

Respiratory and Cardiovascular Effects of Traditional versus Electronic Smoking Among Firefighters in Salah ad-Din/Samarra: An Age-Stratified Cross-sectional Study with Hormonal and Immunological Biomarkers

Omar Thaer Jawad

Department of Biology, College of Education, University of Samarra.

*Correspondence:

Aumrthaarjoad@uosamarra.edu.iq

Received: 09/08/2025

Revised: 11/09/2025

Accepted: 20/09/2022

Published: 30/09/2025

DOI:

2025 This article is an open access distributed under the terms and conditions of the Creative Commons Attribution License (CC BY 4.0)

ABSTRACT

Background: Firefighters face dual respiratory challenges from occupational smoke exposure and personal smoking habits. While electronic cigarettes (e-cigarettes) are often perceived as safer alternatives to traditional cigarettes, their cardiovascular and respiratory effects in occupationally exposed populations remain poorly understood.

Objective: To compare pulmonary function, inflammatory/oxidative stress markers, and cardiovascular indicators among traditional cigarette smokers, e-cigarette users, and non-smokers within an age-stratified firefighter cohort.

Methods: This cross-sectional analytical study enrolled 70 firefighters from Salah ad-Din/Samarra Fire Department, age-stratified into four groups (20-29, 30-39, 40-49, 50-59 years). Each age stratum included 5 traditional smokers, 5 e-cigarette users, and 7-8 non-smoking controls. Primary outcomes included spirometry parameters (FEV1, FVC, FEV1/FVC ratio), fractional exhaled nitric oxide (FeNO), high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), malondialdehyde (MDA), and total antioxidant capacity (TAC). Secondary assessments encompassed cardiovascular risk factors, stress hormones, and occupational exposure metrics.

Results: Traditional smokers demonstrated significantly reduced FEV1/FVC ratio ($76.2 \pm 8.4\%$ vs. $84.1 \pm 5.2\%$ in controls, $p < 0.001$) and elevated FeNO levels (42.3 ± 12.7 ppb vs. 18.6 ± 6.4 ppb, $p < 0.001$). E-cigarette users showed intermediate impairment (FEV1/FVC: $80.7 \pm 7.1\%$, FeNO: 28.9 ± 9.2 ppb). Inflammatory markers were highest in traditional smokers (hs-CRP: 4.8 ± 2.1 mg/L vs. 1.2 ± 0.6 mg/L in controls; IL-6: 3.4 ± 1.8 pg/mL vs. 1.1 ± 0.5 pg/mL), with e-cigarette users showing intermediate elevation. Oxidative stress markers followed similar patterns, with traditional smokers exhibiting highest MDA levels and lowest TAC. Age-stratified analysis revealed amplified smoking effects in older firefighters (≥ 40 years).

Conclusions: Traditional cigarette smoking produces more severe respiratory and systemic inflammatory effects than e-cigarette use among firefighters, though both demonstrate significant impairment compared to non-smokers. The interaction between occupational smoke exposure and personal smoking habits suggests cumulative pulmonary damage that accelerates with age.

Translational Perspective: These findings support targeted smoking cessation interventions in high-risk occupational populations and inform evidence-based policies regarding alternative tobacco products in safety-critical professions.

Conclusions: Even at an early stage of DN, T2DM patients show a pronounced pro-oxidant shift that parallels renal impairment. OS monitoring may help identify high-risk individuals, while intensified glycaemic control and antioxidant support could attenuate renal damage. Prospective studies should validate these findings and assess antioxidant-based interventions.

KEYWORDS : Electronic cigarettes, firefighters, pulmonary function, oxidative stress, occupational health, cardiovascular risk

INTRODUCTION

Firefighters represent a unique occupational population facing dual respiratory challenges: acute and chronic exposure to combustion products during emergency responses, and personal lifestyle factors including tobacco use. The prevalence of smoking among firefighters ranges from 15-25% globally, comparable to or slightly higher than general population rates, despite heightened awareness of respiratory health risks inherent to their profession.

The emergence of electronic cigarettes (e-cigarettes) as putative harm reduction alternatives has complicated the landscape of tobacco-related health risks. While population-level studies suggest reduced toxicant exposure compared to traditional cigarettes, the specific implications for occupationally exposed groups remain unclear. Firefighters encounter complex mixtures of particulates, volatile organic compounds, and combustion byproducts that may interact synergistically with nicotine delivery systems, potentially amplifying respiratory and cardiovascular consequences.

Traditional cigarette smoking impairs pulmonary function through multiple mechanisms: direct epithelial damage, increased airway reactivity, accelerated lung function decline, and elevated risk of chronic obstructive pulmonary disease (COPD). The inflammatory cascade initiated by cigarette smoke involves activation of alveolar macrophages, neutrophil recruitment, and release of pro-inflammatory cytokines including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α). Concurrently, oxidative stress markers such as malondialdehyde (MDA) increase while antioxidant defenses become depleted.

E-cigarettes deliver nicotine through heated aerosols containing propylene glycol, vegetable glycerin, flavorings, and various additives. While avoiding many combustion-related toxicants, emerging evidence suggests e-cigarette aerosols can induce inflammatory responses, oxidative stress, and cardiovascular dysfunction through distinct pathways. Propylene glycol and glycerin thermal decomposition products, flavoring compounds like diacetyl, and metal nanoparticles from heating coils represent potential respiratory irritants.

For firefighters, the interaction between occupational and personal smoke exposure may produce cumulative effects exceeding simple additive models. Occupational exposure primes inflammatory pathways, potentially heightening susceptibility to additional respiratory insults. Furthermore, the cardiovascular demands of firefighting—including extreme physical exertion, heat stress, and psychological stressors—may be particularly problematic in the context of nicotine-induced cardiovascular effects.

The present study addresses this knowledge gap through a comprehensive, age-stratified comparison of respiratory function, inflammatory biomarkers, oxidative stress indicators, and cardiovascular risk factors among firefighters using traditional cigarettes, e-cigarettes, or neither. By controlling for occupational exposure variables and employing sensitive biomarkers, this research aims to inform evidence-based policies and clinical recommendations for tobacco use in safety-critical occupations.

MATERIALS AND METHODS

Study Design and Setting

This cross-sectional analytical study was conducted at the Salah ad-Din/Samarra Fire Department between January and April 2024. The study protocol received approval from the Ethics Committee of the University of Samarra and the Salah ad-Din Health Directorate. All participants provided written informed consent prior to enrollment.

Participants

Inclusion Criteria:

- Active firefighters or fire department personnel
- Minimum one year of service
- Stable health status without acute illness
- Age 20-59 years
- Ability to perform spirometry testing

Exclusion Criteria:

- Respiratory infection within four weeks of testing
- Previously diagnosed severe asthma or advanced COPD
- Acute cardiovascular disease
- Systemic corticosteroid use within two weeks
- Concurrent waterpipe or chewing tobacco use

- Pregnancy
- Inability to abstain from smoking for 8 hours pre-testing

Study Groups

Participants were stratified by age (20-29, 30-39, 40-49, 50-59 years) and smoking status:

- **Traditional smokers:** Daily cigarette use ≥ 6 months, smoking ≥ 5 cigarettes/day
- **E-cigarette users:** Daily e-cigarette use ≥ 6 months, minimal traditional cigarette use (< 1 pack-year lifetime)
- **Non-smokers:** Never-smokers or former smokers with > 5 years cessation

Target enrollment: 5 traditional smokers and 5 e-cigarette users per age stratum ($n=40$), plus 30 non-smoking controls distributed across age groups (total $n=70$).

Primary Outcome Measures

Pulmonary Function: Spirometry was performed using a calibrated spirometer (CareFusion MasterScope, Germany) following ATS/ERS guidelines. Participants performed three acceptable maneuvers with best two FEV1 and FVC values within 150mL. Measured parameters included forced expiratory volume in 1 second (FEV1), forced vital capacity (FVC), FEV1/FVC ratio, peak expiratory flow (PEF), and forced expiratory flow 25-75% (FEF25-75).

Fractional Exhaled Nitric Oxide (FeNO): FeNO was measured using a portable analyzer (NIOX VERO, Circassia, UK) according to ATS guidelines. Participants exhaled at 50 mL/s flow rate after standard preparation.

Inflammatory Biomarkers: High-sensitivity C-reactive protein (hs-CRP) was measured by immunoturbidimetry. Cytokines (IL-6, TNF- α , IL-10) were quantified using commercial ELISA kits (R&D Systems, Minneapolis, MN).

Oxidative Stress Markers:

- Malondialdehyde (MDA): Thiobarbituric acid reactive substances assay
- Total antioxidant capacity (TAC): ABTS radical scavenging assay
- Antioxidant enzymes: Superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase (CAT) activities by spectrophotometric methods

Secondary Outcome Measures

Cardiovascular Assessment:

- Blood pressure (automated oscillometric measurement, three readings)
- Heart rate variability (5-minute ECG recording)
- Lipid profile (total cholesterol, LDL-C, HDL-C, triglycerides)
- Pulse wave velocity (PWV) using applanation tonometry where available

Hormonal Assessment:

- Morning cortisol (8-9 AM collection)
- Plasma catecholamines (epinephrine, norepinephrine)
- Testosterone (males only)

Exposure Validation:

- Serum cotinine (ELISA)
- Exhaled carbon monoxide
- Carboxyhemoglobin percentage
- Fagerström Test for Nicotine Dependence

Occupational Exposure:

- Years of firefighting service
- Number of fire incidents in preceding 6 months
- Days since last significant smoke exposure
- Personal protective equipment compliance

Laboratory Procedures

Participants fasted for 8 hours before blood collection. Morning samples (8-9 AM) were obtained for hormone assessment. Participants abstained from caffeine and nicotine for 8-12 hours prior to testing when possible. Serum and plasma samples were stored at -80°C until batch analysis.

Statistical Analysis

Data normality was assessed using Shapiro-Wilk tests. Continuous variables are presented as mean \pm standard deviation or median (interquartile range) as appropriate. Two-way ANOVA examined effects of age group

(4 levels) and smoking status (3 levels) with Tukey post-hoc corrections. ANCOVA adjusted for potential confounders including BMI, years of service, and HbA1c.

Correlation analyses and multiple regression models assessed relationships between exposure metrics (pack-years, cotinine levels, CO-Hb) and outcome measures. Sensitivity analyses excluded participants with recent occupational exposure (≤ 7 days). Statistical significance was set at $p < 0.05$. Analyses were performed using SPSS version 28.0.

3-RESULTS

Participant Characteristics

Seventy firefighters completed the study protocol (Table 1). Groups were well-matched for age, BMI, and years of firefighting service. Traditional smokers had significantly higher cotinine levels (242.1 ± 89.4 ng/mL) and CO-Hb percentages ($4.8 \pm 1.6\%$) compared to e-cigarette users (cotinine: 156.3 ± 67.2 ng/mL, CO-Hb: $2.1 \pm 0.8\%$) and controls (cotinine: 2.4 ± 1.1 ng/mL, CO-Hb: $0.8 \pm 0.3\%$). Fagerström scores indicated moderate nicotine dependence in traditional smokers (5.8 ± 2.1) versus mild dependence in e-cigarette users (3.2 ± 1.8).

Pulmonary Function Outcomes

Traditional smokers demonstrated significantly impaired spirometry parameters compared to controls (Table 2). FEV1/FVC ratio was reduced by 9.4% ($76.2 \pm 8.4\%$ vs. $84.1 \pm 5.2\%$, $p < 0.001$), with parallel decrements in FEV1 ($87.3 \pm 12.1\%$ vs. $96.8 \pm 8.7\%$ predicted, $p < 0.01$) and FEF25-75 ($72.4 \pm 18.9\%$ vs. $89.6 \pm 14.2\%$ predicted, $p < 0.001$). E-cigarette users showed intermediate impairment, with FEV1/FVC ratio of $80.7 \pm 7.1\%$ ($p < 0.05$ vs. controls, $p < 0.01$ vs. traditional smokers).

Age-stratified analysis revealed progressive worsening of smoking effects with advancing age. Among participants ≥ 40 years, traditional smokers showed FEV1/FVC ratios averaging 11.8% below age-matched controls, compared to 6.2% reduction in younger smokers ($p < 0.01$ for age \times smoking interaction).

Airway Inflammation

FeNO levels were markedly elevated in traditional smokers (42.3 ± 12.7 ppb vs. 18.6 ± 6.4 ppb in controls, $p < 0.001$), with e-cigarette users showing intermediate elevation (28.9 ± 9.2 ppb, $p < 0.001$ vs. controls, $p < 0.01$ vs. traditional smokers). Strong inverse correlations existed between FeNO and FEV1/FVC ratio ($r = -0.68$, $p < 0.001$) and between FeNO and FEF25-75 ($r = -0.71$, $p < 0.001$).

Systemic Inflammatory Markers

Traditional smokers exhibited significantly elevated inflammatory markers (Table 3). hs-CRP levels were 4-fold higher than controls (4.8 ± 2.1 vs. 1.2 ± 0.6 mg/L, $p < 0.001$), while IL-6 concentrations were 3-fold elevated (3.4 ± 1.8 vs. 1.1 ± 0.5 pg/mL, $p < 0.001$). TNF- α showed similar patterns (8.7 ± 3.2 vs. 3.1 ± 1.4 pg/mL, $p < 0.001$). E-cigarette users demonstrated intermediate elevation across all inflammatory markers, with statistical significance versus controls but lower levels than traditional smokers.

Anti-inflammatory IL-10 was paradoxically reduced in both smoking groups, suggesting impaired inflammatory resolution capacity.

Oxidative Stress Assessment

MDA levels, indicating lipid peroxidation, were highest in traditional smokers (3.8 ± 1.2 vs. 1.6 ± 0.7 nmol/mL in controls, $p < 0.001$), with e-cigarette users showing intermediate elevation (2.7 ± 0.9 nmol/mL). TAC was significantly depleted in traditional smokers (1.2 ± 0.4 vs. 2.1 ± 0.6 mmol/L, $p < 0.001$) and moderately reduced in e-cigarette users (1.7 ± 0.5 mmol/L).

Antioxidant enzyme activities followed similar patterns. SOD activity was reduced by 34% in traditional smokers and 18% in e-cigarette users compared to controls. GPx and CAT activities showed comparable decrements, suggesting comprehensive antioxidant system impairment.

Cardiovascular Risk Factors

Traditional smokers exhibited elevated systolic blood pressure (132.4 ± 14.8 vs. 118.6 ± 12.1 mmHg, $p < 0.001$) and reduced heart rate variability (SDNN: 28.3 ± 8.7 vs. 41.2 ± 11.4 ms, $p < 0.001$). Lipid profiles revealed increased LDL-C (142.3 ± 28.7 vs. 116.8 ± 22.4 mg/dL, $p < 0.001$) and reduced HDL-C (38.2 ± 8.9 vs. 48.6 ± 11.2 mg/dL, $p < 0.001$) in traditional smokers. E-cigarette users showed less pronounced but statistically significant changes in these parameters.

PWV measurements (available in 45 participants) indicated increased arterial stiffness in traditional smokers (8.9 ± 1.8 vs. 7.1 ± 1.2 m/s, $p < 0.01$).

Hormonal Profiles

Morning cortisol levels were elevated in traditional smokers (18.7 ± 5.2 vs. 12.1 ± 3.8 μ g/dL, $p < 0.001$), suggesting chronic stress axis activation. Plasma norepinephrine was increased in both smoking groups, with

highest levels in traditional smokers (387 ± 89 vs. 241 ± 67 pg/mL in controls, $p < 0.001$). Testosterone levels (males) were reduced in traditional smokers (412 ± 118 vs. 548 ± 142 ng/dL, $p < 0.01$).

Dose-Response Relationships

Pack-years in traditional smokers correlated significantly with FEV1/FVC ratio ($r = -0.59$, $p < 0.001$), FeNO ($r = 0.52$, $p < 0.01$), hs-CRP ($r = 0.48$, $p < 0.01$), and MDA ($r = 0.44$, $p < 0.05$). Among e-cigarette users, duration of use correlated with FeNO ($r = 0.41$, $p < 0.05$) and IL-6 levels ($r = 0.38$, $p < 0.05$).

Occupational Exposure Interactions

Years of firefighting service independently predicted reduced FEV1/FVC ratio ($\beta = -0.31$, $p < 0.01$) and elevated hs-CRP ($\beta = 0.28$, $p < 0.05$) after adjusting for smoking status. Participants with recent fire exposure (≤ 7 days) showed amplified inflammatory responses across all smoking groups, suggesting synergistic effects between occupational and personal smoke exposure.

Discussion

This comprehensive age-stratified study provides novel insights into the differential health effects of traditional versus electronic cigarette use among firefighters, a population facing unique occupational respiratory challenges. Our findings demonstrate a clear hierarchy of harm: traditional cigarettes produce the most severe impairment across pulmonary, inflammatory, and cardiovascular domains, while e-cigarettes cause intermediate effects that remain significantly worse than non-smoking controls.

Pulmonary Function Implications

The 9.4% reduction in FEV1/FVC ratio among traditional smokers represents clinically meaningful airway obstruction, approaching thresholds associated with increased respiratory symptoms and reduced exercise tolerance. The intermediate impairment in e-cigarette users (4.6% reduction) suggests that while potentially less harmful than traditional cigarettes, e-cigarettes are not benign alternatives. These findings align with emerging literature demonstrating e-cigarette-associated respiratory inflammation and airway remodeling, albeit through mechanisms distinct from combustible tobacco.

The pronounced age \times smoking interaction indicates accelerated lung function decline in older firefighters who smoke, potentially reflecting cumulative damage from dual exposure sources. This finding has particular relevance for career firefighters approaching retirement eligibility, as respiratory impairment may influence fitness-for-duty assessments and pension-related disability determinations.

Inflammatory and Oxidative Stress Pathways

The systemic inflammatory response pattern—characterized by elevated hs-CRP, IL-6, and TNF- α —demonstrates activation of pathways linked to cardiovascular disease risk, insulin resistance, and accelerated aging. The intermediate inflammatory profile in e-cigarette users suggests that while avoiding many combustion-related toxicants, e-cigarette aerosols retain significant pro-inflammatory potential.

The comprehensive oxidative stress assessment reveals concerning depletion of antioxidant defenses in both smoking groups. Given firefighters' occupational exposure to reactive oxygen species from combustion products, personal smoking may overwhelm already-stressed antioxidant systems, potentially accelerating oxidative damage to pulmonary and cardiovascular tissues.

Cardiovascular Risk Stratification

The cardiovascular risk profile associated with traditional smoking—elevated blood pressure, reduced HRV, adverse lipid changes, and increased arterial stiffness—represents a constellation of factors associated with increased myocardial infarction and stroke risk. For firefighters, whose occupational cardiac event rates already exceed general population norms, these additional risk factors may compound an already elevated baseline risk.

The intermediate cardiovascular effects of e-cigarettes align with recent clinical studies suggesting persistent hemodynamic and autonomic impacts despite reduced exposure to combustion products. Nicotine's direct cardiovascular effects, including increased heart rate, blood pressure, and sympathetic activation, likely contribute substantially to these observations.

Hormonal Implications

Elevated cortisol levels in smokers suggest chronic stress axis activation, potentially reflecting both nicotine's direct effects and psychological dependence patterns. The reduced testosterone levels in male traditional smokers may have implications for physical performance, recovery from training, and overall health maintenance in this physically demanding profession.

Occupational Health Considerations

The synergistic effects between occupational smoke exposure and personal smoking habits represent a critical finding for fire service medical programs. Our data suggest that firefighters who smoke may be at

disproportionately high risk for respiratory and cardiovascular complications, potentially justifying enhanced medical surveillance and targeted intervention programs.

The correlation between years of service and adverse health outcomes, independent of smoking status, underscores the cumulative nature of occupational respiratory exposure in firefighting. Combined with personal smoking effects, this suggests need for comprehensive approaches addressing both occupational protection and lifestyle modification.

Clinical and Policy Implications

These findings have several immediate clinical applications. First, they support aggressive smoking cessation interventions specifically tailored for firefighter populations, with emphasis on the additive risks associated with occupational exposure. Second, they suggest that e-cigarettes, while potentially less harmful than traditional cigarettes, should not be promoted as safe alternatives for firefighters given persistent inflammatory and cardiovascular effects.

For fire departments, these data support policies that discourage all forms of tobacco and nicotine use, provide comprehensive cessation resources, and potentially incorporate smoking status into fitness-for-duty assessments. Enhanced respiratory protection measures during fire suppression activities may be particularly important for firefighters who smoke.

Study Limitations

Several limitations merit consideration. The cross-sectional design precludes causal inference and cannot capture longitudinal changes in health parameters. The relatively small sample size may limit detection of subtle effects, particularly in age-stratified analyses. Additionally, while we controlled for recent occupational exposure, lifetime cumulative exposure variability may influence results.

Self-reported smoking behaviors, despite biochemical validation, may be subject to social desirability bias in this health-conscious occupational group. The exclusion of waterpipe users, while necessary for interpretability, may limit generalizability in regions where such use is common.

Future Research Directions

Longitudinal studies tracking firefighters over extended periods could better characterize the trajectory of smoking-related health effects and interaction with occupational exposures. Mechanistic studies examining inflammatory pathway activation and oxidative stress responses to acute smoking and occupational exposure could inform targeted interventions.

Investigation of smoking cessation interventions specifically designed for high-risk occupational populations represents an important translational research priority. Additionally, studies examining genetic polymorphisms affecting nicotine metabolism, inflammatory responses, and antioxidant capacity could enable personalized risk assessment and intervention strategies.

Conclusions

This study provides compelling evidence that traditional cigarette smoking produces severe respiratory and systemic health effects among firefighters that exceed those associated with e-cigarette use, though both forms of nicotine delivery cause significant impairment compared to non-smoking controls. The age-stratified analysis reveals accelerating effects of smoking with advancing age, while the comprehensive biomarker assessment demonstrates activation of inflammatory and oxidative stress pathways linked to cardiovascular disease risk.

For the fire service community, these findings underscore the critical importance of comprehensive tobacco control policies that address all forms of nicotine use. The interaction between occupational smoke exposure and personal smoking habits suggests particular vulnerability among firefighters who smoke, justifying enhanced medical surveillance and targeted intervention programs.

From a broader public health perspective, this research contributes to the growing evidence base regarding e-cigarette health effects in occupationally exposed populations, demonstrating that while potentially less harmful than traditional cigarettes, e-cigarettes are not risk-free alternatives.

Translational Perspective

The present study bridges a critical gap between basic mechanistic research on tobacco-related health effects and clinical applications in high-risk occupational populations. By demonstrating measurable biomarker changes associated with different forms of nicotine delivery in a real-world population facing unique occupational exposures, this research provides actionable evidence for clinical decision-making and policy development.

Immediate Clinical Applications:

1. **Risk Stratification:** The comprehensive biomarker panel provides objective measures for assessing smoking-related health risk in firefighters, supporting individualized medical surveillance and intervention strategies.
2. **Cessation Counseling:** Quantifiable evidence of inflammatory and oxidative stress responses provides powerful counseling tools for healthcare providers working with firefighter populations, demonstrating concrete health benefits of cessation.
3. **Occupational Health Programs:** The synergistic effects between occupational and personal smoke exposure support integration of smoking status into occupational health assessments and fitness-for-duty determinations.

Policy Translation: These findings provide evidence-based support for fire service policies addressing tobacco use, including enhanced smoking cessation resources, restriction of tobacco use in fire stations, and potential integration of smoking status into recruitment and retention decisions for safety-critical positions.

Research Translation: The biomarker framework established in this study provides a model for assessing intervention effectiveness in future smoking cessation trials specifically designed for high-risk occupational populations. The age-stratified approach offers insights into optimal timing for intervention strategies across career trajectories.

Public Health Impact: By demonstrating persistent health risks associated with e-cigarette use in a population already facing elevated baseline risk, this research contributes to evidence-based regulation of alternative tobacco products and supports comprehensive approaches to tobacco control in occupational settings.

Future Translational Opportunities: This work establishes a foundation for developing personalized intervention strategies based on biomarker profiles, occupational exposure history, and genetic factors affecting nicotine metabolism and inflammatory responses. Such approaches could optimize cessation success rates and minimize long-term health risks in safety-critical occupations.

Funding

This research was supported by grants from the University of Samarra Research Council and the Salah ad-Din Health Directorate. The funding sources had no role in study design, data collection, analysis, or manuscript preparation.

Conflict of Interest Statement

The authors declare no conflicts of interest related to this research.

Author Contributions

[Study conception and design], [Data collection and laboratory analyses], [Statistical analysis], [Manuscript preparation], [Critical revision]. All authors reviewed and approved the final manuscript.

Data Availability Statement

Anonymized datasets are available from the corresponding author upon reasonable request and with appropriate ethical approval.

Table 1: Baseline Characteristics of Study Participants

Characteristic	Traditional Smokers (n=20)	E-cigarette Users (n=20)	Non-smokers (n=30)	P-value
Age (years)	38.4 ± 8.7	36.9 ± 9.2	37.8 ± 8.9	0.73
BMI (kg/m ²)	27.2 ± 3.8	26.8 ± 3.4	26.1 ± 3.2	0.41
Years of service	12.8 ± 6.4	11.9 ± 5.8	12.3 ± 6.1	0.85
Systolic BP (mmHg)	132.4 ± 14.8 ^a	124.7 ± 11.2 ^b	118.6 ± 12.1	<0.001
Diastolic BP (mmHg)	84.2 ± 9.1 ^a	79.3 ± 7.8 ^b	75.1 ± 8.2	<0.01
Heart rate (bpm)	78.9 ± 12.3 ^a	74.2 ± 9.7	71.8 ± 10.1	<0.05
Pack-years	14.7 ± 8.9	-	-	-
E-cig duration (months)	-	18.3 ± 12.4	-	-
Fagerström score	5.8 ± 2.1	3.2 ± 1.8	-	<0.001
Cotinine (ng/mL)	242.1 ± 89.4 ^a	156.3 ± 67.2 ^b	2.4 ± 1.1	<0.001
CO-Hb (%)	4.8 ± 1.6 ^a	2.1 ± 0.8 ^b	0.8 ± 0.3	<0.001

^a P<0.05 vs. non-smokers; ^b P<0.05 vs. traditional smokers

Table 2: Pulmonary Function Parameters

Parameter	Traditional Smokers	E-cigarette Users	Non-smokers	P-value
FEV1 (% predicted)	87.3 ± 12.1 ^a	92.8 ± 9.4 ^b	96.8 ± 8.7	<0.01
FVC (% predicted)	91.4 ± 10.8	94.2 ± 8.9	97.1 ± 9.3	0.08
FEV1/FVC (%)	76.2 ± 8.4 ^a	80.7 ± 7.1 ^b	84.1 ± 5.2	<0.001
PEF (% predicted)	83.7 ± 15.2 ^a	89.1 ± 11.8	93.4 ± 12.1	<0.05
FEF25-75 (% predicted)	72.4 ± 18.9 ^a	81.3 ± 14.7 ^b	89.6 ± 14.2	<0.001
FeNO (ppb)	42.3 ± 12.7 ^a	28.9 ± 9.2 ^b	18.6 ± 6.4	<0.001

^a P<0.05 vs. non-smokers; ^b P<0.05 vs. traditional smokers

Table 3: Inflammatory and Oxidative Stress Markers

Biomarker	Traditional Smokers	E-cigarette Users	Non-smokers	P-value
hs-CRP (mg/L)	4.8 ± 2.1 ^a	2.9 ± 1.4 ^b	1.2 ± 0.6	<0.001
IL-6 (pg/mL)	3.4 ± 1.8 ^a	2.1 ± 1.2 ^b	1.1 ± 0.5	<0.001
TNF-α (pg/mL)	8.7 ± 3.2 ^a	5.9 ± 2.4 ^b	3.1 ± 1.4	<0.001
IL-10 (pg/mL)	2.1 ± 0.8 ^a	2.8 ± 0.9 ^b	3.7 ± 1.1	<0.001
MDA (nmol/mL)	3.8 ± 1.2 ^a	2.7 ± 0.9 ^b	1.6 ± 0.7	<0.001
TAC (mmol/L)	1.2 ± 0.4 ^a	1.7 ± 0.5 ^b	2.1 ± 0.6	<0.001
SOD (U/mL)	142.3 ± 28.7 ^a	178.4 ± 32.1 ^b	215.6 ± 41.2	<0.001
GPx (U/mL)	78.9 ± 18.4 ^a	94.2 ± 21.7 ^b	112.8 ± 24.3	<0.001

^a P<0.05 vs. non-smokers; ^b P<0.05 vs. traditional smokers

References

- Jahnke SA, Poston WS, Haddock CK, Murphy B. Firefighting and mental health: experiences of repeated exposure to trauma. *Work*. 2016;53(4):737-744.
- Soteriades ES, Smith RF, Tsismenakis AJ, Baur DM, Kales SN. Cardiovascular disease in US firefighters: a systematic review. *Cardiol Rev*. 2011;19(4):202-215
- Reidel B, Radicioni G, Clapp PW, et al. E-cigarette use causes a unique innate immune response in the lung, involving increased neutrophilic activation and altered mucin secretion. *Am J Respir Crit Care Med*. 2018;197(4):492-501.
- Goniewicz ML, Knysak J, Gawron M, et al. Levels of selected carcinogens and toxicants in vapour from electronic cigarettes. *Tob Control*. 2014;23(2):133-139.
- Bals R, Boyd J, Esposito S, et al. Electronic cigarettes: a task force report from the European Respiratory Society. *Eur Respir J*. 2019;53(2):1801151.
- U.S. Department of Health and Human Services. *The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General*. Atlanta, GA: USDHHS; 2014.
- World Health Organization. *WHO Report on the Global Tobacco Epidemic*. Geneva: WHO; 2021.
- Eisner MD, Anthonisen N, Coultas D, et al. "An official American Thoracic Society public policy statement: Tobacco control in the 21st century." *Am J Respir Crit Care Med*. 2010;182(5):596-606.
- Salvi S. "Tobacco smoking and chronic obstructive pulmonary disease: A review of the evidence." *Monaldi Arch Chest Dis*. 2010;73(2):57-64.
- Barnoya J, Glantz SA. "Cardiovascular effects of secondhand smoke: nearly as large as smoking." *Circulation*. 2005;111(20):2684-2698.
- Goniewicz ML, Knysak J, Gawron M, et al. "Levels of selected carcinogens and toxicants in vapour from electronic cigarettes." *Tob Control*. 2014;23(2):133-139.
- Bals R, Boyd J, Esposito S, et al. "Electronic cigarettes: a task force report from the European Respiratory Society." *Eur Respir J*. 2019;53(2):1801151.
- Reidel B, Radicioni G, Clapp PW, et al. "E-cigarette use causes a unique innate immune response in the lung." *Am J Respir Crit Care Med*. 2018;197(4):492-501.
- Wills TA, Soneji SS, Choi K, Jaspers I, Tam EK. "E-cigarette use and respiratory disorders: an integrative review." *Prev Med*. 2021;153:106766.

10. Glantz SA, Bareham DW. "E-cigarettes: use, effects on smoking, risks, and policy implications." *Annu Rev Public Health*. 2018;39:215-235.
11. Soteriades ES, Smith DL, Tsismenakis AJ, Baur DM, Kales SN. "Cardiovascular disease in US firefighters: a systematic review." *Cardiol Rev*. 2011;19(4):202-215.
12. Jahnke SA, Poston WS, Haddock CK, Murphy B. "Firefighting and mental health: experiences of repeated exposure to trauma." *Work*. 2016;53(4):737-744.
13. Bolstad-Johnson DM, Burgess JL, Crutchfield CD, Storment S, Gerkin R, Wilson JR. "Characterization of firefighter exposures during fire overhaul." *AIHAJ*. 2000;61(5):636-641.
14. Daniels RD, Kubale TL, Yiin JH, et al. "Mortality and cancer incidence in a pooled cohort of US firefighters." *Occup Environ Med*. 2014;71(6):388-397.
15. Ridker PM. "C-reactive protein and the prediction of cardiovascular events among those at intermediate risk: moving an inflammatory hypothesis toward consensus." *J Am Coll Cardiol*. 2007;49(21):2129-2138.
16. Calder PC, Ahluwalia N, Brouns F, et al. "Dietary factors and low-grade inflammation in relation to overweight and obesity." *Br J Nutr*. 2011;106(S3):S5-S78.
17. Rahman I, MacNee W. "Oxidative stress and regulation of glutathione in lung inflammation." *Eur Respir J*. 2000;16(3):534-554.
18. Zuo L, He F, Sergakis GG, et al. "Interrelated role of cigarette smoking, oxidative stress, and immune response in COPD and corresponding treatments." *Am J Physiol Lung Cell Mol Physiol*. 2014;307(3):L205-L218.
19. Rohleder N. "Stimulation of systemic low-grade inflammation by psychosocial stress." *Psychosom Med*. 2014;76(3):181-189.
20. Dai WS, Gutai JP, Kuller LH, Cauley JA. "Cigarette smoking and serum sex hormones in men." *Am J Epidemiol*. 1988;128(4):796-805.