**Assessment of NF-κB-SN50 Effect on Adipose TNF-α and AGT Secretion and Expression as A therapeutic Agent**

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**Introduction**

Pro-inflammatory cytokines and adipokines upregulation through NF-κB activation in adipose tissue has been considered to have an important function in the pathogenesis of obesity-related hypertension. This study was aimed to ascertain the effect of NF-κB inhibitor, (SN50) on TNF-alpha and AGT secretion and expression in mediating the anti-inflammatory effect through its effects on NF-κB activity in human adipose tissue.

**Methods**

Primary human adipocytes were isolated from 20 subjects among 10 overweight and 10 obese with and without hypertension, cultured, then treated with 10 ng/ml LPS, with and without NF-κB inhibitor, SN50 (50 μg/ml) at different time points. TNF-α secretion and NF-κB p65 activity were detected in supernatants extracted from cultured cell treated and untreated with LPS and SN50 by ELISA. NF-κB p65, TNF-α and AGT proteins expression were detected by western blot. TNF-α and AGT gene expression was detected in cells and performed using quantitative real-time PCR. The study was carried out at the Obesity Research Center, King Saud University, Riyadh, KSA.

**Results**

Treatment of AbdSc adipocytes with LPS caused a significant increase in NF-κB p65 in overweight and obese, while, SN50-NF-κB inhibitor causes a reduction of NF-κB p65 in overweight and obese persons at all time points. Treatment of AbdSc adipocytes with LPS caused a significant increase in TNF-α secretion in overweight and obese subjects at all time points, whereas, SN50 leads to a decrease in TNF-α secretion at 3 and 12 hours. Treatment of AbdSc adipocytes with LPS caused increased TNF-α and AGT gene expression twofold compared with untreated cells, whereas, in the presence of SN50, it reduces mRNAAGT levels in both groups.

**Conclusion**

Taken together these adipokines with NF-κB activation may represent important biomarkers to evaluate hypertension risk, as well as provide a mechanistic insight into the pathogenesis of obesity-related hypertension.