

# Airway Hyperresponsiveness in Elite Athletes

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It has been suggested that high-level training could contribute to the development of airway hyperresponsiveness (AHR), but the comparative effects of different sports on airway function remains to be determined. We evaluated 150 nonsmoking volunteers 18 to 55 yr of age; 100 athletes divided into four subgroups of 25 subjects each according to the predominant estimated hydrocaloric characteristic of ambient air inhaled during training: dry air (DA), cold air (CA), humid air (HA) and a mixture of dry and humid air (MA), and 50 sedentary subjects. Each subject had a respiratory questionnaire, a methacholine challenge, allergy skin-prick tests, and heart rate variability recording for evaluation of parasympathetic tone. The athletes had a 49% prevalence of AHR ( $PC_{20} < 16$  mg/ml), with a mean  $PC_{20}$  of 16.9 mg/ml, compared with 28% ( $PC_{20}$ : 35.4) in sedentary subjects ( $p = 0.009$ ). The prevalence (%) of AHR and mean  $PC_{20}$  (mg/ml) varied as followed in the four subgroups of athletes: DA: 32% and 30.9; CA: 52% and 15.8; HA: 76% and 7.3; and MA: 32% and 21.5 ( $p = 0.002$ ). The estimated parasympathetic tone was higher in athletes ( $p < 0.001$ ), but this parameter showed only a weak correlation with  $PC_{20}$  ( $r = -0.17$ ,  $p = 0.04$ ). This study has shown a significantly higher prevalence of AHR in athletes than in the control group because of the higher prevalence in the CA and HA groups. Parasympathetic activity may act as modulator of airway responsiveness, but the increased prevalence of AHR in our athlete population may be related to the type and possibly the content of inhaled air during training.

Physical exertion, particularly when intense and prolonged, causes significant stress to the respiratory system, from the associated hyperventilation and increased airway exposure to contaminants of inhaled air (1–4). Recent studies have generally shown a higher prevalence of airway hyperresponsiveness (AHR) in athletes, particularly those performing winter sports such as cross-country skiing, ice skating, or hockey than in the general population (5–9). More and more reports suggest that many athletes have a physician-made diagnosis of asthma or show an abnormal fall in expiratory flows after exercise challenge tests (10–12).

Most studies showing an increased airway responsiveness have been done using exercise as the provoking stimulus. The possibility that this reflects only a normal response to a supra-physiologic stimulus has been contradicted by recent reports showing that this increased airway response could also occur in athletes after other stimuli such as methacholine (13).

Factors predisposing the athletic population to asthma and AHR have not been confirmed. High-intensity exercise could induce or promote airway inflammation, either from hyperventilation or from an increased airway exposure to inhaled allergens or pollutants (14). Other factors may also be involved in the development of AHR such as a predominance of

the parasympathetic nervous system over the sympathetic one, a phenomenon that has been reported in athletes (15).

Tremendous resources and efforts are invested in developing high levels of performance by athletes. It is therefore mandatory to know whether these subjects are more prone to respiratory diseases such as asthma and, if so, to suggest preventive measures and early treatment. Furthermore, we must identify any sports that may increase the risk of airway diseases. The goal of this study was therefore to determine the prevalence of physician-diagnosed asthma and of AHR to methacholine in different groups of athletes involved in high-level competitions, compared with a control group of sedentary subjects not involved in regular physical activity. We also evaluated the impact of the hydrocaloric characteristics of the air inhaled during exercise and of changes in vagal activity.

## METHODS

### Subjects

One hundred high-level athletes (65 male, 35 female) and 50 sedentary controls (25 male, 25 female) from the Quebec City area agreed to participate in this project. Athletes were classified according to the predominant hydrocaloric characteristics of ambient air inhaled during competitive training: (1) dry air (DA) group ( $n = 25$ ): long-distance running ( $n = 14$ ), mountain biking ( $n = 11$ ); (2) cold air (CA) group ( $n = 25$ ): biathlon ( $n = 1$ ), cross-country skiing ( $n = 13$ ), speed-skating ( $n = 11$ ); (3) humid air (HA) group: swimming ( $n = 25$ ), and (4) mixed air (MA) group: triathlon ( $n = 25$ ), which represents exposure to a combination of dry and humid air. The term "dry air" means not fully saturated and usually warm air. Athletes involved in long-distance running, swimming, and triathlon were recruited through contacts with coaches of local university teams. Cross-country skiers and speed-skaters came from a national team, and the mountain bikers were from a regional team, some competing at the national level. To avoid any selection bias, the athletes were selected randomly from a list provided by the coaches. In order to obtain 100 participants, we had to contact a total of 112 athletes; nine athletes declined participation for various reasons, mostly because of time constraints, and three were excluded for noncompliance to pretest recommendations.

**Inclusion criteria.** All subjects had to be nonsmokers ( $< 1$  pack-year of cigarette smoking and not currently smoking) and from 18 to 55 yr of age. Athletes had to be enrolled in a recognized sports federation program and currently being trained by a professional coach. Sedentary subjects were solicited through advertisements. They could not be involved in sport activities for more than 2 h per week.

**Exclusion criteria.** We excluded subjects with any evidence of respiratory disease other than asthma or rhinitis, clinical evidence of upper respiratory infection during the 3 wk prior to participation, or any condition that could interfere with the proposed evaluation.

The study was approved by the Institution Ethics Committee, and each subject signed an informed consent form.

### Study Design

The cohort of 100 athletes and 50 control subjects was evaluated between November 1997 and May 1998. All subjects were tested in the morning. They were asked to fill out two questionnaires. After a standard physical examination, heart rate variability (HRV) measurements were taken as an indirect measure of parasympathetic tone (16). Baseline spirometry was followed by a methacholine challenge

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and allergen skin prick tests. All subjects were instructed not to participate in any sports and to avoid caffeine for 12 h prior to the day of the tests. Asthmatic patients were given appropriate instructions regarding the washout period for asthma medications before methacholine challenge.

**Questionnaires.** During a one-on-one interview with the examiner, each subject completed a first questionnaire on exercise-induced symptoms, including cough, anterior or posterior rhinorrhea and sensation of secretions in the throat, breathlessness, wheezing, and chest tightness. Then, a more exhaustive questionnaire was filled out by the subject; this included personal and family history of respiratory diseases, allergies, lifestyle, past physician-made diagnosis of asthma, use of asthma medications, respiratory symptoms, past smoking habits and smoking exposure at home, at work, and in recreational environments. For athletes, all sports practiced were recorded in order of priority based on training schedule, average time spent per week, and number of years of training for each.

**Baseline expiratory flows and methacholine challenge.** Baseline expiratory flows were obtained with a wedge spirometer (Vitalograph PFT II; Vitalograph Ltd, Buckingham, UK) according to the standard guidelines of the American Thoracic Society (17). Three reproducible measurements of FEV<sub>1</sub> and FVC were obtained. Airway responsiveness to methacholine was measured according to the method described by Juniper and colleagues (18). After baseline measurements of FEV<sub>1</sub> and FVC, each subject inhaled saline (0.9%) followed by doubling concentrations of methacholine between 0.03 and 128 mg/ml to obtain a > 20% fall in FEV<sub>1</sub>. Methacholine aerosols were generated by a Wright nebulizer with an output of 0.13 ml/min and were inhaled for 2 min at 5-min intervals. The FEV<sub>1</sub> was measured 30, 90, and 180 s after each inhalation or until it started to increase. FVC was also measured at 90 s. The concentration of methacholine inducing a 20% fall in FEV<sub>1</sub> (PC<sub>20</sub>) was obtained by linear interpolation on the log concentration response-curve. Significant airway hyperresponsiveness was defined as a PC<sub>20</sub> < 16 mg/ml (19).

**Allergy skin prick tests.** Atopic status was determined by skin-prick tests with 26 common airborne allergens, including animal danders, house dust mite, mixed trees, mixed grasses, pollens, and molds. The presence of atopy was defined as at least one positive ( $\geq 3$  mm mean wheal diameter at 10 min) response to allergens in the presence of a negative control.

**Heart rate variability.** Autonomic nervous system activity was estimated by using heart rate variability analysis techniques (HRV) (20). This last parameter is the recurrent change in the interval between R waves of the QRS complex obtained on electrocardiographic measurements. This variation from one cardiac cycle to another is considered dependent on both sympathetic and parasympathetic tone modulation. Parasympathetic tone was therefore estimated from the standard deviation of all normal-to-normal intervals (SDNN) from a 5-min segment of ECG recording, a currently recommended time domain measure (16). All subjects were lying in a 40-degree angle bed in a quiet dark room at 24° to 25° C and instructed to rest for 20 to 30 min. Using a Mini-Logger, Series 2000 logger, and software (Mini-Mitter Co., Sunriver, OR), we collected the 5-min bloc of interbeat intervals between the 20th and the 30th minute of the resting period for calculation of SDNN, expressed in milliseconds.

## Data Analysis

Results are expressed as mean  $\pm$  SEM. Mean values of FEV<sub>1</sub>, FVC, log-transformed PC<sub>20</sub> methacholine, and changes in FVC ( $\Delta$ FVC) were compared by ANOVA between sedentary subjects and the four athlete groups. As normality and variance assumption were not encountered for parameters, data were transformed by their ranks to perform statistical analysis. Changes in FVC ( $\Delta$ FVC) for a given concentration of methacholine was defined as the difference between FVC at that concentration and after FVC saline inhalation over the FVC measured after saline  $\times 100$ . Categorical parameters were analyzed with Fisher's exact test. Spearman's rank correlation was used to measure the relationship between parameters. The PC<sub>20</sub> value obtained from the methacholine challenge test, reflecting the degree of AHR, implies that the lower the PC<sub>20</sub> value, the higher the degree of AHR. So a parameter correlating negatively with PC<sub>20</sub> is equivalent to a positive correlation with AHR. Subjects with a PC<sub>20</sub> < 128 mg/ml

were assigned a PC<sub>20</sub> value of 128 mg/ml for statistical purpose. All reported p values were declared significant at the 0.05 level.

## RESULTS

### Subject Characteristics

All recruited subjects completed the evaluation. Mean age ( $\pm$  SEM) for athletes was 24.9  $\pm$  0.9 yr and for the control group, 26.8  $\pm$  1.1 (p > 0.05) (Table 1). Subjects from the DA group were the oldest (mean age, 32.9  $\pm$  2.2 yr), and those from the HA group were the youngest (20.6  $\pm$  1.5 yr, p = 0.0001). These two groups also differed for the mean number of hours spent training each week: subjects from the DA group had the shortest training duration time, with 12.4 h/wk and those from the HA group had the longest, with 19.1 h/wk (p = 0.0002). The mean number of years involved in their respective sport was quite similar among the DA, CA, and HA groups, with 10.0, 11.5 and 10.7 yr, respectively. Only the MA group significantly differed from the others, with 2.7 yr of training duration (p = 0.0001); this can be explained by the fact that triathlon has gained popularity among athletes in Canada only recently.

Prevalence of atopy was high in all groups: DA, CA, HA, MA, and control groups had 80, 88, 84, 52, and 82%, respectively, with the MA group being the only one significantly different from the other groups (p = 0.029). For the whole group of athletes, the prevalence was 76% and not significantly different from that found in the control group (p > 0.05) (Table 1).

When athletes and control subjects were compared for the prevalence of physician-made diagnosis of asthma done prior to study, there was no significant difference between them and control subjects (p > 0.05). When the four groups of athletes were compared, the DA and the CA groups were both significantly different from the others (p = 0.005) (Table 1). Use of asthma medications is summarized in Table 1.

### Respiratory Symptoms

Athletes showed a higher prevalence of cough with exercise than did sedentary subjects (p = 0.0015) (Table 2), and among the four groups of athletes, the CA group showed the highest prevalence (p < 0.0001). The prevalence of rhinorrhea and pharyngeal secretions with exercise was higher in athletes than in the control group (p = 0.01), but between the four groups of athletes there were no significant differences (p > 0.05). Symptoms of breathlessness, wheezing, or chest tightness with exercise were not significantly different between athletes and control subjects (p > 0.05), but among the four groups of athletes, only the CA group had a significantly higher prevalence of symptoms (p = 0.04).

### Expiratory Flows and Methacholine Responsiveness

As shown in Table 3, FEV<sub>1</sub> was normal in all groups. The prevalence of AHR in the athlete group was 49%, with a mean PC<sub>20</sub> (geometric mean) of 16.9 mg/ml, which differed significantly from the control group, with 28% prevalence and a mean PC<sub>20</sub> of 35.4 mg/ml (p = 0.015). This difference in prevalence was also marked for PC<sub>20</sub> < 8 mg/ml (p = 0.037) (Table 3), but even more so for PC<sub>20</sub> < 2 mg/ml (p = 0.0049) (Figure 1). This higher prevalence among the athletes was mostly due to the HA group, where 19 of 25 swimmers were hyperresponsive and had the lowest mean PC<sub>20</sub> (7.3 mg/ml), followed by the CA group where 13 subjects were hyperresponsive (52%, mean PC<sub>20</sub>: 15.8 mg/ml). The DA and MA groups had a prevalence of 32% each, with, respectively, PC<sub>20</sub> of 30.9 and 21.5 mg/ml, showing no significant difference from the control group.

TABLE 1  
SUBJECTS' CHARACTERISTICS\*

	Control Subjects (n = 50)	Athletes				
		All (n = 100)	DA (n = 25)	CA (n = 25)	HA (n = 25)	MA (n = 25)
Sex, M/F	25/25	65/35	24/1	16/9	11/14	14/11
Age, yr*	26.8 (1.1)	24.9 (0.9)	32.9 (2.2)	23.9 (1.7)	20.6 (0.3)	22.8 (0.8)
Hours of training/week*	NA	16.7 (0.5)	12.4 (0.7)	16.6 (1.3)	19.1 (1.3)	13.8 (1.0)
Years involved in this current discipline*	NA	8.3 (0.6)	10.0 (2.0)	11.5 (0.9)	10.7 (0.7)	2.7 (0.4)
Atopy <sup>†</sup>	41 (82)	76 (76)	20 (80)	22 (88)	21 (84)	13 (52)
History of physician-diagnosed asthma <sup>†</sup>	2 (4)	16 (16)	5 (20)	7 (28)	2 (8)	2 (8)
Use of inhaled corticosteroids with short-acting $\beta_2$ -agonist <sup>†</sup>	0 (0)	6 (6)	3 (12)	2 (8)	1 (4)	0 (0)
Use of inhaled corticosteroids alone <sup>†</sup>	1 (2)	1 (1)	0 (0)	1 (4)	0 (0)	0 (0)
Use of inhaled short-acting $\beta_2$ -agonist alone <sup>†</sup>	1 (2)	7 (7)	2 (8)	3 (12)	1 (4)	1 (4)

Definition of abbreviations: CA = Cold Air group; DA = Dry Air group; HA = Humid Air group; MA = Mixed Air group; NA = not applicable.

\* Values are means with SEM shown in parentheses.

<sup>†</sup> Number of subjects, with percentage shown in parentheses.

### Heart Rate Variability

The mean SDNN  $\pm$  SEM was  $88.2 \pm 3.6$  ms for the whole group of athletes compared with  $62.7 \pm 4.8$  ms for the control group ( $p = 0.0001$ ), suggesting higher parasympathetic tone among athletes. The DA, CA, HA, and MA groups presented no significant differences between each other: SDNN were  $77.8 \pm 5.5$ ,  $93.0 \pm 8.1$ ,  $87.8 \pm 7.2$ , and  $94.4 \pm 7.8$  ms, respectively.

### Relationship between AHR and Other Parameters

When data of all subjects were analyzed together ( $n = 150$ ), we observed a significant correlation between PC<sub>20</sub> methacholine and age ( $r = 0.2$ ,  $p = 0.01$ ) and SDNN ( $r = -0.17$ ,  $p = 0.04$ ), but the latter failed to demonstrate significant correlation in athletes only ( $r = -0.15$ ,  $p > 0.05$ ). When all athletes were analyzed together ( $n = 100$ ), there was a significant correlation between PC<sub>20</sub> and the number of years of involvement in their current sport ( $r = -0.2$ ,  $p = 0.04$ ), and if they were analyzed by individual subgroups, it was not significant except for the HA group ( $r = -0.45$ ,  $p = 0.03$ ). SDNN also presented, in all subjects ( $n = 150$ ), a significant correlation with age ( $r = -0.27$ ,  $p = 0.0008$ ). Because age may influence PC<sub>20</sub>, we ensured that the age factor did not explain the correlation between SDNN and PC<sub>20</sub> by using a partial correlation coefficient statistical method.

Finally, the degree of AHR of atopic subjects was not significantly different from those of nonatopic subjects ( $p > 0.05$ ),

but atopic athletes were significantly more prone to airway hyperresponsiveness than were nonatopic athletes ( $p = 0.04$ ).

### DISCUSSION

This study has shown that elite athletes had a significantly higher prevalence of AHR than did a group of sedentary subjects of the same age. This high prevalence of AHR was due to swimmers, and to a lesser extent to those involved in winter sports, suggesting that it is not only exercise itself that predisposes to AHR but possibly the content and the physical characteristics of inhaled air during training. Although there was a correlation between AHR and indirect measures of parasympathetic tone in all subjects, this did not explain the differences in AHR prevalence among the groups of athletes.

Although original in comparing different types of sports according to inhaled air content and looking at the influence of cholinergic tone, our observations are in keeping with many reports published in the last few years (12–14). The 76% prevalence of AHR among swimmers in our study correlates closely with the results of Zwick and colleagues (13) who reported that 11 of 14 competitive swimmers (78.6%) had increased airway response to methacholine compared with five of 14 (35.7%) control subjects (13). Helenius and colleagues (12) studied 213 track and field athletes and 124 control subjects and found that 17% of long-distance runners, 8% of speed and power athletes, and 3% of control subjects had a physician-

TABLE 2  
RESPIRATORY SYMPTOMS\*

	Control Subjects (n = 50)	Athletes				
		All (n = 100)	DA (n = 25)	CA (n = 25)	HA (n = 25)	MA (n = 25)
Cough with exercise	6 (12)	38 (38)	5 (20)	19 (76)	6 (24)	8 (32)
Rhinorrhea and pharyngeal secretions	10 (20)	40 (40)	9 (36)	12 (48)	10 (40)	9 (36)
Breathlessness, wheezing and chest tightness	14 (28)	24 (24)	3 (12)	12 (48)	5 (20)	4 (16)

For definition of abbreviations, see Table 1.

\* Number of subjects, with percentage shown in parentheses.

TABLE 3  
PULMONARY FUNCTION TESTS

	Control Subjects (n = 50)	Athletes				
		All (n = 100)	DA (n = 25)	CA (n = 25)	HA (n = 25)	MA (n = 25)
FEV <sub>1</sub> , % pred	104	108	105	113	113	108
Prevalence of AHR*						
< 16 mg/ml	14 (28)	49 (49)	8 (32)	13 (52)	19 (76)	8 (32)
< 8 mg/ml	9 (18)	35 (35)	5 (20)	5 (20)	15 (60)	8 (32)
> 128 mg/ml	16 (32)	22 (22)	10 (40)	2 (8)	3 (12)	7 (28)
Mean (median) PC <sub>20</sub> (mg/ml)	35.4 (56.9)	16.9 (16.0)	30.9 (69.1)	15.8 (12.9)	7.3 (5.5)	21.5 (54.4)

Definition of abbreviations: AHR = airway hyperresponsiveness. For other definitions, see Table 1.

\* Number of subjects, with percentage shown in parentheses.

made diagnosis of asthma; these investigators suggested that this higher prevalence in athletes might possibly be due to the prolonged hyperventilation and increased exposure to inhaled allergens and irritants during endurance training and competition. In another study comparing 162 athletes with 45 control subjects, the same group of investigators observed that the proportion of subjects with AHR to histamine as defined by a PD<sub>15</sub> FEV<sub>1</sub> ≤ 1.6 mg/ml was higher in swimmers (15 of 42, 35.7%) than in speed and power athletes (nine of 49, 18.5%), long-distance runners (six of 71, 8.5%) or control subjects (five of 45, 11.1%) (14).

The proportion of subjects with AHR in our control group was higher than the ones found in the above-mentioned studies; it suggests a higher prevalence of AHR in our population, as reported in a recent survey of airway response to methacholine in six large Canadian cities, where AHR ranged from 13 to 29% of the population (21). We should also point out that our cutoff for AHR (16 mg/ml) was slightly higher than in these previous studies (19). As control subjects were selected from the general population through advertisements without mentioning specifically that the study was on asthma or AHR, a bias from subjects interested in being tested for respiratory problems is unlikely.

An original aspect of this study is the evaluation of the types of influence different sport disciplines have on airway responsiveness according to ambient air conditions during training. The 52% prevalence of AHR in the CA group is in line with studies suggesting that exercising in a cold environment could increase the prevalence or expression of AHR among athletes (6–8). Mannix and colleagues (6) found a post-exercise fall in FEV<sub>1</sub> > 10% in 43 of 124 (35%) highly trained figure skaters, and a fall > 15% in 19 of them, indicating a prevalence significantly higher than what is usually reported from the general population. Provost-Craig and colleagues (5) documented exercise-induced bronchoconstriction after a 4-min skating program in 30 of 100 competitive skaters. Larson and colleagues (7) found that 33 of 42 elite cross-country skiers (79%) had abnormal respiratory symptoms or AHR to methacholine compared with one of 29 control subjects. Leuppi and colleagues (9), who studied AHR prevalence among Swiss elite ice hockey players and compared them to elite floor-ball players, found that nine of 26 hockey players (35%) had a positive PD<sub>20</sub> methacholine test compared with five of 24 floor-ball players (21%, p < 0.05), whereas AHR was estimated at only 7% in the Swiss population as a whole. Recently, Sue-Chu and colleagues (22) obtained an 80% prevalence of positive methacholine challenge in 44 cross-country skiers of Norway and Sweden compared with none of 12 control subjects. Although various methods have been used in the past to evaluate the prevalence of AHR, most studies have

suggested a high prevalence of AHR among athletes exercising in cold air on a regular basis.

For athletes practicing their sport in indoor ice arenas, however, it might not be cold air alone that is involved in the observed changes, but also air contaminants such as carbon monoxide (CO), nitric oxide (NO), nitrogen dioxide (NO<sub>2</sub>), and various volatile organic compounds (23, 24). These could also contribute to the development of AHR, but the possible risk to respiratory health associated with this type of exposure remains to be more clearly documented.

In our study, the athletes showing the highest prevalence of AHR were swimmers. Although swimming is believed to be beneficial for those suffering from asthma, there is now evidence showing that environmental irritants such as chlorine and its derivatives found in indoor pools (27–29) could contribute to the development and/or expression of symptomatic asthma and AHR (30, 31). Canadian swimmers train and compete primarily in indoor pools; the water is usually sterilized using chloramine or bromine-derived products (31).

On the other hand, we observed that athletes performing in mostly dry air and triathletes had a prevalence of AHR that was not significantly different from that found in the control group. This strongly suggests that exercise or hyperventilation per se may not be the main mechanism involved in the development of AHR and asthma in athletes; environmental factors may influence the type and content of the air inhaled.

As for the difference in AHR prevalence between triathletes and swimmers, the athletes from the MA group were exposed only part time to the environment of the swimmers of the HA group. The difference in prevalence of AHR could be explained by the fact that the athletes from the MA group trained significantly fewer hours in indoor pools than did those of the HA group (approximately 5 compared with 16 h/wk).

Helenius and colleagues (14) have suggested that atopy was more common among athletes, possibly explaining some of the increased AHR often found among athletes as compared with control subjects. The hypothesis that increased penetration of allergens into the athletes' airways come from repeated hyperventilation is an attractive one, but our data show that atopy does not seem to be the main factor explaining the higher prevalence of AHR in athletes. In the whole group of subjects (n = 150), there was no significant difference in AHR between atopic and nonatopic subjects (p = 0.14). However, although our atopic athletes had a higher incidence of AHR, in subgroup analysis, this difference was significant only in the DA group (p < 0.005). Of all subgroups, only the MA group had significantly different percentage in prevalence of atopy (Table 1).

We realize that there is, in comparison with previous studies, a high prevalence of atopy in our group of athletes and

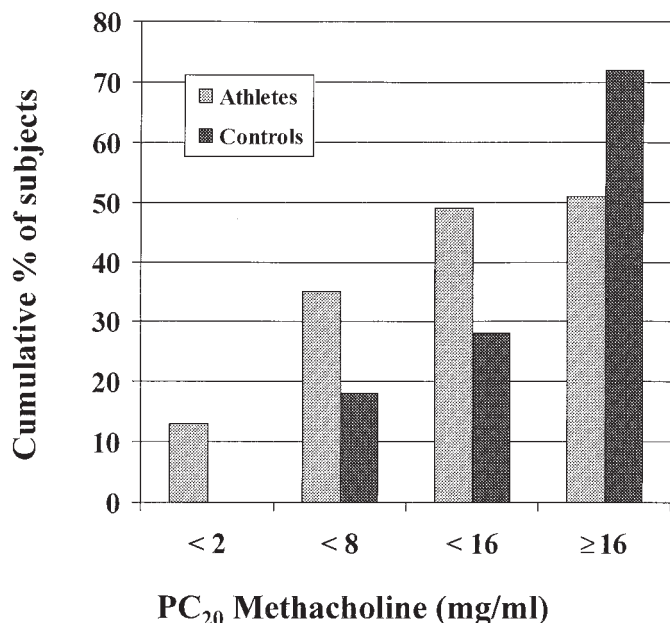


Figure 1. Cumulative percentage of subjects presenting a FEV<sub>1</sub> fall of 20% at concentrations < 2, < 8, < 16, and ≥ 16 mg/ml of methacholine. Lightly shaded columns represent athletes (n = 100) and dark shaded columns represent control subjects (n = 50).

control subjects (Tables 1 and 4). To explain this finding, we think that a series of factors have played a role. They include the population studied, which is mostly young adults, where the prevalence atopy is recognized to be the highest, the definition of atopy and method used, and regional environmental and population-related factors. We found a similar increased prevalence of atopy in our population in recent studies, in keeping with other observations suggesting an increasing prevalence of atopy in North America in the past decades. Finally, even if this observation results from many contributing factors, it does not, however, influence our results (comparison of subject's characteristics) as the method was the same in all subjects.

We looked at the possible role of autonomic nervous system on AHR in athletes. Because there are no established standard values to categorize parasympathetic tone, the recommended measure of SDNN from a 5-min segment provided a noninvasive estimation of sympathovagal balance (16). Our results demonstrate a higher SDNN in athletes, reflecting a stronger parasympathetic tone. Other recommended methods of HRV analysis have been used to assess the parasympathetic tone (16). Using respiratory sinus arrhythmia from R-R interval variation induced by controlled deep breathing, De Meersman (32) obtained a 23.1% increase in efferent parasympathetic activity in athletes after a 7.3% increase in aerobic capacity. Goldsmith and colleagues (15) used the frequency domain analysis of HRV from a 24-h ECG recording to find that trained subjects had substantially greater parasympathetic activity than did untrained ones throughout the 24-h period.

Kallenbach and colleagues (33) found a strong correlation between the magnitude of respiratory sinus arrhythmia and the degree of AHR ( $r = 0.70$ ,  $p < 0.005$ ) in 15 asthmatic subjects, but no significant correlation in 18 normal subjects, suggesting a relationship between enhanced parasympathetic tone and the degree of AHR in asthmatic patients. Although we used a different method, our results agree with those of others on the tendency to observe a higher parasympathetic

TABLE 4

## PREVALENCE OF ATOPY

Positive Skin Tests (n)	Control Subjects (n = 50)	Athletes (n = 100)
0	9	24
1	3	15
2	9	7
3 to 5	15	34
6 to 9	14	20
Total	50	100

tone in athletes and the presence of a relationship between parasympathetic tone and AHR in subjects in general. Although the exact mechanisms are still unknown, it was to be expected, as previously reported, that athletes would have a higher resting parasympathetic tone because of long-term adaptation to exercise training (34, 35). The athlete group, however, failed to show the presence of a relationship between the parasympathetic tone and AHR. This lack of correlation between level of AHR and parasympathetic tone among our athlete population could indicate that the parasympathetic system only acts as modulator of airway sensitivity and does not explain the increased prevalence of AHR in athletes.

Age and family history of asthma were similar in the athlete and the control groups and did not explain the observed differences in AHR. In athletes, however, the duration of involvement in their current sport seemed to be a contributing factor to AHR, whereas the level of competition was not, as no significant difference was observed between all parameters when athletes were divided by competitive caliber.

Finally, the possibility that swimming is preferred as a sport by already asthmatic subjects because of its reduced bronchoconstrictive effect cannot be ruled out, although unlikely as most subjects reported no respiratory problem in the past and had started training at a very young age.

This study has suggested that the increased prevalence of AHR observed in elite athletes is mostly due to the nature and content of the inhaled air and not to the exercise itself or to neurogenic mechanisms. This illustrates the importance of pursuing studies to further investigate the mechanisms involved in the development of asthma and AHR in athletes in order to suggest preventive measures.

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