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ORIGINAL ARTICLE

Salivary bacteria and oral health status in medicated and non-medicated children and adolescents with attention deficit hyperactivity disorder (ADHD)

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Abstract
Objectives Attention deficit hyperactivity disorder (ADHD) is a childhood neurological disorder. Studies have shown that children with ADHD are more prone to caries than those without. The study investigated children diagnosed with ADHD, both with and without pharmacological intervention, and the following: DMFT\dmft, plaque index (PI), mutans streptococci (MS) levels, lactobacilli (LB) levels, salivary flow, salivary buffer capacity, oral hygiene, and diet.
Study design DMFT/dmft index, PI, MS and LB levels, salivary flow, and salivary buffer capacity were examined in three groups of children: ADHD1—diagnosed with ADHD with no pharmacological intervention (N=31), ADHD2—treated with medications for ADHD (N=30), and a healthy control group (N=30). Diet and oral health habits were assessed through questionnaires completed by parents.
Results There were no differences in the DMFT/dmft index, MS and LB counts, salivary buffer capacity, and parent reported diet and oral health behavior between the three groups. Children with ADHD demonstrated a higher plaque index.
Conclusions Although children with ADHD did not report different diet and oral health behavior from children without ADHD, this group had significantly higher levels of plaque than the control group, which combined with hyposalivation may be a risk factor for caries at an older age.
Clinical relevance Medicated and non-medicated ADHD children were similar to control children in their caries rate, MS and LB counts, salivary buffer capacity, and diet and oral health behavior. They differed in the amount of plaque found on their teeth. As a group, ADHD children demonstrated hyposalivation compared with the control.

Keywords ADHD · Oral health status · Mutans streptococci (MS) · Lactobacilli (LB) · Buffer capacity

Introduction

Attention deficit hyperactivity disorder (ADHD) is a chronic, pervasive childhood disorder characterized by inattention, developmentally inappropriate activity level, low frustration tolerance, impulsivity, poor organizational behavior, distractibility, and inability to sustain attention and concentration. ADHD is the most common childhood-onset behavioral disorder, affecting approximately 5 to 10 % of children and adolescents [1]. Boys are affected about eight times more than girls [2]. Genetic and environmental etiologies that implicate the neurotransmitter dopamine have been proposed for ADHD [3]. The literature on brain imaging supports the presence of abnormalities in structure (smaller size) and function (hypoactivation) of critical brain regions related to dopamine. Molecular genetics data clearly support the associations between ADHD and dopamine-related genes (DRD4 and DAT) [3].

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Few studies reported higher DMFT or dmft (D;d—decay, M;m—missing, F;f—filled, T;t—teeth) scores among ADHD children compared with control [4–6]. Broadbent et al. found that children with ADHD had nearly 12 times the odds of having a higher DMF score than children without ADHD [5]. On the other hand, Blomqvist et al. [7] found in their study that children with ADHD did not exhibit statistically significant higher caries prevalence than healthy children.

Most children being treated for ADHD are managed with a combination of behavioral and pharmacologic therapies. Current drugs employed in the treatment include stimulants and non-stimulants. Two kinds of stimulants are available: methylphenidate, a stimulant drug considered to be an indirect dopamine agonist that acts by blocking the reuptake of the neurotransmitter dopamine, and dextroamphetamine [8]. Xerostomia is mentioned in the literature as one of the adverse effects of methylphenidate [9]. While some authors reported that methylphenidate causes dry mouth [10], others did not find any change in saliva flow rate [11].

Hyposalivation or dry mouth reduces salivary buffering capacity, thus making the oral cavity more acidic [12]. The levels of mutans streptococci and lactobacilli, which are major caries pathogens, are higher in patients with hyposalivation [12].

The current study hypothesized that lower salivary flow rates in medicated ADHD children (an outcome of the medicament) would result in lower buffer capacity and higher bacterial count. The purpose of the study was to compare DMFT/dmft, plaque index, buffer capacity, mutans streptococci (MS) and lactobacilli (LB) levels, and oral health behavior in children with ADHD, medicated or not, with those of healthy children.

Materials and methods

The study protocol was approved by the Institutional Human Subjects Ethics Committee of the Hebrew University, Hadassah School of Dental Medicine, Jerusalem, Israel. Informed consent was obtained from all parents or legal guardians of participating children.

This study comprised two groups of children diagnosed with combined type ADHD according to a specific set of symptoms: inattention, hyperactivity, and impulsivity, as described in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition and treated by pediatric neurologists (I.M) in the pediatric department of Hadassah Hospital, Jerusalem. Only healthy children aged 5–18 not taking medications other than methylphenidate or dextroamphetamine were included in the study. Children who took other medication on the day of the check-up (e.g., antibiotics) were excluded from the study. One group (ADHD1) comprised 31 children who did not receive any medication for ADHD at the time of the study. Five of them had received medication in the past. Of them, two stopped after a week due to adverse effects and the other three stopped taking medication at least a few months prior to their participation in the study. The second group (ADHD2) comprised 30 children who were currently being treated with methylphenidate for at least 1 month previous to their participation in the study. All children in both study groups were otherwise healthy. The control group comprised 30 healthy children without ADHD, who attended the clinic of the pediatric dentistry department in the Hadassah School of Dental Medicine in Jerusalem for routine check-ups.

ADHD1 group comprised 24 boys and seven girls; aged 5.9–16.7 years (mean age 10.3±2.8 years). ADHD2 group comprised 19 boys and 11 girls; aged 6.7–17.2 years (mean age 11.8±3.5 years). Thirty children (21 boys and nine girls) comprised the control group; aged 6.0–17.8 years (mean age 10.7±2.9 years). Among the ADHD2 group, 26 were taking Ritalin® hydrochloride and four were taking Concerta®. The duration of pharmacological intervention was 1 to 120 months. The medication was taken between 4 and 7 days a week. Only three children reported poor compliance for the medication.

Clinical examination

Medical history and medicament data for all participants were gathered from medical records. All study and control children were examined by the same investigators (N.B and A.F.N). The dental examination included dentition charting, DMFT/dmft index score, and plaque index (PI) (according to Silness and Loe [13]). Examinations were conducted using a dental mirror [14].

Salivary sampling

Whole saliva samples were collected from the ventral part of the tongue and the oral vestibulum using a sterile cotton pallet. Samples were collected in the morning, at least 2 h after eating. Salivary flow rate was calculated as the amount of saliva collected during 1 min [14]. One sample was collected from each subject.

Saliva analysis was performed using standard bioassay and plating procedures on a caries risk test (CRT) (Ivoclar Vivadent Inc.) used to determine the MS and LB count in saliva by means of selective culture media. The CRT kits were incubated at 37 °C for 48 h. Enumeration of bacterial growth was conducted as semi-quantities ranking according to manufacturer's instructions.

Buffer capacity

Salivary buffer capacity was assessed using pH indicators from CRT (Ivoclar Vivadent Inc.) and was evaluated using a color scale provided by the manufacturer.

Oral health behavior

Parents and participants older than 15 years of age were asked to answer a questionnaire translated from that used by Blomqvist et al. [7] regarding their children's diet and oral health behavior, including complaints about dental pain, dental anxiety, bruxism, regular/irregular eating habits, major beverage consumed during the day, and tooth brushing habits. This questionnaire had been validated in a Swedish study [15]. The dietary habits component included dietary behaviors that had been found to be most predictive of a risk of caries in a clinical setting in the USA [16].

Statistical analysis

Sample size

The statistically significant sample size needed was determined at the design stage of the study. The mean DMFT for participants with ADHD was assumed to be twice that of participants without ADHD (based on Blomqvist et al. [9]). It was assumed that the DMFT for the control group would be 2.5±3 (mean±SD), and for those with ADHD, 4.5±3. With the significance level set at 5 % (one-tailed) and 80 % power, it was calculated that 29 children would be needed for each study group. Accordingly, 31 and 30 participants for the study and control groups respectively were recruited.

Data were analyzed using SPSS software Chicago, IL, USA. The analysis consisted of basic descriptive statistical analysis assays; ANOVA test was used to compare the DMF/dmf, bacterial count, and buffer capacity between the three groups. Chi-square analysis was used to compare plaque index and oral health behavior between the three groups. Kruskal–Wallis test was used to check for correlation between DMF/dmf and plaque index. A general linear model was performed to study oral health outcome (DMFT/dmft, MS counts, and LB counts) between groups. Significance level was set at $p \leq 0.05$.

Results

Study groups and control group were not different in either age or gender (ANOVA (F=2,88)=1.818; p=0.618); χ^2 (df=2)=1.452; p=0.484, respectively). While there was no statistically significant difference (χ^2 (df=4)=9.011; p=0.068) in plaque index between the three groups, significantly higher levels of plaque were found in the ADHD group (ADHD1 + ADHD2) compared with the control group (χ^2 (df=2); p=0.024).

The ADHD2 group had the highest DMFT/dmft index and the ADHD1 group had the lowest DMFT/dmft index, with no significant difference (Table 1; ANOVA (F (2,88)=2.603; p=0.082). No statistically significant difference was found between the groups regarding buffer capacity (χ^2(df=4)=0.416; p=1), MS counts (χ^2(df=2)=1.352; p=0.509) and LB counts (χ^2(df=2)=1.092; p=0.579) (Table 2), dental anxiety (χ^2(df=6)=5.621; p=0.357), bruxism (χ^2 (df=2)=3.365; p=0.264), snack eating habits (χ^2(df=4)=2.298; p=0.706), and oral hygiene regarding the frequency of brushing (χ^2(df=6)=10.190; p=0.161).

Table 1 The DMFT/dmft index

	Number	Mean DMFT/dmft	SD
ADHD1 non-medicated	31	2.55	2.293
ADHD2 medicated	30	4.30	3.807
Control	30	4.10	3.595
Total	91	3.64	3.348

Statistically significant higher counts of mutans streptococci were found in correlation with higher D-decay (p=0.001). Using a general linear model, a correlation was found between buffer capacity and salivary flow rates (p=0.019, Pearson correlation=−0.245), between buffer capacity and MS counts (p=0.045, Pearson correlation=−0.211), and between buffer capacity and LB counts (p=0.008, Pearson correlation=−0.278) (Table 2).

A correlation was found between oral hygiene habits (brushing) and MS counts (Pearson correlation=−0.28; df=1; p=0.007; F=7.51). Consumption of cariogenic drinks was associated with higher DMFT/dmft (df=1; p=0.043; F=4.218)

Discussion

The main finding of the present study was that despite a higher plaque index in the ADHD groups, no significant differences existed in salivary buffer capacity and LB and MS counts between children with ADHD (with or without pharmacological intervention) and the control group (Table 2). The finding that higher plaque index in the ADHD group did not affect the DMFT/dmft index is contrary to previous studies who found a higher prevalence of caries in ADHD children [4, 5].

In spite of reported similar tooth brushing frequency in the ADHD group and the control group, the plaque index of ADHD children was significantly higher. This may be due to less efficient tooth brushing and/or to unreliable survey responds. Blomqvist et al. [9] reported that children with ADHD brushed their teeth less frequently than did children without ADHD and had worse dietary habits, but similar to the present study, they did not find any difference in caries prevalence.

In the present study, higher counts of mutans streptococci were found in correlation with higher D-decay, but no such

Table 2 Salivary buffer capacity and LB and MS counts between children with ADHD and control group

		ADHD1 Non-medicated	ADHD2 medicated	Control	Total
High buffer capacity	Count	19	19	18	56
	%	61.3 %	63.3 %	60 %	61.5 %
Medium buffer capacity	Count	10	10	10	30
	%	32.3 %	33.3 %	33.3 %	33.0 %
Low buffer capacity	Count	2	1	2	5
	%	6.5 %	3.3 %	6.7 %	5.5 %
MS higher than 10^5	Count	22	17	19	58
	%	71 %	56.7 %	63.3 %	63.7 %
MS lower than 10^5	Count	9	13	11	33
	%	29 %	43.3 %	36.7 %	36.3 %
LB higher than 10^5	Count	14	12	16	42
	%	45.2 %	40 %	53.3 %	46.2 %
LB lower than 10^5	Count	17	18	14	49
	%	54.8 %	60 %	46.7 %	53.8 %

MS mutans streptococci, *LB* lactobacilli

correlation was found regarding lactobacilli counts. Tanzer et al. [17] stress the essential role of mutans streptococci in the initiation of caries of smooth surfaces and question lactobacilli's role in induction of lesions. On the contrary, studies based on molecular techniques had found both mutans streptococci and lactobacilli to be dominant in advanced caries [18].

Contrary to the study's hypothesis that medicated ADHD children would have lower salivary flow rates and consequently lower buffer capacity and higher bacterial count, no differences were found between the ADHD groups and the control group regarding buffer capacity and LB and MS counts. A complementary study to the present one [14] found lower unstimulated salivary flow rate in ADHD children and young adults than in the non-ADHD group, which did not result in higher DMFT/dmft scores. The same study found no difference in salivary flow rates between medicated and non-medicated ADHD children. This may explain the findings that contradict the initial hypothesis of the present study. The findings of the present study are in accordance with the claim that the association between poor buffering capacity and caries is weaker than the association between low salivary flow and caries [19]. Also, when salivary pH was evaluated independently of buffer capacity, it was found to be a relatively poor indicator of caries risk [19].

The similarities between ADHD and control group in the bacterial counts, salivary buffering capacity, and oral hygiene reported habits may account for the similarities in DMFT/dmft scores in the present study. Nevertheless, as ADHD children were found to have a higher plaque index, dentists should emphasize the importance of oral hygiene and plaque control to ADHD children and to their caretakers. The correlation found in the present study between oral hygiene habits and MS counts and between cariogenic drinks consumption and higher DMFT/dmft give emphasis to this recommendation.

Strengths and limitations of the study

To our knowledge, only few studies have compared medicated and non-medicated ADHD patients. This study's findings disproved the hypothesis that medicated ADHD children are at a higher risk for carries than non-medicated ADHD children due to hyposalivation that is the presumed to be the outcome of the medications. Also, medicated children may cope better than non-medicated children with the assignment of effective tooth brushing twice a day. The findings of the present study may have clinical implication on carries risk assessments in ADHD patients. On a more theoretical level, further studies are needed to determine if ADHD actually influences salivary status, and if so, through what biological mechanisms. One possible such mechanism may be the dopaminergic receptors system [3, 20, 21]. A limitation of the study is that the reliance on questionnaires to explore oral hygiene habits, as the validity of self-reported questionnaires is less than optimal.

Conclusion

Although children with ADHD did not report different diet and oral health behavior from children without ADHD, this group had significantly higher levels of plaque than the control group, which combined with hyposalivation may be a risk factor for developing caries at an older age.

Conflict of interest The authors deny any conflicts of interest.

Reference

1. Wolraich ML, Hannah JN, Pinnock TY, Baumgaertel A, Brown J (1996) Comparison of diagnostic criteria for attention-deficit hyperactivity disorder in a county-wide sample. J Am Acad Child Adolesc Psychiatry 35:319–324
2. Zametkin AJ, Nordahl TE, Gross M et al (1990) Cerebral glucose metabolism in adults with hyperactivity of childhood onset. New Eng J Med 323:1361–1366
3. Swanson JM, Kinsbourne M, Nigg J et al (2007) Etiologic subtypes of attention-deficit/hyperactivity disorder: brain imaging, molecular genetics and environmental factors and the dopamine hypothesis. Neuropsychol Rev 17:39–59
4. Chandra P, Anandakrishna L, Ray P (2009) Caries experience and oral hygiene status of children suffering from attention deficit hyperactivity disorder. J Clin Pediatr Dent 34:25–29
5. Grooms MT, Keels MA, Roberts MW, McIver FT (2005) Caries experience associated with attention-deficit hyperactivity disorder. J Clin Pediatr Dent 30:3–8
6. Broadbent JM, Ayers KMS, Thomson WM (2004) Is attention deficit/hyperactivity disorder a risk for dental caries? Caries research 38:29–33
7. Blomqvist M, Holmberg K, Fernell E, Ek U, Dahllöf G (2006) Oral health, dental anxiety, and behavior management problems in children with attention deficit/hyperactivity disorder. Eur J Oral Sci 114:385–390
8. Cormier E (2008) Attention deficit/hyperactivity disorder: a review and update. J Pediatr Nurs 23:345–357
9. Blomqvist M, Holmberg K, Fernell E, Ek U, Dahllöf G (2007) Dental caries and oral health behavior in children with attention deficit hyperactivity disorder. Eur J Oral Sci 115:186–191
10. Friedlander AH, Yagiela JA, Paterno VI, Mahler ME (2006) The neuropathology, medical management and dental implications of autism. JADA 137:1517–1527
11. Pataki CS, Carlson GA, Kelly KL, Rapport MD, Biancaniello TM (1993) Side effects of methylphenidate and desipramine alone and in combination in children. J Am Acad Child Adolesc Psychiatry 32:1065–1072
12. Friedlander AH, Friedlander IK (1992) Dental management considerations in children with attention-deficit hyperactivity disorder. J Dent Child 59:196–201
13. Silness J, Löe H (1964) Periodontal disease in pregnancy II. Correlation between oral hygiene and periodontal condition. Acta Odontol Scand 22:121–135
14. Hidas A, Noy FA, Birman N et al (2011) Oral health status, salivary flow rate and salivary quality in children, adolescents and young adults with ADHD. Arch Oral Biol 56:1137–1141
15. Julihn A, Barr Agholme M, Grindefjord M, Modeer T (2006) Risk factors and risk indicators associated with high caries experience in Swedish 19-year-olds. Acta Odontol Scand 64:267–273
16. Mobley CC (2003) Nutrition dental caries. Dental Clin North Am 47:319–336
17. Tanzer JM, Livingston J, Thompson AM (2001) The microbiology of primary dental caries in humans. AM Dent Educ 65:1028–1037
18. Aas JA, Griffen AL, Dardis SR et al (2008) Bacteria of dental caries in primary and permanent teeth in children and young adults. J Clin Microbiol 46:1407–1417
19. Leone CW, Oppenheim FG (2001) Physical and chemical aspects of saliva as indicators of risk for dental caries in humans. J Dent Educ 65:1054–1062
20. Smith KM, Daly M, Fischer M et al (2003) Association of the dopamine beta hydroxylase gene with attention deficit hyperactivity disorder: genetic analysis of the Milwaukee longitudinal study. Am J Med Genet B Neuropsychiatr Genet 119B:77–85
21. Volkow ND, Wang GJ, Kollins SH et al (2009) Evaluating dopamine reward pathway in ADHD. JAMA 302:1084–1091