Human herpesvirus 6 (HHV-6)-associated dysfunction of blood monocytes.

Abstract

HHV-6 is a recently described member of the herpesvirus family. HHV-6-associated marrow failure and interstitial pneumonitis where macrophages are the primary infected cell type have been described in marrow transplant patients (Carrigan, 1991; Drobyski et al., 1993). In recent studies we have shown that exposure of normal human marrow to HHV-6\(_{GS}\) (a type A strain) or several type B strains resulted in suppression of growth factor induced outgrowth of macrophages by > 90% (Burd and Carrigan, 1993). Additional experiments using HHV-6\(_{GS}\) to characterize the effects of the virus on peripheral blood monocytes showed that the respiratory burst capacity of these cells as determined by luminol-enhanced chemiluminescence using phorbol myristate acetate as a trigger was decreased by 83% ± 13% in a series of 5 experiments. The decreased respiratory burst was evident as early as 15 min after exposure to virus. Experiments in which cells were separated on a fluorescence activated cell sorter prior to respiratory burst measurement showed that the decreased respiratory burst was evident as early as 15 min after exposure to virus.
which cells were separated on a fluorescence activated cell sorter prior to respiratory burst assay showed that the response was mediated solely by peripheral blood monocytes. The respiratory burst response of virus-exposed cells to opsonized zymosan was not affected, indicating that the virus may selectively interfere with the protein kinase C pathway of cellular activation. Ultracentrifugation of stock material to remove infectious virus showed that the suppressive factor was associated with the supernatant fraction. These findings suggest that HHV-6 infection may be associated with a defect in one of the major monocyte activation pathways, and this could be of importance with respect to persistent infection by HHV-6 in immune compromised patients.

Keywords
Human herpesvirus 6; Respiratory burst; Monocyte; Signal transduction; Immune suppression
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