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# ELECTRONIC CIGARETTES: LONG TERM CONSEQUENCES ON CARDIAC AND RESPIRATORY HEALTH

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According to recent statistics, the predicted annual growth rate of e-cigarette usage will reach 8.46% (2024–2028). It's currently estimated to be a market of 38.8 million US dollars. The potent component, nicotine, is highly addictive, causes long-term multi-system organ damage, and also increases the risk of developing cancer.

Vaping is considered the modern replacement for smoking. It comprises different components like nicotine, carcinogens, diacetyl, heavy metals, cadmium flavourings, etc. that affect multiple organs. According to Wittenberg, Wolfman et al. (2020) pure nicotine binds to the nicotinic acetylcholine receptors (nAChRs), activating the mesolimbic reward system, leading to dopamine release and addiction. Andreas Diaber et al. (2023) study with evidence based proposition

showed the oxidative stress, endothelial dysfunction and in turn leading to cardiovascular sequelae. Cinical trials prove association of e-cigarette vaping with a higher risk of myocardial infarction with an odds ratio of 2.11[confidence interval (95% CI) 1.14–3.88].

The main cause of concern is the oxidative stress caused by e-cigarette, the presence of acrolein and formaldehyde compounds are major cause in causing a cascade of terrible reactions such as, superoxide O2•−) and the reduction of Nitric Oxide by the guyanyl cyclase process. Renin-angiotensin-aldosterone system (RAAS), catecholamines like noradrenalin and endothelin-1 (ET-1) and the dysregulated synthesis of endothelium-derived hyperpolarizing factor (EDHF) and epoxyeicosatrienoic acids (EETs). ROS-dependent activation of cyclooxygenase (COX) provides the peroxide tone, which dysregulates prostanoid synthesis from arachidonic acid (AA) and increases endoperoxide prostaglandin H2 (PGH2). Vasoconstriction is brought on by the latter process through the thromboxane/PGH2-receptor (TP), primarily as a result of PGH2 accumulation brought on by prostacyclin synthase (PGIS) inactivation and peroxynitrite-dependent nitration (–NO2). Deactivation of the prostacyclin (PGI2)-receptor (IP) results in the loss of vasodilation.

\These oxidative stress reactions also cause a cytokine storm, consisting of MMP-9 (Matrix Metalloprotease-9) and VCAM-1 (Vascular Cell Adhesion Molecule-1), the former leading to atherosclerosis and tissue remodelling in heart and the latter leading to an excess production of ROS. In turn the aforementioned reactions lead to a serum of patients which has less nitric oxide release and it also cause the cells to be more permeable, a strong sign of vascular impairment. Putting things into a sequence, the following biochemical and physiological events cause a barrage of consequences like EVALI (E-cigarette induced lung injury) which is caused by often by Vitamin E acetate and acrolein, furthermore ARF 6 (ADP-ribosylation factor 6) and ROS (Reactive Oxygen Species) combined damage lung microvasculature, finally increased propensity towards cardiac dysfunction and other related events

With a FMD (Flow Mediated Dilation) and FMC (Flow Mediated Constriction) values <5.1%, it has been demonstrated that e-cigarette users are more likely to experience myocardial infarction and that using the product also increases the risk of myocardial fibrosis and re-modelling because to acrolein, EVALI (E-Cigarette associated lung injury) in which exogenous pneumonitis reaction due to increased Vitamin E acetate levels ruin the microvasculature, further investigation has identified ARF 6 mediated pathway activated by acrolein and diacetyl (flavouring agent) as a cause of barrier induced disruption and ROS accumulation leading to pulmonary endothelium disruption and inflammation. Latest developments also suggest that e-cigarettes are getting prevalent among adolescents and teens, thus leading to a lot of problems with holistic health; not only vascular injury is bad for cardiopulmonary system but also for the entire conduction system. Thus, the situation needs to be addressed with utmost caution and awareness.