Inhibiting the Effects of Fetuin-B Upregulation Using TAK-242

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Low birth weight (LBW) offspring have a high risk of developing cardiovascular and renal diseases such as chronic kidney disease and hypertension, which have no preventative measures or cures. Previous research showed that the protein fetuin-B increases pro-inflammatory cytokine concentration, preventing normal nephrogenesis. Research has also shown that the protein TLR-4 promotes pro-inflammatory cytokine concentration, similarly to fetuin-B, and that TAK-242 is a potential TLR-4 inhibitor. The goal of this study was to determine if treatment with fetuin-B causes glomerular degradation and if treatment with both fetuin-B and TAK-242 prevents such degradation. The hypotheses were that treatment with fetuin-B would cause a significant decrease in glomeruli count and treatment with fetuin-B and TAK-242 would not. To test the hypotheses, the glomeruli of kidneys treated with either fetuin-B or fetuin-B and TAK-242 counted. Fetuin-B treatment caused a -59% change in glomeruli count and treatment with both fetuin-B and TAK-242 caused only a -15% change. The hypotheses were supported: the fetuin-B group experienced a significant attenuation in glomeruli count in comparison to controls ($p<.0005$), while the TAK-242 + fetuin-B group did not ($p>0.05$). In the future, TAK-242 treatment during pregnancy could be used to prevent LBW offspring from developing cardiovascular and renal diseases.

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