

## Keywords

Smoking, Liver enzymes, SGPT/ALT, SGOT/AST, Prosthodontic treatment planning, Restorative dentistry, Dental risk assessment

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# Biochemical Effects of Smoking and Their Clinical Significance in Prosthodontic and Restorative Treatment Outcomes

## Abstract

**Objectives;** Cigarette smoking is a clinically relevant risk factor in dentistry because it may influence systemic metabolism, tissue healing, inflammatory response, and medication safety. This study aimed to compare serum glutamate pyruvate transaminase/alanine aminotransferase (SGPT/ALT) and serum glutamate oxaloacetate transaminase/aspartate aminotransferase (SGOT/AST) levels between smokers and non-smokers attending a private dental clinic, and to interpret the clinical significance of these biochemical changes for prosthodontic and restorative dental treatment planning.

**Methods;** A comparative cross-sectional study was conducted among 120 adult male dental patients aged 18–40 years. Participants were divided into two groups: smokers (n = 60) and non-smokers (n = 60). Case histories, smoking status, and smoking severity were recorded. Smokers were categorized as mild, moderate, or severe according to daily cigarette consumption, and smoking exposure was estimated using the Brinkman Index. Venous blood samples were collected, and SGPT/ALT and SGOT/AST levels were estimated using an automated Integra 400 Plus analyser. Data were analysed using IBM SPSS version 25.0, with P < 0.05 considered statistically significant.

**Results;** Smokers showed significantly higher mean SGPT/ALT levels than non-smokers,  $69.38 \pm 19.42$  versus  $16.38 \pm 7.04$ . Similarly, mean SGOT/AST levels were higher among smokers,  $64.09 \pm 20.30$ , compared with non-smokers,  $19.53 \pm 5.05$ . Both differences were statistically highly significant (P < 0.001).

**Conclusions;** Smoking was associated with significantly elevated liver enzyme markers among dental patients, suggesting possible smoking-related hepatic stress.

**Clinical Relevance;** Assessment of smoking history and liver-related biochemical status may support safer medication planning, risk assessment, smoking cessation counselling, and individualized prosthodontic and restorative dental care.

## 1. INTRODUCTION

Smoking of cigarettes is a significant modifiable risk factor with systemic and oral health effects. It releases the body to several toxic chemicals which could impact on a number of organs and biological methods. Cigarette smoking has been reported as having an effect on liver markers in the serum, suggesting that smoking could affect hepatic biochemical status [1]. Smoking in dental practice is important due to its association with oxidative stress, poor vascular response, delayed wound healing, poor host defence, periodontal breakdown and poor prognosis in various treatment settings. These effects are especially important in prosthodontics and restorative dentistry where the treatment's success

relies not just with the restorative material and clinical technique, but also with patient's biological ability to heal tissue, manage inflammation, tolerate their medications, and maintain them long-term.

Cigarette smoking affects the body by damaging the cells with toxic chemicals, including: Nicotine, Carbon. These compounds can lead to elevated oxidative stress and inflammation and cause cell injury. The aryl hydrocarbon receptor has been suggested to be an important pathway for responses to oxidative stress and toxicological regulation [2]. Smoke contains polycyclic aromatic hydrocarbons that are able to bind to receptor mediated pathways controlling xenobiotic metabolism in liver cells [3]. The activity of the cytochrome P450 enzymes could also be altered by smoking, which might affect the metabolism of drugs, toxins, and endogenous compounds [4]. Thus, smoking can be a cause of hepatic metabolic burden and change in liver enzyme markers like serum glutamate pyruvate transaminase/alanine aminotransferase, SGPT/ALT, serum glutamate oxaloacetate transaminase/aspartate aminotransferase, SGOT/AST.

The liver has a pivotal function in detoxification, glycogen storage, xenobiotic metabolism, drug biotransformation and regulates systemic biochemical homeostasis. Changes in liver enzymes can be a marker of some form of liver stress or hepatocellular damage. Smoking also may interact with other systemic risk factors such as alcohol exposure and there are previous reports of synergistic effects of smoking and alcohol consumption on serum gamma-glutamyl transferase levels [5]. Moreover, oxidized plasma proteins have been found to be linked to cigarette smoking dose which further corroborates the role of cigarette smoking in systemic oxidative damage [6]. The mechanisms are significant in dental medicine since many of the drugs used in dentistry such as analgesics, anti-inflammatory drugs, antibiotics, sedatives, and local anaesthesia adjuncts are metabolised partially or completely in the liver.

The intensity and duration of cigarette smoking is clinically relevant as it is probable that cumulative exposure can affect systemic risk. Modelling studies demonstrate that the risk of smoking-related diseases depends upon the total amount of smoking and the intensity of smoking [7]. In young adults, interventions for smoking cessation highlight that smoking is a chronic, behaviour and health issue that needs clinical management and structure [8]. While there has been some research investigating smoking and systemic diseases including cancer, respiratory disease, cardiovascular function and pulmonary outcomes [9–11], the biochemistry of smoking in the context of a patient seeking dental treatment is relatively understudied.

This is a clinically relevant problem in prosthodontics and restorative dentistry. Careful systemic evaluation may be necessary in patients that require some form of fixed or removable prostheses, implant supported restorations, extensive restorative treatment, endodontic-restorative management, periodontal-restorative procedures, or surgical crown-lengthening procedures. Biochemical alteration caused by smoking may affect the healing response, drug planning, postoperative recovery and risk assessment prior to

Monoxide, Tar, Aldehydes, Free Radicals, and Polycyclic Aromatic Hydrocarbons. complex dental rehabilitation.

While it is widely acknowledged that smoking is a risk factor for oral and systemic disease there is little focus in clinical settings on biochemical screening markers in dental patients prior to any prosthodontic and/or restorative procedures. Hence, the present study aimed to compare the SGPT/ALT and SGOT/AST levels of smokers and non-smokers attending the private dental clinic and interpreted the results with regard to prosthodontic and restorative treatment planning. The aim of the present study was therefore to assess the alterations in liver enzyme markers in serum of smokers visiting the dental clinic, and to interpret the clinical significance for prosthodontic and restorative treatment planning. Specifically, the study was designed to compare the SGPT/ALT and SGOT/AST levels of smokers and non-smokers who attended a private Dental Clinic, to assess the distribution of smoking severity among smokers and to discuss the possible clinical relevance of biochemical changes associated with smoking for Dental treatment. The significance of the changes in liver enzyme markers for medication management, healing, systemic risk evaluation, and individual patient decision-making prior to prosthodontic and restorative procedures were emphasized.

## 2. MATERIALS AND METHODS

### 2.1 Study Design and Setting

A comparative cross sectional study was carried out in patients visiting a private dental clinic for routine dental treatment. This study aimed to compare the selected biochemical parameters of liver function with smokers and non-smokers and interpret the clinical significance of these parameters in the context of prosthodontic and restorative dental treatment planning. An evaluation of smoking parameters may be relevant prior to undertaking complex restorative, prosthodontic-restorative, endodontic-restorative, periodontal-restorative or implant-related dentistry procedures since smoking may have effects on systemic metabolism, tissue healing, inflammatory response and drug biotransformation.

### 2.2 Participants

A total of 120 adult male patients aged 18–40 years were included in the study. There were two groups of participants, smokers (n = 60), and non-smokers (n = 60). Data pertaining to history, demographic information, medical history, dental history, smoking, and lifestyle-related information was obtained for all subjects and recorded as case histories. The two groups were chosen from the patients who visited the same private dental clinical environment for dental treatment. The smoking group consisted of people who had smoked for about 4-5 years. The non-smoker group comprised subjects who have not smoked nor been exposed to passive smoking at some point in their lives. The groups were compared with regard to age and

biochemical liver enzyme markers.

### 2.3 Inclusion Criteria

Patients visiting dental clinic in the private sector were included if they were adults male aged between 18-40 years, willing to participate and gave informed consent for the dental treatment. Individuals were included if they had no known systemic disease or medical history which might have influenced liver enzyme activity. Smokers were retained if they had smoked for around 4–5 years. The non-smokers included were those who had never smoked and were not exposed to passive smoking.

### 2.4 Exclusion Criteria

Subjects with known liver disease or any disease that might impact liver enzyme levels were excluded. Patients with systemic diseases, muscle injuries, recent infections, alcohol abuse, drug abuse that could affect liver function, passive smoking and heavy exercise prior to blood sampling were excluded. Liver function markers were excluded from the analysis of participants with dietary or lifestyle factors that could affect liver function markers. Such exclusion criteria were used to minimise confounding factors which could independently affect SGOT/AST and SGPT/ALT levels.

### 2.5 Assessment of Smoking Status and Smoking Severity

The smokers and non-smokers were divided according to case-history and self-reported smoking history. Smokers were then broken down by number of cigarettes smoked daily. Mild smokers were defined as those who smoke less than 2 cigarettes a day. Moderate smokers were defined as those who consumed more than 2 cigarettes a day but not 10. Smokers who smoked over 10 cigarettes a day were considered severe smokers. The Brinkman Index was also used to estimate exposure to smoking. The Brinkman Index was calculated by multiplying the duration of smoking in years by the number of cigarettes smoked per day:

**Brinkman Index = Duration of smoking in years × Number of cigarettes smoked per day**

During the present study, smokers' smoking duration was about 4–5 years. Thus, smoking exposure was primarily interpreted from cigarette consumption per day and the Brinkman

Index values were calculated in this range of smoking-duration.

### 2.6 Biochemical Assessment

Blood samples were withdrawn from each of the participants from the veins to estimate the liver enzyme markers. The blood was collected in 2.0ml of venous blood in aseptic conditions. Serum glutamate oxaloacetate transaminase/aspartate aminotransferase (SGOT/AST) and serum glutamate pyruvate transaminase/alanine aminotransferase (SGPT/ALT) were estimated by using automated Integra 400 Plus analyser. SGOT/AST and SGPT/ALT were chosen as biochemical parameters indicating hepatocellular stress

and hepatic metabolic disturbances. The purpose of this study was to determine if the smokers who were part of this study had different liver enzyme levels than non-smokers, in a dental clinic setting.

### 2.7 Relevance to Prosthodontic and Restorative Dental Treatment Planning

The biochemical findings were discussed in relation to dentistry prosthodontics and its restoration as liver function may have an influence on the metabolism of some of the drugs frequently used in dentistry such as analgesics, anti-inflammatory drugs, antibiotic drugs, sedative and drugs used during the peri-operative care of dental patients. Smoking related biochemical alteration may also be involved in clinical risk assessment as smoking is related to oxidative stress, defective tissue response, impaired wound healing and alteration in host defence.

In this study, SGOT/AST and SGPT/ALT were not directly used as measures of outcomes of prosthodontic or restorative treatment. On the contrary, they were seen as biochemical risk factors that could alert the clinician to the need to thoroughly assess the medical history and carefully plan medications, smoking cessation counseling and medical referral prior to extensive prosthodontic, restorative, periodontal-restorative, endodontic-restorative, and/or implant-related therapy for any smoker.

## 3. RESULTS

In the study, we enrolled 120 adult male dental patients. There were two groups, smokers and non-smokers, of 60 each. The following are the demographic distribution, the severity of smoking and the liver enzyme marker levels.

### 3.1 Participant Characteristics

Table 1 shows the frequency distribution of the participants according to their age. The age group with the greatest number of participants was aged 31-40 years, with 62 (51.6%) participants. The age group 21–30 years was the next largest with 44 participants (36.7%) while 14 participants (11.7%) were in the age group 18–20 years.

Among smokers, 38 participants, 63.3%, belonged to the 31–40-year age group, 18 participants, 6.7% were in the 18-20 year age group, 30.0% were in the 21-30 year age group and 4 participants (6.7%) were in the 18-20 age group. Of non-smokers, 24 participants (40.0%) were from the age group 31–40; 26 participants (43.3%) were from the age group 21–30; and 10 participants (16.7%) were from the age group 18–20.

The mean age of smokers was  $31.90 \pm 5.83$  years, while the mean age of non-smokers was  $28.43 \pm 7.11$  years. The mean ages of smokers and non-smokers were statistically different with  $t = 2.919$  and  $P = 0.003$ . Table shows that older individuals were smokers as compared to non-smokers in this study. Age distribution of smokers and non-smokers dental patients (adult males). The majority of participants were in the 31–40-year age group. The mean age was higher in smokers compared to non-smokers and the

difference was significant.

**Table 1. Age wise distribution of patients**

Age in years	Smokers		Non-smokers		Total	
	No.	%	No.	%	No.	%
18-20	4	6.7	10	16.7	14	11.7
21-30	18	30.0	26	43.3	44	36.7
31-40	38	63.3	24	40.0	62	51.6
Total	60	100.0	60	100.0	120	100.0
Mean ± SD	31.90 ± 5.83		28.43 ± 7.11		30.17 ± 6.71	
P-value	t = 2.919 P = 0.003, S					

**3.2 Smoking Severity**

The distribution of smokers according to smoking severity is shown in Table 2. Of the 60 smokers, 19 (31.7%) were classified as mild smokers, 31 (33.3%) as moderate smokers and 21 (35.0%) as severe smokers. The distribution of this group is such that there were people in it who had varying amounts of cigarette exposure per day. A greater percentage of severe

smokers indicates that there is likely a large number of smokers with greater exposure to tobacco daily, which may be clinically significant when considering systemic biochemical risk prior to treating with prosthodontics and restorative dentistry. Smokers by number of cigarettes per day. The distribution of the smoker group was nearly equal among all the groups (mild, moderate and severe).

**Table 2: Categorization of smokers based on smoking severity**

Categories	Number of smokers	Percentage
Mild (<2 cigarettes/day)	19	31.7%
Moderate (2–10 cigarettes/day)	20	33.3%
Severe (>10 cigarettes/day)	21	35.0%
Total	60	100.0%

**3.3 Comparison of Liver Enzyme Markers Between Smokers and Non-Smokers**

The comparison of the levels of SGPT/ALT and SGOT/AST between the smokers versus non-smokers is shown in Table 3 and graphically shown in figure 1. The mean SGPT/ALT level was higher among smokers than non-smokers. Smokers showed a mean SGPT/ALT value of 69.38 ± 19.42, compared with 16.38 ± 7.04 among non-smokers. This was a statistically very significant difference (P < 0.001). Similarly, the mean SGOT/AST level was higher among smokers than non-smokers. Smokers showed a mean SGOT/AST value of 64.09 ± 20.30, compared with 19.53 ± 5.05 among non-smokers. This difference was also statistically significant (P < 0.001).

These results showed a significant increase of liver enzymes in smokers in the dental clinic compared to non-smokers. The increased SGPT/ALT and SGOT/AST levels were observed, indicating a possible smoking-related liver stress or changes in biochemical status. These findings could be clinically relevant in pre-treatment assessment, medication planning, smoking cessation counselling and risk assessment for extensive dental rehabilitation procedures in the field of prosthodontic and restorative dentistry. An analysis of SGPT/ALT and SGOT/AST levels between smokers and the non-smokers. Liver enzyme markers were significantly increased with smoking as compared to non-smoking, and were quite significant.

**Table 3. Comparison of SGPT and SGOT levels between smokers and non- smokers**

Variables	Number	Smokers	Non-smokers	t-test value, P-value & significance
		Mean ± SD	Mean ± SD	
SGPT	60	69.38± 19.42	16.38± 7.04	t = 13.687, P <0.001, HS
SGOT	60	64.09± 20.30	19.53± 5.05	t = 13.687, P <0.001, HS

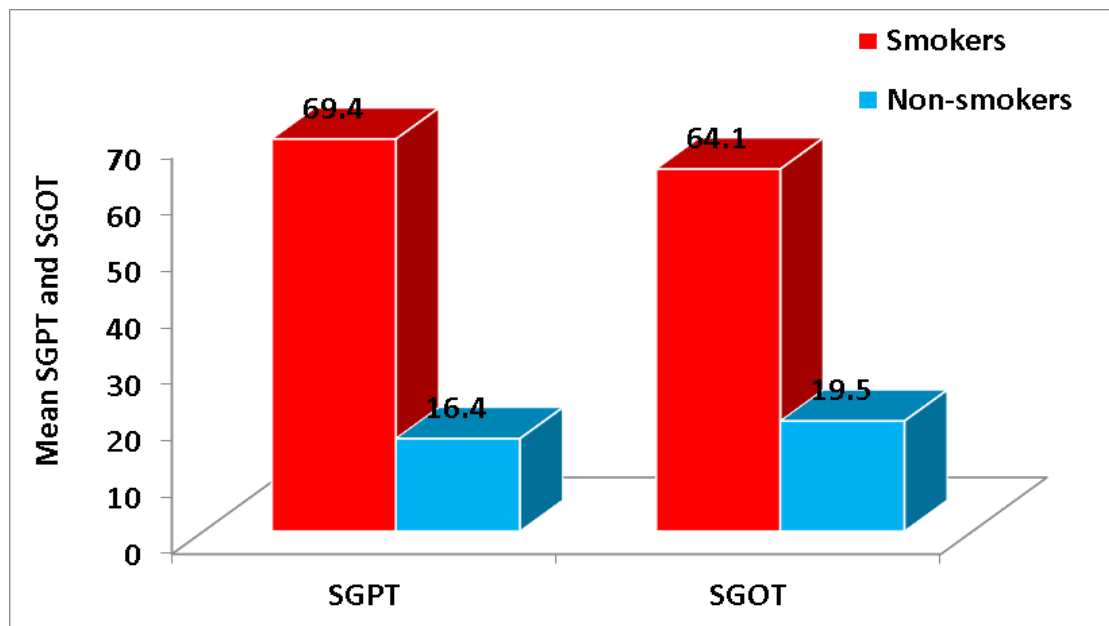


Figure 3. Age distribution of smokers and non-smokers

The number of smokers and non-smokers by age are displayed for each in multiple bar diagram. The age ranges of the highest proportion of smokers (31-40 years) and non-smokers (31-40 years) were also similar.

#### 4. DISCUSSION

The current study was conducted to assess the biochemical effect of smoking, where the SGPT/ALT and SGOT/AST were compared between smokers and non-smokers visiting the private dental clinic. It was observed that the smokers had elevated liver enzyme levels compared to non-smokers, implying that cigarette smoking may be linked to stress in the liver or alteration in the systemic biochemical condition. For the clinical relevance of prosthodontics and restorative dentistry, it is important to consider not only the oral condition but also the systemic health of the patients. In many cases, smoking is a behavior that is sustained and determined by factors such as dependence, duration, past experiences with quitting, and abstinence [12]. It is, therefore, important to include smoking status as a vital component of the patient's case history, particularly when undergoing extensive restorative treatment, prosthodontics, implant dentistry, periodontal-restorative treatment, and endodontic-restorative treatment. There is evidence from other medical disciplines that smoking influences the behavior of diseases. Thus, smoking can be regarded as a systemic risk factor rather than just a behavioral one. Smoke from cigarettes is laden with numerous toxins, such as nicotine, carbon monoxide, aldehydes, free radicals, and polycyclic aromatic hydrocarbons. This could result in oxidative stress, inflammation, cell damage, and abnormal metabolism. Through combined studies of chemistry, toxicology, and clinical medicine, it has been established that emissions of these toxins by cigarette smoke are crucial in increasing smoking-related risks [14]. It follows that the increased SGPT/ALT and SGOT/AST values found in smokers in the current study may be attributed to hepatic stress

resulting from smoking. Studies conducted on smoking abstinence have also demonstrated that sustained abstinence can cause changes in the body [15]. The clinical importance of these results in dentistry is primarily related to risk assessment and therapy planning. The liver is engaged in the metabolism of various drugs that are routinely prescribed by dentists such as analgesics, anti-inflammatory drugs, antibiotics, sedatives, and other supportive drugs. However, an increase in SGPT/ALT and SGOT/AST does not necessarily preclude the initiation of prosthodontic and restorative treatment. Nevertheless, a thorough examination of the medical history, prudent prescription, and consultations with a physician may be recommended. Outcomes of patients treated by means of root canal and surgical endodontic procedures have recently been considered a valuable factor [16]. Periodontal stability is another important factor that affects the success of restorative and prosthodontic treatment procedures. Studies have demonstrated that maintenance has an effect on the survival of teeth that are periodontally compromised, which means that periodontal stability is very crucial in determining the prognosis of any dental procedure [17]. In addition, because smoking can affect the vascularity, immunity, and healing of the tissue, it might be necessary to have a more rigorous periodontal assessment as well as maintenance before and after the restorative and prosthodontic procedures. Modern dental literature has highlighted the negative impact of smoking on dental structures [18]. Apical periodontitis after endodontic-restorative procedures is still an important issue in clinical practice, and systemic or behavioral risk factors may affect the healing process and prognosis [19]. Likewise, social behaviors have been found to affect the clinical success of CAD/CAM ceramic restorations, suggesting that the patient factor should be considered during ceramic and prosthodontic restoration planning [20]. Thus, the current biochemical results provide additional systemic information about the risks associated with

smoking and dental health.

However, the current study must be taken cautiously. The study did not evaluate prosthodontic or restorative treatment results such as success rate of implants, restorative failure, mucosa healing, prosthetic complications, periodontal condition, or endodontic healing. On the other hand, SGPT/ALT and SGOT/AST were employed to indicate a potential systemic change associated with smoking using biochemical methods. In general, the current study demonstrates that patients who visit dental clinics as smokers might have elevated markers of liver enzymes. Such results emphasize the importance of assessing smoking behavior, biochemical knowledge, conservative medication management, smoking cessation, and risk evaluation prior to any complicated prosthodontic or restorative procedures.

## 5. CONCLUSION

It was seen in the current study that the levels of SGPT/ALT and SGOT/AST were markedly higher in smokers as opposed to non-smokers attending a private dental clinic. This implies that cigarette smoking may be related to biochemically induced stress on the liver in dental patients. It can be stated that the elevated level of enzymes is indicative of some sort of metabolic change in the functioning of the liver which is important in dental treatment. It should be noted that this study did not evaluate prosthodontic/restorative treatment results. The role of the liver in the metabolism of various medicines used in the practice of dentistry, such as analgesics, anti-inflammatory medicine, antibiotics, sedatives, and other medicines that can be used as adjuvant therapy, needs to be taken into account. Hence, the patients who smoke and suffer from altered liver enzyme levels will need proper evaluation of their medical history and appropriate medication. In the field of prosthodontics and restorative dentistry, the effects of smoking on biochemistry can also be important for the assessment of the healing process, risks of surgery or restoration, periodontal health, endodontic-restorative prognosis, implants, and maintenance in the long run. The integration of smoking history and smoking severity and biochemistry assessment in pre-treatment evaluation may contribute to safer prosthodontics and restorative treatment.

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