BMC Pulmonary Medicine

RESEARCH

Open Access

The impact of smoked cigarettes' type on the level of reactive oxygen species in physicians with surgical and non-surgical specialization

Artur Nowak[1], Paulina Natalia Kopa-Stojak[1] and Rafał Pawliczak[1*]

Abstract

Background Cigarette smoking remains a pervasive global health concern, contributing to a myriad of debilitating conditions. One critical aspect of its detrimental impact is the induction of oxidative stress (OS). The aim of the study was to identify differences in the level of reactive oxygen species between surgical and non-surgical physicians who smoke different types of cigarettes and their level of nicotine addiction.

Methods The prospective study conducted on surgical and non-surgical physicians who smoke various types of cigarettes worked at the Provincial Multidisciplinary Oncology and Traumatology Centre in Lodz (Poland) and at the Polish Mother's Health Centre Institute in Lodz (Poland). The Fagerström's test for nicotine dependence was used to determine degree of nicotine addiction. The differences in reactive oxygen species level among physicians with surgical and non-surgical specialization who smoke different types of cigarettes was analysed by the Cellular Reactive Oxygen Species Assay Kit.

Results In this study 35.1% of surgical and 40.5% of non-surgical physicians indicated signs of nicotine addiction. The Fagerström score was significantly higher in surgical than non-surgical physician specialties (5.4 ± 1.372 vs. 4.7 ± 1.310; $p = 0.001$). Significantly higher ROS level was observed in conventional cigarette (CS) smokers, e-cigarette (EC) smokers and tobacco heating products (THP) smokers compared to non-smokers ($p < 0.05$). In addition, there was a significantly lower ROS level in THP smokers compared to CS smokers ($p < 0.05$) and EC smokers ($p < 0.05$). There was a significantly higher ROS level in physicians with surgical specialization who smoke CS ($p < 0.001$) and EC ($p = 0.004$) compared to non-surgical physicians.

Conclusions Understanding the influence of the type of smoked cigarettes on oxidative stress in surgical and non-surgical physicians is imperative for targeted interventions and health promotion strategies. The broader goal is mitigating the health risks associated with cigarette smoking, especially within professional domains where stressors may already be heightened. Further investigations are essential for developing tailored approaches to address the nuanced relationships between cigarette types, occupational stress, and oxidative stress in specialized medical fields.

Clinical trial number not applicable.

Keywords Smoking, Reactive oxygen species, Oxidative stress, Physicians

*Correspondence:
Rafał Pawliczak
rafal.pawliczak@csk.umed.lodz.pl

[1]Department of Immunopathology, Faculty of Medicine, Medical University of Lodz, 7/9 Żeligowskiego, Building 2, Room 177, Lodz PL-90-752, Poland

Background

Cigarette smoking remains a global health concern, contributing to a myriad of debilitating conditions. Cigarette smoke is a complex mixture encompassing over 7000 chemicals [1, 2], including: tobacco-specific nitrosamines (TSNAs), polyaromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), carbon monoxide (CO) and metals [3–8]. Moreover, cigarette smoke contains a huge amount of free radicals, which leads to the induction of the oxidative stress (OS) [9, 10]. Oxidative stress may be defined as a state of imbalance between reactive oxygen species (ROS) production and the body's ability to neutralize them [11]. Clinical symptoms of oxidative stress are often nonspecific. In addition, it can indicate hundreds of different diseases, such as pulmonary diseases (i.e. chronic obstructive pulmonary disease (COPD)), cardiovascular diseases (i.e. atherosclerosis, heart attack, stroke, coronary artery disease), metabolic disorders (i.e. type 2 diabetes), inflammatory disorders (i.e. rheumatoid arthritis), kidney diseases (i.e. glomerulo- and tubule-interstitial nephritis, renal failure, proteinuria, uraemia) neurodegenerative disease (i.e. Parkinson's disease, Alzheimer's disease), reproductive disorders (i.e. lower rate of fertilization, implantation and pregnancy) and cancer [12–14].

In addition to classic cigarettes, there are also alternative tobacco products, i.e. electronic cigarettes (ECs) and tobacco heating products (THPs) [15]. E-cigarettes, an electronic nicotine delivery system, heat "e-liquid," which typically contains nicotine as well as flavourings, propylene glycol and/or glycerine to produce aerosols [16, 17]. THP (or heat-not-burn (HnB) products) are electronic tobacco-containing devices that heat tobacco instead of burning it, producing inhalable aerosols containing nicotine and other chemicals [18, 19]. These products have gained popularity based on the belief that they pose fewer risks compared to traditional cigarettes [20]. Due to heating of e-liquid instead of tobacco, e-cigarette aerosol contains significantly lower level of hazardous substances compare to conventional cigarettes [21, 22]. Furthermore, THP aerosol is produced by heating tobacco rather than burning it as in conventional cigarettes at a lower temperature. Therefore, reduction in the amount of tar and other hazardous and potential hazardous constituents (HPHCs) is possible [23, 24]. However, the alternative smoking products are not harmless and may provide negative health consequences, especially among never smokers, adolescents and young adults. Using of such products may leaded to respiratory irritation and airway epithelial injury, endothelial dysfunction, increased activation of leukocytes, increased aggregation of platelets, systemic inflammation and increased arterial stiffness. Moreover, aerosol of such products may contain human carcinogens and/or potential carcinogens [25–28]. Moreover, the aerosol of both e-cigarettes and tobacco heating products contains free radicals, which induce oxidative stress [29, 30].

Smoking has been associated with increased oxidative stress in various populations, including healthcare professionals. Surgeons, who already face high-stress environments and occupational hazards, may be particularly susceptible to the compounded effects of smoking and oxidative stress. Similarly, non-surgical specialists also experience occupational stressors that may interact with the type of cigarettes smoked, further exacerbating oxidative stress levels [31, 32]. However, stress levels can vary within the profession [33, 34]. Understanding how different cigarettes affect oxidative stress in surgical and non-surgical professionals provides valuable information about the broader health consequences of smoking habits [31, 32].

The aim of this study was to determine if smoking as well as type of smoked cigarettes affect the level of oxidative stress in surgical and non-surgical physicians. The secondary aim of the study was to compare oxidative stress level among surgical and non-surgical physicians who smoke different types of cigarettes (including conventional cigarettes, e-cigarettes and tobacco heating products). Finally, the Fagerström's test for nicotine dependence was used to determine degree of nicotine addiction.

Methods

Study design

This prospective study was conducted on surgical and non-surgical physicians who smoke various types of cigarettes (including conventional cigarettes (CS), e-cigarettes (EC) and tobacco heating products (THP)). The objective of this study was to determine potential differences in reactive oxygen species (ROS) level among such group of physicians. The study have been Approved by Ethical Committee of Medical University of Lodz (RNN/74/22/KE). All research was performed in accordance with the Declaration of Helsinki and a good clinical practice guidance.

Definitions

Current smoker is defined as a person who reports any tobacco products use on a daily (every day) or less than daily (not every day, occasionally) basis at the time of survey. Former smoker is defined as a person who reports any tobacco products use on a daily (every day) or less than daily (not every day, occasionally) basis, but quit smoking at least 2 years before the time of survey. Current vaper is defined as a person who reports exclusively electronic cigarettes use on a daily (every day) or less than daily (not every day, occasionally) basis from at least 2 years before the time of survey. Current tobacco heating product smoker is defined as a person who reports exclusively HnB cigarettes use on a daily (every day) or less than daily (not every day, occasionally) basis from at least 2 years before the time of survey. Never smoker is defined as a person who has never smoked any tobacco products at the time of survey.

Patients

For this study a randomly selected group of physicians worked at the Provincial Multidisciplinary Oncology and Traumatology Centre in Lodz (Poland) and at the Polish Mother's Health Centre Institute in Lodz (Poland) was qualified. After obtaining written informed consent from each study participant, physicians were asked to complete anonymous study questionnaire.

The inclusion criteria for the study were: age over 26 years, smoking of different types of cigarettes (conventional, electronic cigarettes or tobacco heating products (exclusive use from at least 2 years)), and surgical or nonsurgical specialization of physicians. As a control group, physicians with no history of smoking any tobacco products were included in this study. Physicians with chronic inflammatory diseases, autoimmune diseases, taking antimicrobial, non-steroidal anti-inflammatory drugs in the last three months, having acute infections, administering antioxidants, vitamins and other products that may affect the antioxidant activity of plasma and also those physicians who were unwilling to participate in the study were excluded from the study.

The sample size $n = 198$, was calculated based on the statistical data on the number of physicians who worked in Lodz, 95% confidence intervals Z score (1.96), margin of error (5%). The population proportion (P) was set as 0.16 in relation to the latest literature data on smoking prevalence among physicians [35]. For at least 198 participants, sampling error was calculated at 6.96%. Based on the sample size ($n = 50$ participants per group; 4 groups), test type (One-Way ANOVA on Ranks), significance level ($\alpha = 0.05$), predicted effect size ($\eta^2 = 0.2$ (large effect size)), statistical power has been calculated at 82.2%.

Questionnaire development and administration

The study was based on a self-developed questionnaire, which consisted of 16 questions. Furthermore, the Fagerström's test for nicotine dependence (FTND) was used. The study questionnaire contained questions on personal data of the study participants, their specialization as well as smoking status, current/past diseases, used medications/allergies and addictions. The study participants were asked to mark the answers in closed questions, or enter personal information (age, height, weight) and type of specialization. In the case of questions about diseases, medications taken and allergies, after marking the option "yes", the participants were asked to indicate them. English translation of the study questionnaire has been presented in Supplementary file 1.

To generalize the results obtained in this research study, the research team decided to enroll physicians (with different specializations) from two large medical centres in Lodz. In addition, the study questionnaire was pre-tested by the research team before being launched. Moreover, the study questionnaire was completed during meeting with the research team member, to ensure that the participants are familiarized with the study questionnaire and how to completed it. In addition, the participants had been informed how data would be handled to ensure anonymity and confidentiality of any personally identifiable information (PII). Privacy of the participants' information was protected during the recruitment of the participants, data collection and analysis, findings reporting/dissemination, data storage and archiving.

Collected data

Study participants were asked to complete an anonymous study questionnaire (multiple-choice, close and open-ended questions and FTND). The questionnaire collected data regarding: personal information (age, gender, height, weight, place of residence of study participants), smoking status (smoking duration and types of smoked tobacco products), education stage (during or with specialization), specialization type (surgical/nonsurgical; type of specialization), current/past diseases, addiction (cigarettes, coffee, alcohol), used medications and allergies.

Fagerström's test for nicotine dependence

The Fagerstrom's test for nicotine dependence was designed to provide an ordinal tool for measure nicotine dependence associated with smoking. This test consisted of yes/no questions (scored: yes = 1, no = 0) and multiple-choice questions (scored 0–3). Question answers are summed to yield a total score from 0 to 10. Score 0–3 means no signs of biological addiction or very low level of nicotine addiction, 4–6 means the presence of signs of biological addiction to nicotine, and 7–10 means the presence of signs of biological and psychological addiction to nicotine [36].

Detection of differences in oxidative stress indicators among physicians

To detect the differences in oxidative stress indicators among physicians with different specialization who smoke different types of cigarettes, 10 ml of blood from cubital vein was collected from the participants into the test tube with heparin and placed in a secured test tube that was used for laboratory tests. To measurement of intracellular hydroxyl activity, hydrogen peroxide and other reactive oxygen species (ROS) in the cytosol, the Cellular Reactive Oxygen Species (ROS) Assay Kit

(Creative Biolabs Inc, NY, USA) was used. The heparin-treated blood samples in a 50-ml tube were diluted with phosphate-buffered saline (PBS) (Sigma Aldrich, MO, USA) in a 1:3 ratio. Gradisol L (Aqua-Med, Lodz, Poland) was introduced into a 15 ml test tube and then blood suspended in PBS was added into in a ratio 1:1 to maintain the phase boundary. Then the tubes were centrifuged for 20 min at 20 °C, 2000 rpm and a ring of interphase mononuclear cells was collected. The collected lymphocytes were twice washed in PBS and centrifuged at 4 °C at 200 g. Each sample of lymphocytes were stained with 100 µl of H2DCFDA working reagent at 50 µM and incubated for 1 h at 37 °C in 96-well plates. Then, cells were washed once in 1x Assay Buffer and treated with hydrogen peroxide (H_2O_2) at 50 µM, which was used as a positive control. In the next step, the cells were incubated for 1 h. Signal intensity was measured using a fluorescence microplate reader at Ex/Em = 485/530 nm. The fluorescence intensity of the sample is directly proportional to the level of ROS in the cytosol. The final results are the mean of three independent measurements.

Statistical analysis

The analysed results were expressed as means ± standard deviations (SD) for continuous variables and numbers and percentages for categorical variables. Normality of distribution of the tested quantitative parameters was verified using the Shapiro-Wilk test. The statistically significant differences in ROS level between surgical and non-surgical specialties as well as differences in Fagerström's score were analysed by Mann-Whitney U test. One-way ANOVA on ranks with Dunn's method was used to analyse differences in ROS level between types of smoked cigarettes Pearson chi^2 test was used to determine linear association between physicians specialization and nicotine addiction (Fagerström's test for nicotine dependence). Odds ratios (OR) with 95% confidence intervals (CI) were calculated to determine the relationship between the physicians' specialization and the nicotine addiction (Fagerström's test for nicotine dependence). The data collected in the study were analysed using a SigmaPlot 12.3 (Systat Software Inc., Palo Alto, CA, USA) software. $p < 0.05$ was considered significant.

Results

Participants' characteristics

Our study involved 215 physicians working in various departments of the Provincial Multidisciplinary Oncology and Traumatology Centre in Lodz (Poland) and at the Polish Mother's Health Centre in Lodz (Poland). 10 physicians did not complete the anonymous research questionnaire, therefore in the final analysis, 205 participants were included.

The analysed group of physicians consisted of 75 (36.6%) women and 130 men (63.4%) with the mean age 40.6 ± 8.813 years, height 1.77 ± 0.091 m, body weight 80.34 ± 12.608 kg and BMI 25.7 ± 3.383. There was a similar number of non-smokers (50 participants (24.4%)), conventional cigarette smokers (55 participants (26.8%)), e-cigarette smokers (50 participants (24.4%)) and tobacco heating product smokers (50 participants (24.4%)) included in this study. Over 2 out of 5 participants had smoked tobacco products for 5–10 years, almost one fourth for 10–20 years, almost 1 out of 5 less than 5 years, 1 out of 10 for 20–30 years and almost 1 out of 20 over 30 years. Tobacco products (75.6% of participants), followed by coffee (68.3% of participants) and alcohol (45.4% of participants) were reported as addictive. Detailed demographic characteristics with analysis of differences between the type of specialization are presented in Table 1.

Nicotine addiction among physicians with surgical and non-surgical specialties

Based on the Fagerström's test for nicotine dependence, 35.1% of surgical and 40.5% of non-surgical specialization of physicians indicated signs of nicotine addiction. Furthermore, the Fagerström score was significantly higher in surgical than non-surgical physician specialties (Fagerström score for surgical: 5.4 ± 1.372; for non-surgical: 4.7 ± 1.310 specialties; mean score: 5.0 ± 1.379; $p = 0.001$). There was a significantly higher Fagerström score in non-surgical conventional cigarettes smokers (5.1 ± 1.499) and e-cigarette smokers (4.82 ± 1.020) compared to tobacco heating product smokers (4.1 ± 1.187; $p < 0.05$). Moreover, higher Fagerström test score among tobacco heating product smokers with surgical specialization compared to non-surgical specialization (5.3 ± 1.030 vs. 4.1 ± 1.187; $p < 0.001$) has been observed (Fig. 1). Pearson correlation coefficient did not reveal any correlation between physicians specialties and nicotine addiction ($p > 0.05$). In addition, there was any statistically significant difference in the odds of nicotine addiction in smokers with surgical ($p > 0.05$) and non-surgical ($p > 0.05$) specialties (Table 2).

The differences in ROS level among smokers of different type of cigarettes

Significantly higher reactive oxygen species (ROS) level was observed in conventional cigarette (CS) smokers (DCF fluorescence intensity for ROS: 21,290.54 ± 3,258.474), e-cigarette (EC) smokers (DCF fluorescence intensity for ROS: 21,722.84 ± 3,459.013) and tobacco heating products (THP) smokers (DCF fluorescence intensity for ROS: 17,585.8 ± 5,125.717) compared to non-smokers (DCF fluorescence intensity for ROS: 11,308 ± 979.175; $p < 0.05$). In addition, there was

Table 1 Participants' characteristic

		Type of specialization (n, %)		
		surgical	non-surgical	total
Sex				
	men	68 (33.2%)	62 (30.2%)	130 (63.4%)
	women	29 (14.2%)	46 (22.4%)	75 (36.6%)
Age (years)*		40.2 ± 8.263	41 ± 9.307	40.6 ± 8.813
Height (m)*		1.78 ± 0.091	1.76 ± 0.091	1.77 ± 0.091
Weight (kg)*		81.1 ± 10.913	79.7 ± 13.973	80.34 ± 12.608
Body mass index (BMI)*		25.6 ± 2.869	25.7 ± 3.798	25.7 ± 3.383
Smoking status				
	non-smoker	20 (9.8%)	30 (14.6%)	50 (24.4%)
	CS smoker	25 (12.2%)	30 (14.6%)	55 (26.8%)
	EC smoker	22 (10.7%)	28 (13.6%)	50 (24.4%)
	THP smoker	23 (11.2%)	27 (13.2%)	50 (24.4%)
Smoking duration				
	< 5 years	9 (5.8%)	20 (12.9%)	29 (18.7%)
	≥ 5 years and < 10 years	33 (21.3%)	33 (21.3%)	66 (42.6%)
	≥ 10 years and < 20 years	22 (14.2%)	15 (9.7%)	37 (23.9%)
	≥ 20 years and < 30 years	7 (4.5%)	9 (5.8%)	16 (10.3%)
	≥ 30 years	1 (0.6%)	6 (3.9%)	7 (4.5%)
Addictives				
	tobacco products	77 (47.3%)	78 (52.7%)	155 (75.6%)
	coffee	75 (36.6%)	65 (31.7%)	140 (68.3%)
	alcohol	52 (25.4%)	41 (20%)	93 (45.4%)
Diseases				
	hypertension	14 (6.8%)	9 (4.4%)	23 (11.2%)
	Hashimoto	4 (2%)	8 (3.9%)	12 (5.8%)
	diabetes mellitus	5 (2.4%)	4 (2%)	9 (4.4%)
	hypothyroidism	2 (1%)	4 (2%)	6 (2.9%)
	hyperthyroidism	1 (0.5%)	0 (0%)	1 (0.5%)
Education stage				
	in the process of specialization	20 (9.8%)	26 (12.7%)	46 (22.4%)
	with specialization	75 (36.6%)	84 (41%)	159 (77.6%)
Specialization				
	urology	10 (4.9%)	0 (0%)	10 (4.9%)
	cardiology	0 (0%)	10 (4.9%)	10 (4.9%)
	vascular surgery	12 (5.8%)	0 (0%)	12 (5.8%)
	oncological surgery	5 (2.4%)	0 (0%)	5 (2.4%)
	neurosurgery	12 (5.8%)	0 (0%)	12 (5.8%)
	intern	0 (0%)	10 (4.9%)	10 (4.9%)
	radiology	0 (0%)	5 (2.4%)	5 (2.4%)
	haematology	0 (0%)	12 (5.8%)	12 (5.8%)
	thoracic surgery	8 (3.9%)	0 (0%)	8 (3.9%)
	anaesthesiology	0 (0%)	21 (10.2%)	21 (10.2%)
	gynaecology	9 ($0.4%)	0 (0%)	9 (4.4%)
	nephrology	0 (0%)	9 (4.4%)	9 (4.4%)
	orthopedy	8 (3.9%)	0 (0%)	8 (3.9%)
	rheumatology	0 (0%)	8 (3.9%)	8 (3.9%)
	surgery	15 (7.3%)	0 (0%)	15 (7.3%)
	neurology	0 (0%)	4 (2%)	4 (2%)
	pediatry	0 (0%)	7 (3.4%)	7 (3.4%)
	neonatology	0 (0%)	5 (2.4%)	5 (2.4%)
	cardiac surgery	5 (0%)	0 (0%)	5 (2.4%)
	gastroenterology	0 (0%)	6 (2.9%)	6 (2.9%)

Table 1 (continued)

	Type of specialization (*n*, %)		
	surgical	non-surgical	total
paediatric surgery	0 (0%)	4 (2%)	4 (2%)
endocrinology	0 (0%)	7 (3.4%)	7 (3.4%)
otorhinolaryngology	6 (2.9%)	0 (0%)	6 (2.9%)
ophthalmology	7 (3.4%)	0 (0%)	7 (3.4%)

* - presented as mean ± SD; Abbreviations: BMI– body mass index, CS– conventional cigarette, EC– e-cigarette, THP– tobacco heating products

a significantly lower ROS level in THP smokers compared to CS smokers ($p < 0.05$) and EC smokers ($p < 0.05$) (Fig. 2).

The differences in ROS level among physicians with surgical and non-surgical specialties

Among surgical specialties physicians, there was a significantly higher level of ROS in CS smokers (DCF fluorescence intensity for ROS: 23,219.12 ± 2,966.909), EC smokers (DCF fluorescence intensity for ROS: 22,840.364 ± 2,698) and THP smokers (DCF fluorescence intensity for ROS: 16,891.407 ± 5,240.548) compared to non-smokers (DCF fluorescence intensity for ROS: 11,737.571 ± 1,168.881) ($p < 0.05$). In addition, there was a higher ROS level in CS and EC smokers with surgical specialties compared to THP smokers with surgical specialties ($p < 0.05$). Among non-surgical specialties physicians, there was a significantly higher level of ROS in CS smokers (DCF fluorescence intensity for ROS: 19,361.96 ± 2,264.147), EC smokers (DCF fluorescence intensity for ROS: 20,844.786 ± 3,772.944) and THP smokers (DCF fluorescence intensity for ROS: 18,255 ± 5,015.239) compared to non-smokers (DCF fluorescence intensity for ROS: 11, 100.552 ± 724.869) ($p < 0.05$). Furthermore, there was a significantly higher ROS level in CS ($p < 0.001$) and EC ($p = 0.004$) smokers with surgical compared to non-surgical specialties (Fig. 3).

Discussion

Our study shown that 35.1% of surgical and 40.5% of non-surgical physicians indicated signs of nicotine addiction. The Fagerström score was significantly higher in surgical than non-surgical physician specialties (5.4 ± 1.372 vs. 4.7 ± 1.310; $p = 0.001$), in non-surgical CS (5.1 ± 1.499) and EC smokers (4.82 ± 1.020) compared to THP smokers (4.1 ± 1.187; $p < 0.05$) and surgical THP smokers compared to non-surgical THP smokers (5.3 ± 1.030 vs. 4.1 ± 1.187; $p < 0.001$). Furthermore, significantly higher ROS level was observed in conventional cigarette (CS) smokers, e-cigarette (EC) smokers and tobacco heating products (THP) smokers compared to non-smokers ($p < 0.05$). In addition, there was a significantly lower ROS level in THP smokers compared to CS smokers ($p < 0.05$) and EC smokers ($p < 0.05$). There was a significantly higher ROS level in physicians with surgical specialization who smoke CS ($p < 0.001$) and EC ($p = 0.004$) compared to non-surgical physicians.

To our knowledge, this is the first research which compared the ROS level among physicians with surgical and non-surgical specializations who smoked different tobacco products. Therefore, we discussed our findings in relation to ROS level in different tobacco products and to results of another studies of ROS concentration in TCs, ECs and THPs smokers. $DCFH_2$ fluorescence method indicated ROS concentration in mainstream cigarette smoke at 18.64–54.81 nmol H_2O_2/l, while 71.21–85.99% of the total amount (14.32–39.03 nmol H_2O_2/l) existed in the gaseous phase [37]. In the another study, total quantities of ROS in the mainstream smoke was 120–150 nmol (regular CS) and 90–110 nmol (light CS) [38]. There was a significantly ($p < 0.05$) higher oxidative stress index (OSI) and total oxidant status in smokers compared to non-smokers [39]. Cigarette smokers have higher serum cotinine level of ROS compared to non-smokers, including: superoxide anion (259.6 ± 63.1 nM vs. 145.3 ± 52.7 nM; $p \le 0.001$), hydroxyl radical (275.3 ± 57.2 nM vs. 149.5 ± 38.1 nM; $p \le 0.001$), singlet oxygen (116.0 ± 47.9 nM vs. 92.1 ± 12.7 nM; $p \le 0.05$), hydrogen peroxide (891.4 ± 153.7 nM vs. 235.1 ± 64.8 nM; $p \le 0.001$), hypochlorite radical (107.2 ± 18.6 nM vs. 53.4 ± 9.5 nM; $p \le 0.001$) and peroxyl radical (138.1 ± 21.5 nM vs. 76.2 ± 11.9 nM; $p \le 0.001$) [40]. The flow cytometry analysis of immune cells subtypes shown greater cellular oxidative stress (COS) in CS smokers compared to non-smokers ($p < 0.05$), as well as to EC smokers ($p < 0.05$). In addition, there was a greater COS in EC smokers compared to non-smokers ($p < 0.05$) [41]. Compared to non-smokers, the moderate to heavy smokers showed significantly higher ROS level in seminal plasma (24.10 ± 18.50 vs. 12.20 ± 8.10 nmol/mg prot; $p < 0.05$). In addition, smoking was positively correlated with the ROS level in seminal plasma ($r = 0.235$; $p < 0.05$), sperm DNA fragmentation index (DFI) in the preconception males ($r = 0.387$, $p < 0.01$) and negatively correlated with the total sperm count (r = -136, $p < 0.05$), progressively motile sperm (PMS) (r = -0.381, $p < 0.01$) and morphologically normal sperm (MNS) (r = -0.218, $p < 0.01$) [42].

$DCFH_2$ fluorescence method determined a significant increase of ROS formation in supra-Ohm EC device

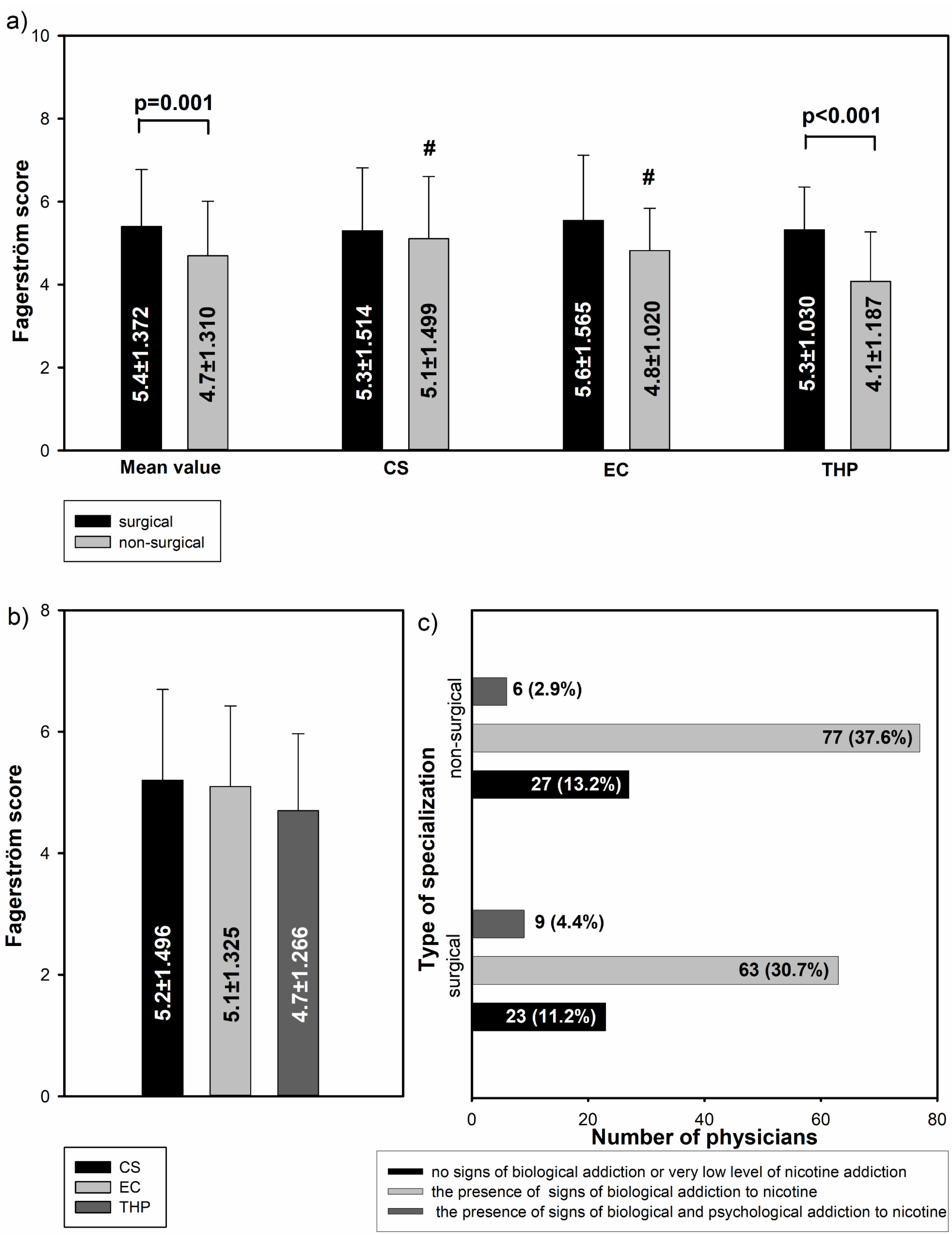

Fig. 1 (See legend on next page.)

(See figure on previous page.)

Fig. 1 Nicotine addiction among physicians with surgical and non-surgical specialization. Fagerström's test for nicotine dependence was performed among 205 physicians with surgical and non-surgical specialization who smoke different types of cigarettes. Differences between Fagerström score (**a-b**) for surgical and non-surgical specialization of physicians was analyzed by Mann-Whitney U test and for different cigarette types by ANOVA on ranks. The Fagerström score was presented as mean ± SD. $p < 0.05$ was considered significant. # for $p < 0.05$ (vs. THP smokers). No signs, biological signs or biological and psychical signs of nicotine addiction (**c**) were presented as a number and percentage of physicians with surgical and non-surgical specialization. Abbreviations: CS– conventional cigarette; EC– e-cigarette; THP– tobacco heating product

($p < 0.01$) and sub-Ohm EC device (SOD) ($p < 0.01$) with the increase of device power. In addition, ROS emission under 200 W in SOD device was similar to those for CS (1.143 ± 0.606 nmol/s vs. 1.240 ± 0.210 nmol/s). There was a significant correlation between ROS flux and power per surface area (P/SA) ($R^2 = 0.78$). Moreover, there was a significant ($p < 005$) decline in a ROS flux with the increase of vegetable glycerine (VG) content [43]. Another study shown, that tobacco-flavour (with 16 mg nicotine) and menthol-flavour (w/out nicotine) EC exposure significantly ($p < 0.05$) increases production of hydrogen peroxide compared to control [44]. Moreover, ROS concentration vary between liquids with different nicotine content and different flavours [45]. Electron spin resonance (ESR) confirmed generation of ROS by e-cigarettes in cellular and acellular systems. In addition, the ROS level was highly dependent on the EC brand, flavour, puffing pattern and device voltage. A novel acellular Trolox-based mass spectrometry method for total ROS and hydrogen peroxide (H_2O_2) detection indicated presence of 1.2–8.9 nmol H_2O_2/puff (approximately 12–68% of total ROS). There was up to 8-times more ROS in small airway epithelial cells (SAEC) exposed to EC aerosol compared to control [46]. Exposure of human bronchial epithelial cells to aqueous aerosol extracts (AqE) of EC leaded to 83% increase in generation of intracellular oxidant species, however oxidative stress was lower than for CS [47]. Human umbilical vein endothelial cells (HUVECs) exposure to 500 µM e-cigarette aerosol extract (EAE) significantly increased ROS level (4.5-fold higher level than control), however such level was lower than for exposure to 500 µM cigarette smoke extract (CSE) (7.8-fold higher level than control) [48]. Another study shown dose-dependent increase in ROS generation (regardless of the flavours) in human induced pluripotent stem cell-derived endothelial cells (hiPSC-ECs) exposed to various EC liquids [49].

$DCFH_2$ fluorescence method shown, that emission of ROS by THP was 85% lower than CS (6.26 ± 2.72 nmol H_2O_2/session vs. 46.83 ± 9.6 nmol H_2O_2/session) [50]. Another study shown a concentration-dependent increase in intracellular oxidant species level ($p < 0.05$) after exposure of NCI-H292 cells to aqueous extract of THP [51]. Animal model (male Sprague Dawley rats exposed for 4 weeks to THP smoke (4 sticks/day)) exposure to THP aerosol indicated a significant increase in ROS generation in S9 ($p < 0.01$) and microsomes ($p < 0.05$) fraction and liver tissue homogenate ($p < 0.01$) compare to unexposed control [52]. Another study showed significantly less amount of radical species in the vapour phase of tobacco heating cigarette (99% less of radical species) and no radical species in the particulate matter phase compared to conventional cigarette [53].

A meta-analysis of the studies on the prevalence of tobacco use among healthcare workers showed worldwide prevalence at 21% (95% CI 20–23%) which varied by World Bank income level ($p = 0.01$). The prevalence in tobacco use was 31% (95% CI: 28–34%) in male and 17% (95% CI: 15–18%) female healthcare workers and varied by income level ($p < 0.01$). The prevalence among physicians was 20% (95% CI: 19–22%) and varied by income level ($p < 0.01$), with higher tobacco use among males (29%, 95% CI: 25–32%) than females (12%, 95% CI: 10–13%) [54]. A cross sectional study of 180 active smoking healthcare workers shown that 72.2% of participants reported occupational stress (including 42.8% of mild, 20.6% of moderate and 8.9% of severe stress). Furthermore, 49.4% of respondents reported mild, 35.6% moderate and 15% severe nicotine dependence. There was a higher Fagerström score for women than men (3.75 vs. 2.83; $p = 0.025$) and significant association between occupational stress and nicotine dependence ($p = 0.011$) [55]. In the study of primary healthcare workers, 77.5% of participants reported the need of smoke/smokeless tobacco within 5–60 min after waking up. In addition, most of them (31.5%) were nervous and stressed. There was a significant ($p \leq 0.05$) association between high nicotine dependence and high perceived stress [56]. In a cross-sectional study of healthcare workers in the western region of the Kingdom of Saudi Arabia 58.3% of physicians had moderate, 33.3% mild and 8.3% severe nicotine dependence. Among physicians who smoke, 64.7% 'want to stop smoking now', 68.8% 'tried to stop smoking during the past year', 47.1% 'received help or advice to help you to stop smoking' and 58.8% 'who smoke less likely to advise patients to stop smoking'. In addition, 94.1% pointed need of specific training on cessation methods [57]. In another study, 18.7% of doctors were smokers. Moreover, very low level of nicotine dependence was reported by 35% of healthcare workers, while 11.9% had very high level of nicotine dependence [58].

Surgical specialists, already exposed to high-stress environments and often faced with occupational hazards, may experience a compounded effect of oxidative

Table 2 Correlation between specialization type and nicotine addiction (Fagerström's test for nicotine dependence)

			surgical				non-surgical			
	Pearson correlation		Odds ratio							
Nicotine addiction signs	**r**	**p**	**OR**	**95% CI**		**p**	**OR**	**95% CI**		***p***
no signs	−0.137	0.0898	0.9820	0.5181	1.8614	0.9556	1.0183	0.5372	1.9303	0.9556
biological	0.0106	0.896	0.8438	0.4680	1.5211	0.5720	1.1852	0.6574	2.1367	0.5720
biological and psychical	0.0671	0.148	1.8140	0.6211	5.2978	0.2761	0.5513	0.1888	1.6101	0.2761

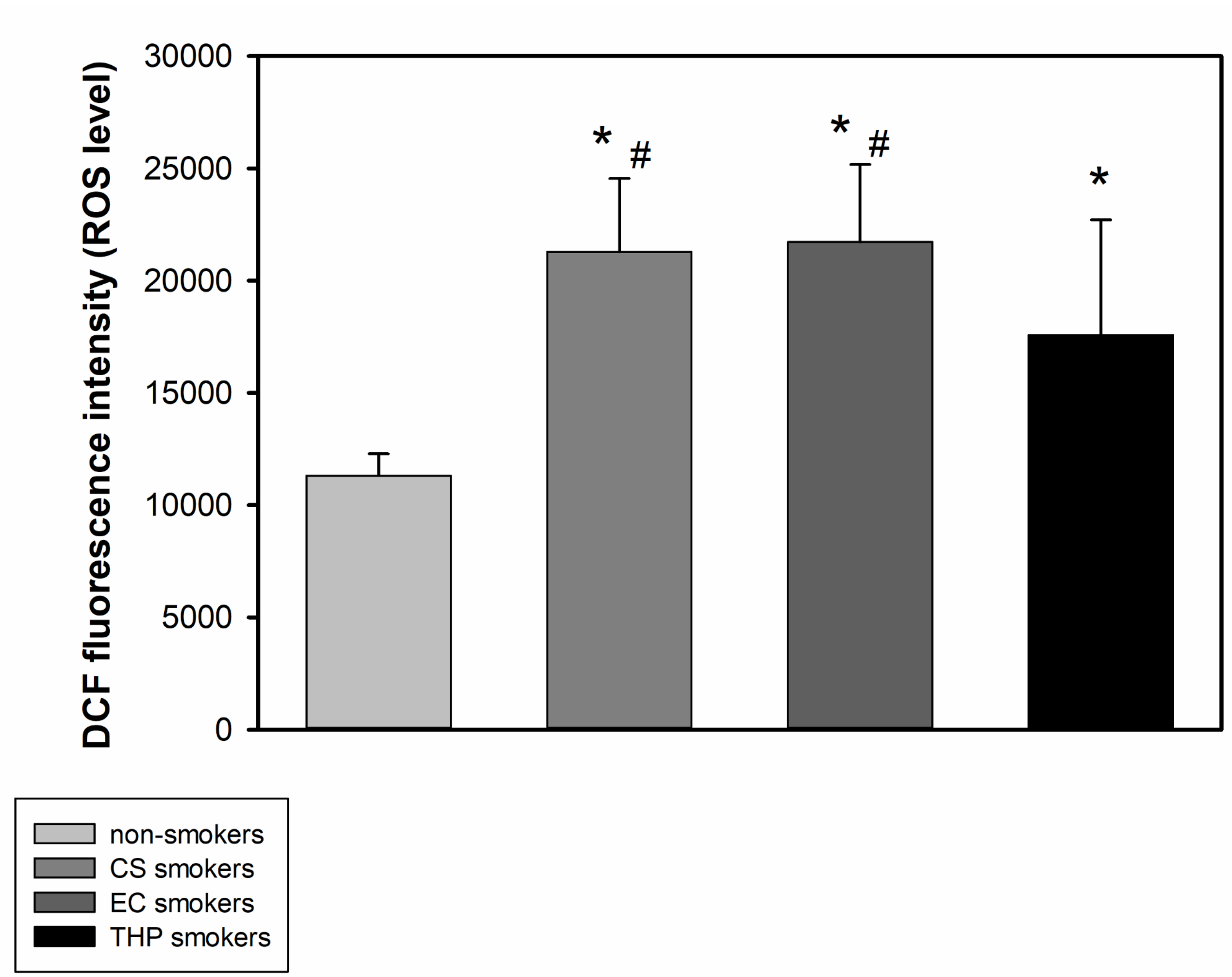

Fig. 2 The differences in ROS level among physicians who smoked different types of cigarettes. DCF fluorescence intensity for ROS were detected in the cytosol of lymphocytes isolated from the blood samples of 205 physicians with surgical and non-surgical specialization who smoke different types of cigarettes by the use of the cell permeation reagent dichlorodihydrofluorescein diacetate (H2DCFDA). The final results are the mean of three independent measurements. The values was presented as mean ± SD. * for $p < 0.05$ (vs. non-smokers); # for $p < 0.05$ (vs. THP smokers). Abbreviations: CS– conventional cigarette; EC– e-cigarette; THP– tobacco heating product

stress when combined with cigarette smoking. The intricate interplay between occupational stress, exposure to smoke, and the specific type of cigarettes smoked creates a complex scenario that demands closer examination [59–61]. Non-surgical specialists, while not directly exposed to smoke, still encounter occupational stressors that may interact with the type of cigarettes smoked [62]. However, stress levels can vary within the profession [33, 34]. Understanding how different cigarettes impact oxidative stress in non-surgical specialists provides valuable insights into the broader health implications of smoking habits [63, 64]. The findings from research in this area contribute to the broader goal of mitigating the health risks associated with cigarette smoking, especially within professional domains where stressors may already be heightened. Further investigations are essential for

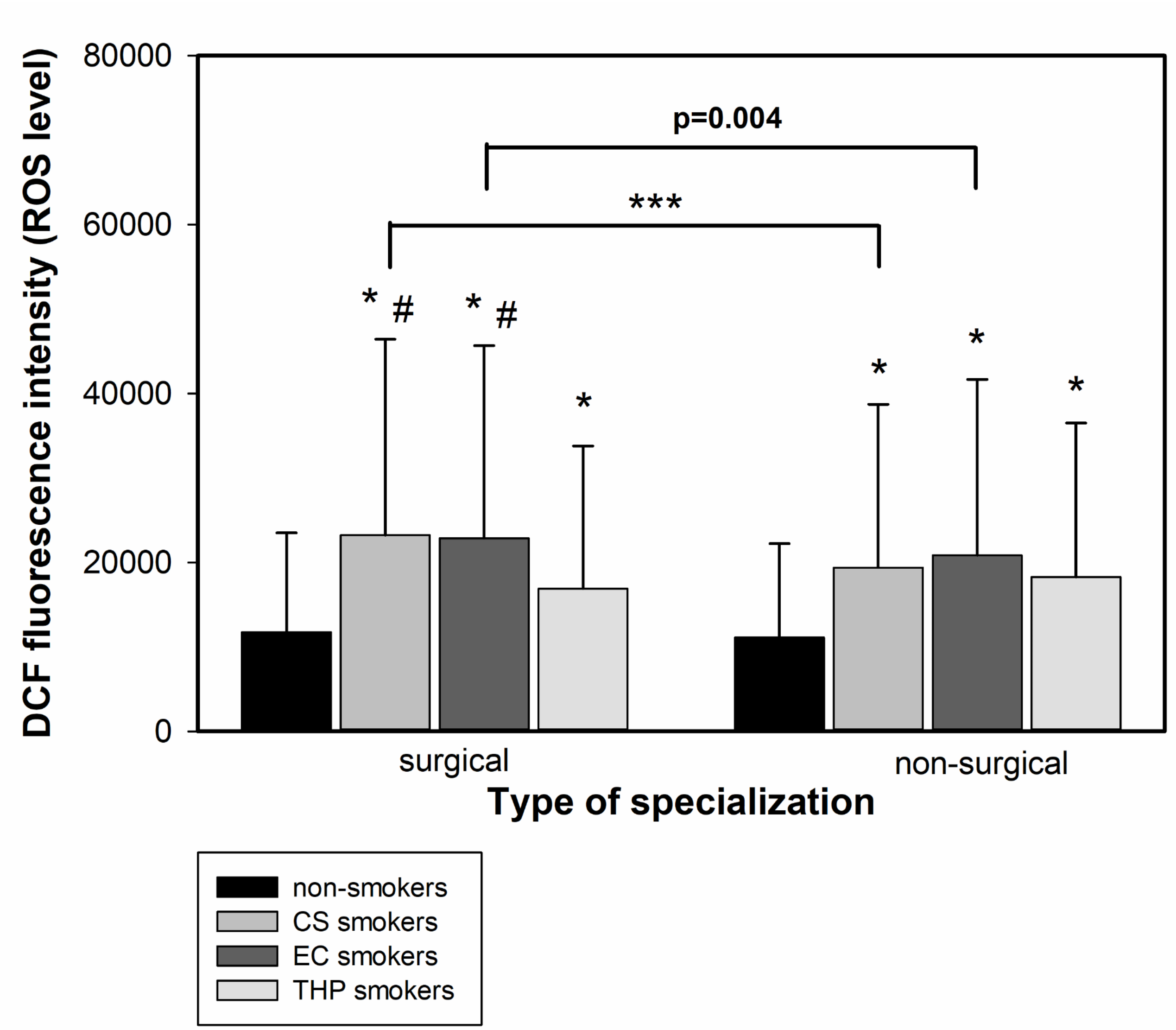

Fig. 3 The differences in ROS level among physicians with surgical and non-surgical specialization. DCF fluorescence intensity for ROS were detected in the cytosol of lymphocytes isolated from the blood samples of 205 physicians with surgical and non-surgical specialization who smoke different types of cigarettes by the use of the cell permeation reagent dichlorodihydrofluorescein diacetate (H2DCFDA). The final results are the mean of three independent measurements. The values was presented as mean ± SD. * for $p<0.05$ (vs. non-smokers); ** for $p<0.01$ (vs. non-smokers); *** for $p<0.001$ (vs. non-smokers) $ for $p<0.05$ (vs. CS smokers); # for $p<0.05$ (vs. THP smokers); Abbreviations: CS– conventional cigarette; EC– e-cigarette; THP– tobacco heating product

developing tailored approaches to address the nuanced relationships. Educating physicians about the harmful effects of cigarette smoking and promoting a healthy lifestyle should be an integral part of medical training. Additionally, addiction support and treatment programs should be available to physicians who want to stop smoking [65–67].

Our study have some limitations. Firstly, the data questionnaire completed by the physicians did not include a question about smoking history, only a question about the type of currently smoked cigarettes. Adding such data would make it possible to check whether study participants smoking e-cigarettes or heated tobacco products were already smokers and switched to using such alternative products, or whether this was their first choice (and analyse the effect of switching between tobacco products on ROS level). Secondly, the questionnaire did not include questions about occupational hazards and occupational stress, which may affect the level of oxidative stress of study participants. Thirdly, nicotine addiction was determined based on the results of the Fagerström test for nicotine dependence. These results could be extended by additionally using the Fagerström tolerance questionnaire. Fourthly, the methodology used in this study did not provide any control for potential confounders, such as diet, exercise, or other lifestyle factors, which could influence ROS levels. Further studies accounting for these variables would strengthen the conclusions. Moreover, clinical significance of such differences in the ROS level in terms of long-term health outcomes should be further explored. Finally, the sample was limited to physicians in one city in Poland, which may limit the generalizability of the results to other regions or populations.

Conclusions

Understanding the influence of the type of smoked cigarettes on oxidative stress in surgical and non-surgical physicians is imperative for targeted interventions and health promotion strategies. The findings from research in this area contribute to the broader goal of mitigating the health risks associated with cigarette smoking, especially within certain professional domains, such as medicine, where stressors can be heightened. Further investigations are essential for developing effective approaches to address the nuanced relationships between cigarette types, occupational stress, and oxidative stress in specialized medical fields.

Abbreviations

AqE	Aqueous aerosol extract
CI	Confidence interval
CO	Carbon monoxide
COPD	Chronic obstructive pulmonary disease
COS	Cellular oxidative stress
CS	Conventional cigarette
CSE	Cigarette smoke extract
DFI	DNA fragmentation index
EAE	E–cigarette aerosol extract
EC	Electronic cigarette
ESR	Electron spin resonance
FTND	Fagerström's test for nicotine dependence
H2DCFDA	Dichlorodihydrofluorescein diacetate
H_2O_2	Hydrogen peroxide
HnB	Heat not–burn
HPHC	Hazardous and potentially hazardous constituent
HUVEC	Human Umbilical Vein Endothelial Cells
OR	Odds ratio
OS	Oxidative stress
OSI	Oxidative stress index
P/SA	Power per surface area
PAH	Polyaromatic hydrocarbon
PBS	Phosphate–buffered saline
PII	Personally identifiable information
PMS	Progressively motile sperm
ROS	Reactive oxygen species
SAEC	Small airway epithelial cells
THP	Tobacco heating product
TSNA	Tobacco–specific nitrosamine
VG	Vegetable glycerin
VOC	Volatile organic compound

Supplementary information

The online version contains supplementary material available at https://doi.org/10.1186/s12890-025-03606-z.

Supplementary Material 1

Acknowledgements
Not applicable.

Author contributions
AN designing study, performing experiments, analysing/interpreting data and writing original draft; PN K-S analysing/interpreting data and writing original draft; RP designing/supervising study, acquiring funding, writing/reviewing and editing of original draft. All authors read and approved the final manuscript.

Funding
This work was supported by Medical University of Lodz Grant 503/0-149-03/503-01-001.

Data availability
All data generated or analysed during this study are included in this published article.

Declarations

Ethics approval and consent to participate
The study have been accepted by Ethical Committee of Medical University of Lodz (RNN/74/22/KE). Participants signed informed consent form before enrolment to the research study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 15 January 2025 / Accepted: 18 March 2025
Published online: 28 March 2025

References

1. National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health. The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General. Atlanta (GA): Centers for Disease Control and Prevention (US); 2014 [cited 2024 Apr 25]. (Reports of the Surgeon General). Available from: http://www.ncbi.nlm.nih.gov/books/NBK179276/
2. Rodgman A, Perfetti TA. The chemical components of tobacco and tobacco smoke. Boca Raton: CRC. 2008;1840.
3. Geiss O, Kotzias D. Tobacco, cigarettes and cigarette smoke. Luxembourg: Institute for Health and Consumer Protection, Directorate-General Joint Research Centre. 2007;4–11.
4. Brunnemann KD, Hoffmann D. Analytical studies on tobacco-specific N-nitrosamines in tobacco and tobacco smoke. Crit Rev Toxicol. 1991;21(4):235–40.
5. Edwards SH, Rossiter LM, Taylor KM, Holman MR, Zhang L, Ding YS, et al. Tobacco-Specific nitrosamines in the tobacco and mainstream smoke of U.S. Commercial cigarettes. Chem Res Toxicol. 2017;30(2):540–51.
6. Vu AT, Taylor KM, Holman MR, Ding YS, Hearn B, Watson CH. Polycyclic aromatic hydrocarbons in the mainstream smoke of popular U.S. Cigarettes. Chem Res Toxicol. 2015;28(8):1616–26.
7. Keith RJ, Fetterman JL, Orimoloye OA, Dardari Z, Lorkiewicz PK, Hamburg NM, et al. Characterization of volatile organic compound metabolites in cigarette smokers, electronic nicotine device users, dual users, and nonusers of tobacco. Nicotine Tob Res. 2019;22(2):264–72.
8. Bernhard D, Rossmann A, Wick G. Metals in cigarette smoke. IUBMB Life. 2005;57(12):805–9.
9. Hoffmann D, Hoffmann I. The changing cigarette, 1950–1995. J Toxicol Environ Health. 1997;50(4):307–64.
10. Valavanidis A, Vlachogianni T, Fiotakis K. Tobacco smoke: involvement of reactive oxygen species and stable free radicals in mechanisms of oxidative damage, carcinogenesis and synergistic effects with other respirable particles. Int J Environ Res Public Health. 2009;6(2):445–62.
11. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease. J Am Coll Cardiol. 2004;43(10):1731–7.

12. Liguori I, Russo G, Curcio F, Bulli G, Aran L, Della-Morte D, et al. Oxidative stress, aging, and diseases. Clin Interv Aging. 2018;13:757–72.
13. Pizzino G, Irrera N, Cucinotta M, Pallio G, Mannino F, Arcoraci V, et al. Oxidative stress: harms and benefits for human health. Oxid Med Cell Longev. 2017;2017:8416763.
14. Budani MC, Carletti E, Tiboni GM. Cigarette smoke is associated with altered expression of antioxidant enzymes in granulosa cells from women undergoing in vitro fertilization. Zygote. 2017;25(3):296–303.
15. Bjurlin MA, Kamecki H, Gordon T, Krajewski W, Matulewicz RS, Małkiewicz B, et al. Alternative tobacco products use and its impact on urologic health– will the lesser evil still be evil? A commentary and review of literature. Cent Eur J Urol. 2021;74(2):152–60.
16. Goniewicz ML, Kuma T, Gawron M, Knysak J, Kosmider L. Nicotine levels in electronic cigarettes. Nicotine Tob Res. 2013;15(1):158–66.
17. DeVito EE, Krishnan-Sarin S. E-cigarettes: impact of E-Liquid components and device characteristics on nicotine exposure. Curr Neuropharmacol. 2018;16(4):438–59.
18. Farsalinos KE, Yannovits N, Sarri T, Voudris V, Poulas K. Nicotine delivery to the aerosol of a Heat-Not-Burn tobacco product: comparison with a tobacco cigarette and E-Cigarettes. Nicotine Tob Res. 2018;20(8):1004–9.
19. Glantz SA. Heated tobacco products: the example of IQOS. Tob Control. 2018;27(Suppl 1):s1–6.
20. Wężyk-Caba I, Kaleta D, Zajdel R, Balwicki Ł, Świątkowska B. Do young people perceive E-Cigarettes and heated tobacco as less harmful than traditional cigarettes?? A survey from Poland. Int J Environ Res Public Health. 2022;19(22):14632.
21. Margham J, McAdam K, Forster M, Liu C, Wright C, Mariner D, et al. Chemical composition of aerosol from an E-Cigarette: A quantitative comparison with cigarette smoke. Chem Res Toxicol. 2016;29(10):1662–78.
22. Kim M. Philip Morris international introduces new heat-not-burn product, IQOS, in South Korea. Tob Control. 2018;27(e1):e76–8.
23. Uguna CN, Snape CE. Should IQOS emissions be considered as smoke and harmful to health?? A review of the chemical evidence. ACS Omega. 2022;7(26):22111–24.
24. Davis B, Williams M, Talbot P. iQOS: evidence of pyrolysis and release of a toxicant from plastic. Tob Control. 2019;28(1):34–41.
25. Marques P, Piqueras L, Sanz MJ. An updated overview of e-cigarette impact on human health. Respir Res. 2021;22(1):151.
26. Cao DJ, Aldy K, Hsu S, McGetrick M, Verbeck G, De Silva I, et al. Review of health consequences of electronic cigarettes and the outbreak of electronic cigarette, or vaping, product Use-Associated lung injury. J Med Toxicol. 2020;16(3):295–310.
27. Znyk M, Jurewicz J, Kaleta D. Exposure to heated tobacco products and adverse health effects, a systematic review. IJERPH. 2021;18(12):6651.
28. Dąbrowska A, Dudzik M, Pyc G, Majewski K, Kowalewski J, Pakos K et al. Impact of heated tobacco products on health– a comparative analysis with traditional cigarettes. J Pre Clin Clin Res. 2024 Jul 26 [cited 2025 Feb 16]; Available from: https://www.jpccr.eu/Impact-of-heated-tobacco-products-on-health-a-comparative-analysis-with-traditional,191338,0,2.html
29. Muthumalage T, Prinz M, Ansah KO, Gerloff J, Sundar IK, Rahman I. Inflammatory and oxidative responses induced by exposure to commonly used e-Cigarette flavoring chemicals and flavored e-Liquids without nicotine. Front Physiol. 2018;8:1130.
30. Sawa M, Ushiyama A, Inaba Y, Hattori K. Increased oxidative stress and effects on inflammatory cytokine secretion by heated tobacco products aerosol exposure to mice. Biochem Biophys Res Commun. 2022;610:43–8.
31. Buyukhatipoglu H, Kirhan I, Dag OF, Turan MN, Vural M, Taskin A, et al. Oxidative stress increased in healthcare workers working 24-Hour On-Call shifts. Am J Med Sci. 2010;340(6):462–7.
32. Rink LC, Oyesanya TO, Adair KC, Humphreys JC, Silva SG, Sexton JB. Stressors among healthcare workers: A summative content analysis. Glob Qual Nurs Res. 2023;10:23333936231161127.
33. LaPorta LD. Occupational stress in oral and maxillofacial surgeons: tendencies, traits, and triggers. Oral Maxillofac Surg Clin North Am. 2010;22(4):495–502.
34. Kang SH, Boo YJ, Lee JS, Ji WB, Yoo BE, You JY. Analysis of the occupational stress of Korean surgeons: A pilot study. J Korean Surg Soc. 2013;84:261–6.
35. Besson A, Tarpin A, Flaudias V, Brousse G, Laporte C, Benson A, et al. Smoking prevalence among physicians: A systematic review and Meta-Analysis. Int J Environ Res Public Health. 2021;18(24):13328.
36. Papadopoulou E, Haidich AB, Mathioudakis A, Tsavlis D, Papadopoulou K, Oikonomidou R, et al. The Fagerström test for nicotine dependence, as a prognostic factor, in current smokers with and without COPD: A cross-sectional study in Northern Greece. Chron Respir Dis. 2024;21:14799731241235213.
37. Huang MF, Lin WL, Ma YC. A study of reactive oxygen species in mainstream of cigarette. Indoor Air. 2005;15(2):135–40.
38. Zhao J, Hopke PK. Concentration of reactive oxygen species (ROS) in mainstream and sidestream cigarette smoke. Aerosol Sci Technol. 2012;46(2):191–7.
39. Ahmadkhaniha R, Yousefian F, Rastkari N. Impact of smoking on oxidant/antioxidant status and oxidative stress index levels in serum of the university students. J Environ Health Sci Eng. 2021;19(1):1043–6.
40. Khojah HM, Ahmed SA. Comparative assessment of individual RONS in serum of smokers compared with non-smokers and their correlation with the lipid profile and antioxidant status. J Int Med Res. 2019;47(12):6223–34.
41. Kelesidis T, Tran E, Arastoo S, Lakhani K, Heymans R, Gornbein J, et al. Elevated cellular oxidative stress in Circulating immune cells in otherwise healthy young people who use electronic cigarettes in a Cross-Sectional Single-Center study: implications for future cardiovascular risk. J Am Heart Assoc. 2020;9(18):e016983.
42. Bao HQ, Sun L, Yang XN, Ding J, Ma SY, Yang L, et al. [Impact of cigarette smoking on sperm quality and seminal plasma ROS in preconception males]. Zhonghua Nan Ke Xue. 2019;25(1):41–5.
43. Haddad C, Salman R, El-Hellani A, Talih S, Shihadeh A, Saliba NA. Reactive oxygen species emissions from Supra- and Sub-Ohm electronic cigarettes. J Anal Toxicol. 2019;43(1):45–50.
44. Lerner CA, Sundar IK, Yao H, Gerloff J, Ossip DJ, McIntosh S et al. Vapors Produced by Electronic Cigarettes and E-Juices with Flavorings Induce Toxicity, Oxidative Stress, and Inflammatory Response in Lung Epithelial Cells and in Mouse Lung. Khan MF, editor. PLOS ONE. 2015;10(2):e0116732.
45. Yogeswaran S, Muthumalage T, Rahman I. Comparative reactive oxygen species (ROS) content among various flavored disposable vape bars, including cool (Iced) flavored bars. Toxics. 2021;9(10):235.
46. Zhao J, Zhang Y, Sisler JD, Shaffer J, Leonard SS, Morris AM, et al. Assessment of reactive oxygen species generated by electronic cigarettes using acellular and cellular approaches. J Hazard Mater. 2018;344:549–57.
47. Taylor M, Carr T, Oke O, Jaunky T, Breheny D, Lowe F, et al. E-cigarette aerosols induce lower oxidative stress in vitro when compared to tobacco smoke. Toxicol Mech Methods. 2016;26(6):465–76.
48. Anderson C, Majeste A, Hanus J, Wang S, E-Cigarette Aerosol. Exposure induces reactive oxygen species, DNA damage, and cell death in vascular endothelial cells. Toxicol Sci. 2016;154(2):332–40.
49. Lee WH, Ong SG, Zhou Y, Tian L, Bae HR, Baker N, et al. Modeling cardiovascular risks of E-Cigarettes with human induced pluripotent stem Cell-Derived endothelial cells. J Am Coll Cardiol. 2019;73(21):2722–37.
50. Salman R, Talih S, El-Hage R, Haddad C, Karaoghlanian N, El-Hellani A et al. Free-Base and Total Nicotine, Reactive Oxygen Species, and Carbonyl Emissions From IQOS, a Heated Tobacco Product. Nicotine & Tobacco Research. 2018 Nov 23 [cited 2019 Feb 2]; Available from: https://academic.oup.com/ntr/advance-article/doi/https://doi.org/10.1093/ntr/nty235/5203659
51. Breheny D, Adamson J, Azzopardi D, Baxter A, Bishop E, Carr T, et al. A novel hybrid tobacco product that delivers a tobacco flavour note with vapour aerosol (Part 2): in vitro biological assessment and comparison with different tobacco-heating products. Food Chem Toxicol. 2017;106(Pt A):533–46.
52. Granata S, Canistro D, Vivarelli F, Morosini C, Rullo L, Mercatante D, et al. Potential harm of IQOS smoke to rat liver. Int J Mol Sci. 2023;24(15):12462.
53. Pryor WA, Church DF, Evans MD, Rice WY, Hayes JR. A comparison of the free radical chemistry of tobacco-burning cigarettes and cigarettes that only heat tobacco. Free Radic Biol Med. 1990;8(3):275–9.
54. Nilan K, McKeever TM, McNeill A, Raw M, Murray RL. Prevalence of tobacco use in healthcare workers: A systematic review and meta-analysis. PLoS ONE. 2019;14(7):e0220168.
55. Aranda C, García Pérez A, González-Aragón Pineda ÁE, Jiménez O. Association between occupational stress and nicotine dependence in health workers. Revista Med Del Instituto Mexicano Del Seguro Social. 2021;59:510–6.
56. Gaur T, Rao DS, Jadhav AA, Dagur LS, Dhaka H, Pathak P, et al. Nicotine dependence, perceived stress, and self-efficacy among primary health care professionals during the times of Covid-19 pandemic– A cross-sectional descriptive study. J Family Med Prim Care. 2021;10(11):4124.
57. Mahdi HA, Elmorsy SA, Melebari LA, Al-Masudi SM, Sharbini DA, Najjar AG, et al. Prevalence and intensity of smoking among healthcare workers and their attitude and behavior towards smoking cessation in the Western region of Saudi Arabia: A Cross-sectional study. Tob Prev Cessat. 2018;4:30.

58. Saglam L, Bayraktar R, Kadioglu EE, Acemoglu H. Smoking prevalance and the degree of nicotine dependence among healthcare workers at the Ataturk university medical facility. Eurasian J Med. 2010;42(2):74–7.
59. Landford WN, Ngaage LM, Lee E, Rasko Y, Yang R, Slezak S, et al. Occupational exposures in the operating room: are surgeons well-equipped? PLoS ONE. 2021;16(7):e0253785.
60. Ryu RC, Behrens PH, Malik AT, Lester JD, Ahmad CS. Are we putting ourselves in danger? Occupational hazards and job safety for orthopaedic surgeons. J Orthop. 2021;24:96–101.
61. Memon AG, Naeem Z, Zaman A, Zahid F. Occupational health related concerns among surgeons. Int J Health Sci (Qassim). 2016;10(2):279–91.
62. Joseph B, Arasu S. Occupational Risks and Health Promotion for Healthcare Workers. In: Haring R, Kickbusch I, Ganten D, Moeti M, editors. Handbook of Global Health. Cham: Springer International Publishing; 2020 [cited 2024 Apr 28]. pp. 1–29. Available from: https://doi.org/10.1007/978-3-030-05325-3_111-1
63. Dai X, Gil GF, Reitsma MB, Ahmad NS, Anderson JA, Bisignano C, et al. Health effects associated with smoking: a burden of proof study. Nat Med. 2022;28(10):2045–55.
64. West R. Tobacco smoking: health impact, prevalence, correlates and interventions. Psychol Health. 2017;32(8):1018–36.
65. Carson KV, Verbiest ME, Crone MR, Brinn MP, Esterman AJ, Assendelft WJ, et al. Training health professionals in smoking cessation. Cochrane Database Syst Rev. 2012;2012(5):CD000214.
66. Zhang G, Zhou J, Luk TT, Lam VSF, Yao Z, Wang MP, et al. The learning outcomes of smoking cessation training in undergraduate nursing students: A systematic review. Nurse Educ Pract. 2024;75:103907.
67. Juranić B, Rakošec Ž, Jakab J, Mikšić Š, Vuletić S, Ivandić M, et al. Prevalence, habits and personal attitudes towards smoking among health care professionals. J Occup Med Toxicol. 2017;12(1):20.

Publisher's note