Public Abstract

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Title: Glomerular Deposition of Homotrimeric Type I Collagen in the COL1A2 Deficient Mouse

Type I collagen is the most abundant structural protein in the body. Type I collagen generally exists as a heterotrimeric protein; however, a homotrimeric isotype of type I collagen has been identified. Our lab currently studies the COL1A2 deficient mouse model which is homozygous for a mutation that causes the mice to synthesize homotrimeric type I collagen exclusively. We recently identified deposition of type I collagen in the kidneys of the COL1A2 deficient mouse model and under normal physiologic conditions such accumulation is pathologic.

The primary goal of this research is to investigate the molecular mechanisms involved in the regulation of type I collagen within the kidney by evaluating synthetic and degradative pathways. As presented here, the accumulation of homotrimeric type I collagen in the kidneys of the COL1A2 deficient mice occurs postnatally and appears to be due to an increase in the synthetic pathway, as well as an alteration in the degradative pathway.

Gaining a further understanding of the mechanisms behind the deposition of collagen within the kidney will hopefully lead to a greater understanding of secondary renal injury seen in the progression to end- stage renal disease, a complication of a variety of diseases, including diabetic and IgA nephropathy. Further, characterization of the role of homotrimeric type I collagen will also lead to a greater understanding of its role in developmental and pathological events.