Effects of E-cigarettes and Traditional Cigarettes on Lungs and Bronchi

Shujin Tian(✉)

Shattuck-St.Mary's School, Faribault, MN 55021, USA
tina.tian@s-sm.org

Abstract. E-cigarettes, considered healthier substitutes for traditional cigarettes, are being used more and more frequently, especially among adolescents. However, there are not many studies that support this claim. Moreover, recent studies have suggested that e-cigarettes are actually as harmful as traditional cigarettes. Considering the increasing trend of people using e-cigarettes, it is necessary to do a study to determine whether or not e-cigarettes are healthier than traditional cigarettes. This paper, through a method of comparing data from recent studies, explores the negative effects of both e-cigarettes and traditional cigarettes on the lungs and bronchi. The paper finds that e-cigarettes are actually harmful to the lungs and bronchial tubes. Besides, its damage level, which is the damage severity e-cigarettes causes to the lungs and bronchial tubes, is approximately the same compared to traditional cigarettes.

Keywords: E-cigarettes · traditional cigarettes · lungs and bronchi · adolescents

1 Introduction

Electrical cigarettes, or e-cigarettes, will deliver an aerosol containing nicotine to users through the heating up of solutions of nicotine, glycerol, flavorings, and other chemicals. It comes up as a healthier substitute for quitting smoking and traditional cigarettes [1]. Nowadays, e-cigarettes are being used more and more frequently, especially among adolescents. According to a report released by the U.S. Food and Drug Administration and the U.S. Centers for Disease Control and Prevention, more than 2 million U.S. middle school and high school students reported using e-cigarettes in 2021 [2]. This number is a massive concern for society. Even though the e-cigarette companies claim that e-cigarettes are harmless to the human body, there is no exact conclusion on whether e-cigarettes are healthier compared to traditional cigarettes.

Whether it is traditional cigarettes or e-cigarettes, their greatest impact on the human body is concentrated in the lungs and bronchi. This has to do with all those chemicals contained in cigarette smoke. The smoke that contains so many chemicals goes through humans' lungs and bronchial tubes and damages the tissue. Scientists have done many studies to try to find out the damage both of them cause, and the controversy continues. In 2018, Reinikovaite V, Rodriguez IE, and Karoor V did a study with a rat model in vivo [3]. They tested whether long-term exposure to e-cigarette vapor would damage the lung

© The Author(s) 2022
G. Ali et al. (Eds.): ISEMSS 2022, ASSEHR 687, pp. 566–573, 2022.
https://doi.org/10.2991/978-2-494069-31-2_70

structure as tobacco smoke does. What they found out is that e-cigarettes and traditional cigarettes have about the same toxicity to the lungs. The long-term exposure to both will lead to significant lung damage. However, Thomas Münzel and his group found in 2020 that e-cigarette vapor is less toxic compared to tobacco smoke [4]. In addition, Higham Andrew found in 2018 that compared to traditional cigarettes, e-cigarettes have less damage to the airway epithelium, which is part of the first line against inhaled particles [5].

With all the varied conclusions, the difference between e-cigarettes and traditional cigarettes remains unclear. This study will analyze data from different studies which explore the damage that e-cigarettes and traditional cigarettes have on the lungs and bronchi. By analyzing the data from those various studies, there will be a more convenient conclusion about whether or not e-cigarettes are a healthier alternative to traditional cigarettes. Most people view e-cigarettes as harmless products (means e-cigarettes will not cause much damage to human body), and this has led to a trend of an increase in the number of e-cigarette users [2]. Considering this trend, especially among adolescents around the world, this study is necessary to do.

2 Traditional Cigarettes

Tobacco and additional chemical additions make up the majority of traditional cigarettes' ingredients. The main cause of smokes' negative health effects is tobacco. Emphysema, a deadly lung illness with severe structural and functional damage to human lungs, is one of the harms brought on by cigarettes. [6] Indranil Gupta and his team conducted a study in 2016 to demonstrate how oxidants in cigarette smoke cause oxidative damage to lung proteins and activate Rtp801, a crucial proinflammatory cellular component that contributes to lung injury. They previously discovered that conventional cigarette smoke directly causes animal lung proteins to deteriorate, making them more vulnerable to proteolytic degradation. This is in line with observations of emphysema brought on by cigarette smoking. However, they were unable to pinpoint the precise cellular components that contribute to this destructive process. They conducted a follow-up study using guinea pigs and discovered that the main cellular pathway for cigarette smoke-induced emphysema, including Rtp801-mediated activation of NF-B and iNOS in the lung and subsequent oxidative -nitrosation damage and lung protein apoptosis, is actually set off by the oxidant in cigarette smoke. Each of the adult male guinea pigs weighed between 300 and 400 g. The guinea pigs were kept in carefully regulated environments with a consistent 12-h daylight cycle and a sufficient amount of food and water. Six guinea pigs from each group were randomly assigned to either smoke exposure or non-smoke exposure. Hematoxylin and eosin was used to stain guinea pig lung slices after the experiment. Then, Lm and DI were used to calculate the amount of alveolar injury.

As can be seen in the figure above, after 28 days of CS exposure, guinea pigs developed alveolar lung damage over time (Fig. 1A, V-VIII), as demonstrated by the mean linear Intercept (Lm) and destruction index (DI) of alveolar air expansion. This lung damage was also accompanied by extensive destruction of key lung structural proteins, elastin, and MMP-9 overexpression. Protein deterioration in emphysema is mediated

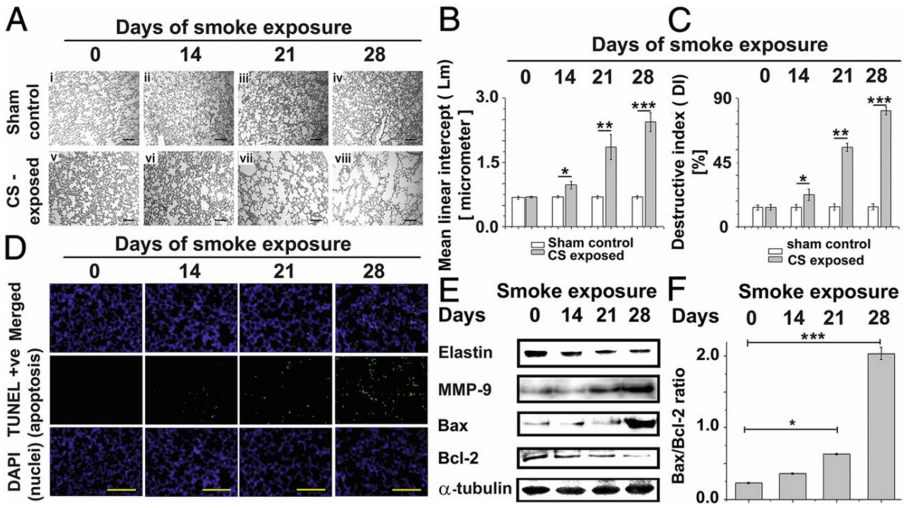

Fig. 1. Comparison of lung sections, Alveolar morphologic changes are represented by Lm, Alveolar morphologic changes were indicated by Di, Representative lung sections of alveolar cell apoptosis in the above treatment group, Immunoblots, Bax or Bcl-2 levels

by primary pulmonary metalloproteinases (30). (Fig. 1E and S1 A and B). This shows that damage to the alveolar lungs is caused by regular cigarettes, which also degrade important lung structural proteins and elastin (Fig. 2).

In addition, a study conducted in 2016 by Cao Chao and his team examined the link between lung cancer and tobacco use. [7] One of the main causes of DNA damage is the oxidative stress brought on by tobacco usage. Numerous malignancies, particularly lung cancers, have been linked to this damage. They compared the levels of oxidative DNA damage responses in lung cancer patients who smoked and those who did not smoke in this study. A chemical known as 8-hydroxydeoxyguanosine was also investigated (8-OHDG). A typical type of DNA damage brought on by oxidative stress is 8-OHDG. Due to this characteristic, 8-OHDG is regarded as a trustworthy indicator of oxidative DNA damage.

In lung cancer patients who smoked and those who did not, they compared the levels of 8-OHDG expression. They initially exposed HBE cells to cigarette smoke extract in order to show that smoking causes oxidative DNA damage in bronchial epithelial cells, and then they looked for the expression of 8-OHDG in the HBE cells. When compared to the control group, they discovered a statistically significant rise in the expression of 8-OHDG cells (62.4 ± 5.3 percent versus 9.5 ± 2.0 percent, P = 0.0007). Flow cytometry added its support to this conclusion.

Mice that had been exposed to cigarette smoke for 12 or 24 weeks in vivo were immunohistochemically stained to look for oxidative lung DNA damage. They found a substantial difference in the percentage of 8-OHDG-positive cells between the group exposed to cigarette smoke and the control group (54.9 ± 5.7 compared 28.3 ± 4.0, P = 0.0026 for 12 weeks; 81.5 ± 4.2 versus 30.5 ± 2.2, P 0.0001 for 24 weeks).

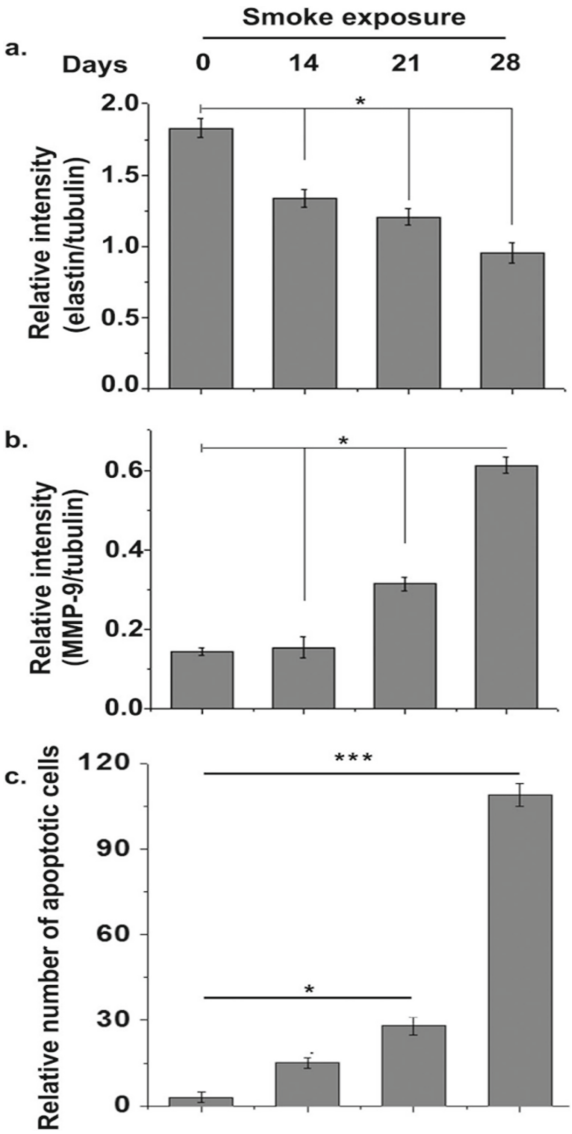

Fig. 2. Histogram describing the relative levels (strengths) of protein elastin (A) and MMP-9 (B), (C) represents the relative number of apoptotic lung cells

With so many studies, it is evident that regular cigarettes are extremely harmful to the body, particularly the lungs and bronchi.

3 E-cigarette

Although the E-cigarette has nothing to do with the combustion of tobacco, it nevertheless causes harm. E-cigarettes create aerosol by heating a liquid that typically contains nicotine flavorings as well as additional, hazardous substances. This aerosol will be inhaled into the lungs of e-cigarette users. It has already been established through research that e-cigarettes are bad for the lungs and bronchi. High levels of free radicals, including reactive oxygen species (ROS) and reactive nitrogen species, are present in e-cigarettes (approximately 1016 molecules each puff) (RNS). Enzymatic antioxidants like glutathione peroxidase and superoxide dismutase will often neutralize these free radicals.

It is impossible to completely neutralize these free radicals when radicals inhale e-cigarettes in excess, though. In reaction to this unsuccessful neutralization, macrophage cells will release cytokines that will induce cellular damage and an inflammatory response. One of these cytokines, interleukin-8, contributes to cellular damage. This rise in interleukin-8 causes the lung tissue to release the metalloprotein-8 matrix. Type 2 collagen breakdown is accelerated by an excess of metalloprotein-8 matrix. The structure and operation of the collagen in the respiratory cartilages are impacted by this deterioration. The likelihood of developing pulmonary fibrosis would rise as a result of this damage to the lung tissue over an extended period of time, making the tissue damage permanent. Different ailments, including lung cancer, cardiovascular issues, and pulmonary hypertension, can be brought on by it [8].

In 2021, a study was carried out by Rivan V. Suryadinata and Bambang Wirjatmadi. The goal of the study was to pinpoint the phases of lung tissue destruction brought on by an increase in free radicals and an inflammatory response. These factors, which indirectly induce an increase in alveolar macrophages, harm the lung tissue. They used a control group of male Wistar rats and exposed some of them to e-cigarettes. After the test, the rat lung tissues were taken out, and a hematoxylin-eosin staining procedure was prepared.

For each preparation, the average number of alveolar macrophages in 10 fields was calculated as part of the assessment. The T test revealed a difference in alveolar macrophage counts between the two groups ($P < 0.001$) between the two groups. The experimental group had considerably more positive cells than the control group (53.26 ± 0.93 vs. 21.86 ± 1.12). Overall, inhaling e-cigarette smoke raises malondialdehyde levels, which raises free radicals in the airways. As a result, lung tissue has been discovered to modify pro-inflammatory cytokines including IL-6 and IL-8, which causes adjustments in the metalloprotein-8 matrix and the beginning of type 2 collagen breakdown.

In 2018, Arunava Ghosh and his team conducted a study to determine the effects of persistent vaping on pulmonary epithelia. Both smokers and those who use e-cigarettes had bronchoscopies. For a proteomic analysis, they also collected bronchial brush biopsies and lavage samples from both groups. In bronchoscopies, it was discovered that e-cigarette smokers' airways appeared friable and erythematous. Additionally, the epithelial cell biopsy samples show that proteins were expressed differently in e-cigarette users. For instance, e-cigarette users had higher levels of CYP1B1 (cytochrome P450 family 1 subfamily B member 1), MUC5AC (mucin 5 AC), and MUC4. Additionally, they

discovered that PG/VG decreased membrane fluidity and impeded protein diffusion, while e-liquids quickly penetrated cells. They came to the conclusion that e-cigarettes have noticeable biological impacts on a person's lungs. The PG/VG base might partially mediate these effects. [9].

These studies demonstrate that using e-cigarettes will cause bronchial and lung tissue damage that is permanent.

4 Comparison

This part will mainly concentrated in the comparison between e-cigarettes and traditional cigarettes. There are two articles that going to be discussed in this part. One articles is a study with different type of smokers, and the other one is a study that get done with mice. Based on the result from both studies, it can be conclude that both e-cigarettes and traditional cigarettes have approximately the same level negative effect on lungs and bronchial.

4.1 Research on Smokers

Researchers have compared e-cigarettes and conventional cigarettes in their investigations. According to Arunava Ghosh and his team, released proteases are biomarkers of damage because they trigger the onset of chronic lung illness when they are out of balance with antiproteases [10]. Based on this, they proposed that, similar to tobacco use, exposure to e-cigarettes would result in an increase in lung protease secretion. They performed a bronchoscopy investigation on smokers, e-cigarette users, and non-smokers. In each of the three groups, BAL protease levels were tested. Additionally, they looked into how various e-liquid ingredients affected important immune cells' in vitro protease secretion.

They discovered that, in contrast to nonsmokers, the levels of the proteins NE, MMP-2, and MMP-9 were significantly higher in smokers and e-cigarette users than in non-smokers, but the levels of the protease were not significantly different. This demonstrated that while protease levels rose, net protease activity rose while antiprotease activity stayed the same. Additionally, they examined the MMP-2 and MMP-9 activity in BAL from e-cigarette users who were not smokers and discovered that it had dramatically increased. In conclusion, both smokers and e-cigarette users had higher amounts of NE protein and functional activity. E-cigarettes can also significantly raise levels of nicotine and other metabolites. This level is comparable to the sputum of a typical smoker (approximately 50 M). Overall, e-cigarette effects are comparable to those of conventional cigarettes.

4.2 Research on Mice

To determine the in vivo effects of e-cigarette smoke in the lung and to compare them to those of tobacco cigarette smoke, Constantinos Glynos and his team conducted a study with the mouse group. [11] There were three separate experimental groups that exposed

people to the smoke from cigarettes, e-cigarettes, and the air. They saved the bronchoalveolar lavage fluid after the experiment. PBS was used to resuspension the obtained cells. The left lung of mice was then removed, and it was stained with hematoxylin-eosin. The following histological findings have been utilized to create a grading system to determine the severity of lung inflammation: capillary congestion, intra-alveolar bleeding, interstitial neutrophil infiltration, intra-alveolar neutrophil infiltration, and localized thickening of alveolar membranes.

For each characteristic, a scale from 0 to 3 will be utilized (0: absence, 1: mild, 2: moderate, 3: most severe) [11] What they discovered is that, in comparison to the air-breathing group, both groups exposed to tobacco cigarettes and e-cigarettes have higher protein carbonyl levels. Additionally, there were more BALF cells overall. In conclusion, exposure to e-cigarettes will cause inflammatory reactions and have a negative impact on the mechanics of the respiratory system. The extra flavor in e-cigarettes also made the negative effects of e-cigarette vapor worse. Lung biology is negatively impacted by both traditional cigarette smoking and e-cigarette use.

5 Conclusion

It can be concluded that e-cigarettes have similar negative effects on the lungs and bronchial tubes. Even though e-cigarettes do not contain tobacco, the aerosol that is produced from e-cigarettes is also very harmful to the human body. Plus, the additional flavor in e-cigarettes can cause problems that tobacco will not. All that data shows that e-cigarettes are not a healthier substitute for traditional cigarettes. Moreover, adolescents who use e-cigarettes will have a higher chance of using tobacco cigarettes in the future. It suggests that the government should have tighter regulations on adolescent use of e-cigarettes and also make more announcements about the harmful effects of e-cigarettes.

There are some flaws in this study. Most of the data mentioned in this study is all from short-term studies. Considering e-cigarettes are still a new product to society, it is understandable. But more long-term studies need to be done on e-cigarettes to test the possible long-term effects they may have.

References

1. Rachel, Grana. “E-Cigarettes: A Scientific Review.” Circulation. 2014. https://www.ahajournals.org/doi/full/10.1161/CIRCULATIONAHA.114.007667
2. “Youth E-Cigarette Use Remains Serious Public Health Concern Amid COVID-19 Pandemic.” Centers for Disease Control and Prevention. 2021 https://www.cdc.gov/media/releases/2021/p0930-e-cigarette.html#:~:text=A%20study%20released%20today%20
3. Viktorija Reinikovaite. “The effects of electronic cigarette vapour on the lung: direct comparison to tobacco smoke.” European Respiratory journal, 2018 https://erj.ersjournals.com/content/51/4/1701661?ctkey=shareline
4. Thomas Münzel. “Effects of tobacco cigarettes, e-cigarettes, and waterpipe smoking on endothelial function and clinical outcomes.” European Heart Journal, Volume 41, Issue 41, 2020, Pages 4057–4070 https://academic.oup.com/eurheartj/article/41/41/4057/5861975?login=true

5. Higham, Andrew et al. “The effect of electronic cigarette and tobacco smoke exposure on COPD bronchial epithelial cell inflammatory responses.” International journal of chronic obstructive pulmonary disease. 2018. vol. 13 989-1000 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5870631/
6. Indranil Gupta et al. “Ascorbate attenuates pulmonary emphysema by inhibiting tobacco smoke and Rtp801-triggered lung protein modification and proteolysis.” PNAS. 2016 https://www.pnas.org/doi/full/10.1073/pnas.1600056113
7. Cao, Chao et al. “Smoking-promoted oxidative DNA damage response is highly correlated to lung carcinogenesis.” Oncotarget. 2016. 18919-26. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4951340/
8. Suryadinata, Rivan V, and Bambang Wirjatmadi. “The Molecular Pathways of Lung Damage by E-Cigarettes in Male Wistar Rats.” Sultan Qaboos University medical journal. 2021. 436-441 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8407909/
9. Ghosh A, Coakley RC, Mascenik T, Rowell TR, Davis ES, Rogers K, Webster MJ, Dang H, Herring LE, Sassano MF, Livraghi-Butrico A, Van Buren SK, Graves LM, Herman MA, Randell SH, Alexis NE, Tarran R. Chronic E-Cigarette Exposure Alters the Human Bronchial Epithelial Proteome. Am J Respir Crit Care Med. 2018. 98(1):67-76. https://pubmed.ncbi.nlm.nih.gov/29481290/
10. Arunava Ghosh et al. “Chronic E-Cigarette Use Increases Neutrophil Elastase and Matrix Metalloprotease Levels in the Lung.” ATS journal. 2019 https://www.atsjournals.org/doi/full/10.1164/rccm.201903-0615OC
11. Constantinos Glynos. “Comparison of the effects of e-cigarette vapor with cigarette smoke on lung function and inflammation in miceConstantinos Glynos.” American physiological society. 2018. https://journals.physiology.org/doi/full/10.1152/ajplung.00389.2017

Open Access This chapter is licensed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License (http://creativecommons.org/licenses/by-nc/4.0/), which permits any noncommercial use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license and indicate if changes were made.

The images or other third party material in this chapter are included in the chapter’s Creative Commons license, unless indicated otherwise in a credit line to the material. If material is not included in the chapter’s Creative Commons license and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder.