



DOI: 10.14744/eer.2025.40469
Eur Eye Res 2026;6(1):11–20

EUROPEAN
EYE
RESEARCH

ORIGINAL ARTICLE

Effects of energy drink consumption on retinal and choroidal structures and pupil dynamics: A multimodal ocular imaging study

 **Mustafa Duran,¹**  **Caner Ozturk,²**  **Sabriye Bolat²**

¹Department of Ophthalmology, Hitit University Erol Olçok Education and Research Hospital, Corum, Türkiye

²Department of Ophthalmology, Hitit University Erol Olçok Education and Research Hospital, Corum, Türkiye

Abstract

Purpose: To evaluate the effects of energy drink (ED) consumption on choroidal thickness (CT), central macular thickness (CMT), choroidal vascularity index (CVI), retinal microvasculature, pupil diameter (PD), intraocular pressure (IOP), central corneal thickness (CCT), and anterior chamber depth (ACD).

Methods: Twenty-seven volunteers with no systemic or ocular diseases were enrolled in this prospective study. CT and CVI by optical coherence tomography (OCT), retinal vessel density (VD) by OCT-angiography, and PD by corneal topography were measured before the ED ingestion. Measurements were performed 1 h and 2 h after ED ingestion again. One week later, the measurements were repeated after the participants consumed 250 mL of water.

Results: There was a significant decrease in temporal CT at 1 h after ED intake compared to baseline ($p=0.005$). Temporal CT was not significantly different from baseline 2 h after ED intake ($p=0.763$). There was a significant increase in temporal CT at both 1 and 2 h after water intake (0.039 and 0.022, respectively). Subfoveal CT was significantly lower at 1 h after ED intake compared to baseline ($p=0.009$), whereas no significant difference in subfoveal CT was found at the end of the 2nd h ($p=0.076$). Water consumption did not affect subfoveal CT ($p=0.473$). There was no statistically significant difference in IOP, CCT, ACD, PD, CMT, nasal CT, CVI, and retinal VD measured at 1 and 2 h after both ED and water consumption compared to baseline ($p>0.05$).

Conclusion: ED consumption resulted in a transient decrease in CT but did not cause significant alterations in other anterior or posterior ocular parameters, including IOP, PD, or retinal microvasculature. These findings suggest that moderate ED intake may induce short-term vascular changes in the choroid without affecting overall retinal or anterior segment structure in healthy individuals.

Keywords: Choroidal thickness; Choroidal vascularity index; Energy drink; Retinal vessel density.

Energy drinks (EDs), widely consumed, especially by students and athletes, are promoted for their effects, such as reducing fatigue, enhancing alertness, and improving performance.^[1,2] These beverages typically contain caffeine, taurine, sugars, and various vitamins.^[3] Caffeine, a central

nervous system stimulant, exerts its effects by antagonising adenosine receptors, while taurine, an amino acid naturally found in the retina, may have vasodilatory properties.^[4,5]

Caffeine intake has been associated with systemic effects, such as increased heart rate and blood pressure, as well as



Cite this article as: Duran M, Ozturk C, Bolat S. Effects of energy drink consumption on retinal and choroidal structures and pupil dynamics: A multimodal ocular imaging study. Eur Eye Res 2026;6(1):11–20.

Correspondence: Caner Öztürk, M.D. Department of Ophthalmology, Hitit University Erol Olçok Education and Research Hospital, Corum, Türkiye

E-mail: canerx6@hotmail.com

Submitted Date: 05.05.2025 **Revised Date:** 11.08.2025 **Accepted Date:** 19.08.2025 **Available Online Date:** 29.04.2026

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ocular changes, including elevated intraocular pressure (IOP) and reduced macular blood flow.^[6] As caffeine and taurine have opposing effects on blood vessels, the combined influence of these two substances on ocular tissues may be complex. The iris, which receives both sympathetic and parasympathetic innervation, may also be affected by ED consumption.^[7]

The development of non-invasive, high-resolution imaging modalities, such as optical coherence tomography (OCT) and OCT-angiography (OCT-A) enables detailed evaluation of retinal and choroidal structures. Enhanced depth imaging (EDI) OCT allows for measurement of choroidal thickness (CT) and calculation of the choroidal vascularity index (CVI), while OCT-A provides insights into microvascular changes in the retina.^[8,9]

This study aimed to investigate the effects of ED consumption on CT, CVI, retinal microvasculature, and pupil diameter (PD) in healthy individuals using multimodal ocular imaging techniques.

Materials and Methods

This prospective study was approved by the institutional ethics committee and conducted in accordance with the Declaration of Helsinki (approval number: 2024-66). Written informed consent was obtained from all participants. The right eyes of 27 healthy subjects were included.

Participants with ocular pathologies (glaucoma, cataract, uveitis, history of intraocular surgery, retinal and corneal abnormalities, >3D myopia or hyperopia), smoking history, pregnancy, metabolic or systemic disease (including hypertension, diabetes mellitus), present drug or alcohol use were excluded. Participants who had consumed caffeinated beverages or chocolate in the previous 12 h were also excluded, as caffeine is known to remain active in the body for up to 3–6 h.^[10]

All participants underwent slit-lamp biomicroscopy, best-corrected visual acuity assessment, and fundus examination. Spherical equivalent (SE) and IOP of the participants were measured with an autorefractometer (Topcon KR-8900; Topcon Corporation, Japan). PD under photopic, mesopic, and scotopic (40 lx illuminance, 4 lx illuminance, 0.4 lx illuminance, respectively) conditions, central corneal thickness (CCT) and anterior chamber depth (ACD) were measured with Sirius topographer (Sirius, Costruzione Strumenti Oftalmici, Italy). Axial length (AL) was obtained with Nidek AL-Scan optic biometry (Nidek Co. Ltd, Japan).

Central macular thickness (CMT), subfoveal CT, and CT at 1000 μ m nasal and temporal to the fovea were measured by

Heidelberg Spectralis OCT (Heidelberg Engineering GmbH; Germany). An experienced examiner measured CT manually from the retinal pigment epithelium to the sclera using the callipers of the OCT device.

The choroidal structures, including the luminal area (LA) and the total choroidal area (TCA), were calculated using the binarization of EDI-OCT images with the ImageJ software program (version 1.50 a; National Institutes of Health, Bethesda, MD, USA). Stromal area was found from the difference of TCA and LA. The CVI values were calculated using the LA/TCA ratio (Fig. 1).

Retinal vessel density (VD) at the superficial and deep capillary plexuses (SCP, DCP) and choriocapillaris (CC) was assessed using 3 \times 3 mm OCT-A scans (Triton OCT, Topcon, Tokyo, Japan) with an early treatment diabetic retinopathy study grid. The central (inner 1 mm) VD and the nasal, inferior, superior, and temporal parafoveal VDs (located between the inner 1 mm and outer 3 mm borders) at the SCP, DCP, and CC levels were evaluated (Fig. 2).

Measurements were obtained at baseline, and at 1 and 2 h after ingestion of 250 mL ED (Red Bull[®], containing 150 mg/L caffeine, 800 mg/L taurine, small amounts of sugar, and vitamins). One week later, the same protocol was repeated with 250 mL of water. All measurements were performed between 13:30 and 15:30 to minimize diurnal variation.

Statistical Analysis

The Statistical Package for the Social Sciences programme V22 (IBM Corp., Armonk, NY, USA) was used for statistical analysis. Shapiro–Wilk test was used to evaluate the normality of the data. Repeated-measures analysis of variance with Greenhouse–Geisser correction was used for normally distributed variables; the Friedman test was applied for non-normally distributed variables. *Post hoc* Bonferroni correction was applied for multiple comparisons. Paired *t*-tests or Wilcoxon signed-rank tests were used for pairwise analyses. Data are presented as mean \pm standard deviation. A $p < 0.05$ was considered statistically significant.

Results

A total of 27 healthy participants, 14 males and 13 females, were included. No significant gender difference was observed ($p = 0.847$). The mean age of the participants was 30.74 ± 5.68 years. Mean systolic and diastolic blood pressures at baseline were 114.62 ± 7.80 and 80.89 ± 5.04 mmHg, respectively. The mean AL was 24.29 ± 0.95 mm.

Table 1 shows CCT, ACD, PD in scotopic, mesopic, and photopic conditions, CT, and CVI values of the participants

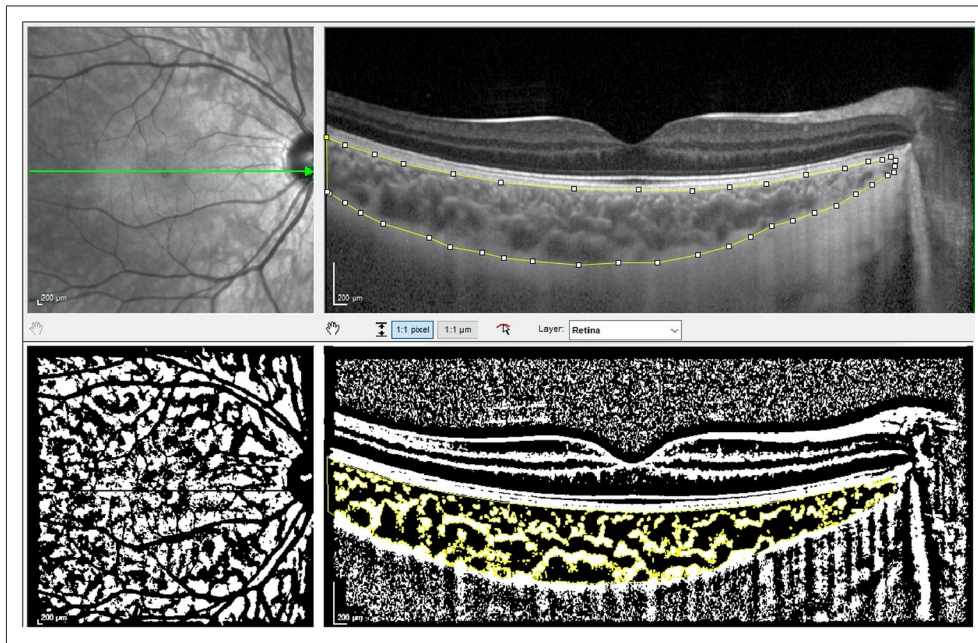


Fig. 1. Marking of the total choroidal area using Image J software. The post-acquisition spectral domain optical coherence tomography image processing demonstrates the binarization of the region of interest utilizing Niblack's auto local threshold technique. Vascular areas appear darker and stromal areas appear lighter.

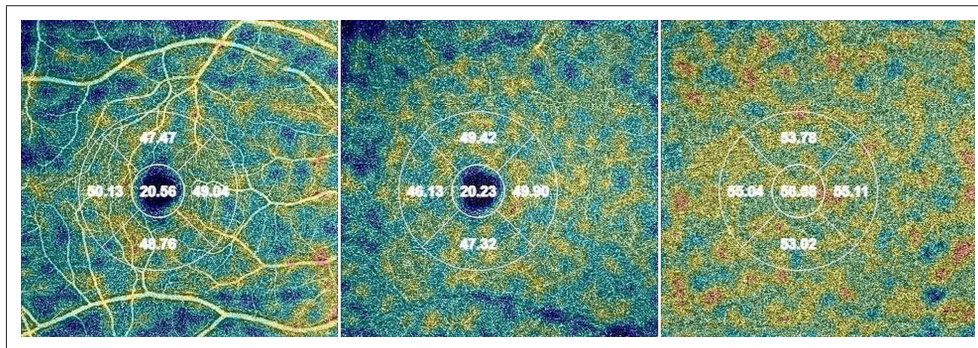


Fig. 2. 1 × 3 mm foveal EDTRS grid showing (a) Superficial vessel density, (b) Deep vessel density, and (c) Coriocabillaris vessel density.

before and after ED and water consumption. Temporal CT decreased significantly 1 h after ED consumption compared to baseline ($p=0.005$), but returned to baseline levels at 2 h ($p=0.763$). Temporal CT was significantly increased at both 1 and 2 h after water intake (0.039 and 0.022, respectively). Subfoveal CT was significantly lower at 1 h after ED consumption compared to baseline ($p=0.009$), whereas no significant difference in subfoveal CT was found at the end of the 2nd h ($p=0.076$). Water consumption did not significantly change subfoveal CT ($p=0.473$) (Fig. 3).

No significant difference was found in SE, IOP, CCT, ACD, PD, CMT, nasal CT, and CVI measured at 1 and 2 h after both ED and water consumption compared to baseline ($p>0.05$).

Table 2 shows the central, superior, inferior, nasal, and temporal parafoveal VDs at the SCP, DCP, and CC levels before and after ED and water consumption. After ED and water consumption, central and parafoveal VD values were not statistically changed ($p>0.05$).

Direct comparison of ocular parameters between ED and water conditions at baseline, 1 h, and 2 h after intake is shown in Table 3. A significant difference in temporal CT at 1 h was observed between ED and water conditions. ED intake caused a decrease in temporal CT, whereas water intake caused an increase. No other parameters, including subfoveal CT, nasal CT, CVI, PD under various lighting conditions, CMT, or VD at the SCP, DCP, and CC levels, showed significant differences between ED and water conditions at any time point (all $p>0.05$).

Table 1. Comparison of SE, IOP, CCT, ACD, PD, CMT, CT, and CVI before and after consumption of energy drink and water

Parameter	Baseline (Mean±SD)	After 1 h (Mean±SD)	After 2 h (Mean±SD)	<i>p</i>	<i>p</i>
SE (Diopters)					
ED	-0.90±0.86	-0.92±0.88	-0.90±0.91	0.952 ^f	
Water	-0.90±0.92	-0.92±0.89	-0.93±0.94	0.620 ^f	
IOP (mmHg)					
ED	16.70±3.22	16.67±2.80	16.67±2.86	0.994 ^f	
Water	17.11±2.33	16.56±2.68	16.70±2.99	0.250 ^f	
CCT (μm)					
ED	540.78±41.40	539.44±41.40	539.26±41.86	0.132 ^f	
Water	540.93±41.93	540.59±42.01	540.44±41.77	0.104 ^f	
ACD (mm)					
ED	3.27±0.22	3.27±0.22	3.27±0.23	0.932 ^f	
Water	3.28±0.22	3.28±0.22	3.26±0.22	0.305 ^f	
Scotopic PD (mm)					
ED	5.68±0.70	5.80±0.76	5.74±0.70	0.583 ^f	
Water	5.78±0.55	5.74±0.69	5.79±0.67	0.890 ^f	
Mesopic PD (mm)					
ED	5.43±0.62	5.51±0.82	5.41±0.75	0.570 ^f	
Water	5.34±0.67	5.32±0.72	5.37±0.75	0.856 ^f	
Photopic PD (mm)					
ED	4.37±0.83	4.26±0.88	4.33±0.89	0.653 ^f	
Water	4.11±0.74	4.08±0.85	4.23±0.71	0.474 ^f	
CMT (μm)					
ED	261.93±14.40	263.37±15.38	262.48±14.71	0.171 ^f	
Water	262.85±14.11	262.70±13.82	261.82±14.95	0.405 ^f	
Temporal CT (μm)					
ED	357.85±76.50	347.93±73.73	358.96±80.73	0.003 ^f	0-1 0.005 ^b 1-2 0.015 ^b 0-2 0.763 ^b
Water	351.41±82.06	363.48±79.74	364.33±91.24	0.010 ^f	0-1 0.039 ^b 1-2 0.843 ^b 0-2 0.022 ^b
Subfoveal CT (μm)					
ED	366.67±85.85	354.56±79.50	361.59±76.95	0.045 ^f	0-1 0.009 ^b 1-2 0.036 ^b 0-2 0.076 ^b
Water	363.63±74.13	363.48±90.33	368.96±90.54	0.473 ^f	
Nasal CT (μm)					
ED	326.52±81.06	322.44±76.43	327.22±74.67	0.495 ^f	
Water	325.41±76.20	330.19±84.54	326.48±81.16	0.648 ^f	
CVI (%)					
ED	0.64±0.02	0.65±0.03	0.64±0.02	0.683 ^f	
Water	0.65±0.02	0.65±0.02	0.65±0.02	0.736 ^f	

^b: Bonferroni correction. ^f: Repeated measures anova. ^f: Friedman test. SE: Spherical equivalent, IOP: Intraocular pressure, CCT: Central corneal thickness, ACD: Anterior chamber depth, PD: Pupil diameter, CMT: Central macular thickness, CT: Choroidal thickness, CVI: Choroidal vascularity index, ED: Energy drink, SD: Standard deviation

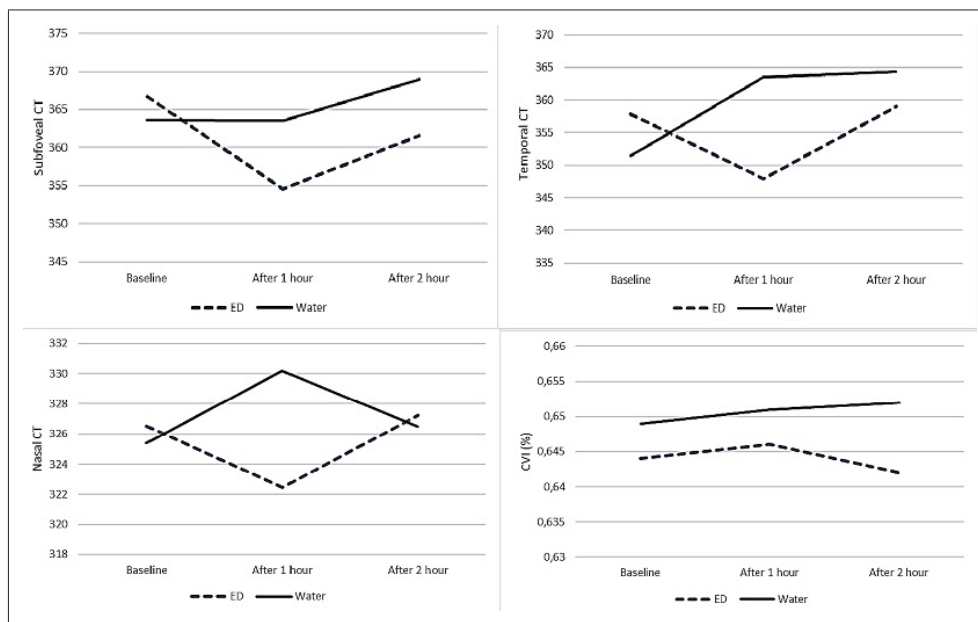


Fig. 3. Time-course changes in choroidal thickness (subfoveal, temporal, nasal) and choroidal vascular index after energy drink and water intake.

Discussion

EDs have gained popularity across all age groups, particularly among young adults, due to their stimulating effects on the central nervous system. Given the increasing concerns about their cardiovascular and neurological impacts, recent studies have also begun to investigate their potential ocular effects. In this context, our study aimed to assess both anterior and posterior segment responses to ED consumption in healthy individuals using multimodal imaging techniques.

Our findings demonstrated that ED consumption did not significantly affect SE, IOP, CCT, ACD, PD, CMT, nasal CT, CVI, or central and parafoveal VD. However, a transient decrease in temporal and subfoveal CT was observed at 1 h following ED intake, which resolved by the 2nd h.

The effect of caffeinated drinks on PD is currently unclear. Redondo *et al.*^[11] found a significant increase in PD 30 min after caffeine intake (4 mg/kg) compared to the placebo drug. A significant increase in PD was also observed by Abokyi *et al.*^[12] in those who consumed 250 mg of caffeine-containing beverages. In contrast, the study by Bardak *et al.*^[13] showed that there was no change in PD after consumption of coffee containing 57 mg of caffeine. The complex effects of caffeine on both the sympathetic and parasympathetic systems may have resulted in contradictory results.^[7] After ED consumption, we found no change in PD under photopic, mesopic, and scotopic light conditions.

The literature on the effect of caffeine on IOP is inconclusive. Jo and Lee^[14] reported that IOP increased significantly after drinking caffeinated EDs in healthy participants, whereas there was no significant change in IOP after drinking caffeine-free beverages. In other studies conducted with healthy participants, it was demonstrated that caffeine did not affect IOP.^[15,16] In a meta-analysis, Li *et al.*^[17] reported that caffeine had no effect on IOP in healthy subjects. However, it increased IOP in patients with open-angle glaucoma and ocular hypertension. In our study, we found no change in IOP after caffeine-containing ED consumption.

It has been shown by color Doppler ultrasound that consumption of caffeine-containing beverages causes narrowing of the arteries supplying the eye, including the ophthalmic artery, central retinal artery, and short posterior nasal ciliary artery.^[18] Terai *et al.*^[19] showed that retinal vessels narrowed 1 h after consuming 200 mg of caffeine. The choroid, a layer rich in vascular structures, can be strongly affected by this vasoconstrictive effect of caffeine. In our study, we found a significant decrease in subfoveal and temporal CT after ED consumption. In a study comprising 30 healthy male subjects, Toprak *et al.*^[20] observed a statistically significant reduction in CT at both 30 min and 1 h following the consumption of caffeine-containing EDs. In contrast, some studies have indicated that ED does not have an impact on CT.^[21,22] The discrepancy in results may be attributed to the varying quantities of caffeine present in the EDs utilized in the

Table 2. Comparison of central and parafoveal vessel densities at the superficial capillary plexus, deep capillary plexus, and choriocapillaris before and after consumption of energy drink and water

Parameters	Baseline (Mean±SD)	After 1 h (Mean±SD)	After 2 h (Mean±SD)	P-value
SCP (%)				
ED				
Central	20.06±4.50	19.87±3.67	19.79±3.92	0.855 ^r
Superior	45.20±3.47	44.89±2.75	45.32±2.91	0.837 ^f
Nasal	46.00±2.44	45.41±2.37	45.64±2.15	0.485 ^r
Inferior	46.11±2.88	45.42±2.42	46.10±2.87	0.253 ^f
Temporal	47.06±2.37	46.14±2.36	46.56±2.57	0.173 ^r
Water				
Central	19.82±4.12	19.63±4.27	20.31±4.56	0.204 ^r
Superior	45.03±3.14	44.74±2.38	45.07±2.32	0.837 ^f
Nasal	44.97±3.10	45.23±2.16	45.91±2.15	0.837 ^f
Inferior	44.66±3.36	45.06±2.80	45.59±2.85	0.195 ^r
Temporal	45.80±3.22	45.90±2.28	46.45±1.76	0.289 ^f
DCP (%)				
ED				
Central	15.95±4.53	15.38±3.87	15.58±4.19	0.638 ^r
Superior	46.65±3.45	47.52±2.12	46.82±2.57	0.335 ^f
Nasal	47.81±2.40	48.01±2.29	47.82±3.75	0.886 ^r
Inferior	47.22±2.61	47.10±1.98	46.77±2.34	0.156 ^f
Temporal	44.92±2.23	44.48±2.40	44.75±2.41	0.740 ^r
Water				
Central	15.53±4.34	15.17±4.15	16.09±4.32	0.108 ^r
Superior	46.39±3.72	46.48±2.31	46.73±2.52	0.335 ^f
Nasal	47.42±2.40	47.51±2.40	47.95±1.93	0.484 ^r
Inferior	45.87±2.78	46.65±2.25	47.33±2.46	0.972 ^f
Temporal	43.91±5.13	43.75±2.74	44.77±2.62	0.071 ^f
CC (%)				
ED				
Central	55.19±3.11	54.87±2.43	55.14±2.84	0.818 ^r
Superior	52.68±2.31	53.13±1.71	52.78±1.89	0.567 ^r
Nasal	52.25±2.29	51.99±2.02	52.70±2.40	0.323 ^r
Inferior	52.90±1.69	53.47±1.96	53.65±1.65	0.254 ^r
Temporal	53.48±1.78	53.42±1.81	52.96±2.42	0.431 ^r
Water				
Central	54.72±2.62	55.14±2.41	55.23±2.72	0.610 ^r
Superior	52.30±1.76	52.01±2.13	52.61±2.14	0.748 ^f
Nasal	52.30±2.14	52.88±2.11	52.71±2.00	0.437 ^r
Inferior	52.97±2.55	53.47±2.03	52.80±2.66	0.964 ^f
Temporal	53.39±1.18	53.34±2.09	53.39±1.94	0.772 ^f

^r: Repeated measures anova, ^f: Friedman test. SCP: Superficial capillary plexus, DCP: Deep capillary plexus, CC: Choriocapillaris, ED: Energy drink, SD: Standart deviation

Table 3. Direct comparison of ocular parameters between energy drink and water conditions at baseline, 1 h, and 2 h after intake

Parameter	ED (mean±SD)	Water (mean±SD)	p
Scotopic PD (mm)			
Baseline	5.68±0.70	5.78±0.55	0.370 ^P
After 1 h	5.80±0.76	5.74±0.69	0.649 ^P
After 2 h	5.74±0.70	5.79±0.67	0.499 ^P
Mesopic PD (mm)			
Baseline	5.43±0.62	5.34±0.67	0.351 ^P
After 1 h	5.51±0.82	5.32±0.72	0.107 ^P
After 2 h	5.41±0.75	5.37±0.75	0.685 ^P
Photopic PD (mm)			
Baseline	4.37±0.83	4.11±0.74	0.107 ^P
After 1 h	4.26±0.88	4.08±0.85	0.188 ^P
After 2 h	4.33±0.89	4.23±0.71	0.330 ^P
CMT (µm)			
Baseline	261.93±14.40	262.85±14.11	0.056 ^W
After 1 h	263.37±15.38	262.70±13.82	0.126 ^W
After 2 h	262.48±14.71	261.82±14.95	0.240 ^W
Temporal CT (µm)			
Baseline	357.85±76.50	351.41±82.06	0.206 ^P
After 1 h	347.93±73.73	363.48±79.74	0.003 ^P
After 2 h	358.96±80.73	364.33±91.24	0.267 ^P
Subfoveal CT (µm)			
Baseline	366.67±85.85	363.63±74.13	0.086 ^P
After 1 h	354.56±79.50	363.48±90.33	0.104 ^P
After 2 h	361.59±76.95	368.96±90.54	0.089 ^P
Nasal CT (µm)			
Baseline	326.52±81.06	325.41±76.20	0.890 ^P
After 1 h	322.44±76.43	330.19±84.54	0.091 ^P
After 2 h	327.22±74.67	326.48±81.16	0.887 ^P
CVI (%)			
Baseline	0.64±0.02	0.65±0.02	0.294 ^P
After 1 h	0.65±0.03	0.65±0.02	0.370 ^P
After 2 h	0.64±0.02	0.65±0.02	0.080 ^P
SCP central (%)			
Baseline	20.06±4.50	19.82±4.12	0.861 ^W
After 1 h	19.87±3.67	19.63±4.27	0.537 ^P
After 2 h	19.79±3.92	20.31±4.56	0.247 ^P
DCP central (%)			
Baseline	15.95±4.53	15.53±4.34	0.426 ^P
After 1 h	15.38±3.87	15.17±4.15	0.630 ^P
After 2 h	15.58±4.19	16.09±4.32	0.320 ^P

Table 3. Continue

Parameter	ED (mean±SD)	Water (mean±SD)	p
CC central (%)			
Baseline	55.19±3.11	54.72±2.62	0.368 ^W
After 1 h	54.87±2.43	55.14±2.41	0.594 ^P
After 2 h	55.14±2.84	55.23±2.72	0.891 ^P

P: Paired t-test, W: Wilcoxon signed-rank test. PD: Pupil diameter, CMT: Central macular thickness, CT: Choroidal thickness, CVI: Choroidal vascularity index, SCP: Superficial capillary plexus, DCP: Deep capillary plexus, CC: Choriocapillaris, ED: Energy drink, SD: Standard deviation

investigation. Another reason why results may vary is that CT can be affected by factors, such as refraction, gender, and age. Our findings are consistent with those reported by Arej *et al.*,^[23] who demonstrated a significant decrease in subfoveal CT 1 h after the ingestion of an ED containing caffeine and taurine. At 4 h post-consumption, subfoveal CT returned to baseline. This transient thinning suggests a transient vasoconstrictive effect, likely due to the caffeine content, which may be counteracted by the vasodilatory influence of taurine over time.

Temporal CT showed a modest but statistically significant increase at both 1 and 2 h after water intake. This finding is consistent with previous reports indicating that systemic hydration can temporarily increase CT by improving ocular perfusion and enlarging the vascular compartment.^[24,25] This effect is probably caused by an increase in blood volume and reduced blood viscosity, which facilitates choroidal filling. In contrast, ED consumption caused a transient CT reduction, presumably due to caffeine-induced vasoconstriction, which was partially offset by the vasodilatory effect of taurine.

CVI allows assessment of choroidal vascularization with higher reliability and lower variability than CT.^[8] Koçak *et al.*^[26] showed that subfoveal CVI was significantly decreased in healthy subjects who consumed coffee containing 75 mg caffeine. To our knowledge, this is the first study to evaluate the effect of ED consumption on CVI. Our results demonstrated that there was no statistically significant change in CVI following ED consumption.

There are only a few studies that show the effect of ED on the retinal microvascular blood flow in using OCT-A. Doğan *et al.*^[21] reported that VD measurements of the parafoveal and perifoveal deep capillary plexus were significantly higher after ED consumption. Karti *et al.*^[27] demonstrated that oral caffeine (200 mg) resulted in a statistically significant decrease in macular flow area (superficial, deep, and CC) and VD. Yılmaz Tugan *et al.*^[28] reported a statistically

significant decrease in VD parameters in all segments of the superficial, deep, and peripapillary macular regions after 200 mg orally caffeine intake. In our study, there was no significant change in superficial, deep, and CC VD values. The reason why the VD values were not affected by the consumption of ED can be attributed to the vasodilatory effect of taurine, in contrast to the vasoconstrictive effect of caffeine. The fact that caffeine and taurine may limit each other's effects in ED may explain the conflicting results in the literature. The absence of significant changes in retinal VD following ED intake may be explained by this opposing pharmacological interplay, as well as the relatively low caffeine dose employed in our study. Together, these observations highlight that the ocular response to ED is both component- and dose-dependent and that certain vascular beds, such as the choroid, may be more sensitive to these acute hemodynamic shifts than others. Although most studies, including ours, suggest that moderate ED consumption does not result in major retinal vascular changes, isolated case reports have described severe ocular events following excessive intake. Gupta *et al.*^[29] reported a case of acute macular neuroretinopathy in a healthy adult shortly after consuming multiple EDs. Similarly, Pagano *et al.*^[30] documented intraretinal hemorrhages and sudden visual loss attributed to high-volume ED ingestion. These cases highlight the need for further investigation into possible individual susceptibility and dose-dependent effects. Importantly, when ED and water conditions were compared directly, only the temporal CT at 1 h differed significantly between the two conditions ($p=0.003$), confirming that this transient thinning was specific to ED consumption rather than a non-specific, time-dependent change. The absence of significant differences between conditions for other ocular parameters, including subfoveal CT, suggests that the vascular response to ED intake may be more localized to certain choroidal regions. This finding supports the idea that caffeine-induced vasoconstriction, potentially moderated by taurine's vasodilatory properties, has a measurable, yet region-specific, impact on choroidal perfusion.

Our study is valuable because it evaluates how ED consumption affects both anterior segment and posterior segment structures of the eye. In addition, it is the first study to evaluate the effect of ED consumption on CVI. Considering the widespread and increasing use of EDs among young adults, understanding their comprehensive ocular effects is clinically important. Our findings contribute to a growing but still limited body of literature examining the specific impact of EDs on ocular parameters.

Study Limitations

Our study also has some limitations. All participants consumed the same amount of ED, regardless of their body weight. Therefore, the amount of caffeine and taurine per kilogram is different for each participant. The ED consumed by the participants in the study contained 37.5 mg/250 mL of caffeine, which is a relatively low amount when compared to other studies that have examined the effects of ED and caffeine on ocular parameters. The low amount of caffeine may have affected the results. Another limitation of our study is the lack of randomization and masking. While the objective nature of OCT and OCT-A measurements reduces the likelihood of measurement bias, the absence of participant masking could theoretically introduce placebo or expectation effects, particularly with regard to subjective parameters. In addition, the relatively small size of our sample and the inclusion of only young, healthy subjects limit the generalizability of the findings to broader populations, including older individuals or those with ocular or systemic diseases. Studies with larger, more diverse populations and varying caffeine doses may reveal additional effects that were not observed in our cohort.

Conclusion

In this study, we investigated the short-term effects of ED consumption on the anterior and posterior structures of the eye using multimodal imaging techniques. While ED intake did not significantly alter in IOP, CCT, ACD, CVI, foveal, and parafoveal VD, our findings revealed that it induced a transient decrease in temporal and subfoveal CT 1 h after consumption. These results suggest that the components of EDs, particularly caffeine, may cause short-term vascular responses in the choroid through sympathetic stimulation and vasoconstriction, while other ocular structures remain unaffected. The resolution of this effect by the 2nd h may reflect compensatory mechanisms or the opposing action of taurine, a vasodilatory component also present in EDs. This study supports the hypothesis that EDs can temporarily influence ocular perfusion, specifically within the choroid, without causing sustained structural changes in healthy individuals. Further studies with longer follow-up periods and varied caffeine doses are needed to better understand the long-term effects of ED consumption on the eyes.

Ethics Committee Approval: This study was approved by The Ethics committee of Hitit University (2044-66 11/09/2024)

Informed Consent: Written informed consent was obtained from the patient for the preparation of this work.

Peer-review: Externally peer-reviewed.

Authorship Contributions: Concept: M.D., C.O., S.B.; Design: M.D., C.O., S.B.; Supervision: M.D., C.O., S.B.; Resource: M.D., C.O., S.B.; Materials: S.B.; Data Collection and/or Processing: M.D., S.B.; Analysis and/or Interpretation: M.D.; Literature Search: C.O.; Writing: C.O.; Critical Reviews: M.D., C.O.

Conflict of Interest: None declared.

Use of AI for Writing Assistance: Not declared.

Financial Disclosure: The authors declared that this study received no financial support.

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