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Supplemental information

STAT3 silencing by an aptamer-based strategy hampers the crosstalk between NSCLC cells and cancer-associated fibroblasts

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Supplemental materials:

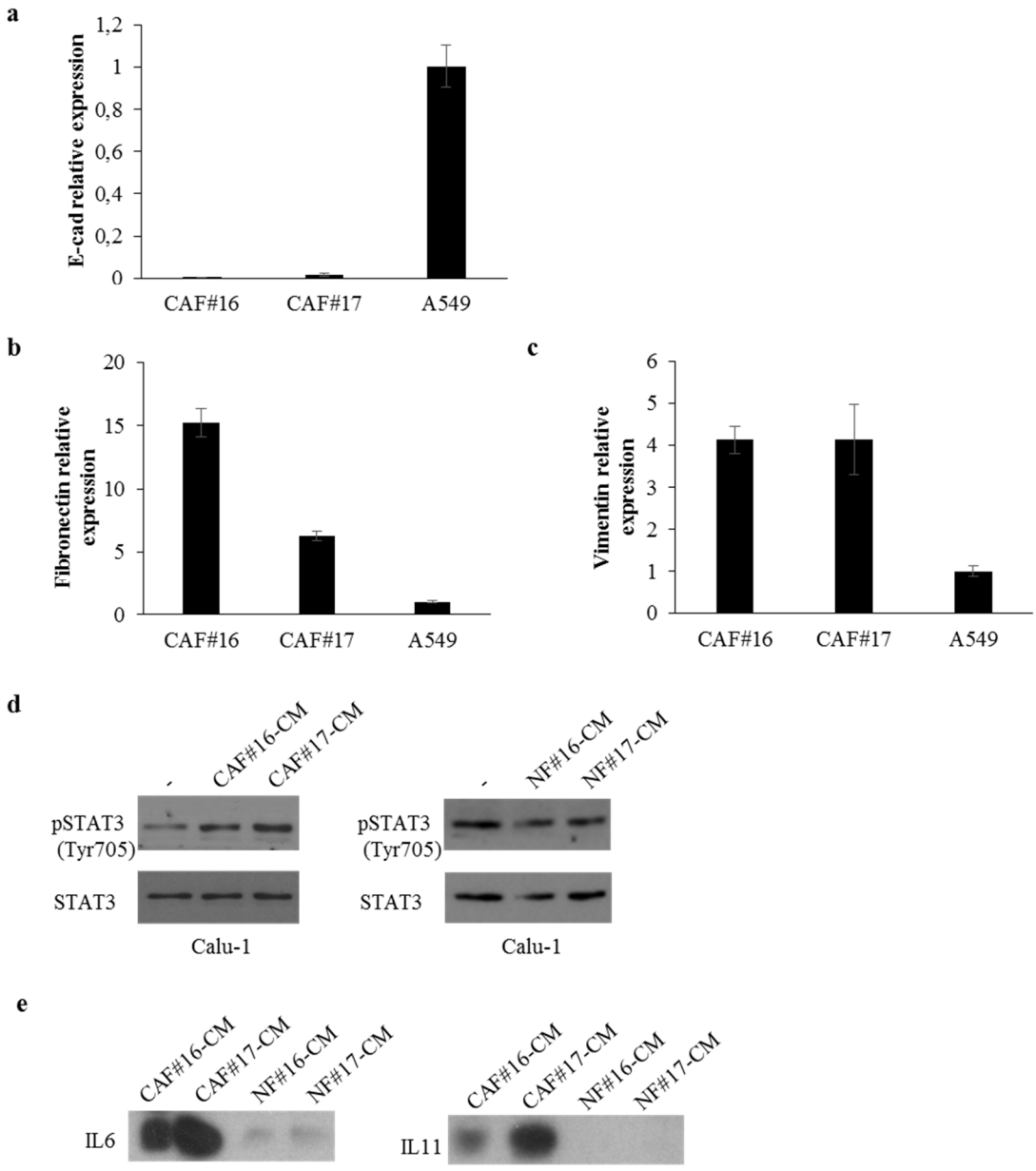

Figure S1. (**a-c**) Levels of E-cad, fibronectin and vimentin were measured by RTqPCR in the indicated cells. (**d**) Levels of phoshpo-STAT3 (pSTAT3) and STAT3 were analysed by immunoblot in Calu-1 cells grown in 1% FBS medium (-) or 1% FBS CAF or NF CMs for 72 hours. (**e**) Immunoblott analyses of IL6 and IL11 levels in CMs derived from indicated CAFs or NFs.

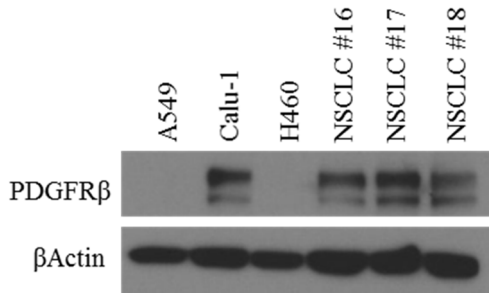

Figure S2. PDGFRβ levels were analyzed by immunoblot in the indicated cell lines. β-Actin antibodies were used as loading control.

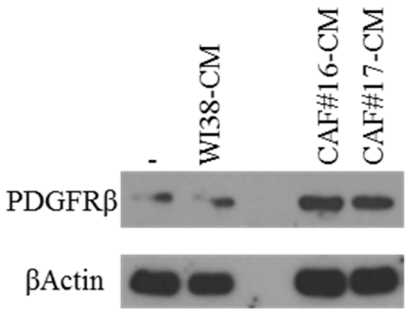

Figure S3. PDGFRβ levels were analyzed by immunoblot in Calu-1 cells grown in 1% FBS medium (-) or 1% CMs from the indicated cell lines. βActin antibodies were used as loading control.

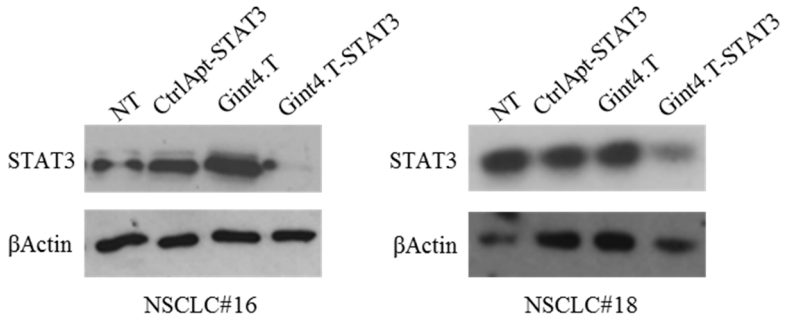

Figure S4. STAT3 levels were analyzed by immunoblot in NSCLC#16 and NSCLC#18 primary cells upon Gint4.T.STAT3 treatment (400nmol/L). βActin antibodies were used as loading control.

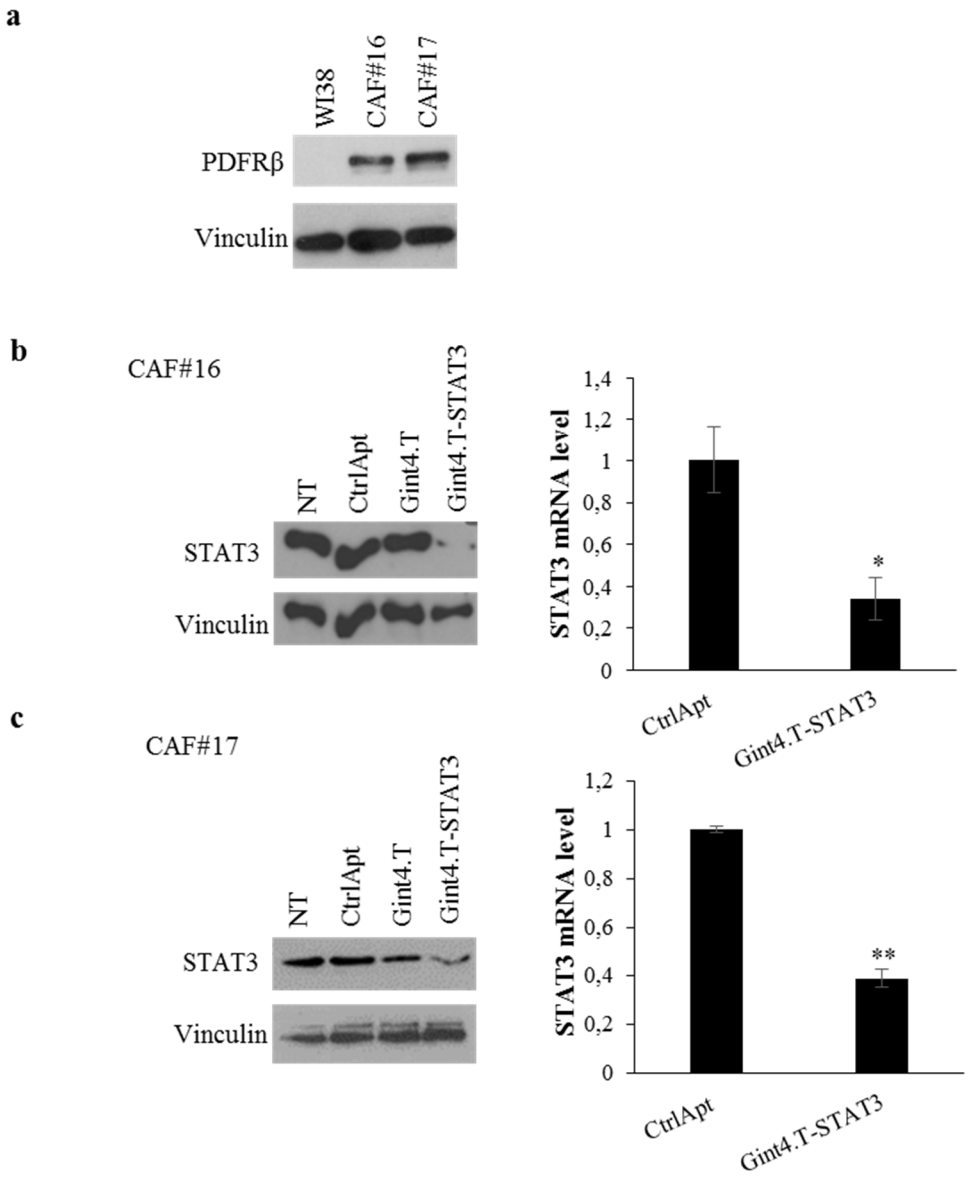

Figure S5. (**a**) PDGFRβ levels in the indicated cell lines. (**b**, **c**) STAT3 levels were analyzed by immunoblot (*left panels*) or RT-qPCR (*right panels*) in CAF#16 and CAF#17 primary cells left untreated or treated with 400 nmol/L CtrlApt, Gint4.T or Gint4.T-STAT3 as indicated. In (**a-c**) Vinculin antibodies were used as loading control