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DOI 10.1055/s-0033-1345137
Int J Sports Med 2013; 34: 1051–1057

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Rüdigerstraße 14
70469 Stuttgart
ISSN 0172-4622

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Diffusion Capacity of the Lung in Young and Old Endurance Athletes

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Key words

- master athletes
- diffusion capacity
- FEV₁
- transfer coefficient

Abstract

Lung diffusion capacity (D_{LCO}) declines with age. A significant proportion of older endurance athletes develop exercise-induced hypoxemia ($SaO_2 < 95\%$). We hypothesised that master endurance athletes have a lower D_{LCO} than age-matched non-athletes. We recruited 33 control (16 young; 17 old) and 29 male endurance athletes (13 young; 16 old) during the World Masters Athletics Indoor Championships, 2012 (Jyväskylä, Finland). To measure D_{LCO} the participant exhaled to residual volume and then quickly inhaled to $\geq 90\%$ total lung capacity from a gas source with 0.3% carbon monoxide. The D_{LCO} and transfer coefficient (K_{CO}) were cor-

rected for the actual haemoglobin concentration. Spirometric function was similar in athletes and age-matched controls. D_{LCO} and K_{CO} were 33% and 25% lower in old and young controls, respectively ($P < 0.001$). Although predicted D_{LCO} and K_{CO} were 11%-points higher in athletes than age-matched controls ($P < 0.001$), they were 23% and 16% lower in old athletes than young controls, respectively ($P < 0.001$). D_{LCO} did not correlate with age-graded performance or weekly training hours. The better lung diffusion capacity in male endurance athletes than age-matched controls might be an adaptation to training, self-selection and/or attrition bias. However, the diffusion capacity of the older athlete is lower than that of the young non-athlete.

accepted after revision
March 15, 2013

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DOI <http://dx.doi.org/10.1055/s-0033-1345137>
Published online:
June 14, 2013
Int J Sports Med 2013; 34:
1051–1057 © Georg Thieme
Verlag KG Stuttgart · New York
ISSN 0172-4622

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Introduction

While cardiovascular and muscular adaptations to endurance exercise do contribute to the enhanced aerobic fitness of athletes, the respiratory system may show no adaptations to regular exercise [13, 14, 23, 27]. This is potentially important as it is in the respiratory system that the exchange of oxygen between the air and blood takes place ensuring an adequate oxygenation of the arterial blood. While aerobic capacity is generally not limited by the lung [13], the absence of any adaptations to endurance training in the respiratory system may contribute to the often observed exercise-induced hypoxemia ($SaO_2 < 95\%$) in endurance athletes [14, 27, 45]. This may be even more pronounced during exercise at a lower relative maximal oxygen uptake in the older endurance athlete [32].

We have recently shown that the forced expiratory volume in 1 s (FEV₁), forced vital capacity (FVC) and peak expiratory flow (PEF) of master athletes are better than that of age-matched sedentary people [12], suggesting that master

endurance athletes are partly exempt from the age-related decline in lung function.

Another factor to consider is the diffusion capacity of the lungs. In sedentary people the diffusion capacity of the lung does not limit exercise capacity. The inability to increase the diffusion capacity with training [34] may explain why the diffusion capacity 1) can limit performance in faster marathon runners [20] and 2) in young fit people predicts the maximal oxygen uptake [46] with athletes having a better lung diffusion capacity than non-athletes [26]. The diffusion capacity decreases, however, with age [19, 39] and is significantly reduced after exercise [8, 24, 27, 43]. In fact, particularly at maximal effort exercise may cause pulmonary oedema [8, 24] and damage the pulmonary blood-gas barrier (BGB) [41]. As a consequence, repeated endurance events over the years may reduce the integrity of the lung and result in a diminished diffusion capacity and cause the exercise-induced hypoxemia that is more prevalent and occurring at a lower maximal oxygen uptake level in older than younger endurance athletes

[32]. We therefore hypothesised that the diffusion capacity of the lung is higher in endurance athletes than age-matched non-athletes, but that this difference is less pronounced in older age. To investigate this we compared the diffusion capacity in male endurance athletes and age-matched non-athletes.

Materials and Methods

Ethical approval

The study was approved by the Ethics Committee of the Central Finland Healthcare District and complied with the guidelines of the *Declaration of Helsinki*. All participants had given written informed consent before inclusion. The study meets the ethical standards set forth by the International Journal of Sports Medicine [16].

Participants

All participants in the study were male Finns. We recruited 16 young control (YC), 17 old control (OC), 13 young endurance athletes (YA) from the local area and 16 endurance master athletes (OA) at the World Masters Athletics Indoor Championships, 2012 (Jyväskylä, Finland). Master athletes are typically defined as 'athletes older than 35 years of age and systematically train for, and compete in, organized forms of sport specifically designed for older adults' [33]. We included men between 20–35 and 65–79 years of age for the young and old groups, respectively. Current smokers were excluded from the study, as well as participants suffering from known chronic respiratory and cardiovascular diseases. Some of the athletes and control participants had smoked in the past, but their spirometric function was in the normal range for the respective groups and their data were included in the analysis. Data were excluded if the forced expiratory volume in 1 s (FEV1) was below the 70% predicted value and the FEV1/Forced Vital Capacity (FEV1/FVC) was below 70% [1], which was the case for one OC.

The athletes competed in endurance events, including long-distance running (1500 m and longer), triathlon, orienteering and cross-country skiing. In all participants the number of training sessions per week and hours per session were assessed by questionnaire and interview and presented as total training hours per week. The age-graded performance (AGP) for the main event of the master athletes was calculated using the WMA Age-grading Calculator:

<http://www.howardgrubb.co.uk/athletics/wmalookup06.html>.

This calculator represents the performance of an individual as a percentage of the world record at his/her age. In other words, if a 24- and a 70-year-old athlete have the same AGP, then the absolute performance of the 24-year-old athlete is better than that of the older athlete. The haemoglobin (Hb) concentration was determined from a small blood sample obtained by a finger prick. A small cuvette containing the blood sample was placed in a Hemo Control (EKF Diagnostics, Germany) and the [Hb] given as $g L^{-1}$.

Spirometry

All experiments were performed using the same spirometer and analysed with integrated software (Vmax SensorMedics, Viasys Healthcare, PA, USA). The calibration of the spirometer was checked on a daily basis according to the American Thoracic Society/European Respiratory Society (ATS/ERS) criteria [25]. All experiments were performed in a seated position and partici-

pants wore a nose clip. First, the (relaxed) vital capacity (VC) was determined. After taking a series of normal breaths, the participant exhaled fully followed by a full inhalation. At least 2 manoeuvres to determine VC were performed, and additional measurements were performed until the 2 highest VCs did not differ by more than 150 mL. After determination of VC, the diffusion capacity for carbon monoxide of the lungs (D_{LCO} in $mmol kPa^{-1} min^{-1}$) was determined as described previously [21]. A simulator test was performed daily, and the system was recalibrated before each individual test. During the test the participant was asked to start with normal respiration followed by a relaxed exhalation to residual volume and a quick inhalation to $\geq 90\%$ VC from the gas source with 0.3% carbon monoxide. After holding his breath for 9–11 s, the participant was asked to exhale. The interval between trials was ≥ 4 min. Trials with excessively high intrapulmonary pressures, excessively short or excessively long breath-holding times were excluded, and the average of at least 2 trials that fulfilled the criteria was used for further analysis. The D_{LCO} was presented unadjusted, adjusted for the actual [Hb] and as percentage predicted ($\%D_{LCOadj_{pred}}$). The D_{LCO} per alveolar volume (V_A) gives the transfer coefficient, K_{CO} . The K_{CO} was presented as percentage predicted K_{CO} (K_{COpred}) and as K_{COpred} adjusted for the actual [Hb] ($K_{COadj_{pred}}$). The V_A was determined with CH_4 tracer gas [21]. After completion of the measurements of the diffusion capacity the participants were asked to perform maximal forced inspiratory and expiratory manoeuvres after a short period of normal breathing to construct flow-volume curves. This procedure was repeated until the criteria set out by the ATS and ERS were met, i.e., a plateau in the expiration or expiration > 6 s, no coughs and inter-test variation for Forced Vital Capacity (FVC in L) and $FEV_1 < 0.15$ L [25]. Whenever participants did not satisfy the criteria, manoeuvres were repeated and checked for a quick start, early peak flow and sufficient expiration [25]. The presented parameters are: FEV_1 (L), Peak Expiratory Flow (PEF: $L \cdot s^{-1}$) and FEV_1/FVC (%). The predicted values for FEV_1 (FEV_{1pred}) and PEF (PEF_{pred}) and D_{LCO} were derived from reference values for Finnish men [38, 39]. That population-based study examined respiratory function in a Finnish population of 296 male and 257 female white-collar workers and labourers. They were life-long non-smokers and between 18 and 65 years of age, had a normal chest X-ray and had regular health checks with the State Railway Health department. People with a history of chronic lung or cardiac disease were excluded. None of the athletes reported suffering from exercise-induced asthma.

Statistics

Statistical analyses were carried out using SPSS 19.0. Differences between groups were compared with a 2-way ANOVA with age (young vs. old) and sport (control vs. endurance athletes) as factors. Relationships between parameters are given as Pearson correlation coefficients. Differences and correlations were considered significant at $P < 0.05$. Values are reported as means \pm SD.

Results

Participant characteristics

Participant characteristics are shown in **Table 1**. The young were taller than the old participants ($P < 0.001$). Athletes had a lower body mass than the age-matched controls ($P = 0.001$). As a result, the Body Mass Index (BMI) of the young was lower than

	YC N=16	YA N=13	OC N=17	OA N=16
Age (yrs)	26±4 (20–35)	29±4 (24–35)	71±4 ¹ (65–78)	73±5 ¹ (65–79)
Body mass (kg)	77.0±11.5 (59.0–98.0)	73.0±7.8 ² (57.0–83.0)	82.6±12.7 (61.0–112.0)	68.6±7.4 ² (56.0–88.0)
Height (m)	1.79±0.06 (1.68–1.89)	1.80±0.06 (1.71–1.88)	1.74±0.07 ¹ (1.58–1.83)	1.71±0.05 ¹ (1.63–1.79)
BMI (kg·m ⁻²)	24.1±3.0 (20.1–29.9)	22.4±1.7 ² (17.6–23.9)	27.3±3.2 ¹ (22.7–34.2)	23.3±1.9 ^{1,2} (20.1–27.5)
AGP (%)	NA	80±8 (66–90) (n=11)	NA	75±9 (55–89)
Training (hrs wk ⁻¹)	4.5±3.2 (1.0–13.0)	13.2±4.7 ^{2,3} (4.8–21.0)	5.3±3.8 (0.5–14.0)	7.3±3.4 (4.0–14.0)
Age started	NA	13.2±3.5 (5.0–18.0)	NA	33.4±15.2 (10.0–59.0)

Table 1 Participant characteristics.

YC: Young Control; YA: Young Athlete; OC: Old Control; OA: Old athlete; BMI: body mass index; AGP: age-graded performance; NA: not applicable; Mean±SD (range); ¹: Age effect, different from Y; ²: Athlete effect, different from C; ³: different from old. All $P < 0.01$

that of the old participants ($P=0.003$), and in both age groups the BMI of the athletes was lower than that of the age-matched control groups ($P < 0.001$). The AGP did not differ significantly between young and old athletes, despite the observation that the YA trained more hours per week than any of the other groups ($P < 0.001$). In line with this, and as we observed previously [12], the weekly training hours did not correlate significantly with the AGP of the athletes. Although the number of training hours did not differ significantly between the old controls and old master athletes, the intensity of physical activity is very low in non-athletes and not at all comparable to that in athletes. In fact, many of the OC included walking in their training hours, while none of the OA did so.

Diffusion capacity

To be able to correct the D_{LCO} for the [Hb] we measured the [Hb] in the blood. The [Hb] was 6% higher in young than in old participants ($P=0.029$) and 7.5% lower in athletes than age-matched controls ($P=0.002$; **Fig. 1**). The D_{LCO} was lower by ~33% in the old than the young participants ($P < 0.001$) and about 15% higher in athletes than in age-matched controls ($P=0.018$; data not shown). The same applied to the D_{LCO} adjusted for the actual [Hb] (age-effect: $P < 0.001$; athlete vs. non-athlete $P=0.001$; **Fig. 2a**). The predicted D_{LCO} adjusted for [Hb] did not differ significantly between young and old participants, but was higher in athletes than the age-matched controls ($P < 0.001$; **Fig. 2b**). The K_{CO} was ~25% lower in old than young participants ($P < 0.001$; data not shown), but the [Hb] adjusted K_{CO} was ~9% higher in athletes than the age-matched controls ($P=0.006$; **Fig. 2c**). The predicted K_{COpred} adjusted for [Hb] was higher in athletes than in age-matched controls ($P=0.001$), but did not differ significantly between young and old participants (**Fig. 2d**).

The average values for spirometry are shown in **Table 2**. The FEV_1 was significantly lower in old than in young participants, irrespective of being an athlete or not ($P < 0.001$; **Table 2**). This decrease amounted to about 36 mL year⁻¹. Even though the FVC was also 24% lower in the old than the young participants ($P < 0.001$), the FEV_1/FVC was 10.5%-points less in the old than the young, irrespective of being an athlete or not ($P < 0.001$; **Table 2**). The V_A was 12% smaller in the old than the young participants ($P=0.002$), while the RV/V_A was 12%-points higher ($P < 0.001$). Almost all differences were as expected to

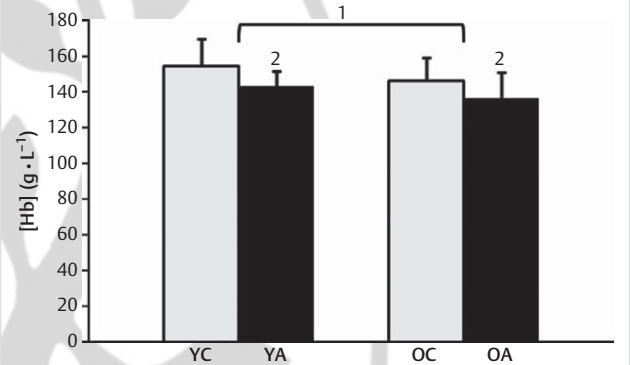


Fig. 1 Haemoglobin concentration [Hb] in the blood of young control (YC), young athletes (YA), old control (OC) and old athletes (OA). 1: Age effect at $P=0.029$; 2: Athlete vs. non-athlete at $P=0.002$.

occur during ageing as reflected by the similar FEV_{1pred} , FVC_{pred} , PEF_{pred} and V_{Apred} in the 4 groups (**Table 2**). The age at which training started (**Table 1**) did not correlate with any of the spirometry parameters (data not shown).

Discussion

Exercise-induced hypoxemia is a common phenomenon in endurance athletes and occurs at lower relative workloads in old than young endurance athletes [13, 14, 32]. Previously we found that the lung function in terms of FEV_1 , FVC and PEF is, if anything, better in master athletes than controls [12]. In this cross-sectional study we observed that also the predicted diffusion capacity of the lung is higher in young and old endurance athletes than age-matched control people. Nevertheless, the difference in diffusion capacity between the young and old men amounted to 33%, in both endurance athletes and controls, and the diffusion capacity of the old endurance athletes was still only 77% of that in the young non-athletes. Exercise-induced reductions in diffusion capacity superimposed on this age-related reduction in resting diffusion capacity [24, 27, 43] may have significant implications for arterial oxygen saturation during exercise and performance. The analysis of world record data suggested that the metabolic power involved in the 10000-m

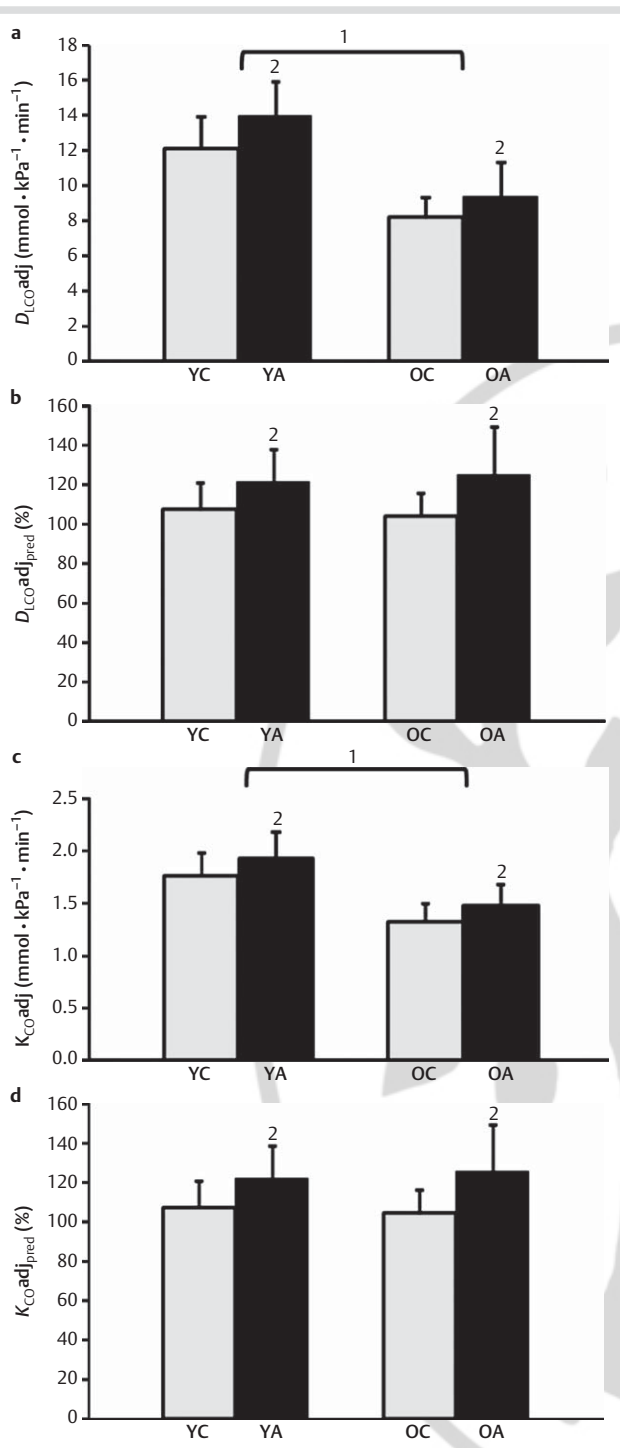


Fig. 2 The diffusion capacity of the lung **a** adjusted for [Hb] (D_{LCOadj} in mmol kPa⁻¹ min⁻¹), **b** predicted D_{LCOadj} ($D_{LCOadjpred}$ in %) **c** D_{LCOadj} for alveolar volume (transfer coefficient K_{COadj}) and **d** predicted K_{COadj} ($K_{COadjpred}$ in %) in young control (YC), young athletes (YA), old control (OC) and old athletes (OA). 1: Age effect at $P < 0.001$; 2: Athlete vs. non-athlete at $P = 0.001$.

run declines from approximately 26 W kg⁻¹ to 20 W kg⁻¹ (23%) between 40 and 75 years [35]. Bearing in mind that there is no great change in running performance between the ages of 20 and 35 [6], it thus seems that the age-related decline in diffusion capacity of the lung is similar to, if not in excess of, the deterioration in aerobic power. It therefore seems likely that the observed

age-related reduction in (resting) lung diffusion capacity does contribute to the reduced metabolic power during endurance events and consequently to the slowing of distance runners at old age [4].

Athlete training and age-graded performance

Both the young and old athletes adhered to a high training volume. In line with previous studies the training volume was lower in old than young endurance athletes, but still considerable [12, 15, 37]. Despite the somewhat lower training volume, the age-graded performance was similar in the young and old athletes, and corresponds with the absence of any correlation between weekly training hours and age-graded performance observed here and in our previous work [12]. The similar age-graded performance in our young and old athletes and that reported for master athletes in previous work [12, 18, 42] indicates that our sample of Master athletes were a 'typical' selection and comparable in age-adjusted athletic achievements with the young athletes. The nature of a cross-sectional study makes it, however, somewhat difficult to discern the impact of athletic training from self-selection, with both the young and old athletes deciding or "selecting" themselves to compete regularly.

Spirometry in Master athletes

Several cross-sectional [3, 28] and longitudinal [30, 31] studies have reported that high physical activity levels attenuate or delay the age-related decline in maximal expiratory flows and lung capacities, further referred to as ventilatory capacity. In our previous cross-sectional work we observed that in absolute terms the age-related decline in FEV₁ and FVC are not attenuated in master athletes [12], but that their better FEV₁ might be due to a higher starting point when young. As a consequence, in relative terms the age-related decline in ventilatory capacity might have been attenuated in master athletes. Here we saw no difference in ventilatory capacity between master athletes and age-matched controls, and in contrast to our expectation the ventilatory capacity of the young athletes was not higher than that of the young controls. Part of the discrepancy could be related to the lower physical activity level of the control group in our previous study than the fairly active controls in the current study. While it is commonly accepted that training in adulthood does not improve ventilatory capacity [13, 14, 23] even after 5 months vigorous endurance training [34], regular swim training at prepubertal age has been shown to improve ventilatory capacity [9]. We did not see a correlation between the age at which training was started and any of the ventilatory parameters. Likewise, no such relationship was evident in elite swimmers [5]. Whatever the cause of the discrepancy between our current and previous study, both studies support the notion that regular training does not improve ventilatory capacity.

While we observed here and previously [12] similar or better ventilatory capacity in athletes than non-athletes, many athletes suffer from exercise-induced asthma or bronchospasm [14] that may contribute to the development of exercise-induced hypoxemia. However, none of our athletes reported suffering from exercise-induced asthma.

Lung diffusion capacity in endurance athletes

We hypothesised that old, like young [26], endurance athletes would have a higher lung diffusion capacity than age-matched controls, but that this difference would be less pronounced at old age. The reason for this suggestion is that exercise can be

	YC N=16	YA N=13	OC N=16	OA N=16
FEV ₁	4.69±0.64 3.36–5.02	4.85±0.84 4.48–5.22	3.14±0.42 ¹ 2.81–3.47	3.24±0.72 ¹ 2.91–3.57
FEV ₁ PRED (%)	97.7±11.4 90.5–104.5	100.0±15.0 92.4–107.6	94.2±12.1 87.3–101.0	98.9±15.9 92.1–105.8
FEV ₁ /FVC	81.5±4.8 78.2–84.8	81.2±4.6 77.5–84.8	72.4±7.4 ¹ 69.1–75.8	69.3±8.5 ¹ 66.0–72.6
PEF	11.6±1.5 10.8–12.3	12.6±1.8 11.8–13.4	10.3±1.4 ¹ 9.5–11.0	10.3±1.5 ¹ 9.5–11.0
PEF _{PRED}	105.1±12.4 98.1–112.2	114.4±16.2 106.6–122.2	107.6±14.0 100.5–114.6	110.2±13.8 103.2–117.2
VC (L)	5.6±0.9 5.2–6.0	5.9±1.1 5.5–6.3	4.3±0.6 ¹ 4.0–4.7	4.4±0.7 ¹ 4.0–4.8
VC _{pred} (%)	99.4±11.4 92.9–105.8	101.5±16.7 94.4–108.7	101.3±0.6 95.0–107.6	106.1±13.1 99.7–112.6
V _A (L)	7.0±1.1 6.4–7.5	7.3±1.2 6.7–7.9	6.2±0.9 ¹ 5.7–6.7	6.4±1.0 ¹ 5.9–6.9
V _{Apred} (%)	91.6±12.2	93.0±13.6	92.1±10.3	97.1±14.1
RV/V _A (%)	22.7±3.1 20.3–25.1	23.6±3.2 21.0–26.3	34.8±4.3 ¹ 32.4–37.2	34.9±7.0 ¹ 32.6–37.3

YC: Young Control; YA: Young Athlete; OC: Old Control; OA: Old athlete; Mean ± SD. FEV₁: Forced Expiratory Volume in 1 s; FEV₁PRED: Predicted FEV₁; FVC/FEV₁: Forced Vital Capacity divided by FEV₁; PEF: Peak Expiratory Flow; PEF_{pred}: Predicted PEF; VC: relaxed vital capacity; VC_{pred}: Predicted relaxed vital capacity; V_A: Alveolar volume; V_{Apred}: Predicted V_A; RV/V_A: Residual volume as percentage of alveolar volume. Lower values are 95% confidence intervals; ¹: Age effect at $P \leq 0.002$

associated with oedema, damage of the blood gas barrier (BGB) and a transient reduction in the diffusion capacity [24,27,41]. To minimize the recurring damage during training sessions the BGB may require a higher tensile strength, which can be realised by thickening of the BGB, as seen in mitral valve disease [41]. Such a thickening of the BGB would reduce the diffusion capacity of the lung. Over the years, the recurring insult on the lung tissue during intense training and competitions may thus cause some reduction in the diffusion capacity that is superimposed on that occurring during normal ageing. In line with this, an inadequate diffusion capacity has been put forward as a potential explanation for the exercise-induced hypoxaemia in, particularly older, endurance athletes [13,14,22,27,32,40]. However, we observed an 11%-point higher, rather than lower, diffusion capacity of the lung in athletes than non-athletes, without any indication that the difference in diffusion capacity was less pronounced in old age.

It could be that the higher diffusion capacity in athletes is the consequence of a self-selection bias. In support of this it was shown in elite swimmers that the enhanced diffusion capacity could be explained by the larger lungs of the elite swimmers and not by a higher transfer coefficient (K_{CO}) [5]. It is thus possible that the higher diffusion capacity in our athletes is an adaptation to training. The absence of any change in lung diffusion capacity after a vigorous 5-month endurance training programme that resulted in a lower heart rate at given absolute workloads [34] does, however, argue against a training adaptation.

There are several factors that at least in theory can contribute to a better diffusion capacity in the endurance athletes, such as a higher [Hb], longer erythrocyte transit time, fewer arteriovenous shunts in the lung, a thinner BGB, larger gas exchange surface area and a better ventilation-perfusion matching. The contribution of shunts to exercise-induced hypoxaemia is negligible, amounting to 0.5% of cardiac output [40], similar to the 0.6% we measured with microspheres in rabbits [11]. It is unlikely that the erythrocyte transit time will limit the diffusion capacity even during exercise [45].

An increase in the [Hb] in the blood was one of the factors that improved lung diffusion capacity at high altitude [2]. Here we show a lower, rather than a higher, [Hb] in athletes than non-athletes, a phenomenon regularly observed and dubbed 'athlete's anaemia' [15,36]. Adjustment for the [Hb] accentuates the difference between athletes and non-athletes. In theory a decrease in the 2,3-diphosphoglycerate (2,3-DPG) concentration would increase the Hb-affinity for oxygen and carbon monoxide and thus result in an increase in the measured diffusion capacity of the lung. However, endurance training induces an increase rather than a decrease in the [2,3-DPG] [7] and hence the lower [Hb] in our study or a shift in the Hb-dissociation curve does not explain the better diffusion capacity in endurance athletes. In fact, acute hypervolemia resulted in improved arterial oxygen pressure in athletes who developed exercise-induced hypoxaemia [44]. The lower [Hb] in the athletes may thus be a consequence of plasma expansion [36] and help to improve the arterial oxygenation.

Another possibility is an increase in alveolar volume and/or lung capillary blood volume which would increase the surface area for gas exchange. While an increase in alveolar volume, but not blood volume, has been seen in response to high altitude [2], we saw no difference in alveolar volume between endurance athletes and non-athletes. Although we did not measure the lung capillary blood volume, it has been shown that endothelin A blockade reduces pulmonary vascular resistance and improves the diffusion capacity [10]. Lower circulating endothelin-1 levels in endurance athletes [29] may thus, via reduction in pulmonary vascular resistance, contribute to the higher diffusion capacity in the endurance athletes. A ventilation-perfusion mismatch is one of the most important contributors to diffusion limitation during exercise [17], and one might speculate that the reduced pulmonary vascular resistance may result in an improved ventilation-perfusion match at rest, and in this way improve the diffusion capacity of the lung of the endurance athlete. While differences in alveolar volume do not seem to contribute to the higher diffusion capacity in endurance athletes than non-

Table 2 Ventilatory characteristics in athletes and controls.

athletes, data in the literature [44] lead to the speculation that it might be due to a larger capillary blood volume and better ventilation-perfusion matching in the endurance athlete, at least at rest.

Ageing

Although the diffusion capacity of the lung was better in young and old endurance athletes than the age-matched controls, the age-related difference in both groups amounted to about 33%. This far exceeded the 11% difference between athletes and non-athletes. In **Fig. 2a** it can be seen that the diffusion capacity of the old endurance athletes was only 77% of that in the young controls. It thus appears that regular training does not attenuate the often described age-related decline in diffusion capacity of the lung [19, 39]. The lower diffusion capacity in old than young athletes, which is further reduced during exercise, may hamper the oxygenation of the blood during endurance exercise at high workloads and hence reduce the ability to perform work requiring aerobic energy metabolism. This would then contribute to the observed lower (aerobic) metabolic power of old than young endurance athletes [35] and contribute to the slowing of the endurance athlete during the ageing process [4]. Since a reduced diffusion capacity contributes significantly to exercise-induced hypoxaemia [13, 14, 27, 32], it is likely that in the absence of a better diffusion capacity in the older athlete than non-athletes the exercise-induced arterial hypoxaemia would have been even more pronounced in the endurance athlete.

Limitations

Although we did not determine whether exercise-induced hypoxaemia occurred in our athletes, it does occur in more than 50% of endurance athletes and increases with age [27]. In one study, for instance, exercise-induced hypoxaemia was experienced by all of the old endurance athletes, but only 8 out of the 10 young athletes [32]. We therefore can assume that a large proportion of the old athletes in the present study would have developed exercise-induced hypoxaemia, despite a better lung diffusion capacity than age-matched controls.

Another limitation of this study is the cross-sectional design that does not allow us to make firm conclusions on whether the better diffusion capacity in endurance athletes is due to their intense training or results from a self-selection bias. Although we have shown here that the diffusion capacity at rest is better in endurance athletes than in a control population, it remains to be seen how the diffusion capacity during exercise in endurance athletes compares to that in exercising non-athletes. Redistribution of blood resulting in a ventilation perfusion miss-match during exercise is considered to be the most important contributor to hypoxaemia during exercise [17], and both ventilation perfusion miss-match and hypoxaemia are reduced by volume expansion [44]. Nevertheless, we have shown here that at least at rest the diffusion capacity and transfer coefficient of the lung are better, rather than poorer, in both young and older endurance athletes than age-matched non-athletes.

Conclusion

Many endurance athletes develop exercise-induced hypoxaemia, which has been suggested to be related to an inadequate diffusion capacity of the lung. The present work shows, however, that both young and old endurance athletes have a better, rather than a lower, lung diffusion capacity and gas transfer coefficient at rest than age-matched controls. We suggest that despite this

benefit, the stark reduction in diffusion capacity with age, which has been reported to reduce even further with exercise, may contribute to the slowing of the older endurance athlete.

Acknowledgements

▼ We appreciate the financial support from the Finnish Academy, Finnish Ministry of Education and Culture, and the Erasmus Exchange Programme of the EU. We are grateful for the instructions and support from Dietmar Herbst and Tommi Seikkula. The help in the recruitment of participants by Eeva Aartolahti, the determination of haemoglobin levels and translation for participants who did not speak English by Matias Rihto and Marianne Suonpää was indispensable. We thank Vesa Lappalainen and the organisers of the WMA 2012 Indoor Championships in Jyväskylä for their support of the study. We owe a big thank you to the participants – without their contribution this study would not have been possible.

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