The impact of swimming speed on respiratory muscle fatigue during front crawl swimming: a role for critical velocity?

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Abstract

The Critical velocity (V\textsubscript{crit}) represents a holistic swimming fatigue threshold and critical stroke rate is thought to coincide with V\textsubscript{crit}. Whether V\textsubscript{crit} and in-turn critical stroke rate, also represent an inspiratory muscle fatigue threshold is not known. Following the determination of V\textsubscript{crit} and critical stroke rate via a two-parameter 200-m and 400-m model, 17 well-trained competitive swimmers (9 males and 8 females) undertook three experimental 200-m front crawl (FC) swims on separate occasions. One experimental swim corresponded to V\textsubscript{crit}, one was 5\% slower than V\textsubscript{crit} (V\textsubscript{crit}\textsubscript{5\%+}) and one 5\% faster (V\textsubscript{crit}\textsubscript{5\%-}) than V\textsubscript{crit}. Swim time, inspiratory and expiratory mouth pressure (P\textsubscript{Imax} and P\textsubscript{Emax}, respectively), stroke rate, stroke length and breathing frequency were recorded during each experimental swim and were compared between swims as well as between genders. Baseline P\textsubscript{Imax} and P\textsubscript{Emax} did not differ between experimental swims or genders. Although P\textsubscript{Emax} decreased significantly by a similar magnitude after each swim (range 5-8\%), P\textsubscript{Imax} was significantly lower in response to swimming at V\textsubscript{crit} (7\%) and V\textsubscript{crit}\textsubscript{5\%-} (22\%) thus indicating inspiratory muscle fatigue. However, the difference in magnitude between velocities was not significant. Only stroke rate and relative swimming velocity were significantly related to the fall in P\textsubscript{Imax} and no correlations were observed between P\textsubscript{Emax} and any other variable. The development of inspiratory muscle fatigue at and above V\textsubscript{crit} is consistent with substantial swim induced metabolic strain with the response similar between males and females. Our data suggest that V\textsubscript{crit} represents a FC inspiratory muscle fatigue threshold when using this two-parameter V\textsubscript{crit} model.

KEY WORDS: breathing muscles, stroke kinematics

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INTRODUCTION

Inspiratory muscle fatigue has been reported following 100-m (Brown and Kilding, 2011), 200-m (Jakovljevic and McConnell, 2009; Lomax and McConnell, 2003), 300-m and 400-m (Thomaidis et al., 2006) front crawl (FC), and 200-m back-stroke, breast-stroke and butterfly swimming (Lomax et al., 2012). However, the swimming intensity and hence swimming speed associated with inspiratory muscle fatigue has received little scientific attention. The few studies that have investigated inspiratory muscle fatigue in swimming have focused on: 1) its occurrence and magnitude following race-paced swimming (Brown and Kilding, 2011; Lomax et al., 2012; Thomaidis et al., 2009) or when swimming at 90-95% of race pace (Jakovljevic and McConnell, 2009; Lomax and McConnell, 2003); 2) how it impacts stroke kinematics such as stroke rate, stroke length and breathing frequency (Jakovljevic and McConnell, 2009; Lomax and Castle, 2011); or 3) the impact of race distance on its development (Brown and Kilding, 2011; Thomaidis et al., 2006). No studies have attempted to identify what the threshold swimming velocity is for inspiratory muscle fatigue to develop. This is surprising given that it can negatively affect stroke kinematics and therefore has implications for the intensity a coach selects when prescribing a training set designed to enhance stroke kinematics (Lomax and Castle, 2011).

Additionally, a sizable number of swimming inspiratory muscle fatigue studies have recruited both male and female swimmers, but with the exception of one study (Santos et al., 2011), none have examined the impact of gender on respiratory muscle strength per se or inspiratory muscle fatigue (Lomax and Castle, 2011; Lomax et al., 2012; Lomax and McConnell, 2003; Thomaidis et al., 2009). This is also surprising given that maximal inspiratory and expiratory mouth pressures are lower in adult (Black and Hyatt, 1969; Gonzales and Scheuermann, 2006; Ozkaplan et al., 2005) and adolescent (Santos et al., 2011; Smyth et al., 1984) females compared to males. Moreover, the rate of inspiratory muscle fatigue development may be age dependent in males (Gonzales and Scheuermann, 2006; Santos et al., 2011).

In terrestrial exercise the development of inspiratory muscle fatigue during prolonged activity is associated with an exercise intensity in excess of 80-85% of maximal oxygen uptake (Johnson et al., 1996; Romer and Polkey, 2008). However, expressing exercise intensity as a percentage of maximal oxygen uptake is impractical for most swimming coaches and athletes and therefore a more practical approach is needed. Both critical velocity (Vcrit) and critical stroke rate may be suitable alternatives.

Vcrit represents the maximal sustainable swimming intensity (Barden and Kell, 2009; Dekkerle et al., 2002; di Prampero et al., 2008) while critical stroke rate, which is thought to coincide with Vcrit, is the highest stroke rate that can be maintained for a prolonged period of time (Dekkerle, 2006). Moreover, Vcrit is the highest intensity that can be sustained without maximal oxygen uptake being obtained (Hill and Ferguson, 1999; Ribeiro et al., 2010) and may elicit approximately 89% of peak oxygen uptake in trained
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As the obtainment of Vcrit (Dekerle et al., 2002) and occurrence of inspiratory muscle fatigue (Jakovljevic and McConnell, 2009; Lomax and Castle, 2011; Lomax and McConnell, 2003) have been shown independently to coincide with 90% of 200-m race pace velocity, there is anecdotal evidence suggesting a possible link between the two (and in-turn critical stroke rate and inspiratory muscle fatigue). Whilst it is obvious to state that as velocity increases so too will the likelihood of developing inspiratory muscle fatigue, it is not known whether Vcrit represents a inspiratory muscle fatigue threshold.

It was the primary aim of this study to determine if the development of inspiratory muscle fatigue was dependent upon the obtainment of Vcrit and critical stroke rate. The secondary aim was to assess whether the development and magnitude of inspiratory muscle fatigue differed between male and female adolescent swimmers. We hypothesised that inspiratory muscle fatigue would be evident following FC swimming at and above Vcrit and critical stroke rate and that the magnitude of inspiratory muscle fatigue experienced would be larger above Vcrit. Based on the findings of Santos et al. (2011) we also hypothesised that there would be no difference in inspiratory muscle fatigue magnitude between genders.

MATERIALS AND METHODOLOGY

Seventeen (9 males, 8 females) competitive adolescent swimmers volunteered for this study. The mean and SD for age, body mass and height can be found in table 1 along with other demographic data. All participants were well-trained swimmers being regional standard or above and all testing took place after a taper phase. Participants and parents/guardians provided written informed assent and consent respectively. The study was approved by the host Institution Biosciences Research and Ethics Committee. Each swimmer completed the study in the same swimming pool, same apparel, and at the same time of day. Pool conditions were standardised throughout (pool temp: 27.6 ± 0.5°C).

Following a standardised swimming warm-up, swimmers undertook a race-paced 200-m FC swim and a race-paced 400-m FC swim in a counter-balanced order from a push start (Ribeiro et al., 2010). The warm-up consisted of 600-m alternating FC and backstroke per length at a pace approximating 60% of 200-m race pace (Lomax and Castle, 2011). Although Vcrit can be calculated based on a number of distances ranging from 50-m to 1,500-m (Dekerle et al., 2010; Dekerle et al., 2002; Garatachea et al., 2006; Zacca and Castro, 2010), we chose the two-parameter 200-m and 400-m model as this is the most pertinent in terms of practicality (Dekerle, 2006; Dekerle et al., 2002; Ribeiro et al., 2010; Wakayoshi et al., 1993). Time taken to complete the flat out 200-m and
400-m swims was used to calculate Vcrit and critical stroke rate (table 1) by modelling the distance (m) time (s) relationship, and the stroke rate (total cycles) time (s) relationship, respectively. The slope of the regression line between swim time in seconds and distance in metres determined Vcrit, while the slope of the regression line between swim time in seconds and stroke rate in seconds determined critical stroke rate (Dekerle, 2006; Dekerle et al., 2002; Ribeiro et al., 2010). This constituted preliminary testing and velocities of the experimental swims were calculated based on Vcrit.

Table 1 Demographic information: mean ± SD

<table>
<thead>
<tr>
<th>Group</th>
<th>Males</th>
<th>Females</th>
</tr>
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<tbody>
<tr>
<td>Age (years)</td>
<td>16.1 ± 1.6</td>
<td>16.6 ± 2.0</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>67.2 ± 7.0</td>
<td>65.2 ± 6.8</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.72 ± 0.07</td>
<td>1.73 ± 0.05</td>
</tr>
<tr>
<td>FVC (l)</td>
<td>4.98 ± 1.03</td>
<td>5.04 ± 1.18</td>
</tr>
<tr>
<td>FEV₁ (l)</td>
<td>4.24 ± 0.90</td>
<td>4.67 ± 0.71</td>
</tr>
<tr>
<td>PImax (cmH₂O)</td>
<td>137 ± 30</td>
<td>140 ± 33</td>
</tr>
<tr>
<td>PEmax (cmH₂O)</td>
<td>135 ± 36</td>
<td>129 ± 40</td>
</tr>
<tr>
<td>200-m time (s)</td>
<td>141.1 ± 10.9</td>
<td>142.0 ± 12.2</td>
</tr>
<tr>
<td>400-m time (s)</td>
<td>293.9 ± 24.1</td>
<td>294.7 ± 28.6</td>
</tr>
<tr>
<td>Vcrit (m/s⁻¹)</td>
<td>1.31 ± 0.11</td>
<td>1.33 ± 0.15</td>
</tr>
<tr>
<td>Critical stroke rate (Hz)</td>
<td>0.68 ± 0.05</td>
<td>0.68 ± 0.04</td>
</tr>
</tbody>
</table>

No significant differences (P>0.05) were evident between males and females.
FVC = forced vital capacity. FEV₁ = forced expired volume in one second.
PImax = maximal inspiratory mouth pressure. PEmax = maximal expiratory mouth pressure. Vcrit = critical velocity.
PImax and PEmax values reflect the mean baseline values across the three experimental swims.

On three separate occasions (separated by at least 24 hours) and following the standardised swimming warm-up described above, swimmers undertook three 200-m FC swims at different velocities in a counter-balanced order. One swim corresponded to Vcrit, one 5% slower than Vcrit (Vcrit5%+) and one 5% faster than Vcrit (Vcrit5%−). Pacing was provided by a swimming metronome placed under the swimmer’s cap and attached to the swimmer’s goggles (FINIS Temp Trainer, FINIS, USA). The metronome was pre-set to beep at a given time interval which corresponded with a set target distance: swimmers were permitted a separate practice swim to familiarise themselves with the required velocity. Additionally, an investigator walked alongside each swimmer on poolside at the predetermined rate and corroborated that the correct pace per length had been achieved. Thus, velocity was fixed per length throughout each swim.

Stroke rate, stroke length, breathing frequency and velocity were determined per length (Cardelli et al., 2000; Lerda and Cardelli, 2002) and averaged per swim. Stroke rate (one right and left arm stroke) was calculated as stroke cycles per second (Hz) and as cycles per minute (cycles/min). The latter was calculated as total cycles divided by time multiplied by 60 (Lomax and Castle, 2011). Additionally, stroke rate as a percentage of critical stroke rate was calculated per 200-m swim. Stroke length
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was calculated as velocity (m s\(^{-1}\)) divided by stroke rate (Hz) (Cardelli et al., 1999; Cardelli et al., 2000) and breathing frequency, which was expressed as breaths per minute (b min\(^{-1}\)), was determined as the number of breaths divided by time in seconds multiplied by 60 (Cardelli et al., 1999).

Forced vital capacity (FVC) and forced expired volume in one second (FEV\(_1\)) were recorded at the start of the study for demographic purposes only (table 1). All measurements were made using a portable spirometer (Micro Spirometer, Micro Medical, Rochester, UK) whilst standing. Values were recorded from total lung capacity and with the nose occluded. Maximal inspiratory and maximal expiratory mouth pressures (PImax and PEmax, respectively) were recorded pre and post each experimental swim using a hand-held respiratory pressure meter (RPM, Micro Medical, Rochester, UK) and were used to assess inspiratory muscle fatigue and expiratory muscle fatigue, respectively. Manoeuvres were made from a standing position on poolside (with the nose occluded) before, and within 60 seconds following, each 200-m swim. PImax was recorded from residual volume whereby swimmers were instructed to fully empty their lungs then inhale as hard and as fast as possible for approximately two to three seconds. PEmax was measured from total lung capacity and swimmers were instructed to fill up their lungs and exhale as hard and as fast as possible for two to three seconds (McConnell, 2007). A separate PImax and PEmax familiarisation session was completed by swimmers before the start of the study. Reliability at performing PImax and PEmax manoeuvres was deemed present when three attempts (60 second rest separating each) within 5% of one another were observed (Lomax and Castle, 2011).

Data were assessed for normality and homogeneity of variance using a Shapiro-Wilk test and Levene’s test, respectively. Intraclass correlation coefficients were used to assess reliability of baseline PImax and PEmax. As only one experimental swim was performed at each prescribed velocity, reliability was assessed within each swim by separating stroke rate, stroke length and breathing frequency into four 50-m blocks. An intraclass correlation coefficient of <.70 was deemed questionable, .70-.80 acceptable, .80-.89 moderate and ≥.90 high (Vincent 2005). The coefficient of variation was used to assess the reproducibility of achieved time versus target time per experimental swim.

Mixed model repeated measures (gender x velocity x time) ANOVAs assessed PImax and PEmax, where velocity had three levels (Vcrit\(_{5\%}\), Vcrit, Vcrit\(_{5\%}\)) and time two levels (pre and post swim). One-way mixed model repeated measures ANOVAs assessed stroke rate, percentage of critical stroke rate, stroke length and breathing frequency. Post hoc analyses were undertaken using paired samples t-tests or repeated measures ANOVAs. Independent t-tests were used to assess for gender differences in baseline respiratory muscle strength, whereby baseline was taken as the mean of the three baseline experimental swim values. Pearson’s correlation coefficient (pooled data) was used to identify any correlations between stroke characteristics, and between inspiratory and expiratory muscle fatigue, and percentage of Vcrit and percentage of critical stroke rate. Effect sizes (Cohen’s d) were calculated where relevant per
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dependent variable. An effect size of 0.2 was deemed small, 0.5 medium and 0.8 large (Cohen, 1988).

Significance was set as $P \leq 0.05$ as a priori, and statistical analyses were conducted using PASW Statistics 18 (Chicago, IL, USA). Unless otherwise stated data are expressed as mean ± SD.

RESULTS

The swimmers’ ability to achieve the target velocity during $V_{crit5\%+}$, $V_{crit}$ and $V_{crit5\%-}$ was excellent with a coefficient of variation between target and achieved time of 0.8, 0.5 and 0.9, respectively (table 2). Similarly, the intraclass correlation coefficients for $P_{max}$ (0.989) and $P_{Emax}$ (0.939) were excellent. The intraclass correlation coefficients for stroke rate (.811 to .942), stroke length (.899 to .944) and breathing frequency (.759 to .839) ranged from acceptable to high.

The occurrence of inspiratory muscle fatigue in response to 200-m swimming depended upon the velocity of the swim ($F=7.855, P=0.003$), however the absolute values for $P_{max}$ and magnitude of inspiratory muscle fatigue were unaffected by gender ($P>0.05$, table 2 and figure 1). $P_{max}$ was unaltered following $V_{crit5\%+}$ ($P=0.434$) but was lower following $V_{crit}$ ($P=0.034, d=0.32$) and $V_{crit5\%-}$ ($P=0.002, d=0.94$). However, the magnitude of inspiratory muscle fatigue did not differ between $V_{crit}$ and $V_{crit5\%}$ ($P=0.187$) despite a medium effect size ($d=0.53$) (table 2). Although $P_{Emax}$ was lower following each swim ($F=10.096, P=0.010$) the magnitude of expiratory muscle fatigue was similar between all three experimental swims ($F=1.544, P=0.238, d$ range of 0.15 to 0.26, table 2). The absolute $P_{Emax}$ values and magnitude of expiratory muscle fatigue were unaffected by gender ($P>0.05$, figure 2).

Figure 1. The magnitude of inspiratory muscle fatigue when swimming at, 5% below, and 5% above, critical velocity
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Notes: Vcrit = critical velocity. Vcrit_{5\%} = 5\% slower than critical velocity. Vcrit_{5\%} = 5\% faster than critical velocity.

As expected the percentage of 200-m race time achieved differed between swims (F=148.058, P<0.001) along with the percentage of critical stroke rate obtained (F=55.236, P<0.001). However, gender had no impact on either (P>0.05) (table 2). Similarly, stroke rate (Hz: F=56.022, P<0.001; cycles/min\(^{-1}\): F=53.372, P<0.001) and stroke length (F=19.090, P<0.001) differed between experimental swims but was unaffected by gender (P>0.05). Specifically, stroke rate was lower during Vcrit_{5\%}+ compared with Vcrit (Hz: P<0.001; cycles/min\(^{-1}\): P<0.001) and Vcrit_{5\%}-. (Hz: P<0.001; cycles/min\(^{-1}\): P<0.001) with Vcrit being lower than Vcrit_{5\%}. (Hz: P=0.006; cycles/min\(^{-1}\): P=0.007). This progressive increase from Vcrit_{5\%}+ to Vcrit_{5\%} was associated with an effect size of 0.23 to 1.18 (Hz) and 0.63 to 2.59 (cycles/min\(^{-1}\)) (table 2). Although stroke length decreased progressively (F=19.090, P<0.001) from Vcrit_{5\%}+ the reduction was only significant between Vcrit and Vcrit_{5\%}+ (P=0.014, d=0.44) and Vcrit and Vcrit_{5\%} (P<0.001, d=1.25). Stroke length was also unaffected by gender (P=0.080) (table 2). Likewise, breathing frequency was unaffected by gender (P=0.504) but did differ

between swims (F=17.773, P<0.001) for Vcrit_{5%+} and Vcrit_{5%-}. (P<0.001, d=1.61) and just missed significance between Vcrit and Vcrit_{5%-} (P=0.051, d=0.83) (table 2).

As no differences were observed between males and females the data were subsequently pooled for all further analyses. As expected the percentage of critical stroke rate obtained differed between swims (F=53.896, P<0.001) with Vcrit_{5%+} being less than Vcrit (P=0.001, d=1.40) and Vcrit_{5%-}. (P<0.001, d=2.79) and Vcrit being less than Vcrit_{5%-}. (P=0.004, d=1.67) (table 2). The development of inspiratory muscle fatigue was not correlated with breathing frequency (r=-.230, P=0.124), stroke length (r=.234, P=0.118) or absolute (m s^{-1}) swimming velocity (r=-.235, P=0.111) but it was correlated with relative swimming velocity when expressed as a percentage of race time (r=-.451, P=0.001) and percentage of Vcrit (r=-.461, P=0.001). Inspiratory muscle fatigue was also correlated with stroke rate (Hz: r=-.377, P=0.010; cycles min^{-1}: r=-.372, P=0.011) and the percentage of critical stroke rate (r=-.407, P=0.005). As expected stroke rate, stroke length, breathing frequency and velocity were correlated (P<0.05).

The development of expiratory muscle fatigue was not correlated with absolute (r=.040, P=0.794) or relative (percentage of race time: r=-.087, P=0.567; percentage of Vcrit: r=-.060, P=0.690) swimming velocity, breathing frequency (r=.083, P=0.589), stroke length (r=.175, P=0.249), stroke rate (Hz and cycles min^{-1}: r=-.093, P=0.545), or the percentage of critical stroke rate (r=-.092, P=0.549).
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Table 2 Group swim stroke, swim time and respiratory muscle characteristics when swimming at, 5% below, and 5% above, critical velocity

<table>
<thead>
<tr>
<th></th>
<th>Vcrit5%+</th>
<th>Vcrit</th>
<th>Vcrit5%-</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>group</td>
<td>males</td>
<td>females</td>
</tr>
<tr>
<td>Target time (s)</td>
<td>161.7 ± 15.2</td>
<td>159.9 ± 20.1</td>
<td>163.8 ± 7.7</td>
</tr>
<tr>
<td>Achieved time (s)</td>
<td>162.1 ± 15.4d</td>
<td>160.6 ± 20.0</td>
<td>163.8 ± 9.1</td>
</tr>
<tr>
<td>Target velocity (m s⁻¹)</td>
<td>1.24 ± 0.34d</td>
<td>1.26 ± 0.14</td>
<td>1.22 ± 0.06</td>
</tr>
<tr>
<td>200-m race time achieved (%)</td>
<td>115 ± 5d</td>
<td>117 ± 5</td>
<td>114 ± 4</td>
</tr>
<tr>
<td>Critical stroke rate (%)</td>
<td>80 ± 9df</td>
<td>77 ± 10</td>
<td>84 ± 5</td>
</tr>
<tr>
<td>Plmax pre swim (cmH₂O)</td>
<td>139 ± 29</td>
<td>146 ± 32</td>
<td>132 ± 27</td>
</tr>
<tr>
<td>Plmax post swim (cmH₂O)</td>
<td>138 ± 30if</td>
<td>143 ± 35</td>
<td>132 ± 26</td>
</tr>
<tr>
<td>PEmaxpre swim (cmH₂O)</td>
<td>132 ± 38</td>
<td>131 ± 49</td>
<td>135 ± 21</td>
</tr>
<tr>
<td>PEmax post swim (cmH₂O)</td>
<td>123 ± 32b</td>
<td>123 ± 42</td>
<td>125 ± 19</td>
</tr>
<tr>
<td>Breathing frequency (b min⁻¹)</td>
<td>25 ± 4d</td>
<td>26 ± 3</td>
<td>24 ± 5</td>
</tr>
<tr>
<td>Stroke length (m cycle⁻¹)</td>
<td>2.24 ± 0.25df</td>
<td>2.26 ± 0.32</td>
<td>2.22 ± 0.14</td>
</tr>
<tr>
<td>Stroke rate (Hz)</td>
<td>0.47 ± 0.29df</td>
<td>0.54 ± 0.07</td>
<td>0.55 ± 0.04</td>
</tr>
<tr>
<td>Stroke rate (cycles min⁻¹)</td>
<td>32.8 ± 3.3d</td>
<td>32.6 ± 3.9</td>
<td>33.0 ± 2.7</td>
</tr>
</tbody>
</table>

Vcrit5%+ = 5% slower than critical velocity. Vcrit = critical velocity. Vcrit5%-. = 5% faster than critical velocity. Critical stroke rate (%) = percentage of critical stroke rate. Plmax = maximal inspiratory mouth pressure. PEmax = maximal expiratory mouth pressure.

Different to pre swim within trial a(p<0.05) b(p<0.01). Different to Vcrit c(p<0.05) d(p<0.01). Different to Vcrit5%-. e(p<0.05) f(p<0.01).

(P=0.051) different to Vcrit5%-. 

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DISCUSSION

Vcrit represents the maximal sustainable swimming velocity (Barden and Kell, 2009; Dekerle et al., 2002; di Prampero et al., 2008) and is indicative of aerobic endurance (Toussaint et al., 1998). Of specific relevance to this study is the suggestion by Wakayoshi et al. (1995) that Vcrit represents a swimming fatigue threshold. This assertion has been supported by Dekerle et al. (2010) who found that swimming time to fatigue was delayed by up to 21 minutes when adopting a pace 5% slower than Vcrit but hastened by up to 23 minutes when swimming 5% faster. Whether this swimming fatigue threshold also represents a respiratory muscle fatigue threshold had not previously been investigated.

Our results indicate that Vcrit does represent an inspiratory muscle fatigue threshold (at least when Vcrit is calculated using the two-parameter 200-m and 400-m model) that once exceeded results in a sizeable (22%) magnitude of inspiratory muscle fatigue (table 2). This magnitude is consistent with the 11-27% typically reported following 200-m FC swimming (Jakovljevic and McConnell, 2009; Lomax et al., 2012; Lomax and McConnell, 2003). As Vcrit reflects the transition from steady-rate to non steady-rate exercise (Dekerle et al., 2010) and the highest intensity that can be sustained without eliciting maximal oxygen uptake (Hill and Ferguson, 1999), it is perhaps not surprising that inspiratory muscle fatigue was most evident above Vcrit but not below it. Indeed, swimming 5% below Vcrit is thought to reflect a heavy but sustainable exercise intensity as evidenced by stable RPE and blood lactate concentrations (Dekerle et al., 2010). In contrast, swimming at Vcrit during interval training sets, or 5% faster than Vcrit to the limit of tolerance, increases blood lactate concentration and RPE (Dekerle et al., 2010; Ribeiro et al., 2010).

The reliance on anaerobic energy pathways and the accumulation of metabolic by-products therefore increases at and above Vcrit and is reflective of the severe exercise intensity domain (Dekerle et al., 2010; Ribeiro et al., 2010). Accompanying this is an increase in total oxygen uptake (Dekerle et al., 2010) and hence the work of breathing (Hong et al., 1969). Despite a clear trend (and medium effect size of 0.53) the magnitude of inspiratory muscle fatigue between Vcrit (7%) and Vcrit5% (22%) was not statistically significant (table 2). However, this does not detract from the practical implications of such a difference. For example, an inspiratory muscle fatigue magnitude of 17-19% is associated with increased stroke rate and breathing frequency and reduced stroke length during fixed velocity FC swimming, as well as a faster rate of lower limb muscle fatigue (Lomax and Castle, 2011; McConnell and Lomax, 2006). Given that the most economical arm coordination occurs at a given velocity when stroke rate is minimised and stroke length is maximised (Wakayoshi et al., 1995) and that both streamlining and propulsion are disrupted by changes in stroke rate, stroke length and breathing frequency (Craig et al., 1985; Seifert et al., 2005; Wakayoshi et al., 1995), the magnitude of inspiratory muscle fatigue experienced in response to Vcrit5% is of practical significance.
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Our data indicate that Vcrit represents an inspiratory muscle fatigue threshold pace that is capable of inducing a statistically significant reduction in PImax (7%) in most swimmers: four males and two females demonstrated no inspiratory muscle fatigue at Vcrit, which together with the small magnitude probably explains the small effect size \((d=0.32)\) at this velocity. Whether the 7% reduction in PImax is of any practical significance is not known. This contrasts with the inspiratory muscle fatigue magnitude associated with Vcrit5%. (Lomax and Castle, 2011). Furthermore, all swimmers exhibited inspiratory muscle fatigue following Vcrit5%. Consequently, if a coach prescribes a training set designed to maximise stroke length and reduce breathing frequency and does so using a target pace above Vcrit, a swimmer's ability to achieve the desired stroke length and breathing frequency is likely to be compromised. Alternatively, if a coach wants to improve a swimmer's ability to maintain stroke length and breathing frequency in the presence of inspiratory muscle fatigue s/he should select a velocity in excess of Vcrit.

It is also important to note that neither the occurrence nor the magnitude of inspiratory muscle fatigue differed between male and female swimmers (figure 1), although there was a non-significant trend for baseline PImax to be higher in males (table 2). All swimmers were of a similar adolescent age (table 1) but the absolute values observed for PImax were substantially higher than that observed in 13-18 year old adolescents (Smyth et al., 1984) and 15-17 year old male and female Brazilian international swimmers (Santos et al., 2011). Indeed our values were 35 to 42 cmH\(_2\)O and 56 to 65 cmH\(_2\)O higher in males and females, respectively. It could therefore be argued that baseline inspiratory muscle strength was uncharacteristically high in our group of adolescent swimmers, and in particular the female swimmers, masking any gender bias. In support of this McConnell (2007) suggested that normal PImax values for healthy young people are typically 110-140 cmH\(_2\)O for males and 90-120 cmH\(_2\)O for females.

The occurrence of inspiratory muscle fatigue was correlated with relative velocity (i.e. percentage of critical velocity and percentage of race time), and absolute stroke rate and relative stroke rate (i.e. percentage of critical stroke rate). As an increase in velocity will naturally cause an increase in stroke rate and decrease in stroke length (Barden and Kell, 2009; Craig et al., 1985; Craig and Pendergast, 1979) the correlations observed in the present study are to be expected. The origin of the correlations between inspiratory muscle fatigue and stroke kinematics are, however, difficult to interpret. For example, given that Vcrit represents a swimming fatigue threshold, swimming above Vcrit might result in local muscular fatigue of the upper body muscles. In this situation the ability to overcome the resistance to forward movement would fall along with force output per stroke and stroke length (Craig et al., 1985). To compensate stroke rate increases but this is likely to exacerbate local muscular fatigue further (Craig and Pendergast, 1979). As accessory inspiratory muscles are critical for supporting breathing during swimming as well as aiding propulsion and stabilization (Kendall et al., 2005; McLeod, 2010), such stroke induced
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fatigue might impact the inspiratory muscles independently of any breathing induced fatigue.

The observation of expiratory muscle fatigue necessitates comment here. Even though the occurrence of expiratory muscle fatigue is a novel finding in swimming, the magnitude was similar between genders (figure 2) and all experimental swims (5-8%, \( P<0.01 \), table 2). Similar to PImax we observed superior strength of the expiratory muscles in both males (by 15-46 cmH\(_2\)O) and females (by 52-68 cmH\(_2\)O) when compared to adolescent elite Brazilian swimmers and healthy adolescents (Santos et al., 2011; Smyth et al., 1984); although our values are consistent with those suggested by McConnell (2007). Nevertheless, the magnitude of expiratory muscle fatigue we observed was substantially smaller than that reported following exhaustive cycling exercise (Taylor et al., 2006) or that shown to exacerbate whole body fatigue (Verges et al., 2007). Furthermore, expiratory muscle fatigue could not be correlated with any of the measured variables. Given that firstly, expiration is aided during exercise by the relaxation of the inspiratory muscles and natural recoil of the lungs (West, 2012), and secondly, that increased pressure around the chest when immersed will actually assist the expiratory process (Lundgren, 1999), one might expect the magnitude of expiratory muscle fatigue to be smaller than inspiratory muscle fatigue during swimming. Nevertheless, the forced expiratory manoeuvre into water might create a small additional expiratory muscle load (Cordain and Stager, 1988) while the hydrostatic pressure and pulmonary engorgement can narrow the large and dependent airways increasing expiratory flow resistance (Lundgren, 1999).

As previous reports have attributed expiratory muscle fatigue during terrestrial exercise to abdominal muscle fatigue (Suzuki et al., 1991; Taylor et al., 2006; Verges et al., 2007), it is relevant to note that the rectus abdominis is activated for over 90% of the FC stroke cycle making it one of the most active muscles (Clarys, 1985). Moreover, the rectus abdominis contributes substantially to the PEmax manoeuvre, so fatigue of this muscle would impact any resultant PEmax value (Gibson, 1995). Despite the attractiveness of attributing expiratory muscle fatigue to reduced rectus abdominus force output we have no evidence to support this. Our assessment of PEmax did not permit the evaluation of individual muscles and was instead a global measure of expiratory muscle function. In addition, the work history of the abdominal muscles is correlated with the magnitude of expiratory muscle fatigue (Taylor et al., 2006). If abdominal muscle fatigue and hence expiratory muscle fatigue was peripheral in origin the magnitude we observed should have increased with velocity, but did not. This raises the possibility that central fatigue caused the reduction in PEmax. We think that this is unlikely because PImax would have been affected in a similar way. Likewise, if dyspnoea or the elevated breathing rate prevented swimmers from obtaining total lung capacity prior to the PEmax manoeuvre, residual volume would also be impeded and hence PImax also affected. Critically however, inspiratory muscle fatigue was absent following Vcrit\(_{5\%}\) and then fell
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progressively thereafter. Thus, we are unable to identify the origin of expiratory muscle fatigue in the present study.

Finally, our decision to base Vcrit on 200-m and 400-m performances requires justification. The intensity associated with Vcrit differs in some (Toubekis et al., 2011; Zacca and Castro, 2010), but not all (Garatachea et al., 2006) studies depending upon the swimming distances used to derive it. Basing Vcrit on distances of 200-m and 400-m is more sustainable than when shorter distances (i.e. 100-m) are included. This is because the exercise intensity associated with the former reflects the lower limit of the severe intensity domain and is sustainable during repetitive training (Ribeiro et al., 2010; Toubekis et al., 2011). Conversely, integrating shorter distances may shift Vcrit to the upper limit of the severe intensity domain and hence require a swimming velocity that is not sustainable (Toubekis et al., 2011). We followed the practical advice of others in selecting a two-parameter distance model. Specifically, the distances of 200-m and 400-m are thought to be the most relevant way for coaches to determine Vcrit (Dekerle, 2006; Dekerle et al., 2002; Ribeiro et al., 2010; Wakayoshi et al., 1993). Thus, our finding that Vcrit represents an inspiratory muscle fatigue transition threshold is specific to the 200-m and 400-m two-parameter model. We cannot rule out the possibility that different results would have been observed had other distances been included in the calculation of Vcrit: this remains to be seen.

CONCLUSIONS

We have provided data demonstrating that inspiratory muscle fatigue is correlated with relative but not absolute FC swimming velocity, and stroke rate. Whilst this finding is obvious and to be expected, the novel aspect is that we were able to determine the relative velocity associated with the development of inspiratory muscle fatigue. Specifically, inspiratory muscle fatigue occurred when swimming at (in some) and above (in all) Vcrit and when stroke rate was at (in some) or above (in all) 92% of critical stroke rate in both male and female adolescent swimmers. Vcrit therefore represents a relative FC velocity which if exceeded by as little as 5% results in a sizeable magnitude of inspiratory muscle fatigue. Importantly, this magnitude is consistent with previous studies demonstrating altered stroke kinematics and a quicker rate of limb muscle fatigue and therefore has important implications for coaches when prescribing intensities for training sets designed to enhance stroke kinematics. Conversely, a small magnitude of expiratory muscle fatigue was evident following Vcrit5%+, Vcrit and Vcrit5%− but its practical significance and origin is unknown.

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