ORIGINAL ARTICLE

High incidence of exercise-induced bronchoconstriction in triathletes of the Swiss national team

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Aim: To assess the progression of bronchial reactivity (BR) and incidence of bronchial hyperreactivity (BH), exercise-induced bronchoconstriction (EIB) and asthma in triathletes over 2 years.

Methods: Subjects were seven athletes from the Swiss national triathlon team (mean (SD) age 24.3 (4.8) years), who initially were not asthmatic, not treated with antiasthmatic medication, and who had performed at international level for more than 3 consecutive years (2001–2003). To assess BR, BH and EIB, subjects ran on a 400 m track for 8 min at intensities equal to the anaerobic threshold. Tests were conducted in ambient temperatures of 4.4 (2.8) °C, –8.8 (2.4) °C and 3.6 (1.5) °C, and humidity of 52 (16)%, 83 (13)% and 93 (2)%. Forced expiratory volume in 1 s (FEV₁) was measured before and at 2, 5, 10 and 15 min after EIB, and 5 min after inhalation of a β_2 agonist. Two methods were used to calculate the incidence: (1) the standard assessment; (2) extrapolation of the decrease in FEV₁ to the BH limit.

Results: BR increased significantly in the seven athletes (FEV₁: year, $p=0.04$; year $\times$ EIB, $p=0.002$; EIB $p<0.001$). Within 2 years, BR had increased significantly and even reached BH in some athletes. Three athletes exhibited BH. After extrapolation of the decrease in FEV₁ in all seven athletes, the limit of 10% by definition for BH was determined to occur within 1.77–4.81 years, resulting in 21–57% of athletes with newly developed BH per year.

Conclusion: Athletes develop EIB quickly, a rate of increase 195–286 times that of the normal rate for development of asthma.

The bronchoconstrictive response to a known dose of provoking agent (eg, exercise at the anaerobic threshold) can be quantified by bronchial reactivity (BR), which reflects the airway responsiveness measured by several lung function tests before and after provocation. In asthmatic people, BR is increased above a defined limit (eg, decrease in forced expiratory volume in 1 s (FEV₁) of more than 10%), which we define as bronchial hyperreactivity (BH). To measure specific BR in athletes, exercise-induced bronchoconstriction (EIB) tests are used.

There is a higher risk of EIB and asthma in high-performance athletes than the general population.^{1–5} The reason for the occurrence of athlete's asthma is not completely understood. Several investigators have suggested that it may be sport specific.^{4–6} Cold environment induces BH, and an improvement in BR is observed in warmer environments.^{7–9} In winter athletes, cold environment is often the cause of increased incidence of BR.^{3 10 11} Chronic exposure to cold air appears to be a key factor.^{3 10–12} However, this hypothesis has not been completely established. For instance, exposure to cold air does not explain observations and documentation of an increase in BR in endurance athletes.^{4–6} Additional factors that may influence BR are mechanical impairment of forced breathing by the athletes,^{13 14} dehydration as a result of increased ventilation,¹⁵ inhalation of harmful chemical irritants,¹⁶ compromised immune function,¹⁷ and possibly an increase in vagal activity.^{18–20} Allergens may also contribute to BR in athletes, especially in those who are predisposed to allergic asthma. Whether athletes are more predisposed to allergens than the general population is unclear.^{1 21} However, there is an association between atopic and exercise-induced asthma²² and asthma in athletes.^{1 21 23} Also, there is a higher incidence of atopic asthma in people with BR during the pollen season.^{24–28} In

contrast with the atopic hypothesis, bronchial lavage of elite athletes with asthma suggests that the response may not be caused by an inflammatory allergic reaction. Instead, it appears to be due to an inflammatory granulocyte reaction dominated by neutrophil-driven asthma.²⁷ We know little about the genesis of athlete's asthma or its significance.

Asthma also appears to persist in less provocative environments. Larsson *et al*² reported that endurance athletes exhibit similar BR during the competitive season in winter and during the recovery season in summer. Moreover, there is evidence of airway inflammation and remodelling in skiers.²⁷ In contrast with these findings, Helenius *et al*²⁹ suggested that athlete's asthma is partly reversible and that it may develop during, and subside after, an active sports career. If athlete's asthma continues after a competitive career, sport at a high performance level may trigger persisting asthma. Thorough epidemiological investigations and therapeutic interventions are long overdue. The high prevalence of athletes with asthma, the potentially lasting occurrence of asthma among athletes, and the unusual form of asthma in athletes challenge the epidemiological evidence. We know very little about the rate of development of athlete's asthma. Verges *et al*³⁰ showed, in a 10-year follow-up study of pulmonary function in three symptomatic elite cross-country skiers, progressive development of airway obstruction at rest throughout their sports careers. To our knowledge, no other studies in high-performance athletes have examined the chronological progression of BR or the incidence of asthma. This study was designed and conducted to assess the longitudinal progression of BR and the incidence of EIB in high-performance triathletes.

Abbreviations: BH, bronchial hyperreactivity; BR, bronchial reactivity; EIB, exercise-induced bronchoconstriction; FEV₁, forced expiratory volume in 1 s.

Table 1 Characteristics of subjects

	2001	2002	2003
Athletes tested			
Total no	36	36	34
Age (years)	25.8 (5.2)	23.8 (5.8)	26.2 (5.6)
Asthmatics (%)	50	64	53
Antiasthmatic therapy (%)	25	33	38
Antiasthmatic therapy (% asthmatics)	50	52	72
Athletes in study			
No in study*	7	7	7
Age (years)	24.3 (4.8)	25.3 (4.8)	26.3 (4.8)

Unless otherwise indicated, values are mean (SD).

*Selected according to the inclusion criteria.

METHODS

Study design

Over 2 years, the chronological progression of BR (three tests) was measured and analysed in triathletes of the Swiss national team. The incidence of EIB was assessed, which is reflected by the rate of occurrence of newly developed BH per year. In addition, the incidence of EIB was extrapolated from the progression of BR over the assessed time period to the limits of BH using several different models.

Protocol

Athletes from the Swiss national triathlon team were interviewed, examined and tested (over 2 years) in the same week (in January) and at the same time of day of the three consecutive years 2001, 2002 and 2003. To be included in the study, athletes were required: (1) not to be asthmatic initially (requirement for assessing incidence); (2) not to receive antiasthmatic treatment during the whole 2 years (requirement for testing EIB); (3) to perform at international level (member of the national team) over the 3 consecutive years (requirement to target top athletes).

Of the 36 athletes initially assessed, only seven met the criteria for the study. These were included in the study and participated in all three tests. Table 1 summarises their anthropometric data and health status.

Athletes were diagnosed as asthmatic if they had a history of obstructive episodes and more than 10% variability in their FEV₁ either during rest or after provocation. Testing, which included physical examination, laboratory tests and other physical diagnostic examinations, was performed during a compulsory team meeting, designated as medical days.

Participation in these annual medical days was required of all athletes of the Swiss national triathlon team. The study was approved by the local ethics committee, and all athletes agreed and gave voluntary informed written consent to participate in the study including publication of the data. Although our sample size was small, it included all athletes of the Swiss national triathlon team, which was internationally successful and included top internationally competitive athletes.

EIB tests

To assess BR and BH, EIB running tests were conducted on a 400 m track-and-field facility for 8 min. Athletes were not allowed to warm up. They were asked to perform at a constant intensity representative of a competitive triathlon that was comparable to the intensity at the anaerobic threshold. Exercise intensity was assessed by heart rate telemetry (Polar Electro OY, S610i, Kempele Finland; accounting for RR intervals) over the entire EIB test in 5 s intervals and kept constant over all three tests. There was no significant difference in mean exercise heart rate for the three exercise tests (table 2).

Ambient conditions at the time of testing each year were significantly different resulting in the mildest conditions for EIB in 2003 (table 2). To measure lung function, a spirometer (Microlab 3000) was used that operates with a bidirectional flow transducer. FEV₁ was assessed before and at 2, 5, 10, and 15 min after exercise. In addition, the effect of a bronchodilator was measured 5 min after inhalation of a β_2 agonist (1 mg terbutaline via aerosol and spacer). BR was assessed and analysed using different methods: (1) the time course of FEV₁ values after exercise defined BR (the lower the values, the higher BR), and these values were used for statistical comparisons; (2) the lowest FEV₁ value after exercise compared with the resting value before exercise and after inhalation defined the degree of BR; (3) the area under the curve was calculated, which reflects the area between the initial FEV₁ value and the FEV₁ values after exercise over 15 min (the larger the area under the curve, the higher the BR). BH was defined as an exercise-induced decrease in FEV₁ of more than 10%.

Incidence of EIB and comparison with general population

To account for the incidence of EIB, two different methods were used (table 3).

Method 1 assessed incidence by dividing the percentage of athletes with newly developed BH (three athletes) by the time period of 2 years. Method 2 estimated the incidence by extrapolating the decrease in FEV₁ to the BH limits using four

Table 2 Exercise testing in the seven athletes who met the criteria for the study

	2001	2002	2003	p Value
EIB tests				
Mean heart rate during EIB (beats/min)	168 (7)	170 (8)	170 (6)	0.89
Temperature (°C)	4.4 (2.8)	-8.8 (2.4)	3.6 (1.5)	<0.001
Humidity (%)	51.8 (15.7)	82.9 (12.9)	92.7 (2.3)	<0.001
Water vapour pressure (mm Hg)	3.41 (1.64)	1.99 (0.54)	5.47 (0.47)	<0.001
BR				
Maximal decrease in FEV ₁				
Reference initial FEV ₁ (%)	2.36 (2.39)	4.34 (3.09)	6.52 (4.02)	0.012
Reference post-inhalation FEV ₁ (%)	4.98 (3.13)	9.12 (2.47)	10.06 (3.33)	<0.001
Area under the curve (% FEV ₁ , 15 min)				
Reference initial FEV ₁ (% $\times$ min)	0.0 (19.4)	35.1 (32.2)	56.7 (50.9)	0.015
Reference post-inhalation FEV ₁ (% $\times$ min)	26.0 (58.6)	102.0 (29.2)	99.5 (32.5)	0.005
BH				
No of athletes	0	2	3	
% of total in study	0	29	43	

BH, bronchial hyperreactivity; BR, bronchial reactivity; EIB, exercise-induced bronchoconstriction; FEV₁, forced expiratory volume in 1 s.

Unless otherwise indicated, values are mean (SD).

Table 3 Incidence of exercise-induced bronchoconstriction

	Method				
	1	2	2	2	2
Assessment or extrapolation	Assess	Extrapol	Extrapol	Extrapol	Extrapol
Extrapolation model number	–	1	2	3	4
No of athletes (total 7)	3	7	7	7	7
BH	Yes	No	No	No	No
BR	Increase	Increase	Increase	Increase	Increase
Origin at zero for decrease in FEV ₁	–	Yes	No	Yes	No
EIB reference initial FEV ₁	–	Yes	Yes	No	No
EIB reference post-inhalation FEV ₁	–	No	No	Yes	Yes
Linear regression					
Y (decrease % FEV ₁)	–	–2.327	–2.327	–5.512	–5.512
X (years)	–	–1.118	–1.118	–2.17	–2.17
Slope	–	–2.081	–2.081	–2.54	–2.54
p Value	–	0.018	0.018	0.222	0.222
Goodness of fit (r ²)	–	0.999	0.999	0.884	0.884
Asthma					
Incidence of EIB (%/year)	22	21	27	25	57
Time to develop EIB (years)	4.65	4.81	3.69	3.94	1.77

Assess, assessment; BH, bronchial hyperreactivity; BR, bronchial reactivity; EIB, exercise-induced bronchoconstriction; Extrapol, extrapolation; FEV₁, forced expiratory volume in 1 s.

Method 1 assessed incidence by dividing the percentage of athletes with newly developed BH (three athletes) by the time period of 2 years. Method 2 estimated the incidence by extrapolating the decrease in FEV₁ to the BH limits using four different models.

different models. We used a linear regression line through the lowest FEV₁ values in each year to extrapolate BH from the group of all seven athletes (see fig 3). In model 1, the origin of the decrease in FEV₁ served as zero, and the initial FEV₁ values were used as reference for EIB to account for the time when EIB occurred in the whole group. In model 2, the origin of the decrease in FEV₁ from the first test was used, and the initial FEV₁ values were used as a reference for EIB. In model 3, the origin of the decrease in FEV₁ was used as zero, and post-inhalation FEV₁ values were used as reference for EIB. In model 4, the origin of the decrease in FEV₁ was used from the first test, and post-inhalation FEV₁ values were used as reference for EIB.

Statistical analysis

The study was analysed using a two-way or one-way analysis of variance with repeated measures. A Tukey post hoc test was used when indicated. To account for the incidence in model 1 to

model 4, linear regression was used (table 3 and fig 3). $p < 0.05$ was considered significant.

RESULTS

Method 1

In three of the seven athletes (table 2), BR increased significantly, which was reflected in the decrease in FEV₁ found with the EIB test (fig 1). All of these athletes complained of typical asthma symptoms and developed BH over the 2-year period. On the basis of these results, 43% of the athletes developed BH within 2 years, and we calculated that BH would be present in the whole group within 4.65 years (table 3).

Method 2

BR increased significantly in all seven monitored athletes (time course of FEV₁, fig 2; maximal decrease in FEV₁, table 2; area FEV₁ by time, table 2). After extrapolation of the decrease in FEV₁ based on the EIB test, the limit of 10% (definition for BH) would occur within 1.77–4.81 years (table 3 and fig 3). With

Figure 1 Exercise-induced bronchoconstriction (EIB) of those athletes (n=3) who were initially not asthmatic and developed bronchial hyperreactivity. Main effects were detected for year ($p < 0.001$), year $\times$ EIB ($p = 0.005$) and EIB ($p = 0.007$). Post hoc comparisons are shown. FEV₁, forced expiratory volume in 1 s.

Figure 2 Exercise-induced bronchoconstriction (EIB) for all athletes (n=7) who were initially not asthmatic. Main effects were detected for year ($p = 0.04$), year $\times$ EIB ($p = 0.002$) and EIB ($p < 0.001$). Post hoc comparisons are shown. FEV₁, forced expiratory volume in 1 s.

regard to the different models (1–4) used to extrapolate the incidence, the risk of developing BH in high-performance athletes is about 25% per year (range 21–57%), which is extraordinarily high (table 3 and fig 3). According to our calculations, a high-performance athlete would develop BH on average in about 4 years; however, because of the small sample size the variations are not reported.

DISCUSSION

The results of our study suggest that triathletes of the Swiss national team increased BR significantly within 2 years. Moreover, these data are clinically relevant, as three of the seven athletes developed BH and typical symptoms of asthma. In addition, on the basis of the assessed incidence (method 1) as well as our extrapolation for BH in the whole group (method 2), we postulate that, on average, athletes develop BH in about 4 years, which is extraordinarily rapidly. A high prevalence of asthma in high-performance athletes has been reported in several studies.^{2–5, 21} Verges *et al*²⁰ reported progressive development of airway obstruction in three cross-country skiers during their sports careers. However, no association was found between development of bronchial obstruction and persistence of detectable BH. No other study has examined the chronological progression of BR or the incidence of EIB in high-performance athletes.

To our knowledge, there are no findings on the incidence of asthma in high-performance athletes in the literature and therefore we cannot compare our results with previous studies. For 3 years we targeted international level competitive athletes as we expected to detect a higher incidence of EIB in this group of subjects.

The incidence of EIB in our triathletes was chiefly calculated using the standard method used in epidemiology (method 1,

assessment of newly developed BH). In addition, different extrapolations of BH based on linear regression through the lowest FEV₁ values of the EIB test (method 2, extrapolation) were performed. The latter method was chosen because measuring BH reduces the data obtained by EIB testing from quantitative assessments of BR to a simple qualitative statement (either BH or no BH). This would not cause a problem if the number of subjects were larger, which is difficult in sport-specific athletes competing at international level. Because of the small number of subjects and the design of our study, it was important to provide a more qualitative tool to examine and interpret the data. The two methods for calculating the incidence (assessment and extrapolation) produced similar conclusions. Our approach using several methods of extrapolation for BH in the seven athletes is valid because it shows a significant linear relationship in models 1 and 2 as well as trends towards linearity in models 3 and 4 during the 2 years. Our results point to aspects that have practical implication for EIB concerning epidemiology, diagnostics and therapy.

Prospective studies of asthma incidence in the general population reveal an incidence of 1.5–2.2 per 1000 persons per year.^{31, 32} If we compare the incidence in the general population with that in our athletes, our athletes show a 195–286 times higher incidence of EIB. Our limited sample size does not allow us to derive a more accurate conclusion. However, these results may reflect a higher risk of developing asthma in athletes, and we strongly suggest that the effect of long-term participation in elite-level endurance sports on BH warrants further investigation. As data from a large number of high-level athletes are hard to collect in event-specific national teams, ideally multicentre studies should be carried out which would provide better answers to these questions.

Figure 3 Method 2, using extrapolation of bronchial hyperreactivity (BH). To extrapolate BH used in models 1–4, linear regression was used through the lowest FEV₁ values for each year (shown as mean) from the group of athletes, who were initially not asthmatic, were not treated, and performed at international competition level for the entire testing period (n=7). The time to develop BH in our group of seven athletes depends on the method and is represented by the lines under the graph: model 1=4.81 years; model 2=3.69 years; model 3=3.94 years; model 4=1.77 years.

What is already known on this topic

- High-performance athletes have a higher risk of exercise-induced bronchial reactivity (BR) and asthma than the general population.
- The reasons are not well understood but they may include sport-specific, environmentally induced (exposure to cold temperatures and pollutants), mechanical impairments of forced breathing and dehydration.
- It has been suggested that BR and asthma may be due to an inflammatory granulocyte reaction dominated by neutrophil-driven asthma.

What this study adds

- We may have underestimated rather than overestimated the increase in exercise-induced BR over the 2 years of our study.
- The importance of convective and evaporative heat loss is not certain; however, osmotic effects and evaporative heat loss appear to be more important factors in exercise-induced BR than convective heat loss.
- If other studies show similar dramatic increases in BR, it may suggest significant clinical concerns for athletes and doctors.
- We do not know if athlete's asthma is preventable or if treatment is useful in initially healthy high-performance athletes.

The progression of BR was highly significant and increased continuously during the 2 years of our study. This was not only statistically significant but also clinically relevant as three of the seven athletes developed BH. It is not known if what is known about asthma in the general population also applies to athlete's asthma. Helenius *et al*²⁹ reported aggravation of eosinophilic airway inflammation in highly trained swimmers who remained active during a 5-year follow-up. The same study reported that BH and asthma attenuated or disappeared in swimmers who had stopped high-level training, suggesting that athlete's asthma may be reversible after a competitive sports career. More longitudinal studies in athletes are required to adequately investigate the long-term development of BH and asthma in athletes during and after their competitive sports careers.

In our study, the prevalence of asthma in triathletes was about five times higher than that in the general population of Switzerland. This is based on the Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) conducted during 1991–1993 in 9651 adults aged 18–60.³³ According to that study, 6.8% of the Swiss population suffers from asthma, and 4.5% suffer from atopic asthma. Our results are in agreement with previous investigations conducted on high-performance athletes.^{2–5, 21} Given the large number of studies on prevalence and incidence of asthma in the general population,^{31–33} we did not include a control group in our study. However, to compare statistically the results for athletes with those of the general population under standard conditions, we recommend that future investigations match athletes with non-athletic control subjects.

BR and BH were assessed using EIB tests because exercise provocation is more specific than chemical provocation in identifying asthma in young people^{34, 35}, although it is less sensitive in identifying BH.^{35, 36} Moreover, field-based exercise tests seem to be more sensitive in detecting EIB than laboratory-based exercise tests.¹³ Lastly, an exercise challenge reflects more accurately than other challenges the specific situation of triathletes. In our study, EIB testing was conducted at a constant running intensity that allowed comparisons from one test to the other. There was no significant difference in absolute humidity between the first and second test (table 2). Therefore, we did not expect to find differences in post-exercise pulmonary function caused by differences in air conditions during the first two tests.⁷ During the third test, however, the air was significantly more humid than during both previous trials (table 2). As humid air has been shown to have a protective effect on EIB,⁷ we would expect an even more dramatic increase in BR during the third test if the air conditions were similar for both previous years. Therefore, our results seem to underestimate rather than overestimate the increase in EIB over the course of the 2 years. Although the relative importance of convective and evaporative heat loss is still unclear, it has been suggested that osmotic effects and evaporative heat loss are more important factors in the induction of EIB than convective heat loss.¹⁵

Further studies in high-performance athletes in different sports are warranted to confirm to what extent BR increases. If other studies were to demonstrate similar dramatic increases in BR, it would augment clinical concerns and warrant doctors to warn elite athletes of the incidence of BR, BH and EIB in high-performance sports. It is not known if athlete's asthma is preventable or whether treatment is useful in healthy high-performance athletes.

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COMMENTARY

This paper fills a void in the literature concerning chronic exposure to exercise stress and how it may adversely affect pulmonary function of triathletes competing at a high level. The use of a predictive equation to track changes in pulmonary function before they become a serious issue might be of great benefit in the future.

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Figure 1 Jon Karlsson.