

HIBBA CHAUDHRY, MUHAMMAD CHAUDHRY, JUSTIN CHUANG, FANG BAI, YING NIE, KOMAL SODHI, JIANG LIU, AND JOSEPH I. SHAPIRO. Marshall University JCE School of Medicine. Huntington, WV 25755, Effects of pNaKtide in 5/6 Partial Nephrectomy mouse model having benefits on Red Blood Cells survival and Cardiac Function.

We have previously shown that  $\text{Na}^+/\text{K}^+$ -ATPase has a signaling function in addition to its pumping function. Interstitial fibrosis in the heart and kidney are hallmark of pathological changes in Chronic Kidney Failure (CKF) patients with Cardiovascular complications. We have also demonstrated that this signaling function amplifies oxidants and increases cellular oxidant stress; conversely the blockage of this signal cascade with a designed peptide, pNaKtide, attenuates oxidant stress. Objective of this study was to investigate the effect of pNaKtide on our mouse model of CKF with 5/6 partial nephrectomy.

We randomly divided animals into 4 groups Sham, PNx, Sham+pNaKtide, and PNx+pNaKtide. Biotin labeled Red Blood Cells (RBCs) were detected using Streptavidin-PE in flow cytometry.

Our results show that pNaKtide ameliorates the phenotypical changes in experimental CKF 5/6 partial nephrectomy (PNx) mouse model. Our PNx model showed non-iron deficient anemia without elevation in the blood pressure compared to Sham surgery animals. Administration of pNaKtide in sham and PNx mice induced an increase in the RBCs half-life and reduced fibrosis in the heart and kidney, which are statically significant.