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## Abstract 2934: GSK3 $\beta$ modulates chemoresistance in epithelial ovarian cancer

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## Abstract

**Introduction:** Epithelial ovarian cancer is one of the most common gynecological malignancies and the fifth most frequent cause of cancer death in women, affecting over 22,000 women annually. Nearly 15,500 affected women die from this disease annually, and chemoresistance from the commonly prescribed platinum-based drug, carboplatin, is a major contributor to this mortality. Previous studies have identified genes with CpG islands that are methylated and transcriptionally silenced in resistant epithelial ovarian cancer patients. One of these genes is GSK3 $\beta$ , an important regulator of apoptosis and cell growth in the Wnt pathway. Thus, understanding the role of GSK3 $\beta$  suppression in chemoresistance of epithelial ovarian cancer can help contribute to more effective treatments for this disease. By performing different assays our study examined the functional role that GSK3 $\beta$  plays in carboplatin chemoresistance.

**Procedure:** Human ovarian surface epithelium (HOSE) 6-3 cell line was utilized, which is characterized as sensitive to carboplatin therapy. The cells were studied in six groups: 1) untreated; 2) treated with Lithium Chloride (LiCl); 3) treated with carboplatin; 4) treated with carboplatin and LiCl; 5) treated with doxorubicin as control; 6) treated with doxorubicin and LiCl as another control. LiCl is known to suppress GSK3 $\beta$  gene expression. We took images of cells using a fluorescence microscope. We also performed the Neutral Red Dye assay that determines cell viability, Vybrant $^{\text{®}}$  MTT Cell Assay which measures amount of non-viable cells, Caspase 3 Assay which measures cell apoptosis, and we did cell counting using a hemocytometer and a light microscope. Data analysis was done by T-tests using Microsoft Excel $^{\text{®}}$ , with  $p < .05$  for significance.

**Summary of Data:** More growth was observed in the carboplatin and LiCl group compared to the carboplatin group alone on microscopy. Neutral Red Dye assay: Compared to the cells exposed to carboplatin alone, those exposed to carboplatin and LiCl were more viable ( $p < 0.01$ ). Vybrant $^{\text{®}}$  MTT Cell Assay: the cells treated with carboplatin and LiCl showed lesser amounts of nonviable cells as compared to the carboplatin alone group ( $p < 0.01$ ). Caspase 3 Assay: the cells treated with carboplatin and LiCl were less apoptotic, compared to cells treated with carboplatin alone ( $p < 0.01$ ). Cell Counting: cells treated with carboplatin and LiCl had significantly more growth compared to the cells treated with carboplatin alone ( $p < 0.01$ ).

**Conclusion:** Our results show that cells with suppressed GSK3 $\beta$  had increased proliferation and reduced apoptosis, strongly suggesting that silenced GSK3 $\beta$  expression contributes to carboplatin resistance and GSK3 $\beta$  expression is vital to carboplatin chemosensitivity. Future in vivo studies could further investigate the role of GSK3 $\beta$  methylation to facilitate the design of potential genome-guided treatments for patients with chemoresistant epithelial ovarian cancer.