



Second-hand aerosol from tobacco and electronic cigarettes: Evaluation of the smoker emission rates and doses and lung cancer risk of passive smokers and vapers

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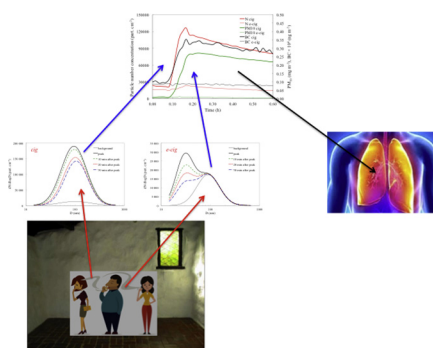
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HIGHLIGHTS

- Smokers present a 4.5-fold higher particle number emission rate than vapers.
- The dose received by passive smokers is up to 15-fold higher than passive vaper one.
- The maximum dose was received by passive smokers and vapers at the 21st generation.
- ELCR for passive smokers is 5 orders of magnitude higher than the passive vaper one.

GRAPHICAL ABSTRACT



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ABSTRACT

Smoking activities still represent the main, and preventable, cause of lung cancer risk worldwide. For this reason, a number of studies were carried out to deepen and better characterize the emission of cigarette-generated mainstream aerosols in order to perform an a-priori evaluation of the particle doses and related lung cancer risks received by active smokers. On the contrary, a gap of knowledge still exists in evaluating the dose and risk received by passive smokers in indoor private micro-environments (e.g. homes). For this purpose, in the present paper, an experimental campaign was performed to evaluate the exposure to second-hand aerosol from conventional and electronic cigarettes and to estimate the consequent dose received by passive smokers/vapers and the related lung cancer risk.

Measurements of exposure levels in terms of particle number, PM₁₀ and black carbon concentrations, as well as particle size distributions, were performed in a naturally ventilated indoor environment during smoking activities of tobacco and electronic cigarettes. The particle emission rates of smokers and vapers, for the different aerosol metrics under investigation, were evaluated. Moreover, for a typical exposure scenario, the dose received by the passive smokers/vapers in a naturally ventilated indoor micro-environment was estimated through a Multiple-Path Particle Dosimetry (MPPD) model able to assess the particle dose received in the different tracts

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of the respiratory systems. Furthermore, on the basis of scientific literature data about mass fraction of carcinogenic compounds contained in cigarette-emitted particles (i.e. Heavy Metals, Benzo-a-pyrene and nitrosamines) and the estimated doses, the excess life cancer risk (ELCR) for passive smokers/vapers was evaluated. Cumulative respiratory doses for passive smokers were up to 15-fold higher than for passive vapers. The ELCR for second-hand smokers was five orders of magnitude larger than for second-hand vapers.

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1. Introduction

1.1. Conventional and electronic cigarette emissions

Tobacco-cigarette smoke has been recognized to increase lung cancer risk by a 5- to 10-fold factor in a dose dependent manner (Schwartz and Cote, 2016). In fact, the International Agency for Research on Cancer (2004) has identified >70 compounds in tobacco smoke with sufficient evidence for carcinogenicity in either laboratory animals or humans and sixteen of them have been classified as group 1 carcinogenic to humans. Some of these group 1 compounds, such as Polycyclic Aromatic Hydrocarbons (PAHs) are emitted by cigarette combustion, others, namely tobacco-specific N-nitrosamines, are mainly produced during the curing process of tobacco (International Agency for Research on Cancer, 2004).

Electronic cigarettes (e-cigs) have been commercialized with the aim of eliminating the intake of such carcinogenic compounds. E-cigs are battery-operated devices, where the liquid contained in a vial is vaporized over an electric heater and the resulting aerosol is inhaled through a mouthpiece. Therefore, e-cigs neither involve combustion nor use cured tobacco leaves. However, several recently published studies have pointed out their health effects and the presence of harmful compounds in their aerosol, even if at lower levels than for tobacco cigarettes (Farsalinos et al., 2014; Goniewicz et al., 2014). Kosmider et al. (2014) found that e-cig vapors contain toxic and carcinogenic carbonyl compounds at levels increasing with battery output voltage. Goniewicz et al. (2014) reported the presence in e-cig aerosol of carbonyl compounds, among which formaldehyde, tobacco-specific N-nitrosamines NNN and NNK and metals, such as Pb, Cd and Ni, at levels 9–450 times lower than in cigarette smoke,

Dosimetry studies on mainstream cigarette (Fuoco et al., 2017; Stabile et al., 2017a) and e-cig aerosols have been carried out also reporting size segregated data per airway generation (Manigrasso et al., 2015a) and as function of the age of the user (hereinafter referred as “vaper”; (Manigrasso et al., 2017a)). On average, 6.25×10^{10} particles are deposited in the human respiratory tree after a single puff of an e-cigarette. This dose represents about 30% of the daily doses of a non-smoking individual (Buonanno et al., 2011a). Moreover, aerosol doses are asymmetrically deposited in the lungs: in the right-upper lung lobe, they are about twice than in left-upper lobe and 20% greater in right-lower lobe than in the left-lower lobe (Manigrasso et al., 2015b).

Azzopardi et al. (2016) studied the cytotoxic effect of tobacco cigarette and e-cig aerosol on human bronchial epithelial cells, reporting the half-maximum effective concentration (EC_{50}) for 60 min exposure. When expressed for deposited mass, e-cig aerosol was significantly (94%) less cytotoxic than conventional cigarette aerosol (60 min deposited mass EC_{50} 52.1 vs. 3.1 $\mu\text{g}/\text{cm}^2$). However, it should be considered that these cell viability data derive from in vitro acute exposure assessments. To date no data from chronic exposure studies are available. The urgent need of such data has been pointed out by Ganapathy et al. (2017). They showed that e-cig aerosol extracts can cause significant levels of DNA damage, even if at levels lower than observed for mainstream smoke extracts. The authors observed increased cellular Reactive Oxygen Species (ROS), and decreased total antioxidant capacity. Moreover, they observed decreased expression of proteins essential for DNA damage repair and pointed out that this could increase cancer risk.

1.2. Second-hand smokers and “vapers”

A further concern related to the e-cigarette consumption is represented by their influence on the indoor air quality and the consequent effects on passively exposed populations (hereinafter referred as second-hand “vapers”). Great part of the studies addressing this issue concludes that harmful compounds are present in the aerosol emitted by e-cigs at lower concentrations than for tobacco cigarettes, but above background concentrations, so that a health risk may be envisaged for the exposed population (Hess et al., 2016). Saffari et al. (2014) performed ambient air sampling in a test room in order to compare second hand exposure to e-cig aerosol and tobacco cigarette smoke. They observed very lower particle-phase emissions from e-cigs than from tobacco cigarette. Black carbon and PAHs abundantly present in tobacco smoke were not detected in e-cig aerosols. Emission rates of organic compounds and of inorganic elements were respectively >100 times and 10 times lower for e-cigs than for tobacco cigarettes, with the exception of Ni and Ag, whose emission rates were higher for e-cigs than for tobacco cigarettes. Similar studies and results were carried out by other authors (McAuley et al., 2012; Schober et al., 2014; Schripp et al., 2013). Moreover, Ballbè et al. (2014) reported nicotine concentrations higher in homes with e-cigarettes users than in nonsmoking control homes and nicotine concentration 5.7 times higher in homes with tobacco cigarette smokers than in homes with e-cigarettes users. Urinary and salivary cotinine concentrations were higher than for control homes. However, no statistically significant differences were observed between e-cig and tobacco cigarette exposed individuals, suggesting similar nicotine uptake. In agreement with these findings, Flouris et al. (2013) reported similar serum cotinine increments for passive exposure to tobacco cigarette smoke and to e-cig aerosol. Moreover, Flouris et al. (2012) showed that second hand exposure to e-cig aerosol does not affect complete blood count markers (blood sample collected 1 h after passive smoking sessions). On the contrary, the authors observed increased white blood cell count, lymphocyte count and granulocyte count in individual exposed to passive tobacco smoke.

1.3. Aims of the work

Dosimetry studies on second-hand exposure to e-cigs aerosols are very sparse. Sosnowski and Kramek-Romanowska (2016) estimated the regional deposition efficiency of secondary e-cig aerosols during passive vaping, based on the expected growth of the exhaled particles. Protano et al. (2016) and Protano et al. (2017) measured the temporal evolution in a test room of the number size distributions of aerosol emitted by tobacco cigarettes, e-cigs and by the newly released IQOS® (an electronic heat-not-burn device) estimating the relevant aerosol deposition doses. Anyway, to date, no study has been performed neither on the distribution per airway generation nor on the size distribution of the aerosol doses deposited in the respiratory tree, due to second hand vaping of e-cigs. Within this context, the purpose of this study is to: i) measure the emission rate of smokers/vapers during smoking/vaping activities, ii) estimate the size segregated and regional resolved aerosol doses deposited in the respiratory systems of individuals exposed to second-hand aerosols emitted by e-cigs and tobacco cigarettes, iii) estimate the related lung cancer risk of individuals exposed to second-hand aerosols on the basis of carcinogenic compounds in cigarette- and e-

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