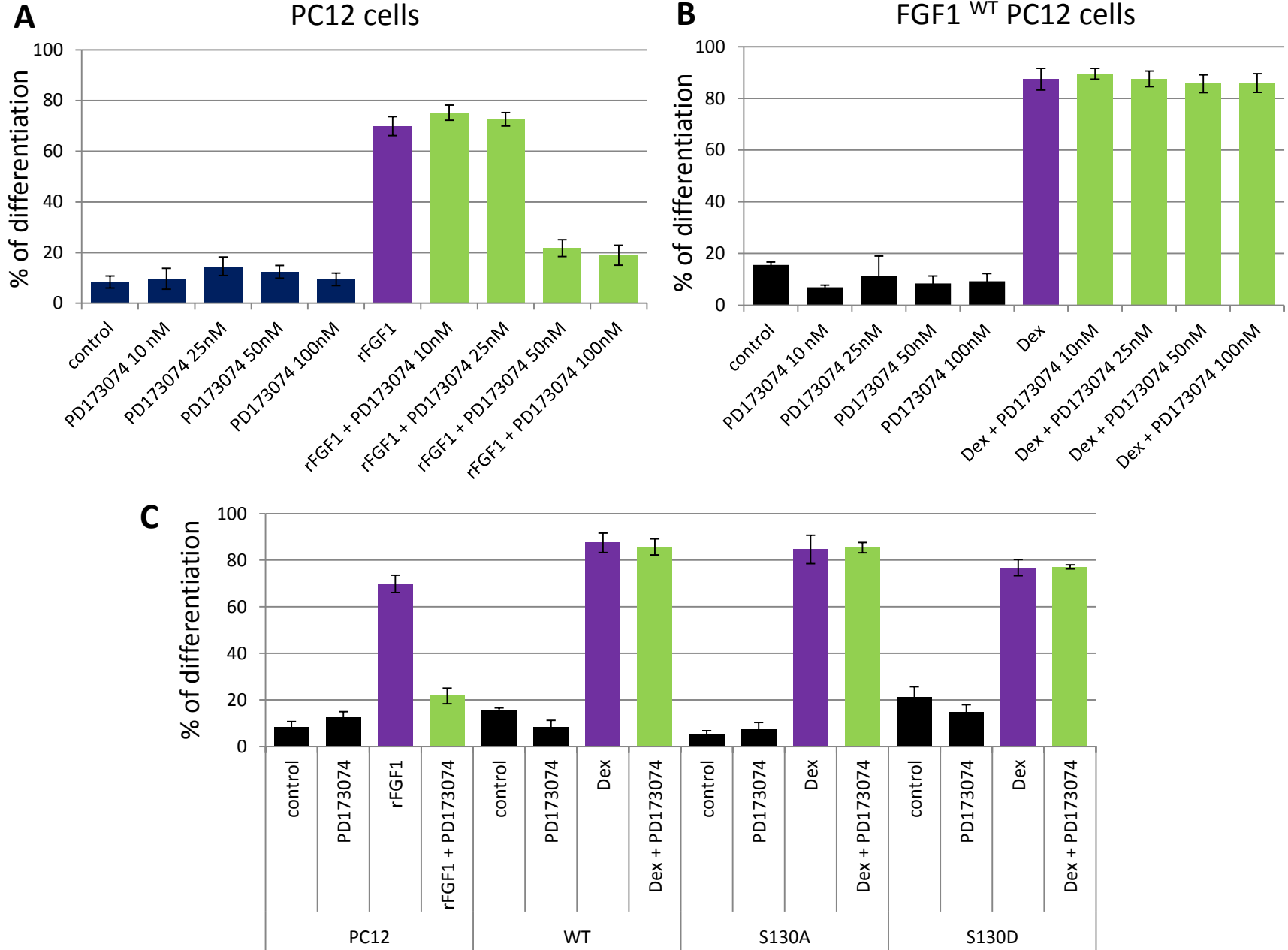
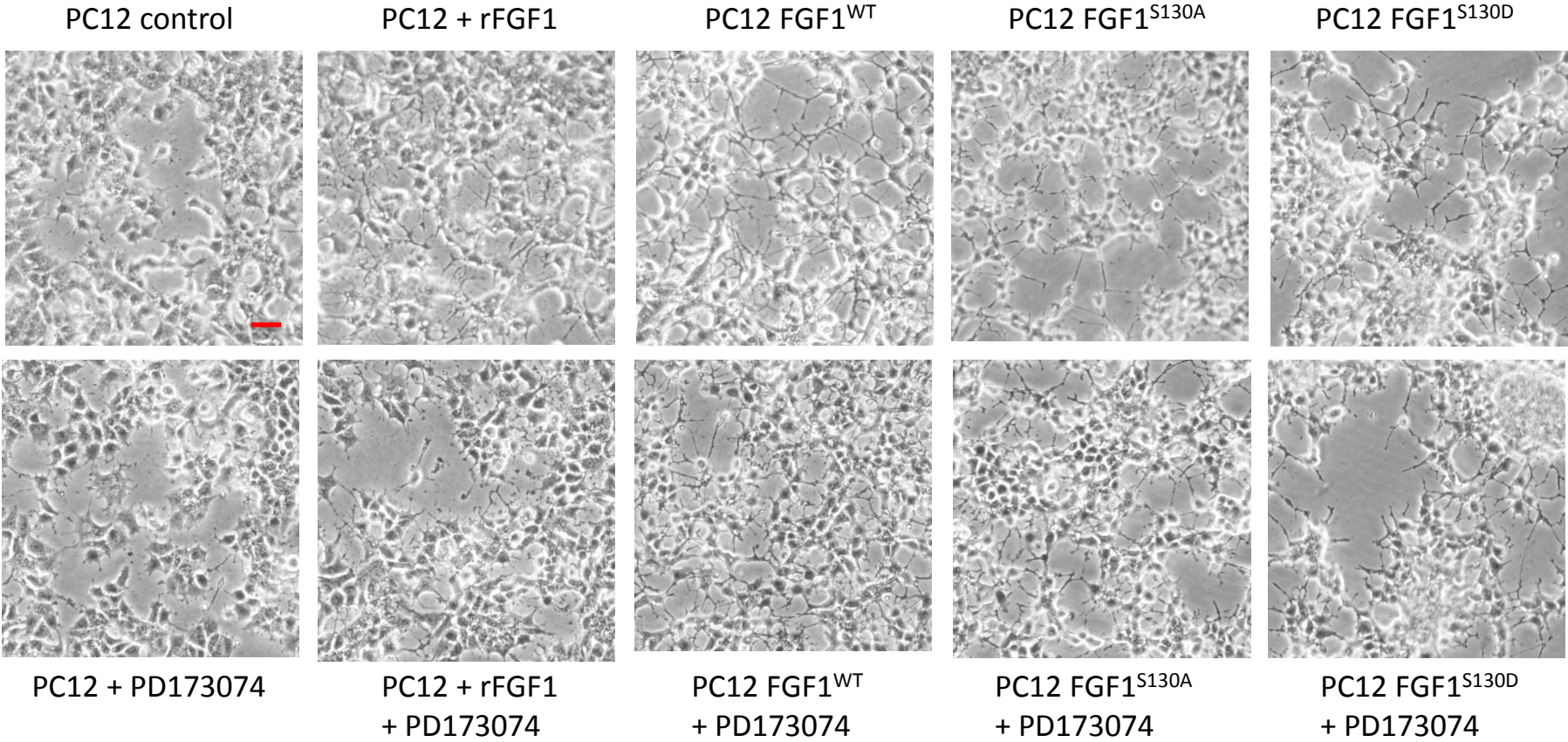


Supplementary Figure 1

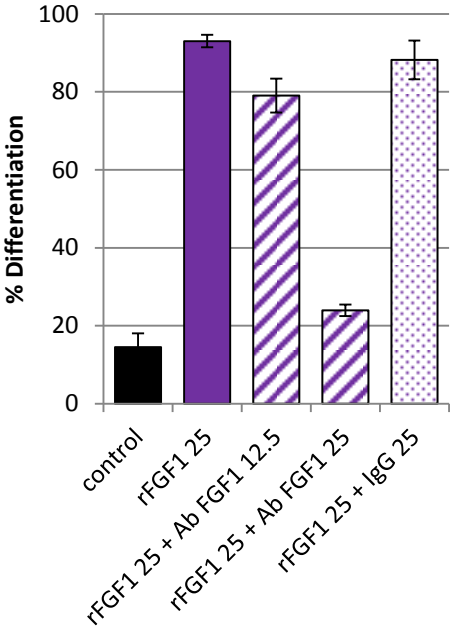


Supplementary Figure 2

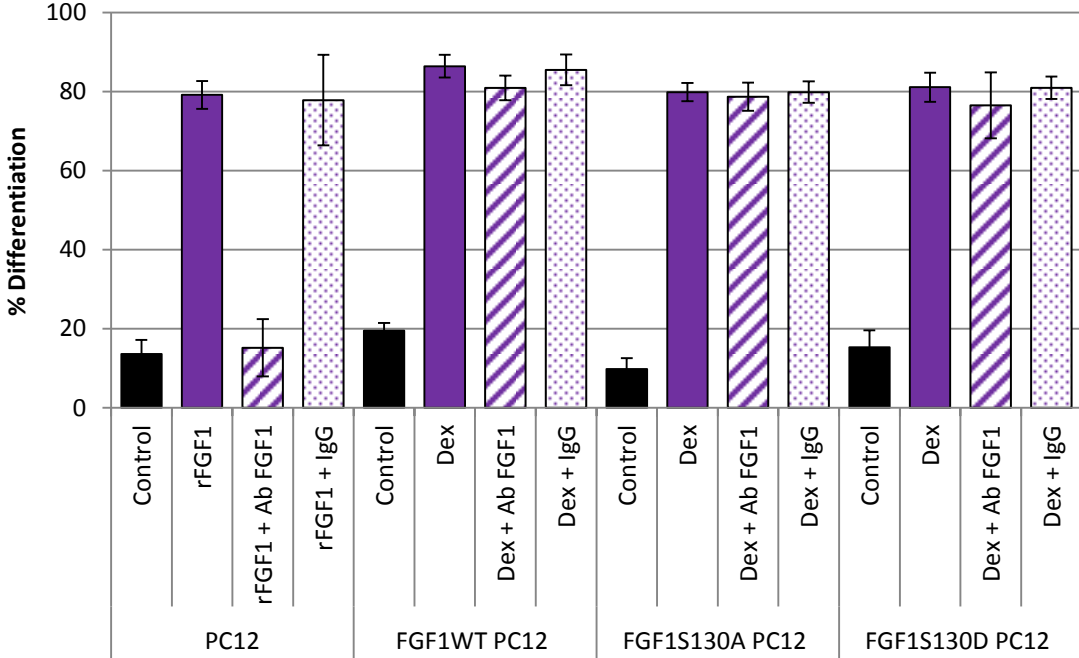


Supplementary Figure 3

A PC12 cells



B FGF1^{WT}, FGF1^{S130A}, FGF1^{S130D} PC12 cell lines



Supplementary Figure 4 (A)

A

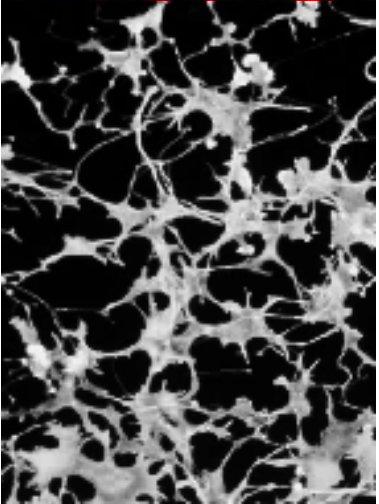
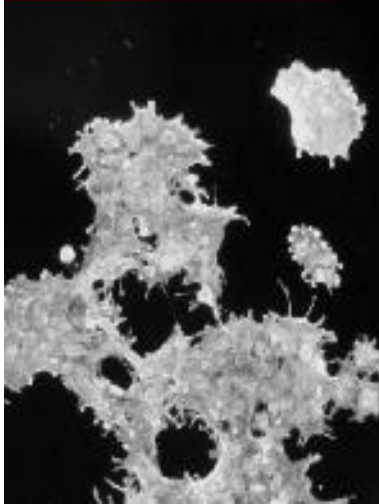
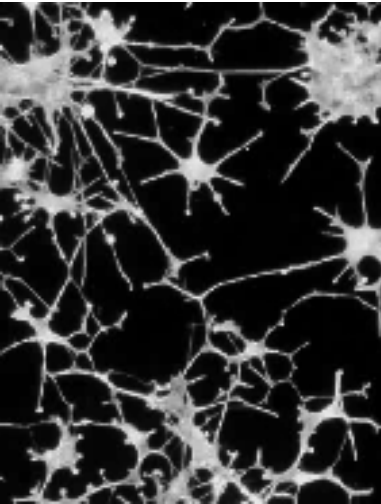
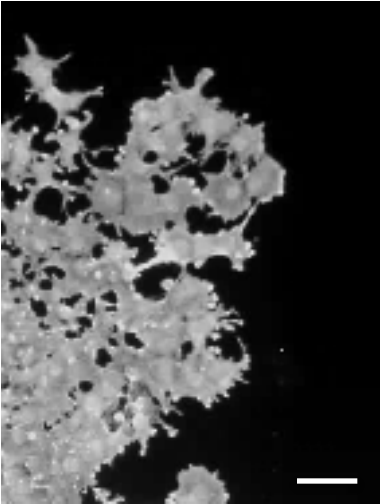
Control

rFGF1

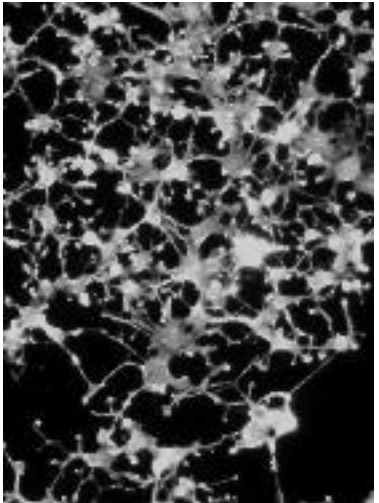
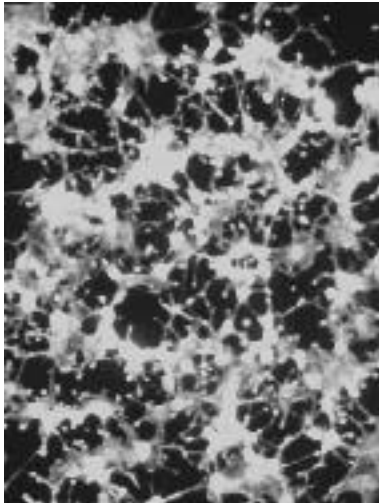
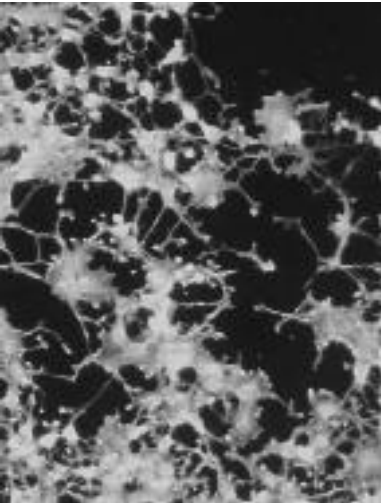
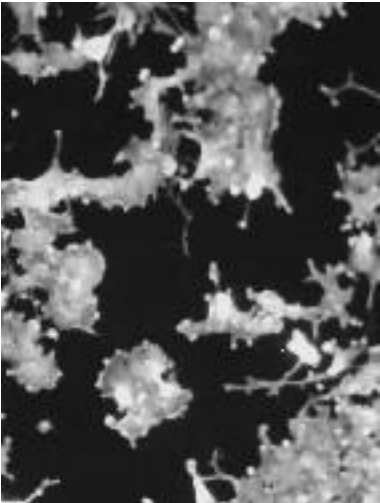
rFGF1 + Ab FGF1

rFGF1 + IgG

PC12



FGF1^{WT}



Control

Dex

Dex + Ab FGF1

Dex + IgG

Supplementary Figure 4 (B)

B

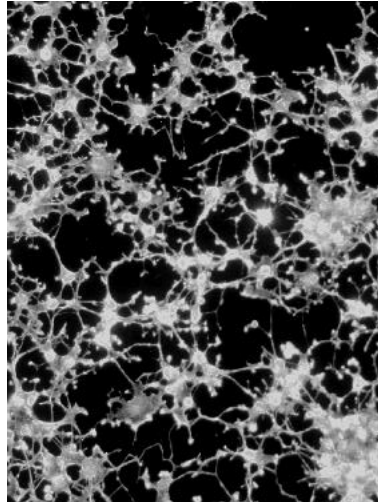
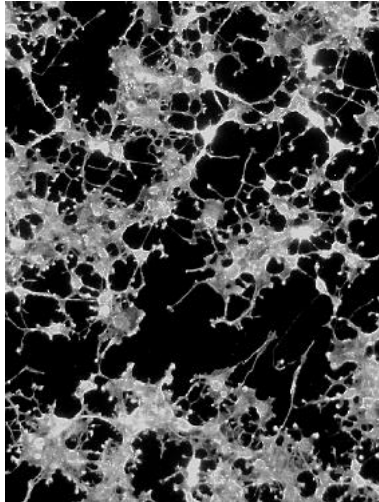
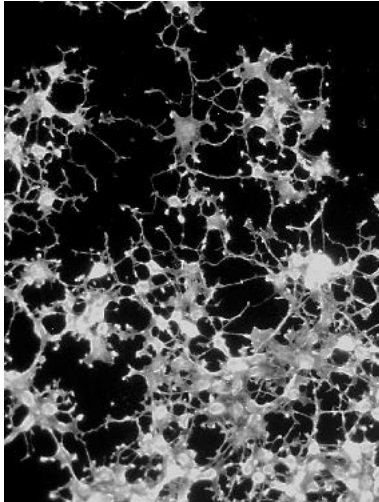
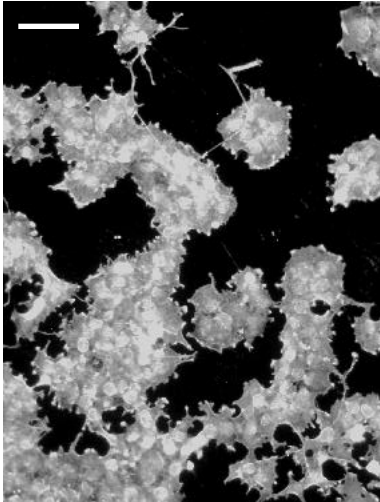
Control

Dex

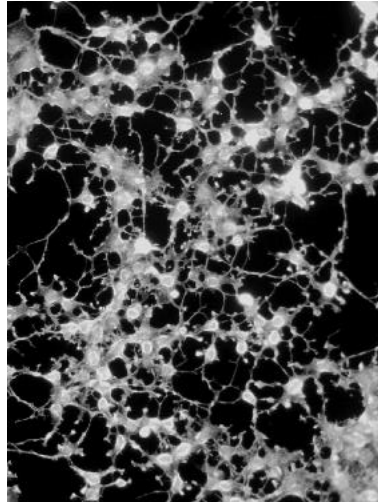
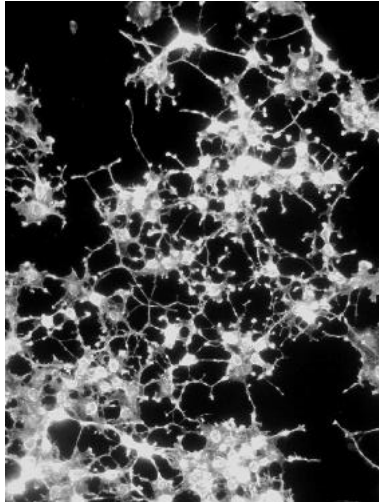
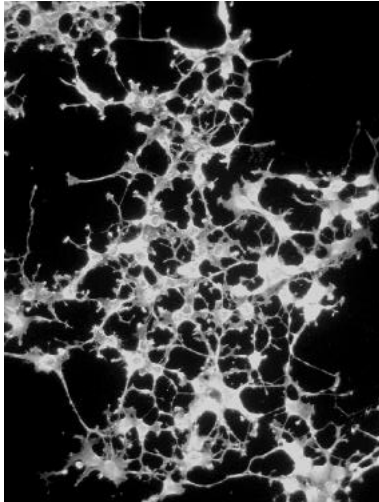
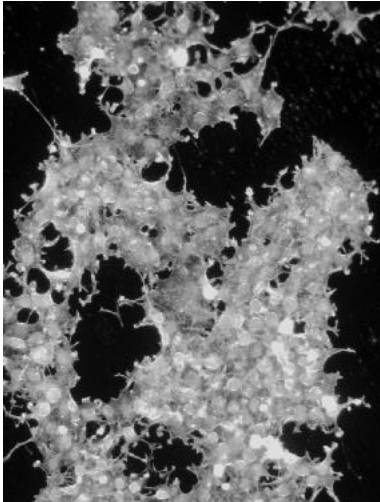
Dex + Ab FGF1

Dex + IgG

FGF1^{S130A}



FGF1^{S130D}



Control

Dex

Dex + Ab FGF1

Dex + IgG

Legends

Supplementary figure 1:

A) PC12 cells were treated with 0, 10, 25, 50 or 100 nM of PD173074 (an inhibitor of FGF receptors) in the absence or presence of 100 ng/ml of recombinant FGF1 and 10 µg/ml Heparin (FGF1 + Heparin is noted rFGF1).

B) FGF1^{WT} PC12 cells were treated with 0, 10, 25, 50 or 100 nM of PD173074 in the absence or presence of dexamethasone.

C) Native and FGF1^{WT}, FGF1^{S130A} and FGF1^{S130D} PC12 cells were treated with 50 nM PD173074, in the presence or not of rFGF1 (PC12 cells) or in the presence of 5 10⁻⁷M dexamethasone (FGF1 transfected PC12 cells).

For A-C, cell morphology was observed after 3 days of treatment and the percentage of cells presenting long and ramified neuritis was determined for each condition.

Supplementary figure 2:

Native, FGF1^{WT}, FGF1^{S130A} and FGF1^{S130D} PC12 cells were cultured in the presence or absence of 50 nM PD173074, with or without rFGF1 (PC12 cells) or in the presence of 5.10⁻⁷M dexamethasone (FGF1 transfected PC12 cells). After 3 days of treatment, cell morphology was observed by microscopy. Representative pictures are presented. Scale bar:50 µm.

Supplementary figure 3:

A) PC12 cells were cultured in 1µ-Slide 8 well ibiTreat (Ibidi GmbH) in the absence or presence of 25 ng/ml of recombinant FGF1 and 10 µg/ml Heparin (FGF1 + Heparin is noted rFGF1), 12.5 or 25 µg/ml of human FGF1 antibody (AF232, R&D systems, noted Ab FGF1) or 25 µg/ml of normal goat IgG control (AB-108-C, R&D systems, noted IgG). After three days of treatment, cells were fixed and permeabilized before performing an actin staining (Rhodamine-Phalloidin, Sigma, 1/200) in order to quantify the percentage of differentiation according to morphological criteria, *i.e.* cells presenting long and ramified neuritis.

B) Native and FGF1^{WT}, FGF1^{S130A} and FGF1^{S130D} PC12 cells were either cultured in the absence or presence of 25 ng/ml rFGF1 and 10 µg/ml of Heparin for native PC12 cells, or treated with 10⁻⁶M dexamethasone for FGF1 transfected PC12 cells. When mentioned, 25 µg/ml of Ab FGF1 or IgG were added. After two days of treatment and actin staining, cell morphology was observed by microscopy and the percentage of differentiation was determined.

Supplementary figure 4:

Native and FGF1^{WT} (A), FGF1^{S130A} and FGF1^{S130D} (B) PC12 cells were cultured either in the absence or presence of 25 ng/ml rFGF1 and 10 µg/ml of Heparin for native PC12 cells, or in the presence of 10⁻⁶M dexamethasone for FGF1 transfected PC12 cells. Cells were additionally treated or not with 25 µg/ml of Ab FGF1 or IgG. After two days of treatment and actin staining, cell morphology was observed by microscopy. Representative pictures are presented. Scale bar: 50 µm.