Induction of COLEC12 expression on myelin-phagocytosing macrophages and in active MS lesions (ongoing).

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Research question and background

MS is an inflammatory, demyelinating disease of the CNS in which foamy macrophages, containing myelin degradation products, are abundantly found. Recent studies have described an altered phenotype of macrophages after myelin phagocytosis. However, how this phenotype influences lesion progression remains largely unclear.

Methods and tissues used

Flow cytometry was used to define the expression of COLEC12 in in vitro experiments. MS lesion tissue was used to assess the expression of COLEC12 in active MS lesions.

Results and conclusion

In our previous study, microarray analysis demonstrated that internalization of myelin by macrophages induces the expression of collectin sub-family member 12 (COLEC12) mRNA in macrophages. In this report we assessed the function of this receptor and determined its expression level in MS lesions. We show that COLEC12 is highly expressed in foamy-appearing perivascular macrophages and parenchymal astrocytes in active MS lesions. Furthermore, *in vitro* experiments revealed that myelin internalization upregulates the expression of COLEC12 in macrophages but not in microglia. However, COLEC12 did not facilitate myelin clearance by macrophages and microglia. Although the function of COLEC12 remains to be resolved, its increased expression in active MS lesions indicates that it may play a role in lesion progression or resolution in MS.