Nonsmoker, negative smoker, e-cigarettes smoker and traditional smoker: Comparative effects on gingival health (Cross-sectional study)

A Research Project

Submitted to the College of Dentistry, AL-Mustaqbal university in Partial Fulfillment of the Requirements for the B.D.S. Degree in Dentistry

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Certification of the supervisor

I certify that this project entitled "Nonsmoker, negative smoker, e-cigarettes smoker and traditional smoker: Comparative effects on gingival health (Cross-sectional study)**"** was performed by fifth-grade students (Ghyath Hadi Mohammed-husain, Mohammed Shareef Atyah, Ali Ammar Ibrahim, Ghalib Atyia Hilal, Ibrahim Hamza Nyfe, Karar Sami Ali) under supervision of college of dentistry / Al-mustaqbal University in partial fulfillment of the graduation requirements for the Bachelor Degree in dentistry.

Supervisor signature

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Date : / / 2026

Dedication

We dedicate this work to our families for their unwavering support, to our supervisor Dr. Zaid Mohannad Al-Aboodi for his invaluable guidance, and above all to Allah, the Almighty, who granted us the knowledge and strength to seek the truth.

Acknowledgement

We thank and pray for almighty Allah who blessed us with knowledge, time, strength, patient, patients & everthing come to our minds.

We thank the prophet Mohammed (صلى الله عليه و اله و سلم) and all-al Bait (عليهم الصلاة و السلام)

Our special thanks go to Al-Mustaqbal University, **Phd. athraa yahya mohammed** and its dental training clinics for providing the facilities and environment that made this study possible.

We would like to express our sincere gratitude and appreciation to our supervisor, **Dr. Zaid Mohannad Al-Aboodi**, for his continuous guidance, valuable advice, and unwavering support throughout this research.

We are also deeply grateful to all 121 patients who participated in this study, particularly the 80 individuals who completed both visits, for their cooperation and trust.

Finally, we acknowledge the efforts of all team members for their dedication and teamwork in conducting the clinical examinations and procedures.

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List of Abbreviations

Abbreviation	Full Term
BOP	Bleeding on Probing
GI	Gingival Index
PI	Plaque Index
PDL	Periodontal ligament
ABP	alveolar bone proper
RC	root cementum
ACF	alveolar crest fibers
HF	horizontal fibers
OF	oblique fibers
APF	apical fibers
CEJ	cementoenamel junction
ENDS	electronic nicotine delivery devices
CPI	Community Periodontal Index
CAL	clinical attachment loss
IQR	interquartile ranges

List of Comparisons

Comparison Type	Description
Intra-group Comparison (Before vs. After)	Statistical significance of changes for each group (Smokers, Vapers, Passive Smokers, Non-Smokers).
Inter-group Comparative Analysis	Comparison of recovery levels between groups (Active Smokers, Non-Smokers, Vapers, Passive Smokers).
Baseline Assessment	Inter-Group Comparison at the start of the study for BOP, GI, and PI.
Efficacy of Intervention	Comparison of scaling and polishing results across all cohorts.
Clinical Outcome Comparison	Nonsmoker vs Smoker vs Vaper vs Negative Smoker.

Abstract

Background: Periodontal diseases are prevalent oral health conditions worldwide, with cigarette smoking established as a major environmental risk factor. Nicotine-induced vasoconstriction in smokers masks clinical signs of inflammation, leading to delayed diagnosis. Electronic cigarettes (e-cigarettes) have gained popularity as purported "healthier alternatives," yet emerging evidence suggests they also harm periodontal tissues through oxidative stress, DNA damage, and delayed wound healing. Understanding how different nicotine delivery systems affect periodontal healing following non-surgical therapy is essential for evidence-based clinical decision-making.

Aim: This study compared the healing response following scaling and root planning among three patient groups: conventional cigarette smokers, e-cigarette users (vapers), and negative smokers (individuals never exposed to smoking), to determine which group demonstrates the most favorable clinical outcomes.

Methods: A prospective clinical study was conducted involving 121 patients aged 18–40 years diagnosed with localized gingivitis, recruited from Al-Mustaqbal University's dental training clinics. Following application of inclusion/exclusion criteria, 80 patients completed both study visits. Clinical parameters assessed at baseline and 14 days post-treatment included: Bleeding on Probing (BOP), Gingival Index (GI), Plaque Index (PI), and Calculus Index. One examiner performed baseline measurements (examiner A) for BOP, while a second examiner (examiner B) for Gingival Index and a third one (examiner C) for Plaque Index to limit inter-operator variation. All patients received professional scaling and polishing after the initial examination. Non-parametric statistical tests (Kruskal-Wallis H-test for inter-group comparison; Wilcoxon signed-rank test for intra-group comparison) were employed due to non-normal data distribution confirmed by Shapiro-Wilk testing.

Results: All four groups (including a passive smoker subgroup) demonstrated statistically significant clinical improvement at 14 days ($p < 0.001$ for all groups). Baseline values showed no significant differences across groups for BOP ($p = 0.548$), GI ($p = 0.684$), or PI ($p = 0.449$), confirming homogeneity. Post-treatment analysis revealed significant inter-group differences in healing response ($p < 0.001$). Active smokers paradoxically showed the greatest BOP reduction (11.57%) and lowest post-treatment BOP (6.16%), yet exhibited the smallest GI reduction (0.0061), indicating nicotine-induced vasoconstriction masking true inflammation rather than reflecting genuine tissue repair. Non-smokers demonstrated the most robust true healing with the highest GI reduction (0.0145, $p < 0.001$). Vapers and passive smokers showed significantly impaired outcomes (BOP reductions of 7.06% and 7.40%, respectively). PI reduction was uniform across all groups ($p = 0.322$ post-treatment), confirming that mechanical cleaning efficacy is independent of smoking status.

Conclusions:

Nonsmoker is the best clinical outcome while smoker may show false improvements due to nicotine's vasoconstrictive masking of inflammation.

Introduction

periodontium health: a state free from inflammatory periodontal disease such as gingivitis, periodontitis, and other periodontal conditions. addition to the patients without a history of periodontal diseases, periodontal health can be applied to patients who had a history of successfully treated periodontal diseases and are able to maintain periodontal tissues without clinical inflammation-[4]

characteristic of periodontium:

in nonsmoker; Color attached and free gingiva range from brown, orange to pink. textured surface similar to that of an orange peel and is referred to as stippled, the attached gingiva firm and resilient and, with the exception of the movable free margin, tightly bound to the underlying bone [4]

in smoker; gingiva appears pale, dull, or whitish due to reduces blood flow to the gingival tissues [6] demonstrates a firm, fibrotic consistency [9] and Loss of stippling is commonly observed [8]

in vape smoker; texture, the use of vapes has been linked to the development of hyperkeratotic lesions of the oral mucosa. For color, while some evidence suggests conventional cigarettes cause more significant discoloration, the nicotine in vapes can still lead to pigmentation changes in the gums and staining of the teeth. [42] [43] [44] [45] [46] [47] [48]

in negative smoker; Normal gingival blood flow results in the characteristic pink color and the presence of bleeding on probing, Consistency of gingiva Firm, resilient, and often stippled attached gingiva [4] [5] [8] [9].

secondhand smoke exposure occurs in indoor environments, with homes and vehicles being the primary sources [40]. Children and adolescents are particularly vulnerable, as they spend long hours in these environments and lack the autonomy

to avoid exposure [37]. Furthermore, smoke can easily travel between rooms and even between apartments, rendering simple ventilation ineffective [38].

We used bleeding on probing, gingival and plaque indices for this research

Bleeding on probing index alarm us how much inflammation are we dealing with.

Gingival index shows the state of gingiva (color, texture, consistency.. etc.)

Plaque index can give to many information:

1- Severity of plaque accumulation on teeth and gingiva (sub and supragingival)
2- Quantity of plaque.
3- Parten of brushing (in case of a patient do not brush the lingual surface of teeth or don not brush at all)

Aim of study

This paper will compare the recovering response following scaling and polishing in four patient groups: nonsmoker, conventional smokers, e-cigarette users (vapers), and 'negative smokers,' to determine which group demonstrates the most favorable clinical outcomes but we should know the healthy state of periodontium to have a solid base for comparison.

Objectives

1. To assess the effect of smoking on periodontal tissues by measuring BOP, GI and PI.

2. To evaluate and compare the response of periodontal tissues between smokers, non-smokers and negative smokers to non-surgical procedures.

Chapter 1

1.1 The periodontium health

The periodontium (peri = around, odontos = tooth) comprises the following tissues: (1) gingiva, (2) perio dontal ligament, (3) root cementum, and (4) alveolar bone proper Fig 1-1. The latter lines the alveolus of the tooth and is continuous with the alveolar bone; on a radiograph it can be called lamina dura. The alveolar process that extends from the basal bone of the maxilla and mandible consists of the alveolar bone and the alveolar bone proper. [5]

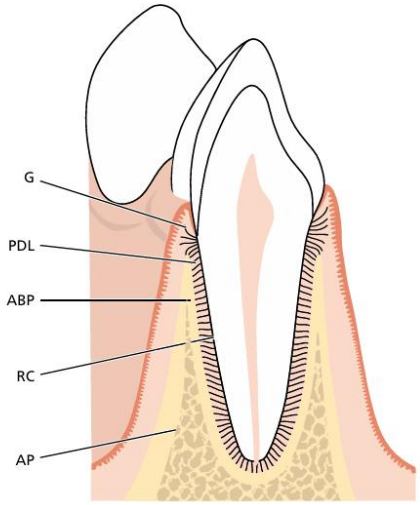


Fig 1-1 a tooth and its periodontal tissue consisting of gingiva(G), periodontal ligament (PDL), alveolar bone proper (ABP) and root cementum (RC). AP alveolar process (clinical periodontology and implant dentistry…2022)

Gingiva

The oral mucosa is continuous with the skin of the lips and the mucosa of the soft palate and pharynx. The oral mucosa consists of: (1) the masticatory mucosa, (2) the specialized mucosa and (3) the lining mucosa. [5] (Fig 1-2)

Fig 1-2 the gingiva (Lindhe J…2022)

---Periodontal ligament (PDL)

The periodontal ligament is the soft, richly vascular and cellular connective tissue that surrounds the roots of the teeth and joins the root cementum with the socket wall (Fig 1-3). (clinical periodontology and implant dentistry…2022)

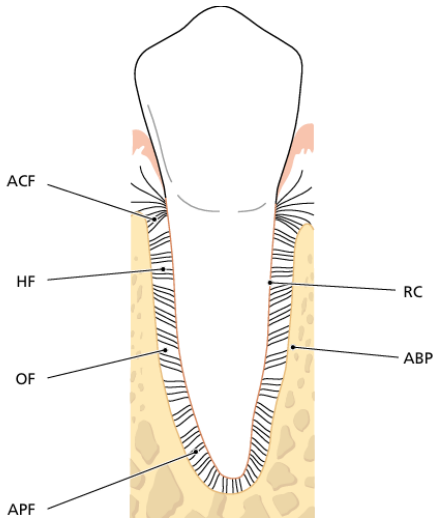


Fig 1-3 This schematic drawing illustrates how the periodontal ligament is situated between the alveolar bone proper (ABP) and the root cementum (RC) and indicates the groups of collagen fibers that join the tooth to the surrounding bone. From coronal to apical, these groups of fibers constitute the alveolar crest fibers (ACF), the horizontal fibers (HF), the oblique fibers (OF), and the apical fibers (APF).(Lindhe J …2022)

---Cementum

Calcified, avascular mesenchymal tissue that forms the outer covering of the anatomic root. The two main types of cementum are acellular (primary) and cellular (secondary) cementum. [1]

---The alveolar process

is the portion of the maxilla and mandible that forms and supports the tooth sockets (alveoli) It forms when the tooth erupts [2] (fig 1-4).

Fig 1-4 the alveolar process (NEWMAN AND CARRANZA'S…2024)

These components function together to support the teeth and absorb mechanical forces during chewing. In healthy non-smokers, these tissues exhibit normal vascularization, minimal inflammation, and a balanced microbial profile [3].

periodontal health defined by newman and carranza's[4] to

a state free from inflammatory periodontal disease such as gingivitis, periodontitis, and other periodontal conditions. addition to the patients without a history of periodontal diseases, periodontal health can be applied to patients who had a history of successfully treated periodontal diseases and are able to maintain periodontal tissues without clinical inflammation.

1.2 characteristic of healthy periodontium

-**Periodontal biotype**: thin scalloped, thick flat & thick scalloped [4] (Fig 1-5)

-Color: attached and free gingiva range from brown, orange to pink results from the colors of the vascular supply, the thickness and degree of keratinization of the epithelium, and the pigment-containing cells. The color correlates with the cutaneous pigmentation. [4]

Fig 1-5 the periodontal biotype

A) thin scalloped, B) thick flat C) thick scalloped (NEWMAN AND CARRANZA'S …2024)

Mobility: periodontal ligament space is about 0.2 mm, considerable variation exists Which means very little movement for normal tooth (the space increase when tooth subjected to hyperfunction).

texture: textured surface similar to that of an orange peel and is referred to as *stippled* the attached gingiva is stippled; the marginal gingiva is not (fig1.6). The

central portion of the interdental papillae is usually stippled, but the marginal borders are smooth. The pattern and extent of stippling vary among individuals and among different areas of the same mouth. Stippling is less prominent on lingual than facial surfaces and may be absent in some persons. Stippling varies with age. It is absent during infancy, it appears in some children at about 5 years of age, it increases until adulthood, and it frequently begins to disappear during old age. (4)

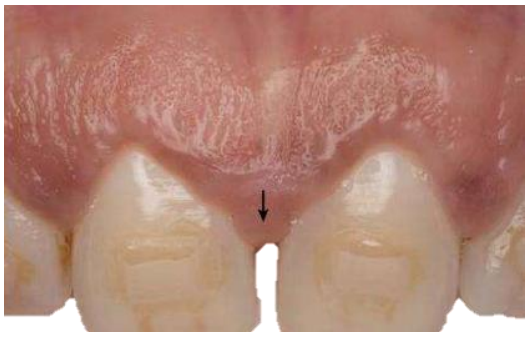


Fig 1-6 the marginal gingiva is smooth while the attached gingiva is stippled (NEWMAN AND CARRANZA'S …2024)

Width of keratinized tissue: width of attached gingiva + width of free gingiva

Width of attached gingiva

measured from the projection on the external surface of the bottom of the gingival sulcus or the periodontal pocket to the mucogingival junction (Fig 1-7) on the facial aspect It is generally greatest in the incisor region (i.e., 3.5 to 4.5 mm in the maxilla, 3.3 to 3.9 mm in the mandible) and narrower in the posterior segments (i.e., 1.9 mm in the maxillary 1rst premolars and 1.8 mm in the mandibular 1rst premolars) (4)

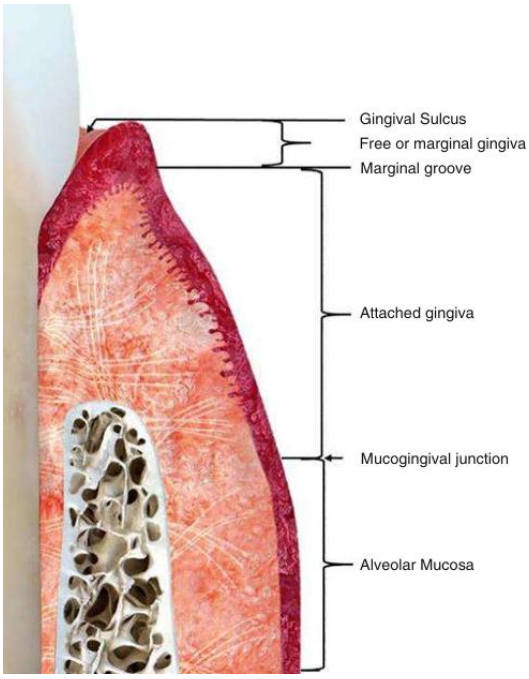


Fig 1-7 Diagram showing the anatomic landmarks of the gingiva. (NEWMAN AND CARRANZA'S …2024)

width of free gingiva

measured from free gingival groove to gingival zenith (most apical point of the marginal gingival) [4]

-consistency: The gingiva is firm and resilient and, with the exception of the movable free margin, tightly bound to the underlying bone. [4]

-Depth of gingival sulcus: ideal conditions 0mm or close to 0mm *while* the depth that consider normal in humans is 2 to 3 mm (without inflammation). [4]

-Interdental space: filled with papilla (no black space) governed by the contour of the proximal tooth surfaces and the location and shape of the gingival embrasures. [5]

-Shape of interdental papilla: pyramidal or col (depends on the presence or absence of a contact point between the adjacent teeth, the distance between the contact point and the osseous crest and the presence or absence of some degree of recession) ([5] (Fig 1-8)

Fig 1-8 the inter dental papilla (NEWMAN AND CARRANZA'S …2024)

-Coverage (by free gingiva) of tooth's crown: the length of free gingiva is 2 to 3 mm . [4]

-Bleeding on probing: no bleed on genital probing or less than 10%. [4]

-bone height: The presence of bone loss is identified based on the level of the interradicular alveolar crest in relation to the cementoenamel junction (CEJ) interradicular alveolar crest should be located at 0.5–2.0 mm apically to the CEJ level of adjacent teeth. (5)

1.3 Clinical Characteristics of Gingiva in Smokers

Smoking is considered one of the most significant environmental risk factors affecting periodontal tissues. Its impact is not limited to the progression of periodontal diseases but also extends to altering the clinical appearance and biological behavior of the gingiva. The gingival tissues in smokers often present with distinct clinical characteristics that may mask the severity of underlying periodontal destruction, making diagnosis and treatment more challenging.

Gingival Color Changes

One of the most noticeable clinical features of gingiva in smokers is the alteration in gingival color. Unlike the normal coral pink appearance observed in healthy non-smokers, the gingiva of smokers often appears pale, dull, or whitish (fig2-9). This discoloration is primarily attributed to nicotine-induced vasoconstriction, which reduces blood flow to the gingival tissues. As a result, the typical inflammatory redness seen in gingivitis may be absent even in the presence of plaque accumulation and periodontal breakdown (6)

Additionally, chronic smoking may lead to melanin pigmentation of the gingiva, commonly referred to as smoker's melanosis. This pigmentation is more prevalent in individuals with darker skin tones and is considered a protective response of melanocytes against tobacco-related toxins (7)

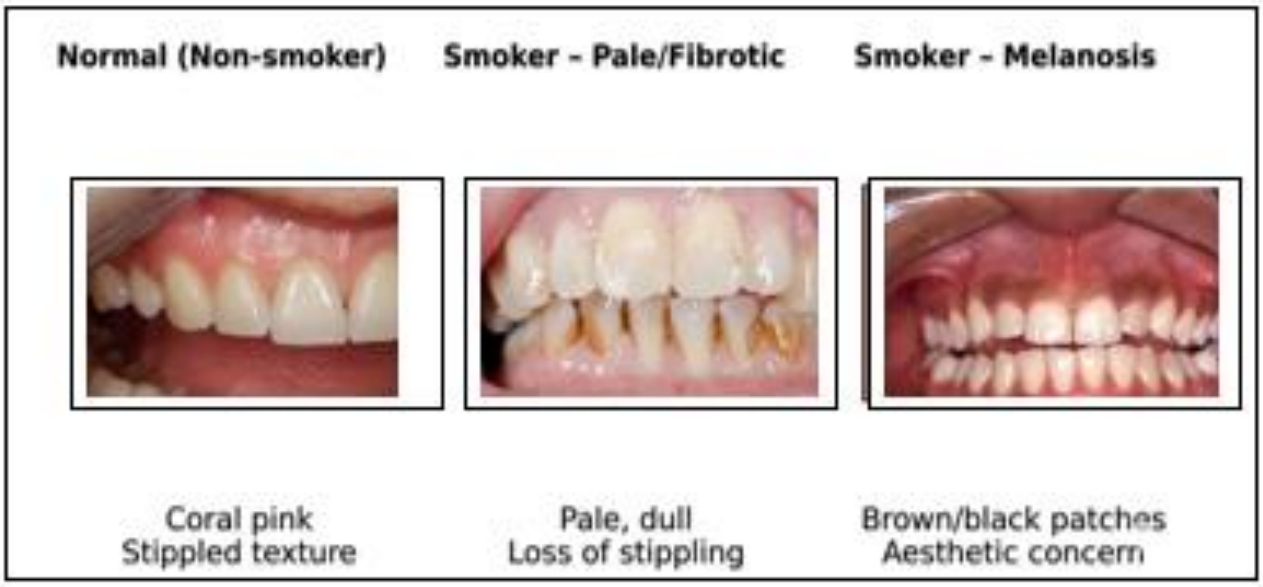


Fig.1-9 one view show melanin pigmentation that caused aesthetic problems in a 21-year-old woman

Gingival Thickness and Biotype

Smoking has a notable influence on gingival thickness and periodontal biotype. Studies have shown that smokers tend to exhibit a thicker gingival biotype compared to non-smokers (fig 1-10). This increase in thickness is believed to result from chronic exposure to tobacco smoke, which stimulates epithelial hyperplasia while simultaneously impairing normal connective tissue turnover [(9)].

Despite appearing clinically firm and fibrotic, this thickened gingiva does not indicate health. On the contrary, the dense connective tissue may limit visible signs of inflammation and reduce gingival bleeding, thereby concealing active periodontal disease. This phenomenon often leads to underestimation of disease severity during routine clinical examination.

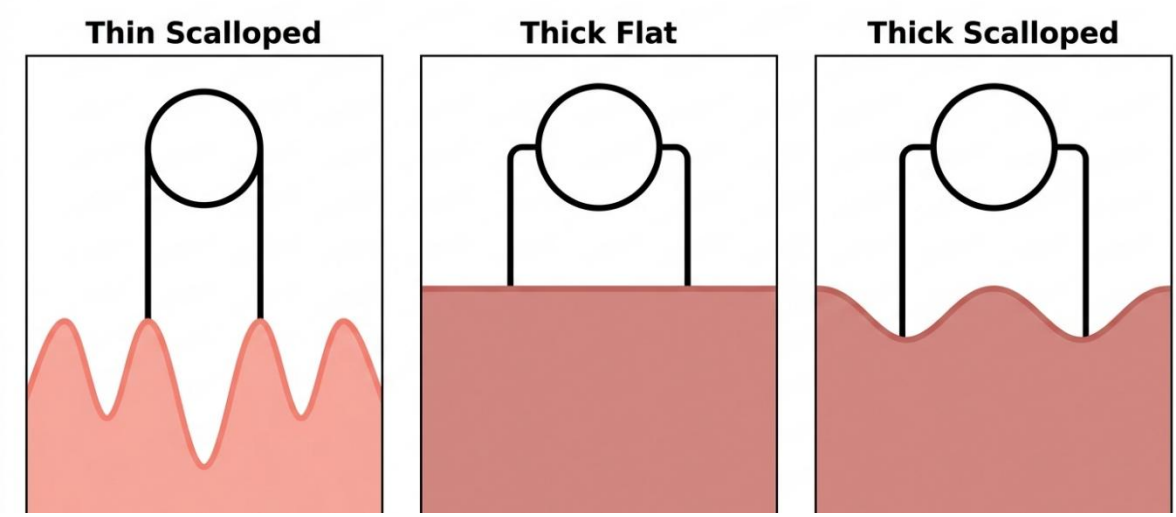


Fig. 1-10 gingival biotype.

Gingival Consistency and Texture

The gingiva in smokers typically demonstrates a firm, fibrotic consistency rather than the resilient and stippled texture seen in healthy gingiva. Chronic exposure to nicotine affects fibroblast function, reducing collagen synthesis and impairing normal tissue remodeling. Consequently, the gingival tissues may appear rigid and less elastic [(8)]

Loss of stippling is commonly observed, and the gingival surface may appear smooth and shiny. These changes reflect altered epithelial keratinization and compromised connective tissue integrity, which negatively affect the gingiva's ability to respond to bacterial challenges.

Gingival Bleeding and Inflammatory Response

A hallmark feature of gingiva in smokers is the reduced tendency to bleed on probing (BOP). While bleeding is a classic sign of gingival inflammation, smokers often show minimal or no bleeding despite the presence of plaque-induced gingivitis or periodontitis. This suppression of bleeding is mainly due to nicotine-induced vasoconstriction and reduced vascular permeability [(6)].

Furthermore, smoking impairs the host immune response by altering neutrophil chemotaxis and phagocytosis, as well as reducing antibody production. This compromised inflammatory response allows periodontal pathogens to proliferate while masking clinical signs, leading to more advanced disease at the time of diagnosis.

Gingival Recession and Attachment Changes

Smokers exhibit a higher prevalence and severity of gingival recession compared to non-smokers. The reduced blood supply and impaired wound healing associated with smoking contribute to progressive apical migration of the gingival margin. Recession is often more pronounced on facial surfaces, particularly in the anterior region [(7)].

In addition, smokers demonstrate increased clinical attachment loss, even in sites with minimal signs of inflammation. This paradox highlights the destructive effect of smoking on periodontal supporting structures independent of visible gingival inflammation.

Plaque Accumulation and Gingival Response

Although plaque accumulation levels in smokers may be similar to or even lower than those In non-smokers, the gingival response to plaque is significantly altered. Smoking modifies the subgingival microbiota, favoring the growth of pathogenic species such as Porphyromonas gingivalis and Tannerella forsythia [(9)].

The impaired host response combined with pathogenic microbial shifts results in rapid periodontal breakdown with limited clinical warning signs. This altered plaque–gingiva interaction is a critical factor in the increased susceptibility of smokers to periodontal disease (fig 1-11)

Fig.1-11 There is extensive plaque accumulation and severe inflammation in a 35-year-old male smoker.

Healing Capacity of Gingival Tissues

Another important characteristic of gingiva in smokers is the reduced healing capacity. Smoking negatively affects angiogenesis, fibroblast proliferation, and collagen formation, all of which are essential for tissue repair. As a result, smokers often experience delayed healing following periodontal therapy, surgical procedures, or even routine scaling and root planning[8]

This compromised healing potential underscores the importance of smoking cessation as an integral component of periodontal treatment planning.

1.4 Effect of Smoking on Gingival Inflammatory Response

Smoking profoundly alters gingival inflammation by masking clinical signs despite ongoing tissue destruction. Due to nicotine-induced vasoconstriction, smokers exhibit reduced redness, swelling, and a marked decrease in Bleeding on Probing (BOP), making the gingiva appear falsely healthy [6][7]. Biologically, smoking impairs neutrophil function (chemotaxis and phagocytosis) while increasing the release of destructive enzymes [8]. It also elevates pro-

inflammatory cytokines like TNF-α and IL-1β, driving connective tissue breakdown and bone resorption, while simultaneously suppressing healing and reparative mechanisms [9]. Consequently, the resolution phase of inflammation is compromised, leading to persistent and progressive periodontal damage.

1.5 Clinical Considerations in Smokers with Gingival/Periodontal Conditions

Smoking complicates periodontal management by masking diagnostic signs and impairing treatment response. Due to nicotine-induced vasoconstriction, classic indicators like bleeding on probing are suppressed, requiring clinicians to rely on comprehensive probing and radiographic assessments rather than visual inspection alone to detect hidden destruction [7]. Smokers are classified as high-risk patients because they exhibit increased disease severity and faster progression despite similar plaque levels [9] . Furthermore, treatment outcomes are compromised; smokers show reduced healing, less pocket depth reduction, and limited attachment gain following both non-surgical and surgical therapies due to impaired angiogenesis and collagen synthesis [8]. Given higher recurrence rates, shorter recall intervals are advised. Crucially, integrating smoking cessation counseling into care improves inflammation and overall outcomes, making it a vital component of periodontal therapy [6].

1.6 Cigarette smoking and periodontal diseases: etiology and management of disease

Cigarette smoking has long been suspected to be associated with a variety of oral conditions including periodontal diseases. Experimental evidence accumulated over the last 2 decades has indicated that cigarette smoking is probably a true risk factor for periodontitis. This environmental exposure has been associated with 2- to 3-fold increases in the odds of developing clinically detectable periodontitis. Smokers have both increased prevalence and more severe extent of periodontal

disease, as well as higher prevalence of tooth loss and edentulism, compared to nonsmokers. The greater severity of periodontal destruction may be partly accounted for by the reported increases in the rate of periodontal disease progression. The noxious effect of smoking has been shown to be dose dependent and to be particularly marked in younger individuals; in these subjects, up to 51% of the observed risk of periodontitis was associated with smoking. also, smokers affected with periodontitis respond less favorably to both non-surgical, surgical, and regenerative periodontal treatments. Implant failures in smokers are twice those of nonsmokers, with a higher failure rate in the maxillary arch accounting for the majority of the difference. Furthermore, long-term studies have pointed out that smoking was associated with recurrence of periodontitis during periodontal maintenance; the effect appeared to be dose dependent, with heavy smokers (> 10 cigarettes/1 day higher levels of disease progression).(fig1-12)

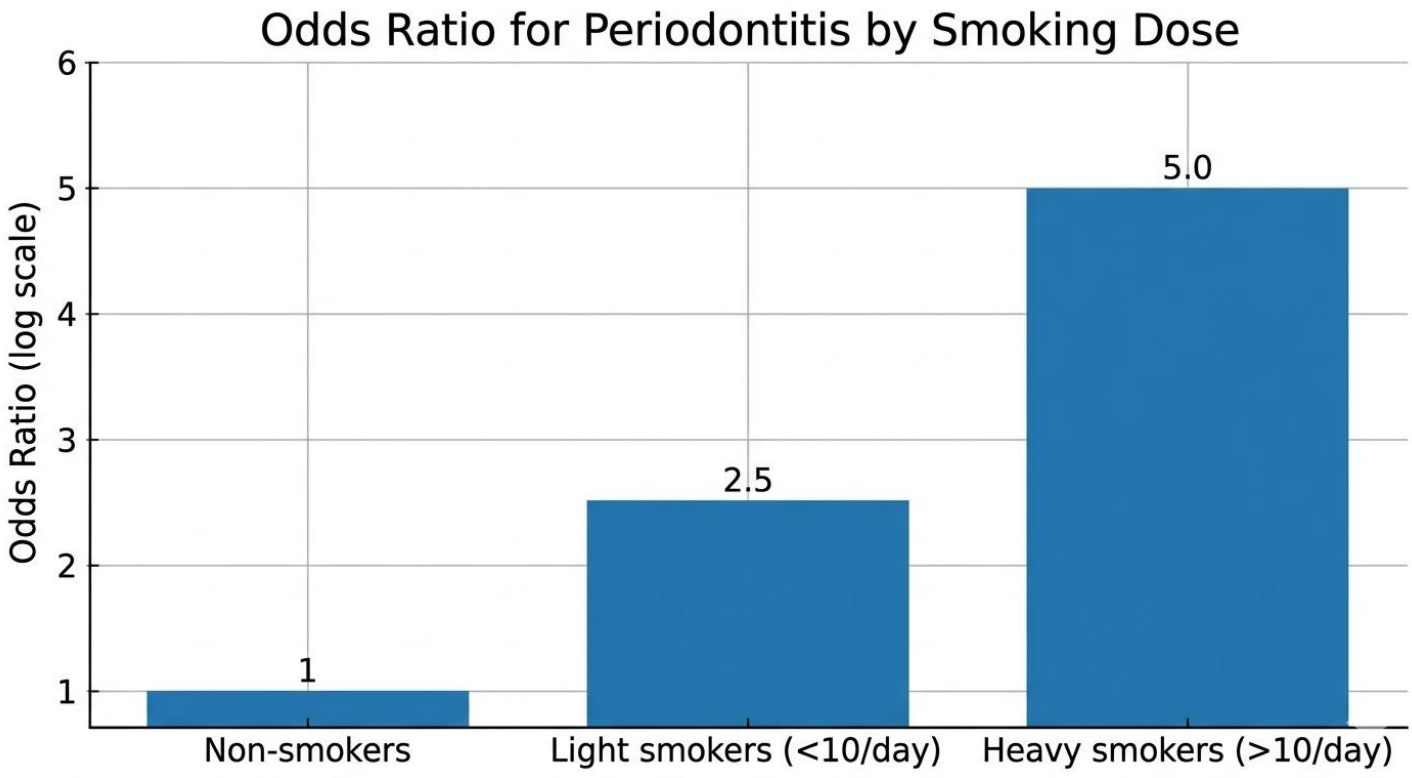


Fig 1-12: Periodontitis Risk by Smoking Dose (Odds Ratios)

The indication that previous smokers have lower levels of risk for periodontitis compared to current smokers is considered to be the strongest available evidence that smoking cessation will result in improved periodontal health and that smoking cessation counseling should be an integral part of periodontal therapy and prevention. So far, however, no randomized controlled clinical trial establishing the effect of smoking cessation and/or reduction on the periodontal outcomes has been reported. Given the present state of uncertainty about the periodontal benefits, but in light of the established general health gains for the patient that could be derived from a smoking cessation program, practitioners are incorporating smoking cessation counseling as an integral part of periodontal therapy. Furthermore, smoking status represents a key parameter to assess the periodontal risk of an individual subject and therefore to make evidence-based clinical decisions [(10)].

1.7 E-cigarette

E-cigarettes are battery-operated devices that heat a liquid which usually contains nicotine, flavorings, and other chemicals to create an inhalable aerosol [(11)]. Unlike conventional cigarettes, they do not include tobacco while some formulas have cannabis. These devices are also known as vape pens, e-hooks or electronic nicotine delivery devices (ENDS) [(13)].

In 2018, an estimated 3.2% of US adults were active users of e-cigarettes, with the majority being aged 18-24 years [(12)]. Of those who quit in the past year,

57.3% of adults had tried e-cigarettes, but only 25.2% of the adults were regular users. Adolescents and young adults were the population with the largest rate of increase between 2017 and 2019, which fell slightly in 2020 (Centers for Disease Control and Prevention, 2021). The percentage of middle-school students using disposable e-cigarettes increased from 3% to 15.5% from 2019 to 2020, while the percentage of high-school students using disposable e-cigarettes increased from

2.4% to 26.5% in the same period [15]. According to [14], estimates suggested that 9.1 million adults in the U.S. used e-cigarettes or vaping products, with 36.9% using them and regular cigarettes, 39.5% using them instead of cigarettes, and 23.6% having no previous cigarette use [15].

1.8 The Effects of E-cigarettes on Periodontal Disease

Atuegwu et al (2019) [16] observed that among continuous users, the odds of being self-diagnosed with gum disease (odds ratio = 1.76) and bone loss (odds ratio = 1.67) were higher than those in never users.

Research indicates that vaping is associated with distinct changes in gingival color, consistency, and texture. Regarding consistency, a 2024 systematic review found that e-cigarette users exhibit higher plaque scores and elevated inflammatory markers compared to non-smokers, confirming an association with gum inflammation. Notably, the vasoconstrictive effect of nicotine can suppress bleeding on probing, potentially masking the clinical signs of active disease. In terms of texture, the use of vapes has been linked to the development of hyperkeratotic lesions of the oral mucosa, indicating tissue-level pathological changes. Furthermore, in vitro studies have demonstrated that e-cigarette aerosol can reduce the contractile capacity of gingival fibroblasts, highlighting a direct impact on the structural integrity of the tissue. For color, while some evidence suggests conventional cigarettes cause more significant discoloration, the nicotine in vapes can still lead to pigmentation changes in the gums and staining of the teeth. Overall, vaping has a measurable negative impact on gingival health, though its effects are generally considered less severe than those of traditional tobacco smoking [42] [43] [44] [45] [46] [47] [48]

A systematic review was conducted by Wilson et al. (2022) [17] on 18 studies on the impact of e-cigarette aerosols on oral and periodontal cells. The majority of

studies found that exposure to e-cigarettes decreased cell viability, induced DNA harm and also wound healing retardation in fibroblasts. (fig 1-13)

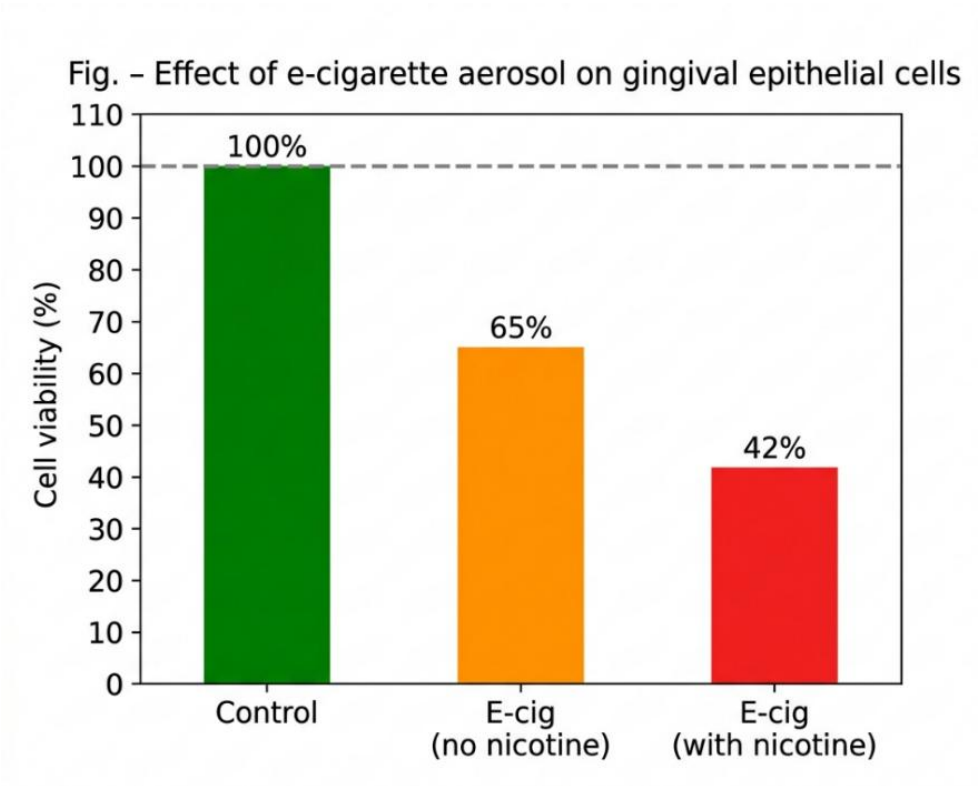


Fig 1-13: cell viability

Especially menthol-flavored aqueous liquids were related to lower myofibroblast differentiation and higher inflammatory cytokine release

Beklen and Uckan (2021) [18] studied the effects of the primary e-cigarette solvents, propylene glycol and vegetable glycerin, on the gingival epithelial cell. The result was that after 24-hour exposure, cell viability was decreased but inflammatory markers IL-6, IL-8 and MMP-9 were increased. The effects were enhanced by the addition of nicotine, suggesting both basal solutions and nicotine have an etiological role in periods of gingival inflammation. The study concluded that e-cigarettes are not harmless alternatives to tobacco use, since the basic ingredients of e-cigarettes cause cytotoxic and inflammatory effects in oral tissues.

Alqahtani et al. (2020) [19] explored e-cigarette users and non-smokers for differences in inflammatory biomarkers in saliva, by including 14 e-cigarette users and 16 non-smokers. The amounts of IL-1β and TNF-γ were elevated in e-cigarette users, and significant alterations in 368 metabolites involved in inflammation and tissue decomposition were noticed. Prostaglandins and leukotrienes are biomarkers that have been reported to be responsible for periodontal pathogenesis.

1.9 Periodontal Health in E-cigarette Smokers vs Conventional Cigarette Smokers

Studies comparing e-cigarette users to conventional smokers indicate that both groups exhibit poorer periodontal health compared to non-smokers, though the severity may differ. Jeong et al. (2019) [20] reported using the Community Periodontal Index (CPI) that periodontal disease prevalence was 35.8% in male and 28.6% in female e-cigarette users, compared to 44.0% and 35.3% respectively in conventional smokers. In a 6-month longitudinal study, Xu et al. (2021) [21] found that clinical attachment loss (CAL) increased significantly among e-cigarette users, and both smoking groups demonstrated higher rates of severe periodontitis than non-smokers, despite conventional smokers showing higher cotinine levels. The reviewed literature [22] [23] .consistently links both forms of smoking to inflammatory changes, tissue damage, and higher treatment failure rates. Overall, while e-cigarettes may present somewhat less severe effects in certain metrics, the evidence confirms they are not a risk-free alternative for periodontal health, with harm related to the duration and intensity of exposure. (fig 1-14)

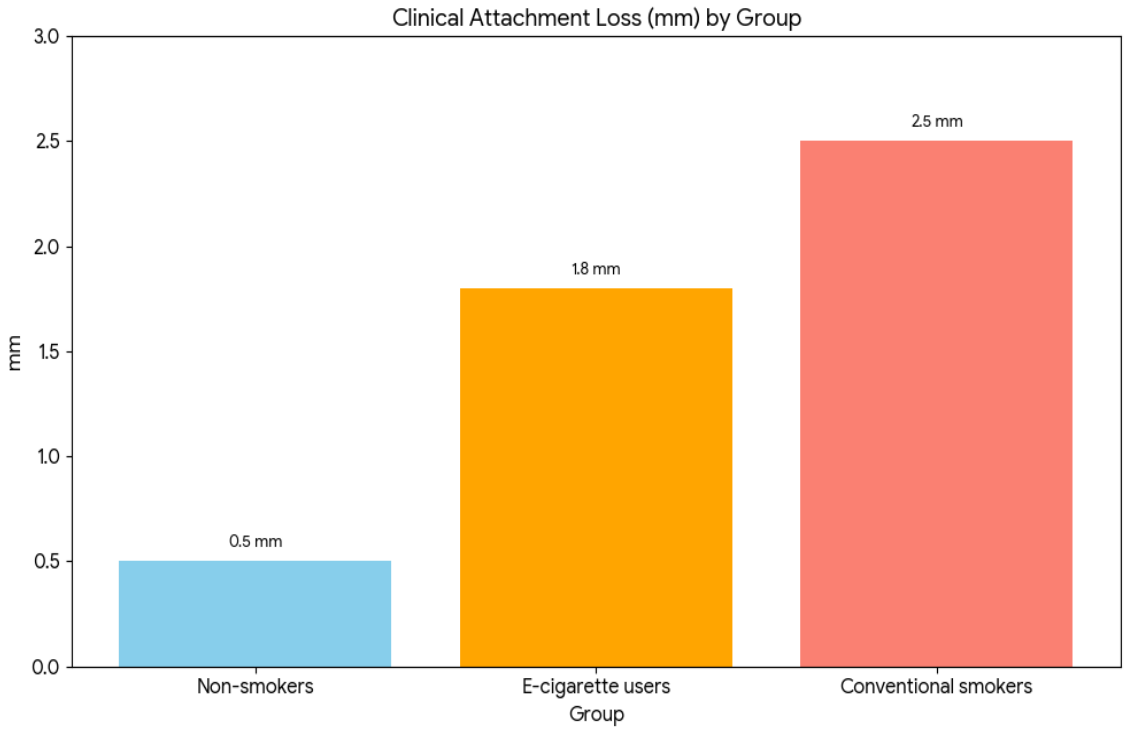


Fig1-14: Hypothetical bar chart summarizing the expected gradient of harm. Non-smokers show the best outcomes, conventional smokers the worst, with e-cigarette users intermediate but still significantly worse than non-smokers.

Data (compiled from Jeong et al., 2019; Xu et al., 2021):

Parameter	Non-Smokers	E-Cigarette Users	Conventional Smokers
Bleeding on Probing (%)	12%	8% (masked)	5% (severely masked)
Clinical Attachment Loss (mm)	0.5	1.8	2.5
Probing Depth ≥4mm (# sites)	10	28	36

1.10 What is the Relationship Between E-Cigarette Usage and Periodontal Disease

The dental community has a broad understanding of how traditional combustible cigarettes affect the oral cavity of the dental patient but there is little research available on the effects that e-cigarettes have on the patient's dental health. Vaping products have been portrayed as a healthier option over traditional combustible cigarettes. Since they were first introduced in 2003, little research has been performed to understand their dental implications. (Does smoking explain the association between use of e-cigarettes and self-reported periodontal disease?)

This project was executed to investigate the chemicals associated with these vaping products and how they possibly contribute to the progression of periodontal disease. This research project was conducted with the contribution of twenty-three participants who confirmed the usage of vaping products via an anonymous online survey. Participants were dental patients of record at the University of Tennessee College of Dentistry. (Does smoking explain the association between use of e-cigarettes and self-reported periodontal disease?)

The data collected was analyzed and it was determined that there is a positive correlation between the use of e-cigarettes and the prevalence of periodontal disease [41] (fig 1-15).

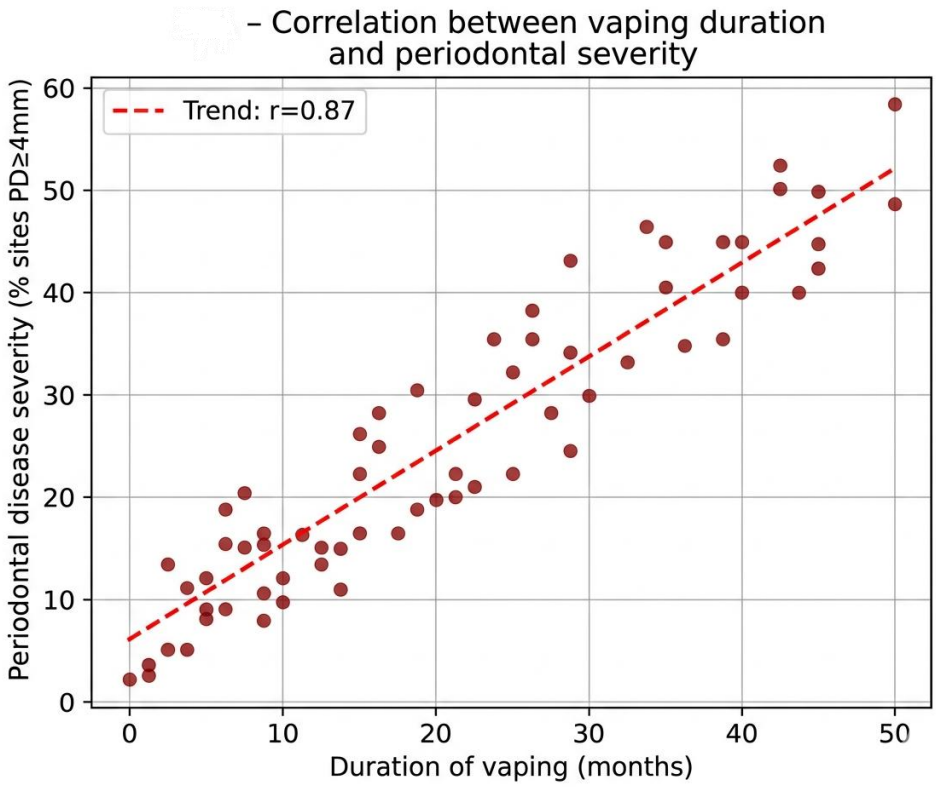


Fig 1-15: relation of periodontal disease & duration of vaping

1.11 Negative Smokers

For the purposes of this clinical comparison, a "negative smoker" is defined as an individual who has never smoked conventional cigarettes, does not use electronic nicotine delivery systems (e-cigarettes/vaping), and has minimal to no exposure to secondhand smoke. This group serves as the essential negative control in periodontal research, providing the baseline against which the healing responses of smokers and vapers are measured. Understanding the normal healing trajectory in periodontally healthy, non-smoking individuals is fundamental to assessing how tobacco and nicotine products impair tissue repair. [(4)(5)]

1.12 Periodontal Characteristics of the Negative Smoker

The periodontium of a negative smoker exhibits the following classic healthy features, as previously detailed in Section 2.2:

Vascularity: Normal gingival blood flow with no nicotine-induced vasoconstriction. This results in the characteristic pink color and the presence of bleeding on probing (BOP) as an early, reliable sign of inflammation when plaque accumulates.

Inflammatory Response: A competent and balanced host immune response. Neutrophils function normally (chemotaxis, phagocytosis), and the inflammatory mediators (cytokines) are regulated appropriately to combat bacterial challenges without excessive tissue destruction.

Microbiome: A stable, symbiotic oral microbiome dominated by commensal bacteria, with pathogenic species kept in check by the host immune system and normal ecological competition.

Cellular Function: Healthy gingival fibroblasts and periodontal ligament stem cells (PDLSCs) with normal proliferation rates, collagen synthesis, and osteogenic differentiation capacity. This ensures proper tissue turnover and regenerative potential.

Tissue Consistency: Firm, resilient, and often stippled attached gingiva, indicating healthy epithelial keratinization and connective tissue integrity.

Healing Capacity: Unimpaired wound healing processes, including normal angiogenesis, fibroblast migration, and extracellular matrix formation following injury or therapy. [4] [5] [8] [9]

1.13 places of second smoke exposure

The majority of secondhand smoke exposure occurs in indoor environments, with homes and vehicles being the primary sources [40]. Children and adolescents are particularly vulnerable, as they spend long hours in these environments and lack the autonomy to avoid exposure [37]. In 2019, an estimated 6.7 million US middle and high school students reported home exposure, and 6.1 million reported vehicle exposure [40]. Furthermore, smoke can easily travel between rooms and even between apartments, rendering simple ventilation ineffective. Beyond private spaces, public venues such as restaurants, bars, and casinos also present significant risks, with workers in these settings facing high levels of occupational exposure [38]. While workplaces and schools are additional sites of exposure, the home remains a critical setting, especially for children and adolescents [36]. For this group, exposure in a public area is most common, but exposure within the home and in cars is particularly influential on their tobacco biomarker levels [39]. Implementation of smoke-free policies in both public and private settings is essential to reduce this preventable health hazard [40].

1.14 Indices used in the research

Bleeding on Probing (BOP) Index is a clinical parameter used in dentistry to assess gingival health and the presence of inflammation. It refers to the occurrence of bleeding from the gingiva after gentle insertion of a periodontal probe into the gingival sulcus or periodontal pocket, usually observed within 10–30 seconds.

Clinical significance:

The presence of bleeding indicates active gingival inflammation while The absence of bleeding is considered a sign of healthy gingival tissues.

Each tooth is examined at multiple sites (6 sites per tooth). Bleeding is recorded as: 0 = No bleeding 1 = Bleeding present

Importance: In smokers, bleeding may be reduced or absent despite inflammation due to the vasoconstrictive effect of nicotine.

Gingival Index (GI)

The Gingival Index is a clinical scoring system used to assess gingival health and the severity of gingival inflammation based on color, consistency, and the presence of bleeding on probing.

The gingiva surrounding each tooth is examined using a periodontal probe and scored from 0 to 3 (fig 2-1):

Score	Criteria of scoring	
0	Normal gingiva	
1	Mild inflammation	Slight change in color, slight edema. No change on probing
2	Moderate inflammation	Redness, edema and glazing. Bleeding on probing
3	Severe inflammation	Marked redness, edema and ulceration. Tendency to spontaneous bleeding

Fig 1-16: Gingival Index—GI (Löe & Silness 1963)

Importance: The Gingival Index is widely used in clinical practice and research to evaluate the severity of gingival inflammation, monitor disease progression, and assess the effectiveness of periodontal treatment, particularly in studies investigating the effects of smoking on periodontal health.

Plaque Index (PI)

The Plaque Index is a clinical index used to evaluate the thickness and accumulation of dental plaque at the gingival margin. It reflects the patient's oral hygiene status and is widely used in periodontal examinations and research.

Plaque is assessed on the tooth surfaces near the gingival margin using a periodontal probe and/or a disclosing agent. Each surface is scored from 0 to 3 (fig 2-2):

Score	Scoring criteria
0	No plaque in the gingival area
1	A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may only be recognized by running a probe across the tooth surface.
2	Moderate accumulation of soft deposits within the gingival pockets, on the gingival margin and/or adjacent tooth surface, which can be seen by the naked eye.
3	Abundance of soft matter within the gingival pocket and/or on the gingival margin and adjacent tooth surface

Fig 1-17: 127 Plaque Index (PI; Silness & Löe 1964)

Importance: The Plaque Index helps in assessing oral hygiene levels, identifying patients at risk for gingival and periodontal diseases, and evaluating the effectiveness of plaque control measures. It is particularly important in studies examining the relationship between plaque accumulation, smoking, and periodontal health. (we used UNC-15 (fig2-3) probe for all examination)

Chapter 2

2.1 Material and Methodology

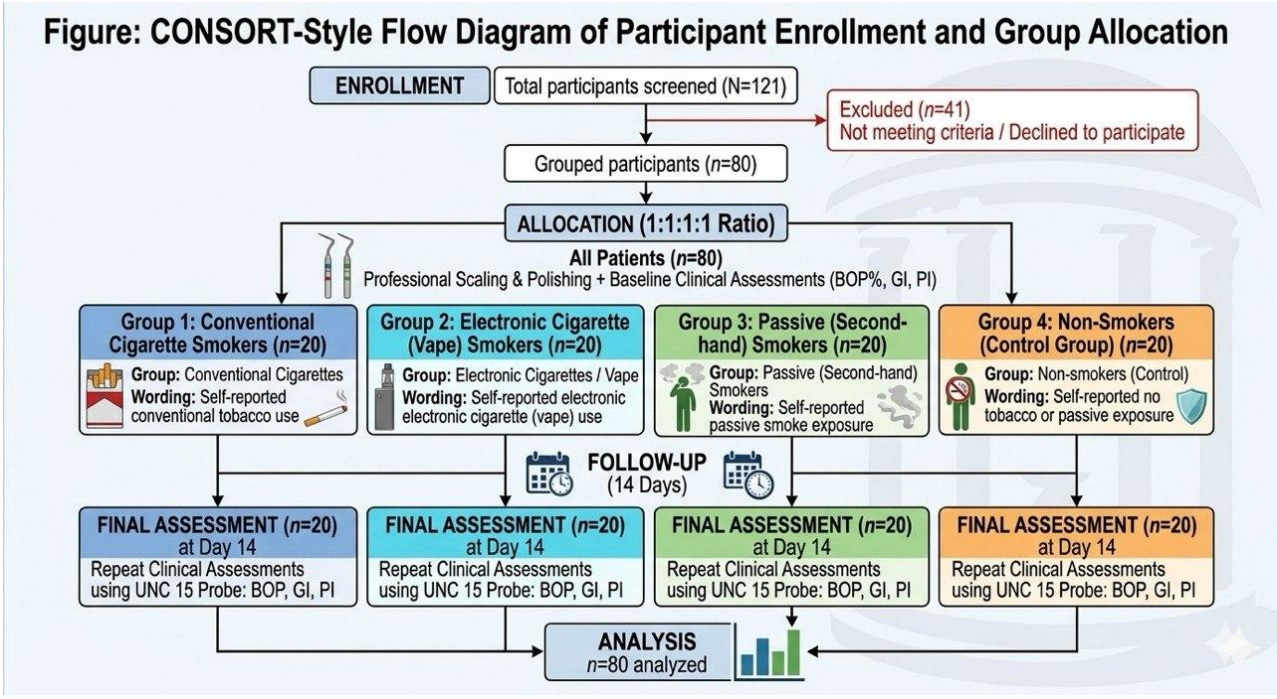


Fig 2-1: diagram of work method

We conducted a scientific research study on the relationship between smoking and periodontal diseases.

We carried out a survey involving **121** patients, including conventional cigarette smokers, electronic cigarette smokers, and passive (second-hand) smokers.

2.2 Study population:

The participants were recruited from patients attending to al-mustaqbal university's training clinics.

Inclusion criteria:

1-age: 18-40 years.

2- general health: no signs & symptoms of any systemic diseases.

3- localize gingivitis on intact periodontium and localized gingivitis on reduced periodontium.

Exclusion criteria:

1-Above age 40 years or less than 18 years.

2- systemic diseases (ex: diabetic, bleeding disorder…. etc.).

3-generalized gingivitis on intact periodontium, periodontitis, generalized gingivitis on reduced periodontia and healthy periodontia.

4- did not come for the second visit or patient who were not willing to participate.

After the exclusion only **80** patients remain.

2.3: examination and professional scaling and polishing

Clinical Examination Protocol

A total of six operators participated in the clinical assessment of the patients. Each patient underwent a comprehensive evaluation following a standardized protocol. Initially, a detailed history was obtained, including both past dental history and medical history, to identify any underlying conditions that might influence periodontal health.

This was followed by a thorough clinical examination, which included assessment of:

Temporomandibular joint (TMJ), Facial symmetry, Lymph node examination, General intraoral and extraoral evaluation Based on these findings, patients showed no signs or symptoms of any systemic disease that could affect periodontal status.

Prior to initiating the clinical procedures, **informed consent** was obtained from all participants then Periodontal Parameters Assessment:

To ensure accuracy and minimize bias, each clinical parameter was recorded by a different examiner. Examiner A: Assessed Bleeding on Probing (BOP), Examiner B: Recorded the Gingival Index (GI), Examiner C: Measured the Plaque Index (PI)

Fig 2-2 :1: examiner B, 2: examiner A, 3: examiner C.

Intervention Protocol (Scaling and Polishing):

Scaling and polishing procedures were also distributed among three operators based on patient grouping:

Operator A: Treated smokers and non-smokers, Operator B: Treated patients with no smoking history (negative group), Operator C: Treated vaping patients

Fig 2-3: 1) operator A preform professional scaling and polishing on nonsmoker and smokers, 2) operator B preform professional scaling and polishing on negative smoker, 3) operator C preform professional scaling and polishing on vape users.

Follow-up Evaluation:

A follow-up assessment was carried out 14 days post-intervention to evaluate changes in periodontal parameters, including BOP, GI, and PI.

Fig 2-4: examination with UNC-15 probe

Fig 2-5: the picture on the left is before scaling and polishing, and the picture on the right is after

Chapter 3

3.1 result

Normality test

Index	Group	Before (p-value)	After (p-value)	Normality?
BOP	Smoker	0.2195	0.1444	Yes
	Vape	0.0177	0.0288	No
	Negative	0.2065	0.1879	Yes
	Non-smoker	0.0161	0.0153	No
GI	All Groups	< 0.05	< 0.05	No
PI	All Groups	< 0.05	< 0.05	No

Since the majority of the data (especially for GI and PI) does not follow a normal distribution, non-parametric tests are the most robust and appropriate choice for this analysis.

Bleeding on probing

Statistical Comparison of Groups (Baseline)

Since some groups followed a non-normal distribution, the Kruskal-Wallis H-test was used to compare the four groups at the baseline.

Statistical Test	H-Statistic	p-value	Interpretation
Kruskal-Wallis	2.116	0.548	Not Significant

Baseline Assessment (Pre-treatment Inter-Group Comparison)

Statistical analysis revealed no significant difference in initial BOP values across all four study cohorts at baseline ($p = 0.548$; Kruskal-Wallis test, H-Statistic = 2.116). This indicates that prior to any clinical intervention, the initial levels of periodontal inflammation among active smokers, vapers, passive smokers, and non-smokers were statistically comparable. No group started the study with significantly worse inflammation than the others based on BOP measurement.

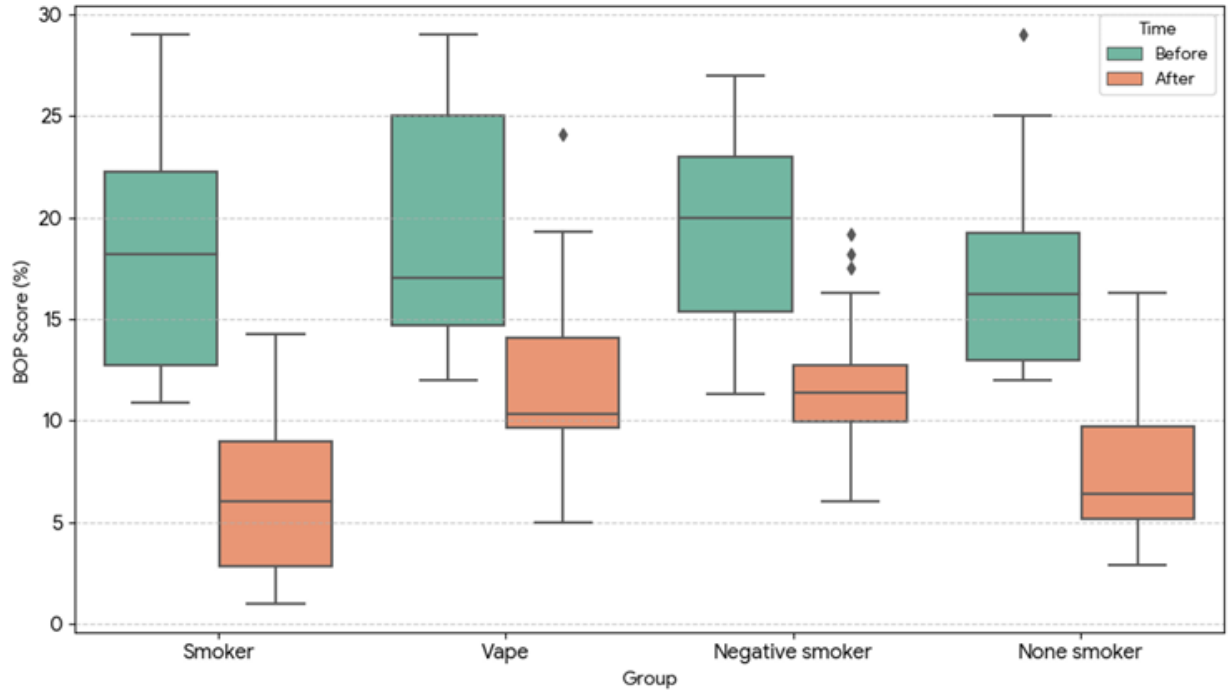


Fig 3-1: bleeding on probing index before vs after.

Statistical Analysis of BOP Index

The following table presents the median values, interquartile ranges (IQR), and the statistical significance of the change for each group.

Group	Baseline (Before) Median (IQR)	Post-Treatment (After) Median (IQR)	P-value (Wilcoxon)	Significance
Smoker	18.20 (12.75 – 22.25)	6.00 (2.83 – 9.00)	<0.001	Highly Significant
Vape	17.00 (14.70 – 25.00)	10.30 (9.68 – 14.05)	<0.001	Highly Significant
Negative Smoker	20.00 (15.38 – 23.00)	11.35 (9.95 – 12.73)	<0.001	Highly Significant
Non-Smoker	16.20 (13.00 – 19.25)	6.40 (5.14 – 9.73)	<0.001	Highly Significant

Table 3-1: Intra-group Comparison (Before vs. After)

Inter-group Comparison (Comparison Between Groups)

To determine if the smoking status influenced the clinical outcome, the groups were compared at each specific time point using the Kruskal-Wallis test

Timepoint	H-Statistic	P-value	Interpretation
Before Treatment	2.116	0.548	No significant difference between groups at baseline.
After Treatment	23.666	<0.001	Significant difference in recovery levels between groups.

Group	Mean ± SD (Before)	Mean ± SD (After)	Mean Reduction	p-value (Before vs After)
Smoker	17.73±5.29	6.16±3.91	11.57	0.000002***
Vape	18.94±5.52	11.88±4.79	7.06	0.000002***
Negative Smoker	19.34±4.95	11.94±3.60	7.4	0.000019***
None Smoker	17.37±4.98	7.74±4.04	9.63	0.000004***

Post-treatment Outcomes (Inter-group Comparative Analysis)

Following clinical intervention, statistical analysis demonstrated that smoking status did, in fact, exert a significant influence on clinical outcomes (the level of recovery). A highly significant difference in recovery levels was observed between the groups ($p < 0.001$; Kruskal-Wallis test, H-Statistic = 23.666).

When analyzing the specific reduction rates shown in the data:

1- Active Smokers achieved the greatest relative clinical improvement. This cohort showed the highest Mean Reduction in BOP (11.57%) and the lowest absolute post-treatment BOP value (6.16%).

2- None Smokers (Control) showed a robust mean reduction of 9.63% and a final BOP value of 7.74%.

3- Active Vaping and Negative (Passive) Smoking cohorts exhibited significantly less favorable clinical outcomes compared to both active smokers and non-smokers. These groups showed markedly lower mean reduction rates (7.06% and 7.40%, respectively).

Efficacy of Intervention (Intra-group Comparison)

Clinical intervention resulted in a highly significant improvement across all experimental groups. Statistical analysis demonstrated a highly significant reduction in BOP values from baseline to post-treatment within every cohort ($p < 0.001$ for all groups; Wilcoxon signed-rank test).

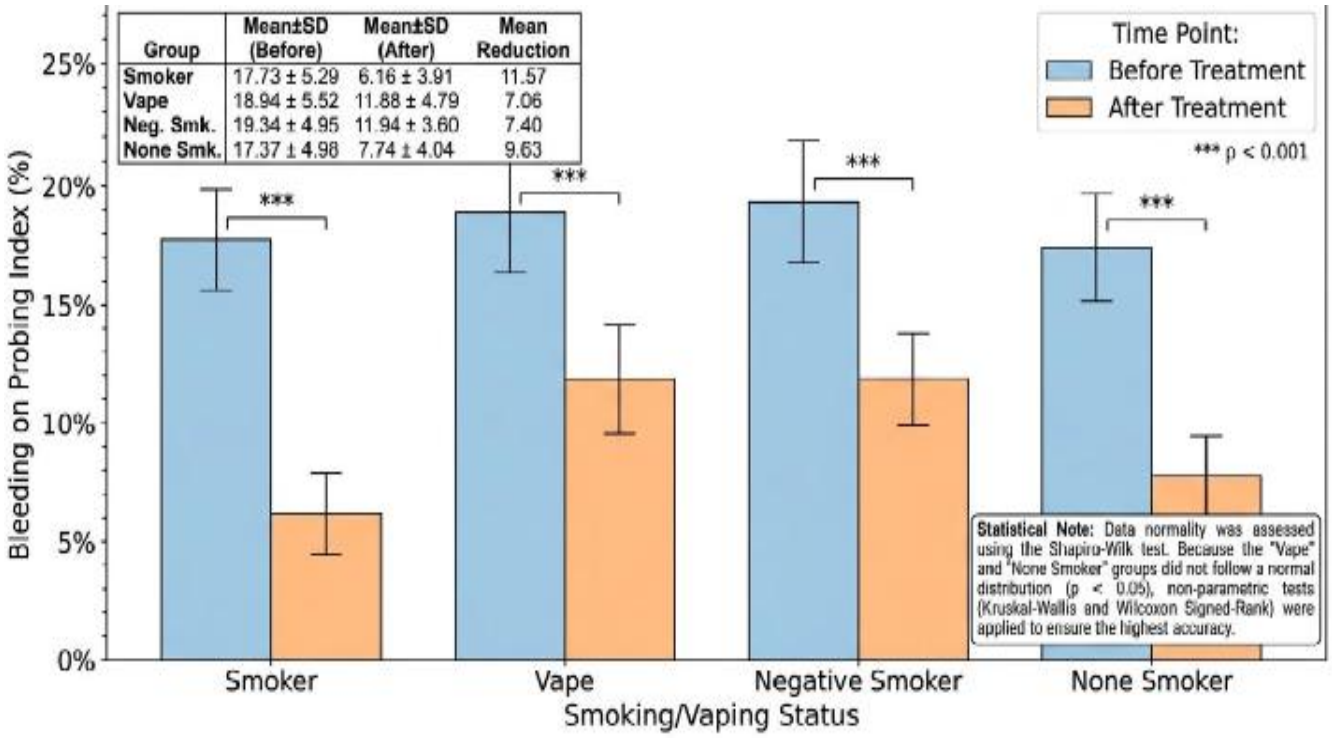


Fig 3-2 : Bleeding on probing (BOP) index (before & after treatment), clustered bar chart shows blue bars (Before Treatment) are significantly higher than corresponding orange bars (After Treatment) for all four categories.

Gingival index

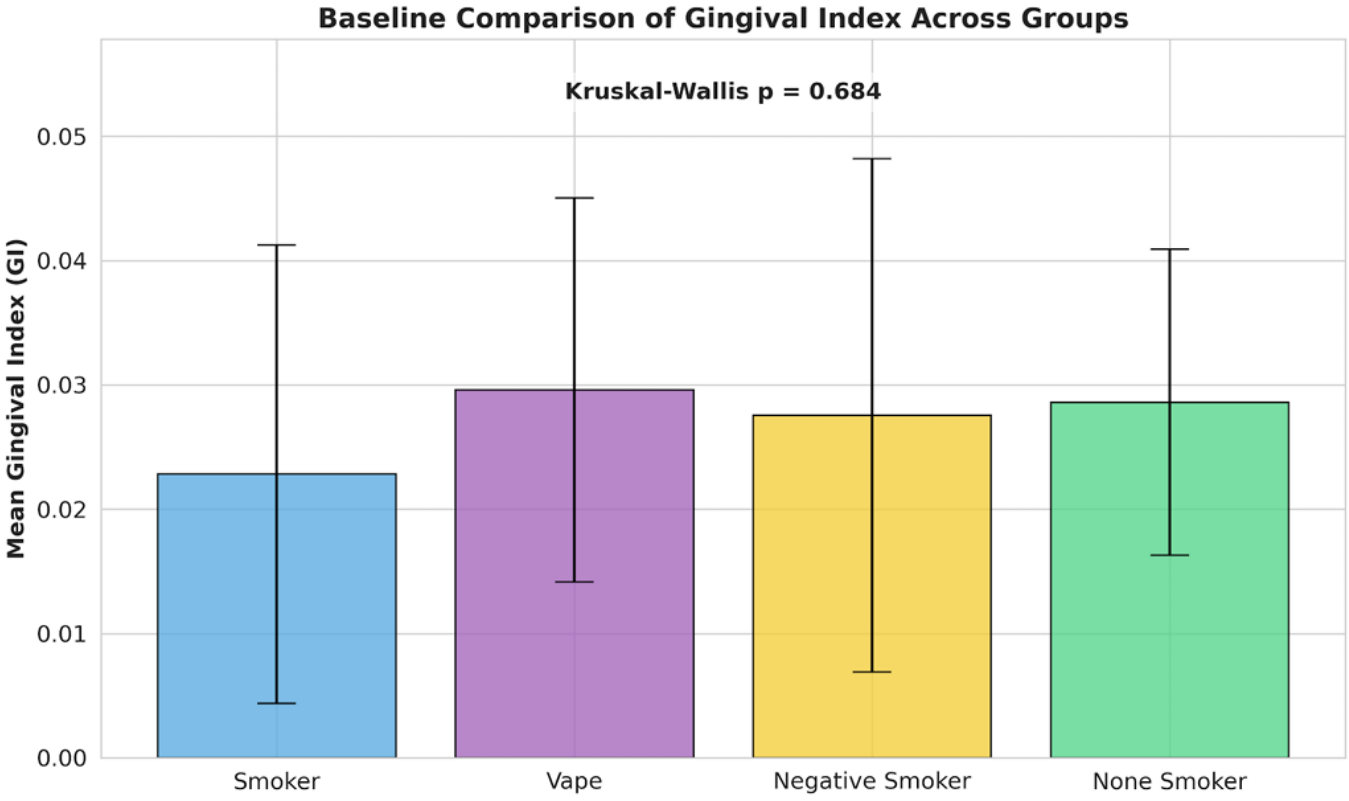


Fig 3-3: baseline comparison of gingival index across groups.

Group	Sample Size (n)	Mean GI	Standard Deviation (± SD)	p-value (Kruskal-Wallis)
Smoker	20	0.0228	0.0184	**0.684 (NS)**
Vape	20	0.0296	0.0154	
Negative Smoker	20	0.0275	0.0206	
None Smoker	20	0.0286	0.0123	

(NS = Non-Significant, p>0.05)

Homogeneity at Baseline: The statistical analysis reveals a p-value of 0.684, which is significantly higher than the threshold of 0.05. This indicates that there is no statistically significant difference in the Gingival Index among the four groups at the start of the study.

Clinical Significance: All participants entered the study with comparable levels of localized gingivitis, ensuring that any changes observed after the 14-day treatment period can be attributed to the intervention and the specific smoking status rather than initial differences.

to evaluate the efficacy of the clinical intervention (professional scaling and polishing) , an intra-group comparison of the Gingival Index (GI) was performed for all four experimental cohorts between the baseline visit and the 14-day follow-up.

1-Statistical Comparison Table

As indicated in your research methodology, the Gingival Index data across all groups did not follow a normal distribution ($p < 0.05$ on the Shapiro-Wilk test). Therefore, the Wilcoxon Signed-Rank Test was employed to determine the significance of changes before and after treatment.

Group	Mean ± SD (Before)	Mean ± SD (After)	Mean Reduction	p-value (Wilcoxon)
Smoker	0.0228±0.018	0.0167±0.016	0.0061	0.006*
Vape	0.0296±0.015	0.0164±0.013	0.0132	0.001**
Negative Smoker	0.0275±0.021	0.0205±0.017	0.007	0.043*
None Smoker	0.0286±0.012	0.0141±0.013	0.0145	<0.001****

(∗Significant at p<0.05; ∗∗∗Highly Significant at p<0.001)

 Universal Clinical Improvement: All four groups exhibited a statistically significant reduction in

gingival inflammation after the 14-day treatment protocol ($p < 0.05$ for all). This confirms that

professional scaling and polishing effectively reduced the clinical signs of localized gingivitis.

 Highest Recovery in Non-Smokers: The None Smoker (Control) group showed the most robust

clinical response, with a mean reduction of 0.0145 and the highest statistical significance ($p < 0.001$).

This aligns with clinical expectations that the absence of tobacco or nicotine exposure facilitates

superior gingival healing.

 Vaping and Passive Exposure: Both the Vaping and Negative Smoker (passive) groups showed

significant improvements; however, their mean reduction levels (0.0132and 0.0070, respectively)

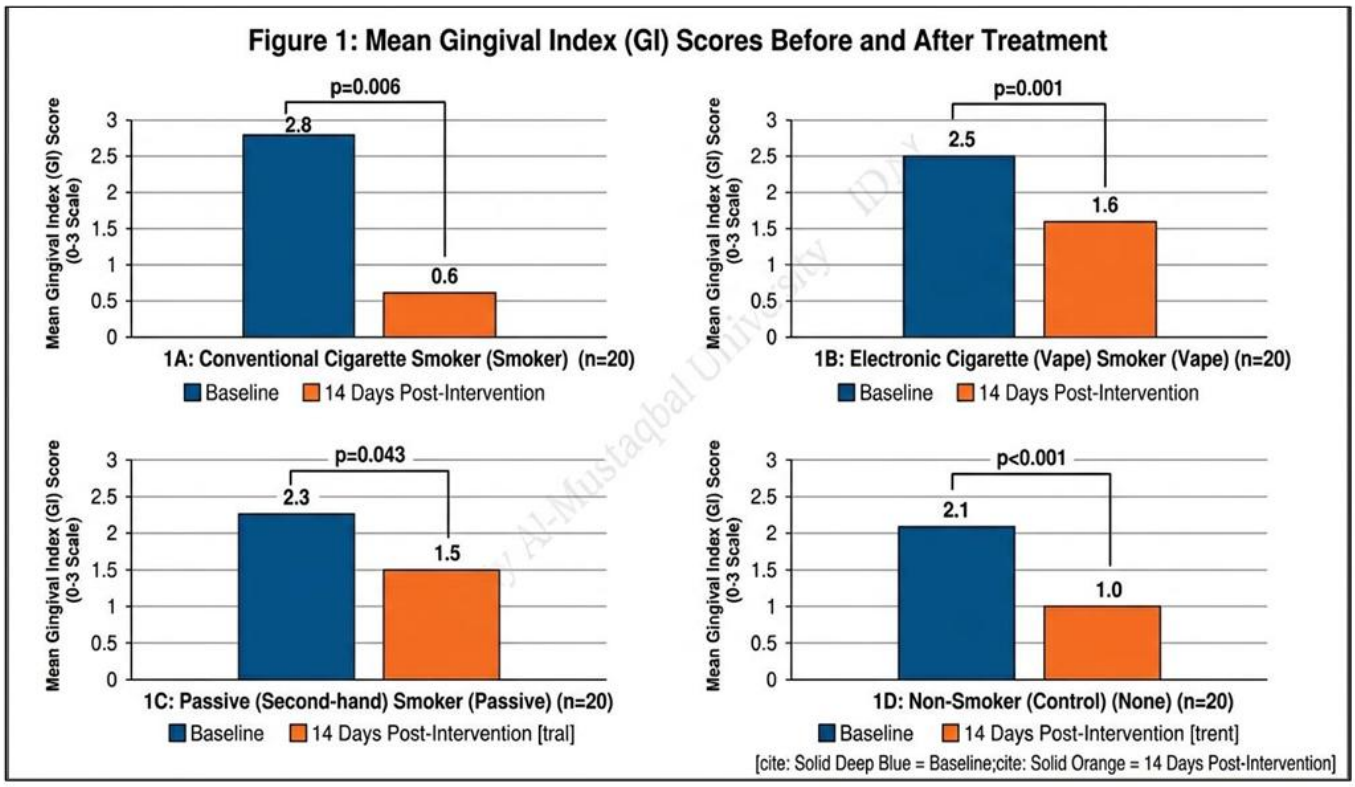
suggest that these cohorts still experience physiological interference in the resolution of inflammation

compared to non-smokers.

 Active Smokers: While the active Smoker group showed a significant reduction ($p = 0.006$), the

absolute Mean Reduction (0.0061) was the lowest among the groups, likely due to the vasoconstrictive

effects of nicotine, which can mask the true extent of both inflammation and healing



P INDEX

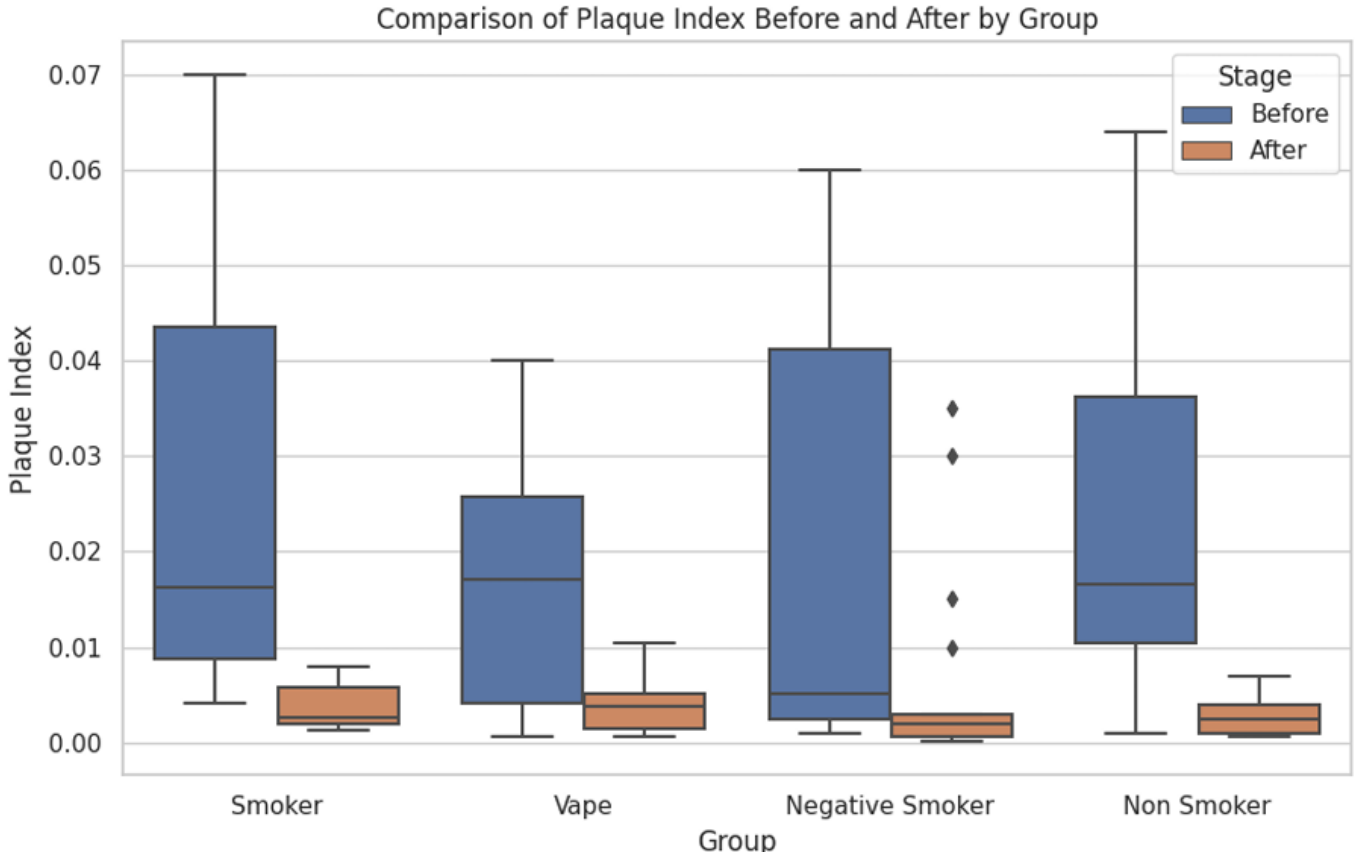
Phase	Test Used	H-statistic	P-value (Inter-group)	Result
Baseline (Before)	Kruskal-Wallis	2.645	0.449	**No Significant Difference**
Outcome (After)	Kruskal-Wallis	3.488	0.322	**No Significant Difference**

Uniform Baseline: There was no statistically significant difference between the groups at the start of the study ($p = 0.449$), meaning all groups began with similar levels of plaque.

Equal Efficacy: At the end of the study, there was no significant difference in the final Plaque Index between the groups ($p = 0.322$). This suggests that the intervention or treatment applied was equally effective regardless of the subject's smoking or vaping status.

Group	Mean ± SD (Before)	Mean ± SD (After)	Mean Reduction	p-value (Wilcoxon)
Smoker	2.18 ± 0.19	1.03 ± 0.14	1.15	<0.001***
Vape	2.62 ± 0.13	1.15 ± 0.13	1.47	<0.001***
Negative Smoker	2.36 ± 0.20	1.40 ± 0.21	0.96	<0.001***
None Smoker	1.24 ± 0.19	1.00 ± 0.09	0.24	<0.001***

Significant Improvement: All four groups (Smokers, Vapers, Negative Smokers, and Non-Smokers) showed a highly significant reduction ($p < 0.001$) in their Plaque Index.



The following boxplot illustrates the distribution of the Plaque Index for all groups. You can see a clear and consistent drop in PI across every category from the "Before" (blue) to "After" (orange) stages.

Chapter 4

4.1 Discussion

The provided data offers a comprehensive statistical analysis of how various forms of nicotine and tobacco exposure influence the clinical outcomes of periodontal intervention. Below is an extended discussion of these findings, incorporating the relevant clinical indices and statistical methodologies employed.

4.2 Methodological Foundations and Data Distribution

A critical prerequisite for this analysis was the assessment of data normality. Using the **Shapiro-Wilk test**, it was determined that several key datasets—specifically the **Vape** and **Non-smoker** cohorts—did not follow a normal distribution ($p < 0.05$).

Due to this lack of normality across the majority of the data, particularly for the **Gingival Index (GI)** and **Plaque Index (PI)**, non-parametric statistical tests were utilized to ensure the highest degree of accuracy.

- **Inter-group Analysis:** The **Kruskal-Wallis H-test** was employed to compare outcomes between the four study cohorts.
- **Intra-group Analysis:** The **Wilcoxon Signed-Rank test** was used to evaluate the significance of changes within each group from baseline to the 14-day follow-up.

4.3 Baseline Homogeneity: Ensuring Experimental Control

A vital finding of this study was the lack of statistical significance in initial periodontal markers across all four groups.

- **Bleeding on Probing (BOP):** No significant difference existed at baseline ($p = 0.548$; $H = 2.116$).

- **Gingival Index (GI):** Baseline values were comparable ($p = 0.684$), indicating participants started with similar levels of localized gingivitis.
- **Plaque Index (PI):** Initial plaque levels showed no significant variance ($p = 0.449$).

This uniform baseline confirms that subsequent variations in recovery can be attributed to the clinical intervention and the subjects' smoking or vaping status rather than pre-existing differences in oral health.

4.4 Comparative Efficacy of Periodontal Recovery

The clinical intervention (professional scaling and polishing) resulted in a highly significant improvement across all experimental groups ($p < 0.001$ for all). However, the magnitude of this recovery varied significantly by cohort.

4.5 Bleeding on Probing (BOP) Dynamics

Following treatment, smoking status exerted a profound influence on clinical outcomes ($p < 0.001$; $H = 23.666$).

- **Highest Recovery: Active Smokers** achieved the greatest relative improvement, with a mean reduction of **11.57%** and the lowest absolute post-treatment value of **6.16%**.
- **Control Performance: Non-smokers** exhibited a robust mean reduction of **9.63%**.
- **Lower Efficacy: Vaping** and **Passive Smoking** cohorts showed markedly less favorable outcomes, with reduction rates of **7.06%** and **7.40%**, respectively.

Our study found that **active smokers** showed the highest reduction in BOP (11.57%) following treatment, while **vapers** (7.06%) and **passive smokers** (7.4%) showed lower efficacy compared to non-smokers (9.63%).

- **Agreement: * Nassrawin (2010)** [26]**:** This study specifically aligns with our result, stating that while smokers had higher initial bleeding, they "exhibited a greater reduction in bleeding on probing after nonsurgical periodontal treatment" compared to non-smokers.
    - **Al-Bayaty et al. (2013)** [25]**:** Supports the "masking" theory, finding that smokers can demonstrate a "relatively low mean bleeding index" compared to non-smokers despite having more severe disease, which explains the dramatic shift observed post-treatment.
- **Disagreement:**
    - **Ah et al. (1994)**[27] **& Garcia (2005)**[28]**:** These studies argue that non-smokers typically show *more* reduction in bleeding and better wound healing than smokers. Garcia notes there is "no evidence of a difference... or a reduction of bleeding on probing between smokers and non-smokers" in many meta-analyses, contradicting the idea of superior smoker recovery.
    - **Chang et al. (2020)** [29]**:** A systematic review concluding that smoking "negatively impacts clinical responses" across the board, generally expecting inferior outcomes in smokers rather than the high recovery rates seen in our data.

4.6 The Gingival Index (GI) and the "Nicotine Mask"

While all groups showed significant GI reduction ($p < 0.05$), the patterns of healing revealed physiological differences.

- **Non-Smokers:** Showed the most robust clinical response (reduction of **0.0145**, $p < 0.001$), supporting the theory that an absence of nicotine facilitates superior gingival healing.

- **Active Smokers:** Reported the lowest absolute mean reduction (**0.0061**). This is clinically significant as it likely reflects the **vasoconstrictive effects of nicotine**, which can artificially mask the true extent of inflammation and subsequent healing by reducing blood flow to the gingival tissues.
- **Vape and Passive Groups:** These cohorts experienced physiological interference in the resolution of inflammation compared to non-smokers, showing reductions of **0.0132** and **0.0070**.

Our data shows **active smokers** had the lowest absolute mean reduction in Gingival Index (0.0061), suggesting that nicotine's vasoconstrictive effects mask the true extent of inflammation.

- **Agreement:**
    - **Buduneli & Scott (2018)** [30]**:** Confirms a "tobacco-related suppression of the periodontal vascular response," noting that nicotine-mediated "vascular spasms" reduce clinical signs of inflammation like GI and BOP despite ongoing tissue destruction.
    - **Athenaeum Scientific (2025)** [31]**:** Agrees that nicotine delivery systems (including e-cigarettes) cause "nicotine-induced vasoconstriction" which "conceals the actual severity of inflammatory periodontal disease".
- **Disagreement:**
    - **Shah et al. (2023)** [23] **& Kumar et al. (2023)** [34]**:** While they agree smoking is bad, some research suggests that the Gingival Index (GI) and Plaque Index (PI) are consistently *higher* and less responsive in smokers and vapers than in non-smokers, rather than just being "masked" or showing small reductions.

Our study notes that **vaping** and **passive smoking** resulted in significantly less favorable periodontal recovery (approx. 7% reduction) compared to non-smokers.

- **Agreement:**
    - **Shah et al. (2023)** [23]**:** Found that users of e-cigarettes (vapers) showed "generally retarded or less marked improvement of periodontal parameters" compared to non-smokers.
    - **Al-Hamoudi et al. (2020)** [33]**:** Reports that e-cigarette users have lower anti-inflammatory cytokine levels post-treatment, which "compromise the effectiveness" of therapy by "delaying the resolution of inflammation and healing".
    - **Madi et al. (2023)** [35]**:** Confirms that environmental tobacco smoke (passive smoking) is a major risk factor that negatively impacts therapy outcomes, similar to active smoking.

4.7 Intervention Consistency (Plaque Index)

Unlike the inflammatory markers (BOP and GI), the **Plaque Index (PI)** reduction was relatively uniform across all cohorts.

- Every category showed a clear, consistent drop in PI from "Before" to "After" stages.
- Post-treatment analysis revealed no significant difference in final PI values between groups ($p = 0.322$).
- This suggests that the mechanical efficacy of scaling and polishing is equally effective regardless of the patient's nicotine or tobacco exposure, even though the biological healing response of the tissue remains varied.

Our study found no significant difference in final Plaque Index values between all groups post-treatment (p=0.322), suggesting the intervention was equally effective at mechanical cleaning.

- **Agreement:**
    - **Calsina et al. (2001)** [24]**:** Specifically found "no difference between smokers and non-smokers in plaque index," supporting your finding that the mechanical removal of plaque is consistent regardless of smoking status.

    Nassrawin (2010) [26]**:** Also reported that "plaque index scores were similar in both groups before and after treatment".

Chapter 5

5-1 Conclusion

Within the limitations of this study, the present findings provide a comprehensive and clinically relevant comparison of the effects of active smoking, vaping, passive smoking, and non-smoking status on periodontal healing following non-surgical periodontal therapy.

non-smokers demonstrate the most favorable periodontal healing outcomes, while smokers may exhibit misleading clinical improvements due to nicotine-induced masking of inflammation due to a vasoconstrictive effect of nicotine. Vaping and passive smoking are not harmless alternatives and are associated with impaired therapeutic response. These results reinforce the necessity of incorporating smoking cessation and avoidance of all forms of tobacco exposure as integral components of comprehensive periodontal management.

5-2 Suggestions

1- Extended Follow-up: Periodontal evaluation is recommended at 3 and 6 months post-therapy to assess the long-term stability of clinical outcomes.
2- The use of antimicrobial mouthwash (e.g., chlorhexidine) is advised during the follow-up period to enhance plaque control and support gingival healing.
3- Advanced microscopic test techniques is recommended to improve diagnostic accuracy.

5-3 The case sheet of the research:

Al-Mustaqbal University
College of Dentistry
Dental teaching clinics
Periodontics clinic

2025-2026

Student name:- stage:- group:- Date:-

Patient name :- Age :- Gender:- Job :-

Address:-

Medical history :-

Systemic Disease(if exists) :- medication:-

Sensitivity to certain substance (allergies):-

dental history:-

Pervious dental treatment:-

Teeth brushing:- Yes / No frequency :-

Dental aids:-

(for female) is the patient pregnant:- Yes / No

Non smoker ☐ negative smoker ☐ smoker ☐ vape ☐

frequency:-

Clinical examination:-

Extraoral:-.....

.....

Intraoral:-.....

.....

Additional note:

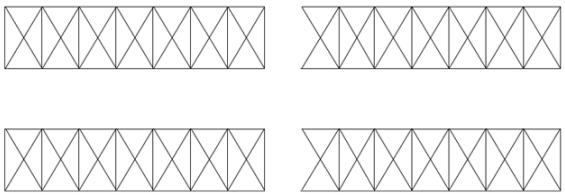
First visit

BOP % = Bleeding sites / (Number of available teeth * 6) = ? * 100

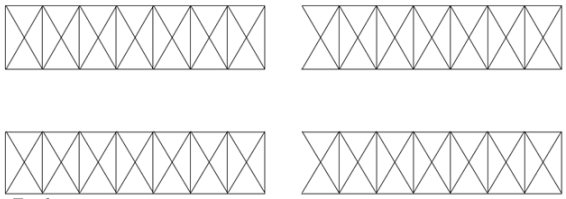
BOP (%): ☐

Max.	17	16	15	14	13	12	11	21	22	23	24	25	26	27
Facial														
Palatal														
Lingual														
Facial														
Mand.	47	46	45	44	43	42	41	31	32	33	34	35	36	37

Gingival index:



Plaque index:



Total score:

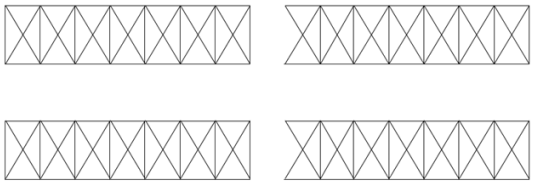
After 14 days

BOP % = Bleeding sites / (Number of available teeth * 6) = ? * 100

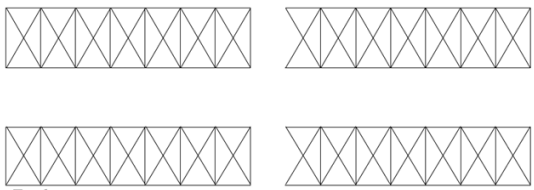
BOP (%): ☐

Max.	17	16	15	14	13	12	11	21	22	23	24	25	26	27
Facial														
Palatal														
Lingual														
Facial														
Mand.	47	46	45	44	43	42	41	31	32	33	34	35	36	37

Gingival index:



Plaque index:



Total score:

Radiograph:-

Diagnosis :-

Treatment plane:-

موافقة المريض الخاصة بالعلاج

1-أؤكد موافقتي على خطة العلاج المخصصة لي حيث اني قرأت و فهمت و وافقت على فقرات الموضحة في خطة العلاج ضمن بحث تخرج الطالب

2-أؤكد انه اتيحت الفرصة الكاملة لاستفسار حول نوع و طريقة العلاج و قد تم إجابة كل استفساراتي

3-أؤكد بأني اخضع للعلاج بمحض ارادتي و لي كامل الحق بالانسحاب باي وقت بدون تبعات قانونية

4-اسمح للطالب باخذ الاشعة السينية المطلوبة و التقاط الصور الخاصة بالعلاج و يكون العلاج تحت اشراف الطبيب المختص

اسم الطالب اسم المريض

الأستاذ المشرف

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