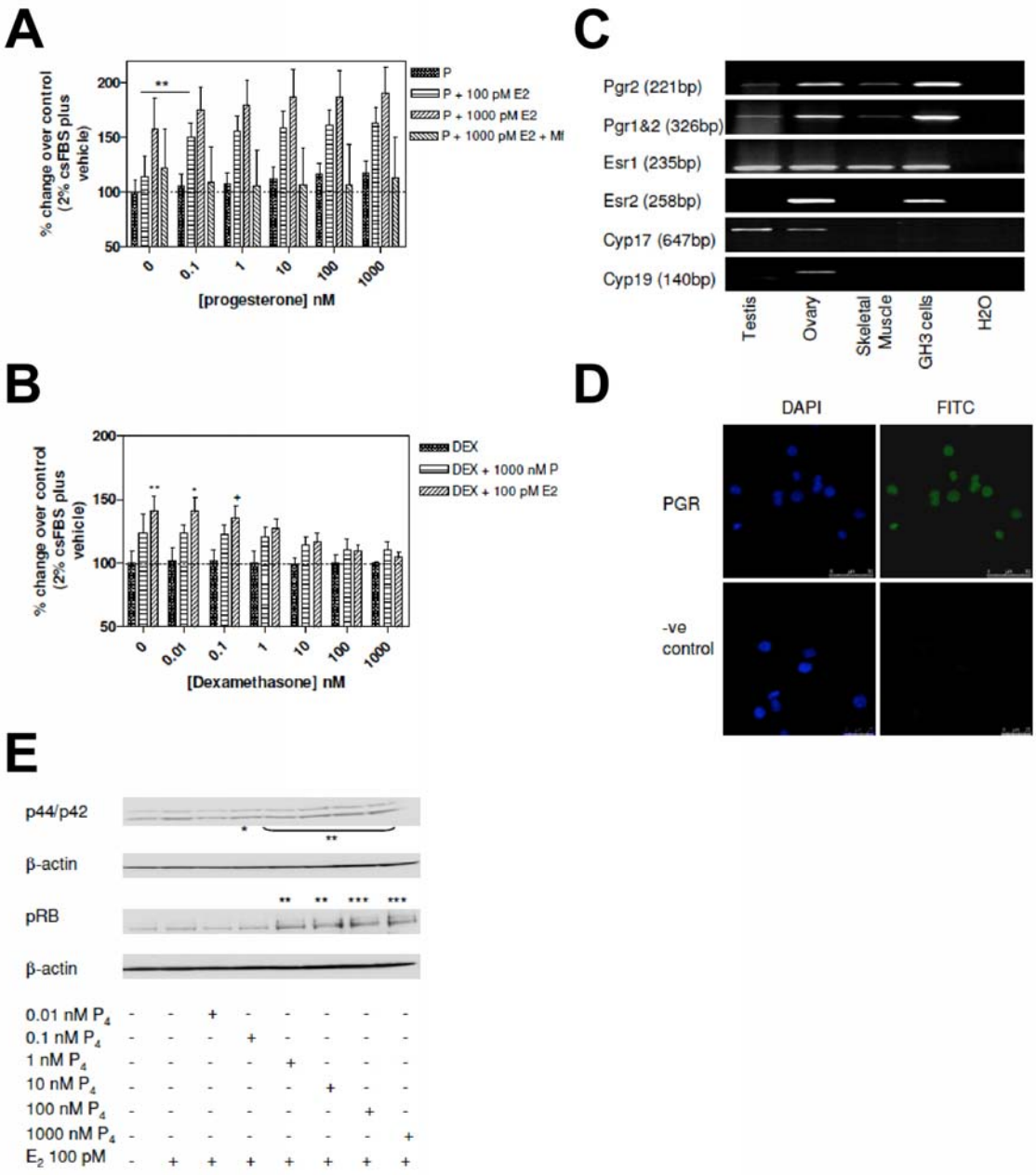

Supplementary Figure 5 Additional data from the experiments with GH3 cells. (A) Effects of 48h progesterone (P4) stimulation on GH3 cell proliferation when co-treated with E2 and/or mifepristone (Mf). The mean cell number in controls was taken as 100%, and those of the other treatments were related to this figure (mean + SEM of 5–6 independent experiments). **, $P < 0.01$ vs basal plus 100 pM P4. (B) Effects of 48 h dexamethasone (DEX) stimulation on GH3 cell proliferation when co-treated with P4 or E2. The mean cell number in controls was taken as 100%, and those of the other treatments were related to this figure (mean + SEM of 6 independent experiments). **, $P \leq 0.01$, compared to basal, #, $P \leq 0.05$, compared to basal. *, $P \leq 0.05$ compared to basal plus 0.01 nM DEX, +, $p \leq 0.05$, compared to basal plus 0.1 nM DEX. (C) Expression of Pgr1 and 2, Esr1 and 2, Cyp17 and Cyp19 mRNAs in rat testis, ovary, skeletal muscle and GH3 cells. The results are representative of 3 individual experiments. (D) Progesterone receptor (PGR) protein localisation in GH3 cells. Immunofluorescence was clearly seen in the nucleus of GH3 when tissues were probed for progesterone receptor. As expected, no fluorescence was observed when primary antibodies were omitted as a negative control. Each panel is representative of 3 individual experiments. (E) pERK and pRB protein levels in GH3 cells in response to P4 and/or E2 stimulation. Western blots were probed with antibodies for phospho p44/p42 (44, 42kDa, pane), and phospho retinoblastoma (pRB, 125kDa). Each panel presents typical Western blots in which the tissue-specific protein expression profile was representative of 3 experiments. The asterisks indicate differences from levels in the absence of P4 in the compiled data (**, $P < 0.01$; ***, $P < 0.001$).