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## Interaction between viruses and the immune system: A pathogenic role for HHV-6A, EBV, and the complement system in multiple sclerosis and age-related macular degeneration; Two hypotheses

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Many microorganisms use a survival strategy based on their interference with the immune system. Some viruses are able to do so by docking to receptors on host cells that are important for proper functioning of the immune system. A well-known example is HIV that uses the CD4 cell surface molecule to enter host lymphocytes and thereby attacking the immune system. A more complicated example is seen in multiple sclerosis (MS) where human herpes virus-6A (HHV-6A) infects astrocytes by docking to the CD46 molecule. Such HHV-6A infection has recently been postulated to enable Epstein-Barr viruses (EBV) to transform latently infected B-lymphocytes leading to the well-known phenomenon of oligoclonal immunoglobulin production and cellular immune response to HHV-6A and EBV as part of pathogenic mechanisms in MS. A more subtle pathogenic mechanism can be seen in the down-regulation of CD46 on astrocytes by the infecting HHV-6A. Since CD46 is central in regulating the complement system, a lack of CD46 leads to hyper-activation of the complement system. In fact, activation of the complement system in brain lesions is one of the pathogenic mechanisms in MS. It is postulated that a similar mechanism is central in the development of age-related macular degeneration (AMD). One of the earliest changes in the retina of AMD patients is the loss of CD46 expression in the retinal pigment epithelium (RPE) in the course of geographic atrophy. Furthermore, CD46 deficient mice spontaneously develop dry-type AMD-like changes in their retina. It is also well known that certain genetic polymorphisms in the complement-inhibiting pathways correlate with higher risks of AMD development. The hypothesis is that HHV-6A infection of the retina leads to down-regulation of CD46 and consequently to hyper-activation of the complement system in the eyes of susceptible individuals.

### Recent Publications

Fierz W (2017) Multiple sclerosis: An example of pathogenic viral interaction? *Viol. J* 14:42.

### Biography

Walter Fierz is a Clinical Immunologist and completed his basic training in Cellular Immunology with experimental work at Transplantation Immunology Unit of the Clinical Research Center in Harrow, London (UK) and at Max-Planck-Society working group for multiple sclerosis research in Würzburg (Germany). He completed his Master's Degree in Health Information Management (MHIM) at Erasmus University in Rotterdam, Netherlands.

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