Training of aerobic and anaerobic fitness in children 
with asthma
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Exercise limitation is often encountered in children with asthma. The primary factor is generally thought to be their relative hyperventilation during exercise, which leads to dyspnea and exercise-induced bronchospasm (EIB). Self-limitation of physical activities then occurs to further avoid these unpleasant feelings. In children with asthma, muscle deconditioning through hypoactivity is thought to explain their low level of physical fitness. Several controlled studies support this concept: (1) children with asthma with a normal forced expiratory volume at one second (FEV₁) have normal habitual physical activity and exercise performance, (2) maximal oxygen uptake (VO₂max) is correlated with physical activity, and (3) physical fitness in children with asthma can be enhanced by reconditioning programs. In contrast, an impaired exercise capacity independent of hypoactivity has been demonstrated in adult and pediatric patients, probably because of overinflated lungs, leading to ventilatory or cardiovascular limitation. As a result, decreased exercise capacity is consistently reported in children with asthma who have basal bronchial obstruction.

On the basis of pathophysiologic concepts, training programs for the aerobic reconditioning of children with asthma have shown well documented benefits: the mean increase in maximal oxygen consumption normalized by body mass is approximately 15%, with the anaerobic threshold shifted toward higher metabolic levels. Until recently, exercise capacity in asthmatic subjects has been studied only in terms of aerobic fitness. Yet it seems that the anaerobic component of exercise performance may also be important, because maximal anaerobic and maximal aerobic work capacities are decreased in

**Objective** To assess the effect of a training protocol on aerobic and anaerobic fitness in children with asthma.

**Study design** Sixteen boys (mean age: 13 years; range: 10-16 years) with mild-to-moderate asthma participated in a rehabilitation program that included 6 weeks of individualized training on a cycle ergometer. Two groups were randomly formed: the control group (CG, n = 7) and the training group (TG, n = 9), which exercised at an intensity set at the heart rate corresponding to the ventilatory threshold, with 1-minute sprints against the maximal aerobic power (MAP) every 4 minutes. Session duration was 45 minutes, 3 sessions per week. Changes in maximal oxygen uptake (VO₂max), MAP, short-term peak power (PP), and pulmonary function were assessed.

**Results** Two patients of the training group did not complete the study. Pulmonary function remained unchanged in both groups. Improvement in both aerobic and anaerobic fitness was significant only in the training group (TG vs CG): VO₂max +18% ± 2.1% versus +9% ± 4.5% (P < .05), MAP +32% ± 5% versus 12% ± 7% (P < .05), PP +21% ± 5.7% versus +8.8% ± 10% (P < .01).

**Conclusion** Exercise training with high-intensity bouts is well tolerated in children with mild-to-moderate asthma. When included in a global rehabilitation program, this type of training improves both aerobic and anaerobic fitness. Anaerobic activities should be considered in sports rehabilitation programs for children with asthma. (J Pediatr 2003;142:179-84)
acclimatization to altitude (1400 m), 6 weeks without any acute episode of wheezing, one year without emergency department visits or hospitalization for acute asthma, and a basal \( \text{FEV}_1 \) > 70% of predicted. The diagnosis of asthma was made on the basis of the following criteria: (1) personal or familial history of allergy; (2) personal history of acute wheezing; (3) reversible airway obstruction documented by lung function testing, ie, improvement of 15%, at least in \( \text{FEV}_1 \) and/or 30% in forced expiratory flow 25-75 by inhaling a bronchodilator; (4) positive specific immunoglobulin E to inhaled allergens by a multi-allergen allergosorbent test (Phadiatop, Pharmacia, Uppsala, Sweden) and/or a cutaneous hypersensitivity to one or several allergens; and (5) no evidence of other lung disease.

A control group of 7 subjects was randomly formed from the 16 volunteers. Two patients of the training group did not complete the study. There were no significant differences in anthropometric characteristics, basal spirometry, and the habitual level of physical activity between the 2 groups (Table I). Nine of the 14 children were taking inhaled steroids.

Informed consent was obtained from the children and their parents before participation in the study; the protocol was approved by our local ethics committee.

**Measurements**

Lean body mass (LBM) was estimated from skinfold thickness measurements using the Durnin and Rahaman formula.\(^{15}\) Sexual maturity was scored with the Tanner indices.\(^{16}\) Lower limb volume (LLV) was evaluated according to the method of Jones and Pearson.\(^{17}\) Weekly physical activity estimates were based on a standardized activity interview\(^{18}\) and expressed in metabolic equivalents (MET). All children underwent pulmonary function testing (System 2800 Autobox, Sensormedics, Anaheim, Calif) at rest, and values were expressed as a percentage of pediatric standards.\(^{19}\)

Aerobic fitness was first assessed by a maximal incremental exercise test on a cycle ergometer (864, Monark-Crescent AB, Varberg, Sweden). A 3-minute warm-up period at

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**Table I. Demographic data for 14 patients**

<table>
<thead>
<tr>
<th></th>
<th>Control group</th>
<th>Training group</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>(n = 7)</td>
<td>(n = 7)</td>
</tr>
<tr>
<td>Mean age (y)</td>
<td>13.9 ± 0.8</td>
<td>14 ± 0.6</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>46.8 ± 3.7</td>
<td>48.9 ± 3.2</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>159.9 ± 5.8</td>
<td>159.4 ± 2.9</td>
</tr>
<tr>
<td>Lean body mass (kg)</td>
<td>42 ± 3.4</td>
<td>42.1 ± 2.3</td>
</tr>
<tr>
<td>Lower limb volume (L)</td>
<td>8.52 ± 0.7</td>
<td>9.45 ± 0.6</td>
</tr>
<tr>
<td>Sexual maturation stages</td>
<td>3 (2-4)</td>
<td>3 (2-4)</td>
</tr>
<tr>
<td>IgE (kUI/L)</td>
<td>1008.1 ± 235</td>
<td>944.7 ± 197</td>
</tr>
<tr>
<td>MET</td>
<td>6896 ± 627</td>
<td>6844 ± 607</td>
</tr>
<tr>
<td>Inhaled corticosteroids</td>
<td>5/7</td>
<td>4/7</td>
</tr>
</tbody>
</table>

*MET*, Metabolic equivalent for weekly physical activity.
30 W was followed by 15- or 30-W increments at 1-minute intervals while the subject pedaled at a constant frequency of 60 rpm. Ventilation and gas exchange were measured with a breath-by-breath automated open circuit system (CPX, Medical Graphics, St Paul, Minn), for the calculation of oxygen consumption (VO2). Exercise continued until at least 3 of the maximal oxygen uptake criteria were obtained: (1) a plateau of VO2 in spite of the increase of workload, (2) maximal heart rate (maximal predicted heart rate ± 5%), (3) respiratory exchange ratio >1.10, and (4) inability to maintain the pedaling frequency (maximal predicted heart rate ± 5%).

The anaerobic threshold (AT) was the disproportionate increase in carbon dioxide output observed during the test was noted. The anaerobic threshold (AT) was defined as the point where the respiratory exchange ratio increased to 1.10.

The maximal pedaling frequency (rpm) was recorded and the linear force-velocity and parabolic force-power relationships were calculated by an automated system that allowed determination of both V from the measurement of pedaling frequency and, for each F, the power corresponding to the product F · V. The accuracy of the pedal revolution duration was 3.3 ms. The test was stopped when there was no longer any increase in the power output in spite of an increasing breaking load. The maximal anaerobic peak power (PP) was calculated from the parabolic force-power relationship. The intercepts of the FV regression line determined Fo and Vo. Fo is the maximal breaking mass the subject could theoretically handle; Vo is the maximal cycling velocity theoretically obtained if the breaking mass was null (Figure).

### Training Programs

The training group exercised 3 times weekly for 6 weeks, with each session consisting of 45 minutes of continuous cycling activity. The design of the training sessions was adapted from Gimenez et al. The target heart rate was individualized and corresponded to the AT level. Every 4 minutes, the subject was asked to sprint for 1 minute against the breaking load corresponding to his MAP. During all sessions, heart rate was continuously monitored with a cardiofrequency meter (Sport Tester PE 3000, Polar Electro, Kempe, Finland). A training instructor and a pulmonologist supervised each session to ensure that the clinical condition was stable and the training procedures were followed.

### Protocol

All subjects underwent clinical examination, anthropometric measurements, ECG and spirometry before entering in the study. They then familiarized themselves with the exercise testing procedures. Each subject performed one incremental exercise test and one FV test, with pre- and postexercise pulmonary function testing. The same evaluation was repeated when the 6 weeks of training were completed. Testing was done blindly regarding the training groups. Peak flow rates were monitored twice daily. All treatment was given under direct medical supervision. Asthma exacerbations with oral corticosteroid use and hospitalization for any reason during the protocol were exclusion criteria.

### Statistical Analysis

Means ± SD values are reported. A Mann-Whitney Wilcoxon rank test was used for between-group comparisons. A 2-way analysis of variance was conducted to evaluate the group × time effect on pulmonary function and fitness parameters. Multiple regression models were applied to assess the relative contribution of the independent variables to the total variance of the fitness variables (VO2max, MAP, PP). These potentially confounding variables were anthropometric and clinical characteristics.
decreased after training only. VO\textsubscript{2} at AT was significantly higher post-training than pretraining. Multiple regression models applied to the whole population (n = 14) revealed that the best subsets model explaining the variance of VO\textsubscript{2}\text{max} and MAP were a linear function of training (\(r^2 = 0.4, P = .0003\)) and a linear combination of sexual maturation and training (\(r^2 = 0.53, P = .006\)), respectively. Anthropometric data, daily activity, initial versus final VO\textsubscript{2}max or MAP measurements, and PFTs did not significantly improve the models when added.

**Table III. Changes in aerobic fitness**

<table>
<thead>
<tr>
<th></th>
<th>Control group (n = 7)</th>
<th>Training group (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>Final</td>
</tr>
<tr>
<td>VO\textsubscript{2}\text{max} (mL/min–1/kgLBM–1)</td>
<td>50.8 ± 1.8</td>
<td>55.4 ± 2.3</td>
</tr>
<tr>
<td>MAP (W/kgLBM–1)</td>
<td>3.28 ± 0.28</td>
<td>3.68 ± 0.23</td>
</tr>
<tr>
<td>HR\text{max} (beats/min–1)</td>
<td>172 ± 5</td>
<td>172 ± 5</td>
</tr>
<tr>
<td>V\text{E}max (L/min–1)</td>
<td>86.1 ± 8.8</td>
<td>87.3 ± 10.4</td>
</tr>
<tr>
<td>VR (%)</td>
<td>11.9 ± 5.4</td>
<td>15.9 ± 5.3</td>
</tr>
<tr>
<td>AT(% of VO\textsubscript{2}\text{max})</td>
<td>54.5 ± 2.4</td>
<td>50.9 ± 3.7</td>
</tr>
<tr>
<td>VO\textsubscript{2} at AT (mL/kg–1)</td>
<td>25.4 ± 2.5</td>
<td>24.9 ± 2.3</td>
</tr>
<tr>
<td>O\textsubscript{2} pulse (mL/beat–1)</td>
<td>12.6 ± 1.3</td>
<td>13.7 ± 1.4</td>
</tr>
</tbody>
</table>

\(\text{VO}_{\text{max}}\), maximal oxygen uptake; \(P_{\text{max}}\), maximal power during VO\textsubscript{2}\text{max} testing; \(HR_{\text{max}}\), maximal heart rate; \(AT\), anaerobic threshold; \(V\text{E}_{\text{max}}\), maximal minute ventilation; \(FR\), ventilatory reserve.

Values are means with SD.

* \(P < .05\).

\† \(P < .01\).

**Table IV. Changes in anaerobic fitness**

<table>
<thead>
<tr>
<th></th>
<th>Control group (n = 7)</th>
<th>Training group (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>Final</td>
</tr>
<tr>
<td>PP (W/kgLBM–1)</td>
<td>11.3 ± 1</td>
<td>12.3 ± 1.2</td>
</tr>
<tr>
<td>Vo (rev/min–1)</td>
<td>172 ± 8.5</td>
<td>177.6 ± 9.6</td>
</tr>
<tr>
<td>Fo (N/kgLBM–1)</td>
<td>2.55 ± 0.0</td>
<td>2.65 ± 0.0</td>
</tr>
<tr>
<td>dFEV\textsubscript{15} (%)</td>
<td>1.4 ± 1</td>
<td>1.4 ± 1</td>
</tr>
</tbody>
</table>

\(PP\), Peak power reached during the force-velocity test; \(Fo\) and \(Vo\), intercepts of the linear force-velocity relationship. \(\text{dFEV}_{15}\), change in FEV\textsubscript{1} 15 minutes after the force-velocity test.

* \(P < .01\).

Table data, sexual maturation, pulmonary function tests (PFTs), initial versus final evaluation (learning effect) and training. The level of statistical significance was set at \(P < .05\).

**RESULTS**

**Tolerance and Side Effects**

The exercise tests were well tolerated. Peak power performed during the FV test was 3-fold higher than MAP. During the training sessions, the maximal drop in peak flow was ~40%. Two subjects did not complete the training program because of a limb fracture unrelated to training in one and fatigue and lack of motivation in the other. The basal PFTs were not modified in these 2 patients and their clinical course remained stable. PFTs and lability of postexercise airways flows were not modified in either group (Table II).

**Aerobic Fitness (Table III)**

No statistical difference was found at the initial evaluation. After 6 weeks, the training group exhibited a significant increase in VO\textsubscript{2}\text{max} (+18 ± 2.1% vs +9 ± 4.5%, \(P < .05\)) and MAP (+32 ± 5% vs 12 ± 7%, \(P < .05\)), whereas no significant changes were observed in the control group. Maximal heart rate was increased and ventilatory reserve was decreased after training only. VO\textsubscript{2} at AT was significantly higher post-training than pretraining. Multiple regression models applied to the whole population (n = 14) revealed that the best subsets model explaining the variance of VO\textsubscript{2}\text{max} and MAP were a linear function of training (\(r^2 = 0.4, P = .0003\)) and a linear combination of sexual maturation and training (\(r^2 = 0.53, P = .006\)), respectively. Anthropometric data, daily activity, initial versus final VO\textsubscript{2}\text{max} or MAP measurements, and PFTs did not significantly improve the models when added.

**Anaerobic Fitness (Table IV)**

Initial evaluation did not reveal any significant difference between the 2 groups for Vo, Fo, and PP; although initial Vo seemed to be lower in the training group (\(P = .4\)). After 6 weeks, the trained group exhibited an improved PP, whereas the control group remained stable (+8.8 ± 9.7% vs +21 ± 6.7%, \(P < .01\)). Multiple regression models applied to the whole population (n = 14) revealed that the best subsets model explaining the variance of PP was a linear combination of height, daily activity, and initial versus final PP measurements (\(r^2 = 0.69, P = .0002\)). Other anthropometric data, training, sexual maturation, and PFTs did not significantly improve the regression models when added.
DISCUSSION

The anaerobic fitness of asthmatic children can be improved by a specific training protocol that includes a combination of ventilatory threshold workloads and high-intensity workloads. This protocol was well tolerated by children with stabilized, mild-to-moderate asthma, which was shown by the absence of significant adverse reactions. One subject of the 7 who completed the training protocol, however, had a poor tolerance attributed to either muscular fatigue or poor motivation. As part of a rehabilitation protocol, a combined aero-anaerobic training program gave specific and significant benefits in terms of physical fitness to those who completed it.

The accurate and reliable evaluation of the effects of physical conditioning is generally difficult in children with asthma. Our children were within normal ranges for daily activity for French children. The most striking point is that children in training programs receive extra attention as they exercise under the supervision of health professionals and are thus in a privileged situation regarding monitoring of their disease. They may therefore benefit from more finely adjusted treatment leading to therapeutic compliance. For this study it was thus very important to ensure a similar level of medical attention to the control group. Our population, recruited from 2 long-stay clinics dedicated to the rehabilitation of asthmatic children, was ideal for this purpose. The daily monitoring of clinical status, physical activity, peak expiratory flow, and therapeutic compliance involved all patients and ensured the optimal care of the control group as well. It is likely that this situation explains the remarkably stable condition of all patients throughout the study. The absence of significant changes in the daily treatment and the PFTs confirmed the clinical stability of the entire population and excluded variability in pulmonary function as an influence on the physical fitness evaluation.

Another major concern regarding the evaluation of pediatric exercise capacity is the child’s commitment during testing. Although specific pediatric criteria for aerobic fitness evaluation are lacking, the peak VO2 measured during an incremental exercise test has been reported to be a valid tool for VO2max determination in children. In our study, the initial VO2max and MAP values were within the range of healthy active boys of similar age, indicating good participation. Measurement of blood acidosis would have been of interest to further confirm the criteria for maximal exercise levels, but we consider this procedure too invasive. Our strongest indication, in fact, of a valid evaluation of aerobic fitness was the finding that all children in both groups met the criteria we set for VO2max determination, especially a respiratory exchange ratio above 1.1. During the FV test, it was easier to verify that maximal power was reached. As stated above, the relationship between force and velocity is quasilinear. Submaximal cycling would have led to aberrant points, which was not the case for any of the subjects tested. All of the PP values observed in our population were in the range of normal pediatric values.

The improvement in aerobic fitness confirmed previous reported findings in children with asthma. Only a few controlled studies are available on the effects of endurance training in children with asthma. Although VO2max and MAP have shown improvement, and AT shifted toward a higher metabolic level in some studies, no significant changes in VO2max, MAP, or AT were observed in others. In our study, the trained children with asthma increased their maximum heart rate and used more of their ventilatory reserve, which suggests improved tolerance to exercise. They shifted their AT toward a higher metabolic level, and training was the main explanatory factor of the variation in the total variance of their VO2max and MAP, suggesting a true aerobic training effect. It is very likely that this improvement can be attributed to muscular conditioning rather than cardiorespiratory adaptation since the PFTs and the O2 pulse remained stable after training: the theoretic maximal ventilatory reserve (35 · FEV1) and the maximal stroke volume as evaluated by the O2 pulse were not significantly modified by the training protocol.

Current data have shown that supramaximal power outputs are generally diminished whatever the type of exercise done by children with mild-to-moderate asthma. Anaerobic fitness as evaluated by the Wingate test, a 30-second all-out cycling test, has been described as either normal or decreased when compared with that of healthy controls. Interestingly, patients with significant basal bronchial obstruction, ie, patients with cystic fibrosis or moderate asthma, have shown diminished maximal power output during the Wingate test. Such a test, however, gives a global picture of aerobic and anaerobic fitness, because the contribution of the aerobic pathway to total energy production is significant in children. Shorter exercise bouts like those of the FV test are mainly anaerobic and glycolytic, and the anaerobic fitness evaluated by this test was also found to be diminished in children with asthma. In our training group, the increase in PP seems related more to an increase in VO2, suggesting a training effect on velocity. Training, however, was a weak explanatory factor of the variation in the total variance of PP, and confounding effects of anthropometry or daily activity cannot be ruled out. The effectiveness of anaerobic training would have probably been more pronounced with supramaximal training loads but the risks and benefits of strength training for children have not yet been extensively studied.

In conclusion, aerobic and anaerobic exercise fitness can both be enhanced by specific training in children with mild-to-moderate asthma. This type of training is well tolerated. When bronchial obstruction is alleviated, the mechanism of muscle conditioning is probably not very different from that of healthy children. Further controlled studies are nevertheless needed to determine whether atopic asthma is associated with a specific pattern of muscle fiber conditioning. Organized sports activities should include short and intense bouts of muscle work and should be proposed to stable asthmatic children.
REFERENCES


