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ORIGINAL RESEARCH

Smoking and Risk of Vision Threatening Complications: A Global Database Analysis

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Purpose: To investigate the association between smoking and the incidence of vision-threatening conditions, including cataract, glaucoma, diabetic retinopathy (DR), age-related macular degeneration (AMD), retinal vascular occlusion, uveitis, and ischemic optic neuropathy (ION) in a large real-world database to provide quantitative risk estimates to inform smoking cessation counseling.

Methods: A retrospective cohort study was conducted using the TriNetX electronic health records platform, analyzing 12,183,254 patients. Patients were categorized into smokers (n = 304,823) and non-smokers (n = 11,878,431), and then a propensity score matching was utilized to balance both groups for demographic features and vascular risk factors. The incidence rates of the studied ocular conditions were then compared over a ten-year period using risk ratios (RR).

Results: Matched cohorts included 300,867 patients per group. Smokers exhibited a significantly higher 10-year risk for all studied ophthalmic outcomes ($p < 0.0001$). The strongest associations were observed for posterior subcapsular cataract (RR 2.60; 95% CI, 2.41–2.82), uveitis (RR 2.43 [2.25–2.63]), and retinal vascular occlusions (CRAO: RR 2.35; CRVO: RR 2.16). Increased risks were also consistent across glaucoma subtypes (RR 1.57–2.47), AMD (RR 1.85), and diabetic retinopathy (RR 1.21).

Conclusion: Smoking is significantly associated with an increased risk of multiple vision-threatening ocular diseases. These findings highlight the need for ophthalmologists and public health professionals to incorporate smoking history into risk stratification and screening programs and to emphasize ocular health during smoking cessation counseling.

Keywords: smoking, counseling, vision loss, vision impairment

Introduction

Smoking exposes the body to over 4000 toxic compounds that induce oxidative stress and promote chronic inflammation in tissues, including ocular tissues, leading to degradation of their integrity. Smoking has been linked to increased retinal ischemia, microvascular dysfunction, and direct toxic effects to the optic nerve. The chemicals released by tobacco smoke inhalation may also damage mitochondrial DNA in the retinal cells contributing to the increased likelihood of their degeneration.[1,2]

Smoking has been strongly linked to the development of various ocular pathologies, including cataract, age-related macular degeneration (AMD), and thyroid eye disease. Other less-established associations include diabetic retinopathy (DR), ischemic optic neuropathy (ION), and retinal vascular occlusions.[2,3] Fear of blindness has been found to be as strong a motivation for quitting among smokers, equivalent to the fear of stroke, lung cancer, and cardiac disorders.[4] Nevertheless, most ophthalmologists do not ask patients about smoking status nor discuss motivation for smoking cessation, and there is a general lack of support at eye care centers for patients willing to quit.[5]

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To provide proper counseling, ophthalmologists should be aware of the magnitude of increased risk for various eye disorders associated with smoking. Conflicting associations could be found in the literature, possibly due to different small sample sizes and population characteristics such as ethnic variation or confounding comorbidities.[3] This study aims to explore the link between smoking and vision-threatening ocular conditions by analyzing the risk magnitude for the major ocular conditions, namely: cataract, glaucoma, AMD, DR uveitis, retinal vascular occlusion, and ION.

Methods

This retrospective multicentric cohort study was conducted using the TriNetX Global Federated Research Network, a large-scale real-world data platform aggregating de-identified electronic medical records (EMRs) from over 200 health-care organizations across the United States and internationally. At the time of data extraction, the US Collaborative Network requested information from 68 HCOs, all of which responded with clinical data.

Ethical approval for this study was acquired from the University of California Riverside Office of Research Integrity (protocol approval number #30442). The research was classified as non-human subjects research with exemption from informed consent (anonymized retrospective database analysis with no intervention nor interaction with patients). The TriNetX network adheres to HIPAA regulations and is ISO 27001:2013 certified.

Patients who had at least one encounter with an ophthalmologist were categorized into two cohorts. Cohort A (non-smokers) consisted of 11,878,431 individuals with no recorded diagnosis, procedure, or laboratory result indicative of tobacco use, as verified through documentation (international classification of diseases [ICD]-10 codes) of general adult medical exams and absence of tobacco-related terms in their EMRs. Cohort B (Smokers) included 304,823 individuals with documented evidence of smoking or tobacco use based on clinical diagnoses (eg, nicotine dependence), or procedures (eg, smoking cessation counseling). Index events were defined as the earliest date on which patients met the inclusion criteria for each cohort. Patients whose index event occurred more than 20 years prior to the analysis were excluded to ensure temporal relevance, although no patients were excluded for this reason in the final cohort.

A 1:1 propensity score matching for age, sex, hypertension, and diabetes mellitus was utilized to create balanced cohorts (Table 1). The primary objective was to evaluate the incidence (new cases) of key vision-threatening ocular diseases over a 10-year observational period (3653 days) following the index event using the respective ICD-10 codes, including cataract (all types), glaucoma (ocular hypertension, open angle glaucoma [OAG], and primary angle closure glaucoma [PACG]), AMD (nonexudative and exudative), DR (non-proliferative [NPDR] and proliferative [PDR]), retinal vein occlusion (RVO, central [CRVO] and branch [BRVO]), retinal artery occlusion (RAO, central [CRAO] and branch [BRAO]), uveitis, and ION.

TriNetX's built-in analytics suite was used for statistical analysis. Risk ratios (RR) and odds ratios (OR) were computed for each outcome. These values were visualized using bar charts comparing disease incidence across cohorts. Confidence intervals (95% CI) and p-values were reported, and significance was determined at a two-sided threshold of $p < 0.001$ for all major outcomes.

Table 1 Propensity Score Matching to Balance Both Cohorts

Variable	Before Matching			After Matching		
	Smokers n = 304,823	Non-Smokers n = 11,878,431	p-value	Smokers n = 300,867	Non-Smokers n = 300,867	p-value
Age at Index (mean ± SD)	50.5 ± 23	44 ± 19.9	<0.0001	50.5 ± 23	50.5 ± 23	0.9932
Males **Females**	50.8% 49.2%	41% 59%	<0.0001	50.8% 49.2%	50.8% 49.2%	0.9979
Hypertension	49.2%	18%	<0.0001	49.2%	49.2%	0.9959
Diabetes Mellitus	31.8%	6.8%	<0.0001	31.8%	31.8%	0.9956

Abbreviation: SD, standard deviation.

Results

A total of 12,183,254 patients were included for 1:1 propensity score matching on demographic features and cardiac risk factors, yielding balanced cohorts of 300,867 individuals each for smoking and non-smoking status across all analyses. The risk and odds ratio for the different outcomes are given in Table 2.

Cataract

Among patients without prior cataract, 9.5% of smokers compared to 5.4% of non-smokers developed new cataract during the 10-year follow-up (RR: 1.77, 95% CI: 1.73–1.8, $p < 0.0001$). The highest risk was observed for the posterior subcapsular type (RR: 2.6), followed by nuclear cataract (RR: 1.91) and cortical cataract (RR: 1.75).

Table 2 Risk and Odds Ratio for Different Outcomes in the Smokers versus Non-Smokers Cohorts

Outcome	Smokers n = 300,867	Non-Smokers n = 300,867	RR (95% CI)	p-value	OR (95% CI)
Cataract	9.515%	5.388%	1.77 (1.73–1.8)	<0.0001	1.85 (1.81–1.89)
Nuclear	7.756%	4.054%	1.91 (1.87–1.96)	<0.0001	1.99 (1.94–2.04)
Cortical	1.434%	0.82%	1.75 (1.66–1.84)	<0.0001	1.76 (1.67–1.85)
PSC	0.753%	0.289%	2.6 (2.41–2.82)	<0.0001	2.62 (2.42–2.83)
Glaucoma	4.301%	2.743%	1.57 (1.52–1.61)	<0.0001	1.59 (1.55–1.64)
Ocular HTN	1.114%	0.51%	2.19 (2.06–2.32)	<0.0001	2.2 (2.07–2.34)
OAG	1.368%	0.869%	1.58 (1.5–1.65)	<0.0001	1.58 (1.51–1.66)
PACG	0.269%	0.109%	2.47 (2.17–2.82)	<0.0001	2.47 (2.17–2.82)
AMD	3.786%	2.051%	1.85 (1.79–1.9)	<0.0001	1.88 (1.82–1.94)
Non-exudative	1.108%	0.57%	1.94 (1.83–2.06)	<0.0001	1.95 (1.84–2.07)
Exudative	0.497%	0.306%	1.62 (1.49–1.76)	<0.0001	1.63 (1.5–1.77)
DR	2.353%	1.944%	1.21 (1.17–1.25)	<0.0001	1.22 (1.17–1.26)
Mild NPDR	1.597%	1.117%	1.37 (1.31–1.43)	<0.0001	1.37 (1.31–1.43)
Moderate NPDR	0.648%	0.4%	1.62 (1.51–1.74)	<0.0001	1.63 (1.51–1.75)
Severe NPDR	0.276%	0.14%	1.98 (1.76–2.22)	<0.0001	1.98 (1.76–2.23)
PDR	0.598%	0.437%	1.37 (1.27–1.47)	<0.0001	1.37 (1.27–1.48)
CRVO	0.197%	0.091%	2.16 (1.87–2.5)	<0.0001	2.16 (1.88–2.5)
BRVO	0.209%	0.127%	1.64 (1.45–1.87)	<0.0001	1.65 (1.45–1.87)
CRAO	0.092%	0.039%	2.35 (1.89–2.91)	<0.0001	2.35 (1.9–2.92)
BRAO	0.101%	0.041%	2.45 (1.99–3.02)	<0.0001	2.46 (1.99–3.03)
Uveitis	0.733%	0.302%	2.43 (2.25–2.63)	<0.0001	2.44 (2.26–2.64)
ION	0.127%	0.075%	1.69 (1.43–1.99)	<0.0001	1.69 (1.43–1.99)

Abbreviations: AMD, age-related macular degeneration; BRAO, branch retinal artery occlusion; BRVO, branch retinal vein occlusion; CI, confidence interval; CRAO, central retinal artery occlusion; CRVO, central retinal vein occlusion; DR, diabetic retinopathy; HTN, hypertension; ION, ischemic optic neuropathy; NPDR, non-proliferative diabetic retinopathy; OAG, open angle glaucoma; OR, odds ratio; PACG, primary angle closure glaucoma; PDR, proliferative diabetic retinopathy; PSC, posterior subcapsular; RR, risk ratio.

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Glaucoma

Overall incidence of glaucoma was 4.3% in smokers compared to 2.7% in non-smokers. The RR was 1.57 (95% CI: 1.52–1.61, $p < 0.0001$). The risk was highest for PACG (RR: 2.47), followed by ocular hypertension (RR: 2.19) and open angle glaucoma (RR: 1.58).

Age-Related Macular Degeneration

The 10-year incidence of AMD was significantly higher among smokers (3.8%) compared to non-smokers (2.1%, RR: 1.85, 95% CI: 1.79–1.9, $p < 0.0001$). Risk was higher for developing non-exudative AMD (RR: 1.85) compared to exudative AMD (RR: 1.62).

Diabetic Retinopathy

Smokers had a significantly higher 10-year incidence of DR (2.4%) compared to non-smokers (1.9%, RR: 1.21, 95% CI: 1.17–1.25, $p < 0.0001$). Risk was significantly higher for all types of NPDR (mild [RR: 1.37], moderate [RR: 1.62], and severe [RR: 1.98]) and for PDR (RR: 1.37).

Retinal Vascular Occlusion

Smoking significantly increased the risk of all forms of retinal vascular occlusion (Figure 1). The highest risk was seen for BRAO (RR: 2.45, 95% CI: 1.99–3.02, $p < 0.0001$), followed by CRAO (RR: 2.35, 1.89–2.91, $p < 0.0001$), CRVO (RR: 2.16, 1.87–2.5, $p < 0.0001$), and BRVO (RR: 1.64, 1.45–1.87, $p < 0.0001$).

Ischemic Optic Neuropathy

Incident ION was diagnosed in 0.13% of smokers versus 0.08% of non-smokers. The RR was 1.69 (95% CI: 1.43–1.99, $p < 0.001$).

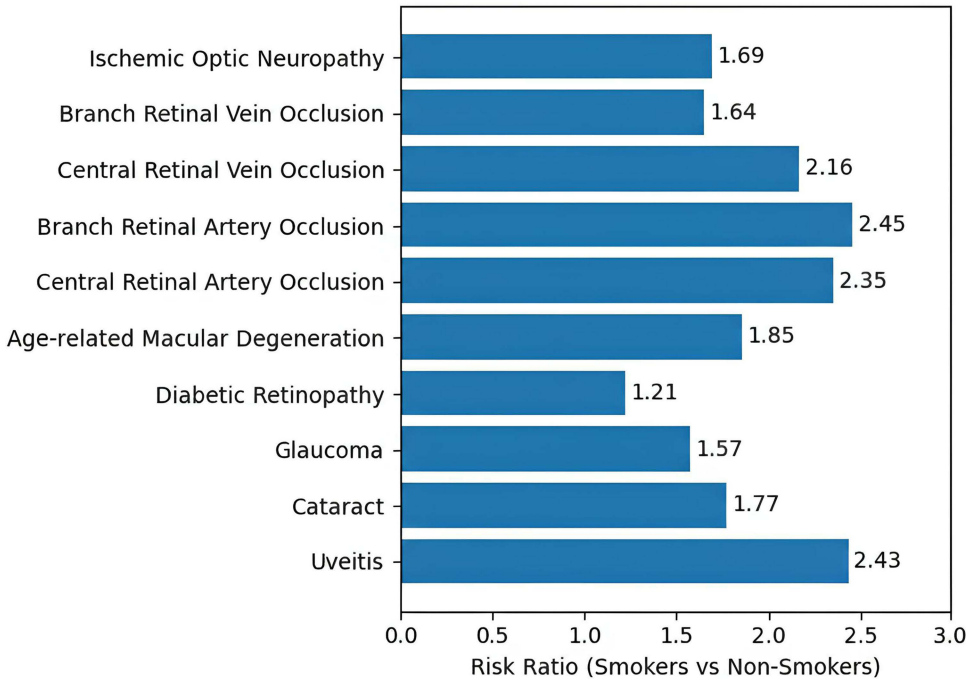

Figure 1 Diagram showing risk ratios for the main outcomes in the smokers group compared to the non-smokers group.

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Uveitis

Smokers had a significantly higher 10-year incidence rate of uveitis compared to non-smokers (0.7% vs. 0.3%, RR: 2.43, 95% CI: 2.25–2.63, $p < 0.0001$).

Discussion

In this large database analysis, we found that smoking was a significant risk factor for the development of the major vision-threatening ocular conditions over 10 years of follow-up, with more than a two-fold increased risk of posterior subcapsular cataract, PACG, ocular hypertension, CRVO, CRAO, BRAO, and uveitis and more than a 1.5-fold increased risk of nuclear and cortical cataract, AMD, OAG, and ION, and 1.2-fold the risk of developing DR. Our findings highlight the need for counseling of ophthalmology patients about smoking cessation and offer numbers that can help in such counseling.

We found an increased risk of cataract among smokers compared to non-smokers (1.8 fold). This is consistent with prior literature supporting that smoking is a significant modifiable risk factor for cataract development, where the odds ratio ranged from 1.6 for posterior subcapsular cataract to 1.8 for nuclear cataract.[1,6] In our analysis, however, the posterior subcapsular subtype had the highest risk. The pathogenesis is likely related to the accumulation of harmful ions like vanadium, aluminum, and cadmium, the increased reactive oxygen species in the lens, and reduced systemic antioxidants. These factors all contribute to opacification and lens protein degradation.[7]

We also found a significant risk of glaucoma among smokers compared to non-smokers (1.6 fold), particularly PACG (2.5 fold). Studies on association between smoking and the risk of glaucoma or ocular hypertension have yielded mixed results. While some studies found a significant positive association with primary OAG,[8,9] other studies reported no association,[10] or even a protective one.[11,12] A recent systematic review and meta-analysis explored the association between smoking and primary OAG risk and found no significant association.[13] However, most of the included studies did not achieve low bias risk, highlighting the need for further research on the topic. The association between smoking and PACG is less studied, and a lack of association between smoking and ACG severity has similarly been reported.[14] Our study, thus, provides exploratory real-world data on a possible association.

Our results indicated a significant association between smoking and both forms of AMD. This aligns with the literature showing an established association between smoking and an increased risk of both the incidence of new AMD and the progression to advanced stages (exudative AMD and geographic atrophy).[15–17] The pathogenesis is suggested to relate to an inflammatory microenvironment with reactive oxygen species, and mitochondrial DNA damage with accumulation of degradation products in the retinal pigment epithelial cells.[18]

The incidence of DR, both NPDR and PDR, was significantly higher among smokers, although the risk was lowest (1.21 fold) compared to other outcomes. Smoking has not been thoroughly investigated in association with DR, but some limited old studies seem to suggest no association.[19–21] A more recent review concluded a significant association between smoking and DR in type 1 but not type 2 diabetes mellitus.[22] The pathogenesis of vascular pathology may be related to the vasoconstrictive properties of nicotine and increased carboxyhemoglobin as a result of smoking cause a reduction in retinal blood flow which exacerbates retinal hypoxia.[23] These effects may also explain the increased risk of retinal vascular occlusion (RVO and RAO) and ION in our study among smokers compared to non-smokers, which is consistent to prior literature reports.[24–26]

We also found that smokers had a significantly higher risk of developing uveitis compared to non-smokers (2.4 fold). This is consistent with findings from a recent meta-analysis that found nearly two-fold increased odds of uveitis among smokers.[27] Taken together, this suggests that smoking is a key modifiable risk factor for uveitis development and carries implications to the counseling of patients suffering intraocular inflammation.

The strength of the study is the large sampling of real-world data that allows the exploration of the weight of each interplaying risk factor. However, the retrospective methodology of this study and the reliance on data from EMRs introduce several limitations. Calculating smoking status based on recorded diagnoses, procedures, or test data may underestimate the study population due to underreporting. Furthermore, stratified risk assessment among present versus former smokers was not possible due to the study's lack of detail regarding smoking quantity, duration, type of tobacco used, and time since cessation.

Even when major cardiovascular risk factors (hypertension and diabetes) were matched using a propensity score, other residual confounding remains a challenge. The smoker-dominated population was more likely to have obesity and dyslipidemia, and unmeasured factors such as alcohol intake, socioeconomic level, dietary intake, or access to eye care may have influenced the outcomes. Finally, given the observational nature of this study, these findings should be interpreted as associations rather than evidence of a direct causal relationship between smoking and the identified ophthalmic conditions.

In conclusion, smoking was significantly associated with an increased risk of multiple vision-threatening ocular diseases. These findings highlight the need for ophthalmologists and public health professionals to incorporate smoking history into risk stratification and screening programs and to emphasize ocular health during smoking cessation counseling.

Data Sharing Statement

The data analyzed in this study are available from the TriNetX registry. Restrictions apply to the availability of these data, which were used under license from the University of California Riverside. Data are available from the authors with the permission of TriNetX.

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Disclosure

The authors report no conflicts of interest in this work.

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