

# VISUAL EVOKED POTENTIALS AND OPTICAL COHERENCE TOMOGRAPHY TO DETECT OPTIC NEURITIS IN C57BL/6

*Silvia Marenna<sup>1</sup>, Su-Chun Huang<sup>1</sup>, Valerio Castoldi<sup>1</sup>, Raffaele d'Isa<sup>1</sup>, Giancarlo Comi<sup>1</sup>, Letizia Leocani<sup>1</sup>*

**<sup>1</sup> INSPE-Institute of Experimental Neurology, University Hospital San Raffaele, Milan**

Experimental autoimmune encephalomyelitis (EAE) is induced through myelin oligodendrocyte glycoprotein (MOG) injection. C57BL/6 mice develop chronic EAE, a common disease model of multiple sclerosis (MS). This animal model permits to study optic neuritis, an acute inflammatory disorder that causes demyelination of the optic nerve, thinning of the retinal nerve fiber layer (RNFL), and death of retinal ganglion cells (RGCs). We aimed at testing the usefulness of non-invasive visual evoked potential (VEP) and optical coherence tomography (OCT) in detecting optic nerve involvement in this model. Ten C57BL/6 were immunized, and VEPs were recorded before the day of immunization and at different time points until 37 dpi. Clinical score was measured daily and healthy control group (n=5) was monitored at the same time points.

In EAE, VEP latency was significantly increased at 11 dpi ( $p=0.026$ ) until 37 dpi ( $p<0.0001$ ), with partial recovery at 23 dpi ( $p=0.195$ ). Amplitude was significantly decreased only at 31 dpi ( $p=0.006$ ). In EAE, NGCC (neuronal ganglion cell complex) decreased significantly at the last time point compared to healthy ( $p=0.008$ ). VEP latency was significantly correlated with NGCC thickness (Pearsons'  $r=-0.661$ ;  $p=0.007$ ). VEP delay preceded clinical EAE motor symptoms (16dpi) and neuroaxonal retinal thinning at OCT. These findings suggests that VEPs can be used as an early biomarker of demyelination in EAE to test new remyelinating treatments, while OCT is suitable for monitoring subsequent neuroaxonal loss for testing neuroprotective strategies.