Genetic and Viral Etiology of Glioblastoma--a Unifying Hypothesis.

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Abstract

Growing body of evidence implicates human cytomegalovirus (HCMV) in the etiology of glioblastoma (GBM). Although HCMV is a ubiquitous herpesvirus, only a minority of those infected develop GBM, suggesting the involvement of host genetic factors in susceptibility to HCMV-induced/spurred GBM. HCMV has evolved a large repertoire of strategies for decreasing the efficacy of the host immune response and interfering with viral clearance. One strategy involves the generation of proteins that have functional properties of the Fcγ receptor (FcγR), which may enable the virus to evade host immunosurveillance by avoiding the effector consequences of antibody binding, such as antibody-dependent cellular cytotoxicity. Results of binding studies involving HCMV-encoded FcγR and genetically different immunoglobulin G proteins suggest that GM genes--genetic determinants of immunoglobulin γ chains--could modulate this viral strategy and thus serve as functional risk factors for the development of GBM, potentially unifying its seemingly disparate infectious, immune, and genetic etiologies. Cancer Epidemiol Biomarkers Prev; 20(6); 1-3. ©2011 AACR.

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