Exercise capacity and exercise-induced bronchoconstriction (EIB) in a cold environment

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KEYWORDS
Peak oxygen uptake; Peak running speed; Lung function; Asthmatic subjects; Cold environmental conditions

Summary

Introduction: Exercise in a cold environment has been reported to increase exercise-induced bronchoconstriction (EIB). However, the effect of a cold environment upon exercise capacity in subjects with EIB has, to our knowledge, not been previously reported.

Purpose: Primary: To examine the influence of changing environmental temperature upon exercise capacity measured by peak oxygen uptake ($\dot{V}O_{2peak}$), peak ventilation ($\dot{V}E_{peak}$) and peak running speed in subjects with diagnosed EIB.

Secondary: To assess the influence of changing environmental temperature upon EIB.

Methods: Twenty subjects (10–45 years old, male/female: 13/7) with EIB underwent exercise testing by running on a treadmill in a climate chamber under standardised, regular conditions, 20.2 ± 1°C (±1.1) and 40.0% (±3.3) relative humidity [mean (±SD)], and in a standardised cold environment, −18.0°C (±1.4) and 39.2% (±3.8) relative humidity in random order on separate days. Oxygen uptake ($\dot{V}O_2$), minute ventilation ($\dot{V}E$), respiratory exchange ratio (RER), heart rate (HR) and running speed were measured during exercise.

Lung function (flow volume loops) was measured before and 1, 3, 6, 10 and 15 min after exercise and 15 min after inhalation of salbutamol.

Results: $\dot{V}O_{2peak}$ decreased 6.5%, from 47.9 (45.0, 50.8) to 44.8 ml kg$^{-1}$ min$^{-1}$ (41.2, 48.4) [mean (95% confidence intervals)] ($p = 0.004$) in the cold environment. Also running speed was significantly lower in the cold environment ($p = 0.02$). No differences were found for $\dot{V}E_{peak}$, RER$_{peak}$ or HR$_{peak}$. The post-exercise reduction in forced expiratory volume in 1 s (FEV$_1$) ($\Delta$FEV$_1$) increased significantly from 24% (19,29) to 31% (24,38), respectively ($p = 0.04$) after exercise in the cold environment. No correlation was found between reduction in $\dot{V}O_{2peak}$ and the increased maximum fall in FEV$_1$ in the cold environment.
Conclusion: Exercise capacity (VO₂ peak and peak running speed) was markedly reduced during exercise in a cold environment whereas EIB increased in subjects suffering from EIB.

Material and methods

Design

The present study has an open randomised, cross-over design with one exercise test performed under standard, regular indoor conditions, temperature of 20 °C and 40.0% relative humidity, and another test in a standardised cold environment, −18 °C and 40% relative humidity on two different days. An interval of at least 48 h was required between the two tests. There were three study days in total. On day one, all subjects underwent an EIB-test to assess if they satisfied the inclusion criterion, reduction in forced expiratory volume in 1 s (FEV₁) ≥ 10% from before to after exercise. If satisfying inclusion criterion the subjects were randomised consecutively to one of the two climate blocks in random order generated by a computer programme. The study could not be blinded because the subjects could immediately feel which climate they went into. The present study was part of a larger study aiming to assess the effect of different environments, altitude and humidity upon exercise capacity and upon EIB in subjects suffering from EIB.

Introduction

Inspiring cold, dry air during exercise is reported to increase exercise-induced bronchoconstriction (EIB) in asthmatic subjects compared with regular, indoor environment and humid environment. 1-3

Most of the previous reports concern the effect of inspiring cold air through a mouthpiece, while the subjects are exposed to regular, laboratory environmental temperature. Only very few studies have investigated the effect of the whole body exposure to cold air upon exercise capacity and/or lung function in asthmatic subjects. 4-8

As far as we know, only three studies have investigated the influence of cold air upon oxygen uptake (VO₂) in asthmatic subjects 4-8 and only one of them has reported on maximum oxygen uptake (VO₂ max). 6 Kallings et al. 7 did not find any differences in VO₂ or other physiological parameters in asthmatic subjects during exercise under room tempered conditions when inhaling cold, dry air as compared with inhaling warm, humid air. Also Sandsund et al. 6 concluded with no differences in VO₂ at submaximal workloads, in VO₂ max or in lung function in seven mild asthmatic subjects between inhaling cold air and warm air in a cold environment during exercise. Eschenbacher et al. 8 found that the workload in watts performed per L min⁻¹ of oxygen consumed was significantly greater during the cold and dry conditions than during hot and humid conditions in eight male asthmatic subjects.

The effect of cold air on physiological parameters in healthy subjects is reported to vary depending on different factors such as type, intensity and duration of exercise, amount of fatty tissue, wind, ambient temperature, clothing, fluctuations in body temperature and energy reserves. 9 Quirion et al. 9 found significantly decreased VO₂ max, maximum workload and time to exhaustion, whereas minute ventilation (VE) did not change during a short exhaustive exercise at −20 and 0 °C as compared with 20 °C in eight healthy males. Sandsund et al. 10 reported increased VE and VO₂ at submaximal workloads in an environment of −15 °C as compared with 23 °C whereas no difference was found for VO₂ max. They suggested that exercise stress increased in a cold environment, probably as a response to increased metabolic demand. Their findings in healthy subjects are supported by Claremont et al. 11

As EIB influences daily life activities and sports activities in children and adolescents, an accurate assessment of EIB is important to enable optimal choice of treatment. EIB is best assessed by a standardised exercise test, commonly used is running on a treadmill for 6–8 min at a submaximal work load. 12,13 Lately it has been maintained that an exercise load corresponding to 95% of maximum heart rate (HR max) is preferable to obtain a high sensitivity. 14 EIB consists of bronchoconstriction occurring immediately or soon after physical exercise triggered by increased ventilation during exercise. 12,14-16 Two main hypotheses have been proposed to explain the relationship between exercise and EIB. Gilbert and McFadden 13 suggested that airway cooling is probably the cause of EIB. Anderson 18 suggested that respiratory water loss due to increased ventilation is the main stimulus to provoke EIB.

Although it has been generally accepted that cold air inhalation increases EIB, this has recently been challenged by Evans et al. 19 They concluded that cold air inhalation had no additive effect upon the severity of EIB after exercise or decrease in lung function after eucapnic voluntary hyper-ventilation.

However, it is not known if cold environment may influence exercise capacity or if there is a relationship between the magnitude of EIB and exercise capacity in subjects with EIB. Such knowledge is needed for giving optimal advice and treatment to asthmatic children and adolescents competing in different sports, especially endurance winter sports. It is also needed in relationship to regular physical training of asthmatic children and adolescents especially in the Scandinavian countries and in other countries with temperature to subartic climate where the winter season can be quite cold.

The aims of the present study were primarily to assess any possible change in exercise capacity measured by peak oxygen uptake (VO₂ peak), peak ventilation (VE peak) and peak running speed during exercise in a cold environment as compared to regular indoor environmental conditions and secondarily to assess the influence of cold environment upon EIB in subjects with diagnosed EIB.
The study was performed according to the principles stated in the Declaration of Helsinki. The Regional Medical Ethics committee approved the study and all subjects signed an informed written consent before inclusion.

**Ambient conditions**

On study days 2 and 3, the subjects performed exercise testing according to identical test procedures. The exercise tests were performed in a conditioned climate chamber (Norwegian Sub diving Techniques A/S, Haugesund, Norway) with relative humidity of 40.0% (±3.3) and temperature 20.2°C (±1.1) [mean(±SD)] on one of the study days and −18°C (±1.4) and relative humidity of 39.2% (±3.8) on the other study day. The barometric pressure during the exercise tests were 98.7 kPa (±1.1) or 740 mmHg (±8).

**Subj ects**

Twenty subjects between 10 and 45 years of age with diagnosed EIB were included in the study. EIB was defined by a reduction in FEV1 of 10% or more from before to after a standardised EIB-test performed under standard, regular conditions. Exclusion criteria consisted of any other diseases or use of any regular medication that might influence test results and any respiratory tract infection during the last 3 weeks before study inclusion. The subjects were also excluded if the baseline FEV1 measurement varied more than 5% between the two test days.

Antiasthmatic medication was withheld according to ERS guidelines. Inhaled short-acting β2-agonists and sodium cromoglicate were withheld for 8 h prior to testing, inhaled long-acting β2-agonists, theophylline and leukotriene antagonists for the last 72 h, anti-histaminic for the last 7 days and orally administered glucocorticosteroids for the last 7 days.

Seventeen of the 20 subjects were atopic as defined by positive skin prick test (SPT). Seven subjects used regular inhaled steroids and ten subjects used regular daily long-acting inhaled β2-agonists. Seventeen subjects used short-acting β2-agonists on demand, one subject used oral theophylline and two subjects used a leukotriene antagonist daily. Four subjects used antihistamines, whereas nine subjects were without any regular asthma medication. Five subjects participated in competitive sports, 14 participated in regular physical activity in school or leisure time, and one subject rarely or never participated in physical activity.

**Lung function**

Lung function was measured by maximally forced expiratory flow volume loops (Mastertab, Erich Jaeger, Germany). FEV1, forced vital capacity (FVC) and forced expiratory flow at 50% of FVC (FEF50) were measured before exercise and 1, 3, 6, 10 and 15 min after exercise and 15 min after inhaled salbutamol (5 mg mL−1; 0.05 mg kg−1). All lung function measurements were performed in a regular, indoor environment outside the climate chamber. All manoeuvres complied with the general acceptability criteria of The European Respiratory Society (ERS). Predicted lung function values, when used, were according to Zapletal et al.23 and Quanjer et al.22

**Exercise test**

EIB was determined by running on a motor-driven treadmill (“Bodyguard” 2313, Sweden) for 8 min at a submaximal work load.12 The inclination of the treadmill was 5.3%. The running speed was adjusted during the first 4 min to achieve a work load corresponding to the maximum speed the subjects were able to maintain the last 4 min, about 95% of estimated HRmax (220 beats min−1-age). If the subjects indicated that higher speed was necessary to achieve exhaustion after 8 min the running speed was also adjusted after 5 and 6 min. The estimated HRmax is elaborated from epidemiological studies, and it is a circumstantial estimation for individual subjects. The standard deviation for maximum heart rate during exercise is ±10 beats min−1. Therefore, the exercise workload was standardised by a combination of 95% of estimated HRmax and the test leader’s evaluation of exhaustion after 8 min. VO2, VE, breathing frequency (BF) and respiratory exchange ratio (RER) were measured 5, 6 and 7 min after starting exercise test. The EIB protocol used in our study is different from a standard, incremental protocol for assessing VO2peak, but has been evaluated in a previous study. A comparison of the EIB protocol and a stepwise protocol showed no difference in VO2peak or VEpeak.24 Douglas bags were used for collecting gas samples of the expired gas.25 The variations reported for the Douglas bag method used with cycle ergometry are 2.3–2.5% for daily variations and 3.3–5.1% for between days variations.26 The Douglas bag system was chosen because the measurements with the automatic equipment were unstable and not reproducible in the cold environment.

The subjects, wearing a nose clip, breathed through a Hans Rudolph mouthpiece (2700 Series; Hans Rudolph Inc, USA). Expiratory gas samples were taken for at least 30 s and analysed for the oxygen and carbon dioxide content (Oxygen analyser model S-3A/1 and Carbon dioxide analyzer model CD-3A; Ametek Inc, USA). The volume, temperature and pressure of the expired gas were measured at the time the air was analysed (“Ventilation measuring system”, model S-430, KL-Engineering, Northridge, California, USA). The heart rate (HR) was recorded electronically and registered every minute (Polar Sports tester PE 300010, Polar Electro Oy, Kempele, Finland).

Maximum percentage reduction in FEV1 after exercise test was calculated by (pre-exercise FEV1−minimum post-exercise FEV1)/(pre-exercise FEV1) × 100%. Minimum post-exercise FEV1 was the lowest recorded value at 1, 3, 6, 10 or 15 min after exercise test. Similar calculations were performed for FEF50 and FVC. The highest recorded HR, VO2, VE, BF, RER and running speed during exercise tests were determined as HRpeak, VO2peak, VEpeak, BFpeak, RERpeak and peak running speed.

Assuming that the inhaled air during exercise is fully saturated with vapour and reaches the temperature of 37°C, the respiratory water loss during the last 3 min of exercise was calculated by using a web-based online calculator designed by the Department of Physics and Astronomy Georgia State University Atlanta, based on
empirical fit for density data (http://hyperphysics.phyastr.gsu.edu/hbase/kinetic/relhum.html 2004).

**Skin prick test**

The skin prick test was performed according to the Nordic guidelines 22 with the following prevalent ambient allergens: mounds (Cladosporium herbarum), house dust mites (Derma-
tohagoideus pteronyssimus), dog dander, cat dander, birch pollen, grass pollen (timothy), mug worth pollen, milk, shrimp and egg (Soluprick, ALK, Copenhagen, Denmark). To be considered allergic to an allergen, a positive skin prick test of at least ++ (1/2 of the reaction to histamine) was required. The size was recorded by measuring (maximum+minimum diameter (mm)) × 2⁻¹.

**Statistical analysis**

Demographics are given as mean values and standard deviation (SD) and results as means with 95% confidence intervals (CI). Differences between the two tests were analysed by Student’s paired t-tests when satisfying normal distribution. Correlation was calculated by Pearson’s correlation coefficient. The bronchoconstrictor response following exercise was measured as the maximum per cent fall in FEV₁ and FEF5₀ after exercise and the area under the curve (AUC) per cent fall of the pre-exercise value in FEV₁ time⁻¹, up to 15-min post-exercise, using the trapezoid rule. Identical analysis was made for FEF5₀. If FEV₁ or FEF5₀ increased from baseline after exercise, the corresponding area was subtracted from the AUC measurements. All tests were two-tailed with a significance level of 5%.

Based upon FEV₁ and VO₂ peak as main variables, with pre-existing knowledge of the variation of these variables and assuming a power of 80%, a sample size of 20 subjects was calculated as necessary to obtain a significance level of 5%. Statistical analyses were performed with Statistical Package for Social Sciences (SPSS) version 11.0.

**Results**

Demographic data and baseline lung function are given in Table 1. Baseline lung function (FEV₁, FEF5₀ and FVC) did not differ significantly on the two test days. VO₂ peak decreased significantly, 6.5%, from 47.9 ml kg⁻¹ min⁻¹ (45.0, 51.8) [mean (95% confidence intervals)] to 44.8 ml kg⁻¹ min⁻¹ (41.2, 48.4), respectively (p = 0.004) during exercise under regular conditions as compared with exercise in the cold environment (Table 2). Four subjects reduced VO₂ peak more than 10%, nine subjects had a reduction between 5 and 10% and six subjects reduced VO₂ peak less than 5% in the cold environment. One subject increased VO₂ peak 5% in the cold environment. Peak running speed was also significantly lower in the cold environment: 10.2 km h⁻¹ (9.5, 11.0) vs. 9.7 km h⁻¹ (8.9, 10.5), respectively (p = 0.02) (Table 3). There were no differences in VE peak, RER peak, HR peak or BF peak during exercise between the two climatic conditions (Table 2). VO₂ was significantly reduced after 5, 6 and 7 min run in the cold environment (p = 0.01 (Fig. 1). The running speed was also significantly lower in the cold environment after 5 and 7 min (p = 0.01 and p = 0.03, respectively) (Fig. 1). No significant differences were found for VE, RER,
BF or HR after 5, 6 and 7 min run between the two climatic conditions.

Maximum reduction in FEV1 and AUC for FEV1 increased significantly after exercise in the cold environment as compared with regular, indoor conditions. Maximum reduction in FEV1 as per cent of baseline lung function after exercise in the cold environment was 31% (24, 38) vs. 24% (19, 29), respectively, after exercise under regular conditions (p = 0.04) (Table 3). AUC for FEV1 was higher after exercise in the cold air, 358 (261, 455) vs. exercise under regular conditions, 250 (182, 317), respectively (p = 0.01) (Table 3).

Increased maximum reduction in FEF50 after exercise in the cold environment was also found; 47% (38, 55) vs. 38% (30, 46), but on the border of significance (p = 0.06). Maximum reduction in FVC as per cent of baseline lung function or AUC for FEF50 did not differ significantly between the climatic conditions (Table 3). Reduction in FEF50 was significantly higher 1 and 6 min after exercise in the cold environment (Fig. 2).

Calculated respiratory water loss during the last 3 min of exercise in the cold environment was 12.5 g (10.8, 14.3) vs. 10.8 g (9.7, 12.0) under regular indoor conditions (p = 0.03).

No significant correlation was found between reduction in lung function after exercise and water loss during the last 3 min of exercise. Nor was there any significant correlation between increased maximum fall in lung function (measured by FEV1 and FEF50) or increased AUC after exercise and reduced VO2peak in the cold environment.

**Discussion**

The present study demonstrated that exercise capacity measured by VO2peak and peak running speed decreased significantly during exercise in a cold environment as compared with regular environmental conditions, whereas VEpeak, RERpeak and BFpeak did not differ in subjects suffering from EIB (Table 2).

Maximum reduction in FEV1 after exercise and AUC for FEV1 increased significantly in the cold environment as compared with exercise under standard, regular conditions. Maximum reduction in FEF50 did not reach statistically significant difference. The increased reduction in FEF50 reached statistical significance only at 1 and 6 min after exercise in the cold environment whereas AUC for FEF50 did not change (Fig. 2 and Table 3). Mean FEF50 at baseline was only 74% and 76% of that predicted (Table 1). This demonstrates the presence of airway obstruction in the peripheral airways in this group of asthmatics. Only seven out of 20 subjects used anti-inflammatory treatment (inhaled steroids).

According to the present study, the differences in VO2 and running speed occur when the subjects were close to their maximal aerobic capacity, the last 3 min of the EIB-test (Table 2 and Fig. 1). No correlation was found between maximum reduction in lung function (FEV1 or FEF50) after exercise or water loss during exercise and the reduced VO2peak in the cold compared to the regular environment. The lack of correlation is possibly due to the number of subjects included. The power is probably too weak to detect any association. Nor can the reduction in VO2 be explained by reduction in VE. No significant difference was found in VE during the last minutes of the tests or in VEpeak (Fig. 1 and Table 2) between the two climatic conditions.

All except three subjects reported spontaneously that breathing during exercise in the cold environment was much more difficult as compared with that in regular conditions. These statements support that the subjects ran slower during the last 4 min of the test with decreased VO2peak in the cold environment. Studies aiming to imitate "real climatic conditions", like the present study, cannot be blinded and psychological factors might influence the results. To minimise these effects, objective measurements and well-standardised test procedures are necessary. In the present study the standardisation of the exercise load was based upon the screening test of the individual subjects aiming a submaximal to maximal exercise load as assessed by HR. The speed of the treadmill thus becomes a measure of performance during the two different climatic conditions.

The measurement of VO2 in the cold environment was challenging because the instruments used for direct and continuously VO2 measurements during exercise did not work in −18 °C. The Douglas Bag System used in the present study is a precise and well-documented instrument, and it is in fact recognised as a "gold standard". The disadvantage using the Douglas Bag system was that the VO2 measurements during the entire exercise period and the feasibility to measure tidal breathing flow volume loops during exercise were missed.

The causes of reduced VO2peak and peak-running speed in the cold environment are unknown. Possibly, an increased

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**Table 3** Difference (Δ) in maximum reduction in FEV1, FEF50 and FVC (% of baseline) and area under curves (AUC) for FEV1 and FEF50 after exercise test in a standardised regular environment, 20.2 °C (±1.1) and 40.0% (±3.3) relative humidity [mean(±SD)] and in a standardised cold environment, −18 °C (±1.4) and 39.2%(±3.8) relative humidity (n = 20).

<table>
<thead>
<tr>
<th>Variables</th>
<th>20 °C</th>
<th>−18 °C</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔFEV1 (%)</td>
<td>24 (19,29)</td>
<td>31 (24,38)</td>
<td>0.04</td>
</tr>
<tr>
<td>ΔFEF50 (%)</td>
<td>38 (30,46)</td>
<td>47 (38,55)</td>
<td>ns</td>
</tr>
<tr>
<td>ΔFVC (%)</td>
<td>15 (11,19)</td>
<td>20 (14,27)</td>
<td>ns</td>
</tr>
<tr>
<td>AUC (FEV1)</td>
<td>250 (182,317)</td>
<td>358 (261,455)</td>
<td>0.01</td>
</tr>
<tr>
<td>AUC (FEF50)</td>
<td>386 (276,495)</td>
<td>485 (364,606)</td>
<td>ns</td>
</tr>
</tbody>
</table>

Values are given as mean with 95% confidence intervals in parentheses. ns = not significant.
strain level, especially for asthmatics starting exercise on a high intensity in a cold environment without warming-up, might reduce the performance. Neither \( \text{HR}_{\text{peak}} \) nor \( \text{RER}_{\text{peak}} \) differed during the two tests and indicate that the subjects achieved equal level of exhaustion even though the running speed was reduced in the cold environment. The subjects were only exposed to the cold environment for 10 min and no freezing or shivering were observed or reported. As they wore warm clothes suited for the cold environment, the decrease in \( \text{VO}_2 \) peak had probably a direct relation to reduced running speed during exercise. An EIB-test with pre-medication of inhaled \( \beta_2 \)-agonists in the cold environment or a control group of EIB-negative subjects might explain if the airway calibre is a possible reason. Our findings are supported by the study from Quirion et al.\(^9\) on healthy subjects. They demonstrated that \( \text{VO}_2 \) max significantly decreased and the VE did not change in \(-20\) and \(0\) °C as compared with that in \(20\) °C, and their subjects reported that submaximal exercise intensities were more tiring in a cold environment as compared with those in a warm environment. They suggested that the net efficiency of exercise at low temperatures is lower than under normal conditions. On the other hand, Sandsund et al.\(^{10}\) reported

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**Figure 1**  Oxygen uptake (\( \text{\text{VO}_2} \)), minute ventilation (\( \text{\text{VE}} \)), heart rate (HR) and running speed after 5, 6 and 7 min exercise test under standardised regular conditions (\( \bullet \)) and under standardised cold conditions (\( \triangle \)) \( (n = 20) \). Results are given as mean with 95\% confidence intervals. (*) = statistical significance.

**Figure 2**  Lung function (FEV\(_1\) and FEF\(_{50}\)) before and 1, 3, 6, 10 and 15 min after exercise and 15 min after inhaled salbutamol in a standardised regular environment (\( \bullet \)) and in a standardised cold environment (\( \square \)) \( (n = 20) \). Results are given as mean with 95\% confidence intervals. (*) = statistical significance.
increased VO₂ in eight healthy male athletes at submaximal exercise intensities in a cold environment compared with those in standard, indoor conditions, but there was no difference in VO₂ max. Time to exhaustion was shorter in the cold environment. They suggested that exercise stress is higher at submaximal exercise intensities in a cold environment in agreement with the reduced running speed during exercise in the present study. Claremont et al.11 tried to explain the same observation by a catecholamine calorigenic effect.

In the studies of asthmatics from Kallings et al.,7 Sandsund et al.6 and Eschenbacher et al.,8 only six, seven and eight subjects, respectively, were included, and their results only serve as pilot studies indicating the need for further investigations. The workload differed markedly between these studies and also from the present study. The workload, ventilation and the demand for oxygen is too low in the study from Kallings et al. and Eschenbacher et al. in order to be able to discover any difference in VO₂ peak as compared with the exercise load at which the difference occurred in the present study.

Sandsund et al.6 found no differences in VO₂ max, VE max, HR peak or blood lactic acid when inhaling cold or warm air during exercise. However, in their study the temperature of the environmental air was −15 °C, the breathing mouth-piece acted as a heat exchanger and increased the inspired cold air to 2 °C. This is most probably not cold enough to observe any differences in lung function or in the physiological variables. Their exercise protocol was in fact not an exercise test for provoking EIB but a stepwise protocol for measuring anaerobic threshold and VO₂ max with a 20 min warming-up period.

The present study confirms previous reports that inhalation of cold air increases EIB in asthmatic subjects.1,2 On the other hand, neither Evans et al.19 nor Sandsund et al.6 could find any additive effect of cold air inhalation upon EIB. The temperature of the inhaled air in their studies was actually −1 and 2 °C, respectively, and probably not cold enough to discover any difference. Evans et al.19 mentioned that lack of exposure to ambient cold air during inhalation may explain the lack of an additive effect.

However, cold environmental conditions seem to aggravate the effect on EIB, and the respiratory water loss significantly increased in the cold environment as compared with that in the regular, indoor conditions. Air of 37 °C fully saturated with vapour contains 44 g H₂O/m³. Air of temperature 20 °C with 40% relative humidity contains 6.9 g H₂O/m³ and air of −18 °C with 40% relative humidity contains 0.01 g H₂O/m³. When the ventilation rates increase during exercise, the water loss increases. These findings indicate that the worsening effect on EIB in asthmatics is partly due to increased water loss and partly due to heat loss and support earlier reports on EIB and cold environment.1,3,7,24,30

Our findings are also supported by Zeitoun et al.5 and Koskela et al.4 Zeitoun et al.5 concluded that facial cooling combined with either cold or warm air inhalation causes the greatest EIB as compared with the isolated challenge with cold air inhalation. They suggested that vagal mechanisms activated by changes in osmolarity play a major role in exercise and cold-induced bronchoconstriction. Koskela et al.4 reported that, for certain stable asthmatic subjects, even a moderate level of exercise can cause bronchoconstriction in climatic conditions similar to a Scandinavian winter. They also found that even sitting in −20 °C caused a greater bronchoconstriction than moderate exercise in room temperature and stated that this could not be explained by hyperventilation-induced airway drying alone, but that the reflex mechanism is more important than was previously thought.4 Boulet and Turcotte30 reported that EIB was influenced by the changes in water content during and after exercise. The recovery period in the present study was in regular, indoor environmental conditions and according to Boulet and Turcotte30 the best recovery environment to protect against EIB.

The choice of including subjects with relatively large range in age was to reflect the period of life extending from schoolage to adulthood, where human beings are physically active and spending time on physical activity. The results from the present study can contribute to giving this group of asthmatics better advice and treatment before exercising in a cold environment. Previous reports have shown that both asthmatic children and asthmatic adults seem to respond equally upon exercise in a cold environment.1,2 In the present study, the subjects below 16 years (n = 6) had the same reduction in VO₂ peak in the cold conditions as the subjects above 16 years (n = 14).

In conclusion, exercising in a cold environment decreases exercise capacity as measured by VO₂ peak and peak running speed, and increases EIB in subjects suffering from EIB. This has important implications for training procedures in a cold environment for patients and athletes with EIB. Although similar effect of a cold environment upon exercise capacity in healthy subjects cannot be excluded.

Acknowledgment

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References

6. Sandsund M, Faerenvik H, Reinertsen RE, Bjermer L. Effects of breathing cold and warm air on lung function and physical


24. Stensrud T, Carlsen KH. Can a test protocol for provoking exercise-induced bronchoconstriction also be used for assessing peak oxygen uptake? 2006; submitted.


