FUDMA Journal of Sciences (FJS)
ISSN online: 2616-1370
ISSN print: 2645 - 2944
Vol. 9 No. 12, December, 2025, pp 194 – 199
DOI: https://doi.org/10.33003/fjs-2025-0912-4055

ACUTE EFFECT OF MAINSTREAM CIGARETTE SMOKE ON HEMODYNAMICS, SERUM NICOTINE, BODY WEIGHT AND ORGAN MASS IN ALBINO RATS

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ABSTRACT

This two-week inhalation study investigated the acute physiological effects of mainstream cigarette smoke (MSCS) on blood pressure, heart rate, body weight and internal organ mass in albino rats. Thirty-two Sprague-Dawley rats (150–200 g; equal sexes) were divided into Control (n = 16) and Smoke-exposed (n = 16) groups. The exposed rats inhaled smoke generated from one standard cigarette per day for 14 consecutive days in a nose–mouth exposure chamber under calibrated suction, while controls received filtered air. Serum nicotine levels were quantified using high-performance liquid chromatography. Blood pressure was measured invasively via carotid cannulation on Days 7 and 14, and internal organs (heart, lungs, liver, kidneys) were weighed post-sacrifice. Data were analysed using Student's and paired t-tests with significance set at $p < 0.05$. Results showed that serum nicotine increased in smoke-exposed rats (~9 ± 1 ng/mL on Day 7) but declined by Day 14 (~5 ± 0.3 ng/mL), suggesting a likely enhancement of metabolic clearance or physiological adaptation. Systolic and mean arterial pressures rose significantly (~15% by Day 14) without a corresponding rise in heart rate. Kidney and lung weights were reduced by about 10% compared with controls, while heart and liver masses remained unchanged. These findings indicate that short-term MSCS exposure elevates arterial pressure and induces organ-specific alterations, particularly in the kidneys and lungs. The decline in serum nicotine likely reflects enhanced hepatic CYP450-mediated metabolism. Therefore, stricter regulation of tobacco smoke constituents and sustained public enlightenment are strongly recommended to mitigate early cardiovascular and organ-level damage.

Keywords: Cigarette, Smoke, Exposure, Nicotine, Organs, Blood Pressure

INTRODUCTION

Cigarette smoking remains one of the leading causes of preventable illness and death around the world. It is strongly linked to heart and lung diseases, which together account for millions of premature deaths every year. In 2024, tobacco smoking was responsible for more than eight million deaths, mainly from cardiovascular complications (World Health Organisation, 2024). Nicotine, the main active and addictive chemical in tobacco smoke and e-cigarette vapor, enters the bloodstream quickly through the lungs. It stimulates specific receptors in the nervous system and adrenal glands, releasing hormones such as adrenaline that tighten blood vessels, raise blood pressure, and alter heart rhythm (Benowitz, 2010).

Studies in both animals and humans have shown that nicotine increases blood pressure within minutes of exposure. In laboratory animals, injections or inhalation of nicotine rapidly elevate arterial pressure, while heart rate may stay steady or briefly decrease because of reflex adjustments (Zhao *et al*., 2020). In human studies, even a short exposure of 20–30 minutes to vaporized nicotine causes noticeable increases in blood pressure, even among non-smokers (Haass & Kübler, 1997). However, while long-term smoking has been widely studied, short-term exposure lasting one to two weeks has received much less attention. Understanding these early responses is essential, because they can reveal how quickly nicotine and cigarette smoke begin to affect key organs and blood circulation.

Experimental work with rodents shows that just a few days of smoke exposure can cause measurable changes in the body. For example, five-day exposures using nose-only chambers have been shown to damage lung cells and increase the production of enzymes involved in inflammation, such as cyclooxygenase-2 (COX-2) and matrix metalloproteinase-12 (MMP-12) (Short Term Systemic Effects, 2018). These enzymes normally help repair tissue but, when over-activated, can worsen injury and inflammation. Short exposures also raise oxidative stress; an imbalance between free radicals and antioxidants, which can damage DNA and affect metabolism in several tissues (Chen *et al*., 2015).

In diabetic rat models, brief smoke inhalation has been found to worsen kidney function. It increases the production of fibronectin and transforming growth factor-β (TGF-β), which are proteins linked to fibrosis, and NADPH oxidase-4 (NOX4), an enzyme that promotes oxidative stress, leading to tissue damage and functional decline (Liu *et al*., 2020). Even exposure to nicotine alone, without the smoke particles, can disrupt normal lung function. Studies report that inhaled nicotine increases fluid accumulation in the lungs (pulmonary edema) and triggers inflammation marked by the release of a signalling protein called high-mobility group box-1 (HMGB1), along with the migration of immune cells known as neutrophils. These responses indicate early tissue stress and occur at nicotine levels similar to those measured in human smokers (Silva-Oliveira *et al*., 2023).

Nicotine also affects the heart and blood vessels. It can cause irregular heartbeat (arrhythmia) by interfering with potassium ion movement in heart cells and can contribute to high blood pressure by narrowing blood vessels. This happens partly because nicotine reduces nitric oxide which is a natural chemical that helps blood vessels relax as well as increases endothelin-1,

a compound that tightens them. Over time, such changes can reduce the ability of arteries to expand properly, a condition known as endothelial dysfunction (Whitehead *et al*., 2021).

In terms of metabolism, nicotine is broken down mainly in the liver through enzymes of the cytochrome P450 (CYP450) system. In rodents, it has a short half-life of about one hour, meaning it clears quickly from the blood. Sensitive analytical techniques, such as high-performance liquid chromatography (HPLC), can detect very small amounts, down to about 1 ng/mL, making them ideal for studying how nicotine levels change over time (Benowitz & Jacob, 2024; Schröder *et al*., 2021).

Based on this background, the present study was designed to quantify serum nicotine concentration after 7 and 14 days of exposure to mainstream cigarette smoke (MSCS) containing about 0.7 mg of nicotine per session using Rothmans cigarettes. It also seeks to measure hemodynamic responses including systolic blood pressure (SBP), mean arterial blood pressure (MABP) and heart rate (HR) through direct monitoring via carotid artery cannulation (Parasurama & Raveendran, 2012). In addition, the study assesses body weight changes and internal organ masses (heart, lungs, kidneys, and liver) to determine how short-term smoke exposure may influence overall physiology.

This combined approach helps explain early responses that occur before visible disease develops. By linking nicotine concentration to blood pressure and organ changes, the study provides insight into how even brief exposure to cigarette smoke can disturb normal body functions. These findings are also highly relevant for e-cigarette users, who may experience similar effects since both traditional and electronic cigarettes deliver nicotine that acts on the same biological pathways. Recent studies show that e-cigarette aerosols and mainstream smoke share comparable effects on blood pressure, oxidative stress, and vascular function (Benowitz, 2010; Silva-Oliveira *et al*., 2023; Whitehead *et al*., 2021).

Overall, this research aims to fill a key knowledge gap by unifying three critical parameters; nicotine kinetics, hemodynamic regulation, and organ responses, within a short-term exposure model. The results are expected to enhance understanding of the early toxicological effects of nicotine and smoke exposure, supporting preventive strategies for both conventional smokers and the rapidly growing population of e-cigarette users.

MATERIALS AND METHODS

Study Design

Animal Housing

Thirty-two (32) adult albino Sprague–Dawley rats (150–200 g; 16 males and 16 females) were randomly assigned (8 M/8 F per group) into Control and Smoke-exposed groups (n = 16 per group). Sample size determination was guided by earlier rodent studies assessing cardiovascular, oxidative, and histological responses to cigarette smoke exposure (Al Arifi *et al*., 2012; Al-Awaida *et al*., 2016; Ovie *et al*., 2021). These studies demonstrated that groups of 6–10 animals per treatment provided adequate statistical power to detect significant physiological effects at $\alpha = 0.05$. Animals were housed under standard laboratory conditions (22–26 °C, 12:12 h light/dark cycle) with unrestricted access to food and water. Animals were acclimatized for one week before exposure.

Cigarettes and Smoke Exposure Protocol

Rothmans cigarettes (British American Tobacco, UK) containing approximately 8 mg tar and 0.7 mg nicotine per stick were used. The choice of Rothmans brand was based on its wide market availability and moderate nicotine content representative of typical commercial cigarettes commonly consumed in Nigeria and other low–middle-income settings (WHO, 2024). The 0.7 mg nicotine yield per cigarette approximates the nicotine dose inhaled by a light-to-moderate human smoker (≈ 0.8 mg/session, corresponding to ≈ 0.015 mg/kg in a 70 kg adult). Scaled to the metabolic rate and body mass of rats, this exposure approximates 0.2–0.25 mg/kg/day nicotine-equivalent, which reflects physiologically relevant human exposure levels (Benowitz & Jacob, 2024). Smoke exposure was conducted in a nose–mouth inhalation chamber, once daily for 10–12 min per rat, for 14 consecutive days. This duration and frequency were chosen to mimic sub-chronic human exposure patterns and to minimize excess oxidative stress beyond physiologically translatable limits. Control animals were handled identically but received filtered air. The nose-only method reduces dermal and oral contamination, ensuring exposure primarily through inhalation.

Serum Nicotine Quantification (HPLC)

On Days 7 and 14, approximately 0.5 mL of blood was collected via the retro-orbital sinus under light isoflurane sedation. Although retro-orbital bleeding is a standard method for small rodents due to its efficiency and reproducibility, it is recognized as invasive and potentially stress-inducing. Therefore, the procedure was performed swiftly by trained personnel under light anaesthesia to minimize discomfort and physiological stress responses that could confound results. Serum samples were extracted with organic solvents and analysed using high-performance liquid chromatography coupled with ultraviolet detection (HPLC–UV; Agilent Technologies, USA). Calibration curves were linear over the expected range, with a detection limit of approximately 0.2 ng/mL.

Invasive Blood Pressure and Heart Rate Measurement

At the end of exposure, invasive hemodynamic parameters were recorded under urethane anaesthesia (1.2 g/kg, i.p.). After tracheostomy, the left carotid artery was cannulated with heparinised saline. Systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were measured via a pressure transducer system, while mean arterial blood pressure (MABP) and pulse pressure (PP) were calculated accordingly. This approach follows established gold-standard protocols for rodent cardiovascular assessment (Parasurama & Raveendran, 2012).

Body and Organ Weight Assessment

Body weights were measured at baseline, Day 7, and Day 14. After final physiological recordings, animals were euthanized and organs (heart, lungs, liver, and kidneys) were excised, blotted dry, and weighed to the nearest 0.01 g to determine organ-to-body weight ratios.

Statistical analysis: All data are presented as mean ± standard error of mean (SEM). Between-group differences (Control vs Smoke) were evaluated using independent Student's t-tests, while within-group changes (Day 7 vs Day 14) were assessed by paired t-tests. Statistical significance was set at $p < 0.05$. Analyses were performed using SPSS version 25.0 and cross-validated in Microsoft Excel.

RESULTS AND DISCUSSION

Table 1: Effect of Cigarette Smoke on the Blood Pressure and Heart Rate of Albino Rats

Blood Pressure Parameter	Control	Test	p-value	Remark
SBP (mmHg)	67.75 ± 3.11	77.25 ± 4.67	< 0.05	Significant
DBP (mmHg)	42.50 ± 3.27	48.00 ± 4.52	> 0.05	Not Significant
PP (mmHg)	25.25 ± 4.11	29.25 ± 2.75	> 0.05	Not Significant
MABP (mmHg)	50.91 ± 2.57	57.75 ± 4.39	< 0.05	Significant
AP (mmHg)	110.25 ± 4.90	125.25 ± 8.78	< 0.05	Significant
HR (beats/min)	375.00 ± 28.72	390.00 ± 17.32	> 0.05	Not Significant

Keys: SBP=Systolic Blood Pressure; DBP=Diastolic Blood Pressure; PP=Pulse Pressure (SBP – DBP); MABP= Main Arterial Blood Pressure (DBP + PP/3); AP=Arterial Blood Pressure

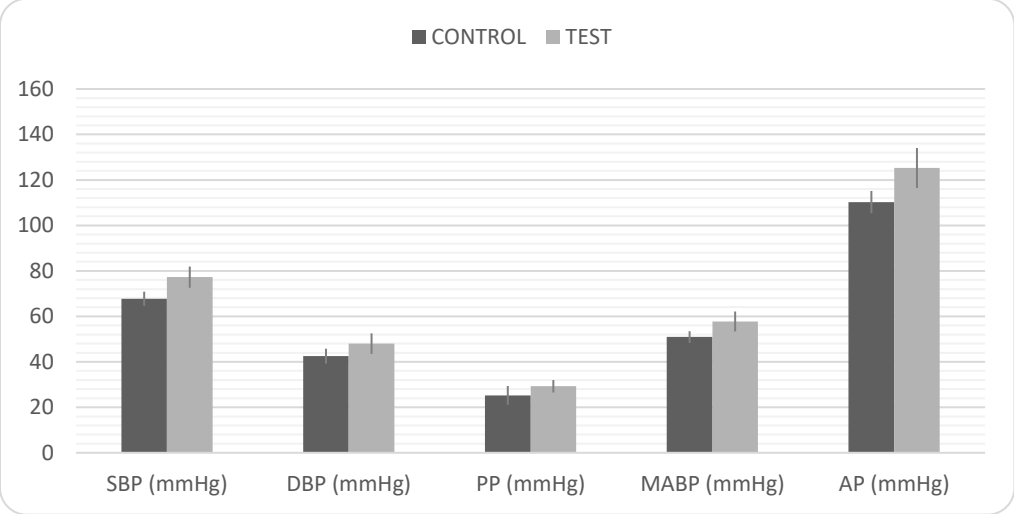


Figure 1: Effect of Cigarette Smoke on the Blood Pressure and Heart Rate of Albino Rats

Exposure to cigarette smoke caused a clear rise in systolic, mean arterial, and total arterial blood pressure in the rats, while diastolic and pulse pressures did not change significantly. These results suggest that cigarette smoke can trigger an early rise in blood pressure. Nicotine, the main active compound in smoke, stimulates nerves in the adrenal gland and blood vessels, leading to the release of adrenaline and other chemicals that increase blood pressure (Liu *et al*., 2019; Zhao *et al*., 2020). However, the heart rate did not increase even though blood pressure rose. This is likely because the rats were anesthetized, and anesthesia can reduce the body's natural reflexes that control heart rate. Normally, when blood pressure increases, the body slows the heart rate to balance circulation (baroreflex). Anesthesia can blunt this reflex, leading to stable heart rate readings despite increased blood pressure (Haass & Kübler, 1997; Zheng *et al*., 2017; Whitehead *et al*., 2021). The true effect of smoke on cardiovascular function can be clearly shown via measuring blood pressure and heart rate in non-anesthetized rats.

Table 2: Effect of Cigarette Smoke on the Weight of Internal Organs

Organ	Control	Test	p-value	Remark
Heart (g)	0.57 ± 0.04	0.50 ± 0.10	> 0.05	Not Significant
Kidney (g)	1.00 ± 0.07	0.80 ± 0.08	< 0.05	Significant
Liver (g)	6.37 ± 0.44	6.07 ± 0.66	> 0.05	Not Significant
Lungs (g)	1.27 ± 0.07	0.95 ± 0.06	< 0.05	Significant

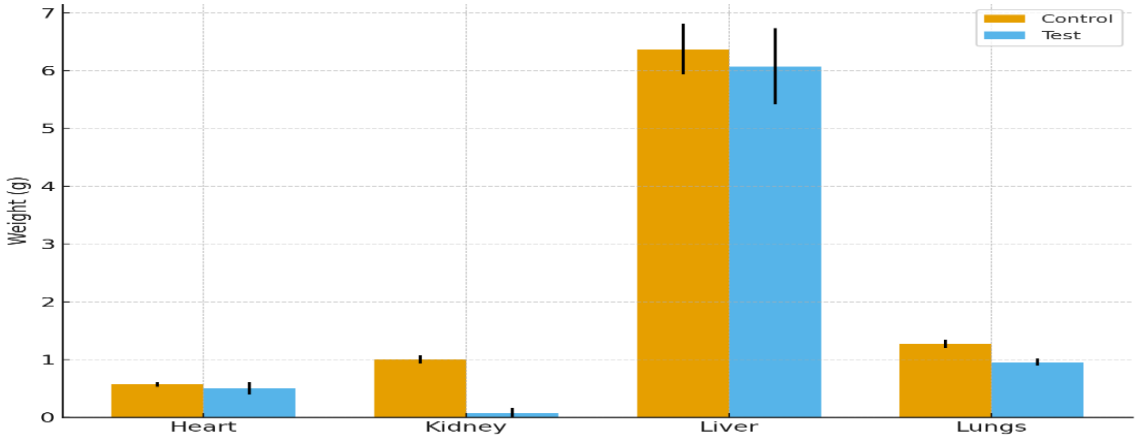


Figure 2: Effect of Cigarette Smoke on the Weights of Internal Organs

Lung and kidney weights were significantly lower in rats exposed to cigarette smoke, while the heart and liver weights showed no meaningful change. The reduction in lung and kidney mass could mean that smoke caused early tissue damage or inflammation. The lungs are directly exposed to smoke and its toxic gases, which can irritate the airways, damage lung cells, and cause inflammation (Ahmad *et al.*, 2018; Chen *et al.*, 2015). The kidneys are also affected indirectly through the bloodstream, which carries harmful chemicals from the smoke that may damage kidney tissue and reduce its size (Jiang *et al.*, 2019; Moraes *et al.*, 2021). Other studies have shown that smoke exposure increases the production of proteins such as TGF-β and NOX4, which promote inflammation and tissue scarring (Hsu *et al.*, 2020). Although this study did not measure these molecules, their involvement is possible.

Table 3: Effect of Cigarette Smoke on Heart Rate

Parameter	Control	Test	p-value	Remark
HR (beats/min)	375.00 ± 28.72	390.00 ± 17.32	> 0.05	Not Significant

Figure 3: Effect of Cigarette Smoke on Heart Rate

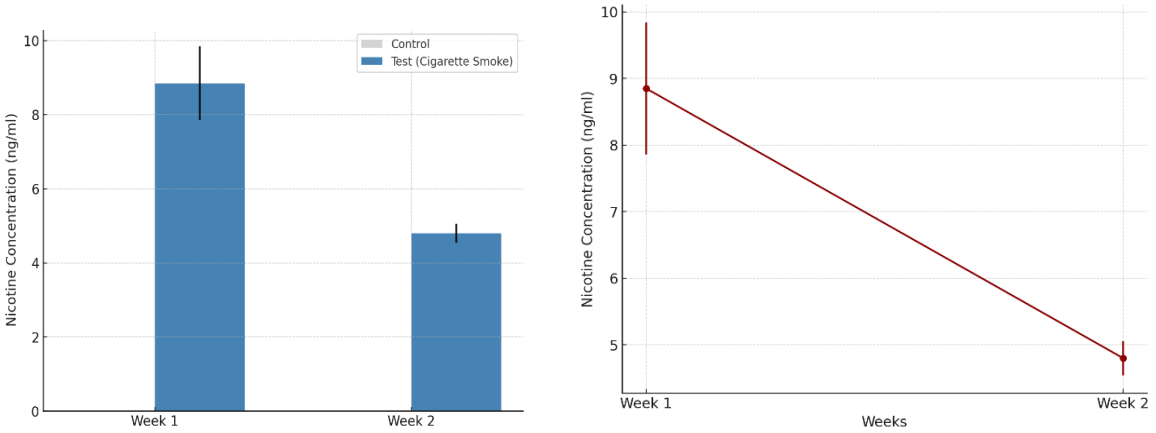
No significant difference in heart rate was found between the two groups. This supports the earlier finding that acute nicotine exposure can increase blood pressure without increasing heart rate. The body's baroreflex system and the use of anesthesia likely kept heart rate stable despite the rise in blood pressure (Smith *et al.*, 2020; Dimitriadis *et al.*, 2022). Long-term smoke exposure, however, can lead to irregular heart rhythms and weaker reflex control (Abdullahi *et al.*, 2020; de Abreu *et al.*, 2021). Therefore, longer studies and measurements of heart rate variability and autonomic balance are needed to understand these changes better.

Table 4a: Concentration of Nicotine in Blood Serum of Rats on Weekly Exposure to Cigarette Smoke

Week	Control	Test	p-value	Remark
1	0.00	8.85 ± 0.99	< 0.05	Significant
2	0.00	4.80 ± 0.26	< 0.05	Significant

Table 4b: Intra-group Weekly Comparison of Nicotine Concentration (ng/mL)

Week 1	Week 2	p-value	Remark
8.85 ± 0.99	4.80 ± 0.26	< 0.05	Significant



(a) (b)

Figure 4: (a) Weekly Nicotine Levels in Blood Serum (b) Intra-Weekly Comparison in Test Group

Nicotine levels in the blood were high after the first week of exposure but dropped significantly by the second week, even though smoke exposure continued. This shows that the bodies of rats were clearing nicotine more quickly over time. One likely reason is that repeated exposure can activate the liver's metabolic enzymes, which break down nicotine faster (Benowitz & Jacob, 2024).

However, this explanation is only a possible mechanism, since this study did not measure the enzyme levels directly. Other factors such as increased kidney elimination or redistribution of nicotine into body tissues could also explain the drop.

Table 5: Effect of Cigarette Smoke on the Body Weight of Albino Rats

Parameter	Control	Test	p-value	Remark
Baseline	142.00 ± 10.67	143.00 ± 16.55	> 0.05	Not Significant
Day 7	158.40 ± 10.21	136.75 ± 7.97	> 0.05	Not Significant

Although there was no statistically significant difference, the rats exposed to cigarette smoke gained less weight compared to the control group. This trend aligns with other short-duration smoke exposure models showing suppressed growth trajectories. Notably, Ovie *et al.* (2021), in a Nigerian study, observed that female Wistar rats exposed to cigarette smoke experienced an initial reduction in weight after the first week of exposure although weight increased over 14 days of exposure. This early weight dip supports the notion of smoke-induced appetite suppression or metabolic impact. The observed pattern in this study is also in tandem with broader global findings such as significant fat mass loss in smoke-exposed mice (Wang *et al*., 2022) or nicotine-induced weight suppression via increased fat metabolism (Rupprecht *et al*., 2018)

CONCLUSION

This study shows that short-term exposure to cigarette smoke can cause clear changes in the body and organs of albino rats. After just two weeks, there was a marked rise in systolic, mean arterial, and total arterial blood pressure, showing that cigarette smoke can quickly affect heart and blood vessel function. The lack of a similar increase in heart rate may be due to anaesthesia, which can reduce the body's natural heart rate response. The decrease in kidney and lung weights suggests early signs of inflammation or tissue damage caused by the toxic substances in cigarette smoke. These organs are especially sensitive because the lungs are directly exposed to smoke, while the kidneys help remove harmful substances from the body. Nicotine levels in the blood were high after one week but dropped by the second week, possibly because the rats' bodies began breaking it down or removing it faster. Although liver enzymes may play a role in this process, this was not directly measured. The slight drop in body weight, though not significant, may indicate metabolic changes or reduced appetite. Overall, the study shows that even brief exposure to cigarette smoke can start harmful changes before visible disease signs appear. Further research with non-anesthetized animals and including tissue and biochemical studies is needed to better understand how these effects develop. These findings confirm that cigarette smoke can harm multiple organs and support continued efforts to reduce tobacco exposure.

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