**Cecchinelli et al., SUPPLEMENTAL MATERIALS**

## Figure 1S

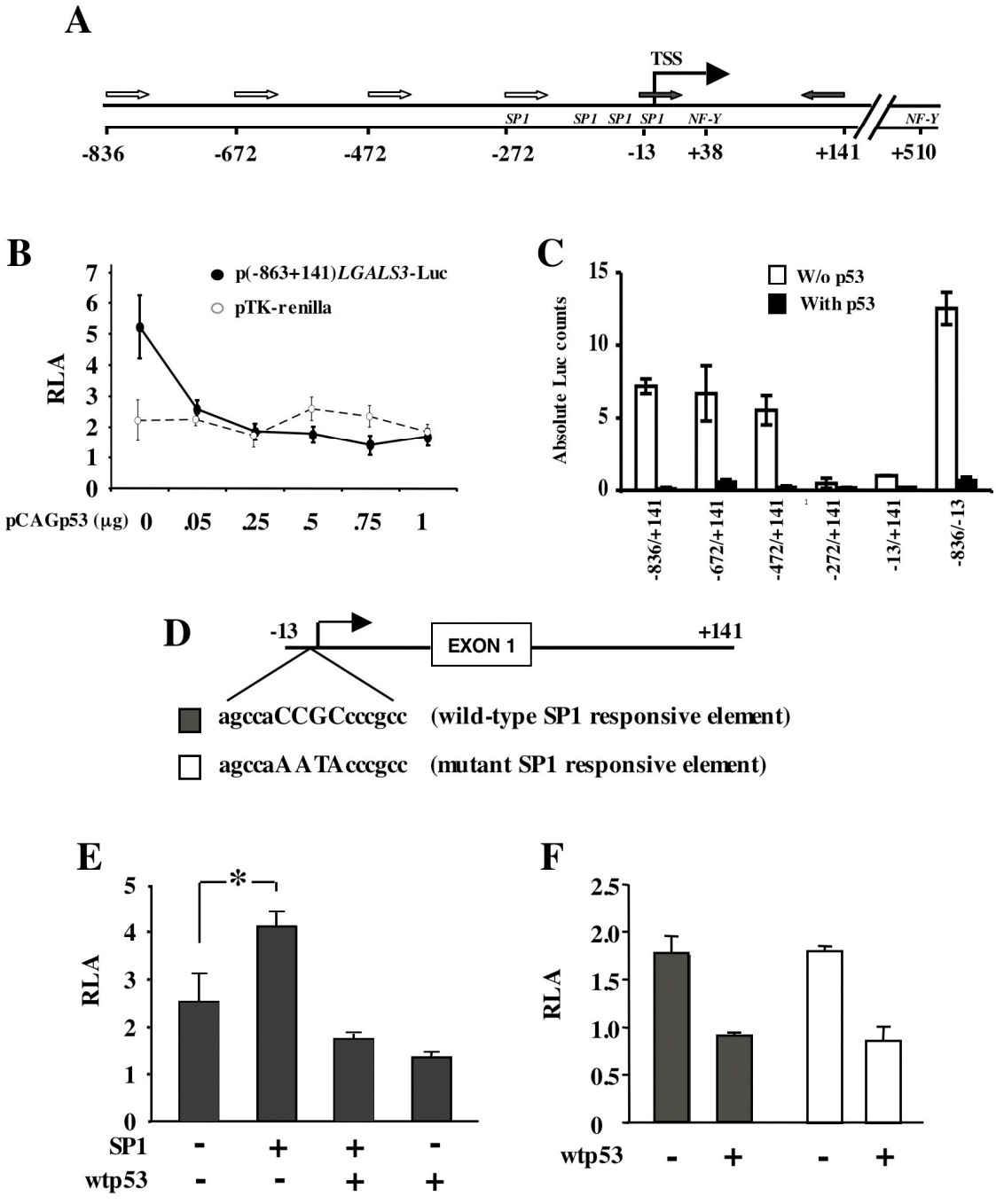

**FIG. 1S.** *LGALS3* promoter characterization**.** (A) Schematic representation of the LGALS3 promoters. The SP1 and the NF-Y binding sites and the transcription start site (TSS) are reported as described (28). Arrows (both back and white) indicate the primers employed to develope the luciferase-reporter vectors. The numbers indicate the position on the promoter at which it primer starts. The black arrows indicate the only couple of primers that was able to amplify the DNA in the ChIP experiments and that was consequently employed for this purpose. (B) Dose-dependent inhibitory effect of wtp53 on the *LGALS3* promoter activity. Luciferase activity on the p53-responsive p(-836/+141)*LGALS3*-Luc (black circles, continuous line) and on the p53-unresponsive pTK-renilla control vector (white circle, dotted line) are shown. Means ± standard deviations of at least seven independent experiments are reported. (C) Absolute luciferase counts relative to the experiments reported in FIG. 2D. (D) Schematic representation of the -13/+141 region of the *LGALS3* promoter. Sequences of the TSS-proximal SP1 site in the wild type (black symbol) and mutant (white symbol) configuration is shown. (E) Coexpression studies were performed in human H1299 cells using the p(-13/+141)*LGALS3*-Luc construct (wild type SP1 sequence, as indicated by the black bars) together with wtp53 and/or SP1 carrying vectors. Means ± standard deviations of at least seven independent experiments are reported. (F) H1299 cells were transfected with the p(-13/+141)*LGALS3*-Luc constructs carrying the wild type (black bars) or the mutant (white bars) SP1 site in the presence or absence of wtp53. Means ± standard deviations of at four independent experiments are reported.