Nicotine induced autonomic nervous system dys-homeostasis is characterized by inadequate sympathetic responsiveness

Dunja Vujčić[1], Matej Šapina[2], Matija Fenrich[3], Dario Brdaric[4], Marko Pirić[3], Dinko Puntarić[5]

1 – University Hospital Centre Osijek

2 – Primary Health Care Centre Đakovo

3 – Faculty of Medicine Osijek

4 – Institute for Public Health of Osijek-Baranya County

5 – Faculty of Medicine Osijek, Department of Public Health

Abstract:

Nicotine is known as a potent autonomic stimulator. As a nicotinic acetylcholine receptor (nAChR) agonist, it directly elicits parasympathomimetic effects. In addition to that, nicotine indirectly evokes sympathetic discharge by means of preganglionic nAChR activation. Since this sympathetic activation is a result of previous cholinergic preganglionic stimulation, we hypothesize that chronic exposure to nicotine may desensitize the preganglionic cholinergic system, thus weakening the appropriate sympathetic response. The aim of our research was to investigate sympathetic activation in long-term young smokers without any previous and present autonomic-related and cardiologic co-morbidity.

The research was conducted on healthy student volunteers (m = 26 and f = 32), divided in smokers and non-smokers. In order to quantify the autonomic activation, we used the non-invasive method of spectral time and frequency domain analysis of Heart Rate Variability (HRV). A continuous ECG was recorded for a period of 100 seconds (BIOPAC Model MP35, sampling rate 500/s). Following the first recording, participants were asked to chew a nicotine gum (Nicorette 4 mg) for 15 minutes, after which an ECG was re-recorded.

We recorded a significant decrease in HRV and an increase in heart rate frequency among non-smokers, and statistically significant absence of high frequency change i.e sympathetic activation in smokers compared to non-smokers ($p = 0.02$).

Our results suggest that smokers lack the appropriate sympathetic activation due to chronic abuse of nicotine. The sustained long-term agonist application may results in various molecular receptoral and post-receptoral adaptations that contribute to reduced nAChR responsiveness, and thus to autonomic dys-homeostasis.

Key words: nicotine; autonomic nervous system; desensitization; autonomic dysregulation