

## Undergraduate Research Symposium May 16, 2014 Mary Gates Hall

### Online Proceedings

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#### POSTER SESSION 2

Commons East, Easel 60

1:00 PM to 2:30 PM

##### **Dickkopf Related Protein-1 Attenuates Connective Tissue Growth Factor Induced Fibrogenic Responses in Pericytes by Multiple Signaling Mechanisms**

*Wen Ying (Joanne) Lee, Senior, Biochemistry*

*Mentor: Jeremy Duffield, Medicine*

*Mentor: Shuyu Ren, Division of Nephrology*

*Mentor: Bryce Johnson, Medicine-Nephrology*

Pericytes are mesenchymal cells that are attached to peritubular capillaries. The activation and differentiation of progenitor pericytes to myofibroblasts leads to matrix deposition, renal scarring, and is a major contributing factor leading to chronic kidney disease after injury. Connective tissue growth factor (CTGF) plays an important role in fibrogenesis by promoting the production and secretion of extracellular matrix components. The goal of this project is to dissect the contribution of CTGF/Wnt signaling pathways in pericytes during the development of kidney fibrosis and to examine the effect of using the canonical Wnt extracellular regulator Dickkopf related protein-1 (DKK-1) on CTGF/Wnt signaling. In-vitro signaling studies on purified kidney pericytes, migration assays, and immunological staining were used to characterize the roles of DKK-1 and CTGF in the regulation of renal fibrosis. Results show that CTGF induces Wnt/ $\beta$ -catenin signaling in pericytes. DKK-1 inhibits CTGF induced fibrotic gene activation, changes the morphology, and the capability of migration of kidney pericytes through JNK MAP kinase, and canonical WNT independent pathway.

MicroRNAs bind to target mRNAs and can inhibit translation and/or facilitate degradation. One of these miRNAs, microRNA-21, plays an important role in kidney disease as it has been shown to increase in animal models of disease. Recent studies have shown that microRNA-21 promotes interstitial kidney disease with fibrosis by silencing metabolic pathways and by promoting reactive oxygen species formation. Mice with microRNA-21 deletion show a decrease in kidney fibrosis. Our hypothesis is that cells isolated from microRNA-21 knock out mice will have decreased expression of cytokines associated with fibrosis. I will look at the expression of reactive oxygen species by live imaging of cells where microRNA-21 has been blocked. Then I will use quantitative polymerase chain reaction and Western blots to analyze protein levels for known molecules associated with fibrosis and inflammation. We expect to see that cells treated with anti-microRNA-21 have a reduction in reactive oxygen species, leading to less fibrosis and inflammation. Blocking microRNA-21 is a potential new therapy to combat kidney disease.

#### POSTER SESSION 3

Commons West, Easel 22

2:30 PM to 4:00 PM

##### **The Role of MicroRNA-21 in Kidney Fibrosis**

*Essence Brandon (Essence) Underwood, Senior, Psychology, Biology (General)*

*Mentor: Ivan Gomez, Nephrology*

*Mentor: Jeremy Duffield, Medicine*

MicroRNAs are a group of highly conserved non-coding RNAs that play critical roles in regulating networks of genes.