

## Review

# Speed endurance training is a powerful stimulus for physiological adaptations and performance improvements of athletes

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The present article reviews the physiological and performance effects of speed endurance training consisting of exercise bouts at near maximal intensities in already trained subjects. Despite a reduction in training volume, speed endurance training of endurance-trained athletes can maintain the oxidative capacity and improve intense short-duration/repeated high-intensity exercise performance lasting 30 s to 4 min, as it occurs in a number of sports. When combined with a basic volume of training including some aerobic high-intensity sessions, speed endurance training is also useful in enhancing performance during longer events, e.g. 40 K cycling and 10 K running. Athletes in team sports involving intense exercise actions and endurance aspects can also

benefit from performing speed endurance training. These improvements don't appear to depend on changes in maximum oxygen uptake ( $\dot{V}O_{2\max}$ ), muscle substrate levels, glycolytic and oxidative enzymes activity, and membrane transport proteins involved in pH regulation. Instead they appear to be related to a reduced energy expenditure during submaximal exercise and a higher expression of muscle  $Na^+, K^+$  pump  $\alpha$ -subunits, which via a higher  $Na^+, K^+$  pump activity during exercise may delay fatigue development during intense exercise. In conclusion, athletes from disciplines involving periods of intense exercise can benefit from the inclusion of speed endurance sessions in their training programs.

The literature contains a high number of studies utilizing training at intensities higher than the one eliciting maximum oxygen uptake ( $\dot{V}O_{2\max}$ ), which have all indistinctively been referred to as sprint, intermittent and interval training (Ross & Leveritt, 2001; Laursen & Jenkins, 2002). It is often not recognized that these represent a broad variety of training protocols and a wide range of exercise intensities. For example, a well-trained cyclist may reach  $\dot{V}O_{2\max}$  at a power output of 400 W and be able to produce a peak power output (PPO) of  $\sim 1200$  W in a 10-s maximal test (Calbet et al., 2003). In this instance,  $\dot{V}O_{2\max}$  is obtained at  $\sim 33\%$  of PPO and the remaining  $\sim 67\%$  represents exercise intensities above the maximal aerobic power (which is defined as supra maximal exercise). Differences in the relative exercise intensity and duration of recovery periods should be taken into account when comparing scientific studies using supramaximal exercise. To clarify the issue, a common terminology would be helpful. Anaerobic training is defined as training where the exercise is performed at intensities above  $\dot{V}O_{2\max}$  and where the primary aim is to stimulate the anaerobic energy production (Bangsbo, 1994). Part of the anaerobic training is classified as speed training,

which consists of maximal short-duration (2–10 s) exercise bouts followed by long recovery periods (50–100 s) (Reilly & Bangsbo, 1998). Speed endurance training is used to describe all the other anaerobic intensities and has been divided into production and maintenance training (Reilly & Bangsbo, 1998). In “production training” the exercise bouts last less than 40 s and are performed at near maximal intensity, while the recovery periods are comparatively long ( $> 5$  times the exercise duration) in order to perform maximally in the subsequent exercise bouts. “Maintenance training” includes exercise bouts of 5–90 s with shorter rest periods in between the intervals ( $\leq 3$  fold longer than the exercise time), resulting in a slightly lower intensity and a progressive accumulation of fatigue as the training continues (Table 1).

The present brief review deals with the effect of speed endurance training on performance and physiological adaptations, but will mainly cover the effect of speed endurance training at near maximal intensities ( $> 70\%$  of maximal intensity) in already trained subjects. For the effects of exercise training at intensities around or slightly higher than the one corresponding to  $\dot{V}O_{2\max}$ , the reader is referred to

Table 1. Types of anaerobic training

Type of anaerobic training	Exercise intensity (% of maximum speed)	Duration of exercise (s)	Duration of recovery	No. of repetitions
Speed	100	2–10	50–100 s	5–20
Speed endurance production	70–100	10–40	> 5 times exercise duration	3–12
Speed endurance maintenance	50–100	5–90	1–3 times exercise duration	2–25

the articles by Laursen and Mujika in the present journal issue. A significant number of studies have examined the muscle adaptations with speed endurance training in untrained subjects (Ross & Leveritt, 2001; Burgomaster et al., 2005; Gibala et al., 2006). However, these are of limited interest in the discussion of how to train athletes, but will be included where they may add information to the data obtained in athletes.

**Physiological response to speed endurance training**

Before discussing its effects, it is useful to present the physiological and metabolic response to speed endurance training. In a study by Mohr et al. (2007), a group of physical active subjects performed eight 30-s runs at ~ 130% of  $\dot{V}O_{2max}$  (158–170 m) separated by 90 s of rest. The heart rate fluctuated with peaks around 97% of the maximal heart rate ( $HR_{max}$ ) and mean heart rate of ~ 84% of  $HR_{max}$ , reflecting a high cardiovascular stimulus. Blood lactate concentration [ $Lac^-$ ] increased progressively during the session from 1.1 to 16.5 mM (Fig. 1a), with muscle [ $Lac^-$ ] rising to ~ 45 mmol/kg d.w. Correspondingly, muscle pH was lowered to 6.98 during the training. Plasma  $K^+$  concentration varied throughout the training session and peaked at 6.3 mmol/L (Fig. 1b). Immediately after the training session muscle creatine phosphate (PCr) and glycogen levels were lowered to 41 and 350 mmol/kg d.w., respectively. In another experiment Bogdanis et al. (1996a) examined the metabolic response when repeating two 30-s maximal cycling bouts separated by 4 min of rest. Muscle pH decreased to 6.69 immediately after the first bout and increased only slightly (6.80) prior to the second sprint, after which it dropped to 6.61. Consistently, muscle [ $Lac^-$ ] rose to 108 and 129 mmol/kg d.w. at the end of the first and second bout, respectively. The muscle PCr levels at the end of the first and second bout were similar (12.6 and 8.8 mmol/kg d.w., respectively) and the PCr was resynthesized to 58.5 mmol/kg d.w. (79% of the resting value) before the second exercise bout. The muscle glycogen content decreased from 327 to 228 and 184 mmol/kg d.w. at the end of the first and the second bout, respectively. Plasma  $NH_3$  concentration was elevated from 29 to 94  $\mu$ mol/L immediately after the first 30-s sprint, and increased

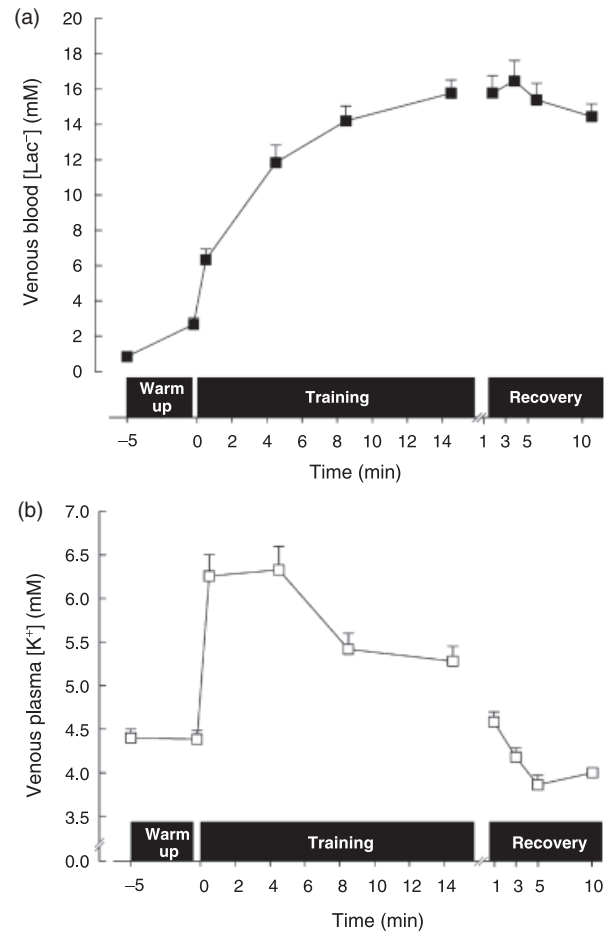


Fig. 1. Venous blood lactate (a) and plasma  $K^+$  concentrations (b) during a speed endurance training session.

throughout recovery peaking at 170  $\mu$ mol/L 3.5 min after sprint 2 (Bogdanis et al., 1995; Bogdanis et al., 1996b). In addition, the calculated ATP turnover was markedly different when repeating 2 30-s maximal sprints. PCr, energy release from glycolysis and aerobic metabolism accounted for 21, 50 and 29%, respectively, of the total energy production during the first sprint, and changed to 20, 36 and 44% in the second bout (Nevill et al., 1994). Consistently, when performing 4 repeated 30-s maximal cycling exercises separated by 4 min of rest, Parolin et al. (1999) observed a decreased substrate phosphorylation by phosphocreatine hydrolysis and glycolysis, and a concomitant shift toward greater reliance on

oxidative phosphorylation. These data taken together with those by Bangsbo et al. (2001), Medbo & Tabata (1989) and Tabata et al. (1997) indicate that speed endurance training taxes both the anaerobic and aerobic energy pathways to a significant extent, with the rate of glycolysis progressively decreasing and the aerobic energy production increasing as the exercise is repeated.

### **Effect of speed endurance training on physical performance in trained human subjects**

#### *Short-term and repeated intense exercise performance*

There is a general consensus that a period of speed endurance training in endurance-trained subjects leads to marked performance improvements during supramaximal and repeated high-intensity exercise lasting less than  $\sim 10$  min (Tables 2 and 3). Houston & Thomson (1977) observed that after a 6-week program involving anaerobic hill running, five endurance-trained men increased the mean distance covered in 2 maximal runs of 60 s and 90 s each by  $\sim 13\%$ . Similarly, Daniels et al. (1978) showed a  $\sim 10\%$  lower time over  $\sim 800$  m ( $\sim 130$  s) when trained runners performed part of their training as 100–600 m sprints. More recently, others reported improved time to exhaustion (11.2%) (Bickham et al., 2006), predicted speed (4.4%) (Hamilton et al., 2006) and mean power (8.7%) (Paton & Hopkins, 2005) during various supramaximal exercises lasting 2–3 min. These changes are, however, lower than those found in other investigations (Table 3). Part of the discrepancies observed could be due to the different testing protocols (i.e. time trial vs time to exhaustion) and measurements (i.e. time, distance, speed, power) utilized. Nevertheless, it appears from Table 2 that the three studies showing the greatest increases in performance used speed endurance production training involving 30-s near maximal intensity bouts with relatively long rest periods ( $> 1:4$ ) (Roberts et al., 1982; Iaia et al., 2008; Bangsbo et al., 2009). In addition, two out of the three had also a concomitant significant reduction in the amount of training. Thus, speed endurance production training with 30-s all-out exercise and  $\sim 3$  min rest is a very potent stimulus and a time efficient strategy for markedly improving performance during very intense short-term exercise, i.e. from 30 s to 3 min duration. Furthermore, these pronounced performance improvements were obtained under conditions of reduced weekly mileage, clearly indicating that high training volumes may not be needed, and in some cases may even be detrimental for improvements during supramaximal exercise performances. This information is relevant for athletes participating in sports such as 400, 800 and 1500 m track events,

1000 m cycling, 100–200 m swimming as well as team sports.

Significant increases in performance are also observed in events lasting 4–6 min with the studies involving shorter recovery times between the intervals (e.g. 30-s exercise separated by 30-s rest periods) (Paton & Hopkins, 2005; Hamilton et al., 2006) showing less pronounced improvements as compared with those using longer resting periods (Shepley et al., 1992; Esfarjani & Laursen, 2007). However, at present there is insufficient data and research comparing speed endurance production vs. maintenance training to provide clear evidence based recommendations about the most appropriate exercise: rest ratio. The recommendations of the type of protocol (intensity, exercise duration and rest) are dependent also upon the type of performance improvements and adaptations an athlete is looking for. In general it appears that “production training” (exercise rest ratio of  $\sim 1:6$ ) is beneficial for improving the ability to perform maximal efforts repeatedly, whereas “maintenance training” (1:1–3) may be effective in increasing the ability to sustain exercise at high intensity.

The effect of speed endurance training has been investigated also in football (soccer). In a series of studies, elite football players underwent 2–10 weeks of intensified training including 1–3 weekly sessions of various speed endurance training (Table 2) and improved their repeated sprint ability (1.9–2.1%) (Ferrari Bravo et al., 2008; Thomassen et al., 2010), maximal aerobic speed (8.1%) (Dupont et al., 2004) and Yo-Yo intermittent recovery (Yo-Yo IR) test (Bangsbo et al., 2008) performance (22–28%) (Ferrari Bravo et al., 2008; Hill-Haas et al., 2009). The latter is in agreement with Iaia et al. (2008) who observed a 19% increase in Yo-Yo IR level 2 performance when for a 4-week period trained runners replaced their endurance training with 3–4 weekly sessions of speed endurance production training. The potency and time-efficiency of very high-intensity training is also evident from a recent study by Thomassen et al. (2010). Trained football players improved their performance during a repeated sprint test, in association with an increased expression of some muscle proteins involved in ion homeostasis, after a 2-week reduction in training volume incorporating 5 aerobic high-intensity and 5 speed endurance training sessions. Thus, speed endurance training is beneficial for people participating in team sports involving intense activities by elevating their ability to recover faster after the intense phases of a match and thereby potentially increasing the number of high-intensity periods during a game. Less clear is the effect of speed endurance training on sprint and jump performance with studies reporting either improved or unchanged performances (Table 3).

Table 2. Effect of speed endurance training on physiological adaptation and performance in trained human subjects

Study	Fitness status and exercise mode	n	Protocol	Intensity	Duration (week)	Changes in training volume	Performance changes	Adaptations	Physiological response to exercise
Bangsbo et al. (2009)	Well-trained runners ( $\dot{V}O_{2\max}$ 63 mL/kg/min, 3 km time 10.4 min, 10 km time 37.3 min)	12	8-12 × 30s-3 min rest, 2-3 × wk	90-95% speed achieved over 30-s run at full maximal effort	6-9	↓ ~ 30% (from ~ 50 to ~ 35 km/wk)	19% ~ 8-min incremental exhaustive test ↑ ~ 6% distance 30-s max test ↑ 36% time to exhaustion 130% $\dot{V}O_{2\max}$ (~ 2 min) ↑ 24% (NS) NKCC1 16% (NS) time to exhaustion 130% $\dot{V}O_{2\max}$ (~ 70 s, EX2) 13.3% 3 km performance 13.1% 10 km performance	↑ 2.7% (NS) $\dot{V}O_{2\max}$ ↑ 68% $Na^+K^+$ pump $\alpha 2$ ↑ 10% (NS) $Na^+K^+$ pump $\beta 1$ ↔ $Na^+K^+$ pump $\alpha 1$ ↔ NHE1, MCT1, MCT4 ↔ CK, PFK, GS, HAD	↔ HR during 3 and 10 k ↑ 3% running economy at 12 km/h ↓ RER at 17 km/h <sup>1</sup> ↑ peak blood [La <sup>-</sup> ] after incremental test ↓ [K <sup>+</sup> ] <sub>iv</sub> after EX1 and EX2
Bickham et al. (2006)	Moderately endurance-trained runners ( $\dot{V}O_{2\max}$ ~ 58 mL/kg/min)	7	4 × (4-8 × 40-100 m sprints - jog 3-5 fold ex time) - 5-min jog between sets, 3 × wk	90-100% maximal	6	Unchanged (~ 50 km/wk aerobic moderate intensity)	↑ 11% time to exhaustion at 110% $\dot{V}O_{2\max}$ (2.3 min)	↓ 2% (NS) $\dot{V}O_{2\max}$ ↑ ~ 45% MCT1 ↔ MCT4 ↓ 11% ST fibers ↑ 14% FT fibers ↔ body mass	
Daniels et al. (1978)	Trained runners ( $\dot{V}O_{2\max}$ ~ 63 mL/kg/min, 800 m time ~ 2 min)	15	5 subjects run half of their training as 100-600 m sprint runs	Not specified	8	↑ From 20-30 to 50-70 km/wk	↑ ~ 10% ~ 800 m performance ↑ ~ 15% ~ 3 km performance	↔ $\dot{V}O_{2\max}$	
Dupont et al. (2004)	Professional football players - running	22	• 12-15 × (40 m-30 s rest), 1 × wk. • 2 × (12-15 × 15 s-15 s rest), 1 × wk	Maximal 120% max aerobic speed	10	Not specifically reported but apparently maintained	18.1% max aerobic speed ↓ 3.5% 40 m sprint time (150% games won)	↔ body fat ↔ body mass ↔ HR	
Esfarjani & Laursen (2007)	Moderately trained runners ( $\dot{V}O_{2\max}$ ~ 52 mL/kg/min, 3000 m time ~ 11 min)	6	12 × 30 s-4.5 min jog, 2 × wk	130% $\dot{V}O_{2\max}$	10	Unchanged (~ 36 km/wk), 2 × 60-min treadmill run at 75% $\dot{V}O_{2\max}$	17.3% 3000 m time trial 17.8% speed at $\dot{V}O_{2\max}$ 132% time to exhaustion at $\dot{V}O_{2\max}$ (~ 6 min)	↑ 6.2% $\dot{V}O_{2\max}$	↑ 4.7% (NS) speed at lactate threshold
Ferrari Bravo et al., (2008)	Sub-elite football players - running ( $\dot{V}O_{2\max}$ ~ 56 mL/kg/min)	13	3 × (6 × 20+20 m sprint -20 s rest) - 3 min rest, 2 × wk	Maximal	7	Maintained	128.1% Yo-Yo IR1 12.1% repeated sprint ability ↓ 0.6% (NS) 10 m sprint time ↔ squat jump height, power ↔ CMJ height, power	↑ 5% $\dot{V}O_{2\max}$ ↑ 3% $V_{T2}$	
Hamilton et al. (2006)	Trained runners ( $\dot{V}O_{2\max}$ 66 mL/kg/min, 5 km time <20 min)	10	3 × (20 explosive 1-leg jumps)+3 × (5 × 30 s-30 s rest), 1-3 × wk	Maximal 65% peak running speed	7	Pre training ≥ 30 km ↓ ~ 17% (3.6-3 h/wk)	14.4% predicted 800 m speed 14.1% predicted 1500 m speed 12.7% peak incremental speed ↔ 2.2% 5 km time trial	↔ body mass ↔ thigh cross-sectional area	↑ 4.0% speed at 4 mM [La <sup>-</sup> ]
Hill-Heas et al. (2009)	Junior elite football players ( $\dot{V}O_{2\max}$ ~ 60 mL/kg/min)	9	• 18 to 20 × 30-60 s 60-90 s rest, 1 × wk • 7 × 34 m-35 s rest+ 2-10 × 5-90 s 15-90 s rest, 1 × wk OR • 30-35 × 10-20 m, 10-40 s rest, 1 × wk	90-95% HR <sub>max</sub> Maximal Maximal	7	Maintained	122% Yo-Yo IR1 ↔ repeated sprint ability 12.5% (NS) ~ 10-min incremental exhaustive test 13.1% (NS) multistage fitness test ↓ 0.9% (NS) 5 m sprint time ↓ 1.5% (NS) 20 m sprint time	↑ 2% (NS) $\dot{V}O_{2\max}$	

## Speed endurance training in trained subjects

Houston and Thomson (1977)	Endurance-trained runners ( $\dot{V}O_{2\max}$ ~ 59 mL/kg/min)	5	3 × (60 s–2 min jog) 3.3% (inclination) +5 × (6 s–24 s rest) 44% +2 × (90 s–3 min jog) 3.3% +15 leg press max rep., 4 × wk	6	Maximal	Similar (~ 35 km/wk). No aerobic exercise except ~ 3 km warm-up	↔ Stair-sprinting power test ↑ 16.7% time to exhaustion steep (20%) treadmill run (~ 50-s) ↑ 13.7% distance 60-s full maximal effort (3.3% grade) ↑ 13.4% distance 90-s full maximal effort (3.3% grade)	13% (NS) $\dot{V}O_{2\max}$ 13.3% (NS) FI fiber ↓ 4% (NS) LDH ↓ 14.8% ATP at rest 14.5% (NS) PCr at rest ↓ 13.7% cholesterol ↔ body fat ↔ body mass	↑ 14% peak blood [ $La^-$ ]
laia et al. (2008)	Moderately endurance-trained runners ( $\dot{V}O_{2\max}$ ~ 55 mL/kg/min, 10 km time ~ 40 min)	8	8–12 × 30 s–3 min rest, 3–4 × wk	4	90–95% speed achieved over 30-s run at full-maximal effort	↓ ~ 64% (from ~ 43 to ~ 15 km/wk) (aerobic exercise only for warm-up and cool down activities)	15% ~ 7-min incremental exhaustive test 17% distance 30-s max test 126% time to exhaustion 130% $\dot{V}O_{2\max}$ (~ 100 s, EX1) ↑ 19% time to exhaustion 130% $\dot{V}O_{2\max}$ (~ 75 s, EX2) ↑ 19% Yo-Yo IR2	↓ 2% (NS) $\dot{V}O_{2\max}$ 129% $Na^+K^+$ pump $\alpha 1$ 117% (NS) $Na^+K^+$ pump $\alpha 2$ ↔ $Na^+K^+$ pump $\beta 1$ 130% NHE1 114% (NS) NKCC1 ↔ $\beta$ <i>in vitro</i> , MCT1, MCT4, ↔ CK, PFK ↔ body mass	↓ $[K^+]_v$ in and after EX1 ↓ glycogen degr. in EX2 ↑ $H^+$ removal after EX1 ↔ PCr, lact glycoген, [ $H^+$ ] at rest and during exercise
laia et al. (2009)	Moderately endurance-trained runners ( $\dot{V}O_{2\max}$ ~ 55 mL/kg/min, 10 km time ~ 40 min)	9	8–12 × 30 s–3 min rest, 3–4 × wk	4	90–95% speed achieved over 30-s run at full maximal effort	↓ ~ 65% (from ~ 43 to ~ 15 km/wk) (aerobic exercise only for warm-up and cool down activities)	↔ 10 km time trial	↓ 2% (NS) $\dot{V}O_{2\max}$ ↔ CS, HAD 141% (NS) UCP3 ↓ 7% (NS) ST fibers ↓ 6% (NS) F1a fibers 144% F1x fibers 17% (NS) No. capillaries	↑ 6–7% running economy ↓ energy expenditure, HR and ventilation during submaximal running ↔ blood [ $La^-$ ] during submaximal running
Laursen et al. (2002)	Highly trained cyclists ( $\dot{V}O_{2\max}$ ~ 64 mL/kg/min)	10	12 × 30 s–4.5 min rec., 2 × wk	4	175% PP	Maintained regular low-intensity training	↑ 3% PP 14.4% 40 km time trial ↔ time to exhaustion at PP (~ 4 min)	13% $\dot{V}O_{2\max}$	
Laursen et al. (2005)	Highly trained cyclists ( $\dot{V}O_{2\max}$ ~ 64 mL/kg/min)	10	12 × 30 s–4.5 min rec., 2 × wk	4	175% PP	Maintained regular low-intensity training		14% (NS) $\dot{V}O_{2\max}$ ↔ plasma protein content ↔ hematoцит ↔ hemoglobin ↔ plasma volume ↑ 17% $VT_1$ 19% $VT_2$ 175% anaerobic capacity	↑ $\dot{V}O_{2\max}$ -peak and HR during 40 km time trial ↔ RER and blood [ $La^-$ ] during 40 km time trial
Paton and Hopkins (2005)	Well-trained cyclists	9	3 × (20 single-leg jumps) + 3 × (5 × 30 s – 30 s rest), 2 min rest, 2–3 × wk	4–5	Maximal	Replaced 20% of their total weekly volume	16.7% PP 18.7% 1 km mean power ↑ 8.4% 4 km mean power	↔ body mass	↓ 3.2% energy expenditure
Roberts et al. (1982)	Regularly active – running	4	8 × 200 m – 2 min rest, 3–4 × wk	5	90% of max speed over 200 m	Not reported	125% 40-s exhaustive run (16 km/h, 15% grade)	↑ ~ 110% PFK ↑ ~ 50% Phos ↑ ~ 50% GAPDH ↑ ~ 50% LDH ↑ ~ 15% MDH ↑ 17% (NS) SDH	

Table 2. (continued)

Study	Fitness status and exercise mode	n	Protocol	Intensity	Duration (week)	Changes in training volume	Performance changes	Adaptations	Physiological response to exercise
Shepley et al. (1992)	Highly trained distance runners ( $\dot{V}O_{2\max}$ ~ 67 mL/kg/min)	9	1–5 × (500 m – 6–7 min walk), 5 × wk	115–120% $\dot{V}O_{2\max}$	1	From 80 to 7.5 km/wk (warm up excluded)	122% treadmill run to exhaustion at ~ 115% $\dot{V}O_{2\max}$ (~ 4 min) 113% (NS) peak torque	↔ $\dot{V}O_{2\max}$ ↑18% CS ↑15% rest glycogen ↑ ~ 5% blood volume ↑ ~ 16% red cell volume ↔ hematocrit	↔ post-exercise plasma [La <sup>-</sup> ]
Stepfo et al. (1999h)	Endurance-trained cyclists ( $\dot{V}O_{2\max}$ ~ 60 mL/kg/min)	4	12 × 30 s–4.5 min rec., 2 × wk	175% PP	3	Not reported	↑ ~ 0.5% (NS) PP ↑ ~ 3.8% (NS) 25 kJ sprint power ↑ ~ 2.3% 40 km (~ 55 min) time trial	↔ HR, carbohydrate oxidation and plasma [La <sup>-</sup> ]	
Tabata et al. (1996)	Physical education students from various sport – cycling ( $\dot{V}O_{2\max}$ ~ 48 mL/kg/min)	7	7–8 × (20 s–10 s rest), 5 × wk	170% $\dot{V}O_{2\max}$	6	Not reported		↑ ~ 15% $\dot{V}O_{2\max}$	
Thomassen et al. (2010)	Elite football players ( $\dot{V}O_{2\max}$ ~ 55 mL/kg/min)	7	• 8 × 2 min – 1 min rest, 2–3 × wk • 10–12 × 25–30 s–2.5–3 min rest, 2 × wk	~ 90% HR <sub>max</sub> 90–95% speed achieved over 30-s run at full maximal effort	2	↓ ~ 30%	11.9% repeated sprint ability 16.1% (NS) Yo-Yo IRI ↔ 20 m sprint time	↑14.5% Na <sup>+</sup> , K <sup>+</sup> pump $\alpha$ 2 127.3% resting phosphorylation status FXVD1 ↑ ~ 18% (NS) Na <sup>+</sup> , K <sup>+</sup> pump $\alpha$ 1 ↔ Na <sup>+</sup> , K <sup>+</sup> pump $\beta$ 1 ↑ ~ 13% (NS) MCT1, MCT4 ↔ NHE1, NKCC1	
Thorstensson et al. (1975)	Moderately trained – running ( $\dot{V}O_{2\max}$ ~ 54 mL/kg/min, 10 km time ~ 40 min)	4	20–40 × (5 s sprint – 25–55 s rest), 3–4 × wk	Maximal	8	Not reported	↓1% (NS) 25 m sprint time ↑12% MVC 19% Jump 14% (NS) Margaria test ↑19% endurance time at 50% MVC (~ 50 s)	136% CK ↑20% MK 130% ATPase ↔ resting ATP, CP 13% (NS) $\dot{V}O_{2\max}$ ↓5% (NS) FTb fibers 12% body mass 13% thigh girth	

ATPase, adenosine triphosphatase; CMJ, counter movement jump; CK, creatine kinase; CS, citrate synthase; FXVD1, Na<sup>+</sup>, K<sup>+</sup> pump accessory and regulatory protein phospholemman; FT, fast twitch; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; HAD, 3-hydroxyacyl-CoA dehydrogenase; HR, heart rate; LDH, lactate dehydrogenase; MCT, monocarboxylate transporters; MDH, malate dehydrogenase; MK, myokinase; MVC, maximal voluntary contraction; NHE1, Na<sup>+</sup>/H<sup>+</sup> exchanger isoform 1; NKCC1, Na<sup>+</sup>-K<sup>+</sup>-2Cl<sup>-</sup> 1 protein co-transporters; PCr, creatine phosphate; PFK, phosphofructokinase; Phos, glycogen phosphorylase; PP, peak aerobic power; RER, respiratory equivalent ratio; SDH, succinate dehydrogenase; ST, slow twitch; UCP3, uncoupling protein 3;  $\dot{V}O_{2\max}$ , maximum oxygen uptake; VT, ventilatory threshold; Yo-Yo IR, Yo-Yo intermittent recovery test; ↑, increased; ↓, decreased; ↔, unchanged; NS, non-significant; wk, week.

### *Medium- to long-term exercise performance*

In the study by Iaia et al. (2009) when already trained runners switched from regular endurance to speed endurance training, the 10 K performance was unaltered despite a 65% reduction in training distance. Apparently, speed endurance training *per se* is sufficient to maintain endurance performance of already trained subjects. This is in accordance with Houmard et al. (1990) who did not find any changes in a 5 K race after a 3-week period in which well-conditioned runners maintained the intensity while reducing their weekly training volume by 70% and frequency by 17%. Bangsbo et al. (2009) even reported an improved performance in 3 and 10 K runs after a 6–9-week period with speed endurance sessions where the total amount of training was reduced by 30%. In addition, other studies on trained cyclists observed a lower time (4–5%) to complete a simulated-laboratory 40 K cycling trial (Stepto et al., 1999a; Laursen et al., 2002), clearly indicating that when combining a basic volume of training including some aerobic high-intensity exercise, together with only 2 weekly sessions of speed endurance training, trained subjects can improve endurance performance. It is interesting to note that in the study by Laursen et al. (2002) the endurance performance was improved after 4 but not after 2 weeks of speed endurance training, which is in contrast with the finding of untrained subjects where pronounced changes were found already after 2 weeks (Burgomaster et al., 2005). Apparently, trained individuals adapt more slowly compared with untrained people. Nonetheless, the improvements observed in endurance events are of a lower magnitude than those reported during intense short-duration exercises (Table 3). The findings in the abovementioned studies taken together with those by Daniels et al. (1978), who observed greater 3 K improvements when combining speed endurance training with augmented training volume (Table 3), suggest that for maximizing performance improvements, distance athletes should also regularly carry out other types of training, e.g. aerobic high-intensity, besides speed endurance training.

### **Effect of speed endurance training on $\dot{V}O_{2\max}$ and energy expenditure in trained human subjects**

Most of the studies on well-trained individuals evaluating the effect of speed endurance production training with an intensity higher than 90% of the maximum have reported unchanged  $\dot{V}O_{2\max}$  values. This suggests that short maximal or near maximal efforts are not sufficient to enhance  $\dot{V}O_{2\max}$ . On the other hand, studies dealing with speed endurance maintenance training where the average intensity was lower, showed  $\dot{V}O_{2\max}$  improvements (Tabata et al.,

1996; Laursen et al., 2002; Ferrari Bravo et al., 2008). However, in two studies the participants were not highly trained (Tabata et al., 1996) and the total weekly volume was maintained (Laursen et al., 2002). Furthermore, in some investigations brief rest periods were used between repetitions, which may have allowed the cardiovascular stimulus to be maintained at a high level for longer durations (Tabata et al., 1996; Ferrari Bravo et al., 2008). Nevertheless, the importance of  $\dot{V}O_{2\max}$  at an elite level is questionable as often no relationship between  $\dot{V}O_{2\max}$  and performance are observed in athletes (Svedenhag & Sjodin, 1985; Coyle, 1995) and in the majority of the studies the improved short- and long-term exercise performances were not associated with  $\dot{V}O_{2\max}$  enhancements (Table 2).

Speed endurance training has been shown to reduce energy expenditure during exercise (Table 3), as is the case after plyometric and sprint training (Paavolainen et al., 1999; Turner et al., 2003; Saunders et al., 2006). In addition, improved performances were associated with a reduced mean net rate of glycogen degradation during supramaximal exercise (Iaia et al., 2009) and an elevated fat oxidation during intense submaximal exercise (Bangsbo et al., 2009). Thus, in trained individuals speed endurance training appears to be a potent stimulus to decrease the net rate of muscle glycogenolysis during submaximal exercise as also observed for untrained subjects (Harmer et al., 2000; Burgomaster et al., 2006).

### **Muscle adaptations to speed endurance training in trained human subjects**

While the alterations in performance occurring after a period of speed endurance training in trained subjects have been extensively investigated, only a few studies have focused on examining the changes in muscle variables with this type of training (Table 2).

#### *Fiber type proportion*

To what extent muscle fibers can shift from slow twitch (ST) to fast twitch (FT) fibers in humans and *vice versa* is still a matter of controversy. Nevertheless, the proportion of ST muscle fibers has been shown to decrease and the relative number of FT has been demonstrated to increase or remain unaltered with anaerobic training (Table 3). In addition, a significant increase in FTx fibers was observed when speed endurance training was accompanied by a severe reduction in training volume (Iaia et al., 2009). Thus, the few studies being performed do not lead to a firm conclusion, but they appear to suggest that speed endurance training of endurance trained

## Iaia & Bangsbo

Table 3. Summary of the effects (expressed as percentage change) of speed endurance training on physiological adaptation and performance in trained human subjects

	Significantly increased (% change)	Maintained (% change)	Significantly decreased (% change)
<b>Anthropometric characteristics</b>			
Body mass	2	0.1, 1, -1.6, 0.3, 0.2, 0.2	
Body fat		-6, -0.7	
Thigh girth	3	1.4	
<b>Exercise performances</b>			
30 s	7, 7		
40-60 s	25, 16.7, 13.7, 19		
1.5-2.5 min	27, 11, 13.4, ~ 10, 4.4, 8.7, 36		
4-6 min	22, 32, 4.1, 8.4	‡	
8-20 min	9*, 3.3, ~ 15, 8.1*, 7.3, 2.7*, 5*, 3*, 6.7*	2.2, 2.5*, 3.1*, ~ 0.5*	
35-60 min	4.4, ~ 2.3, 3.1	0.2	
Repeated intense exercise to exhaustion	19, 28.1, 22	6.1	
Repeated sprint ability	2.1, 1.9	0.2	
Sprint time		-0.6, -0.9, -1.5, 0, -1	-3.5
Jump test	9	0	
<b>Pulmonary variables</b>			
$\dot{V}O_{2max}$	6.2, 3, 5, 15 <sup>†</sup>	3, ~ 0, -2, -2, 3, 0.1, 4, 2.7, 2	
Energy expenditure			-7, -3.2, -3
<b>Blood variables</b>			
Blood/plasma volume	5	1.7	
Cholesterol			-13.7
Hematocrit		3, -0.2	
<b>Muscle substrates</b>			
ATP		6	-14.8
PCr		4.5, -12, -14	
Glycogen	15	-3	
<b>Muscle anaerobic enzymes</b>			
ATPase	30		
CK	36	0.3, 10.4	
GAPDH	50 <sup>†</sup>		
LDH	50 <sup>†</sup>	-4	
MK	20		
PFK	110 <sup>†</sup>	1, 10.5	
Phos	50 <sup>†</sup>		
<b>Muscle oxidative enzymes</b>			
CS	18	-5, -3.5	
HAD		2, -11	
MDH	15 <sup>†</sup>		
SDH		17 <sup>†</sup>	
<b>Muscle membrane transport protein</b>			
MCT1	45	-3, -2, 13.3	
MCT4		3, ~ 8, -10, ~ 13	
Na <sup>+</sup> ,K <sup>+</sup> pump $\alpha$ 1	29	~ -10, ~ 18	
Na <sup>+</sup> ,K <sup>+</sup> pump $\alpha$ 2	68, 14.5	17	
Na <sup>+</sup> ,K <sup>+</sup> pump $\beta$ 1		1, 10, ~ 4	
NHE1	30	1, ~ -4	
NKCC1		14, 24, 0	
<b>Muscle histochemical variables</b>			
Capillarization		7	
Fiber type I		-7	-11
Fiber type IIa	14 (FT)	-6, 3.3 (FT)	
Fiber type IIx	44	-5	
<b>Muscle buffering capacity</b>			
		-3	

\*Incremental test trial.

<sup>†</sup>Physically regularly active.

<sup>‡</sup>Percentage change not reported.

Data taken from studies reported in Table 2.

ATPase, adenosine triphosphatase; CK, creatine kinase; CS, citrate synthase; Fr, Fast Twitch; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; HAD, 3-hydroxyacyl-CoA dehydrogenase; LDH, lactate dehydrogenase; MCT, monocarboxylate transporters; MDH, malate dehydrogenase; MK, myokinase; NHE1, Na<sup>+</sup>/H<sup>+</sup> exchanger isoform 1; NKCC1, Na<sup>+</sup>-K<sup>+</sup>-2Cl<sup>-</sup> 1 protein co-transporters; PCr, creatine phosphate; PFK, phosphofructokinase; Phos, glycogen phosphorylase; SDH, succinate dehydrogenase;  $\dot{V}O_{2max}$ , maximum oxygen uptake.



subjects leads to a higher relative number of FT fibers.

### *Muscle metabolites, enzymes and capillarization*

One study has shown a reduced amount of muscle resting ATP concentration with speed endurance training in trained subjects (Houston & Thomson, 1977) and another an unaltered level (Thorstensson et al., 1975). The difference may be related to the extensive speed endurance training protocol used in the former study which required subjects to run to exhaustion. This is in contrast with Thorstensson et al. (1975) who employed shorter exercise bouts and longer recovery periods. In accordance, sprint training of untrained people has been shown to decrease the resting levels of skeletal muscle adenine nucleotides, if the training was frequent and very intense (Hellsten-Westing et al., 1993). However, it does not appear to have a major negative impact on work capacity as all the studies have shown increases in exercise performance. The muscle concentration of PCr does not appear to be affected by speed endurance training. One study has shown increased levels of muscle glycogen, whereas in another investigation they were maintained (Table 3). Importantly, it is not easy to examine the effect of training on muscle glycogen in athletes as the control level may be influenced by the frequent training sessions. Nevertheless, it is doubtful whether a change in muscle glycogen plays a role in the performance improvement during a single high-intensity exercise bout as muscle glycogen does not appear to be a limiting factor in short-term intense exercise (Bangsbo et al., 1992a). On the other hand, elevated glycogen levels in the working fibers are beneficial in sports with repeated intense exercise such as soccer and rugby (Bangsbo et al., 1992b; Balsom et al., 1999).

The effect of speed endurance training on the activity of enzymes related to the anaerobic metabolism of trained individuals is not clear. Roberts et al. (1982) found that the maximal activity of glycolytic enzymes and creatine kinase (CK) was increased by speed endurance training (Table 3). However, the participants in this study were regularly active but not well trained and studies enrolling trained subjects have not been able to show changes (Iaia et al., 2008; Bangsbo et al., 2009). It is traditionally believed that in order to enhance the activity of metabolic enzymes it is fundamental to utilize an exercise mode that produces high flux through the relevant pathways in the contracting muscles. It was recently reported that CK activity was higher when a group of untrained subjects performed a sprint-training program consisting of 6-s maximal runs separated by 1 min of recovery, whereas it remained unchanged for another group performing 30-s runs at a speed

eliciting  $\sim 130\% \dot{V}O_{2\max}$ , suggesting that a high rate of PCr breakdown is an important stimulus for CK adaptation (Mohr et al., 2007). In agreement, a considerable number of studies on untrained people have shown improvements in anaerobic enzymes activity after periods of training including very short maximal/near maximal exercise bouts (5–30 s) interspersed with relatively long resting periods (45 s to 20 min) (Costill et al., 1979; Jacobs et al., 1987; Linossier et al., 1993; Hellsten-Westing et al., 1993; Linossier et al., 1997; MacDougall et al., 1998; Parra et al., 2000; Rodas et al., 2000). On the other hand, in a study (Barnett et al., 2004) utilizing a training protocol (30-s sprints with 3–4 min of rest) similar to the one used by Iaia et al. (2008) and Bangsbo et al. (2009), the activity of phosphofructokinase (PFK) remained unaltered. Thus, the higher fitness level together with longer exercise periods (i.e. 30s) may explain why no changes in the activity of CK and PFK were observed in the latter two studies (Iaia et al., 2008; Bangsbo et al., 2009). Nevertheless, pronounced alterations in short-term performance were observed despite unaltered levels of enzymes related to anaerobic energy production (Iaia et al., 2008; Bangsbo et al., 2009), suggesting that the changes in the activity of these enzymes are not crucial for work capacity improvements during exercise lasting 30 s to 2 min.

The activity of oxidative enzymes in trained subjects does not appear to be elevated with speed endurance training (Table 3), which is clearly in contrast to a number of studies of untrained subjects (Rodas et al., 2000; Parra et al., 2000; Burgomaster et al., 2005; Burgomaster et al., 2006; Gibala et al., 2006; Burgomaster et al., 2008). This difference may be related to the lower level of enzyme activity in untrained as compared with already trained individuals and to the fact that untrained subjects do respond to almost all types of training stimuli by increasing a large number of muscle proteins. It is interesting to note, however, that in a study with a 65% reduction in training volume, the levels of citrate synthase (CS) and 3-hydroxyacyl-CoA dehydrogenase (HAD) were maintained with regular speed endurance training, indicating that this type of training also in trained humans stimulates the mitochondrial oxidative proteins (Iaia et al., 2009). In accordance, Shepley et al. (1992), investigating highly trained runners during a week of reduced training combined with speed endurance training, found an 18% increase in the CS level in association with improved performance (Table 2). Thus, it appears that speed endurance training is a powerful stimulus to mitochondrial protein synthesis and whether it leads to net synthesis depends on the training status as well as on the frequency and amount of training.

There is limited information about the effect of speed endurance training on muscle capillarization. Iaia et al. (2009) observed a non-significant 7% increase in the number of capillaries per fiber despite a 65% reduction in the total amount of training. The lack of significance may have been due to the fact that the two best trained subjects showed a decrease in the number of capillaries. These findings may suggest that speed endurance training is a stimulus for muscle angiogenesis in trained subjects. However, a basic volume of training may also be necessary in order to increase the capillarization. Further studies are needed to examine this hypothesis.

### *Muscle ion transport proteins*

Among the wide range of potential candidates involved in the fatigue processes, sarcolemmal depolarization due to extracellular  $K^+$  accumulation has been suggested to be of primary importance for fatigue development during high-intensity exercise (Sejersted & Sjogaard, 2000). This hypothesis is based on observations that during dynamic exercise the contracting muscles lose  $K^+$  which progressively accumulates in the extracellular space leading to a depolarization of the membrane potential ( $E_m$ ) (Juel, 1988; Cairns et al., 1995; Cairns et al., 1997). A lower  $E_m$  has been shown in *in vitro* studies (Sjogaard et al., 1985; Juel, 1988; Cairns et al., 1997; Sejersted & Sjogaard, 2000) to cause membrane inexcitability and simultaneous tetanic force reduction probably via a slow inactivation of the voltage-gated  $Na^+$  channels (Ruff et al., 1988; Ruff, 1999). The evidence of  $K^+$  being of importance for fatigue development in humans is related to the findings that muscle interstitial  $K^+$  during intense exercise is elevated to levels  $>10$  mM (Nordsborg et al., 2003; Nielsen et al., 2004), and that exercise training leads to reduced interstitial  $K^+$  accumulation (Nielsen et al., 2004) in association with improved exercise performance and a change in the amount of  $Na^+,K^+$  pumps (Clausen, 2003).

Speed endurance training of trained subjects elevates the expression of either the  $Na^+,K^+$  pump  $\alpha 1$  or  $\alpha 2$  subunits (Iaia et al., 2008; Bangsbo et al., 2009; Thomassen et al., 2010) as well as its accessory and regulatory protein phospholemman (FXD1) (Thomassen et al., 2010), whereas no significant increases have been observed in the  $\beta 1$  subunit (Iaia et al., 2008; Bangsbo et al., 2009; Thomassen et al., 2010). In the first two studies, the changes reported in the  $\alpha 1$  and  $\alpha 2$  subunits were marked (29% and 68%, respectively) and are comparable to those observed in untrained and diabetic subjects after a period of speed endurance and strength training (Dela et al., 2004; Nielsen et al., 2004). These adaptations occurred despite the fact that the trained subjects may

have already had an elevated protein content before the training, as endurance training has been demonstrated to increase the  $Na^+,K^+$  pump subunits (Green et al., 2004). The augmented  $Na^+,K^+$  pump  $\alpha$  subunits may result in a high number of functional pumps and play an important role for the increases in work capacity during intense short-term performance. Indeed, a higher  $Na^+,K^+$  pump maximum activity reduces the net loss of  $K^+$  from the contracting muscles preserving the cell excitability and force production (Sejersted & Sjogaard, 2000). This is supported by the finding that in both studies the elevated expression of the  $Na^+,K^+$  pump  $\alpha$  subunits observed after the speed endurance training period was associated with a decreased accumulation of venous plasma  $K^+$  and an increased performance during repeated supramaximal exercise.

In trained people, isoforms of the muscle monocarboxylate transporters (MCT) have been shown to be unchanged with speed endurance training (Iaia et al., 2008; Bangsbo et al., 2009; Thomassen et al., 2010), which is in contrast to data from untrained subjects (Pilegaard et al., 1999; Juel et al., 2004; Bickham et al., 2006; Burgomaster et al., 2007; Mohr et al., 2007). One study has reported sprint training-induced changes on MCT1 proteins in endurance-trained subjects (Bickham et al., 2006). In contrast to studies reporting no changes, in the latter investigation the subjects maintained a high volume of training ( $\sim 50$  vs  $\sim 15$  and  $\sim 32$  km/week, respectively), which may be part of the explanation for the differences observed.

In the study by Iaia et al. (2008) the *in vitro* measured muscle buffering capacity was not changed. Consistently, muscle and blood lactate concentrations as well as muscle  $H^+$  accumulation were found to be unaltered during repeated intense exhaustive exercise (Iaia et al., 2008). Apparently, improved short-term performance can occur without changes in MCT and buffering capacity. In accordance, Messonnier et al. (2007) observed that subjects with elevated performance levels compared with untrained individuals are "less dependent on the muscle content of proteins involved in buffering mechanisms and lactate/ $H^+$  transport." As such, it appears therefore reasonable to assume that in already trained individuals mechanisms other than changes of pH regulatory systems may be predominant in delaying fatigue development and enhancing work capacity during intense exercise. On the other hand, the expression of the  $Na^+/H^+$  exchanger isoform 1 (NHE1) was enhanced after a speed endurance training period (Iaia et al., 2008). This could have facilitated the  $Na^+$  uptake inside the muscle cell and thus possibly resulted in a higher stimulation of the  $Na^+,K^+$  pump (Ewart & Klip, 1995) causing a hyperpolarization of the  $E_m$ .

## Conclusion

In many sports the time available for training is limited during the season. Coaches and athletes are usually concerned that a reduced amount of training, i.e. number and duration of sessions, may be detrimental for performance and sometimes athletes train more than required. The studies presented in this review provide strong evidence that, although its brevity, speed endurance training leads to performance improvements during several high-intensity short-duration exercises in already trained subjects. Furthermore, despite marked reduction in training volume, the muscle oxidative potential, capillarization and aerobic performance were shown to be unchanged indicating that exercise at near maximal intensity is a powerful stimulus for maintaining physiological adaptation and performance gained from previous endurance training. On the other hand, when combined with a basic amount of aerobic training, speed endurance training has even been reported to improve endurance performance. These changes do not appear to be mediated by elevated  $\dot{V}O_{2\max}$ , higher glycolytic and oxidative enzymes activity, and improved pH regulation. It may instead be related to a lower energy expenditure during exercise and changes in the  $Na^+, K^+$  pump  $\alpha 1$  or  $\alpha 2$  subunits, which via a reduced contraction-induced net loss of  $K^+$  from the working muscles may contribute to preserve cell excitability and maintain force development. However, there may be a number of other factors contributing to the elevated performance. It should be emphasized that none of the studies presented have been performed on elite athletes. Regardless, it appears reasonable to suggest that athletes in sports involving intense exercise may benefit from periodically reducing the amount of workload and undertaking speed endurance training on a regular basis. Nevertheless, further studies are needed to examine the effect of reducing the amount of training and performing speed endurance training over a longer period (several months).

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## Practical implications

For a practical purpose, speed endurance training may be divided into two different forms: production and maintenance. In “production training” the exercise intensity is almost maximal and there is research evidence that this is a very effective way to elicit adaptations in several muscle variables as well as to improve performance during very intense short-duration and repeated high-intensity exercises. In order to maintain a high-intensity throughout the session, the duration of exercise periods should be relatively short but sufficiently longer ( $>10$  s) to stimulate the systems which limit performance. To perform maximally in a subsequent exercise bout, the resting periods should therefore be long enough for muscle to approximate to their pre-exercise state. Thus, exercise periods lasting 10–40 s at near maximal speed separated by rest periods of 1–5 min may be optimal for production training.

In speed endurance maintenance training the recovery times between the intervals are shorter (one to threefolds the exercise time) and the duration of the exercise periods may be longer (5–90 s) compared with speed endurance production training, resulting in an overall lower exercise intensity. This exercise mode leads to a gradual accumulation of fatigue as the training progresses and it has been shown to be useful to improve the ability to sustain intense exercise.

**Key words:** high-intensity intermittent, enzymes, pH, membrane transport proteins,  $Na^+, K^+$  pump.

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