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Assessment of sleep bruxism, orthodontic treatment need, orofacial dysfunctions and salivary biomarkers in asthmatic children

Juliana Neide Amato^a, Rogério Antônio Tuon^{a,b}, Paula Midori Castelo^c,
Maria Beatriz Duarte Gavião^a, Taís de Souza Barbosa^{a,*}

^a Piracicaba Dental School – State University of Campinas, São Paulo, Brazil

^b Municipal Government of Piracicaba, São Paulo, Brazil

^c Federal University of São Paulo, São Paulo, Brazil

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ABSTRACT

Objective: To evaluate the sleep bruxism, malocclusions, orofacial dysfunctions and salivary levels of cortisol and alpha-amylase in asthmatic children.

Design: 108 7–9-yr-old children were selected from Policlinic Santa Teresinha Doutor Antonio Haddad Dib (asthmatics, $n = 53$) and from public schools (controls, $n = 55$), Piracicaba, SP, Brazil. Sleep bruxism diagnosis was confirmed by parental report of grinding sounds and the presence of shiny and polish facets on incisors and/or first permanent molars. The index of orthodontic treatment need was used for occlusion evaluation. Orofacial dysfunctions were evaluated using the nordic orofacial test-screening (NOT-S). Salivary cortisol and alpha-amylase were expressed as “awakening response” (AR), calculated as the difference between levels immediately after awakening and 30 min after waking, and “diurnal decline” (DD), calculated as the difference between levels at 30 min after waking and at bedtime. Data were analyzed using Shapiro–Wilk/Kolmogorov–Smirnov, Chi-square, unpaired t test/Mann–Whitney and paired t /Wilcoxon tests.

Results: Sleep bruxism was more prevalent in children with asthma than controls (47.2% vs. 27.3%, $p < 0.05$). Asthmatics had higher scores of NOT-S total and interview ($p < 0.05$). Dysfunctions on sensory function and chewing and swallowing were more frequent in asthmatics ($p < 0.05$). Salivary cortisol AR on weekend was significantly higher for asthmatics ($p < 0.05$). Salivary cortisol DD was significantly higher on weekday than weekend for controls ($p < 0.05$). There were no significant differences in alpha-amylase values in and between groups.

Conclusions: The presence of asthma in children was associated with sleep bruxism, negative perception of sensory, chewing and swallowing functions, and higher concentrations of salivary cortisol on weekend.

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* Corresponding author at: Faculdade de Odontologia de Piracicaba/UNICAMP – Departamento de Odontologia Infantil – Área de Odontopediatria, Av. Limeira 901, Piracicaba, SP 13414-903, Brazil. Tel.: +55 19 2106 5200/5369.

E-mail address: tais_sb@fop.unicamp.br (T.d.S. Barbosa).

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1. Introduction

Asthma is one the most common chronic illnesses of childhood and accounts for significant morbidity and mortality in children.¹ Asthma impacts approximately 4.9% and 8.9% of children (6–7-yr-old) and adolescents (13–14-yr-old), respectively, in the Brazil² and it is estimated that 60% of cases are intermittent or mild persistent, moderate are 25–30%, and 5–10% of them are severe.³ Asthma is considered a chronic inflammatory disorder of the airway similar to allergic rhinitis in that airway hyperactivity develops, resulting in changes in airway tone and thus airflow. As a consequence of inflammatory response and increased airway resistance in the respiratory system, the subjects suffering from asthma, experience difficulty in breathing. To overcome this difficulty their mode of breathing changes from nasal to oral breathing.⁴ This change in function can trigger modulations in the craniofacial growth patterns.⁵

The tendency to develop asthma is determined both by genetic and environmental influences. Among environmental influences, acute and chronic stress in children was found to be related to asthma symptoms⁶ and the timing of asthma exacerbations. Children who experience acute stress are at increased risk for an asthma exacerbation, and if chronic stress is present, the exacerbation occurs sooner than if chronic stress is absent.^{6,7} Stress is known to activate two major biological systems, the hypothalamus–pituitary–adrenal (HPA) axis and the sympatho-adrenal medullary (SAM) axis. Salivary cortisol levels reflect the activity of the HPA axis,⁸ whereas salivary alpha-amylase activity can be considered a marker of sympathetic activity.⁹ Activation of the HPA axis results in an enhanced secretion of the hormone cortisol,⁸ while alpha-amylase secretion releases upon sympathetic stimulation.⁹ In Wolf et al.¹⁰ study, higher chronic stress was associated with lower daily alpha-amylase output among children with asthma and with flatter cortisol slopes among healthy children.

The importance of understanding the direct consequences of asthma in the structures and functions of the stomatognathic system, as well as the environmental factors associated with the development of asthma are still poorly explored and studied. In this way, the present study aimed to evaluate signs and symptoms of sleep bruxism, orthodontic treatment need, orofacial dysfunctions and salivary levels of cortisol and alpha-amylase in asthmatic children. It is expected that asthmatic children have higher negative effects on morphological and functional characteristics of stomatognathic system, such as malocclusions and orofacial dysfunctions, as consequences of oral breathing; and higher concentrations of salivary cortisol and alpha-amylase as results of stress associated with disease limitations.

2. Materials and methods

2.1. Participants

The sample consisted of 108 children of both genders in the age-range from seven to nine years, selected from Policlinic Santa Teresinha Doutor Antonio Haddad Dib (asthma group,

$n = 53$) and from public schools (control group, $n = 55$) of Piracicaba, SP, Brazil, after anamnesis and clinical examination. The sample size was calculated by Epi info version 6.0.1 software. A standard error of 5%, a 95% confidence interval level, a power test of 80%, and a 4.9% prevalence of medical diagnosis of asthma in 6–7-yr-old Brazilian children² were used for the calculation. A correction factor of 1.2 was used to increase the precision.¹¹ Thus, the minimal sample size required to satisfy the requirements was estimated to be 104 individuals, 52 with asthma and 52 controls. The children and their parents consented to participate in the study, which was approved by the Ethics Committee of the Piracicaba Dental School (protocol no. 010/2011).

2.2. Anamnesis and clinical examination

The following procedures were performed by one calibrated examiner (JNA) at Policlinic Santa Teresinha Doutor Antonio Haddad Dib and public schools for asthma and control groups, respectively. The parents/guardians provided written information by means of a pre-structured questionnaire. The questions verified the pre-natal, natal and post-natal histories, habitation characteristics and actual and past symptoms of asthma, bronchitis or wheezing^{12–19} and parafunctional habits (bottle feeding, pacifier use, finger sucking, nail biting, sleep bruxism, and enuresis nocturna). The questions could be answered by qualitative (yes/no) and quantitative responses (frequency characteristics). These data were complementary to doctor's diagnosis of asthma (RAT), who classified asthma severity as intermittent, mild persistent, moderate persistent and severe persistent.³ The following protocol of medications was used depending on asthma severity:

Intermittent: Beta-2-adrenoreceptor agonist salbutamol (oral spray) during crisis.

Mild persistent: salbutamol (oral spray) during crisis and, eventually, inhaled corticosteroid beclometasone (oral spray) daily (low dose).

Moderate persistent: inhaled corticosteroid beclometasone (oral spray) daily (moderate dose) and salbutamol (oral spray) during crisis.

Severe persistent: inhaled corticosteroid beclometasone (oral spray) daily (high dose) and salbutamol (oral spray) during crisis and, eventually, daily.

2.2.1. Orthodontic treatment need

The index of orthodontic treatment need – dental health component (IOTN-DHC) was used for occlusion evaluation.²⁰ The DHC was examined using a millimetre probe, and the occlusal characteristics of a malocclusion that harm the teeth and adjacent structures were recorded. There are five levels, ranging from Degree 1 (there is no need for treatment) up to Degree 5 (there is a great need for treatment) (Table 1). This index serves as basic guide for impartial judgement of the malocclusion.²¹

2.2.2. Sleep bruxism

The sleep bruxism diagnosis was confirmed taking into account the international classification of sleep disorders

Table 1 – The dental health component of the index of orthodontic treatment need (IOTN).

Grade 5 (need treatment)	
5-i	Impeded eruption of teeth (except for third molars) due to crowding, displacement, the presence of supernumerary teeth, retained deciduous teeth and any pathological cause.
5-h	Extensive hypodontia with restorative implications (more than 1 tooth missing in any quadrant) requiring pre-restorative orthodontics.
5-a	Increased overjet greater than 9 mm.
5-m	Reverse overjet greater than 3–5 mm with reported masticatory and speech difficulties.
5-p	Defects of cleft lip and palate and other craniofacial anomalies.
5-s	Submerged deciduous teeth.
Grade 4 (need treatment)	
4-h	Less extensive hypodontia requiring pre-restorative orthodontics or orthodontic space closure to obviate the need for a prosthesis.
4-a	Increased overjet greater than 6 mm, but less than or equal to 9 mm.
4-b	Reverse overjet greater than 3–5 mm with no masticatory or speech difficulties.
4-m	Reverse overjet greater than 1 mm but less than 3–5 mm with recorded masticatory speech difficulties.
4-c	Anterior or posterior crossbites with greater than 2 mm discrepancy between retruded contact position and intercuspal position.
4-l	Posterior lingual crossbite with no functional occlusal contact in one or both buccal segments.
4-d	Severe contact point displacements greater than 4 mm.
4-e	Extreme lateral or anterior open bites greater than 4 mm.
4-f	Increased and complete overbite with gingival or palatal trauma.
4-t	Partially erupted teeth, tipped and impacted against adjacent teeth.
4-x	Presence of supernumerary teeth.
Grade 3 (borderline need)	
3-a	Increased overjet greater than 3–5 mm, but less than or equal to 6 mm with incompetent lips.
3-b	Reverse overjet greater than 1 mm, but less than or equal to 3–5 mm.
3-c	Anterior or posterior crossbites with greater than 1 mm, but less than or equal to 2 mm discrepancy between retruded contact position and intercuspal position.
3-d	Contact point displacements greater than 2 mm, but less than or equal to 4 mm.
3-e	Lateral or anterior open bite greater than 2 mm, but less than or equal to 4 mm.
3-f	Deep overbite complete on gingival or palatal tissues, but no trauma.
Grade 2 (slight)	
2-a	Increased overjet greater than 3–5 mm, but less than or equal to 6 mm with competent lips.
2-b	Reverse overjet greater than 0 mm but less than or equal to 1 mm.
2-c	Anterior or posterior crossbite with less than or equal to 1 mm discrepancy between retruded contact position and intercuspal position.
2-d	Contact point displacements greater than 1 mm but less than or equal to 2 mm.
2-e	Anterior or posterior open bite greater than 1 mm but less than or equal to 2 mm.
2-f	Increased overbite greater than or equal to 3–5 mm without gingival contact.
2-g	Pre- or post-normal occlusions with no other anomalies (includes up to half a unit discrepancy).
Grade 1 (none)	
1	Extremely minor malocclusions including contact point displacements less than 1 mm.

criteria,²² which considers parental report of grinding sounds (at least three times a week) and at least one of the following characteristics: (1) presence of shiny and polish facets on incisors and/or first permanent molars (based primarily on palatal surface and incisal edges and working cusps, respectively) observed in clinical examination using dental mirror with LED light, taking into account the time of eruption and (2) mandibular discomfort or pain when awakening.

The exclusion criteria were: pain of dental origin, premature tooth loss, anomalies of shape, number, structure and/or changes that might compromise the mesiodistal dimensions, tooth decay, trauma, soft tissue abnormalities, facial trauma, neurological, psychiatric or sleep disorders (e.g., epilepsy and obstructive apnoea syndrome), use of dental prostheses, history or presence of orthodontic treatment and uncooperative behaviour. Participants who did not collect saliva or submitted insufficient/contaminated samples were also excluded.

2.3. Orofacial dysfunctions

Orofacial dysfunctions were evaluated using the NOT-S protocol, which was developed by a working group formed at the Second Nordic Conference on Orofacial Therapy in Gothenburg in 2002 and was translated and culturally adapted to Brazilian Portuguese by Leme et al.^{23,24} (the form in English and in other languages, including Brazilian Portuguese, can be downloaded free of charge from www.mun-h-center.se). This protocol consists of a structured interview and a clinical examination, each part with six domains. In the interview, the following functions are assessed: (I) sensory function, (II) breathing, (III) habits, (IV) chewing and swallowing, (V) drooling, and (VI) dryness of the mouth. In the examination, the following functions are assessed: (1) face at rest, (2) nose breathing, (3) facial expression, (4) masticatory muscle and jaw function, (5) oral motor function, and (6) speech. Each domain contains one to five items, reflecting the complexity of

the specific function. NOT-S was applied individually by the same trained researcher (JNA), in a vacant classroom, with the child seated in an upright position. The NOT-S interview was held by asking the questions in the screening form. To assess orofacial dysfunction in the clinical examination, the subjects were requested to carry out tasks for each item in conjunction with the illustrated manual. Each item has criteria for the respective function. An answer of YES or a task that met the criteria for impaired function resulted in a score of 1, indicating a dysfunction in the scored domain. An answer of NO or a task that did not meet the criteria resulted in a score of 0 (zero). The total score was the sum of the scores for each domain and ranged from 0 to 12.²⁵ The higher the score the worse the orofacial dysfunction.

2.4. Salivary cortisol and alpha-amylase

Stimulated saliva samples were collected at home after the subjects and their parents were given written oral instructions for the collection procedure. Participants were instructed not to eat or drink or brush their teeth in the ½ hour before sampling. They were also asked not to perform physical exercises or caffeinated beverages in the day before sample collection. The participants received six coded plastic tubes containing cotton rolls (Salivettes, Sarstedt, Numbrecht, Germany). The saliva samples were collected during two days, on a weekday and on weekend, and in three different moments, immediately after awakening, 30 min after waking (fasting) and at bedtime. The samples were stored into a refrigerator and, in the next day, they were kept on ice and transported to the laboratory, where they were centrifuged at 3000 rpm for 15 min and stored at -80°C for analysis. To minimize variation, all samples from the same subject were assayed in the same batch (in duplicate) and were measured by the same researcher (TSB).

Salivary cortisol was assayed in 25 μl samples of whole saliva using a highly sensitive commercial enzyme immunoassay kit (product no. 1-3002; Salimetrics, State College, PA, USA) in a microtiter plate and read at 450 nm (EON, Biotek Instruments, Inc., Winooski, VT, USA) according to the manufacturer's directions. Following Granger et al.,²⁶ 8 μl samples of diluted saliva (1:200) were assayed for salivary alpha-amylase activity using a commercially available kinetic reaction kit (product no. 1-1902; Salimetrics, State College, PA, USA) in a microtiter plate and read at 405 nm (37°C) (EON, Biotek Instruments, Inc., Winooski, VT, USA) according to the manufacturer's directions.

Salivary cortisol ($\mu\text{g/dl}$) and alpha-amylase (U/ml) were expressed as “awakening response” (AR), calculated as the difference between levels immediately after awakening and 30 min after waking, and “diurnal decline” (DD), calculated as the difference between levels at 30 min after waking and at bedtime.

2.5. Measurement errors

For assessment of method error, the Kappa (κ) values (dental facets) and intraclass correlation coefficients (ICC) (IOTN-DHC and NOT-S scores) were calculated from the data of subjects not included in the studied sample, on two separate occasions

with an interval of 14 days between them (BioEstat 5.0; Mamirauá, Belém, PA, Brazil).

The assessment of method error showed an acceptable concordance ($\kappa = 0.58$, $n = 10$) for dental facets²⁷ and excellent to perfect agreement for IOTN-DHC (ICC = 0.93, $n = 12$), NOT-S total (ICC = 0.82, $n = 15$), interview (ICC = 0.85) and examination (ICC = 0.90) scores.²⁸

2.6. Statistics

Statistical analysis was performed using SPSS 18.0 (SPSS Inc., Chicago, EUA) and BioEstat 5.0 (Mamirauá, Belém, PA, Brazil) with a 5% significance level. Data normality was assessed using Kolmogorov–Smirnov or Shapiro–Wilk test (where appropriate). The proportions of subjects divided by groups according to gender, IOTN-DHC scores, presence/absence of sleep bruxism, severity of asthma and presence/absence of allergic rhinitis were evaluated using Chi-square test. Differences in NOT-S total, interview and examination scores between asthma and control groups were evaluated by means of Mann–Whitney test. Differences in salivary cortisol/alpha-amylase AR/DD values on weekday/weekend between groups were evaluated using unpaired t or Mann–Whitney tests. Paired t or Wilcoxon tests were used to compare salivary cortisol/alpha-amylase AR/DD on weekday and weekend for each group.

3. Results

Table 2 shows the distribution of the evaluated sample in accordance with gender, IOTN scores, sleep bruxism, severity of asthma and allergic rhinitis. Sleep bruxism was more prevalent in children with asthma than controls (47.2% vs. 27.3%, $p < 0.05$). More than half of the asthmatic children had intermittent severity (50.9%), followed by mild (28.3%), moderate (17.0%) and severe persistent (3.8%) ($p < 0.001$). There were no significant differences in gender, IOTN-DHC scores and allergic rhinitis between groups.

Table 3 shows the values of NOT-S total, interview and examination scores according to groups. The asthma group showed higher scores of NOT-S total and interview than control group ($p < 0.05$). There were no significant differences in NOT-S examination scores between groups.

Distribution of NOT-S domains according to groups is shown in Table 4. Symptoms of oral dysfunctions related to (I) sensory function and (IV) chewing and swallowing were more frequent in asthmatic children than controls ($p < 0.05$).

Table 5 shows the values of salivary cortisol ($\mu\text{g/dl}$) and alpha-amylase (U/ml) according to groups. Salivary cortisol AR on weekend was significantly higher for asthma group than controls ($p < 0.05$). In control group, salivary cortisol DD was significantly higher on weekday than weekend ($p < 0.05$). There were no significant differences in alpha-amylase values in and between groups.

4. Discussion

Sleep bruxism was more frequent in children with asthma than controls (47.2% vs. 27.3%, $p < 0.05$). This is preliminary

Table 2 – Distribution [n (%)] of gender, IOTN scores, sleep bruxism, severity of asthma and allergic rhinitis according to groups.

Characteristics	Asthma group (n = 53)	Control group (n = 55)	p-Value [*]
Gender			
Male	29 (54.7)	21 (38.2)	0.085
Female	24 (45.3)	34 (61.8)	
IOTN-DHC scores			
1	13 (24.5)	11 (20.0)	0.923
2	12 (22.6)	13 (23.6)	
3	15 (28.3)	15 (27.3)	
4	13 (24.5)	16 (29.1)	
Sleep bruxism			
Yes	25 (47.2)	15 (27.3)	0.032
No	28 (52.8)	40 (72.7)	
Asthma severity			
Intermittent	27 (50.9)	–	0.000
Mild persistent	15 (28.3)	–	
Moderate persistent	9 (17.0)	–	
Severe persistent	2 (3.8)	–	
Allergic rhinitis			
Yes	31 (58.5)	–	0.399
No	22 (41.5)	–	

SD: standard deviation; IOTN-DHC: index of orthodontic treatment need – dental health component.

Bold value indicates: Sleep bruxism $p < 0.05$, Asthma severity $p < 0.001$.

^{*} Chi-square test.

evidence that suggests an association between these phenomena. Recent publications have suggested that sleep bruxism may occur concomitantly or secondary to sleep-disordered breathing,^{29–31} and the latter with asthma in children.³² The association between asthma and sleep-disordered breathing seems to be evident from a physiological point of view. In the present study, parental report of sleep disorders (such as obstructive apnoea syndrome) was an exclusion criteria. On the other hand, 18.9% of the evaluated children with asthma reported snoring when sleep, a common symptom of obstructive apnoea syndrome,³³ when interviewed using NOT-S (domain II – breathing). As the current reference standard for diagnosing of sleep-disordered breathing is polysomnography,^{34–37} it is not possible to postulate of a

Table 3 – Mean (SD) scores of NOT-S total, interview and examination according to groups.

NOT-S score	Asthma group (n = 53)	Control group (n = 55)	p-Value [*]
Total [0–12]	3.3 (1.5)	2.7 (1.6)	0.018
Interview [0–6]	2.6 (1.3)	2.1 (1.3)	0.021
Examination [0–6]	0.8 (0.9)	0.7 (0.8)	0.444

SD: standard deviation; IQR: interquartile range; NOT-S: nordic orofacial test-screening.

[] range of possible scores.

Bold value indicates: Total $p < 0.05$, Interview $p < 0.05$.

^{*} Mann-Whitney test.

Table 4 – Distribution of NOT-S domains according to groups.

NOT-S domains	Groups (%)		p-Value [*]
	Asthma (n = 53)	Control (n = 55)	
Interview			
(I) Sensory function	35.8	14.5	0.011
(II) Breathing	18.9	20.0	0.882
(III) Habits	62.3	49.1	0.168
(IV) Chewing and swallowing	84.9	65.5	0.020
(V) Drooling	17.0	21.8	0.525
(VI) Dryness of the mouth	39.6	34.5	0.585
Examination			
(1) Face at rest	18.9	18.2	0.927
(2) Nose breathing	9.4	1.8	0.084
(3) Facial expression	39.6	38.2	0.878
(4) Masticatory muscle and jaw function	9.4	3.6	0.221
(5) Oral motor function	1.9	0.0	0.306
(6) Speech	3.8	3.6	0.970

NOT-S: nordic orofacial test-screening.

Bold value indicates: (I) Sensory function $p < 0.05$, (IV) Chewing and swallowing $p < 0.05$.

^{*} Chi-square test.

co-existing association between asthma and sleep-disordered breathing in the evaluated sample.

Asthma can be classified as intermittent, mild persistent, moderate persistent or severe persistent. In the present study, the majority of children had intermittent asthma (50.9%),

Table 5 – Mean (SD) of salivary cortisol (μg/dl) and alpha-amylase (U/ml) levels according to groups.

	Asthma group (n = 53)	Control group (n = 55)	p-Value [*]
Cortisol AR ^a	0.02 (0.35)	0.00 (0.34)	0.810
Cortisol AR ^b	0.03 (0.16)	–0.03 (0.27)	0.016
	p-Value ^{**} = 0.664	p-Value ^{**} = 0.210	
Cortisol DD ^a	0.25 (0.24)	0.32 (0.22)	0.112
Cortisol DD ^b	0.21 (0.19)	0.21 (0.30)	0.943
	p-Value ^{**} = 0.319	p-Value ^{**} = 0.043	
Alpha-amylase AR ^a	–7.57 (50.46)	–64.32 (333.00)	0.092
Alpha-amylase AR ^b	2.47 (40.09)	–19.35 (120.92)	0.149
	p-Value ^{**} = 0.214	p-Value ^{**} = 0.421	
Alpha-amylase DD ^a	–31.28 (91.75)	–20.85 (91.91)	0.982
Alpha-amylase DD ^b	–25.19 (74.79)	–30.89 (96.08)	0.732
	p-Value ^{**} = 0.807	p-Value ^{**} = 0.523	

IQR: interquartile range; AR: awakening response; DD: diurnal decline.

Bold value indicates: Cortisol AR $p < 0.05$, Cortisol DD $p < 0.05$.

^a Weekday.

^b Weekend.

^{*} Differences between groups (unpaired t/Mann-Whitney test).

^{**} Differences between AR/DD on weekday and weekend for each group (paired t/Wilcoxon test).

followed by mild persistent (28.3%), moderate persistent (17%) and severe persistent (3.8%). It is estimated that 60% of cases are intermittent or mild persistent, moderate are 25–30%, and 5–10% of them are severe.³ More than half of the asthmatic children also had allergic rhinitis (58.5%), corroborating the guidelines of allergic rhinitis and its impact on asthma (ARIA).³⁸ From studies in several countries, the Project ARIA has consistently shown that asthma and rhinitis often coexist within the same patient. There is evidence that the upper and lower airways are affected by a common inflammatory process that can be maintained and amplified by interconnected mechanisms. Consequently, these children usually exhibit increased nasal resistance and may constitute a risk group for changes in dentofacial growth and development.^{5,39,40} In Kumar and Nandlal⁵ study, children diagnosed as having asthma and allergic rhinitis had a significantly deeper palate and there was a tendency to a more accentuated increase of palate depth between the stages of primary and mixed dentition. The lack of difference in IOTN-DHC scores between groups may be related to the limited in ability of the index to assess each type of malocclusion, as it is an objective measure designed to measure the severity of malocclusion.

Asthmatic children had higher NOT-S total and interview scores than controls, suggesting more symptoms of orofacial dysfunctions in the former, especially related to (I) sensory function (35.8% vs. 14.5%) and (IV) chewing and swallowing (84.9% vs. 65.5%). In relation to (I) sensory function domain, 32.1% of asthmatic children reported decreased sensitivity, i.e., difficulty chewing because of putting too much food in mouth, while 5.7% reported gag reflex when tooth brushing (increased sensitivity). A recent literature review showed that high dosage and long duration of inhalation therapy has been closely link with several adverse effects on the oral health.⁴¹ One of these effects is dry mouth or xerostomia observed with use of beta-2 agonists,⁴² anticholinergic inhalers⁴³ and inhaled corticosteroids.⁴⁴ Xerostomia produces taste changes secondary to incomplete food solubilization and by diminished transport of tastant molecules to taste buds.^{45–47} The beta-2 agonists, frequently used by the evaluated sample, can also cause relaxation of smooth muscles such as the lower esophageal sphincter leading to gastro-esophageal reflux symptoms, such as of indigestion, vomiting and heartburn.⁴⁸ Nausea has also been significantly reported in individuals on inhaled corticosteroid therapy.⁴⁹

In relation to (IV) chewing and swallowing domain, the most frequent report was difficulty to eat foods with certain consistencies (36.7%), followed by taking 30 min or more to eat a main meal (35.8%), having cough during meals (20.8%) and swallowing large bites without chewing (17%). Analyzing the results of the speech-language pathology evaluation, Castro et al.⁵⁰ observed worse results of mastication, characterized by unilateral chewing and increased/decreased speed of mastication, for children with asthma when compared to healthy children. These authors also found alterations on swallowing solid food and liquids, characterized by anterior projection of the tongue, stasis in the oral cavity after swallowing and exaggerated contraction of the mentalis muscle in order to compensate for the hypotonicity of the lower lip.

As expected, salivary alpha-amylase and cortisol were found to have opposite daily patterns, reflecting the distinctiveness of

the two parameters being indicators of sympathetic activity versus HPA axis, respectively. In other words, the trajectory of salivary alpha-amylase activity over the course of a day was opposite of that observed for cortisol, with lowest levels 30 min after awakening and increasing levels over the day. Interestingly, only salivary cortisol but not alpha-amylase trajectories differed between children with asthma and healthy children. Although salivary alpha-amylase levels do not differ, compared to healthy children, asthmatic children showed lower salivary alpha-amylase levels throughout the day, corroborating the hypothesis found by Wolf et al.¹⁰ that increasing sympathetic activity may serve a protective function in asthma. With respect to cortisol, the present findings reinforce the evidence linking altered cortisol levels and atopic diseases.⁵¹ Additionally, there were differences in salivary cortisol levels on weekday and weekend among healthy children. Each of these findings will be discussed in more detail below.

In the present study, asthmatic children had higher values than controls on weekend, indicating hyperresponsiveness of the HPA axis. Based on this, it is reasonable to suggest that psychosocial stress experienced during weekday remains activated during weekend for asthmatic children. Psychosocial stress can trigger asthma exacerbations⁵² and it is suggested that psychosocial stress is also involved in asthma development.^{53–55} Regarding to control group, higher salivary cortisol DD was observed on weekday than weekend. According to Cunningham-Bussell et al.⁵⁶ study, a greater daily cortisol amplitude is associated with less responsiveness of limbic regions (hippocampus and amygdala) associated with both stress and fear response. Previous studies have found higher salivary cortisol concentrations in weekday than weekend in adult population due to work stress.^{57–59} In other words, the somewhat paradoxical finding that healthy subjects had higher salivary cortisol DD on weekday than weekend might be caused by chronic stress, and it is possible that the greater duration and accumulation of stressor exposure (e.g., difficulties due to low socioeconomic level and familiar problems) is physiologically important.⁶⁰ Consistent with this explanation, Wolf et al.¹⁰ found a flatter cortisol profile under conditions of chronic stress, higher home life stress and parents having less years of education, in healthy children.

This study has some limitations. To assess sleep bruxism events with higher specificity, and consequently better understand its association with asthma, further studies using polysomnographic records are recommended, despite of its high cost and its difficulty in using in young children. Studies are needed which include other measures of autonomic nervous system activity to test the hypothesis of salivary alpha-amylase indicating sympathetic counter-regulation. Finally, including a questionnaire of stress symptoms and impacts on quality of life would allow researchers to test the predictive value of physiological findings.

5. Conclusions

Asthmatic children presented more signs and symptoms of sleep bruxism, negative perception of sensory, chewing and swallowing functions, and higher concentrations of salivary cortisol on weekend.

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Competing interests

The authors have no conflict of interests.

Ethical approval

This study was approved by the Ethics Committee of the Piracicaba Dental School (protocol no. 010/2011).

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