Supplementary Figure 1: Inhibition of CREB/CREM/ATF transcription factor binding *in vitro* by methylation at the -154 and -129 CpGs. (A) EMSAs with the TxRE1 probe in the presence of methylated and unmethylated competitor oligonucleotides. The TxRE1 probe was incubated with nuclear extracts from BLV-infected ovine PBMCs (10 µg) in the absence of competitor (lanes 1, 6 and 11) or in the presence of increasing concentrations (7.5-, 15-, 30- and 60-fold molar excess) of the homologous unmethylated TxRE1 oligonucleotide (lanes 2 to 5), of the methylated TxRE1-154me oligonucleotide (lanes 7 to 10), or of the heterologous HIV-1 NF-κB oligonucleotide (lanes 12 to 15). The HIV-1 NF-κB oligonucleotide contains the two NF-κB-binding sites of the HIV-1 enhancer (nt +350 to +373) and has been described previously (73). The figure shows the sequence-specific retarded bands of interest. (B) EMSAs with the TxRE2 probe in the presence of methylated and unmethylated competitor oligonucleotides. The TxRE2 probe was incubated with nuclear extracts from BLV-infected ovine PBMCs (10 µg) in the absence of competitor (lanes 1, 6 and 11) or in the presence of increasing concentrations (7.5-, 15-, 30- and 60-fold molar excess) of the homologous unmethylated TxRE2 oligonucleotide (lanes 2 to 5), of the methylated TxRE2-129me oligonucleotide (lanes 7 to 10), or of the heterologous HIV-1 NF-κB oligonucleotide (lanes 12 to 15). The HIV-1 NF-κB oligonucleotide contains the two NF-κB-binding sites of the HIV-1 enhancer (nt +350 to +373) and has been described previously (73). The figure shows the sequence-specific retarded bands of interest.