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Nicotine impact on the structure of adult male guinea pig auditory cortex

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ABSTRACT

Background: A growing body of evidence suggests that chronic cigarette smoking causes detrimental effects on brain morphology.
Aim of work: To study the structural changes in auditory cortex region (Layer V), under the influence of nicotine.
Material and methods: Three animal groups (10 each) were used; group I (control) and groups IIa and IIb received 3 and 6 mg/kg nicotine respectively. The specimens from the auditory cortex were examined using light and electron microscopy and morphometry.
Results: Neurons and blood capillaries of the auditory cortex (layer V), were influenced by chronic nicotine treatment in a dose dependent manner. The neurons and their processes revealed disorganization and dissociation of microtubules. The neuronal cells nucleoli characteristically revealed large fibrillar centers detected by silver stain and ultrastructure. The blood capillaries revealed collapse, irregular lumen, thickened basal lamina, abnormal forms of nuclei and organization of microtubules. Neuroglia revealed marked reactivity. Morphometrically, there was a significant decrease in the thickness of the auditory cortex and the number of light neurons and a significant increase in the number of dark neurons in comparison to the control.
Conclusion: Nicotine affects the integrity of the auditory cortex possibly by reducing metabolic and transcription activities.

1. Introduction

While the lungs seem to be at the center of attention when it comes to the harm that smoking causes, other organs are also quite vulnerable to smoke infused damage. Cigarette smoking has well-documented associations with numerous negative health outcomes including possible effects on the brain. A growing body of evidence suggests that chronic cigarette smoking alone causes detrimental effects on brain morphology, blood flow, neurochemistry, and neurophysiology (Brody, 2005; Domino et al., 2004; Gallinat et al., 2006; Hayee et al., 2003; Neuhaus et al., 2006). Although the link between cigarette smoking and peripheral hearing loss was established over 40 years ago, information on the effects of smoking at the cochlear and auditory central nervous system levels has become available only recently.

The most significant ingredient in tobacco smoke is nicotine, the component that causes addiction. Of all the chemicals found in cigarette smoke, nicotine is responsible for virtually all nACh receptor occupation. Stimulation of these receptors underlies nicotine addiction.

Nicotine is known to influence auditory-cognitive functions such as attention. Systemic administration of nicotine enhances sensory-evoked responses recorded within or near auditory cortex in animals and non-smoking humans (Guha and Pradhan, 1976; Bringmann, 1994; Oldford and Castro-Alamancos, 2003; Methe-rate, 2004; Liang et al., 2006)

In a more direct examination of nicotine's effects on auditory processing, systemic nicotine delivered to nonsmokers transdermally, via a nicotine "patch," did not affect a measure of peripheral auditory function (otoacoustic emissions in the cochlea), but did affect CNS responses to auditory stimuli (Harkrider and Champlin, 2001a,b; Harkrider et al., 2001).

Chronic nicotine use has been reported to impair cognitive auditory processing (Muller et al., 2007). Regarding brain structure, previous studies explored structural differences between smokers and nonsmokers, focusing on regional gray matter (and white matter) volumes as well as densities with voxel

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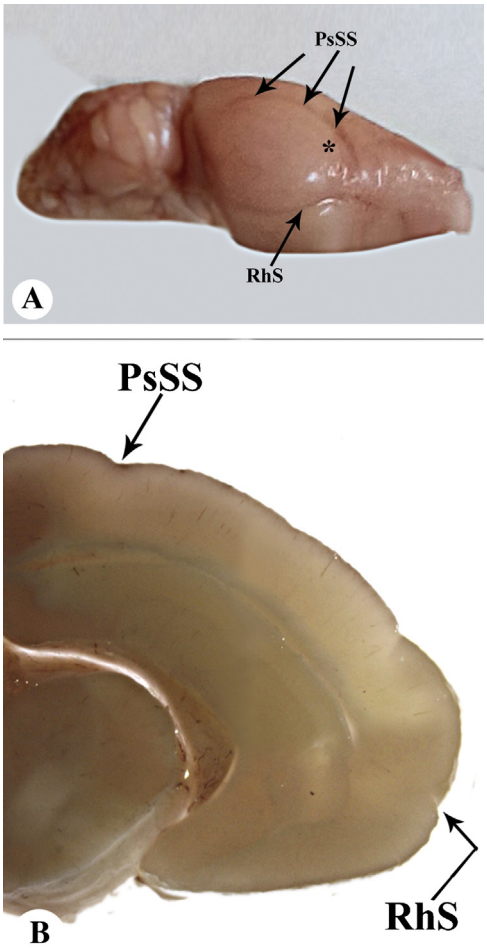

Fig. 1. (A) Lateral view of the guinea pig brain, showing the area of the auditory cortex (*) which is located posterior to the pseudosylvian sulcus (Ps Sv S). Rh S (rhinal sulcus). (1B) Coronal sectionsshowing the pseudosylvian sulcus (Ps Sv S) and the rhinal sulcus (Rh S).

based morphometry (VBM). Overall they found smaller gray matter volumes and densities for smokers. Gazdzinski et al. (2005) showed a reduction in parietal and temporal gray matter, which is in line with findings of Durazzo et al. (2007), reporting smaller temporal, parietal, and neocortical gray matter volume among smokers who were heavy drinkers. Kühn et al. (2010) reported that the brains of smokers are structurally different from those of never-smokers in a dose-dependent manner. They found cortical thinning in medial orbitofrontal cortex in smokers relative to never-smokers. Nicotine administration in animals can affect the morphology of neurons which revealed a decrease in the size of pyramidal cells in hippocampus CA1 area (Bergstrom et al., 2008). Moreover, nicotine administration can decrease the size of perikarion and number of dendritic spines in the prefrontal cortex (Jalili et al., 2014). However, with regard to the impact on auditory cortex, we are not aware of any previous studies focusing on auditory cortical structure in smokers compared to nonsmokers. No study has been conducted on the effects of nicotine on morphological changes in auditory cortex region, so far. Therefore, this study aims to verify the assumption that smoking has harmful effects upon central auditory system structure. Layer V is of particular interest because its cells form part of the projection to the thalamus, sub-thalamic nuclei, and contralateral cortex

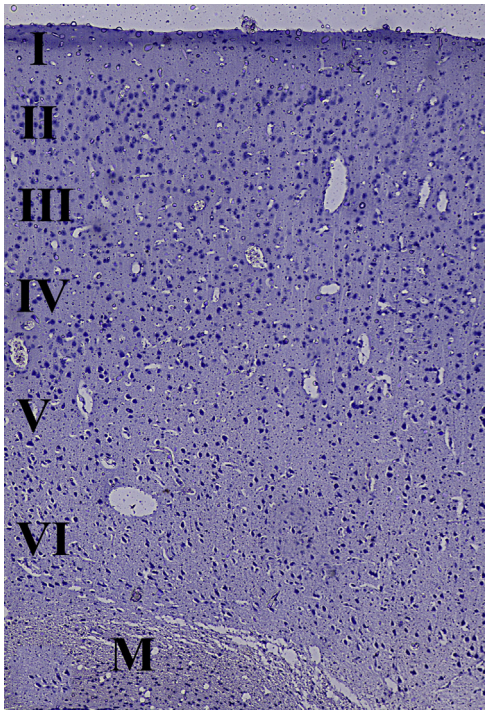

Fig. 2. Histological section in auditory cortex showing the cortical layers. Layer I has very few cell bodies. Layers II and III contain densely packed cells, many of which are pyramidal. A prominent layer IV is dominated by granule cells, which are smaller and rounder and packed more closely than the cells in adjacent layers. Layer V is characterized by a low density of cells, most of which are pyramidal in shape. Layer VI has a higher density of cells than layer V, and the cells are generally smaller compared to those in layer V.M (medulla). (Gallocyanin X 40).

through the corpus callosum. It is the thickest layer (25% of the total) and the pyramidal cells are the main cell type in layer V (cf Paxinos, 2004).

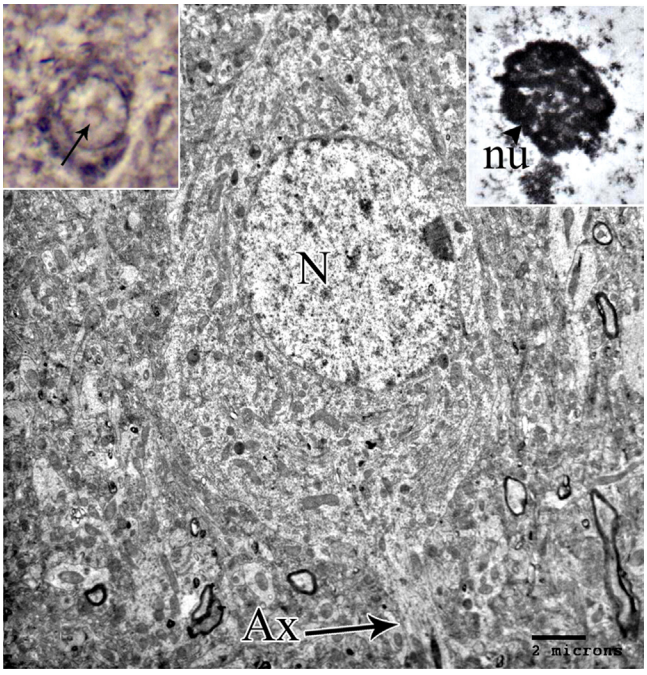

Fig. 3. Shows a pyramidal neuron with euchromatic nucleus (N) and an axonic process (Ax). Note the well-formed Nissle bodies (R) and nucleolus (nu) shown in the upper right inset. (X 3600; 7200). Upper left inset: Paraffin section showing weak staining of the neurofibrils (↑) and the nucleolus (nu) fibrillar center (X 1000).