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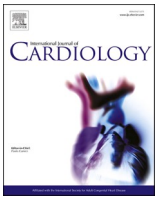

Childhood oral health is associated with the incidence of atherosclerotic cardiovascular disease in adulthood

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ABSTRACT

Background: While adult oral health has been consistently linked to cardiovascular disease, the long-term impact of childhood oral health remains underexplored. Thus, we investigated the association between dental caries and gingivitis in childhood and the incidence of atherosclerotic cardiovascular disease (ASCVD) in adulthood.
Methods: This nationwide Danish cohort study included 568,778 individuals born between 1963 and 1972, with oral health data from the National Child Odontology Registry (1972–1987) and ASCVD outcomes from the National Patient Register (1995–2018). Dental caries and gingivitis were categorized by severity and trajectory across childhood. Cox regression models stratified by sex, education level, and type 2 diabetes status were used to estimate hazard ratios (HRs) and 95 % confidence intervals (CI) for incident ischemic heart disease, myocardial infarction, and ischemic stroke.
Results: Severe childhood dental caries was associated with increased ASCVD incidence in both males (HR 1.32; 95 % CI: 1.18–1.50) and females (HR 1.45; 95 % CI: 1.25–1.68). High gingivitis scores also predicted elevated ASCVD risk (males: HR 1.21; 95 % CI: 1.10–1.32; females: HR 1.31; 95 % CI: 1.14–1.50). Disease trajectories with moderate to severe level oral disease and oral health deterioration were significantly associated with higher ASCVD incidence.
Conclusion: Within the limitations of this study, poor childhood oral health, particularly persistent or worsening dental caries and gingivitis, is associated with an increased risk of ASCVD in adulthood. These findings highlight the potential of early oral health interventions in reducing long-term cardiovascular risk.

1. Introduction

Cardiovascular diseases are amongst the greatest contributors to the global burden of disease [1]. Atherosclerotic cardiovascular diseases (ASCVD), namely Ischemic heart disease (IHD), Myocardial infarction (MI) and Ischemic stroke (IS) are some of the most common causes of premature death from cardiovascular disease [2]. Prevention strategies tackling risk factors in the earlies stages of life (childhood and young adulthood) could reduce the risk of ASCVD in middle age [3]. Thus, identifying common and novel risk factors of cardiometabolic disease viable for early intervention already in childhood could be instrumental in preventing ASCVD.

Oral health is often overlooked in children although its effect on nutritional/behavioural and physical status later in life is well documented [4]. Oral inflammatory conditions such as periodontitis and gingivitis, and dental caries (DC) are amongst the most prevalent diseases worldwide [5], not least in children. Data from the United States Center for Disease Control indicates that between 40 and 60 % of children aged 6 to 9 have had dental caries in either their temporary or permanent dentition [6]. Periodontitis on the other hand is much less common in children, with an estimated prevalence of 0.2–0.5 % [7,8] whilst gingivitis, which could be considered its precursor, instead is one of the most common forms of human inflammation [9].

Oral health and disease are increasingly being linked to systemic

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health through two major pathways: 1) by contributing to systemic low-grade inflammation and 2) bacterial dissemination (i.e. the translocation of bacteria from the oral cavity to the rest of the body) [10–12]. Several studies have found oral disease in adults, periodontal disease in particular, to be associated with an increased risk of a range of cardiovascular outcomes [13]. Few studies have examined the long-term effects of oral disease in children on cardiovascular health. One longitudinal study from Finland on a cohort of 755 individuals followed from childhood to age 35 found signs of oral infection in childhood, measured by prevalent dental caries and periodontal disease, to be associated with increased blood pressure and an increased thickness of the intima media in early adulthood [14]. Another study based on the National Longitudinal Study of Adolescent to Adult Health, followed 11,556 individuals from between ages 12 to 19 to between ages 26 and 32. This study found no significant effect of periodontal disease and tooth loss from dental caries on cardiometabolic risk. It did find deferred dental care during adolescence to be to be associated with increased BMI and blood pressure values, though the magnitude of the latter was judged unlikely to have any clinical significance. A major flaw of this study was its questionnaire based assessment of oral health measures, asking whether the participant had had "gum disease or tooth loss because of cavities in the past 4 weeks" [15], likely resulting in a severe underestimation of the rate of dental disease in the study population. As such, further study of the association between childhood oral health and adulthood cardiovascular disease is warranted.

We hypothesize that childhood dental caries (DC) and gingivitis are associated with incident ASCVD in adulthood, measured by the occurrence of IHD, MI, and/or IS. We expect this association to follow a dose-response pattern, such that higher levels of DC or gingivitis are linked to a higher incidence of ASCVD. Furthermore, we hypothesize that the trajectory of DC and gingivitis, across childhood is associated with the incidence of ASCVD in adulthood. Poor or declining oral health throughout childhood may reflect persistent biological mechanisms or low health literacy, both of which could increase ASCVD risk. Therefore, we expect that individuals with poor or declining oral health trajectories will have a higher incidence of ASCVD in adulthood compared to those with good or improving oral health trajectories.

2. Methods

This nationwide study used Danish registry data with follow-up from January 1st, 1995 to December 31st, 2018. Individuals were followed from January 1st, 1995, or from age 30 (whichever occurred last) and until the first event (receiving an ASCVD-diagnosis as a primary-diagnosis for the first time), censoring (death or disappearance), or end of follow-up.

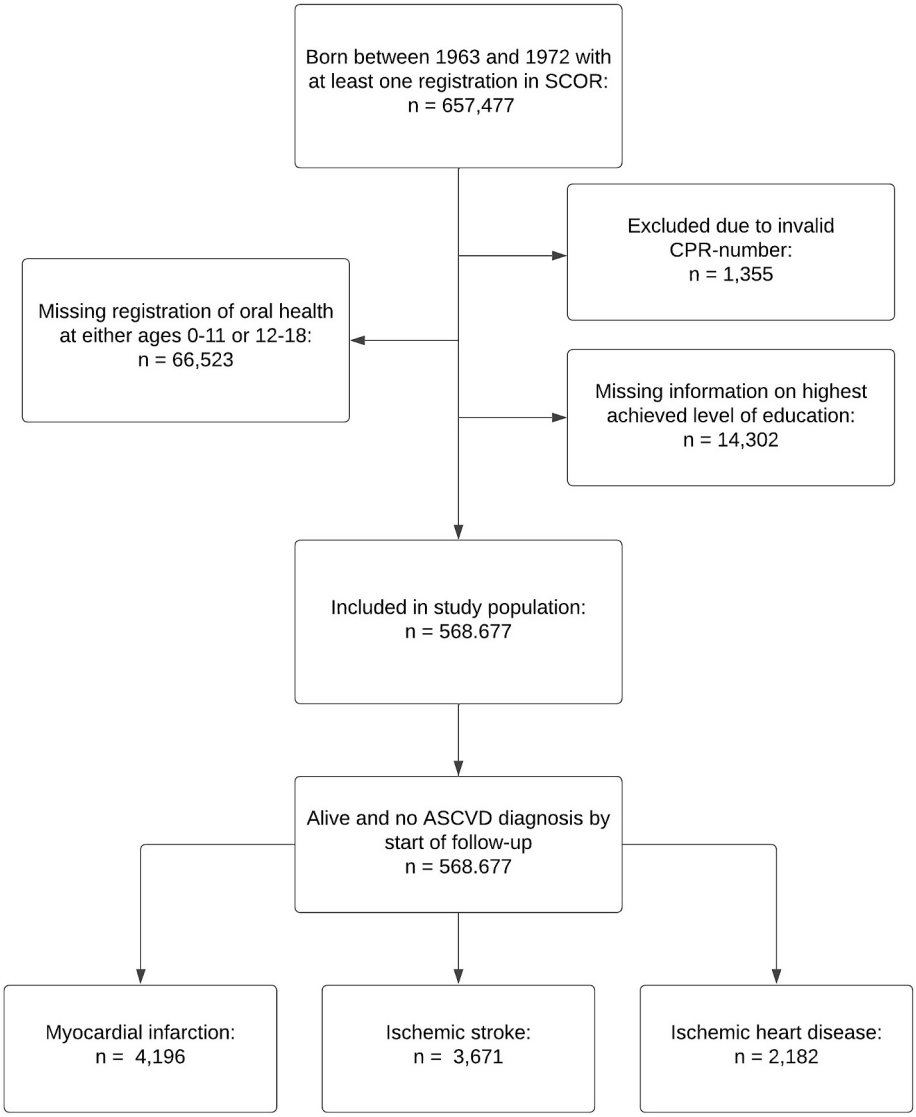

Fig. 1. Selection into the study.

2.1. Inclusion criteria

Participants had to be born between 1963 and 1972, have no ASCVD-diagnoses before baseline, at least one registration of their oral health status before and after the age of 12, information on their highest achieved level of education between age 25 and 30, possess a valid code in the central person registry, and still be alive and residing in Denmark at baseline. Individuals with missing data in any of these variables were excluded from the study (Fig. 1).

2.2. Registries

Data on childhood oral health was collected from the National Child Odontology Registry (SCOR) using registrations from the years 1972–1987. Data on the development of ASCVD during follow-up (1995–2018) was collected from the National Patient Register (LPR). Outcomes of interest were IHD (ICD10: I25/I251/I258/I259), MI (ICD10: I21-I22) and IS (ICD10: I63/I64). From the Central Person Register we gathered data on status (dead, alive, disappeared, or emigrated) in the years 1967–2018, and the highest achieved level of education for each study participant using data from 1981 to 2018. Data was linked across registries using the Danish personal identification number assigned to each individual at birth or immigration.

2.3. Ethical considerations

The use of registry data was approved by the Danish Data Protection Agency via the Office of Research and Innovation, University of Copenhagen (case number 514–0496/20–300), and licensed by the Danish Health Data Authority. All data was anonymized by the Danish Health Data Authority, and all methods carried out in accordance with relevant guidelines and regulations. According to guidelines from the Danish National Committee on Health Research Ethics, registry-based studies do not require ethical approval nor informed consent from the study population.

2.4. Variables

The outcome variable for this study was ASCVD defined as the first registered event of IHD, MI or IS.

DC was measured as the number of decayed, missing, and filled teeth (dmft/DMFT). The scoring system used for the registration of DC in SCOR has been described in detail previously [7]. Based on odontological considerations the highest registered dmft/DMFT in any dentition at any point in time was grouped into dental caries score (DCS):

- Low: 0–4 affected teeth
- Moderate: 5–12 affected teeth
- High: 13–16 affected teeth
- Severe: ≥17 affected teeth

Gingivitis was measured on four index-teeth, and graded using the Silness and Löe gingival index [16]. Summing these provided an index-score for the severity of gingivitis at each visit to the dentist ranging from 0 to 12 [7]. Based on odontological considerations, the highest registered index-score at any visit for each individual the variable gingivitis score (GS) was grouped by:

- Low: Score of 0–4
- Moderate: Score of 5–8
- High: Score of 9–12

Disease trajectories were created based on the highest registered level of DC and gingivitis before and after the age of 12 for each individual and classified as (Supplementary Fig. 1):

Low disease level:

- Stable: DMFT / gingivitis index-score below age 12 same as after age 12 and DCS / GS ≤ Low
- Improving or worsening: DMFT / gingivitis index-score differ before and after age 12 and DCS / GS t ≤ Low

Moderate to severe disease level:

- Stable: DMFT / gingivitis index-score the same before and after age 12 and at least one low DCS / GS > Low
- Improving: DMFT / gingivitis index-score higher before age 12 than after age 12 and at least one DCS / GS > Low
- Worsening: DMFT / gingivitis index-score lower before age 12 than after age 12 and at least one DCS / GS > Low

Educational length was grouped by: Short) mandatory schooling from ages 6–16. Medium) secondary school and vocational education. Long) higher education of any duration.

2.5. Statistical methods

Population characteristics at baseline were described using means and standard deviations for continuous variables, and frequencies and percentages for categorical variables. Hazard ratios (HRs) and 95 % confidence intervals (CIs) for association between childhood oral health and adulthood ASCVD, as well as IHD, MI and IS, were generated using Cox-regression analysis with age during follow-up as the underlying time variable for predefined sex-specific models. Models were stratified by sex, as we hypothesized that the protective effect of oestrogen in females could be a mediator in the associations between oral health and ASCVD. The highest achieved level of education between ages 25–30 for each individual and prevalent type 2 diabetes was used as Cox-strata in all models. Cox-strata was used to satisfy the proportional hazards assumption while accounting for potential confounding from the variables. Models with interaction terms between sex and the oral exposures were created, but the interactions terms were not statistically significant. The assumptions underlying the Cox-model were evaluated by plotting Schoenfeld residuals, log-log curves, and cumulative hazards. No substantial violations of model assumptions were identified. Sensitivity analyses were done for the caries trajectory analyses using active DC cases rather than DMFT. Sensitivity analyses including adulthood income level in the models did not substantially alter the produced effect estimates.

Analyses were run in R v.4.4.1 with Rstudio (IDE version 2024.04.2 + 764 using the packages *prodlim* [17], *survival* [18], *timereg* [19], and *mets* [20]. Reporting followed the STROBE guidelines for cohort-studies.

3. Results

3.1. Study population

The study included 568,778 individuals (291,058 males; 277,720 females). During follow-up 10,049 males and 5705 females developed ASCVD. In males, MI was the most frequent ASCVD-diagnosis (41.8 %), while IS predominated in females (54.3 %). Most participants, regardless of ASCVD status and sex, had moderate levels of dental caries (up to 68 %) and gingivitis (57–68 %) in childhood (Table 1, for visualizations see Supplementary Figs. 2–4).

3.2. Association of Childhood DC and GS with adulthood incidence of ASCVD

Males with a high DCS had a 32 % (hazard ratio = 1.32, 95 % confidence interval [1.18;1.50]) higher incidence of ASCVD compared to those with low DCS. Similarly, males with a high GS had a 21 % (1.21 [1.10;1.32]) higher incidence of ASCVD compared to those with a low GS. In females a high DCS compared to a low DCS was associated with a 45 % (1.45 [1.25;1.68]) higher incidence of ASCVD, while a high GS was associated with a 31 % (1.31 [1.14; 1.50]) higher incidence of ASCVD

Table 1
Study population characteristics.

	Males		Females	
N study participants (%)	Total 291,000	ASCVD 10,049 (3.5)	Total 277,677	ASCVD 5705 (2.1)
Mean age at baseline (SD)	30.1 (0.2)	30.0 (0.2)	30.0 (0.2)	30.0 (0.2)
Mean age at end of follow-up (SD)	49.4 (3.7)	44.4 (5.3)	49.7 (3.3)	43.6 (5.4)
Mean time-on-study (SD)	19.4 (3.7)	14.3 (5.3)	19.7 (3.3)	13.6 (5.4)
N (%) with diagnosis				
IHD	–	2182 (21.7)	–	1112 (18.5)
MI	–	4196 (41.8)	–	1552 (27.2)
IS	–	3671 (36.5)	–	3041 (54.3)
N (%) with DCS				
Low	42,534 (14.6)	1031 (10.3)	36,718 (13.2)	489 (8.6)
Moderate	194,008 (66.7)	6749 (67.2)	188,876 (68.0)	3807 (66.7)
High	45,417 (15.6)	1823 (18.1)	43,181 (15.6)	1127 (19.8)
Severe	9041 (3.1)	446 (4.4)	8902 (3.2)	282 (4.9)
N (%) with GS				
Low	85,857 (29.5)	2601 (25.9)	111,404 (40.1)	1986 (34.8)
Moderate	190,892 (65.6)	6801 (67.7)	158,834 (57.2)	3486 (61.1)
High	14,251 (4.9)	647 (6.4)	7439 (2.7)	233 (4.1)
N (%) with caries trajectory				
Stable, low disease level	17,101 (5.9)	398 (4.0)	14,684 (5.3)	179 (3.1)
Improving or worsening, low disease level	25,433 (8.7)	633 (6.3)	22,034 (7.9)	310 (5.4)
Improving, moderate to severe disease level	167,051 (57.4)	5708 (55.8)	159,005 (57.3)	3164 (55.5)
Worsening, moderate to severe disease level	57,367 (19.7)	2414 (24.0)	60,097 (21.6)	1544 (27.1)
Stable, moderate to severe disease level	24,048 (8.3)	896 (8.9)	21,857 (7.9)	508 (8.9)
Mean years between registrations (SD)	4.7 (2.1)	4.8 (2.2)	4.9 (2.1)	5.0 (2.2)
N (%) with gingivitis trajectory				
Stable, low disease level	38,721 (13.3)	1173 (11.7)	45,341 (16.3)	808 (14.2)
Improving or worsening, low disease level	47,136 (16.2)	1428 (14.2)	66,063 (23.8)	1178 (20.6)
Improving, moderate to severe disease level	100,334 (34.5)	3397 (33.8)	100,779 (36.3)	2029 (35.6)
Worsening, moderate to severe disease level	72,776 (25.0)	2855 (28.4)	45,528 (16.4)	1191 (20.9)
Stable, moderate to severe disease level	32,033 (11.0)	1196 (11.9)	19,966 (7.2)	499 (8.7)
Mean years between registrations (SD)	4.3 (2.1)	4.3 (2.0)	4.4 (2.1)	4.4 (2.1)
N (%) with type 2 diabetes	10,130 (3.5)	651 (6.5)	6953 (2.5)	311 (5.5)

Abbreviations: DCS = dental caries score, GS = gingivitis score, IHD = ischemic heart disease, IS = ischemic stroke, MI = myocardial infarction, N = number, SD = standard deviation.

compared to a low GS (Fig. 2, Supplementary Table 1). No interactions were found between DCS and GS ($p > 0.05$) (Supplementary Table 2). Looking at the individual ASCVD diagnoses, the lowest HRs were found for IHD, while the HRs of MI and IS dependent on childhood oral health were similar to those found in the analyses of total ASCVD. While females had higher HRs for MI, and males for IS (Fig. 2, Supplementary table 3), interactions terms between the oral exposures and sex (not shown) were not found to be statistically significant.

3.3. The Association of Childhood DC and GS Trajectories with Adulthood Incidence of ASCVD

In males, a worsening trajectory of DC in childhood with moderate to severe disease was associated with a 26 % (1.26 [1.14; 1.41]) higher incidence of ASCVD in adulthood compared to males with a stable trajectory and low levels of DC, while a stable DC trajectory with moderate to severe disease was associated with a 21 % (1.21 [1.07; 1.36]) higher incidence of ASCVD compared to a stable trajectory with a low level of disease. In females a worsening DC trajectory with moderate to severe disease in childhood was associated with a 45 % (1.45 [1.24; 1.69]) higher incidence of ASCVD in adulthood and a stable DC trajectory with moderate to severe disease with a 41 % higher incidence of ASCVD (1.41 [1.18; 1.67]), compared to a stable trajectory with low levels of DC (Table 2, Supplementary Fig. 1).

In males, any gingivitis trajectory with at least one observed disease status being moderate to severe was associated with an up to 13 % (1.13 [1.04; 1.22]) higher incidence of ASCVD in adulthood compared to a stable trajectory with a low level of disease. In females a worsening gingivitis trajectory with moderate to severe disease was associated with a 27 % (1.27 [1.16;1.39]) higher incidence of ASCVD in adulthood, and a stable gingivitis trajectory with moderate to severe disease a 25 % (1.25 [1.12; 1.39]) higher incidence of ASCVD in adulthood (Table 2, Supplementary Fig. 1).

4. Discussion

In this study, we found that higher levels of dental caries and gingivitis in childhood were associated with an increased incidence of ASCVD in adulthood compared to low levels of disease. This supports our hypothesis that childhood oral health is linked to later cardiovascular risk. Likewise, our hypothesis regarding oral health trajectories was confirmed, although significant associations were observed only for trajectories involving moderate to severe disease. Altogether, these findings suggest that both disease severity and trajectory of childhood oral disease, may represent early-life risk-factors for the development of ASCVD.

Our findings align with the Finnish study referenced in the introduction, which found that poor childhood oral health was associated with higher blood pressure and an increased thickness of the intima media in adulthood [14]. That study found males with poor childhood oral health had a greater incidence of increased intima media thickness compared to females [14], whereas we observed a higher ASCVD incidence in females relative to childhood oral health. This corresponds with a previous study of ours showing that the incidence of type 2 diabetes associated with childhood DC and gingivitis was higher in females than males [21]. Females have been reported to have a higher cardiovascular risk from factors such as smoking, dietary habits [22,23], indicating that exposures to risk factors may affect the sexes differently. However, interaction terms between sex and our oral exposures showed no indication of effect-modification from sex. Thus, it is also possible that the observed sex differences in our study are due to underlying baseline hazards in males and females. Further research is needed to clarify whether sex-specific differences exist in the impact of childhood oral disease on systemic health and to explore potential mechanisms underlying such differences.

While our findings are based on Danish data, they point to the importance of prioritising childhood oral health internationally. Dental care is the greatest unmet health care need amongst children in the United States, with those from more disadvantaged households being disproportionally affected [24]. In the light of the presents findings this is particularly worrying, as it may put already disadvantaged children at further disadvantage by adding to their total risk of disease and disability later in life. Our models provide modest HRs for the association between oral health and ASCVD, but considering the high baseline

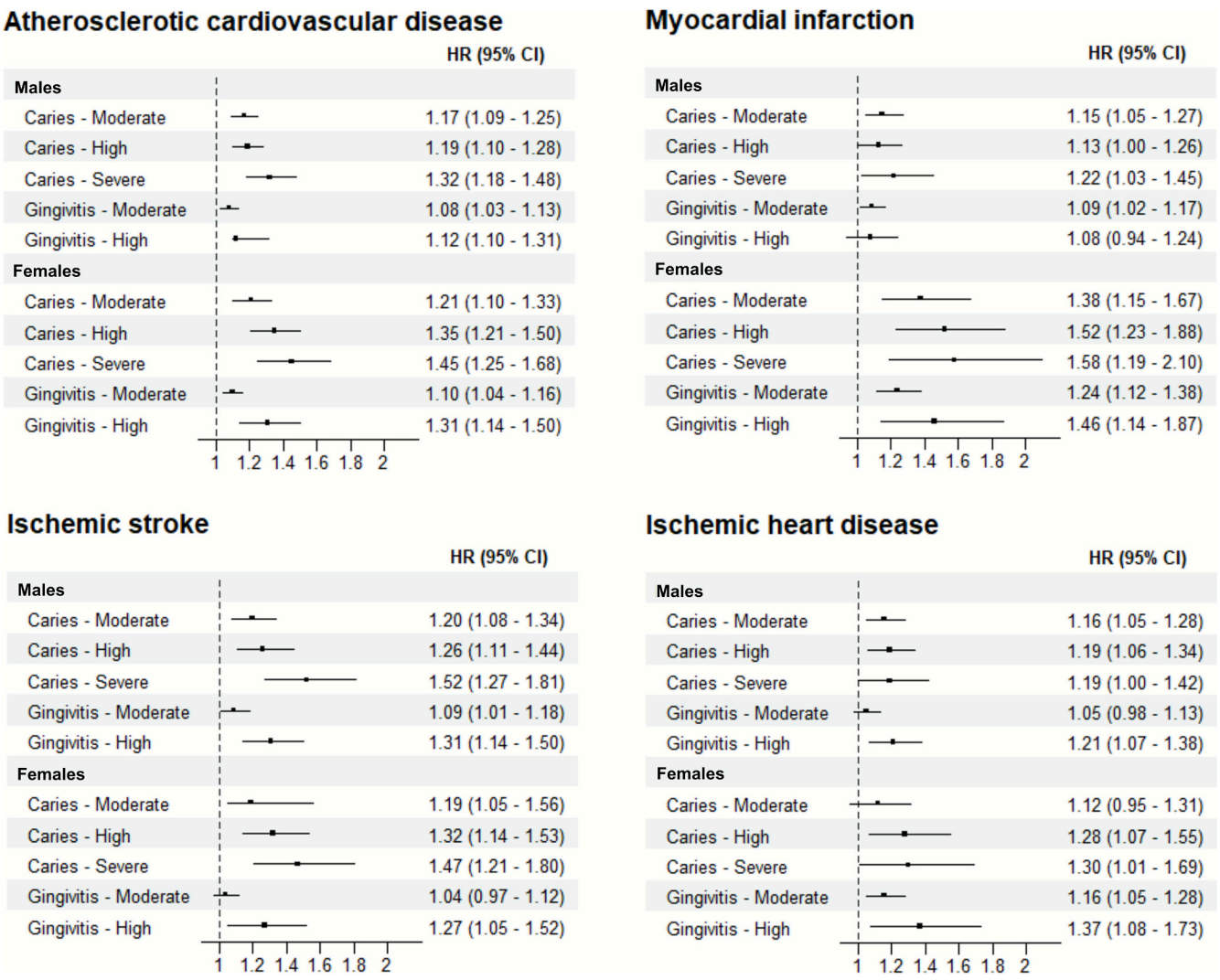

Fig. 2. Adjusted estimates of the association between oral health in childhood and the incidence of ASCVD, and separate diagnoses of IHD, MI and IS in adulthood. Using highest achieved level of education between ages 25 and 30 and prevalent type 2 diabetes as cox-strata. Abbreviations: ASCVD = atherosclerotic cardiovascular disease, CI = confidence interval, DCS = dental caries score, GS = gingivitis score, HR = hazard ratio.

incidence of ASCVD, even modest changes in modifiable risk-factors can substantially shift the disease burden at a population level, for better or for worse. Thus, even with our relatively small HRs, investing in childhood oral health may have downstream benefits far beyond the oral cavity, providing further impetus for governments and other stakeholders to invest more in childhood oral health.

Periodontal diseases are considered a biofilm-induced, inflammation-mediated diseases [25]. They encompass a wide range of disease states, from light gingival bleeding affecting a few teeth - typically associated with poor oral hygiene and biofilm accumulation - to severe, generalised, chronic bleeding. Progression from gingivitis to the more severe periodontitis depends on multiple factors including the host immune-response, and the host's microbial composition [25,26]. The inflammatory response in periodontal disease contribute to an increase in chronic low-grade inflammation, which has been suggested as a key linking oral and cardiovascular health, potentially increasing sensitivity to inflammatory exposures later in life [12]. Supporting this, periodontal treatment has been shown to reduce markers of systemic inflammation and blood pressure in hypertensive patients [27]. Given the inflammatory nature of gingivitis, we would expect the estimated HRs for the association of gingivitis with ASCVD to be higher than those of DC. However, we find DC to be associated with slightly higher HRs of ASCVD than gingivitis in this study. This may reflect indirect mediation through inflammation, as gingivitis often follows severe caries in both clinical and research settings [28].

DC is a localized chemical dissolution of the tooth surface driven by the metabolic activity of the biofilm communities colonizing the tooth surface [29]. It is thought to link to ASCVD through transference of oral bacteria to the systemic circulation, causing inflammation and contributing atherosclerotic plaque formation [30]. Changes in the composition, function and metabolic activities of the oral microbiome - key drivers of oral disease - have previously been associated with atherosclerosis and hypertension [11]. Oral bacteria may promote the formation of arterial biofilm through bacteraemia or the release of pro-inflammatory cytokines from oral inflammatory lesions into the circulatory system [11]. Human studies have identified 23 oral bacterial species in atherosclerotic plaques, some of which are members of the resident oral microbiota commonly associated with periodontitis and infected root canals [31,32]. Thus, poor oral health in childhood and resulting changes in the oral microbiome, could influence systemic disease risk including ASCVD [11]. However, it is beyond the scope of our data to confirm or reject these ideas. Future studies exploring these mechanisms further are needed.

Genetic factors may also contribute to the observed associations between oral health and ASCVD. Although the evidence is mixed, Shungin et al., in a large scale genome wide association study, identified

Table 2
The association between childhood oral health trajectories and the incidence of ASCVD in adulthood.

	Males		Females	
	HR (95 % CI)	p	HR (95 % CI)	p
Dental caries				
Stable, low disease level	Ref	–	Ref	–
Improving or worsening, low disease level	1.02 (0.90; 1.16)	0.72	1.12 (0.93; 1.34)	0.25
Improving, moderate to severe disease level	1.19 (1.08; 1.32)	<0.001	1.31 (1.13; 1.53)	<0.001
Worsening, moderate to severe disease level	1.26 (1.14; 1.41)	<0.001	1.45 (1.24; 1.69)	<0.001
Stable, moderate to severe disease level	1.21 (1.07; 1.36)	0.002	1.41 (1.18; 1.67)	<0.001
Gingivitis				
Stable, low disease level	Ref	–	Ref	–
Improving or worsening, low disease level	0.99 (0.93; 1.08)	0.99	1.02 (0.93; 1.11)	0.69
Improving, moderate to severe disease level	1.08 (1.01; 1.15)	0.03	1.08 (0.99; 1.17)	0.07
Worsening, moderate to severe disease level	1.13 (1.06; 1.21)	<0.001	1.27 (1.16;1.39)	<0.001
Stable, moderate to severe disease level	1.13 (1.04; 1.22)	0.003	1.25 (1.12; 1.39)	<0.001

Using highest achieved level of education between ages 25 and 30 and prevalent type 2 diabetes as cox-strata. Abbreviations: CI = confidence interval, HR = hazard ratio.

overlapping genetic determinants of oral disease – particularly dental caries and periodontitis - and systemic health outcomes. This suggests that some of the biological processes underlying DC may also influence general health, including metabolic and cardiovascular conditions [33]. Again, the data used in our study cannot confirm or refute this. Future studies examining the genetic profiles of the SCOR population could help clarify shared genetic pathways underlying our findings.

4.1. Strengths and limitations

Several limitations should be considered. First, the use of Danish nationwide register data does not allow for direct adjustment of lifestyle factors such as smoking and dietary habits, which may influence both poor oral health and ASCVD, as such information is not available in the registries. Lifestyle factors, including smoking and an obesogenic lifestyle, have been shown to correlate with infrequent toothbrushing, and a lower level of education [34]. Consistent with this, stratification by highest achieved level of education reduced the estimated HRs in our study, suggesting partial control for confounding from lifestyle. Nevertheless, residual confounding is likely. Future studies could leverage Danish cohort data, such as the Danish Blood Donor Study, which includes detailed lifestyle information, though this would involve smaller sample sizes and greater selection bias compared to the present study.

Second, we were unable to account for oral health in adulthood. Studies of the association between childhood and adulthood oral health are lacking, but it is believed that the basis for adulthood oral health is established already in childhood as oral hygiene, and dietary habits along with patterns of dental care attendance are laid down [35,36]. Consequently, the observed associations may be mediated by poor oral health in adulthood, which is an established ASCVD risk factor. However, given the assumed strong correlation between childhood and adulthood oral health, our findings still underscore the potential benefits of improving childhood oral health to reduce ASCVD risk.

Third, the use of dmft/DMFT as the primary measure of dental caries may be considered imprecise, as it does not distinguish active lesions from inactive lesions or fillings. Sensitivity analyses using only active caries lesions yielded similar associations with ASCVD, though group distributions were less balanced. Therefore, dmft/DMFT was retained as the main measure.

Last, the relatively young age of the study population limits the number of ASCVD events and may bias results toward early-onset phenotypes, potentially inflating HR estimates and reducing generalizability.

Strengths of this study include its nationwide design, long follow-up, and detailed childhood oral health measures. The validity of ASCVD diagnoses in the National Patient Register is high [37], and given Denmark's universal healthcare system and minimal private hospital coverage for acute conditions, missing diagnosis data is unlikely. SCOR data accurately reflect historical dental caries prevalence, though gingivitis prevalence may be somewhat underestimated [7]. Overall, generalizability to the Danish population is high, though applicability to populations worldwide may vary.

5. Conclusions

In conclusion we find evidence of an association between DC and gingivitis in childhood and the incidence of ASCVD measured by IHD, MI and/or IS in adulthood. Our data does not, however, establish causality. Studies accounting for lifestyle factors and oral health in adulthood are warranted. Nevertheless, our results suggest that the impact of childhood oral health on adulthood risk of ASCVD is worth investigating further, as it opens a new line of enquiry in understanding the complex causes underlying the development of ASCVD, and potentially provides new avenues of early detection and prevention of ASCVD. To fully leverage this potential, closer collaboration between cardiovascular and dental health professionals is essential. Furthermore, our findings underscore the importance of governmental investment in cross-disciplinary initiatives and in childhood oral health programs to promote oral and cardiovascular health across the lifespan.

Data statement

All SCOR-data as used in the present article are available from The Danish Health Data Authority, albeit with restrictions on the availability of these data. The R-script for cleaning data and generating the presented analyses can be found on GitHub @Niknyg.

CRediT authorship contribution statement

Nikoline Nygaard: Writing – review & editing, Writing – original draft, Visualization, Validation, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Francesco D'Aiuto:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Anne Kirstine Eriksen:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Anja Olsen:** Writing – review & editing, Supervision, Methodology, Data curation, Conceptualization. **Evelina Stankevic:** Writing – review & editing, Methodology, Data curation, Conceptualization. **Lars Ängquist:** Writing – review & editing, Supervision, Methodology, Data curation, Conceptualization. **Torben Hansen:** Writing – review & editing, Supervision, Project administration, Methodology, Funding acquisition, Data curation, Conceptualization. **Daniel Belstrøm:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Merete Markvart:** Writing – review & editing, Supervision, Project administration, Funding acquisition, Data curation, Conceptualization.

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Declaration of competing interest

The authors report no relationships that could be construed as a conflict of interest.

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The graphical abstract was created in BioRender. Nygaard, N. (2026) https://BioRender.com/s66f006.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2025.134151.

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