Visual acuity deficits, particularly those developed in the context of cataracts, have been associated with poorer cognitive performance both presently and in the future. These deficits have been postulated as potential modifiable risk factors for the development of dementia. However, to date, it remains unclear whether the association between cataract-induced visual deficits and the development of dementia is causal or coincidental and whether the increased risk of developing dementia is limited to a specific etiology.

In this context, the study by Ferguson et al represents a significant advancement in the field. Using an innovative approach, Ferguson et al conducted a study with a clinical cohort of 300,000 participants, including neuroimaging and genetic risk score data for more than 30,000 individuals. The study by Ferguson et al not only examines the association between cataract-related or myopia-related visual acuity deficits and the risk of dementia but also evaluates whether an increased genetic risk of developing cataracts or myopia could impact structural neuroimaging and the profile of such an imprint, if it exists.

Ferguson et al found that both an increased genetic risk of developing cataracts and a self-reported history of cataracts were associated with total brain volume, specifically total gray matter volume. This leads to greater brain atrophy in regions not specific to Alzheimer disease and an increased volume of white matter hyperintensities, which is a potential biomarker of cerebral microvascular damage. In contrast, this association was not the same in the case of myopia, as there was no clear association between an increased genetic risk of its development and the risk of dementia.

Therefore, the association of cataracts with brain structure and the risk of dementia seems to exceed the direct association induced by the visual deficit per se (expected damage to tracts and brain regions involved in visual processing). This supports a potential association of visual deficit with neurodegeneration not specific to Alzheimer disease and specific cerebral vascular damage.

In the context of global population aging and the absence of disease-modifying treatments for most causes of cognitive decline, the identification of modifiable risk factors is of vital importance. This is especially true when such risk factors are highly prevalent at the population level, as is the case with visual acuity deficits, which are estimated to affect up to 1 billion people by the year 2050, specifically impacting up to 50% of the institutionalized population with dementia. Until now, it has not been clear whether the association between cataract-induced visual deficits and dementia was coincidental due to the coexistence of common risk factors, such as age, reduced access to health care due to socioeconomic fragility, and lower educational attainment, among others. However, the study by Ferguson et al supports that the association of cataracts with the future risk of dementia goes beyond the coexistence of common risk factors. Therefore, the creation of specific population screening programs for the early detection of visual acuity deficits, particularly cataracts in the late adult population, becomes even more relevant. Early intervention for cataracts would not only improve quality of life immediately but also promote greater integration into socioeducational and occupational activities, with potential improvement of cognitive reserve. Additionally, intervention could have a direct impact on increasing brain reserve and reducing cerebral vascular damage.

There is a need to further explore whether the visual deficit associated with cataracts could constitute a risk factor not only for the development of vascular dementia, the second most frequent cause of dementia, but also specifically for Alzheimer disease. The study by Ferguson et al contradicts some
previous studies that specifically suggested that visual deficits associated with ophthalmological diseases and cardiovascular risk factors specifically increased the risk of Alzheimer disease. However, as Ferguson et al.\(^1\) rightly postulate in their work, many previous studies did not fully control for confounding factors (such as the long-term prevalence of risk factors for both conditions) or had insufficient statistical power. To evaluate the potential causal relationship between cataract-induced visual deficits and the risk of dementia, it would be advisable to have longitudinal data from large cohorts that include not only structural neuroimaging and genetic risk phenotyping but also core amyloid, tau and neurodegeneration biomarker phenotyping for Alzheimer disease.

It would also be advisable to study the association that may exist between untreated visual impairment and other sensory deficits, such as hearing loss, which has been postulated as a risk factor for worse present and future cognitive performance and a poorer quality of life with higher prevalence of affective symptomatology and social withdrawal. It is unknown whether the presence of both may have a synergistic effect on the risk of dementia,\(^5\) regardless of its cause.

Studying the associations of delayed diagnosis and treatment of sensory deficits (and the total or partial resolution of deficit symptoms) with future cognitive performance and mechanisms involved in various causes of dementia, both neurodegenerative, such as Alzheimer disease, and vascular, is also of great interest. Given the significant advancements in the treatment of visual and auditory sensory deficits, if an association is confirmed, it is even more important to address the health gap induced by socioeconomic status, as in most countries, lower income and socioeconomic status are associated with significantly delayed and reduced access to symptomatic and curative treatments for sensory deficits. These could potentially be underestimated risk factors in studies confirming a higher prevalence of dementia in populations with unfavorable socioeconomic status, even when controlling for educational level and classic cardiovascular risk factors, among others.\(^6\)

### Article Information

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### References


