TRABAJO FIN DE GRADO

Grado en Odontología

EL USO DEL TABACO Y SU RELACIÓN CON LA ENFERMEDAD PERIODONTAL

Madrid, curso académico 2024/2025

Número identificativo: 050

RESUMEN

Introducción: este trabajo analiza la relación entre el tabaquismo y la enfermedad periodontal, un problema de salud pública relevante debido a la alta prevalencia del consumo de tabaco y su impacto en la salud bucodental. El tabaco es un factor de riesgo clave en el desarrollo y progresión de la periodontitis, influyendo en la inflamación, la respuesta inmunitaria y la microbiota oral; **Objetivos:** el objetivo principal de este trabajo es analizar si el consumo de tabaco incrementa la prevalencia de la periodontitis en comparación con pacientes no fumadores; **Metodología:** se realizó una revisión sistemática de la literatura científica utilizando bases de datos como PubMed, Medline y la biblioteca CRAI Dulce Chacón, incluyendo artículos de los últimos 25 años; **Resultados:** se incluyeron 10 estudios que mostraron que los fumadores presentaron mayores niveles de pérdida de inserción clínica, profundidad de sondaje, pérdida ósea marginal y una menor respuesta al tratamiento periodontal. También se observó una mayor prevalencia de bacterias periodontopatógenas y niveles elevados de marcadores inflamatorios y estrés oxidativo en comparación con no fumadores; **Conclusiones:** las conclusiones evidencian que el tabaquismo agrava la severidad de la periodontitis, aunque no puede confirmarse que incremente su prevalencia debido a su naturaleza multifactorial. El tabaco actúa como un factor modificador importante. Se destaca la importancia de realizar más estudios clínicos para comprender mejor esta relación y desarrollar estrategias preventivas y terapéuticas más eficaces.

PALABRAS CLAVE

Odontología, tabaco, fumar, enfermedad periodontal, periodontitis.

ABSTRACT

Introduction: this paper analyzes the relationship between smoking and periodontal disease, a public health issue of great relevance due to the high prevalence of tobacco use and its impact on oral health. Tobacco is a key risk factor in the development and progression of periodontal disease, influencing inflammation, immune response, and the oral microbiota; **Objectives:** the main objective is to evaluate whether tobacco consumption increases the prevalence of periodontitis compared to non-smokers; **Methodology:** a systematic review of the scientific literature was carried out using databases such as PubMed, Medline, and the CRAI Dulce Chacón library, including articles from the last 25 years; **Results:** 10 studies were included, showing that smokers had higher levels of clinical attachment loss, probing depth, marginal bone loss, and a poorer response to periodontal treatment. Addiotionally, a higher prevalence of periodontopathogenic bacteria and elevated levels of inflammatory markers and oxidative stress were observed compared to non-smokers; **Conclusions:** the conclusions indicate that smoking worsens the severity of periodontitis, although it cannot be confirmed that it increases its prevalence due to the multifactorial nature of the disease. Tobacco acts as a significant modifying factor. The importance of conducting more clinical studies to better understand this relationship and develop more effective preventive and therapeutic strategies is emphasized.

KEYWORDS

Dentistry, tobacco, smoke, periodontal disease, periodontitis

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1. INTRODUCCIÓN

El consumo de tabaco aumenta el riesgo de casi todas las afecciones orales (1), afectando (entre otras estructuras) al periodonto (2). Cuando éste presenta una inflamación crónica en la que se ven afectados los tejidos de soporte, quiere decir que hay una patología existente denominada enfermedad periodontal (3). Numerosos estudios han evidenciado que el tabaquismo no solo exacerba la inflamación y el daño tisular, sino que también compromete la respuesta inmunitaria (2). En este contexto, el presente trabajo se propone analizar la relación entre el consumo de tabaco y la enfermedad periodontal.

1.1. Tabaco

1.1.1. Definición

El tabaco es una sustancia obtenida de la hoja de *Nicotiana tabacum*, y mezclado con diferentes aditivos para mejorar el sabor y otras características. (4)

El daño causado por el tabaco se debe principalmente a su combustión, proceso en el que se liberan más de 7000 compuestos, de los cuales 100 han sido identificados como perjudiciales o potencialmente peligrosos para la salud. La ignición del tabaco se produce a temperaturas cercanas a los 800. Los datos demuestran que la producción de sustancias dañinas del tabaco se incrementa con el aumento de la temperatura. (5)

Algunas de estas sustancias son: óxido de carbono (CO), dióxido de carbono (CO_2), acetona, amoniaco (NH_3), cianuro de hidrógeno (HCN), metano, propano, metil clorhidrato, metil furano, óxidos de nitrógeno (NOX), nitrospirrolidina, propionaldehido, 2-butano, nicotina, anilina, benzopireno, hidracina, naftalina, 4-(metilnitrosamina)-1-(3-piridil)-1-butanona) (NNK), fenol, pireno, quinolona, stigmasterol, tolueno… (4)

1.1.2. Marco histórico

El tabaco proviene de plantas procedentes de América (alrededor de Perú y Ecuador), donde se empezó a cultivar aproximadamente desde 5000 antes de Cristo (a.C) – 3000 a.C. Cuando los colonizadores españoles llegaron al continente en 1492, su uso ya estaba completamente

extendido por todo el continente, y fue a partir de entonces cuando éste empezó a ser exportado a Europa. (6)

El hábito de fumar seguramente comenzó con la inhalación, ya que los dispositivos más antiguos relacionados con el tabaco estaban hechos para este fin. Sin embargo, también se consumía de muchas otras formas: se masticaba, bebía en infusiones, aplicaba en la piel para eliminar parásitos, y se usaba en rituales y ceremonias religiosas. Se valoraba tanto por sus propiedades medicinales como por su significado espiritual, y se empleaban diversos instrumentos para su consumo, siendo la pipa el método más popular en la antigüedad, cumpliendo roles sociales y culturales. (6)

1.1.3. Tabaco en la actualidad

Actualmente, con el fin de reducir los efectos tóxicos de los cigarrillos convencionales, se han introducido nuevos tipos de cigarrillos, como los productos de tabaco calentado y los cigarrillos electrónicos. Aunque estas opciones pueden mitigar la producción de ciertos compuestos tóxicos, los cigarrillos electrónicos pueden generar nuevas sustancias nocivas (por ejemplo, formaldehído, acetaldehído y acroleína) que no existían en la solución original. Estos compuestos generalmente se producen a través de la oxidación de sus componentes (propilenglicol (PG) y glicerina vegetal (VG)) mediante calentamiento y vaporización. (7)

La Región de Europa tiene una de las tasas más altas de muertes prematuras debidas al consumo de tabaco en el mundo, y la disminución de su prevalencia avanza lentamente, especialmente entre las mujeres. En 2015, la Organización Mundial de la Salud (OMS) estimó que el 18% de las muertes por enfermedades no transmisibles en la región eran atribuibles al tabaco. La prevalencia es mayor en Europa central y oriental, y menor en los países nórdicos y en Asia central. Se prevé que la mitad de los países europeos no logrará reducir el consumo de tabaco en un 30% para 2025, meta global establecida. Además, el tabaquismo es casi el doble entre hombres que entre mujeres, y se observa una mayor prevalencia en personas con niveles socioeconómicos más bajos. (8)

1.1.4. Tabaco y enfermedades sistémicas

El tabaquismo es el principal factor de riesgo conductual, asociado con una mayor tasa de mortalidad y morbilidad, lo que lo convierte en un problema de gran relevancia para la salud pública a nivel global. (1)

Además, el tabaco provoca el desarrollo de múltiples enfermedades en el organismo, siendo las más frecuentes las afecciones respiratorias crónicas, las enfermedades cardiovasculares y distintos tipos de cáncer, además de empeorar patologías ya preexistentes en el organismo. (9)

En los países del este mediterráneos, en 2020 el consumo de tabaco fue responsable de más de 100 000 casos de cáncer y de 82 000 muertes por cáncer por tabaco en población de 30 años o más. (10)

Fumar tiene un profundo impacto en nuestro sistema respiratorio. Las principales enfermedades respiratorias asociadas con el tabaquismo incluyen enfermedad pulmonar obstructiva crónica (EPOC), enfisema, asma bronquial, enfermedades pulmonares intersticiales, cáncer de pulmón, fibrosis pulmonar, etc. El tabaquismo es un factor crucial en la causa y el empeoramiento de muchas enfermedades respiratorias, y el pronóstico de estas empeora en gran medida con el hábito de fumar. Todos los fumadores dañan parte de su arquitectura bronquial debido al tabaquismo y perjudica el funcionamiento del complejo alveolar. La bronquiolitis, una reacción bronquial inflamatoria, a menudo se encuentra en las personas que fuman. (11)

El tabaquismo se considera un factor de riesgo para enfermedades pulmonares como el EPOC, aunque también intervienen en la patogénesis múltiples factores de riesgo ambientales y genéticos. El objetivo principal del humo del cigarrillo inhalado es el epitelio de las vías respiratorias, que funciona como una barrera para las sustancias químicas nocivas inhaladas. El humo del cigarrillo es una fuente exógena de oxidantes y genera especies oxidativas intracelulares que interrumpen procesos celulares como la respiración aeróbica. El estrés oxidativo es inducido por la exposición del epitelio pulmonar al humo del cigarrillo, y el estrés oxidativo crónico se ha relacionado con enfermedades pulmonares. (12)

El consumo de cigarrillos contribuye al desarrollo y progresión de enfermedades vasculares: periféricas, coronarias, accidentes cerebrovasculares y aneurismas de la aorta. Esto se produce por varios mecanismos que aumentan la inflamación, alteran la regulación de los vasos sanguíneos y causan estrés oxidativo. Además, fumar activa las plaquetas, deprime la función endotelial e induce un estado hiperadrenérgico, lo que aumenta el gasto cardíaco, la frecuencia del ritmo cardíaco y los niveles de presión arterial. El riesgo también depende del nivel de

exposición, siendo los fumadores crónicos y más intensos quienes presentan una mayor carga de enfermedades y comorbilidades inducidas por el tabaco. (13)

El tabaco es el principal factor responsable del cáncer a nivel global, y en Estados Unidos (EE.UU) se estima que, por sí solo, contribuye a al menos el 30% de las muertes por esta enfermedad. En particular, fumar está vinculado con aproximadamente el 87 % de los fallecimientos por cáncer de pulmón, el tipo de cáncer con mayor tasa de mortalidad en hombres y mujeres. (14) Aunque existe un amplio conocimiento sobre la relación entre el tabaquismo y el cáncer de pulmón, muchos desconocen que también se vincula con el desarrollo de cáncer en al menos otros 17 órganos, como la laringe, faringe, cavidad oral, cavidad nasal y senos paranasales, esófago, estómago, colon y recto, hígado, páncreas, médula ósea, vejiga, riñón, uréter, cuello uterino y ovario. Esta lista de cánceres causados por el tabaco sigue creciendo a medida que se genera nueva evidencia científica. (4)

La investigación ha determinado muchos de los procesos biológicos a través de los cuales el hábito tabáquico y la inhalación de humo conducen al cáncer. Por ejemplo, los carcinógenos del tabaco se activan metabólicamente en los seres humanos en formas que se unen al ácido desoxirribonucleico (ADN) y crean aductos de ADN, lo que a su vez causa mutaciones en genes importantes para la regulación del crecimiento. Fumar también induce efectos epigenéticos que contribuyen a la carcinogénesis. (14)

1.1.5. Tabaco y cavidad oral

La primera zona del organismo con la que tiene contacto el tabaco es la cavidad oral, por lo que es lógico pensar que muchas de las consecuencias también se observan a nivel de esta parte del organismo: manchas, caries dental, lesiones en mucosas, cáncer oral, enfermedad periodontal y fracasos de implantes, entre otras (15,16).

Actualmente, se acepta que la disminución de la capacidad amortiguadora y el pH más bajo de la saliva de los fumadores, junto con una mayor concentración de *Lactobacilli* y *Streptococcus mutans (S. mutans)*, causa caries dental. Investigaciones recientes realizadas *in vitro* han evidenciado que la nicotina estimula el crecimiento de *S. mutans* en el biofilm. Además, los niveles reducidos de anticuerpos inmunoglobulina A (IgA) en la saliva de los fumadores podrían aumentar su incidencia de caries. (16)

El contacto prolongado del tabaco con la mucosa oral induce una variedad de cambios, los cuales podrían clasificarse como lesiones de la mucosa oral inducidas por el tabaco que tienen menos probabilidades de causar cáncer (como las reacciones liquenoides, leucoedema, melanosis, queratosis, eritema palatal con hiperplasia), lesiones que son potencialmente malignas (leucoplasia, eritroplasia, liquen plano y fibrosis submucosa) y neoplasias malignas inducidas por el tabaquismo (carcinoma de células escamosas). (17)

El tabaquismo se ha asociado también con la coloración de los dientes, restauraciones y prótesis dentales, alteración en las sensaciones del gusto y el olfato, lengua vellosa negra, candidiasis y alveolitis seca tras las extracciones dentales. (16)

Múltiples estudios clínicos y poblacionales demostraron fuertes asociaciones entre el tabaquismo y la enfermedad periodontal, provocando una disminución de la inserción del epitelio y del nivel del hueso alveolar, y pérdida de dientes, incluso independientemente de los niveles de placa dental. (1,4)

La exposición al humo del tabaco perjudica la reacción defensiva del organismo frente a la película microbiana del biofilm oral y, además, aumenta la producción de citocinas y enzimas inflamatorias potencialmente destructivas. Además, se han caracterizado distintos patrones microbianos entre el biofilm de la placa de fumadores de tabaco y no fumadores, lo que sugiere un perfil más patogénico. Finalmente, fumar tabaco parece afectar la capacidad regenerativa de las células encargadas de la salud periodontal, como son los fibroblastos, osteoblastos y cementoblastos, lo que disminuye la formación de nuevo tejido y puede interferir en la eficacia del tratamiento periodontal. (1)

Los fumadores tienen un riesgo aproximadamente 1.69 veces mayor de fracaso de implantes, tanto antes del implante como después de la inserción de la prótesis. El tiempo de exposición y la cantidad de cigarrillos fumados diariamente influyen directamente en el tejido óseo periimplantario, ya que aumentan la concentración de componentes tóxicos. La nicotina es citotóxica para los fibroblastos gingivales y reduce su proliferación, lo que afecta la osteointegración y provoca la deposición de tejido fibroso en la superficie hueso-implante. El tabaquismo afecta la densidad y cantidad de hueso formado alrededor del implante después de 60 días de su colocación, y la inhalación de humo de cigarrillo tiene un impacto más negativo en la osteointegración que la administración subcutánea de nicotina. (2,16)

Los niveles elevados de nicotina y radicales libres influyen en el metabolismo óseo, estimulando el proceso de resorción ósea al reducir el reclutamiento, anclaje, adhesión, propagación, proliferación y diferenciación de los osteoblastos. Esto disminuye la producción de colágeno

necesario para la formación de la matriz extracelular y reduce la calcificación de esta matriz en la superficie del implante. Además, la obstrucción microvascular causada por la nicotina provoca isquemia y una disminución en la proliferación de células sanguíneas, lo que compromete el suministro de sangre y nutrientes en el sitio de curación del implante. También se ha observado que la nicotina sobreestimula las células defensivas del organismo, como monocitos, neutrófilos y macrófagos, para producir mediadores inflamatorios, lo que aumenta la susceptibilidad a infecciones en el sitio quirúrgico. (16,18)

1.2. Enfermedad periodontal

1.2.1. Definición

La enfermedad periodontal es una enfermedad crónica inflamatoria y destructiva, que compromete los tejidos que sostienen el diente, (3) lo que resulta en la pérdida de inserción del diente respecto al periodonto y en la destrucción del hueso alveolar. (19)

El periodonto saludable se adapta firmemente a los tejidos subyacentes, con un margen en forma de filo de cuchillo ubicado a nivel de la unión amelocementaria. Presenta un borde festoneado, más alto en las papilas interdentales, y más bajo en las áreas bucales y linguales. El surco gingival, donde la encía se une al diente, tiene una profundidad de 1 a 3 mm, no presenta sangrado con un leve sondaje y contiene fluido crevicular. La pared lateral del surco constituye el margen gingival libre. La encía adherida, de 1 a 9mm de ancho, es inmóvil, queratinizada y con aspecto punteado, mientras que la mucosa alveolar, situada por debajo de la unión mucogingival, es móvil y no queratinizada. La falta de encía adherida en ciertas zonas puede generar problemas mucogingivales, y cualquier alteración en estas estructuras puede ser indicativo de enfermedad periodontal. (Figura 1) (19)

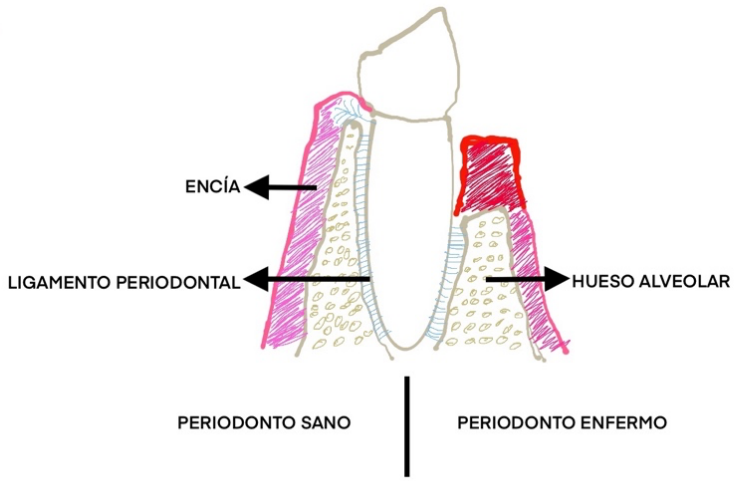

Figura 1. Tejidos periodontales sanos vs con enfermedad periodontal

1.2.2. Marco histórico

Las primeras referencias a la enfermedad periodontal se encontraron en escritos del antiguo Egipto y China, sugiriendo su reconocimiento hace unos 5000 años. Los primeros escritos modernos aparecieron en España en el siglo X. A lo largo de la historia, autores como Pierre Fauchard, considerado el padre de la odontología moderna, y John Hunter, contribuyeron con sus obras. Fauchard describió procedimientos periodontales en su obra *El dentista cirujano* (1728), mientras que Hunter, en *La historia natural de los dientes humanos* (1771) y *Un tratado práctico sobre las enfermedades de los dientes* (1778), estableció las bases científicas del tratamiento odontológico actual. (19)

Anton van Leeuwenhoek fue quien descubrió la presencia de bacterias alrededor de los dientes en el siglo XVII, quien también observó por primera vez el papel protector de las biopelículas sobre las bacterias. Sin embargo, la relación entre las bacterias y la periodontitis no fue plenamente aceptada hasta finales del siglo XIX, cuando se publicaron los trabajos fundamentales sobre la teoría microbiana de la enfermedad realizados por Pasteur, Koch y Lister. Adolph Witzel (1847–1906) fue uno de los primeros en reconocer las bacterias como el origen de la enfermedad periodontal, desafiando la creencia predominante de que su origen estaba relacionado principalmente con factores sistémicos. (19,20)

John W. Riggs (1811–1885) fue líder en el tratamiento de la enfermedad periodontal, hasta el punto de que la periodontitis fue conocida durante un tiempo en EE.UU como "la enfermedad de Riggs". En el siglo XIX, se comprendía poco sobre el origen y el mecanismo patológico de las enfermedades periodontales, por lo que su clasificación se basaba en aspectos clínicos o en teorías sin base científica. En esa época también surgió el término “piorrea alveolar”, usado para describir la periodontitis como una supuesta infección ósea. Esta concepción errónea llevó durante años a tratarla mediante la eliminación quirúrgica del hueso marginal. (19,21)

Es a finales de 1940 cuando se relacionó fumar cigarrillos con la enfermedad periodontal, al estar implicado en formas necrotizantes de la enfermedad periodontal. (16)

1.2.3. Estado actual

Los informes epidemiológicos más recientes indican una prevalencia del 46 % de mala salud periodontal en EE.UU; sin embargo, en este país el reembolso para el cuidado periodontal es

bastante limitado, lo que podría explicar esta prevalencia. Curiosamente, también se informó una prevalencia del 45 % en el Reino Unido, donde el Servicio Nacional de Salud (NHS) apoya financieramente la prevención periodontal y reembolsa los tratamientos periodontales. (22) Por otro lado, otra de las razones por la que la prevalencia de la periodontitis es tan alta, puede ser el resultado de un aumento de la esperanza de vida. (3)

En esta muestra poblacional, aproximadamente el 45 % de las personas afirman realizar chequeos regulares, cepillarse los dientes dos veces diarias y usar dentífrico con flúor. Sin embargo, un análisis detallado de los datos indica que el 52 % de los participantes eran fumadores activos, el 51 % presentaba placa visible, y el 44 % tenía una dieta deficiente que incluía un alto consumo de azúcar. (22)

1.2.4. Clasificación

La clasificación de la periodontitis ha evolucionado durante las últimas décadas. Inicialmente, se basaba en términos clínicos generales, aunque en la década de 1980 se introdujeron categorías de periodontitis del adulto, juvenil y formas rápidamente progresivas. La *American Academy of Periodontology* (AAP) en 1999 introdujo una nueva clasificación, eliminando los términos anteriores y dividiendo la enfermedad periodontal en crónica, agresiva, necrosante y asociada a enfermedades sistémicas. (19)

Tonetti propuso una nueva clasificación de la periodontitis basada en estadios (I-IV) y grados (A-C), la cual evalúa la gravedad y factores de riesgo de la enfermedad periodontal. (Tabla 1 y Tabla 2) (23)

Tabla 1. Clasificación de periodontitis por grados, basada en evidencia directa, indirecta y factores modificadores. Tonetti y colaboradores (cols.). 2018. (23)

		Grado A	**Grado B**	**Grado C**
Evidencia directa	Radiografías o evaluación periodontal en los 5 últimos años	No evidencia de pérdida de hueso/inserción	Pérdida < 2 mm	Pérdida ≥ 2mm
	Pérdida ósea vs. Edad	< 0,25	0,25–1,0	> 1,0
Evidencia indirecta	Fenotipo	Grandes depósitos de biofilm con niveles bajos de destrucción	Destrucción proporcional a los niveles de biofilm	El grado de destrucción supera las expectativas teniendo en cuenta los depósitos de biofilm; patrones clínicos específicos que sugieren periodos de progresión rápida y/o patología de aparición temprana. Falta de respuesta prevista a tratamientos de control bacteriano habituales.
Factores modificadores	Tabaquismo	No fumador	< 10 cigarros/día	≥ 10 cigarros/día
	Diabetes	Normal con/sin diabetes	HbA1c < 7 con diabetes	HbA1c > 7 con diabetes

Tabla 2. Clasificación de periodontitis por estadios, según la gravedad del diagnóstico inicial y la complejidad, sobre la base de factores locales. Tonetti y cols. 2018. (23)

		Estadio I	**Estadio II**	**Estadio III**	**Estadio IV**
Gravedad	CAL interdental en zona de mayor pérdida	1–2 mm	3–4 mm	≥ 5 mm	≥ 5 mm
	Pérdida ósea radiográfica	Tercio coronal (<15 %)	Tercio coronal (15–33 %)	Extensión a tercio medio/apical de raíz	Extensión a tercio medio/apical de raíz
	Pérdida dentaria	Ninguna por periodontitis	Ninguna por periodontitis	≤ 4 dientes perdidos por periodontitis	≥ 5 dientes perdidos por periodontitis
Complejidad	Local	Profundidad de sondaje máxima ≤ 4mm	Profundidad de sondaje máxima ≤ 5 mm	Profundidad de sondaje máxima ≥ 6 mm	Profundidad de sondaje máxima ≥ 6 mm
		Pérdida ósea principalmente horizontal	Pérdida ósea principalmente horizontal	Además de complejidad Estadio II:	Además de complejidad Estadio III:
				Pérdida ósea vertical ≥ 3 mm	Necesidad de rehabilitación debido a:

			Afectación de furca grado II o III	Disfunción masticatoria
			Defecto de cresta moderado	Trauma oclusal secundario (movilidad dentaria ≤ 2)
				Defecto alveolar avanzado
				Colapso de mordida, abanicamiento dental, migraciones dentarias
				Menos de 20 dientes residuales (10 parejas con contacto oclusal)
Extensión y distribución	Añadir a estadio como descriptor	En cada estadio, describir extensión como localizada (> 30% de dientes implicados), generalizada o patrón molar/incisivo		

La AAP, en colaboración con la Federación Europea de Periodoncia (EFP), adoptó este nuevo sistema de clasificación para crear una clasificación actualizada, creada en 2017 y publicada en 2018. Se basa en mediciones del nivel de inserción, la profundidad de sondaje, la pérdida ósea y/o el grado de inflamación, eliminando la distinción entre crónica y agresiva. (Figura 2, Figura 3, Figura 4, Figura 5) (19,24,25)

Salud periodontal, enfermedades y condiciones gingivales.

1. **Salud periodontal y gingival**
   a. Salud gingival clínica en un periodonto intacto
   b. Salud gingival clínica en un periodonto reducido
      I. Paciente de periodontitis estable
      II. Paciente sin periodontitis

2. **Gingivitis inducida por biopelícula dental**
   a. Asociada solamente a biopelícula dental
   b. Mediada por factores de riesgo sistémicos o locales
   c. Agrandamiento gingival inducido por medicamentos

3. **Enfermedades gingivales no inducidas por biopelícula dental**
   a. Trastornos genéticos del desarrollo
   b. Infecciones específicas
   c. Condiciones inflamatorias e inmunes
   d. Procesos reactivos
   e. Neoplasias
   f. Enfermedades endocrinas, nutricionales y metabólicas
   g. Lesiones traumáticas
   h. Pigmentación gingival

Figura 2. Clasificación de Enfermedades y Alteraciones Periodontales y Periimplantarias 2017. Salud periodontal, enfermedades y condiciones gingivales. (25)

Formas de periodontitis

1. **Enfermedades periodontales necrosantes**
   a. Gingivitis necrosante
   b. Periodontitis necrosante
   c. Estomatitis necrosante

2. **Periodontitis como manifestación de enfermedades sistémicas**
   a. La clasificación de estas condiciones debe basarse en la enfermedad sistémica primaria, de acuerdo con los códigos de la clasificación estadística internacional de enfermedades y problemas de salud relacionados (ICD)

3. **Periodontitis**
   a. Estadios basados en la severidad y complejidad de manejo
      I. Estadio I: periodontitis inicial
      II. Estadio II: periodontitis moderada
      III. Estadio III: periodontitis severa con potencial de pérdida dental adicional
      IV. Estadio IV: periodontitis severa con pérdida potencial de la dentición
   b. Extensión y distribución: localizada, generalizada, distribución incisivo-molar
   c. Grados: evidencia o riesgo de progresión rápida, respuesta anticipada al tratamiento
      I. Grado A: tasa lenta de progresión
      II. Grado B: tasa moderada de progresión
      III. Grado C: tasa rápida de progresión

Figura 3. Clasificación de Enfermedades y Alteraciones Periodontales y Periimplantarias 2017. Formas de periodontitis. (25)

Manifestaciones periodontales de las enfermedades sistémicas y condiciones del desarrollo y adquiridas.

1. **Enfermedades sistémicas y condiciones que afectan los tejidos de soporte periodontal**

2. **Otras condiciones periodontales**
   a. Abscesos periodontales
   b. Lesiones endodóntico-periodontales

3. **Deformidades mucogingivales y condiciones alrededor de los dientes**
   a. Fenotipo gingival
   b. Recesión gingival/de tejido blando
   c. Falta de encía
   d. Profundidad vestibular reducida
   e. Frenillo aberrante/posición del músculo
   f. Exceso gingival
   g. Color anormal
   h. Condición de superficie radicular expuesta

4. **Fuerzas oclusales traumáticas**
   a. Trauma oclusal primario
   b. Trauma oclusal secundario
   c. Fuerzas ortodóncicas

5. **Factores protésicos y dentales que modifican o predisponen a las enfermedades gingivales/periodontales inducidas por placa**
   a. Factores localizados relacionados con dientes
   b. Factores localizados relacionados con prótesis dental

Figura 4. Clasificación de Enfermedades y Alteraciones Periodontales y Periimplantarias 2017. Manifestaciones periodontales de las enfermedades sistémicas y condiciones del desarrollo y adquiridas. (25)

Enfermedades y condiciones periimplantarias

1. **Salud periimplantaria**
2. **Mucositis periimplantaria**
3. **Periimplantitis**
4. **Deficiencias de tejidos blandos y duros periimplantarios**

Figura 5. Clasificación de Enfermedades y Alteraciones Periodontales y Periimplantarias 2017. Enfermedades y condiciones periodontales. (25)

1.2.5. Factores de riesgo

Un factor de riesgo es una condición que incrementa la posibilidad de que un suceso ocurra. Puede estar relacionado tanto con el inicio como con la progresión de la enfermedad. Así, el concepto de factor de riesgo está principalmente vinculado a la causa de una enfermedad. Numerosos estudios han demostrado que la presencia de biopelícula oral es un requisito previo para el desarrollo de enfermedades periodontales. Por ejemplo, la diabetes y el tabaquismo son verdaderos factores de riesgo para las enfermedades periodontales. En el contexto de las enfermedades crónicas, la exposición al factor de riesgo tiene un efecto indirecto sobre la probabilidad del evento. (22)

El conocimiento de las enfermedades periodontales ha permitido identificar diversos factores de riesgo, tales como el estilo de vida, las enfermedades sistémicas y la inflamación. Entre ellos, se ha destacado el impacto de la condición socioeconómica durante la infancia que puede influir negativamente en la salud bucal en la adultez. Además, el tabaquismo contribuye al desarrollo de la enfermedad periodontal al afectar a la respuesta inmunitaria, modificar la microbiota subgingival y dificultar la cicatrización de los tejidos, alterando así la homeostasis tisular. (2)

La cantidad de destrucción en los fumadores es mayor que en los exfumadores y los no fumadores, con un riesgo aproximadamente cuatro veces superior de desarrollar periodontitis. Además, los exfumadores son más susceptibles a la periodontitis que los que nunca han fumado.

Existe una relación dosis-dependiente entre el hábito de fumar y la periodontitis. Aproximadamente el 41.9% de los casos de periodontitis se atribuyeron al tabaquismo actual y el 10.9% al tabaquismo previo. Resultados similares se reportaron en estudios que investigaron los efectos del tabaquismo en la pérdida de inserción y en los niveles de la cresta alveolar. (3,16)

En los fumadores actuales y exfumadores se observan niveles elevados de periodontopatógenos en comparación con quienes nunca han fumado. El tabaquismo causa cambios cualitativos en la microflora subgingival, favoreciendo la presencia de periodontopatógenos, como *Aggregatibacter actinomycetemcomitans (A. actinomycetemcomitans)*, *Porphyromonas gingivalis* (*P. gingivalis*) y *Tannerella forsythia (T. forsythia)*. Además, se ha observado un mayor riesgo de infección por *Treponema denticola* (*T. denticola*) en bolsas periodontales. Resultados similares señalan que incluso en bolsas superficiales, los fumadores muestran una mayor colonización bacteriana, lo que sugiere que el tabaco crea un entorno favoranle para la aparición y progresión de la enfermedad periodontal, facilitada además por una respuesta inmune menos eficaz del huésped. (16)

La evidencia epidemiológica indica que la periodontitis es más común en personas con diabetes mellitus no controlada. Además, factores ambientales, junto con los genéticos, influyen en la respuesta inmunitaria y pueden favorecer la progresión de enfermedades inflamatorias crónicas como la periodontitis. Asimismo, los trastornos de salud mental se han relacionado con una mayor gravedad de esta enfermedad, tanto por cambios en los hábitos de higiene y estilo de vida como por la inflamación crónica de bajo grado asociada a estas condiciones.(2)

1.3. Tabaco y enfermedad periodontal

Como se ha mencionado anteriormente, hay varios estudios que demuestran asociación entre tabaco y enfermedad periodontal. (1)

Revisiones sistematizadas encuentran asociación positiva significativa entre el hábito de fumar tabaco y un mayor riesgo de desarrollar periodontitis en estudios longitudinales prospectivos. Además, se estima que la eliminación del tabaquismo podría reducir el riesgo de periodontitis en aproximadamente un 14%, según cálculos basados en la fracción atribuible al riesgo poblacional. (26)

Aunque los mecanismos exactos mediante los que el tabaquismo influye en la incidencia y progresión de la periodontitis aún no se comprenden completamente, se han propuesto diversas hipótesis. Entre ellas, se plantea que el tabaco podría afectar la composición de la microbiota oral (2,26), la respuesta inmune y la capacidad de cicatrización del periodonto (2,26,27). Se sugiere, además, que el hábito de fumar podría inducir cambios en el biofilm subgingival, favoreciendo un incremento en la prevalencia de patógenos periodontales.(2,26)

Los efectos nocivos del tabaco debilitan la función de los tejidos de soporte del diente, lo que provoca pérdida ósea, disminución de inserción y formación de bolsas periodontales, pudiendo llegar a la pérdida dental. Los fumadores presentan un riesgo significativamente mayor de sufrir estos problemas, con un riesgo de enfermedad periodontal entre 5 y 20 veces mayor en comparación con los no fumadores. En fumadores crónicos con alta exposición, el riesgo de enfermedad periodontal grave es equivalente al de desarrollar cáncer de pulmón. (27)

El tabaquismo afecta negativamente la salud periodontal al alterar la respuesta inmune, especialmente por el retraso en la acción de los neutrófilos y el aumento de su actividad destructiva. También incrementa los niveles de interleucinas (IL-1 e IL-6), favoreciendo la resorción ósea mediante el desequilibrio entre el receptor activador del factor nuclear-κβ (RANKL) y y su inhibidor osteoprotegerina (OPG). Además, eleva las concentraciones de elastasa y de metaloproteinasas de matriz (MMP-8 y MMP-9), mientras reduce los inhibidores de proteasas, lo que empeora la cicatrización. Estudios muestran un efecto acumulativo del tabaco, con mayor riesgo de periodontitis en quienes han fumado más tiempo o desde edades tempranas. (26)

Los estudios coinciden en que los fracasos terapéuticos y las recaídas de la enfermedad se observan predominantemente en fumadores, lo que sugiere que el tabaquismo interfiere con un resultado normal esperado tras las terapias periodontales habituales. (27)

1.4. Justificación

Conocer detalladamente los efectos adversos que provoca el tabaco en los tejidos de soporte del diente podrá ayudar a establecer planes preventivos y de tratamiento específicos para pacientes fumadores, cuya salud bucodental está más comprometida que aquellos que no lo son. Debido al impacto de la enfermedad periodontal a nivel oral y a la alta prevalencia de consumo de tabaco a nivel mundial, me veo en la obligación de realizar esta revisión.

2. OBJETIVO

¿En pacientes (P) fumadores (I) en comparación con los pacientes no fumadores (C), es la enfermedad periodontal más prevalente (O)

El objetivo de este trabajo es analizar si el consumo de tabaco en pacientes fumadores aumenta la prevalencia de padecer enfermedad periodontal frente a pacientes no fumadores.

3. MATERIAL Y MÉTODOS

Para la realización de esta revisión sistemática, se han utilizado medios de búsqueda como Pubmed, Medline complete y también se ha consultado la biblioteca CRAI Dulce Chacón, dentro de la cual se ha accedido a las plataformas de Academic Search Ultimate y Dentistry & Oral Science Source simultáneamente.

Para la búsqueda de los artículos, se ha utilizado una búsqueda avanzada en la que se han incluido operadores booleanos: ((((tobacco[MeSH Terms]) OR (smoking[MeSH Terms])) OR (cigarette smoking[MeSH Terms])) AND (periodontal disease[MeSH Terms])) OR (periodontitis[MeSH Terms])

Criterios de inclusión: publicaciones recientes (artículos publicados en los últimos 25 años), revisiones sistemáticas y metaanálisis, estudios transversales, de cohortes, casos y controles y ensayos clínicos sobre el tabaco y la enfermedad periodontal, publicaciones en inglés y español.

Criterios de exclusión: antigüedad (artículos publicados hace más de 25 años), artículos cuyo contenido no pueda visualizarse por completo, falta de relación con el tema del trabajo, estudios en animales.

4. RESULTADOS

Para la búsqueda de los artículos que se incluyeron en los resultados de este trabajo, se utilizó la Biblioteca CRAI Dulce Chacón, buscando simultáneamente en las bases de datos de Academic Search Ultimate y Dentistry & Oral Science Source. Con la búsqueda de (tobacco) AND (periodontitis) aparecieron 953 artículos, de los cuales se excluyeron 521 por tener más de 10 años de antigüedad. De los 432 restantes, se excluyeron 63 artículos duplicados. A continuación, se procedió a la revisión de cada uno de los 369 artículos restantes, descartando 345 de estos por no abordar el tema de interés. De los 24 artículos restantes, se realizó un análisis detallado cada uno de los artículos, de los cuales 10 fueron incluidos para la elaboración de los resultados de este trabajo.

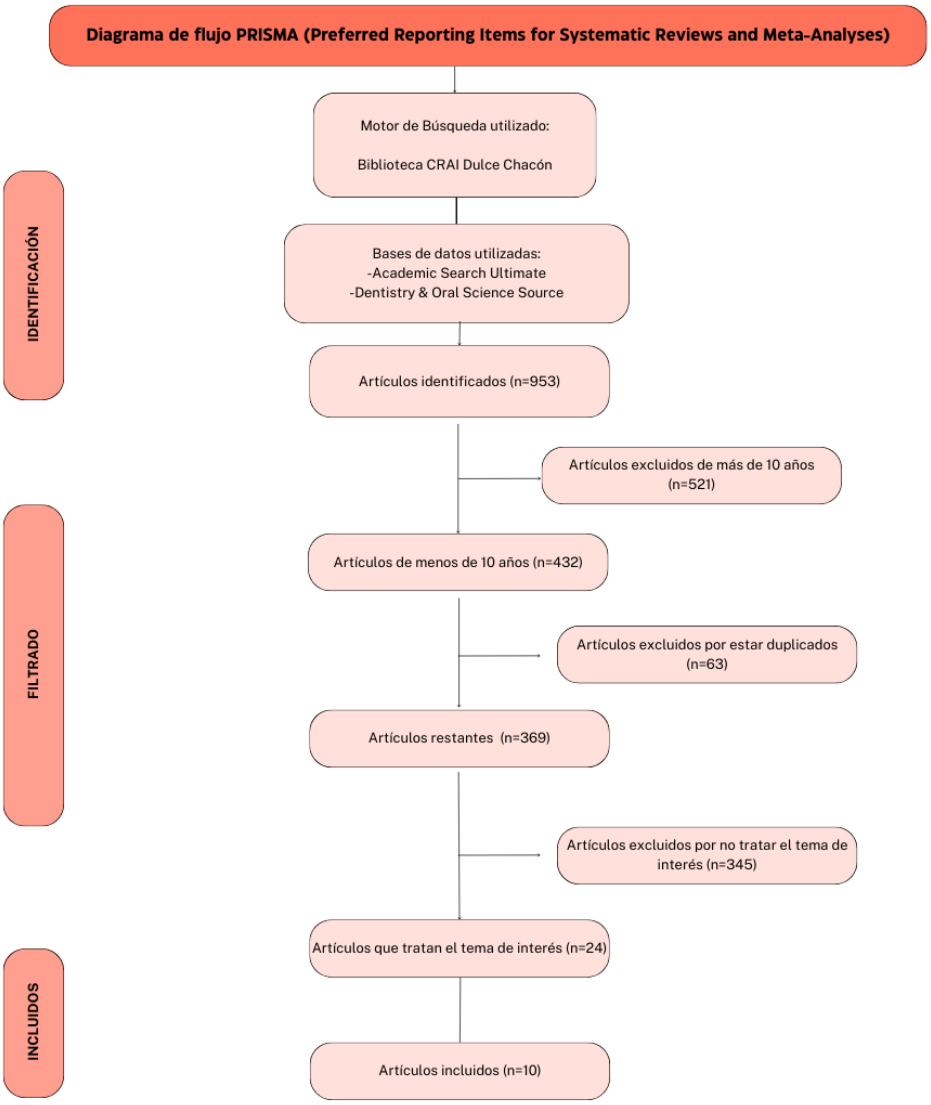

Figura 6. Diagrama de flujo PRISMA 2000. (28)

De los resultados de los presentes estudios, se incluyeron exclusivamente datos de sujetos que consumen tabaco de forma inhalada (casos) y de las personas que no lo consumen (controles). De este modo, se excluyeron aquellos hallazgos que examinan la influencia de otras formas de consumo de tabaco, así como los que evalúan el efecto de sustancias distintas al tabaco en el desarrollo o la progresión de la enfermedad periodontal. Los principales parámetros periodontales evaluados son la pérdida de inserción clínica (CAL), profundidad de sondaje (PD), sangrado al sondaje (BP), índice de placa (IP), índice gingival (IG), recesión gingival (RC), movilidad dental (M), pérdida de hueso marginal (MBL) y nivel de inserción (AL)

En cuanto al ecosistema oral, se han evaluado parámetros bioquímicos (iones como el tiocianato salival (SCN), proteínas como la E-cadherina, proteína C reactiva (prot-CR), IL-8 y MMP-8 y microbiológicos (bacterias como *T. denticola, P. gingivalis* o *A. actinomycetemcomitans).*

Tabla 3. Resultados de estudios incluidos.

Referencia	Casos	Controles	Tipo de estudio	Método de evaluación	Resultados
Hiral y cols. (29)	44	36	Observacional prospectivo	CAL, PD, BP con sonda CPI	CAL fue 1,3mm mayor en fumadores. PD: 4,47 ± 0,59mm en fumadores versus (vs.) 3,15 ± 0,58mm en no fumadores. BP mayor en no fumadores
Goel y cols. (30)	91	349	Observacional prospectivo	CAL y PD con sonda UNC-15	Periodontitis en 85,4% de fumadores vs. 59,6% en no fumadores. Periodontitis severa: 44,6% en fumadores, 15,7% en no fumadores
Mišković y cols. (31)	22	22	Observacional prospectivo	CAL, PD, IG, IP, RC, M, FD con sonda UNC-15	32% fumadores con periodontitis vs. 5% no fumadores. Mayores valores de IG, IP, M, FD en fumadores. PD: 3,42mm vs. 1,88mm; CAL: 3,6mm vs. 2,27mm. Riesgo 4,7 veces mayor por tabaco
Shah y cols. (32)	150	150	Observacional prospectivo	IP, IG y CAL con sonda UNC-15	Valores más altos en fumadores

Aldakheel y cols. (33)	15	15	Observacional prospectivo	PD, CAL, IP, IG, MBL con sonda UNC-15. PCR del biofilm	Más altos niveles de PD, CAL, MBL en fumadores. Mayor presencia de A. actinomycetemcomitans y P. gingivalis.
Ramesh y cols. (34)	15	30	Observacional prospectivo	Análisis inmunohistoquímico de E-cadherina	Menor expresión en fumadores, pero los resultados no son estadísticamente significativos
Hegde y cols. (35)	10	10	Observacional prospectivo	PD, CAL, IG. SCN por espectrofotometría	Fumadores con mayor SCN, PD y CAL
Kanmaz y cols. (36)	23	21	Observacional prospectivo	IP, BOP, PD, CAL con sonda Williams. Cotinina, IL-8, MMP-8 y patógenos periodontales	Fumadores con mayores valores de IL-8, MMP-8, PD, CAL. Mayor número de P. gingivalis, T. forsythia y T. denticola en fumadores
Aziz y cols. (37)	80	51	Observacional prospectivo	CAL, PD, IG, IP con sonda UNC-15. Biomarcadores inflamatorios y estrés oxidativo en sangre	Mejora general, pero respuesta limitada en fumadores

Waseem y cols. (38)	38	62	Observacional prospectivo	PD, IG, BP, AL con sonda CPITN. Prot-CR en sangre	Prot-CR mayor en fumadores. Mejora tras tratamiento en ambos grupos

5. DISCUSIÓN

El papel del hábito tabáquico como un factor de riesgo de la enfermedad periodontal está ampliamente documentado.

Los resultados indican que el tabaquismo tiene un impacto negativo significativo en la salud periodontal, evidenciado por mayores niveles de CAL, PD e IP en fumadores en comparación con no fumadores. Además, los fumadores presentan una mayor prevalencia de bacterias periodontopatógenas como *P. gingivalis*, *T. forsythia* y *T. denticola*, así como niveles elevados de biomarcadores inflamatorios (IL-8, MMP-8, prot-CR) y estrés oxidativo, lo que sugiere un ambiente oral más propicio para la progresión de la periodontitis. A pesar de que el tratamiento periodontal mejora los parámetros clínicos en ambos grupos, los fumadores muestran una respuesta menos efectiva.

Hiral y cols. (29) evidenciaron que los fumadores tienen mayores niveles de CAL y PD en comparación con los no fumadores, lo que coincide con los hallazgos de Goel y cols. (30), quienes reportaron una prevalencia significativamente mayor de periodontitis en fumadores frente a no fumadores, demostrando así una asociación entre tabaquismo y enfermedad periodontal, lo cual apoya la hipótesis de este trabajo. Estos resultados se ven reforzados por el estudio de casos y controles de Calsina y cols. (39), en el que los fumadores también presentaron mayores valores de CAL y PD, además de mayor prevalencia de RC. Los resultados de este último estudio concluyeron que los fumadores tenían 2,7 veces más probabilidades de desarrollar periodontitis en comparación con los no fumadores.

Siguiendo la línea de la hipótesis de este trabajo, el estudio de Kham y cols. (40) también reporta una mayor prevalencia de periodontitis crónica en pacientes fumadores en comparación con los no fumadores. De acuerdo con los resultados del estudio de Tomar y cols. (41), fumar podría ser la causa de más de la mitad de los casos de periodontitis en adultos en los Estados Unidos, habiendo más casos de la enfermedad en fumadores.

Asimismo, el estudio de Mišković y cols. (31) refuerza esta relación, concluyendo que los fumadores de cigarrillos presentan peores condiciones periodontales (tanto en comparación de no fumadores como de fumadores de sistemas de tabaco calentado (IQOS)). Del mismo modo, el estudio de Amarasena y cols. (42) muestra que los consumidores de tabaco (tanto fumado como mascado) presentan mayor pérdida de inserción epitelial que los no fumadores (de 1,38mm y 1,47mm respectivamente).

Otras revisiones de diferentes estudios muestran una abrumadora consistencia entre los resultados de los estudios, indicando que la condición de salud periodontal de los fumadores es significativamente inferior a la de los no fumadores. (43)

Shah y cols. (32) aportan evidencia adicional al observar que los fumadores de más de 10 cigarrillos diarios tenían valores estadísticamente significativos mayores de índices periodontales (IP, IG Y CAL) que los que fumaban menos de 10 cigarrillos diarios, lo que concluye que la periodontitis se ve agravada a mayor consumo del tabaco. De igual manera, el estudio de Khan y cols. (40) concluye que la gravedad de la enfermedad periodontal aumenta con la cantidad y la duración del hábito tabáquico.

Siguiendo esta tendencia, el estudio de Calsina y cols. (39) encontró que los fumadores mostraron mayores PD, RC y CAL en comparación con no fumadores. Sin embargo, presentaron menor BP, posiblemente debido a una respuesta inflamatoria vascular reducida causada por la vasoconstricción inducida por la nicotina. M fue mayor en fumadores, relacionada con la pérdida avanzada de hueso alveolar, aunque las diferencias no fueron significativas. El IP no tuvo diferencias significativas entre fumadores y no fumadores. El tabaquismo durante más de 10 años aumentó aumentó 2.7 veces la probabilidad de desarrollar periodontitis establecida, y 2.3 veces en los exfumadores, además de aumentar significativamente PD, RC y CAL.

Del mismo modo, la evidencia obtenida de estudios transversales y longitudinales confirma de manera sólida que el riesgo de desarrollar enfermedad periodontal, evaluado a través de la pérdida de inserción clínica y la pérdida ósea alveolar, se incrementa con el aumento del consumo de tabaco. Las investigaciones indican que los exfumadores (aquellos que han dejado de fumar por un período de al menos dos años) presentan una menor pérdida de inserción en comparación con los fumadores actuales, aunque mayor que la observada en personas que nunca han fumado. Asimismo, la gravedad de la enfermedad periodontal parece estar directamente relacionada con la cantidad de tabaco consumido. (44)

Por otro lado, Aldakheel y cols. (33) demostraron que los fumadores presentan niveles significativamente más altos de bacterias periodontopatógenas como *A. actinomycetemcomitans* y *P. gingivalis*, además de mayores valores de PD, CAL y MBL, lo que sugiere que el tabaco no solo influye en la inflamación, sino también en la microbiota oral. Estos hallazgos coinciden con los de Kanmaz y cols. (36), quienes también evidenciaron mayor prevalencia de bacterias periodontopatógenas, en este caso *P. gingivalis*, *T. forsythia,* y *T. denticola*.

Sin embargo, la influencia del tabaco en la microbiota oral sigue siendo un tema controversial. Estudios basados en métodos de cultivo tradicionales y técnicas moleculares han reportado resultados contradictorios respecto a su efecto sobre la presencia de patógenos clave (como *P. gingivalis, T. forsithya* y *T. denticola*), mostrando que no hay diferencias significativas en las concentraciones de bacterias patógenas en fumadores respecto a no fumadores. No obstante, investigaciones recientes utilizando análisis metagenómicos han revelado cambios más profundos en la composición bacteriana de los fumadores, incluso en ausencia de enfermedad periodontal. (45,46)

Desde el punto de vista bioquímico, se ha observado que el tabaco induce cambios en la composición química de la cavidad oral, lo que contribuye al desarrollo y progresión de la periodontitis. Kanmaz y cols. (36) encontraron niveles elevados de IL-8 y MMP-8 en fumadores, lo que indica un incremento en la respuesta inflamatoria y en la degradación del tejido conectivo. Hedge y cols. (35) también evidenciaron niveles elevados de SCN en saliva de fumadores con periodontitis.

También se ha encontrado que los fumadores muestran una reducción en diversas citocinas y quimiocinas proinflamatorias, además de una disminución en algunos reguladores de células T y células *natural killer* (NK). (44)

Por otro lado, el tabaquismo impacta el periodonto al inducir también alteraciones en los ácidos grasos 3-OH del lípido A, lo que se asocia con una microflora oral de menor potencial inflamatorio. Estudios muestran que los fumadores tienen niveles reducidos de estos ácidos grasos en comparación con no fumadores con periodontitis crónica, explicando la paradoja de mayor infección periodontal pero menor inflamación clínica en fumadores. Además, el humo del tabaco afecta a *P. gingivalis*, incrementando la expresión del antígeno fimbrial principal (FimA) y promoviendo la formación de biopelículas menos proinflamatorias. Esto podría deberse a adaptaciones de *P. gingivalis* frente al estrés ambiental del humo, alterando factores de virulencia y reduciendo su capacidad inflamatoria. (44)

Diferentes estudios han evaluado la influencia de la cotinina en la enfermedad periodontal, que es el principal metabolito de la degradación de la nicotina. Kanmaz y Jiang evaluaron su influencia. Desde el punto de vista microbiológico, ambos evaluaron la presencia de *P. gingivalis* y *T. denticola*. Mientras que el primer estudio sugiere que la cotinina puede promover la adhesión e invasión de *P. gingivalis* en las células epiteliales, la revisión de Jiang encontró que los fumadores tenían una mayor prevalencia de *T. denticola*, aunque las diferencias no fueron estadísticamente significativas. Esto sugiere que la exposición al tabaco podría no solo favorecer

la colonización de ciertos patógenos, sino también modificar la dinámica microbiana en el entorno subgingival. (36,46)

En términos de inflamación y degradación tisular, ambos estudios analizaron niveles de mediadores inflamatorios. Kanmaz y cols. (40) encontraron niveles más bajos de IL-8 y MMP-8 en fumadores en comparación con los no fumadores, aunque las diferencias no fueron estadísticamente significativas. Esto sugiere que el tabaco podría suprimir la respuesta inflamatoria del huésped, lo que explicaría la menor tendencia al sangrado en fumadores. Esta observación coincide con el estudio de Jiang, (42) que también señala que la cotinina puede reducir la producción de superóxido por los neutrófilos en respuesta a *P. gingivalis*, lo que compromete la capacidad del sistema inmune para controlar la infección.

Investigaciones in vitro también han evidenciado cambios en los niveles de citocinas inflamatorias en el fluido crevicular gingival, alteraciones en la función de las células del sistema inmune y modificaciones en la regulación proteolítica en personas fumadoras. (44)

En términos de respuesta al tratamiento periodontal, Aziz y cols. (37) demostraron que los fumadores responden de manera menos efectiva al tratamiento periodontal, presentando menor reducción en la inflamación y el estrés oxidativo tras el SPR. De manera similar, Waseem y cols. (38) encontraron que los fumadores presentaban niveles de prot-CR antes y después del tratamiento periodontal, lo que confirma el impacto del tabaquismo en la inflamación sistémica y su influencia en la respuesta al tratamiento periodontal.

Los mismos resultados encuentran la revisión de Jiang y cols. (46), los cuales concluyen que el hábito de fumar tiene un impacto negativo en la efectividad del tratamiento periodontal. Aunque tanto fumadores como no fumadores muestran mejoras en los parámetros clínicos tras la terapia, en los fumadores la disminución de bacterias periodontopatógenas es menos significativa y los patógenos tienden a persistir en mayor medida. Además, las personas que fuman presentan una mayor predisposición a la reaparición de biopelículas patogénicas después del SRP.

También apoyan estos resultados la revisión de Leite y cols. (47), quienes encuentran que el SPR beneficia a la mayoría de los participantes, pero el efecto del tratamiento se ve influenciado por la exposición al tabaquismo de manera dosis-dependiente. Los fumadores moderados y los que dejaron de fumar mostraron mejoras en los parámetros periodontales, mientras que los fumadores intensos no experimentaron beneficios significativos.

De igual manera, Borojevic y cols. (44) afirman que el tratamiento periodontal suele ser menos efectivo en fumadores en comparación con los no fumadores. Las investigaciones que analizan la eficacia del control de la enfermedad periodontal y de procedimientos específicos, como los regenerativos, los injertos de tejidos blandos y los relacionados con implantes, han demostrado de forma consistente que el tabaquismo tiene un impacto negativo en las tasas de éxito.

6. CONCLUSIONES

Los datos sugieren que, aunque el tabaquismo agrava significativamente el estado periodontal y favorece formas más severas de periodontitis, y aunque algunos estudios muestren mayor prevalencia de la enfermedad periodontal en fumadores, no puede afirmarse categóricamente que la prevalencia sea mayor en fumadores. La enfermedad periodontal es multifactorial y depende de diversos factores como la higiene oral, el acúmulo de placa, condiciones sistémicas, factores genéticos y sociales. Por tanto, el tabaquismo debe considerarse un factor modificador más que un factor causal exclusivo.

Sería esencial fomentar la realización de más estudios para determinar si la prevalencia de enfermedad periodontal es mayor en fumadores, en concreto ensayos clínicos aleatorizados, ya que constituyen el estándar más alto de evidencia científica en el ámbito clínico. Estos estudios permitirían determinar con mayor certeza si existe realmente una mayor prevalencia de esta patología en pacientes fumadores frente a los no fumadores. La generación de este tipo de evidencia es clave para orientar estrategias de prevención y tratamiento más eficaces y personalizadas en función del hábito tabáquico.

7. SOSTENIBILIDAD

El abordaje de la relación entre el tabaquismo y la enfermedad periodontal debe alinearse con los Objetivos de Desarrollo Sostenible (ODS) de la Organización de las Naciones Unidas (ONU), promoviendo estrategias que beneficien la salud global de manera sostenible.

1. ODS 3: Salud y bienestar. El trabajo se enfoca en la prevención de la enfermedad periodontal relacionada con el tabaquismo, promoviendo la cesación tabáquica y el acceso a tratamientos.
2. ODS 7. Energía asequible y no contaminante. El uso de herramientas digitales para elaborar el trabajo implica un consumo eficiente de electricidad, contribuyendo a un uso responsable de la energía y el acceso a fuentes sostenibles.
3. ODS 10: Reducción de las desigualdades. Es fundamental que los programas de salud bucal y abandono del tabaco estén disponibles para todas las personas, reduciendo las desigualdades y mejorando el bienestar de las poblaciones vulnerables.
4. ODS 12: Producción y consumo responsables. El tabaquismo impacta negativamente en la salud y en el medio ambiente. Reducir su consumo promueve un consumo responsable y conciencia sobre los efectos ambientales.
5. ODS 13: Acción por el clima. La adopción de prácticas sostenibles en odontología, como el uso de materiales ecológicos y tecnologías que minimicen residuos, reduce el impacto ambiental del sector.

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9. ANEXOS

9.1. Anexo 1. Bibliografía

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DOI: 10.1111/prd.12395

REVIEW ARTICLE

Periodontology 2000 WILEY

Oral and periodontal implications of tobacco and nicotine products

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Funding information
United States National Institutes of Health, Grant/Award Number: U54HL147127; California Department of Public Health, Grant/Award Number: 17-10592; Richard Holliday is funded by a UK National Institute for Health Research (NIHR) Clinical Lectureship

Editor: Yvonne Kapila

1 | INTRODUCTION

Tobacco use is responsible for nearly 9 million annual global deaths (approximately 15% of all deaths worldwide). This is more than any other behavioral risk factor and trailing only high systolic blood pressure among all risk factors in its contribution to human mortality.[1] Tobacco smoke disrupts the functioning of nearly every human organ system, causing most deaths through cancer, heart disease, and noncancer respiratory diseases.[2] Health risks extend not only to the person using tobacco but to people involuntarily exposed to smoke (second-hand smoking).[3] Tobacco experimentation typically begins in adolescence, often due to both social influences and tobacco marketing.[4] Later in life, most adult tobacco users find themselves chemically and/or behaviorally dependent on nicotine and unable to quit tobacco use.[2] Yet, despite this well-chronicled destruction, industrially produced tobacco products remain legally sold and marketed in nearly every country, well surpassing US$1 trillion in annual sales.

Owing to combined efforts of public messaging, excise taxes, social norm shifting, and numerous other tobacco control strategies, cigarette smoking prevalence in most high-income countries has declined dramatically in recent decades.[2,5-8] However, as the current number of global deaths would indicate, substantial challenges remain. China, by far, is the single largest consumer of cigarettes and where smoking prevalence, especially among men, remains persistently high.[9] Though smoking prevalence in Africa has historically been low, aggressive tobacco industry efforts on the continent have health experts projecting increased tobacco use over the coming decades.[10] In countries where smoking prevalence has declined, inequalities in tobacco use and cessation have often risen, marked by widened gaps according to socioeconomic disadvantage,[11,12] race/ethnicity,[12] and mental illness,[13] among other factors, exacerbating health inequity.

The most recent decade has also seen an expanding variety of new or emerging tobacco and/or nicotine products brought to market, most prominently electronic cigarettes (commonly called e-cigarettes). Heated tobacco products[14] and nicotine-containing pouches[15] are other examples of an increasingly diverse product landscape. Meanwhile, more permissive laws and regulations have broadened access to cannabis (marijuana) products. Cannabis, though not a tobacco product, is frequently consumed in combination with tobacco and by individuals who also use tobacco.[16,17] Smoke from cannabis products shares many chemical properties with tobacco smoke and has been linked to health problems, including potential cardiovascular[18] and respiratory[19,20] impairment.

For the practicing clinician, providing sound patient recommendations requires knowledge of the general and oral health implications not only associated with smoking cigarettes but also with

[Correction added on 06 September 2021, after first online publication: The copyright line was changed.]

(2) Darby I. Risk factors for periodontitis & peri-implantitis. Periodontol 2000. octubre de 2022;90(1):9-12.doi:10.1111/prd.12447. https://pubmed.ncbi.nlm.nih.gov/35913624/

DOI: 10.1111/prd.12447

REVIEW ARTICLE

Periodontology 2000 WILEY

Risk factors for periodontitis & peri-implantitis

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1 | INTRODUCTION

What is risk? Risk has a number of definitions depending on the point of view or profession from exposure to danger, interaction with uncertainty to the potential for loss in insurance or finance terms. However, in the medical and dental fields it generally means the chances of getting some form of illness.

The management of noncommunicable disease is fundamentally about managing risk and risk factors, both modifiable and nonmodifiable, to prevent either initiation or progression of the disease. A risk factor is a known variable that has a direct detrimental effect by enhancing the disease process or increasing the likelihood of a disease developing.[1] Risk indicators, on the other hand, may be risk factors, but the data are weaker and associated with cross-sectional studies. Risk factors/indicators will be different for every patient, and it is likely that the progression is a result of the interaction of many risk factors, some of which we know about and some of which are still to be discovered. An example of this over the last 20 years is the emerging evidence of the effect of inflammation in mental health disorders and the development of neuro-immunology.[2]

In the same time frame, the understanding of periodontal diseases has improved with identification of more risk factors, such as lifestyle, the interaction with systemic diseases, and the role of the inflammatory burden. The new classification of periodontal disease directly assesses risk and has been left open for the addition of further factors over and above smoking and diabetes.[3] This volume looks at a number of risk factors, known and emerging.

The development of dental implants has revolutionized dentistry. Millions of dental implants are placed each year, and a great number will develop biological complications, including inflammation and bone loss. When we plan for implant outcomes, we are really planning for many years ahead. Getting it "right" at the start is vital to optimal outcomes. It is clear that there are risk factors for peri-implant diseases, but these are less well understood compared with periodontal risk factors. This volume also looks at the known and emerging risk factors for dental implant disease. In addition, the operator placing the implant may also be a risk, which is known, but perhaps under-reported or poorly understood. The clinician also needs to be aware of the materials used and their possible host interactions.

Socioeconomic status has been implicated as a risk indicator for several chronic diseases including periodontitis.[4] Multivariate analysis of data from large databases and cohort studies have demonstrated that socioeconomic status indicators such as income and education have an independent association with periodontitis. Since behavioral factors, such as smoking, only partially explain the association between socioeconomic status adversity and periodontitis, socioeconomic status may be proposed as a true risk factor for periodontitis. Cumulative biologic effects of psychosocial and physical stress during an individual's lifetime, known as allostatic load, have been suggested as an underlying plausible mechanism for the interrelationship between low socioeconomic status and periodontitis. The results of studies on socioeconomic status mobility showed that the childhood socioeconomic status could have important and possibly irreversible impacts on adulthood oral health. In addition, the effect of socioeconomic status on periodontal health might be further complicated by interactions with factors such as sex, race/ethnicity, culture, and occupation. Future investigation of association between socioeconomic status and periodontal health seems warranted to identify high-risk groups and to design public health policies.

Tobacco smoking has been implicated in periodontal pathology through various mechanisms, including perturbations of the inflammatory and host responses to putative periodontal pathogens, alterations in the subgingival microbial communities, and a compromised healing potential of the tissues leading to an imbalance of tissue homeostasis.[5] Critical appraisal of study limitations is required, especially the differences in study protocol designs of (a) early and more recent studies exploring cigarette smoking–induced changes

(3) Alwithanani N. Periodontal Disease and Smoking: Systematic Review. J Pharm Bioallied Sci. julio de 2023;15(Suppl 1):S64-71. doi:10.4103/jpbs.jpbs_516_22. https://pubmed.ncbi.nlm.nih.gov/37654319/

Systematic Review

Periodontal Disease and Smoking: Systematic Review

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ABSTRACT

Introduction: Tobacco has been linked multiple times to many health implications. The relationship between periodontitis and tobacco was thoroughly investigated in this systemic review to evaluate if tobacco specifically smoking impacts the progression of periodontal through impairing vascular and immunity mediators processes. **Materials and Methods:** The manual and electronic literature searches up to 2020 in the databanks of the EMBASE, MEDLINE, PUBMED, and SCOPUS were conducted. The search terms were "periodontitis," "periodontitis diseases," "smoking," "tobacco use," "tobacco," and "cigarette, pipe, and cigar." The types of studies included were restricted to the original studies and human trials. Analyses of subgroups and meta-regression were used to calculate the heterogeneity. **Results:** 15 papers total were considered in the review, however only 14 of them provided information that could be used in the meta-analysis. Smoking raises the incidence of periodontitis by 85% according to pooled adjusted risk ratios (risk ratio 1.845, CI (95%) =1.5, 2.2). The results of a meta-regression analysis showed that age, follow-up intervals, periodontal disease, the severity of periodontitis, criteria used to determine periodontal status, and loss to follow-up accounted for 54.2%, 10.7%, 13.5%, and 2.1% of the variation in study results. **Conclusion:** Smoking has an undesirable impact on periodontal incidence and development. Therefore, when taking the history of the patients at the initial visits the information about the habit of smoking has to be thoroughly noted.

KEYWORDS: *Frequency, oral diseases, periodontitis, smoking*

Submitted: 25-Oct-2022
Revised: 04-Jan-2023
Accepted: 03-Mar-2023
Published: 05-Jul-2023

INTRODUCTION

The consumption of tobacco in the form of smoking is recognized as a substantial risk factor for chronic non-communicable ailments. The majority of deaths worldwide are now caused by diseases associated with smoking.[1,2] In spite of a decline in tobacco usage, estimations indicate that 10% of all fatalities in 2020 will be attributable to smoking.[1] The overall tax revenue from tobacco products is exceeded by health costs associated with cigarette-related ailments.[1] As a result, both individuals and healthcare systems are significantly burdened by the size of tobacco-related expenditures.

The global burden of chronic diseases includes periodontitis since it is a chronic, inflammatory, destructive disorder that affects the tooth's supporting components.[3] Periodontitis eventually results in the loss of a tooth. Oral diseases have an effect on mastication, quality of life, speech, and consequently self-esteem.[4] Dental caries is on the decline, although the severity of periodontitis has not changed since 1990.[3] There are about 700 million cases of severe periodontitis worldwide, according to a meta-analysis of the condition's prevalence.[3] The prevalence of periodontitis is anticipated to rise as a result of longer life expectancies and a marked decline in loss of the tooth caused by caries. We take it for granted that cigarette use and periodontitis are related.

Nevertheless, the preponderance of the data comes from studies that are designed as cross-sectional, which makes it impossible to establish temporal connections. Furthermore, numerous epidemiologic studies on the

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Access this article online
Quick Response Code:
Website: https://journals.lww.com/jpbs
DOI: 10.4103/jpbs.jpbs_516_22

How to cite this article: Alwithanani N. Periodontal disease and smoking: Systematic review. J Pharm Bioall Sci 2023;15:S64-71.

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Efectos tóxicos del tabaco

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Recibido 1 de Octubre de 2003 / Aceptado 29 de Noviembre de 2004

Resumen: A partir de los años 40 se inician investigaciones para relacionar el tabaquismo con la aparición de determinadas enfermedades, principalmente respiratorias y pulmonares. Desde entonces se genera una cascada de información epidemiológica y médica, que termina por considerar el consumo de tabaco como un problema de salud mundial. Como protección y promoción de la salud, la OMS insta a los gobiernos para que desarrollen programas específicos antitabaco. En este artículo se hace una revisión sobre la toxicidad de los componentes químicos del tabaco y los estudios más actuales sobre sus efectos en el organismo.

Palabras clave: Tabaco, nicotina, toxicidad, fumador activo, fumador pasivo.

Abstract: Tobacco toxic effects. As of 1940, investigations begin to relate nocotinism with the appearance of certain illnesses, mainly respiratory and pulmonary diseases. subsequently, a cascade of epidemic and medical information is generated and concludes that the consumption of tobacco is a universal problem. in order to promote and protect health, the oms urges the governments to develop specific antitobacco programs. in this article reviews the toxicity of the chemical components of tobacco and the most current studies regarding the effects of said components on the organism.

Key Words: Tobacco, nicotine, toxicity, smokers, passive smoking.

Introducción

En la elaboración del tabaco se utiliza la hoja de *Nicotiana tabacum* de la que existen cuatro variedades: *brasiliensis, havanensis, virginica y purpúrea*. El tabaco recolectado se mezcla con diferentes sustancias aromatizantes, y se expone al aire o calor artificial. A la hoja obtenida se le añaden aditivos para mejorar el sabor y otras características y se trocea. Esta mezcla se envasa dentro de un cilindro de papel al que se le coloca en un extremo un filtro de celulosa, de mayor o menor porosidad, y que puede, además, contener otros materiales como carbón vegetal, etc [1,2].

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Estudios epidemiológicos han demostrado la mayor incidencia de determinadas enfermedades en personas con el hábito de fumar y en fumadores pasivos [3], y de una mayor morbi-mortalidad. De ahí la importancia del reciente Plan Nacional de Prevención y Control del Tabaquismo [4]. En líneas generales este Plan pretende actuar a diferentes niveles [5]: prevención, control de lugares donde se fuma y potenciación del abandono del tabaco por lo que se financiarán algunos tratamientos farmacológicos para la deshabituación.

Componentes químicos del humo del tabaco

Las sustancias químicas contenidas en las hojas del tabaco son las precursoras de las más de 4000 sustancias que aparecerán en el humo de la combustión, el cual se divide en dos fases:
fase gaseosa y fase sólida o de partículas. La separación de las fases se realiza pasando el humo del tabaco por un filtro tipo Cambridge, formado por agujas de vidrio muy finas que retienen las partículas dejando pasar la fase gaseosa [1]. Posteriormente se identifican las sustancias con espectrometría de masas, cromatografía gaseosa, etc, cuantificando resultados incluso en ng/ml [6]. Algunos de los componentes identificados en la fase gaseosa son los siguientes:

CO, CO_2, acetona, acetonitrilo, acetileno, NH_3, dimetilinitrosamina, HCN, metano, propano, piridina, metil clorhidrato, metil furano, NO_X, nitrospirrolidina, propionaldehido, 2-butano, 3-picolina, 3-binilpiridina, etc. De la fase de partículas se han aislado: nicotina, anilina, benzopireno, catecola, hidracina, naftalina, metil naftalina, metil quinolinas, NNK, fenol, pireno, quinolona, stigmasterol, tolueno, "brea", 2-naftilamina, 4-aminopifenil, etc.

Se observan variaciones cuantitativas de los componentes en los diferentes tipos de cigarros, debido a características del propio cigarro, tipo de filtros, factores de producción , uso de fertilizantes, métodos analíticos, etc. La International Agency for Research on Cancer (IARC) ha incluido algunos agentes químicos procedentes del humo del tabaco en el "Grupo I de carcinógenos humanos": benceno, Cd, As, Ni, Cr, 2-naftil-amino, cloro vinil, 4 aminobifenil y Be. Cuando se usan los piretroides como insecticidas en el cultivo del tabaco, algunos residuos de estos componentes pueden aparecer en el humo del cigarrillo [7,8]. En la tabla 1 se resumen las características de algunos componentes del humos del cigarrillo [9].

Toxicocinética del humo

La combustión del tabaco origina dos corrientes:

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Clínica e Investigación en Arteriosclerosis 34 (2023) 330–338

Sociedad Española de Arteriosclerosis

CLÍNICA E INVESTIGACIÓN EN ARTERIOSCLEROSIS

www.elsevier.es/arterio

REVIEW ARTICLE

Update on new forms of tobacco use

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Received 21 February 2022; accepted 16 March 2022

KEYWORDS
Tobacco use;
Strategy;
Heat-not-burn tobacco;
E-cigarette;
Snus

Abstract Smoking remains the leading cause of morbidity and mortality worldwide. Because of its clear influence on cardiovascular and respiratory diseases, it is an important factor in internal medicine consultations. Although the rate of smoking cessation has been increasing in recent years, there is a percentage of patients who continue to smoke because they are unable or unwilling to quit, despite having tried existing pharmacological and non-pharmacological therapies. For this group of patients there are strategies based on interventions aimed at reducing the negative effects of smoking without the need for complete cessation. In this review it is shown that due to the absence of combustion of organic matter in conventional cigarettes, snus, e-cigarettes and heated tobacco products generate significantly lower levels of toxic substances.

PALABRAS CLAVE
Tabaquismo;
Estrategia;
Tabaco calentado;
Cigarrillo electrónico;
Snus

Actualización sobre las nuevas formas de consumo de tabaco

Resumen El tabaquismo sigue siendo la principal causa de morbimortalidad a nivel mundial. Por su clara influencia en las enfermedades cardiovasculares y respiratorias es un factor importante en la consulta de medicina interna. Aunque la tasa de abandono del hábito tabáquico esta ascendiendo en los últimos años, existe un porcentaje de pacientes que continúan fumando porque no pueden o no quieren cesar el hábito, a pesar de haber probado las terapias

DOI of original article: https://doi.org/10.1016/j.arteri.2022.03.004
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INVITED REVIEW SERIES: TOBACCO AND LUNG HEALTH

History of tobacco and health

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History of tobacco and health
MUSK AW, DE KLERK NH. *Respirology* 2003; **8**: 286–290

Abstract: Tobacco comes from plants that are native to the Americas around Peru and Ecuador, where it has been found since prehistoric times. It was brought back to Europe by early explorers where it was adopted by society and re-exported to the rest of the world as European colonization took place. Smoking tobacco in pipes of one sort or other gave way to handmade and then manufactured cigarettes, especially during the First World War. Smoking rates increased dramatically during the 20th century in developed countries until recently and rates are still increasing in underdeveloped countries. An epidemic of smoking-related diseases has followed the prevalence of smoking. Scientific knowledge of the harmful effects of active tobacco smoking has accumulated during the past 60 years since early descriptions of the increasing prevalence of lung cancer. The first epidemiological studies showing an association between smoking and lung cancer were published in 1950. In 1990 the US Surgeon General concluded that smoking was the most extensively documented cause of disease ever investigated but governments worldwide have been ambivalent and slow in taking action to reduce smoking. Tobacco smoking is now agreed to be a major cause of a vast number of diseases and other adverse effects. Since the 1980s passive smoking including exposure *in utero* has also been implicated as a significant cause of numerous diseases. In response, the tobacco industry has managed to forestall and prevent efforts to control this major health problem.

Key words: adverse effects, cigarettes, smoking, tobacco.

BACKGROUND

Tobacco and mankind have been associated in the same way as food and tea since before history began. Its ancient origins and how it subsequently insinuated itself into modern society have been described in detail by Gately.[1] *Nicotiana tabacum and Nicotiana rustica* are native plants of the Americas having evolved in the Andes around Peru/Ecuador. Men came across them (along with more useful plants such as tomatoes, potatoes, maize, cocoa and rubber) about 18 000 years ago when they migrated to the American continents from Asia across the Bering Straight land bridge. Tobacco is thought to have been cultivated since about 5000–3000 BC. The use of tobacco was universal throughout the American continents (and Cuba) by the time that Christopher Columbus arrived in North America in 1492.[1]

The practice of smoking appears to have arisen from snuffing, as snuffing instruments are among the most ancient tobacco-related artifacts that have been found. However, tobacco was not only sniffed and smoked but chewed, eaten, drunk (like tea), smeared over bodies (to kill lice and other parasites), and used in eye drops and enemas. It was blown into warriors' faces before battle, over fields before planting (it is still used as an insecticide in agriculture) and over women before sex. It was used medicinally for its analgesic and antiseptic properties and as a cure for a variety of ailments. It was offered to the gods and used in religious ceremonies. It had both real and mystical qualities. All sorts of implements were invented and used to administer it but the most enduring method of administration ever since these distant times has been smoking. Tobacco was smoked rolled up in cigars but the most popular method in ancient times

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Environmental Research 185 (2020) 109413

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Development of a standardized new cigarette smoke generating (SNCSG) system for the assessment of chemicals in the smoke of new cigarette types (heat-not-burn (HNB) tobacco and electronic cigarettes (E-Cigs))

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ARTICLE INFO

Keywords:
Standardized new cigarette smoke generating (SNCSG) system
Heat-not-burn (HNB)
Tobacco
Electronic cigarette (E-cig)
Carbonyl group

ABSTRACT

To systematically regulate new types of cigarettes for which their safety has yet to be verified, such as heat-not-burn (HNB) products and electronic cigarettes (E-Cigs), the identification of chemicals in the new cigarette smoke is necessary. However, this is challenging due to the large number of new cigarette types and their different vaporization approaches. To address this issue, we herein report the development of a standardized new cigarette smoke generating (SNCSG) system based on heating-temperature control, which is able to generate smoke for all types of new cigarettes. Validation of the developed system was also carried out through analysis of the carbonyl compounds (e.g., formaldehyde and acetaldehyde) in the new cigarette smoke of HNB products and E-Cigs generated by the SNCSG system under different heating temperatures. The analytical results were used to validate the SNCSG system by comparison with those of previous studies. In all new cigarette smoke samples, the formaldehyde and acetaldehyde concentrations increased dramatically upon increasing the heating temperatures, especially over the reference heating range of each HNB device (mean concentration (μg/cigarette, n = 5 (HNB and E-Cig samples)): formaldehyde = 0.373–5.841 (250–320 °C), and acetaldehyde = 0.088–27.60 (250–320 °C). In the case of the HNB samples, the concentration differences determined by the heating temperatures of the tobacco stick were statistically significant, with p-values (ANOVA) of 1.85E−10 (formaldehyde) and 1.73E−08 (acetaldehyde). In the majority of smoke samples, acrolein and propionaldehyde were detected under relatively high heating temperature conditions (> 250 °C) at 0.50 ± 1.76 μg/(cigarette or 10 μL), while acetone was detected under low heating temperature conditions (< 250 °C) at 0.09 ± 0.17 μg/(cigarette or 10 μL). These results indicate that the developed SNCSG system could be suitable for application in the regulation of new types of cigarettes, regardless of the cigarette type and heating approach.

1. Introduction

Conventional cigarette smoke generated by combusting tobacco at 700–950 °C contains thousands of hazardous compounds, including nicotine and polycyclic aromatic hydrocarbons (Busch et al., 2012; Cai et al., 2003; Kim and Kim, 2015). For the purpose of replacing conventional cigarettes as well as reducing their toxic effects, new cigarette types, such as heat-not-burn (HNB) tobacco and electronic cigarettes (E-Cigs) have been introduced (Dai et al., 2018; Murphy et al., 2018; Uchiyama et al., 2018), whereby the cigarette aerosols of these products are generated by different heating approaches. More specifically, in the case of HNB products, the HNB aerosols are produced by the heating of a tobacco stick using HNB devices. In contrast, E-Cig aerosols are generated from the E-Cig refill solution in the electronically heated cartomizer (atomizer + cartridge).

It is well known that the replacement of conventional cigarettes with new cigarette types, such as those mentioned above, can mitigate the production of toxic compounds through heating of the tobacco or refill solution at low temperatures without combustion. However, E-Cigs can generate new toxic compounds (e.g., formaldehyde, acetaldehyde, and acrolein) that did not exist in the original solution; these compounds are generally produced via oxidation of the E-Cig components (propylene glycol (PG) and vegetable glycerin (VG)) through heating and vaporization (Bekki et al., 2014; Dai et al., 2018; Etter et al., 2013). Since the HNB tobacco sticks also contain PG and/or VG, formaldehyde and acetaldehyde can also be generated from in the HNB

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https://doi.org/10.1016/j.envres.2020.109413
Received 13 August 2019; Received in revised form 22 February 2020; Accepted 19 March 2020
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Review

Tobacco control in Europe: progress and key challenges

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Received 18 June 2021
Accepted 27 September 2021

To cite: Willemsen MC, Mons U, Fernández E. *Tob Control* 2022;**31**:160–163.

ABSTRACT

We discuss progress made with respect to reducing the burden of disease caused by tobacco use within the WHO European Region and outline major issues and challenges regarding ongoing implementation of tobacco control policy. Although 50 of 53 countries in the WHO European Region are parties to the WHO Framework Convention for Tobacco Control (FCTC), smoking prevalence varies tremendously between European countries. While smoking rates are decreasing slowly, faster declines among smokers with a higher socioeconomic status are leading to growing inequalities in tobacco use. Governments in Europe increasingly formulate visions of 'tobacco-free' societies and it is encouraging that the European Commission aims to achieve a tobacco-free Europe in 2040 as part of its Europe's Beating Cancer Plan. While core WHO FCTC measures still have to be fully implemented in many European countries, some countries are implementing more advanced measures such as plain packaging, banning of characterising flavours from tobacco products, tobacco retailer licensing and extensions of smoking bans into spaces such as cars, outdoor areas and public streets. Remaining challenges include protecting tobacco control policymaking from tobacco industry interference, insufficient dedicated budget for scientific research and the need for Europe-wide monitoring data on use of tobacco and nicotine products.

INTRODUCTION

The WHO European Region comprises 53 countries. To date, with the exception of Liechtenstein, Monaco and Switzerland, all WHO European Region countries, including the European Union (EU), are parties to the WHO Framework Convention for Tobacco Control (FCTC). Despite this, the European Region has one of the highest proportion of premature deaths due to tobacco use in the world, while smoking prevalence declines at a relatively slow rate compared with other WHO regions, particularly among women.[1] According to WHO estimates, the proportion of deaths from non-communicable diseases attributable to tobacco use in Europe was 18% in 2015, meaning that almost one in every five of deaths from non-communicable diseases could be avoided if tobacco use was eliminated altogether from the region.[2] As exemplified for lung cancer in a recent modelling study, implementation of evidence-based tobacco control policies at the most comprehensive level could considerably reduce the smoking-attributable disease burden across Europe.[2]

Trends in smoking prevalence

Within the European Region, differences between countries are tremendous, with current male smoking prevalence, for example, varying from nearly 60% in the Russian Federation to around 16% in Iceland in 2015.[3] Overall, smoking prevalence (male and female combined) tends to be highest in Central and Eastern European countries, and lowest in the Nordic countries and in Central Asia (Uzbekistan, Azerbaijan). Overall, smoking rates are decreasing only slowly and projections suggest that half of the countries of the WHO European Region will not reach the global target of 30% tobacco-use prevalence reduction between 2010 and 2025.[3] Besides smoking prevalence on average being nearly twice as high among men than women, there are also apparent disparities in smoking in relation to socioeconomic status (SES) with consistently higher smoking prevalence among people with lower SES across the European Region.[3] As several studies indicated disproportionally faster declines among smokers with a higher socioeconomic position in European countries, relative inequalities appear to have widened in recent years,[4 5] and are projected to increase further.[3] Thus, accelerating the decline of smoking among all population groups should continue to be a high priority in Europe.[6]

Variation in adoption of tobacco control

The adoption and implementation process of tobacco control policies has varied widely across European countries, that is, in strength of policies, the timing of implementation and level of enforcement.[7] The Tobacco Control Scale (TCS) monitors the implementation of tobacco control policies at country level across Europe.[8] The TCS is based on the six cost-effective interventions listed by the World Bank and ranks countries according to their total score.[9] So far, there have been six editions (2005, 2007, 2010, 2013, 2016 and 2019). The policy domains and corresponding scores have slightly varied across consecutive editions, but the total maximum score (100) remained the same. As illustrated by the comparison of TCS scores from 2007 and 2019 (figure 1), countries with scores above 50 in 2007 but which failed to undertake new initiatives fell in their ranking (Sweden, Malta, Belgium, Italy, Estonia, Bulgaria, Poland). A few countries scoring less than 50 in 2007 (Hungary, Portugal, Slovenia, Greece and Austria) advanced to a score above 50 in 2019. It should be noted however that the TCS scores are published every 3 years, so that most recent improvements in tobacco control policies are not yet captured. For example, Belgium and the Netherlands have

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Nicotine and Tobacco Research, 2022, **24**, 864–870
https://doi.org/10.1093/ntr/ntab265
Advance access publication 20 December 2021
Original Investigation

Health Effects of Tobacco at the Global, Regional, and National Levels: Results From the 2019 Global Burden of Disease Study

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Abstract

Introduction: The purpose was to quantify the health effects of tobacco using data from the 2019 Global Burden of Disease study.

Aims and Methods: We collected detailed information on tobacco consumption overall as well as its individual aspects (smoking, secondhand smoke, and chewing tobacco) for the deaths and disability-adjusted life years (DALYs) for all-cause disease, cardiovascular disease, neoplasms, and chronic respiratory diseases, and their age-standardized rates (ASRs).

Results: Tobacco was responsible for 8.71 million deaths and 229.77 million DALYs globally in 2019. The ASRs of all tobacco-related deaths and DALYs declined from 1990 to 2019, to 108.55 deaths per 100 000 population and 2791.04 DALYs per 100 000 population in 2019. During any year the ASRs of all tobacco-related deaths and DALYs were higher in males than in females. The ASRs of all tobacco-related deaths and DALYs were highest in countries with a low-middle sociodemographic index (SDI) and lowest in high-SDI countries in 2019. Cardiovascular disease, neoplasms, and chronic respiratory diseases were the three leading causes of tobacco-related mortality.

Conclusions: Although the ASRs of deaths and DALYs related to tobacco have declined, the absolute number remain high. Tobacco control policies need to be strengthened further in order to reduce the heavy health burden of tobacco.

Implications: This study provides a detailed description on the health effects of tobacco, including maps of the current global burden of tobacco-related disease. Although the ASRs of tobacco-related deaths and DALYs have declined, the absolute numbers remain high—tobacco was responsible for 8.71 million deaths and 229.77 million DALYs globally in 2019. The findings may have implications for tobacco control. Countries where progress has been slower in reducing tobacco-related disease burden should study and consider implementing policies and strategies that have been applied in countries like Singapore which show the greatest declines for recent decades.

Introduction

Tobacco is one of the main risk factors for disability and premature loss of life globally. Its health burden is associated with significant economic costs, namely expenditure on health care and law enforcement, lost productivity, and other direct and indirect costs including harm to others.[1] It is well known from epidemiological studies that the consumption of tobacco products in general, and smoking products in particular, is the main preventable risk factor for the initiation and progression of periodontal diseases, cardiovascular disease, respiratory diseases, and various types of cancer.[2–7] Secondhand smoke also causes many health issues. The main victims are adult females, infants, and children, whose neurological, immune, respiratory, and circulatory systems may be affected.[8] Smokeless tobacco, mainly including chewing tobacco and snuff, has also been associated with adverse health consequences such as periodontal disease, precancerous oral lesions, and cancers of the mouth, kidney, pancreas, and digestive system.[9]

While policies for reducing the consumption of tobacco have been initiated globally, and the prevalence of tobacco use has declined in some parts of the world, tobacco use remains a persistent—and in some cases growing—problem.[10–12] Estimating the prevalence of use and associated burden of disease and mortality at the country, regional, and global levels is critical for quantifying the extent and severity of the burden arising from tobacco use. Such knowledge would inform allocation decisions by governments, policymakers, and funding bodies about service provision and policies, and assist in evaluations of the impacts of such policies.

The 2019 Global Burden of Disease study (GBD 2019) coordinated by the Institute for Health Metrics and Evaluation estimated the burden of diseases, injuries, and risk factors at the global, regional, and national levels. GBD 2019 included tobacco as a risk factor in the forms of smoking, secondhand smoke, and chewing tobacco. The GBD 2019 database includes information on the contribution of tobacco to the disease burden by country, age, and sex, which represents a

Received: January 5, 2021. **Revised:** December 8, 2021. **Accepted:** December 16, 2021

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Nicotine and Tobacco Research, 2023, **25**, 12–18
https://doi.org/10.1093/ntr/ntac179
Advance access publication 27 July 2023
Original Investigation

National and Regional Fraction of Cancer Incidence and Death Attributable to Current Tobacco and Water-Pipe Smoking in the Eastern Mediterranean Countries in 2020

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Abstract

Introduction: We aimed to calculate the Population Attributable Fraction (PAF) of cancers due to tobacco use in the Eastern Mediterranean Region (EMRO), where water-pipe smoking is prevalent but its effect was not considered in previous studies.

Aims and Methods: We applied Levin's formula to estimate PAFs of cancers due to tobacco use (defined as all type tobacco including both cigarette and water-pipe). We also calculated PAF of water-pipe smoking separately. Exposure prevalence data were retrieved from representative national and subnational surveys. Data on cancer incidence and death were also and cancer cases were obtained GLOBOCAN 2020. We also obtained associated relative risks from published meta-analyses.

Results: Of the total 715 658 incident adult cancer cases that were reported in 2020 in EMRO, 14.6% (n = 104 800) was attributable to tobacco smoking (26.9% [n = 92 753]) in men versus 3.3% (n = 12 048) in women. Further, 1.0% of incident adult cancers were attributable to current water-pipe use (n = 6825) (1.7% [n = 5568]) in men versus 0.4% (n = 1257 in women).

Conclusions: PAFs of cancers due to tobacco smoking in EMRO were higher in our study than previous reports. This could be due to the neglected role of water-pipe in previous studies that is a common tobacco smoking method in EMRO. The proportion of cancers attributable to water-pipe smoking in EMRO might be underestimated due to lack of research on the risk of cancers associated with water-pipe smoking and also less developed cancer registries in EMRO.

Implications: In this study, we found higher PAFs for cancers due to tobacco smoking in the Eastern Mediterranean (EMR) region than previous reports. This difference could be due to ignoring the role of water-pipe smoking in previous studies. In 2020, 1% of incident cancers and 1.3% of cancer-related deaths in EMRO were attributable to water-pipe smoking. We also found a big difference in PAFs of cancers due to tobacco and water-pipe smoking across EMRO countries, with Tunisia, Lebanon, and Jordan having the highest, and Djibouti, Sudan, and Somalia having the lowest proportions of cancers attributable to tobacco and water-pipe smoking.

Introduction

Tobacco smoking is a global health challenge as it is a major cause for several non-communicable diseases including cancers and cardiovascular diseases.[1] Particularly, tobacco plays a critical role in the etiology of different cancers and it has a major contribution to the current cancer burden.[2,3] A recent study from the United States indicates that 33.1% of all cancers in this country could be attributed to tobacco smoking.[4]

Tobacco is used in several different types and routes. There is strong evidence on the carcinogenicity of all types and routes of using tobacco.[5–8] The term "tobacco" is often used to refer to manufactured cigarettes in western countries which are the most common type for using tobacco worldwide.[9] Water-pipe (also called hookah, shisha, narghile, hubble-bubble, arghile) is another common tobacco product that was traditionally more used by Middle Eastern population.[10,11] Although the prevalence of cigarette smoking has been declining over the past decade, the prevalence of water-pipe smoking has been increasing through the world.[12] Lack of public knowledge on the harmful effects of water-pipe and its aromatic and flavored smoke seem to be the main drivers in increasing the popularity of water-pipe smoking, particularly among the youth, which places water-pipe smoking as an emerging global health concern.[13,14] Unlike cigarette that is mostly smoked by men in the Middle East, water-pipe is smoked frequently, sometimes on daily basis, by both men and women in the Eastern Mediterranean Region (EMRO).[15,16]

Received: November 20, 2021. Revised: July 16, 2022. Accepted: July 22 2022.

(11) Varghese J, Muntode Gharde P. A Comprehensive Review on the Impacts of Smoking on the Health of an Individual. Cureus. octubre de 2023;15(10):e46532. doi:10.7759/cureus.46532. https://pubmed.ncbi.nlm.nih.gov/37927763/

Cureus
Part of SPRINGER NATURE

Open Access Review Article

Published via DMIHER Center for Advanced Physiotherapy Education & Research

A Comprehensive Review on the Impacts of Smoking on the Health of an Individual

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Received 08/19/2023
Review began 09/17/2023
Review ended 10/01/2023
Published 10/05/2023

DOI: 10.7759/cureus.46532

Abstract

Long-term smoking for several years has been known to cause severe ailments in humans from the beginning. Even after knowing that this dangerous addiction is a life-threatening deal, still, ironically, the prevalence of smoking is more or less not getting reduced to a desirable extent. Those who smoke are becoming miserable because of their habit of smoking. Still, on the other hand, due to passive smoking, many more innocent lives are also adversely affected for no fault. This aspect of smoking, i.e., passive or second-hand smoking, is a fearful complication of smoking which is seldom seen with other modes of addiction. Time and again, numerous researches have highlighted the adverse effects of smoking on the human body and the interference it does bring in one's life. Smoking contributes to the deterioration of many preexisting ailments and depletes many valuable aspects of the human body. Smoking thus has a devastating effect on almost all of the tissues of our body and thus exerts its effect on nearly all the major organs. This review article is made by analysing various findings from many researches conducted across the globe by having a thorough search of Pubmed database, which in turn is the main methodology of the article. This review article aims to provide a simple and subtle understanding of the ill effects of smoking on the human body by serving the readers with a readymade platter of comprehensive knowledge about smoking coupled with efforts to eliminate the associated myths.

Categories: Preventive Medicine, Public Health, Medical Education
Keywords: risk-factors, negative impact, various systems of human body, harmful effects, cigarette smoking

Introduction And Background

Since the beginning of the human race, men have abused various smokes to get euphorbia. Depending on the availability of the smokable stuff, the content being rolled and its effects vary. Numerous types of smoke-producing products are available in the market [1]. Almost all of them, irrespective of the design of the smoke-propelling device, are harmful to the human body. Even after knowing that smoking is a significant cause of several ailments in the community, the prevalence of smoking is ironically increasing daily [2]. The health impacts of smoking have been put forward in the community with the help of various campaigns and other means in simple language [3]. Still, the effect of these campaigns on the education of smoke-related health issues is only short term and in the long run, is not very helpful in making the users of smoke-related products abstain from them in large numbers [4].

Unlike other addictions, one of the most dangerous and feared adverse effects of smoking is that those who smoke are ruining their own life and becoming a risky threat to many more innocent lives around them by paving the way for them to their graves. Passive smoking is a dreaded complication of smoking, and people who have nothing to do with it suffer because of those around them who smoke [5]. Even though smoking is prohibited in public places, the extent to which a smoker pays attention to it is seldom impressive and respectful [6-11]. In families where some member of the family does smoke, other members seldom are spared from the ill effects of passive smoking [12,13]. Strict rules and regulations need to be put forward, and in a way, making sure it is imposed correctly and promptly would solve the issue of passive smoking considerably. Smoking areas exist in many airports, offices, etc., where a person can smoke without letting that smoke reach a non-smoker, and this is a reality already; better strategies like these are needed to prevent passive smoking in our community.

Those who smoke and drink are considered to have more severe health ailments. Drinking and smoking increases the intensity of harm a body needs to bear and is a matter of great concern. The easy availability of smoke-related products near areas where drinks are available is a primary culprit behind this dangerous deed. Enforcements must be planned and carried out successfully to help smokers abstain from this fatal mixture of addictions. Those who smoke need proper care and counselling, and time and over, it has been evident that many smokers benefited and gave up their habit of smoking after such eventful counselling sessions and guidance [14]. The prevalence of such activities needs to be increased and made available to the general public to ensure it is not limited only to the wealthy and affluent class. Media does play a crucial role in the promotion of smoking. Seeing a favorite actor smoking does impact their fans from adopting similar actions [15,16]. Over the past couple of years, the narration of women being smokers has been

How to cite this article
Varghese J, Muntode Gharde P (October 05, 2023) A Comprehensive Review on the Impacts of Smoking on the Health of an Individual. Cureus 15(10): e46532. DOI 10.7759/cureus.46532

(12) Munakata S, Ishimori K, Kitamura N, Ishikawa S, Takanami Y, Ito S. Oxidative stress responses in human bronchial epithelial cells exposed to cigarette smoke and vapor from tobacco- and nicotine-containing products. Regul Toxicol Pharmacol. noviembre de 2018;99:122-8. doi:10.1016/j.yrtph.2018.09.009. https://pubmed.ncbi.nlm.nih.gov/30227175/

Regulatory Toxicology and Pharmacology 99 (2018) 122–128

Contents lists available at ScienceDirect

Regulatory Toxicology and Pharmacology

journal homepage: www.elsevier.com/locate/yrtph

Oxidative stress responses in human bronchial epithelial cells exposed to cigarette smoke and vapor from tobacco- and nicotine-containing products

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ARTICLE INFO

Keywords:
Bronchial epithelial cells
Cigarette smoke
Tobacco vapor
Oxidative stress
Inflammation
In vitro

ABSTRACT

The use of novel tobacco- and nicotine-containing vapor products that do not combust tobacco leaves is on the rise worldwide. The emissions of these products typically contain lower numbers and levels of potentially harmful chemicals compared with conventional cigarette smoke. These vapor products may therefore elicit fewer adverse biological effects. We compared the effects of emissions from different types of such products, i.e., our proprietary novel tobacco vapor product (NTV), a commercially available heat-not-burn tobacco product (HnB), and e-cigarette (E-CIG), and a combustible cigarette in a human bronchial epithelial cell line. The aqueous extract (AqE) of the test product was prepared by bubbling the produced aerosol into medium. Cells were exposed to the AqEs of test products, and then glutathione oxidation, Nrf2 activation, and secretion of IL-8 and GM-CSF were examined. We found that all endpoints were similarly perturbed by exposure to each AqE, but the effective dose ranges were different between cigarette smoke and the tobacco- and nicotine-containing vapors. These results demonstrate that the employed assays detect differences between product exposures, and thus may be useful to understand the relative potential biological effects of tobacco- and nicotine-containing products.

1. Introduction

The use of tobacco- and nicotine-containing vapor products is increasing in many countries, in particular that of e-cigarettes (E-CIGs) that produce vapor by heating a nicotine-containing liquid (Pepper and Brewer, 2013). In addition to E-CIGs, new types of tobacco vapor products have emerged in which the tobacco is heated, but not combusted, during use; these are typically termed heat-not-burn (HnB) products.

Two main types of HnB products are currently available: Tobacco heating systems, which produce vapor using an electronically controlled unit to heat a tobacco stick (Smith et al., 2016), and heated cigarettes, which produce vapor by heating tobacco leaves using carbon (Sakaguchi et al., 2014). Emissions from HnB products have been shown to contain lower levels of potentially harmful constituents than levels in cigarette smoke, consistent with the lack of tobacco combustion in such products (Sakaguchi et al., 2014; Margham et al., 2016; Schaller et al., 2016). Whether this reduction in exposure to potentially harmful chemicals translates into decreased toxicity to the user remains unknown. Elucidating this relationship will be key in the successful development of modified-risk tobacco products designed to decrease the incidence of smoking-related disease (FDA, 2012).

Cigarette smoking is considered a risk factor for lung diseases such as chronic obstructive pulmonary disease, although multiple environmental and genetic risk factors are also involved in the pathogenesis (Eisner et al., 2010). The primary target of inhaled cigarette smoke is the airway epithelium, which functions as a barrier to inhaled harmful chemicals (BeruBe et al., 2009). Cigarette smoke is an exogenous source of oxidants, and generates intracellular oxidative species that disrupt cellular processes such as aerobic respiration (Pryor and Stone, 1993; Waris and Ahsan, 2006). Oxidative stress is induced by exposure of the lung epithelium to cigarette smoke, and chronic oxidative stress has

Abbreviations: AqE, aqueous extract; ARE, antioxidant response element; DMEM, Dulbecco's modified Eagle's medium; E-CIGs, e-cigarettes; EC_{50}, half-maximal effective concentration; FBS, fetal bovine serum; GM-CSF, granulocyte macrophage colony-stimulating factor; GSH, reduced glutathione; GSSG, oxidized glutathione; HnB, heat-not-burn; IL, interleukin; K3R4F, Kentucky 3R4F reference cigarette; KEAP1, Kelch-like ECH-associated protein 1; NRF2, nuclear factor erythroid 2-related factor 2; NTV, novel tobacco vapor product

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https://doi.org/10.1016/j.yrtph.2018.09.009
Received 15 May 2018; Received in revised form 13 August 2018; Accepted 12 September 2018
Available online 15 September 2018

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Cardiovasc Toxicol
DOI 10.1007/s12012-014-9280-0

Tobacco and Cardiovascular Health

Prajeena Mainali · Sadip Pant · Alexis Phillip Rodriguez · Abhishek Deshmukh · Jawahar L. Mehta

Abstract Tobacco consumption has been inextricably intertwined with society and its evolution. At one time, centuries ago, thought to be a sign of refinement and nobility, fortunately, this perception has been changing worldwide. Currently, this change in perception has been so dramatic that laws are enacted to limit tobacco exposure through second-hand smokers. Countless studies continue to emerge on tobacco's healthcare toll to the point that we now consider indisputable facts that smokers have a higher incidence of coronary artery disease, peripheral vascular disease, chronic obstructive pulmonary disease, stroke, among many others. However, there are other less well-known emerging facts that still require close attention such as the effect on the immune and hematopoietic systems. Tobacco smoke is injurious to all major organs in our bodies. With over 30 known carcinogens, it should not be surprising that it affects all aspects of human health. In this chapter, we will focus on the effects of tobacco on cardiovascular health.

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Published online: 16 September 2014

Keywords Tobacco · Atherosclerosis · Hypertension · Coronary artery disease · Coronary revascularization · Aortic aneurysm · Peripheral arterial disease

Introduction

Almost all societies have consumed tobacco for millennia. Cigarette smoking became widely popular among Americans with the arrival of the Europeans, and over the last century, cigarette smoking was accepted as norm. In the last two decades, there has been a shift in attitude toward cigarette smoking, especially after the realization of the social, economic, and medical burden of this habit. Although cigarette smoking has declined markedly in the last decade, in the USA alone, cigarette smoking claims roughly 440,000 deaths per year and about 5.4 million worldwide, which is likely to continue to increase unless cigarette smoking declines further [1, 2]. Cigarette smoking imposes a heavy economic toll on the society worth about $200 billion annually in health care and loss of productivity [1]. Considering these facts, many states as well as the US federal government have run campaigns aimed to educate consumers regarding the adverse effects of cigarette smoking and to implement clean indoor air and other policies designed to reduce smoking and protect people from secondhand smoke.

New initiatives to thwart tobacco consumption include clean indoor air laws mandating smoke free public places and workplaces, age restriction, limiting advertisement, and increase in taxes among many others. These steps have successfully decreased the rate of tobacco sales and consumption; however, smokeless and water pipe tobacco are increasing in popularity. In actuality, these forms of tobacco products are becoming more prevalent in

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Cancer Research

American Association for Cancer Research

Tobacco and Cancer: An American Association for Cancer Research Policy Statement

Kasisomayajula Viswanath, Roy S. Herbst, Stephanie R. Land, Scott J. Leischow, and Peter G. Shields; Writing Committee for the AACR Task Force on Tobacco and Cancer

Executive Summary

The evidence against tobacco use is clear, incontrovertible, and convincing; so is the need for urgent and immediate action to stem the global tide of tobacco-related death and suffering and to improve public health.

The American Association for Cancer Research makes an unequivocal call to all who are concerned about public health to take the following immediate steps:

- Increase the investment in tobacco-related research, commensurate with the enormous toll that tobacco use takes on human health, to provide the scientific evidence to drive the development of effective policies and treatments necessary to dramatically reduce tobacco use and attendant disease.
- Develop new evidence-based strategies to more effectively prevent the initiation of tobacco use, especially for youth and young adults.
- Promote the further development of evidence-based treatments for tobacco cessation, including individualized therapies, and ensure coverage of and access to evidence-based behavioral and pharmacological treatments.
- Develop evidence-based strategies for more effective public communication to prevent, reduce, and eliminate tobacco use and to guide health policies and clinical practice.
- Develop effective, evidence-based policies to reduce disparities across the tobacco continuum among social groups and developed and developing nations.
- Implement to the fullest extent existing evidence-based, systems-wide tobacco control programs to prevent initiation and foster cessation. Adapt and implement appropriate approaches to reduce the growing burden of tobacco use in the developing world.
- Enhance and coordinate surveillance efforts, both in the United States and globally, to monitor tobacco products, tobacco use, and tobacco-related disease, including tobacco use in oncology clinical trials.
- Establish a comprehensive, science-based regulatory framework to evaluate tobacco products and manufacturers' claims.
- Promote research that addresses the following: the potential harms of current and new tobacco products; the impact of altering the levels of addictive components in tobacco products; the identification of risk and risk-reduction measures for current and former tobacco users; enhanced early detection methods for tobacco-related cancers; and effective treatments against tobacco-related cancers tailored to the unique effects of tobacco on cancer.
- Pursue domestic and international economic policies that support tobacco control.
- Urge the United States to ratify the World Health Organization Framework Convention on Tobacco Control. Foster global scientific efforts to support the Framework.
- Work together with stakeholders worldwide, including federal agencies, to develop and implement effective tobacco control strategies and to deter counter-tobacco control efforts by the tobacco industry.

Only such concerted global actions by scientists, policymakers, and advocates together can prevent the invidious impact of tobacco, the use of which is cutting wide swathes of death and disease around the world.

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OPEN ACCESS
doi: 10.31729/jnma.6605

Tobacco and its Relationship with Oral Health

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ABSTRACT

Tobacco and its various forms cause major oral health problems. Tobacco either in smoked or smokeless forms is prevalent in Nepal and counts as a risk factor for the causation of various red and white lesions, premalignant lesions, oral cancers, gingival and periodontal diseases. Tobacco in conjunction with other risk factors adds a potential threat to oral diseases and its timely control is a cure to those threats. This article focuses on tobacco and its forms affecting oral health and also focuses on its prevention and control from the ground to the National level.

Keywords: *mouth neoplasms; oral health; smoking; tobacco.*

INTRODUCTION

Tobacco consumption is one of the major public health problems in the world. Annually, 27,100 premature deaths are attributed to tobacco-related diseases in Nepal. Tobacco along with its harm to oral and overall systemic health also causes a potential threat to human life as well.[1] About 24% of the adult population use tobacco as per data suggested by WHO in 2018 killing more than 8 million people per year around the world. About 80% of the total tobacco users are from low- and middle-income countries like Nepal.[2] Nepal being a developing nation with a low socioeconomic status tobacco use has been a deep-rooted problem. The prevalence of tobacco use is 56.5% in men and 19.5% in women which is higher than in other countries. The figure is higher in marginalized areas of the country.[3] About 14.9% of total mortality (27,100 deaths annually) are attributed to tobacco use in Nepal.[4]

A total of 32 studies and 5 policy documents were reviewed. Findings suggest that tobacco consumption was higher among men, illiterates, older people, people living in rural and mountainous areas, and those who initiated smoking as adolescents. Peer pressure and parental/family smoking were major contributing factors for tobacco initiation.[4] Cigarette smoking, bidi, khaini, areca nut, slaked lime, snuff, gutkha, paan, hookah, chillum, kankad, sulfa; the major form being paan with tobacco and is most popular in the Terai region. Low socio-economy, illiteracy, unskilled manpower, socio-cultural support to some forms of tobacco is tobacco use boosters in Nepal. Its prevalence in teenagers and young adults is no less. Peer pressure, imitation, fantasy, advertisements, and stress encourage todays' adults to have such habits. This ultimately causes oral diseases, malignancy, and even deaths and sadly the future appears worse.

TOBACCO AND ORAL HEALTH

All of the major forms of tobacco used in Nepal have oral health consequences. Both smoked and smokeless tobacco are prevalent in Nepal. Cigarette smoking can cause a spread of adverse oral effects, including gingival recession, impaired healing following periodontal therapy, oral carcinomas, mucosal lesions (e.g., oral leukoplakia, nicotine stomatitis), periodontal disease, premature tooth loss, and tooth staining. The use of smokeless tobacco is associated with increased risks of oral cancer and oral mucosal lesions, Oral cancer being the second most common cancer in Nepal and sixth among the cancer deaths.[5] Smokeless tobacco use also causes oral conditions like gingival keratosis, tooth discolouration, halitosis, enamel erosion, gingival recession, alveolar bone damage, periodontal disease, coronal or root-surface dental caries due to sugars added to the product, and tooth loss.[4]

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Geriatrics Gerontology INTERNATIONAL Medline Indexed

Geriatr Gerontol Int 2014; **14:** *526–540*

REVIEW ARTICLE: EPIDEMIOLOGY, CLINICAL PRACTICE AND HEALTH

Implications of tobacco smoking on the oral health of older adults

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Cigarette smoking is the foremost health risk issue affecting individuals of all age groups globally. It specifically influences the geriatric population as a result of chronic exposure to toxins. Its role in various systemic and oral diseases including cancer, premalignant lesions, periodontitis, tooth loss, dental caries and implant failures is well established. Smoking causes immuno-inflammatory imbalances resulting in increased oxidative stress in the body. The latter hastens the immunosenescence and inflammaging process, which increases the susceptibility to infections. Thus, implementation of smoking cessation programs among older adults is imperative to prevent the development and progression of oral and systemic diseases. The present review focuses on smoking-associated oral health problems in older adults, and the steps required for cessation of the habit. **Geriatr Gerontol Int 2014; 14: 526–540**.

Keywords: chronic periodontitis, dental caries, oral cancer, oral premalignant lesions, smoking, smoking cessation.

Introduction

Tobacco smoking is one of the major preventable risk factors threatening the current and future health of populations across the world. Global trends on tobacco consumption suggest that by 2030, it would be one of the leading causes for death of approximately 8 million people per year residing in both the developed and developing countries.[1] It has been reported that smoking reduces the lifespan of an individual by 7 years.[2] In older adults, the cumulative effects of prolonged smoking increase disease incidence and mortality, accounting for approximately 70% of the 400 000 or more deaths of individuals aged ≥60 years, as a result of chronic obstructive pulmonary disease and lung cancer.[3]

The tobacco can be smoked in various forms, such as cigarettes, bidis, kreteks and shisha. Cigarettes are "nicotine delivery devices," which carry nicotine to the brain as efficiently as an intravenous injection.[4] Bidis are small hand-rolled cigarettes typically smoked in India and other Southeast Asian countries.[5] They produce approximately threefold more carbon monoxide and nicotine, and fivefold more tar. They are implicated in oral, lung, stomach and esophageal cancers. Kreteks are Indonesian clove and tobacco cigarettes that cause acute lung injury.[6] Shisha is tobacco cured with flavorings, and smoked from hookahs primarily in the east Mediterranean region.[7]

Smoking has been strongly associated with cancers, coronary artery disease, Alzheimer's disease, stroke and reduced bone density. These problems are frequently seen in the elderly population.[8] The destructive effects of the aforementioned conditions are mediated through its reservoir of toxic chemicals that directly or indirectly trigger the host immune-inflammatory system thereby damaging the tissues.[9] They increase the production of pro-inflammatory cytokines, such as tumor necrosis factor-α (TNF-α), interleukin (IL)-1, IL-6 and IL-8, and decrease the levels of anti-inflammatory cytokines (e.g. IL-10). Additionally, they activate macrophages and dendritic cells. The levels of immunoglobulin (Ig) E are elevated leading to immune hypersensitivity-type reactions.[10] It even weakens the innate defences against pathogens, alters antigen presentation and promotes autoimmunity.[11]

In older adults, the weakened host immune system becomes further suppressed as a result of the effects of smoking. This increases the risk for systemic as well as oral diseases. The latter include oral cancer, periodontal disease, tooth loss, dental caries and precancerous conditions.[12] Among them, chronic periodontitis has been reported to worsen diabetic control, cardiovascular

Accepted for publication 17 February 2014.

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Review Article

Epidemiology, control and prevention of tobacco induced oral mucosal lesions in India

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Abstract

Oral cancer is one of the leading causes of human morbidity and mortality especially in developing countries like India. Tobacco consumption in smokeless and smoking form along with alcohol is considered as the primary risk factors. Tobacco is a major health challenge with various tobacco products available for use which are known to have deleterious effects on the oral mucosa. The oral lesions caused by tobacco are inclusive of those that are less likely to progress to cancer; lesions with increased tendency to develop into cancer and cancerous lesions. Prevention and control of tobacco induced oral mucosal lesions is the prime requisite currently and mainly involves measures undertaken at primary, secondary and tertiary levels. Primary prevention plays a pivotal role in tobacco induced lesions and steps can be taken at policy level, community as well as individual level. This review paper focuses on the epidemiological data of tobacco induced oral mucosal lesions in India available in the literature with an overview on various strategies for their prevention and control.

Key Words: Epidemiology, oral squamous cell carcinoma, potentially malignant disorders, prevention and control, tobacco induced oral mucosal lesions

Introduction

Oral cavity is prone for a myriad of changes with advancing age as well as a result of the environmental and life style related factors. Oral mucosal lesions can occur as a result of infections, local trauma or irritation, systemic diseases and excessive consumption of tobacco, betel quid and alcohol.[1] The prevalence and incidence rates of oral mucosal lesions are available from various countries, but the information thereby obtained may not always be applicable to Indian population due to the existence of cultural, ethnic and demographic differences. Despite the efforts made by the different groups, establishment of prevalence data related to oral mucosal lesions is meager in Indian literature.[2]

Chewing and smoking of tobacco along with consumption of alcohol beverages have become common social habits in India.[3] Tobacco was introduced in India by the Portuguese nearly 400 years ago and since then it rapidly became a part of socio-cultural milieu in various communities.[4] India is the second largest producer and consumer of tobacco next only to China.[4,5] The prevalence of tobacco use among Indian adults is 35%.[6] Introduced initially in India as a product to be smoked, tobacco gradually began to be used in several other forms such as paan (betel quid) chewing and leaf tobacco.[7] In India, beedi smoking is the most popular form of tobacco smoking and paan with tobacco is a major chewing form. Dry tobacco-areca nut preparations such as paan masala, gutkha and mawa are also popular and highly addictive. It has been estimated that the number of tobacco users in India among those 10 years of age and above is around 250 million.[7]

Alcohol use often co-exists with tobacco consumption. In India, 4.5% of the population use alcohol regularly.[8] The various types of alcoholic beverages used in India are wine, beer, toddy, whisky, gin, rum, brandy, arrack and liqueurs.[9] Epidemiological studies have shown that alcohol consumption in association with tobacco is a significant risk factor for oral pre-cancer and cancer.[10,11] The metabolized product of alcohol, acetaldehyde, is known for its carcinogenic activity. Alcohol also causes an increased rate of penetration of substances from the oral environment across the mucosa due to alteration of mucosal permeability that may play a role in carcinogenesis.[12]

Smoking, drinking and tobacco chewing have been positively associated with oral lesions such as leukoplakia, oral submucous fibrosis and oral lichen planus which have the potential for malignant transformation.[3] Tobacco consumption also remains the most important avoidable risk factor for oral cancer. Tobacco related cancers account for nearly 50% of all cancers in men and 25% in women.[13] Oral squamous cell carcinoma may occur either de novo or from the precursor lesions. As a result, prompt intervention at appropriate levels may aid in prevention and better control of tobacco induced lesions. Keeping in view the major risk factors for oral mucosal lesions and its associated effects, a range of preventive measures could be implemented at primary, secondary or tertiary levels.

Though oral pre-cancer and cancer is widespread in India, epidemiological data from various geographical areas is scarce. The present article attempts to compile the prevalence and incidence rates of tobacco induced oral mucosal lesions in India that is documented in the literature with a note on the different preventive measures that could be implemented at various levels.

Tobacco Induced Oral Mucosal Lesions

Long term contact of tobacco with the oral mucosa induces variety of changes which could be due to the carcinogen itself or as a protective mechanism of the oral cavity. These changes could be categorized as tobacco induced oral mucosal lesions which are less likely to cause cancer, lesions that are potentially malignant and tobacco induced malignancies.

Tobacco Induced Non-Neoplastic Oral Mucosal Lesions

The tobacco induced mucosal lesions which are less likely to cause cancer are betel chewer's mucosa, leukedema, smoker's palate, lichenoid reaction, smoker's melanosis, tobacco

Access this article online

Quick Response Code:

Website: www.indianjcancer.com

DOI: 10.4103/0019-509X.134651

PMID: *******

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Article published online: 2022-12-27

Dental Implant Osseointegration Inhibition by Nicotine through Increasing nAChR, NFATc1 Expression, Osteoclast Numbers, and Decreasing Osteoblast Numbers

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Eur J Dent 2023;17:1189–1193.

Abstract

Objective The success of dental implants is determined by the osteointegration process. Many studies state that smoking cigarettes can inhibit osseointegration, but the inhibition mechanism is still unclear.

The aim of this study was to identify and analyze the effect of nicotine on the inhibition of dental implant osseointegration through the expression of nicotinic acetylcholine receptor (nAChR), nuclear factor of activated T cells cytoplasmic 1 (NFATc1), osteoclast, and osteoblast numbers.

Materials and Methods This study is an experimental study of 16 New Zealand rabbits, randomized across two groups. Group 1 (eight rabbits) was a control group, and group 2 (eight rabbits) was a treatment group. The treatment group was given 2.5 mg/kg body weight/day of nicotine by injection 1 week before placement of the implant until the end of research. Observations were made in the first and the eighth week by measuring the number of osteoblast and osteoclast by immunohistology test and the expression of nAChR and NFATc1 by immunohistochemistry test.

Statistical Analysis Data was analyzed using a one-way analysis of variance and Student's *t*-test. A *p*-value of < 0.05 was considered statistically significant.

Results Significant differences were found between the control and treatment groups ($p < 0.05$). Results showed that nicotine increases the expression of nAChR and decreases the number of osteoblasts and the expression of BMP2 and osteocalcin.

Conclusion Nicotine inhibits the osseointegration of dental implants by increasing nAChR, NFATc1, osteoclast numbers, and decreasing osteoblast numbers.

Keywords
- dental implant
- nicotine
- nAChR
- NFATc1
- osteoclast
- osteoblast
- osseointegration

Introduction

Dental implants are the gold standard for the replacement of missing teeth. Many factors have been recognized as critical for the successful performance of the implants. One of the most important factors is osseointegration—the direct interactions between the implant and the tissues. The osseointegration depends on the osteoblastic activity enhancement around the implant that promotes direct union between the

article published online December 27, 2022

DOI https://doi.org/10.1055/s-0042-1758794.
ISSN 1305-7456.

Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

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doi: 10.1111/j.1834-7819.2009.01140.x

Diagnosis and classification of periodontal disease

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ABSTRACT

Periodontal diseases have been recognized and treated for at least 5000 years. Clinicians have recognized for many years that there are apparent differences in the presentation of periodontal diseases and have attempted to classify these diseases. Systems of classifications of disease have arisen allowing clinicians to develop structures which can be used to identify diseases in relation to aetiology, pathogenesis and treatment. It allows us to organize effective treatment of our patients' diseases. Once a disease has been diagnosed and classified, the aetiology of the condition and appropriate evidence-based treatment is suggested to the clinician. Common systems of classification also allow effective communication between health care professionals using a common language. Early attempts at classification were made on the basis of the clinical characteristics of the diseases or on theories of their aetiology. These attempts were unsupported by any evidence base. As scientific knowledge expanded, conventional pathology formed the basis of classification. More recently, this has been followed by systems of classification based upon our knowledge of the various periodontal infections and the host response to them. Classification of periodontal diseases has, however, proved problematic. Over much of the last century clinicians and researchers have grappled with the problem and have assembled periodically to review or develop the classification of the various forms of periodontal disease as research has expanded our knowledge of these diseases. This has resulted in frequent revisions and changes. A classification, however, should not be regarded as a permanent structure. It must be adaptable to change and evolve with the development of new knowledge. It is expected that systems of classification will change over time. This review examines the past and present classifications of the periodontal diseases.

Keywords: Diagnosis, classification, periodontal diseases, gingivitis, periodontitis.

Abbreviations and acronyms: AST = aspartate aminotransferase; CAL = clinical loss of attachment; GCF = gingival crevicular fluid; LJP = localized juvenile periodontitis; NUG = necrotizing ulcerative gingivitis; NUP = necrotizing ulcerative peridontitis.

INTRODUCTION

Periodontal disease is a disease, or more likely a number of diseases of the periodontal tissues that results in attachment loss and destruction of alveolar bone. The natural history of periodontal disease, in some but not all patients, results in tooth loss.[1] Periodontal disease, however, encompasses a wider spectrum of diseases than just periodontitis and the recognition of these diseases requires a diagnosis be made.

Diagnosis is the recognition of the presence of a disease.[2] Clinical diagnosis of periodontal disease is made by the recognition of various signs and symptoms in the periodontal tissues which herald a departure from health. The diagnosis of periodontal disease demands a firm knowledge of what constitutes periodontal health. The healthy periodontium,[3] of which only the gingival tissues may be directly observed, is described as being stippled, pale pink or coral pink, in the Caucasian (Fig 1), with various degrees of pigmentation in other races. It is tightly adapted to the underlying tissues, with a knife edge margin where it abuts the tooth, The gingival margin is located, in the absence of pathology, at the cemento-enamel junction. It displays a scalloped edge configuration highest interdentally, where it constitutes the interdental papilla and lowest buccally and lingually. There is a gingival crevice where it abuts the tooth which in health is 1–3 mm deep. There is an absence of bleeding from the crevice on gentle probing. The crevice in health will show a small amount of interstitial fluid, gingival crevicular fluid.[4] The lateral wall of the crevice constitutes the free gingival margin. From the most apical extent of the free gingival to the mucogingival junction is the attached gingival which varies in width from 1 to 9 mm and has a stippled surface. It is an immobile tissue tightly bound down to the bone as a mucoperiostium and is a keratinized mucosa well suited to resist injury. Apical from the mucogingival junction

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Received: 12 July 2022 | Revised: 21 August 2022 | Accepted: 6 September 2022
DOI: 10.1111/prd.12473

REVIEW ARTICLE

Periodontology 2000 WILEY

Periodontal microbiology and microbial etiology of periodontal diseases: Historical concepts and contemporary perspectives

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1 | INTRODUCTION

The etiology of a disease refers to the causative trigger(s), whereas pathogenesis refers to the mechanism(s) by which the disease progresses. Over the past century, we have appreciated that periodontitis is of a microbial etiology and an inflammatory pathogenesis, albeit the coordination of the contributing factors for the initiation and progression of the disease may vary from an epidemiological perspective.[1] In other words, while the microbial biofilm developing on the tooth surface constitutes a necessary etiological factor, its mere presence is insufficient for the initiation of the disease. Further risk factors, such as host genetics, lifestyle, stress, and systemic conditions, that dictate the immunopathogenesis are crucial for the transition from a healthy to a diseased state. Such factors will be addressed in other papers within this special issue.

Whether it is one form of disease manifesting with different degrees of progression and severity, or different forms of disease exhibiting similar clinical manifestations, has long been a topic of public curiosity and scientific endeavor for mankind. The historical and contemporary knowledge established by pioneering researchers around the globe has led to paradigm shifts in our understanding of the etiology of the disease. This article discusses the continuum of seminal discoveries in the field, while highlighting the European contribution and its universal impact.

2 | ETIOLOGICAL HYPOTHESES AND MODELS FOR PERIODONTAL DISEASES

At the cradle of European civilization, ancient Greeks had already been able to identify the signs of periodontal disease and used their sense of smell as a diagnostic aid. Hippocrates refers to his scripts that the "evil malodor" is as result of "pitius" and even proposed oral rinsing with a solution of natural herbs as a treatment method.[2] Centuries later, the Romans observed "wobbly" teeth to be a diagnostic sign of the disease, which was attributed to the hard "calculus" deposits on the tooth surface, a dogma that dominated until the 18th century. Then, French pathologist Pierre Fauchard concluded that periodontal pathology is "a distinct type of scurvy" of local rather than systemic causes, whereas later that century, Scottish physiologist and surgeon John Hunter supported that gingival inflammation is the cause of alveolar bone dissolution, while introducing for the first time the term "periodontosis".[3] In late 19th century American dentist John Riggs historically named the disease "pyorrhea alveolaris" (also known as "Riggs' disease"), describing it as a suppurative inflammation of the gingiva and the alveolar process, while strongly advocating for hard calculus as the single local causative factor.[4] This theory coincided with the time of an unparalleled evolution of the scientific field of microbiology, leading to the contemporary notion that bacteria residing within the dental plaque deposits are

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DOI: 10.1111/prd.12303

REVIEW ARTICLE

Periodontology 2000 WILEY

A brief history of periodontics in the United States of America: Pioneers and thought-leaders of the past, and current challenges

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1 | INTRODUCTION

The purposes of this paper are: (1) to summarize important historical events or periods in periodontology in the United States; (2) to highlight some key pioneers and thought-leaders of the past; and (3) to identify the major research and clinical challenges that the field faces in the coming decades. Many of the historical events in periodontology were shaped and influenced by people who were not originally from the United States. This is not surprising because the United States has always been a country of immigrants from diverse backgrounds. There is nothing in periodontology that is uniquely American. Furthermore, most of the new clinical and research developments in periodontology emanate from the work of international teams of scientists and clinicians.

2 | HISTORICAL EVENTS OR PERIODS IN PERIODONTOLOGY IN THE UNITED STATES

2.1 | Periodontology in the 1800s

Throughout the 19th century, clinicians and scholars debated about the causes of, and best treatments for, destructive gum diseases.[1-7] Among the names for periodontitis in the 1800s were pyorrhea alveolaris,[5,8] Riggs Disease,[8] calcic pericementitis,[4] interstitial gingivitis,[7] phagedenic pericementitis,[9] and chronic suppurative pericementitis.[9] The early debates on causation revolved around local vs systemic causes of the disease. The majority of experts in the United States favored the notion that periodontitis was caused by local irritants, such as dental calculus[2-4,10] and bacteria.[5,6] Others argued that pyorrhea alveolaris was caused by a hereditary predisposition,[11] malnutrition,[12] localized deposits of uric acid,[13-16] or a localized expression of occult systemic disease.[17] Most of the theories on causation were supported by little or no evidence.

In the 1800s, the majority of treatments were based on trial-and-error clinical experience and strong opinions. In his historical review of the era, written in 1921, Arthur H. Merritt[8] described the profession's general approach to the treatment of periodontitis:

> Some ineffective attempts at local treatment were made but dependence was largely placed on drugs, acids and certain antiseptics seeming to be favorites. The rank and file of the profession apparently paid little attention to its treatment; people retained their teeth until they became so loose as to cause annoyance, when they were extracted. The dental schools were equally indifferent.

Throughout the 20th century, the evolution of thought, leading to current views of the pathogenesis and treatment of periodontal diseases, was significantly influenced by: (1) major changes in health-care education; (2) emergence of periodontics as a specialty of dentistry; (3) publication of peer-reviewed journals with an emphasis on periodontology; (4) formation of the NIDCR; and (5) expansion of periodontal research programs by the NIDCR.

2.2 | Major changes in health-care education

In 1900, medical education across the United States was of variable quality. At some locations it was a state-of-the-art enterprise and at others the educational offerings in medicine were dismal.Many medical schools had no full-time faculty, and relied on poor hospitals with inadequate laboratory facilities.[18] National educational standards for medical schools did not exist. This nationwide problem was

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Received Date : 21-Jun-2016

Revised Date : 27-Aug-2016

Accepted Date : 21-Sep-2016

Article type : Other

Risk Factors in Periodontology: a Conceptual Framework

Running Title: *Risk factors in Periodontology*

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Received: 2 January 2018 | Revised: 11 February 2018 | Accepted: 11 February 2018

DOI: 10.1002/JPER.18-0006

JOURNAL OF Periodontology

2017 WORLD WORKSHOP

Staging and grading of periodontitis: Framework and proposal of a new classification and case definition

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The proceedings of the workshop were jointly and simultaneously published in the *Journal of Periodontology* and *Journal of Clinical Periodontology*.

Abstract

Background: Authors were assigned the task to develop case definitions for periodontitis in the context of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. The aim of this manuscript is to review evidence and rationale for a revision of the current classification, to provide a framework for case definition that fully implicates state-of-the-art knowledge and can be adapted as new evidence emerges, and to suggest a case definition system that can be implemented in clinical practice, research and epidemiologic surveillance.

Methods: Evidence gathered in four commissioned reviews was analyzed and interpreted with special emphasis to changes with regards to the understanding available prior to the 1999 classification. Authors analyzed case definition systems employed for a variety of chronic diseases and identified key criteria for a classification/case definition of periodontitis.

Results: The manuscript discusses the merits of a periodontitis case definition system based on Staging and Grading and proposes a case definition framework. Stage I to IV of periodontitis is defined based on severity (primarily periodontal breakdown with reference to root length and periodontitis-associated tooth loss), complexity of management (pocket depth, infrabony defects, furcation involvement, tooth hypermobility, masticatory dysfunction) and additionally described as extent (localized or generalized). Grade of periodontitis is estimated with direct or indirect evidence of progression rate in three categories: slow, moderate and rapid progression (Grade A-C). Risk factor analysis is used as grade modifier.

Conclusions: The paper describes a simple matrix based on stage and grade to appropriately define periodontitis in an individual patient. The proposed case definition extends beyond description based on severity to include characterization of biological features of the disease and represents a first step towards adoption of precision medicine concepts to the management of periodontitis. It also provides the necessary framework for introduction of biomarkers in diagnosis and prognosis.

KEYWORDS

aggressive periodontitis, biomarkers, case definition, chronic periodontitis, classification, clinical attachment loss, diagnosis, furcation involvement, grade A periodontitis, grade B periodontitis, grade C

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BMC Oral Health

PROCEEDINGS Open Access

Defining periodontal health

Angelo Mariotti*†, Arthur F Hefti†

From Prevention in practice - making it happen
Cape Town, South Africa. 29 June 2014

Abstract

Assessment of the periodontium has relied exclusively on a variety of physical measurements (e.g., attachment level, probing depth, bone loss, mobility, recession, degree of inflammation, etc.) in relation to various case definitions of periodontal disease. Periodontal health was often an afterthought and was simply defined as the absence of the signs and symptoms of a periodontal disease. Accordingly, these strict and sometimes disparate definitions of periodontal disease have resulted in an idealistic requirement of a pristine periodontium for periodontal health, which makes us all diseased in one way or another. Furthermore, the consequence of not having a realistic definition of health has resulted in potentially questionable recommendations. The aim of this manuscript was to assess the biological, environmental, sociological, economic, educational and psychological relationships that are germane to constructing a paradigm that defines periodontal health using a modified wellness model. The paradigm includes four cardinal characteristics, i.e., 1) a functional dentition, 2) the painless function of a dentition, 3) the stability of the periodontal attachment apparatus, and 4) the psychological and social well-being of the individual. Finally, strategies and policies that advocate periodontal health were appraised.

I'm not sick but I'm not well,
and it's a sin to live so well.
Flagpole Sitta, Harvey Danger

Introduction

Most people use the word "health" casually, in juxtaposition to disease, with no frame of reference. Frequently used terms associated with health include "health benefits", "health promotion", "health prevention", "health care", "health insurance", and "oral health", to name just a few. Clearly, the word health means different things to different people in different situations. An epidemiologist may use mortality data to study the "health" of a population, the economist may discuss the "health" of the economy as it relates to sustainable GDP growth, the stressed-out dental student may wonder whether the final examinations will affect her "health", most likely meaning her sanity, and periodontists are measuring attachment and bone levels when seeking information about periodontal "health". Obviously, these professionals use the same word but with very different meanings. Etymologically, the word "health" was derived from the Old English "hale", meaning wholesome, sound, or well. By and large, the original and broad connotation of this word has prevailed but with modern context.

Although the original Old English meaning of health has survived through the centuries, what constitutes health in the twenty-first century is far more perplexing than we can suppose. What we consider to be healthy has evolved as a result of definitions of health that have varied with the times because society's perception of disease and health have been influenced by our expanding scientific knowledge base as well as our cultural, social, and individual value judgments [1,2]. Despite the continually changing set of pretexts, the definition of health is important because it provides a common reference point to define recurring signs and symptoms that are within a significant standard definition of normal. Therefore, instead of trying to define a set of periodontal diseases whose etiology we cannot comfortably explain, perhaps, a better policy would be to characterize what periodontal health actually constitutes and what can affect it. The goal of the present paper is to present a framework for

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REVISTA MEXICANA DE PERIODONTOLOGÍA

ARTÍCULO ESPECIAL

Vol. IX, Núms. 1-2 • pp 24-27

Este artículo puede ser consultado en versión completa en
www.medigraphic.com/periodontologia

Clasificación de enfermedades periodontales[1]

Alma Alicia Soto Chávez,* Alondra del Carmen Ruiz Gutiérrez,* Vianeth Martínez Rodríguez*

RESUMEN

Desde 1999 hemos utilizado una clasificación de la enfermedad periodontal la cual requería una actualización que se adaptara a los avances actuales. Por primera vez se unen la Academia Americana de Periodontología y la Federación Europea de Periodoncia y deciden trabajar una clasificación universal. Dentro de los aspectos más destacados son: la recategorización de periodontitis con la eliminación de los adjetivos de «agresiva» y «crónica»; la suma de estadios y grados; también se encuentra un apartado para la salud periodontal así como para las enfermedades y condiciones periimplantarias. El resultado de este encuentro entre ambas organizaciones científicas logró un sistema global de clasificación de enfermedades periodontales y periimplantarias en donde ahora el reto actual de todo profesionista del área de la salud es optimizar la planificación integral de los tratamientos así como un enfoque personalizado de atención al paciente. Este documento presenta una visión abreviada de la clasificación.

Palabras clave: Clasificación, periodontitis, gingivitis, periimplantitis.

ABSTRACT

Since 1999 we have used a classification of periodontal disease which required updating to adapt to current advances, for the first time they join the American Academy of Periodontology and the European Federation of Periodontics and decide to work a universal classification. Among the most prominent aspects are: the recategorization of periodontitis with the elimination of the adjectives of «aggressive» and «chronic»; the sum of stages and degrees, there is also a section for periodontal health as well as for diseases and peri-implant conditions. The result of this meeting between the two scientific organizations achieved a global classification system for periodontal and peri-implant diseases where now the current challenge of every health professional is to optimize the integral planning of the treatments as well as a personalized approach of attention to the patient. This document presents an abbreviated view of the classification.

Key words: *Classification, periodontitis, gingivitis, periimplantitis.*

La clasificación de la enfermedad periodontal después de dos décadas se renueva y adapta a las tendencias y avances actuales. En este sentido hay que resaltar varios aspectos, se trata de una clasificación con alcance global, ya que por primera vez se hace un amplio consenso uniéndose las dos organizaciones científicas más importantes del mundo, la Academia Americana de Periodontología (AAP) y la Federación Europea de Periodoncia (EFP por sus siglas en inglés). La clasificación de la enfermedad periodontal y periimplantar se presentó durante el Congreso EuroPerio 2018, realizado en la ciudad de Ámsterdam el 22 de junio del presente año; esta exhaustiva actualización proviene del taller mundial de 2017 realizado en Chicago.

Uno de los objetivos principales es volver universal esta clasificación y que todos los profesionales de la salud sean capaces de definir los individuos sanos de los enfermos y, de estos últimos, identificar la enfermedad, el estadio y grado de la misma. Dentro de los aspectos más destacados incluye: una recategorización de la periodontitis, un apartado para la salud periodontal, así como para las enfermedades y condiciones periimplantarias.

La nueva clasificación de la enfermedad periodontal abarca cuatro grandes grupos, los cuales se conforman de la siguiente manera: Grupo 1: salud periodontal, enfermedades y condiciones gingivales; Grupo 2: periodontitis; Grupo 3: enfermedades sistémicas y condiciones que afectan el tejido de soporte periodontal; y Grupo 4: enfermedades y condiciones periimplantarias. A continuación se presenta una aproximación a cada uno de estos grupos.

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[1] Este documento presenta una visión abreviada de la clasificación, por lo que recomendamos consultar http://perio.org/2017wwdc

Recibido: 10 de junio de 2018. Aceptado: 06 de agosto de 2018.

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American Journal of
Preventive Medicine

REVIEW ARTICLE

Effect of Smoking on Periodontitis: A Systematic Review and Meta-regression

Fábio R.M. Leite, PhD, Gustavo G. Nascimento, PhD, Flemming Scheutz, DrPH, Rodrigo López, DrOdont

Context: The study systematically reviewed articles on the association between tobacco smoking and periodontitis, as it has been hypothesized that smoking affects the course of periodontitis through impairment of immunological and vascular mechanisms.

Evidence acquisition: Searches of articles indexed in PubMed, Scopus, and Embase were performed up to and including May 2017. Search strategy included MeSH and free terms: *periodontitis*, *periodontal diseases*, *smoking*, *tobacco use*, *tobacco*, *tobacco products*, *cigarette*, *pipe*, and *cigar*. Only original prospective longitudinal studies that investigated the association between smoking and periodontitis incidence or progression were included. Results were shown as combined risk ratio. Meta-regression and subgroup analyses were used to explore potential sources of heterogeneity. Analyses were conducted in August 2017.

Evidence synthesis: Twenty-eight studies were included in the review; of these, only 14 presented data that could be included in the meta-analysis. Pooled adjusted risk ratios estimate that smoking increases the risk of periodontitis by 85% (risk ratio=1.85, 95% CI=1.5, 2.2). Meta-regression demonstrated that age explained 54.2% of the variability between studies, time of follow-up explained 13.5%, loss to follow-up 10.7%, criteria used to assess the periodontal status explained 2.1%, and severity of periodontitis explained 16.9%.

Conclusions: Smoking has a detrimental effect on the incidence and progression of periodontitis. Tobacco smoking, therefore, is important information that should be assessed along with other risk factors for periodontitis.

Am J Prev Med 2018;■(■):■■–■■

CONTEXT

Smoking is acknowledged as an important risk factor for noncommunicable chronic diseases. Smoking-related diseases have become one of the leading causes of death in the world.[1,2] Despite a decrease in smoking habits, estimates suggest that in 2020 approximately 10% of all deaths will be related to smoking.[1] Health expenditures attributable to tobacco-related diseases exceed the total tax revenue from tobacco products.[1] Thus, the magnitude of tobacco-related expenditures is a significant burden for both individuals and healthcare systems.

Periodontitis is a chronic destructive inflammatory condition affecting the supporting structures of the teeth and as such, it is listed in the global burden of chronic diseases.[3] In its ultimate stage, periodontitis leads to tooth loss. Both conditions, periodontitis and tooth loss, affect mastication and speech and have an impact on quality of life and self-esteem.[4] In contrast to the decrease of dental caries, severe periodontitis remains unchanged since 1990.[3] Meta-analysis on the prevalence of severe periodontitis shows that approximately 700 million people are affected worldwide.[3] Given the combination of greater life expectancy and a significant reduction in tooth loss because of dental caries, the burden of periodontitis is expected to increase.

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0749-3797/$36.00
https://doi.org/10.1016/j.amepre.2018.02.014

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Odontology (2004) 92:1–8
DOI 10.1007/s10266-004-0043-4

REVIEW ARTICLE

Jan Bergström

Tobacco smoking and chronic destructive periodontal disease

Received: June 4, 2004 / Accepted: June 22, 2004

Abstract Tobacco smoking is the main risk factor associated with chronic destructive periodontal disease. No other known factor can match the strength of smoking in causing harm to the periodontium. The harmful effects manifest themselves by interfering with vascular and immunologic reactions, as well as by undermining the supportive functions of the periodontal tissues. The typical characteristic of smoking-associated periodontal disease is the destruction of the supporting tissues of the teeth, with the ensuing clinical symptoms of bone loss, attachment loss, pocket formation, and eventually tooth loss. A review of the international literature that has accumulated over the past 20 years offers convincing evidence that smokers exhibit greater bone loss and attachment loss, as well as more pronounced frequencies of periodontal pockets, than non-smokers do. In addition, tooth loss is more extensive in smokers. Smoking, thus, considerably increases the risk for destructive periodontal disease. Depending on the definition of disease and the exposure to smoking, the risk is 5- to 20-fold elevated for a smoker compared to a never-smoker. For a smoker exposed to heavy long-life smoking, the risk of attracting destructive periodontal disease is equivalent to that of attracting lung cancer. The outcome of periodontal treatment is less favorable or even unfavorable in smokers. Although long-term studies are rare, available studies unanimously agree that treatment failures and relapse of disease are predominantly seen in smokers. This contention is valid irrespective of treatment modality, suggesting that smoking will interfere with an expected normal outcome following commonplace periodontal therapies. The majority of available studies agree that the subgingival microflora of smokers and non-smokers are no different given other conditions. As a consequence, the elevated morbidity in smokers does *not* depend on particular microflora. The mechanisms behind the destructive effects of smoking on the periodontal tissues, however, are not well understood. It has been speculated that interference with vascular and inflammatory phenomena may be one potential mechanism. Nicotine and carbon monoxide in tobacco smoke negatively influence wound healing. Smoking research over the past two decades has brought new knowledge into the domains of periodontology. Even more so, it has called into question the prevailing paradigm that the disease is primarily related to intraoral factors such as supra- and subgingival infection. Smoking research has revealed that environmental and lifestyle factors are involved in the onset and progression of the disease. Being the result of smoking, destructive periodontal disease shares a common feature with some 40 other diseases or disorders. As a consequence, periodontal disease should be regarded as a systemic disease in the same way as heart disease or lung disease. Thus, chronic destructive periodontal disease in smokers is initiated and driven by smoking. Its progression may or may not be amplified by unavoidable microbial colonization.

Key words Periodontal disease · Periodontal treatment · Risk factor · Smoking · Tobacco

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Introduction

Tobacco smoking is the number one enemy of periodontal health. No other as yet identified factor can parallel the force of smoking as a destroyer of the periodontal tissues. The destructive force of smoking against the periodontal tissues clinically appears in several forms. On one hand, subtle signs of inflammation are suppressed by its interference with vascular and immune reactions and, on the other, the supporting functions are undermined by its destructive action on the bone tissue, the consequences being bone loss, pocket formation, and tooth loss. To describe this latter process, the term "(*chronic*) *destructive periodontal disease*" will be used in the following text. The present review provides an evaluation of the current literature on the

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The PRISMA 2020 statement: an updated guideline for reporting systematic reviews

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Additional material is published online only. To view please visit the journal online.

Cite this as: *BMJ* 2021;372:n71 http://dx.doi.org/10.1136/bmj.n71

Accepted: 4 January 2021

The Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) statement, published in 2009, was designed to help systematic reviewers transparently report why the review was done, what the authors did, and what they found. Over the past decade, advances in systematic review methodology and terminology have necessitated an update to the guideline. The PRISMA 2020 statement replaces the 2009 statement and includes new reporting guidance that reflects advances in methods to identify, select, appraise, and synthesise studies. The structure and presentation of the items have been modified to facilitate implementation. In this article, we present the PRISMA 2020 27-item checklist, an expanded checklist that details reporting recommendations for each item, the PRISMA 2020 abstract checklist, and the revised flow diagrams for original and updated reviews.

SUMMARY POINTS

- To ensure a systematic review is valuable to users, authors should prepare a transparent, complete, and accurate account of why the review was done, what they did, and what they found
- The PRISMA 2020 statement provides updated reporting guidance for systematic reviews that reflects advances in methods to identify, select, appraise, and synthesise studies
- The PRISMA 2020 statement consists of a 27-item checklist, an expanded checklist that details reporting recommendations for each item, the PRISMA 2020 abstract checklist, and revised flow diagrams for original and updated reviews
- We anticipate that the PRISMA 2020 statement will benefit authors, editors, and peer reviewers of systematic reviews, and different users of reviews, including guideline developers, policy makers, healthcare providers, patients, and other stakeholders

Systematic reviews serve many critical roles. They can provide syntheses of the state of knowledge in a field, from which future research priorities can be identified; they can address questions that otherwise could not be answered by individual studies; they can identify problems in primary research that should be rectified in future studies; and they can generate or evaluate theories about how or why phenomena occur. Systematic reviews therefore generate various types of knowledge for different users of reviews (such as patients, healthcare providers, researchers, and policy makers).[1 2] To ensure a systematic review is valuable to users, authors should prepare a transparent, complete, and accurate account of why the review was done, what they did (such as how studies were identified and selected) and what they found (such as characteristics of contributing studies and results of meta-analyses). Up-to-date reporting guidance facilitates authors achieving this.[3]

The Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) statement published in 2009 (hereafter referred to as PRISMA 2009)[4-10] is a reporting guideline designed to address poor reporting of systematic reviews.[11] The PRISMA 2009 statement comprised a checklist of 27 items recommended for reporting in systematic reviews and an "explanation and elaboration" paper[12-16] providing additional reporting guidance for each item, along with exemplars of reporting. The recommendations have been widely endorsed and adopted, as evidenced by its co-publication in multiple journals, citation in over 60 000 reports (Scopus, August 2020), endorsement from almost 200 journals and systematic review organisations, and adoption in various disciplines. Evidence from observational studies suggests that use of the PRISMA 2009 statement is associated with more complete reporting of systematic reviews,[17-20] although more could be done to improve adherence to the guideline.[21]

Many innovations in the conduct of systematic reviews have occurred since publication of the PRISMA 2009 statement. For example, technological advances have enabled the use of natural language processing and machine learning to identify relevant evidence,[22-24] methods have been proposed to

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ATTACHMENT LEVEL IN PERIODONTITIS PATIENTS WITH HABIT OF TOBACCO CONSUMPTION

Original Research

Hiral Parikh , Archita Kikani**, Manish Garnara*** , Mihir Shah**** , Sandip Ladani******

ABSTRACT

Aim : The present study was carried out to compare the periodontal status & pattern of attachment loss in periodontitis patients; who are current smokers, current tobacco chewers & non-smoker and non-tobacco chewers.
Material and Method : 150 male subjects ranging from 18 to 60 years having periodontitis were selected. Smoking and tobacco consumption history was obtained through personal interview. Then patients were divided into 3 group current smokers, current tobacco chewers and non-smoker & non-tobacco chewer. Clinical parameters include loss of attachment, marginal gingival index, probing pocket depth. Female patients, patients of tobacco chewers and smokers (both), past smokers &/or tobacco chewers, patients who had undergone periodontal treatment in last 3 months, patients who have other forms of periodontitis & patients who have systemic history are excluded. Collected data will be analyzed statistically.
Results : The current smokers and tobacco chewers exhibited higher level of attachment loss and probing pocket depth than nontobacco chewer and nonsmokers. The results of analysis showed that the attachment loss in smokers and chewers 1.3mm and 0.7 mm more than nonsmokers and nontobacco chewers. Bleeding on probing was more in nonsmoker and nontobacco chewers than smokers and tobacco chewers.
Conclusion : The overall pattern of attachment loss in smokers exhibited more than tobacco chewers, nonsmokers and non-tobacco chewers. Both tobacco smoke and tobacco components may reduce gingival blood flow and gingival bleeding. This could be either due to vasoconstriction of gingival vessels or to the heavier keratinization of the gingiva in smokers.

Key words: smoking, tobacco, periodontal disease, periodontitis, attachment level.
Received : 11-09-2012 ; Review Completed :18-12-2012 ; Accepted : 02-01-2013

INTRODUCTION

A position paper from the American Academy of Periodontology (1999) concluded that tobacco use in an important factor affecting the prevalence and progression of most forms of periodontitis. The relationship between cigarette smoking and periodontal disease status has been extensively examined. Subjects who smoke cigarettes have a greater risk of exhibiting more extensive and more severe alveolar bone loss [1-4] and greater periodontal attachment loss [4-9] than subjects who never smoked cigarettes. In addition, smokers tend to have greater numbers of deeper periodontal pockets and mean probing pocket depth [10-13]. Early studies suggested that smokers exhibited worse oral hygiene than nonsmokers[14-18]. Furcation involvement at molar teeth is more frequent in smokers than non-smokers[11-13]. Longitudinal studies have shown continued periodontal deterioration in those who smoked throughout the period of investigation6. The severity of smoking associated periodontal destruction appears to be related to the duration of use. A recent study of young male recruits in Spain found that moderate to heavy smoking was associated with accelerated loss of attachment.

The effect of cigarette smoking on periodontal disease status in subjects with different systemic backgrounds has also been examined. When the relationship of IL-1 polymorphism to periodontal disease severity was examined in current and non-smokers, it was found that both smoking and genotype related to an increase risk of tooth loss[12]. It has also been found that diabetics who smoke are at greater risk than diabetic who do not smoke and that diabetics who smoked exhibited significantly more periodontal disease than non-diabetic who smoked[1]. Thus, a picture emerges suggesting that smokers have greater bone loss, greater attachment loss, deeper periodontal pockets and more supra gingival calculus than non-smokers but less bleeding from periodontal sites. Smokers appear to have similar

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Goel *et al. BMC Oral Health* (2021) 21:66
https://doi.org/10.1186/s12903-021-01416-3

BMC Oral Health

RESEARCH ARTICLE Open Access

Current status of periodontitis and its association with tobacco use amongst adult population of Sunsari district, in Nepal

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Abstract

Background: Tobacco products are considered significant, but preventable factors related to initiation and progression of periodontal diseases. We assessed the prevalence of periodontitis and evaluated its association with tobacco use and other factors amongst the adult population of Sunsari district in eastern Nepal.

Methods: A community-based, cross-sectional study was conducted in rural municipalities in the province one of eastern Nepal. A total of 440 adults were interviewed with a set of a pre-tested semi-structured questionnaire. Data on social demographics, adverse oral habits followed by periodontal clinical examination were recorded. Prevalence of periodontitis was assessed by a case definition provided by CDC-AAP. Univariate and multivariate logistic regression analysis was done to measure the association between tobacco use and other factors with periodontitis.

Results: The overall prevalence of periodontitis was found to be 71.6%. Majority (85.4%) of tobacco users had periodontitis and they were significantly associated with the disease and its severity. The study identified age groups, 45–65 years (AOR = 7.58, 95% CI 3.93–14.61), plaque accumulation (AOR = 1.01, 95% CI 1.00–1.02), smoking (AOR = 3.14, 95% CI 1.36–7.27), khaini users (smokeless tobacco, AOR = 2.27, 95% CI 1.12–4.61) and teeth loss (AOR = 2.02, 95% CI 1.21–3.38) as the significant factors associated with periodontitis.

Conclusion: The prevalence of periodontitis is high in the surveyed rural adult population. Cigarette smoking along with the use of smokeless tobacco in the form of khaini were identified as significant factors associated with periodontitis.

Keywords: Periodontitis, Prevalence, Smoking, Smokeless tobacco

Background

Periodontal diseases are a result of a disruption in the host microbial interaction, and are known to be one of the major causes of tooth loss [1]. Overall, this disease affects about 20–50% of the global population [2] and in its severe form, ranks sixth among the most prevalent disorders [3]. Although dental plaque-associated microorganism are the primary etiologic agent, several other factors such as genetic, systemic, immunological, environmental and behavioral factors play an important role in determining the susceptibility of individuals to periodontal diseases [4, 5].

Among the environmental factors, tobacco smoking is considered one of the true risk factors and is known to be independently related to periodontal destruction [6]. The common forms of tobacco smoking are cigarette, beedi, chutta and hooka, with cigarettes being the main product smoked [7]. More than seven thousand toxins are present in tobacco smoke [8] including, carcinogens and addictive psycho-active substances like nicotine,

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Article

Periodontal Health Status in Adults Exposed to Tobacco Heating System Aerosol and Cigarette Smoke vs. Non-Smokers: A Cross-Sectional Study

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Abstract: Tobacco heating systems (THS) are new products on the market, advertised as a less harmful alternative for smokers, in which tobacco is heated and not burned like in conventional cigarettes. This research explored the effect on periodontal tissues in contact with heating and burning tobacco residual products (smoke and tobacco). Methods: The sample included 66 subjects, patients of the Clinic of Dentistry in Rijeka, Croatia, aged 26–56 (median 38), 64% females. Three age- and gender-matched groups were formed (each $N = 22$): non-smokers, classic cigarettes smokers and THS smokers. Probing depth (PD) and clinical attachment loss (CAL) were primary research parameters. Results: Three groups differed in average PD and CAL ($p \leq 0.002$), with cigarette smokers having the highest and non-smokers the lowest values ($p \leq 0.002$). THS consumers generally had lower values of periodontal indices than smokers, but only CAL differed significantly ($p = 0.011$). Periodontal indices CAL and PD were worse in THS consumers than non-smokers, but they did not reach a level of statistical significance. Cigarette smoking was the only predictor of periodontitis (average CAL ≥ 4 mm) in logistic regression models, with an odds ratio of 4.7 (95% confidence interval 1.2–18.3; $p = 0.027$). Conclusions: Exposure to nicotine-containing aerosol of THS in adults has a less harmful effect on periodontal tissues, measurable through periodontal indices (PD and CAL), compared to burning tobacco of conventional cigarettes. THS, presented as an alternative product to classic cigarettes, also has a detrimental effect on the periodontium.

Keywords: cigarette smoking; electronic nicotine delivery system; periodontics; periodontitis; tobacco; smokers

Citation: Mišković, I.; Kuiš, D.; Špalj, S.; Pupovac, A.; Prpić, J. Periodontal Health Status in Adults Exposed to Tobacco Heating System Aerosol and Cigarette Smoke vs. Non-Smokers: A Cross-Sectional Study. *Dent. J.* **2024**, *12*, 26. https://doi.org/10.3390/dj12020026

Academic Editor: Kenneth Markowitz

Received: 7 December 2023
Revised: 21 January 2024
Accepted: 24 January 2024
Published: 29 January 2024

1. Introduction

The link between smoking and periodontitis has been well known for several decades, and a large number of studies demonstrated beyond doubt that cigarette smoke has a marked negative effect on periodontal tissues. The fact that a person is a smoker increases the odds ratio of acquiring periodontal disease by 85% [1].

Tobacco smoke as a product of conventional cigarettes contains a wide variety of harmful, mutagenic and carcinogenic chemicals such as nicotine, carbon monoxide, arsenic, hydrogen cyanide, benzene, reactive radicals, and tobacco-derived nitrosamines [2]. The effects of these cigarette smoke chemicals on the hard and soft tissues of the oral cavity are very well known and documented. Cancer of the oral cavity and pharynx, periodontitis, dental caries, oral pain, and diminished salivary flow are just a few of many conditions

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Original Article

Effect of tobacco smoking on the periodontal health in Parsa, Nepal: A comparative cross-sectional study

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Abstract

Introduction: For ages, smoking tobacco has been a part of cultural practice in different parts of Nepal. For periodontal diseases, one of the important risk factors is tobacco smoking, mainly cigarette smoking.

Objectives: This study was conducted to find out the relationship among tobacco smoking and periodontal disease by means of clinical attachment loss (CAL), plaque score, and gingival score in cigarette smoker patients in comparison with nonsmokers.

Methodology: A comparative cross-sectional study was done among 300 patients who visited the Outpatient Department of Periodontics, M. B. Kedia Dental College, Parsa, Nepal. The patients were divided into two groups, cigarette smokers and nonsmokers. Oral cavity examination was done using plaque index, gingival index, and CAL. Mann–Whitney test was done to determine the relation between tobacco smoker and overall periodontal health.

Results: The result shows that the overall median plaque score of the patients was 1.08, the gingival score of the patients was 1.22, and CAL was 2.04. Furthermore, there was statistical significance between periodontitis and cigarette smoking.

Conclusion: The present study concluded that smoking has a negative effect on periodontium health. More duration of cigarette smoking habit had higher severity of symptoms when compared to nonsmokers.

Keywords: Clinical attachment loss, periodontitis, tobacco smoking

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Submitted: 27-Apr-2023 **Revised:** 04-Sep-2023 **Accepted:** 11-Sep-2023 **Published:** 02-Jan-2024

INTRODUCTION

Periodontal diseases show a multifactorial causation phenomenon.[1] It has cyclic patterns of progression and resolution at any given site.[1] The use of tobacco is one of the prime threats to oral health today. Tobacco is available in different forms, which can be used primarily in smoking, chewing, sucking, and sniffing forms of tobacco. Cigarette smoking is a well-established modifiable risk factor for periodontal diseases.[2] Tobacco smoking is one of the major risk factors for the prevalence, severity, and extension of periodontal tissue breakdown.[3]

When compared to nonsmokers, tobacco/cigarette smokers have more periodontal damage. Smoking negatively affects bone transplantation as well. Smoking

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Quick Response Code:

Website: https://journals.lww.com/jorr

DOI: 10.4103/jorr.jorr_29_23

How to cite this article: Shah R, Singh A, Singh HM, Gupta R, Ghosh S. Effect of tobacco smoking on the periodontal health in Parsa, Nepal: A comparative cross-sectional study. J Oral Res Rev 2024;16:1-5.

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Archives of Oral Biology 117 (2020) 104793

Contents lists available at ScienceDirect

Archives of Oral Biology

journal homepage: www.elsevier.com/locate/archoralbio

Quantification of pathogenic bacteria in the subgingival oral biofilm samples collected from cigarette-smokers, individuals using electronic nicotine delivery systems and non-smokers with and without periodontitis

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ARTICLE INFO

Keywords:
Bacteria
Cigarette smoking
Periodontitis
Electronic nicotine delivery systems
Subgingival oral biofilm

ABSTRACT

Objective: The aim of the present study was to quantify pathogenic bacteria isolated from the subgingival oral-biofilm samples collected from cigarette-smokers and ENDS-users with periodontitis, when compared to non-smokers with and without periodontitis.
Methods: Demographic data was collected using a questionnaire. Periodontal parameters (plaque [PI] and gingival [GI] indices, clinical attachment loss [CAL], probing depth [PD] and marginal bone loss [MBL]) were measured. Subgingival oral bio-film samples were collected and assessed for periodontopathogenic bacteria (*Aggregatibacter actinomycetemcomitans* [*A. actinomycetemcomitans*], *Prevotella intermedia* [*P. intermedia*], *Porphyromonas gingivalis* [*P. gingivalis*], *Tannerella forsythia* [*T. forsythia*] and *Treponema denticola* [*T. denticola*]). Group-comparisons were performed; and $P < 0.01$ were considered statistically significant.
Results: All cigarette-smokers, ENDS-users and non-smokers with periodontitis had Grade-B periodontitis. The CFU/mL of *A. actinomycetemcomitans* ($P < 0.001$) and *P. gingivalis* ($P < 0.001$) were significantly higher among cigarette-smokers ($P < 0.01$) and ENDS-users ($P < 0.01$) than non-smokers with periodontitis. The CFU/mL of *T. denticola* were significantly higher among cigarette-smokers ($P < 0.001$), ENDS-users ($P < 0.001$) and non-smokers with periodontitis ($P < 0.001$) compared with non-smokers without periodontitis. There was no statistically significant difference in the CFU/mL of *P. intermedia* and *T. denticola* among cigarette-smokers, ENDS-users and non-smokers with periodontitis.
Conclusion: Counts of periodontopathogenic bacteria in the subgingival oral-biofilm are comparable among cigarette-smokers and individuals using ENDS.

1. Introduction

Cigarette-smoking is a significant risk-factor of oral diseases such as aggressive and non-aggressive (chronic) periodontitis (Javed, Rahman, & Romanos, 2019; Mullally, 2004; Senturk, Sezgin, Bulut, & Ozdemir, 2018). Tobacco-smokers are usually aware of the deleterious effects of smoking on health (Kawakami, 2000); however, quitting smoking is challenging for these individuals as nicotine (a major component in tobacco) is addictive and often sparks withdrawal symptoms such as anxiety, irritability/anger, restlessness and insomnia (Benowitz, 2010; McLaughlin, Dani, & De Biasi, 2015). Individuals attempting to or after quitting tobacco smoking often use consume nicotine in other forms such as chewing gums, patches, nasal sprays, waterpipes and electronic nicotine delivery systems (ENDS) such as electronic-cigarettes and JUUL (Alahmari, Javed, Ahmed, Romanos, & Al-Kheraif, 2019; Alqahtani, Alqahtani, Shafqat et al., 2019; BinShabaib et al., 2019; Javed et al., 2019; Nardone, Helen, Addo, Meighan, & Benowitz, 2019). Electronic-cigarettes are ENDS in which, a tank filled with a usually flavored nicotine-containing electronic liquid (e-liquid) is connected to a battery. The battery once powered, sets up an electric current that heats the e-liquid to produce vapor that is inhaled (Javed et al., 2019).

Individuals using ENDS (including pregnant women) often perceive

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https://doi.org/10.1016/j.archoralbio.2020.104793
Received 23 January 2020; Received in revised form 30 May 2020; Accepted 31 May 2020

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ORIGINAL RESEARCH

JIDA

Periodontics

Immunolocalization of gingival E-cadherin expression in smokers and non-smokers with chronic periodontitis

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Access this article online

Quick Response Code:

Website: jida.ida.org.in

DOI: 10.33882/jida.14.27157

How to cite this article: P. K. Ramesh, R. Renuka Devi, H Esther Nalini, P Arun Kumar Prasad. Immunolocalization of gingival E-cadherin expression in smokers and non-smokers with chronic periodontitis. JIDA 2020; XIV;12-16. https://doi.org/10.33882/jida.14.27157.

Abstract

Brief Background

Tobacco smoking is a well-known risk factor for periodontitis. It destroys the extracellular matrix proteins and cell-cell interaction proteins (E-cadherin) during the infection or tissue repair.

Materials and Methods

45 subjects were included in the study according to the inclusion criteria. The subjects were divided into three groups: Group A, B and C. Gingival E-cadherin expression was evaluated using Immunohistochemical analysis (IHC)..

Results

The mean value of E-cadherin for Group A, Group B and Group C are 2.5, 1.3 and 1.3 respectively. The healthy subjects are having higher E-cadherin expression than periodontitis and smoker with periodontitis group.

Summary and Conclusions

Expression of E-cadherin observed in the gingival epithelium in groups of periodontitis with and without smokers was not statistically significant.

Key Words

E-cadherin, IHC, Periodontitis, Smoking.

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ORIGINAL RESEARCH

Estimation and correlation of salivary thiocyanate levels in periodontally healthy subjects, smokers, nonsmokers, and gutka-chewers with chronic periodontitis

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ABSTRACT

Aim: This study was conducted to estimate and correlate salivary thiocyanate (SCN) levels in periodontally healthy subjects, smokers, nonsmokers, and gutka-chewers with chronic periodontitis.

Methodology: The study population consisted of 40 systemically healthy subjects in the age group of 18–55 years that was further divided into four groups: Control, smokers, nonsmokers, and gutka-chewers with chronic periodontitis. Gingival index (GI) (Loe and Silness-1963), probing depth (PD), clinical attachment loss was assessed. Estimation of SCN was performed by ultraviolet spectrophotometer at 447 nm wavelength. Statistical analysis was performed using the one-way ANOVAs Welch test and Pearson's correlation test using SPSS version 17 software.

Results: Results showed statistically significant increase in SCN levels in smokers as compared to gutka-chewers with chronic periodontitis, control, and nonsmokers with chronic periodontitis subjects. Significantly higher PD and loss of attachment were seen in smokers group compared with other groups. A negative correlation observed between the GI and thiocyanate levels.

Conclusion: The present study revealed a significant increase in SCN levels in smokers with periodontitis as compared to nonsmokers.

Received : 28-10-15
Review completed : 26-12-15
Accepted : 03-02-16

Key words: Salivary peroxidase, salivary thiocyanate, smokeless tobacco, smoking

Smoking is a major risk factor for chronic periodontitis.[1] Periodontitis is a multifactorial disease which is initiated by plaque and influenced by other factors which also play a role in the pathogenesis and disease progression.[2] Effect of smoking manifests as increased loss of attachment, development, and progress of inflammation, whereas smokeless tobacco leads to/associated with localized gingival recession at the site of tobacco placement.[3-5] Smokeless tobacco is commercially available as gutka, which contains areca nut, slaked lime and spices[6] and more nicotine content than cigarette.[7] Periodontal disease has been primarily diagnosed by clinical and radiographic findings.[8] Saliva can be a valuable source because it contains specific biomarkers for periodontal diseases.[9]

Salivary thiocyanate (SCN) is a metabolic product of cyanide, found in organic and inorganic compounds. They prevent toxic accumulations of hydrogen peroxide (H_2O_2) and hypochlorite (ClO^-), which may be carcinogenic or mutagenic.[10]

SCN concentrations are variable; it ranges from 0.5 to 2 mM in nonsmokers, whereas it can be as high as 6 mM in heavy smokers.[11]

The present study was taken up to estimate and correlate SCN levels in periodontally healthy subjects,

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Access this article online

Quick Response Code:

Website: www.ijdr.in

DOI: 10.4103/0970-9290.179807

How to cite this article: Hegde S, Chatterjee E, Rajesh KS, Arun Kumar MS. Estimation and correlation of salivary thiocyanate levels in periodontally healthy subjects, smokers, nonsmokers, and gutka-chewers with chronic periodontitis. Indian J Dent Res 2016;27:12-4.

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Research Paper

Tobacco Induced Diseases

Microbiological and biochemical findings in relation to clinical periodontal status in active smokers, non-smokers and passive smokers

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ABSTRACT

INTRODUCTION Cigarette users are more susceptible than non-smokers to periodontitis, a bacterial-induced, inflammation-driven, destructive disease of the supporting tissues of the teeth. We hypothesized that clinical periodontal findings and microbiological and/or inflammatory marker levels would be intermediate in those exposed to environmental tobacco smoke compared to active smokers and non-smokers.

METHODS Sixty individuals were recruited from a University periodontal clinic and assigned as non-smokers, active smokers or passive-smokers according to their self reports. Clinical periodontal measurements, comprising plaque index, probing depth (PD), clinical attachment level (CAL) and bleeding on probing, were recorded at six sites per tooth. Cotinine levels were determined in whole saliva samples by EIA. *Treponema denticola* and *Porphyromonas gingivalis* infection was determined by PCR, while matrix metalloproteinase-8 (MMP-8) and interleukin-8 (IL-8) concentrations were determined by ELISA.

RESULTS Study groups were subsequently reassigned in accordance with the cotinine data. The smoker group exhibited higher mean PD and CAL values compared to the non-smoker group ($p<0.05$). Passive-smokers exhibited PD and CAL values smaller than those of the active smokers and greater than those of the non-smokers, but the differences were not statistically significant. PD and CAL values correlated with cotinine concentrations ($p<0.05$). *P. gingivalis* infection was noted in most subjects, irrespective of smoking status. *T. denticola* infection was noted in 4/23 (17.4%) smokers, 0/16 (0%) environmentally-exposed recruits and 2/21 (9.5%) non-smokers. Salivary MMP-8 and IL-8 levels were lower in smokers compared to both non-smokers and passive-smokers but the differences were not significant (all $p>0.05$).

CONCLUSIONS The present clinical periodontal findings provide further support for a negative, dose-related effect of tobacco exposure on periodontal health. The tendency for a more prevalent detection of *T. denticola* and for a suppressed inflammatory response observed in the smokers may partly explain the increased susceptibility to periodontal tissue destruction, but needs to be verified in larger scale studies.

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KEYWORDS
bacteria, cytokines, environmental tobacco smoke, matrix metalloproteinases, saliva

Received: 20 November 2018
Revised: 16 February 2019
Accepted: 18 February 2019

INTRODUCTION

Severe periodontitis leads to loss of teeth and impairs functions of dentition (e.g. mastication, speech, and facial esthetics). Interactions between microbial, immunological, environmental, and genetic risk factors, as well as age, sex, and race affect

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Received: 20th May 2015 Accepted: 11th August 2015 Conflicts of Interest: None
Source of Support: Nil

Original Research

Short-Term Effectiveness of Scaling and Root Planning on Periodontal Parameters, Systemic Inflammatory, and Oxidative Stress Markers in Smokers with Chronic Periodontitis

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How to cite the article:
Aziz AS, Kalekar MG, Benjamin T, Suryakar AN, Patil S, Bijle MN. Short term effectiveness of scaling and root planning on periodontal parameters, systemic inflammatory and oxidative stress markers in smokers with chronic periodontitis. J Int Oral Health 2015;7(10):16-23.
Abstract:
Background: Tobacco smoking is one of the main risk factor associated with periodontitis and has long-term altered inflammatory, immunological, and therapy response. This study comparatively assesses the short-term effectiveness of scaling and root planing (SRP) on clinical periodontal parameters, some systemic inflammatory and oxidative stress (OS) markers between male smokers and nonsmokers with chronic periodontitis (CP).
Materials and Methods: The study groups comprised of 131 males with severe CP (clinical attachment loss [CAL] ≥ 5 mm). They were divided into Group I (n = 51, mean age: 40.9 ± 4.6) without smoking habits (CP), and Group II (n = 80, mean age: 44.1 ± 5.81) with smoking habits (CPSM). The clinical periodontal evaluation was done by measuring gingival index (GI), plaque index, probing depth, and CAL using a UNC-15 probe. The biochemical markers estimated were interleukin (IL)-6, 10, C-reactive protein, total antioxidant capacity (TAOC), RBC - superoxide dismutase, glutathione peroxidase), vitamin C, and malondialdehyde (MDA). SRP was performed on both the study groups with a follow-up after 3 months. The periodontal status and biochemical markers were estimated at baseline and post-treatment.
Results: Smokers with CP showed significantly higher (P < 0.001 for all parameters) periodontal damage and higher systemic inflammatory and OS markers compared to non-smokers with CP (P ≤ 0.05 for TAOC, GPx, Vitamin C and MDA). Post SRP improvements in the mean values (compared to baseline values) were observed in both the groups (P < 0.001 for all parameters). Comparatively, the CPSM group showed significantly lower (P ≤ 0.05 for all parameters) relative % change post SRP in the study parameters than those of CP group.
Conclusions: Smokers with CP exhibited more periodontal damage and higher systemic inflammatory and OS burden than nonsmokers with CP. Under the study condition, SRP was effective in improving periodontal and systemic inflammatory and OS markers in both the groups, although the improvement was lower in smokers than non-smokers. SRP could be a useful supportive therapy in checking periodontitis induced inflammation and OS burden on the systemic health of smokers.

Key Words: Biochemical markers, chronic periodontitis, oxidative stress, root planing smoking

Introduction

Smoking represents a major preventable cause of many human diseases.[1] One among them is chronic destructive periodontal disease.[2] Smokers show severe sub-gingival calculus formation[2] and are more likely to have severe periodontitis compared to the non-smokers.[3] Smoking is also associated with oxidative stress (OS), in which smoke derived oxidants are the major factors in inflammatory reactions. An alteration in antioxidant defenses and upregulation of inflammation by these oxidants may lead to an extensive oxidative damage.[4-6]

With respect to periodontal tissue damage, smoking has a long term chronic effect on the local oral inflammatory and immune response.[7] It has also been implicated as a factor that reduces the effectiveness of periodontal treatment. Smokers respond to scaling and root planing (SRP) less favorably than non-smokers, especially in terms of probing level and bone level.[8] Smoking may alter the healing response through increased activity of proteolytic enzymes directed against the structural elements of periodontium, elevated levels of destructive inflammatory cytokines, and suppression of regenerative functions of periodontium.[9] The primary reason for development of periodontitis is the continued presence of periodontopathogens in the periodontium. Mechanical removal of these pathogens can reduce the inflammatory burden, but smokers show a resistance to lowering of them.[9]

Keeping in mind the above facts, this study comparatively assess the effectiveness of SRP on clinical periodontal

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ORIGINAL ARTICLE

EFFECT OF SMOKING ON C-REACTIVE PROTEIN LEVELS IN CHRONIC PERIODONTITIS

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ABSTRACT

The objective of the study was to compare the effect of smoking on the level of C-reactive protein (CRP) in chronic periodontal disease. The response of periodontal treatment and its effect on the level of the CRP was also observed in both groups.

Hundred patients with chronic periodontitis were taken. Smoking history of the patients was assessed and accordingly, they were categorized as: Group A (smokers) and Group B (non- smokers). Periodontal evaluation was done on every patient and blood samples were collected for noting the levels of CRP. All patients were then given treatment of chronic periodontitis by mechanical removal of calculus as well as prescription of antimicrobials and mouth rinses. Periodontal evaluation and blood samples were repeated three months after the given treatment to check the change in CRP levels.

At Baseline, CRP levels of smoker group was more than non- smoker group. Three months after the given periodontal treatment, the periodontal health was significantly improved. Mean CRP level in both smoker group and non- smoker group was significantly decreased.

Chronic periodontitis has significant effect on CRP levels. Chronic periodontitis may add to the inflammatory burden of an individual and this can be reverted to some extent if appropriate treatment is given. This response will not be affected by adverse effects of smoking.

Key Words: *C- reactive protein, Chronic periodontitis, Smoking.*

INTRODUCTION

Chronic Periodontal infections has been identified as a potential risk factor for systemic pathologies such as cardiovascular disease, atherosclerosis, stroke, diabetes mellitus, preterm labor, stroke, respiratory conditions.[1-3] Fortunately, it is a modifiable risk factor since periodontal disease can be easily prevented and treated.[4]

There is an increasing interest among the researchers to observe role of periodontitis on level of C-reactive protein in diseased as well otherwise healthy individuals.[5,6,7]

C-reactive protein is an acute phase protein synthesized in liver and is normally present as a trace constituent of plasma or serum in diseased as well otherwise healthy individuals. Elevated CRP goes hand in hand with traditional risk factors for heart disease, such as smoking, obesity, high blood pressure or elevated cholesterol, and rarely occurs in their absence. Recently, it is being preferred on ESR (Erythrocyte Sedimentation Rate), as a routine prognostic test and has become valuable as cholesterol level in lipid profile to predict cardiovascular diseases.[8,9,10]

Smoking has been strongly associated as a major etiological factor of the cardiovascular disease, high blood pressure, brain stem infarction[11], and periodontal disease. It is involved in with the periodontal attachment loss resulting in the active progression of disease. The deleterious effects of active smoking on the musculoskeletal system, soft tissue and bone wound healing have also been recorded.[12]

In spite of its hazardous health risks, smoking is very common in Pakistan. One out of every two to three middle-aged men in Pakistan, smoke cigarettes.[13] Several epidemiological studies have proved smoking as primary behavioral risk factor for the increased accumulation of plaque and Periodontal attachment loss.[14,15]

METHODOLOGY

Patient Selection

One hundred patients were included for the conduction of this interventional study visiting the

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Received for Publication: January 8, 2015
Revision Received: March 4, 2015
Revision Accepted: April 1, 2015

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J Clin Periodontol 2002: 29: 771–776
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Journal of Clinical Periodontology
0303-6979

Effects of smoking on periodontal tissues

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Calsina G, Ramón J-M, Echeverría J-J: Effects of smoking on periodontal tissues. J Clin Periodontol 2002; 29: 771–776. © Blackwell Munksgaard, 2002.

Abstract

Background/Aims: Most studies about the association between tobacco and periodontal disease have shown that tobacco negatively affects periodontal tissues, although some authors have failed to demonstrate such association. Very few studies have tried to find out whether the effect of tobacco on periodontal tissues was similar for women and men. The aims of this investigation were to confirm the possible relationship between tobacco consumption and periodontitis, to study the correlation between intensity of smoking and disease severity, and to investigate any differences between genders related to the effects of tobacco consumption in periodontal health.

Material and methods: In this case-control study, 240 dental patients were selected according to previously defined criteria and were divided in two groups according to their periodontal status. Patients with established periodontitis constituted the case group. The remaining patients constituted the control group. Smoking status, probing depth, gingival recession, clinical attachment level, tooth mobility, periodontal bleeding index and plaque index were determined for each participant. Generated data were processed for statistical analysis using multiple comparisons, covariance analysis and logistic regression analysis.

Results: Logistic regression analysis showed that smokers had 2.7 times and former smokers 2.3 times greater probabilities to have established periodontal disease than non-smokers, independent of age, sex and plaque index. Among cases, probing depth, gingival recession and clinical attachment level were greater in smokers than in former smokers or non-smokers, whereas plaque index did not show differences. Bleeding on probing was less evident in smokers than in non-smokers. There was a dose–effect relationship between cigarette consumption and the probability of having advanced periodontal disease. The association between tobacco smoking and periodontal disease was more evident after 10 years of smoking, independent of age, gender and plaque index. Finally, it was observed that tobacco affected periodontal tissues more severely in men than in women.

Conclusions: Smoking is a risk factor strongly associated with periodontitis. The effects of smoking on periodontal tissues depend on the number of cigarettes smoked daily and the duration of the habit. The effect of tobacco on periodontal tissues seems to be more pronounced in men than in women.

Key words: periodontal disease; periodontium; tobacco

Accepted for publication 16 July 2001

Although periodontal diseases are infections caused by dental plaque (Socransky 1970), risk factors could modify the periodontal response to microbial aggression (Page et al. 1985, Grossi et al. 1995, Clarke & Hirsch 1995). Tobacco smoking is considered one of these factors (Bergström & Preber 1994), and was first studied by Pindborg (1947) and Arno et al. (1958). Grossi et al. (1995) found that tobacco smoking was strongly associated with both attachment and bone loss. Smokers are more susceptible than non-smokers to advanced and aggressive forms of periodontitis (Haber et al. 1993, Ketabi & Hirsh 1997). In smokers, there seems to be a relationship between periodontal attachment loss, number of cigarettes smoked daily (Martinez-Canut et al. 1995), and number of years of tobacco consumption (Haber & Kent 1992). Probing depth (Bergström & Eliasson 1987a) and gin-

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Chronic periodontitis and smoking

Prevalence and dose-response relationship

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ABSTRACT

الأهداف: تحديد مدى انتشار وعلاقة الاستجابة للجرعة لالتهاب اللثة المزمن بين المدخنون في باكستان.

الطريقة: أجريت هذه الدراسة المستعرضة بين مشاركين يبحثون عن الرعاية الصحية للأسنان في كلية كراتشي للطب والأسنان، كراتشي، باكستان. بلغ عدد المشاركين ٤٤٣ متوسط العمر 44.3 (± 6.5) وذلك خلال الفترة من أبريل 2011 إلى ديسمبر 2011 بلغت نسبة الذكور 64.7% والأناث 35.2%. وقد أجريت مقابلات مع المشاركين حول البيانات الديموغرافية وعادات صحة الأسنان. اعتبرنا المشاركين الذين يعانون من التهاب اللثة المزمن تراوحت بين 3.5–5.5 ملم جيوب ضحلة، وأكثر من 5.5 ملم جيوب عميقة. قمنا كذلك بتقييم بيانات المشاركين باستخدام التوزيع التكراري للبيانات المبوبة والمتوسط الحسابي (الانحراف المعياري) للبيانات المستمرة.

النتائج: من بين 443 مشارك بلغت نسبة المدخنين 55.1% وغير المدخنين 44.9% على التوالي. ارتبط التدخين بشكل إحصائي بالشباب، والجنس الذكوري، وانخفاض مستوى التعليم. يقدر معدل انتشار التهاب اللثة المزمن لدى المدخنين 81.6%. كما انتشر التهاب اللثة وحدته بين المدخنين بشكل مرتفع مقارنة مع المدخنين بشكل متوسط وخفيف. يصور نموذج متعدد المتغيرات غير المعدل أن المدخنون معرضين 3.5 مرة لالتهاب اللثة المزمن.

الخاتمة: انتشر التهاب اللثة المزمنة بشكل أعلى بين المدخنين . كما اظهرت الدراسة أن المدخنون بشكل مرتفع عرضة بشكل أكثر للإصابة بالتهاب اللثة .

Objectives: To determine the prevalence and dose-response relationship of chronic periodontitis among smokers in Pakistan.

Methods: This is a cross-sectional study among participants seeking dental care in Karachi Medical and Dental College, Karachi, Pakistan. A total of 443 participants with a mean age of 44.3 (±6.5) participated in the study from April 2011 to December 2011. Males comprised 64.7%, and females comprised 35.2%. Participants were interviewed on social demographics and oral habits. Participants with shallow pockets (3.5-5.5 mm) and deep pockets (>5.5 mm) were considered suffering from chronic periodontitis. The characteristics of participants were assessed using frequency distribution for categorical variables and mean (standard deviation) for continuous variables.

Results: Among 443 participants, smokers were distributed as 55.1% and non-smokers as 44.9%. Smoking was found to be significantly related to young adults (p<0.007), male gender (p<0.001), and lower education level (p<0.01). Overall prevalence of chronic periodontitis among smokers was estimated at 81.6%. Heavy smoking was found to have significantly high prevalence (p<0.001) and severity (p<0.001) of periodontitis as compared with moderate and light smokers. The multivariate unadjusted model depicted 3.5 times higher risk of chronic periodontitis among smokers (p<0.001).

Conclusion: Chronic periodontitis had a high prevalence among smokers. Heavy smoking was found to have a higher risk for having periodontitis.

Saudi Med J 2016; Vol. 37 (8): 889-894
doi: 10.15537/smj.2016.8.14223

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Received 30th December 2015. Accepted 2nd June 2016.

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J Periodontol • May 2000

Smoking-Attributable Periodontitis in the United States: Findings From NHANES III

Scott L. Tomar* and Samira Asma†

Background: The principal objectives of this study were to examine the relationship between cigarette smoking and periodontitis and to estimate the proportion of periodontitis in the United States adult population that is attributable to cigarette smoking.

Methods: Data were derived from the Third National Health and Nutrition Examination Survey, a nationally representative multipurpose health survey conducted in 1988 to 1994. Participants were interviewed about tobacco use and examined by dentists trained to use standardized clinical criteria. Analysis was limited to dentate persons aged ≥18 years with complete clinical periodontal data and information on tobacco use and important covariates (n = 12,329). Data were weighted to provide U.S. national estimates, and analyses accounted for the complex sample design. We defined periodontitis as the presence of ≥1 site with clinical periodontal attachment level ≥4 mm apical to the cemento-enamel junction and probing depth ≥4 mm. Current cigarette smokers were those who had smoked ≥100 cigarettes over their lifetime and smoked at the time of the interview; former smokers had smoked ≥100 cigarettes but did not currently smoke; and never smokers had not smoked ≥100 cigarettes in their lifetime.

Results: We found that 27.9% (95% confidence interval [CI]: ±1.8%) of dentate adults were current smokers and 23.3% (95% CI: ±1.2%) were former smokers. Overall, 9.2% (95% CI: ±1.4%) of dentate adults met our case definition for periodontitis, which projects to about 15 million cases of periodontitis among U.S. adults. Modeling with multiple logistic regression revealed that current smokers were about 4 times as likely as persons who had never smoked to have periodontitis (prevalence odds ratio [OR_P] = 3.97; 95% CI, 3.20-4.93), after adjusting for age, gender, race/ethnicity, education, and income:poverty ratio. Former smokers were more likely than persons who had never smoked to have periodontitis (OR_P = 1.68; 95% CI, 1.31-2.17). Among current smokers, there was a dose-response relationship between cigarettes smoked per day and the odds of periodontitis (P <0.000001), ranging from OR_P = 2.79 (95% CI, 1.90-4.10) for ≤9 cigarettes per day to OR_P = 5.88 (95% CI, 4.03-8.58) for ≥31 cigarettes per day. Among former smokers, the odds of periodontitis declined with the number of years since quitting, from OR_P = 3.22 (95% CI, 2.18-4.76) for 0 to 2 years to OR_P = 1.15 (95% CI, 0.83-1.60) for ≥11 years. Applying standard epidemiologic formulas for the attributable fraction for the population, we calculated that 41.9% of periodontitis cases (6.4 million cases) in the U.S. adult population were attributable to current cigarette smoking and 10.9% (1.7 million cases) to former smoking. Among current smokers, 74.8% of their periodontitis was attributable to smoking.

Conclusions: Based on findings from this study and numerous other reports, we conclude that smoking is a major risk factor for periodontitis and may be responsible for more than half of periodontitis cases among adults in the United States. A large proportion of adult periodontitis may be preventable through prevention and cessation of cigarette smoking. *J Periodontol 2000;71:743-751.*

KEY WORDS

National Health and Nutrition Examination Survey; periodontal diseases/epidemiology; periodontitis/epidemiology; smoking/adverse effects; risk assessment; tobacco/adverse effects.

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Community Dent Oral Epidemiol 2002: 30: 115–23
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COMMUNITY DENTISTRY AND ORAL EPIDEMIOLOGY
ISSN 0301-5661

Tobacco use and oral hygiene as risk indicators for periodontitis

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Amarasena N, Ekanayaka AN, Herath L, Miyazaki H. Tobacco use and oral hygiene as risk indicators for periodontitis. Community Dent Oral Epidemiol 2002; 30: 115–23. © Munksgaard, 2002

Abstract – *Objective:* To detect the periodontal status of male smokers and betel chewers in a rural community in Sri Lanka and compare it with that of male non-tobacco users of the same community. *Methods:* A cross-sectional community based study was carried out in a sample of 2277 rural adult males aged 20–60 years, adopting multistage cluster sampling technique. The present analysis was confined to 2178 subjects who were mutually exclusive smokers, betel chewers or non-tobacco users. The periodontal status was assessed by clinical measurement of levels of bacterial plaque (PLI), gingival inflammation (GI) and loss of epithelial attachment (LA). All measurements were carried out on four sites of all teeth present except third molars and the mean values for periodontal parameters were calculated. *Results:* Bivariate analysis revealed that the overall periodontitis levels were significantly higher in betel chewers and smokers than in non-tobacco users. Multiple linear regression analysis showed that there were no significant effects of smoking and betel chewing *per se* on LA, independent of age, socioeconomic status (SES) and whether or not controlled for PLI. The effect of the quantified tobacco use on LA was statistically significant regardless of age, PLI or SES. However, the effect of the quantified tobacco use was considered limited when compared to that of oral hygiene. *Conclusions:* The findings highlighted the importance of oral hygiene in the aetiology of periodontitis while confirming the statistical significance of the quantified tobacco use on LA. Oral hygiene and the quantified tobacco use may be considered as risk indicators for periodontitis.

Key words: betel chewing; developing countries; multiple regression analysis; oral hygiene; periodontitis; risk; smoking; Sri Lanka; tobacco use

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Submitted 17 August 2000;
accepted 6 April 2001

Tobacco use, in particular smoking, has been a prominent theme among periodontal researchers, predominantly of the industrialised countries in the recent past. Accordingly, smoking has been implicated as one of the important environmental factors associated with periodontitis (1–6). The 1996 World Workshop in Periodontics reviewed these studies and a meta-analysis of data from a number of studies confirmed that 'smoking entailed an overall increased risk for severe disease; estimated overall odds ratio 2.82' (6). However, there is a paucity of information concerning tobacco use and periodontitis in developing countries (1–3) like Sri Lanka, where both oral hygiene levels and the severity of periodontitis may be worse than in developed countries (7–10).

The relationship between smokeless tobacco use and periodontitis has also been the subject of scientific inquiry, although there are few such studies in comparison with the many that have examined the association between smoking and periodontitis (1, 2, 11). Furthermore, most investigations into the smokeless tobacco–periodontal relationship have been confined to the use of smokeless tobacco in the form of snuff dipping and tobacco chewing (11). The habit of betel chewing with tobacco is a particular form of smokeless tobacco consumption that is predominantly practised in South Asian countries like Sri Lanka (12–16). It involves chewing a quid that includes betel leaf, lime, areca nut and tobacco. The association between tobacco consumed in this form and oral cancer in the Indian subcontinent has been widely documented over many years (14–16). However, relatively few studies have examined the relationship between this practice and periodontitis (9). There is a need to

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Periodontitis and Smoking: An Evidence-Based Appraisal

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INTRODUCTION

The awareness of the harmful effects of tobacco smoking on many organs and tissues in the body has gradually increased in the general population and bans on smoking in public places are becoming more and more common in many countries. In dentistry the harmfulness of smoking, surprisingly, has gained only limited concern. In spite of the fact that there is substantial knowledge to verify the effects of smoking on oral tissues, dental care in general has devoted very little time and effort to the information about these untoward effects. One reason for this negligence may be that the effects of smoking on oral and, in particular, periodontal health are insufficiently appreciated or underestimated.

Although the first reports on smoking and its potential effects on periodontal health emanate from the early 1950s, a more thorough understanding or acceptance of smoking as a periodontal health risk began with the appearance of 3 independent publications in 1983.[1-3] Since then, a gradually increasing interest in the relationship between smoking and the periodontal health condition has emerged. Over the past 10 to 15 years smoking has gained scientific acceptance as an important risk factor for destructive periodontal disease. In addition, the potential interference of smoking with the outcome of various periodontal therapies has been addressed in a number of investigations. The objective of the present systematic review, therefore, is to give a critical appraisal of the available literature on the subject to establish an evidence base regarding (1) the relationship between smoking and the periodontal health condition and (2) the influence of smoking on periodontal therapy outcome.

METHOD

A search in medical databases using MeSH terms related to "smoking" and "periodontal…" resulted in approximately 1050 hits. A further selection including clinical and epidemiological studies in humans alone resulted in a retrieval of 577 titles. A narrowing to include studies that only used measures of the periodontal destruction such as pocket probing depth, attachment level (or attachment loss), bone height (or bone loss), and tooth frequency further reduced the number of titles. Thus, studies that have used descriptors of inflammatory change alone such as gingival bleeding or gingival index were excluded. In addition, reviews, nonoriginal research articles, and opinionated statements have been excluded. In the final analysis, 129 titles including 105 titles referring to population observational studies and 24 titles referring to periodontal therapy intervention studies have been evaluated.

J Evid Base Dent Pract 2006;6:33-41
1532-3382/$35.00

doi:10.1016/j.jebdp.2005.12.018

RESULTS

Population Studies

Overall, the population studies that have addressed the relationship between smoking and periodontitis include 70 cross-sectional,[1-70] 14 case-control,[71-85] and 21 cohort[86-107] studies. The measures of the periodontal condition that are used as effect measures or endpoints in the population studies are periodontal pocket depth (PPD), clinical attachment level (or loss, CAL), periodontal bone level (or loss, PBL), and number of retained (or lost) teeth. Frequently more than one of these endpoints are used. A majority of population studies is based on 200 or more individuals.

1. Cross-sectional studies The most common type of population study is the cross-sectional study. Almost 70% of the population studies belong to this category representing 19 different nations The number of participants in the cross-sectional studies varies from 82 to 12,329, with an average of 1692 individuals per study. More than 80% of cross-sectional studies are based on 200 or more individuals (Table 1). Altogether, the cross-sectional studies cover a total of 79,444 individuals. Most cross-sectional studies concern a broad age range, and approximately 90% of the studies cover the ages 20 to 70 years or 40 years and above (Table 2). In 56% of the cross-sectional studies endpoint is used and in 34% 2 endpoints are used (Table 3). The most frequently applied endpoints are PPD, used in 53% of studies, and CAL, used in 50% of studies (Table 4).

Irrespective of endpoint, all cross-sectional studies demonstrate a statistically significant association between smoking and an impaired periodontal health condition suggesting that on the average smokers exhibit greater periodontal morbidity than nonsmokers. Statistical significance testing is presented in all studies, and *P* values range from less than .001 to less than .05. A summary of the results appears in Table 5. In 97% of 37 studies using PPD as the only or one of several endpoints, a significant association with smoking is demonstrated. Similarly, all 35 studies that use CAL, all 16 studies that use PBL, and 95% of 19 studies that use number of teeth

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Mat Soc Med. 2012 Dec 24(4): 274-276

Received: 27 September 2012
Accepted: 19 November 2012
Conflict of interest: none declared.

DOI: 10.5455/msm.2012.24.274-276

Smoking and Periodontal Disease

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REVIEW

ABSTRACT

Periodontitis is a group of inflammatory diseases affecting the supporting tissues of the tooth (periodontium). The periodontium consists of four tissues : gingiva, alveolar bone and periodontal ligaments. Tobbaco use is one of the modifiable risk factors and has enormous influance on the development, progres and tretmen results of periodontal disease. The relationship between smoking and periodontal health was investigated as early as the miiddle of last century. Smoking is an independent risk factor for the initiation, extent and severity of periodontal disease. Additionally, smoking can lower the chances for successful tretment. Tretmans in patients with periodontal disease must be focused on understanding the relationship between genetic and environmental factors. Only with individual approach we can identify our pacients risks and achieve better results.
Key words: smoking, periodontal disease.

1. INTRODUCTION

Periodontitis is a group of inflammatory diseases affecting the supporting tissues of the tooth (periodontium). The periodontium consists of four tissues : gingiva, alveolar bone and periodontal ligaments.

The periodontal diseases are highly prevalent and can affect up to 90% of the world wide population. Gingivitis, the mildest form of periodontal disease, is caused by the bacterial biofilm (dental plaque) that accumulates on teeth adjacent to the gingiva (gums).

The simtoms are usualy red, swollen gums who can bleed easily. However, gingivitis does not affect the underlying supporting structures of the teeth and is reversible. When gingivitis is not treated, it can advanece to periodontitis. Periodontitis results in loss of connective tissue and bone support and is a major cause of tooth loss in adults (1). In addition to pathogenic microorganisms in the biofilm, genetic and environmental factors has enormouse influence on development periodontal disease.Tobbaco use is one of the modifiable risk factors and has enormous influance on the development ,progres and tretmen results of periodontal disease.

The American Academy of Periodontology (AAP) has classified periodontitis into aggressive periodontitis (AgP), chronic periodontitis (CP) and periodontitis as a manifestation of systemic diseases (2). Both AgP and CP have a multi-factorial etiology with dental plaque as the initiating factor (3). However the initiation and progression of periodontitis are influenced by other factors including tobbaco use.

2. RELATIONSHIP BETWEEN SMOKING AND PERIODONTAL DISEASE

One-third of the world's adult population are smokers (57% of these are men, 43% are women) . It is predicted that in 20 years this yearly death rate from tobacco use will be more than 10 million people. Smoking in developing countries is rising by more than 3% a year (4). We can assume periodontal diseasees will also rise.

The relationship between smoking and periodontal health was investigated as early as the miiddle of last century. Smoking is an independent risk factor for the initiation, extent and severity of periodontal disease. Additionally, smoking can lower the chances for successful tretment. (Figure 1,2,3,4).

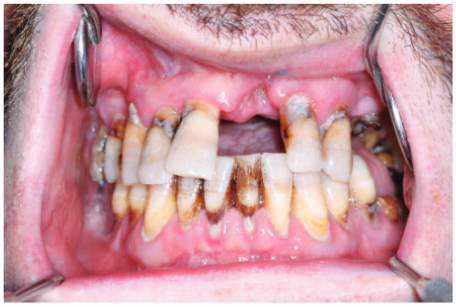

Figure 1. Generalized advanced chronic periodontitis in smooker

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J Clin Periodontol 2001; 28: 212–219
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Journal of Clinical Periodontology
ISSN 0303-6979

Smoking and subgingival microflora in periodontal disease

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Boström L, Bergström J, Dahlén G, Linder LE: Smoking and subgingival microflora in periodontal disease. J Clin Periodontol 2001; 28: 212–219. © Munksgaard, 2001.

Abstract
Aim: The present investigation was undertaken to analyze the influence of smoking on the periodontal disease associated subgingival microflora. The population included 33 smokers and 31 non-smokers in the age range 36–86 years.
Methods: Microbial samples were obtained from 4 sites per patient. The checkerboard DNA-DNA hybridization technology was used for detection of the bacterial species *P. gingivalis*, *P. intermedia*, *P. nigrescens*, *B. forsythus*, *A. actinomycetemcomitans*, *F. nucleatum*, *T. denticola*, *P. micros*, *C. rectus*, *E. corrodens*, *S. noxia* and *S. intermedius*.
Results: Using score 1 as cutoff, contrasting colonized versus non-colonized patients, 8 out of 12 species were detected in ≥90% of both smokers and non-smokers. Using score 4 as cutoff, contrasting heavily colonized patients versus non-colonized and less heavily colonized patients, the detection rates decreased in both smokers and non-smokers. No significant differences in detection rates were observed between smokers and non-smokers. Logistic regression analysis indicated that neither smoking, probing depth nor gingival bleeding influenced the occurrence of the species analyzed. The lack of a smoking exposure dose-response further supported the indication of a limited influence of smoking.
Conclusion: Smoking exerts little, if any, influence on the subgingival occurrence of several of the bacteria most commonly associated with periodontal disease.

Key words: DNA-DNA hybridization; periodontitis; smoking; subgingival microflora

Accepted for publication 22 March 2000

Periodontal disease, a local inflammation in the supporting tissues of the teeth leading to progressive loss of periodontal ligament and bone, is thought to be the result of a disruption of the homeostatic balance between the host response and pathogenic micro-organisms (Haffajee et al. 1991, Genco 1992). The rôle of smoking as a risk factor for periodontal disease is well documented (Bergström & Eliasson 1987, Bergström 1989, Haber et al. 1993, Bergström & Preber 1994, Grossi et al. 1994, Barbour et al. 1997). Studies on the relation between smoking and some subgingival periodontopathogens such as *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis* and *Prevotella intermedia* indicate that smoker and non-smoker patients largely exhibit the same subgingival microflora, suggesting that smoking has limited influence on the microflora involved in periodontal disease (Preber et al. 1992, Stoltenberg et al. 1993, Renvert et al. 1998). It has been reported, however, that smokers are more likely to harbour higher levels of the periodontopathogens *A. actinomycetemcomitans*, *P. gingivalis* and *Bacteroides forsythus* than non-smokers (Zambon et al. 1996). Furthermore, in a recent study on early onset periodontitis, it was reported that smokers harboured higher levels than non-smokers of some periodontopathogens (Kamma et al. 1999).

We have previously investigated the influence of smoking on the occurrence in gingival crevicular fluid (GCF) of *A. actinomycetemcomitans*, *P. gingivalis* and *P. intermedia* and their relations with immune response factors such as tumor necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1β) and IL-6 (Boström et al. 1998, 1999, 2000). Using culture technique, no influence of smoking on these periodontopathogens was observed. The object of the present study was to further extend the analysis of the subgingival microflora associated with periodontal disease using currently available checkerboard DNA-DNA hybridization technology (Socransky et al. 1994). By means of this technology we have investigated the influence of smoking on the GCF content of *P. gingivalis* (*Pg*), *P. intermedia* (*Pi*), *Prevotella nigrescens* (*Pn*), *B. forsythus* (*Bf*), *A. actinomycetemcomitans* (*Aa*), *Fusobacterium nucleatum* (*Fn*), *Treponema dentico-*

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frontiers in Microbiology

REVIEW
published: 29 January 2020
doi: 10.3389/fmicb.2020.00066

The Impact of Smoking on Subgingival Microflora: From Periodontal Health to Disease

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Periodontal disease is one of the most common diseases of the oral cavity affecting up to 90% of the worldwide population. Smoking has been identified as a major risk factor in the development and progression of periodontal disease. It is essential to assess the influence of smoking on subgingival microflora that is the principal etiological factor of the disease to clarify the contribution of smoking to periodontal disease. Therefore, this article reviews the current research findings regarding the impact of smoking on subgingival microflora and discusses several potential mechanisms. Cultivation-based and targeted molecular approaches yield controversial results in determining the presence or absence of smoking-induced differences in the prevalence or levels of certain periodontal pathogens, such as the "red complex." However, substantial changes in the subgingival microflora of smokers, regardless of their periodontal condition (clinical health, gingivitis, or periodontitis), have been demonstrated in recent microbiome studies. Available literature suggests that smoking facilitates early acquisition and colonization of periodontal pathogens, resulting in an "at-risk-for-harm" subgingival microbial community in the healthy periodontium. In periodontal diseases, the subgingival microflora in smokers is characterized by a pathogen-enriched community with lower resilience compared to that in non-smokers, which increases the difficulty of treatment. Biological changes in key pathogens, such as *Porphyromonas gingivalis*, together with the ineffective host immune response for clearance, might contribute to alterations in the subgingival microflora in smokers. Nonetheless, further studies are necessary to provide solid evidence for the underlying mechanisms.

Keywords: periodontal disease, smoking, subgingival microflora, nicotine, microbial diversity

OPEN ACCESS

Edited by:
Giovanna Batoni, University of Pisa, Italy

Reviewed by:
Kesavalu Naidu Lakshmiya, University of Florida, United States
Tomoki Maekawa, Niigata University, Japan

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Specialty section:
This article was submitted to Microbial Symbioses, a section of the journal Frontiers in Microbiology

Received: *25 October 2019*
Accepted: *13 January 2020*
Published: *29 January 2020*

Citation:
Jiang Y, Zhou X, Cheng L and Li M (2020) The Impact of Smoking on Subgingival Microflora: From Periodontal Health to Disease. Front. Microbiol. 11:66. doi: 10.3389/fmicb.2020.00066

INTRODUCTION

Smoking remains a highly prevalent addiction in many populations worldwide despite the increasing awareness of its harmful effects on general health (World Health Organization [WHO], 2018). The number of smokers is >1.1 billion (1 out of 7) globally now, and over 8 million people die annually because of smoking[1]. As one of the five leading risk factors for the global burden of the disease, smoking is responsible for various diseases, including cancer, cardiovascular

[1]https://www.who.int/news-room/fact-sheets/detail/tobacco

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Research Reports: Clinical

Journal of Dental Research
2023, Vol. 102(3) 280–286

DOI: 10.1177/00220345221135100
journals.sagepub.com/home/jdr

Effect of Smoking Exposure on Nonsurgical Periodontal Therapy: 1-Year Follow-up

F.R.M. Leite[1,2,3], R. López[1], J.B. Pajaniaye[1], and G.G. Nascimento[1,2,3]

Abstract

This study investigated the influence of different levels of exposure to smoking on periodontal healing for 12 mo after nonsurgical periodontal therapy and supportive periodontal care every third month. Eighty smokers willing to quit smoking and with periodontitis were included. Participants were offered an individualized voluntary smoking cessation program. Data collection included questionnaires and a full-mouth periodontal examination. Group-based trajectory modeling was used to model smoking trajectories over the follow-up. The effect of smoking trajectory on periodontal parameters over time was estimated with mixed effects modeling. Three smoking patterns were identified: light smokers/quitters ($n = 46$), moderate smokers ($n = 17$), and heavy smokers ($n = 17$). For the periodontal data, the first factor, moderate periodontitis, included the number of sites with clinical attachment levels (CALs) of 4, 5, 6, and 7 mm; periodontal pocket depths (PPDs) of 4, 5, and 6 mm; and bleeding on probing. The second factor, severe periodontitis, consisted of the number of sites with a CAL ≥8 mm and PPD ≥7 mm. Heavy smokers commenced with a higher average CAL of 1.1 mm and 10 more sites with severe periodontitis than light smokers/quitters. While light smokers/quitters and moderate smokers obtained an average improvement of 0.6-mm PPD and 0.7-mm CAL, respectively, heavy smokers experienced 0.5-mm attachment loss. Heavy smokers had only a 50% reduction in the number of sites with moderate periodontitis when compared with light smokers/quitters and moderate smokers. While most participants benefited from nonsurgical periodontal therapy with results affected in a dose-response manner, the therapy had no effect on severe periodontitis among heavy smokers. Smoking cessation should be part of periodontal therapy; otherwise, limited benefits would be observed among heavy smokers, hindering the effect of treatment.

Keywords: periodontitis, periodontal disease, smoking, smoking cessation, chronic periodontitis, tobacco

Introduction

Periodontitis has been described as a chronic, multicausal, inflammatory syndrome with clinical signs of progressive destruction of the tooth-supporting structures (Nascimento et al. 2017). Treatment usually comprises nonsurgical and surgical periodontal therapy, concomitant to establishing a personalized training program for daily plaque removal at home, named *active periodontal therapy*. This stage is followed by supportive periodontal care (SPC), aiming to sustain the active periodontal therapy results. The concept derives from the understanding that periodontitis development is driven solely by local biofilm dysbiosis, which triggers a non–self-resolving chronic inflammatory process. However, periodontitis severity and progression depend on several complex interactions, such as immunological, microbial, genetic, sociological, and environmental factors (Nascimento et al. 2017; Loos and Van Dyke 2020; Buduneli 2021).

After active periodontal therapy, the patient's susceptibility to recurrence of periodontitis is determined. Those more susceptible have been offered SPC sessions with 3- to 4-mo intervals, whereas less susceptible individuals are recommended larger intervals. Severe cases of periodontitis, patients with diabetes, and smokers, for instance, have been classified among the more susceptible group. However, despite the short interval between SPC sessions, studies demonstrated that more susceptible patients had a higher incidence of tooth loss than less susceptible individuals (Eickholz et al. 2008; Costa et al. 2022). The stability of clinical outcomes after active periodontal therapy is usually observed in mild to moderate cases of periodontal diseases (Eickholz et al. 2008). The fact may contribute to a reduction in SPC compliance as the risk for periodontitis and tooth loss increases (Leow et al. 2022). In the more susceptible patients, studies showed that long-term stability of periodontal parameters is rarely achieved; yet, a reduction in the rate of attachment and tooth loss may be

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A supplemental appendix to this article is available online.

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9.2. Anexo 2. Figuras y tablas

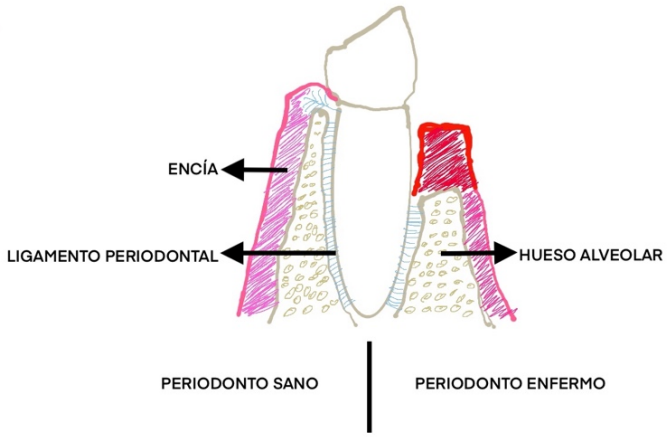

Figura 1. Tejidos periodontales sanos vs con enfermedad periodontal

Salud periodontal, enfermedades y condiciones gingivales.

1. **Salud periodontal y gingival**
   a. Salud gingival clínica en un periodonto intacto
   b. Salud gingival clínica en un periodonto reducido
      I. Paciente de periodontitis estable
      II. Paciente sin periodontitis

2. **Gingivitis inducida por biopelícula dental**
   a. Asociada solamente a biopelícula dental
   b. Mediada por factores de riesgo sistémicos o locales
   c. Agrandamiento gingival inducido por medicamentos

3. **Enfermedades gingivales no inducidas por biopelícula dental**
   a. Trastornos genéticos del desarrollo
   b. Infecciones específicas
   c. Condiciones inflamatorias e inmunes
   d. Procesos reactivos
   e. Neoplasias
   f. Enfermedades endocrinas, nutricionales y metabólicas
   g. Lesiones traumáticas
   h. Pigmentación gingival

Figura 2. Clasificación de Enfermedades y Alteraciones Periodontales y Periimplantarias 2017. Salud periodontal, enfermedades y condiciones gingivales. (25)

Formas de periodontitis

1. **Enfermedades periodontales necrosantes**
   a. Gingivitis necrosante
   b. Periodontitis necrosante
   c. Estomatitis necrosante

2. **Periodontitis como manifestación de enfermedades sistémicas**
   a. La clasificación de estas condiciones debe basarse en la enfermedad sistémica primaria, de acuerdo con los códigos de la clasificación estadística internacional de enfermedades y problemas de salud relacionados (ICD)

3. **Periodontitis**
   a. Estadios basados en la severidad y complejidad de manejo
      I. Estadio I: periodontitis inicial
      II. Estadio II: periodontitis moderada
      III. Estadio III: periodontitis severa con potencial de pérdida dental adicional
      IV. Estadio IV: periodontitis severa con pérdida potencial de la dentición
   b. Extensión y distribución: localizada, generalizada, distribución incisivo-molar
   c. Grados: evidencia o riesgo de progresión rápida, respuesta anticipada al tratamiento
      I. Grado A: tasa lenta de progresión
      II. Grado B: tasa moderada de progresión
      III. Grado C: tasa rápida de progresión

Figura 3. Clasificación de Enfermedades y Alteraciones Periodontales y Periimplantarias 2017. Formas de periodontitis. (25)

Manifestaciones periodontales de las enfermedades sistémicas y condiciones del desarrollo y adquiridas.

1. **Enfermedades sistémicas y condiciones que afectan los tejidos de soporte periodontal**

2. **Otras condiciones periodontales**
   a. Abscesos periodontales
   b. Lesiones endodóntico-periodontales

3. **Deformidades mucogingivales y condiciones alrededor de los dientes**
   a. Fenotipo gingival
   b. Recesión gingival/de tejido blando
   c. Falta de encía
   d. Profundidad vestibular reducida
   e. Frenillo aberrante/posición del músculo
   f. Exceso gingival
   g. Color anormal
   h. Condición de superficie radicular expuesta

4. **Fuerzas oclusales traumáticas**
   a. Trauma oclusal primario
   b. Trauma oclusal secundario
   c. Fuerzas ortodóncicas

5. **Factores protésicos y dentales que modifican o predisponen a las enfermedades gingivales/periodontales inducidas por placa**
   a. Factores localizados relacionados con dientes
   b. Factores localizados relacionados con prótesis dental

Figura 4. Clasificación de Enfermedades y Alteraciones Periodontales y Periimplantarias 2017. Manifestaciones periodontales de las enfermedades sistémicas y condiciones del desarrollo y adquiridas. (25)

Enfermedades y condiciones periimplantarias

1. **Salud periimplantaria**

2. **Mucositis periimplantaria**

3. **Periimplantitis**

4. **Deficiencias de tejidos blandos y duros periimplantarios**

Figura 5. Clasificación de Enfermedades y Alteraciones Periodontales y Periimplantarias 2017. Enfermedades y condiciones periodontales. (25)

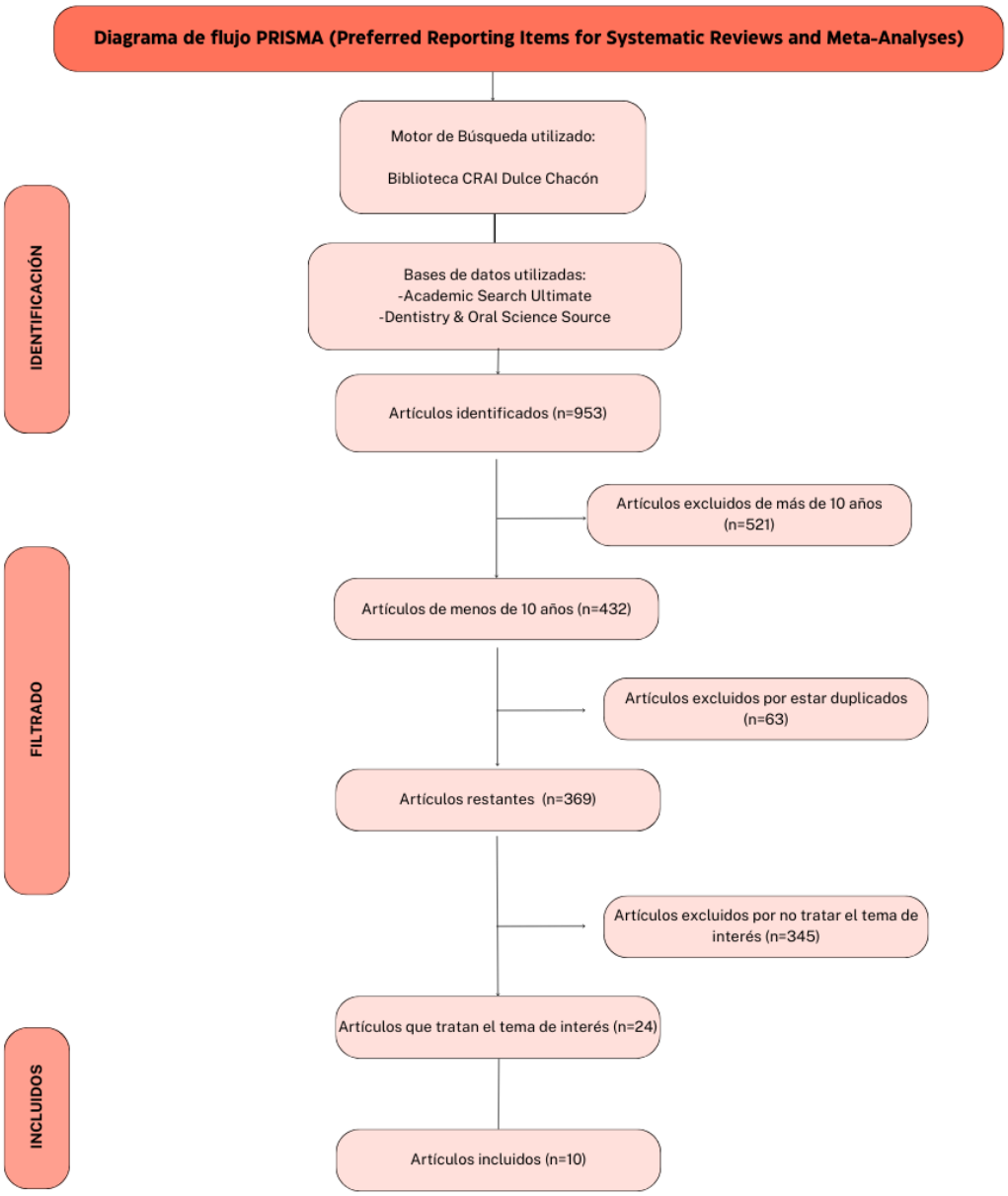

Figura 6. Diagrama de flujo PRISMA 2000. (28)

Tabla 1. Clasificación de periodontitis por grados, basada en evidencia directa, indirecta y factores modificadores. Tonetti y colaboradores (cols.). 2018. (23)

		Grado A	**Grado B**	**Grado C**
Evidencia directa	Radiografías o evaluación periodontal en los 5 últimos años	No evidencia de pérdida de hueso/inserción	Pérdida < 2 mm	Pérdida ≥ 2mm
	Pérdida ósea vs. Edad	< 0,25	0,25–1,0	> 1,0
Evidencia indirecta	Fenotipo	Grandes depósitos de biofilm con niveles bajos de destrucción	Destrucción proporcional a los niveles de biofilm	El grado de destrucción supera las expectativas teniendo en cuenta los depósitos de biofilm; patrones clínicos específicos que sugieren periodos de progresión rápida y/o patología de aparición temprana. Falta de respuesta prevista a tratamientos de control bacteriano habituales.
Factores modificadores	Tabaquismo	No fumador	< 10 cigarros/día	≥ 10 cigarros/día
	Diabetes	Normal con/sin diabetes	HbA1c < 7 con diabetes	HbA1c > 7 con diabetes

Tabla 2. Clasificación de periodontitis por estadios, según la gravedad del diagnóstico inicial y la complejidad, sobre la base de factores locales. Tonetti y cols. 2018. (23)

		Estadio I	**Estadio II**	**Estadio III**	**Estadio IV**
Gravedad	CAL interdental en zona de mayor pérdida	1–2 mm	3–4 mm	≥ 5 mm	≥ 5 mm
	Pérdida ósea radiográfica	Tercio coronal (<15 %)	Tercio coronal (15–33 %)	Extensión a tercio medio/apical de raíz	Extensión a tercio medio/apical de raíz
	Pérdida dentaria	Ninguna por periodontitis	Ninguna por periodontitis	≤ 4 dientes perdidos por periodontitis	≥ 5 dientes perdidos por periodontitis
Complejidad	Local	Profundidad de sondaje máxima ≤ 4mm	Profundidad de sondaje máxima ≤ 5 mm	Profundidad de sondaje máxima ≥ 6 mm	Profundidad de sondaje máxima ≥ 6 mm
		Pérdida ósea principalmente horizontal	Pérdida ósea principalmente horizontal	Además de complejidad Estadio II:	Además de complejidad Estadio III:
				Pérdida ósea vertical ≥ 3 mm	Necesidad de rehabilitación debido a:

			Afectación de furca grado II o III	Disfunción masticatoria
			Defecto de cresta moderado	Trauma oclusal secundario (movilidad dentaria ≤ 2)
				Defecto alveolar avanzado
				Colapso de mordida, abanicamiento dental, migraciones dentarias
				Menos de 20 dientes residuales (10 parejas con contacto oclusal)
Extensión y distribución	Añadir a estadio como descriptor	En cada estadio, describir extensión como localizada (> 30% de dientes implicados), generalizada o patrón molar/incisivo		

Tabla 3. Resultados de estudios incluidos.

Referencia	Casos	Controles	Tipo de estudio	Método de evaluación	Resultados
Hiral y cols. (29)	44	36	Observacional prospectivo	CAL, PD, BP con sonda CPI	CAL fue 1,3mm mayor en fumadores. PD: 4,47 ± 0,59mm en fumadores versus (vs.) 3,15 ± 0,58mm en no fumadores. BP mayor en no fumadores
Goel y cols. (30)	91	349	Observacional prospectivo	CAL y PD con sonda UNC-15	Periodontitis en 85,4% de fumadores vs. 59,6% en no fumadores. Periodontitis severa: 44,6% en fumadores, 15,7% en no fumadores
Mišković y cols. (31)	22	22	Observacional prospectivo	CAL, PD, IG, IP, RC, M, FD con sonda UNC-15	32% fumadores con periodontitis vs. 5% no fumadores. Mayores valores de IG, IP, M, FD en fumadores. PD: 3,42mm vs. 1,88mm; CAL: 3,6mm vs. 2,27mm. Riesgo 4,7 veces mayor por tabaco
Shah y cols. (32)	150	150	Observacional prospectivo	IP, IG y CAL con sonda UNC-15	Valores más altos en fumadores

Aldakheel y cols. (33)	15	15	Observacional prospectivo	PD, CAL, IP, IG, MBL con sonda UNC-15. PCR del biofilm	Más altos niveles de PD, CAL, MBL en fumadores. Mayor presencia de A. actinomycetemcomitans y P. gingivalis.
Ramesh y cols. (34)	15	30	Observacional prospectivo	Análisis inmunohistoquímico de E-cadherina	Menor expresión en fumadores, pero los resultados no son estadísticamente significativos
Hegde y cols. (35)	10	10	Observacional prospectivo	PD, CAL, IG. SCN por espectrofotometría	Fumadores con mayor SCN, PD y CAL
Kanmaz y cols. (36)	23	21	Observacional prospectivo	IP, BOP, PD, CAL con sonda Williams. Cotinina, IL-8, MMP-8 y patógenos periodontales	Fumadores con mayores valores de IL-8, MMP-8, PD, CAL. Mayor número de P. gingivalis, T. forsythia y T. denticola en fumadores
Aziz y cols. (37)	80	51	Observacional prospectivo	CAL, PD, IG, IP con sonda UNC-15. Biomarcadores inflamatorios y estrés oxidativo en sangre	Mejora general, pero respuesta limitada en fumadores

Waseem y cols. (38)	38	62	Observacional prospectivo	PD, IG, BP, AL con sonda CPITN. Prot-CR en sangre	Prot-CR mayor en fumadores. Mejora tras tratamiento en ambos grupos