QUERCETIN LIMITS VASCULAR DYSFUNCTION ASSOCIATED WITH PRESSURE OVERLOAD LEFT VENTRICULAR HYPERTROPHY

Acute administration of the flavonoid Q evokes endothelium-dependent and -independent relaxation of isolated aorta and coronary arteries. We hypothesized that chronic Q-supplementation limits vascular dysfunction associated with LVH. Rats consumed standard chow (C) or chow supplemented with Q for 7 days. Sham surgery or abdominal aortic constriction to produce LVH was performed on all animals. After 14 more days of C or Q consumption, mean blood pressures (MAP) were obtained from the carotid and caudal artery of conscious rats. Next, after excising the aorta, vascular reactivity was assessed using a wire myograph. Compared to sham-operated C-fed animals (n=8), LVH C-fed rats (n=10) had a greater carotid to caudal MAP gradient (5±2 vs 38±5* mmHg), less maximal acetylcholine (ACh) evoked relaxation (10-4 M, 40±4 vs 30±3%*), and less sodium nitroprusside (SNP) evoked relaxation (10-4 M, 71±5 vs 56±3%*) in norepinephrine (NE) precontracted (10-5M) aortic segments. Compared to sham-operated Q-fed animals (n=10), LVH Q-fed rats (n=7) had a greater MAP gradient (7±1 vs 18±4*), but similar maximal ACh evoked relaxation (38±4 vs 44±3%) and SNP evoked relaxation (71±5 vs 71±4%) (*p<0.05 for all). Our results suggest that Q-supplementation limited aortic vascular dysfunction and lessened the carotid to caudal MAP gradient.