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INTENSITY OF SECONDHAND SMOKE EXPOSURE IS ASSOCIATED WITH SUBCLINICAL ELEVATION OF SERUM ALT LEVELS IN HEALTHY YOUNG ADULTS

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ABSTRACT

***Background**: Secondhand smoke exposure contains various toxic compounds that may affect liver function even in young, apparently healthy individuals. Evidence regarding the impact of exposure intensity on liver enzyme activity remains limited.*

***Objective**: This study aimed to analyze the association between secondhand smoke exposure intensity and serum alanine aminotransferase (ALT/SGPT) levels among university students.*

***Methods**: A cross-sectional study was conducted among 99 university students selected using purposive sampling. Exposure intensity was quantified using a structured questionnaire based on frequency and duration of exposure. Serum ALT levels were measured using an enzymatic method with an automated chemistry analyzer. Data were analyzed using Spearman's correlation test.*

***Results**: A significant positive correlation was observed between secondhand smoke exposure intensity and serum ALT levels (r = 0.293, p = 0.003). Although most ALT values remained within normal reference ranges, higher exposure intensity was associated with a gradual increase in ALT levels.*

***Conclusion**: Increased intensity of secondhand smoke exposure is associated with subclinical elevation of serum ALT levels in healthy young adults. These findings highlight the importance of minimizing passive smoke exposure even in non-smoking populations.*

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ABSTRAK

Latar Belakang: Paparan asap rokok pasif mengandung berbagai senyawa toksik yang berpotensi memengaruhi fungsi hati, bahkan pada individu usia muda yang secara klinis tampak sehat. Namun, bukti ilmiah mengenai pengaruh intensitas paparan terhadap aktivitas enzim hati masih terbatas.

Tujuan: Penelitian ini bertujuan untuk menganalisis hubungan antara intensitas paparan asap rokok pasif dan kadar serum alanine aminotransferase (ALT/SGPT) pada mahasiswa.

Metode: Penelitian ini menggunakan desain potong lintang (cross-sectional) dan melibatkan 99 mahasiswa yang dipilih dengan teknik purposive sampling. Intensitas paparan asap rokok diukur menggunakan kuesioner terstruktur berdasarkan frekuensi dan durasi paparan. Kadar serum ALT diperiksa menggunakan metode enzimatik dengan alat kimia klinik otomatis. Analisis data dilakukan menggunakan uji korelasi Spearman.

Hasil: Ditemukan hubungan positif yang signifikan antara intensitas paparan asap rokok pasif dan kadar serum ALT (r = 0,293; p = 0,003). Meskipun sebagian besar nilai ALT masih berada dalam batas rujukan normal, peningkatan intensitas paparan berkaitan dengan kenaikan kadar ALT secara bertahap.

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Kata kunci:
Paparan asap rokok pasif; Alanine aminotransferase; Fungsi hati; Perokok pasif; Dewasa muda

Kesimpulan: Peningkatan intensitas paparan asap rokok pasif berhubungan dengan peningkatan subklinis kadar serum ALT pada dewasa muda yang sehat. Temuan ini menegaskan pentingnya upaya meminimalkan paparan asap rokok pasif, bahkan pada populasi non-perokok.

INTRODUCTION

Secondhand smoke exposure remains a significant environmental health problem, especially among young people who do not smoke but are chronically exposed in their homes and public spaces **(Febiyanti et al., 2025)**. Cigarette smoke contains various toxic compounds, including nicotine, carbon monoxide, and free radicals, which can trigger oxidative stress and systemic inflammatory responses **(Maiyo et al., 2023)**. As the primary organ for metabolism and detoxification, the liver plays a critical role in neutralizing xenobiotic compounds. Consequently, chronic exposure to secondhand smoke may induce cellular stress in hepatocytes and impair liver function, even in the absence of overt clinical symptoms **(Mohajan, 2025)**.

Clinical and subclinical liver function can be evaluated through transaminase enzyme tests, particularly Serum Glutamic Pyruvic Transaminase (SGPT) or alanine aminotransferase (ALT), which are known as sensitive biomarkers for liver cell damage or stress **(Kalas et al., 2021)**. Elevated SGPT levels reflect hepatocyte membrane leakage due to inflammatory processes or oxidative stress **(Sudarwo & Hidayati, 2024)**. Several previous studies have reported increased SGPT levels in active smokers, indicating the toxic effects of cigarette smoke components on liver tissue **(Prambudi et al., 2023)**. However, most of these studies focused on active smokers and used a simple categorical approach, such as comparing smokers and non-smokers, without considering the variation in exposure levels received by individuals.

Toxic constituents of cigarette smoke, such as benzene and other aromatic hydrocarbons, are primarily metabolized in the liver by cytochrome P450 enzymes, particularly CYP2E1 and CYP1A1. During this biotransformation process, these enzymes generate reactive oxygen species (ROS) as by-products, leading to oxidative stress in hepatocytes when ROS production exceeds the cellular antioxidant capacity **(Hsb & Wulandari, 2021)**. Research specifically examining the impact of passive cigarette smoke exposure on liver function is still limited, especially that which uses a quantitative approach to assess cumulative exposure intensity. Several studies have compared passive smokers and moderate smokers without measuring exposure levels continuously, thus failing to describe the dose-response relationship between cigarette smoke exposure and changes in liver function biomarkers **(Afriayani et al., 2023)**. In addition, studies on clinically healthy young populations are still rare, even though in this group changes in liver function are thought to be subclinical and may escape detection if analyzed based solely on exposure status.

Based on this background, this study aims to analyze the relationship between the intensity of passive cigarette smoke exposure and SGPT levels in college students as a clinically healthy young adult population. The novelty of this study lies in the use of quantitative assessment of cigarette smoke exposure intensity based on frequency and duration of exposure, as well as its evaluation of subclinical changes in liver enzymes in young non-smokers. This approach is expected to provide a more comprehensive picture of the initial response of liver function to passive smoke exposure and serve as a scientific basis for efforts to prevent smoke exposure in educational environments.

METHODS

Study Design and Participants

This study employed a quantitative cross-sectional design with comparative and associative approaches to examine differences in serum alanine aminotransferase (ALT/SGPT) levels between students exposed to secondhand cigarette smoke and non-exposed controls, as well as to analyze the association between exposure intensity and ALT levels as an indicator of liver function. The study population consisted of 132 undergraduate students from Politeknik Indonusa Surakarta. A total of 99 participants were selected using purposive sampling, with sample size determined based on the Slovin formula at a 5% margin of error. Participants were classified into two groups: students exposed to secondhand smoke and non-smoking students as the control group. Inclusion criteria were students aged 18–25 years, apparently healthy, willing to participate by providing written informed consent, and not consuming medications known to affect liver function. Exclusion criteria included a history of liver disease, use of hepatotoxic drugs, alcohol consumption within the previous three months, or incomplete participation in study procedures.

Assessment of Secondhand Smoke Exposure

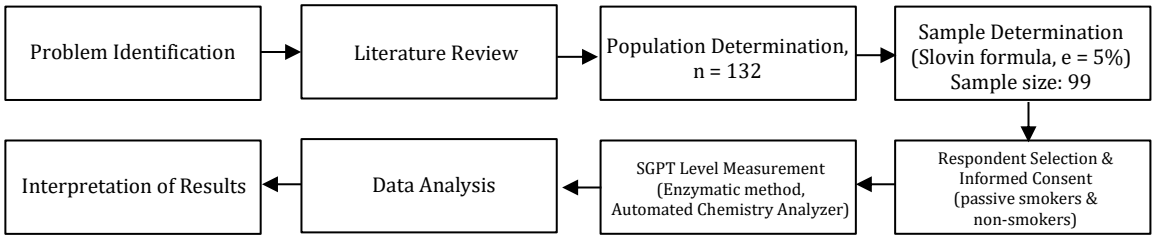
Secondhand smoke exposure was assessed using a structured questionnaire designed to collect information on the frequency and duration of exposure in both residential and non-residential environments. Exposure frequency and duration were scored and summed to generate a total exposure score representing the intensity of secondhand smoke exposure. The total score was analyzed as a continuous variable to evaluate the association between exposure intensity and ALT levels, and also categorized into exposure levels to support comparative analysis.

Laboratory Measurement of ALT (SGPT)

Venous blood samples were collected from the antecubital vein using sterile techniques and vacutainer tubes without anticoagulant. Samples were allowed to clot and centrifuged to obtain serum. Serum ALT levels were measured using an enzymatic method with an automated chemistry analyzer in accordance with standard laboratory operating procedures. All laboratory procedures were conducted by trained medical laboratory personnel using appropriate personal protective equipment to ensure accuracy and biosafety.

Statistical Analysis

Data analysis was performed using statistical software. Descriptive statistics were used to summarize participant characteristics, exposure intensity scores, and ALT levels. Data normality was assessed using the Kolmogorov–Smirnov test. Differences in ALT levels between exposure groups were analyzed using the Kruskal–Wallis test, while the association between secondhand smoke exposure intensity and ALT levels was evaluated using Spearman's rank correlation test. Statistical significance was set at a 95% confidence level.



ETHICAL CONSIDERATIONS

This study was conducted in accordance with the principles of research ethics and received approval from the Health Research Ethics Committee of Universitas Muhammadiyah Purwokerto (Approval No. KEPK/UMP/87/X/2025). Written informed consent was obtained from all participants prior to data collection.

RESULTS AND DISCUSSION

This study demonstrates a significant positive association between secondhand smoke exposure intensity and serum alanine aminotransferase (ALT/SGPT) levels among apparently healthy young adults, indicating the presence of early, subclinical alterations in liver enzyme activity related to environmental tobacco smoke exposure. Although categorical comparisons did not reveal statistically significant differences, quantitative continuous analysis identified a meaningful association between exposure intensity and ALT levels.

Respondent Characteristics

This study involved 99 student respondents aged 18–25 years, with a mean age of 19.3 years. All respondents were in relatively homogeneous health conditions, characterized by no history of liver disease, alcohol consumption, or long-term use of drugs that are potentially hepatotoxic. This homogeneity is important in liver function studies because these factors are known to independently affect serum glutamic pyruvic transaminase (SGPT) levels **(Iffaf, 2024)**.

Table 1. Respondent Characteristics

Characteristics	Category	n	%
Gender	Male	7	7.1
	Female	92	92.9
History of Liver Disease	No	99	100
Alcohol Consumtion	No	99	100
Regular Medication	No	99	100

The gender distribution shows a predominance of female respondents at 92.9% (n = 92), while male respondents accounted for 7.1% (n = 7). Despite the gender imbalance, all respondents met the inclusion criteria for the study and were in the young adult age group, which physiologically has relatively stable liver function. Thus, the SGPT levels obtained in this study can be assumed to represent liver function without the influence of major confounding factors.

Intensity of Cigarette Smoke Exposure

Cigarette smoke exposure was assessed using a total score system based on a combination of frequency and duration of exposure in the home and outside the home. The total exposure score ranged from a minimum of 2 to a maximum of 14, with an average score of 5.80. This score range is then used as the basis for grouping the intensity of cigarette smoke exposure into four categories, namely non-smokers (score ≤ 3), light passive smokers (score 4–6), moderate passive smokers (score 7–10), and heavy passive smokers (score ≥ 11). The distribution of respondents showed that most students were in the light passive smoking category at 45.5% (n = 45), followed by moderate passive smokers at 29.3% (n = 29), non-smokers at 18.2% (n = 18), and heavy passive smokers at 7.1% (n = 7). These findings indicate

that cigarette smoke exposure among students is generally indirect but occurs repeatedly and consistently. This quantitative approach to exposure provides methodological advantages over previous studies. Studies by **(Prambudi et al., 2023)** and **(Yasmeen, 2023)** classified exposure based on active smoker status and number of cigarettes per day, while the study by **(Afriayani et al., 2023)** compared moderate and passive smokers without measuring cumulative exposure intensity. Therefore, this study is able to describe cigarette smoke exposure as a spectrum of intensity that is more representative of environmental exposure conditions.

SGPT Level Overview

Table 1. Descriptive Statistics of SGPT Levels

Variable	Mean (U/L)	Median (U/L)	Minimum (U/L)	Maximum (U/L)
Up to SGPT	11.79	11.00	5	34

Laboratory test results show that the respondents' SGPT levels had an average value of 11.79 U/L with a median of 11.00 U/L, a minimum value of 5 U/L, and a maximum value of 34 U/L. In general, most SGPT values were within the normal reference range. However, the relatively higher maximum value indicates biological variation between individuals.

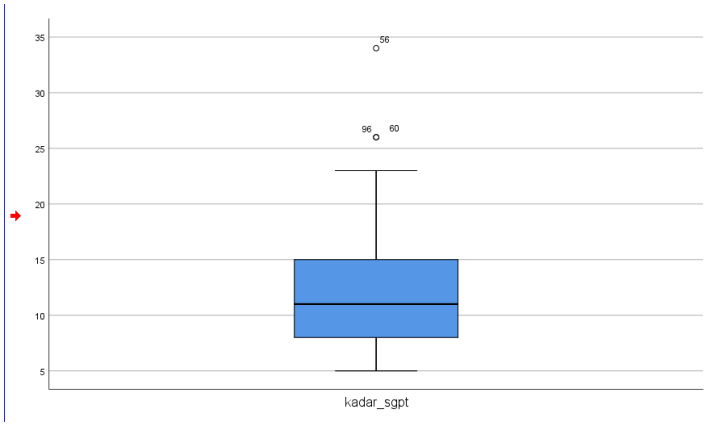


Figure 1. SGPT Level Boxplot

The boxplot visualization shows extreme values, indicating that some respondents experienced increased liver enzyme activity. Physiologically, increased SGPT levels reflect the release of enzymes from hepatocytes into the bloodstream due to cellular stress or minor disturbances in the liver cell membrane **(Ruslim et al., 2025)**. This finding is in line with previous studies stating that exposure to toxic substances in cigarette smoke, such as nicotine and free radicals, can trigger oxidative stress and inflammatory responses in liver tissue **(Labenz et al., 2022)**. However, unlike studies on active smokers, which generally demonstrate a more pronounced increase in SGPT levels, the present study indicates that even passive smokers may exhibit measurable changes in SGPT. These subclinical elevations likely reflect hepatocyte membrane instability, whereby increased membrane permeability allows the leakage of intracellular enzymes such as ALT into the circulation in the absence of overt hepatocellular necrosis.

Statistical Analysis and Data Distribution

Table 2. Kolmogorov-Smirnov Normality Test

Variable	Kolmogorov–Smirnov Statistics	Sig. (p)	Description
Up to SGPT	–	.000	Abnormal

The Kolmogorov–Smirnov normality test showed that the distribution of SGPT levels did not follow a normal distribution ($p = 0.000$; $p < 0.05$). Therefore, further statistical analysis was performed using nonparametric tests. This method was chosen in accordance with statistical analysis principles to maintain the validity and reliability of the research results.

Differences in SGPT Levels Based on Exposure Categories

Table 3. Kruskal–Wallis Test Results for SGPT Levels

Variable	Number of Groups	Statistics H	Sig. (p)
Up to SGPT	4	–	.110

The Kruskal–Wallis test result of $p = 0.110$ shows that there is no statistically significant difference in SGPT levels between categories of cigarette smoke exposure intensity ($p > 0.05$). This finding indicates that categorical exposure analysis is unable to detect significant differences in SGPT. The variation in SGPT levels between groups is still within the normal physiological range.

Relationship between Cigarette Smoke Exposure Intensity and SGPT Levels

Tabel 4. Results of Spearman's Correlation Test between Exposure Scores and SGPT Levels

Variable	Correlation Coefficient (r)	Sig. (p)	Description
Exposure Score – SGPT Level	.293	.003	Significant

In contrast, Spearman's correlation analysis demonstrated a weak but statistically significant positive correlation between secondhand smoke exposure intensity and SGPT levels ($r = 0.293$; $p = 0.003$). This finding indicates that when exposure is evaluated as a continuous variable, increasing frequency and duration of cigarette smoke exposure are associated with a small yet consistent elevation in SGPT levels, reflecting a measurable subclinical hepatic response rather than a pronounced biochemical alteration.

The results of this study indicate a positive and significant relationship between the intensity of passive cigarette smoke exposure and serum alanine aminotransferase (ALT/SGPT) levels in clinically healthy students. These findings indicate an early biological response in liver function due to environmental smoke exposure, even though most ALT values are still within the normal reference range. This suggests that changes in liver function can occur at a subclinical stage before the appearance of obvious clinical manifestations.

Biologically, cigarette smoke contains various toxic components, including nicotine, carbon monoxide (CO), and free radicals, which, once inhaled, enter the systemic circulation and are metabolized in the liver as the primary detoxification organ **(Shabah et al., 2023)**. Repeated exposure to these compounds can trigger oxidative stress in hepatocytes through increased production of reactive oxygen species (ROS) that exceeds the capacity of the cellular antioxidant system **(Gusnirwan & Sangging, 2024)**. This condition causes a disturbance in intracellular redox balance and increased permeability of liver cell membranes, allowing the release of ALT into the circulation without massive cell damage or hepatocyte necrosis. Therefore, the increase in ALT in passive smokers tends to be mild but consistent, reflecting subclinical impairment of liver function. It is important to note that ALT values that are still within the normal reference range do not necessarily indicate the absence of biological effects. In the context of environmental health, small but consistent increases in biochemical biomarkers can serve as an early warning system for the effects of long-term exposure **(Lestari et al., 2021)**. These findings have important implications for early prevention and

public health, as they indicate that exposure to secondhand smoke continues to affect young individuals who appear physiologically healthy.

Compared to studies on active smokers, the increase in ALT in the passive smoking group in this study was indeed milder. Previous studies have reported that active smokers tend to have higher ALT levels and more frequently exceed normal limits due to more intense and direct exposure to toxins. However, this study confirms that in passive smokers, the biological effects remain evident even though they are subclinical. The uniqueness of this study lies in its focus on a healthy young adult population and the use of an intensity-based quantitative approach, which has proven to be more sensitive than a categorical approach in detecting biological relationships with ALT levels.

CONCLUSION

This study demonstrates a significant positive correlation between the intensity of secondhand smoke exposure and serum alanine aminotransferase (ALT/SGPT) levels among healthy young adults. Although most enzyme levels remained within the established normal reference range, a higher intensity of exposure—determined by frequency and duration—was associated with a gradual elevation in ALT activity. These findings suggest an early subclinical response in liver function triggered by environmental tobacco smoke. Consequently, this research confirms that secondhand smoke exposure intensity is a critical environmental determinant of liver health, even in clinically healthy non-smoking populations.

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