Exercise-induced hypoxaemia in master athletes: effects of a polyunsaturated fatty acid diet

Abstract  Exercise-induced hypoxaemia (EIH) has been associated with an oxygen diffusion limitation. Because polyunsaturated fatty acids (PUFA) administration can modify cell membrane fluidity, we hypothesized that the importance of EIH could be reduced after a 6-week PUFA diet. Resting pulmonary functions and a maximal cycling test were performed before and after the diet, in eight master athletes [48 (SD 6 years)]. The partial pressure of O2 in arterial blood (P\textsubscript{a}O\textsubscript{2}), alveolar ventilation (VA) and ideal alveolar-arterial oxygen partial pressure difference (P\textsubscript{(A-a)}O\textsubscript{2}) were obtained at each exercise intensity. The extent of EIH at maximal exercise was significantly lower after PUFA [-17.2 (SEM 1.9) vs -12.9 (SEM 2.2)]. Before PUFA, VA accounted for 50% of the variance in the fall in P\textsubscript{(A-a)}O\textsubscript{2} for intensities below 80% maximal oxygen uptake (VO\textsubscript{2}max) and P\textsubscript{(A-a)}O\textsubscript{2} for 60% between 70% and 100% VO\textsubscript{2}max. After PUFA, the reduction in EIH was highly correlated (r\textsuperscript{2} = 0.85; P<0.001) to resulting changes in P\textsubscript{(A-a)}O\textsubscript{2} and resting pulmonary diffusing capacity (DL\textsubscript{co}/VA) but not with changes in ideal alveolar partial pressure of oxygen. The improvement in EIH following PUFA could be related to an increase in alveolar-arterial oxygen conductance following improved pulmonary diffusion.

Key words  Alveolar-arterial difference in oxygen · Exercise test · Driving component of ventilation · Cell membrane fluidity

Introduction

The mechanisms of exercise-induced arterial hypoxaemia (EIH) are still not clearly understood. Ventilation-perfusion heterogeneity (Wagner et al. 1986, 1989), relative hypoventilation (Johnson and Dempsey 1991; Johnson et al. 1992; Miyachi and Tabata 1992; Powers et al. 1993) and more recently diffusion limitations for pulmonary exchange (Manier et al. 1991, 1993; Schaffartzik et al. 1992, 1993) have been the most commonly proposed explanations. A diffusion limitation may be due to an incomplete equilibration of O2 between alveolar gas and pulmonary capillary blood following a markedly shortened mean pulmonary capillary transit time or to a potential alteration in the integrity of the alveolo-capillary membrane or a combination of both. Recent evidence obtained both in animals (Schaffartzik et al. 1993; Wagner et al. 1989) and in humans (Manier et al. 1991, 1993; Schaffartzik et al. 1992) has suggested an accumulation of pulmonary extravascular water during heavy exercise potentially altering membrane diffusion and or distribution of ventilation.

In addition, it has appeared that increasing age could potentiate the occurrence of EIH (Johnson and Dempsey 1991; Prefaut et al. 1994). In master endurance athletes an exaggerated widening of the alveolo-arterial oxygen pressure gradient and hypoxaemia has been reported during both submaximal and maximal exercise. Changes with age in the mechanical properties of breathing mechanics or of pulmonary diffusing capacity are all possible explanations for this observation. On the other hand, alterations in membrane properties of the red blood cell especially or general cell membrane transport functions have been reported with increasing age (Kinsella 1990; Levin et al. 1992; Tsuda and Masuyama 1990) as well as physical training (Sumi-
kawa et al. 1993) which could contribute to the disturbance in gas exchange. This hypothesis however has not been considered in relation to exercise-induced hypoxaemia. Changes in cell membrane fluidity may be related to the membrane content of polyunsaturated fatty acids (PUFA) which it has been found can be modified by dietary lipid manipulations (Biagi et al. 1991; Kinsella 1990). The beneficial bloodrheological effects of PUFA supplementation have been well demonstrated for several years (Debry and Pelletier 1991; Kinsella 1990) and improvements in red cell deformability and bloodrheological responses to physical exercise have been reported following administration of a PUFA enriched diet (Guezennee et al. 1989).

The present study was designed to examine whether a sustained dietary PUFA supplementation fed to master endurance athletes would reduce the extent of exercise-induced hypoxaemia.

Methods

Subjects

A group of 27 endurance-trained master athletes completed the initial progressive maximal exercise test on a cycle ergometer. Exercise-induced hypoxaemia was found in all the athletes. Reproducibility of exercise-induced hypoxaemia was confirmed through a series of three subsequent tests in 12 of these athletes. From this group, 8 athletes aged 48.1 (SEM 5.6) years (30–71), height 171 (SEM 3.2) cm, body mass 66 (SEM 2.9) kg rigorously selected for strict adherence to their training or dietary regime as well as for the day-to-day exercise response reproducibility were invited to take part in the study. All the subjects were nonsmokers and had been training regularly for 19 (SEM 3) years (range 10–32 years).

At the time of the study, all the subjects had been following a regular endurance training programme [10 (SEM 1) h x week⁻¹] for at least 5 months. A signed, informed consent was obtained from all the participants and ethics approval was received.

Experimental design and procedures

The study was designed as a test-retest protocol, with each subject acting as his own control. A maximal incremental cycling exercise was performed before (control, c) and after a 6-week period of borage oil supplementation with 3.66 g x day⁻¹ of n-6 PUFA containing 62% of gamma linolenic acids (C18:3 n-6). Borage oil (Vivis, Dijon, France) was administered in the form of 12 capsules where PaCO₂ is partial CO₂ pressure in arterial blood.

The 0₂, expired CO₂ (CO₂) and ventilation (Vₐ) as well as tidal volume (V½), breathing frequency (fₐ), R, and end tidal partial pressure of O₂ and CO₂ (PₐO₂, PₐCO₂) were measured continuously throughout the exercise protocol using a breath-by-breath automated exercise metabolic system (CPX, Medical Graphics, St. Paul, Minn.). Average values were obtained over the last 30 s of each exercise intensity simultaneously with arterial blood sampling.

Oxygen haemoglobin saturation was monitored continuously using an ear oximeter (Radiometer, Copenhagen, Denmark) and was also calculated from partial pressure of oxygen in arterial blood PₐO₂ and pH determinations. Ideal alveolar-arterial oxygen partial pressure difference (P(A-a)O₂) was calculated according to the standard equation (Anthonisen and Fleetham 1987): $P_{a}O_2 = (P_{F}O_2 - P_{CO}_2) \cdot (0.2093 + (0.79/R))$; where $P_{F}O_2$ is oxygen fraction in inspired gas, $P_{a}CO_2$ is barometric pressure, and $P_{CO}_2$ is partial CO₂ pressure in arterial blood.

Alveolar ventilation ($V_a$), was also calculated as $VCO_2 \times 0.863/P_{CO}_2$; where $P_{CO}_2$ is partial CO₂ pressure in arterial blood.

For each subject a plot of the dependent variable with respect to $V_O_2$ was generated and the mathematical model of best fit for the curve was used to calculate the value corresponding to each percentage of $V_{O2_{max}}$ from 30% to 100%.

Blood analyses

Blood gases and lactate concentration

Blood samples for arterial blood gases were quickly analysed for $P_{O}_2$, $P_{CO}_2$ and pH at 37°C using the appropriate electrode (Radiometer, Copenhagen, Denmark). The instrument was calibrated before and several times during the course of blood analysis using precision buffers and gases. All $P_{O}_2$ and $P_{CO}_2$ were analysed twice or until two $P_{O}_2$ and $P_{CO}_2$ readings were within 0.27 and 0.2 mmHg respectively. Blood lactate concentration determination was carried out using a lactate analyser (MICRO-ZYM, SGI, Toulouse, France).