

MOG-Ab disease patients do not have damage outside of lesions and no increasing lesions burden over time, supporting the clinical observation of good recovery and no background progression

Lesions, brain volume and normal appearing white matter in demyelinating diseases

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BACKGROUND AND AIMS

Previous non-conventional imaging studies comparing Neuromyelitis Optica Spectrum disorder (NMOSD) and Multiple Sclerosis (MS), has yielded differing results, partly explained by cohort heterogeneity (i.e. antibodies status). No studies are available in MOG-Ab disease (MOGAD).

METHODS

Eighteen relapsing-remitting MS, 18 MOGAD, 19 NMOSD-AQP4 and 18 HC were included. We performed 3T structural-brain-MRI and multi-shell DWI. The brain volume was calculated using SIENAX and FA-voxel-wise statistical analysis was carried out using Tract-Based-Spatial-Statistic (TBSS), both part of FSL. Lesions were segmented manually on an axial-FLAIR.

Table 1: clinical and demographic characteristics of the enrolled participants

	MOGAD	MS	NMOSD-AQP4	HC
Patient, n	18	18	19	18
Mean age at onset \pm SD	41.7 \pm 11	44.3 \pm 6.4	55.6 \pm 13.2	38.9 \pm 13.4
Female, %	52	50	68.4	53
Median Disease duration (range)	2 (0-24)	11 (1-24)	11 (0-24)	NA
Median number of relapse (range)	2 (1-7)	2 (1-6)	2 (1-11)	NA
Median EDSS (range)	1.5 (0-7)	2.3 (0-6)	3 (0-7)	NA
Patients with brain lesions, (%)	70	100	100	NA

RESULTS

Figure 1: Pearson correlation between disease duration and number of lesions

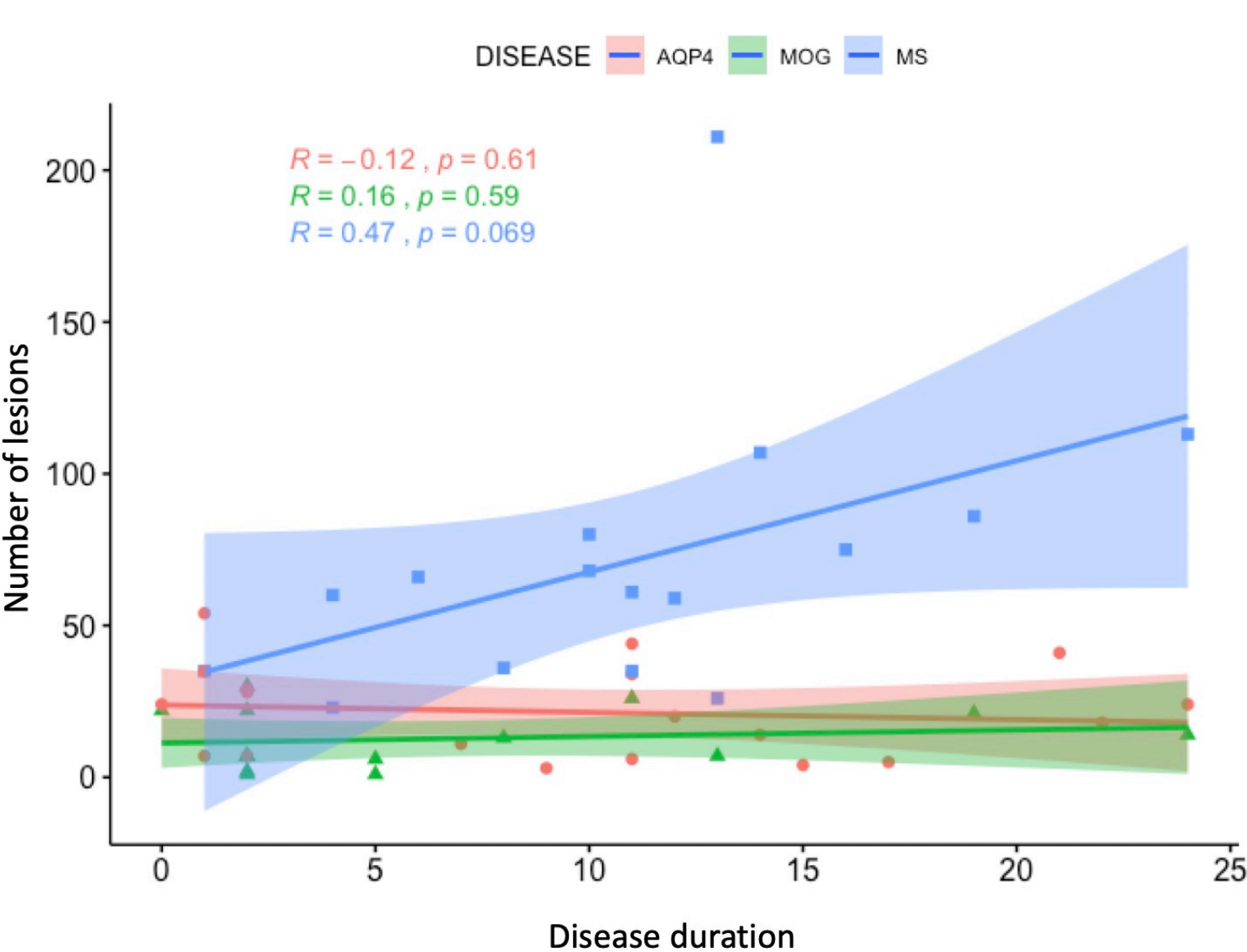


Figure 2: total brain volume

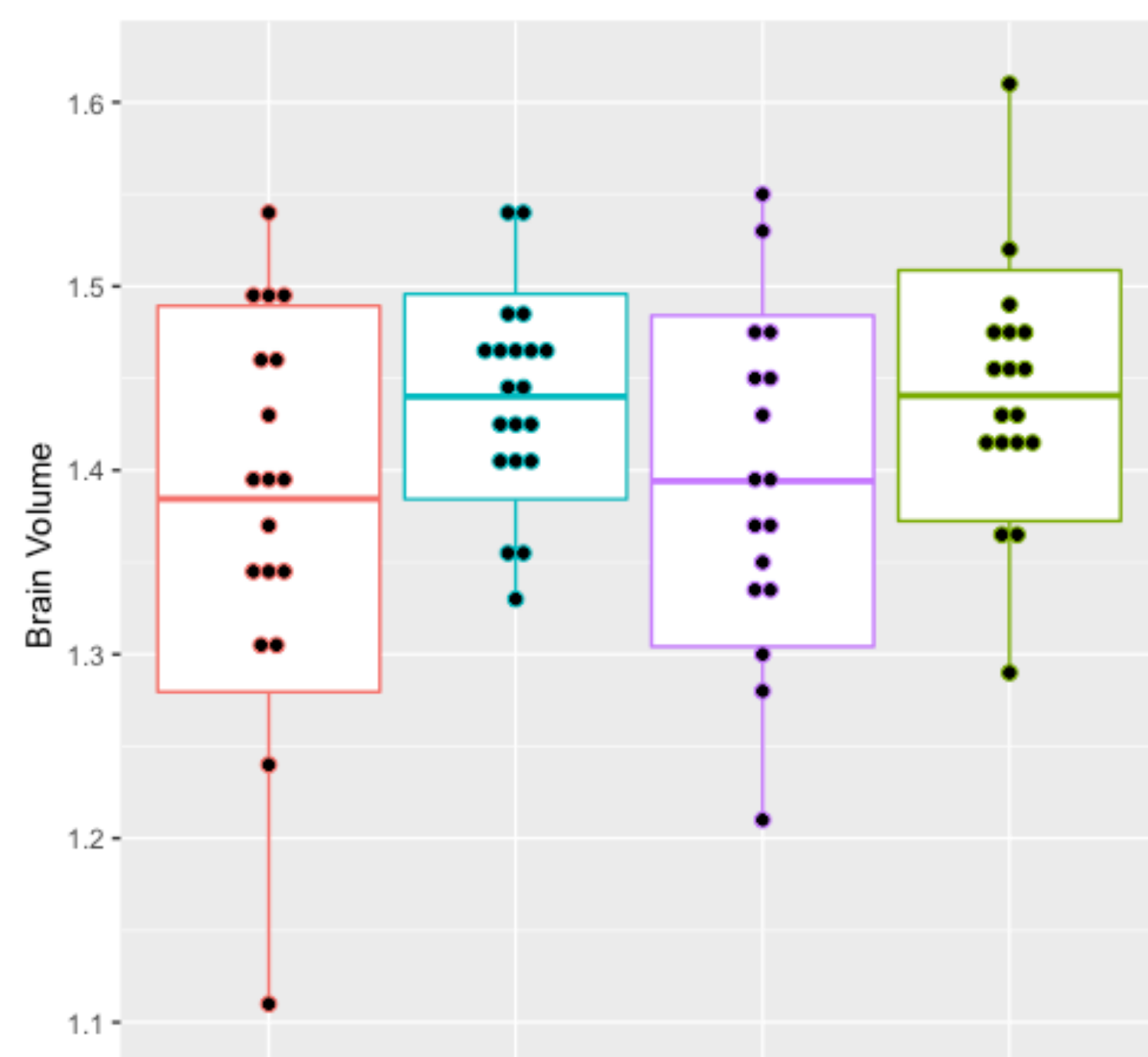
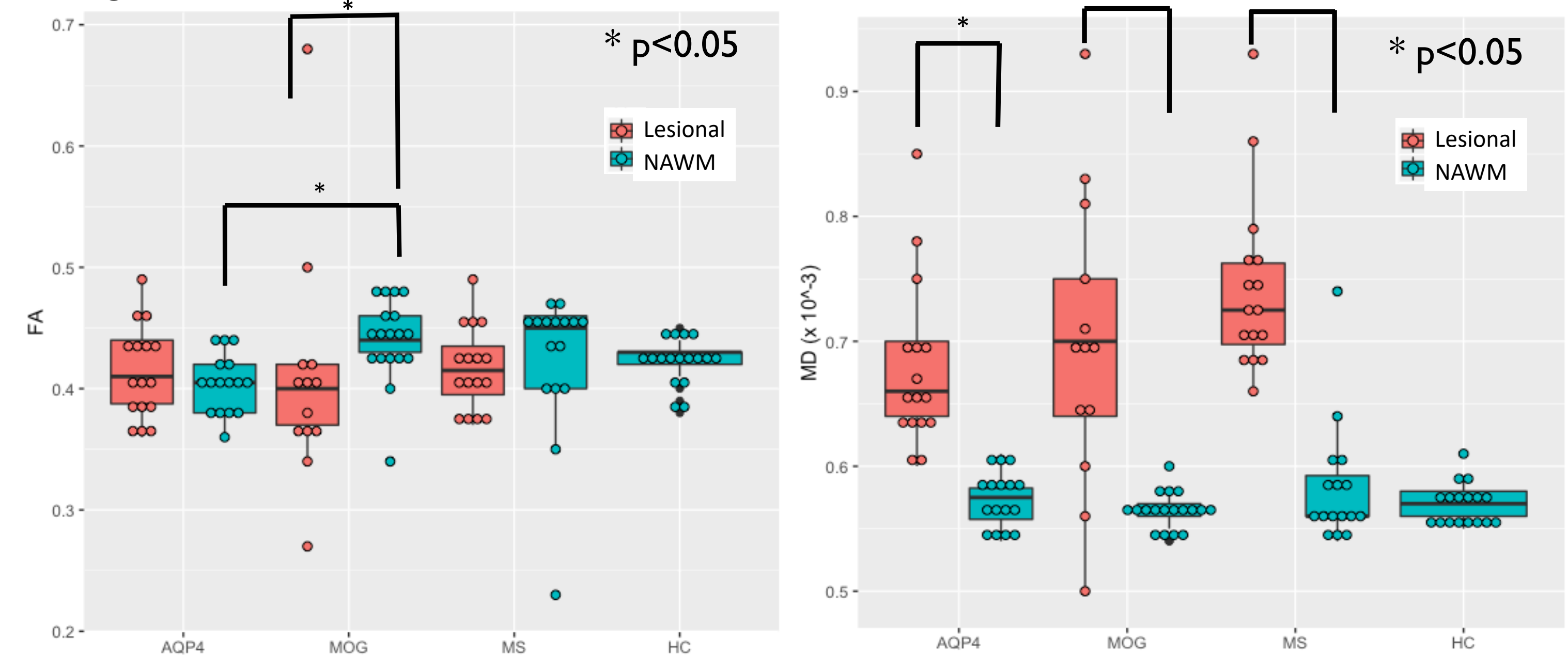
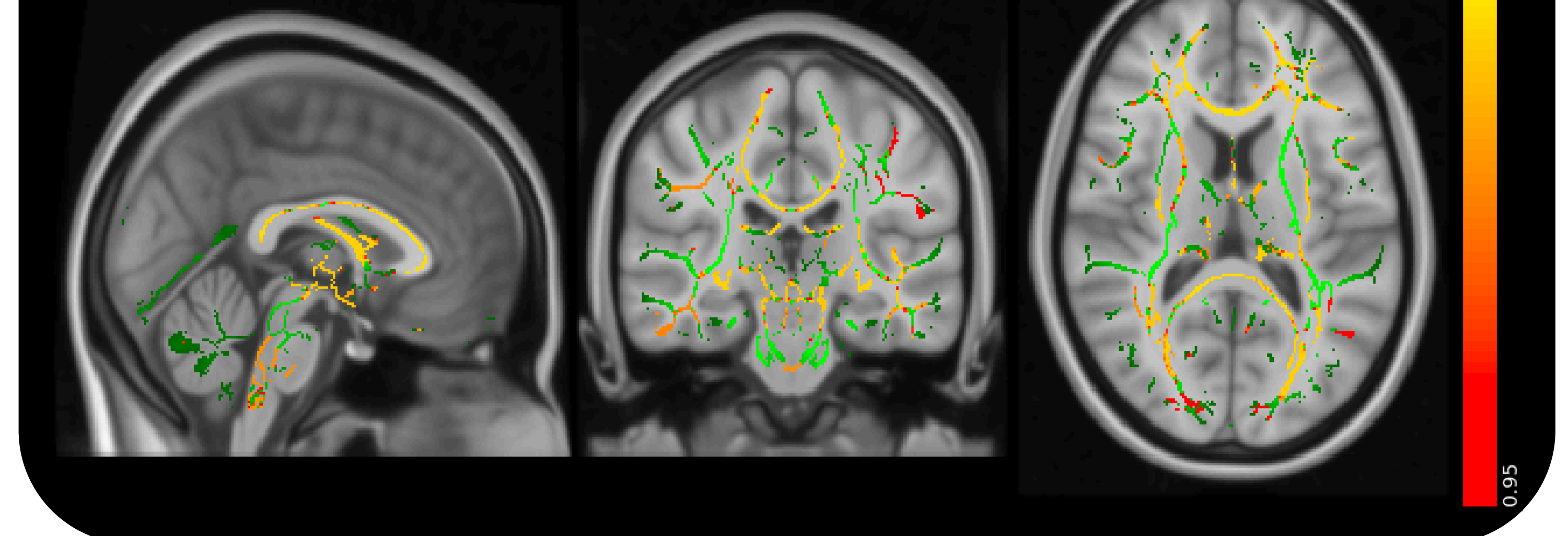


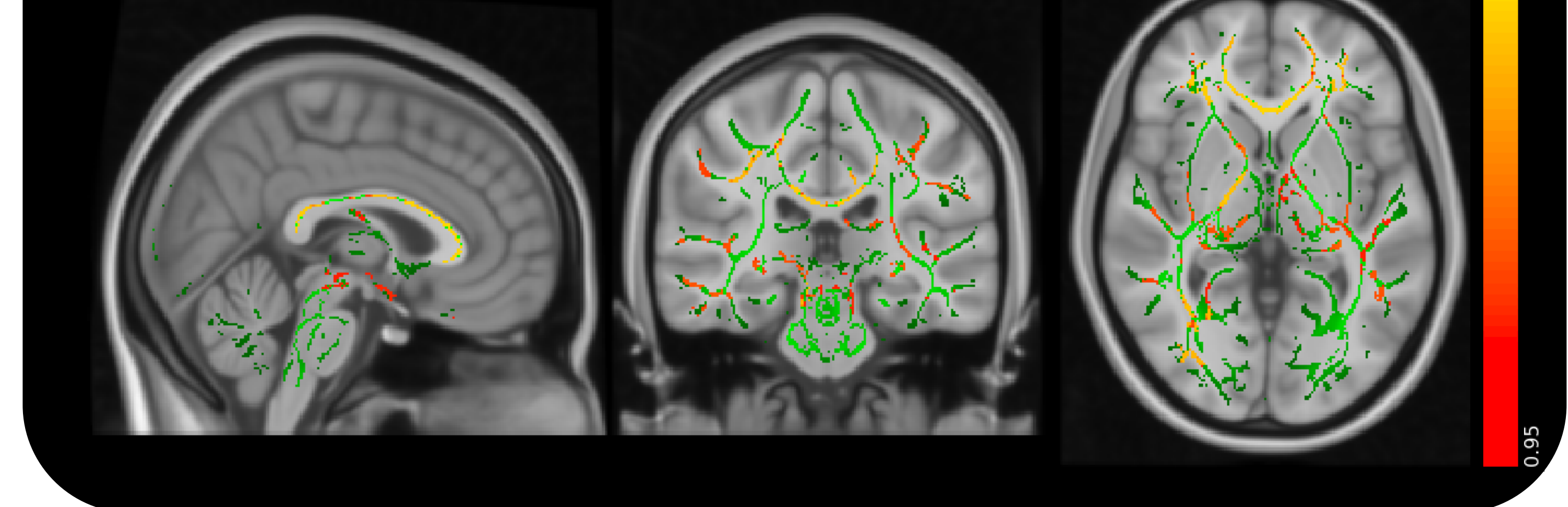
Figure 3 and 4: FA and MD in lesional and non-lesional areas



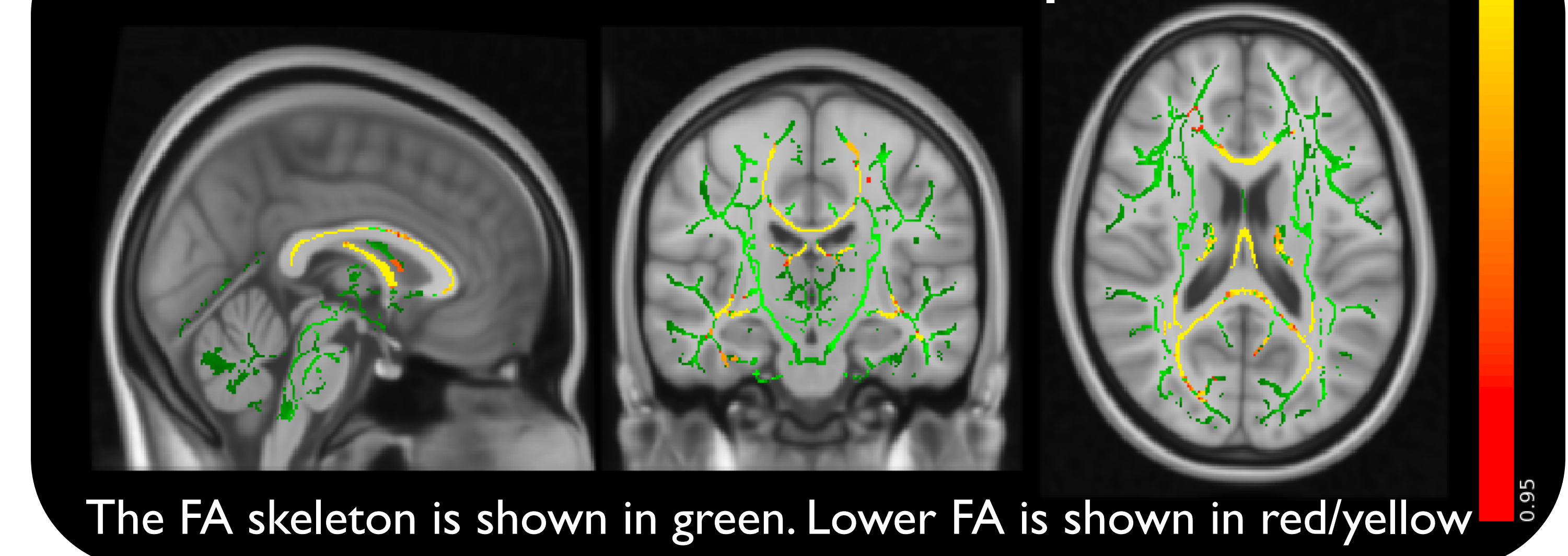
FA is reduced in NMOSD-AQP4 when compared to HC



FA is reduced in NMOSD-AQP4 when compared to MOGAD



FA is reduced in MS when compared to HC



No FA differences in MOGAD disease vs HC

CONCLUSIONS

MOGAD showed normal brain volume, NAWM integrity and fewer lesions, compared to other diseases. FA is able to discriminate between lesional and non-lesional area only in MOGAD. The lack of lesion-load increase with disease duration points towards antibody-mediated disease.

DISCLOSURES AND REFERENCE

S. Messina has received travel grants from Roche and honorarium for advisory work from Biogen. R. Mariano is undertaking graduate studies funded by the Rhodes Trust. M. Leite reported being involved in aquaporin 4 testing, receiving support from the National Health Service National Specialised Commissioning Group for Neuromyelitis Optica and the National Institute for Health Research Oxford Biomedical Research Centre, receiving speaking honoraria from Biogen Idec, and receiving travel grants from Novartis. A. Roca-Fernandez, A. Cavey, R. Everett, S. Reeve, M. Jenkinson have no disclosures. J. Palace is partly funded by highly specialised services to run a national congenital myasthenia and a neuromyelitis optica service. She has received support for scientific meetings and honorariums for advisory work from Merck Serono, Biogen Idec, Novartis, Teva, Chugai Pharma and Bayer Schering, Alexion, Roche, Genzyme, MedImmune, EuroImmun, MedDay, Abide and ARGENX, and grants from Merck Serono, Novartis, Biogen Idec, Teva, Abide and Bayer Schering. Her hospital trust received funds for her role as clinical lead for the RSS, and she has received grants from the MS society and Guthy Jackson Foundation. Reference: Juryńczyk M et al, Pract Neurol. 2019 Jun;19(3):187-195. Myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease: practical considerations

