

Sweat Electrolyte Loss in Asthmatic Children During Exercise in the Heat

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This study assessed sweat electrolyte concentration and losses in asthmatic children during exercise in the heat. Eleven asthmatics (AG; 11 ± 2 years old) and 11 nonasthmatics (CG; 10 ± 1 year old) underwent a maximal progressive cycle-ergometer test. During a second session, participants cycled in the heat (35°C , 60% RH) of a climatic chamber for 30 min at 50–60% of maximal workload. Sweat was collected using sweat patches attached to the dorsal region. No differences were observed in sweat $[\text{Na}^+]$ (AG = 35 ± 12.9 and CG = 43.4 ± 18 mmol/L) and $[\text{Cl}^-]$ (AG = 27.3 ± 10.4 and CG = 38.5 ± 19.1 mmol/L). There was no difference in sweat Na^+ losses (AG = 0.47 ± 0.36 and CG = 0.66 ± 0.68 mmol/kg/h) and Cl^- losses (AG = 0.37 ± 0.29 and CG = 0.59 ± 0.62 mmol/kg/h) between groups. Asthmatic children did not differ from nonasthmatics in their sweat electrolyte concentrations and electrolyte losses.

Some, still inconclusive, indication exists that higher concentrations of sweat sodium ($[\text{Na}^+]$) and chloride ($[\text{Cl}^-]$) can be found in asthmatic patients and not only in those with cystic fibrosis (6,15,18,21,28). None of these studies, however, assessed a group of asthmatics under controlled conditions (e.g., same degree of asthma severity, use of medication, age group, and maturational degree), so there is still inconclusive information regarding this issue. It has also been suggested that high $[\text{Na}^+]$ and $[\text{Cl}^-]$ in the periciliary fluid of the lungs induces hyperresponsiveness and exercise-induced bronchoconstriction (14,24). It is therefore possible that high sweat $[\text{Na}^+]$ and $[\text{Cl}^-]$ reflect a systemic aberration in ion transport among asthmatics, and it might be reflected in sweat ionic composition.

When sweat electrolyte concentration was assessed in boys and girls with different maturational degrees (21), it was observed that two prepubescent boys had a high $[\text{Na}^+]$ in their sweat. These participants were excluded from the study and after further investigation; it was found that both children were asthmatics.

Adults and children with respiratory disease, including asthma, may participate in any sport or exercise activities when they are not symptomatic (1,2). Likewise, studies have reported the trainability and benefits of exercise in asthmatic patients

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(8,27). Thus, if asthmatics, indeed, have high levels of Na^+ and Cl^- in their sweat, this might have two main implications: Asthmatics might be at risk for severe Na^+ losses while exercising in hot weather, and the interpretation of positive sweat tests in asthmatics should be made with caution. Therefore, this study aimed to verify whether asthmatic children lose more Na^+ and Cl^- through sweating than nonasthmatics during a single session of exercise in the heat.

Methods

Subjects

Twenty-two children, 11 asthmatics (AG), and 11 nonasthmatics (CG), Tanner stage 1–3 (29) were recruited. Asthmatic children were recruited at the asthma clinic of the Hospital Presidente Vargas of Porto Alegre. All asthmatic children were classified as having mild-persistent to moderate-persistent asthma according to the Global Initiative for Asthma – GINA (13), and they were under continuous daily treatment with inhaled corticosteroids. The University Ethical Committee approved the study, and parents gave written informed consent. Physical characteristics from both groups are described in Table 1. Experiments took place in Porto Alegre, Brazil, during late autumn and winter of a south Brazilian weather (from March to July) when temperatures ranged from 10 to 20°C. Therefore, participants were assumed not to be acclimatized to the heat. The asthmatics and nonasthmatic children underwent two experimental sessions.

Preliminary Session

During their initial visit to the laboratory, all children answered the ISAAC questionnaire (4) to confirm or refute the diagnoses of asthma. Anthropometric measurements: height (wall stadiometer Seca), weight (Filizola scale, 0.1 kg), and skinfold thickness (Lange caliper) over the triceps, subscapula, abdomen, and the anterior midthigh were measured. Body mass index (BMI) was calculated by using the follow formula: $\text{BMI} = \text{body mass (kg)} / \text{height (m}^2\text{)}$. Maximum exer-

Table 1 Participants' Physical Characteristics

| | Control Group (<i>n</i> = 11, 9♂ and 2♀) | Asthma Group (<i>n</i> = 11, 7♂ and 4♀) | <i>p</i> Value |
|--------------------------|--|---|----------------|
| Age (years) | 11 ± 2 | 10 ± 1 | .203* |
| Tanner | I-III | I-II | |
| Body mass (kg) | 43.1 ± 8.5 | 39.1 ± 13.6 | .133** |
| Height (cm) | 150 ± 1 | 140 ± 1 | .215* |
| Skinfold sum | 77.9 ± 36.7 | 77.6 ± 48.1 | .606** |
| BMI (kg/m ²) | 19.8 ± 2.9 | 19.2 ± 4.4 | .300** |
| Maximal workload (W) | 135 ± 39 | 112.5 ± 27.6 | .164* |

Note. Skinfold sum = triceps, subscapular, abdomen, and thigh.

*Independent *t* test; **Mann-Whitney test.

cise workload was determined using a cycle ergometer (Ergo Fit 169, Spain) via a progressive 2-min stage protocol (16) whereby increments varied from 15 to 25 W depending on subjects' height and gender. Heart rate (HR; Polar S610, Polar Electro Oy, Finland) and rate of perceived exertion (RPE; 5) were monitored at the end of each stage. During this test, participants were instructed to cycle at a cadence up to 60 rpm. For safety reasons, the test was halted when participants were unable to maintain this cadence; HR exceeded 200 bpm or when RPE was greater than 18. Participants completed the test in 8–12 min.

In-Chamber Session

Approximately 1 week later, participants performed the exercise-in-the-heat session. On the day of the experiment, they arrived at the laboratory after having refrained from using beta₂ adrenergic and corticosteroids drugs for 24 hr. Participants emptied their bladders, and to ensure they started the session euhydrated, they drank 250 ml of plain water 30 min before starting the exercise (23). Before entering the climatic chamber (Russells, Holland), in which thermal conditions were 35°C and 60% relative humidity, participants' nude body mass was recorded (Urano, nearest 0.01 kg) and then a 5-min warm-up period began. After this, participants cycled (Ergo Fit 167) for 30 min at 50–60% of their predetermined maximal workload. HR and auricular temperature (AuT; Digital Infrared Ear Thermometer, Microlife, FL) were measured every 5 min. At the end of exercise, nude body mass was measured again.

Sampling, Sweat Analysis, and Calculations

Sweat was collected into sweat patches (3M Tegaderm + pad, ref. 3,582) attached at both sides of the dorsal region, over the spine of the scapula, and ~7 cm lateral from the vertebral column as described by Patterson and colleagues (26). This site was chosen because of its good estimation of whole body sweat electrolyte losses as previously assessed (26). In addition, this site is not easily reached by children's hands during the exercise session, thus avoiding contamination. Before the patch application, participants had their skin cleaned with deionized water before it was thoroughly dried with sterile gauze to avoid contamination. The sweat patches were removed at the end of the in-chamber session and stored into a 20 ml tube.

Sweat [Na⁺] was measured by flame photometry (Corning 400, NY) and [Cl⁻] by spectrophotometry (Hitachi U2000). Sweat volume was determined by the changes in body mass during exercise in the heat and then corrected for exercise duration to determine sweat rate. Sweat electrolyte losses were calculated by multiplying sweat electrolyte concentration by sweat rate and corrected for both body mass and time.

Statistical Treatment

Statistical treatment was done using the Statistical Package for Social Sciences 13.0. A Shapiro-Wilk test was used to verify the normality of data. When nonparametric data were found, data were transformed into logarithmic basis. When data persisted nonparametric, Wilcoxon and Mann-Whitney tests were employed. For between-group analysis, two-way analysis of variance (two-way ANOVA) with

Greenhouse-Geisser corrections where appropriate and independent *t* test were used. Paired-sample *t* test was employed for comparisons within groups. Data are expressed as mean \pm *SD*. Significant differences were considered when $p < .05$.

Results

All asthmatic children answered that they had at least one asthma attack in the previous 12 months, whereas nonasthmatics did not have any symptom of respiratory disease as assessed by ISAAC questionnaire. No differences were found in age, height, body mass, BMI, skinfold sum, and maximal workload between groups (Table 1).

The increase in AuT from rest (AG = $36 \pm 0.3^\circ\text{C}$ and CG = $36.4 \pm 0.3^\circ\text{C}$) to the end (AG = $36.4 \pm 0.3^\circ\text{C}$ and CG = $36.7 \pm 0.4^\circ\text{C}$) of the in-chamber session was similar between the groups ($p = .553$). Values were as follows: $0.37 \pm 0.26^\circ\text{C}$ (AG), $0.28 \pm 0.42^\circ\text{C}$ (CG). The increase in HR from rest (AG = 114 ± 14.8 bpm and CG = 123 ± 11.3 bpm) to the end (AG = 138 ± 16.3 bpm and CG = 147 ± 17.6 bpm) of exercise was also similar ($p = .962$) between groups: 23.9 ± 22.4 bpm in the AG and 24.2 ± 10.9 bpm in the CG. A similarity ($p = .288$) was observed in RPE during exercise in the heat between groups; Borg scale values ranged from 10 to 13 in the AG and from 10 to 14 in the CG.

Sweating rate was similar ($p = .144$) between groups (AG 0.47 ± 0.22 L/h and CG 0.64 ± 0.32 L/h). No differences were found either in sweat $[\text{Na}^+]$ ($p = .423$) and $[\text{Cl}^-]$ ($p = .398$) or in sweat Na^+ ($p = .372$) and Cl^- ($p = .421$) losses between groups (Figure 1).

Discussion

The hypothesis that asthmatic children have higher sweat $[\text{Na}^+]$ and $[\text{Cl}^-]$ than nonasthmatics, although previously suggested (6,15,18,21,28), has never been systematically tested. Furthermore, none of these studies (6,15,18,21,28) had as their main purpose the assessment of sweat electrolyte concentration in asthmatic children. To our knowledge, this was the first study that assessed sweat electrolyte concentration and losses in a group of asthmatic (mild-persistent to moderate-persistent) children during a session of exercise in the heat.

The asthmatics of this study were clinically classified as having mild-persistent to moderate-persistent asthma according to the criterion of GINA (13) and also by answering the ISAAC questionnaire (4). Rosenstein et al. (28) evaluated 271 children with cystic fibrosis in which 8 presented a positive sweat test. After further evaluation, they found that 6 out of these 8 children could have asthma rather than cystic fibrosis. No information was given, however, on the degree of asthma of these children.

To make a representative sweat collection, a session of exercise in the heat was held. According to Vimiero-Gomes et al. (30), exercise-induced sweating is a result of integrated physiological mechanism, and therefore this would provide a more precise sweat sample than via pilocarpine stimulus. Kapranov et al. (18) assessed 156 asthmatics using the palm imprints qualitative method for sweat collection. They found that 24% of the children presented a positive sweat test. When

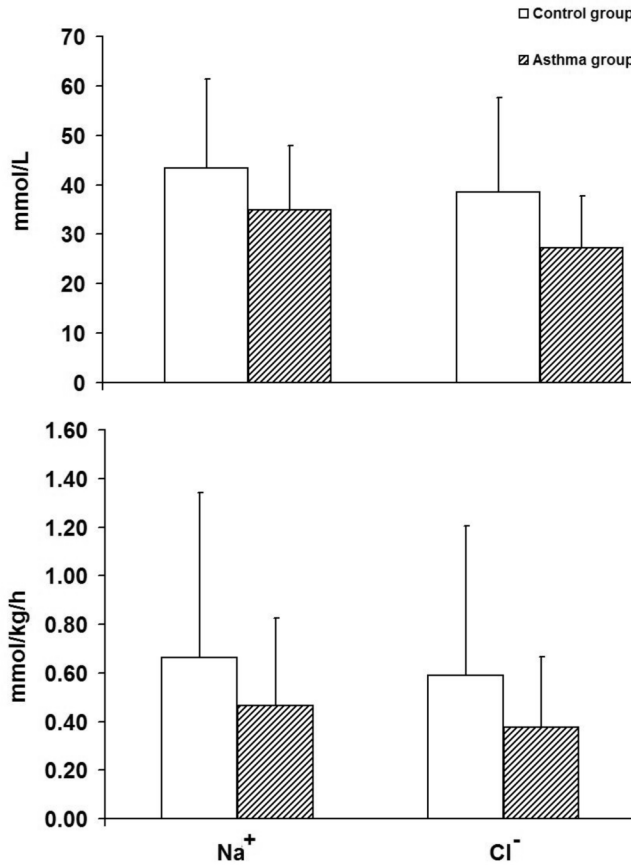


Figure 1 — Sweat [Na⁺] and [Cl⁻] and sweat Na⁺ and Cl⁻ losses. (Mean ± SD).

they quantified the test (pilocarpine iontophoresis), however, in 36 participants at the end of the study, only one child had sweat [Na⁺] higher than 50 mmol/L. It is possible that the way to collect sweat, in the current study induced by exercise, could explain the normal sweat electrolyte concentration in asthmatics.

In the current study, asthmatics presented similar sweat [Na⁺] and [Cl⁻] as compared with nonasthmatics. Gyurkovits et al. (15) verified sweat electrolyte concentration in asthmatic children with cystic fibrosis heterozygosity and in a control group. Both [Na⁺] and [Cl⁻] were low, although this was higher in the asthmatics ([Na⁺] = 31 ± 11.3 mmol/L and [Cl⁻] = 35.8 ± 14.7 mmol/L) than in the control group ([Na⁺] = 25.3 ± 10.6 mmol/L and [Cl⁻] = 28.7 ± 12.5 mmol/L). This inconsistency might be because of the presence of cystic fibrosis heterozygosity in those asthmatic children from Gyurkovits et al. study (15).

A limitation of the current study is the relatively low number of participants, which was mainly because of three reasons: difficulty in finding asthmatic children who accepted taking part in this study; difficulty in finding asthmatic children who

fitted on the asthma clinical criteria (mild persistent to moderate persistent); and time to complete data collection before winter begins in Brazil, which could interfere on children sweat electrolyte composition. Therefore, further investigations should confirm whether these results are consistent with a larger sample size.

Several studies carried out with healthy pediatric population have reported a large variability in sweat electrolyte concentration (3,21,23). This large variability was also found in the current study. According to Meyer and Bar-Or (22), children's sweat $[\text{Na}^+]$ ranges from 30 to 60 $\text{mmol}\cdot\text{L}^{-1}$ and tends to be lower than those of adults. Despite being naturally acclimatized to a tropical weather in the current study, sweat $[\text{Na}^+]$ and $[\text{Cl}^-]$ were within the expected normal range.

As a result of the reduced sweating rate in children, their total sweat Na^+ and Cl^- losses would be expected to be lower than 6.5 mmol/h and 5.5 mmol/h respectively (21). Although it is hard to compare studies that used participants with different degree of acclimatization, sweat Na^+ and Cl^- losses corrected for body mass were somewhat higher in both groups when compared with a study (21) that used a similar exercise protocol for sweat collection in both prepubescent and pubescent children. These data might be explained by the degree of acclimatization of the children from the current study that were naturally acclimatized to a tropical climate and therefore are expected to have a higher absolute sweating rate (3,21). Furthermore, acclimatization causes an increase in eccrine sweat gland responsiveness to aldosterone, thus increasing sweat Na^+ absorption (19).

In the current study, both groups were similar in terms of their maximal exercise workload, suggesting that asthmatics in this study have a similar physical fitness level when compared with the nonasthmatics. The level of physical fitness might affect sweat electrolyte concentration and losses, because high sweat rate is observed in those with high levels of physical fitness (12,25). The extent to which the level of physical fitness improves thermoregulatory effectiveness in children seems to be minor (10,11,20), however.

Another reason that might lead to a normal sweat electrolyte concentration in asthmatic children is the continuous daily medication taken by them. Although asthmatics avoided using medication at least 24 hr before the in-chamber trial, it is possible that the inhaled corticosteroids drug therapy might have resulted in a decreased $[\text{Na}^+]$ in their sweat. The possible mechanism by which this therapy might have affected asthmatics' sweat $[\text{Na}^+]$ is based on the hypothesis that corticosteroid drugs might activate the renin-angiotensin-aldosterone system (7). This might increase Na^+ absorption in the eccrine sweat glands because of an increased concentration of aldosterone and thus reducing its sweat concentration. For ethical reasons, the asthmatics were allowed to keep on with their daily drug therapy until a day before the trials. This limited the generalization of the study for asthmatics who are not mild persistent to moderate persistent. Therefore, further studies should clarify if these results persist when asthmatic children are not under continuous therapy with inhaled corticosteroids.

As children have a higher surface area per unit mass, there are risks such as hyperthermia and severe dehydration when they are exposed to a heat environment (9,11). The AuT fluctuation was too small to cause hyperthermia, however, and the ingestion of 250 ml of plain water 30 min before starting exercise in the climatic chamber was enough to keep all children well hydrated throughout the trial.

Although exercise intensity was sufficient to induce enough sweat for collection, the duration of exercise was too short to cause severe dehydration.

Hydration status of the broncho-pulmonary structures and composition of the airway surface liquid might be assumed to contribute to the maintenance of an efficient function (17). Therefore, even though presenting a normal sweat electrolyte concentration, asthmatics should be advised to drink while exercising in the heat to avoid dehydration and thus avoid risks of enhanced bronchoconstriction.

In conclusion, this study demonstrated that asthmatic and nonasthmatic children have similar sweat $[\text{Na}^+]$ and $[\text{Cl}^-]$, as well as normal Na^+ and Cl^- losses during a single session of exercise in the heat. It suggests that mild-persistent to moderate-persistent asthmatic children do not require special recommendation for electrolyte replenishment while exercising in the heat. In addition, the interpretation of sweat test in these asthmatics might not be affected by their chronic disease.

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