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**ORIGINAL ARTICLE**

# Effect of endurance training on lung function: a one year study

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**Objective:** To identify in a follow up study airway changes occurring during the course of a sport season in healthy endurance athletes training in a Mediterranean region.

**Methods:** Respiratory pattern and function were analysed in 13 healthy endurance trained athletes, either during a maximal exercise test, or at rest and during recovery through respiratory manoeuvres (spirometry and closing volume tests). The exercise test was conducted on three different occasions: during basic endurance training and then during the precompetition and competitive periods.

**Results:** During the competitive period, a slight but non-clinically significant decrease was found in forced vital capacity ( $-3.5\%$ ,  $p = 0.0001$ ) and an increase in slope of phase III ( $+25\%$ ,  $p = 0.0029$ ), both at rest and after exercise. No concomitant reduction in expiratory flow rates was noticed. During maximal exercise there was a tachypnoeic shift over the course of the year (mean (SEM) breathing frequency and tidal volume were respectively 50 (2) cycles/min and 3.13 (0.09) litres during basic endurance training v 55 (3) cycles/min and 2.98 (0.10) litres during the competitive period;  $p < 0.05$ ).

**Conclusions:** This study does not provide significant evidence of lung function impairment in healthy Mediterranean athletes after one year of endurance training.

Over the past few decades, several studies have highlighted that winter sports activities put athletes at risk of asthma and exercise induced bronchoconstriction (EIB).<sup>1–4</sup> This is believed to result from the repetitive recruitment/dehydration of the small airways when large volumes of cold dry air are inhaled.<sup>5</sup> From a theoretical point of view, the colder and/or drier the air and the higher the ventilatory rate, the greater the risk of the small airways becoming dehydrated and damaged. In addition, the higher the ventilatory rate and the longer the period of hyperventilation, the greater the deposition of airborne allergens and other irritants particles in the lower airways. Consequently, not only winter sports athletes, but also endurance trained athletes may be at increased risk of developing asthma/EIB.

Helenius *et al*<sup>6</sup> showed a higher prevalence of physician diagnosed asthma in long distance runners than in controls and speed and power athletes. Among the American Olympians taking part in the Atlanta Summer Games (1996), cyclists most often reported the use of asthma medication, a diagnosis of asthma, or both.<sup>7</sup> Finally, among Italian athletes competing for selection for the Sydney Olympic Games (2000), a higher prevalence of reported asthma and respiratory symptoms was found in aerobic activities.<sup>8</sup>

If long term training in endurance activities seems to trigger asthma-like diseases, it is also established that more transient airway changes can occur during the course of a sport season. In Finnish runners with probable EIB (reduction of 6.5% or more in forced expiratory volume in the first second (FEV<sub>1</sub>)), the airway response to exercise was shown to vary during winter and the pollen season.<sup>9</sup> In recreational joggers from New York, biomarkers of airway inflammation were detected during the summer months only, suggesting a possible continuing inflammatory response in the lungs of athletes exposed to ozone and associated pollutants.<sup>10</sup>

During a normal sports season in Mediterranean regions, endurance trained athletes are never exposed to very cold and dry weather (temperatures rarely drop below zero). However, a large release of airborne allergens often occurs at the beginning of spring. Moreover, because of the photochemical nature of ozone forming reactions, considerable accumulation of ozone

may occur during hot summer days. Finally, the training workload increases from low levels during the basal training period (fall to winter) to high loads during the precompetitive (end of winter to spring) and competitive (summer) periods. Consequently, phases of hyperventilation are more common and longer when the quality of the ambient air deteriorates—that is, when allergens and ozone concentrations peak. Thus one may expect impairment of lung function in these athletes as the season goes on.

The aim of this follow up study was to determine if any functional airway changes occur during the course of a sports season in healthy endurance athletes training in a Mediterranean region. Lung function before and after exercise and ventilatory response to exercise were checked three times in the year. As the level of training and quality of inhaled air can be confounding factors in the development of respiratory disorders in athletes,<sup>5</sup> time and intensity of training, and meteorological, air allergen, and air pollution data were collected at each time point.

**MATERIALS AND METHODS****Subjects****Athletic group**

Thirteen young male endurance trained athletes participated in this study. Six were cyclists and seven were triathletes. They had been training regularly for 7 (1.5) years. The mean (SEM) training volume was 11 (1) hours a week. All were non-smokers and clinically healthy. During the first visit to the laboratory, a medical standardised interview was conducted. None of the athletes reported current asthma, exercise induced asthma, or habitual use of  $\beta_2$  agonists. They were also free from respiratory infection and allergic reactions and none used anti-allergenic agents.

**Control group**

To check on the seasonal variation of pulmonary function in healthy sedentary subjects with no significant episode of

**Abbreviations:** FEF<sub>25–75</sub>, mid maximal expiratory flow rate; FEV<sub>1</sub>, forced expiratory volume in the first second; FVC, forced vital capacity

**Table 1** Metabolic data and power output at maximal exercise during the different evaluation periods in both groups

	Athletes			Controls	
	BET	PrC	C	BET	C
̄V̄O <sub>2</sub> (ml/min/kg)	62.6 (1.3)	64.0 (1.3)	66.3 (1.4)*	45.5 (1.3)†	47.4 (1.3)†
Power output (W)	376 (7)	378 (9)	395 (8)*	264 (17)†	276 (15)†
HR (beats/min)	189 (2)	186 (3)	188 (2)	185 (5)	187 (4)
̄V̄E (l/min)	158.3 (3.8)	159.3 (6.0)	162.0 (6.4)	115.2 (7.8)†	118.9 (5.0)†
f <sub>B</sub> (cycles/min)	50 (2)	51 (3)	55 (3)*	43 (8)	44 (3)†
V <sub>T</sub> (litres)	3.13 (0.09)	3.20 (0.08)	2.98 (0.10)*	2.73 (0.17)†	2.74 (0.31)

Within-group effects: \*significantly different from BET and PrC ( $p<0.05$ ); between-groups effects: †significantly different from the athletic group ( $p<0.05$ ).

BET, Basic endurance training; PrC, precompetition; C, competition; ̄V̄O<sub>2</sub>, oxygen uptake; HR, heart rate; ̄V̄E, minute ventilation; f<sub>B</sub>, breathing frequency; V<sub>T</sub>, tidal volume.

hyperventilation, six control subjects were also tested. All were healthy non-smokers and had a negative medical history. All were free from respiratory infection and nasal allergies during the year of the study. They were not involved in any regular physical activity, exercising fewer than two hours a week in recreational sports.

All subjects gave written consent to participate in the study after the protocol had been fully explained to them. This study was approved by the local ethics committee.

### Study design

Testing in the athletes was carried out to correspond with the major changes in training periods. End of autumn testing (November–December) corresponded to basic endurance training (BET). End of winter testing (February–March) was performed half way through the precompetition period (PrC). Testing during the competition period (C) was in early summer (June–July). Control subjects were investigated twice: at the end of the year (corresponding to BET for the athletic group) and during summer (corresponding to C). At each visit, all the subjects performed a maximal exercise test preceded and followed by respiratory manoeuvres, athletes completed a questionnaire on training habits and respiratory symptoms, and ambient ozone concentrations, airborne allergens, and meteorological data were obtained.

### Questionnaire

Using the formula of Morton *et al*,<sup>11</sup> we evaluated the changes in training load and training volume in athletes over the course of the season. The calculated index—termed the training impulse—was specified quantitatively from two variables: training session duration and the concomitant heart rate. The formula was as follows:

Training impulse (arbitrary units, au) = (duration of training (minutes) × (ΔHR exercise ratio (no units) × (0.64e<sup>1.92 ΔHR exercise ratio</sup>))

where HR = heart rate, and ΔHR exercise ratio = (HR during exercise – HR at rest)/(maximal HR – HR at rest).

During the week preceding each test, athletes were asked to record their heart rate continuously while training using a Polar Sport Tester (Polar Electro, Kempele, Finland). They then noted down these values and the time that they spent training on the questionnaire.

Athletes also completed a questionnaire that asked about the frequency of four common symptoms of exercise induced asthma: cough, wheezing, chest tightness, and breathlessness. For each item, they were asked to evaluate symptom frequency (never (value of 0), sometimes (value of 1), often (value of 2), and always (value of 3)) during and after training and race participation in the month before the laboratory tests. For each of the periods studied, respiratory symptoms were first analysed individually. The addition of all responses then provided a global index of respiratory symptoms for each subject.

### Exercise challenge test

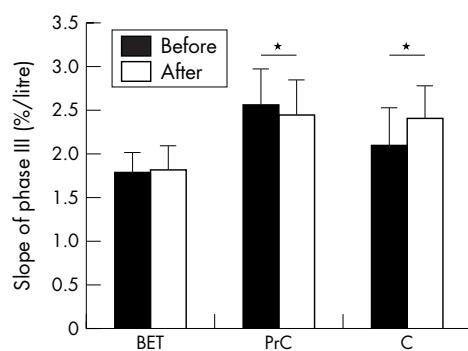
Incremental exercise testing was performed on an electronically braked cycle ergometer (Ergoline, Bitz, Germany). In the athletic group, the starting workload was 60 W and was increased by 30 W/min until exhaustion. In the control group, the starting workload was 30 W plus 30 W/min until exhaustion. All the ventilatory variables were measured continuously using a breath by breath automated exercise metabolic system (Vmax29; SensorMedics, Yorba Linda, California, USA). Heart rate was continuously monitored using a 12 lead electrocardiograph (Marquette Hellige Medical Systems, Milwaukee, Wisconsin, USA). Vo<sub>2</sub>MAX was assumed when at least three of the four following criteria were met: (a) an increase in ̄V̄O<sub>2</sub> of <100 ml/min with the last increase in workload; (b) attainment of age predicted maximal heart rate (210 – (0.65 × age) ± 5%); (c) a respiratory exchange ratio >1.10; (d) an inability to maintain

**Table 2** Pulmonary flow values in the athletic group before and after exercise (at 10 and 35 minutes of recovery) during each of the three periods

	BET			PrC			C		
	Rest	10 min	35 min	Rest	10 min	35 min	Rest	10 min	35 min
FVC (litres)	6.12 (0.17)	6.01 (0.16)	6.16 (0.15)	6.12 (0.15)	6.20 (2.99)	6.24 (0.15)	5.89 (0.14)*	5.90 (0.15)*	5.89 (0.16)*
% FVC theoretical	117 (3)	115 (3)	118 (3)	116 (3)	118 (3)	116 (3)	112 (2)*	112 (3)*	112 (3)*
FEV <sub>1</sub> (litres)	4.95 (0.13)	4.97 (0.13)	5.00 (0.10)	5.05 (0.12)	5.08 (0.14)	5.09 (0.14)	4.90 (0.11)	4.99 (0.14)	4.95 (0.13)
% FEV <sub>1</sub> theoretical	112 (2)	113 (3)	114 (3)	114 (3)	115 (3)	115 (3)	111 (2)	112 (3)	112 (3)
FEV <sub>1</sub> /FVC (in %)	81 (1)	83 (2)	81 (2)	83 (1)	82 (2)	81 (2)	83 (1)*	85 (2)*	84 (1)*
FEF <sub>25–75</sub> (l/s)	4.75 (0.19)	4.92 (0.24)	4.89 (0.24)	5.11 (0.24)	5.13 (0.30)	5.10 (0.28)	5.01 (0.24)	5.18 (0.26)	5.12 (0.24)
% FEF <sub>25–75</sub> theoretical	94 (4)	97 (5)	97 (5)	101 (5)	102 (6)	101 (6)	99 (5)	103 (5)	101 (5)

\*Significantly different from BET and PrC ( $p<0.01$ ).

BET, Basic endurance training; PrC, precompetition; C, competition; FEV<sub>1</sub>, forced expiratory volume in one second; FVC, forced vital capacity; FEF<sub>25–75</sub>, mid maximal expiratory flow rate.



**Figure 1** Slope of phase III values before and after exercise in the athletic group at each period of the year (BET, basic endurance training; PrC, precompetition; C, competition). \*Significantly different from BET ( $p<0.01$ ).

the minimal required pedalling frequency (60 rpm) despite maximum effort and verbal encouragement.

### Pulmonary function

Standard spirometry was carried out before exercise and after 10 and 35 minutes of recovery in all subjects according to the recommendations of the American Thoracic Society<sup>12</sup> with a flow volume spirometer (Vmax29; SensorMedics). Pulmonary measures included forced vital capacity (FVC), FEV<sub>1</sub>, FEV<sub>1</sub>/FVC, and mid maximal expiratory flow rate (FEF<sub>25–75</sub>). Values are expressed as percentages of reference (predicted) values for European adults.<sup>13</sup>

Closing volume tests—that is, single breath O<sub>2</sub> test<sup>14</sup>—were performed in both groups at rest and after 20 minutes of recovery (Vmax29; SensorMedics). The pneumotachograph was filled with 100% O<sub>2</sub> and equipped with a N<sub>2</sub> meter. During these tests, the subjects performed a slow, full vital capacity manoeuvre at inspiratory and expiratory flow rates of about 0.5 l/s. The slope of the N<sub>2</sub> alveolar plateau (slope of phase III) and the closing volume were calculated. Slow vital capacity manoeuvres were repeated at least twice—that is, until the slow vital capacity volumes were within 10% of each other—and the mean was calculated.

### Air characteristics

Pollution data, including ozone (O<sub>3</sub>), nitrogen dioxide (NO<sub>2</sub>), sulphur dioxide (SO<sub>2</sub>), and particulate matter (PM<sub>10</sub>), were taken from Air Languedoc-Roussillon association (<http://www.air-lr.asso.fr>). The concentration of air allergens was obtained from the National Network of Aero-biologic Surveillance (<http://www.rnsa.asso.fr>). Concentrations of allergens were expressed as number per m<sup>3</sup> of air per 24 hours. Daily meteorological data (temperature and relative humidity) were obtained from a station operated by the

Departmental Meteorological Centre of Hérault at Montpellier-Méditerranée airport.

### Data analysis

In the athletic group all the variables were first compared between cyclists ( $n = 6$ ) and triathletes ( $n = 7$ ). As no significant differences were noted, the values were combined and analysed as a whole. Metabolic values and questionnaire responses were compared using one way analysis of variance with repeated measurements or Friedman repeated measures analysis of variance on ranks (for non-Gaussian values). The other data—that is, spirometry and closing volume values—were examined using 2 (rest/post-exercise) or 3 (rest/10 min recovery/35 min recovery)  $\times$  3 (BET/PrC/C) analysis of variance with repeated measures across time. When significant  $F$  values were obtained, multiple comparisons were performed using Student-Newman-Keuls' test to determine where the differences had occurred. Because of the small sample size, exercise test values in the control group were compared throughout the year using Wilcoxon tests. At the beginning (during BET) and at the end of the sports season (during C), physiological and anthropometric variables were compared between athletes and controls using unpaired  $t$  tests (for Gaussian values) and Mann-Whitney tests (for non-Gaussian values). Pulmonary data between groups at different periods of the year were compared by 2 (athletic/control group)  $\times$  2 (rest/post-exercise) or 2  $\times$  3 (rest/10 min recovery/35 min recovery) analysis of variance with repeated measures across time. Meteorological, air allergen, and air pollution data were compared using one way analysis of variance or Kruskal-Wallis analysis of variance on ranks (depending on data distribution). For all comparisons, the level of significance was set as  $p<0.05$  and tests were two sided. Results are expressed as mean (SEM).

## RESULTS

As no significant differences were noted between cyclists and triathletes, a single group was formed (the athletic group). The mean values for this group and the control group are presented below.

### General data

#### Athletic group

At the beginning of the study, mean (SEM) age, height, and weight were 22 (1) years, 179 (1.5) cm, and 70 (1) kg respectively in the athletic group. Training volume increased slightly but not significantly ( $p = 0.0552$ ) between the beginning and the end of the sports season (total training time 9 h 24 min (50 min), 11 h 25 (1 h 02), and 12 h 29 min (1 h 19) a week during BET, PrC, and C respectively). Training load increased significantly between BET and the two following periods (training impulse = 805 (94), 1324 (125), and 1339 (192) au during BET, PrC, and C respectively).

**Table 3** Closing volume and slope of phase III values in athletes and control subjects before and after exercise at the time of the evaluation periods

	Athletes						Controls					
	BET		PrC		C		BET		PrC		C	
	Rest	Recovery	Rest	Recovery	Rest	Recovery	Rest	Recovery	Rest	Recovery	Rest	Recovery
Slope of phase III (%/litre)	1.78 (0.23)	1.82 (0.28)	2.55 (0.42)*	2.45 (0.39)*	2.10 (0.42)*	2.41 (0.36)*	1.38 (0.24)	1.50 (0.20)	1.04 (0.13)†	1.14 (0.15)‡		
Closing volume (%)	8.0 (0.7)	9.4 (0.4)†	7.1 (0.4)	8.5 (0.6)†	8.3 (0.4)	9.0 (0.4)†	11.6 (0.6)	11.6 (1.4)	11.3 (0.7)	10.3 (0.3)		

BET, basic endurance training; PrC, pre-competition; C, competition. Within-group effects: \*significantly different from BET ( $p<0.01$ ); †significantly different from rest ( $p<0.05$ ); between-groups effects: ‡significantly different from the athletic group ( $p<0.01$ ).

**Table 4** Spirometric values in the control group before and after exercise (at 10 and 35 minutes of recovery) at two different times of the year, corresponding to basic endurance training (BET) and competition (C) in athletes

	BET			C		
	Rest	10 min	35 min	Rest	10 min	35 min
FVC (litres)	5.21 (0.17)	5.22 (0.23)	5.36 (0.21)	5.35 (0.15)	5.39 (0.19)	5.35 (0.20)
% FVC theoretical	100 (4)	101 (5)	103 (5)	103 (5)	104 (4)	103 (5)
FEV <sub>1</sub> (litres)	4.33 (0.28)	4.32 (0.25)	4.36 (0.25)	4.33 (0.31)	4.49 (0.30)	4.35 (0.28)
% FEV <sub>1</sub> theoretical	98 (4)	98 (4)	99 (4)	98 (5)	102 (5)	99 (4)
FEV <sub>1</sub> /FVC (%)	83 (4)	83 (4)	82 (4)	81 (5)	83 (4)	82 (5)
FEF <sub>25-75</sub> (l/s)	4.49 (0.66)	4.65 (0.78)	4.54 (0.68)	4.48 (0.82)	4.82 (0.68)	4.52 (0.75)
% FEF <sub>25-75</sub> theoretical	89 (12)	92 (15)	90 (13)	88 (16)	95 (13)	89 (14)

FVC, Forced vital capacity; FEV<sub>1</sub>, forced expiratory volume in one second; FEF<sub>25-75</sub>, mid maximal expiratory flow rate.

### Control group

In this group, mean (SEM) age, height, and weight were 25 (1) years, 177 (3) cm, and 72 (4) kg respectively. Except for age (control subjects were 3 years older than athletes), no significant differences from the athletic group were noted.

### Aerobic capacity

#### Athletic group

Table 1 summarises the maximal exercise data for the athletes.  $\dot{V}O_{2\text{MAX}}$  increased significantly as a function of training (+5.9%). Although maximal ventilation remained close to 160 litres/min throughout the study, the breathing pattern was significantly modified during the competitive phase. Breathing frequency increased from 50 to 55 cycles/min, and tidal volume decreased from 3.13 to 2.98 litres during C testing.

#### Control group

Table 1 presents maximal exercise data for the control group as well. No modification was noted through the year. When compared with the athletic group, a significant difference was noted for  $\dot{V}O_{2\text{MAX}}/\text{kg}$ , minute ventilation, and power output. Tidal volume was significantly higher in the athletic group than the control group only during BET, whereas the opposite happened for breathing frequency—that is, breathing frequency was higher in the athletic group only at the end of the sports season.

### Lung function

#### Athletic group

FVC decreased slightly but significantly during the competitive period, but we found no simultaneous alteration in FEV<sub>1</sub> or FEF<sub>25-75</sub> (table 2). Because FVC decreased with no change in FEV<sub>1</sub>, the Tiffeneau ratio (FEV<sub>1</sub>/FVC) had increased significantly at the end of the sports season.

The slope of phase III increased significantly between BET and the two last periods (fig 1, table 3). Whatever the season

of the year, a significant increase in closing volume values was noticed after exercise (table 3).

Over the course of the study, respiratory symptoms evaluated by the questionnaire occurred only occasionally in most of the athletes. None of the athletes reported permanent symptoms, and only four of them (about 30%) reported at least one frequent symptom. The most often reported symptom was a cough after the race. Self reported respiratory symptoms remained stable through the year: after addition of all items, the mean symptom score was 1.5 (0.3), 1.5 (0.5), and 1.6 (0.4) during training sessions at BET, PrC, and C respectively (maximum value = 12).

#### Control group

Spirometric data before and after exercise remained stable through the year in the control group (table 4). FVC was significantly higher during BET in the athletic group than the untrained group. However, this was no longer evident during C.

No modification in closing volume values was noted during the year in this untrained population (table 3). As the slope of phase III remained unchanged in the control group throughout the year whereas it increased in the athletic group, the difference between the two groups reached a significant level during C (table 3).

### Air data

Table 5 reports all air data. Temperature rose progressively over the course of the year, whereas relative humidity significantly decreased between BET and the two following periods. On the one hand, O<sub>3</sub> values increased progressively, and on the other hand NO<sub>2</sub> and SO<sub>2</sub> values decreased. A peak of airborne allergens was noted during PrC.

### DISCUSSION

This study shows a slight decrease in FVC at rest and after exercise at the end of a sports season conducted in a Mediterranean region, an increase in the slope of phase III (index of exaggerated alveolar ventilation mismatch) half way through the year, but no concomitant reduction in flow rates.

To our knowledge, this is the first longitudinal study of lung function changes in endurance athletes training in a Mediterranean region. Previous studies in other athletic populations found different results. Helenius *et al*<sup>9</sup> noted a change in airway response to exercise in elite Finnish runners between winter and the pollen season. Several factors could explain the discrepancy between our results and those obtained by Helenius *et al*. (a) Mediterranean athletes never train in subfreezing conditions—we found a mean temperature of 7 (0.5) °C during the coldest months (November and December). Therefore the risk of dehydration of their small airways during exercise is not as high as in Finnish runners. (b) All our athletes competed at regional level, whereas 79% of the runners in the study of Helenius *et al* were part of the Finnish national team. The results of

**Table 5** Air data at different times of the year

	BET	PrC	C
Temperature (°C)	7 (0.5)	11 (0.5)†	22 (0.5)*
Relative humidity (%)	69 (3)	60 (3)†	62 (2)†
O <sub>3</sub> (µg/m <sup>3</sup> )	26 (3)	43 (3)†	64 (2)*
NO <sub>2</sub> (µg/m <sup>3</sup> )	42 (4)	38 (3)	18 (1)*
SO <sub>2</sub> (µg/m <sup>3</sup> )	10 (1)	5 (0.6)†	4 (0.1)†
PM <sub>10</sub> (µg/m <sup>3</sup> )	27 (4)	26 (2)	21 (1)
Airborne allergens (grains/m <sup>3</sup> /h)	366 (140)	588 (122)†	283 (39)

\*Significantly different from BET and PrC ( $p<0.05$ ).

†Significantly different from BET ( $p<0.05$ ).

BET, basic endurance training; PrC, precompetition; C, competition; O<sub>3</sub> and NO<sub>2</sub> data are daily one hour maxima; SO<sub>2</sub> and PM<sub>10</sub> are daily means.

**What is already known on this topic**

- The prevalence of asthma/EIB is high in endurance trained athletes
- Asthma may develop during an active sport career
- Transient airways changes can occur during the course of a sports season in runners exposed to extreme environmental conditions—that is, freezing air, allergen surges, peaks of pollution

**What this study adds**

This study shows that over one year of endurance training in a mild and relatively unpolluted environment, healthy athletes are unlikely to develop respiratory dysfunctions as a result of their physical activity.

Nystad *et al*<sup>15</sup> suggest that only extreme training (training volume >20 hours a week) and not moderate training (10–20 hours a week) favours asthma development. (c) The test we performed was not specifically designed to detect airway hyper-responsiveness to exercise (in that case, we should have used a short, one stage protocol, such as that used by Helenius *et al*<sup>9</sup>). Unlike Helenius *et al*, we excluded athletes with a previous medical diagnosis of asthma and/or EIB and those with frequent respiratory symptoms. Our aim was to determine if a year of continuous endurance training triggered respiratory disorders in healthy athletes. Another follow up study focused on this type of population. Considering the increase in ozone during summer time in New York, Kinney *et al*<sup>10</sup> looked at biomarkers of airway inflammation in bronchoalveolar lavage from recreational runners during both summer and winter. They showed signs of airway inflammation only during the summer months, suggesting a possible continuing inflammatory response in the lungs of athletes exposed to ozone and associated pollutants. We did not collect airway inflammatory markers in our study. However, the tachypnoeic shift we noted during exercise at the end of the year may be relevant. Firstly, it is well established that rapid shallow breathing is a common response to O<sub>3</sub> exposure.<sup>16</sup> Secondly, this type of ventilatory pattern is unusual in well trained athletes. Lastly, our ambient O<sub>3</sub> values peaked during the competitive period.

One limit of this interpretation is that the concentration of O<sub>3</sub> never reached the upper exposure limit for European countries—that is, 180 µg/m<sup>3</sup>. This means that other factors such as respiratory muscle fatigue cannot be ruled out. Perret *et al*<sup>17</sup> showed that exhaustive constant load cycling exercise may lead to fatigue of the diaphragm. As the tachypnoeic shift appeared in our study after several months of intensive training, the increase in breathing frequency may also be due to impaired inspiratory muscle contractility. It may be wise to explore further the ventilatory adaptations that occur during the course of a sports season in Mediterranean endurance trained athletes to determine which one of these two factors (ozone exposure or respiratory muscle fatigue) is mainly responsible for the functional changes.

A criticism of this study is the lack of homogeneity in our athletic population. Both triathletes and cyclists were included. Because triathletes are exposed not only to outdoor pollutants and allergens, but also to chlorine compounds in swimming pools, one might argue that the chemical stress on the airways was not the same in the two subpopulations. It has been shown that exposure to chlorine gas and its derivatives can produce inflammation and oedema of the mucous membranes.<sup>18</sup> However, as the airway changes through the year were not significantly different between the triathletes and cyclists, and as most of the triathletes spend less than three hours a week in indoor chlorinated swimming pools, it seems unlikely that this factor affected our results much.

In conclusion, this study does not provide significant evidence of lung function impairment in healthy, Mediterranean, endurance trained athletes after a year of

training. Only minor and non-clinically significant changes were noticed through the season—that is, a slight decrease in FVC and increase in the slope of phase III, both before and after exercise. During maximal exercise, a modification in the breathing pattern was observed during the competitive period—a shift to a rapid shallow breathing—suggesting that the ventilatory adaptation was no longer the same at the end of the sports season. It needs to be determined if this change is mainly linked to increased exposure to ambient ozone or respiratory muscle fatigue.

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