**Modeling the effect of changes in central respiratory-sympathetic coupling on the sympathetic nerve activity after chronic intermittent hypoxia**

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An increased sympathetic nerve activity (SNA) observed after chronic intermittent hypoxia (CIH) was found to be respiratory modulated which suggests the involvement of specific interactions between respiratory and sympathetic systems. However, the sources of SNA respiratory modulation remain unclear. We extended an established computational model of the brainstem respiratory network by incorporating two neural populations representing the rostral and caudal ventrolateral medulla regions receiving inputs from the pons, ventral respiratory column and retrotrapezoid nucleus (RTN) providing the respiratory modulation of SNA. Our simulations predict that CIH may reduce a threshold for generation late-expiratory (late-E) activity in RTN. This was tested experimentally in *in situ* preparations after rats were submitted to CIH (6% O2, every 9 min, 8 h/day) for 10 days. In normocapnia (5% CO2), the CIH rats exhibited synchronized late-E discharges in both SNA and abdominal nerve that were similar to the late-E bursts observed in both nerves in the control rats during high CO2. Under 1% CO2 phrenic nerve activity was abolished in the control group, but not in CIH, indicating a lower apneic threshold for hypocapnia in CIH rats. We suggest that CIH exposure increases the CO2-sensitivity of late-E activity in RTN and SNA contributing to elevated SNA in CIH rats. FAPESP, CNPq, NIH (R01 NS057815, R33 HL087379, R01 NS069220).