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Prevalence of exercise-induced bronchoconstriction in elite Chinese summer sport athletes

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Abstract

Background: Exercise-induced bronchoconstriction (EIB) is a prevalent respiratory condition among summer sport elite athletes, yet epidemiological data from Asian populations remain scarce. The objective of this study was to investigate the prevalence, sport-specific patterns, and physiological characteristics of EIB in Chinese summer sport elite athletes.
Methods: A cross-sectional study of 500 summer sport elite athletes across 17 sports was conducted. Participants underwent standardized exercise challenge testing, spirometry, and serum biomarker assessments (eosinophils, interleukin-5 (IL-5), interleukin-8 (IL-8), Clara Cell protein 16 (CC16), immunoglobulin E (IgE), and uric acid (UA)).
Results: EIB prevalence was 27.6% (138/500), with significant variation across sports: highest in swimming (51.52%) and lowest in wrestling (6.45%). Female athletes were more prevalent than males (31.1% *vs.* 23.7%, $p = 0.030$). Outdoor sports demonstrated higher rates than indoor disciplines (37.4% *vs.* 19.4%, $p = 0.002$). EIB-positive athletes showed pronounced post-exercise declines in forced expiratory volume in 1 s (FEV_1) at 5 min ($p < 0.001$) and elevated inflammatory biomarkers: eosinophils ($p < 0.001$), neutrophils ($p = 0.019$), IL-5 ($p < 0.001$), IL-8 ($p < 0.001$), CC16 ($p < 0.001$), IgE ($p < 0.001$), and UA ($p < 0.001$) *vs.* EIB-negative counterparts.
Conclusion: This is the first large-scale study of Chinese athletes to reveal EIB prevalence exceeding global averages. Distinct risk profiles emerge, associated with gender, athletic level, sport type, and environmental factors. The findings outline the need for targeted screening programs and biomarker-guided management to mitigate respiratory health risks in athletic populations.

Keywords: Exercise-induced bronchoconstriction; FEV1; Elite athlete; China; Asian

1. Introduction

Exercise-induced bronchoconstriction (EIB) is characterized by acute airway constriction following physical activity, which can occur in individuals with or without a history of asthma.[1] EIB is a condition where the airway reacts to various bronchoconstrictive stimuli and contracts more readily to triggers (e.g., dry air). It is primarily due to airway dehydration resulting from sustained high minute ventilation, which raises the osmotic pressure of airway-lining fluids and releases mediators such as histamine and leukotrienes, causing muscle contraction and swelling in the airways.[2] EIB is a common chronic condition among Olympic competitors, negatively

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impacting their athletic performance, health status, and overall well-being.[3] Athletes with healthier lungs can train more effectively, resulting in fewer training interruptions. Thus, appropriately managing respiratory health can optimize athletic performance.[4] Once athletes with EIB receive appropriate treatment, their aerobic performance may return to levels comparable to those of non-EIB athletes.[5] In contrast, untreated EIB can hinder athletic ability and performance, with rare but serious consequences such as sudden death reported.[6−8]

Airway dysfunction in elite athletes deserves equal attention as other occupational lung diseases.[9] The prevalence of EIB is higher among athletes (10%−70%) compared to the general population (4%−12%).[2,10] There has been a gradual increase in the prevalence of EIB among elite athletes. For example, the prevalence of EIB in U.S. Olympic athletes shows an increasing trend, with 9.7% (1976), 16.7% (1996), and 21.9% (2000);[11] EIB in Italian and Finnish Olympians increased from 11.3% and 9.4% in 2004 to 17.2% and 12.6% in 2012, respectively.[12,13] Price et al.[14] conducted a recent meta-analysis indicating that the prevalence of EIB among elite athletes remains at 22%, showing no decrease compared to data from 2004. The World Anti-Doping Agency (WADA) and the International Olympic Committee's Medical Commission (IOC-MC) permit elite international-level athletes with approved Therapeutic Use Exemption Authority (TUE) to utilize medication within specified dosage ranges for therapeutic purposes during both training and competitive events. In 2016, WADA observed a notable increase in the proportion of EIB-TUE applications compared to figures from 2008. The elevated frequency of EIB occurrences and corresponding TUE requests underscores a potential health concern among athletes on a global scale.

Chinese athletes have a long-standing record of international success at the Olympic Games. However, EIB in athletes has not received sufficient attention in China. Some studies report EIB prevalence in Chinese athletes in summer Olympic disciplines (26.5%) and winter Olympic disciplines (36.7%); however, these findings were constrained by limited sample sizes.[15,16] Compared with established EIB management frameworks within the IOC,[17] China lacks systematic protocols for athlete respiratory health. This study, therefore, aimed to: (a) establish the first nationally representative EIB prevalence data in Chinese summer sport elite athletes; (b) identify sport-specific and sex-based risk stratification; and (c) characterize airway inflammation biomarkers across athletic disciplines.

2. Methods

2.1. Study design

This research adopts a cross-sectional analysis structured into four components: participant recruitment, an introductory information survey, blood sample collection, and an exercise challenge test (Fig. 1). The study protocol received ethical approval from the Academic and Ethics Committee of Shanghai University of Sport (Approval number: 102772020RT082) and was conducted in accordance with the principles of the Declaration of Helsinki. Before any testing commenced, all participants provided written informed consent. Patients and/or the public were not involved in the design, conduct, reporting, or dissemination plans of this research. The study focused on elite athletes as a professional cohort, and due to the specific nature of respiratory health assessments in high-performance sports, ethical and logistical constraints precluded direct public involvement. All procedures adhered strictly to institutional protocols for elite athlete populations.

2.2. Study participant recruitment

Participants were recruited from provincial representative teams across various regions of China and 13 national representative teams. The inclusion criteria required participants to meet at least one of the following conditions: (a) active membership in the Chinese National team; (b) at least 1 instance of active participation as a national team athlete between 2019 and 2023; (c) rank within the top 12 in national sports events over the past 3 years (national team selection prioritizes athletes ranked within the top 12 positions in domestic competitions, constituting a strategic cohort for targeted performance development initiatives); (d) rank within the top 8 in national competitions or the top 12 in international championships; or (e) rank equally in competitions of a similar or higher level. Based on the athlete classification framework of the General Administration of Sport of China and internationally recognized standards,[18] athletic level is categorized into Olympic/international level (Tiers 5 and 4, China Certified International Masters), national level (Tier 4, China Certified National Masters), and regional level (Tier 3, China Certified National First-Class). Participants were excluded according to the criteria established by Reis et al.[19] and Graham et al.[20] if they met any of the following conditions: (a) presence of chronic cardiopulmonary diseases (e.g., chronic obstructive pulmonary disease, pulmonary heart disease, known structural heart disease) or other significant medical conditions (e.g., uncontrolled hypertension, depression) that could confound respiratory test results or pose a risk during intense exercise; (b) history of chest surgery within the preceding 6 months; (c) any active lung infection (e.g., pneumonia, bronchitis) or systemic infection within 3 weeks before the examination; (d) cessation of regular training for 2 or more weeks immediately before the examination, to ensure participants were in their typical training state; (e) experience of a moderate or severe upper respiratory tract infection (e.g., common cold) within the week preceding the examination; (f) individuals who reported chest pain or experienced syncope (fainting) during the spirometry maneuver; and (g) individuals who were unable to understand the test instructions or unwilling to cooperate fully with the testing procedures, as determined by the research staff.

Five hundred seventy-nine athletes preliminarily met the inclusion/exclusion criteria, and informed consent was

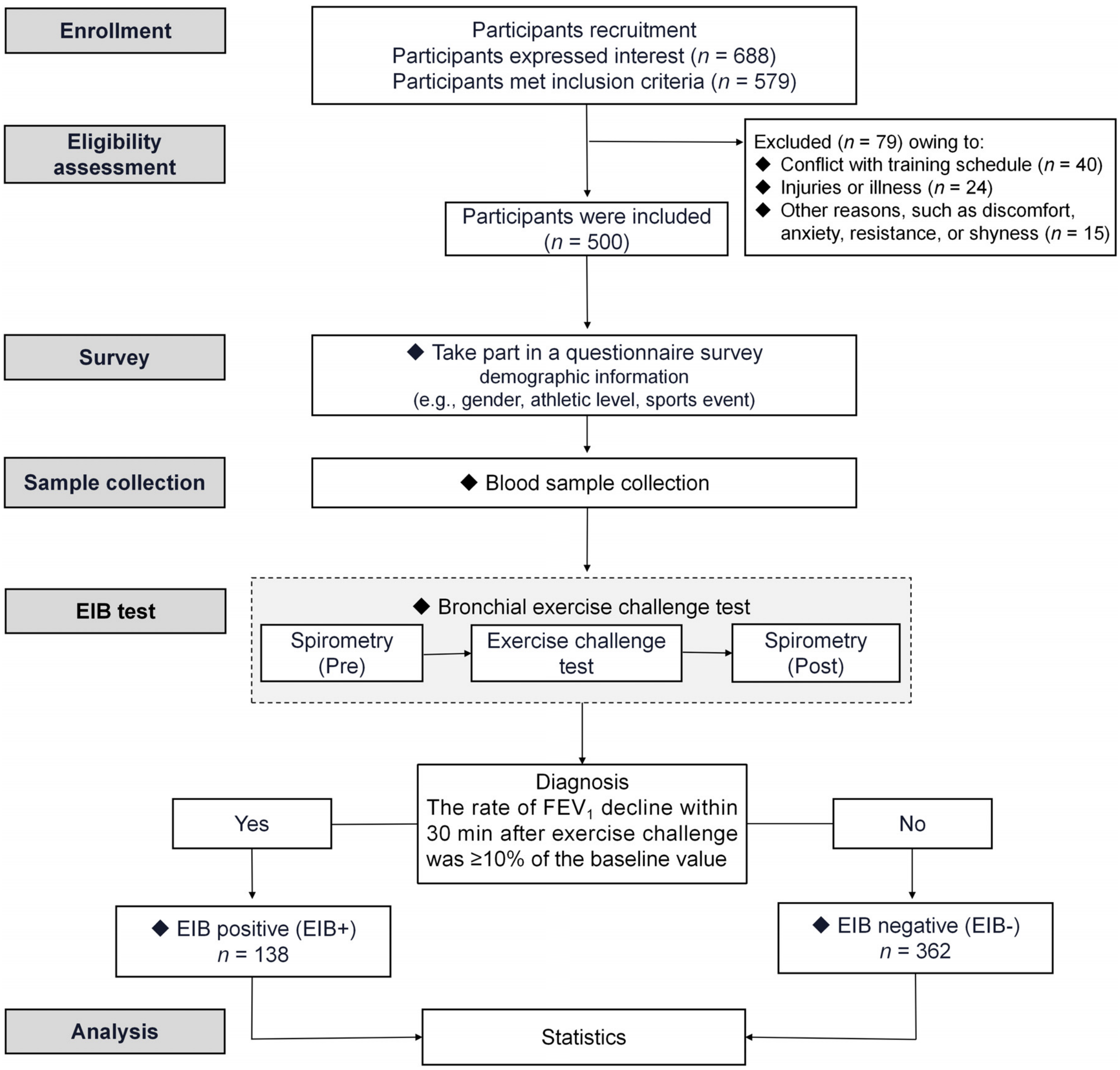

Fig. 1. Flow diagram. EIB = exercise-induced bronchoconstriction; FEV_1 = forced expiratory volume in 1 s.

obtained from each athlete (Supplementary Table 1). Among the 579 athletes who met the criteria, 79 did not participate due to scheduling conflicts with competitions or training ($n = 40$), acute injuries or illnesses *prior to* the test ($n = 24$), or due to personal reasons or voluntary withdrawal ($n = 15$). Finally, 500 athletes (Tables 1 and 2) agreed to participate in our study and signed informed consent forms. According to data from the General Administration of Sport of China (2022), our sample represents 11.3% of athletes of comparable levels within these 17 summer Olympic sports in China.[21]

Table 1
Athlete information.

Athlete information	Mean ± SD
Height (cm)	173.1 ± 8.5
Weight (kg)	61.7 ± 13.1
Age (year)	23.5 ± 4.1
Weekly training time (h)	33.9 ± 5.3
Professional training (year)	6.9 ± 3.4

2.3. Questionnaire survey

As reported in previous studies, an introductory information survey (Supplementary Table 2) was administered using paper questionnaires to gather necessary data on the athletes.[22] The survey included 8 primary aspects, including immediate measurement morphological parameters (e.g., height, body mass), training experience (e.g., training duration, years of participation), history of respiratory diseases (e.g., asthma, chronic obstructive pulmonary disease), symptoms of exercise-induced respiratory discomfort (e.g., wheezing, chest tightness, dyspnea), family history of asthma (restricted to immediate family members), smoking history, allergy history (including food and drug allergies), and EIB diagnosis and treatment information (clinical diagnosis, medication, and EIB-TUE).

Table 2
Questionnaire results.

Question	Yes/Total response (%)
1. You experienced severe coughing, chest tightness, asthma, and dyspnea during exercise.	177/500 (35.3)
2. Allergic to medicine, insect bites/stings, food or anything.	107/500 (21.3)
3. You had a history of smoking.	47/500 (9.4)
4. Your smoking history is ≥3 years.	20/500 (4.0)
5. You had been diagnosed with asthma.	11/500 (2.2)
6. Your family member had been diagnosed with asthma.	4/500 (0.8)
7. You ever used asthma medication.	10/500 (2.0)
8. You were previously diagnosed with EIB.	3/500 (0.6)
9. You had successfully applied for EIB-TUE.	2/500 (0.4)

Abbreviations: EIB = exercise-induced bronchoconstriction; TUE = therapeutic use exemption.

2.4. *Blood sample collection*

In the early morning (6:30−7:30 a.m.) of the test day, 4 mL of blood were collected from the brachial vein of each athlete after fasting for ≥8 h. The samples were stored in ethylene diamine tetraacetic acid (EDTA) Tubes A (1 mL) and B (3 mL). Tube A was used for counting basophils, eosinophils, and neutrophils within 10 min after sampling using a hematology analyzer (Model BC-5150; Mindray, Shenzhen, China). Tube B was used to separate the serum within 30 min after sampling with a centrifuge (Model TG16; Xiangyi, Changsha, China) at 3000 revolutions per minute (rpm) for 15 min. The serum was stored in Ultra-low temperature freezer (Model BDF-86V158; Biobase, Jinan, China). Serum samples were analyzed using a SAL-6000 Modular System (Mindray, Shenzhen, China) and a microplate reader (Thermo Fisher Scientific, Waltham, Massachusetts, MA, USA). Enzyme-linked immunosorbent assays (ELISA) were utilized to measure levels of interleukin-5 (IL-5), interleukin-8 (IL-8), Clara Cell protein 16 (CC16), immunoglobulin E (IgE), uric acid (UA), and cortisol (COR). ELISA kits were obtained from Jiancheng Bioengineering Institute (Nanjing, China), and the tests were conducted strictly according to the manufacturer's instructions. The selection of these specific biomarkers was informed *a priori* by their established roles in EIB pathophysiology: IL-5 and eosinophils for type 2 inflammation, IL-8 and neutrophils for neutrophilic inflammation, CC16 for airway epithelial injury, IgE for atopy, and uric acid for oxidative stress and purine metabolism. Cortisol was included to assess stress response.[23,24]

2.5. *Exercise challenge test*

Athletes were prohibited from strenuous exercise, alcohol consumption, smoking, and eating foods or supplements with known or potential bronchodilatory or EIB-relieving effects (e.g., caffeine-containing products such as coffee, tea, energy drinks, chocolate, caffeine supplements, and fish oil rich in omega-3 fatty acids; all reliever medications such as short-acting beta2-agonists) within 30 h before the test. A comprehensive list was provided to each athlete and their coach to ensure compliance. Two hours before the test, laboratory temperature and humidity were adjusted and maintained at 20−25°C and 40%−50% relative humidity, respectively. One hour before the test, athletes were allowed to enter the laboratory to adapt to the indoor environment. Furthermore, they must wear loose clothing that does not restrict their full chest and abdominal expansion. The test was conducted in the afternoon (2:00−6:00 p.m.), and the athletes were at rest on the morning of the test day. Bronchial provocation was evaluated using the laboratory exercise test method recommended by the American Thoracic Society (ATS)/European Respiratory Society (ERS) for EIB diagnosis, which has been extensively used in related research.[25,26] Athletes wearing nose clips and heart rate belts (Model M430; Polar, Kempele, Finland) ran for 2−3 min with an appropriate initial speed on a treadmill (Model T150; COSMED, Rome, Italy). Next, the running speed was increased by 2 km/h, and the slope was increased by 0.5 every 20 s to the maximal exercise intensity (85% of maximal heart rate (HR_{max}), calculated using the formula: $208 - (0.7 \times \text{age})$),[27] and the running continued for 8 min. The heart rate monitor (Model M430; Polar) was worn only during the exercise challenge to ensure the target intensity was achieved. It was removed immediately post-exercise for all subsequent spirometry to avoid any potential restriction of chest wall expansion. The exercise challenge test is one of the diagnostic methods for EIB recommended by the ATS and the ERS. When the air is relatively dry and the exercise intensity reaches 80%−90% of HR_{max}, it has high sensitivity.[26]

2.6. *Spirometry*

According to the EIB diagnosis method recommended by the ATS, 8 time points were selected: pre-exercise test, 1st, 3rd, 5th, 10th, 15th, 20th, and 30th min after the exercise test.[28] A portable spirometer (Model Chest HI-101, Chestgraph; Chest M.I., Tokyo, Japan) was used to measure the pulmonary function value of the athletes. The test at each time point involved the maximal forced spirometry maneuver to measure forced expiration volume in 1 s (FEV_1), forced vital capacity (FVC), peak expiratory flow (PEF), and forced expiratory flow at 50% of FVC (FEF_{50}). Predicted values were calculated using ERS Global Lung Function Initiative (GLI) equations, with Southern athletes using GLI-2012 South East Asia equations and Northern athletes using GLI-2012 North East Asia equations based on their birth and living regions. During the spirometry maneuver, the athlete performed 2 tidal respirations followed by a deep inspiration until maximal. At that point, they were instructed to exhale forcefully upon hearing the command "blast". Athletes were required to exhale as much air as possible within the shortest time following the "blast" command. Exhalation continued until the command "stop" was given, with a minimum interval of 6 s between the "blast" and "stop" commands. During the interval between the "blast" and "stop" commands, maximal flow-volume variables such as FEV_1, FVC, and PEF were measured during each maximal forced expiration. The ATS and ERS statements were utilized as references to ensure the accuracy of spirometry results.[20] Decline rate (R) was calculated using the following equation:

$$R = \frac{Pre\ exercise\ FEV_1 - Post\ exercise\ FEV_1}{Pre\ exercise\ FEV_1} \times 100\%$$

Athletes were instructed to repeat the spirometry test within 1 min if $R \geq 10\%$, and the lower R-value was considered the official result. A positive result was confirmed in cases where $R \geq 10\%$ was observed for 2 consecutive tests. We did not suspend the experiment if an athlete's FEV_1 decline rate was $\geq$10% to ensure objective and accurate data collection. The laboratory supplies short-acting beta-2 agonist, and its use is determined by a sports medicine clinician based on the athlete's symptoms, with consent from the chief coach and the athlete. The criterion is the standardized threshold endorsed by major international guidelines, including the ATS.[1] This adherence to internationally recognized protocols ensures the validity of our findings and facilitates direct comparability with a substantial body of existing literature that utilizes the same diagnostic criteria and methodology.

2.7. Data analysis

Statistical analysis was performed using SPSS V 25.0 (IBM Corp., Armonk, NY, USA). Variables, except EIB prevalence, were shown as mean ± standard deviation (SD). *Chi*-squared tests compared EIB prevalence by gender (male/female), sports environment (indoor/outdoor), sports type (endurance/team/speed and power/skill), and athletic level (Olympic & national/regional). Bonferroni correction was applied for multiple comparisons. The prevalence data for all subgroups in this study were calculated using the Clopper-Pearson method with 95% confidence intervals (95%CIs) (Fig. 2). Repeated-measure analysis of variance assessed pulmonary function (e.g., FEV_1) between EIB-positive (EIB+) and EIB-negative (EIB−) athletes across 8 time points. Normality of continuous variables (e.g., inflammatory biomarkers) was assessed using a combination of graphical methods (Q−Q plots) and statistical indices (skewness and kurtosis Z-scores). Data were considered normally distributed if the absolute Z-scores for both skewness and kurtosis were <1.96. Normally distributed data are presented as mean ± SD and were compared using independent samples t tests. Non-normally distributed data were log-transformed if applicable. If normality assumptions were met after transformation, results from t tests on transformed data are reported as geometric means with 95%CIs. Otherwise, the Mann-Whitney U test was used, with data presented as median and interquartile range. Homogeneity of variances was verified using Levene's test. Statistical analysis and presentation are consistent with the checklist for statistical assessment of medical papers (CHecklist for statistical Assessment of Medical Papers (CHAMP)).[29]

3. Results

3.1. Symptoms

A total of 94 EIB+ athletes reported experiencing symptoms related to Question 1 (Table 2), while 44 EIB+ athletes reported no symptoms. Among the EIB− athletes, the number of those with symptoms and without symptoms was 83 and 279, respectively. Sensitivity = 68.1%; specificity = 77.1%; positive predictive value = 53.1%; negative predictive value = 86.1%. Although 35.3% of athletes reported respiratory symptoms during exercise, only 68.1% of those with objectively confirmed EIB reported such symptoms. Moreover, 22.9% of athletes without EIB also reported symptoms. This indicates that symptoms reporting is not a reliable indicator of EIB, although symptoms related to EIB highlight the importance of objective testing for diagnosis.[30]

3.2. Prevalence

One hundred and thirty-eight (27.6%) participants had a positive result for exercise challenges. Moreover, they were subsequently diagnosed with EIB by a sports medicine clinician. Fig. 3A shows that the prevalence rates for females were 31.1% (n = 82/264) and males were 23.7% (n = 56/236). The prevalence rates of Olympic & national athletes were 30.4% (n = 72/237) and of regional athletes, 25.1% (n = 66/263); Olympic and national athletes with EIB have more training years than regional athletes with EIB (8.9 ± 3.7 *vs.* 5.6 ± 2.5, $p < 0.001$).

Bonferroni-adjusted for multiple tests, $\alpha = 0.0083$ (0.05/6). Fig. 3B shows the prevalence rates for endurance, team, speed/power, and skill sports, which were 38.4% (n = 58/151), 33.7% (n = 29/86), 20.0% (n = 30/150), and 18.6% (n = 21/113), respectively. The prevalence rates were found to be higher in endurance sports compared to speed/power ($p < 0.001$) and skill sports ($p < 0.001$). The prevalence was also higher in team sports compared to speed/power and skill sports.

Fig. 3C shows that the prevalence rates were higher in outdoor sports (n = 85/227, 37.4%) compared to indoor sports (n = 53/273, 19.4%) ($p = 0.002$, Fig. 3C). Furthermore, the prevalence of EIB in pool sports was 47.2% (n = 25/53), which was more significant than in indoor hall sports at 12.7% (28/220) ($p < 0.001$). The prevalence rate was also higher in outdoor field sports than in indoor hall sports. Analysis depicted in Fig. 3D reveals that swimming had the highest prevalence rate of EIB at 51.52%, while wrestling had the lowest at 6.45%.

3.3. Spirometry

At baseline, the results indicate that the measured FVC, FEV_1, PEF, and FEF_{50} values were lower than

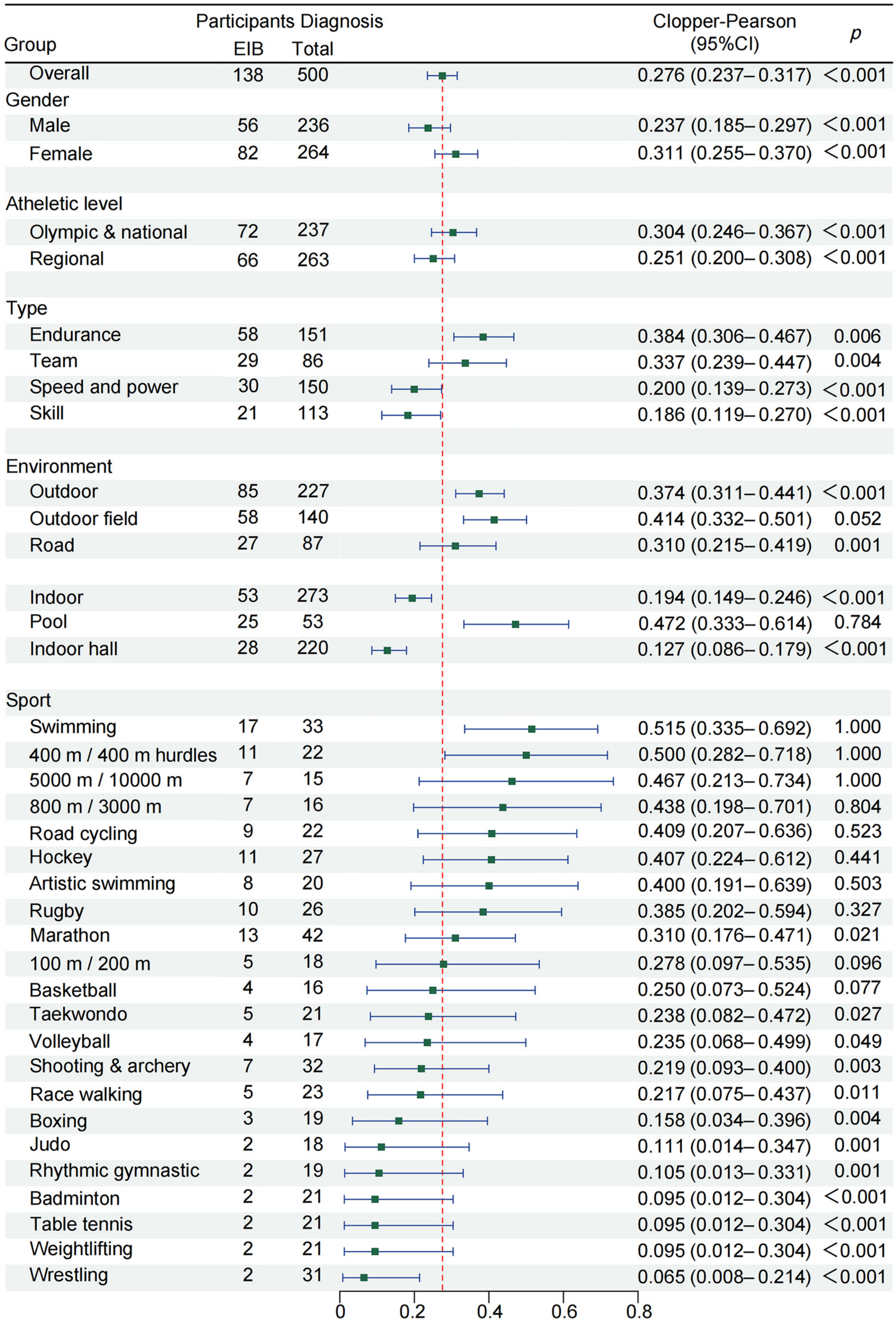

Fig. 2. Stratified comparison of EIB athletes with Clopper-Pearson 95%CIs. Red dotted line stands for overall Clopper-Pearson = 0.276. 95%CI = 95% confidence interval; EIB = exercise induced bronchoconstriction.

predicted. Among measured/predicted ratio, EIB+ athletes had slightly higher FVC (90.2% ± 15.0% *vs.* 89.9% ± 13.0%) but lower FEV_1 (87.2% ± 12.5% *vs.* 88.3% ± 12.3%), PEF (75.1% ± 19.1% *vs.* 81.4% ± 20.7%, $p = 0.011$), and FEF_{50} (73.3% ± 27.6% *vs.* 79.4% ± 26.2%) compared to EIB− athletes. After the exercise challenge test, the *R* values were elevated in EIB+ athletes compared to EIB− athletes at 1st, 3rd, 5th, 10th, 15th, 20th, and 30th min (Fig. 4A). However, we noted no differences in the baseline of FEV_1, FVC, FEV_1/FVC, PEF, and FEF_{50} between EIB+ and EIB− athletes *prior to* the spirometry (Table 4).

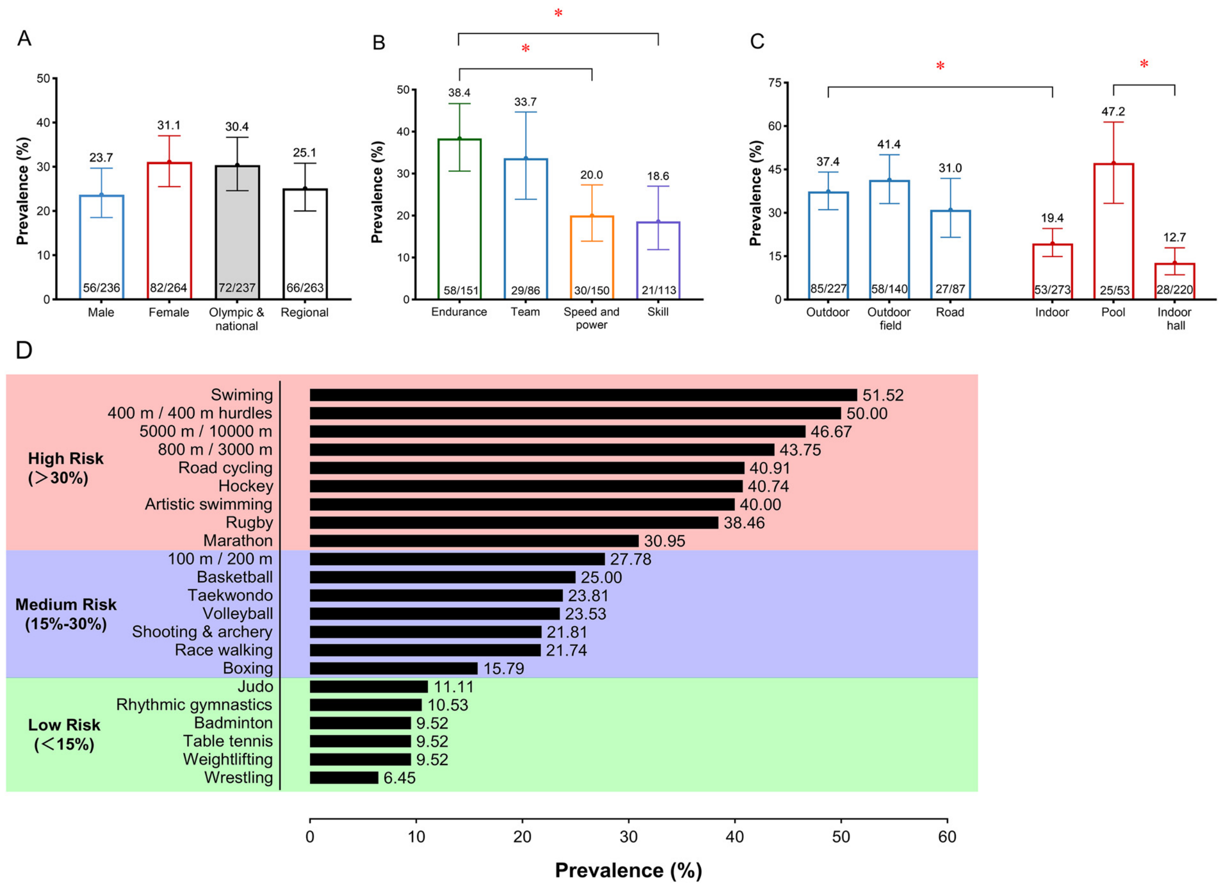

Fig. 3. Prevalence of EIB among athletes by gender, athletic level, sport type, environment, and sport. (A) Gender and athletic level; (B) sport type; (C) sport environment; (D) sport. * $p < 0.0083$. EIB = exercise induced bronchoconstriction.

The FEV_1 of EIB+ athletes was attenuated *vs.* pre-test ($p < 0.05$) at all measurement time-points from 1 min to 20 min post-exercise (Fig. 4A). The FEV_1 of EIB+ athletes was lower than in EIB− athletes at 1st, 3rd, 5th, 10th, and 15th min ($p < 0.05$); FVC decreased in the EIB+ group at 1st min ($p < 0.05$) and in the EIB− group at 1st and 3rd min ($p < 0.05$) (Fig. 4E); FEV_1/FVC ratio in EIB+ athletes dropped at 3rd and 5th ($p < 0.01$), while EIB− athletes showed an increase at 1st min ($p < 0.05$) (Fig. 4F). EIB+ athletes had lower PEF than EIB− athletes at 1st, 3rd, and 5th min ($p < 0.05$), PEF in the EIB+ group decreased at 3rd and 5th min ($p < 0.05$) but increased at 10th and 20th min in the EIB− group ($p < 0.05$) (Table 4). EIB+ athletes were lower FEF_{50} than EIB− athletes at 3rd and 10th min ($p < 0.05$), FEF_{50} in EIB+ athletes decreased at 1st, 3rd, 5th, and 10th min ($p < 0.05$), while EIB− athletes showed a slight increase, remaining higher than EIB+ athletes at 1st, 3rd, and 5th min ($p < 0.05$) (Table 4).

3.4. *Inflammatory and epithelial damage biomarkers*

The inflammatory biomarkers of respiration in EIB+ and EIB− athletes are shown in Tables 5 and 6. As no post-provocation samples were obtained, we could only assess baseline levels. At this baseline time point, eosinophil ($p < 0.001$), neutrophil ($p = 0.019$), and lymphocyte counts ($p = 0.015$) were higher in the EIB+ group over those in the EIB− group. However, we observed no differences in leukocyte count, basophil, monocyte, hemoglobin, erythrocyte, or erythrocyte distribution width between EIB+ and EIB− athletes; cortisol concentrations tended to be higher in the EIB+ group, but the difference was not significant (Table 5). The levels of IL-5, IL-8, CC16, IgE, and UA were significantly higher in EIB+ athletes compared with EIB- athletes (all $p < 0.001$; Table 6).

The inflammatory biomarkers of respiration in EIB athletes who performed in different sports are shown in Fig. 5. As shown in Fig. 5A, the eosinophil count of EIB+ athletes was higher in pool and outdoor field sports. The median basophil count revealed that among EIB+ athletes, levels were higher in those from road sports compared to other environments (Fig. 5B). We noted no differences in other sports. As shown in Fig. 5C, the neutrophil count of EIB+ athletes was higher in indoor hall sports than in pool ($p < 0.001$), field ($p < 0.001$), or road ($p < 0.001$) sports, while the neutrophil count in this group was higher in pool sports than in road sports ($p = 0.002$). As shown in Fig. 5D and E, the serum concentrations of IL-5 and IL-8 in EIB+ athletes in pool and road sports were above those in outdoor field and indoor hall sports ($p < 0.001$ for all comparisons). In addition, IL-5 ($p = 0.008$) and IL-8 levels ($p = 0.027$) were higher in pool sports than in indoor hall sports performed by EIB+ athletes. CC16 was also significantly higher in the road

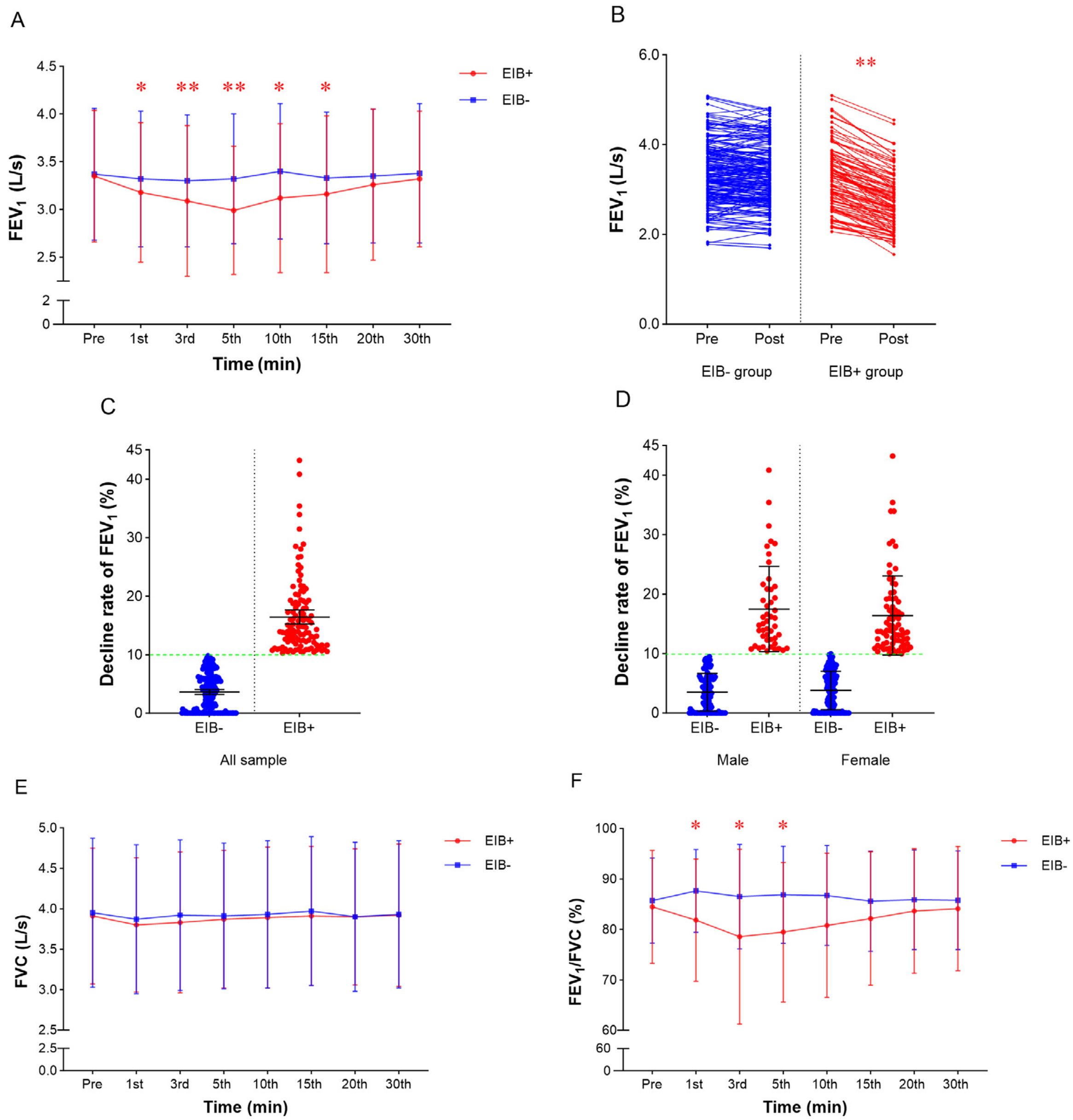

Fig. 4. Comparison of different pulmonary function parameters between EIB+ and EIB−. (A) FEV_1 before and after exercise challenge test; (B) maximum decline of FEV_1 after exercise challenge test between EIB− and EIB+ athletes; (C) individual maximum decline rate of FEV1 after exercise challenge test; (D) maximum decline rate of FEV_1 after exercise challenge test between male and female; (E) FVC before and after exercise challenge test; (F) FEV_1/FVC before and after exercise challenge test. Green dotted line = positive diagnostic criterion for EIB: the decline rate of FEV_1 ≥10%. * $p < 0.05$, ** $p < 0.01$ for comparisons between EIB + and EIB− athletes. EIB = exercise induced bronchoconstriction; EIB− = EIB negative; EIB+ = EIB positive; FEV_1 = force expiratory volume in 1 s; FVC = forced vital capacity; L/s = Liter/second.

($p < 0.001$), pool ($p < 0.001$), and outdoor field ($p = 0.038$) sports than in indoor hall sports (Fig. 5F); and it was higher in road sports than in outdoor field sports ($p < 0.001$).

4. Discussion

In our sample, 27.6% of Chinese athletes had EIB, with prevalence ranging from 6.45% to 51.52% across 17 summer Olympic sports. Olympic and national athletes had a higher EIB rate (30.4%) than regional athletes (25.1%), but this difference was not statistically significant. Female athletes had a higher incidence of EIB (31.1%) than male athletes (23.7%). The most significant decline in FEV_1 for EIB+ athletes occurred 5 min after exercise, while FEV_1 in EIB− athletes was minimally affected by intense physical activity. EIB+ athletes also had significantly higher blood concentrations of eosinophils, neutrophils, lymphocytes, IL-5, IL-8, IgE, CC16, and UA than EIB− athletes.

4.1. Characteristics and differences of EIB prevalence

The prevalence of EIB among summer sport elite athletes in China was 27.6%, which is higher than the prevalence reported in international studies (22%).[13] This regional specificity may stem from geographical, climate, and ethnic variations. Specifically, athlete data from various continents is structured in a hierarchical manner. The prevalence of EIB among athletes in Europe, Finland, Germany, Ireland, Italy, Norway, Spain, Poland, and the UK ranged from 10.1% to 22.4%.[12,31−37] In North America, including the USA and Canada, the prevalence ranged from 16.7% to 18.6%.[22,31] Meanwhile, in Oceania, including Australia and New Zealand, the prevalence ranged from 21.1% to 21.9%.[31] The prevalence in athletes from Tunisia, a North African country, is approximately 13%.[38] From a study involving 593 participants, Asian children exhibited a significantly higher susceptibility to EIB compared to other ethnic groups ($p < 0.01$).[39] Currently, high EIB prevalence among Chinese athletes, possibly extending the observed trend in Asian children, suggests that region and/or ethnicity may influence EIB precursors. Moreover, our observed prevalence may also be influenced by factors beyond race, ethnicity, and climate. First, while our recruitment strategy targeted entire teams rather than symptomatic individuals, a potential selection bias cannot be entirely ruled out. Athletes experiencing respiratory symptoms might have been more motivated to participate, potentially leading to an overestimation of the true prevalence. Second, the overall prevalence may be influenced by China's specific sporting culture and talent development focus. Future multi-national studies with standardized sport-specific sampling frameworks are needed to disentangle the effects of ethnicity, environment, and sport discipline distribution on EIB prevalence.

The 17 sports events in this study fall into 4 categories: endurance, team, speed and power, and motor skills (Table 3). EIB incidence was higher in endurance (38.4%) and team events (33.7%) than in speed and power (20.0%) and skill (18.6%) categories. These findings align with previous EIB studies.[10,40] Selge et al.[35] suggested that the prevalence of EIB varies due to differences in ventilation across sports, with athletes in high-ventilation sports having a higher asthma risk than those in low-ventilation sports. Additionally, exercise intensity has a greater impact on EIB prevalence than exercise duration.[3] Athletes are most at risk of EIB when exercising at 90%−95% of their peak heart rate, explaining its higher occurrence in 400 m/400 m hurdles and 5000 m/10,000 m events than in marathons and race walking.[3] Conversely, while marathon runners do not reach the same minute ventilation, their longer training duration likely results in greater total air ventilation than 400 m runners.

Table 3
Different stratification for corresponding numbers of athletes.

Stratification		Sum number
Gender (total = 500)	Male	236
	Female	264
Athletic level (total = 500)	Olympic/international athlete (Tiers 5 and 4, certified as International Master in China)	41
	National elite athlete (Tier 4, certified as National Master in China)	196
	Regional elite athlete (Tier 3, certified as National Level-1 in China)	263
Sport type (total = 500)	Endurance	151
	Road cycling, Marathon, 20-km race walking, Swimming, 800 m running; 3000 m running; 5000 m running; 10,000 m running	
	Motor skills	113
	Rhythmic gymnastics, Shooting and archery, Badminton, Artistic swimming, Table tennis	
	Speed and power	150
	100 m running, 200 m running, 400 m running, 400-m hurdles, Weightlifting, Boxing, Judo, Taekwondo, Wrestling	
	Team	86
	Basketball, Volleyball, Hockey, Rugby	
Sport environment (total = 500)	**Indoor**	
	Pool	53
	Swimming (33) Artistic swimming (20)	
	Indoor hall	220
	Taekwondo (21), Basketball (16), Volleyball (17), Boxing (19), Judo (18), Table tennis (21), Weightlifting (21), Rhythmic gymnastics (19), Shooting (16), Badminton (21), Wrestling (31)	
	Outdoor	
	Road	87
	Road cycling (22), 20-km race walking (23), Marathon (42)	
	Outdoor field	140
	Hockey (27), Rugby (26), Archery (16), 100 m and 200 m running (18), 400 m running and 400 m hurdles (22), 800 m and 3000 m running (16), 5000 m and 10,000 m running (15)	

Sporting events were also studied and divided into 4 groups based on the environment: indoor (pool 47.2%, indoor hall 12.7%) and outdoor (road 31.0%, outdoor field 41.4%) (Table 4). Outdoor sports had a significantly higher incidence (37.4%) than indoor sports (19.4%), likely due to greater exposure to air pollutants like industrial emissions, haze, dust, NO_2, and SO_2. Giles et al.[41] and McCreanor et al.[42] demonstrate that exercising in high-pollution areas reduces lung function (FEV_1 and FVC) and increases serum markers of airway inflammation and injury compared to low-pollution areas. Swimming pools pose a risk due to disinfectants like sodium hypochlorite and chloramines, which can harm airway tissues, cause inflammation, and trigger EIB. Research indicates a high rate of EIB in swimmers.[22,37,43] Weiler et al.[22] and Dickinson et al.[37] reported swimmers had high EIB rates (29.6%−44%). Our result not only supports the global consensus on swimming as a high-risk discipline but also highlights a high prevalence of EIB among Chinese elite swimmers that warrants attention.

The 2022 Global Initiative for Asthma guidelines indicate that elite athletes are more susceptible to EIB than average athletes.[44] Our study supports this, showing a higher EIB prevalence in Olympic & national athletes (30.4%) than regional athletes (25.1%), though the difference was not statistically significant ($p = 0.206$). The reason may be that Olympic and national athletes have significantly more professional training (8.23 ± 3.57 years *vs.* 5.21 ± 2.59 years, $p < 0.001$). This extended training leads to more significant and prolonged exposure to respiratory stressors, pollutants, and airway issues, increasing their susceptibility to EIB, particularly in sports like swimming.[45]

Gender is one of many variables that affect EIB prevalence. Couillard et al.[46] reported that 66.2% of female athletes had EIB, compared to 35.4% of males. In Olympic athletes, women had EIB rates of 23.7%−26%, while men had 18%−19.1%.[37,47] Our study reveals that EIB is more prevalent in Chinese female athletes (31.1%) than in male athletes (23.7%), consistent with past research. It could be due to women's smaller airway cross-sectional area, leading to more significant airway restriction and increased perception of dyspnea during exercise.[48] Additionally, androgen and estrogen secretion variations affect respiratory function through sex hormone receptors in the central respiratory brain region and nearby respiratory organs.[49] Research in animals and humans shows that progesterone increases airway inflammation and cytokines, exacerbates bronchial hyperreactivity, and is linked to the severity of EIB.[50,51] Notably, there are limited investigations to explain the high EIB prevalence in female athletes, which warrants more attention and protection for this population.

4.2. *Differences and characteristics of spirometry and blood sample*

A decrease in FEV_1 by ≥10% after an exercise challenge test confirms EIB. Our findings revealed that EIB+ athletes experienced their lowest FEV_1 at 5 min post-exercise and their lowest FEV_1/FVC ratio at 3 min post-exercise, indicating

Table 4
Changes in FEV1, FVC, FEV_1/FVC ratio, PEF, and FEF_{50} for all athletes.

	FEV_1 (L/s)		FVC (L/s)		FEV1/FVC (%)		PEF		FEF_{50} (L/s)	
	EIB+	EIB−	EIB+	EIB−	EIB+	EIB−	EIB+	EIB−	EIB+	EIB−
Pre	3.35 ± 0.69	3.37 ± 0.69	3.91 ± 0.84	3.95 ± 0.92	84.47 ± 11.19	85.72 ± 8.41	6.22 ± 2.39	6.45 ± 2.19	3.79 ± 1.04	3.98 ± 1.04
1st min	3.18 ± 0.73$^{\#}$	3.32 ± 0.71*	3.80 ± 0.83$^{\#}$	3.87 ± 0.92†	81.85 ± 12.14	87.66 ± 8.21*,†	6.07 ± 2.31	6.33 ± 2.15	3.45 ± 1.18$^{\#}$	4.04 ± 1.12*
3rd min	3.09 ± 0.79$^{\#\#\#}$	3.30 ± 0.69**	3.83 ± 0.87$^{\#}$	3.92 ± 0.93	78.56 ± 17.35$^{\#\#}$	86.51 ± 10.34*	5.69 ± 2.19$^{\#\#}$	6.45 ± 2.13*	3.21 ± 0.99$^{\#\#\#}$	3.96 ± 1.11**
5th min	2.99 ± 0.67$^{\#\#\#}$	3.32 ± 0.68**	3.87 ± 0.85	3.91 ± 0.90	79.46 ± 13.84$^{\#\#}$	86.87 ± 9.62*	6.00 ± 2.32$^{\#}$	6.44 ± 2.12	3.32 ± 1.16$^{\#\#}$	3.91 ± 1.09*
10th min	3.12 ± 0.78$^{\#\#}$	3.40 ± 0.71*	3.89 ± 0.87	3.93 ± 0.91	80.82 ± 14.26	86.74 ± 9.91	6.05 ± 2.54	6.70 ± 2.29*,†	3.38 ± 0.98$^{\#}$	3.89 ± 1.06
15th min	3.16 ± 0.82$^{\#}$	3.33 ± 0.69*	3.91 ± 0.86	3.97 ± 0.92	82.18 ± 13.22	85.59 ± 9.96	6.24 ± 2.50	6.26 ± 2.07	3.48 ± 1.04	3.92 ± 0.99
20th min	3.26 ± 0.79$^{\#}$	3.35 ± 0.70	3.90 ± 0.84	3.90 ± 0.92	83.66 ± 12.34	85.89 ± 9.88	6.20 ± 2.45	6.57 ± 2.25†	3.56 ± 1.09	4.19 ± 1.11
30th min	3.32 ± 0.71	3.38 ± 0.73	3.92 ± 0.88	3.95 ± 0.92	84.13 ± 12.31	85.78 ± 9.78	6.24 ± 2.50	6.26 ± 2.07	3.55 ± 1.06	4.22 ± 1.13**

* $p < 0.05$, ** $p < 0.01$ for comparisons between healthy athletes and athletes with EIB.
\# $p < 0.05$, \#\# $p < 0.01$, \#\#\# $p < 0.001$ for comparisons with EIB+ athletes before their exercise challenge test.
† $p < 0.05$, for comparisons with healthy athletes before their exercise challenge test.
Abbreviations: EIB = exercise-induced bronchoconstriction; EIB− = EIB negative; EIB+ = EIB positive; FEV_1 = forced expiration volume in 1 s; FEF_{50} = forced expiratory flow at 50% of FVC; FVC = forced vital capacity; L/s = Liter/second; PEF = peak expiratory flow.

Table 5
Blood biochemical indices in EIB+ and EIB− athletes (*t* test).

Variable	EIB+ ($n = 138$)	EIB− ($n = 362$)	*p*
Leukocyte count (10^9/L)	5.76 ± 1.53	5.55 ± 1.39	0.344
Neutrophil count (10^9/L)	3.99 ± 1.92	3.40 ± 1.61*	0.019
Lymphocyte count (10^9/L)	2.53 ± 0.47	2.31 ± 0.55*	0.015
Monocyte count (10^9/L)	0.36 ± 0.11	0.34 ± 0.12	0.461
Hemoglobin (g/L)	138.82 ± 16.01	139.55 ± 12.99	0.811
Erythrocyte (10^{12}/L)	4.55 ± 0.53	4.43 ± 0.42	0.272
Erythrocyte distribution width (%)	12.55 ± 1.33	13.24 ± 8.02	0.591
Cortisol (ng/mL)	389.44 ± 84.10	366.66 ± 82.02	0.338

* $p < 0.05$, for comparisons between EIB+ and EIB− athletes.
Abbreviations: EIB = exercise-induced bronchoconstriction; EIB− = EIB negative; EIB+ = EIB positive.

Table 6
Blood biochemical indices in EIB+ and EIB− athletes (Mann-Whitney *U*).

Variable	Group	Median	IQR	*U*	*p*	*Z*	Effect size (*r*)
Eosinophil count (10^9/L)	EIB+	0.17	0.24	19,899.5	<0.001	3.566	0.16
	EIB−	0.13	0.12				
Basophil count (10^9/L)	EIB+	0.03	0.05	23,892.5	0.529	0.629	0.028
	EIB−	0.03	0.03				
IL-5 (pg/mL)	EIB+	109.25	71.86	11,718	<0.001	8.636	0.389
	EIB−	56.06	43.04				
IL-8 (pg/mL)	EIB+	250.25	185.40	12,179	<0.001	8.862	0.398
	EIB−	151.45	110.23				
CC16 (ng/mL)	EIB+	20.5	14.9	19,429	<0.001	3.734	0.167
	EIB−	19.0	6.8				
IgE (μg/L)	EIB+	3359	676	8236	<0.001	11.596	0.519
	EIB−	2590	721				
UA (μmol/L)	EIB+	393.6	85.3	3551	<0.001	13.030	0.583
	EIB−	252.3	32.7				

Abbreviations: CC16 = Clara Cell protein 16; EIB = exercise-induced bronchoconstriction; EIB− = EIB negative; EIB+ = EIB positive; IgE = immunoglobulin E; IL-5 = interleukin-5; IL-8 = interleukin 8; IQR = interquartile range; UA = uric acid.

airflow restriction, which then gradually improved. EIB− athletes showed no significant changes post-exercise. Beyond the observed decline in FEV_1, our spirometric data revealed a significant post-exercise reduction in FEF_{50} among EIB+ athletes, suggesting concomitant small airway dysfunction. Small airways play a critical role in EIB pathogenesis due to their structural fragility and susceptibility to environmental stimuli.[52] Dysfunction in these airways can lead to early constriction, ventilation heterogeneity, and air trapping, even before large airway involvement.[53] Such isolated small airway involvement, especially in athletes without a clinical asthma history, could represent an early or "pre-EIB" phenotypic stage, preceding the classic large-airway bronchoconstriction measured by FEV_1.[54] According to Poiseuille's law, minor constriction in narrow airways (<2 mm) can exponentially increase resistance,[55] leading to gas trapping and reduced expiratory efficiency. Pulmonary ventilation restriction may stem from 3 factors: (a) Long-term training in EIB athletes frequently leads to airway injury and repair cycles, causing irregular, smooth muscle and airway surface changes, increasing inhalation and exhalation friction; (b) EIB disrupts airway moisture regulation, increasing Muc5ac secretion by goblet cells, resulting in mucus buildup and higher airway resistance; and (c) The most critical issue is airway injury and inflammation from smooth muscle contraction, leading to bronchial stenosis.[56,57]

Airway epithelial injury is key in EIB development in elite athletes.[58] Rapid breathing during exercise increases shear stress, disrupting the airway epithelial barrier and boosting immune stress.[59] CC16, a biomarker of this injury, leaks out due to breakdown of the barrier. Post-ECT (8 min, 80% maximum aerobic speed), urine CC16 levels were about 2.75 times higher in EIB+ than in EIB− athletes.[60] EIB+ swimmers had significantly higher baseline sputum CC16 levels than EIB− swimmers.[61] In our study, we observed higher baseline serum CC16 levels in EIB+ athletes ($p < 0.001$), indicating incomplete airway repair in these athletes. This persistent airway injury and repair cycle lowers the response threshold of airway epithelial cells, leading to hyper-reactivity.[62] Inadequate protection allows allergens, high osmotic pressure, and other stimuli to cause ongoing airway inflammation.[57] Our findings indicate that EIB+ athletes had significantly higher resting blood levels of eosinophils, IL-5, and IgE than EIB− athletes, suggesting greater

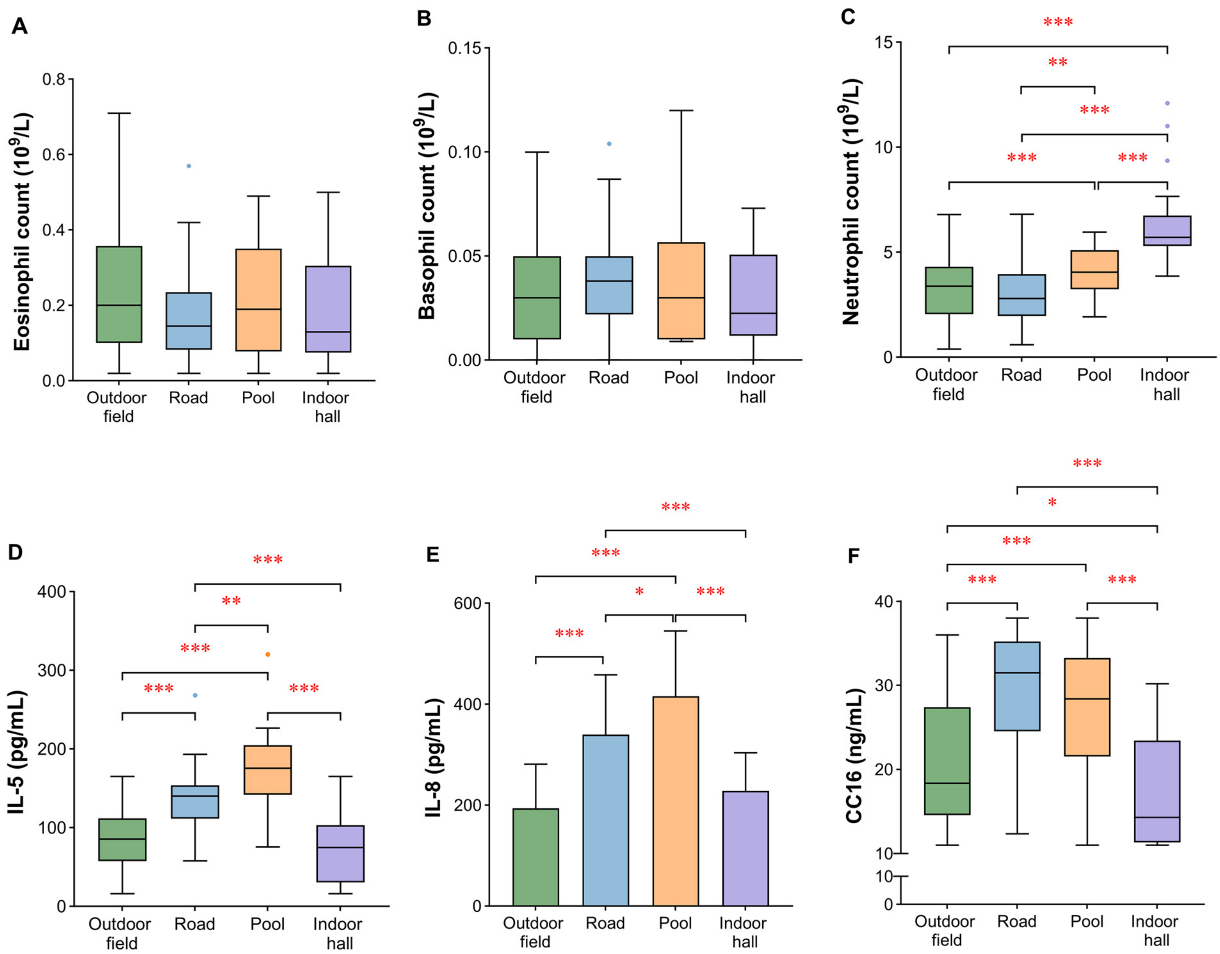

Fig. 5. Comparison of inflammatory and epithelial injury biomarkers in EIB+ athletes in different sports environments. (A) Eosinophil count; (B) basophil count; (C) neutrophil count; (D) IL-5; (E) IL-8; (F) CC16. Data that are not normally distributed between groups are presented using box plots (Tukey method). This graph shows the median and the range from the 25th percentile to the 75th percentile. Normally distributed data (Panel E, IL-8) are presented as bar charts showing mean ± SD. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. CC16 = Clara Cell protein 16; EIB = exercise-induced bronchoconstriction; EIB− = EIB negative; EIB+ = EIB positive; IL-5 = interleukin-5; IL-8 = interleukin 8.

susceptibility to atopic airway inflammation. IL-5 is crucial for eosinophil development, and eosinophils interact with IgE to release inflammatory proteins. EIB+ athletes also had significantly higher levels of neutrophils and IL-8, which initiate and amplify innate immune responses. Thus, the elevated CC16 and inflammatory biomarkers in EIB+ athletes provide a mechanistic link between airway epithelial damage and the chronic inflammatory state that underpins EIB.

EIB+ athletes' blood markers are influenced by their sports environment. Comparing 4 settings (pool, road, indoor hall, and outdoor field), swimmers showed higher levels of eosinophils, CC16, IL-5, and IL-8, indicating more severe airway damage and varied inflammation due to pool exposure. Road athletes exhibit elevated basophils and CC16, indicating serious airway injury likely due to high exercise loads. Indoor stadium athletes have higher neutrophil levels than other groups but lower levels of the inflammatory mediators and airway injury markers, possibly due to exposure to stadium pollutants. Field athletes exhibit elevated eosinophils and basophils, with other indices not showing significant differences. These findings may provide initial insights into the physiological differences of EIB+ athletes based on their sports environments. Our findings of elevated inflammatory biomarkers (eosinophils, IL-5, IL-8, and CC16) and environmental sport-specific risks suggest that EIB in athletes may involve complex gene−environment interactions. Furthermore, the observed airway injury and persistent inflammation raise concerns about potential long-term cardiopulmonary sequelae, particularly in sports with high environmental exposures, such as swimming and outdoor endurance events. These insights underscore the importance of future research to investigate genetic predispositions, longitudinal health outcomes, and personalized treatment strategies tailored to inflammatory phenotypes.

Our study discovered that resting serum UA median levels were higher in EIB+ athletes than in EIB− athletes (393.6 μmol/L *vs.* 252.3 μmol/L, $p < 0.001$), with higher cortisol levels but not significantly higher. Similar findings were reported by Li et al.,[63] who observed a positive correlation between serum uric acid levels and asthma severity and a negative correlation with pulmonary ventilation function ($r = -0.507$, $p < 0.001$) in 217 asthma patients and 142 nonasthma patients. Jiang et al.[64] studied 143 children aged 10−16 and found higher blood cortisol levels in those with asthma. They suggested that airway epithelial apoptosis leads to inflammation, increased purine nucleotide metabolism, and increased xanthine oxidase activity. UA, produced from adenosine triphosphate (ATP) breakdown to xanthine in low-oxygen conditions and decomposed by xanthine oxidase, may be a biomarker for EIB. However, further research is required to confirm this.

A critical finding of this study is the profound underdiagnosis of EIB in Chinese summer sport elite athletes, evidenced by the disparity between the low pre-existing diagnosis rate (2.2% for asthma and 0.6% for EIB) and the high prevalence identified through objective testing (27.6%). This discrepancy likely stems from a systemic lack of awareness, where athletes and coaches often misattribute EIB symptoms (e.g., dyspnea, bronchoconstriction) to inadequate physical conditioning, leading to increased efforts to overcome perceived fitness deficits rather than seeking respiratory care.

The ability of these athletes to reach elite levels despite unmanaged EIB is notable. This may be attributed to exceptional physiological compensation, allowing them to tolerate the condition. However, it more likely suggests that the primary impact of unmanaged EIB is not an absolute barrier to performance but a subtler, cumulative limitation on training quality, recovery, and the ability to consistently achieve peak performance.[5] This aligns with observations that Olympic medalists often exhibit a high prevalence of EIB using appropriate inhaler therapy to manage the condition,[65] implying that optimal control of EIB is one of the key factors in unlocking full athletic potential rather than merely enabling participation. Consequently, the performance ceiling for these athletes may be higher than currently observed. The vast population base in China may also contribute to this phenomenon, as a high attrition rate of athletes whose careers were limited by unmanaged EIB during development would remain unseen in a cross-sectional study of established elites. This underscores the imperative for systematic screening programs to optimize performance and protect long-term respiratory health in current and future athletic cohorts.

An intriguing observation in our study was that baseline spirometric values (FVC, FEV_1, PEF, and FEF_{50}) were consistently lower than those predicted by GLI-2012 reference equations across the entire cohort of elite athletes. This pattern was evident even within the EIB− subgroup, indicating that it is a characteristic of the high-level athletic population itself rather than solely a consequence of high EIB prevalence. We postulate 2 non-exclusive explanations for this finding. First, the GLI equations, derived from general populations, may not fully capture the unique respiratory physiology of elite athletes, whose respiratory systems are highly adapted for efficiency rather than isolated maximal maneuvers. Second, long-term, high-intensity training might induce specific physiological adaptations—such as a more "economical" resting state to preserve a greater ventilatory reserve for exercise, analogous to athletic bradycardia—or subtle airway remodeling due to chronic exposure to high ventilation. This finding underscores the potential need for sport-specific prediction equations in the future and highlights that "lower-than-predicted" values in this unique cohort may represent a specialized functional state rather than dysfunction.

4.3. Study strengths and limitations

This study is the first comprehensive global analysis of EIB prevalence in Chinese athletes, addressing a lack of data on Asian and East Asian athletes. It also provides a foundation for future research examining ethnic disparities in EIB. The "Chest HI-101" portable spirometry device has proven reliable and accurate in EIB studies.[60,66] Controlled lab-based exercise challenge tests ensured that observed bronchoconstriction in athletes was due to exercise alone, not environmental factors. Our lab conditions (20−25˚C, 40%−50% relative humidity) were slightly higher than the 18˚C and 40% humidity recommended for exercise challenges. Hence, the prevalence may change with a more provocative EIB challenge.

The findings of this study must be considered in light of certain limitations. The present findings, based on summer sport athletes, are not generalizable to winter sport athletes, who face different environmental risks. The decision to exclude winter sports was primarily based on logistical feasibility and the need to maintain standardized testing conditions across all participants. This specific scope should be taken into account when interpreting our results. The study involved 17 relatively independent projects at different training or competition stages. Although not conclusively proven, it is hypothesized that EIB prevalence may vary across training phases and be influenced by exercise programs. This study was unable to account for potential variability. Another limitation is that prevalence may differ in response to various challenges; for example, EVH challenges are more provocative than exercise bronchial provocation. Some EIB can be seasonal and affected by training status, which was not controlled, potentially resulting in overlooked athletes. Moreover, the FVC of both EIB+ and EIB− athletes decreased post-exercise. The observed decline in FVC could reflect suboptimal respiratory muscle conditioning during early athletic training, making it challenging for individuals to maintain proper breathing techniques when fatigued, despite their efforts to exhale forcefully. Subjects were retested if their FEV_1 decline rate (R) was $\geq$10%, and EIB+ was confirmed if R was $\geq$10% in 2 consecutive tests, to minimize misdiagnosis due to poor pulmonary function tests. Furthermore, the potential for selection bias must be acknowledged. Participants were recruited from provincial and national teams, which may not be fully representative of all summer sport elite athletes in China. Athletes who agreed to participate might also differ systematically from those who declined (e.g., in their concern about respiratory health), which could influence the generalizability of our prevalence estimates.

5. Conclusion

This large-scale cross-sectional study provides the first comprehensive analysis of EIB prevalence among Chinese summer sport elite athletes. Notably, the vast majority of EIB+ athletes in our cohort had not been previously diagnosed, underscoring a significant gap in respiratory health monitoring within elite athletic populations in China. It revealed a markedly higher rate (27.6%) than global averages (22%). Key findings highlight significant sex disparities (31.1% in females *vs*. 23.7% in males), sport-specific risks (highest in swimming (51.52%) and endurance disciplines), and environmental influences (37.4% in outdoor *vs*. 19.4% in indoor sports). Elevated inflammatory biomarkers (eosinophils, IL-5, IL-8, CC16, IgE, and UA) and pronounced post-exercise FEV_1 decline in EIB+ athletes underscore the interplay of airway injury, inflammation, and hyper-reactivity. The results underscore the need for regular screening of athletes in high-risk sports (e.g., swimming, endurance events) using objective bronchial provocation tests, as well as a phenotype-guided management approach that incorporates inflammatory biomarkers to tailor therapy. Subsequent investigations should prioritize multicenter cohort studies examining gene-environment interactions in EIB pathogenesis, the long-term cardiopulmonary sequelae of sport-specific training modalities, and the comparative effectiveness of phenotype-guided pharmacologic interventions.

Authors' contributions

ZC performed the research protocol, acquired and analyzed the data, performed extensive manuscript writing and revision, and handled all journal correspondence; JD, MAF, and MD participated in the study design and provided expert revision advice; SC supervised manuscript revision, liaised with international co-authors, and polished the language; BG provided senior supervision of the research and revision guidance; ML conceived and executed the study design, wrote the initial draft, secured funding, liaised with experts, supervised and guided manuscript revision. All authors contributed to multiple revisions of the article. All authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

Declaration of competing interest

The authors declare that they have no competing interests.

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Data availability statement

Data are available upon reasonable request.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the authors used Grammarly in order to improve language clarity and readability, polish and refine the English language. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Supplementary materials

Supplementary materials associated with this article can be found in the online version at doi:10.1016/j.jshs.2026.101131.

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