

Oral presentation

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Viral infections as triggers for CNS autoimmune diseases via molecular mimicry

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from Infectious diseases of the nervous system: pathogenesis and worldwide impact
Paris, France. 10–13 September 2008

Published: 23 September 2008

BMC Proceedings 2008, 2(Suppl 1):S29

This abstract is available from: <http://www.biomedcentral.com/1753-6561/2/S1/S29>

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It is well supported by epidemiological evidence that disease exacerbations of CNS autoimmune disorders such as multiple sclerosis can be triggered by viral infections. Similarly, it has been claimed that the development of MS is linked to environmental factors, particularly infections with specific viruses including EBV, HHV-6 and others. Two main mechanisms, by which autoimmune responses against CNS tissue can be induced, have been described, a) molecular mimicry, i.e. similarities in antigenic epitopes of viruses and tissue-specific autoantigens, and b) bystander activation, i.e. the induction of immune responses against autoantigens by unspecific activation of the innate immune system in the context of an infection.

The focus of the presentation will be on the mechanisms of molecular mimicry. This concept, which had originally been described by Fujinami and Oldstone and stipulated identical amino acid sequences between a peptide from self- and a viral protein, has evolved considerably during recent years. With a better understanding of the activation requirements and broader specificity of T cells, it has become clear that molecular mimicry is likely a frequent event and physiological under most circumstances. The context, in which molecular mimicry may become pathogenic and the evidence supporting its involvement in MS will be discussed.