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Epstein–Barr Virus (EBV) Receptors, Complement Receptors, and EBV Infectibility of Different Lymphocyte Fractions of Human Peripheral Blood

II. Epstein–Barr Virus Studies[1]

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In B-cell fractions isolated from human peripheral blood, the frequency of surface immunoglobulin-positive and of complement receptor-positive cells showed a good correlation with the frequency of EBV-binding cells, as detected by membrane fluorescence or by a quantitative bioassay for infectious virus in the absorbed supernatant fluid. There was a close relationship between all three parameters mentioned, the frequency of EBNA-positive cells 2 or 3 days after the infection, and the stimulation of cellular DNA synthesis. So-called O-cell fractions remaining after the removal of nylon adherent and E-rosetting cells contained a certain frequency of complement receptor-positive cells and absorbed EBV to a limited extent, but did not respond to EBV infection with EBNA induction or stimulation of DNA synthesis. None of the T-cell fractions absorbed EBV to a detectable extent. This includes the T_{EA+} fraction that contained a certain proportion of complement receptor-positive cells. It is concluded that the previously demonstrated relationship between EBV receptors and complement receptors on B-lymphoblastoid lines also holds for peripheral B lymphocytes. In these cells, virus absorption is followed by an intracellular infectious process, signaled by the appearance of EBNA and cellular DNA synthesis. O cells carry complement receptors and absorb EBV to a certain extent, but do not respond with EBNA synthesis or DNA stimulation, presumably due to intracellular restrictions. T cells do not bind EBV, and the complement receptors present on some cells of the T_{EA+} fraction do not function as EBV receptors.