Aim: Arterial chemoreflex (AC) is an important cardiorespiratory control mechanism. Previous studies have shown that severe protein restriction enhances AC responses to potassium cyanide (KCN) and that tonic AC activity may play a role for blood pressure maintenance in such animals. The aim of this study was to investigate if AC dysfunctions were reversed after protein restriction recovery. Methods: male Fischer rats (n=16) received a low protein diet (8% casein, PR group) for 35 days post-weaning. From those, a subgroup recovered (n=7) for 70 days under control diet (20% casein, PR-R group). Each group was carried out with its respective control groups totaling 4 groups: PR, control/PR, PR-R and control/PR-R. Body weight was monitored throughout the treatment period. Under ketamine (80mg/kg) plus xylazin (7mg/kg) anesthesia, femoral artery and vein were cannulated for blood pressure (BP) and heart rate (HR) recording and drug injections respectively. AC was stimulated by intravenous injections of KCN (20, 40, 60, 80 and 160 µg/kg). Results: PR and PR-R rats ended up with smaller body weight than its respective controls (111±2g for PR and 199±4g for control/PR; 331±5g for PR-R and 389±7g for control/PR-R; ANOVA two way followed by Bonferroni’s post-test). Baseline HR was higher in PR rats (442±8bpm) compared to control (356±10bpm). No differences were found for BP throughout the groups. Presor (42±6 vs. 18±4 for 60 µg/kg and 55±3 vs. 37±3 for 80 µg/kg) and bradycardic (-163±37 vs. -85±5 for 160 µg/kg) responses to AC activation were higher for PR compared to control/PR rats (Bonferroni´s post-test; p<0.05). AC responses and basal HR were not different between PR-R and control/PR-R rats. Conclusion: cardiovascular responses to AC activation were higher in PR rats and seem to return to normal after protein restriction recovery. These findings suggest that chemoreflex dysfunctions are reversible and may play a role in tonic control of the blood pressure.

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