Early adulthood [10]. It has been hypothesised that EBV can cause observational studies strongly suggest that a predisposition for MS can timing of infection can have adverse consequences and, in this respect, of this have, until now, not been considered. Certainly a delay in the regard to the long co-evolution with humans and the consequences permanent absence of this virus would be extremely uncommon with after birth. Worldwide, the EBV sero-prevalence is high (>95%) so a increasingly occurs in adolescents and young adults rather than soon of relevance until in combination with accumulating disappearances of the disappearance of a given immunogenic member might not become other, at least in part, in their roles in the microbiome/virome so that microbi-organisms/viruses, are obviously able to substitute for each endogenous retroviruses, have been described [9]. Moreover, various micro-organisms/viruses, are obviously able to substitute for each other, at least in part, in their roles in the microbiome/virome so that the disappearance of a given immunogenic member might not become of relevance until in combination with accumulating disappearances of other permanent or transient co-players [7]. EBV is present in all human host immune system [6,7]. It is thus increasingly recognised that the ability of the individual immune system to fight infecting microorganisms and to take part in cancer immune surveillance is highly dependent on the population of endogenous micro-organisms including viruses (the microbiome and virome) and the past history, or ‘biography’, of the host immune system [8]. Linear and non-linear interactions between different co-players of the microbiome/virome, including human endogenous retroviruses, have been described [9]. Moreover, various micro-organisms/viruses, are obviously able to substitute for each other, at least in part, in their roles in the microbiome/virome so that the disappearance of a given immunogenic member might not become of relevance until in combination with accumulating disappearances of other permanent or transient co-players [7]. EBV is present in all human communities, including the industrialized countries where infection increasingly occurs in adolescents and young adults rather than soon after birth. Worldwide, the EBV sero-prevalence is high (>95%) so a permanent absence of this virus would be extremely uncommon with regard to the long co-evolution with humans and the consequences of this have, until now, not been considered. Certainly a delay in the timing of infection can have adverse consequences and, in this respect, observational studies strongly suggest that a predisposition for MS can result when a primary infection with EBV is delayed until teen-age or early adulthood [10]. It has been hypothesised that EBV can cause a subversion of a MS-protective immune reaction which, once subverted or eclipsed, is not easily repaired. An explanation has been given based on the phenomenon of ‘original antigenic sin’ and candidate epitopes on the viral EBNA1 protein have been delineated [6]. In general, however, a causative role of EBV for MS is still not definitively proven and there are also more direct role(s) for EBV in the pathogenesis of MS under consideration [11]. Although much more work needs to be done on the virus-host interactions following EBV infection it is increasingly apparent that such studies will need to take into account the overall composition of the virome as well as the ‘biography’ of the immune system, both of which could have far reaching consequences on the outcome of the infection or immunological significant contact. These considerations lead to the view that the use of a living attenuated vaccine given in early childhood would be far preferable to the use of sub-unit vaccines to be given irrespective of age at vaccination. Other types of agents might, however, be appropriate for the immunotherapy of any established EBV related disease.

References


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