Recent studies have suggested links between Herpesviral infection and periodontopathic bacteria, i.e. Porphyromonas gingivalis. OBJECTIVE: The objective of this study was to investigate effects of bacterial metabolic end products on gammaherpesviruses, Kaposi’s Sarcoma associated Herpesvirus (KSHV) and Epstein-Barr Viruses (EBV). Hypothesis: P. gingivalis metabolic end products, such as butyrate, induce gamma herpes virus reactivation from latency. METHODS: Quantitative real time polymerase chain reaction (QRT PCR) and immunofluorescence used to assay the role of P. gingivalis in reactivation of epithelial and lymphoid cells latently infected with KSHV and EBV. Uninfected lymphoid cells, BJAB, used as a negative control. BCBL1 cells, latently infected with KSHV, or B95-8 cells, latently infected with EBV, were exposed to tetradecanoyl phorbol acetate (TPA), 3mM sodium butyrate (N-BU), or P. gingivalis spent media. In all experiments N-BU and TPA served as positive controls for induction as evidenced by past experiments. Reactivation was also studied in a telomerase immortalized oral epithelial (TIPOE) cell line, generated from gingival extraction tissue, and latently infected with KSHV. Cells were exposed to TPA, N-BU, or P. gingivalis spent media. Reactivation was detected by both increase in viral DNA by QRT PCR and by expression of viral lytic proteins in immunofluorescence assays. RESULTS: Induction of both KSHV and EBV replication was detected in lymphoid and epithelial cells upon exposure to P. gingivalis spent media, to TPA or to 3mM N-BU. Latently infected cells exposed to P. gingivalis exhibited QRT PCR results indicating KSHV and EBV replication levels comparable to those cells exposed to N-BU. CONCLUSION: These results support P. gingivalis as an inducer of gamma herpes viral replication in the oral cavity, suggesting that the organisms act cooperatively. Findings may be central to pathogenesis of both viruses and bacteria in oral disease. Studies supported by USPHS NIDCR grant 1 K23 DE 00460-01. picot@email.unc.edu

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