Ultra-high field MRI: looking through the 'macroscope'

Iris D Kilsdonk,¹ Mike P Wattjes,¹ Jeroen JG Geurts²

The first publications about in vivo application of ultra-high field MRI in multiple sclerosis (MS) appeared in 2008. From then on, the number of studies performed at 7 Tesla (T) has rapidly increased. The main advantage of ultra-high field MRI is its higher signal-to-noise ratio, which can be used to increase spatial resolution greatly. This has led to improved detection of subtle MS pathology, particularly in clinically relevant regions like the grey matter (GM). Another advantage of 7 T MRI is its increased sensitivity to susceptibility effects. This has been used to image additional morphological characteristics of MS lesions.¹ For example, susceptibility-weighted MRI at 7 T has confirmed that MS white matter (WM) lesions are consistently oriented perivascularly, and has furthermore shown that hypointense rims may appear around lesions, and that some lesions appear hypointense, whereas others do not.

Filippi *et al*² provide an up-to-date overview of the major achievements of 7 T MRI so far. The authors discuss improved detection of WM lesions, their particular morphology, improved visibility of GM lesions and the exact location of these lesions, quantification of (novel) brain metabolites and sensitivity to iron accumulation. They conclude that ultra-high field MRI has improved our understanding of MS pathology, but that

implementation in clinical practice is unfortunately still a bridge too far. Its role in the diagnostic process and the monitoring of disease progression and treatment response has not yet been sufficiently established.

Also, valid interpretation of the ultra-high field images can be challenging. For example, hypointense (rims around) lesions on T2*-weighted images have been generally proclaimed to reflect iron-laden macrophages, but a recent report showed that this may not always be true.³ Hypointense rims around WM lesions did not disappear over time at 7 T, decreasing their potential as a method for lesion staging.⁴ Moreover, several studies have focused on finding the 7 T sequence that detects the highest number of cortical lesions. It was even suggested that T2*-weighted imaging at 7 T should be the new 'gold standard' for detection of cortical lesions in MS.⁵ However, when considering the set-up of these studies, at times simultaneously comparing different sequences and different field-strengths, it might be too early to conclusively recommend any of the sequences investigated thus far. Comparison of sequences at 7 T needs to be more systematic and precise in future studies, and results should ideally be compared with the real gold standard, that is, histopathology, at some point. Sensitivity and specificity of many (if not all) 7 T sequences presented have yet to be determined. At 1.5 T, only the tip of the proverbial iceberg is visible when it concerns GM lesions.⁶ How much more of the iceberg do we see at 7 T? And do cortical hyperintensities or hypointensities, as seen through the 7 T 'macroscope', really reflect demyelination under the microscope? This has to be investigated

carefully; with new fields and sequences, new artefacts arise.

These issues aside for now, we very much look forward to the further development of ultra-high field MRI in MS. Better detection of subtle and diffuse pathology and the discovery of new imaging features of MS are likely to further improve the way we look at this complex disease.

Acknowledgements The authors kindly thank Professor Frederik Barkhof for providing the metaphor 'macroscope' to reflect the detailed view 7 T MRI offers.

Contributors IDK, writing and revising manuscript; MPW, writing and revising manuscript; JJG, writing and revising manuscript.

Competing interests None.

Provenance and peer review Commissioned; internally peer reviewed.

To cite Kilsdonk ID, Wattjes MP, Geurts JJG. *J Neurol Neurosurg Psychiatry* 2014;**85**:4.

Received 20 May 2013

Accepted 23 May 2013

Published Online First 11 July 2013

► <http://dx.doi.org/10.1136/jnnp-2013-305246>

J Neurol Neurosurg Psychiatry 2014;**85**:4.
doi:10.1136/jnnp-2013-305601

REFERENCES

- 1 Kilsdonk ID, De Graaf WL, Barkhof F, *et al*. Inflammation high-field magnetic resonance imaging. *Neuroimaging Clin N Am* 2012;**22**:135–57.
- 2 Filippi F, Evangelou N, Kangarlu A, *et al*. *J Neurol Neurosurg Psychiatry* 2014;**85**:60–6.
- 3 Walsh AJ, Lebel RM, Eissa A, *et al*. Multiple sclerosis: validation of MR imaging for quantification and detection of iron. *Radiology* 2013;**267**:531–42.
- 4 Bian W, Harter K, Hammond-Rosenbluth KE, *et al*. A serial in vivo 7T magnetic resonance phase imaging study of white matter lesions in multiple sclerosis. *Mult Scler* 2013;**19**:69–75.
- 5 Nielsen AS, Kinkel RP, Tinelli E, *et al*. Focal cortical lesion detection in multiple sclerosis: 3 Tesla DIR versus 7 tesla FLASH-T2*. *J Magn Reson Imaging* 2012;**35**:537–42.
- 6 Seewann A, Vrenken H, Kooi E, *et al*. Imaging the tip of the iceberg: visualization of cortical lesions in multiple sclerosis. *Mult Scler* 2011;**17**:1202–10.

¹Department of Radiology, VU University Medical Center, Amsterdam, The Netherlands; ²Department of Anatomy and Neurosciences, VU University Medical Center, Amsterdam, The Netherlands

Correspondence to Dr Jeroen Geurts, Department of Anatomy and Neurosciences, VU University Medical Center, Amsterdam 1081 BT, The Netherlands; j.geurts@vumc.nl