.01), myopia, or binary 20/40 vision (eTable 11 in Supplement 1) across most summary MR estimates. There was no evidence of horizontal pleiotropy between AD and cataracts ($\beta = -0.003; P = .39$) (eFigure 3 in Supplement 1).

**Discussion**

We evaluated vision impairments and dementia risk in UKB, using observational and MR approaches. Cataracts and poor visual acuity were associated with dementia risk. Higher genetic risk for cataracts was associated with increased risk of VaD. Analysis using 2-sample MR estimators showed cataracts increased VaD risk 2-fold. Genetic risk for AD was not associated with cataracts, suggesting observational associations are not due to reverse causation. We found evidence that cataracts may be associated with brain atrophy, specifically in global gray matter. In addition, myopia was associated with a decreased risk of dementia, but we found no evidence to support causality. Together, these results are consistent with the hypothesis that cataracts increase the risk of dementia.

![Figure 2. Association Between Cataracts or Cataracts-Genetic Risk Scores (GRS) and Total Brain and Gray Matter Volumes](image-url)
Our results extend current evidence by observing that the association between cataracts and dementia may be causal and that cataracts are associated with brain volumes. Observational studies have found that cataracts were associated with increased dementia risk.4,5,46,47 Some previous work found no association between cataracts and dementia,48,49 but was underpowered. One MR study did not find a strong link between cataracts and AD22 but did not examine other dementia outcomes, such as VaD.

Our findings are strengthened by evaluating multiple ADRD-related outcomes, including neuroimaging markers. Few prior studies have examined associations between vision and ADRD-related neurodegeneration.36,37,50 In our study, cataracts were associated with increased brain atrophy, but we did not find evidence for AD-related neurodegeneration. Because a history of cataracts was associated with VaD and white matter hyperintensity volumes (a marker of small-vessel ischemic disease),51 there may be vascular mechanisms linking cataracts and dementia. Additionally, our observational work suggested an association between diabetic retinopathy and dementia. Future studies will need to confirm underlying mechanisms and should investigate other eye diseases (observationally and in MR studies), including diabetic retinopathy, optic neuropathy, and other vascular causes of eye disease.52 Future studies also should evaluate associations with

Table 3. MR Estimates for Vision Impairment and Dementia

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<tr>
<th>MR estimate</th>
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<td>MR estimate of visual impairment association with dementia</td>
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<td>Genetically predicted association between cataracts and all-cause dementia</td>
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<tr>
<td>Inverse-variance weighted</td>
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<td>MR-Egger</td>
<td>1.37 (0.84-2.23)</td>
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<td>Weighted median</td>
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<td>Genetically predicted association between cataracts and vascular dementia</td>
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<tr>
<td>IVW</td>
<td>1.92 (1.26-2.92)</td>
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<td>MR-Egger</td>
<td>2.33 (0.90-6.02)</td>
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<td>Weighted median</td>
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<td>Weighted mode</td>
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<td>MR estimate of AD association with visual impairment (reverse causality)</td>
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<td>Genetically predicted association between AD and self-reported and ICD-10 diagnoses of cataracts</td>
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<td>Genetically predicted association between AD and binary 20/40 vision</td>
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<td>Weighted mode</td>
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Abbreviations: AD, Alzheimer disease; ICD-10, International Statistical Classification of Diseases and Related Health Problems, 10th Revision; IVW, inverse variance-weighted; MR, mendelian randomization; OR, odds ratio.

* Models adjusted for age, sex, and first 10 principal components to proxy for genetic ancestry.
ADRD pathologic burden, such as amyloid tau neurodegeneration biomarkers, alpha-synucleinopathies, and cerebrovascular markers.\textsuperscript{53-55} Our results are largely consistent with previous literature showing that poor visual acuity\textsuperscript{6-8} is associated with increased dementia risk. However, visual acuity was not associated with any neuroimaging measures, in contrast to another study.\textsuperscript{36} Myopia was associated with a decreased risk of dementia in observational models. Although this is consistent with some prior work,\textsuperscript{56} the finding is likely due to confounding, as the myopia-GRS was not associated with dementia.

The validity of our MR analyses depends on 4 assumptions.\textsuperscript{20,21,43} First, we confirmed that genetic instruments estimate each exposure of interest. Second, while we cannot prove that the independence assumption is met, we have adjusted for the strongest potential confounder: principal components.\textsuperscript{57} Third, we also believe the exclusion restriction assumption holds in our MR models, as there was no evidence for horizontal pleiotropy. Fourth, the monotonicity assumption limits the generalizability of our estimates and may not represent the true effect of cataracts on dementia in different populations.

**Strengths and Limitations**

Our study has important strengths compared with prior work, including the strength of causal inference, a large sample, and an investigation into biological pathways.

This study has some important limitations. First, MR analyses are underpowered, which is reflected in the wide CIs of our MR estimates. However, by also including observational methods and multiple GRS, this study suggests there is a causal effect of cataracts on dementia by triangulation.\textsuperscript{58} Second, the genetic analyses were restricted to individuals with European ancestry, meaning genetic results may not be generalizable to other genetic backgrounds. Third, the present results do not consider monocular vs binocular presentation of cataracts, potential delays in cataract surgery by dementia status, or other factors. Fourth, the individuals in the UKB are relatively young (mean age, 62 years) and healthy,\textsuperscript{59} leading to only a smaller number of cases of ADRD. This study should be replicated in an older cohort with more diverse representation.

**Conclusions**

In this cohort and bidirectional MR study of UKB participants, we added important insights into the link between eye conditions, visual impairment, and dementia risk by combining both observational and MR analyses. We found that cataracts, but not myopia, may increase the risk of dementia, especially VaD. Mendelian randomization and imaging findings suggested that non-AD biological pathways may explain this association. This study lends further support for the hypothesis that treatment or prevention of cataracts may prevent dementia. Clinical screening and treatment of cataracts may be important clinical strategies to improve quality of life. Future studies should test whether vision screening or cataract surgery in those with co-occurring dementia risk factors are effective at reducing the risk of dementia.
Author Contributions: Ms Ferguson and Dr Brenowitz had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Concept and design: Glymour, Andrews, Yaffe, Casaletto, Brenowitz.

Acquisition, analysis, or interpretation of data: Ferguson, Thoma, Buto, Wang, Hoffmann, Choquet, Yaffe, Casaletto, Brenowitz.

Drafting of the manuscript: Ferguson.

Critical review of the manuscript for important intellectual content: All authors.

Statistical analysis: Ferguson, Thoma, Buto, Glymour, Andrews, Casaletto.

Obtained funding: Glymour, Choquet, Brenowitz.

Supervision: Glymour, Choquet, Casaletto, Brenowitz.

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Data Sharing Statement: See Supplement 2.

Additional Contributions: We thank the UK Biobank participants and staff.

REFERENCES


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eTable 11. Summary Reverse Causation MR Estimates for: 1) the Association Between AD and Self-Reported and ICD Diagnoses of Cataracts, 2) the Association Between AD and Myopia, and 3) the Association Between AD and Binary 20/40 Vision

eFigure 1. Association Between 43 Cataracts SNPs and Both Cataracts and Dementia

eFigure 2. Association Between 43 Cataracts SNPs and Both Cataracts and Vascular Dementia

eFigure 3. Association Between 27 AD SNPs and Both Cataracts and Dementia

SUPPLEMENT 2.

Data Sharing Statement