**The role of oral health related quality of life in the association between dental caries and height, weight and BMI among children in Bangladesh**

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**Abstract**

Objectives: To examine whether oral health related quality of life (OHRQoL) explained the negative associations between dental caries and anthropometric measures of child growth among a sample of 5-9-years-old children in Dhaka, Bangladesh, while taking potential confounding factors (maternal education, family income, study setting, child's birth weight, and childhood diseases) into account. In addition, to test whether specific oral impacts had a role in explaining these associations.

Methods: Data collection was conducted via a cross sectional survey among children and their parents from both hospital and school settings in Dhaka. Dental caries and severe consequences of dental caries (defined here as dental sepsis) were the exposure variables and age and sex adjusted height-z-scores (HAZ), weight-z-scores (WAZ) and BMI-z-scores (BAZ) were the outcome variables. OHRQoL was measured using the Bengali version of the Scale of Oral Health Outcomes for 5-year-old children (SOHO-5). First, associations between oral impacts (prevalence of overall impacts and specific items of the SOHO-5) and outcome measures were assessed. Multiple linear regression was used to assess associations between caries and anthropometric measures, adjusted for potential confounders. Oral impacts were then added to test whether their inclusion attenuated the associations between exposures and outcomes. To further investigate potential mediating role of oral impacts, structural equation modelling (SEM) was used to test the hypothesis that dental caries and sepsis were associated with the outcomes directly and also indirectly via oral impacts in general. A similar approach was used to investigate mediation by specific SOHO-5 items.

Results: The sample consisted of 715 children, 73.1% of whom had dental caries, 37.5% presented with sepsis, and 57.3% reported at least one oral impact (SOHO-5 score ≥1). Prevalence of overall oral impacts and also the impact on 'eating difficulty' (a specific item of SOHO-5) were negatively associated with all three outcomes. Dental caries and sepsis was associated with lower HAZ, WAZ and BAZ, and adjustment for overall oral impacts considerably attenuated the associations between 'severe dental caries' and outcomes, and dental sepsis and outcomes. Using SEM, we found significant indirect associations between caries and sepsis and anthropometric measures via oral impacts (except for dental caries and HAZ). Considering specific oral impacts, eating difficulties explained about 44% and 65% of the associations between caries and

anthropometric outcomes, and dental sepsis and anthropometric outcomes, respectively.

Conclusions: Oral impacts, in particular eating difficulties, appear to mediate associations between caries and markers of child growth among this population.

**(Word counts 400)**

## Introduction

Dental caries is one of the most common childhood diseases globally.[1] In 2015, 573 million children (7·8 per cent of the global child population) had untreated dental caries.[2] Despite improvements in most high-income countries over the last decades, the prevalence of dental caries is still high among children from low and middle-income countries (LMICs).[3] Alarmingly, more than 90% of decay remains untreated in those countries[4] due to the inadequate access to and prohibitive cost of treatment. Many children face lifelong, severe consequences of chronic untreated tooth decay. One of these consequences refer to the negative impact of dental caries on children's growth[5,6].

Systematic reviews assessing the association between dental caries and height, weight and BMI (where these anthropometric measures were considered as proxy measures of child growth or nutrition) show that there is no consensus in the literature about the direction of the association (positive, negative and no association).[7–9] Studies from LMICs on populations with high levels of untreated caries support a negative association.[10,11], and we have also shown that dental caries was negatively associated with height, weight and BMI among 5-9-year-old children in Bangladesh.[12]

Several mechanisms have been proposed to explain the adverse impact of dental caries on child growth.[11,13] One of those mechanisms postulates that severe caries can affect children's quality of life and thereby growth.[13] Severe caries may lead to pain and discomfort that affects children physically and psychologically,[14] and leads to a detrimental effect on their overall quality of life.[15,16]. Evidence has demonstrated the negative impact of dental caries on oral health related quality of life (OHRQoL), particularly in terms of compromised physical functioning (for example, eating difficulties, sleeping difficulties),[8] and social and psychological well-being.[17–19] Dental

pain induced eating difficulties may lead to malnutrition and sleeping difficulties may affect growth hormone secretion, which may in turn negatively affect child growth.[8] The social and psychological impacts of caries might affect growth indirectly [20–23] Therefore, OHRQoL may partly explain the negative associations between dental caries and poor child growth.

However, to our knowledge no study has assessed if OHRQoL could explain the associations between dental caries and poor height, weight and BMI among children. This is particularly important in a LMIC country [24] such as Bangladesh, where oral health is not a priority in national health policy, children suffer from untreated dental caries, its impact on their quality of life is often ignored[25] and childhood malnutrition is still a major concern.[26] Therefore, assessing OHRQoL and understanding its influence on indicators of poor child growth could highlight the importance of children's oral health to policy makers to promote oral health and better access to dental care.[27]

The aim of this study was to examine whether OHRQoL partly explained the negative associations between dental caries and anthropometric indicators of child growth among a sample of 5-9-years-old children in Dhaka, Bangladesh, while taking potential confounding factors into account. In addition, we aimed to test whether specific oral impacts had a role in explaining these associations. We hypothesised that dental caries and sepsis would be associated with poorer OHRQoL, which in turn would partly attenuate associations between dental caries and sepsis and age and sex adjusted height, weight and BMI.

**Methods**

A cross-sectional survey was conducted between August and October 2015 among 5-9-year-old children and their parents in Dhaka, Bangladesh. Participants were recruited from two settings (hospital and community). The hospital samples were recruited from children attending the Dhaka Dental College Hospital[28] (the largest public dental hospital in Bangladesh) for treatment. Within the scope of the study, three local primary schools were randomly selected from the schools in the catchment area of the hospital. All children of the eligible age group and their parents were invited to the study. Children who had any self-reported systemic diseases or acute infections, fever or

diarrhea during the week preceding the data collection were excluded. All participants' parents or legal guardians provided written informed consent. Further details of recruitment, sample size calculation and clinical measurements can be found elsewhere.[12] Ethical approval was obtained from the University College London Research Ethics Committee and the National Research Ethics Committee of Bangladesh. A pilot study, conducted in January 2015 with 272 children, demonstrated the feasibility of the procedures adopted for the study.

Age and sex-adjusted z scores for height, weight, and BMI were the outcome variables. Height, weight and BMI were converted to z-scores, namely height-for-age z-scores (HAZ), weight-for-age z-scores (WAZ), and BMI-for-age z-scores (BAZ) using WHO standard growth reference data 2007.[29] Dental caries and severe consequences of dental caries were the main exposures. Dental examinations were conducted visually by three trained and calibrated dentists, following WHO guidance for oral health surveys.[30] The average Kappa was 0.92 for intra-examiner reproducibility and 0.88 for inter-examiner reproducibility, revealing very good agreement. Dental caries was measured using the dmft/ DMFT indices. Severe consequences of dental caries (defined in this study as dental sepsis) was measured using the pufa/ PUFA index.[31] For analytical purposes dental caries was classified into four categories: a caries free group (dmft+DMFT = 0); while children with dental caries experience were divided into three groups: a low caries tertile (dmft+DMFT = 1-2), a moderate caries tertile (dmft+DMFT = 3-5) and a severe caries tertile (dmft+DMFT = 6-15). Dental sepsis was dichotomised into 'no sepsis' (pufa+PUFA = 0) and 'having sepsis' (pufa+PUFA>0).

We considered the role of OHRQoL in the associations between exposures and outcomes. The theoretical model was based on a paper by Sheiham (2006) on the impact of caries, where plausible mechanisms for how dental caries may be associated with underweight and poor growth in young children were put forward, postulating that 'severe caries can affect children's quality of life and thereby growth'.[13] In this study OHRQoL was measured using a cross-culturally adapted and validated Bengali version[25] of the Scale of Oral Health Outcomes for 5-year-old children (SOHO-5).[32] Three trained interviewers conducted face-to-face interviews with the children, using the child version of the SOHO-5 questionnaire. The parental version of the SOHO-5 questionnaire was self-administered by their parents.

We used the data from the child SOHO-5 for the analysis reported in this paper as previous studies have demonstrated that children of this age group can express their symptoms and quality of life.[33] In short, the child version of SOHO-5 contains seven questions to assess oral impacts, including difficulty with eating, drinking, speaking, playing, sleeping, as well as avoiding smiling due to appearance, and avoiding smiling due to pain. All questions were assessed through a 3-point answering scale ('no', 'a little', 'a lot'). The cumulative SOHO-5 score was calculated for the seven items on oral impacts, with a higher score indicating poorer OHRQoL. To determine the overall oral impacts, we dichotomized the SOHO-5 score into those with no oral impacts (SOHO-5 score = 0) and those with at least one oral impact (SOHO-5 score = 1 or higher). In addition, to assess the prevalence of each specific oral impact, we dichotomized each of the seven SOHO-5 items by combining 'a little' and 'a lot'.

Information on socio-economic background, birth weight and childhood diseases was provided by parents through self-administered questionnaires. The following covariates were considered as potential confounders, as they are associated with both child growth[34] and dental caries[35,36] and are not on the direct pathway between them: maternal education ('no formal education', 'primary', 'secondary', 'higher secondary', and 'tertiary'), monthly gross family income in Taka/month (< 8000; 8000 to 20,000; > 20,000 to 30,000; > 30,000 Taka; 1GBP=110 Taka), birth weight (normal, low, and high), and childhood diseases (whether the child had any long-standing illness) (yes, no).

**Data analysis**

Data analysis was performed using Stata version 13 software.[37] Initial descriptive analyses were followed by one-way analysis of variance (ANOVA) and t-tests to assess the bivariate associations between exposures (dental caries and sepsis) and the outcomes (height, weight, and BMI). We then examined associations between the overall prevalence of oral impacts (as the potential mediator) and the exposures and outcomes. Chi-squared tests were carried out to assess the bivariate associations between exposures and oral impacts. Multiple linear regression models were used to test the association between overall prevalence of oral impacts and outcomes, adjusting for dental caries and sepsis and other confounders (maternal education, family income, study setting (hospital and school), birth weight, and childhood diseases. In addition, we conducted separate multiple linear regression models using the prevalence of

specific oral impacts (the seven items of SOHO-5) to assess the role of specific oral impacts following a similar strategy of adjustments to test which specific oral impacts were significantly associated with the outcomes.

Two separate sets of multiple linear regression models (one with dental caries and one with dental sepsis as the exposure) were run for each outcome variable. We modelled these separately as we wanted to test the effect of dental caries and severe consequences of dental caries separately on the outcomes. Each model was adjusted for other confounders. Prevalence of oral impacts was then added to the models to test whether their inclusion attenuated the associations between dental caries and sepsis with the anthropometric outcomes. In addition, we conducted a sensitivity analysis by running the model with active dental decay (d+D) instead of caries experience (dmft + DMFT). While not strictly appropriate for cross-sectional data, we also conducted a mediation analysis to examine if there is any preliminary evidence that oral impacts (overall SOHO-5 score) mediate the associations between the predictor variables (dental caries and sepsis) and the anthropometric outcomes (HAZ, WAZ and BAZ) using Structural Equation Models (SEM). We tested the hypothesis that dental caries or sepsis influences anthropometric outcomes directly and also indirectly via oral impacts. Figure 1 presents the conceptual model that informed our analyses.

Finally, we used a similar approach (initially testing attenuation of the associations using multiple linear regression models followed by mediation analysis using SEM) to investigate the potential mediating role of specific oral impacts that showed significant negative associations with the outcomes in the previous analysis.

All analyses were based on complete data.

**Results**

Among 805 invited children, parental consent was obtained from 797 children. 788 children participated in the survey and 725 (92%) parents returned the questionnaires, of which ten had some missing data. Therefore, the analysis sample included 715 children with complete data.

The characteristics of the sample and bivariate associations between exposures and prevalence of oral impacts, and anthropometric outcomes are presented in Table 1. The

children were between 5 and 9 years old, with a mean age of 7.1 (SD: 1.0) years and 365 (51%) were girls. The mean dmft+DMFT score was 2.8 (95% CI 2.6, 3.0), where active decay (d+D component) constituted more than 70% of the dmft+DMFT score. Nearly 99% of the decay was in deciduous teeth (not shown in the Table). Overall, 26.8% of the study population were caries free, 25.7% had low levels of caries, 30.3% had moderate and 17.1% had severe levels of caries, while 37.5% had dental sepsis (pufa/PUFA>0). The mean (95% CI) HAZ, WAZ and BAZ scores were -0.04 (-0.14, 0.05), -0.22 (-0.34, -0.10) and -0.32 (-0.43, -0.20), respectively. The mean SOHO-5 score was 1.79 (1.62, 1.96) (range: 0 to 14) and the median was 1. The distribution of the SOHO-5 scores was positively skewed, with 42.7% of children having a score of zero (no oral impacts) and 57.3% of children reporting at least one oral impact on their daily life (SOHO-5 score ≥1). Difficulty eating was the most commonly reported oral impact (52%), followed by difficulty sleeping (30%), difficulty drinking (14%), avoiding smiling due to pain (15%) and difficulty speaking (10%) (supplementary Table 2). Children with higher levels of dental caries and those with dental sepsis had higher prevalence of oral impacts and lower mean HAZ, WAZ and BAZ scores.

Table 2 shows associations between the oral impact and outcomes. The results of multiple linear regressions showed that children with at least one oral impact (SOHO-5 score ≥1) had lower WAZ and BAZ than children with no oral impacts, even after adjustment for dental caries or sepsis and other confounders. The associations of specific oral impacts (individual items within the child version of SOHO-5) with the outcomes showed that 'difficulty eating' was the only item that was significantly associated with lower BAZ in the fully adjusted analysis (Table 2). 'Avoiding smiling due to pain' in the SOHO-5 scale showed significant negative associated with anthropometric measures in the unadjusted model, but the associations become non-significant in the fully adjusted model.

**Associations between dental caries / sepsis and outcome measures**

Results of multiple linear regression models showed that there were significant negative associations between dental caries and HAZ, WAZ and BAZ scores, before and after adjustment for potential confounding factors (Table 3a models 1 and 2). Following additional adjustment for oral impacts (model 3), the associations were further attenuated and became non-significant. For example, for children in the 'moderate' and

'severe' dental caries groups, BAZ scores were -0.43 (-0.72, -0.13) and -0.50 (-0.87, -0.13) points lower than the 'no caries' group (model 2), respectively. Additionally, after adjusting for the prevalence of oral impacts, the coefficients for moderate and severe dental caries groups were reduced to -0.10 (-0.48, 0.29) and -0.16 (-0.61, 0.28) respectively (model 3). Sensitivity analyses using active dental decay (d+D component) instead of caries experience as the main exposure confirmed the attenuation of the coefficients after adjusting for oral impacts for WAZ and BAZ (Supplementary Table 1). However, the attenuation was larger when caries experience was considered as the exposure (comparing Table 3a and Supplementary Table 1). Similar inverse associations were found between dental sepsis and all three outcomes (Table 3b). The coefficients for dental sepsis were reduced to -0.15 (-0.37, 0.07), -0.14 (-0.40, 0.12) and -0.07 (-0.34, 0.19) for HAZ, WAZ and BAZ (respectively) after additional adjustment for SOHO-5 and became non-significant (model 3). The complete version of Tables 3a and 3b with coefficients for all the covariates are presented as Supplementary Tables 4 and 5.

The results of the mediation analysis showed that the relationships between dental caries and sepsis with the outcomes were mediated by oral impacts (Table 4). Direct associations were observed between caries and all three outcomes. We also found significant indirect associations between dental caries and WAZ (indirect effect: −0.03; 95% CI −0.05, –0.01) and BAZ (indirect effect: −0.03; 95% CI −0.05, –0.00) via oral impacts. However, the indirect association between dental caries and HAZ was not statistically significant (indirect effect: −0.02; 95% CI −0.04, 0.00). Overall, indirect effects represented 17% of the estimated total effect for HAZ, 21% for WAZ and 24% for BAZ. Oral impacts played an even stronger role in explaining the association between dental sepsis and the outcomes. Significant direct associations were observed between dental sepsis and HAZ and WAZ but not with BAZ. Overall, indirect effects represented 35% of the estimated total effect for HAZ, and 41% for WAZ. The association between dental sepsis and BAZ was almost equally split between direct and indirect pathways, where 47% of the estimated total effect of dental sepsis on BAZ was indirect and 53% direct. The path analytic models are shown in Supplementary Figures 1 and 2.

Examination of specific oral impacts revealed that adjustment for 'eating difficulty' led to attenuation of associations between dental caries / sepsis and anthropometric indicators (model 3 of Supplementary Table 2a and 2b). Structural equation models identified significant indirect associations between caries / sepsis and anthropometric measures via 'eating difficulty'. Eating difficulties explained about 44% (for dental caries) and 65% (for dental sepsis) of these associations (Supplementary Table 3).

**Discussion**

Our study has demonstrated that OHRQoL might partially explain the negative associations between dental caries / sepsis and anthropometric indicators of child growth. Of the specific oral impacts included in the SOHO-5, eating difficulties appeared to explain most of these associations. The associations between severe caries and HAZ, and those of moderate and severe caries and WAZ and BAZ were attenuated after adjusting for prevalence of oral impacts. For HAZ, there was a 25.0% attenuation of the regression coefficient for the severe caries group. For WAZ, the adjustment resulted in 67.4% and 50.8% attenuations of the regression coefficients for the moderate and severe caries groups respectively, and for BAZ the attenuations of the respective regression coefficients were 76.8% and 68.0%. Finally, there were 34.8%, 57.6%, 75.9% attenuations of the regression coefficients for the associations between HAZ, WAZ and BAZ with dental sepsis after the adjustment. Sensitivity analyses using active decay (d+D) as the exposure, rather than overall caries experience, showed a similar pattern of associations but a smaller attenuation of the coefficients after adjusting for oral impacts. It is worth mentioning that very few children receive dental restorations in LMICs.[24] In our study, dental fillings (f+F component) constituted only 6% of the dmft+DMFT score. Children usually seek dental care at an advanced stage of caries, and this may result in pain and contribute to oral impacts.

Mediation analysis showed that oral impacts played a role in explaining these associations. We found statistically significant indirect associations between dental caries / sepsis and all anthropometric outcomes via oral impacts, except for the association between dental caries and HAZ. The indirect paths via oral impacts were strongest for associations between dental sepsis and the outcomes, where 40% or more of the estimated total effect was indirect, while for associations between dental caries and anthropometric outcomes about 20% of the total effect was indirect. This potential

mediation role could be due to the impact of dental caries on specific aspects of daily life that affect the growth of children. Higher caries levels have been shown to be associated with overall poorer OHRQoL, higher subscale scores for oral symptoms, and impacts on functional and emotional well-being.[38–40] Dental caries particularly affects functional aspects of OHRQoL such as food intake and sleep.[23] Alkarimi et al. suggested potential mechanisms as to how these might have negative impacts on child growth.[11] Reduced food intake, as a consequence of eating difficulty due to dental caries and pain, can lead to undernutrition and poor child growth.[41] Similarly, dental pain can negatively impact on sleep and result in reduced slow wave sleep, which may cause disturbance of growth hormone secretion.[22] Looking at specific oral impacts, our mediation analysis suggests that eating difficulty plays a key role in explaining these associations, further highlighting the importance of functional aspects of OHRQoL for child growth. Evidence from randomized controlled trials on the effects of dental caries treatment on quality of life and child growth indirectly supports this interpretation of our findings. Extraction of pulpally involved teeth or rehabilitation of caries was shown to be associated with a significant increase in weight and BMI,[42] as well as improvements in oral impacts.[43–46]

At the same time, we did not find associations between other oral impacts and the outcomes and, as such, these were not considered as part of potential pathways between dental caries and anthropometric measures for this population. For the oral impact on sleeping, it might be that the sleeping pathway via growth hormones might be particularly important at other stages of child growth rather than for the age group (5-9-year-olds) covered in this cross-sectional study. Further longitudinal studies including a wider age range might provide more insight. Psychosocial aspects of oral impacts (i.e. 'avoidance of smiling due to appearance' and 'avoidance of smiling due to pain'), may also indirectly impact on child growth. However, as only a small proportion of the sample reported those oral impacts, it was not possible to clearly explain the observed associations.

It is important to interpret our findings within the limitations of our study. We examined oral impacts as a mediator in the association between dental caries/sepsis and anthropometric measures but acknowledge that the cross-sectional nature of our data limits what conclusions can be drawn from the testing for mediation. Future

longitudinal studies in LMICs should provide more comprehensive evidence for this. We also acknowledge that the direction of the observed associations cannot be ascertained in cross sectional studies. However, the direction from dental caries to lower body weight is supported by evidence from previous studies on weight gain after oral rehabilitation[5,47]. Finally, the study sample was not representative of Bangladeshi children in general.

Our study has several strengths. While most previous cross-sectional studies on child populations investigated associations between dental caries and either anthropometric measures or OHRQoL, we took the approach of examining both OHRQoL and anthropometric measures at the same time. Furthermore, we adjusted for a range of potential confounding factors such as sociodemographic and child health related factors. Our findings provide evidence from an LMIC setting about the importance of dental caries for indicators of child growth (e.g. BMI), a research area where existing relevant evidence is inconclusive and comes primarily from high income settings[48,49]. Furthermore, we shed light on the role of OHRQoL in children, which is often overlooked amongst health care providers in Bangladesh. OHRQoL is a useful tool to communicate with policy-makers, as it reflects the impact of dental caries on children's life such as difficulties eating, sleeping, smiling, and socialising. Our findings show associations between impaired OHRQoL and child growth. Highlighting the potential role of OHRQoL in the association between dental caries and child growth is expected to substantiate the rationale for prevention and early treatment of dental caries of children, as treatment of dental caries could improve OHRQoL[50] and child growth.[5] Future studies in LMICs should focus on developing effective public health interventions in collaboration with related stakeholders to reduce caries prevalence and ensure early treatment, better OHRQoL and growth of children.

## Conclusion

This study has shown that poor OHRQoL could be an important factor in partially explaining the inverse association between dental caries and growth (mostly via eating difficulty) among this study population. To alleviate this problem, oral health should be considered in health improvement policies and efforts should be made to reduce the prevalence of child dental caries, and to improve access to oral health care services.

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Authors Contribution:

MPM: Conceiving, designing and conducting the study, analyzed and interpreted the data and drafted the manuscript.

GT, AH and RW: Contributed in conceiving and designing the study, provided guidance on data analysis and interpretation, and critically reviewing the manuscript and provided comments for revision.

**Ethical approval**

Ethical approval was obtained from University College London Research Ethics Committee and National Research Ethics Committee of Bangladesh. Written permission was obtained from the hospital and school authority.

**Conflicts of interest**

The authors declare that they have no conflicts of interest to disclose.

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Table 1: Oral impacts and anthropometric outcomes by demographic, socioeconomic and clinical characteristics (N=715)

| Variables | Oral impacts n (%) | | | HAZ, WAZ, BAZ Mean (95%CI) | | |
|---|---|---|---|---|---|---|
| | N (%) | No oral impact (SOHO-5 score=0) | Any oral impact (SOHO-5 score≥1) | HAZ | WAZ | BAZ |
| Overall | 715 (100) | 305 (42.7) | 410 (57.3) | -0.04 (-0.14, 0.05) | -0.22 (-0.34, -0.10) | -0.32 (-0.43, -0.20) |
| Sex | | | | | | |
| Boy | 350 (48.9) | 140 (40.0) | 210 (60.0) | -0.17 (-0.31, -0.03) | -0.45 (-0.62, -0.28) | -0.55 (-0.71, -0.38) |
| Girl | 365 (51.0) | 165 (45.2) | 200 (54.8) | 0.08 (-0.05, 0.22) | -0.00 (-0.16, 0.16) | -0.09 (-0.25, 0.06) |
| *p* | | *0.159*[1] | | *0.009*[2] | *<0.001*[2] | *<0.001*[2] |
| Study setting | | | | | | |
| Hospital | 232 (32.4) | 36 (15.5) | 196 (84.5) | -0.68 (-0.83, -0.52) | -0.98 (-1.15, -0.81) | -0.84 (-1.00, -0.68) |
| School | 483 (67.5) | 269 (55.7) | 214 (44.3) | 0.26 (0.15, 0.38) | 0.15 (0.00, 0.29) | -0.06 (-0.21, 0.08) |
| *p* | | *<0.001*[1] | | *<0.001*[2] | *<0.001*[2] | *<0.001*[2] |
| Maternal education | | | | | | |
| No, primary | 170 (23.8) | 51 (30.0) | 119 (70.0) | -0.73 (-0.92, -0.54) | -1.03 (-1.25, -0.82) | -0.88 (-1.09, -0.68) |
| Secondary | 216 (30.2) | 87 (40.3) | 129 (59.7) | -0.11 (-0.28, 0.05) | -0.34 (-0.54, -0.14) | -0.44 (-0.65, -0.24) |
| Higher secondary | 183 (25.6) | 92 (50.3) | 91 (49.7) | 0.21 (0.02, 0.41) | 0.11 (-0.13, 0.35) | -0.06 (-0.30, 0.17) |
| Tertiary | 146 (20.4) | 75 (51.4) | 71 (48.6) | 0.54 (0.34, 0.74) | 0.50 (0.26, 0.73) | 0.21 (-0.05, 0.47) |
| *p* | | *<0.001*[1] | | *<0.001*[3] | *<0.001*[3] | *<0.001*[3] |
| Family income (Taka /month) | | | | | | |

| Variables | | Oral impacts n (%) | | HAZ, WAZ, BAZ Mean (95%CI) | | |
|---|---|---|---|---|---|---|
| | N (%) | No oral impact (SOHO-5 score=0) | Any oral impact (SOHO-5 score≥1) | HAZ | WAZ | BAZ |
| <8 thousand | 105 (14.7) | 43 (40.9) | 62 (59.0) | -0.49 (-0.72, -0.26) | -0.82 (-1.10, -0.53) | -0.79 (-1.07, -0.52) |
| 8-20 thousand | 334 (46.7) | 124 (37.13) | 210 (62.9) | -0.18 (-0.32, -0.04) | -0.45 (-0.62, -0.29) | -0.54 (-0.70, -0.37) |
| >20-30 thousand | 165 (23.1) | 93 (56.4) | 72 (43.6) | 0.41 (0.23, 0.60) | 0.41 (0.17, 0.65) | 0.19 (-0.06, 0.44) |
| >30 thousand | 111 (15.5) | 45 (40.5) | 66 (59.5) | 0.12 (-0.14, 0.39) | 0.11 (-0.18, 0.41) | 0.04 (-0.24, 0.32) |
| *p* | | *0.001*[1] | | *<0.001*[3] | *<0.001*[3] | *<0.001*[3] |
| Dental caries | | | | | | |
| No caries | 192 (26.8) | 186 (96.9) | 6 (3.1) | 0.34 (0.17, 0.52) | 0.33 (0.11, 0.54) | 0.13 (-0.10, 0.36) |
| Mild caries | 184 (25.7) | 80 (43.5) | 104 (56.5) | 0.12 (-0.08, 0.32) | -.012 (-.24, 0.21) | -0.16 (-0.38, 0.07) |
| Moderate caries | 217 (30.3) | 30 (13.8) | 187 (86.2) | -0.20 (-0.37, -0.02) | -0.47 (-0.68, -0.26) | -0.55 (-0.76, -0.35) |
| Severe caries | 122 (17.1) | 9 (7.4) | 113 (92.6) | -0.62 (-0.84, -0.41) | -0.94 (-1.21, -0.67) | -0.84 (-1.09, -0.59) |
| *p* | | *<0.001*[1] | | *<0.001*[3] | *<0.001*[3] | *<0.001*[3] |
| Dental sepsis | | | | | | |
| No sepsis | 447 (62.5) | 284 (63.5) | 21 (7.8) | 0.18 (0.06, 0.30) | 0.07 (-0.07, 0.22) | -0.09 (-0.24, 0.06) |
| Sepsis | 268 (37.5) | 163 (36.5) | 247 (92.2) | -0.41 (-0.57, -0.26) | -0.71 (-0.89, -0.53) | -0.69 (-0.87, -0.52) |
| *p* | | *<0.001*[1] | | *<0.001*[2] | *<0.001*[2] | *<0.001*[2] |

[1] Chi square test, [2] t test, [3] ANOVA

Table 2: Results of linear regression models testing the associations of prevalence of oral impacts with HAZ, WAZ and BAZ and associations of individual items of oral impacts with HAZ, WAZ and BAZ (N= 715)

| Oral impacts (ref. no impact/ no difficulty/ not avoiding) | Models | HAZ Regression coefficient (95% CI) | WAZ Regression coefficient (95% CI) | BAZ Regression coefficient (95% CI) |
|---|---|---|---|---|
| Prevalence of oral impacts (57.34%) | Model 1* | -0.63 (-0.83, -0.44)[a] | -0.95 (-1.17, -0.72)[a] | -0.80 (-1.03, -0.57)[a] |
| | Model 2* | -0.12 (-0.37, -0.13) | -0.37 (-0.66, -0.08)[c] | -0.43 (-0.73, -0.13)[b] |
| | Model 3* | -0.18 (-0.40, 0.04) | -0.44 (-0.70, -0.18)[b] | -0.48 (-0.75, -0.21)[a] |
| Difficulty in eating (52.0%) | Model 1 | -0.49 (-0.71, -0.26) [a] | -0.80 (-1.06, -0.53) [a] | 0.73 (-1.00, -0.47) [a] |
| | Model 2 | -0.04 (-0.21, 0.12) | -0.18 (-0.37, 0.01) | -0.24 (-0.44, -0.04) [c] |
| | Model 3 | -0.06 (-0.22, 0.09) | -0.22 (-0.41, -0.03)[c] | -0.28 (-0.47, -0.08)[b] |
| Difficulty in drinking (14.3%) | Model 1 | 0.15 (-0.13, 0.45) | 0.14 (-0.20, 0.49) | 0.06 ( -0.29, 0.40) |
| | Model 2 | 0.02 (-0.18, 0.22) | 0.03 (-0.20, 0.26) | 0.01 ( -0.23, 0.26) |
| | Model 3 | -0.00 (-0.20, 0.19) | -0.01 (-0.24, 0.22) | 0.01 (-0.25, 0.22) |
| Difficulty in speaking (10.5%) | Model 1 | -0.05 (-0.44, 0.34) | -0.21 (-0.67, 0.25) | -0.22 (-0.69, 0.24) |
| | Model 2 | -0.10 (-0.36, 0.17) | -0.24 (-0.70, 0.22) | -0.25 (-0.71, 0.21) |
| | Model 3 | -0.09 (-0.36, 0.31) | -0.23 (-0.54, 0.08) | -0.23 (-0.56, 0.09) |
| Difficulty in playing (7.3%) | Model 1 | 0.36 (-0.09, 0.82) | 0.48 (-0.06, 1.02) | 0.32 (-0.22, 0.86) |
| | Model 2 | 0.25 (-0.06, 0.57) | 0.45 (0.08, 0.82) [c] | 0.40 (0.01, 0.78)[c] |
| | Model 3 | 0.26 (-0.06, 0.57) | 0.46 (0.09, 0.83)[c] | 0.40 (0.02, 0.78)[c] |
| Difficulty in sleeping (30.2%) | Model 1 | -0.16 (-0.42, 0.10) | -0.09 (-0.40, 0.22) | 0.02 (-0.29, 0.33) |
| | Model 2 | -0.06 (-0.23, 0.11) | -0.00 (-0.20, 0.19) | 0.05 (-0.16, 0.25) |
| | Model 3 | -0.06 (-0.23, 0.10) | -0.02 (-0.21, 0.18) | 0.03 (-0.17, 0.24) |
| Avoiding smiling due to appearance (8.8%) | Model 1 | 0.41 (0.05, 0.77) [c] | 0.52 (0.09, 0.95) [c] | 0.41 (-0.02, 0.84) |

| Oral impacts (ref. no impact/ no difficulty/ not avoiding) | Models | HAZ Regression coefficient (95% CI) | WAZ Regression coefficient (95% CI) | BAZ Regression coefficient (95% CI) |
|---|---|---|---|---|
| | Model 2 | 0.23 (-0.02, 0.48) | 0.31 (0.01, 0.60) [c] | 0.26 (-0.04, 0.57) |
| | Model 3 | 0.24 (0.01, 0.49) | 0.32 (0.02, 0.61) [c] | 0.27 (-0.03, 0.58) |
| Avoiding smiling due to pain (14.6%) | Model 1 | -0.44 (-0.80, -0.09) [c] | -0.60 (-1.02, -0.18) [b] | -0.43 (-0.84, -0.01) [c] |
| | Model 2 | -0.12 (-0.35, 0.12) | -0.20 (-0.48, 0.07) | -0.16 (-0.45, 0.12) |
| | Model 3 | -0.10 (-0.34, 0.13) | -0.18 (-0.46, 0.09) | -0.15 (-0.44, 0.14) |

Model 1* (unadjusted model): outcomes+ prevalence of oral impacts (dichotomized SOHO-5)
Model 2* (adjusted for dental caries and other confounders): outcomes+ prevalence of oral impacts + dental caries+ socio-economic variables (maternal education+ family income) +setting+ birth weight, childhood disease
Model 3* (adjusted for dental sepsis and other confounders): outcome+ prevalence of oral impacts + dental sepsis socio-economic variables (maternal education+ family income)+setting+birth weight, childhood disease

Model 1 (unadjusted model): outcome+ difficulty in eating+ difficulty in drinking+ difficulty in speaking+ difficulty in playing+ difficulty in sleeping+ avoiding smiling due to appearance+ avoiding smiling due to pain
Model 2 (adjusted for dental caries and other confounders): outcome + difficulty in eating+ difficulty in drinking+ difficulty in speaking+ difficulty in playing+ difficulty in sleeping+ avoiding smiling due to appearance+ avoiding smiling due to pain + dental caries+ socio-economic variables (maternal education+ family income) +setting+ birth weight, childhood disease
Model 3 (adjusted for dental sepsis and other confounders): outcome + difficulty in eating+ difficulty in drinking+ difficulty in speaking+ difficulty in playing+ difficulty in sleeping+ avoiding smiling due to appearance+ avoiding smiling due to pain + dental sepsis+ socio-economic variables (maternal education+ family income) +setting+ birth weight, childhood disease

[a]p<0.001; multiple linear regression
[b]p<0.01; multiple linear regression
[c]p<0.05; multiple linear regression

Table 3 (a): Association between dental caries and HAZ, WAZ and BAZ and the potential role of oral impacts (Coefficients with 95% CIs) (N= 715)

| Outcomes | Exposure | Categories | Model 1 Regression coefficient (95% CI) | Model 2 Regression coefficient (95% CI) | Model 3 Regression coefficient (95% CI) |
|---|---|---|---|---|---|
| HAZ | Dental caries (ref: caries-free) | Mild | -0.22 (-0.49,0.04) | -0.07 (-0.32,0.17) | -0.01 (-0.29, 0.27) |
| | | Moderate | -0.54 (-0.79, -0.29)[a] | -0.24 (-0.48, 0.00) | -0.14 (-0.46, 0.17) |
| | | Severe | -0.97 (-1.26, -0.67)[a] | -0.40[b] (-0.69, -0.10) | -0.30 (-0.66, 0.07) |
| WAZ | Dental caries (ref: caries-free) | Mild | -0.34 (-0.65, -0.03) | -0.16 (-0.45, 0.13) | 0.03(-0.30, 0.35) |
| | | Moderate | -0.80 (-1.10, -0.50)[a] | -0.43 (-0.72, -0.15)[b] | -0.14 (-0.51, 0.22) |
| | | Severe | -1.27(-1.62, -0.92)[a] | -0.59 (-0.94, -0.24)[b] | -0.29 (-0.72, 0.13) |
| BAZ | Dental caries (ref: caries-free) | Mild | -0.28 (-0.60, 0.03)[b] | -0.16 (-0.46, 0.14) | 0.05 (-0.29, 0.39) |
| | | Moderate | -0.68 (-0.98, -0.38)[a] | -0.43 (-0.72, -0.13)[b] | -0.10 (-0.48, 0.29) |
| | | Severe | -0.96 (-1.31, -0.61)[a] | -0.50 (-0.87, -0.13)[b] | -0.16 (-0.61, 0.28) |

Model 1: Unadjusted analysis
Model 2: Model 1 + socio-economic variables (maternal education+ family income)+setting+ birth weight, childhood disease;
Model 3: Model 2 + prevalence of oral impacts

Table 3 (b) Association between dental sepsis and HAZ, WAZ and BAZ and the potential role of oral impacts (Coefficients with 95% CIs) (N= 715)

| Outcomes | Exposure | Categories | Model 1 Regression coefficient (95% CI) | Model 2 Regression coefficient (95% CI) | Model 3 Regression coefficient (95% CI) |
|---|---|---|---|---|---|
| HAZ | Dental sepsis (ref: no sepsis: pufa+PUFA=0) | pufa+PUFA≥ 1 | -0.60 (-0.79, -0.40)[a] | -0.23 (-0.42, -0.03)[c] | -0.15 (-0.37, 0.07) |
| WAZ | Dental sepsis (ref: pufa+PUFA=0) | pufa+PUFA≥ 1 | -0.78 (-1.02, -0.55)[a] | -0.33(-0.56, -0.10)[b] | -0.14 (-0.40, 0.12) |
| BAZ | Dental sepsis (ref: pufa+PUFA=0) | pufa+PUFA≥ 1 | -0.60 (-0.84, -0.37)[a] | -0.29 (-0.53, -0.05)[c] | -0.07 (-0.34, 0.19) |

Model 1: Unadjusted analysis
Model 2: Model 1 + socio-economic variables (maternal education+ family income)+ setting+ birth weight, childhood disease;
Model 3: Model 2 + prevalence of oral impacts

[a]p<0.001; multiple linear regression
[b]p<0.01; multiple linear regression
[c]p<0.05; multiple linear regression

Table 4: The role of oral impacts as a mediator on the association between dental caries and sepsis and HAZ, WAZ and BAZ (Coefficients with 95% CIs for direct, indirect and total influence)

| | | Outcomes | | | | | |
|---|---|---|---|---|---|---|---|
| | | HAZ | % | WAZ | % | BAZ | % |
| | | Coefficient (95% CI) | | Coefficient (95% CI) | | Coefficient (95% CI) | |
| Dental caries | Direct | -0.09 (-0.13, -0.06)[a] | 83.8 | -0.12 (-0.16, -0.07)[a] | 79.4 | -0.09 (-0.13, -0.04)[a] | 76.0 |
| | Indirect via oral impacts | -0.02 (-0.04, 0.00) | 16.2 | -0.03 (-0.05, -0.01)[b] | 20.6 | -0.03 (-0.05, -0.00)[c] | 24.0 |
| | Total | -0.11 (-0.15, -0.08)[a] | | -0.15 (-0.19, -0.11)[a] | | -0.11 (-0.15, -0.07)[a] | |
| Dental sepsis | Direct | -0.09 (-0.16, -0.02)[b] | 65.5 | -0.11 (-0.19, -0.03)[b] | 59.0 | -0.07 (-0.15, 0.01) | 52.9 |
| | Indirect via oral impacts | -0.05 (-0.08, -0.02)[b] | 34.5 | -0.07 (-0.11, -0.04)[a] | 41.0 | -0.06 (-0.10, -0.03)[b] | 47.1 |
| | Total | -0.14 (-0.20, -0.08)[a] | | -0.18 (-0.26, -0.11)[a] | | -0.14 (-0.21, -0.06)[a] | |

([a]p<0.001, [b]p<0.01, [c]p<0.05; Structural Equation Models)

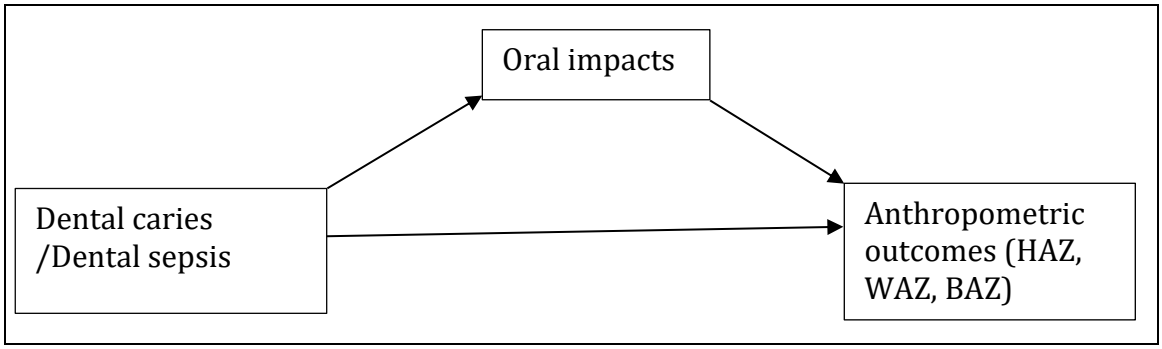


Figure 1 Path diagram for the conceptual model of main exposures, outcomes and potential mediator.