ophthalmological examination, and spectral-domain optical coherence tomography (OCT; Spectralis OCT, software v. 4.0, Heidelberg Engineering, Dossenheim, Germany) imaging to measure RNFL thickness. Subjects were excluded from the study if any of the following were present: glaucoma, optic neuropathy, high ametropia (refractive error spherical equivalent more severe than ±5 dioptres), history of ocular or neurological trauma, or other relevant retinal and/or optic nerve disease.

Fifty-six subjects with treated MS and 35 healthy subjects were included. Mean global (MS: 89.6 ± 15.4 µm, control: 104.3 ± 9.1 µm; p < 0.001) and sectorial RNFL thicknesses were significantly less in the MS group than in the control group (Table 1). Global RNFL thickness was thinnest in MS subjects with a history of ON (79.8 ± 15.9 µm), followed by MS subjects without a history of ON (93.6 ± 13.3 µm), and thickest in the control group (104.3 ± 9.1 µm; all p < 0.001). Additionally, the Spearman rank correlation coefficient (rs) between the number of ON episodes and RNFL thickness was −0.41 in the MS group (p < 0.001). Therefore, MS subjects that had more ON episodes had a thinner RNFL thickness. The area under the receiver operating characteristic curve (AUROC) for global RNFL measurements was 0.83 (95% confidence interval [CI]: 0.66–0.94) for discriminating between healthy subjects and those with MS. Sectorial RNFL thickness measurements had the highest AUROC (0.83, 95% CI: 0.67–0.93), and subsequently the best accuracy, in the superior temporal sector. That means that the superior temporal parapapillary sector is the most affected in MS.

Interestingly, subjects with a higher number of ON episodes had larger RNFL changes than subjects with a lower number of ON episodes. This finding indicates that serial OCT monitoring of patients with MS may provide useful information on disease status, disease activity and treatment efficacy. However, caution should be used to not overlook RNFL changes in eyes classified as ‘within normal limits’, because the software database is made for glaucoma, not for demyelinating disease. Serial testing is always helpful for comparison to baseline values obtained at the beginning of a disease process.

In conclusion, MS subjects without a history of ON had a thinner RNFL than normal subjects. Additionally, RNFL thickness was negatively correlated with the number of prior ON episodes, indicating a larger amount of RNFL damage. Therefore, we recommend that all patients with MS, and not just those with a history of ON, undergo regular RNFL thickness measurement with OCT during the diagnostic process and follow-up.

### Table 1. Global and sectorial RNFL thickness in the control, MS without ON and MS with previous ON groups.

<table>
<thead>
<tr>
<th>Region</th>
<th>Control Mean (SD)</th>
<th>MS – without ON Mean (SD)</th>
<th>MS – with ON Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global</td>
<td>104.3 (9.1)</td>
<td>93.6 (13.3)*</td>
<td>79.8 (15.9)*</td>
</tr>
<tr>
<td>Temporal</td>
<td>76.0 (11.5)</td>
<td>63.7 (14.2)*</td>
<td>48.9 (14.8)*</td>
</tr>
<tr>
<td>Superior temporal</td>
<td>141.6 (19.2)</td>
<td>126.7 (24.9)*</td>
<td>113.0 (25.1)*</td>
</tr>
<tr>
<td>Inferior temporal</td>
<td>146.5 (17.6)</td>
<td>132.3 (23.3)*</td>
<td>107.3 (30.2)*</td>
</tr>
<tr>
<td>Nasal</td>
<td>81.1 (12.7)</td>
<td>75.2 (14.7)</td>
<td>65.2 (16.9)*</td>
</tr>
<tr>
<td>Superior nasal</td>
<td>112.9 (19.4)</td>
<td>99.5 (25.4)*</td>
<td>88.4 (24.0)*</td>
</tr>
<tr>
<td>Inferior nasal</td>
<td>118.6 (20.7)</td>
<td>108.6 (25.5)</td>
<td>98.9 (24.6)*</td>
</tr>
</tbody>
</table>

MS = multiple sclerosis; ON = optic neuritis; RNFL = retinal nerve fibre layer; SD = standard deviation.

* Statistically significant difference (p value < 0.01; adjusted for age) when compared to the control group.

### References


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### Genetic influence on contrast sensitivity in young adults

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riva License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.
Continuous traits were normalized using the inverse normal transformation.

The mean (SD) log contrast sensitivity across all twin pairs was 1.80 (0.06) and the mean (SD) visual acuity was −0.14 (0.12) logMAR. There were no significant differences between the monozygotic and dizygotic groups in contrast sensitivity, visual acuity, age (all p > 0.58), or the proportion of males/females (zip = 1.72, p = 0.19). Contrast sensitivity was moderately heritable, with additive genetic effects explaining 27% of the phenotypic variance (h² = 0.27, SE = 0.07, p = 1.2 × 10⁻⁴), which is consistent with the prior estimates for peak contrast sensitivity in middle-aged men.

The comparably moderate heritability of contrast sensitivity in early and middle adulthood suggests a strong influence of nongenetic, non-ageing-related factors. While these influences may partly reflect measurement error, variations in cognitive ability and/or task engagement, a large proportion likely involves individual-specific environmental experiences during childhood and adolescence (Cronin-Golomb et al. 2007; Baker 2013; Bartholomew et al. 2016). Identifying these experiences is an important direction of future research, as they may be altered to improve visual function in adulthood.

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References


Persisting diplopia after periocular injection of parallel imported Kenalog® (triamcinolone acetonide)

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Editor,

We report four cases with persisting diplopia as an unusual complication after periocular steroid injections with Kenalog Orifarm.

Case 1 A 20-year-old man with bilateral idiopathic pars planitis complicated by cystoid macular oedema (CME) in his left eye. Immediately after a periocular injection with Kenalog Orifarm, the patient developed painless diplopia, which initially was thought to be caused by the