Effects of reduced glutathione microinjection into the nucleus of tractus solitarius in blood pressure maintenance

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Aim: Several studies suggest hypertension as an imbalance between neural networks of excitation or inhibition of neurons within central nervous system and more recently, possibly related to availability of reactive oxygen species, and appointed the nucleus of tractus solitarius (NTS) as the main region of termination of baroreceptors afferent fibers on sympathetic discharge. The present study evaluates the effects of reduced glutathione (GSH) microinjection into the NTS of non-anesthetized rats in the maintenance of blood pressure, considering its possible modulating effect of glutamatergic neurotransmission in the NTS region. Methods and results: Male Fischer 344 rats (250 grams body weight) were anesthetized with ketamine (80mg/kg) and xylazine (7mg/kg) mixture for implant of stainless steel cannulas directed to the NTS and for femoral artery cannulation procedures. The basal levels of blood pressure were recorded during 20 minutes, followed by successive L-glutamate (L-glu, 1nmol/100nL) microinjections for a functional identification of NTS region. After the reestablishment of blood pressure basal levels, GSH was microinjected at a concentration of 20nmol/100nL, followed by a new L-glu microinjection 40 minutes later. The GSH microinjection produced pressor response (17.7 ± 3.2 mmHg, n = 4, p <0.05, Student’s t-test) when compared to vehicle microinjection (2.4 ± 0.8 mmHg, n = 4, p <0.05, Student’s t-test). After GSH microinjection, the responses to a new microinjection of L-glu were virtually abolished (0.7 ± 5.0 mmHg and -6.7 ± 8.6 bpm, n = 4 p <0.05, Student’s t-test) when compared to the initial microinjection to functional identification of NTS region (-50.0 ± 28.3 mmHg and -236.5 ± 33.0 bpm, n = 4 p <0.05, Student’s t-test). Conclusion: The data suggest that changes in the local availability of GSH in the region of the NTS can modulate neuronal activity leading to an increase in mean arterial pressure and a blockade of new responses to exogenous L-glu.

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