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Original Article

Oral Health and Risk of Incident Diabetes: Evidence from a Population-Based Cohort Study

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ABSTRACT

Chronic oral infections, which are widespread and involve ongoing inflammation, may contribute to the development of systemic diseases. This study explored the potential role of long-standing oral conditions as risk factors for various chronic systemic illnesses, seropositive rheumatoid arthritis, including diabetes mellitus, Crohn's disease, ulcerative colitis, and connective tissue diseases, as well as for severe mental disorders such as psychosis and other major psychiatric conditions. This cohort study included 68,273 adults aged 29 and older who visited Helsinki City Health Services for dental care at least once between 2001 and 2002. Participants' records were linked with national registries, including mortality data from Statistics Finland, cancer diagnoses from the Finnish Cancer Registry, and medication reimbursement information from the Finnish Social Insurance Institution, with follow-up continuing until death or the end of 2013. Chronic disease outcomes were identified through the initiation of medications eligible for special reimbursement, indicating partial or full coverage of treatment costs. The diseases examined were diabetes mellitus, seropositive rheumatoid arthritis, connective tissue disorders, Crohn's disease, ulcerative colitis and severe psychiatric disorders. The average follow-up period was 9.8 years. At baseline, approximately 25% of participants had periodontitis, 17% had dental caries, over 70% had apical periodontitis, and 9% had fewer than 24 teeth. Among the chronic systemic conditions studied, only diabetes showed a significant association with oral health indicators. Individuals with 24–27 teeth had a higher incidence rate ratio (IRR) of diabetes (1.21; 95% CI: 1.09–1.33) compared with those having 28 or more teeth, while those with 23 teeth or fewer had an IRR of 1.40 (95% CI: 1.22–1.60). Additionally, the presence of periodontitis (IRR 1.10, 95% CI 1.01–1.20), dental caries (IRR 1.12, 95% CI 1.01–1.23), and apical periodontitis (IRR 1.16, 95% CI 1.04–1.30) was linked to an increased risk of developing diabetes. Findings from our 10-year epidemiological follow-up indicate a link between diabetes and chronic oral diseases, highlighting the importance of coordinated care among healthcare professionals managing these patients.

Keywords: Chronic systemic diseases, Diabetes, Periodontitis, Chronic diseases, Oral infections

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Introduction

Dental caries and periodontitis represent the two most prevalent chronic infectious diseases in the oral cavity. Both are characterized by persistent inflammation and are modulated by numerous behavioral, environmental, and genetic factors [1]. Severe periodontitis affects approximately 10–15% of adults worldwide [2]. Substantial evidence links periodontitis to elevated risk of several systemic chronic conditions—including diabetes [2], inflammatory bowel disease [3], various cancers [4], and cardiovascular diseases—primarily through sustained low-grade systemic inflammation [5-7]. Dental caries, when untreated, frequently progresses to pulp necrosis and apical periodontitis (AP), a condition that can also contribute to systemic

inflammation. In European populations, AP has been reported in 61% of individuals and involves 14% of all teeth, with prevalence increasing markedly with age [8]. Emerging data suggest a moderate but consistent association between endodontic lesions and systemic diseases such as cardiovascular disease and diabetes [9].

Low-grade systemic inflammation and tissue-level inflammatory changes are known to precede the clinical onset of diabetes, contributing to insulin resistance and the subsequent development of the disease and its complications [10, 11]. Given that effective treatments for periodontal disease are widely available, clarifying whether periodontitis causally contributes to diabetes incidence or its severe complications is critical for future prevention strategies [12]. Although some clinical trials and observational studies exist, high-quality longitudinal evidence remains limited [13]. A previous systematic review that included four studies and a total of 22,230 participants concluded that periodontal disease significantly worsens glycaemic control, increases diabetes-related complications, and raises the risk of incident type 2 diabetes (and possibly gestational diabetes) [2]. Because the available evidence at that time was sparse and of variable generalizability, we emphasized the need for large cohort studies with extended follow-up periods.

In a separate systematic review and meta-analysis focused on inflammatory bowel disease (IBD) and oral health, we observed that patients with IBD exhibited significantly higher rates of periodontal disease and poorer overall oral health than individuals without IBD [3]. That meta-analysis, however, was restricted to case-control designs, and we highlighted the necessity of prospective longitudinal studies to establish temporality and potential causality.

Multiple systematic reviews have also provided support for an association between periodontal disease and increased risk of rheumatoid arthritis [14-16]. Most of the epidemiological evidence, however, originates from case-control studies with relatively modest sample sizes. Additional support comes from experimental animal models that demonstrate plausible biological pathways linking periodontal infection to rheumatoid arthritis [17, 18].

We hypothesized that poor oral health and chronic oral infections may precede, exacerbate, or contribute to systemic tissue inflammation implicated in various chronic diseases. Accordingly, the present study examined whether baseline oral health status predicts the subsequent incidence of four distinct systemic conditions: diabetes, inflammatory bowel disease, connective tissue diseases, and psychosis. These outcomes were selected because they are relatively common in the general population, their diagnoses are reliable and uniformly recorded in national registers, and drug-reimbursement entitlement provides an objective marker of incident disease requiring pharmacological treatment. Although clinically diverse, these disorders share potential inflammatory pathways—even psychiatric conditions increasingly appear to involve immune dysregulation. Moreover, individuals with severe mental illness often face socioeconomic disadvantage and poorer somatic health overall; including psychosis therefore serves as an internal specificity control, strengthening causal inference regarding the specific relationship between oral infections and glucose-metabolism disorders when associations differ across outcomes.

The investigation was designed as a large, population-based, prospective register-linked cohort study in which the incidence of the target chronic diseases was defined by the initiation of special drug reimbursement or corresponding entitlement, ensuring high diagnostic validity and complete capture of medically confirmed cases requiring treatment.

Materials and Methods

Study population

The study drew on records from the Helsinki Public Dental Service patient registry to select all persons aged 29 years and older who had at least one routine dental appointment between January 1, 2001, and December 31, 2002. Mortality follow-up and underlying causes of death for these individuals were obtained through automatic record linkage with the national Cause-of-Death Register at Statistics Finland [19], using the unique personal identification number given to every person living in Finland. Death records provided the exact date of death and the primary cause coded with ICD-10. Information on education and socioeconomic position also came from Statistics Finland. Furthermore, the dental records were merged with the nationwide Drug Reimbursement Register kept by the Social Insurance Institution of Finland (SII). This register includes practically all outpatient prescriptions in the country (except for people living in institutions). In Finland, individuals diagnosed with certain serious or chronic illnesses (for example, diabetes) receive special reimbursement entitlement for medication costs based on a doctor's certificate [20]. All cancer cases, including diagnosis date and ICD-O-3 topography and morphology codes [21], were identified from the Finnish Cancer Registry, a population-based registry with near-complete coverage

and high data quality for all cancers diagnosed in Finland since 1953 [22, 23].

During 2001–2002, a total of 71,200 patients attended the Helsinki Public Dental Service. The cohort was limited to those without any previous cancer diagnosis at the time of their first appointment, who survived at least two years afterward, and who had complete records on tooth count and other oral health variables. Treating dentists had carried out comprehensive oral examinations that included caries assessment, periodontal examination (pocket depths, bleeding on probing), and panoramic or bitewing radiographs. Since cancer itself can trigger or alter systemic inflammation [24-27], patients with prior malignancy were excluded to avoid bias. After these restrictions, the analytic cohort comprised 48,609 participants. The observation period for outcomes started exactly two years after the initial dental visit and lasted until the event of interest occurred, December 31, 2013, or death—whichever happened first.

Outcomes

The study outcomes were defined as the onset of selected chronic diseases, identified through the initiation of special medication reimbursement rights. In Finland, patients become eligible for such reimbursements after a physician confirms their diagnosis and need for ongoing treatment [20]. We used both the SII reimbursement categories and the corresponding ICD-10 codes listed within these groups to define the outcomes. The diseases examined included seropositive rheumatoid arthritis (M05), diabetes mellitus (SII code 103), connective tissue disorders (202), Crohn's disease (K50), severe psychotic and other major mental illnesses (112) , ulcerative colitis (K51) and ulcerative colitis and Crohn's disease (208). Severe mental disorders served as a negative control outcome [28]. Participants who already held reimbursement rights for these conditions at baseline were excluded from the analysis of disease incidence.

The measure of exposure and potential confounders

The present study made use of clinical dental data collected during the exposure assessment window, which consisted of all visits occurring within the first two years after the initial appointment. In the Finnish public dental service, treatment procedures are documented using standardized procedure codes issued by the Social Insurance Institution (SII). These codes were employed in the current analysis. The dataset contained detailed procedure codes for treatments related to gingivitis, periodontitis, caries, endodontics, oral surgery, and prosthetics, as well as key indicators of oral health status, including total number of teeth present and several established indices: primary caries lesions (I), number of decayed teeth (DT), decayed/missing/filled teeth (DMFT), and periodontal treatment need based on the Community Periodontal Index (CPI). Periodontitis exposure was operationalized as a dichotomous variable (absent/present) according to whether periodontitis-specific treatment codes had been recorded [24, 25].

Potential confounding factors included sociodemographic variables available for the entire cohort: age, sex, baseline statin use, and socioeconomic status. Socioeconomic status (SES) was derived from occupational classifications provided by Statistics Finland and collapsed into eight broad groups (with an additional "unknown" category). Baseline statin use (ATC code C10A) was ascertained from SII prescription reimbursement records. To adjust for overall oral health beyond periodontitis, the following variables were included: number of teeth (categorized as 0–23, 24–27, or 28–32), primary caries index I (0, 1–2, 3–4, ≥5), decayed teeth DT (0, 1–2, 3–4, ≥5), DMFT index (quartiles: 0–13, 14–18, 19–23, ≥24), CPI score (0–1, 2, 3–4), number of healthy sextants (0, 1, 2–4, 5–6), number of edentulous sextants (0, 1–6), and binary indicators for receipt of various other dental treatments (yes/no) [29, 30]. For the indices I, DT, DMFT, and CPI, the highest value recorded across any visit within the two-year window was used. The count of healthy sextants was taken from the first visit, whereas the count of edentulous sextants was based on the lowest value observed. Because these clinical indices are not documented at every routine or emergency appointment (only at comprehensive examination visits), a proportion of visits lacked this information and were classified as follow-up visits. Individuals without any visit containing complete index data during the two-year window were excluded from the analysis.

Statistical method

Incidence was quantified using incidence rates and analyzed with Poisson regression, with results expressed as incidence rate ratios (IRRs). The model included the following explanatory variables: age, sex, baseline statin use (no/yes), socioeconomic status, I index, DT index, CPI, DMFT index, number of teeth, caries (no/yes), periodontitis (no/yes), and endodontic caries (no/yes). All statistical analyses were performed using the R programming language [31].

Patient involvement and ethical considerations

No patients or members of the public were involved in defining the research question, selecting outcome

measures, or in any aspect of study design, recruitment, conduct, or dissemination of findings. The study protocol was approved by the Ethics Committee of the Faculty of Medicine, University of Helsinki, Finland (01/2014). Necessary data permits were obtained from the Social Insurance Institution of Finland (SII) (68/522/2014), the Finnish Institute for Health and Welfare (THL/1295.05.00/2014), and Statistics Finland (TK-53-1290-14). In accordance with Finnish legislation, this register-based study used fully anonymized data with no direct patient contact; therefore, individual informed consent was not required.

Results and Discussion

The number of participants included in the analyses differed by outcome, ranging from 46,998 individuals followed for diabetes mellitus to 48,223 individuals followed for inflammatory bowel disease (ulcerative colitis and Crohn's disease combined); **(Figure 1; Table 1)**. Average follow-up duration was approximately 9.7 years for diabetes, 9.8 years for connective tissue diseases and severe mental disorders, and 9.9 years for seropositive rheumatoid arthritis and Crohn's disease specifically (9.8 years when ulcerative colitis and Crohn's disease were combined). Variations in cohort size reflect differences in the background prevalence of these chronic conditions, with diabetes being by far the most common.

At baseline (the start of outcome follow-up), roughly 25% of participants had periodontitis, 17% had active caries, more than 70% had at least one apical periodontitis lesion, and 9% had fewer than 24 remaining teeth. The socioeconomic profile of the study cohort closely resembled that of the overall adult population of Helsinki.

Among all chronic conditions examined, only diabetes showed clear associations with oral health indicators **(Table 2; Figure 2)**. Compared with having ≥28 teeth, possessing 24–27 teeth was linked to a 21% higher diabetes incidence (IRR 1.21, 95% CI 1.09–1.33), while having ≤23 teeth was associated with a 40% higher incidence (IRR 1.40, 95% CI 1.22–1.60). Periodontitis (IRR 1.10, 95% CI 1.01–1.20), active caries (IRR 1.12, 95% CI 1.01–1.23), and apical periodontitis (IRR 1.16, 95% CI 1.01–1.30) were also related to increased diabetes risk. Baseline statin use showed the strongest association with subsequent diabetes diagnosis (IRR 2.49, 95% CI 2.10–2.94). Additionally, a higher number of decayed teeth (DT) was linked to diabetes incidence; participants with 3–4 decayed teeth had a 25% greater risk than those with none (IRR 1.25, 95% CI 1.10–1.42).

No consistent associations were found between any oral health variables and the incidence of the other chronic inflammatory or immune-mediated diseases studied.

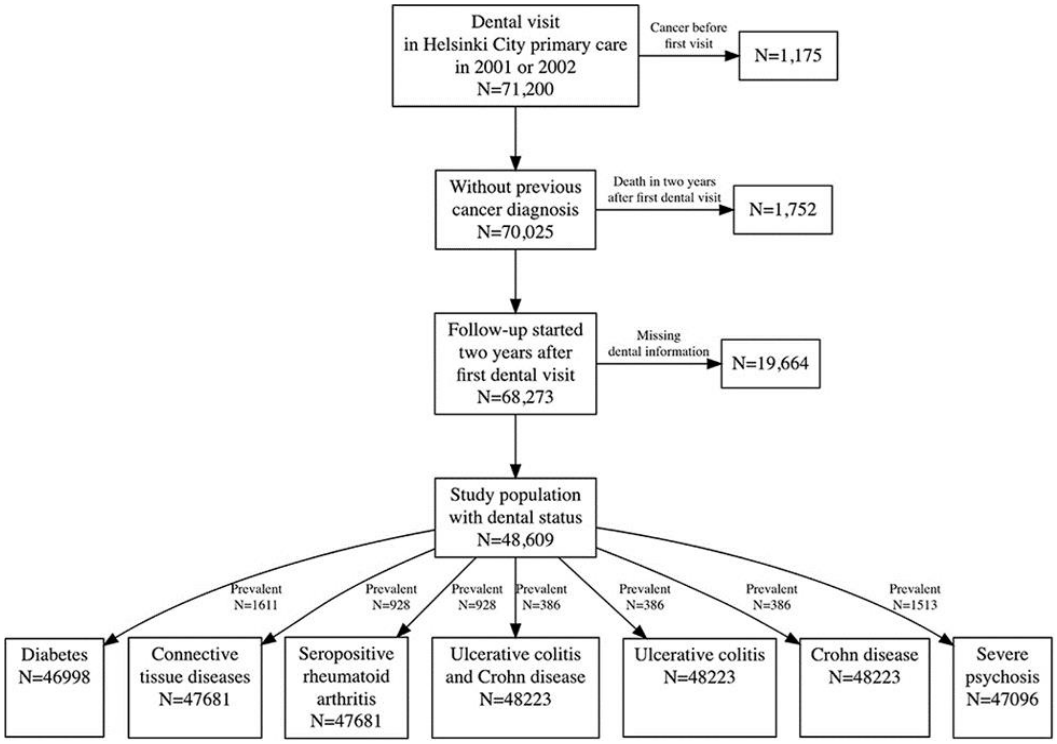

Figure 1. Flow diagram illustrating the construction and selection of the final study cohort.

Table 1. Baseline characteristics of the study participants.

Characteristic	Incident Diabetes (n=46,998)	Incident Connective Tissue Disease (n=47,681)	Incident Seropositive RA (n=47,681)	Incident IBD (n=48,223)	Incident Crohn's Disease (n=48,223)	Incident Ulcerative Colitis (n=48,223)	Incident Severe Psychosis (n=47,096)
Age group, years							
>29 – ≤40	23,832 (50.7%)	23,914 (50.2%)	23,914 (50.2%)	23,951 (49.7%)	23,951 (49.7%)	23,951 (49.7%)	23,623 (50.2%)
>40 – ≤50	14,892 (31.7%)	15,036 (31.5%)	15,036 (31.5%)	15,146 (31.4%)	15,146 (31.4%)	15,146 (31.4%)	14,773 (31.4%)
>50 – ≤60	5,755 (12.2%)	5,969 (12.5%)	5,969 (12.5%)	6,126 (12.7%)	6,126 (12.7%)	6,126 (12.7%)	5,877 (12.5%)
>60 – ≤70	1,016 (2.2%)	1,112 (2.3%)	1,112 (2.3%)	1,208 (2.5%)	1,208 (2.5%)	1,208 (2.5%)	1,124 (2.4%)
>70	1,503 (3.2%)	1,650 (3.5%)	1,650 (3.5%)	1,792 (3.7%)	1,792 (3.7%)	1,792 (3.7%)	1,699 (3.6%)
Sex							
Male	18,531 (39.4%)	19,076 (40.0%)	19,076 (40.0%)	19,191 (39.8%)	19,191 (39.8%)	19,191 (39.8%)	18,728 (39.8%)
Female	28,476 (60.6%)	28,605 (60.0%)	28,605 (60.0%)	29,032 (60.2%)	29,032 (60.2%)	29,032 (60.2%)	28,368 (60.2%)
Statin use							
No	46,144 (98.2%)	46,524 (97.6%)	46,524 (97.6%)	47,008 (97.5%)	47,008 (97.5%)	47,008 (97.5%)	45,932 (97.5%)
Yes	854 (1.8%)	1,157 (2.4%)	1,157 (2.4%)	1,215 (2.5%)	1,215 (2.5%)	1,215 (2.5%)	1,164 (2.5%)
Socioeconomic status							
Upper-level white-collar	10,086 (21.5%)	10,145 (21.3%)	10,145 (21.3%)	10,162 (21.1%)	10,162 (21.1%)	10,162 (21.1%)	10,179 (21.6%)
Entrepreneurs/self-employed	1,425 (3.0%)	1,439 (3.0%)	1,439 (3.0%)	1,444 (3.0%)	1,444 (3.0%)	1,444 (3.0%)	1,437 (3.1%)
Lower-level white-collar	15,254 (32.5%)	15,315 (32.1%)	15,315 (32.1%)	15,416 (32.0%)	15,416 (32.0%)	15,416 (32.0%)	15,398 (32.7%)
Manual workers	8,423 (17.9%)	8,547 (17.9%)	8,547 (17.9%)	8,601 (17.8%)	8,601 (17.8%)	8,601 (17.8%)	8,540 (18.1%)
Unemployed	3,993 (8.5%)	4,087 (8.6%)	4,087 (8.6%)	4,124 (8.6%)	4,124 (8.6%)	4,124 (8.6%)	4,007 (8.5%)
Students	1,486 (3.2%)	1,487 (3.1%)	1,487 (3.1%)	1,500 (3.1%)	1,500 (3.1%)	1,500 (3.1%)	1,439 (3.1%)
Retired	3,868 (8.2%)	4,156 (8.7%)	4,156 (8.7%)	4,458 (9.2%)	4,458 (9.2%)	4,458 (9.2%)	3,640 (7.7%)
Unknown	2,463 (5.2%)	2,505 (5.3%)	2,505 (5.3%)	2,518 (5.2%)	2,518 (5.2%)	2,518 (5.2%)	2,456 (5.2%)
Number of remaining teeth							
28–32	33,515 (71.3%)	33,657 (70.6%)	33,657 (70.6%)	33,823 (70.1%)	33,823 (70.1%)	33,823 (70.1%)	33,307 (70.7%)
24–27	9,173 (19.5%)	9,378 (19.7%)	9,378 (19.7%)	9,522 (19.7%)	9,522 (19.7%)	9,522 (19.7%)	9,267 (19.7%)
0–23	4,319 (9.2%)	4,646 (9.7%)	4,646 (9.7%)	4,878 (10.1%)	4,878 (10.1%)	4,878 (10.1%)	4,522 (9.6%)
Periodontitis (CPI 3–4)	10,637 (22.6%)	11,042 (23.2%)	11,042 (23.2%)	11,225 (23.3%)	11,225 (23.3%)	11,225 (23.3%)	10,798 (22.9%)
Any active caries	7,842 (16.7%)	8,060 (16.9%)	8,060 (16.9%)	8,147 (16.9%)	8,147 (16.9%)	8,147 (16.9%)	7,860 (16.7%)
Apical periodontitis	34,733 (73.9%)	35,310 (74.1%)	35,310 (74.1%)	35,740 (74.1%)	35,740 (74.1%)	35,740 (74.1%)	34,847 (74.0%)

Participant numbers reported for each outcome are restricted to individuals without the condition at baseline. Age categories are non-overlapping, with intervals denoted as (29,40] indicating ages greater than 29 and less than or equal to 40 years (lower bound excluded, upper bound included).2.

Table 2. Incident case counts, incidence rates per 10,000 person-years (including 95% confidence intervals), and both crude and fully adjusted incidence rate ratios (IRR) with their corresponding 95% confidence intervals.

Outcome	Oral health variable	Category	Events (n)	Incidence rate per 10,000 PY (95% CI)	Crude IRR (95% CI)	Adjusted IRR* (95% CI)
Diabetes	Number of teeth	28–32 (reference)	1,412	42.37 (40.19–44.64)	1.00 (reference)	1.00 (reference)
		24–27	650	74.17 (68.57–80.09)	1.75 (1.60–1.92)	**1.21 (1.09–1.33)**
		0–23	471	139.88 (127.53–153.10)	3.30 (2.97–3.66)	**1.40 (1.22–1.60)**
	Periodontitis	No (reference)	1,702	50.06 (47.71–52.50)	1.00 (reference)	1.00 (reference)
		Yes	831	72.55 (67.70–77.65)	1.45 (1.33–1.57)	**1.10 (1.01–1.20)**
	Caries	No (reference)	1,949	51.39 (49.13–53.72)	1.00 (reference)	1.00 (reference)
		Yes	584	77.60 (71.44–84.16)	1.51 (1.38–1.66)	**1.12 (1.01–1.23)**
	Apical periodontitis	No (reference)	478	40.48 (36.93–44.28)	1.00 (reference)	1.00 (reference)
		Yes	2,055	61.09 (58.48–63.79)	1.51 (1.37–1.67)	**1.16 (1.04–1.30)**
Connective tissue diseases	Number of teeth	28–32 (reference)	338	9.97 (8.94–11.10)	1.00 (reference)	1.00 (reference)
		24–27	129	14.07 (11.75–16.72)	1.41 (1.15–1.73)	1.21 (0.97–1.50)
		0–23	43	11.36 (8.22–15.30)	1.14 (0.83–1.56)	1.00 (0.68–1.47)
	Periodontitis	No (reference)	372	10.67 (9.61–11.81)	1.00 (reference)	1.00 (reference)
		Yes	138	11.52 (9.68–13.61)	1.08 (0.89–1.31)	1.09 (0.88–1.34)
	Caries	No (reference)	419	10.77 (9.76–11.85)	1.00 (reference)	1.00 (reference)
		Yes	91	11.48 (9.24–14.09)	1.07 (0.85–1.34)	0.97 (0.76–1.24)
	Apical periodontitis	No (reference)	119	9.89 (8.19–11.83)	1.00 (reference)	1.00 (reference)
		Yes	391	11.23 (10.15–12.40)	1.14 (0.93–1.40)	0.93 (0.73–1.18)
Seropositive rheumatoid arthritis	Number of teeth	28–32 (reference)	67	1.97 (1.53–2.50)	1.00 (reference)	1.00 (reference)
		24–27	29	3.15 (2.11–4.52)	1.60 (1.03–2.47)	1.30 (0.81–2.07)
		0–23	16	4.21 (2.41–6.84)	2.14 (1.24–3.69)	1.78 (0.89–3.57)
	Periodontitis	No (reference)	81	2.31 (1.84–2.88)	1.00 (reference)	1.00 (reference)
		Yes	31	2.58 (1.75–3.66)	1.11 (0.74–1.68)	1.03 (0.66–1.59)

	Caries	No (reference)	91	2.33 (1.88–2.86)	1.00 (reference)	1.00 (reference)
		Yes	21	2.64 (1.63–4.03)	1.13 (0.70–1.82)	1.01 (0.61–1.68)
	Apical periodontitis	No (reference)	29	2.40 (1.61–3.45)	1.00 (reference)	1.00 (reference)
		Yes	83	2.37 (1.89–2.94)	0.99 (0.65–1.51)	0.77 (0.47–1.27)
Ulcerative colitis or Crohn's disease	Number of teeth	28–32 (reference)	141	4.13 (3.48–4.87)	1.00 (reference)	1.00 (reference)
		24–27	36	3.85 (2.70–5.33)	0.93 (0.65–1.35)	1.18 (0.80–1.74)
		0–23	11	2.76 (1.38–4.94)	0.67 (0.36–1.23)	1.35 (0.63–2.90)
	Periodontitis	No (reference)	137	3.88 (3.26–4.59)	1.00 (reference)	1.00 (reference)
		Yes	51	4.19 (3.12–5.51)	1.08 (0.78–1.49)	1.36 (0.97–1.91)
	Caries	No (reference)	159	4.03 (3.43–4.71)	1.00 (reference)	1.00 (reference)
		Yes	29	3.61 (2.41–5.18)	0.89 (0.60–1.33)	0.95 (0.62–1.45)
	Apical periodontitis	No (reference)	52	4.27 (3.19–5.60)	1.00 (reference)	1.00 (reference)
		Yes	136	3.85 (3.23–4.56)	0.90 (0.65–1.24)	0.89 (0.61–1.31)
Crohn's disease	Number of teeth	28–32 (reference)	28	0.82 (0.54–1.18)	1.Concurrent (reference)	1.00 (reference)
		24–27	9	0.96 (0.44–1.83)	1.17 (0.55–2.49)	1.52 (0.68–3.42)
		0–23	1	0.25 (0.01–1.40)	0.31 (0.04–2.25)	0.59 (0.06–5.32)
Ulcerative colitis	Number of teeth	28–32 (reference)	113	3.31 (2.73–3.98)	1.00 (reference)	1.00 (reference)
		24–27	27	2.89 (1.90–4.20)	0.87 (0.57–1.33)	1.10 (0.70–1.71)
		0–23	10	2.51 (1.20–4.61)	0.76 (0.40–1.45)	1.53 (0.67–3.48)
	Periodontitis	No (reference)	108	3.06 (2.51–3.69)	1.00 (reference)	1.00 (reference)
		Yes	42	3.45 (2.49–4.67)	1.13 (0.79–1.61)	1.42 (0.98–2.07)
Severe psychosis	**Number of teeth**	**28–32 (ref)**	**28**	**0.82 (0.54–1.18)**	**1.00 (ref)**	**1.00 (ref)**
		24–27	9	0.96 (0.44–1.83)	1.17 (0.55–2.49)	1.15 (0.93–1.41)
		0–23	1	0.25 (0.01–1.40)	0.31 (0.04–2.25)	0.86 (0.60–1.21)
	Periodontitis	No (ref)	29	0.82 (0.55–1.18)	1.00 (ref)	1.00 (ref)
		Yes	9	0.74 (0.34–1.40)	0.90 (0.43–1.90)	1.04 (0.86–1.25)
	Caries	No (ref)	34	0.86 (0.60–1.20)	1.00 (ref)	1.00 (ref)
		Yes	4	0.50 (0.14–1.27)	0.58 (0.20–1.63)	1.12 (0.91–1.38)

	Apical periodontitis	No (ref)	14	1.15 (0.63–1.93)	1.00 (ref)	1.00 (ref)
		Yes	24	0.68 (0.43–1.01)	0.59 (0.31–1.14)	1.19 (0.95–1.48)

Adjusted using Poisson regression for age, sex, usage of statins in baseline, socioeconomic status, D-index, CPI and i-index.

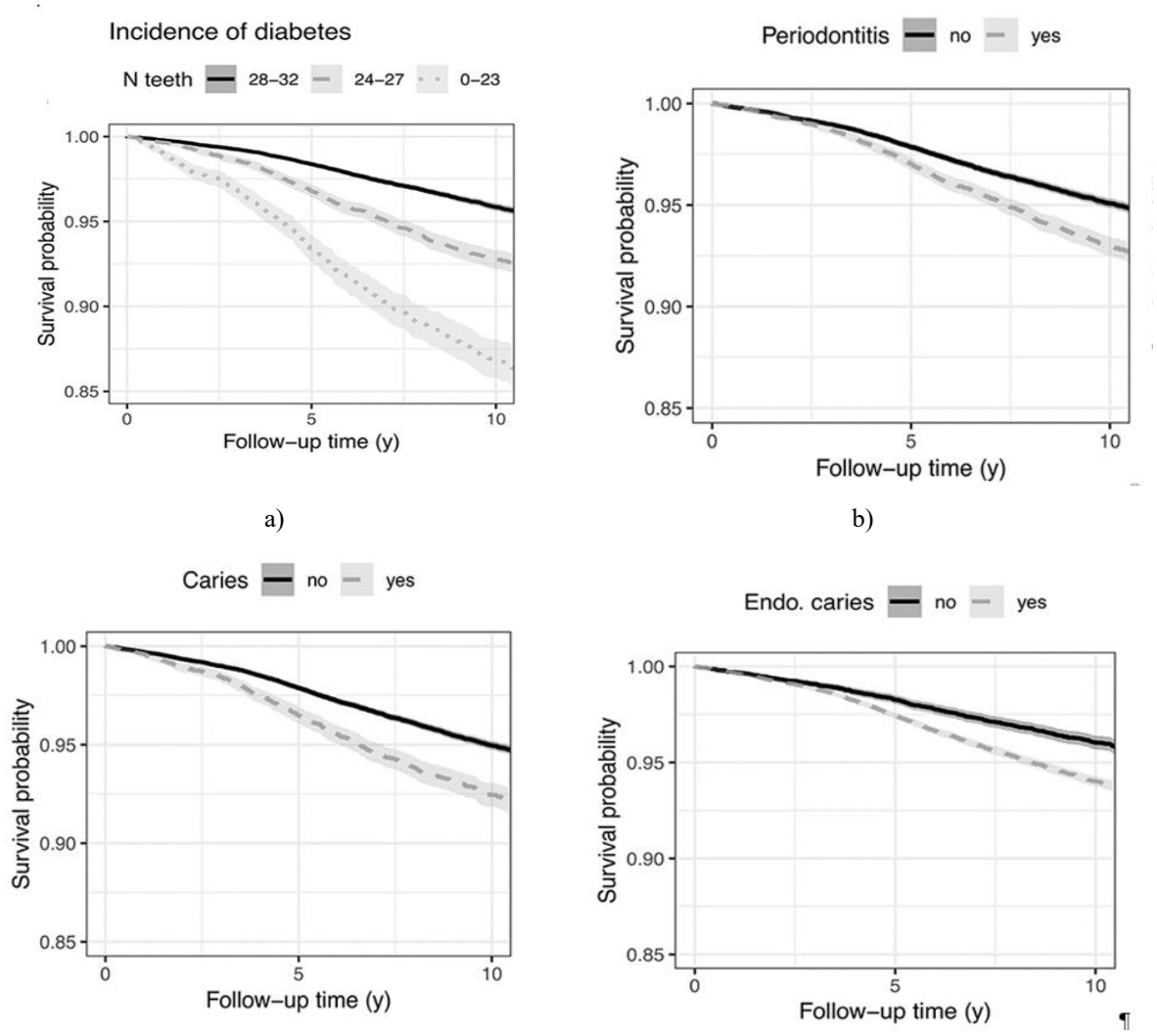

Figure 2. Cumulative incidence of diabetes by oral health status. Kaplan–Meier curves (with 95% confidence bands) stratified by: (a) number of remaining teeth, (b) presence of periodontitis, (c) presence of caries, (d) presence of apical periodontitis (endo/caries).

we estimated the one-year number needed to harm (NNH) for incident diabetes associated with three oral health indicators **(Table 3)**.

Using an additive Poisson model, we derived the 1-year number needed to harm (NNH) for incident type 2 diabetes linked to three measures of poor oral health **(Table 3)**. Periodontitis: NNH = 1,736 (95% CI 903 to 22,103) Dental caries: NNH = 1,342 (95% CI 709 to 12,497) Edentulism (<24 remaining teeth vs ≥28 teeth): NNH = 262 (95% CI 190 to 420).

Table 3. One-year diabetes incidence rates and corresponding numbers needed to harm (NNH) estimated via additive Poisson regression.

Characteristic	Category	NNH (95% Confidence Interval)
Number of teeth	28–32 (≥28 teeth)	Reference
	24–27	1,004 (625 – 2,561)
	0–23	262 (190 – 420)
Clinical periodontitis	No	Reference

	Yes	1,736 (903 – 22,103)
Dental caries	No	Reference
	Yes	1,342 (709 – 12,497)
Apical periodontitis	No	Reference
	Yes	2,052 (1,029 – 349,261)

Adjusted for i-index, gender, socioeconomic status, age, D-index, usage of statins in baseline and CPI.

We analyzed data from a predominantly representative population (N = 68,273) in an observational registry study with a long-term follow-up of 10 years. Incidence was determined based on the initiation of drug treatments for specific conditions, as confirmed through special reimbursement records. Our primary finding was that oral health indicators were associated with diabetes, but not with other chronic diseases. These results highlight a strong link between oral health—particularly periodontal disease—and the metabolic deterioration of glucose regulation.

Strengths and limitations of this study

The study included only individuals who had at least one dental visit in the public dental services of the City of Helsinki during a two-year period. Consequently, people who did not visit public dental care at all, or who used only private dental services, were excluded. During the study period, approximately 36% of all dental care in Finland was provided by the private sector. Since private services are more frequently utilized by higher socioeconomic groups, this exclusion may have shifted the study population slightly toward lower socioeconomic groups. However, because socioeconomic status (SES) data were available for the entire cohort, we were able to confirm that individuals from higher SES groups also used public dental services to a considerable extent **(Table 1)**.

Potential confounding factors such as age, sex, and socioeconomic status were available for all participants and were adjusted for in the analyses. A notable limitation was the absence of data on smoking, alcohol consumption, and dietary habits, all of which are established risk factors for chronic diseases and could potentially confound the observed associations [30]. In our earlier publication, we observed a strong link between baseline periodontitis and subsequent fatal pancreatic cancer, yet no association was found with lung cancer, suggesting that residual confounding by smoking is likely minimal [32]. Nevertheless, future studies with more comprehensive measurement of these behavioral confounders are needed to confirm the present findings.

Diabetes diagnoses were identified through entitlement to special reimbursement for antidiabetic medications, a system that effectively captures nearly all patients receiving pharmacological treatment, as medication is typically initiated at the time of diagnosis [33]. However, individuals with undiagnosed diabetes or those managed solely through lifestyle interventions (without medication) were not identified, and HbA1c values were not available from the registers. This limitation would, if anything, tend to attenuate the observed associations rather than inflate them.

We additionally considered statin use, as a small number of studies have linked statins to a modestly elevated risk of new-onset diabetes. Nevertheless, this risk remains very low in absolute terms and is far outweighed by the substantial reduction in cardiovascular events. In patients with intermediate-to-high cardiovascular risk or established cardiovascular disease, the benefits of statin therapy are firmly established [34]. Beyond lipid lowering, statins exert multiple pleiotropic actions, including anti-inflammatory, immunomodulatory, antioxidant, antithrombotic, endothelium-protective, and pro-angiogenic effects [35].

Several retrospective analyses have shown that individuals with chronic periodontitis who received simvastatin or atorvastatin exhibited significantly better periodontal indices than untreated patients [36, 37]. Atorvastatin has also been reported to improve endothelium-dependent vasodilation in normocholesterolemic smokers independently of lipid changes [38, 39], and recent data indicate statins provide periodontal benefits even in smokers [40].

In the current cohort, statin users displayed a markedly higher incidence rate ratio for diabetes (IRR 2.49, 95% CI 2.10–2.94). This association, however, must be interpreted cautiously because of possible "**Table 2** fallacy" (i.e., over-interpretation of covariates that are not the primary exposure of interest) [41]. The major potential confounders linking chronic oral infections to diabetes include smoking, alcohol consumption, socioeconomic position, age, sex, genetic predisposition, and dietary habits. In the present study, comprehensive adjustment was possible for key sociodemographic variables—age, sex, and socioeconomic status—which were available for the entire cohort.

Outcomes were defined as the initiation of special reimbursement for antidiabetic medication, meaning the clinical diagnosis of diabetes had occurred some time earlier. The time lag between diagnosis and

reimbursement entitlement varies between individuals and may introduce bias if oral health status influences this lag. To minimize this, follow-up began two years after the first recorded dental visit. Diabetes type could only be inferred from treatment: patients treated solely with insulin include both type 1 diabetes cases and long-standing type 2 patients requiring insulin. Distinguishing diabetes subtypes in adults can sometimes be clinically ambiguous. Patients managed only with lifestyle measures were not captured, but Finnish guidelines in place at the time (first published 2007 and subsequently updated) strongly recommended starting metformin alongside lifestyle intervention when indicated [42]. Previous work has shown excellent adherence to these guidelines [43], so nearly all patients with a clinically confirmed diabetes diagnosis were included, making the cohort highly representative.

A key strength of the study lies in the detailed clinical oral health data derived from actual treatment procedure codes, resulting in very low rates of false-positive diagnoses of periodontitis, caries, and apical periodontitis. In contrast, many epidemiological studies suffer from lack of standardized criteria for defining periodontal disease, complicating comparisons across studies [44]. We used number of remaining teeth as a robust cumulative marker of lifelong oral inflammation; tooth loss in adults is predominantly caused by periodontitis or severe caries [45], although some older smaller studies reported caries as the main reason [46, 47]. Severe caries can progress to pulp necrosis and apical periodontitis, thereby sustaining chronic inflammation [48].

The three tooth-number categories (28–32, 24–27, and 0–23) were chosen pragmatically due to the absence of universal consensus. Although the underlying causes of tooth loss overlap between categories, this grouping aids interpretation: 28–32 teeth generally indicates periodontally healthy individuals or removal of third molars only; 24–27 teeth may reflect additional loss due to orthodontic treatment, moderate periodontitis, or caries; whereas 0–23 teeth most likely represents advanced chronic periodontitis or severe caries with substantial cumulative inflammatory burden [45].

Exposure assessment combined procedure codes, number of remaining teeth, oral health indices, initial caries lesions, DMFT scores, and periodontal treatment need based on pocket depth recordings. Periodontitis was defined dichotomously (yes/no) according to registered periodontitis treatment codes during 2001–2002, the period when baseline oral health status was recorded. Overall, the data consistently support associations between periodontitis, dental caries, apical periodontitis, and incident drug-treated diabetes.

Comparisons with other studies

A substantial body of research has explored the biological links between periodontitis and diabetes, with most work concentrating on how diabetes influences periodontal disease mechanisms. Evidence supports a bidirectional relationship in which each condition can amplify the other. Immune dysregulation plays a central role in diabetes and its complications, and systemic shifts in cytokines and matrix metalloproteinases (MMPs) contribute to the development of type 2 diabetes. These alterations are connected to physiological, metabolic, and nutritional factors such as hyperglycemia, accumulation of advanced glycation end products (AGEs), elevated lipid levels, and increased adiposity [44, 49]. Such mechanisms can impair host immune responses and negatively affect periodontal health. Persistent hyperglycemia leads to protein glycation and eventual formation of AGEs. These irreversible modifications have multiple consequences, including disrupted immune regulation and heightened, prolonged inflammation due to impaired resolution of immune responses [44, 49]. These processes contribute to the pathophysiology of periodontitis in individuals with diabetes by interfering with normal tissue healing and repair. When AGEs bind to their cellular receptors, this triggers the release of reactive oxygen species, proinflammatory cytokines, and MMPs. These molecules intensify inflammation and accelerate tissue destruction in the periodontium through an exaggerated immune response and compromised regenerative capacity [50].

In contrast to the extensive research on diabetes-driven effects, limited biological evidence exists on how periodontitis may influence diabetes. Diabetes does not appear to significantly alter periodontal microbial composition, and only weak evidence suggests an effect on glycemic control. However, systemic inflammation originating from periodontitis may disrupt glucose regulation by elevating circulating inflammatory mediators—including tumor necrosis factor-alpha, interleukin-6, MMPs, reactive oxygen species, and acute-phase proteins—which impair insulin signaling, hinder receptor activity, and reduce cellular glucose uptake. In severe periodontitis, these mechanisms may raise serum glucose levels over time, even in individuals without diabetes. Notably, MMP-8 can degrade insulin receptors [11]. In people with diabetes who also have untreated severe periodontitis, optimal glycemic control becomes more difficult, and

the likelihood of diabetes-related complications rises [51].

Several earlier studies have suggested that periodontal treatment may reduce the risk of systemic diseases. Clinical trials have reported that periodontal therapy can slow or alter the progression of systemic conditions, although these studies generally suffered from small sample sizes, varied outcome measures, and short follow-up periods [52, 53]. In a review by Sabharwal *et al.* most of the 23 randomized controlled trials examined showed moderate and consistent improvements in glycemic control in individuals with type 2 diabetes following periodontal treatment. By reducing inflammatory mediators and MMPs in circulation, periodontal therapy may benefit both oral health and systemic metabolic control, contributing to lower average glucose levels and improved lipid profiles [52]. A recent study by D'Aiuto *et al*. also demonstrated that intensive periodontal therapy resulted in a 0.6% reduction in HbA1c after 12 months among 264 participants with type 2 diabetes and moderate-to-severe periodontitis [13].

The present study includes a large and unselected population with representative groups of patients affected by periodontitis and apical periodontitis, followed for a period of 10 years. Although the study design carries inherent limitations, the findings are likely applicable to similar populations with chronic oral conditions. The demonstrated association between chronic oral diseases and diabetes highlights the importance of coordinated care between healthcare providers, particularly collaboration between dental and medical professionals.

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Conflict of Interest: None

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