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Dr Wekerle began by highlighting the problems associated with the investigation of patients with multiple sclerosis. Whilst he thought it very likely that the disease was an autoimmune one, he reminded the audience that we still do not know what the target autoantigen is, have almost no access to the target tissue and, are as yet, unable to transfer the disease with human material. He suggested therefore that most of our knowledge on multiple sclerosis, to date, had to rely on animal models of the disease. He went on to highlight the two animal models that he had worked with; the first is the EAE Lewis rat in which the disease is inducible with myelin or myelin basic protein (MBP) in complete Freund's adjuvant. He pointed out that the T cells and the pathogenesis of this disease were CD4+ T cells which were specific for MBP. In the mouse models that he has worked with, he reported that differences in haplotypes in mice lead to differences in the epitopes of the MBP autoantigen that they recognize so that, for example, the PL/J mice see the 1-11 amino acid sequence of the autoantigen, whereas the SJL/J mice see the 90-101 amino acid sequence. In contrast, the Lewis rat encephalytogenic T cell clones seemed to be seeing the amino acid sequence 68-88.
of MBP. Using these T cell clones it was possible to show that they transferred disease.

A major interest in the group currently is in the role which cells within the central nervous system may play in the process of antigen presentation. In this context, he pointed out that astrocytes and microglial cells can both express class I and class II antigens and, in addition, have antigen presenting capacity. In contrast, oligodendroglial cells, whilst expressing low levels of class I, neither express class II nor have the ability to function as antigen presenting cells with or without the addition of gamma interferon. He showed evidence of MBP specific T cells killing MBP-presenting astrocytes.

Using their panel of encephalytogenic T cell lines and clones in their PLJ mouse model and in the Lewis rat, they demonstrated that all T cell clones which recognize MBP have in common the presence of the V beta 8.2 determinant. Interestingly, despite the sequence homology between the mouse and rat V beta 8.2, the two species seem to recognize different epitopes of MBP.

In a different set of experiments, Dr Wekerle reported on work in the Lewis rat model in which an autoantigen specific CD8 positive T cell population were detectable in mice which had recovered from experimental allergic encephalomyelitis following innoculation with pathogenic CD4 positive T cell lines. These CD8 positive T cells were then allowed to