“Medicine of the Future”

Kidney disease, diabetic kidney disease (DBK) more specifically, is a much too common phrase for many of us. as the effects of DBK gradually take on the kidney, once the disease has affected the kidney there is not a reversible cure. As the leading cause of kidney failure, DBK doesn’t solely affect the kidneys alone. Diabetes are a major concern in the daily life of many and the damage during these high glucose level spikes are not yet specified at a cellular level. It is understood that over time these high levels of glucose cause damage to the small blood vessels in the kidney, where the small filtration units, glomerulus, filter our blood. As these units are damaged and die off, the others are capable of compensating to maintain efficiency, but over time can these units can overcompensate and become damaged as well. But what exactly is causing this damage to the filtration units, and what role does glucose play in? What processes or pathways are being subjected to change; and what does this mean for the cells, tissue, and body as a whole? Dr. Ford Versypt, a faculty member in the Chemical Engineering department at Oklahoma State University, is taking a closer look into the glucose dependent effects on the kidney.

Dr. Ford Versypt’s recent scientific publishing “Mathematical Model for Glucose Dependence of the Local Renin–Angiotensin System in Podocytes”, explains the role of podocytes on Diabetic Kidney Disease. Dr. Versypt walked me through the project and thoroughly informed me on the background of her research as well as the future plans. Because the kidney serves many functions to the body its complexity makes it difficult to determine exactly the cause and effect of the damage. Dr. Ford Versypt used her prior knowledge of kidney disease and delved further into areas of DBK that have not been extensively researched.

Podocytes are specialized cells within the filtration units of the kidney (2). They act as one of the layers responsible for filtering waste out of the blood. Wrapping around the small capillaries, they link together like fingers meshing together, leaving small slits between them for small molecules to filter through. When these cells are damaged or die, they break off, leaving a gap in the filter. This allows bigger molecules to pass through that are not supposed to, leading to proteins being present in the urine. Knowing that these podocytes had recently been found to be affected in high glucose levels, and also these cells being the last line of filtration, Dr. Ford Versypt started here.

Podocytes possess a system that produces a hormone called angiotensin II (ANG II). It has been found that high glucose levels elevate ANG II concentration; triggering podocyte injury (1). Blocking this system and decreasing ANG II could serve as a potential pharmaceutical treatment method. This research looked to quantify glucose dependency of ANG II, creating a model that allows us to predict ANG II concentrations at varying glucose levels (1). By selecting specific parameters, Dr. Ford Versypt was able to generate a sensitivity analysis; giving us the capability to study the impact of pharmaceuticals on ANG II (1).

By looking into these specific cellular pathways at a molecular level, and the cascade of effects they produce on the body, Dr. Ford Versypt hopes “to bridge the gap between chemical understanding and medical understanding”. She emphasized to me that she was really interested in how the effecting damage on specific cells holistically affects the rest of the body. Moving forward the same holds true, attending the Chronic Kidney Disease Drug Development Summit this summer in Boston; Dr. Ford Versypt hopes to expand her breadth and knowledge on kidney disease, while creating new networking and collaboration opportunities for the future.

References

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2. Reiser, Jochen, and Mehmet M. Altintas. “Podocytes.” *F1000Research* 5 (2016): F1000 Faculty Rev–114. *PMC*. Web. 15 Apr. 2018.