The Epstein-Barr virus (EBV) is a well-known example of a human virus which interacts most intimately with the immune system. EBV is a polyclonal B lymphocyte activator, can immortalize B cells and, depending on situations, can also induce different T cell and cytokine activities (1-6). The present paper examines briefly several immunoregulatory aspects of EBV interaction with the immune system, many of which are pertinent to our understanding of immune controls over EBV infections. Various effects of EBV infection on cells of the immune system and their products are thus examined.

Effects of EBV Infection on B Cells: Early observations showed that lymphoblastoid cell lines can be established following EBV infection of human lymphocytes in vitro (7-8). It was later observed that EBV immortalizes B but not T cells (3-4). This is understandable in view of the fact that the EBV receptor is a commonly expressed marker of the human B lymphocyte (4, 29). It was subsequently demonstrated that EBV is in fact a polyclonal B cell activator (1,9) and that it can also induce differentiation of early B-lineage cells (10). These observations are therefore important to understand why some patients with EBV-induced infectious mononucleosis (IM) produce, in addition to EBV-specific antibodies (11), immunomodulatory immunoglobulins (12-13). These immunoglobulins were found to belong to the IgG class (14) and capable of inhibiting interleukin-2 (IL-2) production (15) as well as lymphocyte...
responses to antigens and mitogens (14, 16). Furthermore, recent studies show that human B lymphocytes immortalized in vitro by EBV can make autoantibodies (17-18). This may explain why spontaneously proliferating peripheral blood B lymphocytes from a significant number of EBV-seropositive patients with autoimmune diseases (19) or with IM (20-21) also produce autoantibodies. Another consequence of EBV-induced immortalization of B lymphocytes is the production by these cells of autostimulatory or B cell growth factor(s) (22-23) as well as immunomodulatory substances such as interleukin-1 (IL-1) (24-26) and plasminogen activator (27). It also appears that B lymphocytes can produce interferon alpha (IFN-α) in response to EBV infection (28). The different effects described above concerning the EBV interaction with B lymphocytes are depicted in the figure 1.

**Figure 1.** Diagrammatic presentation of EBV interaction with the B lymphocyte and its products.

**Effects of EBV Infection on T and Natural Killer (NK) Lymphocytes:** In contrast to B cells, the direct effects of EBV infection on T cells are not known. Recent studies by flow cytometry show however that a proportion of CD8-positive (cytotoxic/suppressor) T lymphocytes bear receptors for EBV, without however the virus being able to penetrate or multiply in these cells (30). On the other hand, it has been observed that during the primary infection (i.e. in IM patients) there is a phase of depression of CD4-positive (helper/inducer) cells (31)