LPS-induced expression of the chemokine receptor CCR7 in microglia and EAE

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In the healthy CNS microglia appear as resting ramified cells that continously screen their environment. Whenever the CNS is challenged and/or damaged, microglia are activated by substances that are released during insults. Activated microglia rapidly change into amoeboid macrophage-like cells, which produce various mediators. In addition, activated microglia phagocytose dead or damaged cells and it has been suggested that microglia can also present antigens.
In the periphery, homing of antigen presenting cells (APCs) to the lymphnodes (LNs) is mediated by CCL21, a chemokine expressed in the LNs, and its receptor CCR7 present on mature APCs. The expression of CCR7 in APCs relates to their maturation stage and can, for instance, be triggered in dendritic cells by the bacterial cell wall component lipopolysaccharid (LPS).
In previous studies we demonstrated that unchallenged cultured microglia do not express CCR7. However, since professional APCs can be triggered to express CCR7 by LPS, we wondered wether microglia –as unprofessional APCs in the CNS- show a similar CCR7 induction response.
Therefore, we challenged microglia with LPS and investigated possible expression of CCR7. LPS induced a rapid increase in microglial CCR7 mRNA expression and functional responses to CCR7 ligands. Next, we studied CCR7 expression in experimental allergic encephalomyelitis (EAE), a mouse model for multiple sclerosis. Animals exhibiting clinical symptoms were found to express higher levels of CCR7 when compared to controls and the cellular source for this CCR7 expression are presumably microglia and infiltrating T cells. The results of these experiments suggest that under specific inflammatory conditions, microglia express CCR7, corroborating the idea that microglia can act as APCs.

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