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Minu Pilvankar* (minu.pilvankar@okstate.edu), 127 N Duck St, Apt F13, Stillwater, OK 74075, and **Ashlee N Ford-Versypt** and **Michele A. Higgins**. *Mathematical Modeling of Glucose Dependent Renin-Angiotensin System in Podocytes in Diabetic Kidney Disease*. Preliminary report.

Diabetic kidney disease (DKD) is the primary cause of end-stage renal failure. Hyperglycemia is known to initiate and worsen the pathophysiology of DKD. Podocytes are terminally differentiated renal cells, vital for maintenance of the renal filtration barrier. Podocytes express a local renin-angiotensin system (RAS) that is altered in hyperglycemia. Studies have shown that a RAS peptide, angiotensin II (ANG II), is modulated in hyperglycemic conditions, triggering podocyte loss. DKD progression can be slowed by controlling the ANG II levels to prevent irreversible podocyte loss. However, experimental evidence for glucose-dose-dependency of ANG II is scarce in the literature. Hence, we use mathematical modeling for a better insight into the underlying mechanism. The model describes local RAS network by a system of ordinary differential equations that triggers the synthesis of ANG II. Different parameterization approaches were analyzed to add glucose-dependency through the parameters. Sensitivity analysis was conducted to identify key ANG II-modulating biomarkers. The model was used to study the change in ANG II concentrations with varying glucose levels. The model can be used to study the effect of different ANG II modulating therapies which could be useful for drug development. (Received September 19, 2016)