Fig. S6

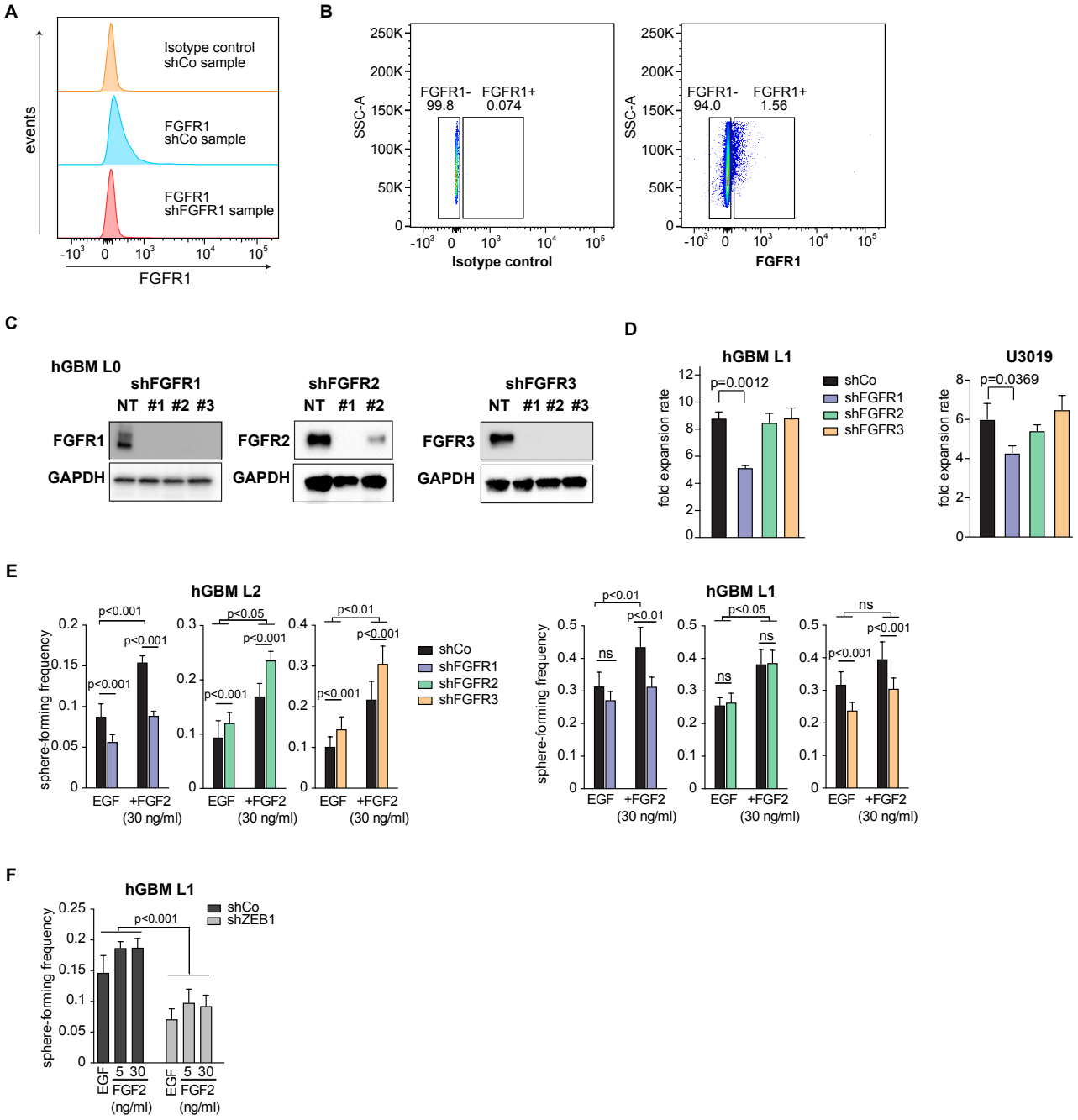

Fig. S6: (A) FGFR1 antibody specificity in flow cytometry. Shown are histograms for staining intensity (x-axis) versus event counts (y-axis) for isotype control, FGFR1-stained samples, and FGFR1 knockdown samples. Note the lack of staining after FGFR1 knockdown. **(B)** Gating strategy for FACS sorting of FGFR1+ and FGFR1- cells. **(C)** Western blot demonstrates specificity of FGFR knockdown constructs. Shown are multiple constructs for FGFR1, -2 and -3. In all cases construct #1 was selected for downstream experiments. **(D)** Loss of FGFR1, but not FGFR2 or -3, affects proliferation in EGF (hGBM L1) and EGF/FGF2 (U3019) treated cells. Bars represent fold-change of expansion rates (n=3 per cell line; $p<0.05$, one-way ANOVA). **(E)** Sphere-forming assays with EGF or EGF+FGF2 reveal that knockdown of FGFR1 consistently blocks self-renewal in two patient-derived GBM cell lines. By contrast, loss of FGFR2 increased self-renewal (hGBM L2), or showed no effect (hGBM L1) in response to FGF2 stimulation. Loss of FGFR3 showed inconsistent effects. **(F)** Loss of ZEB1 also blocked FGF2-induced self-renewal, supporting that ZEB1 is downstream of FGFR1, and sufficient to mediate self-renewal effects of FGF2.