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Novel aspects on the role of potential environmental risk factors in multiple sclerosis and schizophrenia: neuroimmunology meets psychoneuroimmunology

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The exact mechanisms underlying neuroinflammation and neuropathology in multiple sclerosis (MS) are still unknown, but 👃 susceptibility depends on a combination of genetic and environmental risk factors and their interactions. With little influence on genetic predisposition, the importance of modulating environmental risk factors is becoming an area of great interest. There is mounting evidence implicating both late Epstein-Barr virus (EBV) infection and hypovitaminosis-D as key environmental risk factors in MS. We have previously shown that active white matter lesions in the MS brain show signs of innate immune activation, and that latently EBV-infected cells can be found in these areas. We hypothesized that EBV-RNAs (EBERs) may promote an inflammatory milieu within the lesion. We propose that dysregulated EBV infection may be a potential risk factor and contribute to MS disease activity via the stimulation of innate immune responses by EBERs, and/or antigenic mimicry, cross-reactivity of cellular immune responses with "self" brain antigens or via the transactivation of endogenous retroviruses. Another potential environmental risk factor in MS is hypovitaminosis-D. Vitamin-D plays an important role not only in bone homeostasis but also in immunity and control of persistent infections. Hypovitaminosis-D, which is a hallmark of MS cohorts, has been associated with disease activity in MS. I will discuss data, which highlights interdependence between EBV- and vitamin-D status in MS. Several vitamin-D supplementation trials are currently underway to test the effect of vitamin-D supplementation on disability progression in MS. In the final part of my presentation I will focus on psychoneuroimmunology, which studies the interactions between immunity/inflammation and mood, cognition and behaviour. I will discuss novel findings on the role of inflammation and potential environmental risk factors in schizophrenia e.g. hypovitaminosis-D. We propose that an MS-like inflammatory signature may be present in schizophrenia. This area warrants further study as it may highlight novel prevention or treatment strategies.

## **Biography**

Ute-Christiane Meier completed her PhD at the University of Oxford, where she worked on the cytotoxic T-cell control of HIV infection. She continued her studies on persistent virus infections and immunotherapeutic cancer vaccine-strategies within Oxford University, the Edward Jenner Institute and British Biotech in Oxford. She started working in Neuroimmunology in 2007 at the Blizard Institute London. In 2012 she was appointed as lecturer in Neuroimmunology. Her main research interest focusses on the role of environmental risk factors in multiple sclerosis, the topic of her Habilitation at the Ludwig-Maximilian-University Munich and more recently on dysregulated immune responses and the role of inflammation in psychiatric disease.

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