The nitric oxide (NO)-soluble guanylate cyclase (sGC)-cyclic guanosine monophosphate signal transduction pathway is impaired in different cardiovascular diseases including pulmonary hypertension, heart failure and systemic arterial hypertension.

We thus investigated the cardio-renal protective effects of riociguat in two rat models of systemic hypertension: a low-renin model (5/6 nephrectomy and sham-operated controls (Sham-op). Data are expressed as means + standard error of the mean. *p < 0.05 vs control; †p < 0.05 vs riociguat low dose; ‡p < 0.05 vs riociguat high dose; ††p < 0.05 vs riociguat low dose; ‡‡p < 0.05 vs riociguat high dose; †††p < 0.05 vs 5/6 NX; ‡‡‡p < 0.05 vs Sham-op. Results

Systolic blood pressure increased markedly in both uraemic groups compared with sham-operated controls (5/6 NX + riociguat) and sham-operated controls (Sham-op; n = 10) at study end (18 weeks). Data are expressed as means ± standard error of the mean. *p < 0.001 vs Sham-op; †p < 0.05 vs 5/6 NX.

References


Conclusions

We demonstrate for the first time that the novel sGC stimulator riociguat provides potent protection against cardiac and renal target organ damage in two independent rat models of systemic hypertension: a low-renin model (5/6 nephrectomised rats) and a high-renin model (L-NAME-treated renin-transgenic rats).

Our data provide evidence that hypertension-induced left ventricular remodelling – a key finding in hypertension-induced heart failure – could be substantially ameliorated by riociguat.