ORIGINAL ARTICLE



# **The electromyographic threshold in boys and men**

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# **Abstract**

*Background* Children have been shown to have higher lactate (La<sub>Th</sub>) and ventilatory (Ve<sub>Th</sub>) thresholds than adults, which might be explained by lower levels of type-II motorunit (MU) recruitment. However, the electromyographic threshold ( $EMG<sub>Th</sub>$ ), regarded as indicating the onset of accelerated type-II MU recruitment, has been investigated only in adults.

*Purpose* To compare the relative exercise intensity at which the  $EMG<sub>Th</sub>$  occurs in boys versus men.

*Methods* Participants were 21 men  $(23.4 \pm 4.1 \text{ years})$  and 23 boys (11.1  $\pm$  1.1 years), with similar habitual physical activity and peak oxygen consumption  $(VO_2pk)$  (49.7  $\pm$  5.5) vs.  $50.1 \pm 7.4$  ml kg<sup>-1</sup> min<sup>-1</sup>, respectively). Ramped cycle ergometry was conducted to volitional exhaustion with surface EMG recorded from the right and left vastus lateralis muscles throughout the test  $(-10 \text{ min})$ . The composite right–left EMG root mean square  $(EMG<sub>RMS</sub>)$  was then calculated per pedal revolution. The  $EMG<sub>Th</sub>$  was then determined as the exercise intensity at the point of least residual

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Research Institute for Sport and Exercise Sciences, Faculty of Science, Liverpool John Moores University, Liverpool, UK sum of squares for any two regression line divisions of the  $EMG<sub>RMS</sub>$  plot.

*Results* EMG<sub>Th</sub> was detected in 20/21 of the men (95.2 %) and only in 18/23 of the boys (78.3 %). The boys' EMG<sub>Th</sub> was significantly higher than the men's (86.4  $\pm$  9.6 vs. 79.7  $\pm$  10.0 % of peak power output at exhaustion;  $p$  < 0.05). The pattern was similar when EMG<sub>Th</sub> was expressed as percentage of  $VO<sub>2</sub>pk$ .

*Conclusions* The boys' higher  $EMG<sub>Th</sub>$  suggests delayed and hence lesser utilization of type-II MUs in progressive exercise, compared with men. The boys–men  $EMG<sub>Th</sub>$  differences were of similar magnitude as those shown for  $La<sub>Th</sub>$ and  $Ve$ <sub>Th</sub>, further suggesting a common underlying factor.

**Keywords** Children · Exercise · Motor unit activation

# **Abbreviations**



# **Introduction**

Children's response to exercise is physiologically and functionally different from adults'. For example, compared with adults, children demonstrate lower body-sizenormalized maximal isometric strength (Falk et al. [2009\)](#page-7-0) and short-term power (Beneke et al. [2007](#page-7-1); Van Praagh and Dore  $2002$ ), higher ventilatory threshold (Ve<sub>Th</sub>) (Klentrou et al.  $2006$ ) and lactate threshold (La<sub>Th</sub>) (Simon et al. [1981](#page-8-1); Tanaka and Shindo [1985\)](#page-8-2). In terms of neuromuscular function, children have been shown to have lower motorunit (MU) activation of the knee extensor muscles (Blimkie [1989;](#page-7-3) O'Brien et al. [2009,](#page-8-3) [2010\)](#page-8-4), and a lower mean power frequency during sustained maximal voluntary contraction (MVC) (Halin et al. [2003\)](#page-7-4). According to the size principle (Henneman et al. [1965\)](#page-7-5), the lower level of voluntary recruitment implies that children activate their higher threshold, fast-twitch, type-II MUs to a lesser extent than do adults. The lesser activation of type-II MUs and greater reliance on slow-twitch, type-I MUs may, in turn, explain many performance, metabolic, and neuro-motor child– adult differences beyond just isometric muscle strength (Dotan et al. [2012](#page-7-6)). Direct supportive evidence for this differential MU activation hypothesis is lacking due to technical or ethical constraints associated with invasive techniques. Hence, new investigative approaches ought to be explored in attempting to further support or refute the hypothesis.

Currently, no technique is available for directly monitoring the proportion of active type-I versus type-II MUs. Furthermore, invasive procedures that might be available for adults (e.g., needle electrodes) are not ethically acceptable for children. A non-invasive technique that is widely accepted as being capable of discerning the onset of type-II MU activation in adults, is the electromyographic threshold  $(EMG<sub>Th</sub>)$  (Candotti et al. [2008](#page-7-7); Edwards and Lippold [1956](#page-7-8); Hug et al. [2006b,](#page-7-9) [2003;](#page-7-10) Lucia et al. [1999](#page-7-11); Maestu et al. [2006;](#page-7-12) Moritani and deVries [1978](#page-7-13); Moritani et al. [1984](#page-8-5), [1993;](#page-8-6) Nagata et al. [1981](#page-8-7); Petrofsky [1979;](#page-8-8) Tikkanen et al.  $2012$ ). The EMG<sub>Th</sub> is defined as a non-linear increase, or upward inflection, in the EMG signal during progressive exercise of increasing intensity (Miyashita and Kanehisa [1980](#page-7-14); Moritani and deVries [1978\)](#page-7-13). The EMG<sub>Th</sub> concept is supported by evidence of orderly depletion of glycogen first in type-I and  $II_A$ , followed by  $II_{AX}$  ( $II_{AB}$ ), and finally  $II_X$  ( $II_B$ ) muscle fibers at increasing workloads (Vollestad and Blom [1985](#page-8-10)), and by increasing muscle fiber conduction velocity at progressively higher power outputs, suggesting progressive recruitment of large, high-conduction-velocity MUs with increasing muscle force (Farina et al. [2004](#page-7-15)).

In adults, the  $EMG<sub>Th</sub>$  has been investigated in untrained as well as trained individuals, mostly in cycling and running, but also in upper-extremity exercise modes (Bearden

and Moffatt [2001;](#page-7-16) Candotti et al. [2008](#page-7-7); Chwalbinska-Moneta et al. [1994](#page-7-17), [1998;](#page-7-18) Hug et al. [2003](#page-7-10), [2004](#page-7-19), [2006b,](#page-7-9) Lucia et al. [1999;](#page-7-11) Moritani et al. [1993;](#page-8-6) Nagata et al. [1981](#page-8-7); Takaishi et al. [1992](#page-8-11); Taylor and Bronks [1994;](#page-8-12) Tikkanen et al. [2012](#page-8-9); Viitasalo et al. [1985\)](#page-8-13), and in a wide range of muscles (Bearden and Moffatt [2001](#page-7-16); Chwalbinska-Moneta et al. [1994;](#page-7-17) [1998](#page-7-18); Hug et al. [2003,](#page-7-10) [2006b](#page-7-9); Lucia et al. [1999](#page-7-11); Moritani et al. [1984;](#page-8-5) Nagata et al. [1981;](#page-8-7) Takaishi et al. [1992](#page-8-11)). Alongside the EMG<sub>Th</sub>, a number of studies also determined the Ve<sub>Th</sub> (Nagata et al. [1981;](#page-8-7) Tikkanen et al. [2012](#page-8-9)), the La<sub>Th</sub> (Candotti et al. [2008;](#page-7-7) Chwalbinska-Moneta et al. [1998;](#page-7-18) Moritani et al. [1993](#page-8-6); Moritani et al. [1984](#page-8-5); Nagata et al. [1981](#page-8-7)), and the onset of blood lactate accumulation (OBLA) (Tikkanen et al. [2012\)](#page-8-9). These thresholds highly correlated with the  $EMG<sub>Th</sub>$ , although they did not necessarily coincide. This inter-threshold correlation could be expected as increased activation of the more glycolytic type-II MUs, presumed to occur at the  $EMG<sub>Th</sub>$ , implies higher lactate production and accumulation, increased acidosis, and elevated ventilatory drive.

The  $EMG<sub>Th</sub>$  has not been studied in children. Thus, the purpose of this study was to compare the relative exercise intensity at which  $EMG<sub>Th</sub>$  occurs in children versus adults. Given the close relationships between  $EMG<sub>Th</sub>$  and the  $La<sub>Th</sub>$  and Ve<sub>Th</sub> seen in adults, and the observed higher exercise intensity at which the latter thresholds occur in children, it was hypothesized that the  $EMG<sub>Th</sub>$  would also occur at higher relative exercise intensities in children than in adults. Such findings would suggest a different muscle activation regimen, in which children recruit type-II MUs later and to a lesser extent than do adults.

# **Methods**

# Participants

All tests and procedures complied with the Helsinki declaration and were cleared by Brock University's Research Ethics Board. Twenty-three boys, aged 8–13 years, and 21 men, aged 18–32 years, with similar weekly physical activity history and peak oxygen consumption  $(VO<sub>2</sub>pk)$ , volunteered for the study. A summary of participants' characteristics is provided in Table [1.](#page-2-0) Written informed consent was provided by all adult participants and by the boys' parents or guardians prior to enrolment in the study. All boys provided their informed assent to participate. Participants completed questionnaires regarding medical history, physical activity (Godin and Shephard [1985](#page-7-20)) and sport training history. All participants were physically active and all child participants were involved in some form of organized, competitive sports. Boys were significantly smaller than men but no differences were observed in body composition or

<span id="page-2-0"></span>**Table 1** Participants' physical characteristics and training histories

	Men	<b>Boys</b>
n	21	23
Age (year)	$23.4 \pm 4.1$	$11.1 \pm 1.1*$
Mass (kg)	$75.4 \pm 10.4$	$37.1 \pm 7.5^*$
Height (cm)	$181.5 \pm 6.3$	$145.7 \pm 8.6^*$
$\%$ Fat	$14.0 \pm 3.6$	$14.2 \pm 3.2$
Activity score	$79.1 \pm 50.9$	$81.6 \pm 25.3$
Sport training (h week <sup>-1</sup> )	$5.6 \pm 4.8$	$5.4 \pm 2.6$
$VO2pk$ (ml kg min <sup>-1</sup> )	$49.7 \pm 5.5$	$50.1 \pm 7.4^{\#}$
RER at $VO2$ pk	$1.15 \pm 0.06$	$1.05 \pm 0.08$ **
HR at $VO2pk$ (bpm)	$194 \pm 7$	$198 \pm 11^{#}$

Values are Means  $\pm$  1SD

**\*** Significant difference between groups, *p* < 0.05

 $n = 22$  (*VO*<sub>2</sub> data unavailable for one boy)

maximal oxygen consumption. Sexual maturity in the boys, as determined by self-assessment of secondary sex characteristics [pubic hair (Tanner [1962\)](#page-8-14)], ranged from stages 1 to 4, with eight boys at stage 1, eight at stage 2, five at stage 3, and one at stage 4. One participant's sexual maturity value was excluded from analysis due to misreport concerns. Years to age of peak height velocity (PHV) for the boys, as estimated using the Mirwald equation (Mirwald et al. [2002](#page-7-21)), averaged −2.30 ± 0.63 years (*n* = 18; range −0.88 to  $-3.59$ ).

# Experimental protocol

Participants made two laboratory visits. During the first visit, participants were informed of all tests and procedures to take place over both visits. Anthropometric variables such as height, body mass, and percent body fat (%Fat) were measured. Skinfold thickness was measured in triplicate using Harpenden calipers (British Indicators, Herts, England) and the median value at each site was used. Skinfold thickness (triceps and subscapula) was used to estimate percent body fat using age- and maturity-specific equations (Slaughter et al. [1988\)](#page-8-15). Pubertal stage and physical activity and training history were also determined. Participants were then familiarized with the crank-length-adjustable cycle-ergometer (Excalibur Sport, Lode, Groningen, The Netherlands). Seat and handlebar positioning were determined and recorded for re-use in the subsequent visit. Proper crank length was individually determined at 5-mm increments as a function of inseam length. Participants then proceeded to perform a submaximal and maximal progressive cycling test to determine aerobic capacity (see below).

The second visit was scheduled 3–7 days following the first visit and included a maximal progressive cycling test for the determination of the  $EMG<sub>Th</sub>$  (see below).

#### Exercise testing

*Submaximal VO<sub>2</sub> and VO<sub>2</sub> pk tests* (*Visit 1*): Following a 5-min warm-up period, participants commenced an incremental exercise protocol consisting of 3–5 submaximal stages to determine the *VO*<sub>2</sub>-versus-power relationship. Stages were 3.5- and 4-min long for the boys and the men, respectively. Boys started out at 40–60 W and progressed in 15–20 W increments at each subsequent stage. Men started at 80–100 W with 30–40 W increments. These submaximal stages were used to determine the steady-state  $VO<sub>2</sub>$  in several progressive power output levels.

Following the completion of the submaximal stages, participants recovered for 10–20 min. Participants then performed an incremental exercise protocol to volitional exhaustion for the determination of peak aerobic power (PVO<sub>2</sub>pk; the mechanical power output corresponding to *VO*<sub>2</sub>pk). Workload was increased every minute (10 and 20 W min<sup>-1</sup>, for the boys and men, respectively) until volitional exhaustion. Pedaling rate was maintained at a minimum of 80 revolutions per minute (rpm).

HR was determined using a HR monitor (Timex Personal Heart Rate Monitor, Timex Group Inc., Toronto, ON, Canada) throughout the test. Expired gas was collected and analyzed using the Moxus metabolic cart (AEI technologies, PA, USA), calibrated prior to each test. *VO*<sub>2</sub>pk was determined as the average of the highest *VO*<sub>2</sub> values attained over three consecutive 15-s periods.

 $EMG<sub>Th</sub>$  *test* (*Visit 2*): Ryan and Gregor ([1992\)](#page-8-16) showed that the mono-articular muscles gluteus maximus, vastus lateralis (VL), vastus medialis, tibialis anterior, and soleus play a relatively invariant role as primary power producers in cycling. Hug et al. ([2006b\)](#page-7-9) showed VL to be the most consistent of eight cycling-involved muscles in demonstrating the  $EMG<sub>Th</sub>$ . We, therefore, chose VL as our tested muscle.

Following a 5-min warm-up period, participants performed a ramped cycling test to exhaustion. Starting power output was set at  $\sim$ 35–40 % of PVO<sub>2</sub>pk and, based on the previously determined PVO<sub>2</sub>pk, the ramp protocol was set so as to reach volitional exhaustion after ~10 min of exercise. Thus, the mean starting power output for the boys was  $51 \pm 12$  W and power output increased on average 1 W every 6 s. The corresponding values for men were  $103 \pm 21$  W and 1 W every 3 s. Surface EMG, using 10 mm2 , bipolar, Ag/Ag surface electrodes (Delsys 2.1, Delsys Inc., Boston, MA, USA), was used to continuously monitor the VL EMG of each leg throughout the test. An area of each thigh at two-thirds the distance from the anterior superior spina iliaca to the superior border of the patella was shaved (if necessary), abraded with skin preparation gel (Nuprep, Weaver and Company, Aurora, CO, USA), and cleaned with rubbing alcohol. Electrodes were

placed parallel to the direction of muscle fibers at approximately mid-width of the VL and affixed with proprietary double-sided tape. A reference electrode was placed over the spinous process of the seventh cervical vertebra. Participants were instructed and supervised to maintain a pedaling rate as close to 80 rpm as possible throughout the test and verbal encouragement was provided throughout the test and particularly as the participant approached exhaustion.

#### EMG data reduction

EMG signals were band-pass filtered (20–450 Hz) using the Bagnoli-4 (Delsys Inc., Boston, MA, USA) bioamplifier and sampled at a rate of 1,000 Hz using a Computer-Based Oscillograph and Data Acquisition System (EMGworks Acquisition, Delsys Inc., Boston, MA, USA).

EMG data were analyzed using a dedicated computer algorithm created in MATLAB (2013 version; MathWorks Inc., Natick, MA, USA). For each pedal stroke, a succinct EMG burst was defined and recorded for each leg and the record was pruned at the beginning and end of each stroke to remove any partial or incomplete bursts, if any. The pruned waveform was then de-trended to offset any baseline deviation (i.e., set baseline value to zero). The root mean square ( $RMS<sub>EMG</sub>$ ) was then calculated for each stroke throughout the entire EMG trace. The onset and offset of each  $RMS<sub>EMG</sub>$  burst were identified as the points where  $RMS<sub>EMG</sub>$  rose or fell, respectively, above or below 10 % of the mean  $RMS<sub>EMG</sub>$  value of the entire test record. The mean  $RMS<sub>EMG</sub>$  of each burst (*i.e.*, between the onset and offset) was then extracted for  $EMG<sub>Th</sub>$  determination.

## $EMG<sub>Th</sub> Determination$

A composite plot, averaging the  $RMS_{EMG}$  bursts from both legs, was constructed for each participant. This plot consisted of  $RMS<sub>EMG</sub>$  values (one for each pedal stroke) plotted against the test duration. To reduce the internal fluctuation, a trimmed moving average (30-point window in which the lowest 10 and highest 10 values were trimmed off and the mean of the median 10 points was calculated) was applied to the plot (Fig. [1\)](#page-3-0). In cases where a drop in the  $RMS<sub>FMG</sub>$  at the end of the test was observed in conjunction with a sustained fall below 80 rpm in pedaling cadence, the plot was truncated at the point where cadence began to fall. The  $EMG<sub>Th</sub>$  was then determined by a computer algorithm as the point of least residual sum of squares for any two linear regression line divisions of the data, similar to the approach of Hug et al. ([2006a](#page-7-22)).

Since the algorithm would always determine a point of least residual sum of squares, even when no actual threshold exists, an additional criterion was used to qualify a threshold. Based on previous literature (Hug et al. [2006b](#page-7-9)),



<span id="page-3-0"></span>Fig. 1 A representative RMS<sub>EMG</sub> trace of an adult man participant. Note the clear rise of the trace above the +3SD confidence interval



<span id="page-3-1"></span>Fig. 2 A representative RMS<sub>EMG</sub> trace of a boy participant. No  $EMG<sub>Th</sub>$  is identifiable in this trace

 $EMG<sub>Th</sub>$  was expected to occur at relative power outputs higher than ~80 %  $P_{\text{max}}$  in adults, and we hypothesized this to be even higher in children. Based on this assumption, a linear regression line was determined for the initial 70 % of the test duration, corresponding to ~80 % of  $P_{\text{max}}$ (since the  $EMG<sub>Th</sub>$  test commenced at a power output equal to  $\sim$ 35–40 % of PVO<sub>2</sub>pk). That line was extrapolated to the entire test duration. A 3-standard deviation (SD) confidence interval was applied above the trend line and extended to the end of the trace (Fig. [1](#page-3-0)). An  $EMG<sub>Th</sub>$  was then confirmed only if the  $RMS<sub>FMG</sub>$  plot rose and stayed above the confidence limit (*e.g.*, Fig. [1\)](#page-3-0), without descending back to within the confidence interval until the end of the test (*e.g.*, Fig. [2](#page-3-1)). For those participants showing a threshold, the power output at the  $EMG<sub>Th</sub>$  time point was determined. This power output was expressed as a percentage of the peak power

output reached during the EMG<sub>Th</sub> test (% $P_{\text{max}}$ ) and as a percentage of *VO*<sub>2</sub>pk (%*VO*<sub>2</sub>pk), based on the data obtained from the first session's *VO*<sub>2</sub> testing.

## Statistical analysis

All statistical analysis was performed using SPSS v.20 (SPSS Inc., Chicago, IL, USA). The data for all groups are presented as means  $\pm$  1 SD. All data were normally distributed. Group differences in physical characteristics and  $\%P_{\text{max}}$ and  $\%$ *VO*<sub>2</sub>pk at EMG<sub>Th</sub> were assessed using a two-tailed, homoscedastic Student's *t* test. Additionally, group differences between 'Responders' and 'Non-Responders' (see ["Results](#page-4-0)" section) were examined using a two-tailed Student's *t* test. Despite the small sample size, assumptions for normal distribution and homogeneity of variance were met. Pearson's correlations were computed for the boys, men, and the combined groups, between the  $EMG<sub>Th</sub>$  (both as  $%VO<sub>2</sub>pk$ and % $P_{\text{max}}$ ) and  $VO_2pk$  kg BW<sup>-1</sup>, PVO<sub>2</sub>pk kg BW<sup>-1</sup>,  $P_{\text{max}}$  kg BW<sup>-1</sup>, leisure time physical activity, and training hours per week, respectively. The acceptable level of significance for all tests was set at  $p < 0.05$ .

## <span id="page-4-0"></span>**Results**

Physical activity scores and training histories were similar in the two groups (Table [1\)](#page-2-0). Importantly, peak heart rate was, on average, 4 bpm higher in the boys whose *VO*<sub>2</sub>pk was also marginally higher. While none of these differences reached statistical significance they do strongly suggest that the boys had attained a comparable level of exertion to that of the men.

An  $EMG<sub>Th</sub>$  was identified in 20 out of the 21 men (95.2 %) and in 18 of 23 boys (78.3 %) ( $\chi^2_{(1, n=44)} = 2.69$ ,  $p = 0.10$ . In the participants for whom a threshold was identified, group differences showed 6.6 % higher (later)  $EMG<sub>Th</sub>$  % $P<sub>max</sub>$  in the boys compared with the men  $(t_{(36)} = -2.08, p = 0.045)$  (86.4 ± 9.6 % vs. 79.7 ± 10.0 %, respectively; Fig. [3](#page-4-1)). There was also a trend for a higher EMG<sub>Th</sub> %*V*O<sub>2</sub>pk in the boys versus men ( $t_{(35)} = -1.62$ ,  $p = 0.12$ ) (101.2  $\pm$  11.4 vs. 94.8  $\pm$  12.2 %, respectively; Fig. [3\)](#page-4-1) (only 17 boys included.  $VO<sub>2</sub>$  data could not be obtained for one boy).

Correlational analyses revealed that for all participants,  $EMG<sub>Th</sub>$  (% $VO<sub>2</sub>pk$ ) was not correlated with body-massnormalized *V*O<sub>2</sub>pk (*V*O<sub>2</sub>pk kg<sup>-1</sup>) ( $r = -0.18$ ,  $p > 0.05$ ). However, when examined separately for each group, a significant negative correlation was observed in the boys  $(r = -0.51, p = 0.015)$ , but not in the men  $(r = 0.18,$  $p > 0.05$ ). Also, EMG<sub>Th</sub> (%*V*O<sub>2</sub>pk) similarly correlated with the power output at  $VO_2pk kg^{-1} (PVO_2pk kg^{-1})$  in the boys ( $r = -0.52$ ,  $p = 0.03$ ) (Fig. [4\)](#page-4-2), but not in the men. No



<span id="page-4-1"></span>**Fig. 3** Group differences in relative exercise intensity at the  $EMG<sub>Th</sub>$ between boys and men in whom  $EMG<sub>Th</sub>$  was identified ('Responders'). Values are Mean ± 1SD. *\** boys significantly higher than men,  $p < 0.05$ 



<span id="page-4-2"></span>Fig. 4 Correlation between peak aerobic power (PVO<sub>2</sub>pk) and  $EMG<sub>Th</sub>$  as %*V*O2pk for the boys who showed  $EMG<sub>Th</sub>$  ( $n = 17$ ; *VO*<sub>2</sub> data unavailable for one boy)

significant correlations were found between  $EMG_{Th}$  % $P_{max}$ or  $EMG<sub>Th</sub>$  %*VO*<sub>2</sub>pk and training history, or leisure time physical activity.

For further analysis, the boys who demonstrated  $EMG<sub>Th</sub>$ ('Responders',  $n = 18$ ) were compared to those who did not ('Non-responders', *n* = 5) (Table [2\)](#page-5-0). The 'Responders' had significantly longer mean test duration than the 'Nonresponders'  $(p = 0.016)$  as well as significantly greater  $P_{\text{max}}/PVO_2$ pk ratio ( $p = 0.027$ ). There were no statistically significant differences in any other variables. In the men's group, only one participant did not show an  $EMG<sub>Th</sub>$ . He had exceptionally high *V*O<sub>2</sub>pk kg<sup>−1</sup> and P*V*O<sub>2</sub>pk kg<sup>−1</sup> values that were ~2.5 SDs above the group's mean.

Non-Responders
$11.1 \pm 0.8$
$2.52 \pm 0.32$
$34.0 \pm 5.0$
$141.7 \pm 6.4$
$29.2 \pm 3.8$
$74.9 \pm 16.9$
$48.9 \pm 6.1$
$507 \pm 67*$
$3.88 \pm 0.43$
$1.05 \pm 0.09*$

<span id="page-5-0"></span>Table 2 Comparison between EMG<sub>Th</sub> 'Responders' and 'Non-Responders' among the boys

Values are Means  $\pm$  1 SD

\* Significant difference between groups; *p* < 0.05

## **Discussion**

The main findings of this study are that (a)  $EMG<sub>Th</sub>$  was identified in only 78.3 % (18/23) of the boys, while the corresponding value in the men was 95.2 % (20/21); and (b) Among those in whom  $EMG<sub>Th</sub>$  was detected, the boys'  $EMG<sub>Th</sub> occurred at significantly higher relative workloads$ compared with the men. As the  $EMG<sub>Th</sub>$  is widely believed to reflect the onset of accelerated increase in higher-threshold, type-II MU recruitment during progressive exercise (Edwards and Lippold [1956](#page-7-8); Hug et al. [2003,](#page-7-10) [2006b;](#page-7-9) Lucia et al. [1999](#page-7-11); Maestu et al. [2006;](#page-7-12) Moritani and deVries [1978](#page-7-13); Moritani et al. [1993,](#page-8-6) [1984;](#page-8-5) Nagata et al. [1981](#page-8-7); Petrofsky [1979](#page-8-8); Tikkanen et al. [2012\)](#page-8-9), these findings suggest that boys activate their type-II MUs later and for a shorter duration than do men.

The results for our men's group are in close agreement with previous findings (Hug et al. [2006b](#page-7-9); Lucia et al. [1999](#page-7-11); Takaishi et al. [1992](#page-8-11)). However, to our knowledge, this is the first study to investigate the  $EMG<sub>Th</sub>$  in children. Compared with the men, our boys' higher relative  $EMG<sub>Th</sub>$  is in line with our hypothesis, based on known boys–men differences in  $La<sub>Th</sub>$  and Ve<sub>Th</sub>. Our observed differences of 6.6  $%P_{\text{max}}$  and 6.4  $%VO_2pk$  are similar to previously reported boys–men %*V*O<sub>2</sub>pk differences in Ve<sub>Th</sub> [7.2 %, (Klentrou et al. [2006\)](#page-7-2); 8.9 %, (Anderson and Mahon [2007](#page-7-23))] and  $La<sub>Th</sub>$  [9.1 %, (Anderson and Mahon [2007](#page-7-23))] during cycling exercise.

A question arising from our findings is why  $EMG<sub>Th</sub>$ could not be detected in the five 'Non-responder' boys? At exhaustion, the force applied by the participating muscles is considerably lower than MVC for the given pedaling cadence (Greig et al. [1985](#page-7-24); Sargeant et al. [1981\)](#page-8-17). In view of the fact that 50 % of 'Responders' EMG<sub>Th</sub> occurred very

close to exhaustion (>92 % Pmax), it is conceivable that the five 'Non-Responders' terminated their tests before attaining the force level at which  $EMG<sub>Th</sub>$  would have taken place. That is, had they been able to continue exercising longer, their  $EMG<sub>Th</sub>$  would have occurred at their observed power at exhaustion or above it. This suggestion is supported by our finding that, in the 'Responders', body-mass-normalized peak aerobic power (PVO<sub>2</sub>pk/kg) negatively correlated with EMG<sub>Th</sub> %*V*O<sub>2</sub>pk ( $r = -0.52$ ,  $p = 0.03$ ; Fig. [4\)](#page-4-2). That is, in boys with lower maximal power output,  $EMG<sub>Th</sub>$  was attained closer to maximal aerobic power, compared with more powerful boys. This, in turn, suggests that 'Non-responders' may not have been able to produce the power and thus the contractile force required to manifest the  $EMG<sub>Th</sub>$ . Further support for the suggestion is provided by Vollestad and Blom [\(1985](#page-8-10)), Vollestad et al. ([1984](#page-8-18)) who found that activation of type  $II_{AX}$  ( $II_{AB}$ ) and  $II_X$  ( $II_B$ ) MUs in adult men, during progressive cycling, took place only at 91 % *VO*<sub>2</sub>pk and beyond. As shown by Greig et al. (Greig et al. [1985](#page-7-24)), this power output level would require only ~50 % of the MVC pedal force at that cadence.

Accepting the notion that  $EMG<sub>Th</sub>$  could indeed occur at or above the peak force level attained in progressive exercise to exhaustion, means that  $EMG<sub>Th</sub>$  would only be detected in those individuals able to exceed the critical force by the end of the progressive exercise test. It is conceivable then that 'Non-Responders' ended their tests prior to attaining the sufficiently high power output necessary to attain that critical force and elicit  $EMG<sub>Th</sub>$ . Men attain higher relative force and power outputs due to their higher anaerobic capacity compared with the boys (Beneke et al. [2007](#page-7-1); Van Praagh and Dore [2002\)](#page-8-0). Thus, most men produce the muscle force necessary for  $EMG<sub>Th</sub>$  manifestation. For this reason, there was little or no correlation between  $EMG<sub>Th</sub>$  and  $VO<sub>2</sub>pk$  or  $PVO<sub>2</sub>pk$  in the men, unlike in the boys. Support for this notion comes from the fact that compared with 'Non-Responders', 'Responders' had significantly longer test durations and higher Pmax/P*VO*<sub>2</sub>pk ratio (Table [2\)](#page-5-0). That is, 'Responders' were able to employ more anaerobic power after reaching their respective  $PVO<sub>2</sub>pk$ and thus extend their test durations. Presumably, this was facilitated by the 'Responders' greater relative capacity to recruit type-II MUs.

The boys–men difference of 6.6 % in EMG<sub>Th</sub> % $P_{\text{max}}$ (boys = 86.4 %, men = 79.7 %;  $p = 0.045$ ) is likely an underestimate of the true group difference, since it reflects only the 'Responders'. Assigning just 100 % as the EMG<sub>Th</sub>  $%P_{\text{max}}$  values for the six 'Non-Responders' (5 boys, 1 man), the overall boys–men EMG<sub>Th</sub> difference increased by 30 %, to 8.6 % (boys = 89.3, men = 80.7 %;  $p < 0.01$ ). Accepting the possibility that  $EMG<sub>Th</sub>$  could occur at >100 % Pmax intensity, the true boys–men difference could well be even larger.

To illuminate possible underlying distinguishing factors, the 'Responder' and 'Non-responder' boys were compared (Table [2\)](#page-5-0). On average, 'Responders' lasted 18 % longer in their respective  $EMG<sub>Th</sub>$  tests than did 'Non-responders'. Accordingly, their final power output  $(P_{\text{max}})$  was 15.5 % higher, although this difference did not reach statistical significance. Since initial loading and the ramping protocol of the  $EMG<sub>Th</sub>$  test were based on peak aerobic power (PVO<sub>2</sub>pk), >95 % of participants attained a  $P_{\text{max}}$  that was greater than PVO<sub>2</sub>pk. However, while the 'Responders'  $P_{\text{max}}$  was 17 % higher, 'Non-responders'  $P_{\text{max}}$  was only 5 % higher than  $PVO_2pk$  ( $p < 0.03$ ). Again, this suggests that 'Non-responders' were less capable of recruiting the higher-threshold type-II MUs and consequently generating the extra anaerobic power necessary to exceed PVO<sub>2</sub>pk. The 'Non-responders' tended to be younger, lighter, and shorter, have lower lean body mass, be less physically active, and somatically less mature (longer time before PHV). Although none of these differences was statistically significant, the general picture is one of lower maturity level, which may be regarded as consistent with them possessing higher or no detectable  $EMG<sub>Th</sub>$ .

The only man in whom  $EMG<sub>Th</sub>$  could not be detected, had the highest  $VO<sub>2</sub>pk (63.6 ml kg<sup>-1</sup> min<sup>-1</sup>)$  and  $PVO<sub>2</sub>pk$  $(5.14 \text{ W kg}^{-1})$ , both of which were ~2.5 SD above the mean for the group. Since in comparable studies (*e.g*., Hug et al. [2003](#page-7-10), [2006a](#page-7-22), [b](#page-7-9)) the EMG<sub>Th</sub> was detected in 100 % of participants, we suggest that the absence of  $EMG<sub>Th</sub>$  in this individual was not due to error and that his high aerobic capacity could have been due to particularly high type-I muscle fiber composition. Thus, like the boy 'Non-Responders', he too might not have been able to sufficiently engage type-II MUs by the time he reached exhaustion.

Given the previously demonstrated relationships between  $EMG<sub>Th</sub>$  and both  $La<sub>Th</sub>$  and  $Ve<sub>Th</sub>$  in men (Candotti et al. [2008;](#page-7-7) Chwalbinska-Moneta et al. [1998;](#page-7-18) Moritani et al. [1993](#page-8-6); Moritani et al. [1984](#page-8-5); Nagata et al. [1981](#page-8-7); Tikkanen et al. [2012](#page-8-9)), similar relationships presumably exist in boys, as well. In both men and boys, however, there is a persistent difference between exercise intensity at  $La<sub>Th</sub>$  or  $Ve<sub>Th</sub>$  versus that of  $EMG<sub>Th</sub>$ . For example, in men,  $La<sub>Th</sub>$  and  $Ve<sub>Th</sub>$  have been typically found between ~50 and 60 %*V*O<sub>2</sub>pk (Anderson and Mahon [2007;](#page-7-23) Klentrou et al. [2006](#page-7-2); Simon et al. [1986](#page-8-19)), while EMG<sub>Th</sub> has been identified at ~90 % $VO_2$ pk (Lucia et al. [1999;](#page-7-11) Takaishi et al. [1992\)](#page-8-11) and at 94.8 % *VO*<sub>2</sub>pk in the present study. The corresponding values for boys are  $\sim 60-70$  % *V*O<sub>2</sub>pk for La<sub>Th</sub> and Ve<sub>Th</sub> (Anderson and Mahon [2007;](#page-7-23) Klentrou et al. [2006\)](#page-7-2) and 101.2 % *VO*<sub>2</sub>pk for  $EMG<sub>Th</sub>$  (in the present study). While boys' values are generally  $~10$  % higher than the men's, the differences between the  $EMG<sub>Th</sub>$  and the  $La<sub>Th</sub>$  or  $Ve<sub>Th</sub>$  thresholds are rather similar: ~45 % in both groups. A notable exception to this pattern is Candotti et al.'s finding of nearly identical

 $EMG<sub>Th</sub>$  and  $La<sub>Th</sub>$  values in adult recreational cyclists (Candotti et al. [2008\)](#page-7-7). A partial explanation for this discrepancy is the  $La<sub>Th</sub>$  protocol used by Candotti et al. (continuous, 3-min stages), which overestimates  $La<sub>Th</sub>$  due to the delay in the lactate response to the fast changing workloads. Furthermore, the testing protocol was exhaustion limited at ~200 W, likely due to lactate accumulation and well before *VO*<sub>2</sub>pk or P*VO*<sub>2</sub>pk could be reached. Thus, the mean reported EMG<sub>Th</sub> (134 W) occurred at no more than ~67 % of the end-of-protocol power and in reality likely constituted *VO*<sub>2</sub>pk percentage considerably lower than that. Since Hug et al. detected two rather than a single  $EMG<sub>Th</sub>$  in professional cyclists (at 52 and 86 % *VO*<sub>2</sub>pk) (Hug et al. [2003](#page-7-10)), it is also conceivable that the  $EMG<sub>Th</sub>$  reported by Candotti et al. (Candotti et al. [2008\)](#page-7-7) is the first (lower) rather than the second of the two thresholds.

The typical magnitude of the  $EMG_{Th} - La_{Th}$  difference appears to suggest that the  $EMG<sub>Th</sub>$  phenomenon is independent of the factors governing  $La<sub>Th</sub>$  and  $Ve<sub>Th</sub>$ , but the apparent consistency of this difference in men and boys seems to suggest otherwise. Several factors should be considered in explaining the apparent  $EMG<sub>Th</sub> - La<sub>Th</sub>/Ve<sub>Th</sub>$ discrepancy: (a) While  $La<sub>Th</sub>$  and Ve<sub>Th</sub> are systemic, wholebody phenomena, the  $EMG<sub>Th</sub>$  is confined to a single muscle. (b) The metabolic acidosis and increased lactate production that underlie both  $La<sub>Th</sub>$  and  $Ve<sub>Th</sub>$  take place as a function of increasing exercise intensity in the active MUs and could occur regardless of whether or not higher-threshold MUs are involved. The  $EMG<sub>Th</sub>$ , on the other hand, is presumably dependent on type-II MU involvement and could thus occur considerably after  $La<sub>Th</sub>$  or Ve<sub>Th</sub> have already taken place. (c) Based on evidence such as Vollestad and Blom's ([1985\)](#page-8-10) glycogen depletion findings,  $La<sub>Th</sub>$  and  $Ve<sub>Th</sub>$  could be related to increased type- $II_A$  MU recruitment, which occurs at low or moderate intensities. The  $EMG<sub>Th</sub>$ , on the other hand, might be associated with recruitment of the fast-twitch MU types  $II_{AX}$  ( $II_{AB}$ ) and IIx ( $II_{B}$ ), which typically occur in the later part of exhaustive exercise.

It may be of interest to draw an analogy between children and elite adult endurance athletes. Compared with untrained men, boys are characterized by markedly higher thresholds of all three types  $(La_{Th}, Ve_{Th}, EMG_{Th})$ . The same has been shown to be true for endurance athletes, in whom the higher thresholds are attained by extensive training at moderate intensities that, according to the size principle (Henneman et al. [1965\)](#page-7-5) and glycogen depletion data (Vollestad and Blom [1985;](#page-8-10) Vollestad et al. [1984\)](#page-8-18), predominantly recruit type-I and possibly the lower portion of the type- $II_A$  MU pool. Also, such athletes often possess higher type-I MU composition to begin with (Costill et al. [1976](#page-7-25)). Consequently, adult endurance athletes utilize type-I MUs to a greater extent than do non-athletes. The present data indicate that children (boys) are characterized by lower

utilization of type-II MUs and, thus, greater reliance on type-I MUs utilization as is the case in endurance athletes. The difference is that in children this is not due to training or talent, but rather to what we suggest is their lower utilization of type-II MUs.

# **Conclusions**

Our findings suggest that the fundamental nature of MU recruitment in progressive exercise [Henneman's size principle; (Henneman et al. [1965\)](#page-7-5)] is qualitatively similar in boys and men. There is, however, a significant quantitative difference in that the boys'  $EMG<sub>Th</sub>$  occurred at higher exercise intensities compared with the men's. This age-related difference suggests later recruitment of higher threshold MUs in boys and is in line with previous findings of higher relative  $La<sub>Th</sub>$  and  $Ve<sub>Th</sub>$  in boys compared with men. These findings support the child–adult differential MU activation hypothesis. We suggest that this support is particularly insightful since the  $EMG<sub>Th</sub>$  is currently the best non-invasive 'window' into the type of differential MU activation at the core of the hypothesis.

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