The electromyographic threshold in boys and men

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Abstract

*Background:* Children have been shown to have higher lactate- \( (L_{a_{Th}}) \) and ventilatory-thresholds \( (V_{e_{Th}}) \) than adults, which might be explained by lower levels of type-II motor-unit (MU) recruitment. However, the electromyographic threshold \( (EMG_{Th}) \), 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 \( EMG_{Th} \) occurs in boys vs. men. *Methods:* Participants were 21 men \( (23.4\pm4.1y) \) and 23 boys \( (11.1\pm1.1y) \), with similar habitual physical activity and peak oxygen consumption \( (VO_{2pk}) \) \( (49.7\pm5.5 \text{ vs. } 50.1\pm7.4 \text{ ml·kg}^{-1}·\text{min}^{-1}, \) 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 \( (EMG_{RMS}) \) was then calculated per pedal revolution. The \( EMG_{Th} \) was then determined as the exercise intensity at the point of least residual sum of squares for any two-regression-line division of the \( EMG_{RMS} \) plot. *Results:* \( EMG_{Th} \) was detected in 20/21 of the men \( (95.2\%) \) and only in 18/23 of the boys \( (78.3\%) \). The boys’ \( EMG_{Th} \) was significantly higher than the men’s \( (86.4\pm9.6 \text{ vs. } 79.7\pm10.0\% \text{ of peak power-output at exhaustion}; \ p<0.05) \). The pattern was similar when \( EMG_{Th} \) was expressed as percentage of \( VO_{2pk} \). *Conclusions:* The boys’ higher \( EMG_{Th} \) suggests delayed and hence lesser utilisation of type-II MU in progressive exercise, compared with men. The boys–men \( EMG_{Th} \) differences were of similar magnitude to those shown for \( L_{a_{Th}} \) and \( V_{e_{Th}} \), further suggesting a common underlying factor.

*Key words:* Children, Exercise, Motor unit activation
Abbreviations

EMG – Electromyography

EMG$_{Th}$ – ElectroMyoGraphic Threshold

HR – Heart Rate

La$_{th}$ – Lactate Threshold

MU / MUs – Motor Unit / Motor Units

MVC – Maximal Voluntary Contraction

OBLA – Onset of Blood Lactate Accumulation

PHV – Peak Height Velocity

Pmax – Maximal power attained at end of the EMG$_{Th}$ test

PVO$_2$pk – Peak aerobic power (mechanical power output corresponding to VO$_2$pk)

RER – Respiratory Exchange Ratio

RMS – Root Mean Square

SD – Standard Deviation

Ve$_{th}$ – Ventilatory / Gas-Exchange Threshold

VO$_2$ – Oxygen Consumption

VO$_2$pk – Peak oxygen consumption
Introduction

Children’s response to exercise is physiologically and functionally different from adults’. For example, compared with adults, children demonstrate lower body-size-normalized maximal isometric strength (Falk et al. 2009) and short-term power (Beneke et al. 2007; Van Praagh and Dore 2002), higher ventilatory threshold ($V_{\text{et}}$) (Klentrou et al. 2006) and lactate threshold ($L_{\text{at}}$) (Simon et al. 1981; Tanaka and Shindo 1985). In terms of neuromuscular function, children have been shown to have lower motor-unit (MU) activation of the knee extensor muscles (Blimkie 1989; O’Brien et al. 2009, 2010), and a lower mean power frequency during sustained maximal voluntary contraction (MVC) (Halin et al. 2003). According to the size-principle (Henneman et al. 1965), 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 differences between children and adults, beyond isometric muscle strength (Dotan et al. 2012). 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 vs. 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 able to discern the onset of type-II MU activation, in adults, is the electromyographic threshold ($EMG_{\text{et}}$) (Candotti et al. 2008; Edwards and Lippold 1956; Hug et al. 2006b; Hug et al. 2003; Lucia et al. 1999; Maestu et al. 2006; Moritani and deVries 1978;
Moritani et al. 1993; Moritani et al. 1984; Nagata et al. 1981; Petrofsky 1979; Tikkanen et al. 2012). The EMG_{Th} is defined as a non-linear increase, or upward inflection, in the EMG signal during progressive exercise of increasing intensity (Miyashita and Kancheisa 1980; Moritani and deVries 1978). The EMG_{Th} 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 fibres at increasing workloads (Vollestad and Blom 1985), and by increasing muscle fibre conduction velocity at progressively higher power outputs, suggesting progressive recruitment of large, high-conduction-velocity MUs with increasing muscle force (Farina et al. 2004).

In adults, the EMG_{Th} 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; Candotti et al. 2008; Chwalbinska-Moneta et al. 1994; Chwalbinska-Moneta et al. 1998; Hug et al. 2004; Hug et al. 2006b; Hug et al. 2003; Lucia et al. 1999; Moritani et al. 1993; Nagata et al. 1981; Takaishi et al. 1992; Taylor and Bronks 1994; Tikkanen et al. 2012; Viitasalo et al. 1985), and in a wide range of muscles (Bearden and Moffatt 2001; Chwalbinska-Moneta et al. 1994; Chwalbinska-Moneta et al. 1998; Hug et al. 2006b; Hug et al. 2003; Lucia et al. 1999; Moritani et al. 1984; Nagata et al. 1981; Takaishi et al. 1992). Alongside the EMG_{Th}, a number of studies also determined the Ve_{Th} (Nagata et al. 1981; Tikkanen et al. 2012), the La_{Th} (Candotti et al. 2008; Chwalbinska-Moneta et al. 1998; Moritani et al. 1993; Moritani et al. 1984; Nagata et al. 1981), and the onset of blood lactate accumulation (OBLA) (Tikkanen et al. 2012). These thresholds highly correlated with the EMG_{Th}, 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_{Th}, implies higher lactate production and accumulation, increased acidosis, and elevated ventilatory drive.
The EMG_{Th} has not been studied in children. Thus, the purpose of this study was to compare the relative exercise intensity at which EMG_{Th} occurs in children vs. adults. Given the close relationships between EMG_{Th} and the La_{Th} and Ve_{Th} seen in adults, and the observed higher exercise intensity at which the latter thresholds occur in children, it was hypothesized that the EMG_{Th} 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 y, and 21 men, aged 18–32 y, with similar weekly physical activity history and peak oxygen consumption (VO_{2pk}), volunteered for the study. A summary of participants' characteristics is provided in Table 1. 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) 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 maximal oxygen consumption. Sexual maturity in the boys, as determined by self-assessment of secondary sex characteristics [pubic hair (Tanner 1962)], 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), averaged −2.30±0.63 years (n=18; range: −0.88 to −3.59).

[Table 1]

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 1988). Pubertal stage and physical activity and training history were also determined. Participants were then familiarised 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 $\text{EMG}_{\text{Th}}$ (see below).

Exercise Testing

Submaximal $\text{VO}_2$ and $\text{VO}_2$pk tests (Visit 1): Following a 5-minute warm-up period, participants commenced an incremental exercise protocol consisting of 3–5 submaximal stages to determine the $\text{VO}_2$-vs.-power relationship. Stages were 3.5- and 4-min long for the boys and
the men, respectively. Boys started out at 40–60W and progressed in 15–20W increments at each subsequent stage. Men started at 80–100W with 30–40W increments. These submaximal stages were used to determine the steady-state VO₂ 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 (PVO₂pk; the power output, in Watts, corresponding to VO₂pk). Workload was increased every minute (10 and 20 W·min⁻¹, for the boys and men, respectively) until volitional exhaustion. Pedalling rate was maintained at a minimum of 80 revolutions per minute (rpm).

Heart rate was determined using a heart rate monitor (Timex Personal Heart Rate Monitor, Timex Group Inc., Toronto, ON) throughout the test. Expired gas was collected and analyzed using the Moxus metabolic cart (AEI technologies, PA, USA), calibrated prior to each test. VO₂pk was determined as the average of the highest VO₂ values attained over three consecutive 15-s periods.

**EMG₉₉ test (Visit 2):** Ryan & Gregor (Ryan and Gregor 1992) showed that the monoarticular 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.* (Hug *et al.* 2006b) showed VL to be the most consistent of eight cycling-involved muscles in demonstrating the EMG₉₉. We therefore chose VL as our tested muscle.

Following a 5-minute warm-up period, participants performed a ramped incremental cycling test to exhaustion. Starting power output was set at ~35–40% of PVO₂pk and, based on the previously-determined PVO₂pk, 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±12 W and power output increased on average 1 W every 6 s. The corresponding values for men were 103±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), was used to continuously monitor the VL EMG of each leg throughout the test. An area of each thigh at two-thirds on the line from the anterior spina iliaca superior to the superior border of the patella was shaved (if necessary), abraded with skin preparation gel (Nuprep, Weaver and Company, Aurora, CO), and cleaned with rubbing alcohol. Electrodes were placed parallel to the direction of muscle fibres on the medial aspect 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 pedalling 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) bioamplifier and sampled at a rate of 1000 Hz using a Computer-Based Oscillograph and Data Acquisition System (EMGworks Acquisition, Delsys Inc., Boston, MA).

EMG data were analyzed using a dedicated computer algorithm created in MATLAB (2013 version; MathWorks Inc., Natick, MA). 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_{EMG} burst were identified as the points where RMS_{EMG} rose or fell, respectively, above
or below 10% of the mean RMS_{EMG} value of the entire test record. The mean RMS_{EMG} of each
burst (i.e., between the onset and offset) was then extracted for EMG_{Th} determination.

**EMG_{Th} Determination**

A composite plot, averaging the RMS_{EMG} bursts from both legs, was constructed for each
participant. This plot consisted of RMS_{EMG} 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). In cases where a drop in the RMS_{EMG} at
the end of the test was observed in conjunction with a sustained fall below 80 rpm in pedalling
cadence, the plot was truncated at the point where cadence began to fall. The EMG_{Th} 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.* (Hug *et al.*
2006a).

![Figure 1](image)

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), EMG_{Th} was expected to occur at relative power outputs
higher than \(\sim80\% \ P_{\text{max}}\) in adults, and we hypothesise 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 \(\sim80\% \ P_{\text{max}}\) (since the EMG_{Th} test commenced at a power output equal to
\(\sim35–40\% \ P_{\text{VO}_2\text{peak}}\)). 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). An EMG_{Th} was then confirmed only if the RMS_{EMG} plot rose and stayed above the confidence limit (e.g., Fig.1), without descending back to within the confidence interval until the end of the test (e.g., Fig.2). For those participants showing a threshold, the power output at the EMG_{Th} time point was determined. This power output was expressed as a percentage of the peak power output reached during the EMG_{Th} test (%P_{max}) and as a percentage of VO_2pk (%VO_2pk), based on the data obtained from the first session’s VO_2 testing.

[Figure 2]

Statistical Analysis

All statistical analysis was performed using SPSS v.20 (SPSS Inc., Chicago, IL). The data for all groups are presented as means ±1 SD. All data were normally distributed. Group differences in physical characteristics and %P_{max} and %VO_2pk at EMG_{Th} were assessed using a two-tailed, homoscedastic Student's t-test. Additionally, group differences between ‘Responders’ and ‘Non-Responders’ (see Results 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_{Th} (both as %VO_2pk and %P_{max}) and VO_2pk·kgBW^{-1}, PVO_2pk·kgBW^{-1}, P_{max}·kgBW^{-1}, leisure time physical activity, and training hours per week, respectively. The acceptable level of significance for all tests was set at p<0.05.

Results

Physical activity scores and training histories were similar in the two groups (Table 1). Importantly, peak heart rate was, on average, 4 bpm higher in the boys whose VO_2pk 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\textsubscript{Th} 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\textsubscript{Th} %P\textsubscript{max} 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). There was also a trend for a higher EMG\textsubscript{Th} %VO\textsubscript{2pk} in the boys vs. men ($t(35)=-1.62, p=0.12$) (101.2±11.4 \textit{vs.} 94.8±12.2\%, respectively; Fig.3) (only 17 boys included. VO\textsubscript{2} data could not be obtained for one boy).

[Figure 3]

Correlational analyses revealed that for all participants, EMG\textsubscript{Th} (%VO\textsubscript{2pk}) was not correlated with body-mass-normalized VO\textsubscript{2pk} (VO\textsubscript{2pk}·kg\textsuperscript{-1}) ($r=-0.18, p>0.05$). 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\textsubscript{Th} (%VO\textsubscript{2pk}) similarly correlated with the power output at VO\textsubscript{2pk}·kg\textsuperscript{-1} (PVO\textsubscript{2pk}·kg\textsuperscript{-1}) in the boys ($r=-0.52, p=0.03$) (Fig.4), but not in the men. No significant correlations were found between EMG\textsubscript{Th} %P\textsubscript{max} or EMG\textsubscript{Th} %VO\textsubscript{2pk} and training history, or leisure time physical activity.

[Figure 4]

For further analysis, the boys who demonstrated EMG\textsubscript{Th} (‘Responders’, n=18) were compared to those who did not (‘Non-responders’, n=5) (Table 2). The ‘Responders’ had significantly longer mean test duration than the ‘Non-responders’ ($p=0.016$) as well as
significantly greater Pmax/PVO2pk 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 EMGTh. He had exceptionally-high VO2pk·kg⁻¹ and PVO2pk·kg⁻¹ values that were ~2.5 SDs above the group's mean.

[Table 2]

Discussion

The main findings of this study are that a) EMGTh 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 EMGTh was detected, the boys’ EMGTh occurred at significantly higher relative workloads compared with the men. As the EMGTh is widely believed to reflect the onset of accelerated increase in higher-threshold, type-II MU recruitment during progressive exercise (Edwards and Lippold 1956; Hug et al. 2006b; Hug et al. 2003; Lucia et al. 1999; Maestu et al. 2006; Moritani and deVries 1978; Moritani et al. 1993; Moritani et al. 1984; Nagata et al. 1981; Petrofsky 1979; Tikkanen et al. 2012), 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; Lucia et al. 1999; Takaishi et al. 1992). However, to our knowledge, this is the first study to investigate the EMGTh in children. Compared with the men, our boys’ higher relative EMGTh is in line with our hypothesis, based on known boys–men differences in LaTh and VeTh. Our observed differences of 6.6 %Pmax and 6.4 %VO2pk are similar to previously reported boys–men %VO2pk differences in VeTh [7.2%, (Klentrou et al. 2006); 8.9%, (Anderson and Mahon 2007)] and LaTh [9.1%, (Anderson and Mahon 2007)] during cycling exercise.
A question arising from our findings is why EMG\textsubscript{Th} 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 pedalling cadence (Greig et al. 1985; Sargeant et al. 1981). In view of the fact that 50% of ‘Responders’ EMG\textsubscript{Th} occurred very close to exhaustion (>92% P\textsubscript{max}), it is conceivable that the five ‘Non-Responders’ terminated their tests before attaining the force level at which EMG\textsubscript{Th} would have taken place. That is, had they been able to continue exercising longer, their EMG\textsubscript{Th} 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\textsubscript{2pk}/kg) negatively correlated with EMG\textsubscript{Th} %VO\textsubscript{2pk} (r=-0.52, p=0.03; Fig.4). That is, in boys with lower maximal power output, EMG\textsubscript{Th} 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\textsubscript{Th}. Further support for the suggestion is provided by Vollestad & Blom (Vollestad and Blom 1985; Vollestad et al. 1984) who found that activation of type II\textsubscript{AX} (II\textsubscript{AB}) and II\textsubscript{X} (II\textsubscript{B}) MUs in adult men, during progressive cycling, took place only at 91% VO\textsubscript{2pk} and beyond. As shown by Greig \textit{et al.} (Greig et al. 1985), this power output level would require only ~50% of the maximal pedal force at that cadence.

\textbf{Accepting the notion} that EMG\textsubscript{Th} could indeed occur at or above the peak force level attained in progressive exercise to exhaustion, means that EMG\textsubscript{Th} 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\textsubscript{Th}. Men attain higher relative force and power outputs due to their higher anaerobic capacity compared with the boys (Beneke
et al. 2007; Van Praagh and Dore 2002). Thus, most men produce the muscle force necessary for
EMG\textsubscript{Th} manifestation. For this reason, there was little or no correlation between EMG\textsubscript{Th} and
VO\textsubscript{2pk} or PVO\textsubscript{2pk} 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 P\textsuperscript{max}/PVO\textsubscript{2pk} ratio (Table 2). That is, ‘Responders’ were able to employ more anaerobic
power after reaching their respective PVO\textsubscript{2pk} 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\textsubscript{Th} %P\textsuperscript{max} (boys=86.4%, men=79.4%; p=0.045)
is likely an underestimate of group difference as it reflects only the ‘Responders’. Assigning just
100% as the EMG\textsubscript{Th} %P\textsuperscript{max} values for the six ‘Non-Responders’ (5 boys, 1 man), the overall
boys–men EMG\textsubscript{Th} difference increased by 30% to 8.6% (boys=89.3, men=80.7%; p<0.01).
Accepting the possibility that EMG\textsubscript{Th} could occur at >100% P\textsuperscript{max} 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). On average, ‘Responders’ lasted 18% longer in their
respective EMG\textsubscript{Th} tests than did ‘Non-responders’. Accordingly, their final power output (P\textsuperscript{max})
was 15.5% higher, although this difference did not reach statistical significance. Since initial
loading and the ramping protocol of the EMG\textsubscript{Th} test were based on peak aerobic power
(PVO\textsubscript{2pk}), >95% of participants attained a P\textsuperscript{max} that was greater than PVO\textsubscript{2pk}. However, while
the ‘Responders’ P\textsuperscript{max} was 17% higher, ‘Non-responders’ P\textsuperscript{max} was only 5% higher than
PVO\textsubscript{2pk} (p<0.03). Again, this suggests that ‘Non-responders’ were less capable of recruiting the
higher-threshold type-II MUs and generating the extra anaerobic power necessary to exceed
PVO\textsubscript{2pk}. 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$_{Th}$.

The only man in whom EMG$_{Th}$ could not be detected, had the highest VO$_{2pk}$ (63.6 ml·kg$^{-1}$·min$^{-1}$) and PVO$_{2pk}$ (5.14 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. 2006a, b; Hug et al. 2003) the EMG$_{Th}$ was detected in 100% of participants, we suggest that the absence of EMG$_{Th}$ in this individual was not due to error and that his high aerobic capacity could have been due to particularly high type-I muscle-fibre 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$_{Th}$ and both La$_{Th}$ and Ve$_{Th}$ in men (Candotti et al. 2008; Chwalbinska-Moneta et al. 1998; Moritani et al. 1993; Moritani et al. 1984; Nagata et al. 1981; Tikkanen et al. 2012), similar relationships presumably exist in boys, as well. In both men and boys, however, there is a persistent difference between exercise intensity at La$_{Th}$ or Ve$_{Th}$ vs. that of EMG$_{Th}$. For example, in men, La$_{Th}$ and Ve$_{Th}$ have been typically found between ~50–60 %VO$_{2pk}$ (Anderson and Mahon 2007; Klentrou et al. 2006; Simon et al. 1986), while EMG$_{Th}$ has been identified at ~90 %VO$_{2pk}$ (Lucia et al. 1999; Takaishi et al. 1992) and at 94.8 %VO$_{2pk}$ in the present study. The corresponding values for boys are ~60–70 %VO$_{2pk}$ for La$_{Th}$ & Ve$_{Th}$ (Anderson and Mahon 2007; Klentrou et al. 2006) and 101.2 %VO$_{2pk}$ for EMG$_{Th}$ (in the present study). While boys’ values are generally ~10% higher than the men’s, the differences between the EMG$_{Th}$ and the La$_{Th}$ or Ve$_{Th}$ thresholds are rather similar: ~45% in both groups. A notable exception to this pattern is Candotti et al.’s finding of nearly identical EMG$_{Th}$ and La$_{Th}$ values in adult recreational cyclists (Candotti et al.
A partial explanation for this discrepancy is the La_{Th} protocol used by Candotti et al. (continuous, 3-min stages), which overestimates La_{Th} due to the delay in the lactate response to the fast changing workloads. Furthermore, the testing protocol was exhaustion-limited at ~200W, likely due to lactate accumulation and well before VO_2pk or PVO_2pk could be reached. Thus, the mean reported EMG_{Th} (134W) occurred at no more than ~67% of the end-of-protocol power and in reality likely constituted VO_2pk percentage considerably lower than that. Since Hug et al. detected two rather than a single EMG_{Th} in professional cyclists (at 52 and 86% VO_2pk) (Hug et al. 2003), it is conceivable that the EMG_{Th} reported by Candotti et al. (Candotti et al. 2008) 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_{Th} phenomenon is independent of the factors governing La_{Th} and Ve_{Th}, 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_{Th}– La_{Th}/Ve_{Th} discrepancy: a. While La_{Th} and Ve_{Th} are systemic, whole-body phenomena, the EMG_{Th} is confined to a single-muscle. b. The metabolic acidosis and increased lactate production that underlie both La_{Th} and Ve_{Th}, 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. EMG_{Th}, on the other hand, is presumably dependent on type-II MU involvement and could thus occur considerably after La_{Th} or Ve_{Th} have already taken place. c. Based on evidence such as Vollestad & Blom’s glycogen depletion findings (Vollestad and Blom 1985), La_{Th} and Ve_{Th} could be related to increased type-II_A MU recruitment, which occurs at low or moderate intensities. The EMG_{Th}, 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 occurs 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 3 types \( \text{La}_{\text{Th}}, \text{Ve}_{\text{Th}}, \text{EMG}_{\text{Th}} \). 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) and glycogen depletion data (Vollestad and Blom 1985; Vollestad et al. 1984), predominantly recruit type-I and possibly the lower portion of the type-II\( \text{A} \) MU pool. Also, such athletes often possess higher type-I MU composition to begin with (Costill et al. 1976). 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)] is qualitatively similar in boys and men. There is, however, a significant quantitative difference in that the boys’ \( \text{EMG}_{\text{Th}} \) 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 \( \text{La}_{\text{Th}} \) and \( \text{Ve}_{\text{Th}} \) 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 \( \text{EMG}_{\text{Th}} \) is currently the best non-invasive ‘window’ into the type of differential MU activation at the core of the hypothesis.
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Competing Interests

The authors have no competing interests to declare.
Figure Legends

Figure 1. A representative $\text{RMS}_{\text{EMG}}$ trace of an adult man participant. Note the clear rise of the trace above the +3SD confidence interval.

Figure 2. A representative $\text{RMS}_{\text{EMG}}$ trace of a boy participant. No $\text{EMG}_{\text{Th}}$ is identifiable in this trace.

Figure 3. Group differences in relative exercise intensity at the $\text{EMG}_{\text{Th}}$ between boys and men in whom $\text{EMG}_{\text{Th}}$ was identified (‘Responders’). Values are Mean±1SD.

*= boys significantly higher than men, $p<0.05$.

Figure 4. Correlation between peak aerobic power (PVO$$_2$$pk) and $\text{EMG}_{\text{Th}}$ as %VO$$_2$$pk for the boys who showed $\text{EMG}_{\text{Th}}$ (n=17; VO$$_2$$ data unavailable for one boy).
### Tables

**Table 1.** Participants’ physical characteristics and training histories. Values are Means±1SD.

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Boys</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>21</td>
<td>23</td>
</tr>
<tr>
<td>Age (y)</td>
<td>23.4±4.1</td>
<td>11.1±1.1*</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>75.4±10.4</td>
<td>37.1±7.5*</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>181.5±6.3</td>
<td>145.7±8.6*</td>
</tr>
<tr>
<td>%Fat</td>
<td>14.0±3.6</td>
<td>14.2±3.2</td>
</tr>
<tr>
<td>Activity Score</td>
<td>79.1±50.9</td>
<td>81.6±25.3</td>
</tr>
<tr>
<td>Sport training (hours·week⁻¹)</td>
<td>5.6±4.8</td>
<td>5.4±2.6</td>
</tr>
<tr>
<td>VO₂pk (ml·kg·min⁻¹)</td>
<td>49.7±5.5</td>
<td>50.1±7.4</td>
</tr>
<tr>
<td>RER at VO₂pk</td>
<td>1.15±0.06</td>
<td>1.05±0.08*#</td>
</tr>
<tr>
<td>HR at VO₂pk (bpm)</td>
<td>194±7</td>
<td>198±11</td>
</tr>
</tbody>
</table>

* – Significant difference between groups, p<0.05

# – n=22 (VO₂ data unavailable for one boy)
**Table 2.** Comparison between EMG<sub>Th</sub> ‘Responders’ and ‘Non-Responders’ among the boys. Values are Means ±1SD.

<table>
<thead>
<tr>
<th></th>
<th>‘Responders’</th>
<th>‘Non-Responders’</th>
</tr>
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<tbody>
<tr>
<td>n</td>
<td>18</td>
<td>5</td>
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<tr>
<td>Age, years</td>
<td>11.1±1.2</td>
<td>11.1±0.8</td>
</tr>
<tr>
<td>Years to age of PHV</td>
<td>2.26±0.68</td>
<td>2.52±0.32</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>38.0±7.9</td>
<td>34.0±5.0</td>
</tr>
<tr>
<td>Height, cm</td>
<td>146.8±9.0</td>
<td>141.7±6.4</td>
</tr>
<tr>
<td>Lean Body Mass, kg</td>
<td>32.4±6.2</td>
<td>29.2±3.8</td>
</tr>
<tr>
<td>Activity score</td>
<td>83.5±27.3</td>
<td>74.9±16.9</td>
</tr>
<tr>
<td>VO&lt;sub&gt;2&lt;/sub&gt;pk, ml·kg&lt;sup&gt;−1&lt;/sup&gt;·min&lt;sup&gt;−1&lt;/sup&gt;</td>
<td>50.5±7.8</td>
<td>48.9±6.1</td>
</tr>
<tr>
<td>EMG&lt;sub&gt;Th&lt;/sub&gt; test duration, s</td>
<td>598±65</td>
<td>507±67 *</td>
</tr>
<tr>
<td>Pmax/kg, W·kg&lt;sup&gt;−1&lt;/sup&gt;</td>
<td>4.0±0.65</td>
<td>3.88±0.43</td>
</tr>
<tr>
<td>Pmax/PVO&lt;sub&gt;2&lt;/sub&gt;pk ratio</td>
<td>1.17±0.09</td>
<td>1.05±0.09 *</td>
</tr>
</tbody>
</table>

* = Significant difference between groups; p<0.05
Trimmed mean $\text{RMS}_{\text{EMG}}$ does not exceed confidence interval at exhaustion.