ABSTRACT

Ovarian cancer is the fifth leading cause of cancer deaths in women and the most lethal of the gynecological cancers. A cancerous phenotype may be promoted by inappropriate activation of developmental regulatory pathways. Currently, there are very limited ovarian cancer models. The Draper lab has shown that fibroblast growth factor 24 (Fgf24) is required for proliferation, morphogenesis, and differentiation of the early somatic gonad. Fgf24 signaling downstream leads to MAPK and Etv4 activation, both of which are known to be dysregulated in cancer. In addition, Fgf receptor overexpression correlates with poor ovarian cancer prognosis.

METHODS

a) Step 1: Constructing "Entry" plasmids

b) Step 2: Recombining "Entry" plasmids into a "Destination" plasmid

RESULTS

CONCLUSIONS

Thanks to Bruce Draper for allowing me to work in his lab, Matt McFaul for lab mentorship and figure preparation, and Dena Leerberg for edits and revisions. Funding for this project was provided by the NIH via the STAR (Students Training in Advanced Research) Program.

ACKNOWLEDGMENTS

CITATIONS