Supplemental Fig. S1. FISH hybridization with the HPV16 probe labeled in red in different cervical cancer cell lines. CaSki, SiHa and Hela cells present high-number, low-number and none copies of the integrated HP16 genome, respectively.
Supplemental Fig. S2. FISH hybridization with the HBV probe labeled in red in different hepatic carcinoma cell lines. HA22T, SNU354 and ALEX demonstrate integrated HBV genome. The hepatoblastoma cell line HepG2 is used as negative control.
Supplemental Fig. S3. FISH hybridization with the EBV probe labeled in red in different lymphoma cell lines. Raji, Akata and Namalwa present high, intermediate and low-number copies of the EBV genome, respectively. The lymphoma cell line Ramos is used as negative control.
Supplemental Fig. S4. The induction of the EBV lytic cycle in a lymphoma cell line (AKATA) upon addition of anti-IgG to the media caused a major change in the EBV DNA methylome, in just 48 hours, characterized by massive hypomethylating events at the transcription start sites present in the EBV virus. This wave of DNA hypomethylation was accompanied by the restoration of gene expression of the previously methylated genes, which matched our expression microarray data [Yuan, J., E. Cahir-McFarland, B. Zhao, and E. Kieff. 2006. Virus and cell RNAs expressed during Epstein-Barr virus replication. J Virol 80: 2548-2565]. Red and green blocks indicate methylated and unmethylated transcription start sites (TSS), respectively. a, DNA hypomethylating events associated with gene reactivation. b, Unmethylated TSSs with decreased expression. c, Upregulated TSSs.
Supplemental Fig. S5. Depletion of LMP1 by RNA interference in Raji lymphoma cells is associated with reduced levels of DNMT1 and DNMT3b, shown by RT-PCR (A), and hypomethylation of particular CpG sites in the EBV genome, shown by bisulfite genomic sequencing (B). Black squares, methylated CpGs; white squares, unmethylated CpGs.