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Effect of chronic Angiotensin II infusion on plasma TNFalpha and IL 6 levels in adiponectin deficient mice

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Chronic low grade inflammation plays an integral part in the development of many types of cardiovascular diseases. Adipose tissue is a well known source of many proinflammatory mediators. Adiponectin (APN) is one of the few proteins secreted by the adipose tissue that is known to have anti-inflammatory properties. Our research goal is to understand if APN deficiency contributes to a proinflammatory milieu especially when provided with an appropriate stimulus. Angiotensin II (ANGII) is a well known prohypertensive agent. Recently, ANGII has emerged as a growth factor and stimulator of proinflammatory cytokine expression in cardiovascular & renal tissue. The present study evaluates the effect of chronic Angiotensin II infusion on plasma TNFalpha and IL 6 levels. Briefly, male C57BL/6J mice and Adiponectin deficient mice (adipo -/-) were implanted with osmotic pumps containing Angiotensin II (ANGII, 800ng/Kg body weight/min) or Saline. Plasma levels of TNFalpha and IL6 were measured using enzyme linked immunoabsorbent assay (ELISA) kits. After two weeks of chronic infusion of ANGII, plasma TNFalpha levels in adiponectin deficient mice (n=6, 11.85±1.54 pg/ml) were not significantly different from the control C57Bl/6J mice (n=6, 9.88±1.17pg/ml). Plasma IL6 levels were also not significantly different in the two groups. In conclusion, adiponectin deficiency did not result in a proinflammatory milieu in the circulation even in the presence of a inflammatory stimulus such as ANGII.