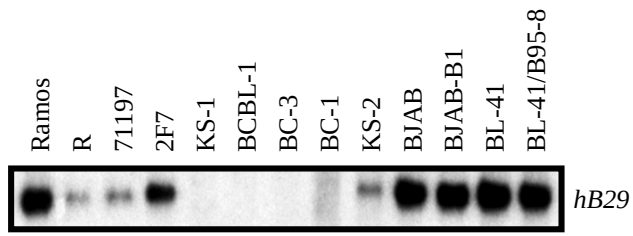
Table 1. Expression of the endogenous *B29* gene in lymphocyte lines

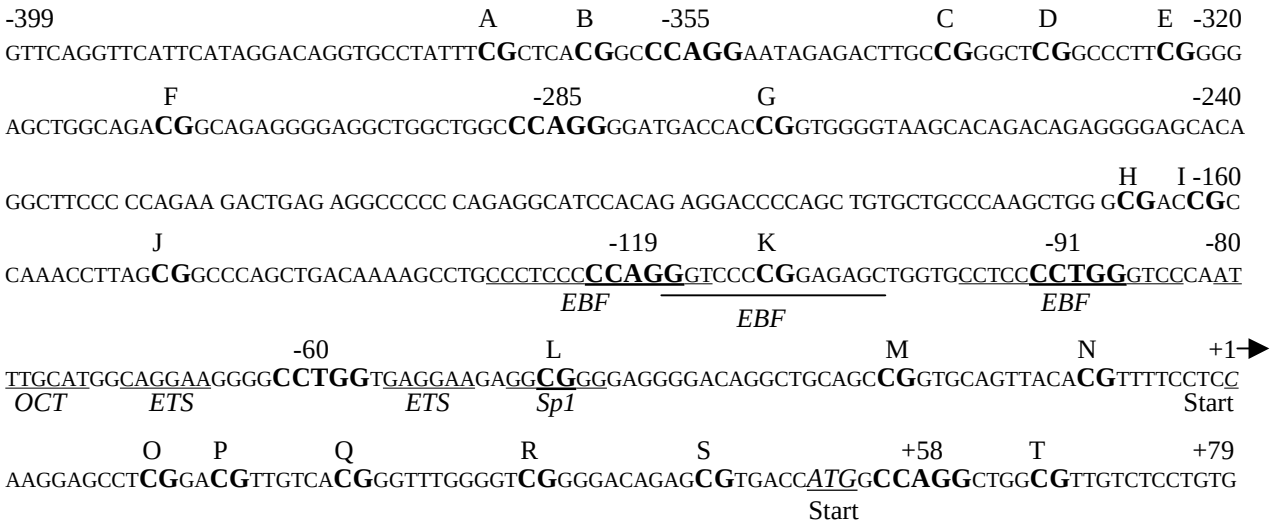
Cell line	*B29* level	γ Herpesvirus	Cell line	*B29* level	γ Herpesvirus
Burkitt lymphomas			**Multiple myelomas**		
BJAB	4.7	neg	AF-10	1.5	ND
BJAB-B1	4.8	EBV	H929	1.4	ND
BL-41	6	neg	KNS11	1.2	ND
BL-41/B95-8	4.7	EBV	MMS1	1.2	ND
Ramos	3.2	neg	RPMI-8226	neg	ND
2F7	2.2	EBV	**Primary effusion lymphomas**		
Diffuse large B cell lymphomas			BC-1	neg	EBV, HHV-8
KS-2	1.4	EBV	BC-3	neg	HHV-8
R	1	EBV	BCBL-1	neg	HHV-8
EBV-immortalized B cell lines			KS-1	neg	HHV-8
61072	1.5	EBV	**T cell leukemia**		
71197	1.1	EBV	Jurkat	neg	neg
75714	2.2	EBV			

Results of Northern blot analyses for *B29* gene expression. BJAB-B1 and BL-41/B95-8 cell lines are Epstein–Barr virus (EBV)-infected subclones of parental lines BJAB and BL-41, respectively. *B29*-negative lines BCBL-1 and BC-3 were also examined with two 35-cycle rounds of *B29* gene-specific nested reverse transcription–PCR and remained negative (data not shown). The *B29* level indicates fold expression compared to an arbitrarily set value of 1.0 for *B29* expression in the diffuse large B cell lymphoma line R, as determined by PhosphorImager analyses. ND, not determined; neg, negative.



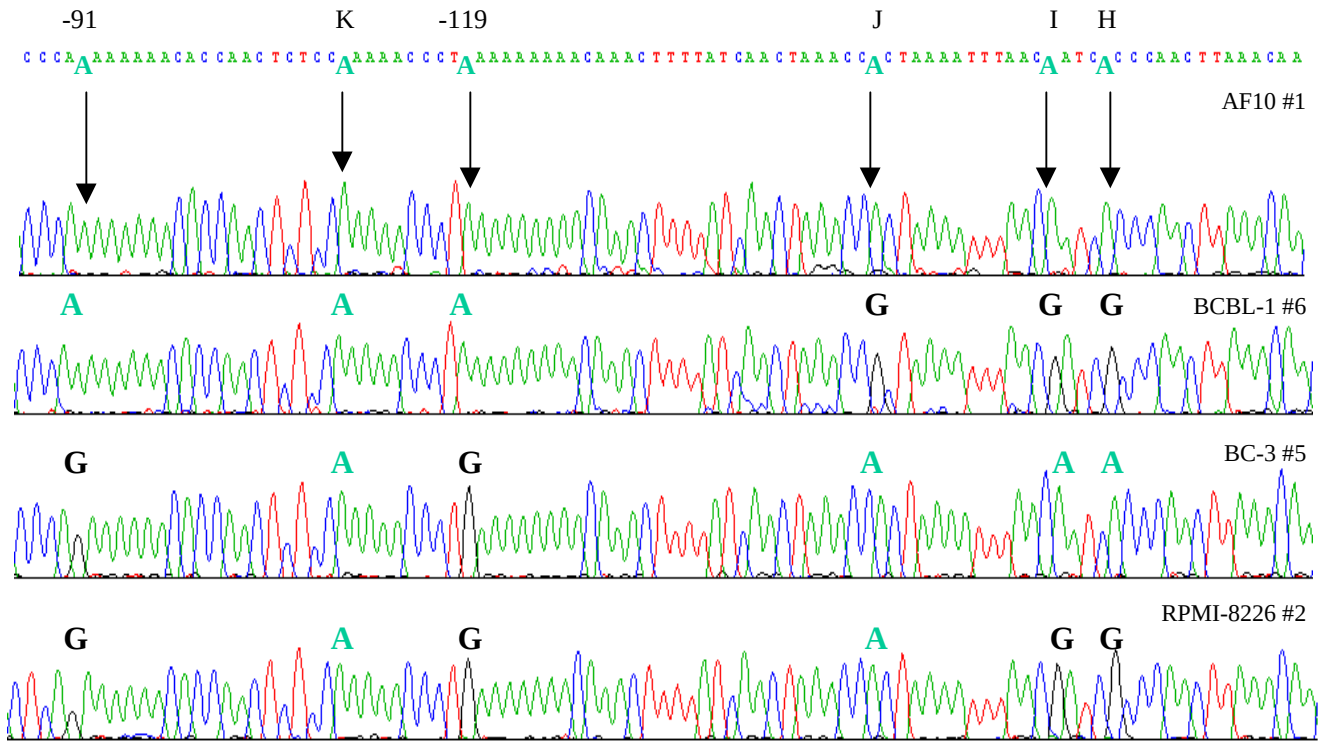
Supplemental Figure 7

Fig. 7. Northern blot analysis of *B29* gene expression in B cell lines. The data are presented in Table 1 from this and additional Northern blots. Ethidium bromide staining of formaldehyde gels showed equal lane loading of total RNA from all B cell lines examined (data not shown). Cell lines used in this study were kindly provided by A. Berk [HeLa; University of California, Los Angeles (UCLA)], S. Plaeger-Marshall (61072, 71197, and 75714; UCLA), B. Bonavida (2F7 and R; UCLA), R. Sun (BCBL-1, BC-1, BC-3, KS-1, BL-41, BL-41/B95-8, BJAB, and BJAB-B1; UCLA), J. Said (KS-2; UCLA), and W. Kuehl (MMS1, H929, Kns11, and AF10; National Cancer Institute). Ramos, RPMI-8226, and Jurkat cell lines were purchased from the American Type Culture Collection.



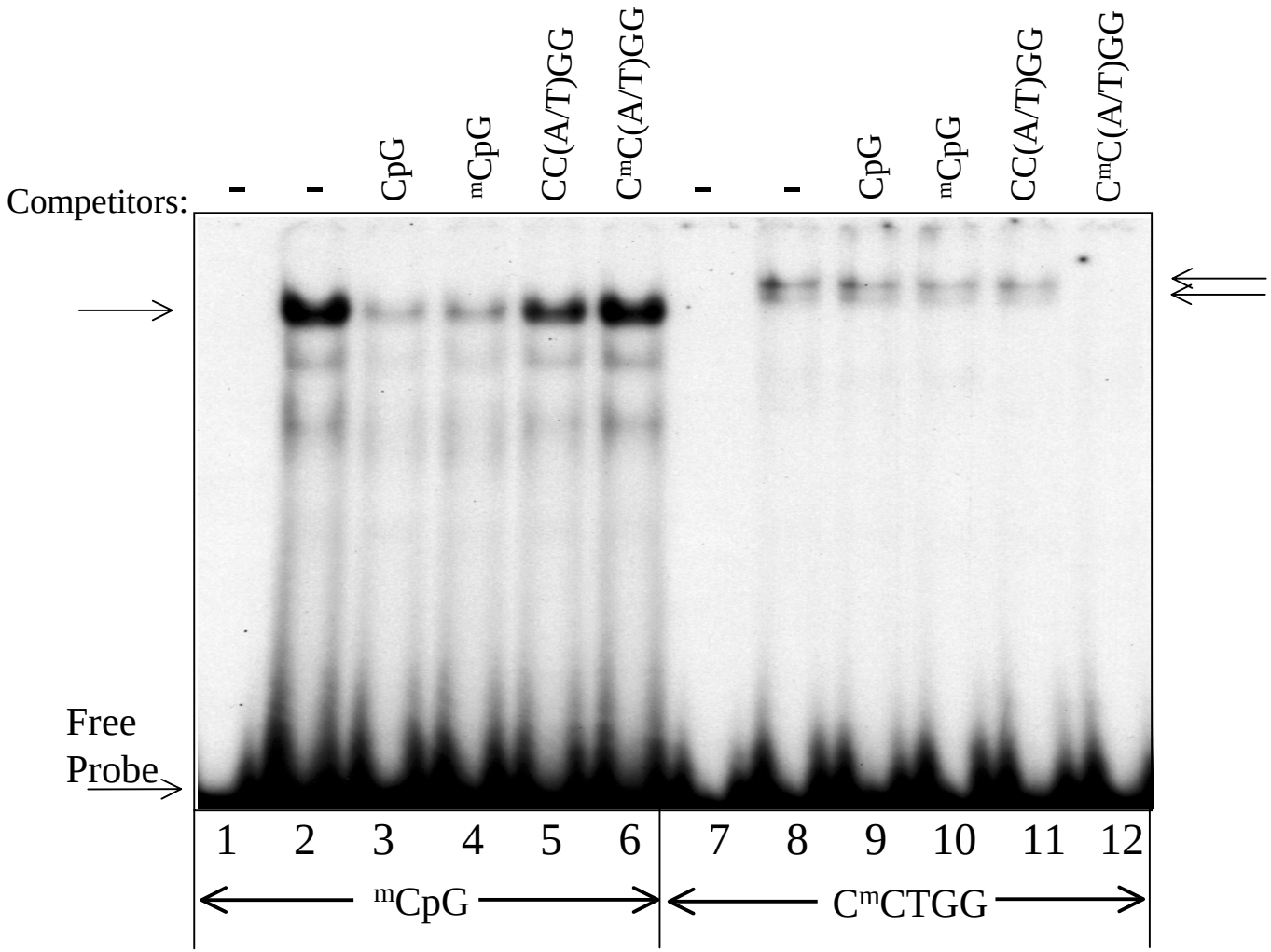
Supplemental Figure 8

Fig. 8. Schematic representation of the human *B29* promoter region examined in this study. CG boldface type indicates positions of CpG dinucleotides. These sites are depicted alphabetically. Numbered bold sequences indicate CC(A/T)GG motifs. Underlined sequences indicate transcription factor-binding sites ADDIN ENRfu [Akerblad, P., Rosberg, M., Leanderson, T. & Sigvardsson, M. (1999) *Mol. Cell. Biol.* **19,** 392–401; Omori, S. A. & Wall, R. (1993) *Proc. Natl. Acad. Sci. USA* **90,** 11723–11727). The major start site of transcription is denoted as +1 with an arrow and the ATG start of translation is denoted +1 in italic typeface.



Supplemental Figure 9

Fig. 9. Primary sequencing data show three distinct patterns of cytosine methylation of the endogenous *B29* promoter. Positions are listed (*Top*) and refer to Fig. 8; the listed sequence is for a completely unmethylated and bisulfite converted sequence. (*Top*) The first sequence shows complete sequence conversion with bisulfite treatment and no evidence of CpG or CC(A/T)GG methylation in *B29*-expressing AF10 clone 2. The second sequence shows selective ^{m}CpG in BCBL-1 clone 6. The third sequence shows selective C^{m}C(A/T)GG in BC-3 clone 5. The bottom sequence shows both ^{m}CpG and C^{m}C(A/T)GG on the same allele in RPMI-8226 clone 2. Primer pairs used to amplify sodium bisulfite treated genomic DNA included: forward primers: A(5′-TTTAGATGTTTGATTTGGGTTTGTGGTTGT-3′), B(5′-GGGTGAGGAATAGTTTAGG ATAGAGGAGT-3′), C(5′-GAGGGGAGGTTGGTTG-GTTTAGGGGATGAT-3′) and D(5′-GGGTAAGTATAGATAGAGGGGAGTATA-GGT-3′); Reverse primers: E(5′-CTATACC TAAAAACAACAACAACAAC-3′), F(5′-CCACCATCCAATAACTAAACAC-3′), G(5′-CCAACCTAACCATAATCAC-3′) and H(5′-ACTACAACCTATCCCCTCCC-3′).



Supplemental Figure 10

Fig. 10. A methylated CpG (^{m}CpG) methylated oligonucleotide does not bind or compete for the $^{m}C^{-91}$ DNA–protein complex. Symmetrically ^{m}CpG- and $^{m}C^{-91}$-methylated (C^{m}CTGG) double-stranded DNA oligonucleotide probes were analyzed by EMSA with using 20 μg of BC-3 primary effusion lymphoma nuclear extracts. Oligonucleotide cold competitor (500-fold) was added, as indicated. Unlabeled arrows denote specific complexes.