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Title: **Inhibiting the Effects of Fetuin-B Using TAK-242**

Abstract:

Low birth weight (LBW) offspring have a high risk of developing cardiovascular and renal diseases such as chronic kidney disease and hypertension. 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, embryonic kidney explants were cultured with either fetuin-B or fetuin-B and TAK-242 and then their glomeruli were 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 ($p = 0.4$). In the future, TAK-242 treatment could be used to prevent LBW offspring from developing cardiovascular and renal diseases during adulthood.