Introduction

- Breast cancer is the most common cancer in women. There were about 249,260 new diagnosed cases in 2016 (Siegel, et al., 2016).
- It has been shown that NDPK-A/B is secreted from breast cancer cell lines but not normal breast cells (Yokdang, et al., 2011). However, the process of NDPK release is not known.
- Exosomes are secreted by cells and can act as signaling mediators on other cells or tissues of the body. NDPK has been shown to be one of the many cargos found within exosomes (Kruger, et al., 2014; Palazzo, et al., 2012).
- NDPK-A/B stimulates P2Y2 receptors (by generating ATP and preserving ADP levels) which transactivates VEGFR-2 even in the absence of VEGF to promote angiogenesis by endothelial cells.

Results

- **Conclusions/Future Directions**
  - Transmission electron microscopy was able to confirm the size and cell surface markers of the secreted exosomes.
  - NDPK-B concentrations based on activity were significantly higher in 231 exosomes than HME1 exosomes.
  - Ellagic acid inhibited NDPK-B activity in the exosomes.
  - Purified exosomes from 231 cells associated with endothelial cells and may represent a mechanistic effect on angiogenetic signaling.
  - NDPK-B induced tubulogenesis was inhibited with 10 µM MR257179 and ellagic acid.
  - NDPK is an exosomal cargo released from breast tumor cells and if found circulating in the blood stream would suggest a role for targeting the metastatic niche before or during metastatic spread.
  - Future directions for this study include:
    - Generating an NDPK-A/B knockout cell line to understand its role in tumorigenesis.
    - Development of a more sensitive immunooassay to quantify secreted NDPK-A/B.
    - Absolution quantitation of NDPK in the exosomes.

References