"PANDER/FAM3B OVEREXPRESSION STIMULATES PROLIFERATION AND INHIBITS CELL DEATH IN MDA-MB-231 BREAST TUMOR CELLS"

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**Background:** PANDER/FAM3B (Pancreatic-derived factor) is a novel cytokine that induces apoptosis in pancreatic beta-cells and regulates the effects of insulin in peripheral tissues. Our previous results reveal that increased PANDER expression in DU145 prostate tumor cells inhibits cell death and promotes tumor growth. **Aims:** We evaluate the role of this cytokine in cell death and proliferation of a breast adenocarcinoma cell line. **Methods:** We used the breast tumor cell line MDA-MB-231-overexpressing PANDER and cells transfected with the empty vector as control. Cell proliferation was inferred by cell growth curve. Cell viability and apoptosis of MDA-MB-231 cells stimulated by several apoptosis inducers (TNF-α plus cycloheximide, etoposide, camptothecin) were measured by using MTT assay and trypan blue exclusion. The gene expression of Bcl-2 family members was quantified by real-time PCR and western Blot. Caspase-3 activity was measured by using fluorometric assays. **Results:** As shown by cell growth analysis, PANDER overexpression increased the rate of proliferation of MDA-MB-231 cells. Comparison of cell viability and DNA fragmentation analysis revealed that overexpression of PANDER inhibits significantly cell death induced by TNF-α plus cycloheximide and etoposide. Both proliferative and survival advantages provided by PANDER are accompanied by increasing expression of anti-apoptotic genes Bcl-2 and Bcl-XL and decreasing of caspase-3 activity. **Conclusions:** In agreement with previously demonstrated role in prostate tumor cells, PANDER also was capable to activates pro-survival mechanisms in MDA-MB-231 breast tumor cells. This antiapoptotic role involves, at least in part, the inhibition of caspase-3 proteolytic activity and the activation of Bcl-2-mediated pathways.