Smoking

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Smoking is the cause of several changes in the structure and function of the brain and constitutes a risk factor for CVA. Extended damage is observed in cortical structures. Namely, long-term nicotine exposure is associated with decreases in frontal and temporal lobes’ and cerebellum’s volume. Subcortical changes, such as grey matter volume and density reduction, may also be present in smoking individuals (Gallinat, Meisenzahl, Jacobsen, Kalus, Bierbrauer, Kienast,... & Staedtgen, 2006). Chronic nicotine exposure may also lead to dysfunction in dopamine transmission (Brody, Mandelkern, Olmstead, Scheibal, Hahn, Shiraga, ... & McCracken, 2006). Literature reports oxidative stress, inflammation, and atherosclerotic processes as potential mechanisms for smoke’s impact on
cognitive performance (Swan, Lessov-Schlaggar, 2007). Consistent to these findings, another study supported that cognitive performance in a working memory task was affected in smokers, as related to the strategies they used during the task, as well as in changes in the underlying brain function (McClernon, Froeliger, Rose, Kozink, Addicott, Sweitzer, ... & Van Wert, 2016).

Smokers are more likely to suffer a cerebrovascular event, with current smokers being at higher risk than ex-smokers (Lee, Forey, Thornton, & Coombs, 2018). Nicotine exposure also increases the risk for subarachnoid hemorrhage, especially in women who are heavy smokers. Former smokers were less at risk than current smokers regardless of gender (Lindbohm, Kaprio, Jousilahti, Salomaa, & Korja, 2016). In another study, smoking combined with hypertension impacted stroke incidence more than expected, especially in females with ischemic stroke (Nordahl, Osler, Frederiksen, Andersen, Prescott, Overvad, ... & Rod, 2014). More recent findings account for smoking and hypertension as independent risk factors. Consistent with the former study, patients with both hypertension and smoking history were at the highest risk for ischemic stroke (Huangfu, Zhu, Zhong, Bu, Zhou, Tian,... & Zhang, 2017). In young men, the higher the dose, the more increased the risk for stroke is. Reducing the number of cigarettes consumed per day reportedly decreased the risk of stroke (Markidan, Cole, Cronin, Merino, Phipps, Wozniak, & Kittner, 2018).

Chronic smoking can also impact the outcome in stroke survivors, as smoking individuals with stroke reportedly face increased risk for post-stroke delirium, longer hospitalization and worse outcome (Lim, Lee, Yoon, Moon, Joo, Huh, & Hong, 2017). Furthermore, substituting smoking with vaping does not decrease the risk for cerebrovascular disease; electronic cigarettes impact the blood-brain barrier integrity, and increase inflammatory responses in the vascular system. Vaping is also related to worse post-stroke injury (Kaisar, Villalba, Prasad, Liles, Sifat, Sajja, ... & Cucullo, 2017). Recent studies have shown that even electronic cigarettes are as likely as the conventional cigarettes to lead in deteriorating effects of oxidative stress (Ikonomidis, Vlastos, Kourea, Kostelli, Varoudi, Pavlidis, ... & Iliodromitis, 2018).

Concludingly, nicotine exposure inflicts structural and functional changes in the brain, which can also reflect upon cognitive performance. Both smoking and vaping increase the risk of stroke, regardless the age of the individual (i.e., young or older adults). Finally, reduction of daily dose of nicotine can help decrease the
risk of stroke in smokers.

References:


SAFE
Stroke Alliance for Europe
THE STROKE PATIENT
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