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ORIGINAL ARTICLE

High sensitivity C-reactive protein and D-dimer levels in a sample of Iraqi cigarettes, shisha and electronic cigarettes smokers

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ABSTRACT

The tobacco pandemic constitutes one of the world's most serious global health hazards, killing about 8 million people each year. Since the emergence of various smoking methods, many claims and controversies have risen regarding the safety of each type of these smoking methods or which of them causes more adverse health effects. We aimed to assess how cigarette, shisha, and vape smoking affect levels of Hs-CRP and D-dimer markers tied to inflammation and clotting compared to Non-smokers. By doing so, we sought to clarify which smoking habit poses the highest cardiovascular or clot-related risk. This study involved the collection of blood samples from 100 cigarettes smokers, 100 shisha smokers, 100 electronic cigarettes (also known as vape) smokers and 200 apparently healthy subjects serving as the control group. Blood levels of Hs-CRP and D-dimer were measured in all of the study subjects. Cigarette smokers had the highest biomarker levels (11.85 mg/L Hs-CRP; 696.46 ng/mL D-dimer), followed by shisha and then vape users. All smoker groups showed far higher risks than non-smokers ($p < 0.000$). Even vaping, often perceived as safer, still raised risks compared to not smoking. The hierarchy is Cigarettes > Shisha > Vape—but no smoking method was risk-free. It is concluded that cigarette smokers are at higher risk for future cardiovascular and thrombotic events compared to shisha and vape smokers. Electronic cigarettes smoking (vaping) is safer compared to cigarettes and shisha smoking. However, even vaping might have an impact or adverse health effect compared to non-smokers.

Keywords HsCRP, D-dimer, Cigarettes, Shisha, Hooka, Vape

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INTRODUCTION

The links between smoking and increased morbidity and mortality have been long established, and current trends indicate that of the one billion smokers worldwide, 500 million

will die prematurely from smoking-related diseases [1]. Smoking has been shown to have harmful effects on numerous organs of the body and the list of diseases where smoking has been recognized as a contributory factor is extensive [2].

Cigarette smoking has been definitively recognized for decades as a primary and significant risk factor in the development of cardiovascular disease (CVD) and atherosclerosis [3,4]. Beyond just structural damage, current medical understanding increasingly emphasizes the critical role of inflammation in CVD. In fact, CVD is now not merely seen as a disease of blocked arteries, but also as one with a substantial inflammatory component, even being characterized by many researchers as fundamentally an inflammatory disease process [5,6]. This inflammatory pathway, triggered by smoking, is not limited to CVD alone.

Chronic smoking shows a strong well-established connection to multiple persistent inflammatory diseases. The list of significant conditions includes chronic obstructive pulmonary disease (COPD) which impacts lung function[7], rheumatoid arthritis which affects joints through autoimmune inflammation, and systemic lupus erythematosus which represents another systemic autoimmune condition[8] as well as inflammatory bowel diseases like Crohn's disease [9]. The exact molecular and cellular processes through which smoking causes and maintains complex inflammatory mechanisms leading to diseases remain unclear, but scientists are increasingly interested in examining inflammatory markers. Scientific studies are investigating specific markers as essential indicators to understand how smoking leads to greater illness rates and death. Our understanding of these inflammatory pathways will enable us to create more effective prevention methods and treatments for diseases caused by smoking.

Tobacco smoked through a hookah endangers both smokers and those who inhale secondhand smoke. The charcoal utilized for tobacco heating creates health hazards through the production of high carbon monoxide levels along with toxic metals and cancer-causing chemicals [10]. The smoke from a hookah retains high levels of toxic agents despite water filtration[11]. Several toxic compounds present in hookah tobacco and smoke have been shown to lead to lung, bladder, and oral cancers as well as clogged arteries and heart disease [10,11].

The tobacco juices from hookahs cause mouth irritation and raise the likelihood of oral cancer development[11,12]. A hookah can transmit infections among its users who share the device. Infants born to mothers who used water pipes throughout pregnancy are born weighing at least $3\frac{1}{2}$ ounces less than infants born to women who did not smoke. Infants born to mothers who smoked hookah show a heightened vulnerability to respiratory illnesses [12].

Hookah smokers often believe their habit is safer than cigarette usage but face similar health dangers as cigarette smokers [10 13]. The nicotine delivery from water pipe smoking operates in the same manner as nicotine affects users who consume tobacco products through other methods [13]. Hookah smokers breathe in higher volumes of damaging substances found in cigarette smoke than traditional cigarette smokers [10,13]. Participants smoke 200 puffs from a hookah during one hour while cigarette smokers average only 20 puffs in

the same time period[10 14 15].

Smoking rates continue to decline [16] while electronic cigarette usage expands [17]. Numerous people who want to stop smoking choose to use electronic cigarettes or vaping for this purpose [18–20]. Although full replacement of ECs for cigarettes leads to smoking cessation[21 22], most EC users engage in dual use. [23 24]. Further,

Research shows that dual use does not lower exposure to toxic substances emitted by combustible cigarettes such as carcinogens [25,26]. The American Cancer Society strongly advises against the combined use of EC devices and cigarettes [27]. Recent research indicates frequent vaping contributes to cardiovascular disease risk [28,29] and newer EC devices deliver nicotine at levels similar to or greater than conventional cigarettes [30] which could heighten the negative impacts from nicotine exposure.

The debate about which smoking method poses greater health risks and produces more harmful effects for smokers remains ongoing. This study sought to analyze Hs-CRP levels and D-dimer values to assess future cardiovascular disease risks and clot formation poten-tial across three distinct smoking groups: traditional cigarette smokers, shisha users, and electronic cigarette smokers [31].

Hs-CRP serves as an indicator of low-grade vascular inflammation while playing a crucial role in forming and tearing atheromatous plaque. High CRP levels can forecast cardiovascular incidents including coronary problems, stroke, peripheral vascular disease, and type 2 diabetes mellitus. This test works together with other assessments to evaluate cardiovascular risk [31].

Protein fragments include D-dimer which serves as the primary product of fibrin breakdown by plasmin during the last stage of clot formation and it's also referred to as fragment D-dimer or fibrin degradation fragment. The body's natural processes of clot formation and breakdown are the only circumstances when D-dimer levels become detectable beyond their normal undetectable state [32].

Pathological conditions trigger significant increases in D-dimer levels within the bloodstream. The biomarker serves as a detection tool in medical evaluations for hyperfibrinolysis by itself or alongside hypercoagulation which might present with vascular thrombosis or not. Diagnostic algorithms for thrombotic disorders now include D-dimer testing to identify venous thromboembolism (VTE), deep vein thrombosis (DVT), pulmonary embolism (PE), and disseminated intravascular coagulation (DIC) as per references [33,34].

METHODS

Study subjects

This cross-sectional study included 500 male participants stratified into four cohorts: (1) 100 cigarette smokers with an average age of 28 ± 4.0 years between ages 21 and 32, (2) 100 hookah smokers averaging 26 ± 3.7 years

ranging from 20 to 32 years old, (3) 100 electronic cigarette users who were 25 ± 2.7 years old with ages between 22 and 30, and (4) 200 healthy non-smokers who served as controls with a mean age of 23 ± 2.9 years within a 20 to 34-year range. The study recruited participants from private medical labs as well as hospitals and primary healthcare centers together with community networks. Before enrollment all subjects gave verbal informed consent. The Scientific Committee of the Department of Molecular and Medical Biotechnology at Al-Nahrain University College of Biotechnology in Baghdad, Iraq approved the study protocol.

Exclusion Criteria:

Participants were excluded based on the following criteria to mitigate confounding effects: The study excluded participants with inflammatory or autoimmune diseases together with those who have diabetes mellitus, pre-existing cardiovascular diseases, recent surgical procedures within the last three months and individuals who use two or more tobacco products concurrently. Selection criteria excluded diseases that could change levels of inflammatory or thrombotic biomarkers such as Hs-CRP and D-dimer. The study excluded dual/triple smokers to ensure clear results by analyzing single smoking types and maintaining uniform exposure levels across participant groups.

Collection of blood samples:

The researchers collected 10 mL venous blood samples from every participant through sterile venipuncture using disposable syringes. The blood was aliquoted into two pre- labeled vacutainer tubes: The blood sample for high-sensitivity C-reactive protein (Hs- CRP) measurements was deposited into a serum-separating gel tube whereas the D-dimer testing used 5 mL of blood in a 3.2% sodium citrate anticoagulant tube. To isolate serum and plasma, a centrifugation process with a force between 1500–3000 ×g for 10–15 minutes was necessary followed by immediate processing within two hours of collection to minimize pre- analytical variability. The measurement of serum Hs-CRP levels was performed using the chemiluminescence immunoassay technique on a Mindray CL-900i device while plasma D-dimer concentrations were evaluated by automated immunoassay using the MINI VIDAS® system according to manufacturer guidelines.

Measurement of Hs-CRP and D-dimer

The Mindray CL-900i chemiluminescence immunoassay system from Mindray Medical International Limited (Shenzhen, China) measured plasma D-dimer levels through the use of reagent kits supplied by the manufacturer. The MINI VIDAS® automated immunoassay platform from bioMérieux Diagnostics in Marcy-l'Étoile, France conducted the analysis of serum high-sensitivity C-reactive protein (Hs-CRP) concentrations using standardized protocols and calibrated assay kits. Each assay included duplicate tests to guarantee precision and achieved quality control by keeping intra- and inter-assay coefficients of variation under 5%.

Statistical Analyses:

The study executed statistical analyses with the assistance of SPSS version 25 developed by IBM Corp. in Armonk, NY, USA. The study report displays continuous variables using the format mean ± standard deviation (SD). The study established a statistical significance threshold using a two-tailed p-value of ≤ 0.05 while selecting inferential tests according to data distribution characteristics and variance uniformity.

RESULTS

Table 1 summarizes the serum high-sensitivity C-reactive protein (Hs-CRP) levels across study groups, whereas Table 2 details plasma D-dimer concentrations. Both tables present comparative data as mean ± standard deviation (SD), with intergroup statistical significance (p
< 0.001) calculated via ANOVA followed by post-hoc Tukey tests.

Table 1. Serum levels of Hs-CRP in the study subjects.

Groups	Number of samples	Hs-CRP(mg/L) (Mean ± SD)	p-value (A vs. B)	p-value (A vs. C)	p-value (B vs. C)	p-value(A, B, C vs. D)
Cigarettes smokers (A)	100	11.85 ± 5.48				
Shisha smokers (B)	100	4.78 ± 1.18	0.000*	0.000*	0.000*	0.000*
Vape smokers (C)	100	2.95 ± 1.70				
Controls (D)	200	1.69 ± 1.2				

*Significant at the levels of (p ≤ 0.05)

According to Table 1 cigarette smokers exhibit the highest serum high-sensitivity C-reactive protein (Hs-CRP) levels at 11.85 ± 5.48 mg/L (p < 0.001) compared to shisha smokers (4.78 ± 1.18 mg/L), vape users (2.95 ± 1.70 mg/L), and non-smoking controls (1.69 ± 1.20 mg/L). The inflammatory marker Hs-CRP was significantly elevated in shisha smokers compared to vape users and non-smoking controls with statistical significance (p < 0.001) which demonstrates progressive inflammation across different smoking methods.

Vape users showed lower Hs-CRP levels compared to traditional tobacco users but their levels at 2.95 mg/L nonetheless surpassed those in non-smokers (p < 0.001). Vaping represents a middle-ground health risk since it produces inflammation lower than cigarettes or shisha though it remains harmful to users. Research shows that patients with Hs-CRP levels exceeding 3 mg/L experience increased cardiovascular risk [35,36]. While vapers maintain levels under the risk threshold their closeness indicates possible long-term damage especially through continuous use.

The results in table (2) show that there was a significant ($p < 0.000$) increase in the levels of D-dimer in the cigarettes smokers compared to shisha, vape and control groups. A significant ($p < 0.000$) increase of D-dimer levels was also found in shisha smokers compared to both vape smokers and the control group. In addition, a significant ($p < 0.000$) increase in D-dimer levels was also found in vape smokers compared to the control group.

Table 2. Serum levels of D-dimer in the study subjects.

Groups	Number of samples	D-dimer(ng/mL) (Mean ± SD)	p-value (A vs. B)	p-value (A vs. C)	p-value (B vs. C)	p-value(A, B, C vs. D)
Cigarettes smokers (A)	100	696.46 ± 37.58				
Shisha smokers (B)	100	310.50 ± 43.94	0.000*	0.000*	0.000*	0.000*
Vape smokers (C)	100	157.91 ± 98.21				
Controls (D)	200	52.58 ± 33.4				

*Significant at the levels of ($p \leq 0.05$)

DISCUSSION

Recent decades have seen important but conflicting research attention toward the connection between smoking and systemic inflammation using serum C-reactive protein (CRP) measurements. Early research with basic assays showed extreme differences in CRP concentrations between smokers and non-smokers but current studies demonstrate subtler patterns. A study from Bangladesh demonstrated increased high-sensitivity CRP levels among male smokers [37], but other research showed no connection[38].

underscoring methodological and demographic variability. In our cohort, cigarette smokers exhibited significantly higher Hs-CRP levels than shisha or vape users ($p < 0.001$), aligning with an Egyptian study that similarly ranked cigarettes > shisha > controls. How- ever, our analysis did not account for smoking duration or intensity, a limitation critical to contextualizing inflammatory burden[39].

The rise of electronic cigarettes (ECs) among youth—driven by perceptions of safety—has outpaced evidence on their health impacts. Despite avoiding tobacco combustion, EC aerosols contain thermal degradation byproducts (e.g., formaldehyde) linked to oxidative stress and endothelial dysfunction[40 41,42 43 44 45 46 47 48 35,36 49 50 51]. Our findings add urgency to this debate: while vapers showed lower Hs-CRP than traditional smokers, their levels still exceeded non-smokers, corroborating prior work. This suggests that vaping, though less inflammatory than cigarettes or shisha, is not benign[52–55].

Clinically, Hs-CRP levels >3 mg/L signal high cardiovascular risk. In our study, cigarette and shisha smokers surpassed this threshold (11.85 mg/L and 4.78 mg/L, respectively), whereas vapers approached it (2.95 mg/L). This gradient—cigarettes > shisha > vape—implies a proportional relationship between smoking modality and cardiovascular risk, though longitudinal data are needed to confirm causality [35,36].

D-dimer, a thrombotic marker, further highlighted cigarettes' unique harm. Levels in cigarette smokers (696.46 ng/mL) dwarfed those in shisha (310.50 ng/mL) and vape users (157.91 ng/mL), aligning with Sudanese data and case reports linking vaping to throm- botic lung injury [55] [56]. These findings position cigarettes as the dominant driver of hyper-coagulability, though shisha and vaping still perturb clotting mechanisms relative to non-smokers [57].

CONCLUSIONS

It is concluded that cigarette smokers are at higher risk for future cardiovascular and thrombotic events compared to shisha and vape smokers. Electronic cigarettes smoking (vaping) is safer compared to cigarettes and shisha smoking. However, even vaping might have an impact or adverse health effect compared to non-smokers.

DECLARATIONS

1. Authors' contributions

All authors have equally contributed to the research.

2. Funding Statement

This research is self-funded

3. Conflict of Interest

The authors declare no conflict of interest

4. Ethical approval

Institutional ethical approval.

REFERENCES

1. Organization WH. WHO report on the global tobacco epidemic, 2008: the MPOWER package; 2008. p. 1–329.
2. of the Surgeon General (US) O, on Smoking O, (US) H. The Health Consequences of Smoking: A Report of the Surgeon General. Atlanta (GA): Centers for Disease Control and Prevention (US); 2004. Reports of the Surgeon General.; 2004. .
3. Ockene IS, Miller NH. Cigarette smoking, cardiovascular disease, and stroke: a statement for healthcare professionals from the American Heart Association. American Heart Association Task Force on Risk Reduction. Circulation. 1997;96(9):3243–3250. 9386200. 10.1161/01.cir.96.9.3243.

4. Smith SC, Milani RV, Arnett DK, Crouse JR, McDermott MM, Rosenson RS, et al. Atherosclerotic Vascular Disease Conference: Writing Group II: risk factors. Circulation . 2004;109(21):2613–2619. 10.1161/01.CIR.0000128519.60762.84.
5. Ajoolabady A, Pratico D, Lin L, Mantzoros CS, Bahijri S, Tuomilehto J, et al. Inflammation in atherosclerosis: pathophysiology and mechanisms. Cell Death Dis. 2024;15(817):817–817. 10.1038/s41419-024-07166-8.
6. Kong P, Cui ZY, Huang XF, Zhang DD, Guo RJ, Han M. Inflammation and atherosclerosis: signaling pathways and therapeutic intervention. Signal Transduct Target Ther. 2022;7(1):131–131. 10.1038/s41392-022-00955-7.
7. Mannino DM, Buist AS. Global burden of COPD: risk factors, prevalence, and future trends. Lancet. 2007;370(9589):765–773. 17765526. 10.1016/S0140-6736(07)61380-4.
8. Majka DS, Holers VM. Cigarette smoking and the risk of systemic lupus erythematosus and rheumatoid arthritis. Ann Rheum Dis. 2006;65(5):561–564. 10.1136/ard.2005.046052.
9. Lakatos PL, Szamosi T, Lakatos L. Smoking in inflammatory bowel diseases: good, bad or ugly? World J Gastroenterol. 2007;13(46):6134–6143. 18069751. 10.3748/wjg.v13.i46.6134.
10. Cobb C, Ward KD, Maziak W, Shihadeh AL, Eissenberg T. Waterpipe Tobacco Smoking: An Emerging Health Crisis in the United States. Am J Health Behav. 2010;34(3):275–285.
11. Ibrahim EEH, MBBS MAEU. Squamous Cell Carcinoma and Keratoacanthomaof the Lower Lips Associated with “Goza” and “Shisha” Smoking. International Journal of Dermatology. 1999;38(2):108–110. 10.1046/j.1365-4362.1999.00448.x.
12. Hookah Smoking: A Growing Threat to Public Health Issue Brief. Smoke free Communities Project; 2011.
13. Benjamin RM. Preventing Tobacco Use Among Youth and Young Adults: A Report of the Surgeon General. Office on Smoking and Health; 2012.
14. Nuwayhid IA, Yamout B, Azar G, Kambris MA. Narghile (hubble-bubble) smoking, low birth weight, and other pregnancy outcomes. American Journal of Epidemiology. 1998;148(4):375–83. 10.1093/oxfordjournals.aje.a009656.
15. Akl EA, Gaddam S, Gunukula SK, Honeiner R, Jaoude PA, Irani J. The Effects of Water-pipe Tobacco Smoking on Health Outcomes: A Systematic Review. International Journal of Epidemiology. 2010;39(3):834–857. 10.1093/ije/dyq002.
16. Cobb CO, Vansickel AR, Blank MD, Jentink K, Travers MJ, Eissenberg T. Indoor air quality in Virginia waterpipe cafes. Tobacco Control. 2012;22(5):338–343. 10.1136/tobaccocontrol-2011-050350.
17. Jamal A, King BA, Neff LJ, Whitmill J, Babb SD, Graffunder CM. Current Cigarette Smoking Among Adults - United States, 2005-2015. MMWR Morb Mortal Wkly Rep . 2016;65(44):1205–1211. 10.15585/mmwr.mm6544a2.
18. King BA, Patel R, Nguyen KH, Dube SR. Trends in awareness and use of elec- tronic cigarettes among US adults, 2010-2013. Nicotine Tob Res. 2015;17(2):219–

227. 10.1093/ntr/ntu191.

19. Mcmillen RC, Gottlieb MA, Shaefer RM, Winickoff JP, Klein JD. Trends in electronic cigarette use among U.S. adults: use is increasing in both smokers and nonsmokers. Nicotine Tob Res. 2015;17(10):1195–1202. 10.1093/ntr/ntu213.
20. Rutten LJ, Blake KD, Agunwamba AA, Grana RA, Wilson PM, Ebbert JO, et al. Use of e-cigarettes among current smokers: associations among reasons for use, quit intentions, and current tobacco use. Nicotine TobRes. 2015;17(10):1228–1234. 10.1093/ntr/ntv003.
21. Zhu SH, Zhuang YL, Wong S, Cummins SE, Tedeschi GJ. E-cigarette use and associated changes in population smoking cessation: evidence from US current population surveys. BMJ. 2017;358. 10.1136/bmj.j3262.
22. Biener L, Hargraves JL. A longitudinal study of electronic cigarette use among a population-based sample of adult smokers: association with smoking cessation and motivation to quit. Nicotine Tob Res. 2015;17(2):127–133. 10.1093/ntr/ntu200.
23. Weaver SR, Huang J, Pechacek TF, Heath JW, Ashley DL, Eriksen MP. Are electronic nicotine delivery systems helping cigarette smokers quit? Evidence from a prospective cohort study of U.S. adult smokers, 2015–2016. PLoS One. 2018;13(7):e0198047–e0198047. 10.1371/journal.pone.0198047.
24. Mirbolouk M, Charkhchi P, Kianoush S, Uddin SMI, Orimoloye OA, Jaber R, et al. Prevalence and Distribution of E-Cigarette Use Among U.S. Adults: Behavioral Risk Factor Surveillance System, 2016. Ann Intern Med . 2016;169(7):429–438. 10.7326/M17-3440.
25. Pasquereau A, Guignard R, Andler R, Nguyen-Thanh V. Electronic cigarettes, quit attempts and smoking cessation: a 6-month follow-up. Addiction. 2017;112(9):1620–1628. 10.1111/add.13869.
26. Fagan P, Pokhrel P, Herzog TA, Moolchan ET, Cassel KD, Franke AA. Sugar and Aldehyde Content in Flavored Electronic Cigarette Liquids. Nicotine Tob Res. 2018;20(8):985–992. 10.1093/ntr/ntx234.
27. American Cancer Society Position Statement on Electronic Cigarettes. American Cancer Society; 2018.
28. Zong H, Hu Z, Li W, Wang M, Zhou Q, Li X, et al. Electronic cigarettes and cardiovascular disease: epidemiological and biological links. Pflugers Arch . 2024;476(6):875–888. 10.1007/s00424-024-02925-0.
29. Espinoza-Derout J, Shao XM, Lao CJ, Hasan KM, Rivera JC, Jordan MC, et al. Electronic Cigarette Use and the Risk of Cardiovascular Diseases. Front Cardiovasc Med. 2022;9. 10.3389/fcvm.2022.879726.
30. Althakfi SH, Hameed AM. Nicotine in electronic cigarettes. Journal of Umm Al- Qura University for Applied Sciences . 2024;10:579–592. 10.1007/s43994-024- 00123-3.
31. Sproston NR, Ashworth JJ. Role of C-Reactive Protein at Sites of Inflammation and Infection. Front Immunol. 2018;9:754–754. 10.3389/fimmu.2018.00754.

32. Yamagata K, Fukuzawa S, Kanno NI, Uchida F, Yanagawa T, Bukawa H. The Association between D-Dimer and Prognosis in the Patients with Oral Cancer. Dent J (Basel) . 2020;8(3):84–84. 10.3390/dj8030084.
33. Beidollahkhani S, Fayedeh F, Shoja A, Nejad EH, Hoseinpour M, Fazlpour F. d- dimer as a biomarker for COVID-19-associated pulmonary thromboembolism: a narrative review from molecular pathways to the imaging findings. Egypt J Bronchol. 2023;17(44):44–44. 10.1186/s43168-023-00221-6.
34. Dahdah A, Jaggers RM, Sreejit G, Johnson J, Kanuri B, Murphy AJ, et al. Immunological Insights into Cigarette Smoking-Induced Cardiovascular Disease Risk. Cells. 2022;11(20):3190–3190. 10.3390/cells11203190.
35. Law KF, Goldklang MP, D'Armiento JM. Vigorous C-Reactive Protein and Alpha1-Antitrypsin Elevations Associated with Vape Pneumonitis. American Journal of Respiratory and Critical Care Medicine. 2020;201:1229–1229. 10.1164/ajrccm-conference.2020.201.1$_{M}$ *eetingAbstracts.A*1229.
36. Moon J, Lee H, Kong M, Kim H, YO. Association Between Electronic Cigarette Use and Levels of High-Sensitivity C-Reactive Protein and Uric Acid. Asia Pac J Public Health . 2020;32(1):35–41. 10.1177/1010539519899777.
37. Das I. Raised C-reactive protein levels in serum from smokers. Clin Chim Acta . 1985;153(1):9–13. 10.1016/0009-8981(85)90133-0.
38. Jahan S, Qaz Q. Serum High Sensitive C - reactive protein in Male Smokers of Bangladesh. Journal of Bangladesh Society of Physiologist. 2015;10(1):36–36. 10.3329/jbsp.v10i1.24617.
39. Aldaham S, Foote JA, Chow HS, Hakim IA. Smoking Status Effect on Inflammatory Markers in a Randomized Trial of Current and Former Heavy Smokers. Int J Inflam . 2015;2015:439396–439396. 10.1155/2015/439396.
40. Diab OA, Abdelrahim EM, Esmail M. Effect of water pipe tobacco smoking on plasma high sensitivity C reactive protein level and endothelial function com- pared to cigarette smoking. The Egyptian Heart Journal. 2015;67(3):233–241. 10.1016/j.ehj.2014.07.004.
41. Ridker PM. Inflammation in atherothrombosis: how to use high-sensitivity C- reactive protein (hsCRP) in clinical practice. Am Heart Hosp J. 2004;2(4):4–9. 15539969.
42. Karakas M, Koenig W. CRP in cardiovascular disease. Herz. 2009;34(8):607–613. 10.1007/s00059-009-3305-7.
43. Traboulsi H, Cherian M, Rjeili A, Preteroti M, Bourbeau J, Smith BM. Inhalation Toxicology of Vaping Products and Implications for Pulmonary Health. Int J Mol Sci. 2020;21(10):3495–3495. 10.3390/ijms21103495.
44. Polosa R, O'Leary R, Tashkin D, Emma R, Caruso M. The effect of e-cigarette aerosol emissions on respiratory health: a narrative review. Expert Rev Respir Med . 2019;13(9):899–915. 10.1080/17476348.2019.1649146.
45. Marques P, Piqueras L, Sanz MJ. An updated overview of e-cigarette impact on human health. Respir Res. 2021;22(151). 10.1186/s12931-021-01737-5.

46. Travis N, Knoll M, Cadham CJ, Cook S, Warner KE, Fleischer NL, et al. Health Effects of Electronic Cigarettes: An Umbrella Review and Methodological Con- siderations. International Journal of Environmental Research and Public Health. 2022;19(15):9054–9054. 10.3390/ijerph19159054.
47. Effah F, Adragna J, Luglio D, Bailey A, Marczylo T, Gordon T. Toxicological assess- ment of E-cigarette flavored E-liquids aerosols using Calu-3 cells: A 3D lung model approach. Toxicology. 2023;500:153683–153683. 10.1016/j.tox.2023.153683.
48. Benam KH. Multidisciplinary approaches in electronic nicotine delivery systems pulmonary toxicology: emergence of living and non-living bioinspired engineered systems. Commun Eng. 2024;3(123):123–123. 10.1038/s44172-024-00276-.
49. Elfadil GA, Elfaki EM, Madani SO, Abdalhabib EK, Elmugadam A. High-Sensitive C-reactive Protein and Atherogenic Lipid Levels in a Group of University Students with Habitual Smoking. Biomed Pharmacol J. 2020;13(3). 10.13005/bpj/2022.
50. Eshraghian EA, Wk AD. A review of constituents identified in e-cigarette liquids and aerosols. Tobacco Prevention & Cessation. 2021;7(10):10–10. 10.18332/tpc/131111.
51. Diab OA, Abdelrahim EM, Esmail M. Effect of water pipe tobacco smoking on plasma high sensitivity C reactive protein level and endothelial function com- pared to cigarette smoking. The Egyptian Heart Journal. 2015;67(3):233–241. 10.1016/j.ehj.2014.07.004.
52. Ridker PM. Inflammation in atherothrombosis: how to use high-sensitivity C- reactive protein (hsCRP) in clinical practice. Am Heart Hosp J. 2004;2(4 Suppl 1):4–9. 15539969.
53. Karakas M, Koenig W. CRP in cardiovascular disease. Herz. 2009;34(8):607–613. 10.1007/s00059-009-3305-7.
54. Martin EM, Clapp PW, Rebuli ME, Pawlak EA, Glista-Baker E, Benowitz NL, et al. E-cigarette use results in suppression of immune and inflammatory-response genes in nasal epithelial cells similar to cigarette smoke. . Am J Physiol Lung Cell Mol Physiol. 2016;311(1):135–144. 10.1152/ajplung.00170.2016.
55. Sy C, Ding J, Elmasry O. A young man with e-cigarette or vaping-associated lung injury. Consultant. 2022;62(1):29–32. 10.25270/con.2021.04.00015.
56. Amin AA, Erica H, Youmna M. Do Not Huff, Puff, or Vape That Stuff: Interstitial Airspace Disease in a Teenager. Case Reports in Pediatrics. 2020;10.1155/2020/8822362.
57. Amir S, Khalil H. Assessment of quantitative D-dimer level among Sudanese smok- ers. International Journal of Information Research and Review. 2015;02(12):1464–1466.