**Chronic Intermittent Hypoxia Conditioning Augments Decrements in Renal Microcirculatory Perfusion During Asphyxia**

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Sleep apnea (SA) is highly prevalent in patients with chronic kidney disease and may contribute to the development and/or progression of this condition. Previous studies suggest that dysregulation of renal hemodynamics and oxygen flux may play a key role in this process. The present study sought to determine how chronic intermittent hypoxia (CIH) associated with SA affects regulation of renal microcirculatory perfusion (RP) and cortical and medullary tissue PO2 as well as expression of genes that could contribute to renal injury. We hypothesized that normoxic tissue PO2 would be reduced after CIH relative to baseline, and that RP and tissue PO2 would be decreased to a greater extent in CIH vs sham during exposure to intermittent asphyxia (IA, FiO2 0.10/FiCO2 0.03). Additionally, we hypothesized that gene programs promoting oxidative stress and fibrosis would be activated by CIH in renal tissue. All physiological variables were measured at baseline (FiO2 0.21) and during exposure to 10 episodes of IA (excluding GFR). Normoxic renal tissue PO2 was significantly lower in CIH vs sham ($p<0.05$). Reductions in RP and renal tissue PO2 during IA occurred in both groups but to a greater extent in CIH ($p<0.05$). Pro-oxidative and pro-fibrotic gene programs were activated in renal tissue from CIH but not sham. In conclusion, CIH adversely affects renal microcirculatory perfusion and oxygen flux during both normoxia and IA and results in changes in renal tissue gene expression. Supported by R15 HL138600-01 and MSRP.