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ORIGINAL ARTICLE

Apostolos Danis · Yannis Kyriazis · Vassilis Klissouras

The effect of training in male prepubertal and pubertal monozygotic twins

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Abstract Nine male pairs of monozygotic twins aged 11–14 years, height 147 (7.6) cm and body mass 39.7 (9.6) kg, participated in this study. Twin zygocity was tested using morphological, dermatoglyphic and hematologic methods, and Tanner's five stages were used for the evaluation of biological maturation. One twin from each pair undertook training for 6 months, three times a week, with running at 85–120% of the lactate anaerobic threshold (LT). Anthropometrics, determination of maximum O_2 uptake ($\dot{V}O_{2max}$), LT and maximal blood lactate concentration ($[La]_{max}$) was carried out before, during and after training. No significant difference existed between the trained twins and their untrained brothers before training. After training, the trained twins increased their $\dot{V}O_{2max}$ (per kg body mass) by 10.6% and their LT by 18.2% ($P < 0.01$), reaching values that differed significantly from those of their untrained brothers [57.5 (3.6) $ml \cdot kg^{-1} \cdot min^{-1}$ vs 55.4 (3.3) $ml \cdot kg^{-1} \cdot min^{-1}$ and 13.4 (1.1) $km \cdot h^{-1}$ vs 12.7 (1.1) $km \cdot h^{-1}$, respectively]. In addition, in the trained twins relative body fat was reduced ($P < 0.05$) from 17.8 to 16.2% and their somatotype altered significantly (decrease of endomorphy and mesomorphy and increase of ectomorphy), while in the untrained twins there was no change in these parameters. Both groups of twins significantly increased their absolute $\dot{V}O_{2max}$ after the 6 months of training, the trained by 14,9% [from 2.08 (0.43) to 2.37 (0.45) $l \cdot min^{-1}$] and the untrained by 10.5% [from 2.10 (0.41) to 2.32 (0.47) $l \cdot min^{-1}$], but no difference was registered between them. A comparison of the intrapair changes in $\dot{V}O_{2max}$ of prepubertal and pubertal twins showed an influence of training in the prepubertal (19.3% vs 5.2%) but not in the pubertal twins (12.7% vs 13.1%). Using analysis of variance, the relative importance of training, heredity and their interaction was evaluated to be 20%, 70% and 10%, respectively, for the change in body fat, 35%, 45% and 20%, respectively, for the change in relative $\dot{V}O_{2max}$ and 25–30%, 50–60% and 15–20%, respectively, for the change in LT. In conclusion, training during pubertal growth can favour aerobic power (depending on body composition) as well as aerobic capacity, but it has no effect on absolute $\dot{V}O_{2max}$. Genetic control seems to have a strong effect on the extent of adaptations, and the genotype—training interaction explains a small, but prominent part of them.

Keywords Maximal aerobic power · Lactate anaerobic threshold · Body composition · Sport genetics · Genotype—training interaction

A. Danis (✉)
Kap. Vangeli 16, 57200 Assiros Thessaloniki, Greece
E-mail: apostolosdanis@yahoo.gr
Fax: +30-3940-61019

V. Klissouras · A. Danis · Y. Kyriazis
Ergophysiology Laboratory,
Department of Physical Education and Sports Science,
University of Athens, Athens, Greece

Introduction

The idea that maximal O_2 uptake ($\dot{V}O_{2max}$) can be improved by training before and during puberty in humans has been investigated in many studies. Some of these studies (Bar-Or and Zwiren 1973; Schmuecker and Hollmann 1973; Kobayashi et al. 1978; Yoshida et al. 1980; Mirwald et al. 1981) did not reveal any training-induced improvement before the onset of puberty, although others (Brown et al. 1972; Weber et al. 1976; Lussier and Buskirk 1977; Vaccaro and Clarke 1978; Gerhardus 1980; Petratis et al. 1983; Rotstein et al. 1986; Mahon and Vaccaro 1994) revealed absolutely the opposite. Similarly, Ekblom (1969), Eriksson (1972), Massicotte and MacNab (1974), Kobayashi et al. (1978), Mirwald et al. (1981) and Mahon and Vaccaro (1989) observed a positive effect of training during puberty, while Daniels and Oldridge (1971), Parizkova and Spynarova (1975), Stewart and Gutin (1976) and Weber et al. (1976) did not find any influence of training. A possible cause of these contradictory findings is the

great increase in $\dot{V}O_{2max}$ observed during pubertal growth, which follows the stage of peak height velocity (Kobayashi et al. 1978; Kemper and Verschuur 1981; Mirwald et al. 1981; Krahenbuhl et al. 1985; Paterson et al. 1986), which is complicated by the deviation between the subjects' chronological and biological age. If groups of the same biological maturation are not compared it is not easy to isolate the training effect from the differentiation caused by growth, especially during the stage of peak growth velocity.

The anaerobic threshold is more sensitive to training than is $\dot{V}O_{2max}$ (Mader et al. 1976; Davis et al. 1979; Denis et al. 1982; Ready and Quinney 1982; Denis et al. 1984). There are few studies of the influence of training during the prepubertal and pubertal stage (Gerhardus 1980; Becker and Vaccaro 1983; Rotstein et al. 1986; Mahon and Vaccaro 1989), but a generally positive effect has been reported.

The maximal blood lactate concentration ($[La]_{max}$) is low in children (Åstrand 1952; Eriksson et al. 1971; Kindermann et al. 1975), probably because of the low activity of the enzyme phosphofructokinase (Eriksson 1972). A significant increase of $[La]_{max}$ in children before and during puberty was observed after training by Eriksson et al. (1973) and Massicote and MacNab (1974), while no change was found by Ekblom (1969) and Weber et al. (1976).

The purpose of the study presented here was to investigate the effect of training just before (prepuberty) and during puberty with a more accurate methodology. The co-twin method was selected, with the following advantages: (1) the same homogeneity in the experimental and control groups, (2) very small differences in the degree of biological maturation between experimental and control subjects and (3) genetic similarity in growth of the experimental and control subjects (Fischbein 1997a, 1997b; Sklad 1997). With regard to the differentiation between the experimental and control twins, "a greater or smaller change on the variables rather than the change of growth" has been defined as an effect of training. If an effect of training were found, it would be interesting to analyse the relative contribution of heredity, training and their interaction.

Methods

Subjects

Nine male monozygotic twin-pairs, 11–14 years old, participated in this study. None of the subjects had participated systematically in sporting activities. They had the same way of life and physical habits and were healthy, without any serious disease in their case history. Their zygocity was established on the basis of morphological and dermatoglyphic similarity, as well as by the identity in red blood cell antigens using the following antibodies: anti-A, -B, -AB, -M, -N, -S, -s, -P, -D, -C, -E, -c, -e, -K, -k, -Fy^a, -Fy^b, -Jk^a, -Jk^b, -Le^a, Le^b, -Lu^a, -Lu^b. When this blood examination was completed, the probability of error in the diagnosis of zygocity was lower than 4% according to Wilson (1980). The biological maturation of the twins was established using Tanner's the five stages of penile and testicular development (Tanner 1962). Three of the twin-pairs were at stage 1 (prepuberty) and the rest were at stages 2–5 (puberty).

Measurements

Measurements, including anthropometry, spirometry and determination of blood lactate concentration ([La]), took place before training, after 3 months of training and again after 6 months of training. An electrocardiogram was taken at rest, followed by a medical examination before any further measurement. There was no case in which any abnormality was found that would be a contraindication for the twins' participation in physical activities.

Anthropometry included measurements of height, body mass, five skinfolds (biceps, triceps, suprailiac, subscapular and calf), two circumferences (flexed arm and calf) and two biepycondilar diameters (humerus and femur). Body fat and lean body mass (LBM) were estimated according to the formula of Durning and Rahaman (1967) for male subjects aged 12.7–15.7 years, using four skinfolds (biceps, triceps, suprailiac and subscapular). Skinfold thickness was measured with a Harpenden calliper. In addition, height, body mass, the two circumferences, the two biepycondilar diameters and four skinfolds (triceps, suprailiac, subscapular and calf) were used to estimate the somatotype (Heath and Carter 1967).

The exercise test protocol included a gradually increasing workload on the treadmill (Quinton, model 644), starting from 8 $km \cdot h^{-1}$and increasing by 2 $km \cdot h^{-1}$ every 4 min to exhaustion. A familiarisation of about 5 min with the running on the treadmill proceeded. O_2 uptake ($\dot{V}O_2$) and heart rate (HR) were measured continuously during the test, while samples of blood from the ear lobe were taken during the last 30 s of every load level (running was interrupted). When the subjects approached exhaustion (respiratory exchange ratio >1.10, HR >200 beats$\cdot min^{-1}$) and were at the end of a work level, the increase in running speed for the next level followed without interruption (to take blood) in order to obtain $\dot{V}O_{2max}$. The gradient of the treadmill was set at a constant 1% during the test for all of the subjects, to compensate for the mean wind resistance of running in the field (Heck et al. 1985).

On a 2nd day, a field test of 300 m running with the simultaneous participation of both twin brothers took place to determine the $[La]_{max}$ (peak). The simultaneous competitive participation of both twins ensured a maximal effort. Blood samples were taken before and after the running at the 1st, 3rd, 5th, 7th, 10th, 14th and 20th min of recovery.

$\dot{V}O_{2max}$ was determined with an automatic open-circuit ergospirometer (MMC Horizon, Sensormedics) as the highest value of $\dot{V}O_2$ that was recorded by the exercise test at exhaustion. Respiratory variables respiratory frequency, pulmonary ventilation, $\dot{V}O_2$ and CO_2 production, as well as the HR (determined using a Philips ECG, AR 100 and MM 210 attached to an ergospirometer) were monitored continuously and recorded every 30 s. Gas calibration (with 16% O_2, 4% CO_2 and 80% N_2) was performed before each measurement. A gas check also followed at the end of each measurement.

Lactate levels were determined with a spectrophotometer (Beckman, model 42) at 365 nm using a modification of the enzymatic method of Boehringer Mannheim (256773; semimicro method). A 20-μl sample of capillary blood was taken from the ear lobe for each determination and emptied into 200 μl of perchloric acid (0.6 N). All samples were centrifuged for 2 min at 12,000 rev$\cdot min^{-1}$ and preserved at $+4^{\circ}C$ for a maximum of 7 days. A double determination was performed for each sample.

LT has been valuated using the variables: running speed, $\dot{V}O_2$ relative to body mass (i.e. $\dot{V}O_2$ per kg body mass; $\dot{V}O_2 \cdot kg^{-1}$), percentage of $\dot{V}O_{2max}$ and HR at the concentration of 4 mmol$\cdot l^{-1}$ [La] (Mader et al. 1976; Sjodin and Jacobs 1981). The estimation of these four variables was accomplished with a linear interpolation between the nearest low and high [La] to 4 mmol$\cdot l^{-1}$, obtained during the incremental exercise test.

Training

One twin from each pair (chosen randomly) participated in the training, while the other was the control twin. Training included running at an intensity of 85–120% of the running speed at LT (75–97% of the $\dot{V}O_{2max}$). Each training session lasted 1–1.5 h and took place three times a week for a total period of 6 months. In the first 3 months the aim of two of those training sessions per week was to improve LT (continuous and interval running at 85–100% of the running speed at LT), whereas the third was to improve the $\dot{V}O_{2max}$ (interval running at 100–120% of the running speed at LT). In the last 3 months, the aim of two of the weekly training sessions was to improve the $\dot{V}O_{2max}$, and only one training session per week was used to improve LT. The exercise intensity was calculated individually and adjusted to the new values of LT by tests after 1.5 and 3 months of training. Training was also set on an individual basis. Both twin-brothers attended the physical education course at school (2–3 times a week). This physical activity has been accepted as a part of the natural motor development of the children. The physical activity of the untrained twins was controlled every week through a questionnaire, and no more physical activity was found to have taken place.

Statistics

Two-way analysis of variance (ANOVA, repeated measures on one factor) was used for the statistical analysis. The two groups (trained and untrained twins) and the three time points of measurements (0, 3 and 6 months of training) were defined as factors 1 and 2, respectively. At significance level $P \leq 0.05$ for factor 2 and/or for the interaction (factor 1 × factor 2), a multiple comparison of mean values followed using the Student-Newman-Keuls-test. If a training effect was ascertained, the relative importance of training, heredity and their interaction was examined using a one-way ANOVA (percentage changes of twin brothers as correlated data). The within-pair variance has been explained as the variance resulting from the influence of training, the between-pair variance as the variance caused by hereditary differences, and the interaction (including the error variance) as the interaction between heredity and training. The intrapair concordance of twins was examined by the intraclass correlation [$R = (B-W)/(B+W)$], where B and W are the between-pair and within-pair variance, respectively (see Falconer 1989).

Results

Before training, no significant differences in any variable were observed between twins of the experimental and the control group. Table 1 shows the anthropometric characteristics of the trained and untrained twins after 3 and after 6 months of training. The two-way ANOVA for these variables gave significant F values ($P \leq 0.05$) only for factor 2 (0, 3 and 6 months of training). Both groups presented a significant increase ($P < 0.001$) in height, body mass and LBM during the 6 months, without any significant difference between them. Body fat exhibited a significant decrease in trained twins ($P < 0.01$), as did the sum of five skinfolds ($P < 0.05$), while no change was observed in their untrained brothers. Similarly, a significant change was noted in the somatotype of only the trained twins, with a decrease in endomorphy ($P < 0.01$), a decrease in mesomorphy ($P < 0.05$) and an increase in ectomorphy ($P < 0.01$).

Table 2 shows the F values of the two-way ANOVA for the variables at maximal effort and LT, and Table 3 gives the mean values for the two groups. The absolute $\dot{V}O_{2max}$ had already increased significantly in the trained twins after 1.5 months of training [6.1 (3.0)%, $P < 0.05$], while a significant increase in their untrained brothers was observed only after 6 months [10.5 (6.6)%, $P < 0.01$]. The mean increase after 6 months of training in the trained twins was 14.9 (7.8)% ($P < 0.01$). Although after 3 months of training the trained twins, but not the untrained brothers, presented a significant increase [7.9 (4.0)%, $P < 0.01$], after 6 months both groups showed a significant increase and did not differ, either in their mean $\dot{V}O_{2max}$ values or in their mean changes (Fig. 1).

The relative $\dot{V}O_{2max} \cdot kg^{-1}$ and per kg LBM ($\dot{V}O_{2max} \cdot kg^{-1}$LBM) had already increased in the trained twins after 1.5 months of training ($P < 0.05$), reaching a

Table 1 Anthropometric characteristics of the twins in the experimental (*E*) and control (*C*) group before (*Pre*), during (*3rd month*) and after (*Post*) training. (*LBM* Lean body mass)

Variable	Pre		3rd month		Post	
	Group	Mean (SD)	Mean (SD)	*P*	Mean (SD)	*P*
Height (cm)	E	147.7 (7.6)	149.7 (8.2)	*	151.9 (8.3)	*
	C	147.4 (8.0)	149.6 (8.6)	*	151.7 (8.9)	*
Bodymass (kg)	E	40.2 (10.4)	41.4 (10.1)	*	41.8 (10.1)	*
	C	39.2 (9.3)	41.2 (9.9)	**	42.2 (10.1)	**
LBM (kg)	E	32.8 (7.3)	34.0 (7.4)	**	34.9 (7.8)	**
	C	32.5 (7.1)	34.1 (7.7)	**	35.1 (7.9)	**
Sum of five skinfolds (mm)	E	44.5 (15.6)	43.7 (16.5)	ns	39.9 (12.7)	*
	C	39.9 (9.8)	40.8 (11.4)	ns	40.6 (10.4)	ns
Fat (%)	E	17.8 (4.1)	17.4 (4.2)	ns	16.2 (3.7)	*
	C	16.8 (2.8)	16.9 (3.2)	ns	16.6 (2.8)	ns
Somatotype						
Endomorphy	E	2.66 (1.05)	2.55 (1.04)	ns	2.23 (0.83)	**
	C	2.34 (0.64)	2.38 (0.72)	ns	2.28 (0.62)	ns
Mesomorphy	E	4.79 (1.11)	4.71 (1.08)	ns	4.61 (1.03)	*
	C	4.78 (0.98)	4.75 (0.98)	ns	4.69 (0.94)	ns
Ectomorphy	E	3.31 (1.27)	3.37 (1.13)	ns	3.74 (1.09)	**
	C	3.44 (0.98)	3.41 (0.93)	ns	3.59 (0.98)	ns

*$P < 0.05$, **$P < 0.01$, ns $P > 0.05$

Table 2 F values [factor 1 (F_1: trained, untrained twins), factor 2 (F_2: 0, 3 and 6 months oftraining) and factor 3 (F_3: interaction F_1xF_2)] in the two-way analysis of variance. ($\dot{V}_E$ Minute ventilation, $\dot{V}O_2$ oxygen uptake, $\dot{V}O_{2max}$ maximum oxygen uptake, $[La]_{max}$ Maximum lactate concentration, *HR* heart rate)

Variables	F_1	*P*	F_2	*P*	F_3	*P*
$\dot{V}_E$ (l·min^{-1})	0.002	ns	26.64	***	1.562	ns
$\dot{V}O_{2max}$ (l·min^{-1})	0.019	ns	53.49	***	1.583	ns
$\dot{V}O_{2max}$ (ml·kg^{-1}·min^{-1})	0.096	ns	22.87	***	7.443	**
$\dot{V}O_{2max}$ (ml·kg LBM·min^{-1})	0.612	ns	14.80	***	4.493	*
HR_{max} (beats·min^{-1})	0.293	ns	15.69	***	1.457	ns
Lactatethreshold of 4 mmol·l^{-1}						
Running speed (km·h^{-1})	0.316	ns	25.89	***	5.507	**
$\dot{V}O_2$ (ml·kg^{-1}·min^{-1})	0.104	ns	3.41	*	2.344	ns
Percentage of $\dot{V}O_{2max}$ (%)	0.325	ns	1.22	ns	0.588	ns
HR (beats·min^{-1})	0.003	ns	1.00	ns	0.007	ns
Running 300 m						
$[La]_{max}$ (mmol·l^{-1})	0.396	ns	4.59	*	0.268	ns
Time (s)	0.024	ns	38.31	***	3.340	ns

*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$, ns $P > 0.05$

Table 3 Mean values and standard deviation of the parameters at maximal effort and at lactate anaerobic threshold of 4 mmol·l^{-1}, before, during and after training in the experimental (E) and control (C) group

Variables	Group	Pre	3rd month		Post	
		Mean (SD)	Mean (SD)	*P*	Mean (SD)	*P*
$\dot{V}_E$ (l·min^{-1})	E	77.16 (16.37)	83.61 (15.35)	*	88.63 (18.36)	**
	C	78.52 (15.36)	85.63 (18.23)	**	86.23 (18.61)	**
$\dot{V}O_{2max}$ (l·min^{-1})	E	2.076 (0.434)	2.234 (0.451)	**	2.369 (0.446)	**
	C	2.098 (0.411)	2.178 (0.425)	ns	2.317 (0.473)	**
$\dot{V}O_{2max}$ (ml·kg^{-1}·min^{-1})	E	52.07 (3.56)	53.94 (3.78)	*	57.49 (3.63)	**
	C	54.02 (3.85)	52.61 (3.79)	ns	55.38 (3.27)	ns
$\dot{V}O_{2max}$ (ml·kg LBM^{-1}·min^{-1})	E	63.30 (3.20)	65.38 (2.84)	*	68.87 (2.96)	**
	C	64.88 (3.75)	63.37 (3.07)	ns	66.47 (2.85)	ns
HR_{max} (beats·min^{-1})	E	205.3 (9.7)	199.0 (8.4)	**	200.2 (9.4)	**
	C	206.1 (11.3)	203.0 (8.1)	**	202.4 (9.5)	**
Lactate threshold of 4 mmol·l^{-1}						
Running speed (km·h^{-1})	E	11.39 (1.32)	13.00 (1.14)	**	13.37 (1.09)	**
	C	11.90 (0.93)	12.43 (0.90)	ns	12.67 (1.06)	ns
$\dot{V}O_2$ (ml·kg^{-1}·min^{-1})	E	45.48 (3.69)	46.84 (4.59)	ns	49.28 (4.03)	**
	C	47.14 (3.71)	45.64 (3.99)	ns	47.27 (4.30)	ns
Percentage of $\dot{V}O_{2max}$ (%)	E	87.4 (4.5)	86.8 (4.9)	ns	85.7 (4.0)	ns
	C	87.3 (4.4)	86.7 (4.3)	ns	85.4 (6.4)	ns
HR (beats·min^{-1})	E	189.1 (7.8)	186.4 (6.8)	ns	185.9 (5.7)	ns
	C	189.4 (11.1)	190.1 (9.1)	ns	187.9 (8.6)	ns
Running 300 m						
$[La]_{max}$ (mmol·l^{-1})	E	9.4 (2.0)	8.8 (1.6)	ns	9.4 (1.6)	ns
	C	9.2 (1.0)	8.2 (0.9)	ns	9.0 (1.6)	ns
Time (s)	E	62.4 (6.8)	58.6 (6.0)	**	55.0 (6.5)	**
	C	60.8 (5.8)	60.1 (6.0)	ns	56.3 (5.0)	**

*$P < 0.05$, **$P < 0.01$, ns $P > 0.05$

10.6 (5.6)% and 9.0 (6.1)% improvement, respectively ($P < 0.01$), after 6 months of training. On the contrary, the untrained twins revealed no change. After 6 months of training there was a significant difference both in the mean changes and in the mean values of the relative $\dot{V}O_{2max}$ between the two groups (Fig. 1).

A significant improvement was observed in the trained twins in running speed at LT of 4 mmol·l^{-1} after 6 months of training [18.2 (10.1)%, $P < 0.01$], an effect that was already in evidence after 1.5 months of training [13.8 (9.5)%, $P < 0.05$]. Control twins showed no significant change during the 6 months of training (Fig. 2). After training, the trained twins demonstrated a significantly higher running speed at LT ($P < 0.05$) than their untrained brothers (Table 3). In addition, a greater transposition of the lactate—running speed curve to the right side was observed in the trained twins compared to their brothers. $\dot{V}O_2$ at LT increased significantly in the trained twins after 6 months of training [8.7 (8.6)%, $P < 0.01$], while no change took place in control twins (Fig. 2). No change was found in the percentage of $\dot{V}O_{2max}$ at LT or in HR at LT during the 6 months of training, in either the trained or in untrained twins.

$[La]_{max}$ in the 300-m run showed no significant change after 3 and after 6 months of training in any of the two groups (Table 3). Neither was there any alteration in the rate of blood lactate disappearance after the 300-m run in the trained or untrained twins after the 6-month training programme.

Table 4 shows the variance of training (within-pair), heredity (between-pair) and their interaction, as well as the proportion of their sum of squares to the total. Of

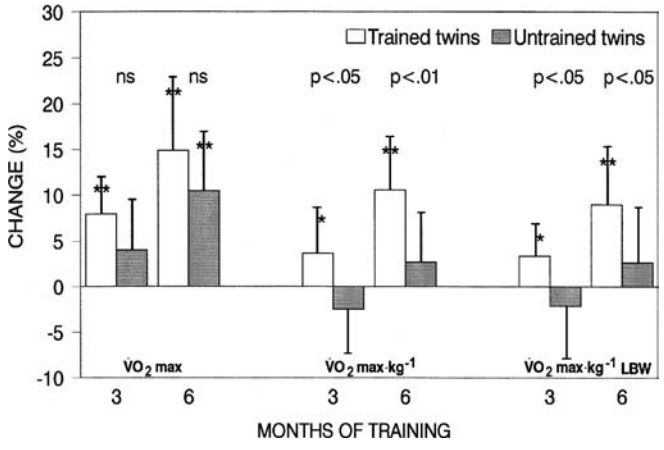

Fig. 1 Changes in maximum O_2 uptake ($\dot{V}O_{2max}$) of the trained twins after 3 and 6 months of training (*white bars*) and in untrained twins during the same time period (*black bars*). (*$P<0.05$, **$P<0.01$; comparison to their initial level. Upper signs: ns:non-significant, $P<0.05$, $P<0.01$; comparison of the changes in trained and untrained twins). (*LBM* Lean body mass, $\dot{V}O_{2max}\cdot kg^{-1}$ $\dot{V}O_{2max}$ per kg body mass)

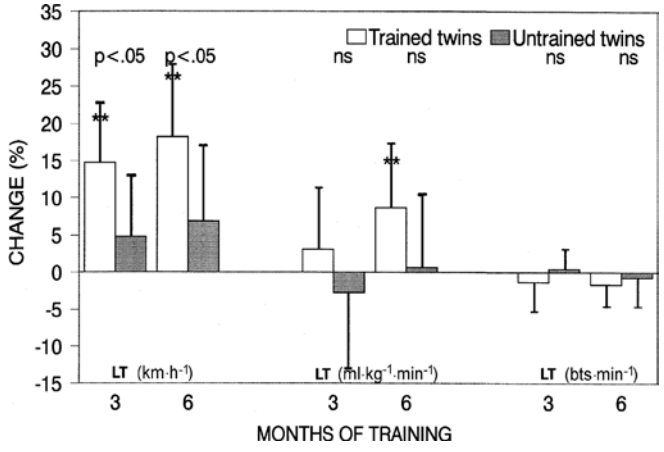

Fig. 2 Changes in the anaerobic lactate threshold (*LT*) in the trained twins after 3 and 6 months of training and in the untrained twins during the same time period. (*$P<0.05$, **$P<0.01$; comparison to their initial level. Upper signs: ns:non-significant, $P<0.05$, $P<0.01$; comparison of the changes in trained and untrained twins)

the change in $\dot{V}O_{2max}\cdot kg^{-1}$, 35% seems to be attributable to training , 45% to hereditary differences and 20% to the interaction of these two variables. In the case of the LT, training appears to account for 25–30% of the change, heredity for 50–60% and the interaction of these two for 15–20% of the change.

The intraclass correlation coefficient (R) remained unchanged after 6 months of training for the variables height (0.97), body mass (0.95), LBM (0.97), body fat (0.84), $\dot{V}O_{2max}$ (0.96) and maximal HR (HR_{max}, 0.93), while it decreased for $\dot{V}O_{2max}\cdot kg^{-1}$ (from 0.71 to 0.55), $\dot{V}O_{2max}\cdot kg^{-1}$ LBM (from 0.73 to 0.32) and running speed at LT (from 0.76 to 0.52), and increased for $\dot{V}O_2$ at LT (from 0.52 to 0.74).

Discussion

No influence of training on stature or the rate of the skeletal growth was observed in this study. This is in agreement with the findings of Parizkova (1970), Reznickova et al. (1981) and Wanne and Valimaki (1983). Ekblom (1969) and Eriksson (1972) found a relatively large increase in the height of trained children, the former without a corresponding increase in the control group, and the latter above the norms of the corresponding population. Both authors implied in the discussion that training favours skeletal growth. However, there is no such evidence when groups of the same biological maturation are compared, as was the case in the study presented here. Biological maturation did not differ in the twin groups, since monozygotic twins exhibit a similar growth rate, with a only small deviation in the onset of puberty (Fischbein 1977a, 1977b; Sklad 1977). Intraclass correlation of the twins' maturity stages gave values of 0.96 and 0.98 before and after training, respectively. It is likely that the large increase in height recorded in the studies of Ekblom and Eriksson was caused by pubertal peak growth velocity in the trained subjects.

Body mass increased in both groups. Although the increase in body mass of the control twins was almost

Table 4 Variance and the relative importance of training, heredity and their interaction in percentage changes of variables. The numbers in parentheses in the left-hand column refere to the month of training at which the changes were observed

Variable	Training		Heredity		Interaction	
	Variance	%	Variance	%	Variance	%
Fat (6)	312.34	20	132.14	69	20.78	11
Sumof five skinfolds (6)	549.00	16	356.24	75	43.88	9
Endomorphy (6)	780.30	21	316.31	68	50.52	11
Mesomorphy (6)	14.41	7	21.34	77	4.57	16
Ectomorphy (6)	798.64	12	625.43	75	110.76	13
$\dot{V}O_{2max}$ (3)	68.19	15	31.67	54	18.19	31
$\dot{V}O_{2max}\cdot kg^{-1}$ (3)	170.08	34	27.18	44	13.38	22
$\dot{V}O_{2max}$ kg^{-1} (6)	278.48	37	43.55	46	16.60	17
$\dot{V}O_{2max}\cdot kg$ LBM^{-1} (3)	136.11	28	25.63	43	17.23	29
$\dot{V}O_{2max}\cdot kgLBM^{-1}$ (6)	179.36	24	48.33	53	21.29	23
LT($km\cdot h^{-1}$) (3)	444.59	30	92.76	50	35.66	20
LT ($km\cdot h^{-1}$) (6)	580.36	26	167.08	60	37.88	14
LT ($ml\cdot kg^{-1}\cdot min^{-1}$) (6)	289.16	17	134.70	65	37.66	18

twice that of their trained brothers, no difference existed between them after training. However, a differentiation was observed in the body composition of the trained twins as an effect of training. Their relative body fat (%) decreased significantly as a result of the decrease of absolute body fat, while the control twins showed the opposite, maintaining their relative body fat constant. Both groups exhibited a similar significant increase in LBM. Thus, this decrease in relative body fat of the trained twins effected a smaller (numerical) increase in their body mass than in their untrained brothers. In addition, Heath-Carter's somatotype analysis revealed this differentiation of body composition in the trained twins to be attributable to a decrease in fatness (endomorphy) and an increase in leanness (ectomorphy). Other authors (Parizkova 1970; Oscai 1973; Reznickova et al. 1981) reported the same effect of training on body fat, but also a positive effect on LBM. This second effect was not observed in the current study.

$\dot{V}O_{2max}$ was increased by 10.5% in untrained twins during the 6-month training programme. A marked increase in $\dot{V}O_{2max}$ is observed during puberty following the peak growth velocity of height (Kobayashi et al. 1978; Kemper and Verschuur 1981; Mirwald et al. 1981; Paterson et al. 1986). By comparison, the greatest values of the annual increases in $\dot{V}O_{2max}$ observed in longitudinal studies with untrained children range from 14–17% (Hermansen and Oseid 1971; Andersen et al. 1978; Bailey et al. 1978) to 22% (Kobayashi et al. 1978), which in all cases were observed at the age of puberty. Thus, the increase in $\dot{V}O_{2max}$ in the control group of the present study over the 6 months of the training programme, which in most cases occurred during puberty, appears to be normal.

After 3 months of training, the trained twins showed a clearly greater increase in $\dot{V}O_{2max}$ than their brothers. However, thereafter, the rate of increase in $\dot{V}O_{2max}$ was similar to that of their untrained brothers. A significant increase in $\dot{V}O_{2max}$ occurred in both twin groups after 6 months, without any difference between them. An effect of training was also found after 3 months that was no longer evident after 6 months. In other words, training effected a fast increase in $\dot{V}O_{2max}$ so that a significant differentiation could be shown after 3 months, but not after 6 months, because of the significant differential rate of growth. The 14.9% increase in trained twins vs the 10.5% increase in their untrained brothers was not statistically significant. On the other hand, a clear effect of training was observed in relative $\dot{V}O_{2max}{\cdot}kg^{-1}$ or $\dot{V}O_{2max}{\cdot}kg^{-1}$LBM: trained twins experienced a 10.6% and 9.0% increase, respectively, after 6 months of training, while no change was observed in their untrained brothers. But this effect of training is partly attributable to the smaller increase in body mass of the trained twins in combination with the decrease in their relative body fat. The improvement of the relative $\dot{V}O_{2max}$ caused by training was combined with the differentiation in body composition in these trained subjects.

A similar improvement (15% in the absolute and 10% in the relative $\dot{V}O_{2max}$) was found by Ekblom (1969) after 6 months of training in 11-year-old boys, without any significant change in the control group. In this study, trained boys had a 55% greater $\dot{V}O_{2max}$ after 32 months of training, and untrained boys a 37% greater $\dot{V}O_{2max}$ in relation to their initial value (namely a maintenance of the difference). Massicotte and MacNab (1974) observed a 15% increase in the absolute $\dot{V}O_{2max}$ and 11% in the relative $\dot{V}O_{2max}$ in one out of three experimental groups with 11- to 13-year-old boys after 6 weeks of training. Eriksson (1972) found a greater improvement (19% in the absolute $\dot{V}O_{2max}$ and 16% in the relative $\dot{V}O_{2max}$) after 4 months of training in 11- to 13-year-old boys, while Mahon and Vaccaro (1989) observed a smaller improvement (9% in the absolute $\dot{V}O_{2max}$ and 7.5% in the relative $\dot{V}O_{2max}$) after 8 weeks of training.

According to Kobayashi et al. (1978) and Mirwald et al. (1981), a remarkable training-induced increase in $\dot{V}O_{2max}$ does not seem to be possible before the onset of pubertal growth, while pubertal growth is a critical period during which higher rates of increase in this parameter (caused by strenuous training or by genetically superior endowment) result in a significantly greater adult value.

The findings of Schmuecker and Hollmann (1973), Bar-Or and Zwiren (1973), Parizkova and Spynarova (1975) and Yoshida et al. (1980) agree on the inability of training to increase $\dot{V}O_{2max}$ before puberty. On the contrary, the results of the research made by Weber et al. (1976) revealed a significant increase in $\dot{V}O_{2max}$ in prepuberty (23.5% vs 11.8%), but the same increase together with growth in puberty (14.2% vs 15.9%) after 10 weeks of training in one of four prepubertal and four pubertal monozygotic twins. Daniels and Oldridge (1971) observed a relatively small increase (22%) in the absolute $\dot{V}O_{2max}$ in 11- to 15-year-old boys after 22 months of training, without any improvement in the relative $\dot{V}O_{2max}$.

No significant change was found by Stewart and Gutin (1976) after 8 weeks of interval training in 10- to 12-year-old boys, while Vaccaro and Clarke (1978) observed a significant increase in the relative $\dot{V}O_{2max}$ (17%) after 7 months of training (swimming) in 9- to 11-year-old boys and girls. A small but significant increase in the relative $\dot{V}O_{2max}$ (7%) was observed by Lussier and Buskirk (1977) in prepubertal boys and girls (growth stage 1) after 3 months of training. A proportionate increase (7% in absolute $\dot{V}O_{2max}$ and 8% in relative $\dot{V}O_{2max}$) was found by Rotstein et al. (1986) after 2 months of training in prepubertal boys (10–11 years old). Mahon and Vaccaro (1994) also observed a significant increase of about 16% in the absolute and 13% in the relative $\dot{V}O_{2max}$ in 8- to 12-year-old boys after 14 weeks of training.

The results of all of this research are unable to establish whether $\dot{V}O_{2max}$ is more or less trainable during puberty and before its onset. In the present study, a

separate comparison of the effects of training in prepubertal (maturity stage 1) and pubertal (maturity stages 2–5) twins gave a surprising result (Fig. 3). The three trained prepubertal twins experienced an increase in their $\dot{V}O_{2max}$ of 19.3%, compared to an increase of 5.2% in their untrained brothers. On the contrary, the other six trained pubertal twins experienced a similar increase (12.7%) to their untrained brothers (13.1%). In these twins, the increase in $\dot{V}O_{2max}$ showed a high correlation with the increase in their stature ($r = 0.93$, $P < 0.05$).

This relationship between the improvement in $\dot{V}O_{2max}$ of prepubertal and pubertal twins explains the small, if non-significant increase in $\dot{V}O_{2max}$ in the total sample of twins above the increase caused by growth, and shows clearly that an effect of training is not possible during the growth spurt associated with puberty. Before the onset of pubertal growth, training can improve the $\dot{V}O_{2max}$, as was also observed by Weber et al. (1976). There is currently no explanation as to why this happens during the period of puberty. Weber et al. (1976) suggest that growth hormone (GH) probably plays a more dominant role in functional adaptability during this growth period than does physical activity. In fact, the mean 24-h concentration of GH increases during puberty in relation to pre- and postpubertal growth, with an augmentation in the amplitude of the pulses rather than in their frequency (Mauras et al. 1987; Albertsson-Wikland and Rosberg 1988; Martha et al. 1989, 1992). Although Fahey et al. (1979) reported no alteration in the response of GH to exercise during the maturity stages of the puberty, an increasing tendency seems to exist between prepubertal and postpubertal subjects (Wirth et al. 1978; Greene et al.1987). Moreover, Weltman et al. (1992) observed an amplification of the pulsatile release of GH after 1 year of training (at an intensity above LT) in female adult subjects. It is unknown whether training has the same effect in children. However, there is no evidence that training during puberty inhibits the pulsatile release of GH or its direct or indirect anabolic action, so that functional adaptability would be limited. It is possible that some genes, which may contribute to the $\dot{V}O_{2max}$ response to training (Dionne et al. 1991; Rivera et al. 1997, 1999), are suppressed under the interaction of androgens and GH during puberty. Thus, the adaptability of $\dot{V}O_{2max}$ in training during this growth period would be either limited or completely inhibited.

In any case, the distribution of the pubertal subjects (in biologically pubertal growth) in the experimental or in the control groups of other publications may explain the conflicting results on the effect of training on $\dot{V}O_{2max}$ during puberty. It is possible that a larger or a smaller number of subjects of "real" pubertal growth in experimental or in control groups will result in a significant or non-significant effect, respectively. More pubertal subjects in the control group challenges a greater differentiation in their group (caused by growth), while fewer pubertal subjects in the experimental group would also seem to effect a greater differentiation in that group. Thus, some researchers have recorded a positive effect, while others have not. The analysis concerning the effect of training on $\dot{V}O_{2max}$ in relation to pubertal growth, as it was described above, can be summarised as in Fig. 4.

It is known that aerobic training can cause a reduction in HR_{max} after aerobic training (Ekblom et al. 1968; Pollock 1973). A similar reduction in the HR_{max} as observed in the present study was observed by Ekblom (1969) and Eriksson and Koch (1973). But this reduction

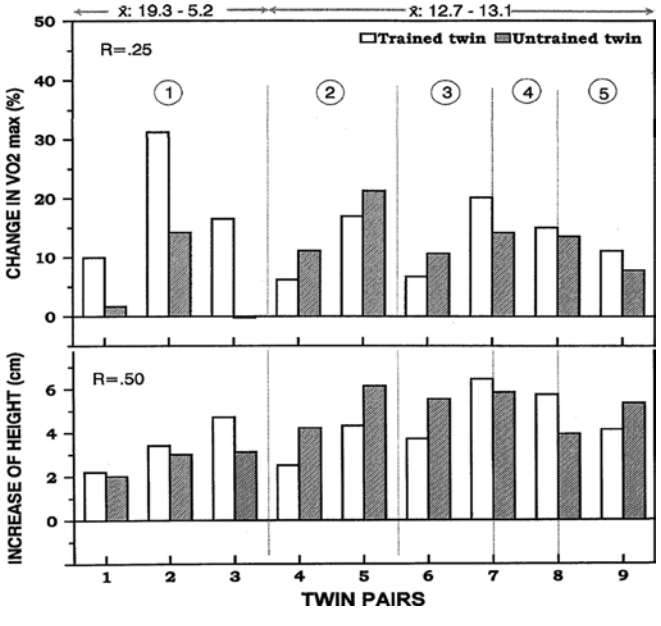

Fig. 3 Individual changes in absolute $\dot{V}O_{2max}$ and body height of each twin (trained and untrained) in relation to their stage of maturity (1–5) during the 6-month training period

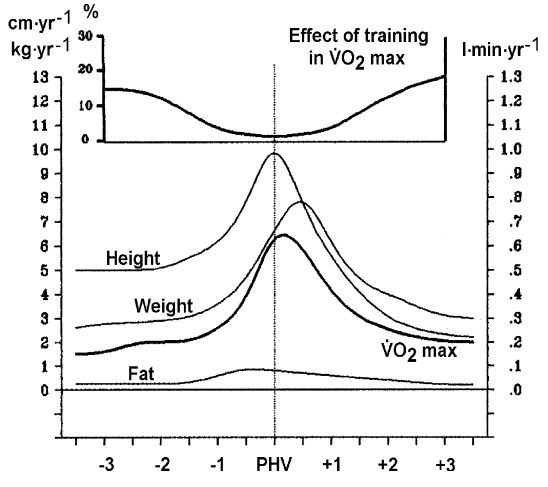

Fig. 4 Possible effect of training on absolute $\dot{V}O_{2max}$ during pubertal growth. The rate of increase per year in height, body mass, fat (left axis) and $\dot{V}O_{2max}$ (right axis) over time (−3 to +3 years) in relation to peak height velocity (*PHV*), as described by Tanner (1962), Kobayashi et al. (1978), Kemper and Verschuur (1981) and Mirwald et al. (1981). The effect of training on $\dot{V}O_{2max}$ during this time period follows an inverse pattern to that of the growth variables

in the twins of the present study is not an effect of training, since the untrained twins also presented a significant reduction. The change in HR_{max} perhaps resulted from the familiarisation of the subjects with the maximal effort tests, thus affording a lower degree of stress and hormonal release in the adrenaline axis.

The improvement in the LT of 4 mmol·l^{-1} occurred relatively quickly (being observed after only 1.5 months) and was independent of growth or maturity stage. Growth during the 6 months of training produced no significant alteration in the LT of the control twins. The 13.8% increase of the running speed at LT after 1.5 months of training, as well as the subsequent increase of 18.2% after 6 months of training show that the running speed at LT is a variable that is more sensitive and adaptable to training than is $\dot{V}O_{2max}$. This improvement in LT was independent of the alterations in $\dot{V}O_{2max}$, as shown by the correlation between their proportional changes (1.5 months: $r = -0.23$; 3 months: $r = 0.49$; 6 months: $r = 0.46$; $P > 0.05$), and is attributable to metabolic economy. Similarly, the increase in $\dot{V}O_2$ at LT is independent of the growth rate; rather, it is defined from the change in $\dot{V}O_{2max}$, maintaining unchanged the percentage of $\dot{V}O_{2max}$ at LT. HR at LT showed the same response to training as did HR_{max} (statistically unchanged); indeed, HR at LT is probably related to HR_{max}, and it follows the change of HR_{max}.

The deficiency of any change in $[La]_{max}$ of the trained twins shows a weakness of prepubertal and pubertal children in that they are unable to improve their lactate production or their tolerance to high [La]. The intensity of training, especially in the later stages, was sufficiently high to provide anaerobic adaptations (interval training with 95–100% of $\dot{V}O_{2max}$), but it did not cause any improvement in the $[La]_{max}$. The mean values of the peak [La] in this test were in the range 8.2–9.4 mmol·l^{-1}, and much of the time differed significantly from those in the laboratory test that were carried out to determine the $\dot{V}O_{2max}$ (7.1–8.5 mmol·l^{-1}). These differences concur with the measurements of Kindermann et al. (1975). The correlation coefficient of the peak lactate concentration between the field and the laboratory tests was also low (0.45–0.59).

In other studies (Ekblom 1969; Eriksson and Koch 1973; Eriksson et al. 1973; Massicotte and MacNab 1974; Weber et al. 1976), [La] was determined by laboratory tests and the mean values in the pre-test were often lower than 8.0 mmol·l^{-1}, giving a significant increase in post-test values after training. Massicotte and MacNab's study is an exception, with a significant increase from 8.0 to 9.6 mmol·l^{-1}. In the studies of Ekblom (1969) and Weber et al. (1976), as well as in the present study, where pre-test mean values were higher than 8.0 mmol·l^{-1}, a non-significant increase was observed. It is also uncertain whether the $[La]_{max}$ can be increased before and during puberty.

In the present study it was also interesting to estimate the contribution of genotype to any significant effect of training. The model of one-way ANOVA was used for this aim. The percentage changes of subjects include differences caused by growth or growth + training, related also to their initial level. Within-pairs variance was also caused by intrapair differences provided by the influence of training. Between-pairs variance resulted from differences between the twins that were due to their growth and was attributed to genetic factors. Interaction or residual variance (including the error variance) indicated a non-linear interplay between heredity and training.

In general, the changes in body fat during prepubertal and pubertal growth seem to have a strong genetic dependence (70–75%), and training can account for only 15–20% of the post-training level (Table 4). Likewise, heredity shapes mainly the changes in $\dot{V}O_{2max}$·kg^{-1} (45%) and LT (50–60%), while training alone plays a subordinate role (35% and 25–30%, respectively). As opposed to the case for body fat, the interaction between heredity and training with respect to $\dot{V}O_{2max}$·kg^{-1} and LT appears to play a substantial role in the changes observed (20% and 15–20%, respectively). This implies that training does not create the same improvement in all genotypes; in about 20% of subjects it provides a non-linear effect. In others words, some genotypes present a low, and some others a high response to training independently to their change because of growth. The present evaluation of the heredity—training interaction with regard to changes in $\dot{V}O_{2max}$·kg^{-1} does not agree with the estimation of 7% by Weber et al. (1976), or that of 50% by Bouchard (1986).

As the Pearson correlation analysis showed, 30% of the interaction variance may be explained by the initial level of $\dot{V}O_{2max}$·kg^{-1} ($r = -0.55$, $P < 0.05$) and 40% by the initial level of LT ($r = -0.65$, $P < 0.01$). The rest of the variance (about 70 or 60%, respectively) may be explained by the differential sensitivity of genotypes to the training stimulus, as presented by Prud'Homme et al. (1984).

It is remarkable to report, for two cases of trained twins, their initial level of $\dot{V}O_{2max}$·kg^{-1} and their improvement in that parameter during the training. One case had a higher value before (58.4 ml·kg^{-1}·min^{-1}) as well as after training (63.8 ml·kg^{-1}·min^{-1}; i.e. an improvement of 9.2%). The other case had a relatively low value (49.6 ml·kg^{-1}·min^{-1}) before training and attained the second, higher value (61.4 ml·kg^{-1}·min^{-1}) after training (i.e. an improvement of 23.8%). By comparing these two cases to the others, two hypotheses were established: (1) it is possible to possess a superior phenotype with respect to aerobic power [i.e. that a superior genotype exists; (Klissouras 1977)] and (2) it is possible to be a high responder to training (Bouchard 1986).

In conclusion, growth at puberty, regardless of structural and functional acceleration, leaves no place for the effective influence of training on variables of maximal effort (e.g. $\dot{V}O_{2max}$, $[La]_{max}$). Training during prepubertal and pubertal growth can control body composition and can favour aerobic power and capacity, as shown in the present study by the changes imposed by

training on $\dot{V}O_{2max}\cdot kg^{-1}$ and the LT. Genetic control seems to play a preponderant role in the expression of adaptations, within which the genotype–training interaction may also have an important contribution to the establishment of an elite phenotype in endurance sports.

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