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Presentation Title:	Protein kinase D1 is associated with membrane trafficking of β-catenin
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Introduction and objectives: The cadherin-catenin complex of proteins plays a critical role in cell-to-cell adhesion and is strongly associated with cancer metastasis and progression. We have previously published that Protein Kinase D1 (PKD1), a serine/threonine kinase interacts with the E-cadherin-catenin complex . The PKD1-E-cadherin interaction results in E-cadherin phosphorylation by PKD1, increased cellular aggregation and decreased motility in prostate cancer (*Cancer Research* 2005, 65: (2), 483-492). In this study we specifically studied the PKD1- β -catenin interaction in prostate cancer.

Methods: PKD1- β -catenin interaction and co localization in prostate cancer cell line and human tissue was determined by confocal Laser Scanning Microscopy (LSM) and/or immunoprecipitation (IP) assays. GST fusion protein pull-down assays were used to demonstrate a direct interaction of β -catenin and PKD1. C4-2 cells were transfected with green fluorescent protein (GFP) fused PKD1 to study the effect of PKD1 activation by Bryostatins 1 on β -catenin localization. Further colocalization experiments were carried out using Golgi and vesicular transport specific antibodies (Golgi Sampler Kit, BD Transduction laboratories, Palo Alto, CA) to study the influence of PKD1 on β -catenin Golgi to membrane trafficking.

Results: The immunoprecipitation experiments performed on E-cadherin positive (LNCaP) and negative (JCA) cancer cell lines suggested cadherin independent PKD1- β -catenin interaction. The GST pull down assay demonstrated direct interaction of PKD1-GST -fusion protein to β -catenin from SW 480 cytoplasmic cell lysates. Co localization experiments demonstrated that PKD1 co-localizes with β -catenin at cell junctions in LNCaP cells and in all five human prostate samples studied. In addition, PKD1 and β -catenin colocalized at trans-Golgi, transport vesicles, and plasma membrane, suggesting that PKD1 is associated membrane trafficking of β -catenin. Activation of PKD1 by Bryostatins 1 caused translocation of β -catenin to the membrane with a consequent decrease in nuclear β -catenin, suggesting a functional role for PKD1- β -catenin interaction in prostate cancer.

Conclusion: Our study has identified a novel and direct interaction between PKD1 and β -catenin resulting in membrane trafficking of β -catenin. This interaction may be functionally important in prostate cancer because PKD1 activation influences membrane trafficking and nuclear localization of β -catenin.

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