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Sommario/riassunto	Epstein-Barr virus (EBV) is a ubiquitous virus that infects 95% of adults worldwide; the vast majority of persons have asymptomatic or non-specific primary infection and no complications associated with EBV during their lifetime. Some persons, particularly those who are infected as adolescents or young adults, develop infectious mononucleosis. EBV infects resting B cells and infection in vitro results in transformation and continuous proliferation of the cells, whereas infection in vivo results in a latent infection in which proliferation of the cells is controlled by virus-specific T cells and NK cells. Certain persons have mutations in genes that result in impaired cellular immunity involving the function of cytotoxic T cells or NK cells that result in impaired responses and failure to control EBV. These persons are at risk for fulminant infectious mononucleosis, EBV-associated hemophagocytosis, EBV B or T cell lymphoma, or other opportunistic infections. These genes encode proteins that are important for a variety of NK and T cell activities: T cell interactions with B cells, NK and T cell activation, NK and T cell cytotoxicity, priming and expansion of virus-specific T cells, and control of T cell apoptosis. For most of these diseases, hematopoietic stem cell transplantation has been the only curative therapy. However, identification of certain immune deficiencies has led to new approaches to therapy such as drugs to inhibit overactive signaling pathways or supplemental magnesium for patients

with mutations in a magnesium transporter. The study of these EBV-associated immune deficiencies identifies the importance of these proteins for the function of T and NK cells and may lead to novel approaches to therapy for EBV diseases.