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Adolescent brain and nicotine

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ABSTRACT

Adolescence is a period of transition between childhood and adulthood that involves major changes in development in the physical, psychological, social and neurobiological domains. During this phase of life, multiple physical and emotional events occur throughout the body, markedly in the central nervous system, regulated by neurohormones, which are fundamental for human development, responsible for changes in thoughts, attitudes and behaviors that culminate in complete maturity for a laborious and reproductive life. Due to the lability of the neurological tissue in this period, external aggressions by means of chemical substances can cause serious and lasting effects. Nicotine is one of the substances considered legal and that more easily the adolescent comes into contact early, and can cause many harm to current and future health. This article presents some characteristics of the development of the adolescent's nervous system and the deleterious actions that can be caused by nicotine in its various forms of social presentation.

Keywords: Adolescence – nicotine – smoking – nicotine addiction

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INTRODUCTION

Adolescence is a period of physical, emotional, and social changes, with significant increases in body size, physical appearance and the development of interpersonal skills necessary for successful integration into society [1]. Adolescence is also characterized by high expression of risk, exploration, search for novelties and sensations, social interaction, and playful behavior [2,3]. This period of great vulnerability, associated with impulsive actions and decisions, is linked to transformations and maturation of the central nervous system (CNS), sensitive to new experiences of plasticity that occurred in the regions of executive control and decision-making [4,5]

Among the main experiences related to the use of legal or illegal substances, smoking and the new emerging tobacco and nicotine products are those that start earlier, reinforced by stimulation and cultural acceptance, ease of access, low cost and ignorance of the negative effects that can cause health [6,7].

NICOTINE

Although nicotine is a psychotropic substance, with a high additive power and widely used worldwide, its use is not considered illegal. Nicotine is a potent parasympathomimetic alkaloid produced in the roots of *Nicotiana tabacum*, a plant found in the American continent from whose leaves it was extracted in 1828 [6,8,9]. In its pure state, nicotine is a colorless, volatile, water-soluble, bioactive alkaloid [9], absorbed by the skin, mucous membranes in the mouth and nose and pulmonary alveoli [10]. Its greatest consumption is through cigarettes that contain, on average, 14 mg, resulting in 1 to 2 mg absorbed per cigarette, although the amount provided by smoking is highly variable and dependent on the type of cigarette and the individual topography of the smoke [11]. The large surface area of the small airways and the alveoli, associated with a pH of 7.4 in the lungs, allow rapid absorption of inhaled nicotine and the transition to the brain in 7 seconds, exerting its pharmacological effects on nicotine acetylcholine receptors and triggering the release of dopamine and other neurotransmitters. The cerebral half-life of nicotine is 52 minutes [6,9,12,13].

Nicotine is a neuroteratogenic substance that alters cell proliferation and differentiation, can induce epigenetic changes in the neural genome and interfere with communication systems between neurons [5,14,15]. Nicotinic cholinergic transmission is a vital process for the functioning of the living organism, but vulnerable to the involvement of nicotine [8,14]. As nicotine exerts complex actions involving

different circuits and structures, even brief exposure can produce lasting changes in the adolescent brain, causing effects on the structure, function, learning and memory of the hippocampus [5,14,16].

The pharmacological action of nicotine occurs by binding to presynaptic cholinergic receptors (nAchRs) located at the terminals of neurons present in the CNS and in the peripheral nervous system [6,17]. When stimulated, these receptors increase the release of several neurotransmitters, such as acetylcholine, norepinephrine, serotonin, dopamine, opioids, glutamate, beta endorphins and gamma-aminobutyric acid, involved in the induction and modulation of neuroplasticity [18-20] The neuronal receptor subtype the most abundant is alpha4 beta 2 nAchR, which has a high affinity for nicotine [5,9] and is found in the shell of the nucleus accumbens, in the ventral tegmental area and in the basolateral amygdala [21,22]. With repeated exposure to nicotine, there is an increase in the number of nAchRs. The activation of the mesolimbic dopaminergic system provides better attention and mood, stimulates memory capacity and reduces muscle tone, which is pleasant for the user, allowing him to continue using this substance and critical to the drug-induced reward system [6,9,23]

ADOLESCENT BRAIN AND NICOTINE

Adolescence is a critical period when drug administration can induce some effects and negatively influence neural, behavioral, and cognitive functions even long after early life exposure [24], because there are physiological, evolutionary, structural, and neurochemical changes, especially in the dopaminergic system, that accompanying the maturing process that occurs in this phase of life. The most important developmental changes in the adolescents' brain are: non-linear reduction in cortical and subcortical grey volume, synaptic pruning, directional organization of white matter, maturation of axons, and myelination resulting in increased efficiency of impulse transduction [5,24,25].

The sensitivity of the CNS to the aggressions of environmental agents depends on their state of development and nicotine use has been associated with deleterious effects in the prefrontal cortex and hippocampal structure, can lead to irreversible decreased cognitive functions, mainly attention, memory and hyperactivity [9,26]. Adolescent smoker demonstrate cognitive and behavioral impairments such as decrease of working memory, impaired serial pattern learning, deficit in attentional performance, increased anxiety and depressive-like behaviors, wake time and reduces both total sleep time and rapid eye movement sleep, inhibits prolactin secretion form the anterior pituitary and increase cortisol concentration [6,24,27-30].

Nicotine during adolescence causes acute and lasting changes in the developing brain, which in addition to the effects on the various systems and functions of the body, can lead to subsequent nicotine dependence. The rapid neuronal response to nicotine positively reinforces smoking behavior because is associated with expectation of increased well-being, and pleasure activities [9]. When dopamine levels fall, the adolescent is bound to seek more nicotine, leading to a vicious cycle that has just turned into addiction [9,31-33].

The age of first cigarette use is critical determinant of nicotine dependence since the earlier it starts, the more difficult is to quitting [5,34]. Also, the early use of nicotine through smoking can act as the gateway to experiment with other illicit drugs such as marijuana, cocaine, opiates, etc. [24].

CONCLUSIONS

Tobacco use is a significant public health concern because it has great potential for the development of addiction [6]. Despite all the knowledge about the damage caused by smoking, prevention and smoking cessation actions in the adolescent care environment are still few, and the damage and dependence on nicotine are a reality for adolescents with the onset of addiction occurring shortly after smoking started [35-37]. It is essential to increase educational actions on the damage caused by smoking, facilitating access to information and making exposure to cigarettes and their derivatives more difficult.

Conflict of interest: none

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