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## LS-14-P-2677 CARNOSINE NEUROPROTECTION OF THE CEREBRAL CORTEX IN MICE EXPOSED TO VANADIUM INHALATION. AN ULTRASTRUCTURAL ANALYSIS

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Vanadium (V) is delivered to the atmosphere by the combustion of petroleum derivates rich in this element. V can induce the formation of reactive oxygen species (ROS) in biological systems and the toxicological effects have been associated with them1. In previous studies we reported neuronal alterations in olfactory bulb and hippocampus in mice exposed to vanadium pentoxide2 ($V_2O_5$). Carnosine (beta-alanyl-l-histidine) is endogenously synthesized in brain and is endowed with antioxidant activity as a scavenger of free radicals3. The present study determined carnosine neuroprotection in mice prefrontal cortex exposed to vanadium with an ultrastructural approach. CD-1 male mice weighing 30±5g were divided into four groups. 1) $V_2O_5$ [0.02M] inhalation one hour twice for a 4-week period. 2) $V_2O_5$ Inhalation plus oral Carnosine treatment (1mg/kg/day) 3) Only Carnosine treatment (1mg/kg/day), and 4) Controls inhaled saline. Animals were sacrificed by pentobarbital overdose, perfused by intracardiac puncture with saline solution and glutaraldehyde (2%); prefrontal cortex was dissected and processed for MET. The samples were analyzed in a Zeiss EM 100. The ultrastructural examination revealed that pyramidal neuron of prefrontal cortex in carnosine group showed undamaged organelles (Fig. 1D) similar to controls (Fig. 1A). In contrast, alterations in pyramidal neuron were observed after vanadium exposure showed dark cells with shrunken soma, pyknotic nucleus and irregularly clumped chromatin, a form of necrotic neuronal death (Fig. 1B). In notably, mice vanadium exposed and treated with carnosine showed a decrease in neuronal death (Fig. 1C) compared with vanadium exposed without carnosine. These results suggest that ultrastructural alterations in pyramidal neuron death induced by vanadium are mediated by oxidative stress and that carnosine may modulate the neurotoxic vanadium action.

References 1. S.S. Soares, et al. Journal of Inorganic Biochemistry, 2007,101, 80-88.

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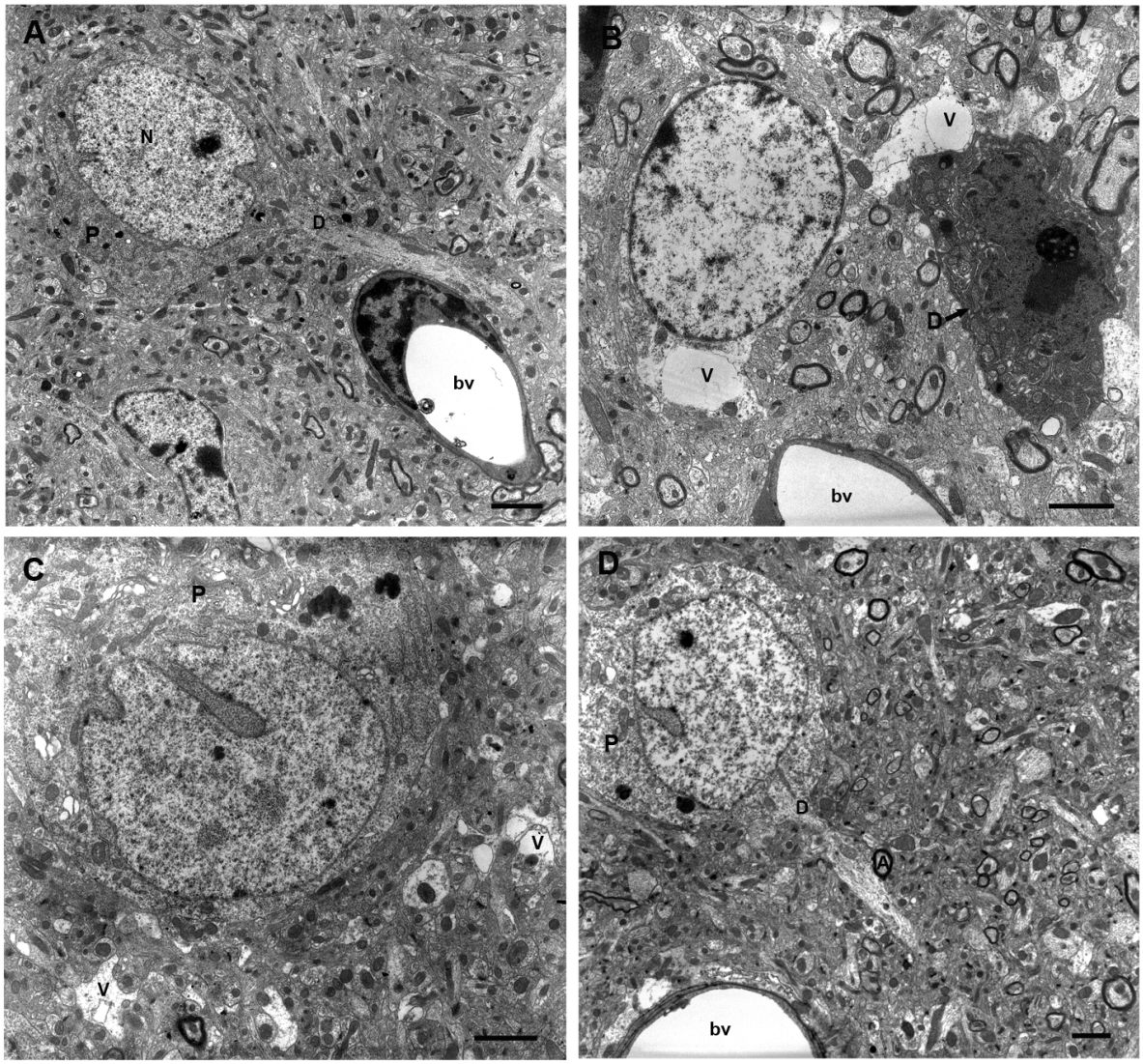


Fig. 1: Prefrontal cortex. A) Pyramidal neuron (P) with nucleus (N),apical dendrite (D) of control mice, B) Dark cell (D) with shrunken soma and vacuolation (V) of exposure vanadium mice, C) Normal pyramidal neuron of exposure vanadium with treatment carnosine mice; and carnosine treatment mice D) with normal axon (A) and blood vessel (bv).Scale bar = 2 µm