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A Comparative Assessment of Liver Function and Coagulation Profiles Among Active and Passive Smokers in Kanpur City

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ABSTRACT

Tobacco smoke exposure, both active and passive, has been implicated in various systemic effects, including hepatic dysfunction and altered coagulation. However, limited data exist on the comparative biochemical impact of active versus passive smoking, particularly in urban Indian settings like Kanpur. To assess and compare liver function tests, coagulation parameters between active and passive smokers. A cross-sectional observational study was conducted among 200 participants aged 20–40 years in Kanpur City, with 100 active smokers and 100 passive smokers. Fasting blood samples were analyzed for liver enzymes and coagulation parameters (PT, aPTT, fibrinogen). Data were statistically analyzed using SPSS, with a significance threshold of $p < 0.05$. Active smokers had significantly elevated ALT (48.6 ± 10.2 U/L), AST (52.1 ± 11.5 U/L), and GGT (65.3 ± 13.2 U/L) levels compared to passive smokers. Coagulation profiles showed a hypercoagulable state in active smokers with shorter PT (11.2 ± 0.9 s) and elevated fibrinogen (420.4 ± 45.3 mg/dL). Passive smokers also showed mild but significant abnormalities across most parameters. All differences were statistically significant ($p < 0.05$). Both active and passive smoking are associated with biochemical alterations affecting liver function and coagulation. While the changes are more severe in active smokers, passive smokers are not exempt from risk. These findings emphasize the need for broader screening, education, and public health interventions to address the risks of second-hand smoke exposure. These alterations represent early biological events that contribute to carcinogenesis, highlighting both active and passive smoking as preventable cancer risk factors.

Keywords: *active smoking; passive smoking; liver function tests; coagulation profile; fibrinogen; second-hand smoke; Kanpur*

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1. INTRODUCTION

Tobacco smoking remains one of the leading preventable causes of morbidity and mortality worldwide. It contributes significantly to a wide spectrum of systemic diseases, including cardiovascular, pulmonary, hepatic, and hematological disorders. Smoking has been shown to induce oxidative stress, systemic inflammation, endothelial dysfunction, and alterations in lipid metabolism and coagulation pathways [1]. While the direct health hazards of active smoking are well-established, increasing evidence also highlights the detrimental effects of passive or second-hand smoke exposure on various physiological systems, including liver function and coagulation profiles [2]. Importantly, these biochemical disturbances are also central to the process of carcinogenesis, as oxidative stress and chronic inflammation can drive DNA damage, impair repair mechanisms, and set the stage for malignant transformation.

Globally, tobacco use is responsible for over 8 million deaths annually, with more than 1.2 million attributed to non-smokers exposed to second-hand smoke [3]. India is the second-largest consumer of tobacco, with approximately 267 million users, out of which nearly 100 million are smokers [4]. According to the Global Adult Tobacco Survey-2 (GATS-2), about 28.6% of Indian adults use tobacco in some form, and 10.7% are exposed to second-hand smoke at home and public places [5]. Among Indian states, Uttar Pradesh shows a significant burden, with a smoking prevalence of approximately 15.4% among adult males and a high level of passive exposure reported in both urban and rural populations [6].

Recent epidemiological data underscore the emerging health risks associated with passive smoking, which has been linked to similar pathophysiological changes as seen in active smokers. Passive smoke exposure has been associated with liver enzyme elevation, altered lipid profiles, increased systemic inflammation, and pro-thrombotic states in both children and adults [7,8]. Despite this, routine clinical assessment and public health screening often overlook the biochemical alterations in passive smokers.

In India, studies assessing liver function and coagulation abnormalities in smokers are limited, and most of them focus primarily on active smokers. Research exploring these changes in passive smokers, particularly within northern Indian regions like Uttar Pradesh, remains sparse [9]. Kanpur, being an industrial and densely populated city, is prone to high levels of environmental tobacco smoke exposure, yet little is known about its biochemical impact on residents, especially those passively exposed.

This study aims to address this gap by assessing and comparing liver function and coagulation profiles among active and passive smokers in Kanpur City. The problem lies in the silent but progressive biochemical damage induced by second-hand smoke, which often goes undiagnosed until clinical disease manifests. Given the increasing burden of non-communicable diseases in India, it is imperative to identify early biochemical markers of risk, particularly in populations with involuntary exposure.

The justification for this study is rooted in the need for evidence-based data on the subclinical effects of smoking, especially passive smoking, to guide preventive and interventional strategies. The primary objectives are to evaluate liver function parameters and coagulation profiles in active and passive smokers and to determine the statistical significance of differences between the two groups.

The findings are expected to contribute valuable insights for clinical surveillance and public health policymaking in similar urban populations.

2. MATERIAL AND METHODOLOGY

This study was conducted as a cross-sectional observational study to compare liver function and coagulation profiles among active and passive smokers residing in Kanpur City. The target population included adults between 20 to 40 years of age who were permanent residents of Kanpur. Participants were categorized into two groups: active smokers with a documented history of smoking, and passive smokers with regular exposure to environmental tobacco smoke. Individuals were included based on age eligibility, residency, smoking exposure, and willingness to provide informed consent.

Participants were excluded if they had known cardiovascular, hepatic, psychiatric, or metabolic disorders, including diabetes mellitus, nephrotic syndrome, or hypertension. Additional exclusion criteria included pregnancy, substance abuse other than tobacco, use of medications affecting lipid or liver profiles, and smoking cessation less than one year before enrollment.

A total of 200 participants were recruited through stratified random sampling, with 100 individuals in each group. Each participant underwent a structured assessment, including lifestyle and health surveys, clinical evaluation, and laboratory investigations. A 5 ml fasting venous blood sample was collected from each participant. Samples were processed for coagulation profiles using a coagulation analyzer and liver using a fully automated biochemistry analyzer.

The primary variables assessed were liver function tests and coagulation parameters. Smoking status and demographic details served as independent variables. Data were analyzed using SPSS software. Descriptive statistics such as means,

standard deviations, and frequency distributions were calculated. Inferential statistics, including t-tests and ANOVA, were used to compare the biochemical parameters between the two groups, with a significance level set at $p < 0.05$.

3. RESULT

A total of 200 participants were included in the study, comprising 100 active smokers and 100 passive smokers. The mean age of participants was comparable between the two groups, with no significant demographic differences.

On evaluating liver function tests, active smokers demonstrated significantly elevated levels of serum alanine transaminase (ALT), aspartate transaminase (AST), and gamma-glutamyl transferase (GGT) compared to passive smokers ($p < 0.05$). Total bilirubin levels were also marginally higher in active smokers, while serum albumin showed no significant difference between the two groups.

Assessment of coagulation parameters showed shortened prothrombin time (PT) and elevated fibrinogen levels in active smokers, indicating a hypercoagulable state. Activated partial thromboplastin time (aPTT) was also reduced in active smokers compared to passive smokers, with all coagulation differences being statistically significant ($p < 0.05$).

Overall, the study demonstrated that active smokers have significantly more pronounced alterations in liver function and coagulation profile compared to passive smokers. However, passive smokers also exhibited subtle but clinically relevant deviations in these biochemical markers, underscoring the adverse health impact of second-hand smoke exposure.

Variable / Parameter	Active Smokers (n=100)	Passive Smokers (n=100)
Age Group (20–30 yrs)	54 (54%)	50 (50%)
Age Group (31–40 yrs)	46 (46%)	50 (50%)
Male	78 (78%)	70 (70%)
Female	22 (22%)	30 (30%)
Urban Residence	65 (65%)	60 (60%)
Rural Residence	35 (35%)	40 (40%)
Elevated ALT	62 (62%)	35 (35%)
Elevated AST	65 (65%)	38 (38%)
Elevated GGT	68 (68%)	41 (41%)
Shortened PT	55 (55%)	30 (30%)
Reduced aPTT	58 (58%)	33 (33%)
Elevated Fibrinogen	63 (63%)	38 (38%)

Table 1: Demographic and Biochemical Characteristics of Study Participants

Parameter	Active Smokers (Mean ± SD)	Passive Smokers (Mean ± SD)	p-value
ALT (U/L)	48.6 ± 10.2	36.4 ± 8.9	< 0.001
AST (U/L)	52.1 ± 11.5	39.3 ± 9.1	< 0.001
GGT (U/L)	65.3 ± 13.2	50.2 ± 11.6	< 0.001
Total Bilirubin (mg/dL)	1.21 ± 0.34	0.91 ± 0.28	0.012
Serum Albumin (g/dL)	4.12 ± 0.40	4.20 ± 0.36	0.287

Table 2: Comparison of Liver Function Profile parameters between Active and Passive Smokers

Parameter	Active Smokers (Mean ± SD)	Passive Smokers (Mean ± SD)	p-value
Prothrombin Time (PT) (s)	11.2 ± 0.9	12.0 ± 1.1	0.003
aPTT (s)	27.5 ± 2.6	30.1 ± 2.9	< 0.001
Fibrinogen (mg/dL)	420.4 ± 45.3	362.8 ± 41.2	< 0.001

Table 3: Comparison of Coagulation parameters between Active and Passive Smokers

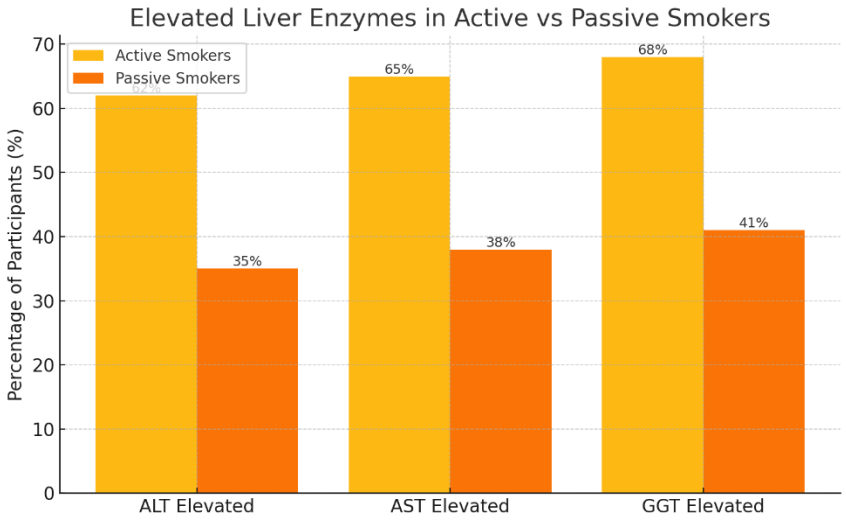

Figure 1: Liver Enzymes status among Active and Passive Smokers

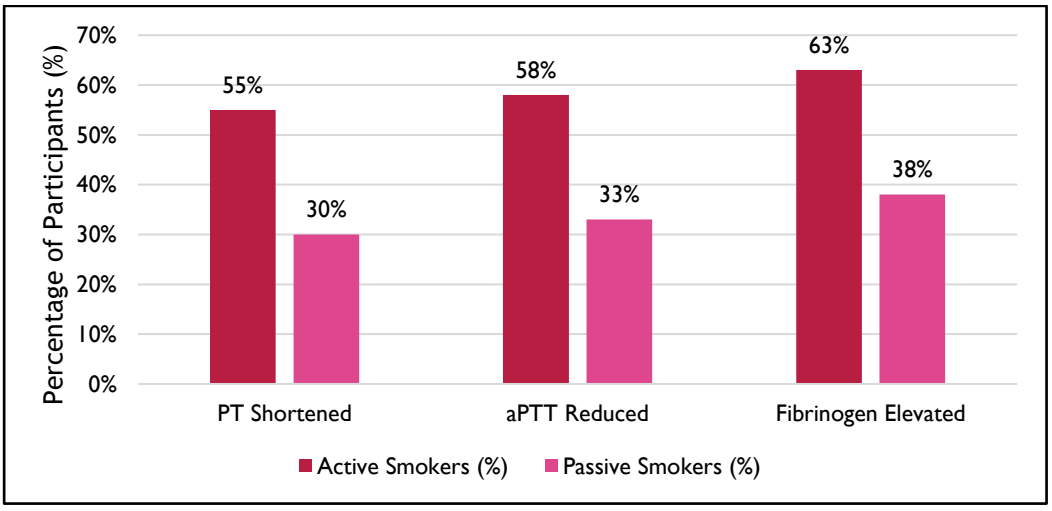

Figure 2: Coagulation Marker Abnormalities status among Active and Passive Smokers

4. DISCUSSION

Active and passive smokers showed considerable biochemical changes in the current study, with active smokers showing greater increases in liver enzymes and hypercoagulable profiles. Increased levels of ALT, AST, and GGT in active smokers (mean ALT: 48.6 ± 10.2 U/L; AST: 52.1 ± 11.5 U/L) relative to passive smokers support previous literature that smoking results in hepatocellular stress caused by oxidative effects [10,11]. Notably, chronic hepatocellular stress is known to play a role in carcinogenesis by causing oxidative DNA damage, malfunction of repair mechanisms, and facilitation of a pro-inflammatory microenvironment. All these processes develop a biological environment in which hepatocytes are prone to

malignant transformation.

The identified coagulation abnormalities, including decreased PT and aPTT and increased fibrinogen in smokers, also manifest a systemic pro-thrombotic state. Although these changes are known risk factors of cardiovascular diseases, they have implications in the pathogenesis of cancer. Hypercoagulability enhances the angiogenesis of the tumor, adhesion of the tumor cells, and metastatic potential. The high increase of fibrinogen level of passive smokers (362.8 ± 41.2 mg/dL) and more significantly in active smokers (420.4 ± 45.3 mg/dL) indicates that low levels of exposure to tobacco smoke could trigger biochemical pathways that predispose to carcinogenesis [12,13].

The evidence taken together supports the conclusion that active and passive tobacco exposure are not only harmful to systemic health but also are key early events in the multistage carcinogenesis process. DNA-level mutations or oncogenic pathways are not the direct focus of our study; however, the biochemical markers measured are critical surrogate markers of physiological stress caused by carcinogens. Our results, by emphasizing quantifiable biochemical alterations, offer evidence of the subclinical but important alterations that might precede malignant change, and thus provide a rationale behind the importance of preventive measures in populations at risk of tobacco smoke exposure.

5. CONCLUSION

As shown in this comparative study, active smokers have greater biochemical impairments in liver function and coagulation profiles than passive smokers; nevertheless, both groups manifest a substantial departure from normal physiology. Such results go beyond mere systemic toxicity: they are early harbingers of mechanisms that are pivotal to carcinogenesis, such as oxidative stress, hepatocellular damage, and pro-thrombotic conditions. Although the direct molecular pathways were not addressed in this study, the evidence is strongly in favor of including the role of active and passive smoking as a factor that contributes to carcinogenic risk.

The importance of regular biochemical monitoring, stringent population health policies on tobacco exposure, and increased awareness of the health hazards of second-hand smoke can be highlighted by the fact that these changes are the precursors in the carcinogenesis process. These primary changes are correctable, and preventive interventions can have the potential to alleviate the burden of tobacco-related carcinogenesis.

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