Abstract: Obesity is a chronic disease characterized by excess body fat, which can lead to other health problems, including insulin resistance, non-alcoholic fatty liver disease, polycystic ovary syndrome, hypertension, dyslipidemia, sleep apnea, asthma, heart attack, stroke, atherosclerosis and metabolic syndrome. Currently, obesity and dental caries are major public health concerns and dietary habits are a very important common component of their etiological factors, showing some correlation with the sociodemographic characteristics of individuals presenting these diseases. In relation to caries experience, the literature suggests a correlation between obesity and dental caries in children and adolescents, in primary and/or permanent dentition, though divergent results exist regarding assessment based on the method recommended by the WHO (1997), i.e., restricted to carious lesions with cavitation. Some studies indicate greater prevalence of proximal carious lesions in obese adolescents compared with those with normal weight.
Salivary changes, such as the concentrations of phosphate, sialic acid, proteins and immunoglobulins and in peroxidase activity could explain the increased probability of obese children presenting greater risk of dental caries. Thus, it is important to consider the contribution of salivary parameters in caries experience of overweight children and adolescents and the implementation of preventive measures in this population.

**Keywords:** obesity; overweight; dental caries; saliva; child; adolescent

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1. Introduction

Childhood obesity is one of the most serious public health challenges of the 21st century [1]. The problem is global and is steadily affecting many low- and middle-income countries, particularly in urban settings, and prevalence has increased at an alarming rate. Worldwide, in 2010, the number of overweight children under the age of five is estimated to be over 42 million, and close to 35 million of these are living in developing countries. Overweight and obese children are likely to remain obese into adulthood and more likely to develop noncommunicable diseases, such as diabetes and cardiovascular diseases, at a younger age. Childhood overweight and obesity, and their related diseases require high priority [1].

The adverse effects of obesity are related not only to total body weight, but also the distribution of stored fat. Central or visceral obesity, characterized by the accumulation of fat in the trunk and the abdominal cavity, is associated with a greatly increased risk of numerous diseases, in contrast to excessive accumulation of fat diffusely distributed throughout the subcutaneous tissue [1]. Obesity is associated with several chronic diseases, including insulin resistance, non-alcoholic fatty liver disease, polycystic ovary syndrome, hypertension, dyslipidemia, sleep apnea, asthma, thrombosis, heart attack, stroke, atherosclerosis and metabolic syndrome [2–5].

The increase in adipose tissue characteristic of the development of obesity causes changes in the release of important hormones and cytokines (adipokines), including adiponectin, leptin, TNF-alpha and IL-6 [6]. These adipokines can act on cells of the immune system, modifying their function. The greater the quantity of body fat, the lower the adiponectin production, which is associated with obesity and insulin resistance [7]. As a result of diminished production, adiponectin possesses anti-inflammatory action and inhibits the action of TNF-α, IL-6 and the consequent induction of IL-10 production [8]. Leptin is another hormone secreted by adipocytes. A study of overweight children and adolescents showed that BMI is strongly associated with leptin plasma levels [9]. Research has shown that leptin acts similarly to cytokines modulating the function of leukocytes. Galgani *et al.* (2010) demonstrated that leptin modulates the survival of CD4+ T lymphocytes through leptin-specific receptors by stimulating the production of Th1 and Th17 cytokines [10]. The cytokine TNF-alpha, released in large quantities by adipose tissue in obesity, can induce an increase in the systemic inflammatory response and induce apoptosis of some circulating cells, altering the immune homeostasis [11]. IL-6 is produced by adipocytes, monocytes, macrophages, lymphocytes and fibroblasts and stimulates T cell proliferation, activation, apoptosis and cytotoxicity. Besides activating
the immune system, the increase in IL-6 induces hepatic synthesis of acute phase proteins and increases the activity of the hypothalamic-pituitary-adrenal axis, altering metabolic responses [12].

Adipose tissue acts as an endocrine organ directly modulating the function of leukocytes; therefore, any change in the number and size of adipocytes can promote imbalance in the immune homeostasis.

2. Obesity and Dental Caries

According to Mathus-Vliegen et al. (2007), obesity (BMI ≥ 30 kg/m²) is related to several aspects of oral health, including dental caries [13]. Dental caries and obesity are multifactorial diseases related to poor eating habits and show a close relationship with the sociodemographic characteristics of individuals presenting these diseases [14].

The literature seems to confirm a positive correlation between dental caries and BMI, and obesogenic behavior, including snacking habits in early childhood, could be a predictor for caries development in adolescence [15]. Thus, changes in lifestyle and eating habits observed since the mid 1990s, such as the increased intake of carbohydrate-rich and high-calorie foods, could have contributed to the increase in dental caries prevalence and obesity in recent years [16,17]. It is important to emphasize that it is well established that controlling the consumption of fermentable carbohydrates and sugars in the diet, in association with good oral hygiene, can reduce the incidence of dental caries in obese children [18].

Conflicting results have been reported by studies linking obesity and caries experience based on the method recommended by the WHO (1997), i.e., limited to the detection of caries lesions with cavitation [19–24]. Few clinical studies have demonstrated the relationship between obesity and dental caries in children [14,15,25,26] and adolescents [27] or even in both dentitions, as verified by Sharma and Hegde (2009), who reported that overweight/obese children presented increased dental caries prevalence in both dentitions compared with normal weight children, in relation to the dmf and DMF-T indices [28].

Regarding the dmf/DMF-S index, some studies have observed a higher number of proximal caries in both children and adolescents with obesity. Hilgers et al. (2006) assessed children aged 8 to 11 years-old and concluded that a high BMI is associated with increased incidence of proximal caries in permanent molars [29]. Similarly, Alm et al. (2008) investigated the prevalence of proximal caries in the posterior teeth of children aged 15 years-old and its association with the rate of age-specific body mass (isoBMI), observing that overweight/obese adolescents presented greater prevalence of proximal caries lesions than those of normal weight [15].

In 2006, a systematic review of the literature conducted by Kantovitz et al., included seven studies published between 1984 and 2004. Only five studies involved samples of children and of these, only three contained high levels of evidence [30]. Only one study determined a positive correlation between dental caries and obesity in a sample of 842 children aged 6 to 11 years old [25]. Another study reported no correlation in a sample of more than five thousand children aged 3 years old [31], while yet another was unable to predict caries experience based on BMI in more than 500 children aged 5 to 13 years old [32]. Thus, the review concluded that randomized controlled trials are required to demonstrate a possible relationship between dental caries and obesity.
More recently, another systematic review of the literature conducted by Hooley et al. (2012) affirmed that there is evidence to suggest that dental caries are associated with both low and high BMI [33]. Although the exact nature of these associations remains unclear, it is possible that different factors are involved in the development of caries in children with high and low BMI and with different socioeconomic profiles. Such findings suggest the implementation of combined strategies to control both caries and obesity simultaneously. However, the elaboration of additional studies is required to evaluate whether associations can be determined between these diseases and among their predictors. Special attention should be given to longitudinal studies that aim to evaluate the association between early childhood caries and health outcomes in adolescence and adulthood. The inclusion of young children (aged 0–6 years-old) in the samples, together with dietary habits and health-related behaviors developed during the preschool phase of growth and family influences on the development of these patterns also require analysis.

3. Obesity and Salivary Parameters

Saliva properties are essential for the protection of the oral cavity, the oropharyngeal and gastrointestinal epithelia, and to moisten the soft and hard tissues of the cavity [34]. The functions of protecting the teeth, achieved by the buffer capacity, maintenance of supersaturated calcium and phosphate concentrations in relation to hydroxyapatite, participation in the formation of acquired pellicle and antimicrobial and digestive activities are related to specific components of this fluid. Given its multiple actions, saliva is an extremely important fluid for both the systemic and oral health of individuals [35,36].

The fluid compound present in the oral cavity in contact with the teeth and mucous, referred to as whole saliva, is composed of secretions from the parotid, submandibular, sublingual and minor salivary glands. The saliva produced by the parotid gland is purely serous and when stimulated, a fine, aqueous saliva is observed that is rich in amylase and accounts for 25% of the total saliva production. The submandibular glands are mixed glands, which secrete saliva that is more viscous and rich in mucin, and are responsible for almost 70% of the saliva produced. The sublingual glands, the smallest among the principal salivary glands, are exclusively mucosal. The minor salivary glands produce less than 10% of the total volume of saliva and are responsible for most of the secretion of salivary proteins [37].

Regarding oral health, the rate of unstimulated salivary flow is more important than when stimulated [36]. The contribution of the parotid glands to unstimulated whole saliva is 25%, the submandibular glands contribute 60%, the sublingual glands 8% and the minor salivary glands 7%. Factors that affect the rate of unstimulated salivary flow include the degree of hydration, circadian rhythm and the use of certain drugs [37].

Diminished salivary flow is considered an important factor for dental caries [38]. In a cross-sectional study, Modeer et al. (2010) observed that childhood obesity is associated with reduced flow and dental caries, similar to that observed in obese adults by Flink et al. (2008), suggesting that inflammatory mediators play an important role in hypofunction of the salivary glands in obese individuals [39,40].
Potentially, the degree of hydration is the most important factor in the rate of salivary flow. When the water content of a person diminishes by 8%, the rate of salivary flow is reduced to virtually zero [41]. Osmolarity is the number of osmotically active solute particles contained in 1 liter of solution and is expressed in milliosmol of solute particles per kilogram of water. Osmolarity has been used to evaluate dehydration and shows a significant correlation with salivary flow [42]. Salivary osmolality is a noninvasive method for evaluating saliva volume and concentration in a real-time, with excellent accuracy and reliability. It presents less variation than individual salivary flow obtained by the collection of unstimulated saliva [43] and greater discriminatory capacity. It is worth emphasizing that the available literature seems to contain no reports concerning the relationship between salivary osmolality and obesity in childhood and adolescence.

Regarding salivary antimicrobial factors, these are divided into nonimmune (lysozyme, peroxidase, lactoferrin, amylase and agglutinins) and immune agents (immunoglobulin A, G and M) [44]. Overweight and obese children showed changes in the concentrations of phosphate, sialic acid and proteins and in peroxidase activity, favoring the development of dental caries [45].

IgA secretion by the parotid is 500-fold greater compared with serum [46,47], but 77% of salivary IgA is derived from serum [48]. Salivary IgA is responsible for immune response in the oral cavity, inhibiting the adhesion of Gram-negative bacteria on the epithelium and neutralizing certain bacterial toxins [49], thus providing a primary barrier to infection and bacterial colonizaton in the oral environment. Regarding periodontal disease, a proportional relationship exists between the concentration of IgA and periodontal disease [46,50]. The accumulation of biofilm facilitates an increase in Actinobacillus actinomycetemcomitans, which stimulates B cells to produce IgA and is also secreted in the gingival fluid [46].

Inflammatory and immunological reactions to dental biofilms are essential in determining the pathological process, because these reactions often prevent the proliferation of bacteria and/or their dissemination. Therefore, bacterial invasion of host tissue determines the development of gingivitis and periodontitis [51].

According Ranadheer et al. (2011), increased levels of sIgA provide a mechanism of protection against caries in caries-active individuals and have an important role in the control of dental caries [52]. Thaweboon et al. (2008) reported that children with rampant caries showed elevated levels of sIgA in the oral cavity, suggesting that the increase in these levels is related to host exposure to cariogenic microorganisms [53].

Data from clinical studies provide information concerning the relationship between obesity and immune function, showing that obesity promotes systemic inflammation [54,55]. Zuniga-Torres et al. (2009) observed a positive correlation between IgA and IgM with skinfold thickness ($r = 0.192$, $p = 0.041$; $r = 0.221$, $p = 0.018$, respectively) and total fat measured by bioimpedance ($r = 0.243$, $p = 0.009$) [56]. Pallaro et al. (2002) compared 155 obese children aged 6–13 years-old with a similar group of non-obese from the same economic class, by collecting blood (C3c and IgA) and saliva (sIgA) samples, and observed lower levels of sIgA in the latter group [57].

The development of a line of research involving inflammatory biomarkers to evaluate the risk of caries and other oral diseases in children and adolescents who are overweight or obese is now fundamental [58].
4. Final Considerations

Obesity and dental caries are major public health concerns, and their etiological factors are intricately linked to dietary habits and are correlated with certain sociodemographic characteristics of individuals presenting these diseases.

Analysis of the changes in the constitution of saliva, such as phosphate, sialic acid, protein and immunoglobulin concentrations and peroxidase activity, could assist in determining why obese children seem to show greater risk of dental caries. The contribution of salivary parameters in caries experience of overweight children and adolescents should be considered, together with the implementation of preventive measures in this population.

Increased calorie intake associated with sugars and carbohydrates, especially when associated with physical inactivity, has serious implications for childhood obesity. Thus, dietary counseling is an important complement to oral health education. Given the impact on health, dental professionals should help identify patients at risk of obesity and provide counseling and referral when appropriate.

Future research that assesses the associations between different BMI indices, salivary parameters and dental caries experience, determined by clinical criteria that consider lesions in the early stages of development, and that explore related socioeconomic factors is recommended.

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Conflicts of Interest

The authors declare no conflict of interest.

References


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