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COMPARATIVE ANALYSIS OF HEPATIC ENZYME LEVELS AND COAGULATION PARAMETERS IN ACTIVE AND PASSIVE SMOKERS: A POPULATION-BASED STUDY IN KANPUR, INDIA

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ABSTRACT

Tobacco smoking, both active and passive, poses major public health risks in India, especially in urban areas like Kanpur, where high smoking prevalence and pollution coexist. This study examines the effects of tobacco exposure on hepatic enzymes and coagulation parameters—key markers of liver function and cardiovascular health. To compare hepatic enzyme levels (SGOT, SGPT, ALP) and coagulation parameters (PT, APTT, fibrinogen) between active and passive smokers in a population-based cohort from Kanpur, India. A cross-sectional study was conducted involving 200 adults aged 20–40 years: 100 active smokers (≥1 cigarette/day for ≥6 months) and 100 passive smokers (exposed to secondhand smoke ≥15 minutes, ≥2 times/week for 1 year). Blood samples were collected in plain vials for liver enzymes (analyzed via BioSystems A25) and in sodium citrate vials for coagulation tests (semi-automated coagulometer, Clauss method). Independent t-tests were applied, with $p < 0.05$ considered statistically significant. Active smokers had significantly elevated hepatic enzyme levels (SGOT: 95.93 ± 20.97 vs. 21.35 ± 3.96 IU/L; SGPT: 96.64 ± 21.4 vs. 21.86 ± 4.07 IU/L; ALP: 160.5 ± 21.17 vs. 86.75 ± 21.17 IU/L) and a more procoagulant profile (fibrinogen: 371.27 ± 21.97 vs. 282.55 ± 4.6 mg/dL; PT and APTT significantly shorter; all $p < 0.001$).

KEYWORDS: Smoking, Secondhand Smoke, Hepatic Enzymes, Coagulation Parameters, Hypercoagulability.

1. INTRODUCTION

Tobacco smoking, a major public health concern, is a leading cause of preventable morbidity and mortality worldwide (1). In India, where smoking prevalence remains high, both active and passive smoking are significant contributors to various health complications, including cardiovascular and hepatic disorders (2). Active smoking involves direct inhalation of tobacco smoke, while passive smoking, or exposure to secondhand smoke, entails involuntary inhalation of environmental tobacco smoke by non-smokers (3). Both forms of exposure have been associated with systemic physiological changes, including alterations in hepatic function (4) and coagulation pathways (5), which may predispose individuals to liver dysfunction and thrombotic events (6).

Hepatic enzymes, such as serum glutamic-oxaloacetic transaminase (SGOT), serum glutamic-pyruvic transaminase (SGPT), and alkaline phosphatase (ALP), serve as critical biomarkers of liver health (7). Elevated levels of these enzymes are indicative of hepatocellular injury or cholestatic dysfunction, often linked to oxidative stress and inflammation induced by toxic constituents of tobacco smoke, such as polycyclic aromatic hydrocarbons and reactive oxygen species (8). Active smokers are exposed to higher concentrations of these toxins, potentially leading to more pronounced hepatic damage compared to passive smokers (9). However, the extent to which passive smoking affects hepatic enzyme levels remains underexplored, particularly in densely populated urban settings like Kanpur, India, where environmental tobacco smoke exposure is prevalent due to high smoking rates and crowded living conditions.

Coagulation parameters, including prothrombin time (PT), activated partial thromboplastin time (APTT), and fibrinogen levels, are essential indicators of hemostatic function (10). Tobacco smoke contains procoagulant compounds, such as nicotine and carbon monoxide, which may disrupt the balance of coagulation and fibrinolysis, increasing the risk of thrombotic events (11). Active smoking has been consistently linked to hypercoagulability, characterized by shortened PT and APTT and elevated fibrinogen levels. Passive smoking, while less studied, may also induce similar changes through chronic low-level exposure to these compounds, potentially contributing to cardiovascular risk (12). Understanding the differential impact of active versus passive smoking on these parameters is crucial for assessing the broader health implications of tobacco exposure in urban populations.

Kanpur, a major industrial city in India, represents a unique setting for studying the effects of tobacco exposure due to its high prevalence of smoking, significant air pollution, and socioeconomic factors that may exacerbate health disparities. Despite the known associations between smoking and adverse health outcomes, few studies have systematically compared the effects of active and passive smoking on hepatic enzyme levels and coagulation parameters in a population-based context. This study aims to address this gap by evaluating SGOT, SGPT, ALP, PT, APTT, and fibrinogen levels in active and passive smokers in Kanpur, providing insights into the comparative health impacts of direct and indirect tobacco exposure. Such data are essential for informing targeted public health interventions and policies aimed at reducing the burden of tobacco-related diseases in India.

2. MATERIALS AND METHODS

Study Design: This study employs a cross-sectional observational design to evaluate the effects of active and passive smoking on hepatic enzyme levels and coagulation parameters among residents of Kanpur, India. Data were collected at a single time point to simultaneously assess hepatic and coagulation markers in active and passive smokers, providing a snapshot of their physiological status in relation to tobacco exposure.

Ethical Approval: Ethical clearance was obtained from the Human Ethical Committee, CSJM University, Kanpur (HEC Reference no. 2024-Jun-006). All participants provided written informed consent prior to enrollment.

Study Population: The study population consists of adult residents of Kanpur, aged 20–40 years, categorized based on smoking exposure into two groups: active smokers and passive smokers.

Study Groups and Sample Size: The study enrolled a total of 200 participants, divided equally into two groups using a stratified random sampling method: 100 active smokers (Group A) and 100 passive smokers (Group B).

Selection Criteria: Participants were selected based on predefined inclusion and exclusion criteria. Adults aged 20–40 years, residing in Kanpur, India, for at least one year were included. Active smokers were defined as individuals smoking ≥1 cigarette daily for ≥6 months, and passive smokers were those exposed to secondhand smoke for ≥15 minutes twice weekly for one year, without active smoking history. Written informed consent was required. Exclusions included individuals <20 or >40 years, non-residents of Kanpur, those with cardiovascular diseases, liver disorders, diabetes mellitus, nephrotic syndrome, hypertension, or autoimmune diseases, ex-smokers with <1 year of cessation, pregnant women, and those unwilling to consent.

Data Collection Procedure: Data were collected using a structured questionnaire to capture demographic details, lifestyle factors, and tobacco exposure history. Blood samples were obtained via

venipuncture. Samples were collected in plain vials for hepatic enzyme analysis and sodium citrate vials for coagulation parameter assessment. Serum was separated by centrifugation at 3000 rpm for 10 minutes, aliquoted, and analyzed. Sample collection adhered to World Health Organization guidelines for biosafety and sample handling.

Analytical Methods: Hepatic enzyme levels and coagulation parameters were analyzed using standardized laboratory techniques. SGOT, SGPT, and ALP were measured using the Kinetic Enzymatic Assay based on the IFCC method, performed on the BioSystems A25 automated biochemistry analyzer. PT and APTT were assessed using a semi-automated coagulometer, while fibrinogen levels were determined via the Clauss method. All assays were conducted in a certified laboratory with internal and external quality control measures to ensure accuracy and reproducibility.

Statistical Analysis: Data were analyzed using IBM SPSS Statistics version 26.0. Descriptive statistics were reported as mean ± standard deviation (SD) for continuous variables. Normality of data distribution was assessed using the Shapiro-Wilk test. Differences in hepatic enzyme levels (SGOT, SGPT, ALP) and coagulation parameters (PT, APTT, fibrinogen) between active and passive smoker groups were evaluated using an independent samples t-test. A p-value <0.05 was considered statistically significant.

3. RESULTS

3.1. Gender Distribution of Active and Passive Smokers

In the active smoker group, comprising 100 participants, 77% (n=77) were male, and 23% (n=23) were female. In the passive smoker group, also consisting of 100 participants, 72% (n=72) were male, and 28% (n=28) were female. The active smoker group showed a marginally higher male predominance compared to the passive smoker group, as depicted in Figure 1.

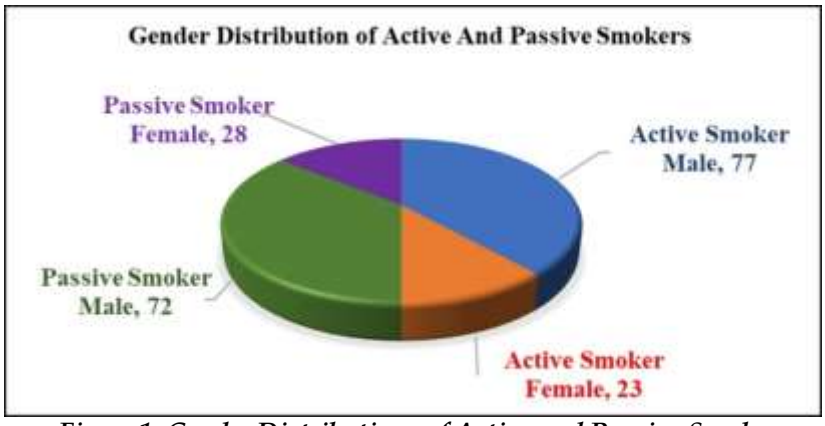

Figure 1: Gender Distributions of Active and Passive Smokers.

Gender-wise distribution of active and passive smokers. The pie chart shows a higher proportion of male smokers compared to females in both active and passive smoking groups.

3.2. Age Distribution of Active and Passive Smokers

The mean age of the active smoker group was 29.99 ± 3.59 years, with males averaging 30.27 ± 3.52 years and females 29.04 ± 3.74 years, indicating slightly older males with lower age variability than females. In the passive smoker group, the overall mean age was 28.57 ± 5.71 years, with males at 28.85 ± 5.60 years and females at 27.86 ± 6.04 years, suggesting that females were younger and had greater age variability than males. Overall, passive smokers displayed greater age variability than active smokers, as illustrated in Figure 2.

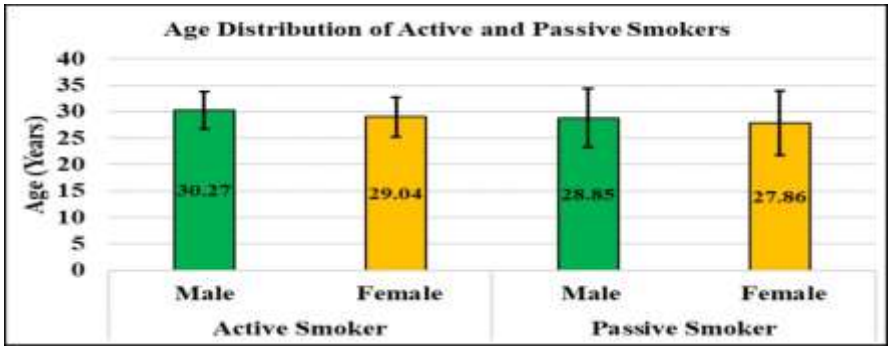

Figure 2: Age Distribution of Active and Passive Smokers.

Age distribution of active and passive smokers among males and females. The bars represent mean age (±SD), showing slightly higher mean age in active smokers compared to passive smokers.

3.3. Hepatic Enzyme in Active and Passive Smokers

The analysis of hepatic enzyme levels revealed significant differences between active and passive smokers. The mean serum glutamic-pyruvic transaminase (SGPT) level in active smokers was 96.64 ± 21.4 IU/L, significantly higher than the 21.86 ± 4.07 IU/L observed in passive smokers ($t = 34.32$, $p < 0.001$). Similarly, serum glutamic-oxaloacetic transaminase (SGOT) levels were markedly elevated in active smokers at 95.93 ± 20.97 IU/L compared to 21.35 ± 3.96 IU/L in passive smokers ($t = 34.95$, $p < 0.001$). Alkaline phosphatase (ALP) levels also showed a significant difference, with active smokers recording 160.5 ± 21.17 IU/L and passive smokers 86.75 ± 21.17 IU/L ($t = 34.26$, $p < 0.001$). These findings indicate that active smokers exhibit significantly higher hepatic enzyme levels compared to passive smokers, suggesting a greater degree of hepatocellular stress or damage associated with direct tobacco exposure, as illustrated in Table 1 and Figure 3.

Table 1: Hepatic Enzyme Levels in Active and Passive Smokers

Parameter	Active Smokers (Mean SD)	Passive Smokers (Mean SD)	t-value	p-value
SGPT (IU/L)	96.64 ± 21.4	21.86 ± 4.07	34.32	<0.001**
SGOT (IU/L)	95.93 ± 20.97	21.35 ± 3.96	34.95	<0.001**
ALP (IU/L)	160.5 ± 21.17	86.75 ± 21.17	34.26	<0.001**

All values are presented as mean ± standard deviation (SD). SGPT: Serum Glutamic-Pyruvic Transaminase; SGOT: Serum Glutamic-Oxaloacetic Transaminase; ALP: Alkaline Phosphatase; IU/L: International Units per Liter. An independent samples t-test was performed to compare hepatic enzyme levels between active and passive smokers. The t-value represents the t-test statistic, and the p-value indicates statistical significance. A p-value < 0.05 was considered significant; ** denotes highly significant results ($p < 0.001$).

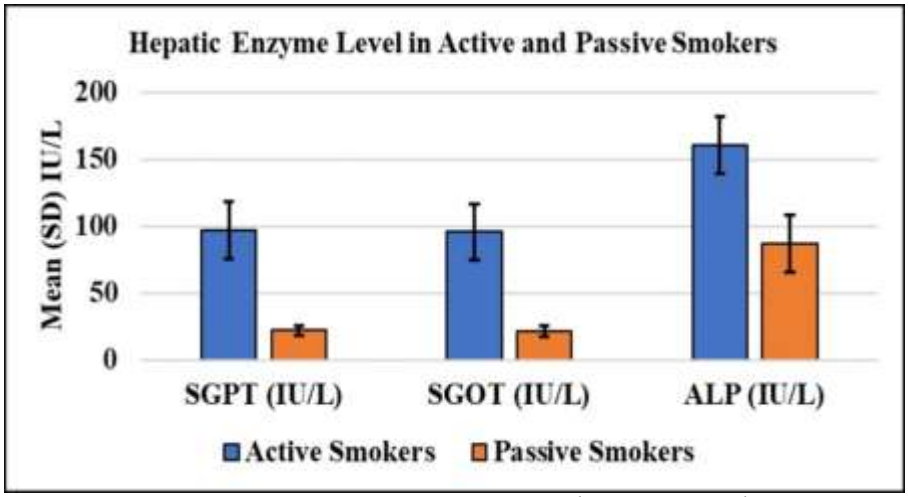

Figure 3: Hepatic Enzyme in Active and Passive Smokers.

Comparison of hepatic enzyme levels (SGPT, SGOT, and ALP) between active and passive smokers. Active smokers show significantly higher mean enzyme levels, indicating greater hepatic stress compared to passive smokers.

3.4. Coagulation Parameters in Active and Passive Smokers

The analysis of coagulation parameters revealed significant differences between active and passive smokers. Fibrinogen levels were markedly higher in active smokers, with a mean of 371.27 ± 21.97 mg/dL, compared to 282.55 ± 4.6 mg/dL in passive smokers ($t = 39.52$, $p < 0.001$), indicating a pronounced procoagulant state in active smokers. PT was slightly shorter in active smokers at 12.43 ± 0.67 seconds compared to 12.95 ± 0.65 seconds in passive smokers ($t = -5.65$, $p < 0.001$), suggesting faster clotting in the extrinsic pathway among active smokers. Similarly, APTT was significantly reduced in active smokers (mean 30.59 ± 1.81 seconds) compared with passive smokers (32.71 ± 1.81 seconds; $t = -8.28$, $p < 0.001$), reflecting enhanced intrinsic pathway activity. These findings indicate that active smoking is associated with a stronger hypercoagulable state compared to passive smoking, as evidenced by elevated fibrinogen

and shortened PT and APTT, as illustrated in Table 2 and Figure 4.

Table 2: Coagulation Parameters in Active and Passive Smokers

Parameter	Active Smokers (Mean SD)	Passive Smokers (Mean SD)	t-value	p-value
Fibrinogen (mg/dL)	371.27 ± 21.97	282.55 ± 4.6	39.52	<0.001**
PT (Sec)	12.43 ± 0.67	12.95 ± 0.65	-5.65	<0.001**
APTT (Sec)	30.59 ± 1.81	32.71 ± 1.81	-8.28	<0.001**

All values are presented as mean ± standard deviation (SD). PT: Prothrombin Time; APTT: Activated Partial Thromboplastin Time. An independent samples t-test was used to compare coagulation parameters between active and passive smokers. The t-value represents the t-test statistic, and the p-value indicates statistical significance. A p-value < 0.05 was considered significant; ** denotes highly significant results (p < 0.001).

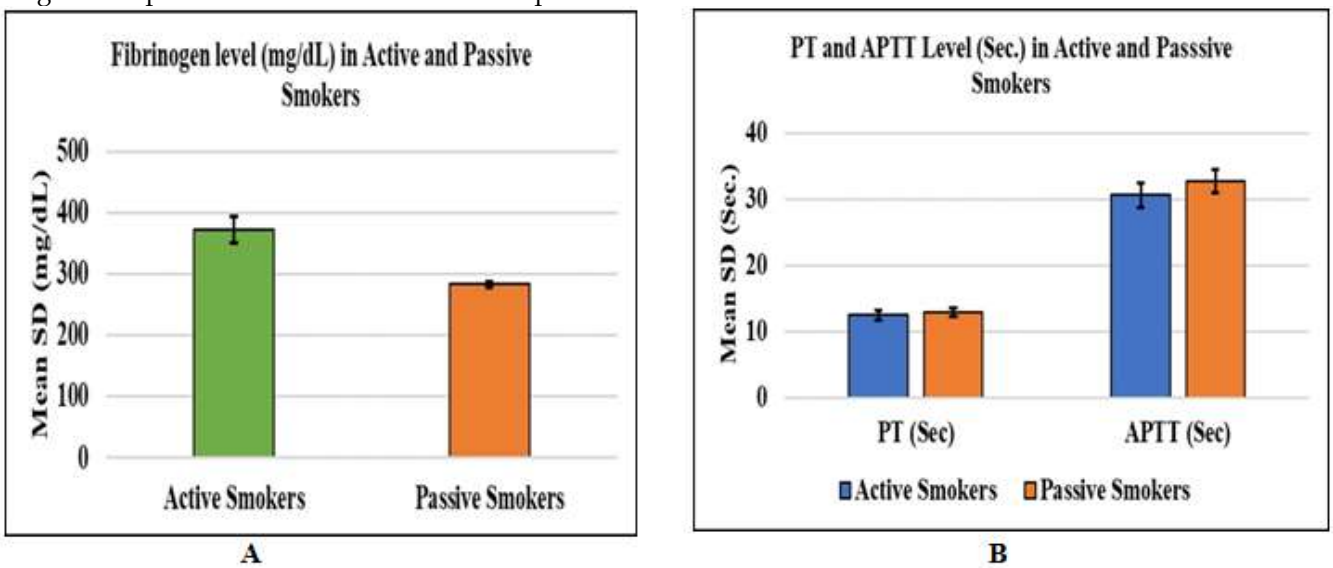

Figure 4: (a) Fibrinogen levels (mg/dL) in active and passive smokers; (b) Prothrombin Time (PT) and Activated Partial Thromboplastin Time (APTT) values (seconds) in active and passive smokers. Active smokers demonstrate higher fibrinogen levels and shorter PT and APTT, indicating a more pronounced hypercoagulable state.

4. DISCUSSION

The demographic profile of the study participants showed a male predominance in both active (77% male) and passive (72% male) smoker groups, reflecting the higher prevalence of smoking among males in India. This is consistent with the GATS India 2016–2017, which reported that 19% of adult males smoke compared to only 2% of females (13). The slightly higher male representation in active smokers aligns with cultural and social patterns in Kanpur, where male smoking rates are elevated due to socioeconomic and occupational factors. The mean age of active smokers (29.99 ± 3.59 years) was slightly higher than that of passive smokers (28.57 ± 5.71 years), with passive smokers showing greater age variability. This variability may reflect the broader exposure scenarios for passive smokers, including household and workplace settings, which affect a wider age range. These demographic patterns are comparable to those reported by Mishra et al. (2020) in a study of urban Indian populations, where active smokers were predominantly male and slightly older than passive smokers (14).

This study found significantly higher hepatic enzyme levels in active smokers compared to passive smokers in Kanpur, India, with SGOT (95.93 ± 20.97 vs. 21.35 ± 3.96 IU/L), SGPT (96.64 ± 21.4 vs. 21.86 ± 4.07 IU/L), and ALP (160.5 ± 21.17 vs. 86.75 ± 21.17 IU/L), showing marked differences (all p < 0.001). These elevations in active smokers suggest hepatocellular stress from tobacco smoke toxins, such as polycyclic aromatic hydrocarbons and reactive oxygen species. Passive smokers had milder elevations, above normal ranges (SGOT/SGPT < 40 IU/L, ALP 20–140 IU/L), indicating subclinical hepatic impact from secondhand smoke. Our findings align with those of Afroz et al. (2024), who reported higher SGOT, SGPT, and ALP levels in smokers, with greater elevations in heavy smokers (15). Similarly, Liu et al. (2013) linked heavy smoking to increased NAFLD risk (OR 2.29, 95% CI 1.30–4.03) and passive smoking to a 25% higher NAFLD risk in

women (OR 1.25, 95% CI 1.05–1.50) (16). However, Wannamethee and Shaper (2010) found smoking increased ALP but not AST after adjusting for confounders like alcohol and CRP, possibly due to differing population characteristics (17). Kanpur's high tobacco use and pollution may amplify these effects, emphasizing the need for smoking cessation interventions.

Active smokers exhibited markedly higher fibrinogen levels (371.27 ± 21.97 mg/dL vs. 282.55 ± 4.6 mg/dL, $p < 0.001$), shorter prothrombin time (PT) (12.43 ± 0.67 seconds vs. 12.95 ± 0.65 seconds, $p < 0.001$), and reduced activated partial thromboplastin time (APTT) (30.59 ± 1.81 seconds vs. 32.71 ± 1.81 seconds, $p < 0.001$) compared to passive smokers. The elevated fibrinogen levels in active smokers align with Hunter et al. (2001), who reported significantly higher fibrinogen synthesis rates in chronic smokers (22.7 ± 1.3 mg/kg/day) compared to non-smokers (16.0 ± 1.3 mg/kg/day, $p < 0.01$), with a strong correlation between plasma fibrinogen levels and synthesis rates ($r = 0.65$, $p = 0.04$) (18). Similarly, Jefferis et al. (2010) found higher fibrinogen levels in active smokers and a dose-dependent increase in passive smokers with higher serum cotinine levels, with differences between high and low cotinine levels being one-third to one-half those of active smokers (19). Our passive smokers' fibrinogen levels (282.55 ± 4.6 mg/dL), slightly above typical non-smoker ranges (200–400 mg/dL), support these findings, suggesting a modest procoagulant effect from secondhand smoke. The shorter PT and APTT in active smokers indicate enhanced extrinsic and intrinsic coagulation pathway activity, consistent with Das et al. (2024), who reported significantly reduced PT (11.56 ± 0.87 vs. 13.07 ± 0.77 seconds, $p < 0.001$) and APTT (28.42 ± 2.00 vs. 31.50 ± 1.00 seconds, $p < 0.001$) in smokers compared to non-smokers, with negative correlations between smoking duration and both parameters (20). Elkhalifa et al. (2018) also observed shorter PT in smokers (12.9 ± 1.2 vs. 13.7 ± 1.04 seconds, $p < 0.000$) but found no significant APTT difference, possibly due to population or methodological differences (21). Our results, showing shorter APTT in active smokers, align with Ahmad et al. (2015), who noted increased prothrombotic factors like tissue factor and von Willebrand factor in smokers, contributing to a hypercoagulable state (22). Passive smokers' PT and APTT values, within normal ranges (PT: 11.84–14.16 seconds; APTT: 29.60–35.70 seconds), suggest minimal impact, though slightly shorter times compared to non-smoker norms corroborate Jefferis et al. (2010), indicating subtle coagulation activation from secondhand smoke (19). The hypercoagulable state in active smokers, driven by elevated fibrinogen and shortened PT and APTT, underscores their increased cardiovascular risk, particularly in Kanpur's high-tobacco-use environment. Passive smokers' milder changes highlight the dose-dependent nature of tobacco exposure. These findings emphasize the need for smoking cessation interventions to mitigate thrombotic risks in urban Indian populations.

5. LIMITATIONS

This study has certain limitations. First, the cross-sectional study design restricts the ability to establish causal relationships between smoking exposure and the observed biochemical changes. Second, smoking status and passive smoke exposure were assessed partly through self-reporting, which may introduce recall or reporting bias. Third, the study sample was limited to a single urban setting in Kanpur, which may reduce the generalizability of the findings to other regions or populations.

6. CONCLUSION

In conclusion, active smokers in Kanpur exhibit significantly higher hepatic enzyme levels and a more pronounced procoagulant state compared to passive smokers, as evidenced by elevated SGOT, SGPT, ALP, and fibrinogen, and shortened PT and APTT. Passive smokers show milder alterations, indicating a dose-dependent effect of tobacco exposure. These findings align with prior research and highlight the need for targeted interventions to reduce both active and passive smoking in urban India to mitigate hepatic and cardiovascular risks.These findings also emphasize the need for stronger community-level tobacco control programs, public-health education, and policies promoting smoke-free environments in households, workplaces, and public spaces. Implementing such measures may help reduce both active and passive smoke exposure in urban populations. Future research using objective exposure biomarkers, such as serum cotinine levels, along with multicentric sampling, would further enhance the validity and generalizability of the results.

7. ETHICAL COMPLIANCE

The study received ethical approval from the Human Ethical Committee, CSJM University, Kanpur (HEC Ref. No.: 2024-Jun-006). Written informed consent was obtained from all participants. The authors declare no funding support and no competing interests. All data are included within the manuscript and available on request.

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Consent to Participate: - Yes
Consent to Publish: - Yes
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Competing Interests: No Competing Interests
Availability of data and materials: All data are available in the manuscript file.
Conflict of Interest- No conflict of interest

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