Citation:

Bird, S, Theakston, S, Owen, A and Nevill, A 2003, 'Characteristics associated with 10-km running performance among a group of highly trained male endurance runners age 21-63 years', Journal of Aging and Physical Activity, vol. 11, no. 3, pp. 333-350.

Journal of Aging and Physical Activity, 2003, 11, 333-350 © 2003 Human Kinetics Publishers, Inc.

Characteristics Associated With 10-km Running Performance Among a Group of Highly Trained Male Endurance Runners Age 21–63 Years

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This study assessed physiological and cardiac factors associated with 10-km running performance in a group of highly trained endurance runners age 21–63 years. Participants (N=37) underwent a resting echocardiograph and incremental treadmill running test. They also provided information on their recent 10-km races. Data were analyzed using "best subsets" multiple regression. Declines with age were found for 10-km running speed ($0.26 \, \mathrm{m \cdot s^{-1} \cdot decade^{-1}}$), maximum heart rate (4 beats/decade), VO_{2peak} (6 ml · kg⁻¹ · min⁻¹ · decade⁻¹), velocity at lactate threshold (1 m · s⁻¹ · decade⁻¹), and VO_{2} at lactate threshold (4 ml · kg⁻¹ · min⁻¹ · decade⁻¹). The percentage of VO_{2peak} at which lactate threshold occurred increased with age by 1.5% per decade. The rate of change of displacement of the atrioventricular plane at the left free wall and septum both declined by 1 cm · s⁻¹ · decade⁻¹. The best single predictor of 10-km running speed was velocity at lactate threshold.

Key Words: endurance runners, aging, cardiac, 10-km running speed

Studies with young adults and endurance athletes under the age of 40 years have suggested that maximum oxygen utilization (VO_{2max}, ml·kg⁻¹·min⁻¹) is a strong predictor of running speed for distances such as 5 and 10 km (Morgan, Baldini, Martin, & Kohrt, 1989; Morgan, Martin, & Kohrt, 1986; Nicholson & Sleivert, 2001; Ramsbottom, Nute, & William, 1987; Ramsbottom, Williams, Kerwin, & Nute, 1992). Nonetheless, in relatively homogeneous groups, VO_{2max} and race times have been shown to have only a low to moderate correlation (Conley & Krahenbuhl, 1980; Sjödin & Svedenhag, 1985; Tanaka, Takeshima, Kato, Niihata, & Ueda, 1990), and VO₂ at lactate threshold is a better predictor of performance (Fay, Londeree, LaFontaine, & Volek, 1989; Kumagai et al., 1982). Likewise, velocity at lactate threshold has been shown to be a good predictor of

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running speed for 3 km (Grant, Craig, Wilson, & Aitchinson, 1997), as has velocity at individual anaerobic threshold for a range of distances from 1,500 m to marathon (Bassett & Howley, 2000; Roecker, Schotte, Niess, Horstmann, & Dickhuth, 1998).

Although a high VO_{2max} is a characteristic of highly trained endurance runners, what limits VO_{2max} and causes fatigue during endurance running is a matter of debate. The "classical" viewpoint, that it is limitations in maximal cardiac output and the consequent oxygen delivery that dictate VO_{2max}, has been subjected to recent discussion in the literature (Bassett & Howley, 1997; Bergh, Ekblom, & Åstrand, 2000; Noakes, 1988, 1998; Richardson, Harms, Grassi, & Hepple, 1999), with Noakes proposing that the available evidence suggests that it is the capacity of the skeletal muscle to work that limits oxygen utilization, and not vice versa. Furthermore, and in the context of this study, Paavolainen, Nummela, Rusko, and Häkkinen (1999) also present evidence for musculoskeletal factors rather than cardiovascular limitations being a cause of fatigue in 10-km running and hence a potential determinant of 10-km running speed.

In master runners, the aging aspect adds a further dimension to the elucidation of performance predictors, with changes in selected peak physiological responses being linked to the seemingly unpreventable declines in endurance running speed. In summary, it is reported that after the age of 30 years, there is a 6%/decade decline in running speed of highly trained male endurance runners over distances such as 10 km (Fuchi, Iwaoka, Higuchi, & Kobayashi, 1989; Joyner, 1993). In accordance with the classical viewpoint that oxygen delivery is the limiting factor for VO (Bassett & Howley, 2000), this decline in running speed has been largely attributed to a concomitant reduction in VO_{2max} (Fuchi et al.; Maharam, Bauman, Kalman, Skolnik, & Perle, 1999; Rivera et al., 1989), via an age-linked reduction in maximum cardiac output (Q_{max}; Fuchi et al.; Green & Crouse, 1993; Rivera et al.) caused by a decline in maximum heart rate (Fuchi et al.; Green & Crouse; Heath, Hagberg, Ehsani, & Holloszy, 1981; Rivera et al.). If these results are interpreted via the converse viewpoint, however (Noakes, 1998; Paavolainen et al., 1999) that it is oxygen use by the skeletal muscle that limits VO_{2max}—an alternative explanation is that the decline in VO_{2max} is caused by reduced functional capacity of the muscle, which results in a reduced maximal workload and consequently a lower maximal oxygen requirement. This, as a consequence of the lower oxygen requirements, means that a lower maximum heart rate is capable of attaining the necessary Q_{max}. Hence, via this alternative explanation, the reduced running speed, VO_{2max}, and maximum heart rate are consequences of reductions in the muscles' working capacity, and not oxygen delivery.

When investigating the physiology of aging endurance runners, researchers have inevitably focused on the widely used measurements of respiratory gases and blood lactate. This has produced some equivocal although not entirely contradictory results that appear to comply with the findings for younger participants. For example, in their work with master athletes, Takeshima and Tanaka (1995) found VO₂ at lactate threshold to be the best predictor for performance over 10 km, whereas Wiswell et al. (2000) reported the single best predictor of performance over 10 km to be VO_{2max}, with VO₂ at lactate threshold (L/min) improving the prediction in female but not male runners. Nonetheless, as indicated earlier, there is potential for further investigation into other factors that might help elucidate the causes of the observed decline in running speed with increasing age. One of these areas is the cardiology of aging runners. Studies involving relatively young adults have reported cardiac adaptations including hypertrophy to be characteristic of highly trained endurance athletes (Cohen & Segal, 1985; George, Wjolfe, Burggraf, & Norman, 1995; Green & Crouse, 1993; Schmidt-Truckäss et al., 2000; Turpeinen et al., 1996; Whyte, Sharma, George, & McKenna, 1999). Furthermore, some have attempted to relate cardiac measurements to peak VO₂ (Takahashi et al., 2001). Despite a number of these cardiac factors being affected by aging in the general population (Owen, 1999), there appears to have been little attempt to assess them in an older athletic population or to incorporate cardiac measurements into the predictive equations for endurance running speed.

Consequently, the aims of this study were to assess selected cardiac and physiological factors in a group of highly trained endurance runners covering a wide span of ages, in order to determine which factors altered with increasing age and to produce a model for predicting running speed during 10-km races.

Methods

The local research-ethics committee approved the study. An information sheet was provided at each of the procedures, and immediately before their involvement in each of the assessment sessions, the participants signed a consent form.

PARTICIPANTS

A total of 40 male endurance runners volunteered to participate in the study. Via a questionnaire, participants provided data on their competitive history, performances in 10-km races during the past 6 months, and their training over the preceding 3 months (Table 1). To quantify the standard of performance relative to his age, each participant's recent (preceding 6 months) fastest time in a ratified 10km race was converted into his average speed for the distance (m/s) and expressed as a percentage of the best ever U.S. 10-km speed for his age (USA Track & Field, 2001). The minimum standard for inclusion in the study was a 10-km running speed greater than or equal to 65% of the best ever U.S. 10-km running speed for one's age. Of the initial volunteers, 37 fulfilled this criterion. All participants competed regularly over the distance of 10 km and had 1-40 years competitive experience. They ranged in standard from good club runners to national age-group champions and Olympic athletes. The group was then ranked and divided via a median split. Participants in the fastest group (Group A: high-caliber runners, age range 21-61 years) had completed 10-km races at speeds at least 85% of the running speed for the best ever U.S. 10-km speed for their age, and the slower group (Group B: lowcaliber runners, age range 23-63 years) ran at speeds 65-85% of the fastest ever U.S. 10-km race speed for their age.

ASSESSMENT OF ANTHROPOMETRIC VARIABLES

See Table 2 for assessment of anthropometric variables. All participants had their height and body mass assessed, using clinical scales and a stadiometer (Seca 710, Hamburg, Germany). This information was then used to calculate surface area, using the equation of DuBois and DuBois (1916).

Table 1 Data Relating to Training and 10-km Performance, $M \pm SD$ (range)

Group	·n	Best 10-km time,in preceding 6 months, min	Speed of 10 km relative to best ever U.S., %	Weckly training distance, km (minimum- maximum)	Years of competition (minimum-maximum)
A	18	33.9 ± 3.3	90.2 ± 4.2	99 ± 43 (40–193)	12.6 ± 8.5 (2-40)
B	19	38.8 ± 3.5	77.9 ± 6.1	98 ± 50 (40–233)	13.7 ± 10.9 (1-30)
All	37	36.4 ± 4.2	83.6 ± 8.1	99 ± 46 (40–233)	13.1 ± 9.6 (1-40)

Note. Group A (high-caliber runners) had recent 10-km race speeds that were at least 85% of the running speed for the best ever U.S. 10-km speed for their age. Group B (low-caliber runners) had recent 10-km race speeds that were 65–85% of the fastest ever U.S. 10-km race speed for their age.

Table 2 Anthropometric Values for Runners $(M \pm SD)$

Group	п	Age, yr	Body mass, kg	Height, m	Surface area, m²
Α	18 -	39.3 ± 13.6	69.2 ± 7.8	1.77 ± .06	1.85 ± 0.11
В	19	39.4 ± 9.1	71.6 ± 9.9	1.78 ± 0.08	1.89 ± 0.16
All	37	39.4 ± 11.3	70.4 ± 8.9	1.78 ± 0.07	1.87 ± 0.14
All	37	39.4 ± 11.3	70.4 ± 8.9	1.78 ± 0.07	

Note. Group A (high-caliber runners) had recent 10-km race speeds that were at least 85% of the running speed for the best ever U.S. 10-km speed for their age. Group B (low-caliber runners) had recent 10-km race speeds that were 65–85% of the fastest ever U.S. 10-km race speed for their age.

ECHOCARDIOGRAPHIC ASSESSMENT

See Table 3 for echocardiographic assessment of the participants. Participants presented themselves to the cardiology department of the local hospital in a normal rested state, not having trained that day. The same experienced cardiologist performed all echocardiographic assessments, using a Hewlett-Packard Sonos 1000 echocardiograph (McMinniville, OR). After reclining into a semirecumbent position, all participants had a standard echocardiogram performed. Left-ventricular measurements were made using the Penn convention (Devereux & Reichek, 1977). The motion of the left-ventricular atrioventricular plane was assessed at the left free wall-and at the septum from the apical four-chamber view (Owen, 1999). From this the following were ascertained: left-ventricular mass (Devereux &

Table 3 Summary of Cardiac Measurements for Runners and p Values for Group Effect (Group A vs. Group B)

Factor	Group	n	p $M \pm SD$	for group effect Group A vs. Group B
Left-ventricular mass, g	all	32	299 ± 85	.868
	Α	16	296 ± 101	
	В	16	302 ± 70	
Left-ventricular mass, g/kg	all	32	4.23 ± 1.08	.774
, _ , _ ,	Α	16	4.29 ± 1.25	
	В	16	4.17 ± 0.91	
Left-ventricular mass, g/kg0.67	all	32	17.21 ± 4.49	.893
	Α	16	17.32 ± 5.30	
	В	16	17.10 ± 3.67	
Ld, cm	all	37	1.66 ± 0.26	.522
•	A	18	1.63 ± 0.24	
	В	19	1.69 ± 0.28	
Lv, cm/s	ali	37	11.2 ± 2.5	.729
,	A	18	11.1 ± 3.0	
	В	19	11.3 ± 1.8	
Cd, cm	all	36	1.43 ± 0.25	.839
•	Α	18	1.43 ± 0.21	
	В	18	1.42 ± 0.30	
Cv, cm/s	all	36	8.0 ± 2.4	.663
•	A	18	7.9 ± 2.3	
	·B	18	8.1 ± 2.5	

Note. L = left free wall; d = total displacement of the atrioventricular plane; v = early rate of change of displacement; C = central septum; LT = lactate threshold. Group A (high-caliber runners) had recent 10-km race speeds that were at least 85% of the running speed for the best ever U.S. 10-km speed for their age. Group B (low-caliber runners) had recent 10-km race speeds that were 65–85% of the fastest ever U.S. 10-km race speed for their age.

Reichek), total displacement of the atrioventricular plane at the left free wall and the septum, and early rate of change of displacement at the left free wall and the septum.

INCREMENTAL TREADMILL TEST

See Table 4 for incremental treadmill test results. For the incremental treadmill test of peak oxygen uptake (VO_{2peak}) and lactate threshold, the participants presented at the laboratory in a rested state, not having raced or undertaken exhaustive training such as intervals or an exhaustive-threshold run for at least 72 hr. They then

Table 4 Summary of Physiological and 10-km Performance for Group Effect (Group A vs. Group B)

Factor	Group	n	$M \pm SD$	for group effec Group A vs. Group B
Pactor				
10-km speed, m/s	all	17	4.63 ± 0.53	<.001
to kin speed, mil	A	18	4.96 ± 0.47	
	В	19	4.32 ± 0.37	
Velocity at LT, km/hr	all	37	14.9 ± 1.6	<.001
, 5100103 110 22, 111111	Α	18	15.8 ± 1.6	
	В	19	14.1 ± 1.1	
VO _{2peak} , ml · kg ⁻¹ · min ⁻¹	all	37	62.8 ± 9.5	.004
2peak*	Α	18	65.8 ± 9.8	
	В	19	60.0 ± 8.5	
VO ₂ at LT, ml · kg ⁻¹ · min ⁻¹	all	37	50.6 ± 6.3	.015
702 11 227, 111	Α	18	52.4 ± 6.2	
	В	19	48.9 ± 6.0	
VO _{2peak} at LT	all	37	80.9 ± 4.8	.255
2pcak	A	18	80.0 ± 4.5	
	В	19	81.8 ± 5.0	
Peak heart rate, beats/min	all	37	177 ± 11	.655
,	Α	18	175 ± 11	
••	В	19	178 ± 12	

Note. LT = lactate threshold.

completed a health questionnaire and a training and competition questionnaire. After a short warm-up and familiarization on the treadmill, the test commenced. The protocol used was discontinuous, involving five to six 4-min stages at increments of 1, 1.5, or 2 km/hr. The treadmill (Woodway XELG2, Weil-am-Rhein, Germany) was set at a 1% gradient throughout (Jones & Doust, 1996). The speed of the stages and the size of the increments were determined, based on knowledge of the participant's recent running speeds in 10-km races, to ensure that at least five stages were completed.

The participants were fitted with Polar SportsTester heart-rate monitors (Polar Elektro Oy, Kempele, Finland) set to record at 5-s intervals throughout the test. Respiratory measurements were recorded at 30-s intervals using a Covox gas analyzer (Fitness Research Systems, Exeter, UK), low-resistance Falconia 35-mm ducting (Baxter, Woodhouse and Taylor, Macclesfield, UK), and low-resistance Tshaped mouthpieces with one-way valves (Hans Rudolf, Kansas City, MO) supported by a headset.

After 3 min and 30 s of each stage, the participant indicated his rating of perceived exertion (Borg, 1982). At the end of each 4-min stage, the participant stopped running and straddled the moving belt while a 20-µl finger-prick blood sample was taken to analyze for lactate concentration, using a Biosen 5030L analyzer (EKF Industrie-Elektronik Gmbh, Magdeburg, Germany; Davison et al., 2000).

The test continued until volitional exhaustion, with the participant indicating when he felt that he could only continue for 1 more minute, at which point he was given verbal encouragement to finish this last minute. Immediately after the cessation of the test, a final blood sample was taken for lactate analysis.

Peak VO₂ was established by calculating the mean VO₂ of the last two 30-s intervals of the final stage. Given the debate over the presence of a genuine plateau in oxygen consumption and the consistency with which it is observed (Noakes, 1998), it was decided to refer to the highest oxygen consumption as peak VO2, although the values attained fulfilled at least three of the criteria for VO_{2max} as described by Bird and Davison (1997). The lactate threshold was determined using the D_{max} method, which is reported to be the strongest predictor of 10-km running velocity by Nicholson and Sleivert (2001). The D_{max} method plots the lactate concentration against VO2 and then calculates the point that yields the maximal distance from the curve to a straight line drawn between two points on the extremes of the curve (Cheng et al., 1992).

STATISTICAL ANALYSIS

Cardiac and physiological factors were correlated with age and the participants' best recent 10-km running speeds using Pearson's product-moment correlations. Differences in physiological and cardiac variables between high-caliber and lowcaliber runners were analyzed using ANCOVA, with group as a between-participants factor and age as a covariate (SPSS 10), and where relevant this provided further analysis of the potential association with age. Alpha was set at .05.

Multiple linear regression was used to identify the physiological and cardiac variables that were best able to predict 10-km running speed. Clearly, not all the physiological and cardiac variables are required to predict 10-km running performance. It is likely that a subset of the predictor variables, that is, a "reduced model," will more than adequately predict performance.

There are a number of useful methods available for choosing such reduced models (backward and forward stepwise regression, e.g.), but the most comprehensive and thorough method of identifying an appropriate reduced model is to explore all possible combinations of subsets using MINITAB's "BREG" multiple-regression routine (see Atkinson & Nevill, 2001), which adopts the maximum coefficient of determination R^2 as the criterion, by first examining all one-predictor regression models and then selecting the two models giving the largest R^2 . Next, the analysis examines all two-predictor models, selects the two models with the largest R^2 , and then displays criterion information on these two models. This process continues until the model contains all the available predictors. For each model, the MINITAB output provides information based on four criteria: R^2 , the adjusted R^2 (adj R^2), Mallows's criterion, and the standard deviation of errors about the regression line (s). For further information about MINITAB's BREG "best subsets" procedure, see Hocking (1976) and Goodnight (1979).

Results

Anthropometric, cardiac, physiological, training, and 10-km-running data for the participants are presented in Tables 1-6.

The running speed of the high-caliber runners (Group A) was on average 0.64 m/s faster than that of the low-caliber runners (Group B; p < .001). Running speed (m/s) declined by 0.26 m·s⁻¹·decade⁻¹ (r = .561, p < .001), with no group-by-age interaction, indicating a similar decline in speed with age for the two groups. Maximum heart rate was not statistically significantly different between the two groups. It declined by an average of 4 beats/decade (r = .415, p < .05), with no statistically significant age-by-group interaction, indicating a similar decline in both groups. The peak VO, (ml·kg-1·min-1) of Group A averaged 5-6 ml·kg-1· min^{-1} higher than that of Group B (p < .01). The rate of decline with age did not differ between groups and was in the region of 6 ml \cdot kg⁻¹ \cdot min⁻¹ \cdot decade⁻¹ (r = .813, p <.001). Velocity at lactate threshold was greater in Group A by an average of 1.7 m/s (p < .001), with both groups displaying a similar decline of just less than 1 m · s⁻¹ · decade⁻¹ (r = .686, p < .001). Oxygen utilization at lactate threshold (ml·kg⁻¹·min⁻¹) showed a similar response, with Group A averaging around 3.5 ml · kg-1 · min-1 higher than Group B (p < .05). The rate of decline with age was around 4 ml·kg⁻¹. min^{-1} · decade⁻¹ (r = .723, p < .001), with no statistically significant difference in the rate of decline between the groups. The average percent peak VO, at which lactate threshold occurred was 81%. This did not differ between groups and increased with age in both groups by just over 1.5% per decade (r = .399, p <.05).

Left-ventricular mass did not correlate with age (p > .05) when expressed as absolute mass (g) or when scaled to body mass (g/kg BM or g/kg BM^{0.67}) or surface area (g/m²; Table 6). Nor did left-ventricular mass (scaled or unscaled) correlate (p > .05) with years of competition or average weekly training distance (Table 6). There was a significant relationship between body mass and left-ventricular mass: Left ventricular mass (g) = 4.2066(kg body mass) + 2.2158 (r = .463, p < .05). Higher mileage runners had proportionately higher left-ventricular-mass to bodymass-1.0 ratios.

For the whole group, the early rates of change of displacement of the atrioventricular plane at the left free wall and septum both declined with age by an average of 1 cm \cdot s⁻¹ · decade⁻¹ (r = .424, p < .01, and r = .506, p < .01, respectively), but neither was affected by the standard of the runner (Group A vs. Group B). There was no correlation between age and total displacement of the atrioventricular plane at the left free wall (r = .063, p > .05) or the septum (r = .093, p > .05), and neither was affected by the standard of runner (p > .05). Runners who ran a greater distance per week (>75 km) had a smaller total displacement of the atrioventricular plane at the septum than did lower mileage runners (<75 km/wk; 1.54 ± 0.27 vs. 1.34 ± 0.20 cm, p < .05), but when body-mass differences (cm/kg) were included in this, the difference in total displacement of the atrioventricular plane at the septum disappeared (p > .05). Displacement and early rate of change of the atrioventricular plane at the left free wall and septum did not differ between the high- and low-distance training groups (p > .05). This was also the case when total displacement and early rate of change of the atrioventricular plane at the left free wall and septum were scaled for body mass (kg; p > .05).

Table 5 Summary of the Fac	of the factors Correlated with Age	Malen M	iui Age				
Factor correlated with age	Group	z.	p for Age × Group interaction	Equation; Group $B = 1$	R2	p for age as covariate	SS
10-km running speed, m/s Maximum heart rate, beats/min Left-ventricular mass, g Lv, cm/s Cv, cm/s VO _{2peak} , ml·kg ⁻¹ ·min ⁻¹ Velocity at LT, km/hr VO ₂ at LT, ml·kg ⁻¹ ·min ⁻¹ % VO ₂ at LT, ml·kg ⁻¹ ·min ⁻¹		37 36 32 37 37 37 37 37	254 .859 .958 .958 .371 .371 .372 .372	5.67 - 0.026(age) - 0.640(group) 193 - 0.42(age) 272 + 0.72(age) 14.8 - 0.0918(age) 12.1 - 0.105(age) 90.7 - 0.634(age) - 5.6(group) 19.6 - 0.0962(age) - 1.7(group) 68.1 - 0.40(age) - 3.5(group) 74.3 + 0.168(age)	.688 .172 .008 .180 .256 .671 .760 .601	<001.012.037.009.002<001<001<001<001<001<001	0.3047 10.52 86.73 2.255 2.076 5.599 0.8053 4.087

early rate of change of displacement; C = central septum; LT = lactate threshold.

Correlation Table for Cardiac Measurements With Age, Performance, Competitive History, and Training Table 6

Cd, cm	0.093	0.033	0.094	0.281	-0.125
	0.591	0.850	0.587	0.102	0.481
	36	36	36	35	34
Ld, cm	-0.063	0.066	0.023	0.113	-0.054
	0.710	0.700	0.892	0.510	0.757
	37	37	37	36	35
. Cv, cm/s	-0.506**	0.384*	0.087	-0.100	0.050
	0.002	0.021	0.614	0.568	0.779
	36	36	36	35	34
Lv, cin/s	0424**	0.327*	0.119	-0.114	0.105
	0.009	0.048	0.485	0.508	0.548
	37	37	37	36	35
Left- ventricular mass/surface area, g/m²	0.087 0.635 32	0.099 0.588 32	0.163 0.372 32	0.273 0.131 32	0.138 0.451 32
Left-	0.045	0.131	0.183	0.254	0.169
ventricular	0.807	0.474	0.316	0.160	0.355
mass, g/kg ^{2,67}	32	32	32	32	32
Left-	0.023	0.162	0.216	0.243	0.200
ventricular	0.902	0.375	0.234	0.180	0.272
mass, g/kg	32	32	32	32	32
Left- ventricular mass, g	0.087 0.637 32	0.061 0.740 32			0.102 0.579 32
	Age (yr) Pearson correlation p (2-tailed)	Speed (m/s) Pearson correlation p (2-tailed)	% U.S. best speed Pearson correlation p (2-tailed)	Competitive experience (yr) Pearson correlation p (2-tailed) n	Training distance (km/wk) Pearson correlation p (2-tailed) n

= left free wall; ν = early rate of change of displacement; C = central septum; d = total displacement of the atnoventricular plane. 5. **p < .01.

Years of competition did not significantly correlate with total displacement or early rate of change of the atrioventricular plane at the left free wall or septum (Table 6), nor was there any difference when the group was divided by a median split into those who had been competing for up to 12 years and those with more than 13 years competitive experience (p > .05), nor for left-ventricular mass (g, g/kg BM, g/kg BM^{0.67}, or g/m²; p > .05).

Physiological and anthropometric measurements derived from the incremental treadmill test considered for inclusion in the regression model for predicting 10km running speed were peak VO₂ (subject to scaling), body mass (kg), maximum heart rate (beats/min), percent maximum heart rate at lactate threshold, percent peak VO, at lactate threshold, VO, at lactate threshold, final running speed (m/s), and velocity at lactate threshold (m/s). Cardiac measures derived from the echocardiographic assessment considered for inclusion in the predictive model were leftventricular mass, total displacement of the atrioventricular plane at the left free wall (Ld) and septum (Cd), and the early rate of change of displacement of the atrioventricular plane at the left free wall (L ν) and septum (C ν). We acknowledge, however, that when considering these cardiac measurements for inclusion, they were semirecumbent resting values, and that this might limit their applicability to cardiac function during strenuous exercise such as 10-km races. Other factors incorporated into the analysis were age (years), years of competitive experience, and average weekly training mileage. The predictive model derived was for the combined subgroups of high- and low-caliber runners.

The single strongest predictor of running speed was velocity at lactate threshold: Speed (m/s) = $0.334 + (0.288 \times \text{velocity})$ at lactate threshold, m/s). This explained 75% of the predicted running speed (s = .27). MINITAB's BREG bestsubsets regression-analysis routine, however, identified the following parsimonious model to predict 10-km running speed (containing only significant predictor variables, p < .05): Speed (m/s) = $3.18 + (0.200 \times \text{velocity at lactate threshold, m/s})$ s) – $(0.0149 \times \text{maximum heart rate, beats/min}) + (0.189 \times \text{peak VO}_{2peak}, L/min}) +$ (0.0495 × early rate of change of displacement of the atrioventricular plane at the septum, cm/s) - 0.209 (low-caliber runners).

This model increased the R^2 to 88% (velocity at lactate threshold p < .001, maximum heart rate p < .001, $VO_{2peak}(L/min) p = .002$, and the early rate of change of displacement of the atrioventricular plane at the septum p = .010; caliber of runner p = .029, s = .19). Note that the value 3.18 m/s represents the intercept for highcaliber runners (as a baseline), and the low-caliber runners are 0.209 m/s below this value.

Discussion

The decline in running speed with increasing age for this cross-sectional group of athletes was around 5% per decade (0.26 m·s⁻¹·decade⁻¹) and is therefore similar to those previously reported by Joyner (1993) on U.S. age-record performances and Fuchi et al. (1989) in their cross-sectional study of Japanese runners. The decline in VO_{2peak} with increasing age of around 6 ml \cdot kg⁻¹ \cdot min⁻¹ \cdot decade⁻¹ was also comparable with the 4.4 ml \cdot kg⁻¹ \cdot min⁻¹ \cdot decade⁻¹ reported by Fuchi et al. and the 5-7% per decade decline reported for highly trained male athletes by Rogers, Hagberg, Martin, Ehsani, and Holloszy (1990) and Trappe, Costill, Vakovich,

Jones, and Melhan (1996). The decline in maximum heart rate of around 4 beats · min⁻¹ · decade⁻¹ is similar to the 3.2% per decade reported by Fuchi et al. and the 5 beats · min⁻¹ · decade⁻¹ decline in a 22-year longitudinal study conducted by Trappe et al. Nonetheless, given the cross-sectional nature of this study, caution is needed when interpreting the results, as previously indicated.

The relatively low maximum heart rate for the younger runners indicated by the correlation and the relatively low rate of decline of around 4 beats · min⁻¹ · decade⁻¹ is an interesting finding, the causes of which can be speculated on. For example, if a characteristic of a highly trained "elite" endurance runner is a suppressed maximum heart rate, it is not surprising that the rate of decline should be less than that observed in nonathletic populations, because they will be declining from the relatively low values of the elite 20- to 30-year-olds. Also, although the study design endeavored to include elite individuals across the age range, it is possible that the older elites were not currently undertaking the same volume and intensity of training as were those from the younger end of the age spectrum. Consequently, any potential effect of training in suppressing the maximum heart rate could be less in the older runners. The overall effect of this would be an ameliorated decline in maximum heart rate as the age-related decline is partly compensated for by the reduced suppression caused by training.

So, although we acknowledge the cross-sectional nature of the study, the decline in maximum heart rate of 4 beats · min⁻¹ · decade⁻¹ from an intercept of 193 beats/min for this group suggests that the commonly applied 6.5 or 10 beats · min⁻¹ · decade⁻¹ decline in maximum heart rate (Londeree & Moeschberger, 1982; Maritz, Morrison, Peter, Strydom, & Wyndham, 1961) might not be applicable in competitive athletes, although the standard error of around 10 beats/min was similar to the variation reported by Londeree and Moeschberger. These results therefore concur with those of Rogers et al. (1990), who concluded that the decline in maximal heart rate was less in master athletes than is typically observed within the general population.

As indicated previously, the intercept (at age 0 years) for the plot of maximum heart rate (beats/min) with age (years) for this study (Table 5) was 193 beats/min, which is below the 200 and 220 beats/min that are used in commonly applied algorithms for the general population (Maritz et al., 1961, and Londeree & Moeschberger, 1982, respectively). This suggests that highly trained endurance athletes might have a lower maximum heart rate than do nonathletic individuals. Indeed, as an example of this, during the past 6 years we have frequently tested an Olympic endurance runner with a VO_{2max} >80 ml · kg⁻¹ · min⁻¹, who at the age of 22 years had a peak heart rate of around 175 beats/min. Similar examples of young elite endurance runners with relatively low maximum heart rates were reported by Bailey and Davies (1999).

Furthermore, in our study the model produced for predicting 10-km running speed associated a fast running speed with a relatively low maximum heart rate. Such findings add further interest to the debate on what limits VO_{2max} and running speed in endurance events and what causes their decline with increasing age. The view that it is oxygen delivery rather than oxygen extraction, which primarily limits VO_{2max} (Bassett & Howley, 2000), would link Q_{max} , the product of maximum heart rate and stroke volume, to VO_{2max} . This by implication has meant that the observed reduction in maximum heart rate has been at least partly attributed as the cause of

the age-related decline in VO_{2max} (Tate, Hyek, & Taffet, 1994). Nonetheless, the findings of the current study argue against the importance of a high maximum heart rate for attaining a high VO_{2max}. Indeed, not only do the data provide individual cases that dispute this assumption but also, although not being statistically significant (*p* = .284), the average maximum heart rate for Group A was not higher but tended to be 4 beats/min lower than for Group B participants of comparable age, this being despite Group A having a higher VO_{2max} and running speed than Group B. Therefore, although there is a concomitant decline in maximum heart rate, VO_{2max}, and 10-km running speed with increasing age in highly trained endurance runners, as indicated in this and other studies, it might be a mistake to assume that the decline in maximum heart rate is the cause of the decline in VO_{2max} and running speed (Wiebe, Gledhill, Jamnik, & Ferguson, 1999). Indeed, according to Noakes's (1998) arguments, the decline in maximum heart rate might be the *consequence* of a lower VO_{2max}, not the *cause*.

In addition, although maximum stroke volume and left-ventricular volume were not assessed in this study, the role of Q_{max} might be further questioned in aging athletes by the failure of left-ventricular mass to correlate with age, peak VO_2 , or performance, either as an absolute value (g) or when scaled for body mass (g/kg BM or g/kg BM^{0.67}). Alternative explanations for declines in running speed with increasing age suggest peripheral factors such as reduction in muscle mass, lean body mass, and muscle power (Green & Patla, 1992; Hawkins, Marcell, Jaque, & Wiswell, 2001; Noakes, 1988). These reduce the oxygen-utilizing capacity of the muscles, and hence the demand for oxygen is reduced, a fact that would be exhibited as a lower VO_{2max} and possibly a reduced maximum heart rate but would not be caused by a lower maximum heart rate. Future studies that include the assessment of maximum stroke volume, left-ventricular volume, and Q_{max} are therefore warranted to determine their potential influence on VO_{2max} and performance.

A further point of interest from this study was the increase in the percent VO_{2peak} at which lactate threshold occurred with increasing age. This has been reported in a few other studies (Coggan et al., 1990; Wiswell et al., 2000), and whether it is linked to changes in lactate production or lactate clearance is not possible to say, but it does suggest peripheral adaptations relating to performance. This again brings into question the role of oxygen delivery in determining the running speed of older athletes in endurance events and suggests that researchers need to investigate peripheral factors that might change with age and could limit maximal workloads, which as a result would limit VO_{2max}, rather than vice versa.

The decline in early rate of change of displacement of the atrioventricular plane with increasing age in nonathletic individuals has previously been reported by Owen (1999) but has not, to our knowledge, been assessed in highly trained athletes. The rate of decline in our athletic population was $1 \text{ cm} \cdot \text{s}^{-1} \cdot \text{decade}^{-1}$, which is similar to the findings of Owen for participants over 50 years of age and thus suggests that long-term endurance training has no effect on this aspect of age-related changes in cardiac function.

The finding that velocity at lactate threshold was the strongest single predictor of running speed for 10 km agrees with those of Bassett and Howley (2000) and is in accordance with other previous work that found velocity at lactate threshold or individual anaerobic threshold to correlate strongly with 10-km running speed (Allen, Seals, Hurley, Ehsani, & Hagberg, 1985; Costill, Thomason, & Roberts,

1973; Nicholson & Sleivert, 2001; Roecker et al., 1998). Unsurprisingly, and in agreement with previously published work (Costill et al.; Foster, Costill, Daniels, & Fink, 1978; Takeshima & Tanaka, 1995), VO_{2peak} (ml·kg⁻¹·min⁻¹), VO_{2peak} (L/min), and VO_2 at lactate threshold (ml·kg⁻¹·min⁻¹) were also strong predictors of running speed in this study (r=.773, p<.001; r=.573, p<.001; r=.663, p<.001, respectively) but in our study were factored out of the prediction model by the dominance of velocity at lactate threshold. The inclusion of VO_{2peak} relative to body mass as a predictor of running speed in our model hence agrees with previous research that has studied heterogeneous groups (Costill et al.; Foster et al.) but differs in that in previous studies the standard was diverse but with a similar age range; in the present study the standard was similar relative to age, and the diversity of performance was largely related to the broad age range. Unlike the model of Takeshima and Tanaka, including age, per se, in our model did not improve the strength of prediction.

Including the early rate of change of displacement of the atrioventricular plane of the septum $(C\nu)$ in the prediction equation is a factor not previously identified and might be associated with the concomitant age-related decline in both running speed and $C\nu$ with increasing age. It should also be noted that although it provided a statistically significant contribution (p=.010), its addition to the predictive model only increased the model's predictive power by 1% and hence should be viewed with caution.

In summary, the results of this study have produced a model for predicting 10-km running speed in highly trained endurance runners across a broad span of ages. In proposing the model we acknowledge that although it strongly predicts running speed in the derivation sample it has yet to be evaluated on an independent sample, and therefore its applicability to 10-km runners in general is unknown. The model includes some previously reported predictors of performance such as running speed at lactate threshold and maximum oxygen utilization, as well as some additional factors (maximum heart rate and rate of relaxation of the left ventricle). Nonetheless, some of the results, in particular the data on maximum heart rate, do not appear to comply easily with the belief that it is oxygen delivery that limits performance in endurance-running events. The findings therefore suggest that further work is needed to assess the potential contribution of age-related changes in the maximal working capacity of skeletal muscle and the influences on age-related declines in endurance-running speed.

To attain real-world validity, 10-km races were used as an indicator of endurance-running performance, but because many different races were included, with differences in terrain and climatic conditions, some variability was almost certainly introduced. Hence, the predictive strength of the model in the derivation sample ($R^2 = 88\%$) might have been even stronger if the same 10-km race had been used. In terms of the future application of the model with an independent sample running in races not used in the derivation of the model, the production of a model derived from different races would seem more applicable.

The negative association between maximum heart rate and 10-km running speed raises some interesting questions concerning the contribution of maximum heart rate to performance, particularly the extent to which the decline in maximum heart rate with age might or might not be a causal factor in the decline of VO_{2max} with age. The inclusion of the rate of relaxation of the left ventricle in the predictive

equation is a new and interesting addition that warrants further investigation, as does the confirmation of the previously reported finding of an increase in the percent VO_{2peak} at which lactate threshold occurs.

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