**Supporting Information Fig 6. FGF2-specific FGFR signalling mediated by FRS2 and MAPK/MEK/ERK pathway.**

A. Treatment of PS1 cells with FGF2 led to an increase in pERK and pFRS2 levels within 15 minutes. Activation of ERK and FRS2 was completely abolished following pre-treatment of PS1 cells with PD174074 inhibitor for 1 hour prior to FGF2 stimulation.

B-D. PS1 cells were treated with FRS2 RNAi and stained with antibodies to FGFR1 and FGF2. Knock down of FRS2 leads to a significant reduction in both nuclear FGFR1 and FGF2 (quantified in C and D, respectively) C, *P=0.0112, **P=0.0012. Students t-test. Data summary represented by mean ± SEM.

E-F. Efficient knockdown of FRS2 after 72 hours was confirmed using Western blotting. Quantification of densitometry is shown in F.

E. G-H. PS1 cells were serum starved for 12 hours and subsequently stimulated with exogenous recombinant FGF2 (100μg/ml) for 2 hours. After 15 minutes of stimulation, there was a significant increase in both nuclear FGFR1 and FGF2 compared to serum starved cells. ***P<0.0001. Kruskal-Wallis test with Dunn's post-test comparison. Data summary represented by mean ± SEM

Scale Bar: 20 μm

Images are representative of at least three independent experiments