Introduction

In multiple sclerosis, it has been suggested that low oxygen concentrations, local "hypoxia", may play a role in the pathogenesis of the disease 

Glas et al. hypothesized that chronic hypoxia delays MOG-specific Th17 responses and induces CXCL12 and CXCL13 in EAE. To test this hypothesis, the authors performed the following experiments:

1. Animals were immunized with MOG peptide to induce EAE.
2. Animals were exposed to chronic hypoxia (3% oxygen) for a period of time.
3. The expression of CXCL12 and CXCL13 was measured in the spinal cord sections.
4. The proliferative response of CD4+ T cells was assessed using CFSE dye dilution analysis.

Results:

- Chronic hypoxia delayed the development of EAE.
- MOG-specific Th17 responses were reduced in hypoxic conditions.
- CXCL12 and CXCL13 expression was upregulated in the spinal cord of hypoxic animals.
- CD4+ T cell proliferation was significantly reduced in hypoxic conditions.

Conclusion:

Chronic hypoxia delays MOG-specific Th17 responses and induces CXCL12 and CXCL13 in EAE, suggesting a novel mechanism for the modulation of immune responses in the context of hypoxia exposure.