10.1071/RD15145_AC

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Supplementary Material

Retinoic acid triggers *c-kit* gene expression in spermatogonial stem cells through an enhanceosome constituted between transcription factor binding sites for retinoic acid response element (RARE), spleen focus forming virus proviral integration oncogene (SPFI1) (PU.1) and E26 transformation-specific (ETS)

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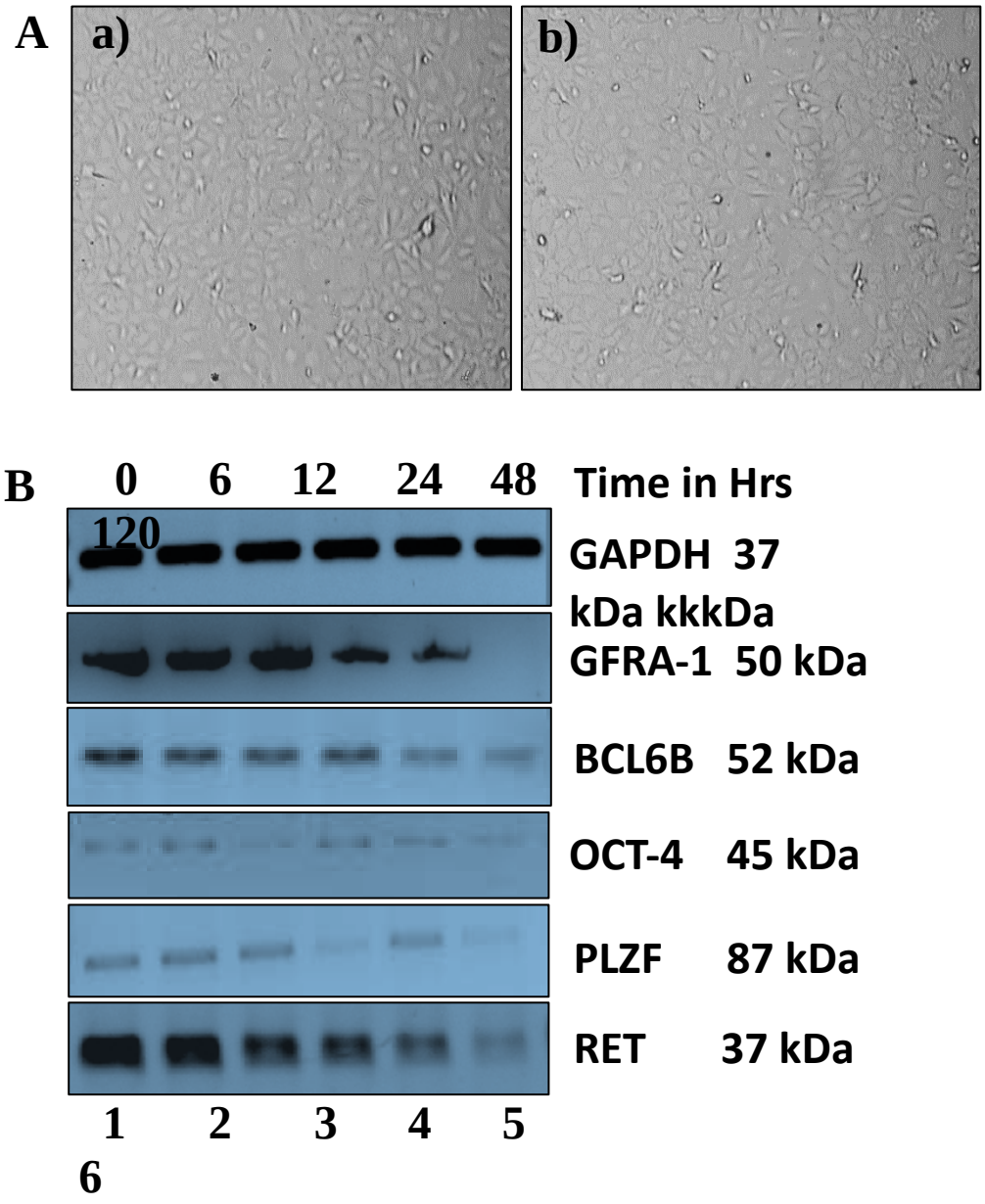

Fig. S1. (A) No morphological differences were seen in C18-4 cells with RA treatment. (a) C18-4 cells Control 24 h. (b) C18-4 cells RA (1 µM) treated 24 h. (B) Representative immunoblot for SSC undifferentiated markers GFRA-1, BCL6B, OCT-4, PLZF, RET and loading control GAPDH assessed in C18-4 cells to RA treatment.

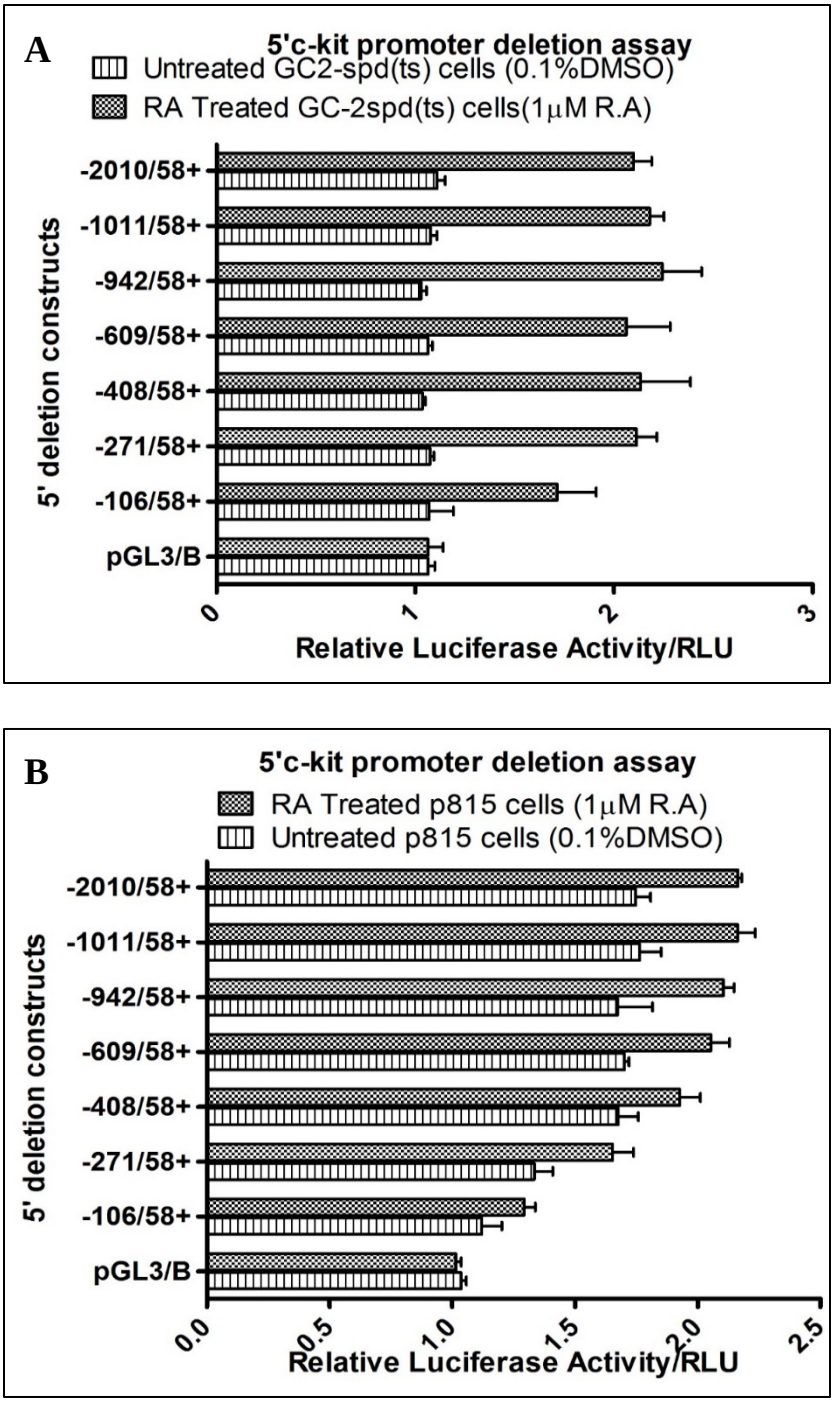

Fig. S2. Promoter deletion assay performed with GC2spd(ts) cells (A) and P815 cells (B) constructs described in materials and methods. The bar values indicates luciferase activity levels of the respective reporter constructs. All transient transfection studies were conducted in triplicate on three separate occasions, with similar results. Luciferase activity (right) was normalised to that of Renilla luciferase. The data reported in the chart represent the means of three replicate experiments.

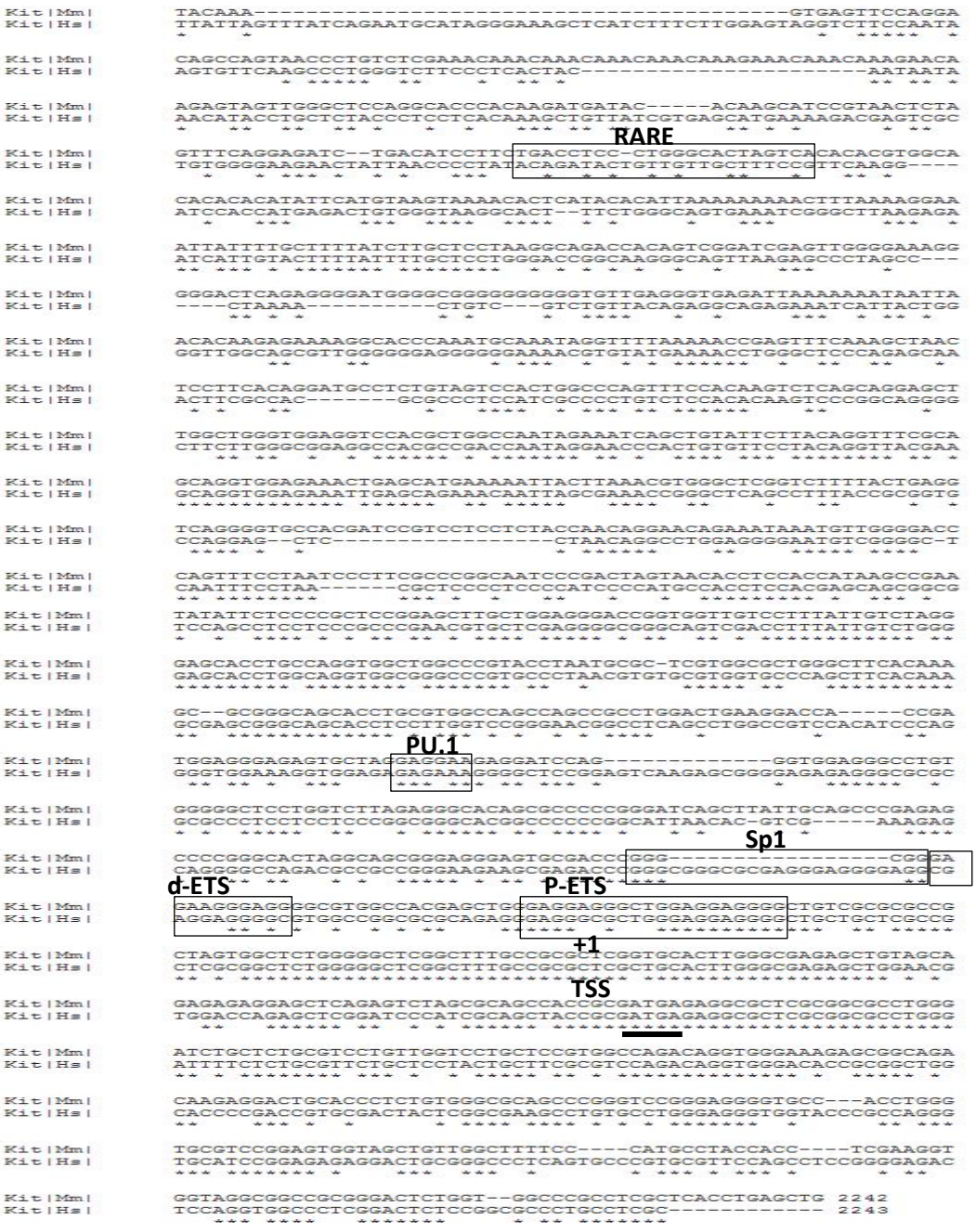

Fig. S3. ClustalW alignment of mouse and human c-kit promoter region. Boxed are the identified transcription factor binding sites. TSS- Transcription start site +1 and ATG - Translation start site underlined.

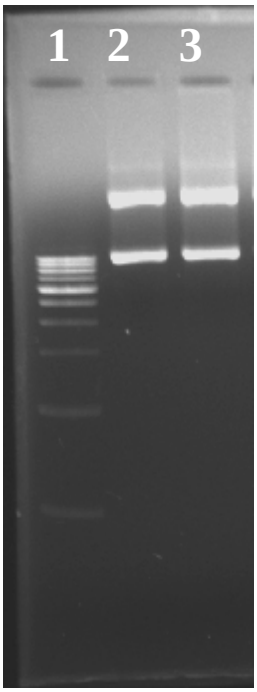

Fig. S4. CBP/p300 overexpression plasmid. 1, 1KB DNA ladder; 2,3, *p*cDNA3β-FLAG-CBP-HA upper band (12.6 Kb) and FLAG-CBP-N602-HA lower band (9.2) Kb.