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1 Original article

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3 **Importance of dimensional changes on glycolytic metabolism during growth**

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30 **AUTHOR CONTRIBUTION STATEMENT**

31 AD, SR, CT, and HM conceived and designed research. AD, SR, JB, CT and HM conducted experiments and
32 collected data. AD, SR, QDL and HM analysed data. AD, SR, NA, CT and HM wrote the manuscript. AD, SR,
33 JB, QDL, NA, CT and HM provided critical revisions important for intellectual content of the finished
34 manuscript, approved the final version of the manuscript, and agree to be accountable for all aspects of the work
35 in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately
36 investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for
37 authorship are listed.

38

39 **CONSENT TO PARTICIPATE**

40 Written informed consent was obtained from all individual included in the study, and from their parents or legal
41 guardians.

42

43 **CONSENT TO PUBLISH**

44 Participants (and their parents or legal guardians) signed informed consent regarding publishing their data.

45

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49

50 **CONFLICT OF INTEREST**

51 The authors declare that they have no conflict of interest.

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52 **ABSTRACT**

53 **Purpose:** The aim of the present study was to investigate (i) how glycolytic metabolism assessed by
54 accumulated oxygen deficit (AOD_{gly}) and blood metabolic responses (lactate and pH) resulting from high-
55 intensity exercise change during growth, and (ii) how lean body mass (LBM) influences AOD_{gly} and its
56 relationship with blood markers.

57 **Methods:** Thirty-six 11- to 17-year-olds performed a 60-s all-out test on a rowing ergometer. Allometric
58 modelling was used to investigate the influence of LBM and LBM + maturity offset (MO) on AOD_{gly} and its
59 relationship with the extreme post-exercise blood values of lactate ($[La]_{max}$) and pH (pH_{min}) obtained during the
60 recovery period.

61 **Results:** AOD_{gly} and $[La]_{max}$ increased while pH_{min} decreased linearly with LBM and MO ($r^2 = 0.46$ to 0.72 , $p <$
62 0.001). Moreover, AOD_{gly} was positively correlated to $[La]_{max}$ ($r^2 = 0.75$, $p < 0.001$) and negatively correlated to
63 pH_{min} ($r^2 = 0.77$, $p < 0.001$). When AOD_{gly} was scaled for LBM, the coefficients of the relationships with blood
64 markers drastically decreased by three to four times ($[La]_{max}$: $r^2 = 0.24$, $p = 0.002$; pH_{min} : $r^2 = 0.30$, $p < 0.001$).

65 Furthermore, by scaling AOD_{gly} for LBM + MO, the correlation coefficients with blood markers became even
66 lower ($[La]_{max}$: $r^2 = 0.12$, $p = 0.037$; pH_{min} : $r^2 = 0.18$, $p = 0.009$). However, MO-related additional changes
67 accounted much less than LBM for the relationships between AOD_{gly} and blood markers.

68 **Conclusion:** The results challenge previous reports of maturation-related differences in glycolytic energy
69 turnover and suggest that changes in lean body mass are a more powerful influence than maturity status on
70 glycolytic metabolism during growth.

71

72 **Keywords:** accumulated oxygen deficit, lactate, allometric modelling, maturation, rowing, adolescent

73 **ABBREVIATIONS**

AOD _{gly}	Glycolysis-derived accumulated oxygen deficit
AOD _{tot}	Total accumulated oxygen deficit
APHV	Age at peak height velocity
[BE] _{min}	Minimal base excess concentration
BF	Body fat
BM	Body mass
BMI	Body mass index
CA	Chronological age
[HCO ₃ ⁻] _{min}	Minimal bicarbonate concentration
HR _{max}	Maximal heart rate
LBM	Lean body mass
[La] _{max}	Maximal lactate concentration
MO	Maturity offset
MPO	Mean power output
pH _{min}	Minimal pH
P \dot{V} O _{2max}	Power at maximal oxygen uptake
OE _{phos+ox}	phosphagen- and blood O ₂ stores-derived oxygen equivalent
\dot{V} O _{2max}	Maximal oxygen uptake

74

75 INTRODUCTION

76 Following their finding of lower muscle lactate concentration resulting from maximal exercise in 13.6-
77 year-old boys compared with young men, Eriksson et al. (1971) suggested that glycolytic activity was lower in
78 children than adults. Furthermore, a lower activity level of muscle phosphofructokinase was found in 11- to 13-
79 year-old boys as compared with 17- to 58-year-old adults (Eriksson et al. 1973; Gollnick et al. 1972). Other
80 studies also reported lower glycolytic enzyme activities (lactate dehydrogenase, aldolase, pyruvate kinase) in 3-
81 to 17-year-old children compared with 29- to 54-year-old adults (Berg et al. 1986; Kaczor et al. 2005). In
82 addition, considering the significant positive relationships between testicular volume index and exercise-induced
83 muscle lactate accumulation (Eriksson et al. 1971) and between salivary/serum testosterone concentration and
84 peak blood lactate responses, some authors supported the contention that glycolysis is dependent on maturity
85 status (Falgairette et al. 1991; Fellmann et al. 1988)

86 However, conclusions from early studies of the potential immaturity of glycolytic activity before
87 puberty must be interpreted cautiously, as these studies were not systematically designed to test the effects of
88 maturation and employed small sample numbers with a lack of continuum throughout the maturation process. In
89 metabolic studies, children were mostly categorised as pre-pubertal and post-pubertal and issues related to the
90 effect of maturation during the circumpubertal period have not been comprehensively addressed (Ratel et al.
91 2002). Also, early metabolic studies did not integrate dimensional changes into data interpretation from
92 childhood into adolescence. Yet, scientific evidence highlighted the importance of total working muscle mass
93 and muscle power output on lactate production in humans (Jensen-Urstad et al. 1994). In mammals, it has been
94 also shown that activities of enzymes functioning in anaerobic glycogenolysis (glycogen phosphorylase,
95 pyruvate kinase and lactate dehydrogenase) increase with increasing body mass (Emmett and Hochachka 1981).
96 Moreover, while nonoxidative energy supply assessed by the accumulated oxygen deficit (AOD) was found to
97 be positively associated with muscle mass involved during exercise (Bangsbø et al. 1993), it was also found to
98 be correlated to the quantity of lactate accumulated after all-out exercise in adult rowers (Maciejewski et al.
99 2013) and muscle lactate concentration following maximal exercise in pubertal children (Eriksson et al. 1971).
100 This suggests that there might be a dimensional effect on glycolysis-derived accumulated oxygen deficit
101 (AOD_{gly}), metabolic by-products accumulation and their interrelation throughout growth, but this remains to be
102 proven.

103 Taken together, the lower blood metabolic disturbances (i.e., lower blood lactate accumulation and
104 higher pH) resulting from high-intensity exercise in young children (Ratel et al. 2002) could be related to their

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105 lower glycolytic energy supply or reduced AOD_{gly} (Carlson and Naughton 1993) owing to their smaller body
106 dimensions, e.g., lean body mass (LBM). Such arguments suggest that with LBM accounted for there might not
107 be significant maturation-related differences in glycolytic energy turnover between pre-pubertal, mid-pubertal
108 and post-pubertal children, contrary to what has been previously reported (Eriksson et al. 1971). However, direct
109 evidence showing the respective influence of LBM and maturity status on the differences in AOD_{gly} and the
110 potentially associated blood metabolic disturbances (i.e., lactate, pH) between pre-, mid- and post-pubertal
111 children is still lacking.

112 Therefore, the purpose of the present study was to determine whether the increase in glycolytic
113 metabolism during growth, assessed by AOD_{gly} and blood metabolic responses (i.e., increased blood lactate
114 accumulation and decreased pH) is principally influenced by concomitant changes in LBM. We hypothesise that
115 the lower blood lactate accumulation and higher pH resulting from high-intensity exercise in younger children
116 could be explained by a reduced amount of energy released from glycolysis (i.e., AOD_{gly}) owing to a smaller
117 LBM rather than maturity status. A proportional allometric modelling approach will be used to check our
118 assumptions by controlling for the effects of LBM and maturity status.

119

120 MATERIALS AND METHODS

121 Participants

122 Thirty-six male competitive rowers aged from 11 to 17 years volunteered to participate in the present
123 study. All participants trained three to six times per week (i.e., two to four “on-water” training sessions and one
124 to two physical training sessions) preceding the experiments. None of the participants had a family history of
125 cardiovascular disease or was using any medication. The present study was approved by an institutional ethics
126 review board (Comité d'Éthique pour la Recherche en Sciences et Techniques des Activités Physiques et
127 Sportives – CERSTAPS, n° 2017-29-11-20) and conformed to the standards of use of human participants in
128 research as outlined in the *Sixth Declaration of Helsinki*. The participants were informed of the experimental
129 procedures and gave their written assent before any testing was conducted. In addition, the written informed
130 consent was obtained from the parents or legal guardians of the participants.

131

132 Experimental procedure

133 Volunteers were tested in two experimental sessions separated by at least 48 hours. Participants were
134 instructed not to undertake any strenuous activity during the 24 hours preceding each session. The first session

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135 was dedicated to gathering participants' physical characteristics (anthropometric measurements, body
136 composition and maturity status) and maximal oxygen uptake ($\dot{V}O_{2max}$) assessment. During the second session,
137 the volunteers performed a 60-s all-out test. The two exercise sessions were carried out on a rowing ergometer
138 (Model D, Concept2, Morrisville, VT, USA). The young rowers were fully familiarised with the equipment. The
139 computer of the ergometer continuously delivered the power output and stroke rates (in W and min^{-1} ,
140 respectively). The resistance factor was set by the investigators between 100 and 130 according to age and the
141 expertise level of young rowers. The same resistance factor was kept for both tests. Verbal encouragement was
142 systematically provided by the investigators during each exercise session.

143

144 **Experimental measurements**

145 *Session 1: Anthropometric characteristics and body composition.* Body mass (BM in kg) was measured using a
146 digital weight scale with a precision of ± 0.01 kg (Seca 899, Seca, Germany). Standing height (in m) was
147 assessed using a stadiometer with a precision of ± 1 mm (Seca 213, Seca, Germany). Sitting height (in m) was
148 also measured with the stadiometer while the participants sat on the floor with their back against a wall. Body
149 mass index (BMI) was subsequently calculated using a standard formula, as follows: mass divided by height
150 squared (in $\text{kg}\cdot\text{m}^{-2}$). Skinfold thicknesses were measured in triplicate at the triceps and subscapular sites using a
151 Harpenden calliper (British Indicators Ltd, St Albans, UK). The measurements were taken by the same
152 experienced investigator on the right side of the body to reduce variability in the results. Body fat percentage (BF
153 in %) and lean body mass (LBM in kg) were determined using Slaughter's et al. equations (Slaughter et al.
154 1988). These equations are specific to sex, ethnicity and maturity status, and are recommended for assessing BF
155 and LBM in children 8-18 years of age.

156

157 *Maturity status.* Maturity offset (MO in years) was determined to assess somatic maturity (i.e., years to (from)
158 age at peak height velocity, APHV) by using chronological age (CA), BM, standing height and sitting height. Its
159 calculation was based on sex-specific regression equations according to the method proposed by Mirwald et al.
160 (2002). Children were categorised by their maturity status (pre-, mid- or post-PHV) into discrete bands based on
161 their MO (pre-PHV: < -1 , mid-PHV: -1 to $+1$, post-PHV: > 1) (Birat et al. 2020).

162

163 *Maximal oxygen uptake test.* Each participant performed a progressive test to exhaustion to determine maximal
164 O_2 uptake ($\dot{V}O_{2max}$ in $\text{L}\cdot\text{min}^{-1}$). The initial power was set between 40 and 80 W during the first five minutes and

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165 the power was incremented by 10-30 W every three minutes according to age and the expertise level of
166 participants (Maciejewski et al. 2013). Arterialised capillary blood samples (20 μL) were taken from the earlobe
167 at rest and every step to measure the time course of blood lactate concentration. Whole blood lactate
168 concentrations ($[\text{La}]$ in $\text{mmol}\cdot\text{L}^{-1}$) were determined enzymatically using a Biosen C-Line Clinic lactate analyser
169 (EFK Diagnostics GmbH, Barleben, Germany).

170 Oxygen uptake, carbon dioxide output and ventilation were continuously monitored with a breath-by-breath
171 analyser (Quark CPET, Cosmed, Italy). Heart rate was continuously recorded with a heart rate monitor (HRM-
172 Dual, Garmin, Kansas, USA). The mechanical power output corresponding to $\dot{V}\text{O}_{2\text{max}}$ ($P\dot{V}\text{O}_{2\text{max}}$ in W) and the
173 linear relationship between $\dot{V}\text{O}_2$ and power output were also assessed. $\dot{V}\text{O}_{2\text{max}}$ was considered to be reached
174 during the last step when at least two of the following criteria were met: (i) $\dot{V}\text{O}_2$ levelling-off, (ii) maximal
175 respiratory exchange ratio ($\text{RER}_{\text{max}} \geq 1.1$), (iii) maximal HR ($\text{HR}_{\text{max}} \geq 95\%$ of the age-predicted HR_{max} ($208.6 -$
176 $0.7 \cdot \text{age}$) (age in years) (Shargal et al. 2015) and (iv) blood lactate concentration higher than $8 \text{ mmol}\cdot\text{L}^{-1}$.

177

178 *Session 2: 60-s all-out test.* After a standardised 20-min warm-up at about $130\text{-}140 \text{ beats}\cdot\text{min}^{-1}$ and two short
179 sprints (10-s) in the last five minutes, all participants performed a 60-s all-out test. This test was followed by a
180 10-min sitting recovery. Cardio-respiratory parameters were continuously measured using a breath-by-breath
181 analyser (Quark CPET, Cosmed, Italy). Capillary arterialised blood samples ($80 \mu\text{L}$) were drawn from the
182 earlobe and collected after warm-up and at 1, 3, 5, and 8 min post-exercise to measure the time course of pH, and
183 lactate ($[\text{La}]$ in $\text{mmol}\cdot\text{L}^{-1}$), bicarbonate ($[\text{HCO}_3^-]$ in $\text{mmol}\cdot\text{L}^{-1}$) and base-excess ($[\text{BE}]$ in $\text{mmol}\cdot\text{L}^{-1}$)
184 concentrations. Blood $[\text{La}]$ was determined enzymatically using the same lactate analyser as in the first session
185 while pH, $[\text{HCO}_3^-]$ and $[\text{BE}]$ were measured by direct potentiometry using an i-STAT® handheld analyser
186 (Abbott Point of Care, Princeton, USA) immediately after collection. The maximal lactate concentration ($[\text{La}]_{\text{max}}$
187 in $\text{mmol}\cdot\text{L}^{-1}$), the minimal pH value (pH_{min}), and the minimal concentrations of bicarbonate ($[\text{HCO}_3^-]_{\text{min}}$ in
188 $\text{mmol}\cdot\text{L}^{-1}$) and base-excess ($[\text{BE}]_{\text{min}}$ in $\text{mmol}\cdot\text{L}^{-1}$) were identified. Mean power output (MPO in W) was
189 calculated over the entire test. In addition, total accumulated oxygen deficit (AOD_{tot} in $\text{L O}_2 \text{ Eq.}$) and glycolysis-
190 derived accumulated oxygen deficit (AOD_{gly} in $\text{L O}_2 \text{ Eq.}$) were individually determined according to the
191 procedures described below.

192

193

194

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195 **Measurements and calculations**

196 *Total accumulated oxygen deficit.* AOD_{tot} was determined by subtracting accumulated O_2 uptake (the measured
197 O_2 uptake integrated over time) from accumulated O_2 demand (the estimated O_2 demand integrated over time).
198 In accordance with Green and Dawson (1996), oxygen demand was extrapolated using the equation of the $\dot{V}O_2$ -
199 power output linear regression obtained in session 1 and considering the individual value of $\dot{V}O_{2rest}$ (i.e., $\dot{V}O_2$
200 measured during three min before the test) for a power output equal to 0 W. Because the present study concerned
201 all-out exercise, O_2 demand was calculated from instantaneous power output (recorded stroke by stroke) rather
202 than mean power output sustained during exercise (i.e., MPO) as initially proposed by Medbø et al. (1988).

203
204 *Glycolysis-derived AOD.* AOD_{gly} was assessed by subtracting phosphagen- and blood O_2 stores-derived oxygen
205 equivalent ($OE_{phos+ox}$) to AOD_{tot} . $OE_{phos+ox}$ was evaluated from the integral of the fast component of $\dot{V}O_2$
206 recovery kinetic using a bi-exponential 4-parameter model, as previously done (Beneke et al. 2002; di Prampero
207 1981):

$$209 \quad y = A_1 \cdot e^{-\frac{t}{\tau_1}} + A_2 \cdot e^{-\frac{t}{\tau_2}} + y_0 \quad (\text{Eq. 1})$$

210
211 where A_1 , A_2 (in $L \cdot \text{min}^{-1}$) are the amplitudes of fast and slow components, respectively; τ_1 and τ_2 the
212 corresponding time constants (in min), and y_0 (in $L \cdot \text{min}^{-1}$) the asymptotic resting $\dot{V}O_2$ at time $\rightarrow \infty$.
213 Fitted $\dot{V}O_2$ data were then integrated over time from the start of recovery to τ_1 value, and resting $\dot{V}O_2$ value
214 integrated over the same time was subtracted.

215
216 *Allometric modelling procedures.* Allometric approach is used to remove any dimensional effect on parameters,
217 and thereby allow fair comparisons between populations of different body dimensions and compositions. As the
218 large range of LBM in the studied population (30.1 to 78.7 kg) may have influenced the capacity to supply
219 glycolytic nonoxidative energy (i.e., AOD_{gly}), we further investigated the influence of LBM on the relationships
220 between AOD_{gly} and extreme blood metabolic responses (i.e., $[La]_{max}$, pH_{min} , $[HCO_3^-]_{min}$ and $[BE]_{min}$), by
221 analysing these relationships with LBM as scaling factor through an allometric modelling procedure. The
222 allometric relationship obtained between LBM and AOD_{gly} was based on the general allometric equation (Nevill
223 et al. 1992):

224

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225
$$AOD_{gly} = a_1 \cdot LBM^{b_1} \quad (\text{Eq. 2})$$

226

227 where a_1 is the proportionality coefficient and b_1 the scaling factor associated with LBM. The resultant power
228 function ratio $AOD_{gly} \cdot LBM^{b_1}$ is allegedly free from the confounding influence of LBM. The statistical approach
229 to allometry is to use a simple logarithmic transformation as follows:

230

231
$$\log(AOD_{gly}) = b_1 \cdot \log(LBM) + \log(a_1) \quad (\text{Eq. 3})$$

232

233 where b_1 is the slope of the linear regression. This slope is calculated by regression analysis, where b_1 in the
234 regression output is equal to the scaling factor and the inverse log of $\log a_1$ is equivalent to the constant a_1 in the
235 Eq. 2.

236 If this first methodological approach totally accounts for the effects of LBM on AOD_{gly} , it is important to
237 underline that LBM could also be influenced by individual maturity status. As a result, we also used the
238 multiplicative allometric model proposed by Nevill and Holder (1994) including the maturity indicator as a
239 second factor within an exponential term in addition to the LBM component. This procedure takes into account
240 both LBM and the effect of MO on LBM on AOD_{gly} as follows:

241

242
$$AOD_{gly} = LBM^{b_2} \cdot \exp(a_2 + c \cdot MO) \quad (\text{Eq. 4})$$

243

244 where a_2 is the proportionality coefficient, b_2 and c are the scaling factors associated with LBM and MO,
245 respectively.

246

247 The statistical approach to allometry is to use a multiple logarithmic transformation (as previously done by
248 Carvalho et al. 2012), as follows:

249

250
$$\log(AOD_{gly}) = b_2 \cdot \log(LBM) + a_2 + c \cdot MO \quad (\text{Eq. 5})$$

251

252 where b_2 and c are the slopes of the multiple linear regression. These slopes are calculated by ordinary multiple
253 regression analysis where b_2 and c are equal to the scaling factors and the inverse log of $\log a_2$ is equivalent to
254 the constant a_2 in Eq. 4.

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255 **Statistical analysis**

256 Analyses were performed using OriginPro 2020 software (OriginLab, Massachusetts, USA). Descriptive
257 statistics were expressed as mean \pm standard deviation (SD) by maturity groups (pre, mid, post). Differences
258 between maturity groups were analysed using the non-parametric Kruskal-Wallis test. Mann-Whitney test was
259 completed for pairwise comparisons when the Kruskal-Wallis test revealed a significant effect. Linear regression
260 models between MO, LBM, AOD_{gly}, and blood parameters (i.e., [La]_{max}, pH_{min}, [HCO₃⁻]_{min} and [BE]_{min}) were
261 fitted by the least-squares method. The squared Bravais-Pearson correlation coefficients (r^2) of these linear
262 regression models were calculated. The statistical significance level was set at 5% (i.e. $p < 0.05$).

263

264 **RESULTS**

265 *Participants' physical and physiological characteristics*

266 The physical characteristics and aerobic fitness of participants are detailed by maturity groups in Table 1. CA,
267 MO, standing height, BM, BMI, LBM, $\dot{V}O_{2max}$ (L·min⁻¹) and $\dot{P}\dot{V}O_{2max}$ significantly increased while BF
268 significantly decreased from the pre-PHV to post-PHV group ($p < 0.001$).

269

270 ***Table 1 around here***

271

272 *60-s all-out test*

273 Mechanical and physiological parameters obtained from the 60-s all-out test (i.e., MPO, AOD_{tot}, AOD_{gly},
274 OE_{phos+ox}, [La]_{max}, pH_{min}, [HCO₃⁻]_{min}, [BE]_{min}) are detailed by maturity groups in Table 2. MPO, AOD_{tot}, AOD_{gly}
275 OE_{phos+ox}, and [La]_{max} significantly increased while pH_{min}, [HCO₃⁻]_{min} and [BE]_{min} significantly decreased after
276 the 60-s all-out exercise from the pre-PHV to post-PHV group ($p < 0.001$).

277

278 ***Table 2 around here***

279

280 *Allometric modelling exponents*

281 Allometric scaling exponents a_1 and b_1 obtained by the simple procedure (i.e., Eq. 2) were -2.40 and 1.60,
282 respectively. Allometric scaling exponents a_2 , b_2 and c obtained from the multiple procedure (i.e., Eq. 4) were -
283 3.96, 1.19 and 0.08, respectively.

284

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285 *Correlations between variables*

286 Relationships between metabolic responses, MO and LBM are displayed in Table 3. AOD_{gly}, [La]_{max}, pH_{min},
287 [HCO₃⁻]_{min} and [BE]_{min} were significantly correlated to MO and LBM ($p < 0.001$ for all) (Table 3).

288

289 ***Table 3 around here***

290

291 In addition, [La]_{max} ($r^2 = 0.75$, $p < 0.001$), pH_{min} ($r^2 = 0.77$, $p < 0.001$), [HCO₃⁻]_{min} ($r^2 = 0.69$, $p < 0.001$) and
292 [BE]_{min} ($r^2 = 0.74$, $p < 0.001$) were highly correlated to AOD_{gly} (Figures 1A, 1B, 1C, 1D, respectively). However,
293 when AOD_{gly} was scaled for LBM^{1.60} the determination coefficients of the relationships with blood markers
294 drastically decreased by three to four times ([La]_{max}: $r^2 = 0.24$, $p = 0.02$; pH_{min}: $r^2 = 0.30$, $p < 0.001$; [HCO₃⁻]_{min}:
295 $r^2 = 0.19$, $p = 0.008$; [BE]_{min}: $r^2 = 0.18$, $p = 0.009$) (Figures 1E, 1F, 1G, 1H, respectively). Furthermore, by
296 considering the additional effect of MO on LBM, the correlation coefficients between AOD_{gly} and blood markers
297 became even lower ([La]_{max}: $r^2 = 0.12$, $p = 0.037$; pH_{min}: $r^2 = 0.18$, $p = 0.009$; [HCO₃⁻]_{min}: $r^2 = 0.11$, $p = 0.051$;
298 [BE]_{min}: $r^2 = 0.10$, $p = 0.061$) (Figures 1I, 1J, 1K, 1L, respectively).

299

300 ***Figure 1 around here***

301

302 **DISCUSSION**

303 The present study aimed to investigate (i) how glycolytic metabolism assessed by accumulated oxygen
304 deficit (AOD_{gly}) and blood metabolic responses (i.e., lactate and pH) changes during growth and (ii) how
305 concomitant changes in lean body mass (LBM) influence AOD_{gly} and its relationship with lactate accumulation
306 and pH reduction. The main results were consistent with our hypotheses since (i) AOD_{gly} and blood metabolic
307 responses concurrently increased with maturity status (MO) and lean body mass (LBM), and were correlated,
308 and (ii) concomitant changes in LBM accounted much more than maturity status for AOD_{gly} and its relationship
309 with lactate accumulation and pH reduction. The results indicate that with LBM accounted for there may not be
310 significant maturation-related differences in glycolytic energy turnover, contrary to what has been previously
311 mentioned.

312

313 In the present study, MO and LBM highly accounted for the increment of AOD_{gly} (71% and 61%,
314 respectively (Table 3). This finding is consistent with previous cross-sectional studies showing that (i) total AOD

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315 is significantly lower in children than adolescents and adults during high-intensity exercise (Carlson and
316 Naughton 1993; Naughton et al. 1997) and (ii) the kinetics of O₂ uptake at the onset of high-intensity exercise
317 are faster in children compared with adults (Armon et al. 1991). The results suggest that prepubertal children
318 may adapt their oxidative metabolism faster than their older counterparts to meet the higher energy requirements
319 and, hence, have a lower need for nonoxidative metabolism at the onset of high-intensity exercise. However, the
320 present data indicate for the first time a progressive increase in glycolysis-specifically derived AOD from
321 childhood into adolescence and this is associated with LBM and MO (Table 3).

322
323 The blood metabolic responses (i.e., [La]_{max}, pH_{min}, [HCO₃⁻]_{min}, [BE]_{min}) were also highly associated with LBM
324 and MO (Table 3). These findings are consistent with previous studies showing lower end-of-exercise blood [La]
325 and higher pH in prepubertal boys than men, either after a Wingate test (Hebestreit et al. 1996) or following
326 repeated cycle sprints (Ratel et al. 2002). However, in the present study, the metabolic stress elicited by high-
327 intensity exercise during rowing was higher than from other forms of exercise in children. Indeed, in previous
328 studies, high-intensity exercises yielded post-exercise peak blood [La] from 7.7 to 8.4 mmol·L⁻¹ during cycling
329 (Falgairrette et al. 1991) or 7.8 to 11.0 mmol·L⁻¹ during running (Paterson et al. 1986) between the ages of 10 and
330 15 years while data from our population ranged from 10.2 to 15.9 mmol·L⁻¹ (Table 2). Similarly, end-of-exercise
331 blood pH values of 7.36-7.37 in 10-year-old boys and 7.28 in 15-year-old male adolescents after repeated sprints
332 have been reported (Kappenstein et al. 2015; Ratel et al. 2004) while values obtained in the present study were
333 7.24 in pre-pubertal boys and 7.13 in post-pubertal boys. Such discrepancies could be ascribed to differences in
334 body mass engaged during exercise as blood metabolic responses were found to be highly associated with LBM.
335 Even if the body mass is not supported in rowing, it involves a greater muscle mass during exercise [about 85%
336 of the total muscle mass (Mader et al. 1988; Maciejewski et al. 2013)], than other modalities of exercise such as
337 cycling [about 25 to 30% of body mass (Gastin 2001)], or running (Bangsbø et al. 1993). The greater muscle
338 mass involved in rowing could elicit a greater glycolytic energy supply and thereby a higher blood lactate
339 accumulation and pH or [HCO₃⁻] reduction (Bangsbø et al. 1993; Saltin 1990). However, direct evidence
340 showing the effect of exercise mode on blood metabolic responses during high-intensity exercise during growth
341 and maturation is still lacking.

342
343 Another interesting point is that AOD_{gly} was highly correlated to [La]_{max}, pH_{min}, [HCO₃⁻]_{min} and [BE]_{min}
344 (Figures 1A, 1B, 1C, 1D). The higher the AOD_{gly}, the greater glycolytic by-products accumulation found during

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345 growth. This finding is novel as no previous studies have analysed the relationships between the (total and
346 glycolysis-derived) accumulated O₂ deficit and blood acid-base disturbance in response to high-intensity
347 exercise during growth. Only Naughton et al. (1997) evaluated total AOD and plasma markers of anaerobic
348 metabolism (lactate, pH, ammonium) during treadmill runs at 120-130% of $\dot{V}O_{2max}$ in 14-year-old trained male
349 and female adolescents but these authors did not establish any relationship between the parameters. In adults,
350 previous studies have provided contradictory data on these relationships; some authors failed to find significant
351 correlations between [La]_{max} and total AOD in elite athletes (Bangsbø et al. 1993) while others found an
352 association between the quantity of lactate accumulated and total AOD in well-trained rowers (Maciejewski et
353 al. 2013). This discrepancy could be related to methodological issues given that [La]_{max} reflects the balance
354 between production and removal of lactate at the time of measurement while the quantity of lactate accumulated
355 is an estimate of total production considering the quantity of lactate removed from the body. Although in the
356 present study the quantity of lactate accumulated was not assessed and [La]_{max} was measured, AOD_{gly} highly
357 accounted for the variance of blood lactate concentration (i.e., 75%). This was also the case for the other blood
358 metabolic responses (i.e., 77%, 69% and 74% for pH_{min}, [HCO₃⁻]_{min} and [BE]_{min}, respectively).

359 In the present study, the allometric exponent calculated to reduce the effect of LBM on AOD_{gly} was
360 positive (1.60), supporting the notion that AOD_{gly} increased with LBM. However, this allometric exponent was
361 clearly higher than the coefficients usually calculated between body mass and $\dot{V}O_{2max}$ (e.g., 0.75) (Armstrong
362 and Welsman 2019; Nevill et al. 1992) or between LBM and $\dot{V}O_{2max}$ (i.e., 1.13) in the present study (Table 1).
363 This allometric exponent is consistent with the high body size allometric exponents reported during high-
364 intensity exercises, as for instance in adolescent rowers during a 30-s all-out test (i.e., 1.24) (Maciejewski et al.
365 2016). Consequently, body size exponents could be higher in anaerobic vs aerobic exercise conditions, and
366 anaerobic metabolic responses could be greater in heavier than lighter individuals, as it is the case for AOD_{gly} in
367 the present study. This could be ascribed to additional anaerobic energy required to move (lean) body mass
368 during all-out exercise compared to aerobic exercise, particularly in heavier individuals.

369
370 The results of the present study suggest that LBM is the main influence on the AOD_{gly} and its
371 relationship with blood parameters. When the effect of LBM was considered in the relationships between
372 AOD_{gly} and [La]_{max}, pH_{min}, [HCO₃⁻]_{min} and [BE]_{min}, the coefficients of determination drastically decreased by
373 three to four times with respect to those obtained from absolute AOD_{gly} values (Figures 1E, 1F, 1G, 1H). The
374 additional effect of MO on these relationships accounted for much less than LBM, as the coefficients of

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375 determination decreased to a lesser extent (Figures 1I, 1J, 1K, 1L). Taken together, this indicates that there
376 would be an associated-cumulative effect of maturity status in addition to the significant influence of LBM on
377 AOD_{gly} and its relationship with blood metabolic responses. However, LBM could be considered as the main
378 influence on these metabolic changes.

379

380 Several considerations should be mentioned in this study. The number of years the rowers have been
381 training prior to the study was different between younger and older participants. However, this difference should
382 not affect the conclusions of the present study since aerobic fitness evaluated from $\dot{V}O_{2max}$ normalised for
383 LBM^{1.13} was not significantly different between the maturity status groups (Table 1). Moreover, although
384 validation studies of maturity offset indicated several limitations for the prediction of biological maturation
385 (Fransen et al. 2018), the level of accuracy of this method was found to be sufficient to assign a maturational
386 classification with a standard error of 0.5 years between the ages of 8 and 16 years (Mirwald et al. 2002). If we
387 had used the Tanner staging criteria based on pubic hair and testicular volume, we could not have analysed the
388 effect of maturation on AOD_{gly} and its relationship with blood markers from the allometric method, as these
389 criteria are categorical variables while maturity offset is a continuous discrete variable. Another point of
390 consideration is that girls were not evaluated. Sex-related differences on AOD_{gly} and blood responses could be
391 expected during puberty, as body composition (whole-body lean vs fat mass) changes significantly over this
392 period between males and females (Van Praagh and Dore 2002), but this remains to be proven.

393

394 In conclusion, AOD_{gly} and blood metabolic responses in boys linearly increased with maturity status
395 and lean body mass. Lactate accumulation and pH reduction resulting from high-intensity exercise were found to
396 be highly associated with the amount of energy released from glycolysis. Changes in lean body mass during
397 growth accounted much more than maturity status for glycolysis-derived accumulated oxygen deficit and its
398 relationship with lactate accumulation and pH reduction. The results challenge previous reports of maturation-
399 related differences in glycolytic energy turnover and suggest that changes in lean body mass are a more powerful
400 influence than maturity status on glycolytic metabolism during growth.

401

402 In the occupational, exercise or sporting context, the results of the present study may prove relevant
403 since it has often been considered that anaerobic glycolysis is immature in pre-pubertal children (Eriksson et al.
404 1971) and it is, therefore, useless to propose physical activities soliciting the glycolytic process to improve

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405 anaerobic capacity before puberty (Ratel and Martin 2012). This work could lead physical educators, teachers,
406 students and coaches who have to train young people to reconsider these conclusions, as dimensional changes
407 could mainly account for glycolytic activity during growth. However, this work needs to be pursued in girls,
408 since body composition (whole-body lean vs fat mass) changes significantly during growth between boys and
409 girls (Tanner et al. 1981), and lean body mass could act differently on glycolytic metabolism between both
410 sexes.

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518 **Table 1:** Physical characteristics and aerobic fitness of the different maturity groups (n = 36) (mean \pm SD).

	Pre-PHV (n = 8)	Mid-PHV (n = 11)	Post-PHV (n = 17)
Chronological age (years)	12.4 \pm 0.9	13.7 \pm 0.7 **	16.2 \pm 0.9 *** \$\$\$
MO (years)	-1.7 \pm 0.6	0.0 \pm 0.5 ***	2.2 \pm 0.6 *** \$\$\$
Standing height (m)	1.55 \pm 0.09	1.70 \pm 0.05 **	1.80 \pm 0.07 *** \$\$\$
BM (kg)	43.6 \pm 6.2	60.7 \pm 3.3 ***	70.5 \pm 6.8 *** \$\$\$
BMI (kg·m ⁻²)	18.0 \pm 1.3	21.0 \pm 1.1 ***	21.7 \pm 1.6 ***
BF (%)	15.1 \pm 4.0	10.6 \pm 5.3	8.6 \pm 3.1 ***
LBM (kg)	37.0 \pm 5.0	54.4 \pm 4.9 ***	64.4 \pm 6.0 *** \$\$\$
$\dot{V}O_{2max}$ (L·min ⁻¹)	2.36 \pm 0.43	3.47 \pm 0.55 ***	4.54 \pm 0.28 *** \$\$\$
$\dot{V}O_{2max}$ (mL·min ⁻¹ ·LBM ^{-1.13})	40 \pm 5	38 \pm 5	41 \pm 4
P $\dot{V}O_{2max}$ (W)	126 \pm 24	204 \pm 40 **	272 \pm 25 *** \$\$\$
HR _{max} (bpm)	203 \pm 9	206 \pm 7	200 \pm 6

519 **: p < 0.01; ***: p < 0.001 from Pre ; \$\$\$: p < 0.001 from Mid.

520 MO: maturity offset; BM: body mass; BMI: body mass index; BF: body fat percentage; LBM: lean body mass;

521 $\dot{V}O_{2max}$ (mL·min⁻¹·kg^{-1.13} LBM): $\dot{V}O_{2max}$ normalized for LBM^{1.13}, allometric exponent was calculated from the522 following equation: $\log(\dot{V}O_{2max}) = b_I \cdot \log(LBM) + \log(a_I)$ (see section *Allometric modelling procedures* for523 further explanations); P $\dot{V}O_{2max}$ power corresponding to $\dot{V}O_{2max}$; HR_{max}: maximal heart rate.

524 **Table 2:** Mechanical and physiological parameters obtained from the 60-s all-out test by maturity group (n = 36)
 525 (mean ± SD).

526

	Pre-PHV (n = 8)	Mid-PHV (n = 11)	Post-PHV (n = 17)
MPO (W)	198 ± 37	343 ± 60 ***	515 ± 45 *** \$\$\$
AOD _{tot} (L O ₂ Eq.)	2.08 ± 0.45	3.39 ± 0.60 ***	5.23 ± 0.68 *** \$\$\$
AOD _{gly} (L O ₂ Eq.)	1.29 ± 0.33	2.10 ± 0.49 ***	3.39 ± 0.73 *** \$\$\$
OE _{phos+ox} (L)	0.79 ± 0.18	1.29 ± 0.44 **	1.84 ± 0.38 *** \$\$\$
AOD _{gly} (% AOD _{tot})	61.7 ± 6.1	61.9 ± 11.4	64.4 ± 7.5
OE _{phos+ox} (% AOD _{tot})	38.3 ± 6.1	38.1 ± 11.4	35.6 ± 7.5
[La] _{max} (mmol·L ⁻¹)	10.2 ± 1.5	12.3 ± 1.1 **	15.9 ± 1.8 *** \$\$\$
pH _{min} (-)	7.24 ± 0.03	7.21 ± 0.03 **	7.13 ± 0.04 *** \$\$\$
[HCO ₃ ⁻] _{min} (mmol·L ⁻¹)	13.5 ± 1.3	11.3 ± 1.3 **	8.7 ± 1.3 *** \$\$\$
[BE] _{min} (mmol·L ⁻¹)	-13.3 ± 2.0	-16.6 ± 1.7 **	-20.3 ± 1.9 *** \$\$\$

527 **: p < 0.01; ***: p < 0.001 from Pre ; \$\$\$: p < 0.001 from Mid.

528 MPO: mean power output calculated over the entire test; AOD_{tot}: total accumulated oxygen deficit; AOD_{gly}:
 529 glycolysis-derived accumulated oxygen deficit; OE_{phos+ox}: phosphagen- and blood O₂ stores-derived oxygen
 530 equivalent; [La]_{max}: maximal blood lactate concentration; pH_{min}: minimal blood pH; [HCO₃⁻]_{min}: minimal blood
 531 bicarbonate concentration; [BE]_{min}: minimal blood base excess concentration.

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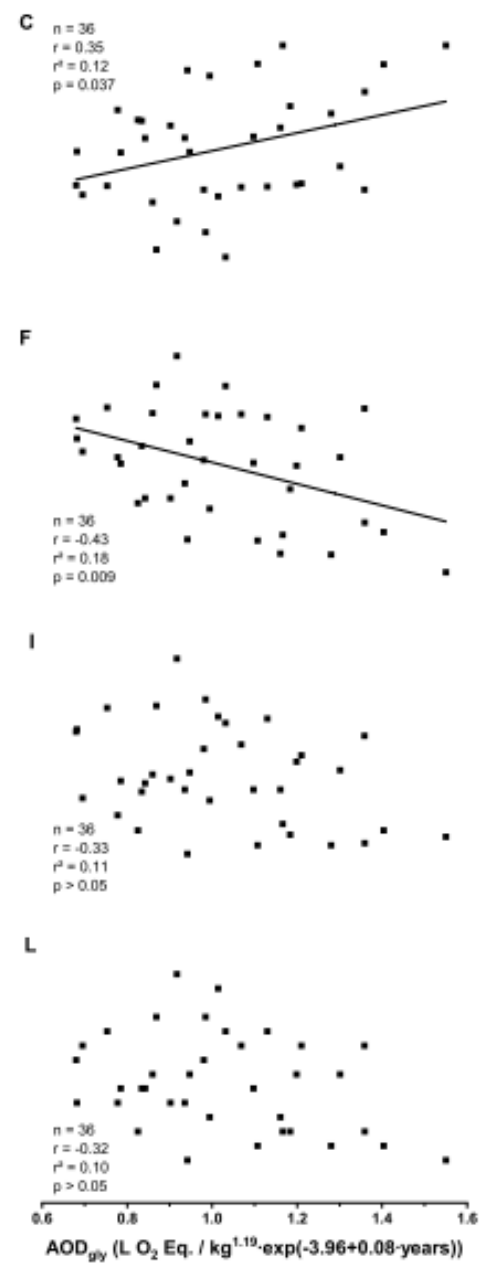
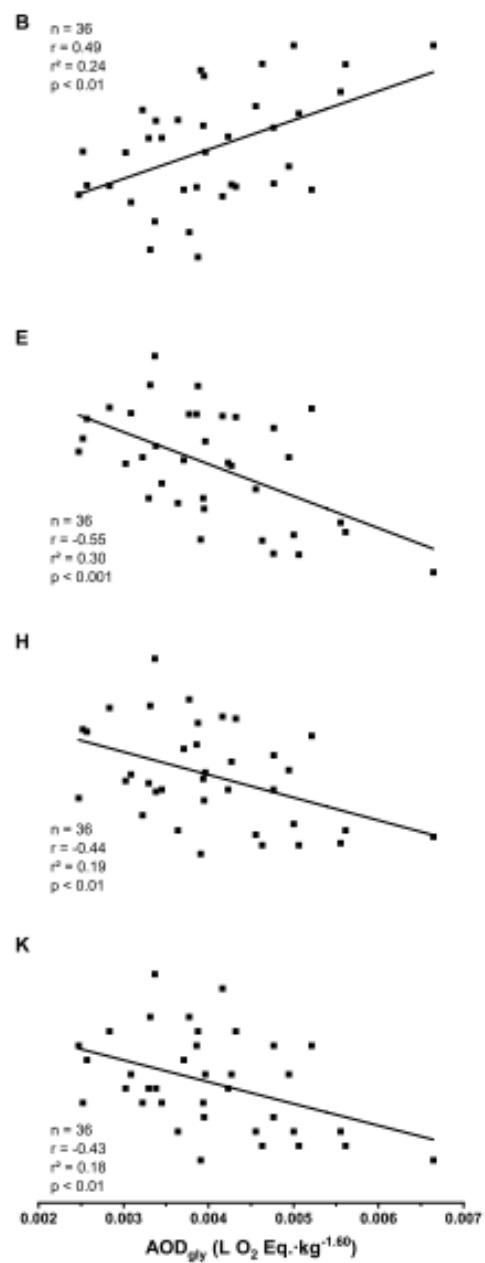
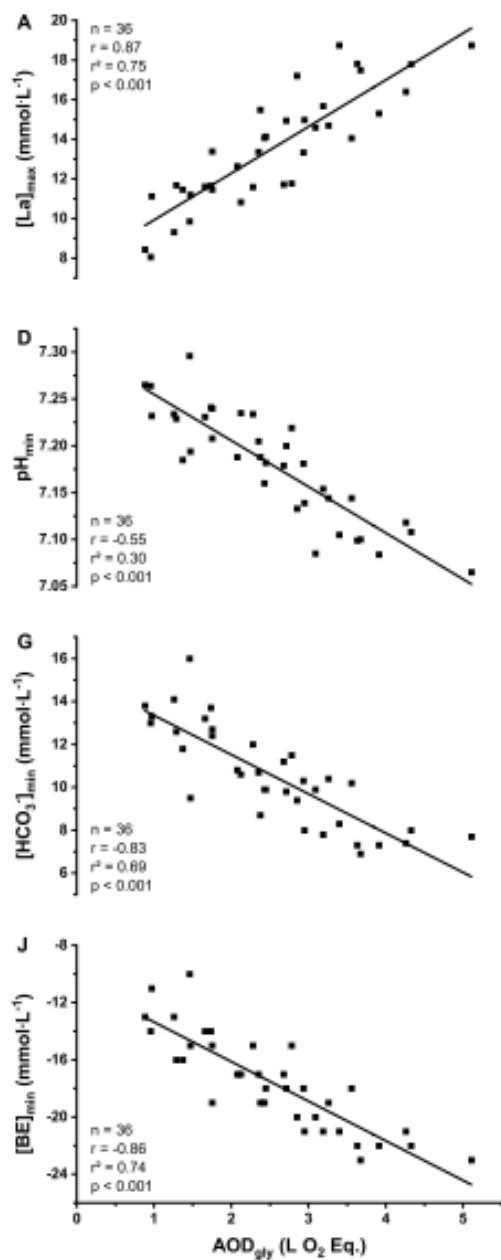
532 **Table 3:** Relationships between metabolic responses, maturity offset and lean body mass (n = 36).

	LBM (kg)	AOD _{gly} (L O ₂ Eq.)	[La] _{max} (mmol·L ⁻¹)	pH _{min} (-)	[HCO ₃ ⁻] _{min} (mmol·L ⁻¹)	[BE] _{min} (mmol·L ⁻¹)
MO (years)	r = 0.91 p < 0.001	r = 0.84 p < 0.001	r = 0.85 p < 0.001	r = -0.78 p < 0.001	r = -0.78 p < 0.001	r = -0.82 p < 0.001
LBM (kg)	-	r = 0.78 p < 0.001	r = 0.78 p < 0.001	r = -0.68 p < 0.001	r = -0.77 p < 0.001	r = -0.79 p < 0.001

533 MO: maturity offset; LBM: lean body mass; AOD_{gly}: glycolysis-derived accumulated oxygen deficit; [La]_{max}:

534 maximal blood lactate concentration; pH_{min}: minimal blood pH; [HCO₃⁻]_{min}: minimal blood bicarbonate

535 concentration; [BE]_{min}: minimal blood base excess concentration.



537 **FIGURE LEGEND**

538 **Figure 1:** Correlations between blood markers and glycolysis-derived accumulated oxygen deficit (AOD_{gly})
539 expressed in absolute value (Panels A to D), scaled for $LBM^{1.60}$ (Panels E to H) and scaled for $LBM + MO$ (Panels
540 I to L); $n = 36$.
541 $[La]_{max}$: maximal blood lactate concentration; pH_{min} : minimal blood pH; $[HCO_3^-]_{min}$: minimal blood bicarbonate
542 concentration; $[BE]_{min}$: minimal blood base excess concentration; LBM: lean body mass; MO: maturity offset.