Regular physical exercise is known to lower the incidence of age-related eye diseases. We aimed to assess the acute chorioretinal alterations in older adults following intense physical strain. Seventeen senior elite athletes were recruited who underwent an aerobic exercise on a cycle ergometer and macular scanning by optical coherence tomography. A significant thinning of the entire retina was observed 1 min after exercise, followed by a thickening at 5 min, after which the thickness returned to baseline. This trend was similar in almost every single retinal layer, although a significant change was observed only in the inner retina. Choroidal thickness changes were neither significant nor did they correlate with the thickness changes of intraretinal layers. The mechanism of how these immediate retinal changes chronically impact age-related sight-threatening pathologies that, in turn, result in a substantially reduced quality of life warrants further investigation on nontrained older adults as well.

**Keywords:** senior athletes, retina, optical coherence tomography, image segmentation, physical strain

Regular physical activity has been proven to have several favorable cardiovascular (CV) effects. Beyond its beneficial effect on obesity, hypertension, serum lipoprotein, systemic inflammation, and endothelial dysfunction (Warburton et al., 2006), it has an independent impact on reducing CV risk (Dioszegi et al., 2021) and, thus, reducing mortality (Nocon et al., 2008).

Due to the reduction of oxidative stress, regular exercise ameliorates age-related arterial stiffness that is known to have a relationship with Type 2 diabetes and sedentary lifestyle, which is a possible biomarker of CV risk (Dioszegi et al., 2021; Madden et al., 2009; Shibata et al., 2018). Moreover, in older adults, even a relatively short, 3-month aerobic training intervention seems to have a positive effect on lowering CV risk (Madden et al., 2009).

As the retina is highly vulnerable to oxidative damage due to its high oxygen consumption, reactive oxidative species developing with aging may cause several degenerative diseases, such as age-related macular degeneration (AMD) and glaucoma, and may play a role in diabetic retinopathy as well (Kim et al., 2015; Sanvicens & Cotter, 2006). However, vigorous physical exercise has been postulated to have a protective effect against AMD (McGuinness et al., 2016; Williams, 2009b), and a greater cardiorespiratory fitness is suggested to decrease this risk or the possibility of cataract formation (Williams, 2009a).

Beyond the higher CV risk due to inactivity, increased abdominal fat, overweight, elevated blood pressure (BP) and serum lipoproteins, systemic inflammation, and endothelial dysfunction are also supposed to play a possible role in the pathogenesis of AMD (Snow & Seddon, 1999). A modification of lifestyle through a healthy diet, physical activity, and smoking cessation may decrease the risk for early AMD about threefold (Mares et al., 2011). Furthermore, an active lifestyle (regular activity three or more times/week) itself showed a protective effect on the incidence of neovascular AMD, possibly due to a decrease in systemic inflammation and endothelial dysfunction (Knudson et al., 2006). It is also known that regular treadmill exercise exerts an antioxidative effect on the retinae of naturally aged mice (Kim et al., 2015), can delay or prevent diabetes-induced apoptosis in retinal cells of rats (Kim et al., 2013), and protects retinal cells against light-induced retinal degeneration in mice (Lawson et al., 2014).

Although increasing evidence is available in the ophthalmic effects of regular exercise, there is only little information on the acute effects of physical exercise on the eye. There is evidence in healthy adults that critical flicker frequency, an indirect measure of optic nerve function, increases after cycling and is maintained for at least 30 min (Maciejewska et al., 2020). Following yoga exercise, an intraocular pressure (IOP) reduction has been shown with an increase in macular thickness compared with preexercise (Galina et al., 2020). Interestingly, subfoveal choroidal blood flow is doubled in patients with central serous chorioretinopathy compared with healthy controls after isometric exercise (Titt et al., 2005).

However, in older adults, there is no information available on the acute chorioretinal effects of physical activity. Therefore, in this present study, we aimed to assess the morphological alterations of the posterior pole in senior elite athletes following short intense
physical exercise using noninvasive spectral-domain optical coherence tomography (OCT) imaging.

Materials and Methods

The study has been approved by the Semmelweis University Regional and Institutional Committee of Science and Research Ethics (272/2013), and written consent was obtained from all participants in accordance with the Declaration of Helsinki.

Members of the Hungarian Senior National Team (of track and field, over 50 years) were enrolled in this prospective study. Each participant declared to have conducted intensive physical activity (described as an increase in heart rate [HR] to age-matched vita maxima) regularly, at least two times a week, for 10 or more years. Each participant completed a questionnaire for general and ophthalmic history and the type and regularity of sports activity. Anthropometric variables (e.g., height, weight) were recorded. Body mass index was calculated as the ratio of weight to height in meters squared (kg/m²). In the case of chronic diseases (such as hypertension or hypercholesterolemia), they had to be well controlled; a valid sports medical certificate issued by a board-certified sports physician was required for enrollment. The participants were asked to refrain from caffeine consumption on the study day.

Study participants went through a detailed examination including autorefractometry, best corrected visual acuity (measured on Early Treatment Diabetic Retinopathy Study [ETDRS] Chart), and biomicroscopic evaluation of the anterior and posterior segments; pupil dilation was carried out by one drop of tropicamide (5 mg/ml). A Spectralis spectral-domain OCT (Heidelberg Engineering, Heidelberg, Germany) was used for performing volumetric OCT scans of the macula, applying the “Enhanced Depth Imaging” protocol to optimally visualize the choroid. The option of posterior pole imaging with a setting of 30° (Horizontally) × 25° (Vertically) with automatic real time of 20 was applied, containing 61 scans. Required signal strength value of the scans was at least 30 to optimize image quality.

Exclusion criteria for the participants were the history of previous ophthalmic or uncontrolled systemic diseases, ocular injury or intraocular surgery within the past 6 months, abnormal appearance of the macula or the optic disk on biomicroscopic examination, best corrected visual acuity worse than 20/25, and refractive error for far distance in ophthalmic history over ± 3D spherical equivalent.

Each participant performed a stepwise incremental exercise trial until exhaustion (age-matched vita maxima, see later) or reaching a peak in systolic BP (exceeding 180 mmHg) on a cycle ergometer (Ergoline Ergoselect 200; Ergoline GmbH Bitz). The strain was initiated at 0 W, and the resistance was increased every 2 min by 25 W, whereas the pedal turn was held constant at 60/min. The maximum power achieved (final absolute work rate) in the last load step was considered in the evaluation. The performance of each subject was expressed as the power-to-weight ratio (given in W/kg), which allowed a neutral comparison between participants (Aigner, 1986).

Before and during the whole exercise and the recovery period, HR was monitored by a Polar Rs400 monitor (Polar Electro Oy) to avoid reaching the maximum physiological age-related HR (calculated as 180/min–age, according to the Maffetone formula, to provide an age-matched submaximal strain) (Fox et al., 1971). We chose the Maffetone formula (as opposed, e.g., to the Tanaka formula [Tanaka et al., 2001]) as its calculated HR levels were somewhat lower, which served the CV safety of our study subjects. BP was monitored by an Omron M6 Comfort automatic cuff sphygmomanometer (Omron Healthcare Co. Ltd.) before, during, and 1, 5, and 30 min after the exercise test.

OCT imaging was performed 1, 5, 15, 30, and 60 min following the exercise. For each test, the eye tracker function was used to provide identical imaging of the retina during the course of the study. The baseline scans were set as reference, and subsequent mapping took place at identical points using the same protocol.

As described in detail previously, the raw OCT data were exported from the OCT device and processed using a custom-built semiautomatic software (OCTRIMA 3D; Tian et al., 2015). This software runs on a MATLAB platform (The MathWorks Inc.) and collects thickness data of seven single retinal layers, the total macular volume, and the choroid from the volumetric mapping of the macula according to their reflectivity. As the software allows semiautomatic image processing, during the review of the segmentation result, the automatic designation of the boundaries of the layers can be corrected manually when necessary. The high reproducibility of the OCTRIMA 3D segmentation of macular OCT scans has been previously confirmed in healthy participants (DeBuc et al., 2009).

The thickness data of the total retina and of the following layers in the nine ETDRS regions (i.e., in the central subfield and in the superior, nasal, inferior, and temporal regions in the inner and the outer rings as well) were recorded: the retinal nerve fiber layer (RNFL); the complex layer of the ganglion cell and inner plexiform layer (GCL + IPL); the inner nuclear layer (INL); the outer plexiform layer (OPL); the complex layer containing the Henle fibers, outer nuclear layer, external limiting membrane, and the myoid zone of the photoreceptors (ONL + IS); the complex layer of the ellipsoid zone and the outer segment of the photoreceptors (ELZ + OS); the complex layer containing the interdigitation zone, the retinal pigment epithelium, and Bruch’s membrane (IDZ + RPE); and the choroid (CRC), consisting of the choriocapillaris, Sattler’s layer, and Haller’s layer as far as the chorioidal–scleral juncure (Figure 1). This nomenclature follows the recommendation of the International Nomenclature for Optical Coherence Topography Panel (Staurenghi et al., 2014).

We also assessed the physiologically different parts of the retina as composite layers, such as the ganglion cell complex (GCC, consisting of the RNFL+GCL+IPL), the inner retina (IR, GCC+INL), a complex containing the cellular elements of the photoreceptor layer (PRL, ONL+IS+ELZ+OS), and the outer retina (OR, OPL+ONL+IS+ELZ+OS+IDZ+RPE).

The OCT segmentations were performed by the same experienced graders (ISZ and CSA), supervised by a third experienced grader (GMS). In cases of discordance, GMS reviewed the images and corrected the segmentation. After the image processing step, the thickness data of the retinal layers and choroid were recorded in the nine ETDRS regions, which was used to compute thickness in four regions: for the total macula (T), the central subfield (1 mm in diameter, C), and the inner (I) and outer (O) macular rings (with diameters of 3 and 6 mm, I and O, respectively; Figure 2).

Statistical analyses were carried out by SPSS Statistics for Windows, version 17.0 software. The Shapiro–Wilk test was used for normality testing; for normally distributed variables, parametric tests were used, and continuous data are reported as mean and standard deviation. The change of layer thickness from the baseline was calculated for each time point, and one-way analysis of variance test was performed for all variables, followed by Dunn’s post hoc test for the pairwise comparison between the thickness data at different time points and the baseline measurements. Pearson correlation was calculated to assess the correlation of retinal thickness changes, and multiple linear regression models with stepwise method were executed to assess which layers were changing together. The Pearson correlation for performance and layer thickness changes at the first and fifth minute was also
calculated to assess the effect of strain intensity and the observed retinal changes. Due to the high number of comparisons, the level of significance was set at .001; results with a $p$ value between .001 and .05 were interpreted as missed significance.

Results

Seventeen eyes of 17 senior elite athletes (11 men and 6 women) were enrolled in the study. A flowchart showing screened and excluded athletes is presented in Figure 3. No otherwise eligible participants needed to be excluded during the study.

Each participant declared to have performed regular intensive physical activity at least twice a week (mean 6.1 ± 2.8 hr/wk) in the past 10 years. The mean duration of doing sports was 48.2 ± 18.3 years. Most study athletes regularly took part at national, European, and world championships at a senior level, as well, with outstanding results. The demographic data of the study participants are shown in Table 1, and a detailed description of the participants is presented in Supplementary Table S1 (available online).

The baseline thickness data of the different retinal layers are shown in Table 2. There was no significant correlation between demographic characteristics and baseline layer thicknesses; height, weight, body mass index, and gender had no effect on baseline thickness data. Age showed a trend toward positive correlation (missed significant statistical values) with baseline RNFL in every region except in the central subfield, with ONL + IS and PRL in the center and OR and PRL in the inner ring.

In addition, a trend toward positive correlation with a missed significance was observed between BCVA and ONL + IS and also PRL in the center. Performance showed a tendency toward negative correlation with RNFL in the outer ring and ELZ + OS in every region except in the outer ring, whereas a trend toward negative correlation
Table 1  Descriptive Statistics of the Study Participants

<table>
<thead>
<tr>
<th></th>
<th>Senior athletes (N = 17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>67.9 (7.4)</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>11/6</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.7 (0.07)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72.5 (13.2)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.7 (3.3)</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>131.2 (17.1)</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>80.9 (12.8)</td>
</tr>
<tr>
<td>HR (1/min)</td>
<td>71.5 (18.5)</td>
</tr>
<tr>
<td>SE (D)</td>
<td>+0.6 (1.1)</td>
</tr>
<tr>
<td>BCVA (logMAR)</td>
<td>−0.1 (0.1)</td>
</tr>
</tbody>
</table>

Note. All data are presented as mean (SD). BMI = body mass index; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; SE = spherical equivalent; BCVA = best corrected visual acuity; logMAR = logarithm of the minimum angle of resolution.

Table 2  Baseline Layer Thickness Data of the Senior Elite Athletes for Total Thickness of the Entire Macula (T), in the Central Subfield (C), Inner Ring (I), and Outer Ring (O)

<table>
<thead>
<tr>
<th>Layers</th>
<th>T</th>
<th>C</th>
<th>I</th>
<th>O</th>
</tr>
</thead>
<tbody>
<tr>
<td>RNFL</td>
<td>37.1 (3.4)</td>
<td>15.3 (1.0)</td>
<td>26.6 (2.3)</td>
<td>41.0 (3.9)</td>
</tr>
<tr>
<td>GCL + IPL</td>
<td>71.3 (5.4)</td>
<td>40.6 (11.8)</td>
<td>93.2 (7.2)</td>
<td>65.9 (5.1)</td>
</tr>
<tr>
<td>INL</td>
<td>33.0 (2.2)</td>
<td>22.4 (5.5)</td>
<td>41.4 (3.6)</td>
<td>31.0 (2.2)</td>
</tr>
<tr>
<td>OPL</td>
<td>25.4 (2.2)</td>
<td>18.9 (2.9)</td>
<td>26.4 (2.1)</td>
<td>25.3 (2.6)</td>
</tr>
<tr>
<td>ONL + IS</td>
<td>75.3 (7.9)</td>
<td>118.3</td>
<td>88.7 (9.4)</td>
<td>69.7 (7.8)</td>
</tr>
<tr>
<td>ELZ + IS</td>
<td>34.7 (2.1)</td>
<td>33.2 (2.5)</td>
<td>33.5 (2.6)</td>
<td>35.2 (2.0)</td>
</tr>
<tr>
<td>IDZ + RPE</td>
<td>32.0 (3.1)</td>
<td>40.0 (4.4)</td>
<td>34.9 (3.7)</td>
<td>30.9 (2.9)</td>
</tr>
<tr>
<td>Composite layers</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GCC</td>
<td>108.4 (7.1)</td>
<td>55.9 (12.6)</td>
<td>119.9 (8.5)</td>
<td>107.0 (7.0)</td>
</tr>
<tr>
<td>IR</td>
<td>141.5 (8.4)</td>
<td>78.4 (17.2)</td>
<td>161.2 (11.0)</td>
<td>138.0 (8.2)</td>
</tr>
<tr>
<td>PRL</td>
<td>110.0 (7.7)</td>
<td>151.4 (11.9)</td>
<td>122.2 (9.1)</td>
<td>104.9 (7.5)</td>
</tr>
<tr>
<td>OR</td>
<td>167.4 (9.0)</td>
<td>210.4 (12.4)</td>
<td>183.6 (10.4)</td>
<td>161.1 (9.1)</td>
</tr>
<tr>
<td>TR</td>
<td>308.9 (14.1)</td>
<td>288.7 (23.3)</td>
<td>344.8 (16.0)</td>
<td>299.0</td>
</tr>
<tr>
<td>Choroid</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CRC</td>
<td>252.0 (77.0)</td>
<td>279.0 (87.4)</td>
<td>272.0 (86.5)</td>
<td>245.1 (74.2)</td>
</tr>
</tbody>
</table>

Note. The data are shown as mean (SD). RNFL = retinal nerve fiber layer; GCL + IPL = complex layer of the ganglion cell and inner plexiform layer; INL = inner nuclear layer; OPL = the outer plexiform layer; ONL + IS = outer nuclear layer, external limiting membrane and the myoid zone of the photoreceptors; ELZ + OS = ellipsoid zone and the outer segment of the photoreceptors; IDZ + RPE = interdigitation zone, the retinal pigment epithelium, and Bruch’s membrane; GCC = ganglion cell complex; IR = inner retina; PRL = photoreceptor layer; OR = outer retina; CRC = choroid.

was observed with the INL in the inner ring. Maximal HR seemed to negatively correlate with PRL in every region except in the inner ring, similarly to the ONL + IS in the outer ring and the total macula.

BP had a trend toward positive correlation with the OPL in the outer ring and the total macula. These data are presented in Table 3.

A significant thinning of the total retina was observed 1 min postexercise (−1.6 ± 1.1 μm, p < .001), which was followed by a trend toward thickening at 5 min with missed significance (1.5 ± 1.0 μm, p < .05). By 30 min, the total retinal thickness approached the baseline. These changes were significant in the inner and outer ring as well with thinning at 1 min (−1.7 ± 1.2 μm, p = .001, −1.5 ± 1.2 μm, p < .001) followed by a missed significant thickening at 5 min (+1.1 ± 1.1 μm, p < .05, +1.0 ± 1.1 μm, p < .05, respectively). In the central subfield, the thinning was only significant at 5 min (1.5 ± 1.1 μm, p < .05; Figure 4).

This trend was present in all single retinal layers, but significant changes were detected only in the GCL + IPL layer complex at 1 min with missed significance at 5 min (−0.4 ± 0.4 μm, p < .001 and +0.2 ± 0.1 μm, p < .05) in the outer ring (−0.4 ± 0.5 μm, p < .01). See Supplementary Figure S1 (available online).

Among the composite layers, no significant changes were observed. The trend observed in the case of the total retina has also appeared in the composite layers; however, the changes were not statistically significant (Supplementary Figure S2; data are shown in Supplementary Table S3 [available online]).

There was no significant change in choroidal thickness; however, we could detect a tendency toward thinning at 1 and 5 min postexercise, which was followed by a thinning at 15 min and then a slight thinning, which continued after 60 min. This trend was observed in every region but the central subfield where, after a thickening at 1 min and thinning at 5 min, an oversensitized thickening was detected at 15 min, which was followed by a slow decrease (Supplementary Figure S3 [available online]). The absolute changes in choroidal thickness did not show any correlation with the thickness changes of the intraretinal layers.

No significant correlation was observed between the layer thickness changes from baseline to Minute 1 and Minute 5 measurements and the power-to-weight ratio. Missed significant correlations were found at Minute 1 for the RNFL in the outer ring and INL in the inner ring (r = −.527, p = .032 and r = .556 and p = .025) and at Minute 5 for the INL and OPL in the inner ring and ELZ + IS in the central subfield (r = −.596, p = .012; r = −.562, p = .019; r = −.580, p = .015, respectively).

Discussion

With increasing age, the prevalence of multiple ocular pathologies, such as AMD, glaucoma, cataract, or diabetic retinopathy, rises significantly as many of these diseases are influenced by vascular alterations and CV risk factors (Ong et al., 2018). Although previous data suggest the beneficial effect of chronic physical exercise in reducing the incidence and progression of these pathologies (Chong Seong et al., 2019; Knudtson et al., 2006; Meier et al., 2018; Ong et al., 2018), there is little known about the acute retinal structural effects of physical strain in older adults. Therefore, in the present study, we assessed the chorioretinal morphological alterations due to a short, intense, age-matched vita maxima physical exercise in physically active and well-trained older adults.

Our results point toward an acute response in the retina with thinning at 1 min, followed by an immediate refractory thickening of the total retina by 5 min postexercise, which then gradually returns to baseline. Although this trend was also present in the
single nuclear layers of the retina, mostly in the outer retina, these changes did not reach statistical significance at the level of segmentation results. Nonetheless, these changes resemble the observations made in young adults undergoing vita maxima strain wherein the same acute significant thinning was described, followed by significant thickening of the total retina and the nuclear layers (Szalai et al., 2022).

In the young adult population, it has been postulated that the reason behind the observations might be twofold. First, the systolic increase in BP may trigger vasoconstriction in the retinal arteriolar index due to the increased thickness of the arterial wall has been previously suggested to lead to a decrease in retinal vascular density along with a decreased vascular density in the deep retinal layers of myopic eyes (Alnawaiseh et al., 2017), and may then result in refractory thickening. It is also known from experimental results in mice that hypoxia causes a shortening of the flexible nuclear layers. Vera et al. (2018) have described an IOP peak acutely after exercise lasting at least 30 min. According to Sayin et al. (2015), subfoveal choroidal thickness for the shortening of the outer retinal layers, although such an acute change at the metabolic level would rather be unlikely (Ebner et al., 2021). Another possible mechanism could be the acute rise in IOP following physical exercise that, then, leads to a mechanical compression of the outer retina and the otherwise more flexible nuclear layers. Vera et al. (2018) have described an IOP peak acutely after physical strain that returned back to normal in trained subjects after 5 min and took somewhat longer in untrained subjects.

The reason behind the somewhat different acute response to heavy physical strain between junior and senior athletes could be manyfold. First, a slightly different strain methodology was used in the two studies, using the Maffetone formula of 180/min–age for maximal HR in the older adults due to CV safety. This, in turn, might have influenced the power-to-weight ratio values, which were markedly higher in the young adults, resulting in less sympathetic response to the exercise in the senior athletes. Second, in case the acute changes in IOP might play a role in the acute thinning observed at 1 min (either per se or by complementing the vasogenic response), it might be postulated that acute exercise leads to less IOP increase in the senior cohort. However, the elevation of BP during exercise in the elite senior athletes was very similar to that in young athletes due to the controlled conditions in the senior cohort to avoid CV complications (Szalai et al., 2022).

A third possibility for the differences between the retinal response in our senior cohort compared with our previously reported data in young adults could be the age-related changes in retinal microvascular dynamics. An increased augmentation index due to the increased thickness of the arterial wall has been previously suggested to lead to a decrease in retinal vascular responses (Kotliar et al., 2008; Seshadri et al., 2016). In turn, increased aortic wall thickness and stiffness has been reported to result in a more pronounced increase in BP after isometric challenge in older people compared with young adults (Hartog et al., 2018). Besides this, the vascular constriction and the amplitude of the arterial dilatation decreased in healthy older adults in the retina after flicker light triggering compared with younger or middle-aged people (Seshadri et al., 2016).

Similar to the findings in young adults, we could not observe any correlation between retinal and choroidal thickness changes postexercise; also, there was no obvious trend in choroidal changes in this period due to the large deviation of the measurements. Some studies have found a significant decrease in macular and optic nerve head flow density along with a decreased vascular density in the deep retinal layers of myopic eyes (Alnawaiseh et al., 2017), and increased vascular density was detected following 30 min of rest after physical exercise in the whole retina in children with myopia and emmetropia as well (Li et al., 2021). These observations could also explain the trend toward an acute thinning of the retina observed postexercise in our cohort. However, choroidal response to physical strain is controversial in the light of previous studies. In children, Li et al. (2021) found choroidal thinning after exercise lasting at least 30 min. According to Sayin et al. (2015), subfoveal choroidal

### Table 3: Correlations Between Demographic Characteristics and Baseline Layer Thickness Values

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>BCVA</th>
<th>PWR</th>
<th>SBP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$r$</td>
<td>$p$</td>
<td>$r$</td>
<td>$p$</td>
</tr>
<tr>
<td>RNFL_T</td>
<td>.497</td>
<td>.042*</td>
<td>.305</td>
<td>.233</td>
</tr>
<tr>
<td>RNFL_I</td>
<td>.496</td>
<td>.043*</td>
<td>.341</td>
<td>.180</td>
</tr>
<tr>
<td>RNFL_O</td>
<td>.485</td>
<td>.048*</td>
<td>.291</td>
<td>.258</td>
</tr>
<tr>
<td>INL_I</td>
<td>-.318</td>
<td>.213</td>
<td>-.139</td>
<td>.594</td>
</tr>
<tr>
<td>OPL_T</td>
<td>-.026</td>
<td>.922</td>
<td>.072</td>
<td>.784</td>
</tr>
<tr>
<td>OPL_O</td>
<td>-.073</td>
<td>.780</td>
<td>.052</td>
<td>.842</td>
</tr>
<tr>
<td>ONL + IS_C</td>
<td>.541</td>
<td>.025*</td>
<td>.495</td>
<td>.043*</td>
</tr>
<tr>
<td>ELZ + OS_T</td>
<td>.096</td>
<td>.713</td>
<td>.280</td>
<td>.276</td>
</tr>
<tr>
<td>ELZ + OS_C</td>
<td>.068</td>
<td>.795</td>
<td>.074</td>
<td>.779</td>
</tr>
<tr>
<td>ELZ + OS_I</td>
<td>.182</td>
<td>.484</td>
<td>.249</td>
<td>.335</td>
</tr>
<tr>
<td>OR_I</td>
<td>.510</td>
<td>.037*</td>
<td>.405</td>
<td>.107</td>
</tr>
<tr>
<td>PRL_C</td>
<td>.561</td>
<td>.019*</td>
<td>.516</td>
<td>.034*</td>
</tr>
<tr>
<td>PRL_I</td>
<td>.523</td>
<td>.031*</td>
<td>.412</td>
<td>.100</td>
</tr>
</tbody>
</table>

Note. The Pearson correlation coefficients with $p$ values below are shown in italic and marked with * in the case of a missed significant correlation. Only layers with at least one missed significant correlation are shown. (For the abbreviation of the layers see Figure 1.) BCVA = best corrected visual acuity (Snellen); PWR = power to weight ratio (W/kg); SBP = systolic blood pressure (mmHg).
Figure 4 — Changes of total retinal thickness of senior elite athletes over time following cycle ergometer strain. Data are shown in the total macular area (T), the central subfield (C), and the inner (I) and outer ring (O). For mean and SD values, see the Supplementary Table S2 (available online). *p < .05 (missed significance). **p < .001 (significant).
thickness increased 5 min after a 10-min low-impact, moderate-intensity exercise. In the study by Alwassia et al. (2013) involving 60-year-old healthy adults, only the systolic BP increased after physical exercise with no changes in choroidal thickness on spectral-domain OCT. Similarly, Kinoshita et al. (2016) found no alterations in the central choroidal thickness and total cross sectional choroidal area immediately and 10 min after a mild dynamic exercise in healthy adults (mean age: 39.3 ± 9.6 years) despite the increased BP, HR, and mean ocular perfusion pressure.

When comparing previous studies with our work, three important differences need to be emphasized beyond the similarity of the methods. First, we applied an age-matched vita maxima-type exercise in our cohort wherein the intensity of the exercise was gradually increased until exhaustion or reaching the maximum physiological age-related HR or a peak in systolic BP (exceeding 180 mmHg), which is a particularly intense load. Still, our older adult subjects were rather submaximally strained for the sake of CV safety. Second, the thickness of the choroid was measured not only at a single given points but also for the whole volume, which provides a more precise analysis. Third, our senior athletes had been doing sports on a regular basis, which may have a strong effect on the microvasculature of the posterior pole in terms of having a trained CV system and, thus, a “trained eye” (Szalai et al., 2020).

The positive chronic effect of physical exercise on the eye and, in particular, the retina has been reported previously in several aspects. In mice, treadmill exercise could prevent photoreceptor degeneration due to light-induced phototoxicity and even in models of hereditary photoreceptor dystrophies (Hanif et al., 2015; Lawson et al., 2014; Zhang et al., 2019). As a possible reason behind this protective effect in these animal studies, the production of brain-derived neurotrophic factor has been postulated. In humans, the neuroprotective effect of exercise (on cognitive and motor functions) has been assessed in several studies. In healthy young and older individuals, aerobic physical activity improved cognitive development, academic achievement, and even psychosocial functioning (Lees & Hopkins, 2013; Tomporowski et al., 2008). In people with schizophrenia, aerobic exercise slowed down the physical function decline and improved the cognitive function (Massa et al., 2020).

Physical activity also had a benefit on motor and cognitive functions of people with neurologic diseases (such as Parkinson’s disease, Alzheimer’s disease, and stroke; Cruise et al., 2011; Filippini et al., 2010; Quaney et al., 2009; Vreugdenhil et al., 2012). Aerobic exercise is also suggested to be associated with better self-reported visual function and vision-related quality-of-life, working neuroprotectively in patients with retinitis (Levinson et al., 2017).

Epidemiological studies also support the positive influence of regular physical exercise on the prevalence of age-related eye diseases. Longitudinal studies in runners have shown a decreased incidence in cataract and AMD development along with reduced glaucoma prevalence (Williams, 2009a, 2009b, 2009c). One possible cause of these observations could be a positive effect on systemic risk factors (such as age-related weight gain, high plasma triglyceride concentrations, risk for diabetes, and hypertension) due to the vigorous exercise. However, in the case of AMD and glaucoma, the risk reduction was independent of cardiorespiratory fitness and adiposity, hypertension, or other systemic factors, whereas improved insulin resistance and lower BP due to vigorous physical activity and better cardiorespiratory fitness may, in turn, lead to lower IOP. Interestingly, in a South Korean national survey the prevalence of glaucoma was higher among participants with daily vigorous exercise (Lin et al., 2017). The difference in these observations could lie in the different physiological backgrounds of physical exercise, namely the longtime nature of marathon running versus a relatively short, vigorous exercise.

It is important to note the limitations of our work. First, we aimed to perform a pilot study to observe chorioretinal changes in senior elite athletes and to gain data that can help the planning of future studies. Second, we recruited a special cohort of senior elite athletes who were regularly controlled at the Department of Health Sciences and Sports Medicine at the University of Physical Education in Budapest, Hungary. It may well be that our participants, along with having a trained CV system and only minimal comorbidities, had a “trained eye” that reacts differently to acute physical strain compared with an age-related control group with obviously more severe comorbidities. Third, we did not measure axial length due to technical reasons, although it might have influenced the segmentation values (Szisgy et al., 2015). To avoid this, we assessed the changes of layer thickness values compared with baseline instead of comparing means. Finally, as previously mentioned, IOP might have influenced our results (Vera et al., 2018); however, it would have been technically challenging to perform applanation or noncontact tonometry parallel with the OCT examination during and after the physical strain, especially in the first 5 min of the recovery, and rebound tonometry was not available in our lab. Finally, caffeine consumption and sleep deprivation may possibly alter eye physiology. We explicitly asked study participants to refrain from caffeine consumption on the study day. Older adults are known to have a reduced sleep need; most of our study participants were not actively working anymore, and as such, one would expect that sleep deprivation did not pose any bias to our measurements.

We believe our work sheds light on the acute retinal structural changes following physical exercise in well-trained people over 50 years of age, although further studies are warranted in ordinary, nontrained older adults as well, with various, age-related ocular comorbidities. This, along with our observations in young adults, could pave the way to better understand the pathophysiology of retinal vascular diseases that are gaining increasing importance in our aging societies. There is an increasing body of evidence that regular physical activity may protect against these diseases; however, the exact mechanism is not yet known. Our observations and the applied methodology could help to further assess these pathologies and may also catalyze the implementation of guidelines for a sight-preserving active lifestyle.

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