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COMPARATIVE ANALYSIS OF PHYSIOLOGICAL AND HISTOLOGICAL EFFECTS OF E-CIGARETTE VS. CONVENTIONAL SMOKING IN ALBINO MICE

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Abstract
This study compares the physiological and histological effects of e-cigarette (T1) and conventional cigarette (T2) exposure in albino mice over nine weeks. Both groups showed decreased hemoglobin levels, with T1 exhibiting higher white blood cell and platelet counts. Testosterone was elevated in T2, while glucose and estrogen levels increased in T1 but decreased in T2. Histological analysis revealed myocardial damage, glomerulosclerosis, liver cell ballooning, and lung emphysema in both groups, more severe in T2. Both exposures caused weight loss, especially in males. Findings suggest that while both vaping and smoking adversely affect health, e-cigarettes may cause greater immune and hormonal disruption alongside significant blood and tissue damage.

INTRODUCTION

Tobacco remains a significant public concern which produces major health issues (He et al., 2022). Electronic cigarettes, e-cigarettes or vapes are battery-powered electronic devices which are designed to deliver nicotine or other chemicals in the form of aerosol or vapour. It has gained much popularity since its introduction among youth and individuals who are attempting to quit traditional smoking (Grana et al., 2014; Jackson et al., 2025). Unlike traditional cigarettes e-cigarettes use a heating element to vaporize the e-liquid or vape juice. The e-liquid often contains nicotine, flavourings, glycol, propylene and vegetable glycerin (Goniewicz et al., 2012). However, concerns have arisen due to health issues, especially variability in product design, chemical composition and long-term effects (Jackson et al., 2025). Further, the increasing trends in youth and early exposure to nicotine raise significant public health alarms. It also obstructed for tobacco control initiative.

Research studying the long-term health impacts of their usage remains scarce due rising popularity (Hartmann-Boyce et al., 2021). A lot of studies are still arguing about how safe e-cigarettes are compared to regular cigarettes (Ceasar et al., 2024). The public health effects and regulatory guidelines on e-cigarettes depend on the assessment of their relative toxicity compared to traditional combustible products (Gordon et al., 2022). E-cigarette aerosols contain toxic substances such as formaldehyde, acetaldehyde, acrolein and nickel together with lead (Cheng, 2014). The toxic compounds found in e-cigarette aerosols lead to respiratory tract inflammation, and oxidative stress that causes chronic bronchitis, decreased lung function and asthma-like conditions (Gotts et al., 2019). The act of inhaling nicotine through e-cigarette

vapour causes heart rate elevation of blood pressure (Benowitz & Burbank, 2016). E-cigarette is also linked with the outbreak of EVALI (e-cigarette or vaping-associated lung injury) became a serious health issue in 2019 in the United States leading to lung inflammation and damage (Layden et al., 2020). Another emerging concern is the appeal of flavoured e-liquids to adolescents and young adults, which not only increases the risk of nicotine addiction but may also act as a gateway to conventional cigarette smoking. The long-term effects of e-cigarettes are largely unknown and new research on vaporized chemical exposure impacts requires more constant surveillance.

Nicotine, the primary addictive component in both conventional and electronic cigarettes, plays a crucial role in dependence and systemic toxicity(Latnikova & Asiru, 2021; Dennison Himmelfarb et al., 2025). The chemical substance operates specifically on nicotinic acetylcholine receptors found in both the central and peripheral nervous systems, resulting in addiction behaviour reinforcement while simultaneously influencing metabolic processes and cardiovascular function (Hurst et al., 2013)(Picciotto et al., 2008). Studies reveal that prolonged nicotine exposure leads to synaptic transmission and neuroplasticity changes as well as mood problems, which create anxiety about ongoing neurological damage (Brooks & Henderson, 2021; Hajdusianek et al., 2021; Airagnes et al., 2024). Further, the use of nicotine also leads to metabolic disorders such as insulin resistance and glucose dysregulation (Milovanovic et al., 2018; Cai & Bidulescu, 2023).

There is an urgent need for the evaluation of toxic compounds between standard and electronic cigarettes through controlled laboratory tests. Therefore, this study aimed to compare the toxicological effects of both traditional and e-cigarettes in albino mice model.

Materials and methods

Experimental model and Acclimatization

Experimental model, Albino mice weighting 25±5 grams were purchased from the University of Veterinary and Animal Sciences, Lahore and transported to the Applied zoology lab at the Zoology Department, University of Okara, Renala Khurd. Before trail, mice were screened for any pathogenic infection and then. acclimatized to laboratory environment for 10 days under 12-h light/dark cycles (Figure 1). All animals were cared for in compliance with the established criteria of the Ethical and Review Committee University of Okara, Pakistan.

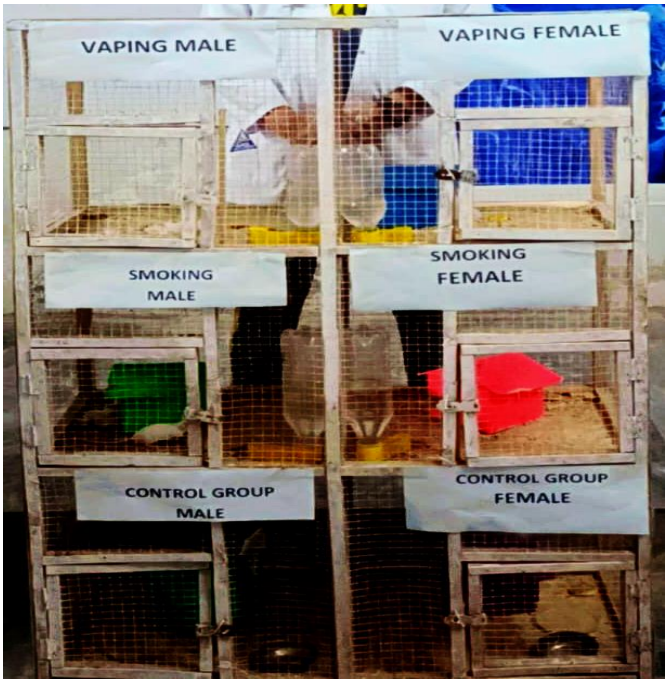


Figure 1 Cage used during the experiment

Experimental design

The mice were divided into three groups one control group two electronic smoking (T1) and Conventional smoking (T2). Each group contained 6 mice (3♂, 3♀). The expose chamber was made up of glass with a hole. The control group chamber had fresh air supply. Smoke was applied using an electronic pump in both the T1 and T2 chambers for 10 minutes each day. T1 chamber had traditional cigarette smoke whereas T2 had berry Bomb strawberry flavour 60ml E-liquid with 50mg nicotine. Both experimental groups were exposed to smoke for 6 days in a week for 9 weeks (Figure 2).

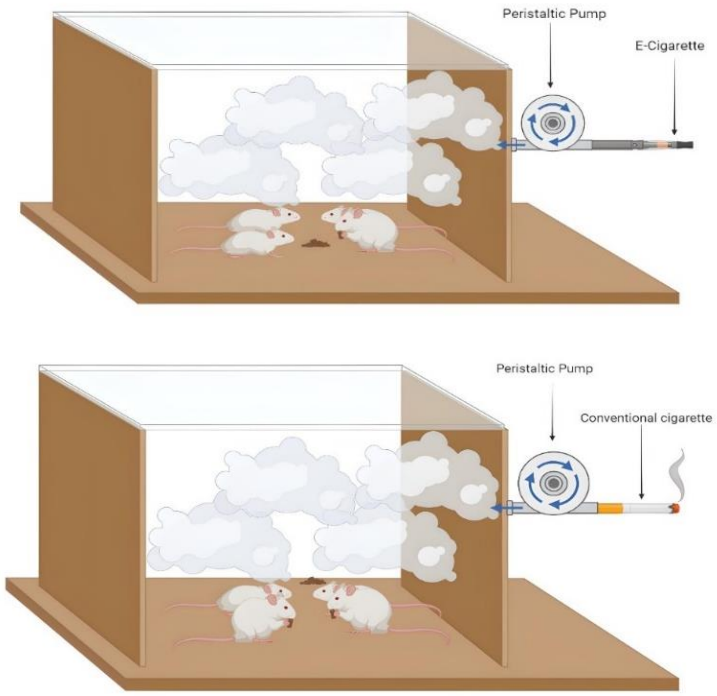


Figure 2 A general experimental design

Sampling collection

Initial and final weight was measured in each group after 9 weeks of trail.

Haematological and serological study

The mice from each group were anaesthetized with ketamine (100 mg/kg) and xylazine (10 mg/kg). Blood was drawn from the tail vein in an EDTA-filled blood collection tube. Then the blood was subjected to haematological analysis such as WBC (x 109 /L), Lymphocytes (%), Granulocytes (%), Haemoglobin (g/dl), MCH (pg), MCHC (g/dl), RBC (x1012/L), MCV (fL), HCT (%), RDW (%), Platelets (x109 /L), MPV (fL), PDWa (fL), PCT (%), and P-LCR % haematological analyser (MYTHIC 18). Other serological parameters such as ALT, AST, Glucose, T3, testosterone, urea, oestrogen and creatinine were measured using automated chemistry analyser (Microlab 300 chemistry analyser) (Umar et al., 2024; Aslam et al., 2025a).

Histology

After dissection, the Liver, Heart, Kidney and Lungs tissues were extracted from each mice in all groups and placed in 10% formalin solution. Histological analysis was carried out by following the protocol of Spencer et al. (2012). The microscopic study was carried out through Trinocular light microscope (model) to observe and record any histological changes in both treatment groups. for the histological analysis and photomicrographs were taken (Khan et al., 2024).

Statistical analysis

Data was presented in the form of Mean±SD Using Graph Pad 9 (San Deigo, CA). The significant difference was determined using a one-way ANOVA and Tukey's post hoc analysis. P values less than 0.05 were deemed significantly different.

Results

Growth

The growth results reveal notable decreases in body weight were recorded in both male and female mice exposed to vaping and conventional cigarette smoke. Specifically, male mice in both the vaping and conventional smoking groups exhibit gradual decreases in body weight over the nine weeks, with

fluctuations observed throughout. Female mice in these groups also display fluctuations in body weight, although the changes are less pronounced compared to males. The research indicates that both vaping and conventional cigarette smoke affect growth reduction in comparison with the control group, but the latter appeared to show fewer effects. The conventional smoking exposure seems to lead to greater body weight reduction in comparison to electronic vaping. The male subjects in the conventional smoking group began showing visible body weight decline at week 2 after exposure; the female subjects in this group demonstrated milder body weight fluctuations. The vapers report steady weight loss patterns across gender throughout the research period as their group tracks a linear descent that lessens gradually, while the conventional smokers show abrupt weight changes that bypass their vaping counterparts over time. A significant finding in this research includes the recorded physiological stress, which mainly affected male mice within both vapor and conventional smoking groups. A single female mouse passed away during week 4 from the vaping exposure group, thus affecting measurement data collection after this point.

Table 1 Body weight (grams) comparison between control, T1 and T2

Week	Gender	Control (Mean ± SD)	Vaping (T1) (Mean ± SD)	C. Smoking (T2) (Mean ± SD)	F-value	p-value
W-1	Male	35.13 ± 1.17	33.12 ± 1.24	33.91 ± 0.17	2.09	0.2044
W-1	Female	34.32 ± 0.65	33.76 ± 1.37	33.67 ± 0.31	0.31	0.7451
W-2	Male	33.75 ± 0.97	29.92 ± 1.18	31.01 ± 0.32	9.64	0.0134*
W-2	Female	32.57 ± 0.60	31.54 ± 0.78	29.41 ± 0.59	11.8	0.0083**
W-3	Male	34.34 ± 0.60	29.61 ± 0.60	29.98 ± 1.44	14.85	0.0048**
W-3	Female	34.79 ± 0.58	28.86 ± 1.36	28.92 ± 1.76	13.12	0.0064**
W-4	Male	36.08 ± 0.26	26.53 ± 1.31	29.68 ± 0.98	52.21	0.0002***
W-4	Female	34.93 ± 0.23	34.71 ± 0.62	30.74 ± 0.10	74.35	0.0001***
W-5	Male	35.43 ± 0.73	26.03 ± 0.54	30.33 ± 1.11	64.96	0.0001***
W-5	Female	34.23 ± 0.64	33.90 ± 1.38	30.15 ± 1.39	7.27	0.0249*
W-6	Male	36.95 ± 0.31	19.89 ± 0.20	27.64 ± 0.76	611.11	<0.00001****
W-6	Female	34.61 ± 0.84	33.17 ± 0.12	29.94 ± 0.77	26.24	0.0011**
W-7	Male	36.39 ± 0.95	20.81 ± 0.25	28.04 ± 0.31	346.75	<0.00001****
W-7	Female	35.37 ± 1.73	32.29 ± 0.24	30.35 ± 0.40	11.93	0.0081**
W-8	Male	37.92 ± 1.03	18.46 ± 0.59	27.42 ± 1.18	203.73	<0.00001****
W-8	Female	36.40 ± 0.42	31.95 ± 0.71	29.36 ± 0.61	72.21	0.0001***
W-9	Male	37.68 ± 1.90	21.69 ± 0.67	26.90 ± 0.72	87.02	<0.00001****
W-9	Female	37.83 ± 1.16	31.20 ± 0.57	28.49 ± 1.43	37.26	0.0004***

(*) for $p < 0.05$ (significant), (**) for $p < 0.01$ (highly significant),(***) for $p < 0.001$ (very highly significant), and (****) for $p < 0.0001$ (extremely significant).

Haematology

The levels of both haemoglobin and haematocrit decreased in both groups. White blood cell counts in the e-cigarette group were significantly higher than in conventional smokers, which showed stronger inflammatory responses. The e-cigarette smokers had notably elevated lymphocyte counts, but conventional cigarette users displayed increased levels of monocytes together with reduced granulocyte counts. The e-cigarette group had the highest platelet count, yet conventional smoking caused a major decrease in platelet numbers. The mean platelet volume, platelet crit levels, and platelet distribution width measurements were all lower in the smoking groups. The use of e-cigarettes led to lower red cell distribution width values when compared to

conventional cigarette users, who showed a more limited decrease in the same variable. The levels of mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration went up in both groups of smokers, but the rise was bigger in the regular smokers (Table 2).

Table 2: Comparison of haematological parameters between control, e-cigarette smoking, and conventional smoking groups

Parameters	Control	T1	T2	F Value	P Value
HGB(HB%) (g/dl)	6.81±0.09	6.67±0.356	5.982±0.059	21.08	0.0001***
WBC(TLC)(10^3/ul)	0.854±0.1	3.13±0.11	1.798±0.104	583.7	<0.0001****
LYM%	70.79±0.16	85.56±0.218	69.574±0.394	5224	<0.0001****
MON%	15.70±0.11	8.588±0.366	19.548±0.178	2624	<0.0001****
GRA%	13.536±0.1	5.674±0.155	10.486±0.199	3238	<0.0001****
RBC (10^6/ul)	4.906±0.13	4.568±0.112	4.7±0.249	4.698	0.0311*
HCT%	25.54±0.46	24.316±0.35	23.45±0.273	40.97	<0.0001****
MCV (um^3)	52.28±0.06	52.402±0.29	51.15±0.065	78.41	<0.0001****
MCH%	13.66±0.25	14.466±0.17	14.716±0.427	16.46	0.0004***
MCHC(g/dl)	26.364±0.2	27.78±0.24	29.2±0.10	280	<0.0001****
RDW%	25.62±0.35	19.26±0.79	21.87±1.063	81.24	<0.0001****
RDW-SD (um^3)	51.61±0.18	41.68±0.54	39.646±0.282	1523	<0.0001****
Platelets (10^3/ul	511.8±1.79	552.4±5.14	325.89±0.26	7357	<0.0001****
MPV (um^3)	6.092±0.08	5.23±0.122	4.58±0.345	61.28	<0.0001****
PCT%	0.312±0	0.24±0.038	0.16±0.037	30.72	<0.0001****
PDW%	13.42±0.45	12.464±0.09	13.874±0.123	34.55	<0.0001****

Metabolism

Glucose levels and the concentration of thyroid hormone were significantly different in all groups. There was a significant increase in the level of glucose in both smoking groups. However, thyroid hormone concentration was significantly higher in the conventional smoking group. (Figure 3).

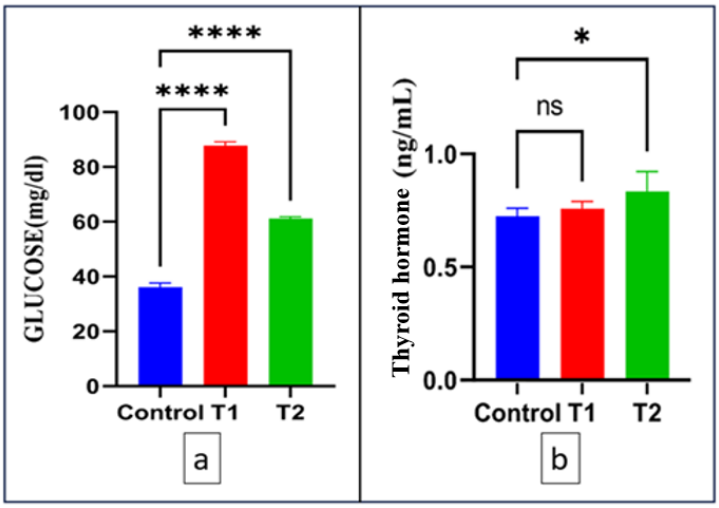


Figure 3: Comparison of glucose and thyroid hormone levels among e-cigarette and conventional smoking groups

Estrogen and Testosterone Levels

The e-cigarette group displayed higher estrogen levels than both the control group and the conventional smoking group. However, the testosterone level was higher in the conventional smoking group (Figure 4).

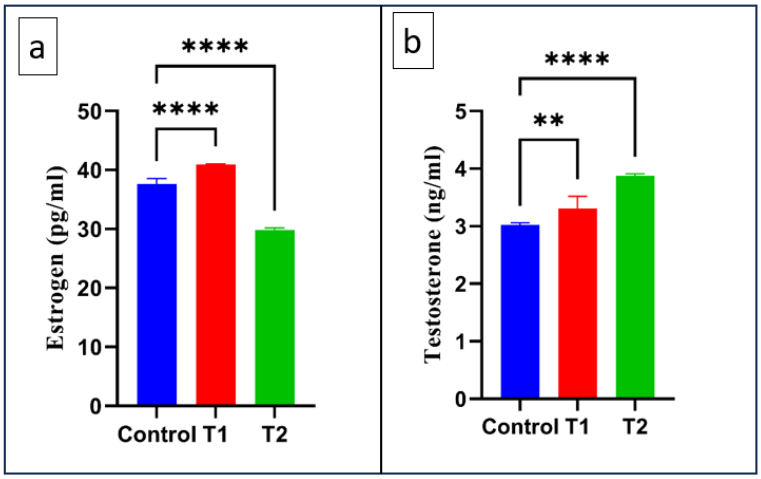
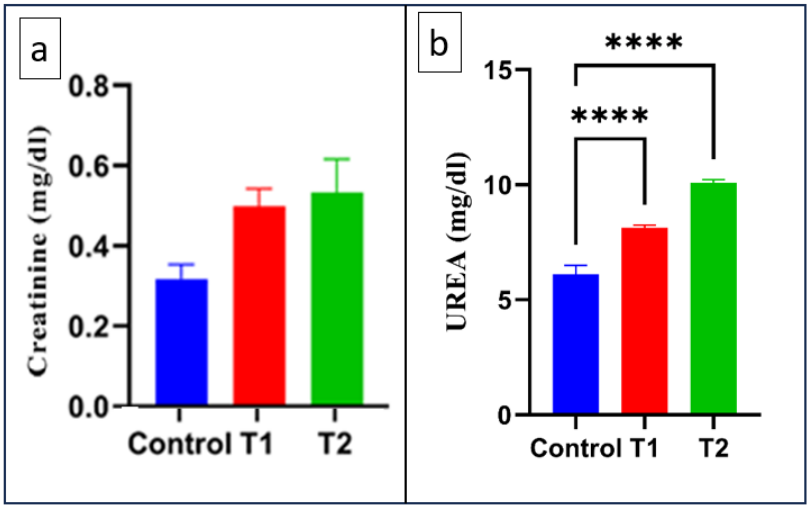


Figure 4: Effects of E-Cigarette and Conventional Smoking on Estrogen and Testosterone Levels

Liver and Kidney Function Biomarkers

The analysis of kidney function biomarkers showed that both creatinine and urea levels were significantly higher in the e-cigarette (T1) and conventional smoking (T2) compared to the control, with the highest in the conventional smoking group. Regarding liver function, aspartate aminotransferase (AST) levels were significantly elevated in the e-cigarette group compared to both the control and conventional smoking groups. However, alanine aminotransferase (ALT) levels increased in the conventional smoking group (Figure 5).



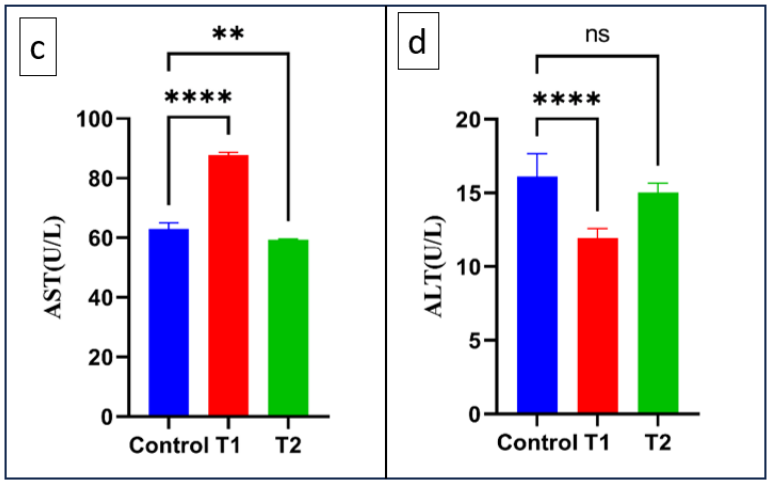


Figure 5: Comparison of kidney and liver function biomarkers between e-cigarette smoking

Histopathology

E-cigarette smoking group showed presence of myocardial interstitial fibrosis (black arrow), collagen and extracellular matrix elements, built up in heart interstitial spaces. further, e-cigarettes shared similar alterations with conventional cigarettes, as the muscle fibers displayed either shattered muscle fibers (yellow arrow) or loose muscle fibres with expanded space (blue arrow). This is a sign of both an architectural problem and a tissue disorder in the heart (figure 3).

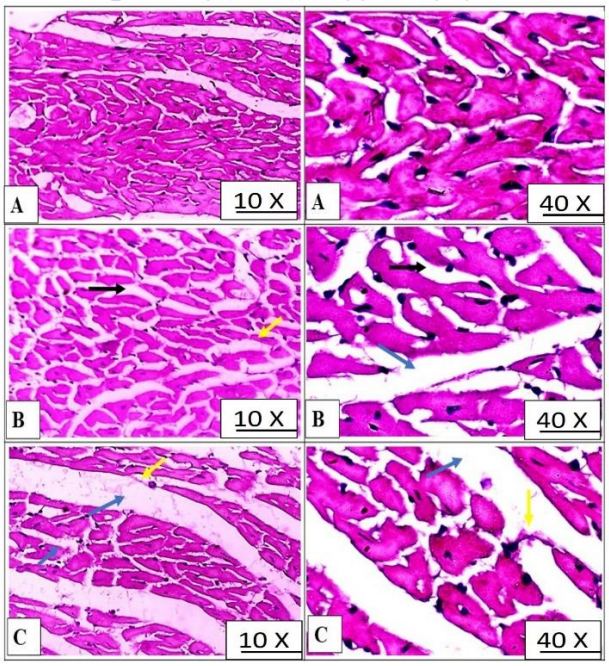


Figure 3: Histopathological comparison of cardiac tissues in mice exposed to electronic and conventional cigarette smoking. (A) The control (B) E-cigarette smoking (C) Conventional smoking group

In kidney histology, figure 4 (B) showed glomerular scarring or glomerulosclerosis (black arrow), interstitial fibrosis (red arrow) and mesangial expansion (yellow area) in the e-cigarette smoking group. In the conventional smoking group, mesangial enlargement (yellow arrow) and glomerulosclerosis (black arrow) were observed.

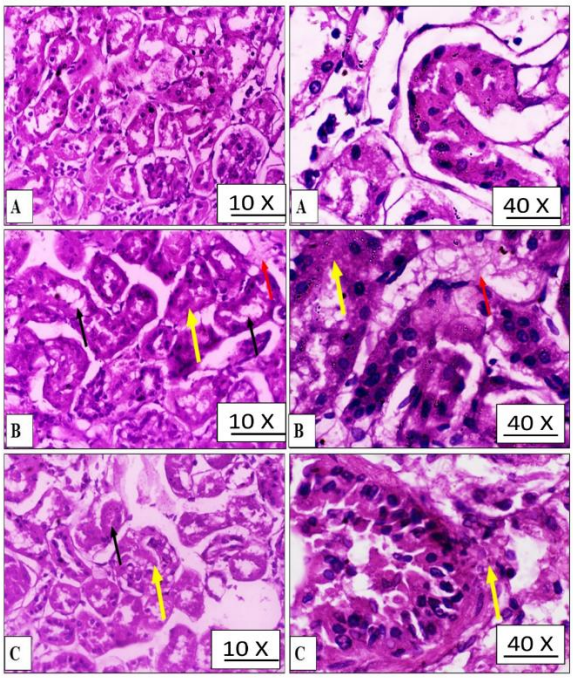


Figure 4: Histological analysis of kidney tissues in mice exposed to electronic and conventional cigarette smoking. (A) Control group (B) E-cigarette smoking (C) Conventional smoking group

The e-cigarette exposure induced emphysema (black arrow) with bigger air spaces, tissue hyperplasia (blue arrow) and hypertrophy (red arrow). The conventional smoking mice group exhibited substantial changes such as the buildup of fibrosis (yellow arrow) in the lung parenchyma, intimal hyperplasia (green arrow) and hyperplasia (blue arrow) as shown in Figure 5.

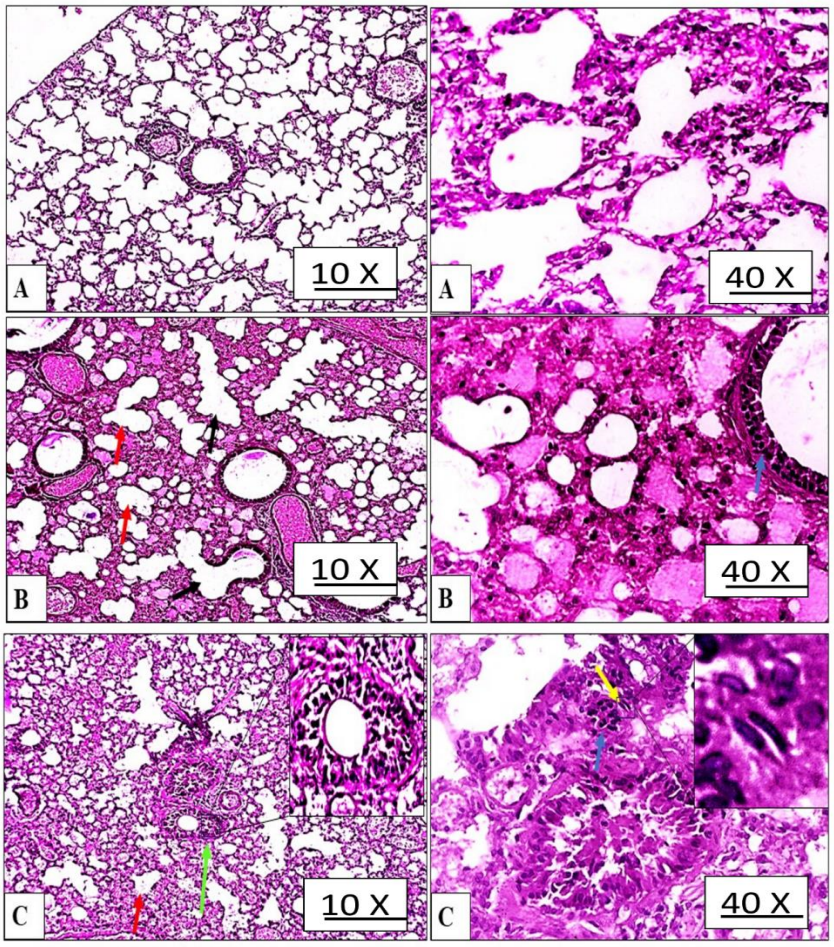


Figure 5: Histopathological analysis of lung tissues in mice exposed to electronic and conventional cigarette smoking. (A) Control (B) E-cigarette smoking (C) Conventional smoking group

The histological examination of liver tissues in the e-cigarette exposure group showed hepatocellular ballooning (red arrow), indicating hepatocyte swelling and vacuolization. Further, degenerative changes in hepatocytes (yellow arrow) and dilated portal vein (grey arrow) were observed in the e-cigarette group. Alterations were seen in the liver histology of the mice in the traditional smoking group such as the presence of lipid droplets (black arrow), hepatocellular ballooning (red arrow) and degenerative alterations in hepatocytes (yellow arrow) suggested liver injury. Furthermore, the green arrow representing fibrosis indicates the accumulation of collagen and other extracellular matrix elements in the liver parenchyma (figure 6).

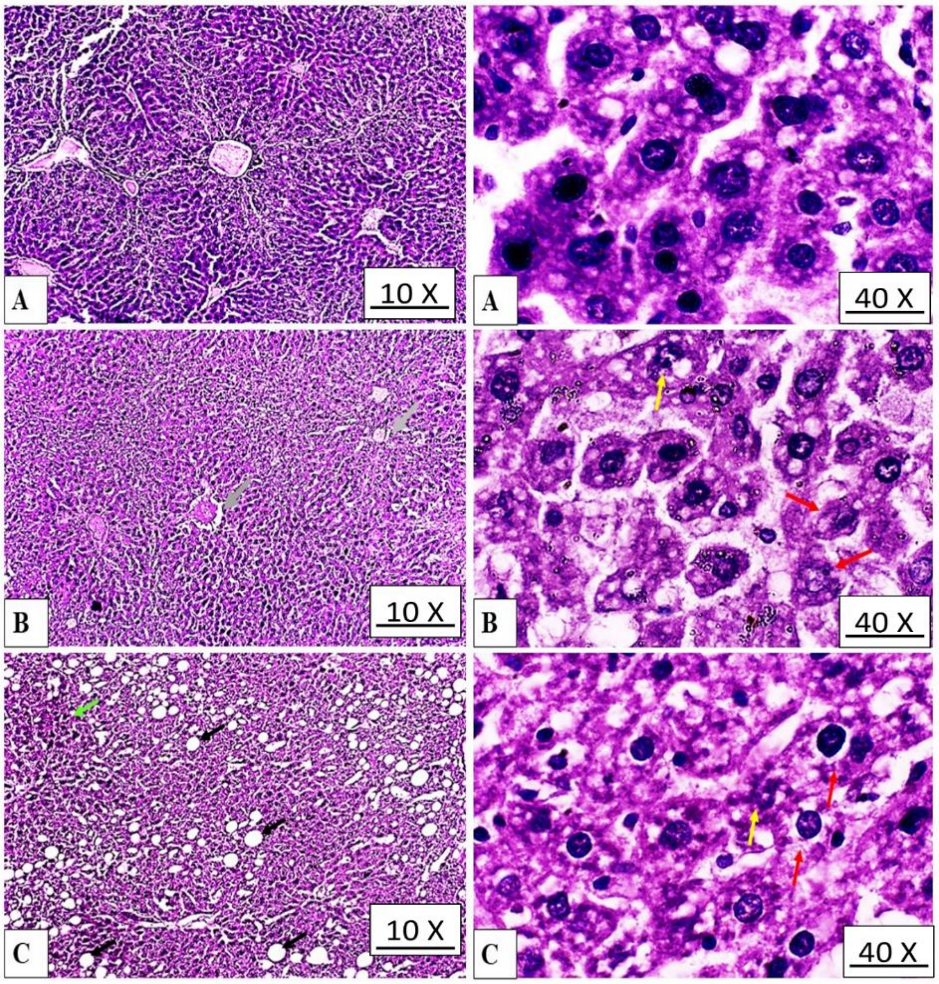


Figure: Histopathological analysis of liver tissues in mice exposed to electronic and conventional cigarette smoking. (A) Control (B) E-cigarette smoking group (C) Conventional smoking group

Discussion

E-cigarettes gained much popularity in very short period of time; thus researchers are under pressure to design controlled trials of lung function that compare the effects of "classical" CS with those of e-vapors cigarettes. Acute toxicological profile of e-liquid cigarette refill components has only been addressed in a small number of in vitro investigations using murine and human lung epithelial cell lines, fibroblasts, and stem cell (Bahl et al., 2012; Farsalinos et al., 2013; Romagna et al., 2013). Chun et al. (2017) reviewed that Primary bronchial epithelial cell exposed directly to e-vapor cig glycerol/propylene glycol showed less severe oxidative stress than cells exposed to traditional cigarette. Further evidence suggests that e-cigarette flavour compounds are responsible for the harmful effects on pulmonary fibroblasts.

The current study aimed to investigate and compared the effects of electronic smoking and conventional cigarette smoking on the albino mice model. The levels of both haemoglobin and haematocrit decreased in both treated groups. White blood cell counts in the e-cigarette group were significantly higher than in conventional smokers, which showed stronger inflammatory responses. The e-cigarette smoking group had notably elevated lymphocyte counts, but conventional cigarette displayed increased levels of monocytes. Research revealed that smoking habits affected red blood cell counts, but traditional cigarette exposure reduced red blood cells to a larger

extent than e-cigarettes did. These results imply that systemic inflammatory reactions and haematological alterations in mice may be brought on by electronic cigarette smoking. A possible reason for the immunological response or inflammatory reaction, as seen by the rise of white blood cell count and lymphocyte percentage, might be exposure to aerosols from electronic cigarettes (Kalininskiy et al., 2021; Umar et al., 2025). Further, these results are in consistent with the findings of Sharif et al. (2014) who found increased concentration of white blood cells, Platelet, Haematocrit, mean corpuscular volume, cholesterol, triglycerides and low density lipoprotein cholesterol in mice exposed to nicotine. Another possible cause of the cardiovascular problems linked to e-cigarette use is an elevated platelet count, which may suggest a pro-thrombotic condition or increased platelet activation (Alarabi et al., 2022). Changes in MCH and MCHC levels may indicate changes in erythropoiesis or oxygen-carrying ability, which might explain variations in haemoglobin concentration and red blood cell shape (Malenica et al., 2017; Aslam et al., 2025b).

Mean corpuscular haemoglobin (MCH) levels also increased significantly in conventional cigarettes smoke, suggesting the change in shape of red blood cell and haemoglobin content may have changed. Interestingly, conventional smoke lower the platelet count compared to control possibly due to the fact that traditional cigarette smoking may damage platelet synthesis or function (Qasim et al., 2018). E-cigarettes smoking group had significantly higher levels of ALT and AST, glucose, T3, testosterone, urea, creatinine, and oestrogen similar to findings of Sharif et al. (2014), who found that administering nicotine reduces total proteins, albumin, and bilirubin levels while increasing ALT, AST, and ALP levels. This rise in liver enzyme suggests hepatotoxic effects of cigarette smoke. In addition, the conventional smoking group had much higher glucose and thyroid hormone levels, which may point to insulin resistance or changes in glucose metabolism and thyroid function. The conventional smoking group also had significantly higher levels of urea and creatinine indicating renal damage or malfunction.

The findings from the body weight comparison among the control group, vaping (T1), and conventional smoking (T2) groups over nine weeks reveal notable trends and implications. Both the vaping and conventional smoking groups exhibited a decline in body weight compared to the control group, suggesting potential adverse effects on growth and development. This weight loss trend was more pronounced in the vaping group, particularly in male mice, where body weight decreased substantially from around 32.67 to 21.33 grams over the study period. Similarly, in the conventional smoking group, male mice showed a significant decrease in body weight from approximately 34 to 27 grams. Female mice in both groups also experienced weight loss, albeit to a lesser extent.

There was continuous and gradual increase in body weight of male and female mice in control group but there was gradual decrease in body weight of male and female mice in both electronic and conventional smoking group. The decrease in weight is associated with food intake. The mice decreased the food intake during the exposure period. The decrease in food intake was due to effect of nicotine present in the smoke. Mangubat et al. (2012) found that nicotine reduced food intake and raised energy expenditure in mice on a high-fat diet (HFD), but nicotine withdrawal caused weight gain because energy expenditure decreased. In both groups each mice loss almost 10g of weight and there was one fatality recorded in electronic smoking and 2 fatalities were recorded in conventional smoking group. It was surprising to note that all the fatalities recorded in females. Smoking causes a number of changes in the body, including a slower metabolism, less food intake, and perhaps harmful effects of the chemicals in cigarette smoke, all of which contributed to the nine-week weight loss in both groups.

E-cigarette smoking group showed presence of myocardial interstitial fibrosis, collagen and extracellular matrix elements, built up in heart interstitial spaces. further, e-cigarettes shared similar alterations with conventional cigarettes, as the muscle fibers displayed either shattered muscle fibers or loose muscle fibres with expanded space. This is a sign of both an architectural problem and a tissue disorder in the heart. The initiation of interstitial fibrosis in reaction to electronic cigarette smoke and has multiple possible contributing factors. E-cigarette

aerosols generate oxidative stress, inflammation, and endothelial dysfunction because they transmit various harmful reactive substances, including nicotine, alongside propylene glycol and flavorings (Hariri et al., 2022). Long-term exposure to harmful chemicals causes fibroblasts to become active and collagen to build up, which changes the heart tissue in a way that is similar to fibrosis. Researchers have found that nicotine causes more collagen to be made and more fibroblasts to multiply, which speeds up the development of interstitial disc disease (Suryadinata et al., 2022). These morphological changes indicate how smoking cigarettes regularly and using e-cigarettes causes both structural damages and cardiac injuries. Due to necrosis, myocyte degeneration, and cellular integrity loss, the gaps between loose muscle fibers are getting bigger. This is a sign of both an architectural problem and a tissue disorder in the heart. Myocardial damage and cell death triggered by exposure to cigarette smoke can be observed through muscle fiber fractures, while myocardial damage or cell death caused by ischemia or oxidative Traditional tobacco usage along with e-cigarettes generates oxidative stress and mechanical stress that damage cardiac cell structure, which leads to documented histological effects (Nucci et al., 2019).

In kidney histology, glomerular scarring or glomerulosclerosis, interstitial and mesangial expansion in the e-cigarette smoking group. In the conventional smoking group, mesangial enlargement and glomerulosclerosis were observed. These finding were in consistent with Jiang et al. (2019) who found mesangial expansion glomerulosclerosis and interstitial fibrosis of the kidney exposed to smoking. These findings indicate potential renal damage or dysfunction associated with smoking. Glomerulosclerosis occurs due to chronic kidney disease and glomerular basement membrane gets harder and thicker. Kidney interstitial fibrosis cause tissue damaged and scarred over time (Lee et al., 2021). Due to more collagen and other extracellular matrix components in the renal interstitium. The problems in the mesangial matrix make it hard for the glomeruli to filter (El Mokdad, 2023).

In lung tissue, e-cigarette exposure induced emphysema with bigger air spaces, tissue hyperplasia and hypertrophy. The conventional smoking mice group exhibited substantial changes such as the buildup of fibrosis in the lung parenchyma, intimal hyperplasia and hyperplasia. Smoking seems to have negative impacts on the structure and function of the lungs, as shown by these alterations (Yoshida & Tuder, 2007).

The histological examination of liver tissues in the e-cigarette exposure group showed hepatocellular ballooning, indicating hepatocyte swelling and vacuolization. Further, degenerative changes in hepatocytes and dilated portal vein. Alterations were seen in the liver histology of the mice in the traditional smoking group such as the presence of lipid droplets, hepatocellular ballooning and degenerative alterations in hepatocytes suggested liver injury. Furthermore, fibrosis was also recorded which indicates the accumulation of collagen and other extracellular matrix elements in the liver parenchyma. These results point to the possibility of hepatotoxic consequences and liver damage linked to smoking (Hasan et al., 2019; Shaffique et al., 2024).

Conclusion

This study concluded that both e-cigarette and conventional cigarette smoke had significant adverse impact on albino mice as evident by progressive reduction in body weight, haemotological disruption, change metabolic and hormonal level and tissue damage. Male had more weight loss than female in both groups. Hematological analysis revealed e-cigarettes significantly increase the level of white blood cell and lymphocyte counts, while conventional smoking increased monocyte levels and severely decreased platelet counts. Metabolically, hyperglycemia is experienced in both groups however, conventional smoking significantly increased the T3 and testosterone level. Liver and kidney markers are significantly higher in conventional smoking group. Histological evaluation further confirmed tissue level damage in heart, kidney, liver and lung. Future research should focus dose-dependent and long-term effects of vaping and conventional smoking with focus on molecular mechanism and gender-specific responses. Expanding sample sizes and dosage groups also recommended.

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N/A

Conflict of Interest
No conflict of interest.

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No funding information available.

HIGHLIGHTS

- E-cigarettes increase WBCs and platelets, disrupting immune and blood functions more.
- Both smoke types alter hormones; estrogen up in T1, testosterone up in T2 mice.
- Histology reveals organ damage; T2 group shows more severe structural changes.

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