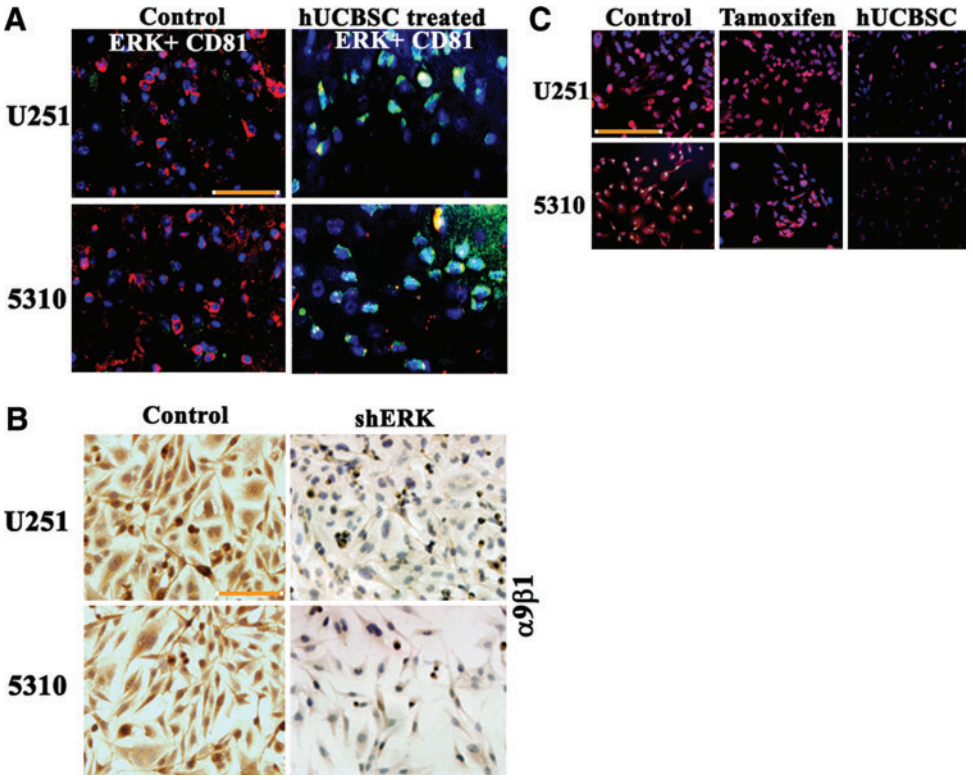
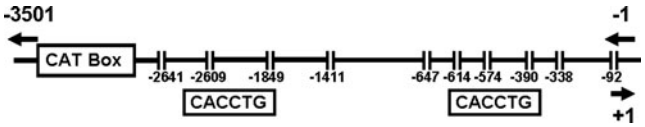
Supplementary Data



SUPPLEMENTARY FIG. S1. hUCBSC treatment downregulates ERK expression *in vivo*. **(A)** Nude mice with pre-established intracranial human glioma tumors were left alone or treated with hUCBSC. Immunohistochemistry was performed in single and cocultures of U251 and 5310 cells and hUCBSC to study the expression of ERK and CD81. ERK is conjugated with Alexa Fluor-594 (*red*), and CD81 is conjugated with Alexa Flour-488 (*green*). Bar = 100 μm ($n = 3$). **(B)** DAB staining to study the expression levels of α9β1 in shERK-transfected samples of U251 and 5310 cells. *Brown* staining is indicative of the expression levels. The cells were counterstained with hematoxylin for nuclear staining. Bar = 100 μm. **(C)** ERK translocation assay. U251 and 5310 alone or in coculture with hUCBSC in the ratio of 1:1 were plated at 1×10^5 cells/well in a 96-well plate and grown in their respective medium overnight. For positive control, the cells were treated with 20 μM Tamoxifen for 1 h. The cells were then processed for immunostaining with ERK/mitogen-activated protein kinase antibody as per standard protocols. Retarding of the translocation of ERK from the cytoplasm to the nucleus as a result of hUCBSC treatment is evident when compared to controls. hUCBSC, human umbilical cord blood stem cell; ERK, extracellular signal-regulated kinase.



SUPPLEMENTARY FIG. S2. Identification of putative c-Myc binding sites in ERK promoter. Around 5,000 bases upstream from the transcription initiation start site were taken and analyzed for the presence of c-Myc binding sites using Transfac (V.0. 2.0) (Beverly, MA). The promoter binding sites were authenticated by annotating the obtained binding sites using AGGLEN (Promo) software version 3.0.2 (Barcelona, Spain). The blocks indicate the presence of putative c-Myc binding sites.