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Infectious disease and oligodendrocyte death as triggers of anti-CNS autoimmunity

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Multiple sclerosis (MS) is a T cell-mediated demyelinating inflammatory disorder of the central nervous system (CNS). To understand the disease initiation phase we used different modelling approaches. As it was suggested that neurodegeneration independent from inflammation could be a disease trigger, release or exposure of antigens by dying oligodendrocytes (ODCs) may play a role. To test this we developed a mouse model (oDTR) that allows us to force ODCs into death by expression of a diphtheria toxin receptor (DTR) and injection of diphtheria toxin (DT). The resulting demyelinating disease was characterized by ataxia, tremor, development of a hunchback, and cachexia. Nevertheless, none of the sick animals showed spontaneous autoimmune attack against myelin, neither alone nor using different autoimmunity-supporting settings like depletion of regulatory T cellsor application of anti-myelin antibodies. It therefore seemed that certain signals were missing in this setup and we started studying danger signal pathways, such as toll-like receptors (TLRs). Recent studies from animal models and from human patients suggested that inappropriate activation of TLR pathways may lead to the initiationof autoimmune responses. We have been studying the role of TLRs for the initiation of anti-CNS autoimmunity in the mouse model of MS, experimental autoimmune encephalomyelitis (EAE). In light of the emerging evidence for the role of the microbiome in initiating and driving autoimmune disease, we consider our observations to be highly relevant for the understanding of how MS starts or is triggered.

Biography

Thorsten Buch has obtained his PhD at the University of Cologneinvestigating thymic tolerance mechanisms in the laboratory of Klaus Rajewsky. During his career he has als performed research at the University of California, Davis and the University of Zurich. He is now Professor for for Molecular Microbiology and Infection Immunology at the Technische Universität München, heading a research team investigating tolerance and tolerance break in mouse models. Further he heads the transcriptomics and sequencing as well as the transgenesis units.

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