

## MULTIPLE SCLEROSIS

# Blood-based biomarkers provide insight into progressive MS

According to two studies published recently in *Neurology*, biomarkers in blood could distinguish between the different clinical phenotypes of multiple sclerosis (MS), and elucidate the role of inflammation in progressive MS.

“Conversion from relapsing–remitting MS (RRMS) to secondary progressive MS (SPMS) occurs subtly and is difficult to define clinically,” explains Daniel Anthony. His research group used metabolomic profiling with high-resolution nuclear magnetic resonance (NMR) spectroscopy and multivariate statistical pattern recognition to identify conversion biomarkers in blood samples.

“We can diagnose MS with 100% sensitivity and predict progression from RRMS to SPMS with 90% sensitivity and 80% specificity,” Anthony summarizes. He contends that, in addition to the clinical benefit accrued from rapid and accurate staging, NMR-based metabolomics could aid the diagnosis of a broader spectrum of neurological diseases, including detection of brain metastasis.

Another study suggests that inflammation, particularly T helper cells that produce IL-17

(T<sub>H</sub>17), contributes to disease progression in SPMS. Benjamin Segal and co-workers analysed peripheral mononuclear cells that produce IL-17 and IFN- $\gamma$ , and proinflammatory factors in plasma from patients with RRMS, SPMS and healthy controls for 12 months. These measures were combined with serial brain MRI.

Patients with SPMS showed elevated levels of IL-17-induced chemokines, which activate and recruit innate monocytes and neutrophils. Furthermore, the increased expression of chemokines correlated with MRI lesion burden.

“The results could revise current concepts regarding the factors that drive tissue damage during SPMS, thereby redirecting strategies for biomarker discovery and drug development in progressive disease,” concludes Segal.

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**Original articles** Dickens, A. M. *et al.*  
A type 2 biomarker separates relapsing–remitting from secondary progressive multiple sclerosis. *Neurology* doi:10.1212/WNL.0000000000000905 | Huber, A. K. *et al.* Dysregulation of the IL-23/IL-17 axis and myeloid factors in secondary progressive MS. *Neurology* doi:10.1212/WNL.0000000000000908