

Clinical science

Statin use is associated with lower risk of open-angle glaucoma incidence and treatment escalation

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ABSTRACT

Background Retrospective cohort study using the TriNetX database to explore the relationship between statin use and glaucoma.

Methods The open-angle glaucoma (OAG) incidence analysis consisted of individuals over 40 years of age with statin use and ophthalmological follow-up within 1 year prior. The OAG treatment analysis consisted of individuals with glaucoma and concurrent statin use. Outcomes included rates of OAG onset, topical ocular hypotensive prescription, incisional glaucoma surgery, minimally invasive glaucoma surgery (MIGS) and selective laser trabeculoplasty (SLT).

Results 210 701 matched patients were included in the OAG incidence analysis and 29 598 in the OAG treatment analysis. At 5 years, statin use was associated with lower risk of OAG incidence (HR 0.85 (95% CI 0.82 to 0.89)), as well as treatment escalation to incisional glaucoma surgery (HR 0.64 (95% CI 0.58 to 0.71)), MIGS (HR 0.45 (95% CI 0.41 to 0.50)) or SLT (HR 0.40 (95% CI 0.36 to 0.44)) and lower rates of topical ocular hypotensive prescription (HR 0.89 (95% CI 0.86 to 0.91)). At 10 years, statin use was associated with lower risk of OAG incidence (HR 0.90 (95% CI 0.87 to 0.93)), escalation to incisional glaucoma surgery (HR 0.68 (95% CI 0.63 to 0.75)), MIGS (HR 0.54 (95% CI 0.49 to 0.59)) or SLT (HR 0.49 (95% CI 0.45 to 0.52)) and lower rates of topical ocular hypotensive prescription (HR 0.88 (95% CI 0.86 to 0.91)).

Conclusions Our results suggest that statin use is associated with lower risk of glaucoma onset and treatment escalation. Prospective trials are needed to further elucidate the relationship between statin usage and glaucoma.

INTRODUCTION

Glaucoma is the leading cause of irreversible blindness worldwide, characterised by permanent damage to the optic nerve, with over 111 million people estimated to be affected by 2040. Currently, intraocular pressure (IOP) is the only modifiable risk factor proven to be associated with glaucoma; several studies have shown that IOP lowering not only slows glaucoma progression but also reduces its incidence in patients with ocular hypertension.^{1–3} As a result, current treatment for glaucoma aims to lower IOP. Despite IOP lowering, however, many glaucoma patients continue to worsen.^{3–5} It has been postulated that this may be due to factors other than IOP that also contribute to glaucomatous optic nerve damage.^{6–8}

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Statins have been shown to have neuroprotective effects, raising interest in their use in neurodegenerative conditions. Glaucoma, a disease involving damage to the optic nerve, is the leading cause of irreversible blindness worldwide.

WHAT THIS STUDY ADDS

⇒ Our findings suggest that statin usage is associated with both lower incidence of primary open-angle glaucoma as well as decreased treatment escalation.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Statin usage should be considered in the long-term treatment and management of glaucoma.

The cellular mechanisms behind the characteristic loss of retinal ganglion cells and optic nerve damage of glaucoma are still poorly understood, and many systemic diseases are associated with a higher risk of developing glaucoma.^{19–11} Experimental models have suggested that several factors have a role in its pathogenesis, including but not limited to neuroinflammation, immune cell activity, biomechanical stress to axons and axonal transport, and oxidative damage.^{12–14} Whether their potentially deleterious effects in causing optic nerve damage can be ameliorated by various neuroprotective agents is an area of increasing investigation.^{15–17} In this regard, statins (3-hydroxy-3-methylglutaryl-coenzyme A reductase inhibitors), traditionally used in the management of hypercholesterolaemia, also have been receiving increasing attention for their potential neuroprotective capabilities.¹⁸

Although the neuroprotective effects of statins have been documented in some neurodegenerative diseases,¹⁹ their role in glaucoma progression and incidence is poorly understood.^{20–22} Existing population-based studies and meta-analyses have yielded inconsistent results, largely due to differences in the definition of open-angle glaucoma (OAG) severity progression, study duration and study populations.^{23–26} However, given that statins are generally well-tolerated and have few side effects, further research to delineate their role in glaucoma is warranted. In this study, we leverage a large electronic medical records database to explore the effects of statin use on glaucoma incidence and



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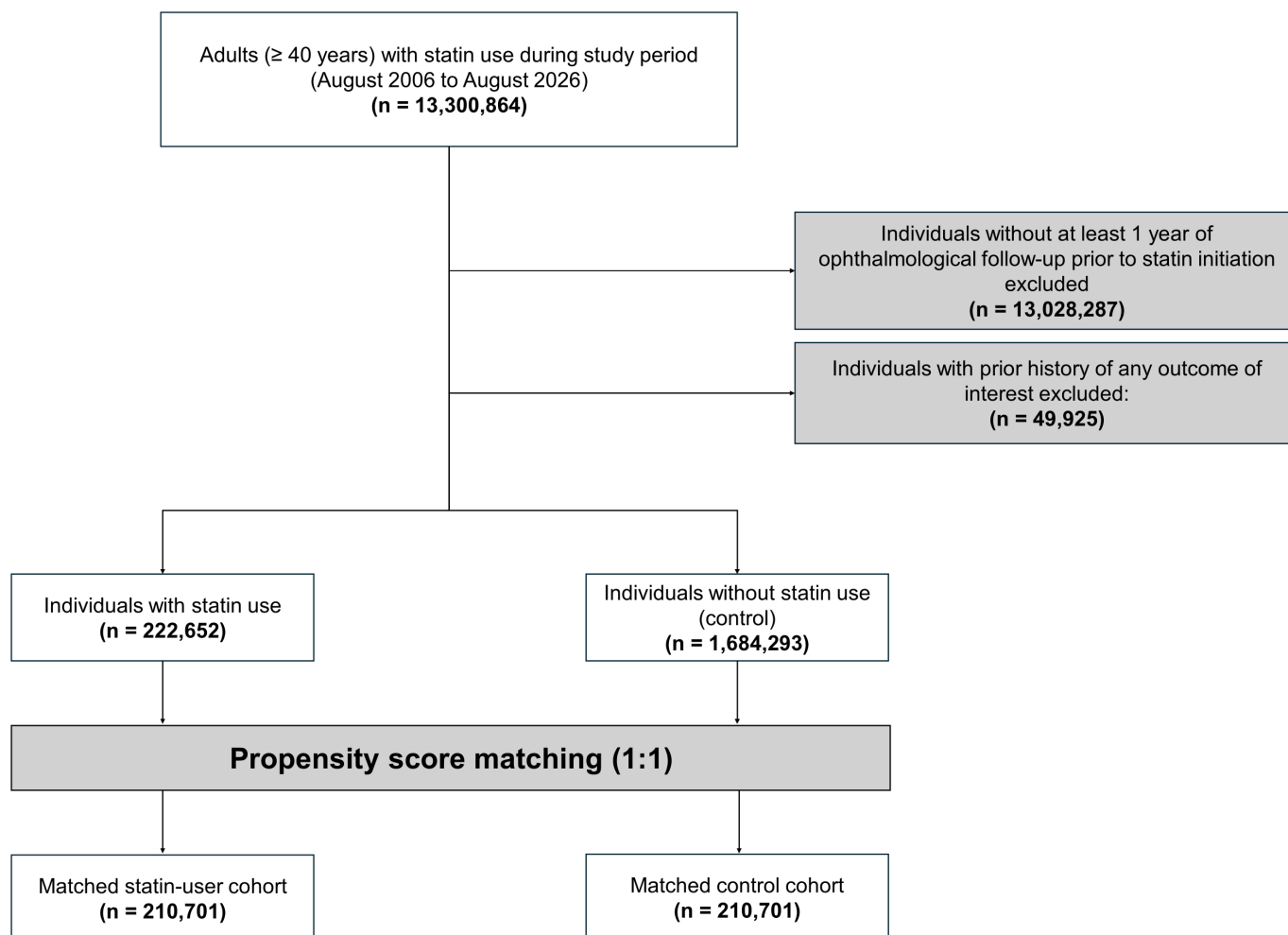


Figure 1 Study cohort flow diagram for open-angle glaucoma incidence analysis.

treatment escalation. We hypothesised that statin use is associated with decreased risk of both onset and treatment escalation of OAG.

METHODS

Data source

This retrospective study analysed data from the Research Network in the TriNetX Analytics platform, a federated health research network spanning 115 healthcare organisations and 165 million individuals.²⁷ Deidentified electronic medical records data included demographics, procedures, medications, diagnoses, laboratory results and visits. Demographic characteristics (sex, race and ethnicity) were self-reported. The study adhered to the tenets of the Declaration of Helsinki and Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines.²⁸ The study period was 8 August 2006 to 8 August 2026. Analyses were conducted in August 2026.

Study population

Cohort selection for the OAG incidence analysis is detailed in [figure 1](#). Individuals over 40 years of age with a record of statin usage were identified using the RxNorm codes 83367 (atorvastatin), 301542 (rosuvastatin), 36567 (simvastatin), 42463 (pravastatin), 6472 (lovastatin), 861634 (pitavastatin) and 41127 (fluvastatin). Individuals were required to have a history of ophthalmology follow-up within 1 year prior to their first recorded statin usage after 40 years of age. The index event

for the statin-user cohort was deemed to be the first record of statin usage after 40 years of age. The control cohort consisted of individuals over 40 years of age with a record of ophthalmology follow-up and at least 1 year of prior follow-up data within TriNetX. Individuals with any record of statin usage were excluded. The index event for the control cohort was deemed to be the first ophthalmology visit after 40 years of age. Individuals who experienced any outcomes of interest before the index date were excluded from the analysis.

Cohort selection for the OAG treatment escalation analysis is detailed in [figure 2](#). Individuals over 40 years of age with a record of statin usage were once again identified using the same RxNorm codes from the incidence analysis. Individuals were required to have a history of OAG diagnosis (*International Classification of Diseases, 10th Revision* [ICD-10] code H40.1) prior to their first recorded statin usage after 40 years of age. The index event for the statin-user cohort was deemed to be the first record of statin usage after 40 years of age and OAG diagnosis. The control cohort consisted of individuals over 40 years of age with an OAG diagnosis and no record of statin usage. The index event for the control cohort was deemed to be the first OAG diagnosis after 40 years of age. Individuals who experienced any outcomes of interest before the index date were excluded from the analysis.

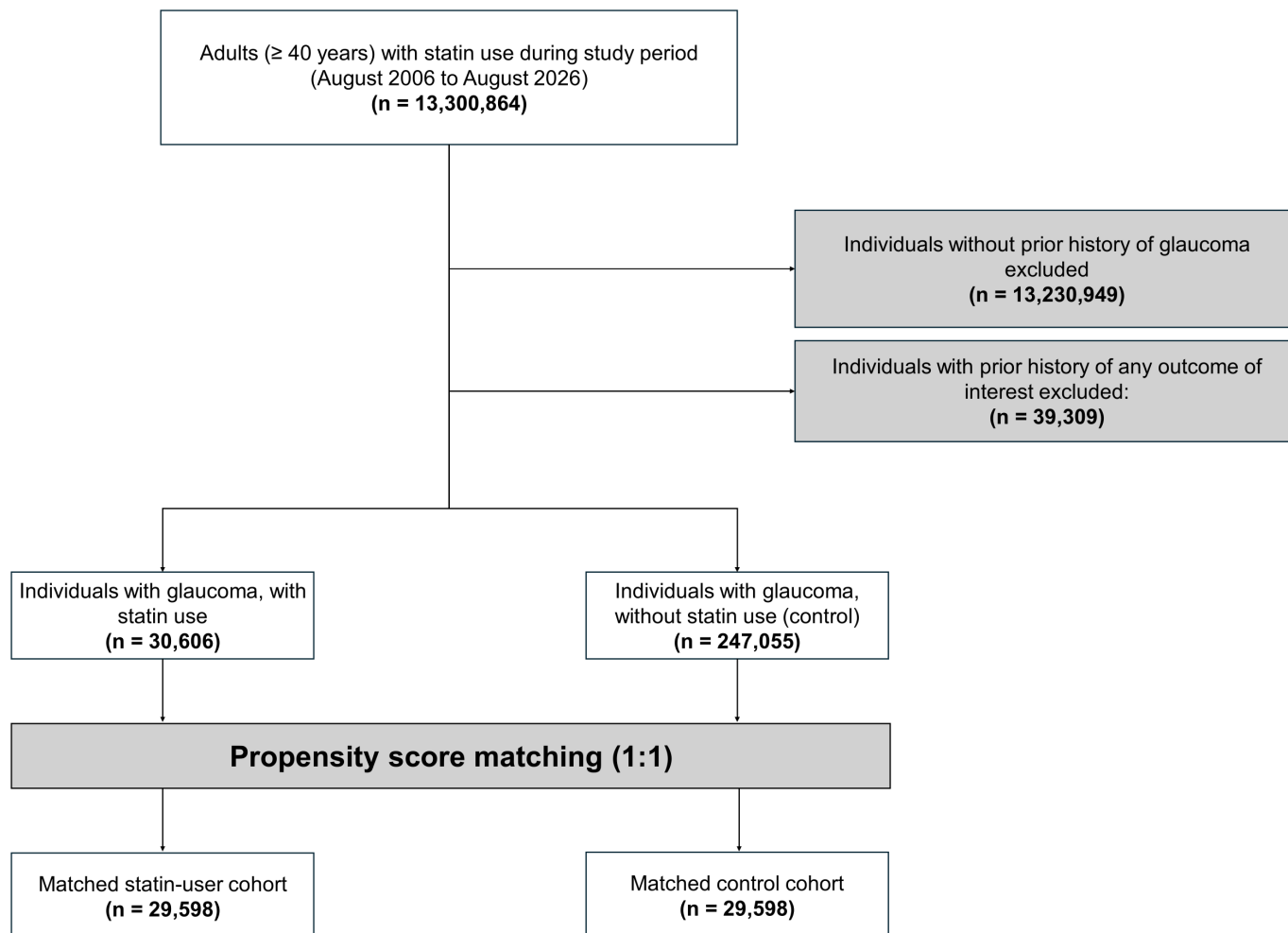


Figure 2 Study cohort flow diagram for open-angle glaucoma treatment escalation analysis.

Outcomes of interest

The primary outcome of interest for the incidence analysis was the rate of a new ICD-10 diagnosis of OAG (H40.1). The primary outcomes of interest for the treatment analysis were topical ocular hypotensive prescription, incisional glaucoma surgery, minimally invasive glaucoma surgery (MIGS) and selective laser trabeculoplasty (SLT). A full list of codes used in the analysis can be found in online supplemental digital content table 1.

Statistical analysis and covariates

Propensity score matching (1:1) was performed to match statin and non-statin user cohorts to adjust for confounders. This was done using greedy nearest-neighbour matching with a calliper distance of 0.1 pooled SD of the logit of the propensity score. Groups were considered well-matched if the standardised mean difference was <0.1 for all covariates. Matched covariates included age, sex, race, ethnicity, diabetes, chronic kidney disease, ischaemic heart disease, myopia, hypertensive disease, socioeconomic and psychosocial health hazards, hyperlipidaemia, nicotine dependence, tobacco use, thyroid disorders, diabetes with ophthalmic complications, ophthalmology services and procedures, systemic corticosteroids, body mass index, haemoglobin A1c, outpatient visits, inpatient visits and emergency department visits (online supplemental digital content table 1).

All statistical analyses were conducted using the built-in functions of the TriNetX platform. Kaplan-Meier curves were generated to estimate survival probability between cohorts, and

log-rank tests were used to estimate differences in survival. Cox proportional hazards regression models were used to calculate HRs with 95% CIs for all outcomes. Statistical significance was defined as a two-sided $p < 0.05$.

Multiple subanalyses were conducted to assess differential effects of individual statins on the incidence and treatment escalation of OAG. Pitavastatin and fluvastatin were not included in the incidence subanalysis due to insufficient cohort sizes, while lovastatin, pitavastatin and fluvastatin were not included in the treatment subanalysis.

RESULTS

OAG incidence

The statin-user cohort included 222 652 individuals (mean (SD) age 62.9 (10.6) years, 56% female, 67% White, 17% Black or African American and 11% Hispanic or Latino), while the non-statin-user cohort included 1 684 293 individuals (mean (SD) age 59.1 (11.2) years, 60% female, 67% White, 15% Black or African American and 10% Hispanic or Latino). After propensity score matching, 210 701 individuals remained in each cohort, with all covariates well balanced (online supplemental digital content table 2).

At 5 years, the statin cohort had 4771 (2.3%) new OAG diagnoses (96.87% survival) and the non-statin cohort had 5491 (2.6%) new OAG diagnoses (96.46% survival) ($p < 0.001$). Statin use was associated with a lower risk of OAG onset (HR 0.85

Table 1 Rates of open-angle glaucoma incidence and treatment escalation in statin users versus controls at 5-year follow-up

Analysis	Outcome	Statin, n (%)	Control, n (%)	HR (95% CI)
Incidence (n=210 701)	Open-angle glaucoma	4771 (2.3)	5491 (2.6)	0.85 (0.82 to 0.89)
Treatment (n=29 598)	Topical ocular hypotensive prescription	11 671 (39.4)	13 191 (44.6)	0.89 (0.86 to 0.91)
	Incisional glaucoma surgery	633 (2.1)	994 (3.4)	0.64 (0.58 to 0.71)
	MIGS	512 (1.7)	1121 (3.8)	0.45 (0.41 to 0.50)
	SLT	665 (2.2)	1656 (5.6)	0.40 (0.36 to 0.44)

MIGS, minimally invasive glaucoma surgery; SLT, selective laser trabeculoplasty.

(95% CI 0.82 to 0.89)) (table 1). The statin cohort had an average follow-up time of 1257 (SD=652) days and the control cohort had an average follow-up time of 1255 (SD=653) days. At 10 years, the statin cohort had 6238 (3.0%) new OAG diagnoses (94.46% survival) and the non-statin cohort had 6794 (3.2%) new OAG diagnoses (94.22% survival) ($p<0.001$) (figure 3). Statin use was associated with a lower risk of OAG onset (HR 0.90 (95% CI 0.87 to 0.93)) (table 2). The statin cohort had an average follow-up time of 1784 (SD=1241) days and the control cohort had an average follow-up time of 1770 (SD=1231) days.

OAG treatment escalation

The statin-user cohort included 30 606 individuals (mean (SD) age 69.6 (9.3) years, 53% female, 55% White, 27% Black or African American and 6% Hispanic or Latino), while the non-statin-user cohort included 247 055 individuals (mean (SD) age 65.8 (10.7) years, 54% female, 49% White, 22% Black or African American and 5% Hispanic or Latino). After propensity score matching, 29 598 individuals remained in each cohort, with all covariates well balanced (online supplemental digital content table 3).

At 5 years, statin use was associated with a lower risk of incisional glaucoma surgery (HR 0.64 (95% CI 0.58 to 0.71)), MIGS (HR 0.45 (95% CI 0.41 to 0.50)), SLT (HR 0.40 (95% CI 0.36 to 0.44)) and topical ocular hypotensive prescription (HR 0.89 (95% CI 0.86 to 0.91)) (table 1). The statin cohort had an average follow-up time of 1170 (SD=675) days and the control cohort had an average follow-up time of 1175 (SD=703) days. At 10 years, statin use continued to be associated with a lower risk of incisional glaucoma surgery (HR 0.68 (95% CI 0.63 to 0.75)), MIGS (HR 0.54 (95% CI 0.49 to 0.59)), SLT (HR 0.49 (95% CI 0.45 to 0.52)) and topical ocular hypotensive prescription (HR 0.88 (95% CI 0.86 to 0.91)) (table 2). The statin cohort had an average follow-up time of 1601 (SD=1213) days and the control cohort had an average follow-up time of 1645 (SD=1261) days.

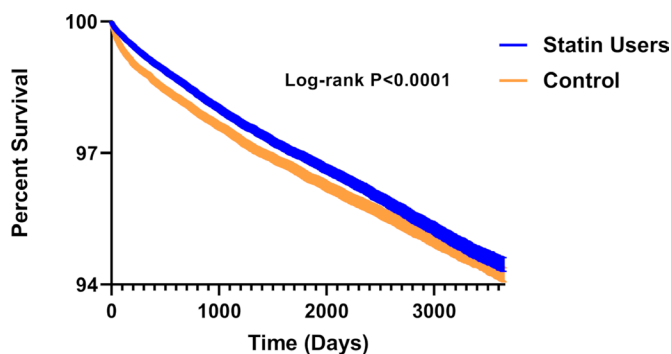


Figure 3 Kaplan-Meier survival analysis of open-angle glaucoma-free survival among patients receiving statins versus no statins in a 10-year follow-up.

Effects of individual statins

When comparing atorvastatin-only users to those without a history of statin use, atorvastatin use was associated with lower risks of OAG incidence (HR 0.83 (95% CI 0.78 to 0.88)), incisional glaucoma surgery (HR 0.67 (95% CI 0.58 to 0.78)), MIGS (HR 0.57 (95% CI 0.49 to 0.65)), SLT (HR 0.44 (95% CI 0.38 to 0.50)) and topical ocular hypotensive prescription (HR 0.88 (95% CI 0.85 to 0.92)) (online supplemental digital content tables 4, 5). Results were consistent in the rosuvastatin-only group. In simvastatin-only (HR 1.04 (95% CI 0.91 to 1.17)) and pravastatin-only (HR 0.97 (95% CI 0.84 to 1.11)) users, the risks of developing incident OAG did not differ significantly between exposure and control groups (online supplemental digital content tables 4, 5). The risks of needing incisional glaucoma surgery or topical ocular hypotensives also did not differ significantly; however, the risks of undergoing MIGS or SLT were significantly lower in the simvastatin-only and pravastatin-only groups when compared with controls.

DISCUSSION

This large retrospective cohort study identified significant associations between statin use and lower risks of both OAG onset and treatment escalation. Specifically, at 5 years, statin use was associated with a 15% reduced risk of OAG incidence and a 36% reduced risk of incisional glaucoma surgery, 55% reduced risk of MIGS, 60% reduced risk of SLT and 11% reduced risk of topical ocular hypotensive prescription in patients with existing glaucoma. Similarly, at 10 years, statin use was associated with a 10% reduced risk of OAG incidence and a 32% reduced risk of incisional glaucoma surgery, 46% reduced risk of MIGS, 51% reduced risk of SLT and 12% reduced risk of topical ocular hypotensive prescription in those with existing glaucoma. Subanalyses assessing individual statins showed consistent results in those who only used atorvastatin and those who only used rosuvastatin. Results varied in those who only used simvastatin, pravastatin or lovastatin; this may have been due to the significantly smaller cohort sizes.

Our findings are consistent with the results of Marcus *et al* (HR 0.46 (95% CI 0.23 to 0.94)) who conducted a prospective cohort study and McGwin *et al* (OR 0.60 (95% CI 0.39 to 0.92)) who conducted a case-control study with a clinical database.^{29 30} Marcus *et al* also investigated the protective effects of statins while controlling for IOP levels, citing a trial by Nagaoka *et al* in which statin use was found to be associated with lowered IOP.³¹ In their analysis, Marcus *et al* found no effect of statins on IOP. Several other studies have also found significant protective associations between statin use and glaucoma, with effect sizes similar to the ones seen in our analyses. Stein *et al* reported that patients with hyperlipidaemia and on statins experienced a slightly decreased risk of developing OAG (HR 0.92 (95% CI 0.87 to 0.98)).²³ Talwar *et al* found a dosage-independent protective effect of statins against OAG incidence (HR 0.79 (95% CI 0.66

Table 2 Rates of open-angle glaucoma incidence and treatment escalation in statin users versus controls at 10-year follow-up

Analysis	Outcome	Statin, n (%)	Control, n (%)	HR (95% CI)
Incidence (n=210 701)	Open-angle glaucoma	6238 (3.0)	6794 (3.2)	0.90 (0.87 to 0.93)
		Statin, n (%)	Control, n (%)	HR (95% CI)
Treatment (n=29 598)	Topical ocular hypotensive prescription	12 565 (43.5)	14 110 (47.7)	0.88 (0.86 to 0.91)
	Incisional glaucoma surgery	804 (2.7)	1179 (4.0)	0.68 (0.63 to 0.75)
	MIGS	721 (2.4)	1323 (4.5)	0.54 (0.49 to 0.59)
	SLT	949 (3.2)	1931 (6.5)	0.49 (0.45 to 0.52)

MIGS, minimally invasive glaucoma surgery; SLT, selective laser trabeculoplasty.

to 0.96)).³² A recent literature review and meta-analysis by Yuan *et al* also reported a slight protective effect against OAG incidence (RR 0.95 (95% CI 0.93 to 0.98)) but found inconsistent results between statin use and OAG progression.²⁶ With regard to disease progression, De Castro *et al* found that statin use was associated with slower progression of optic nerve damage.³³ Other studies have also found a beneficial effect of statins on retinal blood supply and visual field deterioration.^{20 31 34 35} However, there is also evidence suggesting that long-term statin use could be associated with elevated IOP and increased glaucoma risk.^{24 25 36 37} Many of these studies suggest that duration of use and dosage is positively correlated with glaucoma risk, which is somewhat reflected in the slightly decreasing protective effects we found at 10 years compared with 5 years.

The inconsistency of the results found in the existing literature is likely due to differences in study design, study populations, medication dosage and duration of study periods. Most importantly, studies have adopted varying definitions of the outcomes of interest; for example, OAG progression has been defined by markers such as retinal blood flow and visual field changes.^{20 34 35 38} Large deidentified databases without access to granular ophthalmological data pose several difficulties when attempting to study disease progression. While it is not a true proxy for disease progression, we chose to measure rates of treatment escalation. This instead provides an alternative perspective into assessing how statin therapy can affect long-term management of glaucoma. Our study found significantly reduced risks of undergoing MIGS, SLT and incisional glaucoma surgery in statin users as well as reduced rates of topical ocular hypotensive prescription, suggesting its potential role in modulating glaucoma disease severity. The strengths of this study also include large sample sizes and extended study periods of 5 and 10 years, providing further insight into the time-dependent effects of statin usage on glaucoma.

The possible mechanisms behind the association between statin use and glaucoma have been discussed for some time. Statins can selectively bind and inhibit the $\beta 2$ integrin leucocyte function antigen-1, a surface antigen expressed on all leukocytes that is crucial to T-cell activation and leucocyte tissue infiltration.³⁹ They can also promote differentiation of T-cells towards the anti-inflammatory $T_H 2$ phenotype while downregulating the pro-inflammatory $T_H 1$ phenotype and MHC (major histocompatibility complex) class I and II expression.^{40 41} The role of the immune system in glaucoma is not fully understood, given that the eye is an immune-privileged tissue, but the presence of auto-antibodies to optic nerve head glycosaminoglycans and increased levels of certain CD8⁺ T-cells and IL-2 in glaucoma patients suggest that it may play an important role.⁴² Statins can also activate the neuroprotective protein kinase B/Akt in endothelial cells, leading to phosphorylation and activation of endothelial nitric oxide synthase.⁴³ This increase in nitric oxide (NO) levels

and subsequent vasodilation can improve retinal blood flow and prevent ischaemic optic nerve damage.³¹ At the same time, statins can downregulate the activities of neuronal nitric oxide synthase and inducible nitric oxide synthase, preventing over-production of NO and oxidative damage.⁴⁴ In addition, statins also downregulate the production of endothelin-1, further improving blood flow.⁴⁵ Finally, a study on porcine eyes found that atorvastatin-perfused eyes had increased aqueous humour outflow, likely due to a blocking of the Rho/ROCK pathway and relaxation of trabecular meshwork cells.⁴⁶

Several important limitations of this study should be noted. The TriNetX database does not report certain ophthalmological exam data such as IOP, visual acuity, visual field tests or optic nerve imaging studies. We were therefore unable to assess OAG progression and severity at a more granular, patient-level scale. Additionally, TriNetX does not provide details on each surgery, rendering us unable to determine whether surgeries were truly performed due to increased disease severity. It is entirely possible that these procedures were done in other contexts such as medication non-adherence or intolerance. Consequently, using the rate of glaucoma treatment escalation as an indicator for disease progression only provides a rough estimate of overall cohort glaucoma severity. We were also unable to perform analyses that controlled for IOP levels, a significant limitation given the evidence in the literature of statins having an IOP-modulating effect. While we did use propensity score matching to reduce bias from confounding variables, the matching is limited to observable variables that can be encoded in the electronic health records. It is possible that residual confounding from variables such as medication adherence and statin dosages was unaccounted for and affected results, further emphasised by the lengthy study period. We were also unable to account for the usage of topical ocular hypotensives during the study period, which reduce glaucoma progression. Additionally, the database's reliance on ICD-10 codes and *Current Procedural Terminology* codes inherently exposes the study to risks of diagnostic misclassification, diagnostic underreporting or overreporting, and misclassification of study outcomes. Finally, due to the observational nature of this study, causality cannot be inferred.

Nonetheless, these findings add to the growing literature surrounding the use of neuroprotective agents that complement ocular hypotensive medications or can be used independently of them to treat and prevent glaucoma. Prospective randomised trials are needed to better understand the effects of statin use on glaucoma incidence and treatment.

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