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Influence of environmental temperature and humidity on the acute ventilatory response to exercise in asthmatic adolescents

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Objective: This study aimed to analyse the influence of moderate changes in air temperature and humidity on the acute ventilatory response to exercise in asthmatic children. ***Design:*** 7 asthmatics over 15 years old (Experimental Group, EG), and 7 healthy subjects with the same characteristics (Control Group, CG) performed an exercise protocol in 2 different environments, interspaced by 48 hours: *hot+humid environment (HH,* temperature: 32.6±0.4ºC, relative humidity: 42.4±1.6%) and an environment with *less humidity+lower temperature (LHLT,* temperature: 24.0±0.9ºC, relative humidity: 36.3±1.3%). Steady state values of cardio-respiratory parameters were monitored during exercise, being the forced expiratory volume in 1st second (FEV_1) and peak expiratory flow (PEF) obtained immediately before exercise and after 5 minutes of recovery. ***Results:*** The EG experienced a post-exercise decrease of FEV1 in LHLT (90.6±9.6%, $p<0.05$) and HH (95.2±5.8%, $p<0.05$) while CG did not show post-exercise changes in both environments (103.7±11.2% vs. 101.2±4.9%, respectively). The EG showed a post-exercise PEF decrease in the LTLH (94.6±8.8%, $p<0.05$) and no changes in the HH environment (99.7±4.2%), while CG did not show significant changes in both environments (102.9±12.7% vs. 107.0±9.8%). ***Conclusion:*** Results allow concluding that ventilatory response to exercise is influenced by air temperature and humidity, with a HH environment being more favourable for asthmatics.

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Key Words: Bronchoconstriction; FEV1; PEF; asthma; airways response

INTRODUCTION

Asthma is a chronic inflammatory disorder of the respiratory airways affecting people of all ages, and constitutes a serious public health problem worldwide (6). Such a chronic inflammation is invariably associated with injury and repair of the bronchial epithelium known as remodelling (11). Inflammation, remodelling, and altered neural control of the airways are responsible for both recurrent exacerbations of asthma and increasingly permanent airflow obstruction (11, 29, 34). Excessive airway narrowing is caused by altered smooth muscle behaviour, in close interaction with swelling of the airway walls, parenchyma retractile forces, and enhanced intraluminal secretions (29, 38). All these functional and structural changes are associated with the characteristic symptoms of asthma – cough, chest tightness, and wheezing –and have a significant impact on patients' daily lives, on their families and also on society (1, 24, 29). Recent epidemiological studies show an increase in the prevalence of asthma, mainly in industrial countries (12, 25, 37). The reasons for this increase may depend on host factors (e.g., genetic disposition) or on environmental factors like air pollution or contact with allergens (6, 22, 29).

Physical exercise is probably the most common trigger for brief episodes of symptoms, and is assumed to induce airflow limitations in most asthmatic children and young adults (16, 24, 29, 33). Exercise-induced asthma (EIA) is defined as an

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intermittent narrowing of the airways, generally associated with respiratory symptoms (chest tightness, cough, wheezing and dyspnoea), occurring after 3 to 10 minutes of vigorous exercise with a maximal severity during 5 to 15 minutes after the end of the exercise (9, 14, 16, 24, 33). The definitive diagnosis of EIA is confirmed by the measurement of pre- and post-exercise expiratory flows documenting either a 15% fall in the forced expiratory volume in 1 second (FEV1), or a ≥15 to 20% fall in peak expiratory flow (PEF) (9, 24, 29). Some types of physical exercise have been associated with the occurrence of bronchial symptoms and asthma (5, 15, 17). For instance, demanding activities such as basketball or soccer could cause more severe attacks than less vigorous ones such as baseball or jogging (33). The mechanisms of exercise-induced airflow limitations seem to be related to changes in the respiratory mucosa induced by hyperventilation (9, 29). The heat loss from the airways during exercise, and possibly its post-exercise rewarming may contribute to the exercise-induced bronchoconstriction (EIB) (27). Additionally, the concomitant dehydration from the respiratory mucosa during exercise leads to an increased interstitial osmolarity, which may also contribute to bronchoconstriction (4, 36). So, the risk of EIB in asthmatically predisposed subjects seems to be higher with greater ventilation rates and the cooler and drier the inspired air is (23). The incidence of EIA in physically demanding cold-weather sports like competitive figure skating and ice hockey has been found to occur in up to 30 to 35% of the participants (32). In contrast, swimming is often recommended to asthmatic individuals, because it improves the functionality of respiratory muscles and, moreover, it seems to have a concomitant beneficial effect on the prevalence of asthma exacerbations (14, 26), supporting the idea that the risk of EIB would be smaller in warm and humid environments. This topic, however, remains controversial since the chlorified water of swimming pools has been suspected as a potential trigger factor for some asthmatic patients (7, 8, 20, 21). In fact, the higher asthma incidence observed in industrialised countries has recently been linked to the exposition to chloride (7, 8, 30).

Although clinical and epidemiological data suggest an influence of humidity and temperature of the inspired air on the bronchial response of asthmatic subjects during exercise, some of those studies did not accurately control the intensity of the exercise (2, 13), raising speculation of whether the experienced exercise overload was comparable for all subjects. Additionally, most of the studies did not include a control group (2, 10, 19, 39), which may lead to doubts about whether asthma per se has conditioned the observed results. Moreover, since the main targeted age group of these studies has been adults (10, 19, 39), any extrapolation to childhood/adolescence might be questionable regarding the different lung maturation. Considering the higher incidence of asthma in youngsters (30) and the fact that only the works of Amirav and co-workers (2, 3) have focused on this age group, a scarcity of scientific data can be identified. Additionally, since the main environmental trigger factors, i.e., temperature and humidity, were tested separately (10, 28, 39) it would be useful to analyse these two variables simultaneously because of their synergic effect on water and heat loss by the airways (31, 33). It also appears important to estimate the airway responsiveness to exercise within moderate environmental ranges of temperature and humidity, trying to avoid extreme temperatures and humidity conditions used by others (2, 3).

So, the aim of this study was to analyse the influence of moderate changes in air temperature and humidity simultaneously on the acute ventilatory response to exercise in asthmatic children. To overcome the above referred to methodological limitations, we used a 15 minute progressive exercise trial on a cycle ergometer at 3 different workload intensities, and we collected data related to heart rate, respiratory quotient, minute ventilation and oxygen uptake in order to ensure that physiological exercise repercussions were the same in both environments. The tests were done in a "normal" climatic environment (in a gymnasium) and in a hot and humid environment (swimming pool); for the latter, direct chloride exposition was avoided.

MATERIAL AND METHODS

Subjects

A group of 14 subjects over 15 years old, without limitations to exercise, was divided in 2 subgroups: Experimental Group (n=7), including subjects with the diagnosis of bronchial asthma without exacerbations over the last month, being followed at the Immunology and Allergy Unit of the local Hospital; and the Control Group (n=7), including

Table 1. Sample age, gender proportion and anthropometrics

	Experimental Group (mean±sd)	Control Group (mean±sd)
Age (years)	15.3±1.8	15.4±2.4
Gender (♀;♂)	1;6	1;6
Weight (kg)	57.4±8.2	58.4±12.4
Height (m)	1.69±0.09	1.67±0.12
BMI (kg/m2)	20.0±1.7	20.7±2.4

Legend: ♀ - girs; ♂ - boys; BMI – Body Mass Index.

Table 2. Mean±standard deviation of cardio-respiratory parameters (percentage of variation) measured in both groups during steady state exercise at different workloads and environments.

Group		Control Group			Experimental Group		
Workload		30 W	60 W	120 W	30 W	60 W	120 W
RF (%)	LHLT	114.3±9.2 [a]	128.9±2.0 [a]	158.9±4.1 [a]	123.7±2.1 [a]	140.0±3.5 [a]	187.7±7.4 [a]
	HH	117.1±11.4 [a]	132.6±2.0 [a]	166.7±4.2 [a]	135.1±2.6 [a]	156.1±3.6 [a,b]	226.2±8.6 [a,b]
TV (%)	LHLT	131.5±1.2 [a]	165.3±2.3 [a]	228.3±4.4 [a]	124.7±1.0 [a]	151.1±1.6 [a]	200.5±3.4 [a]
	HH	138.4±3.0 [a]	172.8±3.8 [a]	249.6±9.7 [a]	130.4±2.3 [a]	169.5±5.7 [a]	230.2±10.2 [a]
VO2(%)	LHLT	178.2±17.3 [a]	255.6±18.3 [a]	412.0±48.3 [a]	161.1±20.1 [a]	228.6±34.6 [a]	350.7 ±68.5 [a]
	HH	177.7±25.3 [a]	254.9±36.6 [a]	410.4±87.1 [a]	171.9 ±26.1 [a]	242.5 ±46.3 [a]	386.4 ±96.6 [a]
RQ (%)	LHLT	105.2±2.9 [a]	110.2±2.8 [a]	120.7±7.7 [a]	105.7±3.0 [a]	109.5 ±4.4 [a]	121.0±8.0 [a]
	HH	105.3±4.4 [a]	102.4±3.6 [a,b]	118.5±14.3 [a]	107.6±4.1 [a]	105.6 ±3.7 [a]	128.9±14.5 [a]
HRmax(%)	LHLT	52.1±5.2 [a]	60.5±6.6 [a]	78.6±5.9 [a]	50.2±5.9 [a]	59.7±8.2 [a]	76.9±10.2 [a]
	HH	52.9±6.6 [a]	62.5±7.5 [a,b]	80.4±5.9 [a]	51.9±6.9 [a]	62.5±8.2 [a]	80.1±10.1 [a,b]

Legend: HH - hot and humid environment; LHLT - less humidity and lower temperature environment; RF - respiratory frequency; TV - tidal volume; VO2 – oxygen uptake; RQ – Respiratory quotient; HRmax – maximal heart rate. a $p<0.05$ longitudinal analysis (between workloads); b $p<0.05$ transversal analysis (between environments).

healthy subjects with the same age, gender and body mass index and daily physical activity levels (Table 1). The study was approved by the local hospital's Ethics Commission and all subjects participated voluntarily with written informed consent of their parents, according to the Helsinki Declaration.

Study design

Study participants were enrolled in an exercise program in 2 different environments: a hot and humid environment (HH) with a mean temperature of 32.6±0.4ºC and relative humidity of 42.4±1.6%, and an environment with less humidity and a lower temperature (LHLT), with a mean temperature of 24.0±0.9ºC and relative humidity of 36.3±1.3%. The interspaced time between these two experimental conditions was at least 48 hours.

Exercise Protocol: For the HH and LHLT environments all participants underwent a 15 minute progressive exercise trial on a calibrated cycloergometer (*Monark 828 E – Ergomedic*), at 3 different workloads (30, 60 e 120 watts) of 5 minutes each, followed by 5 minutes of recovery (Figure 1).

Analysed variables: Heart rate (HR), respiratory quotient (RQ), tidal volume (TV), respiratory frequency (RF) and oxygen uptake (VO_2) (*Cosmed K4b2, Italy*) were monitored during the exercise protocol. For data analysis only steady-state values registered during the last minute of each workload were considered (Figure 1). For each environment, the forced expiratory volume in the first second (FEV_1) and peak expiratory flow (PEF) was obtained immediately before exercise and after 5 minutes of recovery (Figure 1), using *Cosmed K4b2* spirometry software following the American Thoracic Society guidelines [1]. Taking into account the pre-exercise values, the relative variation (%) of RQ, RF, TV, and VO_2 induced by the acute exercise was calculated and was used for intergroup comparisons. Regarding the HR absolute values registered during steady state of each bout of exercise, it was calculated for each subject their percentage of the theoretical maximum heart rate, being the percentage used for intergroup comparisons.

Statistical analysis

Data was analysed using statistical software (SPSS, version 15.0). Means and standard deviations were calculated for all variables. Regarding the reduced sample size of each group, comparisons between

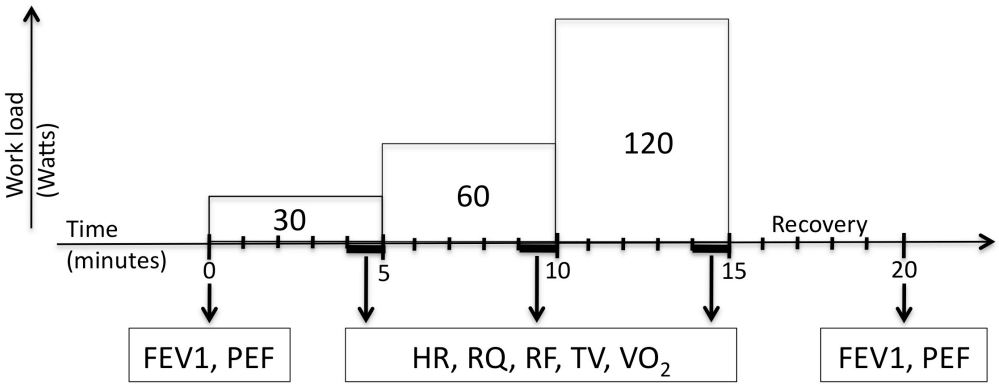

Figure 1. Experimental procedures followed in each tested environment. HR - heart rate; RF - respiratory frequency; RQ – Respiratory quotient; TV - tidal volume; VO2 – oxygen uptake; FEV1 – forced expiratory volume in one second; PEF – peak expiratory flow.

groups and/or environments were performed using non-parametric tests. Within each group, the comparisons between environments were assessed by the Wilcoxon test using the relative variations of each variable. The Mann-Witney test was used to establish comparisons between groups for each specific environment. The significance level was set at alpha=0.05.

RESULTS

Cardio-respiratory parameters

Data from all variables obtained during exercise under both environments for experimental and control groups are shown in Table 2. Comparing environments, we did not find statistical differences of RF, TV, VO2, RQ, and HR between groups for each workload, supporting the assumption that the performed workloads were identical for environments and groups.

Forced expiratory volume in the first second (FEV1)

Asthmatic subjects experienced a significant post-exercise decrease of FEV1 in both the LTLH (90.6±9.6%, $p<0.05$) and HH environments (95.2±5.8%, $p<0.05$) while healthy subjects did not show any significant post-exercise variation of this parameter in either environment (103.7±7.2% vs. 101.2±4.9%, respectively) (Figure 2). The EG FEV1 response observed in the LTLH environment was significantly different ($p<0.05$) from the response observed in CG under the same environment. When comparing the exercise response of both groups among environments, the asthmatic subjects experienced a larger post exercise decrease of FEV1 in the LTLH than in the HH environment ($p<0.05$) but no changes were registered among environments in CG (Figure 2).

Peak expiratory flow (PEF)

The asthmatic subjects showed a post-exercise PEF decrease in the LTLH (94.6±8.8%, $p<0.05$) and no changes in the HH environment (99.7±4.2%). In opposition, the healthy subjects did not experience significant changes in either environment (102.9±12.7% vs. 107.0±9.8%, respectively) (Figure 2). In asthmatic subjects, the PEF exercise response in the HH and LTLH environments was significantly different but no changes were registered for healthy subjects among environments (Figure 2).

DISCUSSION

The results of the present study suggest that the acute ventilatory response to exercise in asthmatic children is influenced by moderate changes of environmental conditions such as air temperature and humidity. Moreover, our data also show that the ventilatory response to exercise in asthmatic children seems to be different from their healthy counterparts.

Concerning FEV1, we found significant differences in FEV1 in asthmatics before and after exercise: in the LTLH environment, exercise resulted in a 9.4% fall of FEV1 and in the HH environment FEV1 fell 4.8% ($p<0.05$). In opposition, we did not find statistical differences in FEV1 in healthy subjects before and after exercise in both environments. Comparing both environments, a higher FEV1 fall was observed for asthmatics in LTLH than in HH after exercise. It is important to highlight that asthmatics did not interrupt medication over the evaluation days, which may have contributed to

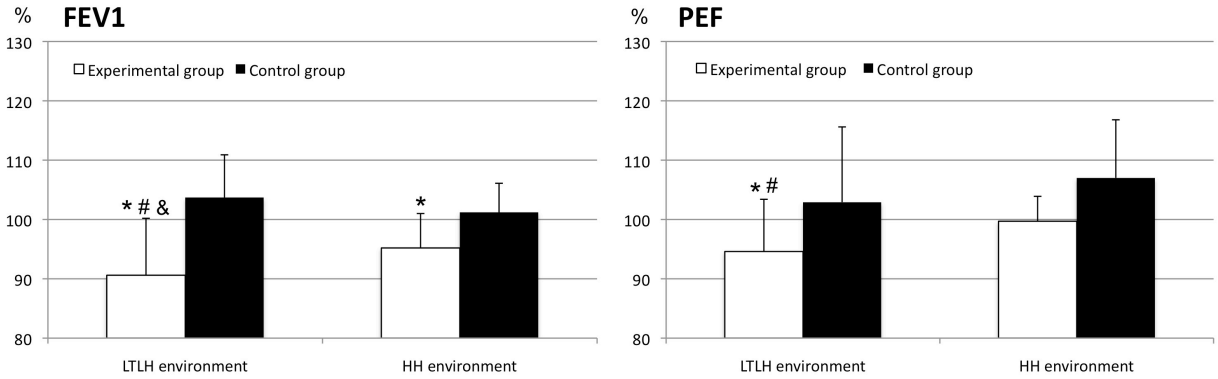

Figure 2. Percentage of variation (of pre-exercise values) of Peak Expiratory Flow and Forced Expiratory Volume in first second with exercise in a hot humid environment and in a less humidity and lower temperature environment in experimental (asthmatic) and control (healthy) groups. Values are mean±standard deviation. FEV1 - Forced Expiratory Volume in first second; PEF - Peak Expiratory Flow; HH - hot humid environment; LHLT - less humidity and lower temperature environment. * - $p<0.05$ vs. pre-exercise values; & - $p<0.05$ vs. control group; # - $p<0.05$ vs. HH environment.

smaller post-exercise decreases in FEV1. Indeed, in this group, the different magnitude of PEF falls observed after exercise in both environments supports the lesser tendency to exercise-induced bronchoconstriction in HH. Kallings and colleagues [19] found similar results in their study, observing that a 6 minute exercise of breathing hot and humid air induced a lower PEF fall (6.1±2%) than the same exercise while breathing cold and dry air (19.4±6% PEF fall).

It is also important to highlight that the majority of experimental designs described in the literature normally used higher temperatures and relative humidity ranges than those employed in our study. In fact, Amirav (2) observed that in young asthmatics, a 6 minute exercise of breathing dry and cold air induced a 39.2±8.5% FEV1 fall while breathing hot and humid air did not induce signals of bronchoconstriction. Another study has also shown a 48±5% FEV1 fall in asthmatic children after exercise in cold and dry environments, which did not occur in hot and humid environments or in the control group (3). Also, Eschenbacher (13) found that after 12 minutes of exercise FEV1 fell 20-21% in cold+dry, cold+humid and hot+dry environments in asthmatics; only in the hot+humid environment they did not find any evidence of bronchoconstriction.

When comparing groups, one could see that the response of asthmatic and healthy children to exercise showed different behaviours: asthmatic subjects showed signals of a bronchoconstriction response while healthy individuals evidenced signals of bronchodilatation. Indeed, we found statistical differences between asthmatics and healthy in the post-exercise FEV1 fall in the LTLH environment ($p<0.05$). In this environment, exercise apparently induced different airway responses concerning the groups: in asthmatics, FEV1 fell 9.4%, suggesting exercise-induced bronchoconstriction, while in the healthy, FEV1 increased 3.7%, suggesting a slight exercise-induced bronchodilatation. These results are concordant with Eschenbacher (13), who observed a 6% post-exercise FEV1 increase in healthy subjects. The explanation for this bronchodilatation involves an increase in catecholamines and of sympathetic tonus with exercise (24). In asthmatics, catecholamines apparently only have a protective effect against bronchoconstriction at least at the beginning of the exercise, restraining airway heat and water losses (3, 9); however, as the exercise progresses, the heat and water loss will gradually increase, overwhelming catecholamine-induced bronchodilatation and favouring the appearance of bronchoconstriction during the progression of exercise (9, 18, 2)]. Some recent studies are trying to demonstrate an association between respiratory heat and moisture loss and inflammation, suggesting that an increase in airway mucosal blood flow associated with airway inflammation in asthmatics would lead to a detectable increase in respiratory heat and moisture loss (31).

In the LTLH environment, we also found statistical differences between groups concerning the post-exercise fall of FEV1 ($p<0.05$); however, when compared to HH the asthmatic subjects evidenced a smaller bronchoconstriction response, suggesting that loss of bronchial temperature and/or water might be smaller in the HH environment. In fact, the degree of mucosal hydration of the bronchial tree seems to be affected by the rate of water evaporation and regulated by the ion composition and osmolarity of the mucosal secretions, the geometry of the

airways, the activity of the cilia, and the hydrostatic pressure of the mucosal blood vessels (35, 40). Therefore, the different airway response to HH and LTLH observed in asthmatic subjects might be explained mainly by local airway factors closely associated with the environmental conditions and not by hormonal or neural factors (3). This assumption is supported by the cardio-respiratory parameters analysis during exercise. It is important to highlight the absence of statistical differences between healthy and asthmatics regarding HR, RQ, VO2, RF, and TV, which is suggestive of a similar physiological impact of exercise in both groups (3, 39). On the other hand, we did not find any differences between environments regarding FEV1 and PEF variation with exercise in healthy subjects, suggesting that those differences found in asthmatics are necessarily due to intrinsic airway properties leading to an altered bronchial response to exercise.

Our results lead us to conclude that acute ventilatory responses to exercise seem to be influenced by moderate variations in air temperature and humidity and that this response is different in asthmatics and healthy subjects. Therefore, it appears that exercising in a HH environment could be more favourable for asthmatics than in a LTLH environment in terms of bronchoconstriction.

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