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Original research

The acute effects of single cup of coffee on ocular biometric parameters in healthy subjects

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Abstract

Purpose: To evaluate ocular biometric changes in healthy subjects after caffeine consumption from a cup of coffee. *Methods*: A total of 36 subjects were included in this prospective observational study. Axial length (AL) and anterior segment parameters including aqueous depth (AD), anterior chamber depth (ACD), lens thickness (LT), and central corneal thickness (CCT) were measured with optic biometry, Lenstar LS 900 (Haag-Streit, Inc., Koeniz, Switzerland) before and 1 and 4 h after ingesting a cup of coffee (60 mg caffeine/ 100 mL).

Results: Mean age of the participants was 30.05 ± 7.43 years (range, 19-45). At baseline, 1st, and 4th hour, AL values were 23.9 ± 1.04 mm, 23.91 ± 1.04 mm, and 23.89 ± 1.04 mm, respectively, and no significant difference was observed (P>0.05). At baseline, 1st, and 4th hour, AD values were 3.06 ± 0.3 mm, 3.11 ± 0.3 mm, and 3.09 ± 0.3 mm, and ACD values were 3.6 ± 0.32 , 3.66 ± 0.31 , and 3.64 ± 0.31 , respectively. AD and ACD values were significantly greater than baseline at 1st and 4th hours following coffee ingestion. Coffee intake caused a significant reduction in LT, compared with baseline and at the 1st and 4th hours which were 3.76 ± 0.28 mm, 3.69 ± 0.32 mm, and 3.72 ± 0.27 mm, respectively. No statistically significant difference was determined in between the 3 measurements in terms of CCT (P>0.05).

Conclusion: Caffeine causes a significant increase in AD and ACD and a significant decrease in LT following oral intake, for at least 4 h. Copyright © 2019, Iranian Society of Ophthalmology. Production and hosting by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Keywords: Axial length; Anterior chamber depth; Caffeine; Central corneal thickness; Coffee; Lens thickness

Introduction

Coffee, a rich source of caffeine, is frequently consumed by people every day. A cup of coffee generally contains 60 mg-300 mg of caffeine, depending on the different styles of preparation.¹ Because of its hydrophobic properties, caffeine passes all biological membranes and, after oral intake, widely distributes to all organs including the nervous system and the eyes.² Although very limited data are known about the pharmacokinetics of caffeine in ocular tissues,² several studies investigated the clinical effects of caffeine in the eye. Coffee

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was attributed to decreased ocular blood flow via constricting retinal arterioles and venules,³ decreased choroidal thickness,⁴ -although controversial-increased intraocular pressure (IOP),⁵ and enlarged the pupils.⁶

The evaluation of axial length (AL) and anterior segment parameters including anterior chamber depth (ACD) and aqueous depth (AD), lens thickness (LT), and central corneal thickness (CCT) provides valuable information in understanding the mechanism of ocular pathologies, risk assessment, and monitoring of several diseases and preoperative calculations for cataract and refractive surgeries.⁷ Ocular structures such as iris muscle, ciliary body and zonules, and choroidal and conjunctival vessels receive autonomic innervation.⁸ Thus, caffeine, which is known to have an intensive effect on sympathetic and parasympathetic nerve activity, might be expected to alter several ocular parameters.

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The effects of coffee on IOP,⁹ choroidal thickness,¹⁰ pupillary response,¹¹ ocular perfusion,⁵ and macular degeneration¹² have been widely assessed in previous studies, however no study so far has investigated the association of coffee intake with AL, ACD, AD, and LT. In this study, we aimed to evaluate the potential acute changes in the ocular biometric measurements after ingestion of a cup of coffee.

Methods

In this prospective, observational study, 36 healthy subjects were recruited from the staff at our department. All subjects were informed about the goals of the study, and informed consent was obtained. This study followed the tenets of the Declaration of Helsinki, and the protocol was approved by the local Ethics Committee.

Each subject underwent a standard ophthalmological examination including best corrected visual acuity (BCVA), slitlamp examination, Goldmann applanation tonometer, pachymetry, and indirect retinoscopy to exclude any undiagnosed ocular disease. Exclusion criteria included any corneal, lenticular or ocular surface abnormalities, any history of glaucoma or ocular surgery or ocular trauma, and refractive errors exceeding ± 2.0 diopters. Chronic coffee consumers (>one cup of coffee/daily, regularly) were not allowed to participate in the study. Ocular biometric parameters including AL, AD, ACD, LT, and CCT were measured with non-contact ocular biometry, Lenstar LS 900 (Haag-Streit, Inc., Koeniz, Switzerland). Five readings were taken for each eye. After omitting the highest and lowest values, the mean of the other three readings was used for analysis.

All subjects were advised not to ingest either caffeinecontaining beverages or chocolate 24 h prior to basal measurements. All measurements were performed under the same room illumination and under natural pupil size. After the placement of the head in the suitable position, the patient was asked to look at the blue fixation light to control accommodation. To avoid diurnal variations, all basal measurements were performed at 9.00 a.m. in the morning by the same experienced technician, and thereafter, subjects received one cup of coffee (60 mg caffeine/100 mL). Ocular biometric measurements were repeated at the 1st and 4th hours after coffee consumption.

The measurements of ocular biometric parameters from the right eyes were used for the analyses. All statistical tests were performed using statistical software package IBM SPSS version 23 (IBM Corp., Armonk, NY, USA). The normal distribution of the data was confirmed for each variable using a Kolmogorov-Smirnov test (P > 0.05). The results are expressed as means \pm standard deviation (SD). One-way repeated measures ANOVA and paired *t*-test were used in analyzing data. The results were considered significant at P < 0.05 and a confidence interval of 95%.

Results

The mean age of the participants was 30.05 ± 7.43 ranging from 19 to 45 years. Of the 36 subjects, 21 were female

(58.3%), and 15 subjects were male (41.7%). Mean ocular biometric parameter values of subjects at baseline and at 1st and 4th hours of coffee consumption are listed in Table 1.

We did not observe any statistically significant difference between baseline, 1st hour, and 4th hour mean AL values (PALbaseline vs AL1st hour = 0.11, ALbaseline vs AL4th hour = 0.13, AL1st hour vs AL4th hour = 0.11).

The AD was significantly greater at the 1st hour of coffee intake than baseline. At the 4th hour, AD was slightly reduced but still significantly greater when compared with baseline value (*P* ADbaseline vs AD1st hour = 0.02, ADbaseline vs AD4th hour = 0.03, AD1st hour vs AD4th hour = 0.1).

Mean ACD both at the 1st and 4th hours was significantly greater than the baseline. There was no significant difference between the 1st and 4th-hour values (*P* ACD*baseline* vs ACD*1st hour* = 0.005, ACD*baseline* vs ACD*4th hour* = 0.008, ACD*1st hour* vs ACD*4th hour* = 0.13).

Coffee intake caused a significant reduction in LT, compared with baseline, at 1st and 4th hours. No significant difference was observed between the 1st and 4th-hour values (*P* LT*baseline* vs LT*1st hour* = 0.005, LT*baseline* vs LT*4th hour* = 0.02, LT*1st hour* vs LT*4th hour* = 0.14). Additionally, after adjustment for age, the similar statistical findings were obtained (*P* LT*baseline* vs LT*1st hour* = 0.004, LT*baseline* vs LT*4th hour* = 0.13).

No statistically significant difference was determined between the 3 measurements in terms of CCT (*P* CCT*baseline* vs CCT1st hour = 0.9, CCT*baseline* vs CCT4th hour = 0.06, CCT1st hour vs CCT4th hour = 0.7).

Discussion

In this study, we have observed a significant increase in both AD and ACD, and a significant reduction in LT for at least 4 h after healthy subjects drank 60 mg caffeinecontaining coffee. On the other hand, AL and CCT remained unchanged.

Caffeine, a methylated derivative of xanthine, was commonly consumed by a wide range of population for its central nervous system stimulant effect. Major systemic effects of caffeine include increased blood pressure, decreased heart rate, and reduced cerebral blood flow.¹³ Caffeine additionally decreases retinal blood flow by increasing the resistivity factor of retinal vessels, and it has been shown that shortly after oral intake, caffeine causes a significant reduction in choroidal thickness.^{4,14}

Table	1					
Mean	anterior segment parameters	at baseline,	1st, and	4th hour	of the	study.

	Baseline	1st hour	4th hour
AL (mean \pm SD) mm	23.9 ± 1.04	23.91 ± 1.04	23.89 ± 1.04
AD (mean \pm SD) mm	3.06 ± 0.3	3.11 ± 0.3	3.09 ± 0.3
ACD (mean \pm SD) mm	3.6 ± 0.32	3.66 ± 0.31	3.64 ± 0.31
LT (mean \pm SD) mm	3.76 ± 0.28	3.69 ± 0.32	3.72 ± 0.27
CCT (mean \pm SD) μ m	545.75 ± 39.53	545.8 ± 40.01	545.16 ± 38.89

AL: Axial length; AD: Aqueous depth; ACD: Anterior chamber depth; LT: Lens thickness; CCT: Central corneal thickness.

It is controversial that acute intake of caffeine has an impact on IOP. Current literature showed that caffeine had different effects on IOP in different groups of subjects. A transient elevation of IOP has been reported after oral caffeine consumption in patients with different types of glaucoma and occasionally in healthy eyes.^{15,16} Some researchers claimed that coffee consumption has no effect on IOP in the healthy population unlike results in an elevation of IOP in patients with glaucoma.¹⁷ In contrast, Chandra et al. reported that caffeine has no significant effect on IOP even in patients with glaucoma. The mechanisms that caffeine has been suggested to cause elevation of IOP include promoting aqueous production, either via increasing intracellular cyclic adenosine monophosphate (AMP) or increasing hydrostatic pressure, or inhibiting outflow via adenosine receptors.⁵ It is important to assess how the structures of the anterior chamber and angle affect IOP. Shallow ACD was reported as a strong risk factor for angle closure glaucoma.^{18,19} Until recently, there were no clear data about the effects of coffee intake on AD or ACD.

Several previous studies have suggested that caffeine may be helpful in inhibiting oxidative stress in the lens with the consequence of attenuating cataract formation.²⁰ In a recent study, caffeine accumulation was shown in lens capsule and lens epithelial cells in a dose-dependent manner after oral intake.²¹ Caffeine was reported to inhibit phosphodiesterase in the lens metabolism of galactosemic rats with a reactive oxygen species scavengering antioxidant mechanism that provides an anti-cataractogenic effect.²² Additionally, topicallyapplied caffeine was reported to protect against ultraviolet radiation induced cataract.²³ Since the clinical importance of LT after oral intake of caffeine is still unclear, more work is needed to elucidate the effects of caffeine on lens structure.

Our study suggested that a single cup of coffee increases ACD and decreases LT of individuals who are not regular caffeine consumers. Caffeine was previously reported as a central and autonomic nervous system stimulant.²⁴ Therefore, the expected effect is a sympathetic activation of the intrinsic muscles of the eye, as well. The sympathetic innervation of ciliary muscle is mediated by $\beta 2$ adrenergic receptors which lead to ciliary muscle relaxation and thus a thinner lens. The taut form of the suspensory ligaments may possibly pull irislens diaphragm to a more posterior dimension. As a result, a wider angle and a deeper anterior chamber may occur. However, recent studies demonstrated controversial results to explain a possible mechanism. Abokyi et al. observed paradoxical effects of caffeine: both dilatation of pupils and on the other hand, an enhanced accommodation amplitude.⁶ Moreover, Kirshner and Schmid observed a negative correlation between caffeine intake and reading improvement which was an unexpected performance of a neurostimulator.²⁵ They accused increased aberrations due to pupil dilatation for the decreased vision performance, and this hypothesis was supported by a study which has reported increased trefoil and higher order spherical aberrations after a single administration of coffee intake in non-regular consumers.¹¹ Apart from the previous studies, the result of this study may explain decreased

near vision performance by a decreased LT which results in a lower refractive power.

The evaluation of CCT is of paramount importance for the diagnosis and follow-up of several eye disorders such as glaucoma and corneal ectatic diseases. In the current study, differences in CCT values before and after oral coffee intake were not statistically significant. No clinical study exists in the literature regarding the impact of oral caffeine administration on corneal morphology and thickness. Kujawa-Hadryś et al. demonstrated that caffeine causes changes of collagen fiber pattern in Bowman's membrane and corneal stroma²⁶ and decreases the total thickness of developing chick embryo cornea.²⁷ Evereklioglu et al. reported some teratogenic effects on newborn rat cornea with excessive gestational caffeine intake.²⁸ Moreover, it has been demonstrated that caffeine inhibits ultraviolet irradiation-induced apoptosis in corneal epithelial cells.²⁹ It would seem that the effect of caffeine on corneal structure should be carefully evaluated with future clinical studies.

It is known that when accommodation is active there is a decrease of the ACD and an increase of the LT.³⁰⁻³² The classic theory of Helmholtz and Fincham indicates that, during accommodation, the anterior surface of the lens becomes steeper and moves forward (increasing its thickness), causing a decrease of the ACD.³³ In recent studies, LT and ACD changes were compared in near and far fixation conditions by IOL Master 700.34,35 Mean LT was increased and ACD was decreased under near fixated condition as coherent with the classical theory. The repeatibility of IOL Master 700 device was also determined, and the LT and ACD values were more prone to fluctuate in non-presbyopic group due to small accommodative alterations.³⁶ However, this situation did not cause a significant change in intraocular lens (IOL) calculations. Lenstar 900 device which is used in this study urges patients to fixate on the measurement beam, and in case of a fixation loss, the device itself detects this situation and resumes again when the fixation is re-gained. So, with the blue light fixation, even in an accommodating scenario, the LT and ACD change occurs similarly in both measurements. Thus, the accommodation effect on measurements is negligible. Based on the readings in the Table 1, the evidence indicates that the lens-iris diaphragm movements were similar in both measurements, and the ACD and LT alteration amounts were nearly same. Besides, the measurements were performed 5 times in every 3 sessions, and in order to increase the accuracy of the results, the highest and lowest numbers were excluded, and the average of the 3 measurements were statistically analyzed. We also repeated the analysis for LT after adjustment for age, which did not reveal a significance. However, for a further study, another sample group included only presbyopic participants would be reasonable to compare with nonpresbyopic ones, in order to detect the effect of age on anterior chamber measurements.

Our study has a number of limitations. The number of the participants was relatively small, and there was no control group. We only observed the changes after a single administration of coffee (60 mg caffeine/100 mL). Future studies

should investigate the impact of caffeine on ocular biometric parameters after oral intake of various doses of coffee. Our study was designed to measure several ocular biometric parameters at 1st and 4th hour of oral coffee ingestion because caffeine reaches maximum blood levels after 30-120 min following oral intake, and the half-life is 3-6 h.³⁷ We measured the study parameters maximally 4 h later than oral coffee intake, and we did not extend the experiment beyond. Lastly, coffee is not equivalent to caffeine. The effects on ocular biometric parameters attributed to coffee ingestion may not be the sole acts of caffeine as there are other active molecules in coffee including furfural, formaldehyde, and acrolein.³⁸ A future study may be designed with oral caffeine capsules to demonstrate the pure effect of caffeine on ocular biometric measurements.

This study was performed in a non-chronic coffee consumer group with regards to the current studies. Wilhelm et al. reported that caffeine has no effect on pupil size in chronic consumers.³⁹ Moreover, Bardak et al. performed a similar study to determine the effect of single cup of coffee on aberrometry changes in a non-chronic consumer group.¹¹ On the other hand, an up-to-date study comparing the measurements of chronic and non-chronic coffee consumers might be valuable for the literature.

AL is the distance from the anterior corneal surface to an interference peak corresponding to the retinal pigment epithelium.⁴⁰ Changes in accommodation and IOP have both been found to be associated with short-term changes in AL.^{34,41–43} Additionally, researchers suggested that diurnal variations in AL⁴⁴ may be mediated by the changes in choroidal thickness.⁴⁵ As we mentioned before, IOP, accommodation, and choroidal thickness are prone to change with caffeine intake. In the current study, we could not observe a significant difference in AL before and after coffee drinking.

In conclusion, in the current study, we evaluated the effects of coffee intake on ocular biometric parameters in healthy subjects and showed that ingestion of a single cup of coffee results in a significant increase in AD and ACD and a significant decrease in LT. The importance of these changes on clinical practice is unknown, and further research is warranted to better clarify the impact of caffeine on ocular biometric measurements.

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