

Physical Inactivity Dependent Neuroplasticity in the Rat RVLM: A Link to Neurogenic Hypertension

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From an evolutionary standpoint human beings have relied upon considerable levels of physical activity to survive. Thus, it is possible that physical inactivity in modern societies is a pathological agent that contributes to cardiovascular diseases such as hypertension. The Mueller laboratory is investigating mechanisms of neuronal plasticity by which physical inactivity may lead to hypertension. The rostral ventrolateral medulla (RVLM) is a brainstem region known to regulate the sympathetic control of blood pressure. Overactivity of RVLM neurons has been implicated in hypertension. Previously the Mueller laboratory reported that sedentary (versus physically active) rats exhibit increased cardiovascular responses to RVLM excitation. Since differences in neuronal structure may contribute to enhanced responses, the aim of this project was to perform an initial investigation into the structure of RVLM neurons from physically active (treadmill-trained for 8-10 weeks) or sedentary rats. Cholera toxin β -subunit (CtB) was injected at the thoracic level of the intermediolateral cell column of the spinal cord to retrogradely label RVLM neurons. CtB immunohistochemistry was performed and neurons were reconstructed with an Olympus microscope and NeuroLucida tracing software (MicroBrightfield). Interesting trends were seen in the structure of CtB-labeled neurons in the RVLM. For example, somas were smaller on average in sedentary animals ($171.7 \pm 8.1 \mu\text{m}^2$, n=2) compared to treadmill-trained rats ($215.0 \pm .04 \mu\text{m}^2$, n=2). Since reduced cell size could indicate enhanced excitability, we propose that neuronal plasticity may contribute to enhanced excitability of RVLM neurons and the increased incidence of hypertension in sedentary versus physically active individuals.

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