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RESEARCH ARTICLE

# Does smoking affect intraocular pressure? A cross-sectional study

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## ABSTRACT

**Background:** Tobacco smoking is linked to ocular conditions such as cataract, age-related macular degeneration, and glaucoma. Glaucoma is the most common cause of irreversible blindness and the second most common cause of blindness. Elevated intraocular pressure (IOP) is a major, modifiable risk factor for glaucoma. **Aims and Objectives:** This study was undertaken to determine the effect of tobacco smoking on IOP. **Materials and Methods**: A total of 200 apparently healthy males (100 smokers and 100 non-smokers) of 20–40 years were recruited. Smokers were categorized based on smoking index. IOP was recorded using Schiotz tonometer. Blood pressure (BP) was recorded using mercury sphygmomanometer. **Results:** Mean age of the smokers was 28.71 years and that of the non-smokers was 29.31 years. Majority of the smokers ($n$ = 79) used cigarettes and most of them ($n$ = 55) smoked 1–10 cigarettes per day. More than half the smokers were light smokers. Bidi smokers totalled 30 in number and 30% of them ($n$ = 9) smoked 21–30 bidis a day. Mean IOP in the right eye and left eye in smokers was 14.68 mmHg and 15.69 mmHg, respectively, whereas they were 14.45 mmHg and 15.11 mmHg, respectively, in the non-smokers. 5% of smokers showed elevated IOP. Mean systolic BP among smokers was 119.52 mmHg and in non-smokers; it was 118.92 mmHg. Mean diastolic BP among smokers was 79.82 mmHg, whereas in non-smokers, it was 79.48 mmHg. **Conclusions:** Tobacco smoking is not associated with raised IOP according to our study and tobacco smokers are not at a greater risk for developing elevated IOP.

**KEY WORDS:** Tobacco; Cigarette; Bidi; Intraocular Pressure; Schiotz Tonometer; Smoking Index

## INTRODUCTION

Tobacco smoking is the most common method of tobacco consumption and tobacco is the most commonly smoked substance. Tobacco is smoked in the forms of bidis, cigarettes, cigars, hookah, kreteks, pipe smoking, etc.[1] India is the world's second largest tobacco producing country and the world's third largest consumer of tobacco.[2] Of the total Indian population, of about 1.2 billion people[3] around 20.4% of males and 1.9% of females are smokers.[4]

There are approximately 4000 chemicals in cigarettes, including over 60 carcinogens.[5] Smoking leads to an increase in the incidence of smoke-related illnesses including various adverse cardiovascular events like coronary heart disease and respiratory diseases like chronic obstructive pulmonary disease.[6] Chronic cigarette use is a known risk factor for several ocular vascular pathologies such as hypertensive retinopathy, age-related macular degeneration, and anterior ischemic optic neuropathy.[7] Tobacco smoking is a widespread modifiable risk factor for many ocular disorders such as cataracts, age-related macular degeneration, and glaucoma.[8] Glaucoma comprises a group of diseases, leading to optic nerve damage and loss of vision. At present, the only well-established modifiable risk factor

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is an elevated intraocular pressure (IOP).[9] Glaucoma is the second major cause of blindness and the most common cause of irreversible blindness worldwide.[10] In India, the estimated number of glaucoma cases is 12 million, around one-fifth the global burden of glaucoma.[11] The 2001 National Blindness survey reports glaucoma as the third major cause of blindness in India, responsible for 5.9% of blindness.[12] Primary open-angle glaucoma (POAG) is the most common form of glaucoma and elevated IOP is one of its established risk factors.[13] Studies have associated tobacco smoking with IOP. As IOP is widely regarded as the most important modifiable risk factor associated with the development of glaucoma, factors that influence IOP and its measurement are of great relevance in understanding the pathogenesis of glaucoma and in reducing the burden of blindness.

Many studies have tried to assess the effects of tobacco smoking on the intraocular pressure but most of them have been conducted abroad, and there are few such studies in India. Furthermore, there is no consensus on the results of these investigations. Hence, this study was undertaken to determine the effect of tobacco smoking on IOP.

## MATERIALS AND METHODS

The present study was conducted in the Department of Physiology, Dr. B. R. Ambedkar Medical College, Bengaluru, after obtaining ethical clearance from the Institutional Ethics Committee.

Volunteers for the study were solicited from those attending the various outpatient departments of the medical college hospital. A total of 200 participants (100 smokers and 100 non-smokers) who consented to take part in the study were inducted based on inclusion and exclusion criteria. Inclusion criteria were males of 20–40 years of age. Females were not included in the study as the effects of smoking on gender are inconstant. Smokers were defined as current smokers (either cigarette or bidi) with variable smoking history and who had been smoking continuously for 1 month before recruitment. Non-smokers were those who had never smoked or consumed tobacco in any other forms. Exclusion criteria were a history of use of any alternate mode of tobacco, diabetes, hypertension and family history of hypertension, use of diuretics or beta-blockers, inflammatory eye conditions, contact lens wearers, and history of allergy to lidocaine. Detailed information including age, occupation, and medical and personal history including smoking and other habits and family history of diabetes, hypertension, refractive errors, and glaucoma were gathered and noted in a pro forma. Based on the information gathered, the subjects were included in the study. Smoking was quantified using smoking index (SI), which was calculated by multiplying the number of cigarettes or bidis smoked per day with the duration of smoking.[14,15]

Smokers were classified as light smokers, moderate smokers, and heavy smokers based on their SI as follows:
SI 1–100 - light smoker
SI 101–300 - moderate smoker
SI >301 - heavy smoker.[14,15]

### Recording the IOP

IOP was recorded using a Schiotz indentation tonometer. Before measuring the IOP, the tonometer was calibrated and sterilized. Calibration was done by placing the instrument's footplate on the artificial cornea provided with the instrument. With the footplate resting on the stand, a correctly calibrated instrument will have a scale reading of zero. If the reading was not zero, it was readjusted to zero. Following calibration, the footplate was sterilized with spirit.

Procedure was explained to the subjects and consent was obtained. With the subject in supine position, a topical anesthesia (procaine) was administered to the eyes. After 2–3 min, the eye to be measured was carefully opened using the left index finger and the thumb while ensuring no pressure was applied which could artificially increase the IOP. The subject was asked to fix his gaze at the ceiling so that the cornea was exposed with minimized movement during the measurements. Tonometer was held by the handle such that the wing lay between the index and the second fingers, respectively, and the other wing was held with the thumb. The scale reading was adjusted to face the investigator's view with the investigator standing behind the subject's head. The instrument was gently lowered for the footplate to rest squarely on the cornea. The handle was depressed downward. The instrument was held perpendicular to the eye to allow the plunger to move freely, indenting the cornea. The degree of indentation is measured by the movement of a needle on the scale. The reading on the scale which indicated the displacement of the pointer was noted. IOP was recorded first in the right eye (RE) and then in the left eye (LE). The procedure was repeated thrice consecutively on both eyes. Average of three readings was computed separately for each eye. Average scale reading and plunger weight were then converted into IOP in mmHg using Friedenwald nomogram. After each use, the tonometer plunger and footplate were rinsed with water followed by spirit and wiped dry with lint-free material. After the procedure, a prophylactic antibiotic, ciprofloxacin eye drops were instilled in both eyes to prevent infections.[16,17] To rule out any acute effects of tobacco smoking, subjects were instructed not to smoke for at least 1 h before recording of IOP.

Normal IOP in healthy young adults ranges from 10 to 21 mmHg.[18] Deviation from this range either above or below implies ocular hypertension or hypotension, respectively. Systemic arterial blood pressure (BP) was recorded using a mercury sphygmomanometer, in the right upper limb, after a rest period of 30 min, with the subject lying supine. Hypertension was defined as an average (calculated from

three measurements) systolic BP (SBP) ≥140 mmHg or an average diastolic BP (DBP) ≥90 mmHg.[19]

## RESULTS

Collected data were analyzed to assess the mean age, mean IOP in the right and LE, and mean SBP and DBP. Statistical Package for the Social Sciences for Windows version 22 was used. Simple descriptive statistical methods (mean and standard deviation) were used to describe numerical data of the sample. Frequency and percentage were used to present categorical values.

A comparison of variables between smokers and non-smokers performed using the independent *t*-test for quantitative variables and the Chi-square test for categorical variables. $P < 0.05$ was considered as statistically significant.

A total of 200 apparently healthy males of the age group of 20–40 years participated in the study, 100 of whom were smokers. Mean age of smokers was 29.31 ± 5.97 years and that of non-smokers was 28.71 ± 6.75 years. Majority of smokers ($n = 34$) and non-smokers ($n = 40$) belonged to the 20–25 years age group. Difference in the mean age between smokers and non-smokers was not statistically significant by *t*-test ($P > 0.05$). The two groups were homogeneous.

About 70% of smokers ($n = 70$) used cigarettes exclusively, 21% of smokers ($n = 21$) used bidis exclusively, and 9% of smokers ($n = 9$) used both cigarettes and bidis. Distribution of the smokers based on the number of cigarettes or bidis smoked per day is given in Figures 1 and 2. Majority of the cigarette smokers smoked 1–10 cigarettes per day, whereas majority of the bidi smokers smoked 21–30 bidis per day. 51% ($n = 51$) of smokers were light smokers, 21% ($n = 21$) were moderate smokers, and 28% ($n = 28$) were heavy smokers. Majority of smokers were light smokers.

Smokers had a mean IOP of 14.68 ± 3.18 mmHg in the RE and a mean IOP of 15.69 ± 3.77 mmHg in the LE. Mean IOP in the RE of non-smokers was 14.45 ± 2.85 mmHg, and in the LE, it was 15.11 ± 2.75 mm Hg. There was no statistically significant difference in the mean IOP between the RE and the LE in both smokers and non-smokers ($P > 0.05$). Hence, only mean IOP of the LE has been considered for discussion. Difference in the mean IOP between smokers and non-smokers too was not statistically significant ($P > 0.05$). 5% ($n = 5$) of smokers showed elevated IOP (>21 mmHg), whereas none of the non-smokers showed higher IOP.

Mean SBP among smokers was 119.52 ± 6.99 mmHg, and in non-smokers, it was 118.92 ± 7.45 mmHg. Mean DBP among smokers was 79.82 ± 5.08 mmHg, whereas in non-smokers, it was 79.48 ± 3.04 mmHg. Difference in the mean SBP and mean DBP among the smokers and non-smokers was statistically insignificant ($P > 0.05$).

About 27% ($n = 27$) of smokers had SBP >120 mmHg and 29% ($n = 29$) of smokers had DBP >80 mmHg. 20% of ($n = 20$) non-smokers had SBP >120 mmHg and 7% ($n = 7$) of non-smokers had DBP >80 mmHg.

Majority of the smokers with IOP>21 mmHg were >30 years of age, heavy smokers with SBP <120 mmHg and DBP >80 mmHg [Table 1].

The difference in proportion of smokers with IOP >21 mmHg did not show any statistically significant association with age, SI, SBP, or DBP [Table 2].

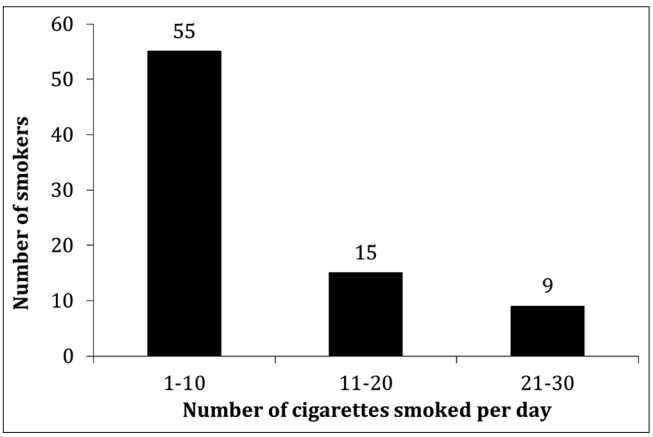


**Figure 1:** Distribution of smokers based on the number of cigarettes smoked per day

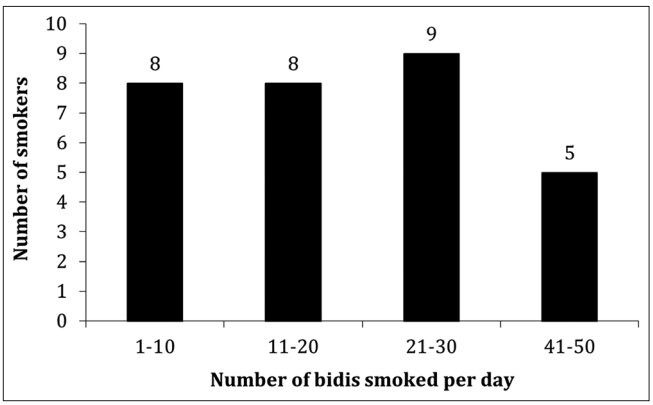


**Figure 2:** Distribution of smokers based on the number of bidis smoked per day

**Table 1:** Comparison of variables between smokers and non-smokers

| Variables | Non smokers (*n*=100) | Smokers (*n*=100) | *P* value |
|---|---|---|---|
| Age (years) | 28.71±6.7 | 29.31±5.97 | 0.506 |
| RE IOP (mmHg) | 14.45±2.85 | 14.68±3.18 | 0.578 |
| LE IOP (mmHg) | 15.11±2.75 | 15.69±3.77 | 0.218 |
| SBP (mmHg) | 118.92±7.45 | 119.52±6.99 | 0.557 |
| DBP (mmHg) | 79.48±3.04 | 79.82±5.08 | 0.566 |

DBP: Diastolic blood pressure, SBP: Systolic blood pressure, IOP: Intraocular pressure

**Table 2:** Association of smokers with IOP>21 mmHg with variables

| Variables | Number of smokers with IOP>21 mmHg | Number of smokers with IOP≤21 mmHg | *P* value |
|---|---|---|---|
| Age in years | | | |
| <30 | 2 | 59 | 0.323 |
| >30 | 3 | 36 | not significant |
| SI | | | |
| <300 | 2 | 72 | 0.102 |
| >300 | 3 | 28 | not significant |
| SBP (mmHg) | | | |
| <120 | 4 | 69 | 0.718 |
| >120 | 1 | 26 | Not significant |
| DBP (mmHg) | | | |
| <80 | 2 | 69 | 0.117 |
| >80 | 3 | 26 | not significant |

DBP: Diastolic blood pressure, SBP: Systolic blood pressure, IOP: Intraocular pressure, SI: Smoking index

**Table 3:** Correlation of SI with the variables

| Variables | Correlation coefficient | *P* value |
|---|---|---|
| RE IOP | 0.153 | 0.132 |
| LE IOP | 0.087 | 0.389 |
| SBP | −0.015 | 0.884 |
| DBP | 0.077 | 0.448 |

DBP: Diastolic blood pressure, SBP: Systolic blood pressure, IOP: Intraocular pressure, SI: Smoking index

Correlation of SI with IOP of RE, IOP of LE, SBP, and DBP shows that the RE IOP, LE IOP, and DBP exhibit a very weak positive correlation with SI but are not statistically significant. SBP shows a very negative correlation with SI but is not statistically significant [Table 3].

## DISCUSSION

Our study reports almost identical mean IOP for smokers and non-smokers. This reporting concurs with the observations of a Denmark study which also reported an identical mean IOP for smokers and non-smokers.[20] Our study did not find any significant relationship between tobacco smoking and IOP. This finding agrees with the results of a study conducted in Toronto which showed the average tonometer readings were closely similar in smokers, exsmokers, and non-smokers and multiple regression analysis showed similar age coefficients for the three categories.[21]

Among the smokers with a higher IOP, most of them showed high SI (heavy smokers). This concurs with observations of a Canadian study, where pressures were slightly higher in the small subgroup of subjects with very high cigarette consumption.[22]

A prospective follow-up study conducted from 1980 to 1986, respectively, to 1996, reported that neither current smokers nor exsmokers were at greater risk for POAG than those who had never smoked and that heavy smokers were not at an increased risk of developing POAG.[23] According to a systematic review, there was little evidence for association between smoking and POAG.[24] The Beaver dam eye study reported that there was no significant effect of iris color, refractive error, cigarette smoking, or alcohol consumption on IOP.[25] These reported findings concur with the results of our study.

Our study did not find any significant correlation between smoking and BP, and we would like to put forth the following hypothesis in support of the findings of the present study.

Aqueous humor is produced in the ciliary processes of the ciliary bodies by two mechanisms - by active secretion (80%) and through ultrafiltration of plasma (20%). Rate of filtration is influenced by BP in the ciliary body capillaries and plasma oncotic pressure. Aqueous humor outflow is through two pathways. Most of the aqueous outflow occurs through trabecular network and canal of Schlemm passing through progressively smaller pores which make up trabecular network and through the cells lining the wall of the canal. The canal communicates directly with the episcleral vein. Around 20% of the outflow occurs through the uveoscleral route.

Aqueous humor production is largely constant. A rise in IOP will be compensated to some degree by an increased rate of aqueous humor drainage. When the capacity of the trabecular drainage system is reduced or the episcleral venous pressure is raised, IOP will rise. Hence, the most common reason for increased IOP is due to the reduction in the drainage of the aqueous humor rather than increased production.[26]

High BP may raise IOP by increasing ultrafiltration of aqueous humor through elevation of ciliary artery pressure. Systemic illnesses associated with older age group are another factor influencing IOP. Our study subjects belonged

to the age range of 20–40 years. Further, in our study, there were no known hypertensives; hence, the effect of long-term hypertension on IOP is not seen in the present study.[27-30]

The presence of luminal constrictions before irregularly dilated capillaries in the anterior ciliary processes suggests precapillary sphincters. These sphincters respond to muscarinic/adrenergic receptors, thereby regulating the formation of aqueous humor. Ciliary processes have been demonstrated to contain both adrenergic and cholinergic receptors. Cholinergic receptors are of two broad classes - muscarinic cholinergic receptors and nicotinic cholinergic receptors (nAChRs). The nAChRs are further classified as nicotinic $N_M$ receptor and nicotinic $N_N$ receptor which are found at neuromuscular junctions in skeletal muscles and in autonomic ganglia, adrenal medulla, and the central nervous system, respectively.[31] Hence, there are no nicotinic receptors in the ciliary body/ciliary processes/ciliary epithelium. As there are no nicotinic receptors in the ciliary epithelium, nicotine from tobacco has no effect on the site of production of aqueous humor. Thereby, in our study, we did not find any significant actions of nicotine influencing the production of aqueous humor and thereby affecting the IOP. Heavy smoking induces degenerative changes in blood vessel wall of small arteries, represented by arteriosclerosis. These degenerative changes of small arteries may lead to an increased BP which by increasing ultrafiltration could lead to raised IOP. A large percent of the study subjects were light-to-moderate smokers. Hence, the vascular changes induced by heaving smoking, leading to a higher IOP could not be demonstrated.[32,33]

In the small percentage of the subjects who were heavy smokers, higher IOP readings could probably be attributed to the degenerative changes in the ciliary arteries induced by their higher levels of smoking. Vasoactive substances in cigarettes and bidis may compromise the vascular system by increasing blood viscosity and inducing vasospasms, which could lead to an increased IOP.[34] Increased blood viscosity in the episcleral veins raise the IOP by increasing resistance to outflow of aqueous into the veins. Smoking induced blood hyperviscosity is seen over long duration of smoking. As most of the smokers in our study were light-to-moderate smokers, we were unable to record this effect of smoking tobacco on IOP.

Any external pressure on episcleral veins such as by contraction of the extraocular muscles or mechanical pressure due to increased orbital fat as seen in Graves' ophthalmopathy, constrict the veins and lead to increased IOP. As any systemic disorder including any endocrinal disorder was the exclusion criteria in our study, extraorbital fat deposition compressing the eyeballs and giving rise to a higher IOP could not be demonstrated.

Disturbance of the physiological balance between the prooxidants and antioxidants modulates the generation of oxidative stress. Increased amount of free radicals, beyond the neutralizing capacity of normal detoxification systems, leads to oxidative stress.[35]

We assume in the majority of our subjects, the balance between the free radical generation and their detoxification has been maintained, probably because the number of cigarettes smoked is not high enough to disturb the balance or production of antioxidants has not yet been affected by tobacco smoking. Hence, the effect of oxidative stress on IOP is not seen in our subjects, and there has been no increase in IOP among the smokers.

### Limitations

The sample size in our study was relatively small, and further studies involving larger sample sizes are needed to examine the association between smoking and IOP. Our study was a cross-sectional study, wherein the obtained data do not allow for conclusions about causal relations.

## CONCLUSIONS

Tobacco smoking does not have an effect on the IOP in the age group which was studied, and tobacco smokers are not at a greater risk for developing elevated IOP as compared to non-smokers. 5% of smokers with high SI had elevated IOP (up to 24.4 mmHg). In them, we conclude that raised IOP could be due to other contributory factors not included in our study. Further studies with a larger sample size incorporating a wider age range of subjects and longer follow-up are needed to confirm our findings before strong conclusion can be drawn that tobacco smoking does not affect IOP.

## REFERENCES

1. O'Connor RJ. Non-cigarette tobacco products: What have we learned and where are we headed? Tob Control 2012;21:181-90.
2. Available from: http://www.searo.who.int/india/topics/tobacco/en. [Last accessed on 2018 Jul 02].
3. Available from: https://www.census2011.co.in. [Last accessed on 2018 Jul 02].
4. Available from: https://www.tobaccoatlas.org/wp-content/uploads/pdf/india-country-facts.pdf. [Last accessed on 2018 Jul 02].
5. International Agency for Research on Cancer. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Lyon (France): IARC; 2004.
6. Hasan A, Ansari N, Parvez A, Beg M, Bhargava R. Understanding the relation between COPD and coronary artery disease. J Indian Acad Clin Med 2014;15:120-4.
7. Solberg Y, Rosner M, Belkin M. The association between cigarette smoking and ocular diseases. Surv Ophthalmol 1998;42:535-47.
8. Cheng AC, Pang CP, Leung AT, Chua JK, Fan DS, Lam DS. The association between cigarette smoking and ocular diseases. Hong Kong Med J 2000;6:195-202.

9. Gordon MO, Beiser JA, Brandt JD, Heuer DK, Higginbotham EJ, Johnson CA, *et al*. The ocular hypertension treatment study: Baseline factors that predict the onset of primary open-angle glaucoma. Arch Ophthalmol 2002;120:714-20.
10. Pascolini D, Mariotti SP. Global estimates of visual impairment: 2010. Br J Ophthalmol 2012;96:614-8.
11. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. Br J Ophthalmol 2006;90:262-7.
12. Murthy GV, Gupta SK, Bachani D, Jose R, John N. Current estimates of blindness in India. Br J Ophthalmol 2005;89:257-60.
13. Buys YM, Harasymowycz P, Gaspo R, Kwok K, Hutnik CM, Blondeau P, *et al*. Comparison of newly diagnosed ocular hypertension and open-angle glaucoma: Ocular variables, risk factors, and disease severity. J Ophthalmol 2011;2012:757106.
14. Singh N, Aggarwal AN, Gupta D, Behera D, Jindal SK. Quantified smoking status and non-small cell lung cancer stage at presentation: Analysis of a North Indian cohort and a systematic review of literature. J Thorac Dis 2012;4:474-84.
15. Jindal SK, Malik SK, Dhand R, Gujral JS, Malik AK, Datta BN. Bronchogenic carcinoma in Northern India. Thorax 1982;37:343-7.
16. Timothy CO, Nneli RO. The effects of cigarette smoking on intraocular pressure and arterial blood pressure of normotensive young Nigerian male adults. Niger J Physiol Sci 2007;22:31-5.
17. Afshan A, Bhutkar MV, Reddy R, Patil RB. Effect of chronic cigarette smoking on intraocular pressure and audio-visual reaction time. Int J Biol Med Res 2012;3:1760-3.
18. Khurana I. Text Book of Medical Physiology. 1st ed. Noida: Elsevier; 2006. p. 335, 1183.
19. Li G, Wang H, Wang K, Wang W, Dong F, Qian Y, *et al*. The association between smoking and blood pressure in men: A cross-sectional study. BMC Public Health 2017;17:797.
20. Bahna SL, Bjerkedal T. Smoking and intraocular pressure. Acta Ophthalmol (Copenh) 1975;53:328-34.
21. Shephard RJ, Ponsford E, Basu PK, LaBarre R. Effects of cigarette smoking on IOP and vision. Br J Ophthalmol 1978;62:682-7.
22. Morgan RW, Drance SM. Chronic open-angle glaucoma and ocular hypertension. An epidemiological study. Br J Ophthalmol 1975;59:211-5.
23. Kang JH, Pasquale LR, Rosner BA, Willett WC, Egan KM, Faberowski N, *et al*. Prospective study of cigarette smoking and the risk of primary open-angle glaucoma. Arch Ophthalmol 2003;121:1762-8.
24. Edwards R, Thornton J, Ajit R, Harrison RA, Kelly SP. Cigarette smoking and primary open angle glaucoma: A systematic review. J Glaucoma 2008;17:558-66.
25. Klein BE, Klein R, Linton KL. Intraocular pressure in an American community. The Beaver dam eye study. Invest Ophthalmol Vis Sci 1992;33:2224-8.
26. Winkler NS, Fautsch MP. Effects of prostaglandin analogues on aqueous humor outflow pathways. J Ocul Pharm Ther 2014;30:102-9.
27. Shiose Y. The aging effect on intraocular pressure in an apparently normal population. Arch Ophthalmol 1984;102:883-7.
28. Mori K, Ando F, Nomura H, Sato Y, Shimokata H. Relationship between intraocular pressure and obesity in Japan. Int J Epidemiol 2000;29:661-6.
29. Wu SY, Leske MC. Associations with intraocular pressure in the Barbados eye study. Arch Ophthalmol 1997;115:1572-6.
30. Ganley JP. Epidemiological aspects of ocular hypertension. Surv Ophthalmol 1980;25:130-5.
31. Golan DE, Tashjian AH Jr., Armstrong EJ, Armstrong AW. Principles of autonomic and peripheral nervous system pharmacology, Table 8-1, characteristics of cholinergic receptor subtypes. IIB. In: Principles of Pharmacology – The Pathophysiologic Basis of Drug Therapy. 2nd ed. Philadelphia, PA: Lippincott, William and Wilkins; 2008. p. 112.
32. Frohlich ED. Hemodynamics of hypertension. In: Genest J, Koiw E, Kuchel O, editors. Hypertension. New York: McGraw-Hill Book Company; 1977. p. 15-49.
33. Van Auker MD. Pathophysiology of hypertension in the elderly. Am J Geriatr Cardiol 2000;9:16-26.
34. Ritch R. Complementary therapy for the treatment of glaucoma: A perspective. Ophthalmol Clin N Am 2005;18:597-609.
35. Halliwell B, Gutteridge JM. Free radicals and antioxidant protection: Mechanisms and significance in toxicology and disease. Hum Toxicol 1988;7:7-13.


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