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Review Article

Prevention of Early Childhood Caries along with Dental Caries in Children and Adolescents Specifically Associated with Obesity in Modern Day Epidemic of Obesity with Oral Salivary Biomarkers-A Short Review

Kulvinder Kochar Kaur[1]*, Gautam Allahbadia[2] and Mandeep Singh[3]

[1]*Scientific Director, Dr Kulvinder Kaur Centre for Human Reproduction 721, Jalandhar, Punjab, India*
[2]*Scientific Director, Ex-Rotunda-A Centre for Human Reproduction 672, Bandra, Mumbai, India*
[3]*Consultant Neurologist, Swami Satyanand Hospital, Jalandhar, Punjab, India*

***Corresponding Author:** Kulvinder Kochar Kaur, Scientific Director, Dr Kulvinder Kaur Centre for Human Reproduction 721, Jalandhar, Punjab, India.

Received: October 01, 2026; **Published:** October 23, 2026

Abstract

Earlier we reviewed role of poor oral hygiene with obesity, association of obesity, diabetes mellitus and oral health, prevention of childhood obesity with breast feeding's role, correlation of poor oral health (POH) with obesity in extremes of age with role of excessive sugars in childhood obesity and oral salivary molecule use for prognosis of cardiovascular events like acute myocardial infarction, obesity and oral health-emphasis on early childhood caries (ECC) and trying to implement prevention strategies right from neonatal age besides updating on other causes of the same. With the escalating obesity worldwide there is escalating incidence of ECC as well as dental caries in children in addition to, adolescents with poor eating behaviours along with escalated consumption of sugary foods, as well as beverages which apart from leading to obesity result in ECC in addition to predisposes the generation further impacting bacterial ecology, inclusive of the colonisation of *Streptococcus mutans*, whose existence is a robust anticipator of future caries incidence in young children as well as cariogenic dietary fashion guarantees shaping acidogenic bacterial populations in the oral cavity. Thereby right at childhood stage eating habits have to be targeted with salivary biomarkers guiding in therapy.

Keywords: *Early Childhood Caries (ECC); Dental Caries; Children; Adolescents; Acidogenic Bacterial Populations; Streptococcus mutans*

Introduction

Dental caries is a prevalent disease with plethora of etiological factors as well as a worldwide public health botheration [1]. It starts with the demineralization of hard dental tissues by organic acids generated from cariogenic bacteria in dental plaque biofilms [2]. i) Cariogenic bacteria, ii) Substrates, iii) Host factors (Inclusive of the (a) quality in addition to (b) quantity of saliva), along with iv) Time are the four imperative factors that directly account for the generation of dental caries [3]. Additionally, factors for instance v) Socioeconomic status along with vi) Oral hygiene practices further possess a part [4,5]. Obesity is one other public health botheration with an escalating worldwide prevalence. Adipose tissue (AT) liberates numerous adipokines for instance i) Interleukin-6 (IL-6) as well as ii) Tumor necrosis factor-α (TNF-α), resulting in low grade inflammation in the body [6]. Central obesity, has been further acknowledged as i) Abdominal or ii) Visceral obesity, is an escalated accrual of abdominal (specifically visceral) fat, that is intricately associated with metabolic changes

in addition to an escalated risk of variable diseases, for instance i) Type 2 diabetes (T2D) along with ii) cardiovascular disease (CVD) [7]. Adolescent obesity is a cardiometabolic risk factor that escalates adult mortality [8]. Owing to obesity as well as dental caries share plethora of risk factors, their associations have been substantially assessed. An earlier meta-analysis illustrated a positive association amongst dental caries in addition to obesity in preschoolers [9]. Earlier studies by Peng., *et al*. [10], from Deng's group further observed that caries encounter was correlated with central obesity in 5-year-old children [10] as well as 12-year-old students [11] in Hong Kong. Further follow-ups of the 12-year-old cohort at the ages of 15 along with 18 years corroborated a longitudinal correlation amongst dental caries in addition to, central obesity [12]. Nevertheless, the mechanistic modes that lie beneath continue to be uncharted. A new insight of the association amongst oral health as well as adiposity status emphasizes the requirement for further research utilization of combined strategies to evaluate oral health status in addition to adiposity status crosstalk to generate biomarkers of oral health status (i.e. dental caries encounter) [13]. One probable indicator of such association is differences in salivary characteristics [14]. Saliva possesses variable meaningful working, inclusive of i) Buffering, ii) Lubricating, iii) Hampering microorganisms, iv) Sustenance of oral hygiene, v) Digesting food, along with vi) Immune defense [15]. Salivary quantities of inflammatory adipokines for instance IL-6 as well as TNF-α have been escalatingly evaluated in reference to oral in addition to systemic situations, along with both have been documented to be escalated in patients with dental caries [16]. One earlier study suggested a plausible association amongst salivary IL-6 quantities, dental caries, in addition to obesity; the median salivary IL-6 quantities were documented to be i) The maximum frequent in children with obesity as well as cavitated carious disfigurements, ii) with subsequent frequent in the ones with normal weight in addition to cavitated carious disfigurements, along with iii) followed by the ones with normal weight as well as iv) without cavitated carious disfigurements [17]. Nevertheless, that study was inclusive of group sizes which were not even as well as did not totally aid in plausible confounding factors, that might restrict the generalizability of the observations. Until now, clarification have not been yielded by studies on such biomarkers regarding the association amongst dental caries as well as obesity [16]. Thus, requirement exists to isolate greater particular biomarkers.

Apart from frequently documented adipokines, researchers have illustrated escalating significance in the ancient enzymes phospholipases A_2, which catalyze the hydrolysis of phospholipids as well as generate bioactive mediators implicated in inflammation in addition to metabolic controlling. Certain phospholipases A_2 family members have been associated with obesity, along with systemic diseases, illustrating probability in the form of diagnostic or prognostic markers [18]. Amongst them, i) Lipoprotein-associated phospholipase A_2 (Lp-PLA_2) as well as ii) Group IIA secretory phospholipase A_2 (sPLA_2-IIA) delineate inflammatory markers linked to both obesity [19-21] in addition to atherosclerotic cardiovascular disease (CVD) [22], along with have further been involved in oral inflammation [23,24]. Recently, the expression of Lp-PLA_2 was corroborated in the minor salivary glands of patients with primary Sjogren's syndrome in addition to controls with sicca complaints [25], suggesting that Lp-PLA_2 is locally generated in salivary tissues, along with might be germane to salivary biomarker studies. Serum amyloid A (SAA) is a pro-inflammatory adipokine [26] correlated with plethora of oral diseases, for instance chronic periapical periodontitis [27]. Despite, such markers were estimated in saliva [28,29], maximum research has concentrated on their circulating quantities in blood samples. Their correlation with i) Dental caries, ii) Obesity, along with iii) Salivary gland working continues to be uncharted. as well as Inflammatory alterations in salivary glands might lie beneath changed salivary profiles found in persons with obesity. For instance, inflammatory cell infiltration has been estimated in the salivary glands of patients with early-stage Sjogren's syndrome, despite the lack of meaningful tissue damage, indicating that inflammation possesses the capability of impacting acinar cell working as well as salivary liberation [30]. Correspondingly, obese Zucker rats illustrate escalated quantities of inflammatory markers in salivary glands in addition to generated unchecked dental caries [31]. While such models vary from human adolescents, they embrace the posit that obesity- associated low-grade inflammation possesses the capability of causing dysfunctional salivary gland working, hence impacting saliva constitution as well as caries generation [32]. In reference to this, i) Lp-PLA_2, ii) sPLA_2-IIA in addition to iii) SAA, in the form of inflammation-associated markers, might yield understanding into the obesity-caries association via salivary mechanistic modes. The objective of the present study was to evaluate the relationship amongst salivary profiles

(physicochemical criterion, inclusive of i) Flow rates, ii) pH, as well as buffering capability; in addition to biomarker quantities, inclusive of i) Lp-PLA_2, ii) sPLA_2-IIA, iii) SAA, iv) IL-6, along with v) cariogenic bacteria (*Streptococcus mutans*), vi) dental caries, as well as vii) adiposity status (central obesity) in Hong Kong adolescents.

Thus Deng., *et al*. [33] conducted a cross-sectional study implicating 184 adolescents aged 12-15 years. Anthropometric determinations inclusive of as well as dental assessments were performed. The participants were sex- in addition to age-matched, along with classified into four groups: 1) Normal waist-to-height ratio (WHtR) without caries (NW+NC), 2) High WHtR as well as without caries (HW+NC), 3) With normal WHtR as well as caries (NW+C), or 4) High WHtR with caries (HW+C). Unstimulated in addition to stimulated saliva samples were acquired to examine i) Salivary flow rates, ii) pH, along with iii) Buffering capability. Salivary biomarkers, inclusive of i) Lipoprotein-associated phospholipase A_2 (Lp-PLA_2), ii) Secretory phospholipase A_2 group IIA (sPLA_2-IIA), iii) Interleukin-6, as well as iv) Serum amyloid A, were quantified by utilization of enzyme-linked immunosorbent assay (ELISA). Quantitative real-time polymerase chain reaction (qPCR) was utilized to estimate *Streptococcus mutans* in addition to total bacterial load.

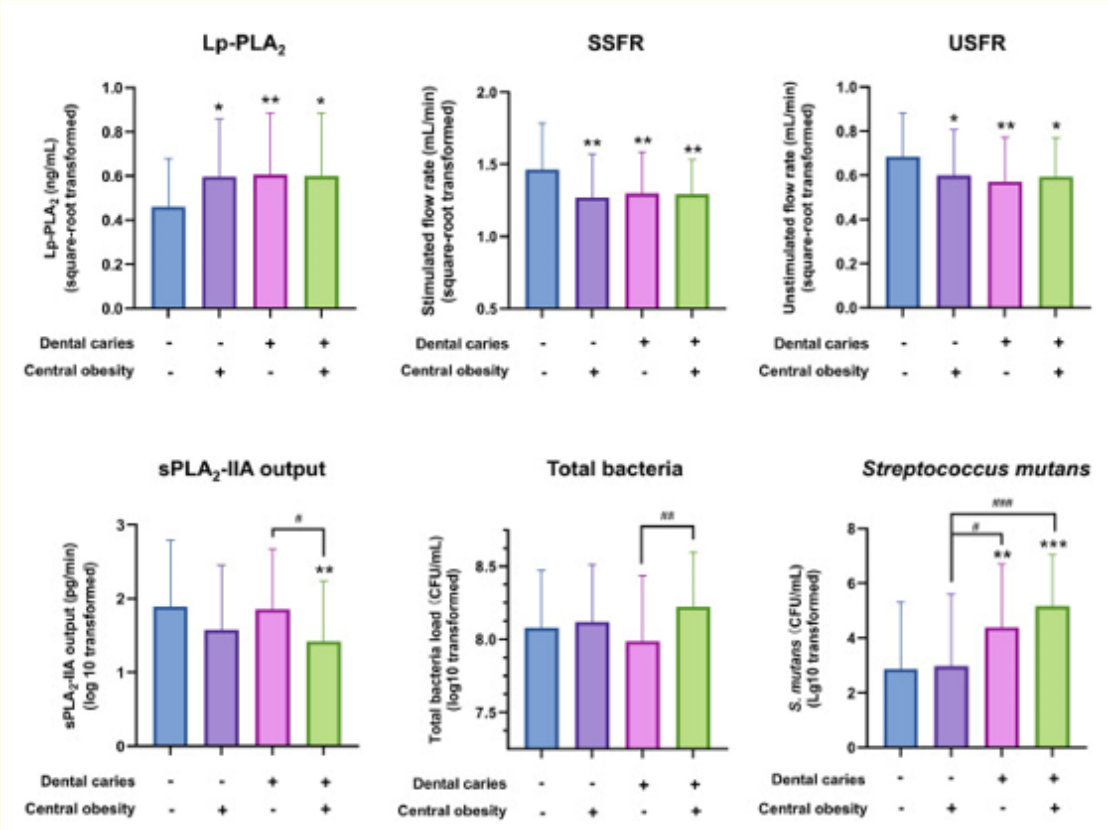


Figure 1: *Courtesy ref no 33-Comparisons of salivary parameters and biomarker levels in adolescents with dental caries and central obesity. Data are presented as mean ± standard deviation; #P < 0.05, ##P < 0.01, ###P < 0.001; *P < 0.05, **P < 0.01, ***P < 0.001, vs. caries-free controls without central obesity. Lp-PLA2, lipoprotein-associated phospholipase A2; sPLA2-IIA, group IIA secretory phospholipase A2; SSFR, stimulated salivary flow rate; USFR, unstimulated salivary flow rate.*

They observed meaningfully variable i) Salivary Lp-PLA_2 quantities, ii) sPLA_2-IIA output, iii) Total bacterial load, iv) *Streptococcus mutans* enrichment, as well as v) Flow rates were found among the four groups. The NW+C, HW+NC, in addition to HW+C groups possessed meaningfully greater salivary Lp-PLA_2 quantities, along with meaningfully lesser salivary flow rates in contrast to the NW+NC group. The HW+C group possessed a meaningfully lesser salivary sPLA_2-IIA output in addition to greater total salivary bacterial load in contrast to the NW+C group. Therefore, conclusions drawn were that both dental caries as well as central obesity are correlated with escalated Lp-PLA_2 in addition to diminished salivary gland liberation working. Adolescents with both disorders illustrated i) Diminished salivary sPLA_2-IIA output as well as ii) Escalated salivary bacterial load, that might indicate jeopardized oral antimicrobial status that makes further evaluation mandatory. The clinical significance of this study was that changes in salivary profiles might yield preparatory understanding into the mechanistic modes that associates obesity in addition oral health, plausibly guiding regarding the generation of greater customized oral health programs for children along with adolescents with obesity.

Earlier we reviewed role of poor oral hygiene with obesity, association of obesity, diabetes mellitus and oral health, prevention of childhood obesity with breast feeding's role, correlation of poor oral health (POH) with obesity in extremes of age with role of excessive sugars in childhood obesity and oral salivary molecule use for prognosis of cardiovascular events like acute myocardial infarction, "obesity and oral health-emphasis on early childhood caries (ECC) and trying to implement prevention strategies right from neonatal age besides updating on other causes of the same [34-38].

We acknowledge that ECC (Early Childhood Caries) possesses a plethora of etiological factors, where dietary factors possess a specifically important part. Early childhood delineates a pivotal time course at the time of which exposure to variable foods in addition to flavours meaningfully shapes dietary preferences as well as future eating behaviours. Early dietary fashions possess the capability of further impacting bacterial ecology, inclusive of the colonisation of *Streptococcus mutans*, whose existence is a robust anticipator of future caries incidence in young children. Early exposure to sugary foods, as well as beverages possess the capacity of, hence, possessing meaningful sequelae, laying the groundwork for future cariogenic dietary fashion or guaranteeing shaping acidogenic bacterial populations in the oral cavity.

The American Heart Association advocates preventing added sugars in food along with beverages for children below two years of age. Additionally, the American Academy of Pediatrics recommends against installation of fruit juices prior to 12 months of age in addition to advocates restricting their ingestion to no greater than weekly four instances for children amongst one to three years age.

ECC further shares overlapping risk factors with other noncommunicable diseases (NCDs), for instance CVD, type 2 diabetes (T2DM), as well as obesity. Hence, approaches in reference to the avoidance of dental caries need to possess the objective of apart from guaranteeing appropriate oral hygiene nonetheless further facilitating proper dietary habits from early childhood—implying, restricted consumption of sugar-sweetened beverages (SSBs) in addition to refined foods.

In reference to that, parents possess the capacity of, along with need to possess a vital part by offering their children healthier dietary choices in addition to behaviours.

Hence we need to take into account ECC in the form of a condition whose initiation—as well as resolution—implicates the family milieu: with children acquiring habits from the ones who look after their needs in addition to the ones regulating quality in addition to b) quantity of their food consumption. Despite alteration a family's dietary habits is bothersome, we need to acknowledge altering a family's dietary habits that arbitration is needed in ECC cases—specifically early forms—imperatively need a strategy which takes into account the family [39].

Conclusion

Here we have emphasized on the development of poor oral health (POH) right from childhood and adolescents with probability in the form of diagnostic or prognostic markers in obesity associated with dental caries. Additionally, the significance of avoidance of ECC in childhood for prevention of dental caries in adolescents as well as stringent controlling of child's eating habits for avoidance of both obesity in addition to ECC starting right at the level of family eating environment.

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Volume 25 Issue 11 November 2026
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