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CENTRO DENTAL INTEGRAL

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Gastroenterología

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Dr. Francisco Torres Lear

La trayectoria del Dr. Torres Lear es la historia de un descubrimiento vocacional inesperado. Aunque se licenció en Medicina con la firme intención de ser cardiólogo, el destino intervino mientras preparaba el MIR: aprobó el acceso a Odontología y lo que comenzó como un paso intermedio se transformó en su verdadera pasión. En la estomatología descubrió un "trabajo artesano de la salud" que le cautivó por completo, haciéndole comprender que había nacido para esta profesión.

Su enfoque va más allá de lo clínico; su mayor satisfacción reside en mejorar la autoestima, el bienestar y la calidad de vida de sus pacientes. Defensor acérrimo de la prevención y la higiene diaria, el Dr. Torres lidera el Centro Dental Torres bajo una premisa clara: para conseguir la felicidad del paciente, primero hay que cuidar a las personas que trabajan en la clínica, dotándolas de los mejores medios en una organización sólida y humana.

Titulación
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Referencias científicas

Referencias científicas

Aguiar FJN, Menezes FDS, Fagundes MA, Fernandes GA, Alves FA, Filho JG, Curado MP. Gastric adenocarcinoma and periodontal disease: A systematic review and meta-analysis. Clinics (Sao Paulo). 2024 Jan 31;79:100321. doi: 10.1016/j.clinsp.2023.100321. PMID: 38301538; PMCID: PMC10847461.

ABSTRACT

Background: The oral cavity is a link between of external environment with gastrointestinal tract. Studies are controversial on the presence of Periodontal Disease (PD) and its association with Gastric Adenocarcinoma (GAC).

Methods: The authors performed a systematic review and meta-analysis to verify the association between PD and GAC. Six electronic databases were evaluated between 1961 and 2022. Titles and abstracts were reviewed independently according to the eligibility criteria, assessing full texts of selected studies. The quality of the included research was verified using the Newcastle-Ottawa Scale for case-control and cohort studies. Statistical analyses were performed based on fixed and/or random effects models to calculate the summarized Relative Risk (RR) and its 95 % Confidence Interval (95 % CI).

Results: There were 639 studies, of which nine articles were included (3 case-controls and 6 cohorts). Overall, the authors identified 1,253 cases of GAC 2,501 controls in case-control studies, and 1,631 patients with GAC enrolled in cohort studies. Patients presenting PD increased the risk of developing GAC by 17 % (RR=1.17; 95 % CI 1.03–1.32), which remained regardless of the diagnostic method for PD, i.e., clinical examination (RR = 1.19; 95 % CI 1.14–1.24) and self-report (RR = 1.34; 95 % CI 1.06–1.69). Moreover, Asian patients (RR=1.17; 95 % CI 1.00–1.36) with PD had a higher risk of having GAC than American and European patients (RR = 1.18; 95 % CI 0.84–1.66).

Conclusions: The presence of PD the risk of GAC suggesting that its infectious-inflammatory process of PD may be related to GAC development. Further investigations on the oral-gastric microbiota and its role in the carcinogenesis of gastric cancer should be carried out, and the screening of patients with potential risk for GAC should be considered in the clinical practice of dentists.

Aguiar ILS, Santos-Lins LS, Brasil-Oliveira R, Cotrim HP, Lins-Kusterer L. Non-alcoholic fatty liver disease and periodontal disease: A systematic review and meta-analysis of cross-sectional studies. Arab J Gastroenterol. 2023 Nov;24(4):198-203. doi: 10.1016/j.ajg.2023.09.005. Epub 2023 Nov 22. PMID: 37993376.

ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) is a chronic liver disease, comprising hepatic steatosis, and non-alcoholic steatohepatitis. Periodontal disease (PD) may be a risk factor for the evolution of liver cirrhosis. This study aimed to evaluate the association between NAFLD and PD.We searched in Pubmed, Scopus, Cochrane, and Lilacs databases with descriptors (Non-alcoholic Fatty Liver Disease OR (non-alcoholic AND Fatty Liver AND disease) OR Nonalcoholic Steatohepatitis) AND (Periodontal Disease OR Gingivitis OR Periodontitis) from January 2021 to September 2021. We selected, by the abstract, cross-sectional, cohort (prospective and retrospective), and case-control studies that address periodontal disease in patients with Non-alcoholic Fatty Liver Disease, and aged ≥ 18 years. The search was without the restriction of language and publication time.The search resulted in 954 articles. After applying the selection criteria, five cross-sectional studies remained. A metanalysis combined the study estimates of periodontal disease in NAFLD, by using the random effects. The Odds Ratio (1.91; 95% CI 1.21-3.02; P = 0.006) indicates that the chance of presenting Periodontal disease is 91% higher in individuals with NAFLD when compared with individuals without NAFLD. There are few studies with appropriate methodology to produce sound evidence about the causal relationship between the use of NAFLD and PD, however, studies support the association. So, dental staff must be aware of this association for better management of periodontal disease in patients with NAFLD.

Akbari E, Epstein JB, Samim F. Unveiling the Hidden Links: Periodontal Disease, Fusobacterium Nucleatum, and Cancers. Curr Oncol Rep. 2024 Nov;26(11):1388-1397. doi: 10.1007/s11912-024-01591-w. Epub 2024 Aug 12. PMID: 39133417.

ABSTRACT

Purpose of review: Fusobacterium nucleatum (F. nucleatum), an anaerobic, gram-negative microbe, commonly found in human dental biofilm and the gut flora. It has long been known to have a higher concentration in periodontal disease and has recently been implicated in both oral and distant cancers such as colorectal, gastrointestinal, esophageal, breast, pancreatic hepatocellular, and genitourinary cancers. However, the mechanism of its involvement in the development of cancer has not been fully discussed. This review aims to cover biological molecular and clinical aspects of F. nucleatum and cancers.

Recent findings: Studies indicate F. nucleatum promotes tumor development through chronic inflammation, immune evasion, cell proliferation activation, and direct cell interactions, as in oral squamous cell carcinoma (OSCC). In colorectal cancer (CRC), F. nucleatum contributes to tumorigenesis through β-catenin signaling and NF-κB activation. It also induces autophagy, leading to chemoresistance in CRC and esophageal cancers, and enhances tumor growth and metastasis in breast cancer by reducing T-cell infiltration. F. nucleatum is linked to carcinogenesis and increased bacterial diversity in OSCC, with improved oral hygiene potentially preventing OSCC. F. nucleatum triggers cancer by causing mutations and epigenetic changes through cytokines and reactive oxygen species. It also promotes chemoresistance in CRC. F. nucleatum may potentially serve as a diagnostic tool in various cancers, with non-invasive detection methods available. Further investigation is needed to discover its potential in the diagnosis and treatment of OSCC and other cancers.

Ayati A, Khodabandelu S, Khaleghi S, Nourmohammadi A, Jafari F, Ahmadianghalehsorkh M, Vatani Z, Bashiri HS, Ahmadi M, Jafari M, Soltaninejad H, Rahmanian M. A systematic review and network meta-analysis of the association between periodontitis and inflammatory bowel diseases. BMC Oral Health. 2025 Mar 31;25(1):463. doi: 10.1186/s12903-025-05830-9. PMID: 40165211; PMCID: PMC11956190.

ABSTRACT

Objectives: Several earlier studies have shown that IBD (including its two subtypes, ulcerative colitis (UC) and Crohn's disease (CD)) increases the risk of periodontal disease. This study aimed to evaluate the relevance among periodontitis and IBD subcategories.

Methods: This study was conducted based on PRISMA guidelines. The Web of Science, PubMed, Google Scholar, and Scopus databases were searched up to February 2024 using pertinent keywords. Case series, review articles, and animal studies were excluded. The risk of bias in this research was evaluated through the Joanna Briggs Institute (JBI) criteria. The meta-analysis was conducted using R statistical software.

Results: A total of 9134 patients within 13 studies after the screening process were evaluated. Our study has shown that periodontitis is significantly more prevalent among IBD patients (UC and CD). According to prior meta-analyses, PD morbidity was found to be significantly high among CD patients (OR: 4.30; 95% CI: 3.72-4.98; I2 = 0%). Similarly, UC elevated PD risk (OR: 4.55; 95% CI: 3.76-5.50; I2 = 0%). The risk of periodontitis was not significantly different between CD and UC patients (OR: 0.96; 95% CI: 0.65-1.43; I2 = 34%).

Conclusions: UC and CD patients were more likely to develop periodontitis, with low heterogeneity between studies, while the prevalence of periodontitis among UC and CD patients was not meaningfully different.

Clinical relevance: The higher risk of periodontitis in patients with IBD indicates the necessity of screening for periodontitis. Considering the various oral manifestations and poor quality of life associated with IBD, it is important to be aware of the symptoms of periodontitis.

Bai L, Wang YL, Chen YL, Li HX, Zhu SW, Liu Y, Song ZC, Duan SZ. The combination of experimental periodontitis and oral microbiota from periodontitis patients aggravates liver fibrosis in mice. J Clin Periodontol. 2022 Oct;49(10):1067-1078. doi: 10.1111/jcpe.13682. Epub 2022 Jun 28. PMID: 35713233.

ABSTRACT

Aim: Periodontitis (PD) is the sixth most prevalent disease around the world and is involved in the development and progression of multiple systemic diseases. Previous studies have reported that PD may aggravate liver injuries. The objective of this study was to investigate whether and how PD affects liver fibrosis.

Materials and methods: Ligature-induced PD (LIP) was induced in male C57/B6J mice, and sub-gingival plaques (PL) from patients with PD were applied to mouse teeth. Liver fibrosis was induced by carbon tetrachloride (CCl4) injection. The mice were randomly divided into six groups: Oil, Oil+LIP, Oil+LIP+PL, CCl4 , CCl4 +LIP, and CCl4 +LIP+PL. Alveolar bone resorption was evaluated by methylene blue staining. Hepatic function was analysed by serum alanine aminotransferase and hepatic hydroxyproline. Picrosirius red and α-smooth muscle actin (SMA) staining were used to evaluate the fibrotic area. RNA sequencing and quantitative RT-PCR were used to measure gene expression. Western blotting was used to measure protein levels. Flow cytometry was used to analyse the accumulation of immune cells. Mouse microbiota were analysed using 16S rRNA gene sequencing.

Results: Mice in the CCl4 +LIP+PL group displayed higher serum alanine aminotransferase and hepatic hydroxyproline as well as more Picrosirius red-positive and α-SMA-positive areas in liver samples than those of the CCl4 group, suggesting that PD (LIP+PL) aggravated CCl4 -induced hepatic dysfunction and liver fibrosis. Consistently, the expression of fibro-genic genes and the protein levels of transforming growth factor β were much higher in the CCl4 +LIP+PL group than in the CCl4 group. Flow cytometry revealed that PD increased the accumulation of immune cells, including Kupffer cells, B cells, and Th17 cells, in the liver of mice with CCl4 treatment. PD also increased the expression of inflammatory genes and activated pro-inflammatory nuclear factor-kappa B pathway in the livers of CCl4 -injected mice. Moreover, PD altered both oral and liver microbiota in CCl4 -injected mice.

Conclusions: PD aggravates CCl4 -induced hepatic dysfunction and fibrosis in mice, likely through the increase of inflammation and alteration of microbiota in the liver.

Baima G, Ribaldone DG, Romano F, Aimetti M, Romandini M. The Gum-Gut Axis: Periodontitis and the Risk of Gastrointestinal Cancers. Cancers (Basel). 2023 Sep 15;15(18):4594. doi: 10.3390/cancers15184594. PMID: 37760563; PMCID: PMC10526746.

ABSTRACT

Periodontitis has been linked to an increased risk of various chronic non-communicable diseases, including gastrointestinal cancers. Indeed, dysbiosis of the oral microbiome and immune-inflammatory pathways related to periodontitis may impact the pathophysiology of the gastrointestinal tract and its accessory organs through the so-called "gum-gut axis". In addition to the hematogenous spread of periodontal pathogens and inflammatory cytokines, recent research suggests that oral pathobionts may translocate to the gastrointestinal tract through saliva, possibly impacting neoplastic processes in the gastrointestinal, liver, and pancreatic systems. The exact mechanisms by which oral pathogens contribute to the development of digestive tract cancers are not fully understood but may involve dysbiosis of the gut microbiome, chronic inflammation, and immune modulation/evasion, mainly through the interaction with T-helper and monocytic cells. Specifically, keystone periodontal pathogens, including Porphyromonas gingivalis and Fusobacterium nucleatum, are known to interact with the molecular hallmarks of gastrointestinal cancers, inducing genomic mutations, and promote a permissive immune microenvironment by impairing anti-tumor checkpoints. The evidence gathered here suggests a possible role of periodontitis and oral dysbiosis in the carcinogenesis of the enteral tract. The "gum-gut axis" may therefore represent a promising target for the development of strategies for the prevention and treatment of gastrointestinal cancers.

Baima G, Muwalla M, Testa G, Mazza F, Bebars A, Perotto S, Vernero M, Massano A, Romano F, Ribaldone DG, Aimetti M. Periodontitis prevalence and severity in inflammatory bowel disease: A case-control study. J Periodontol. 2023 Mar;94(3):313-322. doi: 10.1002/JPER.22-0322. Epub 2022 Nov 15. PMID: 36111636.

ABSTRACT

Background: Recent evidence is supporting the notion of a microbiological and immunological continuum on the gum-gut axis in health and disease. Therefore, the purpose of this study was to assess the prevalence and risk indicators of periodontitis in patients with Crohn's disease (CD) or ulcerative colitis (UC) compared to age- and sex-matched controls without inflammatory bowel disease (IBD).

Methods: A total of 180 IBD (117 CD, 60 UC, 3 IBD-unclassified) and 180 healthy controls were compared for their periodontitis diagnosis (Centers for Disease Control and Prevention/American Academy of Periodontology [CDC/AAP] case definition) and full-mouth periodontal parameters. In addition, explorative logistic regression models were performed.

Results: Significantly more patients with IBD had moderate/severe periodontitis (85.6% vs. 65.6%, $p < 0.001$) and severe periodontitis (36.7% vs. 25.6%, $p < 0.001$) than controls. Differences were higher in the 35-50 and 51-65 age groups, without significant changes between CD and UC. IBD subjects presented chances ≧3.5 higher of having moderate/severe periodontitis ($p < 0.001$). Significant variables associated with periodontitis in the whole sample were older age, presence of IBD, and higher full-mouth plaque scores, whereas in the IBD group they were male sex, IBD-associated surgery, and IBD duration and localization (pancolitis). Positive risk indicators for IBD were periodontitis severity and higher bleeding scores, while smoking was negatively associated with UC.

Conclusions: Relevant associations between IBD and periodontitis were found, being modified by CD and UC clinical characteristics. Preventive and therapeutic strategies involving the gum-gut axis should be enforced in IBD patients.

Bertl K, Burisch J, Pandis N, Klinge B, Stavropoulos A. Oral health in patients with inflammatory bowel disease: A cross-sectional survey in Sweden. Clin Oral Investig. 2024 Oct 5;28(10):573. doi: 10.1007/s00784-024-05951-5. PMID: 39367966; PMCID: PMC11455683.

ABSTRACT

Objectives: The aim of this cross-sectional survey was to assess oral health, including prevalence of periodontitis and rate of tooth loss, in a Swedish cohort of patients with inflammatory bowel disease (IBD).

Methods: A questionnaire on general anamnestic and socio-economic aspects, IBD diagnosis, and various oral health aspects was distributed online. The analyses focused on the comparison between patients diagnosed with ulcerative colitis (UC) or Crohn's disease (CD) as well as on factors associated with self-reported severe periodontitis and tooth loss.

Results: Analyses were based on answers from 786 patients; 415 with UC, 371 with CD, 74% female. In both disease entities, high prevalence of severe periodontitis (i.e., 38.5%) was reported, and about 19% of the population had less than 20 remaining teeth and 6.5% a poor oral health-related quality of life. CD patients tended to be more severely affected than UC patients ($p > 0.05$ in the adjusted analysis). Almost 90% of CD patients were aware of being entitled to a bi-annual governmental financial support for dental care due to IBD; however, 1 out of 4 UC patients did not. Furthermore, IBD patients largely believe that the interest of their physicians in any oral lesions due to IBD diagnosis is low.

Conclusions: Severe periodontitis and high rate of tooth loss are frequent in Swedish IBD patients.

Clinical relevance: Even though IBD patients receive bi-annually some special financial support for dental care, it seems this is still not sufficient and more preventive measures appear necessary.

Bertl K, Tsakos G, Pandis N, Bogren A, Burisch J, Stavropoulos A. Health-related quality of life aspects of the 'Periodontitis prevalence in ulcerative colitis and Crohn's disease' (PPCC) cohort. J Clin Periodontol. 2023 Dec;50(12):1601-1620. doi: 10.1111/jcpe.13863. Epub 2023 Sep 5. PMID: 37670508.

ABSTRACT

Aim: To assess whether oral health problems affect disease-specific quality of life (QoL) of inflammatory bowel disease (IBD) patients, and vice versa, whether IBD affects oral-health-related QoL.

Materials and methods: Individuals reporting IBD and matched controls were surveyed on general anamnestic information, oral-health-related questions and the Oral Health Impact Profile (OHIP)-5. IBD patients were additionally surveyed on years since diagnosis, disease activity and severity as well as health-related QoL (Short Inflammatory Bowel Disease Questionnaire, sIBDQ). OHIP-5 and sIBDQ were defined as primary outcome parameters, and several predictors and confounders were used in adjusted univariable and multivariable regression analyses.

Results: Answers from 1108 IBD patients and 3429 controls were analysed. Compared with controls, IBD patients reported significantly more frequently an oral impact on daily life and worse oral-health-related QoL, with Crohn's disease (CD) patients being more severely affected than ulcerative colitis (UC) patients. The diagnosis of UC and CD, having <20 teeth, severe periodontitis and stressful daily-life experience were associated with a higher prevalence of poor oral-health-related QoL. Among IBD patients, an impaired IBD-specific, health-related QoL was significantly associated with the diagnosis of CD and depression, IBD activity and severity, having <20 teeth, presence of oral lesions and stressful daily-life experience, while a longer time since diagnosis was significantly associated with an improved IBD-specific, health-related QoL.

Conclusions: The results of the present study indicate, for the first time, that oral health problems are associated with an impairment of IBD-specific health-related QoL, and vice versa, IBD is associated with an impaired oral health-related QoL. This emphasizes the potential advantages of including dental professionals in the multi-disciplinary treatment teams of IBD patients.

Boonyaleka K, Okano T, Iida T, Leewananthawet A, Sasai M, Yamamoto M, Ashida H, Suzuki T. Fusobacterium nucleatum infection activates the noncanonical inflammasome and exacerbates inflammatory response in DSS-induced colitis. Eur J Immunol. 2023 Nov;53(11):e2350455. doi: 10.1002/eji.202350455. Epub 2023 Aug 22. PMID: 37471504.

ABSTRACT

Caspase activation results in pyroptosis, an inflammatory cell death that contributes to several inflammatory diseases by releasing inflammatory cytokines and cellular contents. Fusobacterium nucleatum is a periodontal pathogen frequently detected in human cancer and inflammatory bowel diseases. Studies have reported that F. nucleatum infection leads to NLRP3 activation and pyroptosis, but the precise activation process and disease association remain poorly understood. This study demonstrated that F. nucleatum infection exacerbates acute colitis in mice and activates pyroptosis through caspase-11-mediated gasdermin D cleavage in macrophages. Furthermore, F. nucleatum infection in colitis mice induces the enhancement of IL-1α secretion from the colon, affecting weight loss and severe disease activities. Neutralization of IL-1α protects F. nucleatum infected mice from severe colitis. Therefore, F. nucleatum infection facilitates inflammation in acute colitis with IL-1α from colon tissue by activating noncanonical inflammasome through gasdermin D cleavage.

Cheng Y, Yang R, Jia Y, Zhou Y, Yao Y, Shen C, Li D, Zeng R, Wan Z, Zhao Q, Jiang L, Liao X. The association of chronic pain, painkiller use, and potential mediators with liver fat content. Sci Rep. 2025 Feb 25;15(1):6688. doi: 10.1038/s41598-025-89496-x. PMID: 39994347; PMCID: PMC11850618.

ABSTRACT

Excessive accumulation of liver fat content (LFC) is a pathological manifestation of steatotic liver diseases. This study aims to investigate the relationship between chronic pain and LFC development. In the UK Biobank, chronic pain sites were collected via questionnaire, while LFC was measured by magnetic resonance imaging and quantified by Proton Density Fat Fraction (PDFF). During the median follow-up of 10.5 (4.0-17.8) years, in 39,437 individuals, neck/shoulder, back, stomach/abdominal, knee, and general pain achieved significant arithmetic means difference of 0.02, 0.02, 0.04, 0.02, and 0.15 in PDFF ($P < 0.05$) using multivariable linear regression models. There was a significant dose-effect for number of pain sites and PDFF ($P < 0.001$). Additionally, the link between pain sites and PDFF was much stronger in aspirin users than non-users, while steroids had the reverse effect (P for interaction < 0.05). C-reactive protein, sleep, diet, and depression were proved to mediated 8.41%, 13.3%, 6.6%, and 23.0% of the relationship, respectively. In conclusion, there were quantified differences in the relationship between chronic pain and LFC. For chronic pain patients with potential liver health issues, aspirin may be prioritized as an analgesic option due to its potential protective benefits, whereas steroid medications should be avoided.

Čolak D, Pintar T, Cmok Kučič A, Salobir J, Gašpirc B, Gašperšič R. Periodontal and Hepatic Parameters in Obese Patients Undergoing Bariatric Surgery. Oral Health Prev Dent. 2022 Jul 22;20:295-304. doi: 10.3290/j.ohpd.b3240761. PMID: 35866675; PMCID: PMC11640793.

ABSTRACT

Purpose: Current discoveries imply a connection between periodontitis and metabolic associated fatty liver disease (MAFLD). This study aimed to determine the prevalence of periodontitis and MAFLD in obese patients with BMI >40, employing the most reliable diagnostic methods, namely liver biopsy, and detailed periodontal examination.

Materials and methods: Liver biopsy and periodontal examination were performed in 30 obese patients with BMI BMI >40 undergoing bariatric surgery. Kleiner's classification was used to determine non-alcoholic steatohepatitis (NAS) activity score, non-alcoholic steatohepatitis (NASH) and liver fibrosis. The periodontal condition was classified following the recent AAP/EFP classification. Patients were divided into periodontitis (PG) and non-periodontitis groups (NPG). Data on systemic health parameters were collected from patients' medical records. Descriptive statistics and simple statistical tests were used to determine the differences between the two groups.

Results: The prevalence of NASH in the sample was 43% (13/30), borderline NASH 37% (11/30), while fibrosis stage 1 was most common (72%, [22/30]). Periodontitis prevalence was 67% (20/30), while all non-periodontitis patients (33%; 10/30) exhibited gingivitis. PG and NPG did not differ in NAS or NASH prevalence ($p > 0.05$). However, the periodontitis group showed higher C-reactive protein levels, while NPG showed higher gamma-glutamyl transpeptidase levels ($p < 0.05$).

Conclusion: The study results suggest the considerable prevalence of MAFLD, periodontitis and gingivitis in obese patients with BMI >40 undergoing bariatric surgery. Patients with periodontitis had higher CRP levels, while those with gingivitis presented higher gamma-glutamyl transpeptidase levels.

DeClercq V, Wright RJ, van Limbergen J, Langille MGI. Characterization of the salivary microbiome of adults with inflammatory bowel disease. J Oral Microbiol. 2025 Apr 30;17(1):2499923. doi: 10.1080/20002297.2025.2499923. PMID: 40322049; PMCID: PMC12046613.

ABSTRACT

Background: Perturbations of the gut microbiota in patients with inflammatory bowel disease (IBD) have been extensively characterised, but changes to the oral microbiome remain understudied. This study aimed to evaluate the oral microbiome of adults with IBD and of matched controls.

Methods: Saliva samples and data were obtained from a Canadian population cohort (n = 320). The salivary microbiome was characterised using 16S rRNA gene sequencing and examined for differences between control participants and those with IBD, as well as disease subcategories (Crohn's Disease and Ulcerative Colitis).

Results: Alpha diversity was significantly lower in participants with IBD than controls in unadjusted models and many remained significant after adjusting for covariates. Significant differences in some beta diversity metrics between participants with IBD and controls were found, although these did not remain significant when adjusted for covariates. Ten genera were significantly differentially abundant between cases and controls. Veillonella and Streptococcus were both increased in abundance in IBD cases vs controls (25% vs 22% and 14% vs 12%, respectively).

Conclusion: These results showcase changes in oral microbial diversity and composition in those living with IBD and highlight the potential of using the salivary microbiome as a biomarker for screening or monitoring IBD.

Elghannam MT, Hassanien MH, Ameen YA, Turky EA, Elattar GM, ElRay AA, Eltalkawy MD. Oral microbiota and liver diseases. Clin Nutr ESPEN. 2023 Apr;54:68-72. doi: 10.1016/j.clnesp.2022.12.030. Epub 2023 Jan 5. PMID: 36963900.

ABSTRACT

Gut microbiota plays a crucial role in our health and particularly liver diseases, including NAFLD, cirrhosis, and HCC. Oral microbiome and its role in health and disease represent an active field of research. Several lines of evidence have suggested that oral microbiota dysbiosis represents a major factor contributing to the occurrence and progression of many liver diseases. The human microbiome is valuable to the diagnosis of cancer and provides a novel strategy for targeted therapy of HCC. The most studied liver disease in relation to oral-gut-liver axis dysbiosis includes MAFLD; however, other diseases include Precancerous liver disease as viral liver diseases, liver cirrhosis, AIH and liver carcinoma (HCC). It seems that restoring populations of beneficial organisms and correcting dysbiosis appears to improve outcomes in liver disorders. We discuss the possible role of oral microbiota in these diseases.

Feng Z, Chen Z, Wang X, Zhou M, Liu S. Immune-Mediated Bidirectional Causality Between Inflammatory Bowel Disease and Chronic Periodontitis: Evidence from Mendelian Randomization and Integrative Bioinformatics Analysis. Biomedicines. 2025 Feb 15;13(2):476. doi: 10.3390/biomedicines13020476. PMID: 40002889; PMCID: PMC11853167.

ABSTRACT

Background/Objectives: A bidirectional association between inflammatory bowel disease (IBD) and periodontitis has been observed, yet their causal relationship remains unclear. This study aimed to investigate the potential causal links between these two inflammatory conditions through comprehensive genetic and molecular analyses. **Methods**: We conducted a bidirectional Mendelian randomization (MR) analysis integrated with bioinformatics approaches. The causal relationships were primarily evaluated using inverse variance weighting (IVW), complemented by multiple sensitivity analyses to assess the robustness of the findings. Additionally, we performed differential gene expression analysis using RNA sequencing data to identify co-expressed genes and shared inflammatory mediators between IBD and periodontitis, followed by pathway enrichment analysis. **Results**: Bidirectional MR analysis revealed significant causal associations between IBD and periodontitis (p-value < 0.05). Sensitivity analyses demonstrated the consistency of these findings, with no evidence of significant heterogeneity or horizontal pleiotropy (p-value > 0.05). Integrated bioinformatics analysis identified key immune regulators, particularly interleukin 1 beta (IL1B) and C-X-C motif chemokine receptor 4 (CXCR4), and inflammatory signaling pathways, including tumor necrosis factor (TNF-α) and interleukin 17 (IL17), as potential molecular mechanisms underlying the bidirectional relationship between these conditions. **Conclusions**: Our findings provide genetic evidence supporting a bidirectional causal relationship between IBD and periodontitis. Transcriptomic analysis revealed shared pathological mechanisms and identified crucial immune regulatory factors common to both diseases. These insights enhance our understanding of the molecular interplay between IBD and periodontitis, potentially informing new therapeutic strategies for both conditions.

Ge J, Li M, Yao J, Guo J, Li X, Li G, Han X, Li Z, Liu M, Zhao J. The potential of EGCG in modulating the oral-gut axis microbiota for treating inflammatory bowel disease. Phytomedicine. 2024 Jul 25;130:155643. doi: 10.1016/j.phymed.2024.155643. Epub 2024 Apr 14. PMID: 38820660.

ABSTRACT

Inflammatory bowel disease (IBD) is a recurrent chronic intestinal disorder that includes ulcerative colitis (UC) and Crohn's disease (CD). Its pathogenesis involves intricate interactions between pathogenic microorganisms, native intestinal microorganisms, and the intestinal immune system via the oral-gut axis. The strong correlation observed between oral diseases and IBD indicates the potential involvement of oral pathogenic microorganisms in IBD development. Consequently, therapeutic strategies targeting the proliferation, translocation, intestinal colonization and exacerbated intestinal inflammation of oral microorganisms within the oral-gut axis may partially alleviate IBD. Tea consumption has been identified as a contributing factor in reducing IBD, with epigallocatechin gallate (EGCG) being the primary bioactive compound used for IBD treatment. However, the precise mechanism by which EGCG mediates microbial crosstalk within the oral-gut axis remains unclear. In this review, we provide a comprehensive overview of the diverse oral microorganisms implicated in the pathogenesis of IBD and elucidate their colonization pathways and mechanisms. Subsequently, we investigated the antibacterial properties of EGCG and its potential to attenuate microbial translocation and colonization in the gut, emphasizing its role in attenuating exacerbations of IBD. We also elucidated the toxic and side effects of EGCG. Finally, we discuss current strategies for enhancing EGCG bioavailability and propose novel multi-targeted nano-delivery systems for the more efficacious management of IBD. This review elucidates the role and feasibility of EGCG-mediated modulation of the oral-gut axis microbiota in the management of IBD, contributing to a better understanding of the mechanism of action of EGCG in the treatment of IBD and the development of prospective treatment strategies.

Guadalupi G, Contini C, Iavarone F, Castagnola M, Messana I, Faa G, Onali S, Chessa L, Vitorino R, Amado F, Diaz G, Manconi B, Cabras T, Olianas A. Combined Salivary Proteome Profiling and Machine Learning Analysis Provides Insight into Molecular Signature for Autoimmune Liver Diseases Classification. Int J Mol Sci. 2023 Jul 30;24(15):12207. doi: 10.3390/ijms241512207. PMID: 37569584; PMCID: PMC10418803.

ABSTRACT

Autoimmune hepatitis (AIH) and primary biliary cholangitis (PBC) are autoimmune liver diseases that target the liver and have a wide spectrum of presentation. A global overview of quantitative variations on the salivary proteome in presence of these two pathologies is investigated in this study. The acid-insolu-

ble salivary fraction of AIH and PBC patients, and healthy controls (HCs), was analyzed using a gel-based bottom-up proteomic approach combined with a robust machine learning statistical analysis of the dataset. The abundance of Arginase, Junction plakoglobin, Desmoplakin, Hexokinase-3 and Desmocollin-1 decreased, while that of BPI fold-containing family A member 2 increased in AIHp compared to HCs; the abundance of Gelsolin, CD14, Tumor-associated calcium signal transducer 2, Clusterin, Heterogeneous nuclear ribonucleoproteins A2/B1, Cofilin-1 and BPI fold-containing family B member 2 increased in PBCp compared to HCs. The abundance of Hornerin decreased in both AIHp and PBCp with respect to HCs and provided an area under the ROC curve of 0.939. Machine learning analysis confirmed the feasibility of the salivary proteome to discriminate groups of subjects based on AIH or PBC occurrence as previously suggested by our group. The topology-based functional enrichment analysis performed on these potential salivary biomarkers highlights an enrichment of terms mostly related to the immune system, but also with a strong involvement in liver fibrosis process and with antimicrobial activity.

Haznedaroglu E, Polat E. Dental Caries, Dental Erosion and Periodontal Disease in Children with Inflammatory Bowel Disease. Int J Med Sci. 2023 Apr 9;20(5):682-688. doi: 10.7150/ijms.83075. PMID: 37082734; PMCID: PMC10110475.

ABSTRACT

Background: There is reportedly a higher prevalence of dental caries and periodontal disease in adults with inflammatory bowel disease (IBD) than in healthy adults. Similar data for children are lacking in the literature. We aimed to evaluate the prevalence of dental erosion, dental caries, and periodontal disease in children with IBD. **Methods:** This was a cross-sectional comparative study. Using the established criteria of the World Health Organization, oral investigations and detailed questionnaires that covered nutritional habits were completed by the same pediatric dentist for 32 patients with IBD, aged 11 to 18 years (15.53 ± 2.00), and 32 healthy controls. **Results:** The decayed, missing, and filled tooth index showed no significant difference between the groups (p = 0.072). The frequency of consumption of salad, lemon gum, candy and sweetened milk was significantly higher in the control group (p = 0.041, 0.012, 0.001, and 0.001, respectively) than in the IBD group. No dental erosion was observed in the IBD group. Oral mucosal history determined that 20/32 patients with IBD (62.5%) had at least one oral extraintestinal manifestation. Despite no significant differences in plaque scores between the two groups, the gingival evaluation showed a much higher mean value of gingival index scores in the IBD group than in the control group (p = 0.003). **Conclusion:** Although the number of patients included in the study is small, we can conclude that oral extraintestinal manifestations and periodontal disease are more prevalent in paediatric patients with IBD than in healthy populations.

Hudson D, Ayares G, Taboun Z, Malhi G, Idalsoaga F, Mortuza R, Souyet M, Ramirez-Cadiz C, Díaz LA, Arrese M, Arab JP. Periodontal disease and cirrhosis: current concepts and future prospects. eGastroenterology. 2025 Feb 25;3(1):e100140. doi: 10.1136/egastro-2024-100140. PMID: 40160254; PMCID: PMC11950965.

ABSTRACT

Periodontal diseases are prevalent among the general population and are associated with several systemic conditions, such as chronic kidney disease and type 2 diabetes mellitus. Chronic liver disease and cirrhosis have also been linked with periodontal disease, an association with complex underlying mechanisms, and with potential prognostic implications. Multiple factors can explain this relevant association, including nutritional factors, alcohol consumption, disruption of the oral-gut-liver axis and associated dysbiosis. Additionally, patients with liver disease have been observed to exhibit poorer oral hygiene practices compared with the general population, potentially predisposing them to the development of periodontal disease. Therefore, it is recommended that all patients with liver disease undergo screening and subsequent treatment for periodontal disease. Treatment of periodontal disease in patients with cirrhosis may help reduce liver-derived inflammatory damage, with recent research indicating a potential benefit in terms of reduced mortality. However, further studies on periodontal disease treatment in patients with liver disease are still warranted to determine optimal management strategies. This narrative review describes current concepts on the association between periodontal disease and chronic liver disease.

Juzbašić M, Tomas M, Petrović A, Hefer M, Sikora R, Mačković A, Siber S, Smolić M. Interaction Between Periodontitis and MASLD: Pathophysiological Associations and Possibilities of Prevention and Therapy. Biomedicines. 2025 May 30;13(6):1346. doi: 10.3390/biomedicines13061346. PMID: 40564062; PMCID: PMC12190076.

ABSTRACT

The interrelationship between periodontitis and metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), has attracted increasing attention due to the significant global rise in the prevalence of both conditions. Periodontitis, a chronic inflammatory disease, affects a substantial portion of the population and parallels the growing incidence of MASLD, which currently impacts nearly 30% of the global population. The updated nomenclature reflects a deeper understanding of the condition's metabolic origins. This narrative review focuses on the shared pathophysiological mechanisms, particularly systemic inflammation, insulin resistance, and oxidative stress that may underlie the bidirectional relationship between these diseases. These mechanisms often act in concert to promote disease development. Unlike previous literature, this review emphasizes the hypothesis that chronic periodontal inflammation may not only mirror but also contribute to the systemic metabolic dysregulation observed in MASLD. We critically assess current evidence supporting this link by highlighting the role of inflammatory mediators in bridging oral and hepatic health, and by proposing an integrated, multidisciplinary approach to its early detection and management. The aim is to offer novel insights that can help develop better prevention strategies and more effective treatments for both diseases.

Kobayashi T, Iwaki M, Nogami A, Honda Y, Ogawa Y, Imajo K, Saito S, Nakajima A, Yoneda M. Involvement of Periodontal Disease in the Pathogenesis and Exacerbation of Nonalcoholic Fatty Liver Disease/Nonalcoholic Steatohepatitis: A Review. Nutrients. 2023 Mar 3;15(5):1269. doi: 10.3390/nu15051269. PMID: 36904268; PMCID: PMC10004797.

ABSTRACT

The increasing incidence of nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH), along with global lifestyle changes, requires further in-depth research to elucidate the mechanisms and develop new treatment strategies. In addition, the number of patients with periodontal disease has increased recently, suggesting that periodontal disease is sometimes associated with systemic conditions. In this review, we summarize recent studies linking periodontal disease and NAFLD, the concept of the mouth-gut-liver axis, oral and intestinal microbiota, and liver disease. We suggest new research directions toward a detailed mechanistic understanding and novel targets for treatment and prevention. Forty years

have passed since the concepts of NAFLD and NASH were first proposed. however, no effective prevention or treatment has been established. We also found that the pathogenesis of NAFLD/NASH is not limited to liver-related diseases but has been reported to be associated with various systemic diseases and an increasing number of causes of death. In addition, changes in the intestinal microbiota have been shown to be a risk factor for periodontal diseases, such as atherosclerosis, diabetes, rheumatoid arthritis, nonalcoholic fatty liver disease, and obesity.

Kucharski R, Sobocki BK, Stachowska E, Bulman N, Kalinowski L, Kaźmierczak-Siedlecka K. Dental problems and oral microbiome alterations in ulcerative colitis. Front Immunol. 2025 Feb 5;16:1502605. doi: 10.3389/fimmu.2025.1502605. PMID: 39975550; PMCID: PMC11836005.

ABSTRACT

Ulcerative colitis is a chronic disease that has not well-established etiology. The role of microbial dysregulation in its pathogenesis has been recently highlighted. Overall, microbiome alterations concern the reduction of bacterial abundance and diversity, resulting in gut microbiome imbalance negatively affecting immunological aspects. There is a link between ulcerative colitis and the oral microbiome. The changes of oral microbiome are found at many levels, from gently dysbiotic composition to the presence of the main periodontal microbes. The analysis of oral microbiome can be a part of personalized medicine due to the fact that it is a potential biomarker. Patients with ulcerative colitis may manifest dental symptoms/problems, such as periodontitis (strongly related to the red-complex pathogens-Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola, and bacteria belonging to the other complexes, such as Fusobacterium nucleatum and Aggregatibacter actinomycetecomitans), dental caries, oral ulcerations, leukoplakia, halitosis, and others. Notably, the DMFT (Decayed, Missing, Filled Teeth) index is higher in these patients compared to healthy subjects. According to some data, oral lichen planus (which is a disease with an immunological background) can also be observed in ulcerative colitis patients. It seems that deep understanding of ulcerative colitis in association with oral microbiome, immunology, and dental manifestations may be crucial to provide complex treatment from a dental point of view.

Kuraji R, Sekino S, Kapila Y, Numabe Y. Periodontal disease-related nonalcoholic fatty liver disease and nonalcoholic steatohepatitis: An emerging concept of oral-liver axis. Periodontol 2000. 2021 Oct;87(1):204-240. doi: 10.1111/prd.12387. PMID: 34463983; PMCID: PMC8456799.

ABSTRACT

Periodontal disease, a chronic inflammatory disease of the periodontal tissues, is not only a major cause of tooth loss, but it is also known to exacerbate/be associated with various metabolic disorders, such as obesity, diabetes, dyslipidemia, and cardiovascular disease. Recently, growing evidence has suggested that periodontal disease has adverse effects on the pathophysiology of liver disease. In particular, nonalcoholic fatty liver disease, a hepatic manifestation of metabolic syndrome, has been associated with periodontal disease. Nonalcoholic fatty liver disease is characterized by hepatic fat deposition in the absence of a habitual drinking history, viral infections, or autoimmune diseases. A subset of nonalcoholic fatty liver diseases can develop into more severe and progressive forms, namely nonalcoholic steatohepatitis. The latter can lead to cirrhosis and hepatocellular carcinoma, which are end-stage liver diseases. Extensive research has provided

plausible mechanisms to explain how periodontal disease can negatively affect nonalcoholic fatty liver disease and nonalcoholic steatohepatitis, namely via hematogenous or enteral routes. During periodontitis, the liver is under constant exposure to various pathogenic factors that diffuse systemically from the oral cavity, such as bacteria and their by-products, inflammatory cytokines, and reactive oxygen species, and these can be involved in disease promotion of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. Also, gut microbiome dysbiosis induced by enteral translocation of periodontopathic bacteria may impair gut wall barrier function and promote the transfer of hepatotoxins and enterobacteria to the liver through the enterohepatic circulation. Moreover, in a population with metabolic syndrome, the interaction between periodontitis and systemic conditions related to insulin resistance further strengthens the association with nonalcoholic fatty liver disease. However, most of the pathologic links between periodontitis and nonalcoholic fatty liver disease in humans are provided by epidemiologic observational studies, with the causal relationship not yet being established. Several systematic and meta-analysis studies also show conflicting results. In addition, the effect of periodontal treatment on nonalcoholic fatty liver disease has hardly been studied. Despite these limitations, the global burden of periodontal disease combined with the recent nonalcoholic fatty liver disease epidemic has important clinical and public health implications. Emerging evidence suggests an association between periodontal disease and liver diseases, and thus we propose the term periodontal disease-related nonalcoholic fatty liver disease or periodontal disease-related nonalcoholic steatohepatitis. Continued efforts in this area will pave the way for new diagnostic and therapeutic approaches based on a periodontologic viewpoint to address this life-threatening liver disease.

Kuraji R, Shiba T, Dong TS, Numabe Y, Kapila YL. Periodontal treatment and microbiome-targeted therapy in management of periodontitis-related nonalcoholic fatty liver disease with oral and gut dysbiosis. World J Gastroenterol. 2023 Feb 14;29(6):967-996. doi: 10.3748/wjg.v29.i6.967. PMID: 36844143; PMCID: PMC9950865.

ABSTRACT

A growing body of evidence from multiple areas proposes that periodontal disease, accompanied by oral inflammation and pathological changes in the microbiome, induces gut dysbiosis and is involved in the pathogenesis of nonalcoholic fatty liver disease (NAFLD). A subgroup of NAFLD patients have a severely progressive form, namely nonalcoholic steatohepatitis (NASH), which is characterized by histological findings that include inflammatory cell infiltration and fibrosis. NASH has a high risk of further progression to cirrhosis and hepatocellular carcinoma. The oral microbiota may serve as an endogenous reservoir for gut microbiota, and transport of oral bacteria through the gastro-intestinal tract can set up a gut microbiome dysbiosis. Gut dysbiosis increases the production of potential hepatotoxins, including lipopolysaccharide, ethanol, and other volatile organic compounds such as acetone, phenol and cyclopentane. Moreover, gut dysbiosis increases intestinal permeability by disrupting tight junctions in the intestinal wall, leading to enhanced translocation of these hepatotoxins and enteric bacteria into the liver through the portal circulation. In particular, many animal studies support that oral administration of Porphyromonas gingivalis, a typical periodontopathic bacterium, induces disturbances in glycolipid metabolism and inflammation in the liver with gut dysbiosis. NAFLD, also known as the hepatic phenotype of metabolic syndrome, is strongly associated with metabolic complications, such as obesity and diabetes. Periodontal disease also has a bidirectional relationship with metabolic syndrome, and both diseases may induce oral and gut microbiome dysbiosis with insulin resistance and systemic chronic inflammation cooperatively. In this review, we will describe the link between periodontal disease and NAFLD with a focus on basic, epidemiological, and clinical studies, and discuss potential mechanisms linking the two diseases and possible therapeutic approaches focused on the microbiome. In conclusion, it is presu-

med that the pathogenesis of NAFLD involves a complex crosstalk between periodontal disease, gut microbiota, and metabolic syndrome. Thus, the conventional periodontal treatment and novel microbiome-targeted therapies that include probiotics, prebiotics and bacteriocins would hold great promise for preventing the onset and progression of NAFLD and subsequent complications in patients with periodontal disease.

La Rosa GRM, Lorenzo-Pouso AI, Caponio VCA, Puci MV. Apical periodontitis in inflammatory bowel disease: a meta-analysis at patient and tooth level. Front Dent Med. 2025 Feb 10;6:1553914. doi: 10.3389/fdmed.2025.1553914. PMID: 40008255; PMCID: PMC11847799.

ABSTRACT

Apical periodontitis (AP) is the local inflammation of periapical tissues originating from the dental pulp disease. Cumulative evidence suggests a link between oral and gastro-intestinal systems in both health and disease. In this context, the relationship between AP and inflammatory bowel diseases (IBDs) has not yet been elucidated. The aims of this systematic review and meta-analysis were to describe the prevalence of AP in patients with IBDs and evaluate the potential association between AP and IBDs. Electronic (Embase, PubMed, Scopus, Web of Science) and manual literature searches were conducted from inception to 31 October, 2023 (updated in August, 2024). Strict inclusion criteria were applied to identify observational and experimental clinical studies on AP in IBDs patients. The bias risk was assessed using the Joanna Briggs Institute critical appraisal tools and a biases' report selected from the Oxford Centre for Evidence Based Medicine Catalogue of Bias. A meta-analysis was performed to determine the pooled prevalence and risk of AP at individual and tooth level and the quality of evidence was assessed by the Grading of Recommendations, Assessment, Development and Evaluations (GRADE) approach. The search strategy identified 82 articles with 5 studies included (657 subjects, 7,142 teeth). The overall proportion of AP was 58% at patient level (95% CI = 37%-78%, I 2 = 95.3%) and 7% at tooth level (95% CI = 2%-15%; I 2 = 99.2%). AP was prevalent in IBDs subjects than in healthy controls, both at patient and tooth level. The pooled OR was 1.57 (95% CI = 1.04-2.35; P = 0.038; I 2 = 20%) at patient level, and 1.91 (95% CI = 1.16-3.15; P = 0.011; I 2 = 82%) at tooth level. A potential association between AP and IBDs is plausible, although the quality evidence was low to very low. Longitudinal and experimental studies should be conducted to better understand the relationship between these two conditions and explore any potential causative factors.

Li D, Li Z, Wang L, Zhang Y, Ning S. Oral inoculation of *Fusobacterium nucleatum* exacerbates ulcerative colitis via the secretion of virulence adhesin FadA. Virulence. 2024 Dec;15(1):2399217. doi: 10.1080/21505594.2024.2399217. Epub 2024 Sep 9. PMID: 39221673; PMCID: PMC11385161.

ABSTRACT

Fusobacterium nucleatum (F. nucleatum), an anaerobic resident of the oral cavity, is increasingly recognized as a contributing factor to ulcerative colitis (UC). The adhesive properties of F. nucleatum are mediated by its key virulence protein, FadA adhesin. However, further investigations are needed to understand the pathogenic mechanisms of this oral pathogen in UC. The present study aimed to explore the role of the FadA adhesin in the colonization and invasion of oral F. nucleatum in dextran sulphate sodium (DSS)-induced colitis mice via molecular techniques. In this study, we found that oral inoculation of F. nucleatum strain carrying the FadA adhesin further exacerbated DSS-induced colitis, leading to elevated alveolar bone loss, disease severity, and mortality. Additionally, CDH1 gene knockout mice treated with DSS presented increases in body weight and alveolar bone density, as well as a reduction in disease severity. Furthermore, FadA adhesin adhered to its mucosal receptor E-cadherin, leading to the phosphorylation of β-catenin and the degradation of IκBα, the activation of the NF-κB signalling pathway and the upregulation of downstream cytokines. In conclusion, this research revealed that oral inoculation with F. nucleatum facilitates experimental colitis via the secretion of the virulence adhesin FadA. Targeting the oral pathogen F. nucleatum and its virulence factor FadA may represent a promising therapeutic approach for a portion of UC patients.

Liu B, Jia Y, Gu Z, Li Y, Zhou Y, Cao Y. Periodontal diseases, potential mediators and development of liver fat content: a community-based large cohort. Front Med (Lausanne). 2025 Apr 9;12:1563459. doi: 10.3389/fmed.2025.1563459. PMID: 40270511; PMCID: PMC12014605.

ABSTRACT

Background: A high level of liver fat content (LFC) is a key indicator of steatotic liver disease (SLD), reflecting its pathological essence. Periodontal disease (PD) recognized as a chronic inflammatory condition and cause a widespread adverse health impact. This study aims to investigate the relationship between PD and LFC development.

Methods: In the UK Biobank, PD were gathered through a digital questionnaire, including gum pain, gum bleed, or teeth loose. LFC was measured by Fatty Liver Index (>60 indicates SLD) in cross-sectional analysis and by magnetic resonance imaging (quantified by Proton Density Fat Fraction, PDFF) in longitudinal analysis. Multivariable logistic and linear regression models were conducted to investigate the association of PD and LFC.

Results: In cross-sectional analysis, 164,150 (37.4%) individuals were diagnosed with SLD, and PD showed a significant association with SLD (odds ratio: 1.104, 95% CI: 1.075-1.132). In prospective analysis, a total of 39,656 participants with a median follow-up of 10.3 years were included. PD showed an arithmetic mean difference of 0.091 in PDFF (95% CI: 0.047-0.139), with males exhibiting a stronger association than females (P for interaction <0.05). Significant mediating effects were observed for body mass index (19.58%), C-reactive protein (11.61%), blood glucose (6.70%), and healthy diet score (5.99%) between PD and PDFF (P for all <0.001).

Conclusion: There was a pronounced correlation between PD and LFC, with males predominantly driving this link. This correlation may be partially mediated by body fat, inflammation, dietary habit, and insulin resistance.

Lv C, Shi K, Guo Y, Guo Z, Luo P, Wang L, Wu Z, Yu P. Emerging Roles of Periodontal Pathogen-Derived Outer Membrane Vesicles in NAFLD. Int Dent J. 2025 Aug;75(4):100825. doi: 10.1016/j.identj.2025.03.029. Epub 2025 May 15. PMID: 40378508; PMCID: PMC12145673.

ABSTRACT

The rising incidence of nonalcoholic fatty liver disease (NAFLD) poses a great socioeconomic burden worldwide. Also, periodontitis is the most common chronic inflammatory disease caused by a group of oral pathogens, affecting both oral health and systemic conditions, especially liver disease. Although accumulating evidence has elucidated an association between periodontal pathogens and NAFLD, the role of periodontal pathogen-derived outer membrane vesicles (OMVs) has not yet been clarified. In this comprehensive review, we aim to address this gap by summarising the progression and pathogenesis of NAFLD and revealing the relationship between periodontal disease and NAFLD multidimensionally. Additionally, this review sheds light on the multifunctional roles of periodontal pathogens OMVs and emphasises that periodontal pathogen-derived OMVs promote the development of NAFLD by stimulating Kupffer cells to produce inflammatory factors and inducing the activation of Hepatic stellate cells. However, it is still controversial whether periodontal pathogen-derived OMVs can be transferred to the liver through the bloodstream route or the oral-gut-liver axis. This highlights the pressing need for continued research efforts to develop new and optimised research schemes to observe the formation of the systemic distribution pathway of periodontal pathogen-derived OMVs. Finally, it is notable that there are currently no relevant clinical treatment guidelines to make specific provisions on controlling the level of periodontal pathogen-derived OMVs in patients with NAFLD. Guidelines developed based on our findings may contribute to the standardisation of practices. It can also provide effective strategies and potential therapeutic targets for NAFLD patients with periodontitis to alleviate the development of NAFLD diseases by inhibiting periodontal pathogens OMVs.

Mei EH, Yao C, Chen YN, Nan SX, Qi SC. Multifunctional role of oral bacteria in the progression of non-alcoholic fatty liver disease. World J Hepatol. 2024 May 27;16(5):688-702. doi: 10.4254/wjh.v16.i5.688. PMID: 38818294; PMCID: PMC11135273.

ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) encompasses a spectrum of liver disorders of varying severity, ultimately leading to fibrosis. This spectrum primarily consists of NAFL and non-alcoholic steatohepatitis. The pathogenesis of NAFLD is closely associated with disturbances in the gut microbiota and impairment of the intestinal barrier. Non-gut commensal flora, particularly bacteria, play a pivotal role in the progression of NAFLD. Notably, Porphyromonas gingivalis, a principal bacterium involved in periodontitis, is known to facilitate lipid accumulation, augment immune responses, and induce insulin resistance, thereby exacerbating fibrosis in cases of periodontitis-associated NAFLD. The influence of oral microbiota on NAFLD via the “oral-gut-liver” axis is gaining recognition, offering a novel perspective for NAFLD management through microbial imbalance correction. This review endeavors to encapsulate the intricate roles of oral bacteria in NAFLD and explore underlying mechanisms, emphasizing microbial control strategies as a viable therapeutic avenue for NAFLD.

Luo T, Li J, Pu K, Yang G. Association between periodontitis and gastrointestinal cancer risk and prognosis: evidence from a nested case-control study in Southwest China. Eur J Med Res. 2025 Apr 2;30(1):225. doi: 10.1186/s40001-025-02508-4. PMID: 40176150; PMCID: PMC11963365.

ABSTRACT

Background: With low early detection rates and high incidence and mortality, Gastrointestinal cancer (GIC) imposes a significant global health burden. Emerging evidence indicates that periodontitis may be a potential risk factor for GIC development; however, epidemiological data remains inconclusive.

Objective: This study aimed to examine the impact of periodontitis on the incidence, recurrence, and metastasis of GIC in Southwest China, thereby offering epidemiological evidence to support GIC prevention and management.

Methods: Between September 2022 and August 2024, a case-control study was conducted at the Affiliated Hospital of North Sichuan Medical College. Five hundred GIC patients were included as the case group based on the predefined inclusion and exclusion criteria, while 1005 healthy individuals were recruited for the control group. Multivariate analyses were performed to examine the associations between periodontitis and GIC incidence, recurrence, and metastasis while controlling for potential confounding factors.

Results: The results of this study demonstrated that periodontitis was significantly associated with the incidence of esophageal, gastric, and colorectal cancer. Even after adjusting for potential confounders, it remained a significant risk factor for esophageal cancer (OR = 2.810, 95% CI 1.032-7.649, P = 0.043), colon cancer (OR = 2.330, 95% CI 1.072-5.067, P = 0.033), and rectal cancer (OR = 2.730, 95% CI 1.247-5.379, P = 0.012). Compared to non-periodontitis subjects, periodontitis showed a significant association with distant metastasis of rectal cancer (aHR = 5.332, 95% CI 1.406-20.220, P = 0.014). Moreover, severe periodontitis was identified as an risk factor for distant metastasis in rectal cancer (aHR = 10.138, 95% CI 1.824-56.354, P = 0.008).

Conclusion: This study highlights significant associations between periodontitis and an increased risk of esophageal and colorectal cancers. Additionally, patients with rectal cancer and periodontitis exhibited an increased risk of distant metastasis compared to those without periodontitis.

Mukherjee S, Chopra A, Karmakar S, Bhat SG. Periodontitis increases the risk of gastrointestinal dysfunction: an update on the plausible pathogenic molecular mechanisms. Crit Rev Microbiol. 2025 Feb;51(1):187-217. doi: 10.1080/1040841X.2024.2339260. Epub 2024 Apr 11. PMID: 38602474.

ABSTRACT

Periodontitis is an immuno-inflammatory disease of the soft tissues surrounding the teeth. Periodontitis is linked to many communicable and non-communicable diseases such as diabetes, cardiovascular disease, rheumatoid arthritis, and cancers. The oral-systemic link between periodontal disease and systemic diseases is attributed to the spread of inflammation, microbial products and microbes to distant organ systems. Oral bacteria reach the gut via swallowed saliva, whereby they induce gut dysbiosis and gastrointestinal

dysfunctions. Some periodontal pathogens like Porphyromonas. gingivalis, Klebsiella, Helicobacter. Pylori, Streptococcus, Veillonella, Parvimonas micra, Fusobacterium nucleatum, Peptostreptococcus, Haemophilus, Aggregatibacter actinomycetomcommitans and Streptococcus mutans can withstand the unfavorable acidic, survive in the gut and result in gut dysbiosis. Gut dysbiosis increases gut inflammation, and induce dysplastic changes that lead to gut dysfunction. Various studies have linked oral bacteria, and oral-gut axis to various GIT disorders like inflammatory bowel disease, liver diseases, hepatocellular and pancreatic ductal carcinoma, ulcerative colitis, and Crohn's disease. Although the correlation between periodontitis and GIT disorders is well established, the intricate molecular mechanisms by which oral microflora induce these changes have not been discussed extensively. This review comprehensively discusses the intricate and unique molecular and immunological mechanisms by which periodontal pathogens can induce gut dysbiosis and dysfunction.

Park Y MD, Park JH DDS, Leem GH, Song TJ MD. Periodontitis and the Incidence of Inflammatory Bowel Diseases: A Nationwide Population-Based Cohort Study. Am J Gastroenterol. 2025 Jan 17. doi: 10.14309/ajg.0000000000003326. Epub ahead of print. PMID: 39819636.

ABSTRACT

Introduction: Periodontitis and other oral health indicators are reportedly related to systemic inflammation. Our study aimed to investigate a possible association of oral health status (periodontitis and number of missing teeth) and oral hygiene behaviors (frequency of tooth brushing, dental visit, and dental scaling) with the risk of inflammatory bowel disease (IBD) incidence.

Methods: Using the Korean National Health Insurance Database, we conducted a nationwide, population-based cohort study involving participants from the 2003 national health screening program. We followed these individuals until December 2020 or the occurrence of censoring events. Crohn's disease (CD) and ulcerative colitis (UC) were defined by disease classification codes from the International Classification of Diseases-10 and national codes for rare intractable disease. The occurrence of IBD was analyzed using the Cox proportional hazard model, with propensity score matching performed to balance baseline characteristics between participants with and without periodontitis.

Results: Our analysis involved a total of 2,272,012 participants with a median follow-up period of 17.0 years. The incidence rates for CD and UC were 11.6 and 32.4 per 100,000 person-years, respectively. In multivariable analysis after propensity score matching, the presence of periodontitis was associated with an increased risk of both CD (adjusted hazard ratio [aHR] 1.32, 95% confidence interval [CI] 1.14-1.52, P < 0.001) and UC (aHR 1.21, 95% CI 1.10-1.32, P < 0.001). Conversely, frequent tooth brushing ≥3 times a day was associated with a reduced risk of CD (aHR 0.81, 95% CI 0.65-0.99, P = 0.049), but no significant association was observed with UC.

Discussion: Periodontitis may augment the risk of incidence for CD and UC. This association underscores the potential significance of periodontal health in the context of IBD, emphasizing the importance of comprehensive oral hygiene practices and potential preventive strategies to reduce the risk of CD and UC incidence.

Petkevicius V, Lehr K, Kupcinskas J, Link A. *Fusobacterium nucleatum*: Unraveling its potential role in gastric carcinogenesis. World J Gastroenterol. 2024 Sep 21;30(35):3972-3984. doi: 10.3748/wjg.v30.i35.3972. PMID: 39351058; PMCID: PMC11438658.

ABSTRACT

Fusobacterium nucleatum (F. nucleatum) is a Gram-negative anaerobic bacterium that plays a key role in the development of oral inflammation, such as periodontitis and gingivitis. In the last 10 years, F. nucleatum has been identified as a prevalent bacterium associated with colorectal adenocarcinoma and has also been linked to cancer progression, metastasis and poor disease outcome. While the role of F. nucleatum in colon carcinogenesis has been intensively studied, its role in gastric carcinogenesis is still poorly understood. Although Helicobacter pylori infection has historically been recognized as the strongest risk factor for the development of gastric cancer (GC), with recent advances in DNA sequencing technology, other members of the gastric microbial community, and F. nucleatum in particular, have received increasing attention. In this review, we summarize the existing knowledge on the involvement of F. nucleatum in gastric carcinogenesis and address the potential translational and clinical significance of F. nucleatum in GC.

Pignatelli P, Nuccio F, Piattelli A, Curia MC. The Role of *Fusobacterium nucleatum* in Oral and Colorectal Carcinogenesis. Microorganisms. 2023 Sep 20;11(9):2358. doi: 10.3390/microorganisms11092358. PMID: 37764202; PMCID: PMC10537357.

ABSTRACT

In recent years, several studies have suggested a strong association of microorganisms with several human cancers. Two periodontopathogenic species in particular have been mentioned frequently: Fusobacterium nucleatum (F. nucleatum) and Porphyromonas gingivalis. Chronic periodontal disease has been reported to be a risk factor for oral squamous cell carcinoma (OSCC), colorectal cancer (CRC) and pancreatic cancer. F. nucleatum is a Gram-negative anaerobic bacterium that lives in the oral cavity, urogenital, intestinal and upper digestive tract. It plays a significant role as a co-aggregation factor, with almost all bacterial species that participate in oral plaque formation acting as a bridge between early and late colonizers. F. nucleatum, gives an important inflammatory contribution to tumorigenesis progression and is associated with epithelial-derived malignancies, such as OSCC and CRC. F. nucleatum produces an adhesion protein, FadA, which binds to VE-cadherin on endothelial cells and to E-cadherins on epithelial cells. The last binding activates oncogenic pathways, such as Wnt/βcatenin, in oral and colorectal carcinogenesis. F. nucleatum also affects immune response because its Fap2 protein interacts with an immune receptor named TIGIT present on some T cells and natural killer cells inhibiting immune cells activities. Morover, F. nucleatum release outer membrane vesicles (OMVs), which induce the production of proinflammatory cytokines and initiating inflammation. F. nucleatum migrates from the oral cavity and reaches the colon hematogenously but it is not known if in the bloodstream it reaches the CRC as free, erythrocyte-bound bacteria or in OMV. F. nucleatum abundance in CRC tissue has been inversely correlated with overall survival (OS). The prevention and treatment of periodontal disease through the improvement of oral hygiene should be included in cancer prevention protocols. FadA virulence factors may also serve as novel targets for therapeutic intervention of oral and colorectal cancer.

Pischke S, Ashouri MM, Peters U, Shiprov A, Schulze Zur Wiesch J, Sterneck M, Fischer F, Huebener P, Mader M, Fischer L, Fründt T, Aarabi G, Beikler T. High incidence of periodontitis in patients with ascitic decompensated cirrhosis. World J Hepatol. 2023 Dec 27;15(12):1325-1332. doi: 10.4254/wjh.v15.i12.1325. PMID: 38223419; PMCID: PMC10784813.

ABSTRACT

Background: Periodontitis has been associated with various liver diseases. However, the relevance of periodontitis in the progression of decompensated cirrhosis remains inconclusive. In particular, it is unclear whether the common periodontitis pathogens, Porphyromonas gingivalis (P. gingivalis) and Actinobacillus actinomycetemcomitans (A. actinomycetemcomitans), can be detected not only in the oral mucosa but also in ascites and stool.

Aim: To investigate the significance of periodontitis, P. gingivalis, and A. actinomycetemcomitans in cirrhosis patients with ascitic decompensation.

Methods: This prospective study was conducted at the University Hospital Hamburg-Eppendorf, a tertiary center in Northern Germany. A cohort of 27 patients with ascitic decompensated liver cirrhosis underwent dental examinations to assess the association between periodontitis and various clinical parameters of cirrhosis, as well as patient outcomes. PCR was used to test gingival samples, ascites, and stool for the presence of P. gingivalis and A. actinomycetemcomitans. Gingival samples were collected by probing the deepest gum pocket of a sextant and wiping them on a cotton swab.

Results: Periodontitis was diagnosed in 22 out of 27 (82%) ascite patients, which is significantly more common than in a control cohort of 100 unselected patients (59%, P = 0.04). P. gingivalis was detected in the gingiva of six patients, and one of them also had P. gingivalis in their stool. However, P. gingivalis was not found in the ascites of any patient. Five out of six patients with P. gingivalis had periodontitis (83%). A. actinomycetemcomitans was not detected in any sample. Patients without periodontitis had a significantly higher mortality rate compared to those with periodontitis, and survival (Kaplan-Meier analysis) was longer in patients with periodontitis (P = 0.02). Transplant-free survival was also more common in patients with periodontitis compared to those without (63% vs 0%, P = 0.02).

Conclusion: Decompensated cirrhotic patients frequently suffer from periodontitis. However, there was no evidence of the translocation of P. gingivalis or A. actinomycetemcomitans into ascites. The survival of cirrhotic patients with periodontitis was not reduced.

Qiao F, Li X, Liu Y, Zhang S, Liu D, Li C. Periodontitis and NAFLD-related diseases: A bidirectional two-sample Mendelian randomization study. Oral Dis. 2024 Jul;30(5):3452-3461. doi: 10.1111/odi.14785. Epub 2023 Oct 25. PMID: 37877540.

ABSTRACT

Background: Epidemiological studies have shown an association between periodontitis and nonalcoholic fatty liver disease (NAFLD)-related diseases. However, a causal relationship between these two diseases remains unclear. To examine the causal relationship between these two diseases, we conducted a bidirectional two-sample Mendelian randomization (MR) analysis using genetic markers as proxies.

Methods: Statistical summary was obtained from a large genome-wide association study (GWAS) on NAFLD (N = 342,499), nonalcoholic steatohepatitis (NASH, N = 342,499), fibrosis (N = 339,081), cirrhosis (N = 342,499), fibrosis/cirrhosis (N = 334,553), and periodontitis (N = 34,615) in the European ancestry. The inverse variance weighted (IVW) method was used as the main method to estimate the bidirectional association. Sensitivity analysis was performed to evaluate the rigidity of the results.

Results: Limited evidence indicated positive causal associations between genetically predicted NAFLD and periodontitis (IVW odds ratio [OR], 1.094; 95% confidence interval [CI], 1.006-1.189; p = 0.036) and between cirrhosis and periodontitis (IVW OR, 1.138; 95% CI, 1.001-1.294; p = 0.048). However, the opposite trend did not indicate a causative effect of periodontitis on NAFLD-related diseases. The sensitivity analysis revealed no obvious pleiotropy or heterogeneity.

Conclusions: Our MR analysis provides new evidence in favor of the moderate causal impact of NAFLD on periodontitis. The causal effects of periodontitis on NAFLD-related diseases warrant further investigation.

Qing X, Zhang C, Zhong Z, Zhang T, Wang L, Fang S, Jiang T, Luo X, Yang Y, Song G, Wei W. Causal Association Analysis of Periodontitis and Inflammatory Bowel Disease: A Bidirectional Mendelian Randomization Study. Inflamm Bowel Dis. 2024 Aug 1;30(8):1251-1257. doi: 10.1093/ibd/izad188. PMID: 38408068; PMCID: PMC11291616.

ABSTRACT

Background: Periodontitis has been reported to be associated with inflammatory bowel disease (IBD), including ulcerative colitis (UC), and Crohn's disease (CD). However, the causality of these 2 diseases remains unclear. We conducted bidirectional Mendelian randomization (MR) to investigate the causal relationship between periodontitis and IBD.

Methods: We obtained the genome-wide association study (GWAS) summary data of European populations from FinnGen database (for IBD) and a published article (for periodontitis), from which independent single nucleotide polymorphisms were selected as instrumental variables. Inverse variance-weighted (IVW), MR-Egger, and weighted median (WM) methods were utilized for MR analysis. Heterogeneity or pleiotropy was detected through Cochran's Q test and MR-Egger intercept, respectively. Outlier was identified with MR-PRESSO (Mendelian Randomization Pleiotropy RESidual Sum and Outlier) and leave-one-out analysis. All statistical analyses were performed with R 4.2.1 and the packages of TwoSampleMR version 0.5.6.

Results: Genetic prediction showed that periodontitis was the risk factor of UC (odds ratio [OR], 1.13; 95% confidence interval [CI], 1.01-1.26; P = .027), rather than of CD (OR, 0.92; 95% CI, 0.74-1.15; P = .456) and IBD (OR, 0.96; 95% CI, 0.81-1.13; P = .619). To the contrary, CD, not UC or IBD, resulted in exacerbating periodontitis in terms of the results of the IVW (OR, 1.09; 95% CI, 1.01-1.17; P = .021) and WM (OR, 1.10; 95% CI, 1.01-1.20; P = .030) methods. Heterogeneity or pleiotropy was acceptable.

Conclusions: Our results indicated that CD was the risk factor for periodontitis; conversely, periodontitis was responsible for the exacerbation of UC, enhancing the existence of mouth-gut axis. Patients with UC should pay more attention to periodontal health, while patients with periodontitis should actively pay close heed to intestinal health.

Rodrigues C, Gomes ATPC, Leal J, Pereira P, Lopes PC, Mendes K, Correia MJ, Veiga N, Rosa N, Soares C, Ministro P. Oral health in inflammatory bowel disease: the overlooked impact and the potential role of salivary calprotectin. BMC Oral Health. 2025 May 15;25(1):729. doi: 10.1186/s12903-025-06064-5. PMID: 40375227; PMCID: PMC12082920.

ABSTRACT

Background: Inflammatory Bowel Disease (IBD), a chronic condition characterized by gastrointestinal inflammation, is influenced by genetic and environmental factors. Emerging evidence suggests a “mouth-gut axis,” with the oral cavity reflecting extra-intestinal manifestations of IBD. This study evaluated the oral health status of IBD patients and the potential of salivary calprotectin (SCP) as a biomarker for assessing IBD activity and oral health.

Methods: Oral health was assessed in 100 IBD patients [60 with Crohn’s disease (CD) and 40 with ulcerative colitis (UC)] and 14 controls. Evaluations included the Decayed, Missing, and Filled Teeth (DMFT) Score, Periodontal Diagnosis and the need for dental or prosthetic treatment. Saliva and stool samples were collected to measure SCP and faecal calprotectin (FCP) levels using the Elia Calprotectin 2 Test. IBD activity was evaluated with FCP, the Harvey-Bradshaw Index for CD, and the Partial Mayo Score for UC.

Results: The DMFT index mean was comparable between IBD patients (mean 7.99, SD 7.73) and controls (mean 10.00, SD 6.49). However, periodontal disease was significantly more prevalent in IBD patients (57% in CD, 70% in UC) than in controls (29%), with severe cases (stages III/IV) more frequent in IBD. Additionally, 89% of IBD patients required dental treatment, and 39% needed prosthetic rehabilitation. SCP levels showed no significant correlation with disease activity or oral health status, while FCP correlated with C-reactive protein and erythrocyte sedimentation rate.

Conclusions: This study underscores the need for improved oral health management in IBD patients and suggests that SCP may not be a reliable biomarker for monitoring IBD or periodontal disease.

Ruan Z, Xie J, Yu J, Yin L, Nesheli DN, Ye W. The association between poor dental health and gastric cancer risk: a nationwide cohort and sibling-controlled study. BMC Med. 2025 Jul 21;23(1):434. doi: 10.1186/s12916-025-04273-x. PMID: 40691605; PMCID: PMC12282018.

ABSTRACT

Background: Poor dental health has been linked to an increased risk of gastric cancer (GC), but previous studies were limited by their retrospective design and relatively small sample size.

Methods: We followed a nationwide cohort of 5,888,034 Swedish adults over the age of 19 who visited a dentist between 2009 and 2016. Additionally, a nested case-control study was conducted by comparing incident GC cases to their siblings. Cox regression analyses, using attained age as the timescale and adjusting for potential confounders, were performed to evaluate the association between various dental health conditions and the risk of GC. In addition, we stratified our analyses by sex and age and conducted various sensitivity analyses to ensure the robustness of our findings.

Results: Over an average follow-up of 6.4 years, we identified 3993 new GC cases, including 1241 cardia GC and 2752 non-cardia GC. Compared to individuals with healthy teeth, those with periodontitis had an 11% and 25% increased risk of GC and cardia GC, respectively. The positive associations between odontogenic inflammation and the risk of GC were consistent in sibling-controlled analyses. We also observed a dose-response relationship between the number of remaining teeth and the risk of GC, with fewer teeth associated with higher risks. Additionally, we did not find significant interactions between dental inflammatory conditions and the number of remaining teeth in relation to the risk of GC or its subtypes. Our findings were consistent across different sex and age subgroups and in sensitivity analyses.

Conclusions: This study provides the largest prospective cohort study evidence to date, along with the first sibling-controlled comparisons, supporting the association between poor dental health and GC risk. Promoting dental health in the general population could have significant public health implications in preventing this disease.

Sato S, Kamata Y, Kessoku T, Shimizu T, Kobayashi T, Kurihashi T, Takashiba S, Hatanaka K, Hamada N, Kodama T, Higurashi T, Taguri M, Yoneda M, Usuda H, Wada K, Nakajima A, Morozumi T, Minabe M. A cross-sectional study assessing the relationship between non-alcoholic fatty liver disease and periodontal disease. Sci Rep. 2022 Aug 10;12(1):13621. doi: 10.1038/s41598-022-17917-2. PMID: 35948584; PMCID: PMC9365789.

ABSTRACT

The risk factors for non-alcoholic fatty liver disease (NAFLD) progression are not completely known. Porphyromonas gingivalis infection is a risk factor for systemic diseases. We investigated the association of P. gingivalis infection with the risk of non-alcoholic steatohepatitis progression. Here, hematological tests, periodontal examination, and saliva collection were performed for 164 patients with NAFLD. P. gingivalis was identified in saliva using polymerase chain reaction. Hepatic steatosis and stiffness were evaluated using vibration-controlled transient elastography (VCTE) and magnetic resonance imaging. In patients with NAFLD,

P. gingivalis positivity (P. gingivalis ratio ≥ 0.01%) in saliva correlated with liver stiffness determined using magnetic resonance elastography (MRE; $p < 0.0001$). A P. gingivalis ratio of 0.01% corresponds to 100,000 cells/mL and indicates the proportion of P. gingivalis in the total number of bacteria in the oral cavity. Patients with NAFLD and advanced fibrosis on MRE showed significantly elevated endotoxin activity; those who had > 10 periodontal pockets with depths ≥ 4 mm had significantly increased hepatic stiffness on both VCTE and MRE.

Sulaiman Y, Pacauskienė IM, Šadzevičienė R, Anuzyte R. Oral and Gut Microbiota Dysbiosis Due to Periodontitis: Systemic Implications and Links to Gastrointestinal Cancer: A Narrative Review. Medicina (Kaunas). 2024 Aug 29;60(9):1416. doi: 10.3390/medicina60091416. PMID: 39336457; PMCID: PMC11433653.

ABSTRACT

Periodontitis can disrupt oral and gut microbiota, leading to dysbiosis that affects overall systemic health. Besides the spread of periodontal pathogens by the hematogenous route, they can also be translocated into the gastrointestinal tract, possibly intervening in the neoplastic process in the gastrointestinal tract. This manuscript reviews the relationship between oral and gut microbiota due to periodontitis, discussing systemic health implications and potential links to gastrointestinal cancer. This article highlights the significance and effect of dysbiosis in the gut, emphasizing the importance of maintaining oral health to prevent systemic diseases. Lastly, it will go through therapeutic innovations such as probiotics and oral microbiota analysis tools for systemic disease detection. These findings will mark the integration of oral health management in clinical practice to lower systemic disease risk and improve overall patient outcomes. Aim of work: This manuscript aims to unravel the pathological interaction between oral and gut microbiota and their bidirectional effect on systemic diseases. Materials and methods: The review was performed using the MEDLINE and ScienceDirect databases. Reviewed articles were published in English between the year 2015 and 2024. The search used keywords such as ("oral microbiota" AND "periodontal disease") OR ("oral microbiota" AND "gastrointestinal cancer") OR ("Porphyromonas gingivalis" AND "periodontal disease") OR ("Helicobacter pylori" AND "gastric cancer") OR ("gut microbiome" AND "inflammatory bowel disease") OR ("oral microbiome" AND "systemic diseases"). Conclusions: The dysbiotic change in the oral cavity due to periodontitis is linked directly and indirectly to systemic diseases such as IBS, neurodegenerative diseases, muscle joint diseases, respiratory infections, and gastrointestinal cancer; this underscores the importance of maintaining oral hygiene for prophylaxis of oral diseases and the prevention of systemic diseases. A better understanding of the interconnections between oral health and systemic diseases will integrate oral health management to offer new prevention, diagnostic, and treatment opportunities to improve overall patient outcomes.

Tan Q, Ma X, Yang B, Liu Y, Xie Y, Wang X, Yuan W, Ma J. Periodontitis pathogen Porphyromonas gingivalis promotes pancreatic tumorigenesis via neutrophil elastase from tumor-associated neutrophils. Gut Microbes. 2022 Jan-Dec;14(1):2073785. doi: 10.1080/19490976.2022.2073785. PMID: 35549648; PMCID: PMC9116393.

ABSTRACT

Intratumor microbiome shapes the immune system and influences the outcome of various tumors. Porphyromonas gingivalis (P. gingivalis), the keystone periodontal pathogen, is highly epidemically connected with pancreatic cancer (PC). However, its causative role and the underlining mechanism in promoting PC oncogenesis remain unclear. Here, we illustrated the landscape of intratumor microbiome and its bacterial correlation with oral cavity in PC patients, where P. gingivalis presented both in the oral cavity and tumor tissues. When exposed to P. gingivalis, tumor development was accelerated in orthotopic and subcutaneous PC mouse model, and the cancerous pancreas exhibited a neutrophils-dominated proinflammatory tumor microenvironment. Mechanistically, the intratumoral P. gingivalis promoted PC progression via elevating the secretion of neutrophilic chemokines and neutrophil elastase (NE). Collectively, our study disclosed the bacterial link between periodontitis and PC, and revealed a previously unrecognized mechanism of P. gingivalis in PC pathophysiology, hinting at therapeutic implications.

Tanwar H, Gnanasekaran JM, Allison D, Chuang LS, He X, Aimetti M, Baima G, Costalonga M, Cross RK, Sears C, Mehandru S, Cho J, Colombel JF, Raufman JP, Thumbigere-Math V. Unravelling the Oral-Gut Axis: Interconnection Between Periodontitis and Inflammatory Bowel Disease, Current Challenges, and Future Perspective. J Crohns Colitis. 2024 Aug 14;18(8):1319-1341. doi: 10.1093/ecco-jcc/jjae028. PMID: 38417137; PMCID: PMC11324343.

ABSTRACT

As the opposite ends of the orodigestive tract, the oral cavity and the intestine share anatomical, microbial, and immunological ties that have bidirectional health implications. A growing body of evidence suggests an interconnection between oral pathologies and inflammatory bowel disease [IBD], implying a shift from the traditional concept of independent diseases to a complex, reciprocal cycle. This review outlines the evidence supporting an 'oral-gut' axis, marked by a higher prevalence of periodontitis and other oral conditions in IBD patients and vice versa. We present an in-depth examination of the interconnection between oral pathologies and IBD, highlighting the shared microbiological and immunological pathways, and proposing a 'multi-hit' hypothesis in the pathogenesis of periodontitis-mediated intestinal inflammation. Furthermore, the review underscores the critical need for a collaborative approach between dentists and gastroenterologists to provide holistic oral-systemic healthcare.

Vegda HS, Patel B, Girdhar GA, Pathan MSH, Ahmad R, Haque M, Sinha S, Kumar S. Role of Nonalcoholic Fatty Liver Disease in Periodontitis: A Bidirectional Relationship. Cureus. 2024 Jul 3;16(7):e63775. doi: 10.7759/cureus.63775. PMID: 39100036; PMCID: PMC11297857.

ABSTRACT

Nonalcoholic fatty liver disease (NAFLD) and periodontitis share common risk factors such as obesity, insulin resistance (IR), and dyslipidemia, which contribute to systemic inflammation. It has been suggested that a bidirectional relationship exists between NAFLD and periodontitis, indicating that one condition may exacerbate the other. NAFLD is characterized by excessive fat deposition in the liver and is associated with low-grade chronic inflammation. There are several risk factors for the development of NAFLD, including gender, geriatric community, race, ethnicity, poor sleep quality and sleep deprivation, physical activity, nutritional status, dysbiosis gut microbiota, increased oxidative stress, overweight, obesity, higher body mass index (BMI), IR, type 2 diabetes mellitus (T2DM), metabolic syndrome (MetS), dyslipidemia (hypercholesterolemia), and sarcopenia (decreased skeletal muscle mass). This systemic inflammation can contribute to the progression of periodontitis by impairing immune responses and exacerbating the inflammatory processes in the

periodontal tissues. Furthermore, individuals with NAFLD often exhibit altered lipid metabolism, which may affect oral microbiota composition, leading to dysbiosis and increased susceptibility to periodontal disease. Conversely, periodontitis has been linked to the progression of NAFLD through mechanisms involving systemic inflammation and oxidative stress. Chronic periodontal inflammation can release pro-inflammatory cytokines and bacterial toxins into the bloodstream, contributing to liver inflammation and exacerbating hepatic steatosis. Moreover, periodontitis-induced oxidative stress may promote hepatic lipid accumulation and IR, further aggravating NAFLD. The interplay between NAFLD and periodontitis underscores the importance of comprehensive management strategies targeting both conditions. Lifestyle modifications such as regular exercise, a healthy diet, and proper oral hygiene practices are crucial for preventing and managing these interconnected diseases. Additionally, interdisciplinary collaboration between hepatologists and periodontists is essential for optimizing patient care and improving outcomes in individuals with NAFLD and periodontitis.

Wang A, Zhai Z, Ding Y, Wei J, Wei Z, Cao H. The oral-gut microbiome axis in inflammatory bowel disease: from inside to insight. Front Immunol. 2024 Jul 26;15:1430001. doi: 10.3389/fimmu.2024.1430001. PMID: 39131163; PMCID: PMC11310172.

ABSTRACT

Inflammatory bowel disease (IBD) is an idiopathic and persistent inflammatory illness of the bowels, leading to a substantial burden on both society and patients due to its high incidence and recurrence. The pathogenesis of IBD is multifaceted, partly attributed to the imbalance of immune responses toward the gut microbiota. There is a correlation between the severity of the disease and the imbalance in the oral microbiota, which has been discovered in recent research highlighting the role of oral microbes in the development of IBD. In addition, various oral conditions, such as angular cheilitis and periodontitis, are common extraintestinal manifestations (EIMs) of IBD and are associated with the severity of colonic inflammation. However, it is still unclear exactly how the oral microbiota contributes to the pathogenesis of IBD. This review sheds light on the probable causal involvement of oral microbiota in intestinal inflammation by providing an overview of the evidence, developments, and future directions regarding the relationship between oral microbiota and IBD. Changes in the oral microbiota can serve as markers for IBD, aiding in early diagnosis and predicting disease progression. Promising advances in probiotic-mediated oral microbiome modification and antibiotic-targeted eradication of specific oral pathogens hold potential to prevent IBD recurrence.

Wang B, Deng J, Donati V, Merali N, Frampton AE, Giovannetti E, Deng D. The Roles and Interactions of Porphyromonas gingivalis and Fusobacterium nucleatum in Oral and Gastrointestinal Carcinogenesis: A Narrative Review. Pathogens. 2024 Jan 20;13(1):93. doi: 10.3390/pathogens13010093. PMID: 38276166; PMCID: PMC10820765.

ABSTRACT

Epidemiological studies have spotlighted the intricate relationship between individual oral bacteria and tumor occurrence. Porphyromonas gingivalis and Fusobacteria nucleatum, which are known periodontal pathogens, have emerged as extensively studied participants with potential pathogenic abilities in carcinogenesis. However, the complex dynamics arising from interactions between these two pathogens were less addressed. This narrative review aims to summarize the current knowledge on the prevalence and mechanism implications of P. gingivalis and F. nucleatum in the carcinogenesis of oral squamous cell carcinoma (OSCC), colorectal cancer (CRC), and pancreatic ductal adenocarcinoma (PDAC). In particular, it explores

the clinical and experimental evidence on the interplay between P. gingivalis and F. nucleatum in affecting oral and gastrointestinal carcinogenesis. P. gingivalis and F. nucleatum, which are recognized as keystone or bridging bacteria, were identified in multiple clinical studies simultaneously. The prevalence of both bacteria species correlated with cancer development progression, emphasizing the potential impact of the collaboration. Regrettably, there was insufficient experimental evidence to demonstrate the synergistic function. We further propose a hypothesis to elucidate the underlying mechanisms, offering a promising avenue for future research in this dynamic and evolving field.

Wang Q, Gu WJ, Ning FL, Sun M, Zhao ZG, Abe MU, Li ZN, Zhang CD. Association between Periodontal Diseases and the Risk of Site-Specific Gastrointestinal Cancers: A Systematic Review and Meta-Analysis. J Dent Res. 2024 Sep;103(10):962-972. doi: 10.1177/00220345241263768. Epub 2024 Aug 26. PMID: 39185624.

ABSTRACT

The association between periodontal diseases and the risk of gastrointestinal cancers, especially site-specific gastrointestinal cancers, remains unclear. Here, we comprehensively searched PubMed, EMBASE, Web of Science, and Google Scholar from inception to April 2024 to identify relevant studies. The pooled hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated with a random-effects model. Subgroup analyses and sensitivity analyses were conducted to confirm the robustness of the main findings in different populations. This study was reported according to PRISMA 2020 guidelines. In total, we identified 19 studies, including 16.6 million participants. Individuals with periodontal diseases had an increased risk of overall gastrointestinal cancers compared with those without periodontal diseases (HR 1.31, 95% CI 1.16-1.49). Periodontal diseases significantly increased the risk of esophageal cancer by 39% (HR 1.39, 95% CI 1.15-1.68), gastric cancer by 13% (HR 1.13, 95% CI 1.01-1.26), colorectal cancer by 21% (HR 1.21, 95% CI 1.05-1.39), pancreatic cancer by 35% (HR 1.35, 95% CI 1.00-1.82), and liver cancer by 9% (HR 1.09, 95% CI 1.04-1.13). The risk of gastrointestinal cancers was significantly increased by periodontitis (HR 1.45, 95% CI 1.14-1.85), gingivitis (HR 1.03, 95% CI 1.01-1.04), and periodontitis/gingivitis (HR 1.27, 95% CI 1.07-1.51). Furthermore, severe periodontal diseases showed a significantly increased risk of gastrointestinal cancer (HR 1.79, 95% CI 1.07-2.99). Results of sensitivity analyses for site-specific gastrointestinal cancers were robust with the main findings. In summary, periodontal diseases, especially severe periodontitis, increase the risk of overall and site-specific gastrointestinal cancers. Interventions to prevent and manage periodontal diseases may reduce the risk of developing gastrointestinal cancers.

Wang Z, Wei M, Wan J, He W, Zhou J, Zhang Y, Liu Y, Liu Y, Liu D, Zhu Q, Wang X, Wu K. Oral Microbiota Dysbiosis Initiated by Chronic Colitis and the Possible Role in Oral Mucosa Changes. Oral Dis. 2025 Jun 19. doi: 10.1111/odi.15344. Epub ahead of print. PMID: 40534416.

ABSTRACT

Objective: To investigate oral microbiota dysbiosis and cytopathological changes in oral mucosa of murine chronic colitis model and the correlation between them.

Methods: Dextran sodium sulfate (DSS) induced chronic colitis was established in SPF C57BL/6 male mice, oral microbiome characterization was performed using 16S rRNA gene sequencing, and cytopathological and immunohistochemistry assessment was performed in oral mucosa.

Results: When chronic colitis was induced, the overall microbial composition of the oral microbiome was altered with increased abundance in phylum Proteobacteria (82.2%), Actinobacteria (2.6%) and decreased abundance in Firmicutes (12.7%), Bacteroidetes (1.1%). Among the top 10 most abundance genera, Streptococcus was the only genera significantly decreased in colitis mice oral cavity. Meanwhile, oral epithelial hyperplasia was identified in the murine chronic colitis model, and the ki67 expression was significantly upregulated in oral epithelium ($p < 0.05$). The chronic course of colitis did not lead to obvious inflammatory infiltration in the oral mucosa. Spearman analysis indicated a strong inverse correlation ($r = -0.52$, $p = 0.03$) between oral Streptococcus and epithelium thickness.

Conclusions: The chronic colitis mice displayed epithelial hyperplasia in the oral mucosa without obvious inflammatory infiltration, which might be associated with oral dysbiosis, especially a decreased abundance of Streptococcus.

Xiang Z, Li X, Wang X, Deng B, He H, Xu M, Wu X, Tan C, Liu Y, Yu B, Zhang J, Dong W. Fusobacterium nucleatum exacerbates colitis via STAT3 activation induced by Acetyl-CoA accumulation. Gut Microbes. 2025 Dec;17(1):2489070. doi: 10.1080/19490976.2025.2489070. Epub 2025 Apr 11. PMID: 40212016.

ABSTRACT

Fusobacterium nucleatum (F. nucleatum) has emerged as a potential contributor to ulcerative colitis (UC) pathogenesis, although the specific mechanisms remain incompletely understood. This study demonstrates that F. nucleatum promotes colitis by disrupting intestinal barrier integrity, inducing apoptosis in epithelial cells, and modulating inflammatory pathways. Furthermore, we demonstrate that F. nucleatum promotes STAT3 acetylation at K685, followed by phosphorylation at Y705, thereby enhancing its transcriptional activity and exacerbating colitis severity. Additionally, F. nucleatum-mediated upregulation of acetyl-CoA levels is responsible for STAT3 acetylation, linking metabolic processes to UC pathophysiology. Pharmacological inhibition of acetyl-CoA production effectively mitigates F. nucleatum-induced colitis in experimental models, suggesting potential therapeutic strategies targeting these pathways. These findings unveil a novel regulatory pathway in F. nucleatum-associated UC progression and offer new insights for future UC prevention and treatment.

Xu J, Zhang Y, Fang XH, Liu Y, Huang YB, Ke ZL, Wang Y, Zhang YF, Zhang Y, Zhou JH, Su HT, Chen N, Liu YL. The oral bacterial microbiota facilitates the stratification for ulcerative colitis patients with oral ulcers. Ann Clin Microbiol Antimicrob. 2023 Nov 9;22(1):99. doi: 10.1186/s12941-023-00646-3. PMID: 37946238; PMCID: PMC10633958.

ABSTRACT

Background: Clinically, a large part of inflammatory bowel disease (IBD) patients is complicated by oral lesions. Although previous studies proved oral microbial dysbiosis in IBD patients, the bacterial community in the gastrointestinal (GI) tract of those IBD patients combined with oral ulcers has not been profiled yet.

Methods: In this study, we enrolled four groups of subjects, including healthy controls (CON), oral ulcer patients (OU), and ulcerative colitis patients with (UC_OU) and without (UC) oral ulcers. Bio-samples from three GI niches containing salivary, buccal, and fecal samples, were collected for 16S rRNA V3-V4 region sequencing. Bacterial abundance and related bio-functions were compared, and data showed that the fecal microbiota was more potent than salivary and buccal microbes in shaping the host immune system. ~ 22 UC and 10 UC_OU 5-aminosalicylate (5-ASA) routine treated patients were followed-up for six months; according to their treatment response (a decrease in the endoscopic Mayo score), they were further sub-grouped as responding and non-responding patients.

Results: We found those UC patients complicated with oral ulcers presented weaker treatment response, and three oral bacterial genera, i.e., Fusobacterium, Oribacterium, and Campylobacter, might be connected with treatment responding. Additionally, the salivary microbiome could be an indicator of treatment responding in 5-ASA routine treatment rather than buccal or fecal ones.

Conclusions: The fecal microbiota had a strong effect on the host's immune indices, while the oral bacterial microbiota could help stratification for ulcerative colitis patients with oral ulcers. Additionally, the oral microbiota had the potential role in reflecting the treatment response of UC patients. Three oral bacteria genera (Fusobacterium, Oribacterium, and Campylobacter) might be involved in UC patients with oral ulcers lacking treatment responses, and monitoring oral microbiota may be meaningful in assessing the therapeutic response in UC patients.

Yamazaki K, Kamada N. Exploring the oral-gut linkage: Interrelationship between oral and systemic diseases. Mucosal Immunol. 2024 Feb;17(1):147-153. doi: 10.1016/j.mucimm.2023.11.006. Epub 2023 Nov 24. PMID: 38007003; PMCID: PMC11222583.

ABSTRACT

The oral cavity harbors a diverse microbiota that plays a significant role in maintaining homeostasis. Disruption of this balance can lead to various oral diseases, including periodontitis. Accumulating evidence suggests a connection between periodontitis and extra-oral diseases such as cardiovascular disease, rheumatoid arthritis, obesity, and diabetes. During periodontitis, oral bacteria enter the bloodstream directly, impacting extra-oral organs. Furthermore, recent studies have uncovered another pathway, the direct oral-gut axis, where oral bacteria translocate to the gut through an enteral route, influencing gut microbiota and metabolism. Oral pathobionts associated with exacerbation of periodontal disease are implicated in gut pathology, including inflammatory bowel disease and colorectal cancer through ectopic gut colonization. Furthermore, oral bacteria can provoke host immune responses, leading to colitis and other inflammatory diseases. Conversely, mechanisms by which extra-oral conditions exacerbate oral diseases, such as periodontitis, are also beginning to be elucidated. This review discusses the bidirectional interrelationship between oral and systemic diseases based on the oral-gut linkage.

Yu J, Lyu J, Zhu T, Li Y, Xia H, Liu Q, Li L, Chen B. Oral-gut axis in inflammation: periodontitis exacerbates ulcerative colitis via microbial dysbiosis and barrier disruption. BMC Oral Health. 2025 Jun 3;25(1):894. doi: 10.1186/s12903-025-06269-8. PMID: 40462053; PMCID: PMC12135269.

ABSTRACT

Background: Periodontitis is a chronic inflammatory disease, having significant impact on systemic conditions. Ulcerative colitis (UC) is a chronic relapsing inflammatory disorder of the intestines. Studies have suggested a potential association between periodontitis and UC. This study aims to elucidate the influence of periodontitis on the progression of UC and to uncover the potential mechanistic pathways involved.

Methods: A total of 20 male C57BL/6J mice were randomly assigned to four groups: Sham, Periodontitis (P), UC, and Periodontitis + UC (P-UC). A chronic UC model was induced by alternating oral administration of 1% and 0.5% Dextran Sulfate Sodium Salt (DSS) solution, while periodontitis was induced by ligatures. Disease severity was accessed using Disease Activity Index (DAI), histopathology, and intestinal permeability assays. Gut microbiota and periodontal microbiota was analyzed using 16S rRNA sequencing. Tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6) were measured by quantitative PCR (qPCR) to evaluate the systemic inflammation burden. Zonula occludens-1 (ZO-1) and occludin in intestinal tissues were assessed using qPCR and immunohistochemistry. Correlation analyses were performed between periodontal destruction indices and markers.

Results: A chronic UC model closely resembling clinical conditions was successfully established. The P-UC group exhibited earlier and more pronounced body weight loss than the UC group. Colonic inflammation was exacerbated, with significantly elevated TNF-α and IL-6 expression ($P < 0.05$). In the P-UC group, intestinal barrier disruption was evident with reduced occludin protein levels ($P < 0.01$) and increased intestinal permeability ($P < 0.05$), indicated by serum diamine oxidase (DAO). Both the P-UC and UC groups exhibited notable dysbiosis of the gut microbiota, with the P-UC group showing significantly higher abundance of UC-associated bacteria, such as Muribaculum and Allobaculum ($P < 0.05$), compared to the UC group. A trend toward reduced abundance of the gut-protective bacterium Akkermansia was also observed ($P = 0.06$). Pearson correlation analysis confirmed the association between periodontitis and intestinal inflammation, suggesting that intestinal barrier dysfunction and gut microbiota dysbiosis may be key mediators in periodontitis-induced UC exacerbation.

Conclusion: Periodontitis may exacerbate UC by increasing harmful gut bacteria, reducing beneficial bacteria, and promoting the secretion of pro-inflammatory cytokines, thereby disrupting the intestinal barrier and worsening UC severity.

Zhang Y, Sun C, Song EJ, Liang M, Shi T, Min M, Sun Y. Is periodontitis a risk indicator for gastrointestinal cancers? A meta-analysis of cohort studies. J Clin Periodontol. 2020 Feb;47(2):134-147. doi: 10.1111/jcpe.13217. Epub 2019 Nov 25. PMID: 31697412.

ABSTRACT

Objective: To evaluate the association between periodontitis and the incidence and mortality of gastrointestinal cancer.

Method: A comprehensive literature search was conducted to identify all relevant studies published prior to April 2019 according to the established inclusion criteria. The pooled hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated with a random-effects model.

Results: We identified 10 studies with 26 estimates of the relationship between periodontitis and gastrointestinal cancer. The HR for the incidence of gastrointestinal cancer in periodontitis was 1.23 (95% CI: 1.10-1.37). Subgroup analyses showed that periodontitis was associated with an increased risk of gastrointestinal cancers in prospective cohort studies and high-quality studies, North American individuals, and individuals 18 years or older, as well as when the dental status was self-reported and when the study was adjusted for smoking. A meta-analysis of nine reports demonstrated that periodontitis was associated with increased mortality from gastrointestinal cancer (HR = 1.59, 95% CI: 1.16-2.16). Additionally, periodontitis was associated with mortality from pancreatic cancer (HR = 2.20, 95% CI: 1.44-3.37); thus, periodontitis may be a risk factor for pancreatic cancer.

Conclusion: Our meta-analysis demonstrated that periodontitis may be a risk factor for gastrointestinal cancers. Additional prospective cohort studies are warranted to confirm these findings.

Zhang Y, Zhang S. Oral microbiota and biliary tract cancers: unveiling hidden mechanistic links. Front Oncol. 2025 Jun 11;15:1585923. doi: 10.3389/fonc.2025.1585923. PMID: 40567604; PMCID: PMC12187648.

ABSTRACT

Biliary tract cancers (BTCs), a group of rare aggressive malignancies, posed significant clinical challenges due to late diagnosis and limited therapies. While gut microbiota had been extensively studied in gastrointestinal cancers, the role of oral microbiota-a primary microbial reservoir entering the digestive system-remained poorly understood. Emerging evidence indicated that oral bacteria might affect biliary carcinogenesis through direct colonization, immune modulation, and metabolic interactions via the oral-gut-liver axis. This narrative review analyzed current research connecting oral microbial imbalance with BTCs. It explored how bacterial translocation, inflammatory metabolites, and immune alterations could promote cancer development. Established BTC risk factors-including gallstones, primary sclerosing cholangitis, cirrhosis, and H. pylori infection-were evaluated for their associations with oral microbiota changes. Epidemiological studies revealed that periodontal disease and poor oral hygiene elevated BTC risk. Sequencing analyses identified oral-origin bacteria (Prevotella, Fusobacterium, Streptococcus) in bile and tumor tissues, suggesting microbial migration through swallowing or bloodstream. Mechanistic investigations showed microbial components (e.g., lipopolysaccharides, membrane vesicles) activated inflammatory pathways (TLR4/NF-κB, STAT3) and modified immune checkpoints, while metabolites potentially altered biliary cell metabolism. Different studies have found variable changes in oral microbiota in the presence of BTCs, thus a novel “biphasic dysbiosis” hypothesis was proposed to explain differing oral microbial diversity patterns across BTC subtypes. Despite progress, critical knowledge gaps persisted regarding causality, spatial microbial variations, and functional impacts of metabolites in BTCs. Future research was recommended to employ multi-omics approaches, single-cell analysis, and AI tools to enhance early detection and prevention strategies.

Zheng Z, Jin W, Guo W, Jin Z, Zuo Y. Oral Fusobacterium nucleatum exacerbates ulcerative colitis via the oral-gut axis: mechanisms and therapeutic implications. Front Cell Infect Microbiol. 2025 Apr 7;15:1564169. doi: 10.3389/fcimb.2025.1564169. PMID: 40260115; PMCID: PMC12009839.

ABSTRACT

Background: Fusobacterium nucleatum (F. nucleatum) is an anaerobic bacterium known for its association with periodontal disease and oral infections. It has been implicated in the development of gastrointestinal diseases such as inflammatory bowel disease and colorectal cancer. Ulcerative colitis (UC), which is characterized by chronic inflammation of the colon, is a condition of unknown etiology with a rising incidence rate, significantly affecting the quality of life for patients. The increased intestinal permeability during UC may facilitate the adherence or invasion of F. nucleatum into the damaged intestinal barrier, leading to exacerbated inflammation.

Methods: This article introduces the concept of the oral-gut axis, reviewing existing literature to analyze the role of F. nucleatum in the pathogenesis of UC and exploring its potential pathogenic mechanisms. It also summarizes the latest advances in treating patients with UC who have F. nucleatum and looks forward to prospective therapeutic strategies and the translational prospects of F. nucleatum within the oral-gut axis.

Results: F. nucleatum may be a key player in the pathogenesis of UC, likely due to its invasiveness during periods of increased intestinal permeability. The paper also discusses innovative approaches for the prevention and management of UC exacerbated by F. nucleatum, paving the way for more effective treatment of UC.

Conclusion: The review offers new insights into the complex relationship between the oral microbiome and intestinal diseases, enhancing our understanding of their dynamic interactions. There is a paucity of literature on therapeutic approaches, indicating a need for further clinical research.

Zhong Y, Kang X, Bai X, Pu B, Smerin D, Zhao L, Xiong X. The Oral-Gut-Brain Axis: The Influence of Microbes as a Link of Periodontitis With Ischemic Stroke. CNS Neurosci Ther. 2024 Dec;30(12):e70152. doi: 10.1111/cns.70152. PMID: 39675010; PMCID: PMC11646473.

ABSTRACT

Periodontitis, a non-communicable chronic inflammation disease resulting from dysbiosis of the oral microbiota, has been demonstrated to have a positive association with the risk of ischemic stroke (IS). The major periodontal pathogens contribute to the progression of stroke-related risk factors such as obesity, diabetes, atherosclerosis, and hypertension. Transcriptional changes in periodontitis pathogens have been detected in oral samples from stroke patients, suggesting a new conceptual framework involving microorganisms. The bidirectional regulation between the gut and the central nervous system (CNS) is mediated by interactions between intestinal microflora and brain cells. The connection between the oral cavity and gut through microbiota indicates that the oral microbial community may play a role in mediating complex communication between the oral cavity and the CNS; however, underlying mechanisms have yet to be fully understood. In this review, we present an overview of key concepts and potential mechanisms of interaction between the oral-gut-brain axis based on previous research, focusing on how the oral microbiome (especially the periodontal pathogens) impacts IS and its risk factors, as well as the mediating role of immune system homeostasis, and providing potential preventive and therapeutic approaches.

Zhou LJ, Chen BY, Li YL, Duan SZ. [Oral Microbiome and Systemic Diseases]. Sichuan Da Xue Xue Bao Yi Xue Ban. 2023 Jan;54(1):1-6. Chinese. doi: 10.12182/20230160504. PMID: 36647635; PMCID: PMC10409018.

ABSTRACT

As one of the most diverse microbial communities within the human body, the oral microbiome is an important component that contributes to the maintenance of human health. The microbial composition of different sites in the oral cavity varies significantly and a dynamic equilibrium is maintained through communications with the environment and oral and distal organs of the host. It has been reported that there is significant correlation between dysbiotic oral microbiome and the occurrence or progression of a variety of systemic diseases. In this review, we summarized recent advances in research on the relationship between oral microbiome and systemic health, focusing on the interaction and pathological mechanisms between oral microbiome and systemic health and hoping to provide new avenues for the early prevention and clinical diagnosis and treatment of systemic diseases.

Zilberstein NF, Engen PA, Swanson GR, Naqib A, Post Z, Alutto J, Green SJ, Shaikh M, Lawrence K, Adnan D, Zhang L, Voigt RM, Schwartz J, Keshavarzian A. The Bidirectional Effects of Periodontal Disease and Oral Dysbiosis on Gut Inflammation in Inflammatory Bowel Disease. J Crohns Colitis. 2025 Apr 4;19(4):jjae162. doi: 10.1093/ecco-jcc/jjae162. PMID: 39447062; PMCID: PMC12041420.

ABSTRACT

Background and aims: Inflammatory bowel disease (IBD) flares can lead to excessive morbidity and mortality. This study aimed to determine whether oral dysbiosis/periodontal disease (PD) is common in IBD and is associated with disease activity in IBD.

Methods: This single-center, prospective, cross-sectional, proof-of-concept, and observational study assessed the frequency of periodontal inflammatory disease and interrogated oral and stool microbiota using 16S rRNA gene amplicon sequencing of active-IBD (aIBD), inactive-IBD (iIBD), and healthy controls (HC). Questionnaires assessed diet, alcohol usage, oral hygiene behavior, and disease activity. A subset of participants underwent comprehensive dental examinations to evaluate PD.

Results: Periodontal disease was severer in aIBD subjects than in HC, as aIBD had poorer quality diets (lower Mediterranean diet scores) than iIBD and HC. Significant differences in microbial community structure were observed in unstimulated saliva, stimulated saliva, gingiva, and stool samples, primarily between aIBD and HC. Saliva from aIBD had higher relative abundances of putative oral pathobionts from the genera Streptococcus, Granulicatella, Rothia, and Actinomyces relative to HC, despite similar oral hygiene behaviors between groups.

Conclusions: Our study suggests that patients with aIBD have severer periodontal disorders and higher relative abundances of putative 'pro-inflammatory' microbiota in their oral cavity, despite normal oral hygiene behaviors. Our data are consistent with the potential presence of an oral-gut inflammatory axis that could trigger IBD flare-ups in at-risk patients. Routine dental health assessments in all IBD patients should be encouraged as part of the health maintenance of IBD and as a potential strategy to decrease the risk of IBD flares.

Živić M, Zdravković N, Stojanović B, Milošević B, Todorović Ž, Adamović M, Zdravković N. Association of Periodontal Disease with Activity of Crohn's Disease. Medicina (Kaunas). 2023 Dec 12;59(12):2154. doi: 10.3390/medicina59122154. PMID: 38138256; PMCID: PMC10744647.

ABSTRACT

Introduction: Crohn's disease (CD) is a chronic inflammatory granulomatous disease that can affect the entire gastrointestinal tract. It is characterized by various extraintestinal manifestations (EIMs), of which oral manifestations (OMs) are often possible. One of the possible OMs is periodontal disease (PD), a chronic inflammatory condition of the supporting tissues of the teeth. This study aimed to show the existence of a mutual relationship between the clinical activity of PD and the clinical and endoscopic activity of CD.

Materials and methods: One clinical and two endoscopic indexes were used for the assessment of CD activity and clinical attachment loss (CAL), bleeding on probing (BOP), pocket probing depth (PPD), and radiographic bone loss (RBL) in a dental panoramic tomogram to assess PD in CD patients.

Results: A total of 38 patients underwent the entire study process, of which 20 patients had CD and 18 patients had CD and PD. Considering all CD activity scores, there were 26 patients with active disease; half of them had PD, and 85.7% of operated patients had active CD. The values of CAL, PPD, BOP, and RBL were higher in active CD patients than those in remission, except for BOP when comparing to the CDAI score, which was higher in those in remission of CD.

Conclusion: The results of this study indicate that there is a connection between the activity of CD and worse conditions of the supporting tissues of the gums in the oral cavity, so it is important to keep in mind the necessity of referring patients with CD to a dentist for timely and adequate therapeutic measures.

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Conclusiones destacadas

Conclusiones destacadas

1. Relación bidireccional: periodontitis y enfermedades digestivas

2. Eje oral-intestinal y disbiosis microbiana

3. Periodontitis y enfermedad inflamatoria intestinal (EII)

4. Periodontitis y cánceres gastrointestinales

5. Relación con hígado graso no alcohólico (NAFLD)

6. Cánceres biliares y eje oral-hígado

7. Mecanismos inmunológicos y vías inflamatorias

8. Implicaciones clínicas y preventivas

1. RELACIÓN BIDIRECCIONAL: PERIODONTITIS Y ENFERMEDADES DIGESTIVAS

- La periodontitis no solo afecta a la cavidad oral, sino que **actúa como un disparador sistémico**, especialmente sobre el eje digestivo.
- Se documenta una **bidireccionalidad clara** con enfermedades como EII, NAFLD, cánceres digestivos y alteraciones hepáticas.
- Diversos estudios confirman que el estado inflamatorio y la disbiosis inducida por periodontitis pueden **agravar o predisponer** a enfermedades digestivas, y a su vez, estas patologías digestivas pueden dificultar el control periodontal.

2. EJE ORAL-INTESTINAL Y DISBIOSIS MICROBIANA

- La migración de bacterias orales patógenas (P. gingivalis, F. nucleatum) hacia el intestino contribuye a la **disbiosis intestinal** y a alteraciones inmunológicas que favorecen inflamación crónica.
- Se ha demostrado que esta disbiosis impacta directamente en la **integridad de la barrera intestinal**, aumentando la permeabilidad (leaky gut) y facilitando el paso de endotoxinas bacterianas al sistema sistémico.
- También se identifica el impacto cruzado desde el intestino: **la colitis crónica puede alterar el microbioma oral**, con signos histológicos en mucosa oral sin inflamación aparente.

3. PERIODONTITIS Y ENFERMEDAD INFLAMATORIA INTESTINAL (EII)

- Existe una **mayor prevalencia de periodontitis en pacientes con EII**, en especial con colitis ulcerosa activa.
- Se han detectado **microbiotas orales alteradas** en pacientes con brote de EII, con menor abundancia de Streptococcus y mayor presencia de patobiontes como Fusobacterium y Campylobacter.
- La inflamación oral puede contribuir a la inflamación intestinal mediante activación inmune, disbiosis y daño epitelial, exacerbando los síntomas y reduciendo la respuesta al tratamiento con 5-ASA.

4. PERIODONTITIS Y CÁNCERES GASTROINTESTINALES

- La evidencia más robusta asocia la periodontitis con **aumento del riesgo de cáncer colorrectal, gástrico, pancreático, esofágico y hepático**.
- Se destacan:
  - Meta-análisis con más de 16 millones de participantes mostrando **incrementos del 13% al 39%** en riesgo según localización tumoral.
  - Detección de **bacterias orales en tejido tumoral**, como F. nucleatum en colon y P. gingivalis en páncreas.
  - Vías patogénicas: activación de STAT3, disrupción de barrera intestinal, y **secreción de elastasa neutrofílica** en tumores.

5. RELACIÓN CON HÍGADO GRASO NO ALCOHÓLICO (NAFLD)

- Estudios recientes muestran una **relación bidireccional entre periodontitis y NAFLD**, compartiendo factores como obesidad, resistencia a la insulina y dislipidemia.
- La inflamación periodontal crónica favorece la **liberación sistémica de citoquinas y endotoxinas**, lo que empeora el estado hepático.
- NAFLD, a su vez, puede alterar la microbiota oral y la inmunorregulación local, empeorando la periodontitis.

6. CÁNCERES BILIARES Y EJE ORAL-HÍGADO

- Nuevas hipótesis como la **"disbiosis bifásica"** proponen que el perfil microbiano oral podría variar según subtipo tumoral biliar.
- Se han hallado bacterias orales en muestras biliares y tumorales, con evidencia de migración por vía enteral o sanguínea.
- Se proponen mecanismos como la activación del eje **TLR4/NF-κB** o **STAT3**, y efectos inmunomoduladores de metabolitos bacterianos.

7. MECANISMOS INMUNOLÓGICOS Y VÍAS INFLAMATORIAS

- Implicación directa de rutas como **NF-κB, IL-6, TNF-α, STAT3**, y efectos sobre moléculas de unión epitelial como occludina o ZO-1.
- La periodontitis induce una respuesta sistémica que favorece inflamación de bajo grado, estrés oxidativo, y **activación inmune persistente**.
- Varios estudios resaltan el potencial diagnóstico y terapéutico de medir cambios en la microbiota oral como **biomarcadores de enfermedades digestivas**.

8. IMPLICACIONES CLÍNICAS Y PREVENTIVAS

Los datos respaldan la necesidad de:

- **Evaluación periodontal rutinaria en pacientes con EII, NAFLD o riesgo oncológico digestivo**.
- Estrategias integradas de salud bucodental y medicina digestiva.
- Potenciar la formación de digestólogos sobre el impacto oral-sistémico.

Tema	Papers incluidos
Relación bidireccional oral-digestiva	Yamazaki 2024, Zhou 2023, Zhong 2024
Disbiosis y eje oral-intestinal	Yu 2025, Zheng 2025, Zilberstein 2025
Periodontitis y EII	Yu 2025, Zilberstein 2025, Živić 2023, Zheng 2025
Periodontitis y cáncer digestivo	Zhang 2020, Zhang & Zhang 2025, otros incluidos previamente
NAFLD y daño hepático	Matsuda 2023 (previo), artículos integrados en la narrativa del resumen
Cánceres biliares	Zhang & Zhang 2025
Vías inmunológicas	Todos los anteriores: NF-κB, IL-6, TNF-α, STAT3, occludina, ZO-1, DAO…
Implicaciones clínicas	Derivado de todos los estudios con enfoque preventivo o diagnóstico

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Conclusiones destacadas individuales

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Conclusiones destacadas individuales

AGUIAR ET AL., 2024, Q1

Tema: Asociación entre periodontitis y adenocarcinoma gástrico.

Contenido: Meta-análisis de 9 estudios (3 casos–control y 6 cohortes) muestra que los pacientes con periodontitis tienen un riesgo 17 % mayor de desarrollar cáncer gástrico (RR = 1.17). El riesgo se mantiene con diagnóstico clínico o autoinformado, siendo especialmente evidente en pacientes asiáticos.

Resumen: La periodontitis se asocia con mayor riesgo de adenocarcinoma gástrico, lo que sugiere un posible papel de la inflamación oral en la carcinogénesis digestiva.

AGUIAR ILS ET AL., 2023, Q1

Tema: Relación entre NAFLD y enfermedad periodontal.

Contenido: Meta-análisis de 5 estudios transversales muestra que los pacientes con hígado graso no alcohólico tienen un 91 % más de probabilidad de presentar periodontitis (OR = 1.91). La calidad metodológica es limitada, pero la asociación es consistente.

Resumen: Se refuerza la conexión entre NAFLD y periodontitis, destacando la necesidad de control periodontal en estos pacientes.

AKBARI ET AL., 2024, Q1

Tema: Rol de Fusobacterium nucleatum en cánceres digestivos y sistémicos.

Contenido: Revisión sobre cómo F. nucleatum, presente en biofilm periodontal, contribuye a varios tipos de cáncer mediante inflamación crónica, evasión inmune, señalización β-catenina y resistencia a quimioterapia. También se discuten sus posibles usos diagnósticos.

Resumen: F. nucleatum actúa como vínculo entre periodontitis y cánceres digestivos, siendo biomarcador potencial y objetivo terapéutico.

AYATI ET AL., 2025, Q1

Tema: Asociación entre periodontitis y EII (CU y Crohn).

Contenido: Meta-análisis con más de 9 000 pacientes muestra que tanto la colitis ulcerosa (OR = 4.55) como la enfermedad de Crohn (OR = 4.30) duplican o cuadruplican el riesgo de periodontitis. No hay diferencia significativa entre ambas.

Resumen: La EII incrementa significativamente el riesgo de enfermedad periodontal, sugiriendo un vínculo bidireccional clínicamente relevante.

BAI ET AL., 2022, Q1

Tema: Efecto de la periodontitis sobre la fibrosis hepática en modelo murino.

Contenido: En ratones con fibrosis hepática inducida, la presencia de periodontitis y microbiota oral humana agravó notablemente el daño hepático, la inflamación y la activación de genes profibróticos, incluyendo TGF-β y células Th17.

Resumen: La periodontitis agrava la fibrosis hepática a través de mecanismos inmunes e inflamatorios, reforzando el eje oral-hígado.

BAIMA ET AL., 2023, Q1

Tema: Eje encía-intestino y riesgo de cáncer gastrointestinal.

Contenido: Revisión que explica cómo patógenos periodontales como P. gingivalis y F. nucleatum favorecen inflamación, disbiosis y evasión inmune, contribuyendo a mutaciones y progresión tumoral en el tracto digestivo.

Resumen: La disbiosis oral y la periodontitis pueden facilitar la carcinogénesis digestiva, posicionando el eje “gum–gut” como objetivo preventivo.

BAIMA ET AL., 2023, Q1

Tema: Prevalencia de periodontitis en pacientes con EII.

Contenido: Estudio caso–control con 180 pacientes con EII y 180 controles muestra mayor prevalencia de periodontitis severa en EII (36.7 % vs. 25.6 %). El riesgo aumenta con duración de la enfermedad y cirugía previa.

Resumen: La EII se asocia a mayor prevalencia y severidad de periodontitis, lo que justifica su inclusión en protocolos preventivos.

BERTL ET AL., 2024, Q1

Tema: Salud oral en pacientes suecos con EII.

Contenido: Encuesta nacional (n=786) muestra que el 38.5 % de los pacientes con CU o Crohn reportan periodontitis severa y hasta un 19 % tiene <20 dientes. Muchos pacientes no perciben interés de sus médicos en salud oral.

Resumen: La enfermedad periodontal es común en EII y está infravalorada en el manejo clínico actual.

BERTL ET AL., 2023, Q1

Tema: Impacto de la periodontitis en la calidad de vida de pacientes con EII.

Contenido: Encuesta a más de 1 100 pacientes con EII y 3 400 controles. Los pacientes con EII reportan peor calidad de vida oral y general. Factores asociados: <20 dientes, lesiones orales, depresión, enfermedad activa.

Resumen: La periodontitis empeora la calidad de vida en pacientes con EII y viceversa, indicando necesidad de atención dental integrada.

BOONYALEKA ET AL., 2023, Q1

Tema: Efecto de Fusobacterium nucleatum en colitis y activación inflamatoria.

Contenido: En modelo murino de colitis, F. nucleatum activa inflamasoma no canónico vía caspasa-11, agrava inflamación y promueve secreción de IL-1α. Neutralizar esta interleucina mejora la evolución de la colitis.

Resumen: F. nucleatum agrava la inflamación intestinal, apoyando su papel patogénico en colitis y enfermedades digestivas.

CHENG ET AL., 2025, Q1

Tema: Dolor crónico, analgésicos y grasa hepática.

Contenido: En un análisis de 39.437 individuos del UK Biobank, se observó que el dolor crónico en múltiples localizaciones se asocia con mayor contenido graso hepático (PDFF), especialmente en usuarios de aspirina. La proteína C reactiva, el sueño, la dieta y la depresión fueron mediadores parciales.

Resumen: El dolor crónico se asocia a esteatosis hepática, y la aspirina podría ser preferible como analgésico en estos pacientes por su efecto protector.

ČOLAK ET AL., 2022, Q1

Tema: Periodontitis y salud hepática en pacientes con obesidad mórbida.

Contenido: En pacientes sometidos a cirugía bariátrica (n=30), se encontró una prevalencia alta tanto de MAFLD como de periodontitis. Aquellos con periodontitis mostraron niveles elevados de PCR, sugiriendo mayor inflamación sistémica.

Resumen: En obesos mórbidos, periodontitis se relaciona con marcadores inflamatorios hepáticos, lo que refuerza el eje periodonto–hígado.

DECLERCQ ET AL., 2025, Q1

Tema: Microbioma salival en pacientes con EII.

Contenido: En una cohorte canadiense, pacientes con EII mostraron menor diversidad alfa y alteraciones en la abundancia de géneros como Veillonella y Streptococcus. Se sugiere utilidad diagnóstica del microbioma salival en EII.

Resumen: El microbioma oral cambia en EII y podría servir como biomarcador no invasivo para cribado o seguimiento.

ELGHANNAM ET AL., 2023, Q1

Tema: Disbiosis oral y enfermedades hepáticas.

Contenido: Revisión sobre cómo la disbiosis oral contribuye a la progresión de MAFLD, hepatitis viral, cirrosis y hepatocarcinoma. Destaca el valor terapéutico de restaurar el equilibrio oral-gastrointestinal.

Resumen: La microbiota oral alterada influye en enfermedades hepáticas y representa una diana potencial de intervención.

FENG ET AL., 2025, Q1

Tema: Causalidad genética entre periodontitis y EII.

Contenido: Estudio de aleatorización mendeliana y transcriptómica revela una relación causal bidireccional entre EII y periodontitis, mediada por IL-1β, CXCR4, TNF-α e IL-17.

Resumen: EII y periodontitis comparten rutas inmunológicas causales, lo que refuerza su vínculo clínico y terapéutico.

GE ET AL., 2024, Q1

Tema: EGCG y modulación del eje oral–intestinal en EII.

Contenido: Revisión sobre el papel del epigalocatequina galato (EGCG) del té verde en reducir colonización bacteriana oral en el intestino y modular la inflamación de la EII. Se exploran mecanismos, toxicidad y nuevos sistemas de liberación.

Resumen: EGCG podría modular la microbiota oral-gut y ser coadyuvante útil en el tratamiento de la EII.

GUADALUPI ET AL., 2023, Q1

Tema: Proteoma salival y diagnóstico de enfermedades hepáticas autoinmunes.

Contenido: Perfil proteómico salival y machine learning permitieron diferenciar pacientes con hepatitis autoinmune y colangitis biliar primaria. Se identificaron proteínas clave asociadas a fibrosis y respuesta inmune.

Resumen: El análisis del proteoma salival permite discriminar enfermedades hepáticas autoinmunes con alta precisión diagnóstica.

HAZNEDAROGLU ET AL., 2023, Q1

Tema: Enfermedad periodontal en niños con EII.

Contenido: Estudio en 32 niños con EII vs. controles muestra mayor gingivitis y manifestaciones orales extraintestinales (62.5 %) en pacientes con EII. No hubo diferencias en caries ni erosión.

Resumen: La enfermedad periodontal y las lesiones orales son más frecuentes en niños con EII, justificando su seguimiento odontológico.

HUDSON ET AL., 2025, Q1

Tema: Periodontitis y cirrosis hepática.

Contenido: Revisión narrativa de los mecanismos de asociación entre periodontitis y cirrosis: disbiosis, inflamación sistémica y menor higiene oral. Se plantea que el tratamiento periodontal puede reducir la mortalidad hepática.

Resumen: La periodontitis influye negativamente en la evolución de la cirrosis, y tratarla puede tener beneficios pronósticos.

JUZBAŠIĆ ET AL., 2025, Q1

Tema: Asociación entre periodontitis y MASLD.

Contenido: Revisión sobre mecanismos compartidos entre MASLD y periodontitis: inflamación crónica, resistencia a insulina y estrés oxidativo. Se propone un enfoque integrado para diagnóstico y tratamiento precoz.

Resumen: MASLD y periodontitis comparten base inflamatoria y requieren manejo multidisciplinar para mejorar resultados clínicos.

KOBAYASHI ET AL., 2023, Q1

Tema: Rol de la periodontitis en el desarrollo y agravamiento del NAFLD/NASH.

Contenido: Revisión centrada en el eje boca–intestino–hígado y la relación entre microbiota oral e intestinal con la patogénesis de NAFLD y NASH. Se analizan vías inflamatorias, disbiosis y mecanismos inmunológicos, además de sugerir nuevas líneas de tratamiento.

Resumen: La periodontitis contribuye al desarrollo de NAFLD/NASH y debe considerarse en su prevención y manejo integral.

KUCHARSKI ET AL., 2025, Q1

Tema: Disbiosis oral y síntomas dentales en colitis ulcerosa.

Contenido: Se describe la alteración de la microbiota oral en colitis ulcerosa, con aumento de patógenos periodontales y mayor prevalencia de periodontitis, caries y manifestaciones como halitosis o úlceras orales. Se propone su valor como biomarcador.

Resumen: La microbiota oral alterada en colitis ulcerosa puede reflejar actividad inmunológica y orientar el tratamiento integral.

KURAJI ET AL., 2021, Q1

Tema: Concepto emergente de NAFLD/NASH asociada a periodontitis.

Contenido: Revisión de mecanismos por los que la periodontitis afecta al hígado: bacteriemia, disbiosis, activación inmune, permeabilidad intestinal. Se propone el término “periodontitis-relacionada con NAFLD/NASH” como nuevo fenotipo.

Resumen: La periodontitis contribuye a la progresión hepática y representa un fenotipo emergente de NAFLD/NASH.

KURAJI ET AL., 2023, Q1

Tema: Terapias periodontales y del microbioma en NAFLD relacionada con periodontitis.

Contenido: Se detallan los efectos de la disbiosis oral–intestinal en la inflamación hepática y se exploran nuevas terapias: tratamiento periodontal clásico, probióticos, prebióticos y bacteriocinas como herramientas frente al NAFLD.

Resumen: El control periodontal y terapias del microbioma pueden ser clave en el tratamiento del NAFLD relacionado con periodontitis.

LA ROSA ET AL., 2025, Q1

Tema: Periodontitis apical y EII.

Contenido: Meta-análisis muestra mayor prevalencia de periodontitis apical (AP) en pacientes con EII. El riesgo se duplica a nivel dental (OR = 1.91) y aumenta 1.6 veces a nivel de paciente. La calidad de la evidencia es baja pero consistente.

Resumen: La AP es más común en pacientes con EII, apoyando el vínculo entre enfermedad oral y digestiva.

LI ET AL., 2024, Q1

Tema: Papel de F. nucleatum y su adhesina FadA en colitis ulcerosa.

Contenido: En ratones con colitis, la inoculación oral de F. nucleatum con FadA exacerba la enfermedad mediante activación de NF-κB y degradación de IκBα. El gen CDH1 (E-cadherina) parece ser clave en esta interacción.

Resumen: La virulencia de F. nucleatum agrava la colitis ulcerosa; FadA puede ser un objetivo terapéutico.

LIU ET AL., 2025, Q1

Tema: Periodontitis y grasa hepática en cohortes poblacionales.

Contenido: En el UK Biobank, la periodontitis se asocia con mayor contenido graso hepático tanto en análisis transversal como longitudinal. Se identificaron mediadores como IMC, PCR, glucemia y dieta.

Resumen: La periodontitis predice aumento de grasa hepática, especialmente en varones, y está mediada por inflamación y factores metabólicos.

LV ET AL., 2025, Q1

Tema: Vesículas de membrana externa (OMVs) y NAFLD.

Contenido: Revisión sobre cómo OMVs derivadas de bacterias periodontales como P. gingivalis inducen activación de células de Kupffer y estrelladas hepáticas, promoviendo inflamación y fibrosis hepática.

Resumen: Las OMVs de patógenos periodontales pueden ser clave en la progresión del NAFLD y representar una nueva diana terapéutica.

MEI ET AL., 2024, Q1

Tema: Bacterias orales en la progresión del NAFLD.

Contenido: Revisión que explica cómo bacterias como P. gingivalis favorecen acumulación lipídica, inflamación e insulinorresistencia, contribuyendo a fibrosis hepática. Se destaca el eje oral–intestinal–hepático como vía clave.

Resumen: Las bacterias periodontales exacerban el NAFLD, y su control podría prevenir progresión a NASH.

LUO ET AL., 2025, Q1

Tema: Periodontitis y riesgo de cáncer gastrointestinal.

Contenido: Estudio caso-control con más de 1.500 participantes muestra que la periodontitis aumenta el riesgo de cáncer de esófago, colon y recto. Además, se asocia a mayor riesgo de metástasis en cáncer rectal.

Resumen: La periodontitis se relaciona con mayor incidencia y peor pronóstico en varios cánceres digestivos.

MUKHERJEE ET AL., 2025, Q1

Tema: Mecanismos moleculares entre periodontitis y disfunción gastrointestinal.

Contenido: Revisión extensa que conecta la diseminación de bacterias periodontales al tubo digestivo con disbiosis intestinal, inflamación y alteraciones epiteliales. Se detallan especies orales implicadas y vías inmunológicas que afectan al intestino, el hígado y el páncreas.

Resumen: La periodontitis contribuye a la disfunción gastrointestinal mediante mecanismos inmunológicos y microbianos bien caracterizados.

PARK ET AL., 2025, Q1

Tema: Periodontitis y riesgo de enfermedad inflamatoria intestinal.

Contenido: Estudio poblacional coreano con más de 2 millones de personas. La periodontitis se asocia con mayor incidencia de Crohn (HR 1.32) y colitis ulcerosa (HR 1.21). Cepillarse ≥3 veces/día reduce el riesgo de Crohn.

Resumen: La salud oral influye en el riesgo de EII, destacando la prevención periodontal como estrategia útil.

PETKEVICIUS ET AL., 2024, Q1

Tema: Fusobacterium nucleatum en la carcinogénesis gástrica.

Contenido: Revisión sobre el papel emergente de F. nucleatum en cáncer gástrico. Aporta evidencia molecular y epidemiológica, incluyendo posibles dianas terapéuticas. Se analiza su interacción con la microbiota gástrica y su rol como cofactor del cáncer.

Resumen: F. nucleatum podría tener un papel relevante en cáncer gástrico, similar a su rol en cáncer colorrectal.

PIGNATELLI ET AL., 2023, Q1

Tema: F. nucleatum en cáncer oral y colorrectal.

Contenido: Revisión sobre mecanismos oncogénicos: adhesión FadA activa vías Wnt/β-catenina y la proteína Fap2 inhibe la respuesta inmune. Se destaca su migración hematógena desde la cavidad oral.

Resumen: F. nucleatum es un puente microbiano entre enfermedad periodontal y cáncer digestivo.

PISCHKE ET AL., 2023, Q1

Tema: Periodontitis en cirrosis hepática descompensada.

Contenido: Estudio prospectivo en 27 pacientes con ascitis. El 82% tenía periodontitis. Se detectó P. gingivalis en encías y en heces de un paciente, pero no en ascitis. Curiosamente, los pacientes con periodontitis mostraron mayor supervivencia.

Resumen: La periodontitis es muy prevalente en cirróticos, aunque no se detecta translocación al líquido ascítico.

QIAO ET AL., 2024, Q1

Tema: Relación causal entre NAFLD y periodontitis.

Contenido: Análisis bidireccional de randomización mendeliana (MR) en más de 340.000 individuos. NAFLD y cirrosis aumentan modestamente el riesgo de periodontitis, pero no se demostró causalidad inversa.

Resumen: El hígado graso podría contribuir al desarrollo de periodontitis; se necesita más investigación sobre la causalidad inversa.

QING ET AL., 2024, Q1

Tema: Relación causal entre periodontitis y EII.

Contenido: Análisis MR revela que la periodontitis aumenta el riesgo de colitis ulcerosa (OR 1.13), mientras que la enfermedad de Crohn aumenta el riesgo de periodontitis. Reforzando el eje boca-intestino.

Resumen: Existe una asociación bidireccional entre periodontitis y EII, especialmente entre periodontitis y colitis ulcerosa.

RODRIGUES ET AL., 2025, Q1

Tema: Salud oral en pacientes con EII y calprotectina salival.

Contenido: En 100 pacientes con EII, se detectó mayor prevalencia de periodontitis (hasta 70%). La necesidad de tratamiento dental fue del 89%. La calprotectina salival no se correlacionó con actividad de EII.

Resumen: La periodontitis es frecuente en EII, pero la calprotectina salival no es útil como biomarcador.

RUAN ET AL., 2025, Q1

Tema: Salud dental y riesgo de cáncer gástrico.

Contenido: Estudio en más de 5,8 millones de suecos muestra que la periodontitis aumenta un 11% el riesgo de cáncer gástrico (25% para cáncer de cardias). Se confirma en análisis con hermanos.

Resumen: La mala salud dental, incluida la periodontitis, eleva el riesgo de cáncer gástrico de forma consistente.

SATO ET AL., 2022, Q1

Tema: P. gingivalis y rigidez hepática en NAFLD.

Contenido: Estudio transversal con 164 pacientes con NAFLD. La presencia salival de P. gingivalis correlaciona con mayor rigidez hepática medida por elastografía. También se asocia con endotoxemia y profundidad de sondaje periodontal.

Resumen: P. gingivalis puede contribuir a la fibrosis hepática en NAFLD, incluso en estadios subclínicos.

SULAIMAN ET AL., 2024, Q1

Tema: Disbiosis oral-intestinal por periodontitis y su relación con cáncer digestivo.

Contenido: Esta revisión destaca cómo la disbiosis causada por periodontitis afecta al eje oral-intestinal, con implicaciones en enfermedades sistémicas y en el desarrollo de cáncer gastrointestinal. Se exploran mecanismos de translocación microbiana, efectos sistémicos inflamatorios y nuevas herramientas diagnósticas como análisis de microbiota oral y uso de probióticos.

Resumen: La disbiosis oral inducida por periodontitis puede intervenir en cáncer gastrointestinal; mantener la salud oral es clave en prevención sistémica.

TAN ET AL., 2022, Q1

Tema: P. gingivalis y cáncer de páncreas.

Contenido: En modelos murinos, la exposición a P. gingivalis acelera el desarrollo tumoral pancreático al activar quimiocinas neutrofílicas y aumentar la elastasa de neutrófilos. El patógeno fue identificado tanto en la cavidad oral como en tejido tumoral pancreático, indicando conexión directa.

Resumen: P. gingivalis puede promover el cáncer de páncreas modulando el microambiente tumoral inmunológico.

TANWAR ET AL., 2024, Q1

Tema: Conexión entre periodontitis y EII.

Contenido: Esta revisión exhaustiva propone el eje oral-intestinal como una vía bidireccional clave en la EII. Se exploran mecanismos compartidos, prevalencia cruzada de patologías y necesidad de colaboración clínico-odontológica. Se presenta una hipótesis “multi-hit” en la patogénesis.

Resumen: Periodontitis y EII comparten rutas microbianas e inmunológicas; se recomienda abordaje conjunto médico-odontológico.

VEGDA ET AL., 2024, Q2

Tema: Relación bidireccional entre NAFLD y periodontitis.

Contenido: Revisión que explora cómo la inflamación sistémica, disbiosis, resistencia a la insulina y el estrés oxidativo vinculan la periodontitis con el hígado graso no alcohólico. Cada patología puede exacerbar la otra, generando un ciclo inflamatorio recíproco.

Resumen: Periodontitis y NAFLD se retroalimentan a través de la inflamación sistémica; su manejo conjunto mejora los resultados clínicos.

WANG A ET AL., 2024, Q1

Tema: Papel de la microbiota oral en la EII.

Contenido: Revisión que analiza cómo el desequilibrio de la microbiota oral actúa como manifestación extraintestinal y modulador de la inflamación colónica en EII. Propone la modificación probiótica de la microbiota oral como vía terapéutica futura.

Resumen: La microbiota oral influye en la EII y puede ser clave para predecir recaídas o intervenir terapéuticamente.

WANG B ET AL., 2024, Q1

Tema: P. gingivalis y F. nucleatum en cáncer oral y digestivo.

Contenido: Revisión sobre la interacción sinérgica entre ambos patógenos en la carcinogénesis oral, colorrectal y pancreática. Se analizan mecanismos compartidos de adhesión, evasión inmune y estimulación inflamatoria.

Resumen: P. gingivalis y F. nucleatum podrían potenciarse mutuamente en la progresión de cánceres gastrointestinales.

WANG Q ET AL., 2024, Q1

Tema: Meta-análisis sobre periodontitis y riesgo de cáncer digestivo.

Contenido: Revisión sistemática de 19 estudios con más de 16 millones de participantes. Se confirma que la periodontitis incrementa el riesgo de varios cánceres: esófago (HR 1.39), estómago (HR 1.13), colon (HR 1.21), páncreas (HR 1.35) e hígado (HR 1.09).

Resumen: La periodontitis aumenta el riesgo de múltiples cánceres gastrointestinales, especialmente en sus formas severas.

WANG Z ET AL., 2025, Q1

Tema: Colitis crónica y alteraciones en la mucosa oral.

Contenido: En ratones con colitis crónica inducida, se observa disbiosis oral (descenso de Streptococcus) y cambios hiperplásicos epiteliales orales sin inflamación evidente. Esto sugiere una conexión indirecta entre colitis y cambios orales.

Resumen: La colitis crónica altera la microbiota y estructura epitelial oral, reflejando el impacto intestinal sobre la boca.

XIANG ET AL., 2025, Q1

Tema: F. nucleatum y colitis vía activación de STAT3.

Contenido: En modelos experimentales, F. nucleatum aumenta la colitis al inducir acumulación de acetil-CoA y activación de STAT3. La inhibición farmacológica de estas rutas mitiga la enfermedad, lo que sugiere nuevas dianas terapéuticas.

Resumen: F. nucleatum exacerba la colitis mediante rutas metabólicas específicas; hay potencial para terapias dirigidas.

XU ET AL., 2023, Q1

Tema: Microbiota oral y respuesta terapéutica en colitis ulcerosa con úlceras orales.

Contenido: En UC con úlceras orales, se identifican tres géneros bacterianos (Fusobacterium, Oribacterium, Campylobacter) asociados a menor respuesta al tratamiento. La microbiota salival podría ser un marcador predictivo útil.

Resumen: La microbiota oral podría predecir respuesta a tratamiento en pacientes con UC y lesiones orales.

YAMAZAKI & KAMADA, 2024, Q1

Tema: Conexión oral-intestinal como vía clave en enfermedades sistémicas.

Contenido: Este artículo revisa el papel de la microbiota oral en enfermedades sistémicas, mostrando cómo bacterias orales pueden llegar al intestino por vía entérica, alterar el microbioma intestinal y contribuir a patologías como EII o cáncer colorrectal. Además, plantea una relación bidireccional, donde enfermedades sistémicas también agravan la periodontitis.

Resumen: La disbiosis oral puede inducir inflamación intestinal y sistémica, lo que refuerza el enfoque integral de la salud oral en medicina interna y digestiva.

YU ET AL., 2025, Q1

Tema: Periodontitis empeora la colitis ulcerosa a través del eje oral-intestinal.

Contenido: En un modelo murino, se demostró que la periodontitis agrava la colitis ulcerosa al aumentar bacterias perjudiciales, reducir barrera intestinal y elevar citocinas proinflamatorias. La disbiosis compartida y la correlación entre destrucción periodontal y daño intestinal fortalecen la evidencia.

Resumen: Periodontitis favorece la progresión de la colitis ulcerosa; tratar la enfermedad periodontal podría ser coadyuvante en pacientes con EII.

ZHANG Y ET AL., 2020, Q1

Tema: Meta-análisis sobre riesgo de cáncer gastrointestinal y periodontitis.

Contenido: En una revisión de 10 estudios, la periodontitis se asoció con mayor incidencia y mortalidad por cáncer gastrointestinal, especialmente páncreas y colorrectal. El HR para cáncer fue de 1.23, y para mortalidad 1.59, con asociación también en estudios ajustados por tabaquismo.

Resumen: La periodontitis es un factor de riesgo para cánceres digestivos, apoyando su inclusión en estrategias preventivas oncológicas.

ZHANG Y & ZHANG S, 2025, Q1

Tema: Relación entre microbiota oral y cáncer de vías biliares.

Contenido: Se propone el eje oral-gut-hígado como ruta patógena en cánceres biliares, mostrando cómo bacterias como Fusobacterium y Prevotella llegan al hígado, inducen inflamación (vía TLR4/STAT3) y alteran el metabolismo celular. Se sugiere un patrón de disbiosis bifásica según el tipo de BTC.

Resumen: La microbiota oral podría influir en la carcinogénesis biliar, lo que abre vías para prevención y detección precoz.

ZHENG Z ET AL., 2025, Q1

Tema: Papel de Fusobacterium nucleatum en la colitis ulcerosa.

Contenido: Esta revisión muestra cómo F. nucleatum, presente en la cavidad oral, puede adherirse al intestino inflamado durante brotes de colitis, empeorando la patología. También se analizan estrategias terapéuticas específicas contra esta bacteria.

Resumen: F. nucleatum conecta salud oral e intestinal; su control podría ser clave en el tratamiento de la colitis ulcerosa.

ZHONG Y ET AL., 2024, Q1

Tema: Eje oral-gut-cerebro en la patogenia del ictus isquémico.

Contenido: Los patógenos periodontales pueden influir en factores de riesgo de ictus (obesidad, aterosclerosis, HTA) y alterar la microbiota intestinal, modulando la respuesta inmune cerebral. Se resalta la posible vía oral-intestino-cerebro.

Resumen: La salud oral podría impactar en el riesgo de ictus a través de vías inflamatorias e inmunológicas mediadas por el intestino.

ZHOU LJ ET AL., 2023, Q1

Tema: Revisión general sobre microbioma oral y enfermedades sistémicas.

Contenido: Este artículo repasa las evidencias que vinculan la disbiosis oral con patologías como diabetes, enfermedades cardiovasculares y digestivas, explicando los mecanismos de translocación bacteriana y activación inmune sistémica.

Resumen: El microbioma oral es un modulador clave de la salud sistémica, y su alteración puede tener efectos a distancia.

ZILBERSTEIN NF ET AL., 2025, Q1

Tema: Relación entre periodontitis y actividad de EII.

Contenido: En pacientes con EII activa se detectaron peores índices periodontales y mayor abundancia de bacterias proinflamatorias orales, a pesar de similar higiene oral. Esto apoya la existencia de un eje inflamatorio oral-intestinal.

Resumen: La periodontitis puede contribuir a los brotes de EII; su control puede mejorar el curso de la enfermedad digestiva.

ŽIVIĆ M ET AL., 2023, Q1

Tema: Actividad de Crohn asociada a peor estado periodontal.

Contenido: En pacientes con Crohn, los indicadores periodontales fueron peores cuanto mayor era la actividad clínica y endoscópica de la enfermedad. Se sugiere una interacción bidireccional entre inflamación intestinal y oral.

Resumen: Evaluar y tratar la salud periodontal puede ser útil en el manejo integral de pacientes con Crohn.

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04

Preguntas y Respuestas

04

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Preguntas y respuestas

1. ¿DE VERDAD LO QUE PASA EN LA BOCA AFECTA AL INTESTINO?

Sí. La boca es el primer tramo del aparato digestivo. Lo que hay allí (bacterias, inflamación, restos) influye en lo que pasa más abajo. Hablamos del "eje oral-intestinal", una vía de comunicación que cada vez se entiende mejor.

2. ¿QUÉ ES ESE EJE ORAL-INTESTINAL DEL QUE SE HABLA TANTO?

Es la conexión entre las bacterias de la boca y las del intestino. Bacterias como Porphyromonas gingivalis o Fusobacterium nucleatum, típicas de las encías enfermas, son capaces de llegar al colon y alterar la flora intestinal, favoreciendo inflamación y enfermedades.

3. ¿UNA PERIODONTITIS PUEDE EMPEORAR UNA ENFERMEDAD DE CROHN O UNA COLITIS ULCEROSA?

Sí. Los pacientes con encías inflamadas tienen más brotes, más actividad de la enfermedad y peor respuesta a los tratamientos. Cuidar la boca es parte del tratamiento integral.

4. ¿Y AL REVÉS? ¿TENER CROHN O COLITIS AFECTA A LA BOCA?

Mucho. Aparecen aftas recurrentes, gingivitis rebelde, lengua inflamada y caries más frecuentes. Es una relación de doble dirección: cuanto peor está uno, peor el otro.

5. ¿LAS ENCÍAS INFLAMADAS PUEDEN AUMENTAR EL RIESGO DE CÁNCER DIGESTIVO?

Se ha descrito una asociación epidemiológica entre periodontitis y cáncer de colon, páncreas y esófago. Hablamos de un factor de riesgo asociado, no de una causa demostrada. Como es modificable, conviene tenerlo en cuenta, sin alarmar: tener periodontitis no significa que vaya a aparecer un cáncer, pero cuidarla resta un factor más a la suma del riesgo.

6. ¿QUIÉN ES FUSOBACTERIUM NUCLEATUM Y POR QUÉ HABLAN TANTO DE ELLA?

Es una bacteria oral que se ha encontrado dentro de tumores de colon y en lesiones de colitis ulcerosa. Favorece la inflamación y la progresión tumoral. La boca actúa como su reservorio principal.

7. ¿LA BOCA INFLUYE TAMBIÉN EN EL HÍGADO?

Sí. En el hígado graso no alcohólico se han encontrado bacterias orales que llegan por la sangre y empeoran la inflamación hepática. En cirróticos, mejorar la higiene oral reduce las endotoxinas que sobrecargan el hígado.

8. ¿Y EN LA ÚLCERA DE ESTÓMAGO? ¿TIENE QUE VER CON LA BOCA?

Algunos estudios han identificado *Helicobacter pylori* en la placa dental, lo que ha llevado a plantear la boca como posible reservorio en personas que se reinfectan tras el tratamiento. Es una hipótesis interesante, todavía en investigación, no una certeza clínica establecida.

9. ¿ES VERDAD QUE CEPILLARSE BIEN LOS DIENTES AYUDA AL INTESTINO?

Rotundamente sí. Una boca sana reduce la cantidad de bacterias y de inflamación que llega al resto del cuerpo, incluido el intestino. Es una medida sencilla, barata y muy eficaz.

10. ¿QUÉ PASA CON LA MICROBIOTA ORAL DURANTE UNA COLITIS O UN CROHN?

Se desequilibra. Aparecen más bacterias proinflamatorias y menos de las protectoras. Ese desequilibrio (disbiosis) alimenta los brotes y dificulta la recuperación de la mucosa.

11. ¿LOS PROBIÓTICOS SIRVEN PARA LA BOCA Y PARA EL INTESTINO?

Algunos sí. Hay cepas que ayudan a reducir la inflamación oral y mejorar el equilibrio intestinal. No son una solución mágica, pero pueden ser un apoyo útil bajo recomendación profesional.

12. ¿INFLUYE LA DIETA EN ESTE VÍNCULO?

Muchísimo. Una dieta rica en azúcares y ultraprocesados favorece la disbiosis en boca e intestino. La dieta mediterránea, con fibra, fruta, verdura y aceite de oliva, beneficia a ambos a la vez.

13. ¿EL TABACO Y EL ALCOHOL EMPEORAN LAS DOS COSAS?

Sí. Aumentan la inflamación, alteran la microbiota, empeoran la periodontitis y elevan el riesgo de cáncer digestivo. Reducirlos es de las medidas con mayor rentabilidad para el conjunto del aparato digestivo.

14. ¿CÓMO PUEDO SABER SI MI PERIODONTITIS ESTÁ EMPEORANDO MIS PROBLEMAS DIGESTIVOS?

Si pese a un buen tratamiento gastroenterológico siguen los brotes, las aftas o la inflamación, conviene revisar la boca. Una analítica con PCR, una exploración periodontal y, si procede, un análisis de microbiota pueden dar pistas claras.

15. ¿DEBERÍA EL GASTROENTERÓLOGO PREGUNTAR POR MI SALUD BUCAL?

Sí, debería. Igual que pregunta por el tabaco o el alcohol. Sangrado al cepillarse, mal aliento o aftas frecuentes son datos clínicos relevantes para el manejo digestivo.

16. ¿Y EL DENTISTA, DEBERÍA PREGUNTAR POR MI INTESTINO?

También. Una encía inflamada en alguien con diarreas crónicas, dolor abdominal o pérdida de peso debe encender alguna alarma. La salud no se reparte por especialidades; el cuerpo es uno solo.

17. ¿ES SEGURO HACERSE LIMPIEZAS DENTALES SI TENGO ENFERMEDAD INFLAMATORIA INTESTINAL?

Sí, y muy recomendable. En fase de brote agudo se evitan procedimientos agresivos, pero el control periodontal habitual es totalmente compatible y beneficioso.

18. ¿LAS BACTERIAS DE LA BOCA PUEDEN ESCONDERSE EN PÓLIPOS DEL COLON?

Se han encontrado bacterias de origen oral, sobre todo *Fusobacterium nucleatum*, en pólipos y tumores de colon. Es un hallazgo consistente en varios estudios y refuerza la idea de que la boca puede actuar como reservorio de patógenos relevantes para el tubo digestivo. El significado clínico exacto sigue estudiándose.

19. ¿POR QUÉ TANTOS PACIENTES CON REFLUJO TIENEN PROBLEMAS DENTALES?

Porque el ácido del estómago erosiona el esmalte y altera el equilibrio bacteriano. Aparece sensibilidad, desgaste y más caries, sobre todo en la cara interna de los dientes. Tratar el reflujo y proteger el esmalte es clave.

20. ¿QUÉ PAPEL TIENEN LAS AFTAS? ¿CUÁNDO DEBEN PREOCUPAR?

Las aftas puntuales son frecuentes y banales. Pero las que se repiten, duran más de dos semanas o aparecen junto a diarrea, sangre en heces o cansancio, deben evaluarse: pueden estar avisando de una enfermedad inflamatoria intestinal o celíaca.

21. ¿Y LA ENFERMEDAD CELÍACA TIENE MARCADORES EN LA BOCA?

Sí. Hipoplasia del esmalte en niños, aftas recurrentes y retraso en la erupción dentaria son hallazgos clásicos. A veces el dentista es el primero en sospecharla.

22. ¿LA BOCA PUEDE AFECTAR AL SISTEMA NERVIOSO A TRAVÉS DEL INTESTINO?

Es lo que se llama eje boca-intestino-cerebro y es una línea de investigación activa. Los datos preliminares sugieren que la inflamación crónica originada en la boca o en el intestino podría influir en el cansancio, el ánimo y la función cognitiva. Por ahora hablamos de asociaciones y mecanismos plausibles, no de una relación causa-efecto demostrada.

23. ¿SIRVE EL CEPILLADO ELÉCTRICO MÁS QUE EL MANUAL EN ESTOS CASOS?

Sí, en general. Los cepillos eléctricos, sobre todo los oscilantes-rotatorios, retiran más placa y reducen mejor la inflamación. En pacientes con destreza limitada o con enfermedades crónicas son una buena inversión.

24. ¿ES MUY CARO PREVENIR TODO ESTO?

No. Una revisión y limpieza anual cuesta mucho menos que tratar un brote de Crohn, una endoscopia o un ingreso. La prevención bucal es de las medidas más rentables en cualquier consulta digestiva.

25. ¿CUÁNDO DEBERÍA DERIVAR UN GASTROENTERÓLOGO A UN PACIENTE AL DENTISTA?

Cuando vea sangrado al cepillarse, mal aliento persistente, aftas recurrentes, encías retraídas o dientes móviles. También antes de iniciar inmunosupresores o biológicos.

26. ¿PUEDE ANALIZARSE LA SALIVA PARA DETECTAR ENFERMEDADES DIGESTIVAS?

Es una línea de investigación activa. Se están estudiando marcadores en saliva relacionados con inflamación intestinal y con ciertos cánceres digestivos. Algunos resultados son prometedores, pero todavía no son pruebas de uso clínico habitual.

27. ¿Y SI LA DISBIOSIS ORAL YA ESTÁ INSTAURADA, SE PUEDE REVERTIR?

Sí, casi siempre. Con limpieza profesional, buena higiene diaria, colutorios específicos durante un tiempo y cambios en la dieta, el equilibrio se recupera en semanas o meses.

28. ¿QUÉ PAPEL JUEGA EL ESTRÉS EN ESTE VÍNCULO?

Importante. Empeora la inflamación, altera la microbiota oral e intestinal y debilita defensas. Cuidar el sueño y el estrés es parte del tratamiento, aunque no lo recetemos en una caja.

29. ¿QUÉ CONSEJO DARÍAS A ALGUIEN CON ENFERMEDAD DIGESTIVA CRÓNICA?

Que vea su boca como un órgano más del aparato digestivo. Cepillarse bien, ir al dentista una vez al año y avisar de cualquier cambio puede marcar la diferencia en el control de su enfermedad.

30. ¿QUÉ MENSAJE FINAL DEJARÍAS A QUIEN ESCUCHA ESTO?

Que un cepillo de dientes es también una herramienta de salud digestiva. La boca abre el tubo digestivo: cuidarla es cuidar todo lo que viene después.

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