Neurotoxic effect of sodium tellurite in the rat temporal lobe

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INTRODUCTION AND METHODS. Previous experimental studies on the intoxication with sodium tellurite indicated that tellurium produced segmental, peripheral neuropathy (2), and pathological changes in myelin of the CNS (3). It has been postulated that transient demyelination is related to the inhibition of squalene epoxydase activity, which results in the block of cholesterol synthesis following tellurium intoxication (1). The aim of this study were comparative ultrastructural observations on the tellurite toxic effect in the temporal cortex and in the hippocampus, in two different phylogenetic structures of the rat temporal lobe. Twenty adult, male, white Wistar rats were intoxicated sc. with 0.1 or 0.25 mg/kg of Te+4 for 5 consecutive days. Temporal cortex and hippocampal CA-1 zone were harvested for electron microscopic studies. RESULTS AND DISCUSSION. Our studies showed a dose dependent vulnerability in the temporal lobe structures. Oedematous astrocytes and astrocytic processes especially around vessels and some swollen synapses were observed. Only few neurones demonstrated Golgi complex and RER enlargement. In the hippocampus additionally to above pathological changes were observed condensed nuclear chromatine and enlarged cytoplasmic channels in oligodendrocytes. Only myelinated fibres of larger diameter showed degenerative changes very often. Myelin lamellae and axons were damaged. Our ultrastructural studies indicate on different neurotoxic effect of sodium tellurite in different phylogenetic structures of the temporal lobe especially as myelin fibre degenerative changes may be related to the primal damage of oligodendrocytes only in the rat hippocampus.

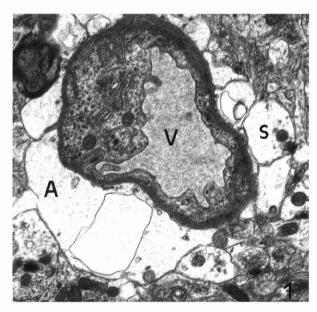


Fig. 1. Temporal cortex. Magn. x 15,000. 5 time repeated dose 0.25 mg/kg Te+4 Swollen synapses (S) and astrocytic processes (A) around a vessel (V).

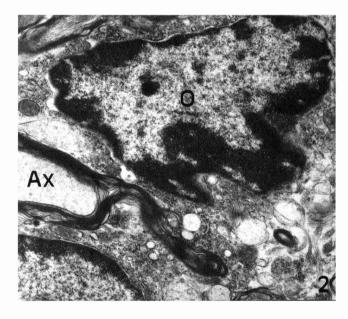


Fig. 2. Hippocampus (CA-1 zone). Magn. x 15,000. Oligodendrocyte (O) with condensed nuclear chromatine and enlarged cytoplasmic channels. Thick neuronal fibre with partially changed myelin lamellae and with lucent axoplasm (Ax).

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