

## **IL-17A and its homologs IL-25/IL-17E recruit the c-RAF/S6 kinase pathway and the generation of pro-oncogenic LMW-E in breast cancer cells**

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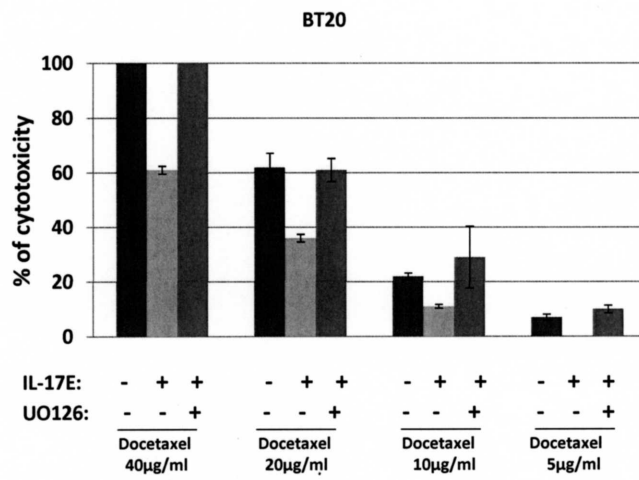
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**Conflict of interest:** G.A., N.B., J-F.E. and A.B are cofounders and shareholders of OREGA Biotech. JB is employee of OREGA Biotech and S.M., S.C., Y.M., C.G., F.A., E.L. and J.G. declare no conflict of interest

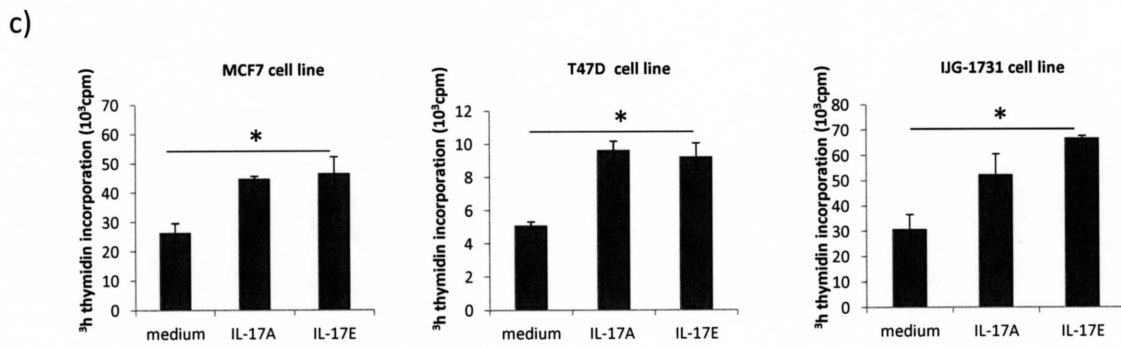
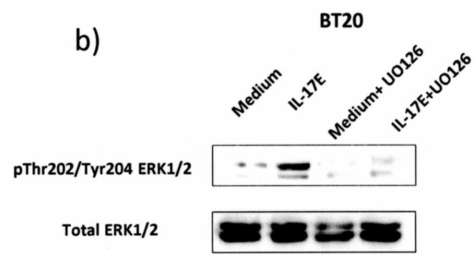
**Supp Figure 1: IL-17E-induced resistance to Docetaxel is dependent of ERK1/2 activation.** BT20 cells were stimulated with recombinant IL-17E at 10ng/ml alone or in association with the MEK inhibitor U0126 at 10 $\mu$ M 24h before adding the drug. Cell death is measured after 7h of culture. Data shown are representative of 2 independent experiments.

**Supp Figure 2: IL-17E induces the phosphorylation of ERK1/2 and cell proliferation** (A) Western blot analysis of pThr202/pTyr204 Erk1/2 was performed on MDA-MB468 stimulated for 20 min with recombinant IL-17A or IL-17E at 10ng/ml alone. (B) Western blot analysis of pErk1/2 was performed on BT20 cells stimulated for 30 min with recombinant IL-17E at 10ng/ml alone or in association with the MEK inhibitor U0126 at 10 $\mu$ M 24h before adding the drug. The figure shows one representative experiment from two independent experiments.

(C) MCF7, T47D, and IJG-1731 breast cancer cell lines were cultured in complete medium supplemented with 20ng/ml of recombinant human IL-17A or IL-17E. Cell proliferation was assessed at 72h using tritiated thymidine ([<sup>3</sup>H]-TdR) incorporation protocol. Data are the mean  $\pm$  SEM of two independent experiments, each performed in hexaplicates (\*  $P < 0.05$ ).



Supp Fig1: IL-17E-induced resistance to Docetaxel is dependent of ERK1/2 activation



Supp Fig2: IL-17E induces the phosphorylation of ERK1/2 and cell proliferation