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Title: Investigate which PKC isoform play an important role in human lung cancer cells against TRAIL-induced apoptosis

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Body: Tumor nercrosis factor-related apoptosis-inducing ligand (TRAIL) is a promising anticancer agent as it selectively kills tumor cells but spares normal cells. Resistance to TRAIL by tumor cells limits its therapeutic use in clinic. TRAIL-induced apoptosis is mediated by binding with death receptors, DR4 and DR5, but the decoy receptors (DcR1 and DcR2) on the surface of tumor cells collapse TRAIL-mediated apoptosis. Recently, several articles have demonstrated that some tumor cell lines exhibit TRAIL-resistance and this resistance may be not associated with decoy receptors. Some receptors indicated that PKC activation was an important factor for cell to protect apoptosis from TRAIL. Therefore, we analyzed the sensitivity of nine human non-small lung cancer (NSCLC) cell lines to TRAIL. We found the expression of PKCε is higher in TRAIL-resistant cell lines (H1355 and H520) than those of in TRAIL-sensitive cell lines (H460 and H358). In order to investigate the role of PKCε in TRAIL-resistant cell lines, we used PKCε shRNA to block PKCε activity, and then observed their effect on TRAIL-induced apoptosis. Knockdown of PKCε expression by shRNA resulted in enhanced sensitivity to TRAIL in H1355 and H520 cell lines. Some references pointed out that PKCε is an oncogene. In this research, we investigated the role of PKCε gene in lung cancer cells. We found out when used PKCε shRNA to suppress PKCε expression, it would decrease cell growth and increase sensitivity to TRAIL-induced apoptosis. Finally, the similar results were observed in animal tumor model.