



Prifysgol  
Abertawe  
Swansea  
University

**Investigating the effect of sex, maturity, training  
status, and physical activity on performance and  
health-related parameters in children, adolescents,  
and adults**

**Adam William Harry Runacres**  
**MSc BSc (Hons)**

Submitted to Swansea University in fulfilment of the requirements for  
the Degree of Doctor of Philosophy

Swansea University  
School of Sport and Exercise Science

2021

## **Abstract**

In 2018, 48% of young people in Wales engaged in sport  $\geq 3$  times a week. However, questions remain regarding the influence of sex and maturation on aerobic and anaerobic trainability. Indeed, many earlier studies failed to appropriately account for physical activity (PA), confounding the interpretation of training *per se*. Moreover, there is a paucity of literature examining the long-term effects of training.

**Chapter 4** revealed that, irrespective of maturity, trained youth had a higher maximal oxygen uptake ( $\dot{V}O_{2\max}$ ) than their untrained counterparts but, importantly, the magnitude of training-related difference was higher in girls than boys. Given the well-established sex-differences in the decline of PA levels with age, **Chapter 5** explored the role of PA on  $\dot{V}O_{2\max}$  using compositional analyses. This demonstrated that, for the same change in PA, girls had a greater predicted change in absolute, and scaled,  $\dot{V}O_{2\max}$ . As the trainability, and kinetic determinants, of sprint performance have received little attention compared to aerobic fitness in youth, this was explored in **Chapters 6 and 7**. In **Chapter 6**, training was associated with a greater peak power and force, depending on maturity, with only post-pubertal participants demonstrating significant increases in performance. Using a repeated sprint protocol, mechanical efficiency was found to be more important than absolute force production for performance in **Chapter 7**, highlighting key training targets. Finally, using a narrative review and meta-analytical approach, **Chapter 8** found significant inter-sport differences in all-cause, cardiovascular disease, and cancer mortality in former elite athletes, suggesting that sport type influences the long-term effects of training.

Overall, this thesis highlights the distinct determinants of aerobic and anaerobic performance, with sex and maturity exerting different, and independent, effects. Moreover, the paucity of data available in girls was highlighted, with conclusions regarding the long-term effects of training in females largely precluded.

## Declarations & Statements

### DECLARATION

This work has not previously been accepted in substance for any degree and is not being concurrently submitted in candidature for any degree.

Signed .....  ..... (candidate)

Date .....08/09/2021.....

### STATEMENT 1

This thesis is the result of my own investigations, except where otherwise stated. Other sources are acknowledged by footnotes giving explicit references. A bibliography is appended.

Signed .....  ..... (candidate)

Date .....08/09/2021.....

### STATEMENT 2

I hereby give consent for my thesis, if accepted, to be available for photocopying and for inter-library loan, and for the title and summary to be made available to outside organisations.

Signed .....  ..... (candidate)

Date .....08/09/2021.....

### STATEMENT 3

The University's ethical procedures have been followed and, where appropriate, that ethical approval has been granted.

Signed.....  .....(candidate)

Date.....08/09/2021.....

## Table of Contents

Abstract.....	i
Declarations & Statements .....	ii
Acknowledgements.....	viii
List of Tables .....	ix
List of Figures.....	x
Abbreviations .....	xi
Scientific Outputs and Public Engagement .....	xv
<b>Chapter 1 - Introduction .....</b>	<b>17</b>
1.1 Experimental Study Aims .....	24
<b>Chapter 2 - Literature Review .....</b>	<b>26</b>
<b>2.1 Influence of training on aerobic fitness in youth .....</b>	<b>26</b>
<i>2.1.1 Influence of maturity on the responses to training stimuli .....</i>	<i>28</i>
<b>2.2 Potential effect of Physical Activity on <math>\dot{V}O_{2\max}</math>.....</b>	<b>33</b>
<b>2.3 Influence of sex on the trainability of <math>\dot{V}O_{2\max}</math>.....</b>	<b>36</b>
<b>2.4 Mechanisms underpinning training-, sex- and maturity-related differences in <math>\dot{V}O_{2\max}</math> .....</b>	<b>39</b>
<i>2.4.1 Haemodynamic mechanisms.....</i>	<i>41</i>
<i>2.4.2 Muscle Deoxygenation kinetics .....</i>	<i>44</i>
<b>2.5 Influence of sex and maturity on anaerobic responses to training in youth .....</b>	<b>46</b>
<i>2.5.1 Over-ground sprint running .....</i>	<i>50</i>
<i>2.5.2 Repeated Sprint Performance .....</i>	<i>53</i>
<b>2.6 Long-term consequences of training.....</b>	<b>55</b>
<i>2.6.1 Adult Literature.....</i>	<i>55</i>
<b>2.7 Overall Conclusions .....</b>	<b>58</b>
<b>Chapter 3 – General Methodology .....</b>	<b>60</b>

<b>Chapter 4 (Study 1) – Effects of sex and training status on the cardiopulmonary responses and muscle deoxygenation kinetics during incremental exercise in children and adolescents.....</b>	<b>91</b>
4.1 Introduction .....	91
4.2 Methods .....	93
4.2.1 Experimental Procedures .....	94
4.2.2 Data Analysis .....	95
4.2.3 Statistical Analyses .....	97
4.3 Results .....	97
4.3.1 Influence of training status .....	97
4.3.2 Influence of sex.....	98
4.3.3 Interaction effects .....	98
4.4 Discussion .....	102
<b>Chapter 5 (Study 2) - The combined associations of physical activity, sedentary time and sleep on <math>\dot{V}O_{2max}</math> in trained and untrained children and adolescents: A novel five-part compositional analysis approach .....</b>	<b>109</b>
5.1 Introduction .....	109
5.2 Methods .....	111
5.2.1 Experimental Procedures .....	111
5.2.2 Data Analyses .....	112
5.2.3 Statistical Analyses .....	114
5.3 Results .....	114
5.4 Discussion .....	125
<b>Chapter 6 (Study 3) – The effect of sex, maturity, and training status on maximal sprint performance kinetics .....</b>	<b>131</b>
6.1 Introduction .....	131
6.2 Methods .....	133
6.2.1 Experimental Procedures .....	133

6.2.2 Biomechanical Modelling.....	134
6.2.3 Statistical Analyses .....	135
6.3 Results .....	136
6.3.1 Influence of training status .....	136
6.3.2 Influence of Sex .....	136
6.3.3 Influence of Maturity .....	137
6.3.4 Interaction of sex, maturity, and training status.....	137
6.4 Discussion .....	142
<b>Chapter 7 (Study 4) – Understanding the kinetics of repeated sprint ability using radar technology in national level adolescent hockey players .....</b>	<b>147</b>
7.1 Introduction .....	147
7.2 Methods .....	149
7.2.1 Experimental Procedures .....	149
7.2.2 Statistics .....	150
7.3 Results .....	151
7.3.1 Biomechanical determinants of repeated sprints .....	151
7.4 Discussion .....	155
<b>Chapter 8 (Study 5) – Health consequences of an elite sporting career – long-term detriment or long-term gain? A meta-analysis of 165,000 former athletes .....</b>	<b>160</b>
8.1 Introduction .....	160
8.2 Methods .....	161
8.2.1 Data sources, literature search and inclusion criteria .....	161
8.2.2 Data Extraction .....	163
8.2.3 Statistics .....	164
8.3 Results .....	165
8.3.1 Meta-Regression and Sensitivity Analyses .....	167
8.4 Discussion .....	174
8.4.1 All-Cause Mortality .....	174

8.4.2 Sport-specific Mortality .....	175
8.4.3 Cardiovascular Disease Mortality .....	177
8.4.4 Cancer Mortality .....	179
8.4.5 Limitations and Conclusions .....	180
<b>Chapter 9 – Synthesis .....</b>	<b>182</b>
9.1 Sex and maturity effects on trainability in youth .....	182
9.2 Experimental Study Strengths .....	189
9.3 Experimental Study Weaknesses.....	190
9.4 Future Research Directions .....	192
9.5 Overall Conclusions .....	194
<b>Chapter 10 – References.....</b>	<b>196</b>
<b>Chapter 11 – Appendices.....</b>	<b>227</b>
Chapters 4 and 5 .....	227
Parental Information Sheet .....	227
11.1.2 Parental Consent Form.....	229
11.1.3 Pre-Screening Medical Questionnaire .....	230
11.1.4 Athlete Participant Information Sheet .....	232
11.1.5 Control Participants Information Sheet .....	234
11.1.6 Participant Assent Form.....	236
Chapter 5 – Additional Material.....	237
11.2.1 Smallest worthwhile change table .....	237
11.2.2 Ternary Plots – SED, LPA, MPA – Scaled $\dot{V}O_{2max}$ .....	238
11.2.3 Ternary Plots – SED, LPA, VPA – Scaled $\dot{V}O_{2max}$ .....	239
11.2.4 Ternary Plots – SED, LPA, MPA – Absolute $\dot{V}O_{2max}$ .....	240
11.2.5 Ternary Plots – SED, LPA, VPA – Absolute $\dot{V}O_{2max}$ .....	241
11.3 Chapter 6 .....	242
11.3.1 Parental Information Sheet .....	242

11.3.2 Parent/Guardian Consent Form .....	244
11.3.4 Participant Information Sheet .....	245
11.3.5 Participant Assent Form.....	247
11.4 Chapter 7 .....	248
11.4.1 Parental Information Sheet .....	248
11.4.2 Parent/Guardian Consent Form .....	250
11.4.3 Participant Information Sheet .....	251
11.4.4 Participant Assent Form.....	253
11.5 Chapter 8 .....	254
11.5.1 Male cardiovascular disease (CVD) mortality forest plot .....	254
11.5.2 Male cancer mortality forest plot.....	255
11.5.3 Funnel Plots .....	256
11.5.3.1 All-Cause Mortality Funnel Plot.....	256
11.5.2.2 Cardiovascular Disease Mortality Funnel Plot .....	257
11.5.2.3 Cancer Mortality Funnel Plot.....	258



## **Acknowledgements**

Firstly, I would like to give a huge thank you to my two academic supervisor's Dr Melitta McNarry and Dr Kelly Mackintosh for your help and guidance over the last four years as I completed my MSc and now PhD. You have both allowed me to pursue my research areas of interest and were always there to put me back on the right track when I inevitably went off on tangents!

Secondly, I would like to thank my company supervisors at Sport Wales Tim Evans and Dr Rachel Hughes. Without your help facilitating the research and linking me with contacts across the organisation the process would have been a lot harder. Moreover, this work is part-funded by the European Social Fund (ESF) through the European Union's Convergence programme administered by the Welsh Government, and without the assistance of this scholarship I would not have been able to complete this research program.

Next, I would like to thank my mum, without your assurance and support I would have found carrying on through certain hard times of data collection much harder, and my brother Daniel who is always there for a laugh (at his expense) when I need it. I would also like to thank my girlfriend Zoe, living and working with someone that understands the demands of a PhD made this process a whole lot easier. Finally I would like to thank all of the MSc and PhD students in the A-STEM office I have had the pleasure of meeting over the last four years. Your support, willingness to talk through problems, and on the odd occasion provide much needed procrastination made those early mornings and late nights a lot more tolerable.

A final thanks to all of the participants and their parents who facilitated this research process without whom none of this research would not have been possible

## List of Tables

<b>Table 3.1</b> – Distribution of participants included within <b>Chapters 4, 5, 6</b> and <b>7</b> .....	61
<b>Table 4.1</b> Descriptive Characteristics.....	98
<b>Table 4.2</b> Pulmonary gas exchange and hemodynamic responses to incremental ramp exercise according to training status and sex.....	99
<b>Table 4.3</b> Muscle deoxygenation variables from the incremental ramp exercise according to training status and sex.....	100
<b>Table 5.1</b> Participant descriptives.....	113
<b>Table 5.2</b> Geometric means for the whole sample.....	114
<b>Table 5.3</b> Pair-wise log ratio variation matrix in the full sample.....	115
<b>Table 5.4</b> Model isometric log ratio parameters for $\dot{V}O_{2max}$ and allometrically scaled $\dot{V}O_{2max}$ .....	115
<b>Table 5.5</b> Change matrices of reallocating 10 minutes from the behaviour in columns to the behaviour in the rows on $\dot{V}O_{2max}$ ( $l \cdot min^{-1}$ ) and scaled $\dot{V}O_{2max}$ ( $ml \cdot kg^{-b} \cdot min^{-1}$ ) in trained children and adolescents, presented as percentage change.....	116
<b>Table 5.6</b> Change matrices of reallocating 10 minutes from the behaviour in columns to the behaviour in the rows on $\dot{V}O_{2max}$ ( $l \cdot min^{-1}$ ) and scaled $\dot{V}O_{2max}$ ( $ml \cdot kg^{-b} \cdot min^{-1}$ ) in untrained children and adolescents, presented as percentage change.....	119
<b>Table 6.1</b> Anthropometric characteristics for trained and untrained participants.....	121
<b>Table 6.2</b> 30 m sprint performance variables in boys.....	136
<b>Table 6.3</b> 30 m sprint performance variables in girls.....	137
<b>Table 6.4</b> Predictor variables for 30 m time for each maturity group.....	138
<b>Table 7.1</b> Participant Descriptives.....	139
<b>Table 7.2</b> Sprint variables form each of the 5 x 15 m sprint repetitions across all five sprints.....	149
<b>Table 7.3</b> Biomechanical determinants of 15 m sprint time for each repetition.....	151
<b>Table 8.1</b> Key information about the studies included within the meta-analysis.....	152
<b>Table 8.2</b> Sport-specific all-cause, CVD and cancer pooled-SMR's.....	166

## List of Figures

<b>Figure 3.1</b> Schematic diagram for the determination of $\dot{V}O_{2max}$ from an incremental ramp test and the supramaximal validation bout.....	65
<b>Figure 3.2</b> The relationship between a) $\dot{V}O_{2max}$ and body mass and b) the relationship between allometrically scaled $\dot{V}O_{2max}$ and body mass.....	67
<b>Figure 3.3</b> The calculation of a) the V-Slope method used to determine the gas exchange threshold (GET) during the incremental ramp test and b) The calculation of the mean response time (MRT) and Gain.....	68
<b>Figure 3.4</b> Representative sigmoidal [HHb] responses when expressed against a) work rate (W), b) relative work rate (% Max), c) $\dot{V}O_2$ ( $l \cdot min^{-1}$ ) and d) relative $\dot{V}O_2$ (% $\dot{V}O_{2max}$ ).....	76
<b>Figure 3.5</b> A graphical representation of the $v_h(t)$ curve modelled for each sprint repetition from which power and force variables were estimated.....	82
<b>Figure 5.1</b> Ternary heat plots of all physical activity behaviours with expected $\dot{V}O_{2max}$ values for all sub-groups with a) trained athletes; b) untrained controls; c) all boys; d) all girls; e) pre-PHV children; f) circa-PHV adolescents; and g) post-PHV adolescents.....	117
<b>Figure 5.2</b> Ternary plots of all physical activity behaviours with expected scaled $\dot{V}O_{2max}$ values for all sub-groups with a) trained athletes; b) untrained controls; c) all boys; d) all girls; e) pre-PHV children; f) circa-PHV adolescents; and g) post-PHV adolescents.....	118
<b>Figure 7.1</b> Post-processed representative traces from the five sprint repetitions for a typical participant showing a) velocity-time profile, b) power-velocity profile and c) the force velocity profile.....	150
<b>Figure 8.1</b> Schematic flow diagram of the systematic review and meta-analysis process.....	161
<b>Figure 8.2</b> Male all-cause mortality forest plot.....	164
<b>Figure 8.3</b> Female all-cause mortality forest plot.....	165

## Abbreviations

a	Amplitude
A(OD)	Light Omitting Diode
ATP	Adenosine Triphosphate
$a-\bar{v}O_{2diff}$	Arteriovenous difference
beats·min <sup>-1</sup>	Beats per Minute
BF	Body Fat
BMI	Body Mass Index
BSA	Body Surface Area
c -	Constant
Ca	Oxygen Concentration in the arteries
Cv	Oxygen Concentration in the veins
CIET	Continuous Intensity Endurance Training
CHD	Coronary Heart Disease
CI	Confidence Interval
CON	Control
CP	Creatine Phosphate
CTI	Contractility Index
CV	Coefficient of Variation
CVD	Cardiovascular Disease
Deoxy[HHb+Mb]	Concentration of deoxygenated haemoglobin
DEXA	Dual x-ray absorptiometry
DPF	Differential Pathway Factor
ECG	Electrocardiogram
EE	Energy Expenditure
END	Endurance
EMG	Electromyography
FFA	Free fatty acids
FFM	Fat Free Mass
F-v-P	Force velocity Power profiling
G	Amount of Light lost due to scattering

GET	Gas Exchange Threshold
GH	Growth Hormone
GP	Grulich-Pyle
HHb	Haemoglobin
HIIT	High Intensity Interval Training
HR	Heart Rate
HR <sub>max</sub>	Maximum heart rate
I	Amount of Emergent Light
I <sub>0</sub>	Light Source
ICC	Intra-class correlation coefficient
IGF-1	Insulin like Growth Factor 1
k	Mathematical Constant
Kg	Kilograms
Kg·m <sup>2</sup>	Kilograms per meters squared
L	Radiance of Light
LLM	Lean Muscle Mass
LLVM	Lower Limb Muscle Volume
LL	Leg Length
L·min <sup>-1</sup>	Litres per minute
LPA	Light Intensity Physical Activity
LTAD	Long-term Athlete Development
LVM	Left Ventricular Mass
m	Meters
Mb	Myoglobin
MET	Metabolic Equivalent
MIIT	Moderate Intensity Interval Training
ml·min <sup>-1</sup>	Millilitres per minute
ml·kg <sup>-1</sup> ·min <sup>-1</sup>	Millilitres Of oxygen per Kilogram per Minute
ml·kg <sup>-b</sup> ·min <sup>-1</sup>	Millilitres of Oxygen per Kilogram (to the logarithmic exponent) per Minute
ml·m <sup>2</sup>	Millilitres per meter squared
MPA	Moderate intensity Physical Activity
MRI	Magnetic Resonance Imaging

MRT	Mean Response Time
MVPA	Moderate-to-Vigorous Intensity Physical Activity
NGB	National Governing Body
NHS	National Health Service
NIRS	Near Infra-red Spectroscopy
O <sub>2</sub>	Oxygen
OBLA	Onset of Blood Lactate Accumulation
OR	Odds Ratio
oxy[HHb+Mb]	Concentration of Oxygenated Haemoglobin
PCr	Phosphocreatine
PHV	Peak Height Velocity
P <sub>mv</sub> $\dot{V}O_2$	Partial Pressure of Oxygen in the Microvasculature
P <sub>mito</sub> O <sub>2</sub>	Partial Pressure of Oxygen in the Mitochondria
POW	Power
PP	Pulse Pressure
PWT	Posterior Wall Thickness
$\dot{Q}$	Cardiac Output
$\dot{Q}_{max}$	Maximum Cardiac Output
RER	Respiratory Exchange Ratio
RPM	Revolutions per Minute
RR	Risk Ratio
s	Seconds
SBJ	Standing Broad Jump
SEE	Standard Error of the Estimate
SEM	Standard Error of the Mean
SJ	Squat Jump
SMR	Standardised Mortality Ratio
SPMR	Standardised Proportional Mortality Ratio
SED	Sedentary Time
SV	Stroke Volume
SV <sub>i</sub>	Stroke Volume Index
SV <sub>max</sub>	Maximum Stroke Volume
SWT	Septal Wall Thickness

TFIT	Thoracic Flow Inversion Time
total[HHb+Mb]	Total of Oxygenated and Deoxygenated
TW2	Tanner-Whitehouse Method 2
TW3	Tanner Whitehouse Method 3
UK	United Kingdom
USA	United States of America
$\dot{V}O_2$	Volume of oxygen
$VO_E$	Pulmonary Ventilation
$v_H(t)$	Velocity-time Curve
$v_{hmax}$	Maximal Horizontal Acceleration
$v_{peak}$	Peak Velocity
VJ	Vertical Jump
VPA	Vigorous Intensity Physical Activity
W	Watts
WADA	World Ant-Doping Association
WnT	Wingate
Z	Bioimpedance signal
%	Percentage
$\varphi$	Influence Rate
$\delta$	Optical Penetration Depth
$\Omega$	Solid Angle
[cyto <sub>ox</sub> ]	Concentration of Cytochrome Oxidase
$\epsilon$	Specific Extinction Coefficient
[c]	Concentration of Chromospheres
$\mu_s$	Scattering Coefficient
$\mu_a$	Absorption Coefficient
$\Delta$	Change
$\tau$	Acceleration Time Constant

## **Scientific Outputs and Public Engagement**

### **Publications:**

Runacres, A., Mackintosh, K.A. & McNarry, M.A. (2020) Health consequences of an elite sporting career – long-term detriment or long-term gain? A meta-analysis of 165,000 former athletes. *Sports Medicine*.

### **Conferences:**

Poster Presentation at 1st Pan Wales Sports and Exercise Conference. *The effect of Constant Intensity Endurance Training (CIET) and High Intensity Interval Training (HIIT) on aerobic and anaerobic parameters in youth*. Swansea UK, April 2017.

Oral Presentation at the 2nd Pan Wales Sports and Exercise Conference. *The Influence of Training Type and Maturational Status on Aerobic and Anaerobic Adaptations over a Three-Month Training Cycle*. Bangor UK, May 2018.

Oral Presentation at the 3rd Pan Wales Sports and Exercise Conference. *The influence of changing physical activity (PA) patterns on peak  $\dot{V}O_2$  in children and adolescents: A Compositional Analysis Approach*. Cardiff UK, May 2019.

### **Public Engagement:**

Research Engagement and Feedback Sessions with Participating Schools. Swansea UK.

Sport Wales Presentation. *Examining the long-term benefits of intensive exercise*. Sport Wales, Cardiff, UK



# **Chapter 1**

## **Introduction**

## **Chapter 1 - Introduction**

Exercise is paramount for the current health and well-being, irrespective of age, though arguably more important during childhood and adolescence given that exercise habits in children are strongly associated with exercise levels in adulthood (Armstrong, 2007; Armstrong & Welsman, 2020c; Baquet, Van Praagh, & Berthoin, 2003; Matos & Winsley, 2007). Exercise is associated with numerous health-related benefits, including, but not limited to, improved physical (Armstrong & Welsman, 2020c; McNarry et al., 2014a; McNarry, Mackintosh, & Stoedefalke, 2014b) and mental health (Eddolls, McNarry, Stratton, Winn, & Mackintosh, 2017; Sabato, Walch, & Caine, 2016), enhanced social well-being (Mountjoy, 2008; Pene & Toutou, 2009), and a reduced risk of cardiovascular disease (CVD; Coombes, Law, Lancashire, & Fasset, 2015; Kaminsky et al., 2019) and all-cause mortality (Garatachea et al., 2014; Lemez & Baker, 2015). Creating a more active nation has been at the forefront of governmental policies for decades (UK Government, 2015), with the latest available data indicating that almost half of children and adolescents under 17 years of age in England and Wales participate in extra-curricular sport at least three times a week (Sport England, 2019; Sport Wales, 2018). Driven by this increase in participation, young athletes are increasingly being enrolled in long-term athlete development (LTAD) to ensure the continuation of elite athlete sporting success (Till, Emmonds, & Jones, 2019). Consequently, young athletes are now training earlier, longer, and at a greater intensity than ever before (Till et al., 2019). Despite this, fundamental questions remain regarding the influence of training during youth, the factors that influence it and its long-term implications on health.

The effect of different training methodologies on various components of fitness has received substantial attention (Armstrong & Welsman, 2020c; Baquet et al., 2003; Cao, Quan, & Zhuang, 2019; Costigan, Eather, Plotnikoff, Taaffe, & Lubans, 2015; McNarry & Jones, 2014) but, despite decades of research, there remains little consensus as to whether endurance training or high-intensity interval training (HIIT) is more effective at obtaining favourable training-related adaptations (Cao et al., 2019). Such discrepancies can be largely attributed to inter-study methodological inconsistencies, including a failure to appropriately account for the pubertal status of the participants within training studies (Kobayashi et al., 1978; Stojmenović et al.,

2018) or failing to include no-exercise comparator groups to enable the concomitant effects of growth and maturation to be accounted for (Faude, Schnittker, Schulte-Zurhasen, Muller, & Meyer, 2013; Sperlich et al., 2011). Indeed, accounting for puberty, the process of growth and sexual maturation in the transition from childhood into adulthood (Rogol, Roemmich, & Clark, 2002), is imperative, as pubertal status has been purported to be strongly linked to improvements in athletic performance for nearly 40 years.

In a seminal paper, Katch (1983) theorised that pubertal adolescents experience a period of accelerated adaptation to training, mediated by increases in circulating androgenic hormones, with negligible adaptations to training observed prior to this point, termed the 'maturational threshold'. Whilst a strong theoretical argument can be made for the presence of the maturational threshold, recent empirical evidence refutes the hypothesis, with pre-pubertal children demonstrating similar trainability to pubertal and post-pubertal adolescents (Armstrong, 2015; Cahill et al., 2020; McNarry et al., 2014b; McNarry, Welsman, & Jones, 2011b; Moran, Sandercock, Rumpf, & Parry, 2016). However, there are a few methodological limitations which may contribute to our lack of understanding regarding the optimal training methodology, and how this interacts with sex and maturation. More specifically, discrepancies between training and intervention exercise modalities (Stoedefalke, Armstrong, Kirby, & Welsman, 2000) likely mask meaningful performance differences. Additionally early training intervention studies in girls were of insufficient intensity (Welsman, Armstrong, Chedzoy, & Withers, 1996; Welsman, Armstrong, & Withers, 1997; Williams, Armstrong, & Powell, 2000), with later research suggesting children and adolescents require a vigorous exercise stimulus to elicit a significant response in peak  $\dot{V}O_2$  (Baquet et al., 2003; Cao et al., 2019; Carazo-Vargas & Moncada-Jiménez, 2015; Foster et al., 2015; Sperlich et al., 2011). Furthermore, a major limitation with much of the training literature, which is often overlooked, is the failure to consider habitual physical activity (PA) levels. Indeed, the most common definition of a control group within training studies is that the participants were not engaged in a formal exercise program (Mahon & Vaccaro, 1989; Mandigout, Lecoq, Courteix, Guenon, & Obert, 2000; Rowland & Boyajian, 1995), but this does not preclude the participants from

being highly physically active, thus potentially confounding the interpretation of the effect of training per se.

The effect of habitual PA and sedentary time (SED) on peak  $\dot{V}O_2$  levels in children and adolescents is equivocal (Armstrong, 2013; Armstrong, Tomkinson, & Ekelund, 2011; Dencker & Andersen, 2011; Dencker et al., 2006; Ekelund et al., 2001; Fenster, Freedson, Washburn, & Ellison, 1989), but paradoxically they are the two single most researched parameters in paediatric exercise science. Whilst accelerometers have enhanced our understanding of PA and SED in youth, the vast majority of research has focused solely on moderate-to-vigorous physical activity (MVPA), which typically encompasses just 4% of the 24-hour day (Chastin, Palarea-Albaladejo, Dontje, & Skelton, 2015). Interpretation of this data is further compounded by the reliance on conventional statistics which typically assume independence between variables (Pearson, 1896), potentially leading to spurious associations with health outcomes (Chastin et al., 2015). In order to account for the constrained and co-dependent nature of PA and SED, a compositional approach may be more appropriate as it considers each movement behaviour (i.e. SED, light physical activity, MPA, VPA, and sleep) relative to the remaining composition (Carson, Tremblay, Chaput, & Chastin, 2016; Carson, Tremblay, Chaput, McGregor, & Chastin, 2019; Dumuid et al., 2018a). Therefore, compositional analysis allows for the constrained, and co-dependent, nature of movement data, enabling the exploration of the independent and interactive effects of movement behaviours on outcome variables (Chastin et al., 2015; Dumuid et al., 2018a). The relationship between PA, SED, and peak  $\dot{V}O_2$  was examined using compositional analyses in a large sample of Canadian children ( $n = 4,169$ ), revealing that whilst the overall PA composition was significantly related to peak  $\dot{V}O_2$ , the reallocation of time from one movement behaviour to another had negligible effect on predicted peak  $\dot{V}O_2$  (Carson et al., 2016). However, it is pertinent to note that peak  $\dot{V}O_2$  in the study of Carson et al. (2016) was assessed using a field-based measure which has questionable reliability (Armstrong & Welsman, 2020b). Moreover, there was a lack of training and maturity status assessment, both of which significantly influence peak  $\dot{V}O_2$  (McNarry & Jones, 2014), along with the pooling of data from boys and girls, potentially confounding the interpretation of the results.

Therefore, future research is warranted to address these limitations and to provide more generalisable, and impactful, insights.

The effect of maturity upon peak  $\dot{V}O_2$  is well established, but key questions still remain regarding the influence of sex on training responses to exercise stimuli (Armstrong & McNarry, 2016). More specifically, girls and boys differ greatly in maturation timing, tempo and duration (Rogol et al., 2002) and have increasingly different body compositions which may potentially alter the response to training stimuli during adolescence (Armstrong & McNarry, 2016; Armstrong & Welsman, 2019b, 2020a). Tentative comparisons suggest that pre-pubertal girls experience a similar degree of improvement in peak  $\dot{V}O_2$  to boys in response to a training stimuli, irrespective of modality (7.8% - 9.1%; McManus, Armstrong, & Williams, 1997; McNarry et al., 2011b). Interestingly, despite their seemingly similar trainability, girls have a lower peak  $\dot{V}O_2$  compared to their male counterparts, even when allometrically scaled for body mass (Armstrong & Welsman, 2019a; Armstrong & Welsman, 2019b; Winsley, Fulford, Roberts, Welsman, & Armstrong, 2009). This sex-related divergence in peak  $\dot{V}O_2$  increases with maturity, with sex differences in absolute peak  $\dot{V}O_2$  increasing from ~12.8% in pre-pubertal children (Winsley et al., 2009) to ~30% in post-pubertal adolescents and adults (Armstrong & Welsman, 2019b). Although there are sex differences in body composition and PA levels, especially during the mid-teenage years (Bitar, Vernet, Coudert, & Vermorel, 2000; Ekelund et al., 2001), these sex differences are unlikely to explain the sexual dimorphism of peak  $\dot{V}O_2$  in its entirety, and more insight could be gained by examining the mechanistic basis for peak  $\dot{V}O_2$  development (Armstrong & Welsman, 2019a; Armstrong & Welsman, 2020a; McNarry et al., 2015; McNarry et al., 2014b; Winsley et al., 2009).

Fick (1870b) highlighted that both oxygen delivery and extraction are important cardiovascular components for peak  $\dot{V}O_2$  development. Oxygen delivery is mediated through central mechanisms, namely stroke volume ( $SV$ , the amount of blood ejected from the heart with every beat) and cardiac output ( $\dot{Q}$ , the amount of blood ejected from the heart every minute). Evidence indicates that boys typically have a larger  $SV$  and  $\dot{Q}$  than girls, irrespective of maturity or training status, but that this sex difference is ameliorated when appropriately normalised for body size (Armstrong & Welsman, 2019a; Armstrong & Welsman, 2020a; Nottin et al., 2002; Obert et al., 2003; Vinet et

al., 2003; Winsley et al., 2009). Consequently, differences in peripheral oxygen extraction at the level of the muscle seem more influential, with evidence derived from near infra-red spectroscopy (NIRS) suggesting that differences in muscle oxygen extraction can explain up to 12% of the variance in peak  $\dot{V}O_2$  in pre-pubertal children (McNarry et al., 2015). However, the majority of research examining sex differences in oxygen delivery and extraction, have been conducted in pre-pubertal children (McNarry et al., 2015; Obert et al., 2005; Obert et al., 2003; Winsley et al., 2009) and thus whether these differences are evident in circa- and post-pubertal adolescents remains to be elucidated.

Whilst the mechanisms underpinning sex and maturity differences in peak  $\dot{V}O_2$  are starting to be elucidated, sex differences in the anaerobic performance capacity of children and adolescents has received substantially less attention (Doré et al., 2005; Van Praagh, 2000; Van Praagh & Doré, 2002). This may be due, at least in part, to a lack of ‘gold standard’ measure (Ingle & Tolfrey, 2013; Watt, Hopkins, & Snow, 2002) and researchers considering anaerobic performance a performance as opposed to a health-related outcome. Nevertheless, over-ground sprint running has become an increasingly popular performance assessment measure over the last decade and is commonly incorporated into LTAD programs and talent identification batteries (Meyers, Oliver, Hughes, Cronin, & Lloyd, 2015; Meyers, Oliver, Hughes, Lloyd, & Cronin, 2016, 2017a; Nagahara et al., 2019; Papaiakovou et al., 2009; Philippaerts et al., 2006). Nonetheless, there remains debate regarding the most appropriate sprint protocol, with suggestions that repeated-sprint protocols may be more ecologically valid and may offer more insight into the fatiguing mechanisms of anaerobic performances (Mendez-Villanueva et al., 2010; Mendez-Villanueva, Hamer, & Bishop, 2007; Mujika, Spencer, Santisteban, Gioriena, & Bishop, 2009; Philippaerts et al., 2006; Rommers et al., 2018). The evidence from both singular and repeated sprints indicates that sprint speed during adolescence is also sexually dimorphic, with girls displaying an almost linear increase in sprint speed until the age of 15 years (Nagahara et al., 2019; Papaiakovou et al., 2009), whereas boys experience an accelerated rate of adaptation in the six months surrounding peak height velocity (Philippaerts et al., 2006).

The majority of research investigating anaerobic sprint performance in youth to date has focused on the influence of internal and external stimuli on a single, summative, outcome variable in the form of maximal sprint speed (Morin, 2018; Morin, Jeannin, Chevallier, & Belli, 2006; Rossi, Slotala, Samozino, Morin, & Edouard, 2017) but this provides little insight to the development or maintenance of speed throughout a sprinting bout. Furthermore, in those studies that have explored the kinetic determinants of sprint performance, this has largely been derived using non-motorised treadmills (Rumpf et al., 2015a; Rumpf, Cronin, Oliver, & Hughes, 2013; Rumpf, Cronin, Oliver, & Hughes, 2015b) or force platforms (Nagahara et al., 2019) which have limited ecological validity. Recent developments in technology now enable for the near instantaneous ( $> 46$  Hz) quantification of velocity data from which the underlying kinetics can be modelled using macroscopic biomechanical models (Rossi et al., 2017; Samozino et al., 2016; Simperingham, Cronin, & Ross, 2016). However, these techniques have not been used in a paediatric population and thus evidence regarding sprint kinetics are sparse (Meyers, Oliver, Hughes, Lloyd, & Cronin, 2017b). A greater understanding of the kinetic determinants of single and repeated sprint performance may enhance our understanding of fatiguing mechanisms, allowing greater specificity in training interventions. Moreover, more insight into the underlying kinetics in field-based settings may increase the success of talent identification batteries.

The effects of training during childhood and adolescence on long-term health in adulthood has received limited attention, at least in part, due to the difficulty in conducting longitudinal studies and controlling for all confounding factors. Therefore, future research is needed to establish the long-term effects of exercise in children. Nevertheless, in adults, a linear dose-response relationship has traditionally been assumed between the amount of exercise performed and all-cause mortality, CVD and cancer related mortality (Blair et al., 1989; Lee, Hsieh, & Paffenbarger Jr, 1995; Paffenbarger & Lee, 1998). However, recent large-scale epidemiological studies have challenged this assumption, suggesting that the exercise-longevity relationship may be 'J' shaped, with exercise above a certain volume and/or intensity deleterious to long-term health (M. Armstrong, Green, Reeves, Beral, & Cairns, 2015a; Merghani, Malhotra, & Sharma, 2016; O'Keefe et al., 2012; Schnohr, O'Keefe, Marott, Lange, &

Jensen, 2015). Of concern, elite athletes often train at levels far exceeding those reported in epidemiological studies (Antero-Jacquemin et al., 2014; Bianco et al., 2007), raising questions regarding the long-term health implications of such training. Whilst the most recent systematic reviews and meta-analyses concluded that former elite athletes live longer and have a lower incidence of CVD and cancer mortality than the general population (Garatachea et al., 2014; Lemez & Baker, 2015; Teramoto & Bungum, 2010b), it is pertinent to note that these reviews did not account for potential between sport differences in all-cause, CVD and cancer mortality. Controlling for sport, and thus training types, is crucial given the significant variations in training methodologies used and their potentially divergent effects on health.

Therefore, this thesis sought to investigate the influence of sex, maturity, training status, and physical activity levels, using novel methods and analysis techniques, on aerobic and sprint performance parameters during childhood and adolescence.



## 1.1 Experimental Study Aims

### Study 1 (Chapter 4):

To investigate the influence of sex on aerobic fitness, and the underpinning mechanisms, in youth.

### Study 2 (Chapter 5):

To examine independent, and interactive, effects of five movement behaviours (i.e. SED, LPA, MPA, VPA, and sleep) on absolute and allometrically scaled  $\dot{V}O_{2\max}$ , accounting for sex, maturity, and training status.

### Study 3 (Chapter 6):

To determine whether the kinetics associated with maximal sprint performance differ according to sex, maturity, and training status.

### Study 4 (Chapter 7):

To investigate the mechanisms of fatigue during repeated over-ground sprints using a combination of radar technology and macroscopic biomechanical modelling in trained children and adolescents.

### Study 5 (Chapter 8):

To examine the relationship between chronic intensive exercise training and all-cause, CVD and cancer mortality in former elite athletes, according to sport type, in comparison to their non-elite counterparts.

# **Chapter 2**

## **Literature Review**

## **Chapter 2 - Literature Review**

Exercise is strongly associated with numerous short- and long-term health-related parameters (Armstrong, 2007; Armstrong & Welsman, 2020c; Väistö et al., 2019). Specifically, regular exercise has been associated with an improved aerobic fitness (Armstrong, 2007; Cao et al., 2019), mental health (Eddolls et al., 2017), and a reduced risk of cardiovascular disease (CVD), cancer, and all-cause mortality (Garatachea et al., 2014; Imboden et al., 2018; Lemez & Baker, 2015). Engaging children and adolescents in regular sports participation has therefore been a long-term target for governing authorities for many years (UK Government, 2015). Encouragingly, the latest available statistics indicate that youth sports participation within the UK is increasing, with 47% of children and adolescents under 17 years in England and Wales now participating in extra-curricular sport at least three times per week (Sport England, 2019; Sport Wales, 2018). As a consequence of this increase in participation, more children and adolescents are being enrolled into long-term athlete development (LTAD) programs to facilitate the continuation of international sporting success (Till et al., 2019). Indeed, elite junior athletes are training longer, more intensively, and more specifically, than ever before (Green, 2006; Till et al., 2019; Williams, 2016), despite fundamental questions remaining regarding the influence of sex (Armstrong & McNarry, 2016; McNarry et al., 2015; Winsley et al., 2009) and the optimal training methodology to engender favourable adaptations (Armstrong & McNarry, 2016; Katch, 1983; McNarry et al., 2014a; Rowland, 1997).

### **2.1 Influence of training on aerobic fitness in youth**

The optimal training method to elicit the most favourable performance adaptations remains to be elucidated (Cao et al., 2019; Carazo-Vargas & Moncada-Jiménez, 2015; Sperlich et al., 2011; Stoeckel et al., 2000). The most researched parameter in paediatric exercise science is aerobic fitness and, specifically, peak oxygen uptake ( $\dot{V}O_2$ ) which is defined as the highest oxygen uptake that can be achieved despite further increases in work rate (Hill & Lupton, 1923). Peak  $\dot{V}O_2$  is the term most accepted within the paediatric literature due to the absence of a plateau in  $\dot{V}O_2$  in approximately 60 - 80% of children and adolescents (Armstrong & Welsman, 2020c; Barker, Williams, Jones, & Armstrong, 2009). Peak  $\dot{V}O_2$  is strongly associated with athletic performance, particularly in endurance and team sports (Armstrong et al.,

2011; Armstrong & Welsman, 2020c; Sperlich et al., 2011; Sperlich et al., 2010), as well as with both current and long-term health. It is therefore imperative to promote peak  $\dot{V}O_2$  during childhood and adolescence (Carson et al., 2016; Hurtig-Wennlof, Ruiz, Harro, & Sjoström, 2007; Mintjens et al., 2018).

The most widely used training method to enhance peak  $\dot{V}O_2$  has been endurance exercise, characterised by periods of constant-intensity exercise (typically  $\leq 75\%$  maximal heart rate;  $HR_{max}$ ) maintained for at least 30 minutes, three times a week (Baquet et al., 2003). Early studies in young children (9 – 10 years) reported conflicting results regarding the influence of such training in children, with some (Massicotte & Macnab, 1974; McManus et al., 1997; Tolfrey, Campbell, & Batterham, 1998), but not all (Stoedefalke et al., 2000; Welsman et al., 1996; Welsman et al., 1997; Williams et al., 2000), studies reporting significant differences in peak  $\dot{V}O_2$  to be elicited by endurance training. Indeed, Welsman et al. (1997) and Williams et al. (2000) compared the effectiveness of two different aerobic interventions in young girls and boys, respectively, compared to no exercise control groups. Both studies reported minimal increases in peak  $\dot{V}O_2$  after the eight-week interventions ( $0.03 \text{ l}\cdot\text{min}^{-1}$  to  $0.13 \text{ l}\cdot\text{min}^{-1}$ ), which may be due, at least in part, to discordant intervention and testing exercise modalities used in these studies. Specificity of exercise modalities is important when conducting training interventions as different exercise modalities may mask physiologically meaningful changes in performance. Alternatively, it could also be postulated that the exercise stimuli were not intense enough to elicit a significant physiological response (Costigan et al., 2015; Massicotte & Macnab, 1974). Indeed, it was demonstrated by Massicotte and Macnab (1974) that an intensity of at least  $180 \text{ beats}\cdot\text{min}^{-1}$  was required to significantly improve peak  $\dot{V}O_2$  in young boys. Of importance, lower intensities matched for total workload did not elicit any significant improvements in peak  $\dot{V}O_2$  (Massicotte & Macnab, 1974). This is supported by a review of endurance training interventions which suggested that whilst endurance training elicits a 5 – 6% improvement in peak  $\dot{V}O_2$  over eight weeks, a greater magnitude of change (8 – 10%) was associated with an intensity of  $\geq 180 \text{ beats}\cdot\text{min}^{-1}$  (Baquet et al., 2003). Nevertheless, Baquet et al. (2003) only identified eight studies in girls, and thus few conclusions could be drawn regarding the effect of sex on the responses to endurance training.

Given the potential importance of training intensity, more recent studies have investigated the relative effectiveness of endurance and high-intensity interval training (HIIT) interventions. Typically, HIIT is characterised by periods of high-intensity exercise ( $\geq 85\%$   $HR_{max}$ ) interspersed with periods of low-intensity recovery (Laursen & Jenkins, 2002). Sperlich et al (2011), one of the first studies to directly compare a HIIT and endurance intervention, found that peak  $\dot{V}O_2$  and 1,000 m running time only significantly improved (both +11%) after a five-week HIIT intervention in male youth footballers ( $13.5 \pm 0.4$  years). Similar results were also reported following an eight-week HIIT intervention in trained adolescent footballers, independent of sex (Foster et al., 2015). Contrastingly, Faude et al. (2013) reported that peak  $\dot{V}O_2$  increased similarly regardless of training modality in a unique randomised cross-over design trial in elite German footballers ( $15.9 \pm 0.8$  years), though these findings must be interpreted with caution given the relatively small sample size ( $n = 20$ ) who also completed all arms of the study. Of note, all paediatric research exploring the differing effects of training interventions is limited to footballers, thus whether the same effects are manifest according to training type in different sports remains to be elucidated.

A common limitation amongst studies comparing training interventions is a lack of account for maturational status. This is crucial when assessing the effectiveness of training interventions due to the potential for androgenic hormones to exaggerate the response to training stimuli (Armstrong, 2007; Katch, 1983; Massicotte & Macnab, 1974; Rowland, 1997). Moreover, the onset and duration of puberty can vary significantly between children, even if they are of the same sex and ethnic background (Marshall & Tanner, 1969, 1970; Rogol et al., 2002; Tanner, Whitehouse, Marshall, & Carter, 1975). Therefore, participants of the same chronological age may not be of the same maturational status, potentially leading to erroneous conclusions regarding the effect of training.

### *2.1.1 Influence of maturity on the responses to training stimuli*

Puberty refers to the process of growth and sexual maturation during the transition from child to adulthood, and is characterised by periods of rapid growth and exponential increases in circulating androgenic hormones, namely testosterone (males) and oestrogen (females; Rogol, 2002). Puberty is a hugely dynamic time, with rapid, sex-dependent, changes in body size and composition occurring over relatively

short periods (Rogol, 1994, 2002; Rogol et al., 2002). An area within paediatric research which has received renewed interest over the last decade is the concept of a maturational threshold, first proposed by Katch (1983). The maturational threshold hypothesis suggests that at the onset of puberty the trainability of children and adolescents is significantly increased beyond those seen pre-puberty (Katch, 1983). This 'threshold' or 'trigger point' is purported to be mediated by the influx of androgenic hormones in the years surrounding peak height velocity (PHV), accelerating adaptations to training stimuli (Katch, 1983). This potential increased trainability presents a theoretical 'window of opportunity' for one to three years surrounding PHV in which greater performance benefits may be engendered (Rowland, 1997). Whilst a strong theoretical argument can be made for the existence of a maturational threshold for peak  $\dot{V}O_2$  (Boisseau & Delamarche, 2000; Mero, Jaakkola, & Komi, 1990; Metaxas et al., 2014; Van Praagh, 2000), there is very little empirical evidence to support it for aerobic parameters (Armstrong & McNarry, 2016; McNarry & Jones, 2014; McNarry et al., 2014b; Rowland, 1997).

Baquet et al. (2002) examined the influence of a seven-week HIIT intervention in 33 (20 girls) pre-pubertal children, determined using tanner staging, compared to a no-exercise control group (n = 20, 10 girls). The HIIT group significantly improved their absolute and relative peak  $\dot{V}O_2$  by 9.2% and 8.2%, respectively, though these results should be interpreted with caution given their prediction from a field-based 20 m shuttle run test, shown to have questionable validity (Armstrong & Welsman, 2020b). Similarly, Barker et al. (2014) reported that just two weeks of HIIT can elicit a 5% increase in peak  $\dot{V}O_2$  in post-pubertal adolescents. Of note, the magnitude of training response in Barker et al. (2014) was similar to, or in some cases exceeded, the magnitude of response observed over eight weeks of running-based interval training interventions in pre-pubertal children (Baquet, Gamelin, Mucci, Thevent, & van Praagh, 2010; Williams et al., 2000). However, whether these observations are due to training intensity, or suggest that participants have to be post-pubertal to be physiologically able to respond to training stimuli, remains to be established. McNarry et al. (2011b) reported the peak  $\dot{V}O_2$  of habitually trained swimmers (n = 23) compared to maturity- and sex-matched untrained girls (n = 36) and concluded that the magnitude of difference was similar between pre-, circa- and post-pubertal

participants, despite increases in training volume with age. Moreover, these results provide evidence that the maturational threshold hypothesis may be refuted in girls as pre-pubertal girls demonstrated similar degrees of trainability to their more mature peers (McNarry et al., 2011b).

Although cross-sectional, Weber et al. (1976) and Danis et al. (2003) utilised case-control study designs using monozygotic twins, thereby accounting for the genetic influence on peak  $\dot{V}O_2$  (Stratton & Williams, 2006). Both studies examined the effects of an endurance training intervention on peak  $\dot{V}O_2$  in one twin whilst the other acted as a control. Interestingly, both studies reported that during the pubertal years, boy's trainability was blunted when compared to younger children or older adolescents (Danis et al., 2003; Weber et al., 1976). Potential reasons for this 'dampened trainability' included the suppression of certain genes during puberty in the presence of growth hormones (GH), but this highly speculative. Alternatively, it may also be possible that the control twins engaged in a similar amount of physical activity (PA), and thus experienced concomitant increases in peak  $\dot{V}O_2$  compared to their trained twin. However, habitual PA was not assessed in either study and is often overlooked in training studies, potentially due to the equivocal evidence in the pediatric literature surrounding PA levels and peak  $\dot{V}O_2$  (Armstrong et al., 2011; Carson et al., 2016; Dencker & Andersen, 2011; Dencker et al., 2007; Ekelund et al., 2001). The reliance on ratio scaling could have also produced spurious associations between training and maturity, especially during puberty where increases in body mass often occur at a greater rate than improvements in peak  $\dot{V}O_2$ , potentially masking the effect of training (Nevill, Bate, & Holder, 2006; Nevill, Holder, Baxter-Jones, Round, & Jones, 1998; Tanner, 1949; Welsman & Armstrong, 2019).

The continued use of ratio scaling, despite its recognised limitations, may be one of the key reasons why the effect of training during maturity still remains debated (Armstrong & McNarry, 2016). Specifically, a number of prominent studies, dating as far back as Tanner (1949), infer that the use of ratio scaling peak  $\dot{V}O_2$  (i.e. dividing peak  $\dot{V}O_2$  ( $\text{ml}\cdot\text{min}^{-1}$ ) by body mass (kg; units:  $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) is not sufficiently statistically robust to fully account for growth and maturation (Armstrong, 2007; Armstrong & Welsman, 2019b; Cunha et al., 2011; Cunha et al., 2016; Welsman & Armstrong, 2019). The fallacy of ratio scaling was demonstrated by Welsman and

Armstrong (2019) who indicated a negative relationship between body mass and ratio-scaled peak  $\dot{V}O_2$  in a data set including over 1,700 incremental ramp tests. Moreover, simply dividing peak  $\dot{V}O_2$  by body mass assumes that body mass and peak  $\dot{V}O_2$  are directly proportional, which is not the case, especially during maturation (Katch, 1983; Tanner, 1949; Welsman & Armstrong, 2019). Allometric scaling seeks to alleviate some of these limitations by utilising log-linear regressions to remove the influence of body mass (Nevill et al., 2006; Nevill et al., 1998), and allows a population-specific exponent to be calculated which accurately describes the relationship between peak  $\dot{V}O_2$  and body mass.

Allometric scaling was first used by Nevill et al. (1998) who reported allometrically scaled peak  $\dot{V}O_2$  increased with age and maturity in boys, but not in girls, contrary to research at the time utilising ratio scaled data (Cunningham, Paterson, Blimkie, & Donner, 1984; Kobayashi et al., 1978; Paterson, McLellan, Stella, & Cunningham, 1987; Rowland, Vanderburgh, & Cunningham, 1997b). Contrastingly, Cunha et al. (2016) calculated lower-limb muscle volume (LLMV) using ultrasound and reported no significant difference between pre- ( $100.1 \pm 7.9 \text{ ml}\cdot\text{kg}^{-b(\text{LLMV})}\cdot\text{min}^{-1}$ ), circa- ( $107.5 \pm 9.6 \text{ ml}\cdot\text{kg}^{-b(\text{LLMV})}\cdot\text{min}^{-1}$ ) or post-pubertal ( $108.0 \pm 10.3 \text{ ml}\cdot\text{kg}^{-b(\text{LLMV})}\cdot\text{min}^{-1}$ ) adolescents after peak  $\dot{V}O_2$  was scaled to LLMV. However, it may be pertinent to note that the biggest change in allometrically scaled peak  $\dot{V}O_2$  occurred between pre- and circa-pubertal adolescents. Similar observations in male youth footballers have been reported by Doncaster et al. (2018) and dos Santos and colleagues (2012; 2014) when allometrically scaling by lean body mass. However, without the use of a control group it is unclear whether the increases from pre-pubertal to pubertal children is training or maturity related (Armstrong & Welsman, 2019b; Armstrong & Welsman, 2019d). Runacres, Mackintosh & McNarry (2019b) investigated the influence of a three-month training cycle on peak  $\dot{V}O_2$  in a group of male trained footballers and endurance runners, compared to a no-exercise control group. Pre- and post-intervention trained participants had a significantly higher peak  $\dot{V}O_2$  than their untrained counterparts, even after allometrically scaling for body mass (Runacres et al., 2019b), but there was no significant difference in the magnitude of change between maturity groups. Nevertheless, akin to all cross-sectional and intervention study designs, Runacres et al. (2019b) only offers a glimpse into the influence of maturity on responses to training



stimuli, and thus greater insights may be gained from longitudinal studies assessing the same participants over successive years.

Early longitudinal studies on peak  $\dot{V}O_2$  were conflicting, with Kobayashi et al. (1978) reporting that peak  $\dot{V}O_2$  did not significantly increase before PHV in fifty untrained controls and six highly-trained long-distance runners, measured annually for five-years between the ages of 9 – 13 years. However, it is pertinent to note that Kobayashi et al. (1978) did not appropriately account for maturity status, missing the age of PHV for most participants. Similarly, in a ten-year longitudinal study of Canadian children, Mirwald et al. (1981) reported that training differences in peak  $\dot{V}O_2$  were not apparent until approximately 14 years of age, coinciding with PHV. Contrastingly, Baxter-Jones et al. (1993) assessed peak  $\dot{V}O_2$  in 271 youth athletes (126 girls) annually for three years, reporting only boy's peak  $\dot{V}O_2$  increased with maturity, and training adaptations were evident in pre-pubertal children, contradicting previous studies (Cunningham et al., 1984; Kobayashi et al., 1978; Mirwald et al., 1981). Whilst Baxter-Jones et al. (1993) was novel in its longitudinal design, the reliance on ratio scaling potentially confounds any meaningful interpretation (Tanner, 1949; Welsman & Armstrong, 2019), and the omission of a control group precludes inferences as to whether increases in peak  $\dot{V}O_2$  were training or maturity related. Additionally, and perhaps most importantly, the results of Baxter-Jones et al. (1993) must be interpreted with caution due to the reliance on secondary criteria to determine a maximal peak  $\dot{V}O_2$  (Barker et al., 2009). Traditional secondary criteria used to validate a maximal effort in adults, including a blood lactate  $\geq 6$  mmol, HR within 95% age predicted max, and an RER  $\geq 1.15$ , have been found to underestimate peak  $\dot{V}O_2$  by as much as 20%, if they are achieved at all (Barker et al., 2009). Therefore, reliance on secondary criteria to validate a maximal effort could lead to the acceptance of sub-maximal peak  $\dot{V}O_2$  values, resulting in erroneous conclusions regarding fitness, intervention effectiveness and/or maturational influences on trainability. To circumvent these issues, it is recommend that participants are asked to complete a supramaximal validation bout but, despite its recommendation, high reliability, and validity (Barker et al., 2009; Poole & Jones, 2017; Schaun, 2017), this technique remains underutilised in paediatric research and therefore the accuracy of many peak  $\dot{V}O_2$  measurements may be questionable.

McNarry et al. (2014b) incorporated an allometric scaling in their three-year longitudinal study involving 19 trained swimmers and 15 untrained controls. McNarry et al. (2014b) reported that trained pre-pubertal children demonstrated a higher allometrically scaled peak  $\dot{V}O_2$  at all time-points, suggesting maturity does not need to occur before a significant training response is observed. However, a maturational threshold may have occurred post-PHV, given that even at the endpoint of the study all children were still pre- or early pubertal (year 3 maturity offset: swimmers:  $-0.7 \pm 0.5$  years and controls:  $-1.2 \pm 1.0$  years). Moreover, the data from boys and girls were pooled for analysis, precluding any sex differences in training responses being elucidated (McNarry et al., 2014b). Nevertheless, McNarry et al. (2014b) provide evidence, utilising robust methodological and statistical measures, that refutes the maturational threshold hypothesis. However, one common limitation with all training studies discussed so far is the lack of consideration of habitual PA levels on peak  $\dot{V}O_2$ . Indeed, the inclusion criteria for the control, or untrained, groups in training studies is that participants are not involved within formal exercise programs (Baquet, Berthonin, Gerbeaux, & Van Praagh, 2001; Milanović, Sporiš, & Weston, 2015; Runacres et al., 2019b), but that does not preclude them being physically active. Indeed, this could help explain, at least in part, the variance between different training studies and the different effects of maturity reported.

## **2.2 Potential effect of Physical Activity on $\dot{V}O_{2\max}$**

Physical activity is defined as any bodily movement that results in energy expenditure above resting (Ekelund et al., 2001), with movement intensity quantified in relation to rest by using the metric of metabolic equivalents (METs; Ainsworth et al., 2000). Although PA and peak  $\dot{V}O_2$  are the two most researched parameters in paediatric exercise science over the last 30 years, their relationship remains contentious (Armstrong, 2013; Dencker et al., 2006; Fenster et al., 1989; Latt et al., 2013). The interaction between PA and peak  $\dot{V}O_2$  in children and adolescents was first investigated by Fenster et al. (1989) who assessed PA using accelerometry and peak  $\dot{V}O_2$  using an incremental treadmill test to exhaustion in 6- to 8-year old children. Fenster et al. (1989) reported a significant positive correlation ( $r^2 = 0.59$ ,  $p < 0.05$ ) between PA and peak  $\dot{V}O_2$ . However, Fenster et al. (1989) only measured PA for one day and thus the reliability is highly questionable. Armstrong et al. (1991a) measured

peak  $\dot{V}O_2$  and habitual PA, using HR monitoring over three school days, in 253 children (199 boys;  $13.2 \pm 1.3$  years) and reported no significant correlations between habitual PA and peak  $\dot{V}O_2$ . Similarly, Ekelund et al. (2001) reported no relationship between habitual PA, quantified using three-day HR monitoring, and treadmill-determined peak  $\dot{V}O_2$  after accounting for maturational and body fat differences between participants. Nevertheless, it is pertinent to note that whilst the quantification of PA based on HR is highly reliable for determining resting energy expenditure, higher intensities are less accurate (Schutz, Weinsier, & Hunter, 2001). Indeed, training has been consistently reported to lower submaximal HR values, therefore the use of fixed HR thresholds could over/under estimate exercise intensities in diverse populations (Schutz et al., 2001).

A consistent finding within the paediatric literature when using accelerometry to quantify PA, sedentary time (SED) and sleep is that vigorous intensity PA (VPA) is more strongly associated with peak  $\dot{V}O_2$  than any other movement behaviour (Dencker & Andersen, 2011; Dencker et al., 2007; Dencker et al., 2006; Gutin, Yin, Humphries, & Barbeau, 2005; Latt et al., 2013). Indeed, measuring PA in 421 adolescents (16 years) using a hip-worn accelerometer over five consecutive days, Gutin et al. (2005) found that whilst peak  $\dot{V}O_2$  was correlated with MPA ( $r^2 = 0.30$ ,  $p < 0.01$ ), the association with VPA was stronger ( $r^2 = 0.43$ ,  $p < 0.01$ ). Similarly, Dencker et al. (2006) and Latt et al. (2013) reported that time spent in VPA explained approximately 9.0% and 15.8% of the variance in peak  $\dot{V}O_2$  and relative peak  $\dot{V}O_2$ , respectively. However, these results must be interpreted with caution given the relatively small amount of VPA engaged in by the participants across the studies ( $5.0 - 35.0 \text{ mins} \cdot \text{day}^{-1}$ ), equating to just 0.3 – 2.5% of the 24-hour period. Consequently, a compositional approach which utilises all available PA data (Chastin et al., 2015; Dumuid et al., 2018a) may be more appropriate to delineate this relationship.

Compositional analysis techniques have recently been introduced and utilised in PA research, providing a new perspective on the influence of PA, SED and sleep on health parameters in children (Carson et al., 2016; Carson et al., 2019; Dumuid et al., 2018b; Väistö et al., 2019). Compositional analysis techniques alleviate many of the limitations of traditional statistical methods such as correlational statistics, which can not infer causality (Hopkins, Marshall, Batterham, & Hanin, 2009), and the use of

predictive linear regressions which assume independence between variables (Chastin et al., 2015). More specifically, the assumption of independence is violated when working with movement data (i.e. SED, LPA, MPA, VPA, sleep) as increases in one behaviour must come at the detriment of another, given that there is only a finite amount of time (1,440 minutes) within a day (Dumuid et al., 2018a). Compositional analysis techniques overcome this using isometric log transformations of the PA data (Chastin et al., 2015; Dumuid et al., 2018a), allowing for the individual and combined effects of movement behaviours to be explored. Additionally, compositional analysis techniques have the ability to predict an 'optimal' movement composition for outcome variables (Chastin et al., 2015), thus it could be used in a performance setting to improve athletic parameters, although this remains unexplored.

The first study to use compositional analyses in a large sample of children ( $n = 4,169$ ;  $11.4 \pm 0.1$  years) reported that the overall PA composition explained ~38% of the variance in estimated aerobic fitness (Carson et al., 2016). Moreover, SED was negatively associated with predicted peak  $\dot{V}O_2$  ( $\beta = -32.8$ ,  $p > 0.01$ ), whilst, conversely, MVPA was positively associated with predicted aerobic capacity ( $\beta = 22.0$ ,  $p > 0.01$ ). Nevertheless, when predictive modelling was employed to explore the effects of 10 minutes re-allocation of time to or from MVPA to one of the other movement behaviours (SED, LPA, sleep), there were negligible effects on predicted aerobic capacity (0.01 – 0.05%; Carson et al., 2016). The authors postulated this negligible change indicated the powerful influence of SED on this critical health and performance indicator (Carson et al., 2016) but, alternatively, it could reflect certain methodological limitations. More specifically, aerobic fitness was estimated using a field-based measure (the Canadian Aerobic Fitness Test), which has questionable validity, and the maturity and training status were not determined. Furthermore, the pooling of data from boys and girls potentially confounds the results (Armstrong, 2007; Carson et al., 2016; Carson et al., 2019; Chastin et al., 2015; Lynch et al., 2019; McNarry & Jones, 2014). However, perhaps the biggest limitation, was the pooling of MPA and VPA into MVPA, thereby potentially masking the importance of intensity in the relationship between PA and peak  $\dot{V}O_2$  (Dencker & Andersen, 2011; Dencker et al., 2006; Gutin et al., 2005; Latt et al., 2013).

Future research is urgently needed to examine the influence of PA on peak  $\dot{V}O_2$  development in children and adolescents. More specifically, PA and training both exert powerful influences on physiological parameters (Al-Mallah, Sakr, & Al-Qunaibet, 2018; Carson et al., 2016; Carson et al., 2019; Chastin et al., 2015; Imboden et al., 2018; Pollock, Duggal, Lazarus, Lord, & Harridge, 2018), thus without the consideration of PA it is impossible to delineate whether increases in performance are training or PA related. Indeed, using a four-part composition of waking hours (i.e. SED, LPA, MPA, VPA) in 2,500 children and adolescents, Carson et al. (2019) reported that increased levels of VPA were more strongly associated with a favourable BMI z-score, waist circumference, diastolic blood pressure, and HDL-cholesterol, than MPA. Consequently, it could be postulated that VPA specifically could be more important than MVPA for the development of peak  $\dot{V}O_2$  in youth given the meaningful associations with cardiovascular risk factors in youth (Carson et al., 2019; Väistö et al., 2019). However, research has not explored the effect of MPA and VPA separately on rigorously determined peak  $\dot{V}O_2$  using compositional analyses techniques, accounting for sex, maturity and training status.

### **2.3 Influence of sex on the trainability of $\dot{V}O_{2max}$**

Whilst the effects of maturity on the trainability of peak  $\dot{V}O_2$  are starting to be elucidated, a key area where substantial questions remain is the effect of sex on the interaction between training and maturity (Armstrong & McNarry, 2016). Indeed, there is a clear male bias within the literature, with a paucity of well-designed studies comparing the trainability of boys and girls across the maturational spectrum (Armstrong & McNarry, 2016; Armstrong & Welsman, 2019b). Consequently, it remains unclear whether the findings in boys can be applied to girls given their distinct physiological differences, not least in the timing and tempo of maturity (Rogol, 2002; Rogol et al., 2002). Specifically, puberty typically begins around the ages of 11 and 13 in girls and boys, respectively, but boys experience a two-year longer growth period than girls (Rogol, 1994, 2002). Moreover, body composition changes are highly sexually dimorphic; whilst pre-pubertal boys and girls display similar fat free mass (FFM) accrual rates between the ages of 5 to 10 years, thereafter boys gain FFM at a greater rate (He et al., 2004; Rogol et al., 2002). This difference in FFM accrual is

thought to be mediated by boy's higher concentration of circulating testosterone, but may also be a consequence of boys' higher habitual PA levels (Bitar et al., 2000; Dumith, Gigante, Domingues, & Kohl III, 2011), especially in the teenage years (Dumith et al., 2011), which may potentially exaggerate the sexual dimorphism in peak  $\dot{V}O_2$  during adolescence. Thus, it could be postulated that training responses in girls may be higher in girls due to the comparatively lower baseline peak  $\dot{V}O_2$  and habitual PA levels (McNarry & Jones, 2014). Therefore, whether the sex differences in anthropometrics and PA translate to differences in aerobic fitness, and modulate the responses to training stimuli, remains to be determined.

Tentative comparisons regarding the effect of training suggest that pre-pubertal girls experience a similar level of improvement in peak  $\dot{V}O_2$  in response to a training stimuli (7.8% - 9.1%; McManus et al., 1997; McNarry et al., 2011b) as those reported elsewhere in pre-pubertal boys (4.1 - 11.1%; Baquet et al., 2002; McManus, Cheng, Leung, Yung, & MacFarlane, 2005), irrespective of exercise type or modality. However, research is equivocal on the trainability of young girls, with other studies reporting no significant response to training stimuli when compared to age-, sex- and maturity-matched controls (Stoedefalke et al., 2000; Welsman et al., 1996; Welsman et al., 1997). These contradictory findings could be explained, at least in part, by the varying baseline fitness levels of the participants across the studies (McNarry & Jones, 2014), the volume of training conducted and/or a discordant training and testing exercise modality (Stoedefalke et al., 2000). Indeed, when peak  $\dot{V}O_2$  is rigorously determined, evidence in girls suggests that training differences remain fairly consistent across maturity stages (14.4% - 17.9%; McNarry et al., 2014b), increasing in an almost linear manner as has been observed in boys (19.6% - 20.1%; Bitar et al., 2000; Paterson et al., 1987). Taken together, emerging evidence refutes the maturational threshold hypothesis in both boys and girls, although this evidence is inconsistent.

In 118 active children (40 girls,  $11.5 \pm 0.5$  years) assessed annually for three years, Malina et al. (1997) found that only boys demonstrated maturity-associated variations in absolute peak  $\dot{V}O_2$  after co-varying for body mass. However, it is pertinent to note the different methods of maturity assessment between boys and girls, with boys grouped by growth rates and girls grouped by age at menarche (Malina et al., 1997). Whilst somatic and sexual maturity are highly correlated, they are independent

measures of maturity (Roemmich et al., 1998; Rogol, 2002) and are therefore not directly comparable. Nonetheless, Baxter-Jones et al. (1993) reported that the significant increase in peak  $\dot{V}O_2$  in the post-pubertal maturity stage was only observed in boys, with girls experiencing a plateau, suggesting that the development of peak  $\dot{V}O_2$  may be sex dependent. Contrastingly, Armstrong and Welsman (2001) reported a significant maturational influence on peak  $\dot{V}O_2$ , after accounting for FFM, in a sample of 132 children (49 girls) followed annually from 11 – 13 years, with a subset (n = 63) followed-up at 17 years. Interestingly, both age and maturity were significant predictors of peak  $\dot{V}O_2$ , suggesting that maturity alone cannot explain the development of peak  $\dot{V}O_2$ . Additionally, when haemoglobin concentration was introduced into the predictive model, this was not significant indicating that oxygen carrying capacity does not limit the development of peak  $\dot{V}O_2$  in girls.

One of the key considerations when assessing the sex differences in peak  $\dot{V}O_2$  is body composition (Armstrong & Welsman, 2019b; Armstrong & Welsman, 2020c; McNarry et al., 2015; Winsley et al., 2009). Greater insights may therefore be gained by scaling peak  $\dot{V}O_2$  by lean body mass (LBM) or FFM. Recognising this, Winsley et al. (2009) determined peak  $\dot{V}O_2$  in 18 (9 boys) pre-pubertal children and found that boys still displayed a ~12.8% higher peak  $\dot{V}O_2$ , even after allometrically scaling for LBM. Similarly, McNarry et al. (2015) reported a sex difference of ~18% in absolute peak  $\dot{V}O_2$  in 52 recreationally active pre-pubertal children, with the sex difference persisting even after allometrically scaling for body mass (~16.2%) and FFM (~11.7%). However, sex differences in LBM and FFM are not typically manifest until the pubertal or post-pubertal maturational stages (He et al., 2004; Rogol et al., 2002), and thus it would have been interesting if pubertal or post-pubertal boys and girls were also included within McNarry et al. (2015) and Winsley et al. (2009).

Armstrong and Welsman (2019b) recently published data from over 1,000 peak  $\dot{V}O_2$  tests on 372 (181 girls) children and adolescents, assessed annually for three consecutive years, and reported that boys had a 10% – 15% higher peak  $\dot{V}O_2$  than girls after accounting for age, body mass and FFM, similar to the sex difference reported elsewhere in pre-pubertal children (McNarry et al., 2015; Winsley et al., 2009). However, Armstrong and Welsman (2019b) utilised sex-specific longitudinal modelling, which relies heavily on the completeness of the dataset, thus the lack of

detail provided on the dataset completeness by the authors means applicability of these results to other populations remains to be elucidated. Despite the limitations of Armstrong and Welsman (2019b), longitudinal data analysis facilitates greater insights into the interaction between training and maturity, and offers the strongest evidence regarding the influence of training and maturity on peak  $\dot{V}O_2$  development. Overall, given that sex differences appear to persist beyond LBM, it seems unlikely that the sexually dimorphic development of peak  $\dot{V}O_2$  is exclusively attributable to differences in body composition. Research is therefore warranted investigating the underpinning mechanisms, namely haemodynamic and muscle deoxygenation parameters, to potentially allow for a greater insight into the sex-specific development of peak  $\dot{V}O_2$ , and how this interacts with training and maturity.

#### **2.4 Mechanisms underpinning training-, sex- and maturity-related differences in $\dot{V}O_{2max}$**

Peak  $\dot{V}O_2$  reflects the coordinated response of numerous physiological processes, two of the most important of which are oxygen delivery and oxygen extraction at local muscle sites as reflected by the Fick equation. The balance between oxygen delivery and extraction was first described by Adolf Fick (1855, 1870b) who described peak  $\dot{V}O_2$  as the product of cardiac output ( $\dot{Q}$ ) and arteriovenous difference ( $a-\bar{v}O_{2diff}$ ), where  $\dot{Q}$  is defined as the amount of blood leaving the left ventricle per minute ( $l \cdot min^{-1}$ ) and  $a-\bar{v}O_{2diff}$  is the difference in the oxygen concentration of the blood in the arteries and veins, providing an insight into peripheral oxygen extraction (Fick, 1870b). Given the rapid and sexually-dimorphic development of peak  $\dot{V}O_2$  during growth and maturation, the relative determinants of peak  $\dot{V}O_2$  may also differ with sex, training, and maturity status. Therefore, a greater understanding of children's oxygen delivery and extraction capabilities could further our understanding of the mechanisms underpinning peak  $\dot{V}O_2$  development, and reveal which mechanism, if any, is the main driver behind sex-, training- and maturity-related differences.

Oxygen delivery to the working muscles during exercise is determined by both the pulmonary and haemodynamic response to stimuli (Kohzuki, 2018). Concomitant with the growth and maturation of skeletal muscle, the size of the lungs increases from child to adulthood, with young adults lung capacity on average three-to-four times greater than a pre-pubertal child (Piccioni et al., 2015). Lung development during



childhood and adolescence is directly proportional to chest dimensions but lung function is dependent on numerous factors including age, sex, stature, and ethnicity (Miller et al. 2005). The two most commonly measured pulmonary factors are forced vital capacity (FVC), the amount of air that can be forcibly exhaled from the lungs measured using spirometry, and the forced expiratory volume in one second (FEV1). During puberty, FVC almost quadruples in healthy individuals (Lum et al. 2010), facilitated by increases in muscular strength, changes in the shape and stiffness of the thorax, and the number and size of alveoli in the lungs (Miller et al. 2005). Airflow, measured using FEV1, is influenced by the calibre of the airways and in some pulmonary diseases such as asthma and cystic fibrosis this can lead to arterial hypoxaemia (DiDario & Becker, 2005). Arterial hypoxaemia is defined as a partial pressure in arterial blood pressure (PaO<sub>2</sub>) less than 80 mmHg while breathing ambient air (DiDario & Becker, 2005). The lack of circulating oxygen severely limits exercise tolerance with arterial hypoxaemia limiting the transfer of oxygen to the working muscles leading to a greater reliance on anaerobic metabolism to sustain exercise demands.

Arterial hypoxaemia can also be a consequence of poor diffusion rates of oxygen across the alveoli to the haemoglobin in the blood as described by Ficks Law (1873). Ficks law states that oxygen diffusion is dependent upon the difference in pressure between the oxygen in the lungs and blood, the area of the lungs, the diffusion constant, and alveolar wall thickness (Fick, 1873; Hopper et al. 1991). With age, the surface area of the lungs increases by 150% from 8 – 18 years and the alveolar wall thickness decreases, making oxygen diffusion more efficient (Hopper et al. 1991). Indeed, the maximum diffusion rate increases from 1.80 l·min<sup>-1</sup> at 9 years to 3.77 l·min<sup>-1</sup> in young adulthood (Hancox & Rasmussen, 2018) and the minimum capillary transfer time for one molecule of oxygen is 0.45 s. Moreover, these adaptations which occur with growth and maturation also increase the perfusion rate, allowing greater expiration of carbon dioxide and waste products from exercise (DiDario & Becker, 2005). As lung size, capacity, and air flow increases, resting ventilation rate also decreases, with adults typically breathing 10-15 times·minute<sup>-1</sup> compared to 20-30 times·minute<sup>-1</sup> for a pre-pubertal child (Piccioni et al., 2015). This lower resting ventilation rate allows for a greater response to exercise as maximal ventilation rate

remains unchanged between children and adults (Piccioni et al. 2015). However, the importance of pulmonary determinants of  $\dot{V}O_{2\max}$  has been disputed in recent years; without sufficient diffusion into the blood, and transport to the working muscles,  $\dot{V}O_{2\max}$  would not be achieved. Therefore, the haemodynamic mechanisms underpinning  $\dot{V}O_{2\max}$  may allow greater insights to be made on the influence of sex, and maturity.

#### *2.4.1 Haemodynamic mechanisms*

Cardiac output, the product of stroke volume (SV; the amount of blood ejected from the left ventricle per beat) and HR, increases almost linearly with age, mediated by increases in cardiac size and change in cardiac morphology (Obert et al., 2003; Vinet et al., 2003). Both cross-sectional and longitudinal studies have demonstrated that  $HR_{\max}$  is independent of sex, training or maturity status (Armstrong & Welsman, 2020a; Armstrong & Welsman, 2020c; McNarry et al., 2015; McNarry et al., 2014b), and therefore SV is suggested to be the primary factor underpinning the increases in  $\dot{Q}$  typically observed with age. Increases in SV with age are purported to be mediated by morphological changes to the myocardium, including increases in left ventricular mass (LVM) and posterior and septal wall thickness (mm; Eisenmann et al., 2000; Milicevic, Fabecic-Sabadi, Rudan, Kokos, & Lukanovic, 1997). Indeed, LVM has been reported to increase by 8.6% and 15.3% per year between the ages of 11 and 14 in girls and boys, respectively, in a three-year longitudinal study of Croatian adolescents (Milicevic et al., 1997). Moreover, posterior wall thickness and septal wall thickness increased by 21.5% and 15.9%, respectively, between 9 – 18 years old in boys, with girls experiencing a lesser magnitude of change in posterior wall thickness (13.9%) and septal wall thickness (6.5%) over the same time-period (Eisenmann et al., 2000). Consequently, these sexually-dimorphic changes in the structural properties of the myocardium may partly explain the sex differences in  $SV_{\max}$  and  $\dot{Q}_{\max}$  reported with growth and maturity.

The apparent sex differences in the structural adaptations that occur during growth and maturation are suggested to be attributable to differences in FFM, stature and resting blood pressure according to sex (Eisenmann et al., 2000; Milicevic et al., 1997). Increases in resting blood pressure enhance LVM through an increased pre-loading capacity and shearing stress, facilitating hypertrophy of the myocardium (Eisenmann

et al., 2000; Rowland & Green, 1988). Additionally, FFM is highly correlated with cardiac size in children ( $r^2 = 0.77$ ,  $p < 0.01$ ), with a greater FFM and stature not only eliciting a greater oxygen delivery, but also augmenting the venous return to the myocardium, thereby increasing the pre- and after-load conditions of the heart (Eisenmann et al., 2000; Vinet et al., 2003). Indeed, when expressed relative to LBM, sex differences in the  $SV_{\max}$  and  $\dot{Q}_{\max}$  of children and adolescents are ameliorated (Armstrong & Welsman, 2019a; Vinet et al., 2003; Winsley et al., 2009). Moreover, body dimensional differences seem to explain child-adult differences in  $SV_{\max}$  and  $\dot{Q}_{\max}$  ( $l \cdot \min^{-1} \cdot m^{-2}$ ; Marwood, Roche, Rowland, Garrard, & Unnithan, 2010; Rowland, 1997; Rowland & Green, 1988). Specifically, when allometrically scaling by body surface area, neither  $SV_{\max}$  nor  $\dot{Q}_{\max}$  ( $10.5$  vs  $10.1$   $l \cdot \min^{-1} \cdot m^{-2}$ ,  $p > 0.05$ ; Rowland et al., 2000b) were significantly different in pre-pubertal girls ( $11.7 \pm 0.7$  years) and young adult women ( $27.4 \pm 2.3$  years). It is pertinent to note the interstudy discrepancies in the scaling parameter used, with some allometrically scaling  $SV_{\max}$  and  $\dot{Q}_{\max}$  by body surface area (McNarry et al., 2014b; McNarry et al., 2011b; Rowland et al., 1997b) and others by LBM (Obert et al., 2003; Vinet et al., 2003). Scaling  $SV_{\max}$  by LBM allows for a greater control of body composition differences compared to body surface area, and therefore direct comparisons between these two scaling methods are not advisable (Armstrong & Welsman, 2020a).

The absence of significant differences in the  $SV_{\max}$  and  $\dot{Q}_{\max}$  of pre-pubertal could be attributable to a lack of cardiac growth (Eisenmann et al., 2000; Milicevic et al., 1997) and/or to an absence of circulating testosterone in pre-pubertal children which is needed to stimulate hypertrophy in cardiac cells (Hayward, Webb, & Collins, 2001), and thus support the maturational threshold hypothesis (Rowland & Boyajian, 1995; Rowland & Obert, 2012; Rowland, Popowski, & Ferrone, 1997a). Alternatively, the small difference in  $SV_{\max}$  and  $\dot{Q}_{\max}$  between trained and untrained pre-pubertal children may be suggestive that oxygen delivery is not limiting exercise and therefore there is no stimulus to increase cardiac capacity in pre-pubertal children (Rowland & Green, 1988; Rowland et al., 2000b). Whilst the influence of training, sex and maturity is questionable once  $SV_{\max}$  and  $\dot{Q}_{\max}$  appropriately scaled, training status does appear to exert a significant impact on the SV response profile during exercise (McNarry et al., 2014b; McNarry et al., 2011b). Specifically, studies have reported untrained

children and adolescents to demonstrate a SV plateau at 40-50% peak  $\dot{V}O_2$ , whereas trained adolescents demonstrate a near-linear increase in SV with  $\dot{V}O_2$  until exhaustion (Armstrong & Welsman, 2019a; Armstrong & Welsman, 2020a; McNarry et al., 2014b; McNarry et al., 2011b), irrespective of maturity.

In untrained children, it is postulated that SV may rise to an ‘optimal’ level in untrained children with HR facilitating any further increase in  $\dot{Q}$  required to match the metabolic demand (Armstrong & Welsman, 2020a; Rowland, Goff, Martel, & Ferrone, 2000a; Rowland & Unnithan, 2013). The mechanistic basis for the near-linear increase in SV in trained youth is unclear, however, an enhanced diastolic filling capacity, coupled with an increased venous return, an increased left ventricular dimension, and an increased muscular  $O_2$  extraction have all been suggested (D'Ascenzi et al., 2019; McNarry et al., 2014b; McNarry et al., 2011b). However, given Obert et al. (2003) and Nottin et al. (2002) reported no influence of training on the SV pattern in less intensively endurance-trained children, there may be a training volume and/or intensity threshold above which the SV response pattern is altered. Specifically, the participants in Obert et al. (2003) and Nottin et al. (2002) were only recreationally active compared to the trained swimmers in McNarry et al. (2014b; 2011b) who were training at least six hours a week for the preceding two years prior to study entry. Therefore, a more intensive and/or chronic training stimuli may be necessary to engender differing SV response profiles to exercise.

Given that absolute  $SV_{max}$  and  $\dot{Q}_{max}$  increase naturally with growth and maturation, a control group is necessary to differentiate any functional training adaptations in youth athletes (Armstrong, 2007). Obert et al. (2003) reported increases in  $SV_{max}$  after a 13-week endurance training intervention in both girls and boys, although it may be worth noting that the  $SV_{max}$  in boys increased to a greater extent than observed in the girls (15% vs 11%, respectively). The rise in  $SV_{max}$  in both sexes was mediated by an increase in left ventricular end diastolic diameter (LVEDd) and LVM, suggesting that training-induced adaptations are independent of sex (Obert et al., 2003). However, one limitation associated with Obert et al. (2003) is the failure to account for maturity, with the age of 10 – 11 years associated with the transition to pubertal status in many girls (Marshall & Tanner, 1969; Roemmich et al., 1998). Consequently, whether the cardiovascular determinants of training are independent of sex, or girls experienced

maturity-, not training-, related increases in  $SV_{\max}$  and the myocardium is not possible to delineate. McNarry et al. (2011b) reported significantly higher scaled  $SV_{\max}$  and  $\dot{Q}_{\max}$  in trained pubertal and post-pubertal girls compared to their untrained counterparts, however, interestingly, there was no significant difference between trained and untrained pre-pubertal girls. Similarly, trained and untrained pre-pubertal boys demonstrate small differences in  $SV_{\max}$  and  $\dot{Q}_{\max}$  once scaled by LBM or body surface area (Armstrong & Welsman, 2019a; Bossone, Vriza, Bodini, & Rubenfire, 2004; Fellmann & Coudert, 1994b; Forbregd, Aloyseus, Berg, & Greve, 2019). Moreover, McNarry et al. (2014b) reported no significant differences over time in  $SV_{\max}$  and  $\dot{Q}_{\max}$  in either trained or untrained children over the course of a three-year longitudinal study. Similarly, Winsley et al. (2009) reported pre-pubertal boys to have a 12.8% higher peak  $\dot{V}O_2$  than pre-pubertal girls when scaled by LBM but no sex differences for either  $SV_{\max}$  or  $\dot{Q}_{\max}$ . Therefore, sex and maturity differences in peak  $\dot{V}O_2$  seem unlikely to be mediated through central mechanisms and peripheral adaptations facilitating enhanced oxygen extraction may be of greater importance.

#### 2.4.2 Muscle Deoxygenation kinetics

Recent advances in technology have allowed paediatric researchers to non-invasively examine muscle deoxygenation kinetics using near infrared spectroscopy (NIRS), typically examined at the *m. vastus lateralis* (Barstow, 2019; Breese, Saynor, Barker, Armstrong, & Williams, 2019; Marwood et al., 2010; Willcocks, Williams, Barker, Fulford, & Armstrong, 2010). The infrared light utilised is typically within the 700 – 900 nanometre (nm) wavelength (Barstow, 2019), which allows the detection of light-absorbing chromophores, namely haemoglobin (HHb) and myoglobin (Mb; Barstow, 2019; Boone, Koppo, Barstow, & Bouckaert, 2009; La Mantia, Neidert, & Kluess, 2018; Ryan, Southern, Reynolds, & McCully, 2013). NIRS therefore has the potential to offer insights into physiological differences in local muscle microvasculature associated with sex, training and/or maturity status (Barstow, 2019; Boone et al., 2009). NIRS devices have a high measurement resolution, allowing for real-time quantification of muscle deoxygenation kinetics that can be normalised to end-exercise values and modelled against work rate and  $\dot{V}O_2$  to enable comparisons between populations (Barstow, 2019; Boone et al., 2009; McNarry et al., 2015). Consequently, if muscle deoxygenation kinetics are assessed alongside SV,  $\dot{Q}$ , and the

$a-\bar{v}O_{2\text{diff}}$ , a holistic assessment can be obtained of the balance between oxygen delivery ( $SV, \dot{Q}$ ) and extraction ( $a-\bar{v}O_{2\text{diff}}$  and muscle deoxygenation) during exercise.

Willcocks et al. (2010) reported no significant differences between 13 year-old children and adults in the concentration of deoxyhemoglobin ([HHb]) or phosphocreatine (PCr) response at the onset of heavy-intensity constant work rate exercise, assessed via NIRS and  $^{31}\text{P}$ -magnetic resonance spectroscopy ( $^{31}\text{P}$ -MRS), respectively. However, despite an initial matching of oxidative capacity in children and adults, the mean response time (MRT) of muscle deoxygenation was significantly faster in children ( $22 \pm 4$  s vs  $27 \pm 7$  s, respectively). This was attributed to an impaired oxygen delivery in children, indicating a poorer matching of oxygen delivery to extraction which is thought to improve with age due to androgenic hormones increasing haemoglobin levels within the blood (Thomsen, Riis, Krabbe, & Christiansen, 1986; Vinet et al., 2003). Nevertheless, the association between circulating androgens and haemoglobin is weak (Armstrong & Welsman, 2001; Vinet et al., 2003), and is therefore unlikely to contribute significantly to the age-related differences in localised muscle oxygen extraction (Ratel, Tonson, Cozzone, & Bendahan, 2010; Ratel, Tonson, Le Fur, Cozzone, & Bendahan, 2008).

The results of Willcocks et al. (2010) are supported by those of Breese et al. (2019) who reported a decrease in the MRT of the tissue oxygenation index (TOI, %) during transitions to moderate and very-heavy cycling transitions in children and adolescents. The slowing of the [HHb] response with advancing age was attributed to a greater muscle mass in the older participants, facilitating a greater local  $O_2$  extraction, thus initiating a right-ward shift of the [HHb] response (Breese et al., 2019). However, the novel finding of Breese et al. (2019) was that during the transition from moderate to very-heavy intensity exercise the slower pulmonary  $\dot{V}O_2$  kinetics were a result of a slower microvascular blood flow, not oxygen delivery (Breese et al., 2019). Endurance training augments this relationship, engendering a right-ward shift in both children (McNarry et al., 2011b) and young adults (Boone et al., 2009), which has been attributed to an increased abundance of type I muscle fibres and an increased  $SV_{\text{max}}$  and  $\dot{Q}_{\text{max}}$  increasing oxygen delivery capacity (McNarry et al., 2014b; McNarry, Welsman, & Jones, 2011c). More specifically, at low-to-moderate work rates, blood flow is primarily distributed to slow twitch muscle fibres (R. Armstrong & Laughlin,

1984), with this response enhanced by training (R. Armstrong & Laughlin, 1984). Therefore, the rightward-shift in the [HHb] response with training is likely mediated by differences in muscle fibre types and increases in regional blood flow.

In one of the few studies to use NIRS in children during incremental exercise, McNarry et al. (2015) examined the acute muscle deoxygenation response of the *m. vastus lateralis* in 52 (21 girls;  $9.9 \pm 0.6$  years) pre-pubertal children during a ramp test to exhaustion. The primary finding was that muscle deoxygenation kinetics explained ~12% of the variance in peak  $\dot{V}O_2$  between boys and girls after accounting for FFM, GET and percentage of body fat in pre-pubertal children (McNarry et al., 2015). Interestingly, utilising multi-level modelling techniques, Armstrong et al. (2019a) reported that after accounting for LBM and  $SV_{\max}$  there was still an ~4% sex difference in peak  $\dot{V}O_2$ , with a similar discrepancy in peak  $\dot{V}O_2$  (~5%) when LBM,  $\dot{Q}_{\max}$  and  $a-\bar{v}O_{2\text{diff}}$  were controlled for (Armstrong & Welsman, 2020a). Consequently, it could be postulated that the remaining unexplored variance in these studies (4 – 5%) may have been explained if a more sensitive measure of muscular deoxygenation kinetics was utilised, as opposed to the  $a-\bar{v}O_{2\text{diff}}$ . However, given that McNarry et al. (2015) only included pre-pubertal children, whether a divergent [HHb] response persists in pubertal and post-pubertal children remains to be determined.

## **2.5 Influence of sex and maturity on anaerobic responses to training in youth**

Anaerobic performances offer an indication of the body's ability to generate energy production through non-oxidative pathways to meet the metabolic demands of strenuous exercise (Armstrong & Welsman, 2020c). Anaerobic trainability has received substantially less attention in the paediatric literature than aerobic fitness (Armstrong & Welsman, 2020c; Van Praagh, 2000; Van Praagh & Doré, 2002), despite the observation that children's PA is often short and sporadic, and therefore may be anaerobic by nature (Holman, Carson, & Janssen, 2011; Whooten, Kerem, & Stanley, 2019).

The potential for a maturational threshold in anaerobic performance is arguably stronger than for aerobic parameters. Specifically, surrounding PHV, boys and girls experience a ten- and five-fold increase in circulating testosterone, respectively

(Fellmann & Coudert, 1994a), which has been demonstrated to be a potent stimulant for LBM accrual and muscular hypertrophy in children (Campbell & Mbizo, 2006; Fellmann & Coudert, 1994a; Thomsen et al., 1986; Vingren et al., 2010). Additionally, it is now accepted that the glycolytic energy system matures with age during childhood, facilitating a greater anaerobic capacity (Armstrong & Welsman, 2019b; Van Praagh, 2000; Van Praagh & Doré, 2002). The main driver behind the development of the glycolytic energy system during maturation is an increase in the number, and distribution of, type II muscle fibres (Fournier et al., 1982; Van Praagh, 2000). Additionally, the increase in muscle mass with maturity enhances the storage capacity for intramuscular adenosine triphosphate and creatine phosphate, two of the rate-limiting compounds in anaerobic glycolysis (Eriksson, 1972, 1980; Van Praagh, 2000). Given these marked changes associated with growth and maturation, it therefore seems plausible that a maturational threshold may be manifest in any response to exercise reliant on these systems.

Strength development during growth and maturation increases linearly in boys until the age of 12 – 13 years, after which a rapid development in explosive (i.e. jumping activities) and isokinetic (i.e. handgrip strength) strength is apparent in both the lower and upper extremities (Buenen & Thomis, 2000; Van Praagh, 2000; Van Praagh & Doré, 2002). Specifically, the greatest strength gains in boys are evident from – 1.5 to + 0.5 years PHV, suggestive of a strong maturational effect thought to be mediated by increases in androgenic sex hormones that facilitate muscular hypertrophy by enabling intramuscular protein synthesis (Buenen & Thomis, 2000; Rogol, 2002; Rogol et al., 2002; Van Praagh & Doré, 2002). Indeed, early-maturing boys have consistently been reported to outperform ‘normal’ and late-maturing boys in a variety of strength-based protocols, with maturity remaining a significant predictor of strength even after accounting for stature, body mass and age differences between participants (Buenen et al., 1998; Buenen & Thomis, 2000; Malina & Bouchard, 1991). Alongside developments in strength, peak power ( $P_{\text{peak}}$ ), the product of force and velocity (Armstrong & Welsman, 2019c), has also been shown to increase non-linearly, with periods of accelerated adaptation evident around the time of PHV (Armstrong & Welsman, 2019c; Doré et al., 2005; Martin et al., 2003). Specifically, Doré et al. (2005) reported a significant increase in  $P_{\text{peak}}$  from 14 years in boys, coinciding with



PHV and facilitated by a significantly greater lower leg muscle volume (LLMV) compared to pre-pubertal children, although changes in muscle metabolism may also have contributed (Armstrong, Barker, & McManus, 2015b; Armstrong & Barker, 2012b).

Given the potential maturational influence on strength performance in boys, resistance training interventions have been utilised to accentuate these changes, although resistance training still remains somewhat controversial in children and adolescents (Behringer, von Heede, & Mester, 2010). Resistance training is a type of exercise that requires the musculature to contract against an opposing force generated by some form of additional resistance (Behringer et al., 2010). Forty two studies were pooled in a meta-analysis by Behringer et al. (2010), demonstrating a large effect of resistance training on multiple strength parameters in pubertal and post-pubertal adolescents (effect sizes (ES) both 1.91). Interestingly, sub-analyses revealed that the effect size in pre-pubertal children was ~50% less for the same relative exercise stimulus (ES: 0.81). This suggests that pre-pubertal children are less trainable from resistance exercise than their more mature peers, supporting the maturational threshold hypothesis. Moreover, there were no significant trainability differences between boys and girls, irrespective of maturity, suggesting they are equally trainable, although this should be interpreted with caution given the scant literature on girls (Behringer et al., 2010).

Whilst there is limited evidence, research indicates that strength increases almost linearly until the age of 15 years after which it plateaus, with no empirical evidence for an adolescent growth spurt or 'boost' in performance (Behringer et al., 2010; Doré, Bedu, & Van Praagh, 2008; Doré et al., 2005; Van Praagh & Doré, 2002). In one of the only large scale studies examining sex differences, Doré et al. (2005) determined peak power ( $P_{\text{peak}}$ ) in 1,113 participants (583 girls) during a cycling Wingate (WnT) test. No sex differences were observed until the age of 13 years, after which  $P_{\text{peak}}$  was consistently higher in boys than girls. These sex differences persisted even after  $P_{\text{peak}}$  was allometrically scaled to LLMV and were attributed to differences in the number of, and the ability to recruit, type II muscle fibres. However, it is pertinent to note that the evidence regarding sex differences in muscle fibre types, beyond those associated with training, are contentious and sparse due to the highly-invasive nature of muscle

biopsies (Eriksson, 1980). Additionally, in a study of early, late and on-time maturing girls, there was no significant difference between any group for shoulder press or handgrip strength performance (Malina & Bouchard, 1991), further suggesting that the development of anaerobic performance may also be sexually dimorphic.

Potential reasons for the scarce research on anaerobic performance in children and adolescents may be a lack of ‘gold-standard’ measure and that anaerobic parameters are predominantly considered a performance measure, as opposed to a health-related outcome (McNarry & Jones, 2014; Van Praagh, 2000). The most common method of anaerobic performance assessment is the cycling WnT, which traditionally involves participants cycling maximally against a set resistance of 7.5% of their body mass for 30 s (Grodjinovsky, Inbar, Dotan, & Bar-Or, 1980). However, concerns have been raised regarding the optimal flywheel resistance needed to generate maximal power outputs (Doré et al., 2003; Watt et al., 2002), with numerous modifications utilised (Armstrong & Welsman, 2019b; Armstrong & Welsman, 2020c), which preclude direct inter-study comparisons (Hopkins et al., 2009; Watt et al., 2002). Perhaps the largest limitation of the cycling WnT test, however, is the large contribution of oxidative phosphorylation to adenosine triphosphate (ATP) resynthesis during the cycling WnT test in children and adolescents (Chia, 2006). Specifically, Chia et al. (2006) reported that aerobic metabolism contributed significantly to total energy metabolism (~70%) during a 30 s cycling WnT, questioning whether this is truly a test of ‘anaerobic’ performance.

Given the associated limitations with jumping test batteries and the cycling WnT test, over-ground sprint running has becoming an increasingly popular method of anaerobic performance assessment over the last decade (Abbasian, Gholamian, Attarzadeh, Khabazan, & Khodadadi, 2011; Gist, Fedewa, Dishman, & Cureton, 2013; Low, Harsley, Shaw, & Peart, 2015; Mendez-Villanueva et al., 2010; Meyers et al., 2015; Meyers et al., 2017a, 2017b; Mujika et al., 2009; Papaiakovou et al., 2009). Indeed, sprint performance is highly correlated to sports performance, particularly in team sports (Abbasian et al., 2011; Mujika et al., 2009; Papaiakovou et al., 2009), and is routinely assessed as part of training programs and as talent identification test batteries (Lloyd & Oliver, 2012; Lloyd et al., 2015; Unnithan, White, Georgiou, Iga, & Drust, 2012). Additionally, sprint performances are easily comparable between populations

and studies, are highly reliable (ICC: 0.96 - 0.99, CV: 0.7 - 1.9%; Ingle & Tolfrey, 2013; Runacres, Bezodis, Mackintosh, & McNarry, 2019a), and the simple data collection methods facilitate large-scale cross-sectional (Mendez-Villanueva et al., 2010; Meyers et al., 2015; Meyers et al., 2017a; Morin, Edouard, & Samozino, 2011) and longitudinal studies (Meyers et al., 2016). However, evidence regarding the determinants of sprint performance and its progression with respect to training, sex, and maturity remains in its infancy.

### *2.5.1 Over-ground sprint running*

Catley and Tomkinson (2013) reported 50 m sprint-performance reference ranges derived from a pooled sample of 85,347 Australian boys and girls aged 11-15 years, highlighting an almost linear increase with age. Such findings indicate a minimal influence of maturity on sprint performance. However, no insight was provided into the potential underlying mechanisms of sprint performance, which may change with age and maturity (Rumpf et al., 2015b; Rumpf, Cronin, Pinder, Oliver, & Hughes, 2012). Indeed, Meyers and colleagues (Meyers et al., 2015; Meyers et al., 2016, 2017a, 2017b) have investigated the development of sprint performance in boys, accounting for age, maturity and potential spatio-temporal determinants, namely stride frequency and step length. Maximal sprint speed was faster in pubertal adolescents compared to pre-pubertal children (Pre-Pubertal peak speed:  $\sim 6.3 \text{ m}\cdot\text{s}^{-1}$  vs  $6.7 \text{ m}\cdot\text{s}^{-1}$  in pubertal group; Meyers et al., 2015), with the increase in maximal sprint speed attributed to increases in stride length due to increases in leg length, thereby reducing the reliance on step frequency with age and maturity (Meyers et al., 2015). Stepwise linear regressions showed that step frequency explained  $\sim 58\%$  of the variance in maximal sprint speed in pre-pubertal boys, whereas step length explained the largest portion of variance in post-pubertal boys ( $\sim 54\%$ ; Meyers et al., 2017a). It is pertinent to note that stride length is highly dependent on leg length, thus if not normalised to stature, spurious associations may be observed that could mask the importance of stride length (Rumpf et al., 2015b). Nevertheless, when stride length was normalised, significant differences were still evident between pre-pubertal children, pubertal and post-pubertal adolescents (Rumpf et al., 2015b), potentially suggesting that other factors may also impact sprint performance during growth and maturation in boys.

The majority of research on spatio-temporal and kinematic sprint variables has focused exclusively on boys and thus the determinants of sprint performance in girls remain unclear (Nagahara et al., 2019; Papaiakovou et al., 2009; Vanderka & Kampmiller, 2013). In one of the few studies investigating sex differences in sprint performance, Papaiakovou et al. (2009) found that boys outperformed girls at all ages, with girls displaying a plateau in peak velocity ( $V_{\text{peak}}$ ) from the age of ~13 years. The results of Papaiakovou et al. (2009) are remarkably similar to those of other studies assessing the development of sprint performance in untrained girls, which similarly report a plateau in performance from the ages of 12.7 years (Nagahara et al., 2019) and 13.5 years (Vanderka & Kampmiller, 2013). In contrast, a plateau in  $V_{\text{peak}}$  is not observed in boys until the age of 15 years (Meyers et al., 2015; Meyers et al., 2017a). This later plateau in boys has been attributed to a greater increase in muscle size and cross-sectional area in boys, compared to girls, with concomitant neuromuscular adaptations facilitating increased recruitment of type II muscle fibres (Armstrong, 2007; Doré et al., 2005; Dotan et al., 2012; Van Praagh & Doré, 2002). However, it is noteworthy that the respective ages of 13 and 15 years in girls and boys coincide with the transition to post-pubertal status and, consequently, these observations could therefore be maturity related.

Given the potential maturational influence on sprint performance, training during this time could be postulated to accelerate the natural increases in sprint performance. Rumpf et al. (2015a) observed a significant reduction in sprint times after six weeks of resistance training in mid- to post-pubertal, but not pre-pubertal, children. This increased velocity was associated with an increase of 15.2% and 14.1% in  $P_{\text{peak}}$  and peak force ( $F_{\text{peak}}$ ), respectively. More recently, Cahill et al. (2020) found that greater training-related gains in 20 m sprint time (20mT) were observed in a heavy-resistance training group (enough load to elicit a 75% reduction in  $V_{\text{peak}}$ ) compared to a moderate (50%  $V_{\text{max}}$  decrement), light (25%  $V_{\text{max}}$  decrement) or an unresisted training group, irrespective of maturational status. Whilst these findings appear to indicate that a greater stimulus is necessary to significantly improve 20mT, irrespective of maturity, it is worth highlighting that neither of these studies specifically explored the effect of maturity, with the pooling of participants in Rumpf et al. (2015a) and the omission of a circa-pubertal group in Cahill et al. (2020) precluding further interpretation.

Moreover, neither study considered the effect of training in girls and thus the potential interaction of sex with training and maturity on the anaerobic response to training in youth remains to be elucidated.

Rumpf et al. (2012) conducted a review into the most effective training methods to improve maximal sprint speed, concluding similar performance gains were elicited in pre- and post-pubertal boys irrespective of training methodology. In contrast, the magnitude of training gains were ~50% lower in pubertal compared to pre- or post-pubertal adolescents, which the authors suggested was likely to reflect the phenomenon of adolescent awkwardness (Buenen et al., 1998; Lloyd et al., 2015). Adolescent awkwardness is a period of plateaus, or declines, in performance attributed to temporary disruptions in motor co-ordination and neural pathways (Buenen et al., 1998). Whilst the mechanisms underpinning adolescent awkwardness remain to be fully elucidated, differences in segmental growth rates in relation to the trunk seem likely to explain at least some of the variance (Buenen et al., 1998; Rumpf et al., 2015b). Other researchers argue that impairments in motor co-ordination during adolescence are a consequence of impaired and/or delayed sensorimotor function, including neurocognitive processing and the regulation of postural control (Quatman-Yates, Quatman, Meszaros, Paterno, & Hewett, 2012). Nevertheless, it seems plausible that it is a combination of both these hypotheses, with differing growth rates causing sensorimotor impairment, but a causal relationship is yet to be established. However, adolescent awkwardness is only reported in some (Buenen et al., 1998; Papaiakevou et al., 2009; Rumpf et al., 2012) and not all (Abbasian et al., 2011; Mendez-Villanueva et al., 2010; Meyers et al., 2016, 2017a; Philippaerts et al., 2006) studies examining sprint performance, and thus more research is warranted.

The majority of research to date has only considered maximal sprint speed, thereby providing little insight into the underlying kinetics (i.e. force and power) of sprint performance; understanding the kinetic determinants of sprint performance may enhance our understanding of the development of sprint performance with training, and the effects of sex and maturity. One possible reason for the lack of clarity on the underlying kinetics of sprint performance is the poor measurement resolution in field-based methodologies (i.e. photocells and timing gates). Studies utilising these methodologies have relied on averaging over sections of, if not the whole, sprint

(Fitzsimons, Dawson, Ward, & Wilkinson, 1993; Mendez-Villanueva et al., 2010; Meyers et al., 2015; Papaiakevou et al., 2009). This lack of measurement resolution has increased the reliance on laboratory-based assessments, including non-motorised treadmills (Rumpf et al., 2015a; Rumpf et al., 2013; Rumpf et al., 2015b) and force platform (Nagahara et al., 2019) derived data, which have questionable ecological validity and applicability (Turley, Rogers, Harper, Kujawa, & Wilmore, 1995).

Recent developments in technology, in combination with advances in macroscopic biomechanical models (Samozino et al., 2016), now enable near-instantaneous quantification of the velocity-time trace from which the underlying kinetics can be modelled (Rossi et al., 2017; Samozino et al., 2016). This enhanced measurement resolution, coupled with simple field-based data collection methods, potentially facilitates large cohort studies. Therefore, the combination of both these methodologies has the ability to enhance our understanding of the underlying kinetics, and the interactive effects of sex, training and maturation on sprint development. Indeed, Rossi et al. (2017), the only study utilising the combination of both these methods in children and adolescents to date, reported that  $P_{\text{peak}}$ , and 30 m time increases with age, but  $F_{\text{peak}}$  was similar between children and adolescents. Interestingly, however, the mechanical efficiency of force application ( $D_{\text{RF}}$ ) was significantly improved in adolescents compared to children, and is a greater predictor of sprint performance than total force production in adults (Morin et al., 2011). However, the lack of maturity assessment, the pooling of boys and girls despite their distinctly different physiology, and the ambiguous quantification of training, precludes further interpretations. Nevertheless, Rossi et al. (2017) demonstrates the potential of these methods to enhance our understanding of sprint development with respect to training, sex and maturity and warrants further investigation.

### *2.5.2 Repeated Sprint Performance*

Whilst singular, over-ground sprints are reliable (Runacres et al., 2019a) and familiar to youth athletes, some researchers argue that repeated sprint ability is more indicative of real-world match-play performances, especially in team sports (Mendez-Villanueva et al., 2010; Mujika et al., 2009; Philippaerts et al., 2006; Spencer, Pyne, & Mujika, 2011). Moreover, repeated sprint performance provides the ability to explore the determinants of fatigue which is not possible from a single sprint.

When repeated sprint ability (RSA) was assessed in a sample of 61 highly-trained footballers over ten 30 m sprints, interspersed with 30 s passive recovery, the mean sprint time significantly decreased with advancing age (Mendez-Villanueva et al., 2010). Sub-analyses revealed that there was a similar magnitude of change between age-groups, indicating that RSA may increase in a near-linear fashion with age, in direct contrast to single-sprint performance (Mendez-Villanueva et al., 2010). This conclusion is supported by the findings of Spencer et al. (2011) who used six, 30 m sprints in 119 highly-trained footballers and found that total sprint time decreased from  $33.1 \pm 1.8$  s to  $28.7 \pm 0.6$  s and  $26.2 \pm 0.8$  s in children under 11, 14 and 18 years, respectively. It must be noted however, that the absence of a corresponding control group in Mendez-Villanueva et al. (2010) or Spencer et al. (2011) means it is not possible to discern whether the observed increases in RSA are specifically training related or may reflect a concomitant effect of growth and maturation.

Philippaerts et al. (2006) reported that the mean yearly improvements in RSA in trained footballers deemed pre- and post-pubertal were 0.1 s and 0.2 s per year, respectively, whereas the mean improvement for pubertal footballers was 0.9 s. Interestingly, whilst Philippaerts et al. (2006) reported a greater magnitude of change in pubertal adolescents, Mujika et al. (2009) found that the decrement in performance over repeated sprints, quantified by fatigue index (FI), remained relatively constant (4.0 – 5.5%). Nonetheless, the findings of Mujika et al. (2009) should be interpreted with caution as FI has repeatedly demonstrated moderate-to-poor reliability (CV: 20 – 30%; Girard, Mendez-Villanueva, & Bishop, 2011; Spencer, Fitzsimons, Dawson, Bishop, & Goodman, 2006). This is perhaps unsurprising given the commonly applied method for quantifying performance decrements in is  $((\text{total sprint time} / \text{ideal sprint time}) \times 100) - 100$ , which offers, at best, a limited insight into the magnitude of fatigue during repeated sprint efforts (Mujika et al., 2009; Spencer et al., 2006; Spencer et al., 2011). Indeed, the FI provides no indication as to the changes in the underlying kinetics that lead to these performance decrements which therefore precludes specific interventions being implemented to improve RSA in youth athletes. However, there is no available literature examining the RSA of girls, and so the effect of sex on training, and maturity, remain unclear. Therefore, due to this lack of clarity, the underlying

mechanisms contributing to fatigue during repeated bouts of over-ground sprints, and how this may differ by training, sex, and maturity status, remain unexplored.

## **2.6 Long-term consequences of training**

Whilst the immediate adaptations to training stimuli in children are starting to be elucidated, one area which has received little attention is the long-term effect of intensive training in childhood on adult health. Nonetheless, the scarce literature generally denotes a linear dose-response relationship between exercise and future health (Attard, Hering, Howard, & Gorden-Larsen, 2013; Mika & Fleshner, 2016; Riner & Sellhorst, 2013). Conversely, others have reported that the effect of training during childhood on adult health is weak, with the maintenance of physical fitness a stronger predictor than prior training history (Hasselstrøm, Hansen, Froberg, & Andersen, 2002). With conflicting evidence in children it may be prudent to examine the literature on the long-term health consequences of an elite sporting career. Indeed, many elite athletes engage in intensive training from very young ages (Gonzalez, Johnson, Fedoruk, Posner, & Bowers, 2018; Green, 2006; Williams, 2016), and, with careers spanning over 40 years in some cases they may be a more appropriate population to study long-term impact. Given that the aim of LTAD programmes is to prepare young athletes for an elite sporting career, understanding the consequences of these exercise behaviours is of paramount importance for coaches, NGB's, and policy makers alike.

### *2.6.1 Adult Literature*

Traditionally, the dose-response relationship between exercise and all-cause mortality was assumed to be linear in adults, with higher levels of exercise associated with a more favourable all-cause, CVD and cancer mortality risk (Blair et al., 1989; Lee et al., 1995; Paffenbarger & Lee, 1998). Indeed, in 17,815 Harvard alumni tracked for an average of 15 years, it was found that former college athletes were less likely to die from CVD before the age of 50 years compared to their previously sedentary counterparts (Blair et al., 1989; Lee et al., 1995; Paffenbarger & Lee, 1998). Moreover, Harvard alumni who engaged in 'regular exercise' and expended  $\geq 2,000$  active calories a week gained a 1.5-year survival benefit by the age of 80 years and a 25%



reduction in CVD incidence (Blair et al., 1989; Lee et al., 1995; Paffenbarger & Lee, 1998).

More recent epidemiological studies have challenged the findings of the Harvard alumni studies, suggesting there may be an upper limit beyond which exercise and physical activity become deleterious to health (M. Armstrong et al., 2015a; Mohlenkamp, Lehman, & Breuckmann, 2008; Schnohr et al., 2015). Specifically, the Copenhagen Heart study, in which 1,098 joggers and 3,950 non-joggers were followed over 12 years ( $48.8 \pm 13.8$  years at baseline), reported that strenuous joggers were at a two-fold higher risk of all-cause mortality compared to non-joggers (Hazard Ratio: 1.97, 95% Confidence Intervals (CI): 0.48 – 8.14), with these findings further exaggerated when expressed in relation to light joggers (Hazard Ratio: 9.08 95% CI: 1.87 – 44.01; Schnohr et al., 2015). However, it is pertinent to note that the small number of deaths in both groups (28 joggers, 128 non-joggers) may have skewed the associations reported. Nonetheless, the results of the Copenhagen Heart Study (2015) were corroborated by a large prospective study of 1.1 million females ( $55.8 \pm 4.7$  years) in the UK who were tracked for 9 years. Specifically, Armstrong et al. (2015a) reported that women who engaged in daily strenuous (exercise intensity  $\geq 6$  METs) exercise had no significant reduction in the incidence of coronary heart disease (Risk Ratio (RR): 0.89, 95% CI: 0.84-0.93) when compared to women who rarely or never engaged in exercise, even after accounting for BMI, smoking, alcohol intake, and socioeconomic status. Furthermore, recreational German marathon runners aged  $57.2 \pm 5.7$  years followed over a two-year period had a similar incidence of cardiovascular (CV) events compared to a group with established coronary heart disease (Mohlenkamp et al., 2008). Taken together, these studies suggest that the exercise-health relationship may be 'J' shaped, with exercise intensities, and volumes, above a certain threshold becoming deleterious to health. It is therefore potentially important to consider that elite athletes generally train at levels far in excess of those reported in epidemiological studies, thereby raising the question as to the effects of such volumes and/or intensities of exercise on longevity in former athletes.

Whilst there is extensive research regarding the health consequences of an elite sporting career, two systematic reviews (Lemez & Baker, 2015; Teramoto & Bungum, 2010a) and a meta-analysis (Garatachea et al., 2014) have sought to assimilate this

evidence. Collectively, these reviews conclude that elite male athletes live longer than the general male population and have a lower incidence of both CVD and cancer mortality (Garatachea et al., 2014; Lemez & Baker, 2015; Teramoto & Bungum, 2010a). However, there is a paucity of literature in former female athletes, precluding inferences as to whether males and females experience similar long-term benefits. Moreover, neither of the reviews (Lemez & Baker, 2015; Teramoto & Bungum, 2010a) sought to elucidate the potential influence of sport type on mortality differences. Sport type may moderate the relationship between intensive training and long-term health outcomes for many reasons, not least the considerable inter-sport differences in the type and volume of training, and the physiological and anthropometric characteristics associated with success (Castanheira et al., 2017; McKendry, Breen, Shad, & Greig, 2018; Venckunas, Simonavicius, & Marcinkeviciene, 2016; Williams, 2016). For example, there are considerable differences in physique according to sport, with power sports generally associated with a higher BMI, which is independently related to an increased risk of CVD and all-cause mortality (Baron, Hein, Lehman, & Gersic, 2012; Keller, 2019; U. M. Kujala, Kaprio, Taimela, & Sarna, 1994b; Lehman, Hein, Baron, & Gersic, 2012; Wang et al., 1994). Indeed, American footballers who had a playing BMI of  $\geq 30 \text{ kg}\cdot\text{m}^{-2}$  were reported to be at a two-fold higher risk of CVD mortality compared to those players with a BMI of  $\leq 25 \text{ kg}\cdot\text{m}^{-2}$  (Baron et al., 2012). Nevertheless, it is pertinent to note that the literature reporting inter-sport health outcomes is inconsistent, with some studies reporting no differences (Kettunen et al., 2015; Kontro, Sarna, Kaprio, & Kujala, 2018) and others only reporting a survival benefit up to a certain age (Gajda, Smigielski, Smigielski, Pakos, & Drygas, 2018; Mackay et al., 2019; Schnohr, 1971a). Determining the long-term health consequences of intensive exercise training is of paramount importance given the increased sports participation in youth (Sport England, 2019; Sport Wales, 2018). Such findings can not only inform training schedules and contribute to ongoing debate surrounding early specialisation (Mostafavifar, Best, & Myer, 2012; Till et al., 2019; Williams, 2016) but also inform strategies to enhance the long-term health of those athletes.

## 2.7 Overall Conclusions

Overall, fundamental questions persist regarding the influence of sex, maturity, and training status and how they interact to improve performance parameters in both the short- and long-term. Moreover, the effect of PA, SED, and sleep on  $\dot{V}O_{2\max}$  still remains to be elucidated, with the majority of the literature confounded by methodological and statistical inconsistencies unable to account for the constrained, and co-dependent, nature of PA data.

Evidence suggests that both training and maturation independently increase aerobic and anaerobic parameters during adolescence, but, after appropriate scaling techniques are employed, pre-pubertal children demonstrate similar levels of aerobic and anaerobic trainability to pubertal and post-pubertal adolescents, refuting the maturational threshold hypothesis. However, what remains less clear is the mediating effect of sex on the maturity and training interaction during childhood and adolescence. Physical activity levels are unequivocally related to an improved health and quality of life in all populations, however, the relationship between physical activity and  $\dot{V}O_{2\max}$  remains contentious despite being two of the most researched topics in paediatric exercise science. This could be due to the reliance on correlations, which only infer causality, and use of inappropriate predictive modelling techniques, which fail to appropriately account for the high co-dependency between movement activity behaviours. Compositional analyses have the ability to overcome these limitations and provide new insights into the relationships between PA, SED, and sleep on  $\dot{V}O_{2\max}$  in trained and untrained youth. Additionally, there is a clear lack of understanding on the sport-specific long-term effects of chronic intensive training, especially in women, which needs addressing urgently so interventions can be implemented, if necessary, to improve the long-term health of former elite athletes.

# **Chapter 3**

## **Methodology**

## **Chapter 3 – General Methodology**

The experimental investigations within this thesis comprise of five experimental chapters. **Chapters 4 and 5** were comprised of the same  $\dot{V}O_{2max}$  and seven-day physical activity (PA) data. Data collection took place either in the Swansea University Applied Sports Technology Exercise and Medicine (A-STEM) research laboratories or at athlete training sessions at various locations across Wales. Prior to any contact with potential participants, all studies were approved by the institutional ethics committee at Swansea University and all experimental procedures were conducted in accord with the Declaration of Helsinki.

### **3.1 Participants**

The trained group in **Chapters 4, 5, 6 and 7** was comprised of national standard athletes involved within long-term athlete development (LTAD) programs overseen by their sport's national governing body (NGB). All trained participants were recruited through the NGB sending emails with participants and parent information sheets to all national squad players and parents with interested parents and athletes then contacting the research team. The trained group was formed of Hockey players, Gymnasts, Cyclists, and Soccer players. All athletes were training an average of  $10 \pm 2$  hours $\cdot$ week $^{-1}$  over four sessions and competed in a competition/match most weekends throughout the competitive season. More specifically, the hockey and football players predominantly engaged in a mixture of endurance and high intensity interval training prior to the commencement of sessions and moving into small-sided games and/or tactical-awareness elements. The gymnasts were all  $< 10$  years old and training in excess of 16 hours $\cdot$ week $^{-1}$  which involved a mixture of strength, technique, and conditioning sessions. Finally, the cyclists and triathletes were engaged in endurance-based training sessions 3 days $\cdot$ week $^{-1}$ , with the fourth day consisting of high intensity repetitions ( $< 30$  s) with periods of active recovery. All participants had been training for an average of  $3.5 \pm 1.5$  years before entry into the studies. Participants were recruited via emails circulated by the NGB inviting the young athletes to be involved within the respective study. Interested parents/guardians and participants were subsequently sent respective information sheets. The inclusion criteria for the trained group were i) part of a national training group, ii) were training in excess of 8

hours·week<sup>-1</sup> and iii) had been training for at least a year. Trained participants were excluded if they had any injury or illness or they had been in the NGB set up for less than a year.

All untrained participants were recruited from local schools across South Wales, or through the university, and were not regularly physically active. Control participants were recruited through local schools through letters being distributed to all school pupils, with control participants also recruited via university emails and word of mouth. The inclusion for control group participants were i) were not habitually active and ii) were aged between 8-18 years old. Control participants were excluded from the study if they i) were part of any formal training regime and ii) they had a pre-existing injury or illness which would stop them from any part of the study. The breakdown of all participants across these four experimental chapters is displayed in Table 3.1.

**Table 3.1** – Distribution of participants included within **Chapters 4, 5, 6 and 7**

	<b>Total Number Recruited per Group</b>	<b>Chapter 4 (n)</b>	<b>Chapter 5 (n)</b>	<b>Chapter 6 (n)</b>	<b>Chapter 7 (n)</b>
All athlete data was collected during 2017-2020 at regular intervals throughout the year	Hockey Players (n = 147)	56	56	147	20
	Football Players (n = 42)	42	42	-	-
	Triathletes (n = 4)	4	4	-	-
	Welsh Gymnasts (n = 3)	3	3	-	-
	Welsh Cyclist (n = 5)	5	5	-	-
All control data was collected during 2018-2019 in line with athlete time- points	Controls - GCSE PE Students (n = 47)	41	47	-	-
	Controls - Other (n = 113)	40	83	113	-

n = Number of participants recruited into each chapter before exclusion criteria were applied.

Prior to participants being accepted into the studies, parent/guardian consent (for those under 16 years) and child assent or participant consent (those over 16 years) were obtained and a pre-screening medical questionnaire completed. Parent/guardian consent was obtained either online (using a custom-built consent form on Survey Monkey, **Chapters 6 and 7**) or in person prior to data collection (**Chapters 4 and 5**). Participants were excluded from the experimental studies within this thesis if they had any known cardiovascular, metabolic or kidney disease, or any other condition that would have prevented them from completing all aspects of the experimental protocol.

Written participant assent was obtained on the day of testing prior to data collection. Participants were asked to arrive to all testing sessions having refrained from performing strenuous exercise in the preceding 24 hours, having had no caffeine in the past 12 hours, and having not eaten a large meal in the last two hours. These criteria were implemented to standardise experimental procedures across studies.

### 3.2 Experimental Procedures

#### *3.2.1 Anthropometric Measurements*

Prior to any other measures being taken, all anthropometric variables were assessed. The participant's stature was measured with shoes off using a Holtain Stadiometer (Holtain, Crymych, Dyfed, UK) to the nearest 0.1 cm. The participant was instructed to stand tall, keep the chin level and look straight ahead. Sitting stature was measured to the nearest 0.1 cm using a Harpenden sitting height table (Holtain, Crymych, Dyfed, UK), adjusted to ensure a 90° flexion of the knee with feet rested on the foot bar. Participants were encouraged to sit up straight, with the chin flat looking straight ahead, and a measure of sitting height recorded after the participant had inhaled and exhaled forcefully. If a sitting height table was not available (i.e. in field testing), sitting height was measured using a portable Holtain stadiometer (Holtain Ltd, Crymych, Dyfed, Wales). Body mass was recorded using electronic scales (Seca 803, Seca, Chino, CA, USA), accurate to the nearest 0.1 kg. Body mass was recorded bare foot and with all heavy items removed from pockets and any excess clothing (i.e. tracksuit trousers) removed. Additionally, date of birth was collected, with decimal age calculated from the day of the assessment.

Given the importance of accounting for sexual maturation within this thesis several different options were considered. A popular method of assessing maturity within children and adolescents is the tanner stages of pubic hair and development of the sexual organs (Marshall & Tanner, 1969, 1970). Tanner stages have been shown to be both valid and reliable in assessing young people's maturity, with Leone and Comtois (2007) reporting high reliability and validity in a group of elite junior athletes (spearman rank  $\geq 0.86$ ,  $p < 0.05$ ;  $\geq 75\%$  agreement with a qualified physician). However, some researchers have questioned the applicability of this method given that these pubertal indices were created from longitudinal data in Caucasian children in the

1950's and therefore may not be representative of today's population or other ethnicities (Coleman & Coleman, 2002). Bone age, and thus skeletal age, is another, less common and more invasive method of maturity assessment which involves the bones of the hand and wrist being measured using dual x-ray absorptiometry (DEXA) scans. The two most common methods are the Greulich-Pyle (GP; Greulich & Pyle, 1959) method and the Tanner-Whitehouse 2 (TW2; Tanner et al., 1983) assessments. However, each of these equations are associated with certain limitations and, irrespective of the equation used, the standard error of estimate (SEE) is approximately one year for girls from 5 – 14 years and boys from 5 – 16 years. Additionally, the radiation exposure rendered DEXA scans for the assessment of maturity in healthy children unfeasible. Consequently, maturity stage was estimated using the anthropometric-based, sex-specific maturity offset equations of Mirwald et al. (2002b), which predict the time in years a child is from their peak height velocity (PHV), to the nearest 0.1 years. The sex-specific maturity offset equations are as follows:

$$\begin{aligned} \text{Boys Maturity Offset (years)} = & -9.236 + (0.0002708 \cdot (LL \cdot SH)) - \\ & (0.001663 \cdot (AG \cdot LL)) + (0.007216 \cdot (AG \cdot SH)) + (0.2292 \cdot (WT \div HT)) \quad (1) \end{aligned}$$

$$\begin{aligned} \text{Girls Maturity Offset (years)} = & -9.376 + (0.0001882 \cdot (LL \cdot SH)) + \\ & (0.0022 \cdot (AG \cdot LL)) + (0.005841 \cdot (AG \cdot SH)) - (0.002658 \cdot (AG \cdot WT)) + \\ & (0.07693 \cdot (WT \div HT)) \quad (2) \end{aligned}$$

with

$$LL = HT - SH \quad (3)$$

where *LL* is leg length (cm), *SH* is sitting height (cm), *AG* is decimal age, *WT* is body mass (kg) and *HT* is height (cm). Participants were subsequently classified as pre-PHV if more than one-year away from PHV, circa-PHV if within a year of PHV and post-PHV if more than one-year post PHV. It is pertinent to note that both sex-specific equations have a standard error (SE) of  $\pm 0.59$  years and  $\pm 0.57$  years for boys and girls, respectively (R. L. Mirwald et al., 2002b).



### 3.2 $\dot{V}O_{2max}$ Assessment

$\dot{V}O_{2max}$  was measured using an incremental ramp test to volitional exhaustion using a Lode cycle ergometer (Lode Excalibur Sport, Groningen, Netherlands) in **Chapters 4 and 7**. Specifically, following a three-minute warm-up at 10 watts (W), the work rate then increased by 20 – 25  $W \cdot \text{min}^{-1}$ , depending on the participant's age ( $\leq 11$  years = 20  $W \cdot \text{min}^{-1}$ ,  $\geq 11.1$  years = 25  $W \cdot \text{min}^{-1}$ ). All participants were instructed to maintain a cadence between 60 and 80 revolutions per minute (rpm), with volitional exhaustion defined as when the cadence consistently fell below 50 rpm, despite strong verbal encouragement.

Exercise modality significantly influences peak  $\dot{V}O_2$  achieved, with a higher peak  $\dot{V}O_2$  in children and adolescents reported using a treadmill when compared to cycle ergometry (Loftin, Sothorn, Warren, & Udall, 2004; Turley et al., 1995). Nevertheless, Loftin et al. (2004) reported peak  $\dot{V}O_2$  derived from cycle ergometry to be equally reliable to that derived from a treadmill (intraclass correlation coefficient 0.98 versus 0.97, respectively), and Turley et al. (1995) reported a higher reliability, and less variability, using a stationary bike peak  $\dot{V}O_2$  protocol, in comparison to a treadmill equivalent (coefficient of variation 4.4% versus 5.6%, respectively;  $p < 0.05$ ). Therefore, given the lower upper body movement artefact associated with cycle exercise, this was deemed the most appropriate modality.

Inspired and expired air were collected on a breath-by-breath basis throughout the incremental ramp protocol using a Vyntus metabolic cart (VYAIRES medical Ltd, Mettawa, IL, USA). It is pertinent to note that Kantaras (2018) reported a high standard error of measurement (SEM) for  $\dot{V}O_2$  and carbon dioxide production ( $\dot{V}CO_2$ ) values both equating to 1.3%, with questionable test-retest reliability also reported (ICC: 0.70 – 0.80). However, the assumption of normality, fundamental to the calculation of SEM, was violated, and therefore these findings should be interpreted with caution. To mitigate any potential reliability issues, the volume calibration was re-run after every test, with gas calibration run every four hours, in line with manufacturer recommendations. Furthermore, the reliability of the Vyntus gas analyser is superior to other commercially available models, including the MetaMax (CV  $\geq 2.8\%$ ;

Macfarlane & Wong, 2012) and the Oxycon mobile ( $CV \geq 4.1\%$ ; Rosdahl, Gullstrand, Sailer-Eriksson, Johansson, & Schantz, 2010).

A  $\dot{V}O_{2max}$  was accepted to reflect a maximal effort when there was a visible  $\dot{V}O_2$  plateau ( $< 150 \text{ ml}\cdot\text{min}^{-1}$  increase in  $\dot{V}O_2$  despite an increasing work rate; Rowland, Lee, & Cunningham, 1992). Heart rate (HR) and the respiratory exchange ratio (RER) were monitored, but not used to determine a maximal effort due to the issues highlighted by Barker et al. (2009). More specifically, reliance on secondary criteria to determine a maximal effort in paediatric populations can underestimate  $\dot{V}O_{2max}$  by as much as 20%, if they are reached at all (Barker et al., 2009). Therefore, after five minutes of active recovery and 10 minutes of passive rest, a supramaximal validation bout was performed to verify whether a maximal effort had been provided. Workloads ranging from 105-130% of peak power have previously been used for supramaximal bouts, though no significant differences have been reported to be elicited by different supramaximal protocols (Schaun, 2017). In accord with the majority of previous studies, a step-transition to 105% of peak power was utilised in the current studies (Barker et al., 2009; Poole & Jones, 2017; Rossiter, Kowalchuk, & Whipp, 1985). Breath-by-breath gas exchange variables were measured throughout the supramaximal validation bout, with participants instructed to maintain a cadence of  $\geq 50 \text{ rpm}$  for as long as possible.  $\dot{V}O_{2max}$  was validated if there was  $\leq 2.5\%$  difference between the maximum 10s stationary  $\dot{V}O_2$  recorded between the incremental ramp test and the supramaximal validation bout (Figure 3.1). In situations where there was a  $\geq 2.5\%$  difference the participants were excluded from further analysis.

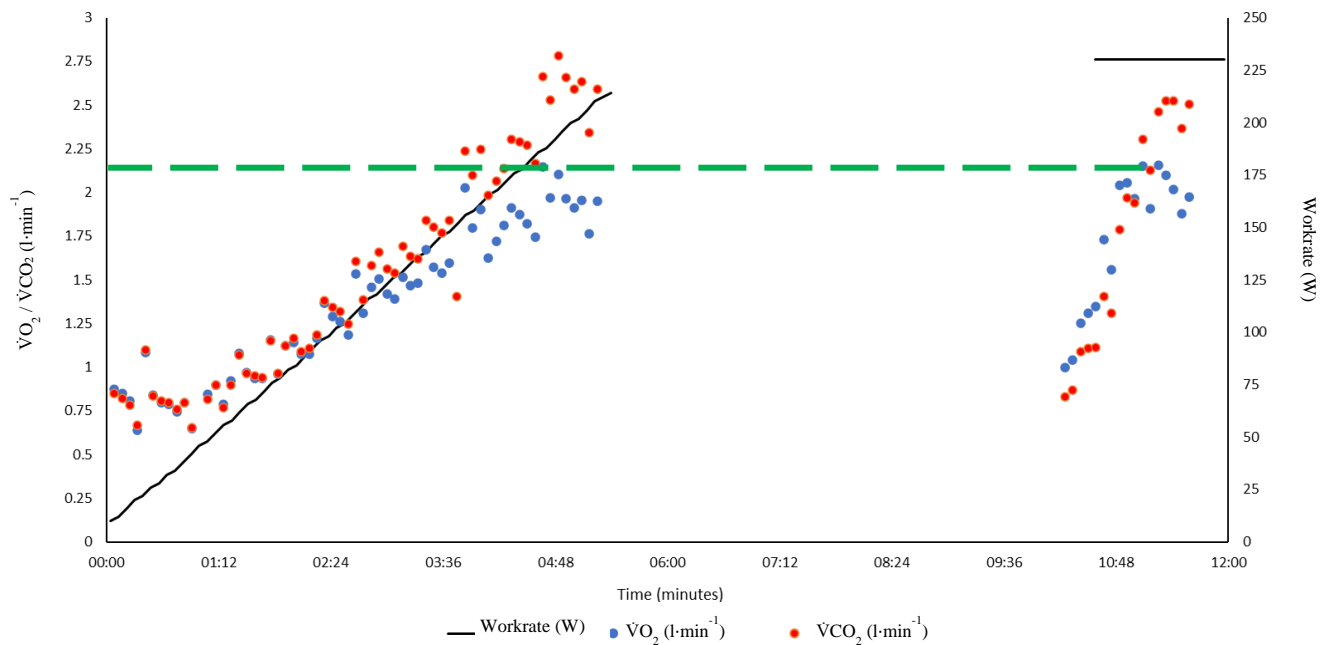


Figure 3.1: Schematic diagram for the determination of  $\dot{V}O_{2max}$  from an incremental ramp test and the supramaximal validation bout. If the difference in  $\dot{V}O_2$  values between the incremental test and supramaximal validation bout was  $< 2.5\%$  then a 'true'  $\dot{V}O_{2max}$  was obtained.

To aid comparisons between training, maturity, and sex groups,  $\dot{V}O_{2max}$  was expressed relative to body mass. Specifically,  $\dot{V}O_{2max}$  underwent two forms of scaling, ratio ( $ml \cdot kg \cdot min^{-1}$ ; equation 4) and allometric ( $ml \cdot kg^{-b} \cdot min^{-1}$ ; equation 5). Allometric scaling is considered more robust than traditional ratio scaling, which assumes a linear relationship between body mass and  $\dot{V}O_{2max}$ , thereby potentially creating spurious associations (Nevill et al., 2006; Tanner, 1949; Welsman & Armstrong, 2019). Moreover, a negative association may still exist between body mass and  $\dot{V}O_{2max}$  following ratio scaling, with heavier, more mature individuals often unfairly penalised (Cunha et al., 2011; Nevill et al., 2006; Nevill et al., 1998), which may mask physiologically meaningful findings. Conversely, allometric scaling calculates a population-specific exponent, removing any association with body mass, and subsequently enabling inter-population comparisons (Cunha et al., 2011; Cunha et al., 2016; McNarry et al., 2014b; Nevill et al., 2006; Nevill et al., 1998; Runacres et al., 2019b). Allometrically scaled  $\dot{V}O_{2max}$  was determined by log transforming body mass (kg) and peak  $\dot{V}O_2$  ( $ml \cdot min^{-1}$ ). Subsequently, a log-linear regression model was generated, and the resulting beta coefficients used as allometric exponents ( $b$ ) when the data was back-transformed into original units.

$$\text{Ratio Scaled } \dot{V}O_{2max} (ml \cdot kg \cdot min^{-1}) = VO_{2max} (ml \cdot min^{-1}) \div BM \quad (4)$$

$$\text{Allometrically Scaled } \dot{V}O_{2max} (ml \cdot kg^{-b} \cdot min^{-1}) = VO_{2max} (ml \cdot min^{-1}) \div BM^b \quad (5)$$

where *BM* is the body mass of the participant and *b* is the scaling coefficient derived from the logarithmic linear regression. To check that the *b* coefficient successfully controlled for the effect of body mass, the data went through two quality check phases. Firstly, the data was graphed to visually inspect the relationship (Figure 3.2) and secondly Pearson's correlations were run to ensure that the relationship between body mass and  $\dot{V}O_{2max}$  were no longer significant.

The gas exchange threshold (GET), defined as the point where  $\dot{V}CO_2$  begins to rise disproportionately to  $\dot{V}O_2$ , was calculated using the V-slope method first described by Beaver, Wasserman and Whipp (1986). The raw  $\dot{V}O_2$  and  $\dot{V}CO_2$  data were averaged into 10-second bins prior to any analysis taking place. The V-slope method uses basic linear regression analysis techniques to identify the point at which  $\dot{V}CO_2$  begins to increase exponentially compared to  $\dot{V}O_2$  (Figure 3.3). This rise in  $\dot{V}CO_2$  is triggered by the buffering of hydrogen ( $H^+$ ) ions as a consequence of incomplete glycolysis (Meyer, Lucia, Earnest, & Kindermann, 2005; Spurway, 1992), thus the GET can provide valuable information, non-invasively, on the sub-maximal threshold associated with the transition from moderate- to heavy-intensity exercise. Whilst the gas exchange and lactate thresholds are two well recognised physiological parameters, there remains debate as to whether they are equivalent (Hebestreit, Staschen, & Hebestreit, 2000; Mucci et al., 2013; Pfitzinger & Freedson, 1997; Ratel & Martin, 2012). To aid comparisons between populations, the relative GET was also calculated by expressing the GET as a percentage of  $\dot{V}O_{2max}$ .

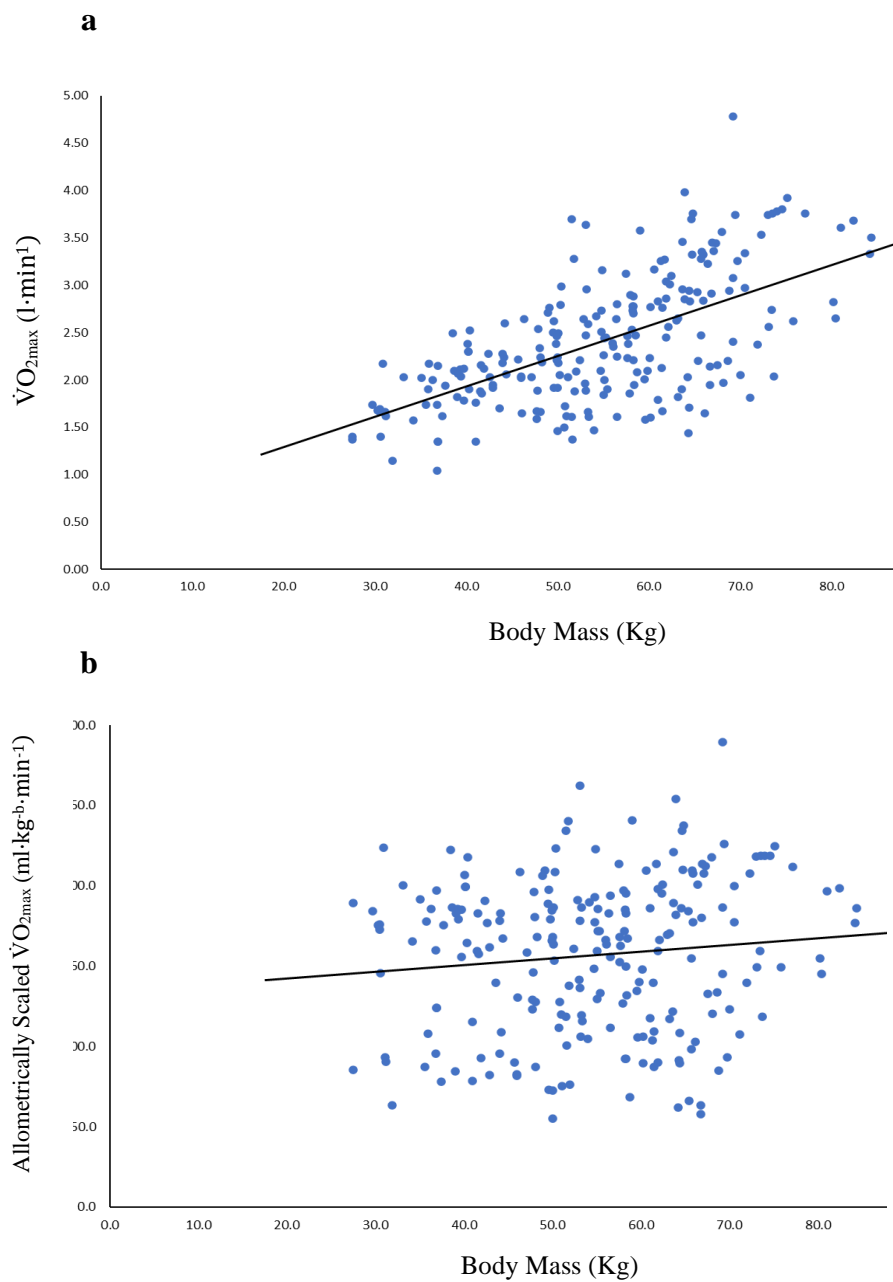


Figure 3.2 The relationship between a)  $\dot{V}O_{2\max}$  and body mass and b) allometrically scaled  $\dot{V}O_{2\max}$  and body mass

The mean response time (MRT) was calculated from the incremental ramp test using the methods described by Barstow and colleagues (Barstow, Jones, Nguyen, & Casaburi, 1999) to estimate the kinetic  $\dot{V}O_2$  response to exercise. Mean response time was defined as the point of intersection between baseline  $\dot{V}O_2$  (average of the final minute of the warm-up) and a backwards extrapolation of the  $\dot{V}O_2$  – time (s) slope from one minute into the ramp-forcing function to steady state  $\dot{V}O_2$  (Figure 3.3b). The

response gain was also calculated according to the average change in  $\dot{V}O_2$  per Watt increase from one minute into the ramp-forcing function to the GET (s1), and from the GET to  $\dot{V}O_{2max}$  (s2), and across the whole incremental ramp slope (sT).

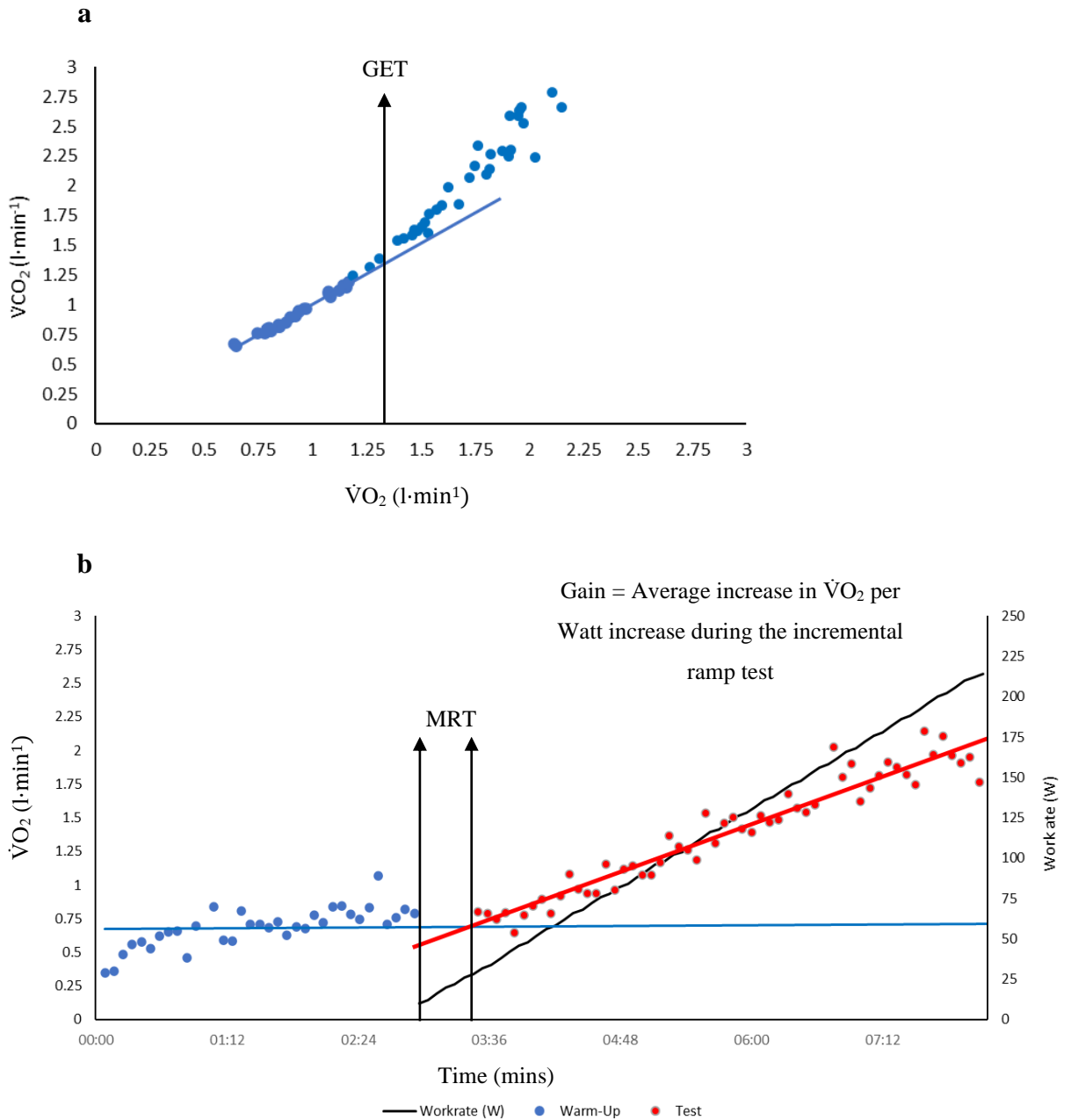


Figure 3.3 The calculation of a) the V-Slope method used to determine the gas exchange threshold (GET) during the incremental ramp test and b) The calculation of the mean response time (MRT) and Gain

### 3.2.3 Haemodynamic Parameters

Haemodynamic parameters were assessed to gain an insight into the functional properties of the myocardium and whether these were affected by training status, sex or maturity in **Chapter 4**. Whilst haemodynamic parameters have been suggested to positively correlate with  $\dot{V}O_{2\max}$  (Armstrong, 2007; Armstrong & McNarry, 2016; Logan, Harris, Duncan, & Schofield, 2014), the mechanisms underpinning changes in peak  $\dot{V}O_2$  remain poorly understood in children and adolescents. Traditionally, electrocardiograms (ECG) were considered the most reliable method to assess stroke volume (SV), HR, and cardiac output ( $\dot{Q}$ ). However, the accuracy of using ECGs during stress tests has been questioned in recent years (Siegler et al., 2011). Specifically, ECG traces are extremely sensitive to movement artefacts as ECGs measure the electrical signal from the brain to the heart and therefore muscle contractions (Odman & Oberg, 1982) or, indeed, movement of the electrodes in relation to the skin (Odman & Oberg, 1982), can influence the ECG trace if not filtered appropriately (Lollgen, 2012; Odman & Oberg, 1982; Siegler et al., 2011). Whilst numerous filtering algorithms have been developed (Khambete, Brown, & Smallwood, 2000; Klijn & Kloprogge, 1974; Nagai, Anzai, & Wang, 2017; Wiklund et al., 2007), there is little consensus as to the most appropriate algorithm for use in paediatric populations (Luo & Johnston, 2010). This lack of clarity, coupled with the high expense and time-intensive set-up, suggest that ECGs are not appropriate for measuring hemodynamic parameters during exercise in a paediatric population.

Doppler echocardiography has been used to measure cardiac variables during paediatric exercise tests, with CV's as low as 5.6% reported for inter-trial variation (Moulinier et al., 1991). However, as noted by Rowland and Obert (2012), there are a number of potential sources of error associated with the Doppler technique, including aortic diameter changes during exercise. This is crucial given that SV is calculated from the aortic cross-sectional diameter (Rowland & Obert, 2012), therefore any changes during exercise will lead to spurious results and an under- or over-estimation of the true SV (Rowland & Obert, 2012). Moreover, a failure to map the full blood flow velocity profile, as well as the relatively expensive equipment, suggests that Doppler echocardiography may not be appropriate for quantifying haemodynamic parameters during exercise in children and adolescents. Conversely, bioelectrical

impedance devices are relatively cheap, as well as being non-invasive, and have been frequently utilised to estimate beat-to-beat changes in cardiac output since the 1960's in paediatric populations (Charloux et al., 2000; Kubicek, Karnegis, Patterson, Witsoe, & Mattson, 1966; J Welsman, Bywater, Farr, Welford, & Armstrong, 2005). However, the widespread use of these devices has been limited due to variable accuracy and reliability of thoracic bioelectrical impedance devices (Charloux et al., 2000; J Welsman et al., 2005). Nonetheless, the PhysioFlow (Mantec, Paris, France) utilises more sophisticated predictive algorithms to minimise such methodological inaccuracies (Charloux et al., 2000), demonstrating highly reliable and accurate estimates of HR, SV and predicted  $\dot{Q}$  in children ( $ICC \geq 0.86$ ,  $CV \leq 9.3\%$ ; J Welsman et al., 2005). Overall, the PhysioFlow is a relatively simple, non-invasive and cost-effective system that provides automated measurement of beat-by-beat haemodynamic variables.

A six-electrode PhysioFlow was used throughout the peak  $\dot{V}O_2$  protocol in **Chapters 4 and 5** and to estimate HR and SV, thereby enabling  $\dot{Q}$  to be calculated. The electrodes were placed on the forehead, left side of the neck, the xiphoid process, the left side of the ribs on the fourth intercostal space, with the final two electrodes placed on the centre of the back. These placements were in accord with the recommendations of Welsman et al. (2005).

The automatic calibration process of the PhysioFlow was recorded over 60 heart beats, in a rested state to attain the highest impedance during systole ( $Z_{max} - Z_{min}$ ), and the rate of variation at rest ( $Z_{max} - Z_{min} / dt_{max}$ ), also known as the contractility index (CTI; Charloux et al., 2000). This enabled the calculation of the thoracic flow inversion time (TFIT) which is the derivative of the raw impedance signal and defined as the time interval between the start of the cardiac cycle (QRS complex) and the lowest velocity of blood ejected from the heart, after the peak ejection velocity (Charloux et al., 2000). The TFIT was subsequently weighted using the algorithm:

$$W(TFIT) = TFIT + HR + PP \quad (6)$$

where  $PP$  is pulse pressure (systolic blood pressure – diastolic blood pressure). Aortic compliance contributes to the signal waveform and a linear relationship has been established according to the SV:PP ratio. Pulse pressure was calculated by manually



entering the resting systolic and diastolic blood pressure values derived following five minutes passive rest using an automated blood pressure monitor (Omron MX3, Milton Keynes, UK). The Omron MX3 blood pressure monitor has been demonstrated to be accurate in adolescents to within 2 and 1 mmHg for systolic and diastolic blood pressure, respectively (Christofaro et al., 2009). Two blood pressure measurements were taken, with the mean systolic/diastolic pressure calculated and utilised in subsequent analyses. The SV index for calibration ( $SV_{i_{cal}}$ ) was subsequently computed by:

$$SV_{i_{cal}} = k * \left[ \frac{dZ}{dt_{max}} \div \frac{Z_{max}}{Z_{min}} \right] * W(TFIT_{cal}) \quad (7)$$

where  $k$  is a constant and  $_{cal}$  indicates the parameters measured during the calibration phase. Thereafter, during the data acquisition phase, SV index was calculated using:

$$SV_i = SV_{i_{cal}} * \sqrt[3]{\frac{CTI}{CTI_{max}}} * \frac{TFIT_{cal}}{TFIT} \quad (8)$$

Beat-by-beat  $\dot{Q}$  could therefore be calculated according to:

$$\dot{Q} = HR * SV_i * BSA \quad (9)$$

with

$$BSA = 0.024265 * BM^{0.5378} * H^{0.3964} \quad (10)$$

$$SV_i = \frac{SV}{BSA} \quad (11)$$

where  $\dot{Q}$  is cardiac output ( $l \cdot \text{min}^{-1}$ ), HR is heart rate (bpm),  $SV_i$  is the SV index ( $\text{ml} \cdot \text{m}^{-2}$ ), BSA is body surface area ( $\text{kg} \cdot \text{m}^{-2}$ ), BM is body mass (kg) and H is height (cm). The BSA equation was proposed by Haycock et al. (1978) and has been validated in infants, children and adults. Maximum stroke volume ( $SV_{max}$ ), maximum cardiac output ( $\dot{Q}_{max}$ ) and maximum heart rate ( $HR_{max}$ ) were defined as the highest 10-second mean value recorded during the incremental ramp test. Additionally, the peak arteriovenous difference ( $a-\bar{v}O_2 \text{ diff}$ ) was calculated to estimate the balance between oxygen delivery and extraction by rearrangement of the Fick (1870a) equation:

$$\text{Peak } a - \bar{v}O_2 \text{ difference} = \frac{\text{Peak } VO_2}{\text{Peak } Q} \quad (12)$$

### 3.2.4 Muscle Deoxygenation Characteristics

Muscle deoxygenation, utilised in **Chapter 4**, reflects the balance of oxygen delivery and extraction, potentially allowing for training, sex and maturational differences in the ability to match oxygen delivery to demand to be elucidated (Barstow, 2019; Boone et al., 2009; Eriksson, 1972; Eriksson, Gollnick, & Saltin, 1973; Gurley, Shang, & Yu, 2012). A plethora of techniques have been utilised to quantify muscle  $\dot{V}O_2$  ( $m\dot{V}O_2$ ), at both rest and during exercise, including arterial-venous catheterisation (Gurley et al., 2012) and doppler ultrasound (Sako, Hamaoka, Higuchi, Kurosawa, & Katsumura, 2001). However, a major limitation with arterial-venous catheterisation is the reliance on highly invasive techniques. Moreover, catheterisation techniques only offer a regional overview of oxygen delivery and extraction as numerous muscles are often supplied via the specific artery/vein (Gurley et al., 2012). Thus, it is not possible to delineate microvascular level changes, which may be critical in furthering our understanding of the mechanisms underpinning changes in peak  $\dot{V}O_2$  during growth and maturation. Similarly, whilst doppler ultrasound has the ability to penetrate deep into muscular tissue (Gurley et al., 2012; Sako et al., 2001), it is limited to detecting larger vessels, consequently providing little information on a microvascular level (Gurley et al., 2012). Therefore, catheterisation and doppler ultrasound were deemed inappropriate for paediatric populations given their inability to elucidate microvascular changes at specific muscle sites during exercise.

One method of  $m\dot{V}O_2$  assessment, which has been increasingly utilised over the last decade in paediatric exercise science due to being non-invasive and relatively inexpensive in comparison to other techniques, is near infrared spectroscopy (NIRS; Barstow, 2019; Breese et al., 2019; McNarry et al., 2015). NIRS can estimate  $m\dot{V}O_2$  by utilising near infrared light in the 700-900 nanometre (nm) wavelength, calculated using the diffusion approximation of the transport theory of light, expressed as:

$$\varphi = \varphi_0 \exp\left(-\frac{x}{\delta}\right), \varphi = \int_0^{4\pi} L d \Omega \quad (13)$$

where  $\varphi$  is the influence rate and is indicative of the flux of energy over a sphere divided by its cross-section (Stolik, Delgado, Pérez, & Anasagasti, 2000),  $x$  is the

distance from the light emitting surface,  $\delta$  is the optical penetration depth,  $L$  is the radiance of the light (Stolik et al., 2000) and  $\Omega$  represents the solid angle. Subsequently, the use of this formula enables the detection of light-absorbing chromophores in biological tissues (Barstow, 2019). NIRS can therefore provide valuable insights into the local microvasculature of muscles at rest, and during exercise, by using the Beer-Lambert Laws of light attenuation (equations 16 and 17). Moreover, in children and adolescents, NIRS has been demonstrated to have excellent intra- (ICC: 0.83 – 0.99, CV  $\leq$  9.8%; Leclair et al., 2010; Ryan et al., 2013) and inter-day reliability (CV = 4.4%; Mantia, Neidert, & Kluess, 2018).

Throughout the incremental ramp test protocol, muscle oxygenation was measured using a commercially available NIRS device (PortaMon, Artinis Medical Systems, Einsteinweg, Netherlands), placed on the m. *vastus lateralis* of the dominant leg, in accord with previous studies (Breese et al., 2019; McNarry et al., 2015). The m. *vastus lateralis* is the dominant muscle utilised during cycling (Kordi et al., 2020), thus more subtle changes in  $m\dot{V}O_2$  may be evident than at other muscular sites. The NIRS device was secured to the leg using sports tape, with a black cloth placed over the NIRS device to attenuate the penetration of ambient light, thereby improving signal quality. Prior to the commencement of the incremental ramp protocol, the NIRS device was zeroed with the participant seated in a stationary position on the cycle ergometer. The intensity of incident and transmitted light was recorded at 10 Hz throughout the protocol, and was used to estimate the relative concentrations, compared to baseline, of oxygenated, deoxygenated and total haemoglobin (HHb).

#### 3.2.4.1 NIRS variables and data processing

Under resting conditions, delivery of  $O_2$  to the muscle, and its relationship to  $m\dot{V}O_2$ , is described by integration of the Fick (1855, 1870a) equations of perfusive (equation 14) and diffusive (equation 15)  $O_2$  delivery.

$$\dot{V}O_2 = \dot{B} * (C_a - C_v)O_2 \quad (14)$$

$$\dot{V}O_2 = DO_2 * (P_{mv}\dot{V}O_2 - P_{mito}O_2) \quad (15)$$

where  $\dot{B}$  is blood flow,  $C_a$  is the  $O_2$  concentration in the arteries,  $C_v$  is the concentration of  $O_2$  in the veins,  $DO_2$  is the diffusivity of  $O_2$ , and  $P_{mv}\dot{V}O_2$  and  $P_{mito}O_2$

represent the partial pressure of O<sub>2</sub> in the microvasculature and the mitochondria, respectively. Previous studies have suggested that NIRS devices can detect the three main light-absorbing chromospheres: haemoglobin (HHb), myoglobin (Mb) and cytochrome oxidase (cyto<sub>ox</sub>; Balaban, Mootha, & Arai, 1996; Barstow, 2019). However, the concentration of [cyto<sub>ox</sub>] is only thought to be approximately 5% the concentration of HHb and Mb and, therefore, its impact upon the NIRS signal is deemed negligible (Balaban et al., 1996). Moreover, both HHb and Mb contain iron, which influences the amount of light absorbed/reflected depending on their O<sub>2</sub> content, consequently their relative concentrations cannot be distinguished, thus the NIRS signal is interpreted as a combination of HHb and Mb. The NIRS signal allows three main variables of interest to be determined, namely, the concentration of oxygenated haemoglobin and myoglobin (oxy[HHb+Mb]), deoxygenated haemoglobin and myoglobin (deoxy[HHb+Mb]) and total [HHb+Mb] (calculated as the sum of oxy[HHb+Mb] and deoxy[HHb+Mb]). When at rest with the *m. vastus lateralis* stationary, all light emitted by the NIRS device is either reflected back to the light emitting device or it is absorbed. This relationship can be described by the Beer-Lambert Law of light attenuation:

$$A(OD) = \log\left(\frac{I}{I_0}\right) = \epsilon * [c] * L \quad (16)$$

where A is light attenuation, I is the amount of emergent light, I<sub>0</sub> is the light source,  $\epsilon$  is the specific extinction coefficient, [c] is concentration of chromospheres, and L is the distance the light has to travel. However, one critical limitation of the Beer-Lambert law is the assumption that all light is contained, and no light is lost due to scattering. This is particularly important when measuring m $\dot{V}O_2$  during exercise as the muscle is constantly changing shape, length and width and thus light is scattered away from the source (Barstow, 2019; Ferreira, Koga, & Barstow, 2007). Subsequently, the Beer-Lambert Law was modified to account for situations where scattering occurs and is described as:

$$A = \log\left(\frac{I}{I_0}\right) = \epsilon * [c] * L * DPF + G \quad (17)$$

with:

$$DPF = \frac{\sqrt{3\mu_s}}{\sqrt{\mu_a}} \quad (18)$$

where  $DPF$  is the differential pathlength factor,  $G$  is the amount of light lost due to scattering,  $\mu_s$  is the scattering coefficient and  $\mu_a$  is the absorption coefficient. The  $DPF$  of the PortaMon (Artinis Medical Systems, Einsteinweg, Netherlands) NIRS device was 4.00, allowing the infra-red light to penetrate ~3 millimetres below the surface of the skin.

Prior to further analyses, the raw NIRS signal was averaged into five-second bins and then scaled from 0% (average of the three-minute warm up at 10 W) to 100% (the highest five second value achieved during the ramp test), in accord with previous studies (McNarry et al., 2015). The [HHb] response was modelled against absolute work rate (W), relative work rate (% of max power achieved during the ramp test), absolute  $\dot{V}O_2$  ( $l \cdot \text{min}^{-1}$ ) and relative  $\dot{V}O_2$  (% of peak  $\dot{V}O_2$ ; McNarry et al., 2015). In line with previous recommendations, the  $\dot{V}O_2$  response was back-shifted by 20 seconds to account for the lung to muscle transit time (McNarry et al., 2015). Subsequently, the profile of the % change ( $\Delta$ ) in [HHb] was modelled using a sigmoidal function, as outlined in McNarry et al. (2015). The sigmoidal model was fitted using:

$$Y = \frac{a}{(1 + e^{-(c+dx)})} \quad (19)$$

where  $a$  is the amplitude of the sigmoid function,  $c$  is a constant which is dependent upon the slope of the sigmoid function ( $d$ ), where  $c/d$  yields the  $x$  value at which 50% of the amplitude is reached. Additionally, the plateau was also reported, which was defined as the  $x$  value at the lower 95% confidence limit of  $a$  (McNarry et al., 2015). A graphical representation of the sigmoidal response profile when expressed against absolute and relative work rate is shown in Figure 3.4.



Figure 3.4 – Representative sigmoidal [HHb] responses when expressed against work rate (W; Left graph), and b) relative work rate (% Max)

### 3.2.5 Anaerobic Parameters

The lack of a ‘gold-standard’ anaerobic measure has resulted in a plethora of anaerobic tests being utilised in paediatric populations, limiting inter-study comparisons due to the different performance outcomes they provide (Ingle & Tolfrey, 2013; Van Praagh, 2000; Van Praagh & Doré, 2002). Athletic performance has been assessed using a variety of different jumping methodologies, namely vertical jumps (VJs), standing broad jumps (SBJs), squat jumps (SJs) and counter-movement jumps (CMJs; Doré et al., 2008; Ingle & Tolfrey, 2013). Whilst these jumping batteries have demonstrated high reliability (ICC: 0.93-0.95, CV: 4.0 – 5.3%; Fernandez-Santos, Ruiz, Cohen, Gonzalez-Montesinos, & Castro-Piñero, 2015; Ingle & Tolfrey, 2013), they only offer a snap-shot of short-term explosive muscular power, and their application to real-world athletic performances is disputed. Indeed, only moderate correlations have been established between SBJ and VJ performance and 30 m sprint performance ( $r^2 = 0.48$ ) in 84 male adolescents ( $14 \pm 1$  years; Hammami, Makhoulouf, Chtara, Padulo, & Chaouachi, 2015).

The most cited measurement of anaerobic performance is the 30-second cycling Wingate (WnT) test which allows for the quantification of peak power ( $P_{peak}$ ), mean power ( $P_{mean}$ ), and fatigue index (FI) over the test duration (Abbasian et al., 2011; Beneke, Hutler, & Leithauser, 2007; Naughton, Carlson, & Fairweather, 1992). The cycling WnT remains a popular method of anaerobic assessment due to the ability to account for differences in body mass, which is accumulated rapidly and differentially

during puberty according to sex (Fellmann & Coudert, 1994b; Rogol et al., 2002). However, reliability and practical issues have been raised regarding the implementation of the WnT in children and adolescents (Doré et al., 2003; Hopkins et al., 2009; Hopkins, Schabert, & Hawley, 2001; Ingle & Tolfrey, 2013; Van Praagh & Doré, 2002; Watt et al., 2002). Specifically, the optimal flywheel resistance for peak power generation (Watt et al., 2002) and the applicability of the WnT to real-world sporting contexts remains debated (Chia, 2006). Perhaps the biggest limitation, however, is the large aerobic energy system contribution throughout the 30-second test. Indeed, Chia (2006) reported 73% and 67% of the total energy consumption to be derived from oxidative phosphorylation during the WnT in girls and boys, respectively and, therefore, the WnT's ability to measure anaerobic performance remains debated.

Given the associated limitations with jumping batteries and the cycling WnT, anaerobic performance was assessed in **Chapter 5** using a 30 m sprint. Over-ground sprinting was chosen as the primary measure of anaerobic performance as it has been reported to be the most reliable anaerobic assessment method currently available (ICC: 0.99, CV: 1.3%; Ingle & Tolfrey, 2013). Moreover, sprinting, with the use of biomechanical modelling, allows for quantification of power and force parameters, giving a more holistic measure of anaerobic performance, whilst retaining high ecological validity (Runacres et al., 2019a). Furthermore, the data collection methods are quick, easy, inexpensive, and can be conducted in field settings, thereby facilitating large cohort studies. Velocity was recorded using a STALKER ATS II (STALKER radar, Plano, Texas, USA) radar gun in **Chapter 5**, which measured velocity over the entirety of the sprint at 46.875 Hz. The radar gun was chosen over the more widely used photocells primarily due to the increased measurement resolution provided meaning that data did not have to be averaged over sections of, or the entire, sprint (Mendez-Villanueva et al., 2010; Meyers et al., 2015; Mujika et al., 2009; Papaikakovou et al., 2009). Instead, a near-instantaneous velocity-time curve was created from which power and velocity could be modelled.

Prior to the undertaking of the maximal 30 m sprint, all participants completed a five-minute warm-up, terminating with a 30 m sprint which simultaneously served as a familiarisation trial. All participants started from a two-point standing start to minimise vertical displacement during the early phase of the sprint and were instructed

to start sprinting using auditory cues ('3...2...1...GO'). All participants completed two maximal sprints over a distance of 35 m, with at least two-minutes rest in between each sprint. The finish line was placed at 35 m to avoid premature deceleration at 30 m, as utilised in previous studies (Meyers et al., 2015; Meyers et al., 2017a), enhancing the fit of the mono-exponential velocity-time curve.

Radar technology has the ability to measure both inbound and outbound velocities, lending itself to potentially determine kinetic differences between repeated sprints, which could enhance our understanding of fatigue mechanisms during sprint running as explored in **Chapter 7**. Repeated sprint ability was assessed using a 5 x 20m shuttle sprint protocol, commonly used within the literature as part of talent identification test batteries (Philippaerts et al., 2006). Repeated sprints, irrespective of protocol, are deemed highly reliable (ICC: 0.98, CV: 0.7 - 2.7%; Oliver, Williams, & Armstrong, 2006; Temfemo et al., 2011). The standardised five-minute warm-up used for the repeated sprint assessment (**Chapter 7**) mirrored that of **Chapter 6**, except it ended with 3 x 10 m sprints by means of a relative familiarisation trial. Participants were instructed to complete 5 x 20 m shuttle runs continuously, completing all repetitions without rest and at maximum effort. Whilst it could be argued that incorporating periods of rest between repeated sprints is more indicative of team sport performances (Mendez-Villanueva et al., 2010; Mujika et al., 2009; Philippaerts et al., 2006), the incorporation of rest periods allows for aerobic recovery, potentially questioning whether protocols of this type are wholly anaerobic. Although participants completed repeated shuttles over a distance of 20 m, velocity, power and force properties were only modelled over 15 m in each direction to minimise deceleration influencing the fit of the velocity-time curve.

### *3.2.6 Force-velocity-Power Profiling*

The raw velocity data was analysed using the novel analysis method of Force-velocity-Power (F-v-P) profiling which allows the computation of power (W) and force properties (N) from a basic velocity ( $v_h$ ) – time (t) trace and basic anthropometric measures of height (m) and body mass (kg; Samozino et al., 2016). F-v-P profiling was validated in the original study against force plate data (Samozino et al., 2016), currently the gold standard for assessing ground reaction forces and power complexes



during over-ground running, in nine elite sprinters ( $23.9 \pm 3.4$  years). The model demonstrated a high correlation for all variables ( $r^2 \geq 0.95$ ;  $p > 0.01$ ), with a small standard error of measurement (SEM; all variables  $\leq 5\%$ ). It must be noted that the current biomechanical model has not been validated in children and adolescents, however, the validity of this measure should be maintained as it has been previously demonstrated that when a maximal acceleration is performed, the  $v_H(t)$  curve follows a mono-exponential profile regardless of age or running proficiency (Morin et al., 2011; Morin et al., 2006). One limitation which must be acknowledged is that the mono-exponential fitting of the  $v_H(t)$  curve does not account for deceleration once peak velocity ( $v_{peak}$ ) has been achieved. Therefore, techniques must be implemented to maintain a maximal sprint over the entire distance to ensure an accurate representation of the kinetics can be modelled, such as asking participants to sprint a longer distance than that over which the kinetic profiles are modelled (Meyers et al., 2015; Runacres et al., 2019a). However, no study to date has compared the differences in reliability between different finishing line distances, so each trial was visually checked for deceleration before data processing commenced.

Despite its strong theoretical validity underpinning F-v-P profiling, Simperingham et al. (2017) reported only moderate intra-day reliability in a group of recreationally active young adults ( $18.6 \pm 0.6$  years). This was postulated to be due to the lack of training specificity to block starts, with more competent athletes able to successfully replicate the co-ordination of a large number of degrees of freedom. This seems plausible as all variables derived from F-v-P profiling in trained and untrained children and adolescents ( $14.1 \pm 2.6$  years) were deemed highly reliable ( $ICC \geq 0.75$ ,  $CV \leq 10\%$ ) when sprint accelerations were performed from a two-point standing start (Runacres et al., 2019a). Therefore, this method was deemed appropriate given its high reliability and the ease of comparison between populations, potentially allowing a maturational and training effects to be identified.

### 3.2.7 F-v-P Mathematical Modelling

The raw velocity data was fitted with a mono-exponential curve to produce a smooth  $v_h(t)$  curve expressed as:

$$v_h(t) = v_{hmax} \cdot (1 - e^{-\frac{t}{\tau}}) \quad (20)$$

where  $v_{hmax}$  is the maximal acceleration reached at the end of the acceleration and  $\tau$  is the acceleration time constant (s). Following integration and differentiation of equation 20, the horizontal position (displacement,  $X_h(t)$ ) and the acceleration ( $\alpha_h(t)$ ) of the bodies centre of mass (COM) can be obtained using equations 21 and 22, respectively. One fundamental assumption associated with F-v-P profiling is that the velocity data is representative of COM motion, and that the human body can be modelled as a complete system represented by its COM.

$$X_h(t) = V_{hmax} \cdot \left( t + \tau \cdot e^{-\frac{t}{\tau}} \right) - v_{hmax} \cdot \tau \quad (21)$$

$$\alpha_h(t) = \left( \frac{v_{max}}{\tau} \right) \cdot e^{-\frac{t}{\tau}} \quad (22)$$

When the fundamental laws of dynamics are applied, the antero-posterior ground reaction force (GRF) applied to the COM in the horizontal direction ( $F_h$ ) can be modelled over time as:

$$F_h(t) = m \cdot \alpha_h(t) + F_{aero}(t) \quad (23)$$

where  $m$  is body mass (kg) and  $F_{aero}(t)$  is the aerodynamic drag the runner must overcome. Aerodynamic drag is directly proportional to the square of the air velocity relative to the participant:

$$F_{aero}(t) = k \cdot (v_h(t) - v_w)^2 \quad (24)$$

where  $v_w$  is the wind velocity and  $k$  is the aerodynamic friction coefficient. This can be estimated from values of air density ( $p$ ,  $\text{kg}\cdot\text{m}^{-3}$ ), the frontal area of the runner ( $Af$ ,  $\text{m}^2$ ) and a drag coefficient ( $Cd = 0.9$ ; Samozino et al., 2016; van Ingen-Schenau, Jacobs, & de Koning, 1991):

$$k = 0.5 \cdot p \cdot Af \cdot Cd \quad (25)$$

with

$$p = p_0 \cdot \frac{Pb}{760} \cdot \frac{273}{273+T^\circ} \quad (26)$$

$$Af = (0.2025 \cdot h^{0.725} \cdot m^{0.425}) \cdot 0.266 \quad (27)$$

where  $p_0 = 1.293 \text{ kg}\cdot\text{m}^{-3}$  is the air density at 760 Torr and 273°K,  $Pb$  is the barometric pressure (in Torr),  $T^\circ$  is the ambient air temperature in which the sprint took place (in

°C),  $h$  and  $m$  are the stature (m) and the body mass (kg) of the sprinter, respectively. Consequently, the power applied to the COM in the horizontal direction can be modelled as:

$$P_h = F_h \cdot v_h \quad (28)$$

Given that all participants started sprinting using a standing start, the mean net vertical acceleration of the COM is quasi-null throughout the acceleration phase of the sprint (Morin et al., 2006). Therefore, if the fundamental laws of dynamics are applied, the average GRF in the vertical direction ( $F_v$ , N) can be modelled over time equal to body mass (Morin et al., 2006):

$$F_v(t) = m \cdot g \quad (29)$$

where  $g$  is gravitational acceleration ( $9.81 \text{ m}\cdot\text{s}^{-1}$ ). The mechanical effectiveness of force application ( $D_{RF}$ ) of the runner can be quantified over the entire acceleration phase by the slope of the linear decrease in the ratio of forces (equation 30) with increasing velocity.

$$\text{Ratio of Forces} = \frac{F_H}{\sqrt{(F_H \cdot F_v)^2}} \quad (30)$$

To date,  $D_{RF}$  values have been modelled for  $t > 0.3$  seconds so only the second step onwards is analysed to remove the initial flight-time from push-off (Samozino, 2018). It is pertinent to note that the 0.3 seconds was derived from block starts. Given that the participants within this thesis started from standing starts,  $t > 0.3$  seconds may not be optimal but was adopted in the absence of evidence-based alternatives. A graphical schematic of the mono-exponential  $v_h(t)$  curve from which the variables were calculated is shown in Figure 3.5.

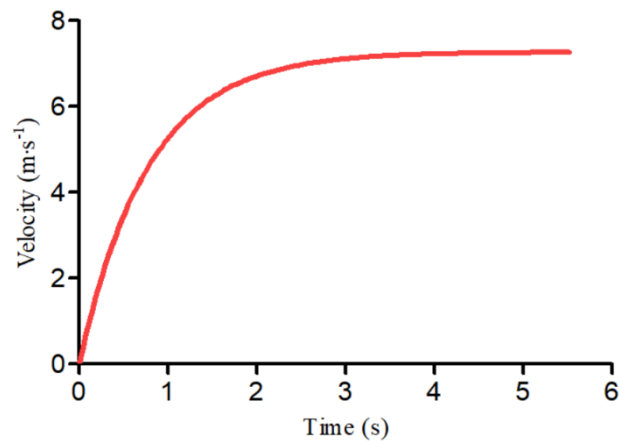


Figure 3.5 – A graphical representation of the  $v_h(t)$  curve modelled for each sprint repetition

In **Chapter 6**,  $v_{\text{peak}}$  ( $\text{m}\cdot\text{s}^{-1}$ ) was interpolated to 0.1s intervals then determined from the mono-exponential  $v_H(t)$  curve, with mean velocity ( $v_{\text{mean}}$ ;  $\text{m}\cdot\text{s}^{-1}$ ) determined as the average velocity across the duration of the sprint defined as the point at which  $x_h(t)$  first exceeded 30 m, which also provided the 30 m sprint time (30mT). All power variables were also interpolated to 0.1 second intervals and  $P_{\text{peak}}$  (W) determined as the highest power output during the sprint, with the time at which  $P_{\text{peak}}$  occurred determining the time to peak power ( $t_{P_{\text{peak}}}$ ; s). Mean power ( $P_{\text{mean}}$ ; W) was determined as the average power output from the start of the  $v_H(t)$  curve until 30mT was reached. Peak horizontal force ( $F_{\text{peak}}$ ; N) underwent the same interpolation as velocity and power, with  $F_{\text{peak}}$  and  $F_{\text{mean}}$  denoting the highest and mean force production during the sprint, respectively. Fatigue rate (FR;  $\text{W}\cdot\text{s}^{-1}$ ) was calculated by averaging the power decline every second from  $P_{\text{peak}}$  until 30mT. Relative peak power ( $R_{P_{\text{peak}}}$ ;  $\text{W}\cdot\text{Kg}^{-1}$ ), relative mean power ( $R_{P_{\text{mean}}}$ ;  $\text{W}\cdot\text{Kg}^{-1}$ ) and relative peak horizontal force ( $R_{F_{\text{peak}}}$ ;  $\text{N}\cdot\text{Kg}^{-1}$ ) were obtained by dividing the values of  $P_{\text{peak}}$ ,  $P_{\text{mean}}$  and  $F_{\text{peak}}$  by the participants body mass (kg). Finally,  $P_{\text{peak}}$  and  $F_{\text{peak}}$  were allometrically scaled by body mass using methods reported elsewhere and described above (Nevill et al., 2006; Nevill et al., 1998).

The repeated sprint data collected in **Chapter 6** was split into five sections, with the end of each sprint determined as when  $x_h(t)$  first exceeded 20 m. Subsequently, the start of each new sprint was assigned time 0 and the same procedures listed above

employed to calculate all variables, with the exception that all variables were averaged from the sprint start until  $x_h(t) = 15\text{m}$ , which also gave 15 m sprint time (15mT).

### *3.2.8 Physical Activity Monitoring*

Habitual physical activity (PA) and sedentary time (SED) were assessed in **Chapters 4 and 5**. The currently accepted ‘gold standard’ for habitual PA assessment is doubly labelled water which assesses energy expenditure engendered by bodily movements (Goran, Poehlman, & Danforth-Jr, 1994). However, the doubly labelled water technique was developed for use in adults (Goran et al., 1994) and has yet to be robustly validated in paediatric populations. Moreover, doubly labelled water uses radioactive isotopes, raising ethical concerns when working with paediatric populations (Plasqui & Westerterp, 2007), is very expensive, and limited to four days recording time (Goran et al., 1994). Much like subjective measures of PA assessment, doubly labelled water only offers a macroscopic overview of energy expenditure (Ottevaere et al., 2011; Plasqui & Westerterp, 2007), with no information provided regarding the pattern or composition of PA which could be crucial for performance and health (Carson et al., 2016; Chastin et al., 2015; Dumuid et al., 2018a).

Accelerometers allow for second-by-second quantification of PA over a finite period, allowing for the exploration of differences in the composition, and pattern, of physical activity accrual between populations (Carson et al., 2016; Rowlands et al., 2018b). Whilst the optimal accelerometer wear location is still debated, the wrist and hip placements are the most common (Fairclough et al., 2016; Herrmann, Barreira, Kang, & Ainsworth, 2014; Rich et al., 2013). Wrist-worn placements have been shown to increase overall wear-time compared to hip-mounted accelerometers (Fairclough et al., 2016; McLellan, Arthur, & Buchan, 2018; Scott et al., 2017). Conversely, hip-worn accelerometers have been reported to offer superior accuracy in quantifying SED due to their ability to differentiate between sitting, lying and reclining positions (Lynch et al., 2019). Despite these differences, PA studies comparing wrist and hip wear locations have shown good agreement between sites ( $r^2$ : 0.81 – 0.88; Fairclough et al., 2016; McLellan et al., 2018; Scott et al., 2017) irrespective of intensity and total activity accumulated. Consequently, given the superior accuracy of hip-worn

accelerometers in quantifying SED, a hip-worn triaxial GT3X+ (ActiGraph, Pensacola, Florida, USA) was utilised to measure PA and ST levels.

The ActiGraph GT3X+ accelerometer was initialised using ActiLife v6.13.4 (ActiLife, ActiGraph, Pensacola, Florida, USA) at 100 Hz for seven consecutive days and nights to gather an indication of PA, SED, and sleep patterns. The start time for each accelerometer recording was set to midnight the day after the testing session to ensure start and end time standardisation between participants. Potential seasonal variation associated with accelerometry data collection (Carson & Spence, 2010) was accounted for by spreading the data collection periods for **Chapters 4** and **5** were evenly throughout the year, for both athletes and controls. Consequently, all potential seasonal variation in PA and SED data was alleviated and accounted for within the subsequent analyses. Whilst it could be argued that a compensation effect following a vigorous exercise stimulus may be evident in PA behaviours (Goodman, Mackett, & Paskins, 2011), there is scant empirical evidence in children to support this notion. Nevertheless, all data on the first day was compared to the following six-days of accelerometry and removed from analyses if significantly less PA, and more SED, was evident in order to gain a representative overview of PA and SED levels. The significance of the data was tested using a repeated t-test of day one and the aggregate PA and SED levels of the remaining six days that met the wear-time criteria. Of the 250 participants involved within **Chapters 4** and **5** this happened on five occasions.

A wear-time log was provided to participants along with the accelerometer, with participants asked to record the time, and reason why, they removed the monitor over the course of the seven-day measurement period on wear-time log. Accelerometers were collected after the seven-day recording and the raw data was then downloaded using ActiLife into one-second epochs and the wear-time determined. Wear-time is defined as the minimum amount of time participants are required to wear the monitor for to provide a representative insight of PA and SED on each day and across the week (Fairclough et al., 2016; Kristensen et al., 2010; Rowlands et al., 2018b; Scott et al., 2017; Trost, Pate, Freedson, Sallis, & Taylor, 2000). Trost et al. (2000) suggested that only three days of habitual PA monitoring are needed to achieve an ICC of 0.80. Moreover, a wear-time of  $\geq 8$  hours·day<sup>-1</sup> on any three recording days has demonstrated excellent reliability (ICC: 0.90; Couto et al., 2014; Herrmann et al.,

2014; Rich et al., 2013) in large scale PA studies in children. Whilst it could be argued that a compensatory effect following a vigorous exercise stimulus may be evident in PA behaviours (Goodman, Mackett, & Paskins, 2011), there is scant empirical evidence in children to support this notion. Nevertheless, a repeated measures ANOVA was conducted to ascertain whether there were any significant differences in PA and SED between the first day, and all subsequent days. In the instances where this was present (7% of participants in **Chapters 4 and 5**), day one was removed and the wear-time criteria applied to the remaining six-days. As such, a wear-time criteria of  $\geq 8$  hours per day on any three-days was used, with non-wear time defined as any period  $\geq 20$  minutes of consecutive zeroes (Couto et al., 2014; Herrmann et al., 2014; Love, Adams, Atkin, & van Sluijs, 2019a).

Cut-points are a means of classifying movement intensity that allow for the quantification of time spent in sleep, SED, light intensity PA (LPA), moderate intensity PA (MPA) and vigorous intensity PA (VPA; Trost, Loprinzi, Moore, & Pfeiffer, 2011). Despite the need for a consensus to ensure studies are comparable, there are currently multiple different cut-points for children and adolescents using hip-worn accelerometers (Evenson, Catellier, Gill, Ondrak, & McMurray, 2008; Freedson, Pober, & Janz, 2005; Mattocks et al., 2007; Puyau, Adolph, Vohra, & Butte, 2002; Treuth et al., 2004). Trost et al. (2011) compared five of these cut-points against indirect calorimetry, concluding that only the Evenson cut-points (2008) displayed good agreement across all intensities, which have therefore been applied within this thesis. The Evenson cut-points are defined as: SED:  $\leq 100$  counts per 15 seconds; MPA:  $\geq 2,296$  counts per 15 seconds; and VPA:  $\geq 4,012$  counts per 15 seconds. LPA is defined as the time spent between the SED and MPA cut points. Sleep was identified using the Sadeh et al. (1994a) sleep algorithm which has been shown to be the most reliable and valid sleep algorithm for quantifying sleep from GT3X+ data in children and adolescents (Kinder et al., 2012).

### 3.3 Statistical Analyses

#### 3.3.1 Compositional Analysis of Physical Activity Data

All compositional analyses, performed in **Chapter 7**, were conducted in R (<https://cran.r-project.org/>) using the compositions package (version 1.40-2) and its dependencies (Chastin et al., 2015). First, time in all PA behaviours was normalised into a proportion of the total time (1,440 minutes = 1 day), following which the geometric mean was calculated and the variation matrix determined (Carson et al., 2016; Chastin et al., 2015). The variance matrix is a measure of distribution, derived by quantifying the variation between pair-wise log ratios (i.e.  $\ln(\text{LPA}/\text{MPA})$ ; Carson et al., 2016; Dumuid et al., 2018a). Values can range from -1 to 1, with the ratios tending towards 0 indicating high co-dependency (Carson et al., 2016; Chastin et al., 2015). The composition model of the five components derived from the accelerometer data were then expressed in isolation and relative to all other behaviours using isometric log ratio (ILR) transformations (Dumuid et al., 2018a). Isometric log ratios were chosen over additive or centred log ratio approaches as the latter do not allow for the quantification of singular movement behaviours in relation to each other or for the assumption of independence between variables to be met, respectively (Dumuid et al., 2018a). Therefore, ILR transformations were conducted according to the following formula:

$$z = [z_1, z_2, \dots, z_{D-1}] = \sqrt{\frac{D-1}{D}} \ln \left( \frac{X_1}{\sqrt{\prod_{k=2}^D X_k}} \right), \sqrt{\frac{D-2}{D-1}} \ln \left( \frac{X_2}{\sqrt{\prod_{k=3}^D X_k}} \right), \dots, \sqrt{\frac{D-j}{D-j+1}} \ln \left( \frac{X_j}{\sqrt{\prod_{k=D-j+1}^D X_k}} \right), \frac{1}{\sqrt{2}} \ln \left( \frac{X_{D-1}}{X_D} \right) \quad (31)$$

with rearrangement of the equation showing:

$$c_1 = \sqrt{\frac{D-1}{D}} z_1 \quad (32)$$

where  $D$  is the number of components,  $z$  a dimensional real vector, and  $x$  is the minutes per day spent in that behaviour (Dumuid et al., 2018a). Each ILR composition demonstrates the relative effect of a singular movement behaviour in relation to all other movement behaviours (Dumuid et al., 2018a). Moreover, the additional



compositions ( $C_2, C_3, \dots, C_{n-1}$ ) contain no relative information about the previous composition (Dumuid et al., 2018a). Thus, all compositions can be considered independent and their individual associations, and influences, on outcome variables can be measured using multiple linear regression techniques (Chastin et al., 2015). The ILR multiple linear regression for  $n$  compositions can be expressed as:

$$i = 1, 2, \dots, n, \text{ where } X_i = [X_{i1}, X_{i2}, \dots, X_{iD}] \text{ with } \sum_{j=1}^D X_{ij} = 1$$

$$y_i = \beta_0 + \sum_{j=1}^{D-1} \beta_j z_{ij} + \epsilon_i \quad (33)$$

where

$$z_{ij} = \sqrt{\frac{D-j}{D-j+1}} \ln \left( \frac{X_{ij}}{\sqrt[D-1]{\prod_{k=j+1}^D X_{ik}}} \right) \text{ for } j = 1, 2, \dots, D-1 \quad (34)$$

where  $\beta_0$  represents the intercept, with the regression coefficient ( $\beta_1$ ) representing the change in  $y$  (outcome variable) when the first ILR co-ordinate is changed, and the remaining co-ordinates kept constant to ensure that  $\sum_{j=1}^D X_{ij} = 1$  remains true (Dumuid et al., 2018a). Therefore, when this is applied to physical activity and sleep data, the five-part composition becomes:

$$ilr_1 = \sqrt{\frac{4}{5}} \ln \left( \frac{Sleep}{\sqrt[4]{(SED, LPA, MPA, VPA)}} \cdot \frac{1+r}{1-s} \right)$$

$$ilr_2 = \sqrt{\frac{3}{4}} \ln \left( \frac{SED}{\sqrt[3]{(LPA, MPA, VPA)}} \cdot \frac{1-s}{1-s} \right),$$

$$ilr_3 = \sqrt{\frac{2}{3}} \ln \left( \frac{LPA}{\sqrt[2]{(MPA, VPA)}} \cdot \frac{1-s}{1-s} \right)$$

$$ilr_4 = \sqrt{\frac{1}{2}} \ln \left( \frac{MPA}{VPA} \cdot \frac{1-s}{1-s} \right) \quad (35)$$

However, the example in equation 35 only gives information for when sleep is the numerator on the first ILR co-ordinate, thus all models underwent sequential rotation to allow all movement behaviours to be expressed against all other movement

behaviours (Carson et al., 2016; Chastin et al., 2015; Dumuid et al., 2018a). All model coefficients were reported, with positive and negative coefficients indicating a positive or negative effect on the outcome variable (y) when time in that behaviour was increased (Dumuid et al., 2018a). Moreover, sex, maturity and training status were entered as covariates in each ILR model to account for differences in PA compositions between groups, improving the accuracy of the predictive models. The predicted change in the outcome variable of interest ( $\Delta y$ ) when the numerator of the first part of the composition ( $X_1$ ) increases (i.e. 10 minutes extra sleep) can be modelled using equations 36 and 37 when time spent in another movement behaviour is decreased by the same magnitude (i.e. 10 minutes less LPA). This is necessary to ensure that the finite whole (i.e. 1,440 minutes) is kept constant.

$$\Delta\hat{y} = \beta_1 \cdot \sqrt{\frac{D-1}{D}} \cdot \ln\left(\frac{1+r}{1-s}\right) \quad (36)$$

where

$$-1 < r < \frac{1-X_1}{X_1} \text{ and } s = r \cdot \frac{X_1}{1-X_1} \quad (37)$$

where  $r$  is the magnitude of increase in the composition numerator and  $s$  is the magnitude of decline in subsequent denominators. Thus, compositional analysis allows for the absolute and percentage change in  $y$  to be determined when time is allocated to, and from, that behaviour and re-allocated to other physical activity behaviours or sleep. Change matrices were produced to illustrate the predictive changes in absolute and scaled peak  $\dot{V}O_2$  when systematically re-allocating 10 minutes from one movement behaviour to all other movement behaviours (Carson et al., 2016; Chastin et al., 2015). All predictive changes were presented as percentage changes relative to the compositional mean, with significant changes defined as any change greater than the group-specific smallest worthwhile change (Hopkins, 2000; Hopkins et al., 2009), calculated as:

$$SWC(\%) = \frac{\text{Group Mean}}{(0.2 * \text{Group SD})} \quad (38)$$

# Chapter 4

Effects of sex, training, and  
maturity status on the  
cardiopulmonary responses and  
muscle deoxygenation kinetics  
during incremental exercise

# **Chapter 4 (Study 1) – Effects of sex and training status on the cardiopulmonary responses and muscle deoxygenation kinetics during incremental exercise in children and adolescents**

## **4.1 Introduction**

In 2018, 48% of children and adolescents in Wales participated in extra-curricular sport three or more times a week, an increase of 4% compared to 2015 (Sport Wales, 2018). The upward trend in sport participation is encouraging given the health benefits associated with exercise during childhood and adolescence (Armstrong, 2007; Eddolls et al., 2017; Imboden et al., 2018; Lemez & Baker, 2015). However, despite this widespread participation in sport, and the training associated with it, fundamental questions remain regarding the physiological responses to training in youth (Armstrong & McNarry, 2016). Indeed, one area that has received renewed interest over the last decade is the concept of a maturational threshold which suggests that pubertal children may experience an accelerated adaptation to training stimuli relative to their pre-pubertal counterparts, mediated by increases in circulating androgenic hormones (Katch, 1983). The existence of a maturational threshold remains highly debated (Armstrong & Welsman, 2019a; Armstrong & Welsman, 2019b; Cunha et al., 2011; McNarry et al., 2014b; Runacres et al., 2019b), with suggestions that it may be dependent on the specific parameter in question (Van Praagh, 2000) or, possibly sex.

Despite the research and practical interest in the influence of maturity on the training responses in youth (Armstrong, 2015, 2017; Doncaster et al., 2018; Rowland, 1997), few studies have considered the interaction of training with the concomitant effects of growth and maturation according to sex. Puberty is highly sexually dimorphic, with significant differences in the timing and tempo of maturity onset and hormonal milieu (Rogol, 2002; Rogol et al., 2002). Whilst no studies have specifically sought to compare the influence of training in boys and girls, marked differences were apparent in the early literature which suggested that, in contrast to boys (Baxter-Jones et al., 1993; Cunningham et al., 1984; Kobayashi et al., 1978; Rowland et al., 1997b),

training was not associated with significant gains in pre-pubertal girls (Stoedefalke et al., 2000; Welsman et al., 1996; Welsman et al., 1997).

More recent studies have suggested these findings are more likely to reflect methodological factors, such as differences in training and testing modalities and an insufficient training stimulus, rather than a physiological inability to respond to training (Armstrong, 2007, 2015; Massicotte & Macnab, 1974; McNarry & Jones, 2014). Indeed, when peak  $\dot{V}O_2$  is rigorously determined, studies report that girls experience a similar degree of trainability to their male counterparts (Armstrong & Welsman, 2019b; McNarry et al., 2014b; McNarry et al., 2011b). Specifically, McNarry et al. (2011b) found trained pre-pubertal girls to have a 17.5% greater peak  $\dot{V}O_2$  than their untrained counterparts, with similar training differences in absolute peak  $\dot{V}O_2$  in pubertal (21.5%) and post-pubertal (17.5%) adolescents. These observed improvements were proposed to be mediated by an increased gas exchange threshold (GET), an absence of a plateau in the stroke volume (SV) response and a rightward shift of the deoxygenated [HHb] response during ramp exercise (McNarry et al., 2011b). Whether similar mechanisms are responsible for the training related increases in peak  $\dot{V}O_2$  reported in boys largely remains to be established. Whilst morphological and functional myocardial adaptations have been reported in boys (Obert et al., 2003; Rowland et al., 1997a; Rowland & Unnithan, 2013; Vinet et al., 2003), no studies have investigated the influence of training on the peripheral oxygen extraction during incremental ramp exercise.

Studies investigating the effect of sex on the development of peak  $\dot{V}O_2$  suggest that sexual dimorphism is evident even in pre-pubertal children (Winsley et al., 2009). Specifically, when peak  $\dot{V}O_2$  is normalised for body mass, pre-pubertal boys have been shown to have a 10-15% greater peak  $\dot{V}O_2$  than girls (Armstrong & Welsman, 2020c), which may be attributable to a higher oxygen delivery capacity mediated by a greater maximal stroke volume ( $SV_{max}$ ) and, consequently, cardiac output ( $\dot{Q}_{max}$ ). However, one of the key considerations when comparing boys and girls is the differing body compositions, with girls having a higher percentage body fat than boys from 10 years of age (J. Wells, 2007), which questions the utility of ratio scaling by body mass (Tanner, 1949; Welsman & Armstrong, 2019). Nonetheless, even when pre-pubertal boys and girls are matched for lean body mass, boys are still reported to demonstrate

an ~15% higher peak  $\dot{V}O_2$  (Winsley et al., 2009). Interestingly, Winsley et al. (2009) also reported no sex differences in  $SV_{\max}$ ,  $Q_{\max}$ , or the structural properties of the myocardium, with a similar left ventricular mass and left ventricular end diastolic volume in both sexes. This therefore contradicts previous suggestions that the sex differences may be related to differences in oxygen delivery, with Winsley et al. (2009) suggesting that these sex differences may be attributable to sexual dimorphism in the ability to extract  $O_2$  at the working muscles. More specifically, a greater maximal arteriovenous difference ( $a-\bar{v}O_{2\text{diff}}$ ), an indicator of peripheral oxygen extraction, was observed in boys. However, the  $a-\bar{v}O_{2\text{diff}}$  only offers a macroscopic overview of peripheral oxygen extraction in contrast to the microvasculature insights afforded by near infra-red spectroscopy (NIRS; Barstow, 2019). Nonetheless, these findings were corroborated by McNarry et al. (2015) who reported that the plateau of the deoxygenated haemoglobin ([HHb]) response during ramp exercise explained ~12% of the variance in peak  $\dot{V}O_2$  between sexes after accounting for fat free mass (FFM), the GET and body fatness. However, it is pertinent to note that the majority of the participants in both of these studies were pre-pubertal, precluding inferences as to the relative contribution of the oxygen delivery and extraction to potential sex differences in peak  $\dot{V}O_2$  in pubertal and post-pubertal adolescents (McNarry et al., 2015).

Therefore, the aim of this study was to investigate the influence of training on the aerobic fitness of youth and whether this, or the mechanisms underpinning it, differ according to sex.

#### 4.2 Methods

Ethics approval was granted by the institutional research ethics committee, with all research practices conforming to the Declaration of Helsinki. Written parent/guardian consent and participant assent were obtained, and a pre-screening medical questionnaire was completed prior to any testing. Participants were excluded if they had any known pre-existing cardiovascular, metabolic, kidney, or other condition that would prevent them from completing the experimental procedures.

Trained children and adolescents were recruited through the respective sport's National Governing Body, with all of the trained children and adolescents' part of a long-term athlete development programme for Hockey or Football. All trained participants completed an average of  $10 \pm 5$  hours of training per week and had been training for at least two years prior to study entry. Untrained participants were recruited from local schools across south Wales and were not engaged in any formal sports. The final sample consisted of 187 participants, of which 108 were trained (43 girls; age:  $14.3 \pm 1.8$  years) and 79 were untrained (36 girls; age:  $14.7 \pm 1.7$  years).

#### *4.2.1 Experimental Procedures*

All participants were required to attend the research labs at Swansea University Bay Campus once to complete the study. On arrival, blood pressure was recorded after five minutes of rest using an automated blood pressure monitor (Omron MX3, Milton Keynes, UK). Stature and sitting stature were then measured to the nearest 0.1 cm using a Holtain Stadiometer (Holtain, Crymych, Dyfed, UK), and body mass recorded to the nearest 0.1 kg using electronic scales (Seca 803, Seca, Chino, CA, USA). Maturity status was estimated using the equations of Mirwald et al. (2002b), with participants  $\geq 1$  year from, between -0.99 and 0.99 years from, and  $\geq 1$  years post peak height velocity classified as pre-, circa- and post-pubertal, respectively.

$\dot{V}O_{2\max}$  was assessed using an incremental ramp test to volitional exhaustion on a cycle ergometer (Lode Excalibur Sport, Groningen, Netherlands). Specifically, following a three-minute warm-up at 10 W, the resistance increased by 20 - 25  $W \cdot \text{min}^{-1}$ , depending on the participant's age. All participants were instructed to maintain a cadence of 60 – 80 revolutions per minute (rpm) throughout the test, with volitional exhaustion defined as when participants could not maintain a cadence above 50 rpm, despite strong verbal encouragement. Inspired and expired air were collected on a breath-by-breath basis using a Vyntus Metabolic Cart (VYAIR medical Ltd, Mettawa, IL, USA), with beat-by-beat heart rate (HR), stroke volume (SV) and estimated cardiac output ( $\dot{Q}$ ) assessed using a thoracic bioelectrical impedance device (Physioflow, Paris, France). The six electrodes were placed according to the recommendations of Welsman et al. (2005), which has been demonstrated to provide accurate and reliable results during exercise in children. Finally, muscle deoxygenation was assessed throughout the exercise protocol using a portable near-infrared spectroscopy (NIRS)

device (PortaMon, Artinis Medical Systems, Einsteinweg, Netherlands), placed on the *m. vastus lateralis* of the dominant leg, as per previous studies (Breese et al., 2019; McNarry et al., 2015). The NIRS device was secured to the *m. vastus lateralis* using sports tape, with blackout cloths also used to prevent ambient light from distorting the NIRS signal. The NIRS device was zeroed whilst the participant was seated and relaxed on the cycle ergometer, prior to data acquisition. The Vyntus, PhysioFlow and Portamon were all calibrated in line with manufacturer instructions prior to each peak  $\dot{V}O_2$  test.

To verify a maximal effort during the incremental ramp test, all participants completed a supramaximal validation bout after 15 minutes of rest. This bout involved participants completing three minutes at 10 W before undergoing a near instantaneous transition to 105% of the peak power achieved during the incremental ramp test. Participants were instructed to maintain a cadence > 50 rpm for as long as possible, with gas exchange continuously measured. For the subsequent seven days, participant's habitual physical activity was assessed at 100 Hz using a GT3X accelerometer (ActiGraph, Pensacola, Florida, USA) worn on the right hip. All participants were instructed to wear the accelerometer for 24 hours a day. Participants were also asked to complete a sleep log detailing periods of monitor removal, waking time, and bedtime. This aided with further analysis and minimised the misclassification of non-wear time as sedentary time (SED) or sleep.

#### 4.2.2 Data Analysis

The raw  $\dot{V}O_2$  data from the incremental ramp test and supramaximal validation bout were averaged into 10 s bins, with  $\dot{V}O_{2max}$  defined as the highest 10 s moving average during the incremental ramp test or the supramaximal validation bout. If there was  $\leq 2.5\%$  difference between the two tests  $\dot{V}O_{2max}$  was deemed to have been reached. In instances where this was violated the highest 10 s  $\dot{V}O_2$  recording was carried forward for analysis. To aid comparisons according to sex, maturity and training status,  $\dot{V}O_{2max}$  was allometrically scaled by body mass, using methods detailed elsewhere (Nevill et al., 2006; Nevill et al., 1998). Allometric scaling was chosen over the more commonly utilised ratio scaling, which has been consistently shown to penalise heavier, more mature children, thus creating spurious results (Welsman & Armstrong, 2019). The GET was determined using the V-slope method (Beaver et al., 1986) and defined as



the point at which carbon dioxide output ( $\dot{V}CO_2$ ) rose disproportionately to  $\dot{V}O_2$ . The GET was expressed in absolute terms ( $l \cdot \text{min}^{-1}$ ) and as a percentage of peak  $\dot{V}O_2$ . The kinetics of the initial  $\dot{V}O_2$  response were quantified using the mean response time (MRT). This was determined as the time from the onset of the ramp forcing function to the intersection point of the baseline  $\dot{V}O_2$  and a backwards extrapolation of the slope of  $\dot{V}O_2$  as a function of time (Barstow et al., 1999). Finally, the  $O_2$  cost of exercise was quantified according to the gain, calculated as the average change in  $\dot{V}O_2$  per W over the entire ramp forcing function (Barstow et al., 1999).

The data obtained from the PhysioFlow were averaged into 15 s bins and maximum heart rate ( $HR_{\text{max}}$ ), stroke volume ( $SV_{\text{max}}$ ) and cardiac output ( $\dot{Q}_{\text{max}}$ ) defined as the highest 15 s moving average throughout the incremental ramp test.  $SV_{\text{max}}$  and  $\dot{Q}_{\text{max}}$  were subsequently allometrically scaled to body surface area (BSA), estimated according to the predictive equations of Haycock et al. (1978). Additionally, to estimate the balance between  $O_2$  delivery and extraction, the peak  $a-\bar{v}O_{2\text{diff}}$  was calculated by rearrangement of the Fick equation (Peak  $\dot{V}O_2 / \dot{Q}_{\text{max}}$ ; Fick, 1870a). Prior to analysis, the NIRS-derived [HHb] was averaged into 5 s bins, baseline corrected and normalised to end-exercise values. A sigmoidal function was then used to ascertain the relationship between [HHb] and peak  $\dot{V}O_2$  and work rate in both absolute and relative terms. A sigmoidal function was used to determine the [HHb] relationship with work rate and  $\dot{V}O_2$  as it provided a superior fit compared to a double-linear model, as reported previously (McNarry et al., 2015).

The accelerometer data was downloaded into 15 s epochs using ActiLife (v6.13.4.0, ActiGraph, Pensacola, Florida, USA) to allow the Evenson (2008) cut-points to be applied which have been consistently demonstrated to accurately quantify sedentary time (SED) and moderate-to-vigorous physical activities (MVPA; Migueles et al., 2017; Trost et al., 2011). Wear-time was set to  $> 8 \text{ hours} \cdot \text{day}^{-1}$  on any three days, shown to provide an accurate and reliable estimation of children's physical activity (Herrmann et al., 2014; Rich et al., 2013), with non-wear time defined as  $> 20$  minutes of consecutive zeroes.

### 4.2.3 Statistical Analyses

All statistical analyses were conducted in SPSS (version 26, IBM, Portsmouth, UK), with values presented as mean  $\pm$  SD. An ANCOVA, covarying for maturity status and physical activity level, was used to establish the effect of sex and training status, and any interactions. Cohens  $d$  was also calculated, with  $\leq 0.20$ ,  $\geq 0.21 - \leq 0.60$ ,  $\geq 0.61 - \leq 0.80$ , and  $\geq 0.81$  considered a trivial, moderate, large, and very large effect, respectively.

### 4.3 Results

Of the 187 participants, 169 reached  $\dot{V}O_{2\max}$  during the incremental ramp test with the remaining 18 participants, evenly distributed across sex and training groups, achieving a higher  $\dot{V}O_2$  value during the supramaximal validation bout, and excluded from subsequent analyses. The  $b$  exponent for allometrically scaling  $\dot{V}O_{2\max}$  by body mass was 0.67 (95%CI: 0.65 – 0.69). There were no significant sex- or training-related differences in any anthropometric variable when accounting for maturity (Table 4.1). However, boys engaged in significantly more MVPA than girls, irrespective of training status ( $F_{(1,186)} = 6.8$ ,  $p > 0.05$ ,  $d = 0.23$ ), with no significant differences in SED.

#### 4.3.1 Influence of training status

Trained children and adolescents had a higher  $\dot{V}O_{2\max}$  than their untrained counterparts ( $F_{(1, 168)} = 16.0$ ,  $p < 0.01$ ,  $d = 0.32$ ), which persisted even after allometrically scaling  $\dot{V}O_{2\max}$  for body mass ( $F_{(1,168)} = 15.2$ ,  $p < 0.01$ ,  $d = 0.81$ ; Table 4.2). Furthermore, trained athletes had a higher maximal power ( $F_{(1,168)} = 26.4$ ,  $p < 0.01$ ,  $d = 0.25$ ),  $SV_{\max}$  ( $F_{(1,168)} = 1.8$ ,  $p < 0.05$ ,  $d = 0.04$ ) and  $\dot{Q}_{\max}$  ( $F_{(1,168)} = 2.4$ ,  $p < 0.05$ ,  $d = 0.01$ ), and a faster MRT ( $F_{(1,168)} = 7.9$ ,  $p < 0.01$ ,  $d = 0.83$ ), than their untrained counterparts. Contrastingly, there was no significant difference in the absolute or relative GET, gain,  $HR_{\max}$ , maximal  $a-\bar{v}O_{2\text{diff}}$  or allometrically scaled  $SV_{\max}$  or  $\dot{Q}_{\max}$  (all  $p > 0.05$ ).

Trained youth had a less steep [HHb] slope when expressed against absolute  $\dot{V}O_2$  ( $F_{(1,168)} = 8.6$ ,  $p < 0.01$ ,  $d = 0.54$ ). However, when expressed against work rate (absolute and relative) or relative  $\dot{V}O_2$ , no significant differences were evident (Table 4.3). Trained children and adolescents had a higher  $c/d$  and plateau than untrained

children and adolescents, irrespective of whether [HHb] was expressed against absolute work rate ( $p < 0.01$ ) or  $\dot{V}O_2$  ( $p < 0.01$ ). However, when [HHb] was expressed against relative  $\dot{V}O_2$ , these differences were ameliorated. Contrastingly, both the  $c/d$  and plateau remained higher in the trained than untrained participants when expressed against relative work rate ( $c/d$ :  $F_{(1,168)} = 13.5$ ,  $p < 0.01$ ,  $d = 0.92$ ; plateau:  $F_{(1,168)} = 14.3$ ,  $p < 0.01$ ,  $d = 0.30$ ).

#### 4.3.2 Influence of sex

Boys had a higher absolute ( $F_{(1,168)} = 11.7$ ,  $p < 0.01$ ,  $d = 0.81$ ) and allometrically scaled  $\dot{V}O_{2\max}$  ( $F_{(1,168)} = 9.4$ ,  $p < 0.01$ ,  $d = 1.09$ ) and absolute GET ( $F_{(1,158)} = 7.8$ ,  $p < 0.01$ ,  $d = 0.65$ ; Table 4.2) than girls. However, when the GET was expressed relative to peak  $\dot{V}O_2$ , no sex difference was evident ( $p > 0.83$ ). Boys were also characterised by a slower MRT ( $F_{(1,168)} = 6.5$ ,  $p < 0.01$ ,  $d = 0.54$ ), a greater gain ( $F_{(1,168)} = 5.7$ ,  $p < 0.01$ ,  $d = 0.55$ ), peak power ( $F_{(1,168)} = 5.2$ ,  $p < 0.05$ ,  $d = 0.28$ ) and maximal  $a\text{-}\bar{v}O_{2\text{diff}}$  ( $F_{(1,168)} = 4.6$ ,  $p < 0.05$ ,  $d = 0.65$ ). However, no sex differences were found for either absolute or scaled  $\dot{Q}_{\max}$  ( $p > 0.45$ ),  $SV_{\max}$  ( $p > 0.22$ ), or  $HR_{\max}$  ( $p > 0.24$ ).

When [HHb] was expressed against absolute  $\dot{V}O_2$ , boys had a higher  $c/d$  ( $F_{(1,168)} = 4.3$ ,  $p < 0.05$ ,  $d = 0.55$ ) and plateau ( $F_{(1,168)} = 4.9$ ,  $p < 0.05$ ,  $d = 0.44$ ). However, when [HHb] was a function of relative  $\dot{V}O_2$ , no sex differences were found (Table 4.3). Similarly, when [HHb] was expressed against absolute work rate, boys had a higher  $c/d$  ( $F_{(1,168)} = 2.9$ ,  $p < 0.05$ ,  $d = 0.33$ ) and plateau ( $F_{(1,158)} = 2.8$ ,  $p < 0.05$ ,  $d = 0.19$ ) compared to girls, but no differences were evident when [HHb] was expressed against relative work rate. There were no significant differences in the amplitude or slope of the [HHb] response expressed against any variable between boys and girls.

#### 4.3.3 Interaction effects

Significant sex and training interactions were found for allometrically scaled  $\dot{V}O_{2\max}$  ( $F_{(1,168)} = 8.0$ ,  $p < 0.01$ ),  $SV_{\max}$  ( $F_{(1,168)} = 2.9$ ,  $p < 0.05$ ) and  $\dot{Q}_{\max}$  ( $F_{(1,168)} = 5.4$ ,  $p < 0.05$ ). More specifically, there was a greater difference between trained and untrained boys than girls for allometrically scaled  $\dot{V}O_{2\max}$  (18.4% vs 6.9%). Contrastingly, there was a greater difference between trained and untrained girls than observed in boys for  $SV_{\max}$  (20.0% vs 9.3%) and  $\dot{Q}_{\max}$  (13.5% vs 9.0%). None of the haemodynamic interaction effects persisted when allometrically scaled variables were considered. A

significant sex and training interaction was evident when [HHb] was expressed against absolute  $\dot{V}O_2$ , with a greater difference was found in the *c/d* (20.6% vs 44.8%) and plateau (11.4% vs 15.9%) of trained and untrained girls than boys, respectively. However, when expressed against relative  $\dot{V}O_2$  no interaction effects persisted.

**Table 4.1** – Descriptive characteristics

	Trained (n = 90)		Untrained (n = 79)	
	Boys (n = 47)	Girls (n = 43)	Boys (n = 43)	Girls (n = 36)
Age (years)	14.2 ± 1.9	14.4 ± 1.7	14.8 ± 1.4	14.8 ± 1.4
Height (m)	1.64 ± 0.15	1.62 ± 0.09	1.68 ± 0.11	1.61 ± 0.08
Weight (kg)	52.4 ± 12.9	53.2 ± 9.3	60.0 ± 12.4	54.3 ± 10.4
BMI (kg·m <sup>-2</sup> )	19.3 ± 2.4	20.2 ± 2.2	21.0 ± 3.4	20.6 ± 2.9
Maturity Offset (years)	0.21 ± 1.78	0.54 ± 1.22	0.63 ± 1.31	0.61 ± 1.38
MVPA (mins·day <sup>-1</sup> )	58.6 ± 24.4	48.2 ± 19.0*	50.1 ± 13.0	42.6 ± 12.8*
SED (mins·day <sup>-1</sup> )	554.3 ± 88.6	541.3 ± 74.4	561.3 ± 78.0	578.0 ± 105.4

All values are presented as mean ± SD, BMI = Body Mass Index, MVPA = Moderate-to-Vigorous Physical Activity, SED = Sedentary Time. \* Indicates a significant difference compared to boys

**Table 4.2 - Pulmonary gas exchange and hemodynamic responses to incremental ramp exercise according to training status and sex**

	Trained		Untrained	
	Boys	Girls	Boys	Girls
Peak $\dot{V}O_2$ ( $l \cdot \text{min}^{-1}$ )	2.64 ± 0.70*#	2.11 ± 0.40*	2.39 ± 0.50#	1.90 ± 0.52
Allometrically Scaled Peak $\dot{V}O_2$ ( $\text{ml} \cdot \text{kg}^{-b} \cdot \text{min}^{-1}$ )	192.2 ± 28.6*#	152.5 ± 23.5*	162.3 ± 30.2#	142.6 ± 36.5
GET ( $l \cdot \text{min}^{-1}$ )	1.55 ± 0.49#	1.25 ± 0.26	1.43 ± 0.34#	1.25 ± 0.36
Relative GET (% $\dot{V}O_2$ peak)	59.4 ± 10.3	60.2 ± 10.8	60.4 ± 11.7	64.0 ± 12.5
MRT (s)	32.9 ± 16.2*#	30.7 ± 16.8*	37.3 ± 14.3#	33.8 ± 14.5
Gain ( $\text{ml} \cdot \text{min}^{-1} \cdot \text{W}^{-1}$ )	8.8 ± 2.1#	7.6 ± 1.5	8.7 ± 2.2#	7.8 ± 1.9
Peak Power (W)	219 ± 66*#	205 ± 43*	200 ± 45#	181 ± 43
Cardiac Variables				
$HR_{\text{max}}$ ( $\text{beats} \cdot \text{min}^{-1}$ )	199 ± 11	194 ± 11	193 ± 13	192 ± 13
$SV_{\text{max}}$ (ml)	134.5 ± 53.9*#	130.1 ± 35.3*	123.0 ± 47.4#	108.4 ± 29.3
Allometrically Scaled $SV_{\text{max}}$ ( $\text{ml} \cdot \text{m}^{-b}$ )	74.9 ± 26.9	72.6 ± 22.2	73.8 ± 22.2	71.9 ± 19.8
$\dot{Q}_{\text{max}}$ ( $l \cdot \text{min}^{-1}$ )	21.9 ± 8.5*	20.5 ± 6.6*	19.3 ± 6.8	18.8 ± 5.1
Allometrically Scaled $\dot{Q}_{\text{max}}$ ( $l \cdot \text{m}^{-b} \cdot \text{min}^{-1}$ )	12.5 ± 4.0	12.0 ± 4.2	12.3 ± 3.6	11.8 ± 3.9
a- $\bar{v}O_2$ diff ( $\text{ml} \cdot \text{dl}^{-1}$ )	14.6 ± 6.7#	10.3 ± 2.4	12.9 ± 4.7#	10.8 ± 2.6

GET = Gas Exchange Threshold, MRT = Mean Response Time,  $HR_{\text{max}}$  = Maximum Heart Rate,  $SV_{\text{max}}$  = Maximum Stroke Volume,  $\dot{Q}_{\text{max}}$  = Maximum Cardiac Output, a- $\bar{v}O_2$  diff = arteriovenous difference. All values presented as mean ± SD, \* Indicates a significant difference between training groups within a sex. # Indicates a significant difference between sexes within a training group.

**Table 4.3** – Muscle deoxygenation variables from the incremental ramp exercise according to training status and sex

		Trained		Untrained	
		Boys	Girls	Boys	Girls
[HHb] vs	a (%)	95.9 ± 10.6	97.9 ± 9.5	94.8 ± 6.8	95.0 ± 8.6
absolute $\dot{V}O_2$	d	2.80 ± 1.50*	2.67 ± 1.55*	3.28 ± 2.45	4.10 ± 3.56
	c/d	1.58 ± 0.49*#	1.39 ± 0.45*	1.31 ± 0.58	0.96 ± 0.42
	Plateau	2.05 ± 0.44*#	1.68 ± 0.56*	1.84 ± 0.50	1.45 ± 0.34
[HHb] vs relative	a (%)	95.9 ± 10.6	97.9 ± 29.5	94.8 ± 6.8	95.0 ± 8.6
$\dot{V}O_2$	d	0.07 ± 0.05	0.05 ± 0.03	0.09 ± 0.07	0.06 ± 0.05
(% Peak $\dot{V}O_2$ )	c/d	63.4 ± 15.5	58.4 ± 9.4	54.8 ± 19.0	46.0 ± 13.7
	Plateau	78.1 ± 12.6	71.4 ± 15.5	76.9 ± 16.9	74.9 ± 18.0
[HHb] vs Watts	a (%)	98.5 ± 36.7	95.4 ± 30.2	94.9 ± 11.6	93.6 ± 15.4
(W)	d	0.02 ± 0.02*	0.02 ± 0.01*	0.01 ± 0.01	0.03 ± 0.01
	c/d	100 ± 29*#	91 ± 25*	81 ± 41	70 ± 30
	Plateau	162 ± 64*#	151 ± 63*	131 ± 53	110 ± 31
[HHb] vs Watts	a (%)	98.5 ± 36.7	95.4 ± 30.2	94.9 ± 11.6	93.6 ± 15.4
(% Peak Power)	d	0.03 ± 0.02	0.04 ± 0.02	0.02 ± 0.01	0.04 ± 0.03
	c/d	49.8 ± 17.2	47.8 ± 16.8	36.5 ± 17.0	40.0 ± 16.8
	Plateau	80.8 ± 16.4*	70.6 ± 19.7*	68.8 ± 22.0	60.7 ± 20.3

[HHb] = concentration of haemoglobin, a = sigmoidal amplitude, d = sigmoidal slope, c/d = Value at the mid-point of the sigmoidal response, Plateau = Value at the lower 95% confidence interval of the amplitude. All values presented as mean ± SD, \* Indicates a significant difference between trained and untrained within a sex. # Indicates a significant sex difference within the same training group.

#### 4.4 Discussion

The primary aim of this study was to explore the role of sex in determining the effect of training on  $\dot{V}O_{2\max}$ , haemodynamic variables and oxygen extraction parameters, accounting for maturity. The key finding was that the influence of training during youth was dependent on sex, with greater training-related differences in allometrically scaled  $\dot{V}O_{2\max}$  of girls compared to their male counterparts. Despite this increased magnitude of training-related differences, boys still demonstrated a greater absolute, and scaled,  $\dot{V}O_{2\max}$  than girls, irrespective of training status. The mechanisms underpinning this greater aerobic capacity in boys appear to be related to differences in oxygen extraction, as there was no difference between any haemodynamic ( $SV_{\max}$  and  $\dot{Q}_{\max}$ ) response to exercise according to training status once normalised to account for body size. However, boys had a significantly higher [HHb] plateau when expressed against absolute  $\dot{V}O_2$  and work rate although these sex differences were ameliorated when expressed as a function of relative  $\dot{V}O_2$  and work rate. This therefore suggests that boys and girls have similar levels of oxygen extraction for the same relative sub-maximal work rate and  $\dot{V}O_2$ . These findings therefore provide novel insights into the comparative trainability of youth according to sex which should be considered in long-term athlete development plans.

Training is associated with a significantly higher  $\dot{V}O_{2\max}$  in youth, irrespective of maturity status, and therefore represents a potent stimulus to aerobic fitness (Armstrong, 2015; Baquet et al., 2003; Cao et al., 2019; McNarry et al., 2014b; McNarry et al., 2011b). In accord with previous studies (Cunha et al., 2011; Cunha et al., 2016; McNarry et al., 2014b; McNarry et al., 2011b). Whilst the difference in  $\dot{V}O_{2\max}$  between the training groups is smaller than observed in many training studies (Armstrong, 2015; Cunha et al., 2011; McNarry et al., 2015; Sperlich et al., 2010), the  $\dot{V}O_{2\max}$  values of the untrained group are similar to normative data reported elsewhere for youth in the United Kingdom (Armstrong & Welsman, 2019b; Armstrong, Williams, Balding, Gentle, & Kirby, 1991b). More specifically, Armstrong et al. (1991b) reported male adolescents ( $13.9 \pm 0.3$  years) with no formal exercise training had a  $\dot{V}O_{2\max}$  of  $2.26 \pm 0.59$  l·min<sup>-1</sup>, with adolescent girls ( $14.0 \pm 0.3$  years) achieved a  $\dot{V}O_{2\max}$  of  $1.78 \pm 0.29$  l·min<sup>-1</sup>, similar to the untrained values in the present study (boys:  $2.39 \pm 0.50$  l·min<sup>-1</sup>; girls:  $1.90 \pm 0.52$  l·min<sup>-1</sup>). Whilst the  $\dot{V}O_{2\max}$  values in the

present study are slightly higher than those reported by Armstrong et al. (1991b) it is worth noting that no supramaximal validation bout was performed and so the discontinuous incremental ramp protocol used may have underestimated participants 'true'  $\dot{V}O_{2max}$ . Given the similarities to previous untrained children and adolescents, the results of this study should be interpreted as a reflection of the effect of sex in moderately trained athletes, as oppose to elite level junior athletes.

The present study refutes suggestions that there may be a maturational threshold below which significant responses to training stimuli do not occur (Katch, 1983). However, it is pertinent to note that some studies have failed to find a significant influence of training in pre-pubertal children (Stoedefalke et al., 2000; Welsman et al., 1996; Welsman et al., 1997; Williams et al., 2000). Whilst these contradictory findings are likely attributable to methodological factors, such as a lack of adequate training stimulus (Stoedefalke et al., 2000; Welsman et al., 1996; Welsman et al., 1997) or inappropriate testing modalities for a given training type (Stoedefalke et al., 2000), the contradictory findings may also be related to the influence of sex. Indeed, the majority of early studies reporting no training effects were in girls (Stoedefalke et al., 2000; Welsman et al., 1996; Welsman et al., 1997), with a paucity of studies available directly comparing the trainability of pre-pubertal boys and girls, precluding further interpretations. In the current study, a significant training and sex interaction was observed for allometrically scaled  $\dot{V}O_{2max}$ , suggesting that girls may display a greater magnitude of change in response to a training stimulus compared to boys. This could be due to girls lower baseline fitness levels as higher initial levels are known to attenuate the response to training (McNarry & Jones, 2014). However, this seems unlikely to fully explain such an interaction given the habitually trained nature of these participants and differences in physical activity levels were covaried for in all analyses.

In the current study, boys had a significantly higher  $\dot{V}O_{2max}$  compared to girls (20.2%), even after allometrically scaling for body mass (16.8%). These findings are congruent with previous research in which boys of varying training statuses had a 12.8 – 22.5% higher peak  $\dot{V}O_2$  whether expressed in absolute terms (Bitar et al., 2000; Winsley et al., 2009) or allometrically scaled to body mass (Rowland, 1997; Winsley et al., 2009). The mechanisms underpinning these sex differences in peak  $\dot{V}O_2$  still remain to be



fully elucidated (Armstrong & Welsman, 2019b). Specifically, Rowland et al. (2000a) and Vinet et al. (2003) found a higher  $SV_{\max}$  when quantified by Doppler echocardiography in boys, whereas, in accord with the current study, Winsley et al. (2009) reported no significant differences in  $SV_{\max}$  or  $\dot{Q}_{\max}$  when assessed using bioelectrical thoracic impedance in pre-pubertal children matched for lean body mass. Furthermore, there was no difference in the SV response profile between any group in the present study. Such findings corroborate those of Obert et al. (2003) and Nottin et al. (2002), but contradict those of McNarry et al. (2014b) and may reflect the lower training volume and history of the present participants compared to those in McNarry et al. (2014b) and Rowland et al. (2000b). Alternatively, they may also be attributable a greater change in the peripheral vasculature, as opposed to central oxygen delivery, in response to training, as suggested by Obert et al. (2003).

Extending the findings of Winsley et al. (2009), the present study suggests that changes in oxygen extraction may be more important to the development of peak  $\dot{V}O_2$  and the sexual dimorphism demonstrated. In accord with this conclusion, McNarry et al. (2015) reported sex differences in muscle deoxygenation kinetics, with pre-pubertal girls demonstrating a greater rate of change in the [HHb] response compared to boys. The results of the current study corroborate these results and extend them across the maturational range, with boys, irrespective of training, demonstrating a higher  $c/d$  and plateau when [HHb] was expressed against absolute  $\dot{V}O_2$  or work rate. These observed sex differences may be related to a lower muscle oxidative capacity in girls or to a lower ability to redistribute blood to the metabolically active myocytes at working muscles (Barstow, 2019; Boone et al., 2009; Harper, Ferreira, Lutjemeier, Townsend, & Barstow, 2006). More specifically, research has reported that a greater proportion of blood flow in women is directed to the respiratory muscles (Smith, Hageman, Harms, Poole, & Musch, 2017). Moreover, women have been reported to have a higher cost of breathing compared to men and consequently may have less of a reserve to redistribute blood, and subsequently oxygen, to the peripheral vasculature (Smith et al., 2017), but there is scant research investigating whether this is also true in children.

Despite sex differences in the [HHb] response when expressed against absolute work rate and  $\dot{V}O_2$ , when the [HHb] response was expressed against relative  $\dot{V}O_2$  or work rate, the sex difference was ameliorated. This therefore suggests that for the same

relative sub-maximal work rate, boys and girls have a similar response pattern (Barstow, 2019; Boone et al., 2009), but the normalisation of  $\dot{V}O_2$  and work rate to peak values does not necessarily indicate boys and girls experience the same relative intensity of exercise. Indeed, the intensity of exercise is dependent upon the GET and critical power thresholds (Anderson & Mahon, 2007; Beaver et al., 1986; Mahon & Cheatham, 2002), which should be considered in future work. Nevertheless, given the similarity in the GET between boys and girls, irrespective of training status, this study tentatively suggests that oxygen extraction capacity at sub-maximal intensities remains similar throughout maturity in boys and girls. Therefore, further research is required to determine the mechanistic basis for the sex differences in peak  $\dot{V}O_2$ .

Few studies have considered the effect of sex, or its interaction with training, on the sub-maximal parameters of aerobic fitness, with the exception of the GET for which evidence consistently reports that, when normalised to peak  $\dot{V}O_2$ , no sex differences are manifest (Mahon & Cheatham, 2002; McNarry et al., 2011b; Obert et al., 2000; Reybrouck, Weymans, Stijns, Knops, & Van der Hauwaert, 1985). Given the sex differences evident in peak  $\dot{V}O_2$ , the absence of sex-related differences in the GET is interesting and suggests that the mechanisms for the greater peak  $\dot{V}O_2$  in boys are unrelated to those that determine the GET. The current findings that the GET was not affected by training status agree with the findings of McNarry et al. (2011b) who similarly reported that the relative GET was similar during lower body exercise irrespective of maturity status. Similarly, the MRT of the trained participants was significantly faster than their untrained counterparts in accordance with the majority of evidence available regarding the influence of training on the dynamic  $\dot{V}O_2$  response to constant work rate exercise (Breese et al., 2019; Breese et al., 2010; Marwood et al., 2010; Willcocks et al., 2010), although it is pertinent to note that the  $\dot{V}O_2$  response to incremental ramp and constant work rate exercise, and its determinants, could be dissociated. Whilst the mechanistic basis of training related adaptations on  $\dot{V}O_2$  kinetics remain largely to be resolved (McNarry, 2019) purported mechanisms include a faster  $O_2$  delivery (Marwood et al., 2010) and an increased muscular oxidative enzyme and mitochondrial volume (Eriksson, 1980; McNarry, 2019).

The similar gain in the trained youth is however counter-intuitive, with previous studies in adults suggesting that the proportion of type I fibres and possibly fitness are

associated with a greater gain (Barstow et al. 1999). It is therefore interesting to note the greater gain reported in the boys in the current study irrespective of training status, compared to girls. This finding is in accord with the suggestions of van Praagh et al. (2000) and Doré et al. (2005) that boys are generally characterised by a greater proportion of type I muscle fibres. Given the discordance in the present training and testing modality, the discrepant findings with regard to the influence of training on the submaximal parameters of aerobic fitness may also be methodological, rather than physiological, but further research is needed before further conclusions can be drawn.

Whilst there are strengths associated with the present study, including a large sample of trained and untrained children and adolescents, the attainment of a true  $\dot{V}O_{2\max}$ , and the use of appropriate scaling techniques, there are some limitations that must be acknowledged. First, given the sex differences in body composition, more insight may have been gained by scaling by fat free mass or lean body mass. Further, all trained children and adolescents were part of similar training regimes, namely football and hockey, precluding any inferences being drawn regarding the effect of different training types on the magnitude of responses. Additionally, the interpretation of the [HHb] signal has specific methodological limitations, including the generalisability of response dynamics from a singular localised area to the whole muscle (Barstow, 2019), and variations in adiposity between boys and girls possibly contributing to signal differences (Gurley et al., 2012; La Mantia et al., 2018). Moreover, whilst research has suggested that a seven-day measurement period is indicative of children and adolescents habitual physical activity levels, others have suggested they may not reflect habitual physical levels appropriately (Troost et al. 2015) and therefore these results should be interpreted with caution. Finally, not splitting sex and training groups by maturity precludes inferences as to whether the observed differences are consistent across maturity, or whether there are periods of divergence.

In conclusion, boys had a greater peak  $\dot{V}O_2$  than girls, irrespective of training status, which does not appear to be related to differences in oxygen delivery as  $SV_{\max}$  and  $\dot{Q}_{\max}$  were similar between sexes when appropriately normalised to body surface area. This study indicates that the sex and training differences in peak  $\dot{V}O_2$  may rather be due to an enhanced oxygen extraction at the working muscles in boys, irrespective of training status. Future research should seek to establish sex differences across

individual maturational stages, and investigate the potential mechanisms underpinning peak  $\dot{V}O_2$  development in youth.

# Chapter 5

The combined associations of physical activity, sedentary time, and sleep on  $\dot{V}O_{2\max}$  in trained and untrained children and adolescents:

A novel five-part compositional analysis approach

## **Chapter 5 (Study 2) - The combined associations of physical activity, sedentary time and sleep on $\dot{V}O_{2max}$ in trained and untrained children and adolescents: A novel five-part compositional analysis approach**

### **5.1 Introduction**

Poor maximal oxygen uptake ( $\dot{V}O_{2max}$ ) has been associated with an increased risk of cardiovascular and metabolic disease, leading to an increased likelihood of premature mortality across the lifespan (Hallal et al., 2012; Laukkanen et al., 2016; Lee et al., 1995; Paffenbarger & Lee, 1998).  $\dot{V}O_{2max}$ , defined as the highest rate of oxygen consumption despite further increases in work rate (Hill & Lupton, 1923), is also key to athletic performance, with youth athletes consistently reported to have a greater  $\dot{V}O_{2max}$  than their untrained counterparts (Armstrong, 2017; Carazo-Vargas & Moncada-Jiménez, 2015; McNarry et al., 2014b; McNarry, Welshman, & Jones, 2011a). Whilst training is well-established to improve  $\dot{V}O_{2max}$  in youth athletes (Armstrong, 2015; Armstrong & Barker, 2011; Baquet et al., 2003; Carazo-Vargas & Moncada-Jiménez, 2015), what remains less clear is the influence of habitual physical activity (PA) and sedentary time (SED) on  $\dot{V}O_{2max}$ . More specifically, some studies have reported a significant association between PA and  $\dot{V}O_{2max}$  (Dencker & Andersen, 2011; Dencker et al., 2007; Dencker et al., 2006; Gutin et al., 2005; Latt et al., 2013), whereas others argue that children and adolescents rarely experience PA of a sufficient duration and intensity to significantly influence  $\dot{V}O_{2max}$  (Armstrong et al., 2011; Armstrong & Welsman, 2019d).

Moderate-to-vigorous PA (MVPA) is perhaps the most widely used PA metric in children and adolescents (Carson et al., 2019; Chastin et al., 2015; Ekelund et al., 2001; Lynch et al., 2019). However, combining MPA and VPA may potentially mask the importance of the intensity of physical activity for improving  $\dot{V}O_{2max}$ . Indeed, training studies consistently show that significant improvements in absolute, and allometrically scaled,  $\dot{V}O_{2max}$  only occur when the intensity is sufficiently vigorous (Baquet et al., 2003; Cao et al., 2019; Milanović et al., 2015), with Gutin et al. (2005) reporting a stronger association between VPA and  $\dot{V}O_{2max}$  ( $r^2 = 0.43$ ,  $p < 0.01$ ) than

MPA ( $r^2 = 0.30$ ,  $p < 0.01$ ) in adolescents. These findings have subsequently been corroborated by both Dencker et al. (2006) and Latt et al. (2013) who reported that the amount of time spent in VPA explained 9.0 – 15.8% of the variance in  $\dot{V}O_{2max}$  in children and adolescents. However, the use of ratio scaling to account for the effect of body mass on  $\dot{V}O_{2max}$  in these earlier studies largely precludes meaningful interpretations, especially with regard to the influence of maturity and/or sex, given the consistent evidence that ratio scaling  $\dot{V}O_{2max}$  penalises heavier, more mature, individuals (Nevill et al., 2006; Tanner, 1949; Welsman & Armstrong, 2019). Additionally, the reliance on correlational statistics, which cannot infer causality (Hopkins et al., 2009; Pearson, 1896), and the use of predictive linear regressions, which assume independence between variables (Chastin et al., 2015), are inappropriate to account for the constrained and co-dependent nature of PA data, potentially creating spurious associations.

Compositional analysis allows all movement behaviours to be expressed as a proportion of a finite period, enabling the individual, and combined, effects of movement behaviours on outcome variables to be established (Carson et al., 2016; Carson et al., 2019; Chastin et al., 2015). Thus, compositional analysis could provide novel insights into the influence of movement behaviour intensity and volume on peak  $\dot{V}O_2$ . Carson et al. (2016) found that the overall PA composition explained ~38% of the variance in the  $\dot{V}O_{2max}$  of 4,169 Canadian children and adolescents (8 – 17 years). Despite this, when 10 minutes of time was allocated to, or removed from, MVPA, there was a negligible effect on  $\dot{V}O_{2max}$ , with predicted changes ranging from 0.03% - 0.05% (Carson et al., 2016). These insignificant changes in aerobic fitness could be attributed to the independent effect of SED, highlighting the need for an integrated, as opposed to segregated, approach to fully examine the relationship between PA, SED and  $\dot{V}O_{2max}$  (Carson et al., 2016). However, whilst Carson et al. (2016) provided the first insights using a compositional approach to the relationship between PA and  $\dot{V}O_{2max}$ , certain methodological limitations mean further research is warranted to establish the relationship between PA and  $\dot{V}O_{2max}$ . Specifically, the estimation of  $\dot{V}O_{2max}$  from a field-based test which is likely to misrepresent true cardiorespiratory fitness (Armstrong & Welsman, 2020b), the pooling of data from boys and girls despite the well-established physiological differences (Armstrong, 2007; Bitar et al.,

2000; Rogol, 2002), and the failure to account for maturity or training status differences between participants limit the interpretation of these earlier results.

Therefore, the aim of this study was to examine the independent, and interactive, effects of the five movement behaviours on  $\dot{V}O_{2\max}$ . The second aim was to explore the effect of baseline fitness, sex, and maturity on the predicted changes in  $\dot{V}O_{2\max}$  elicited by changing PA compositions.

## 5.2 Methods

Ethics approval was granted by the institutional research ethics committee prior to the commencement of data collection and the study conformed to the Declaration of Helsinki. Before participants were accepted into the study, written informed parental consent and participant assent were obtained, along with all parents completing a pre-screening medical questionnaire on behalf of their child. Participants were excluded if they had known cardiovascular, metabolic, kidney, or any other disease that meant they would not have been able to complete the exercise protocol. The trained children and adolescents were all national level athletes who were part of a long-term athlete development (LTAD) program overseen by the national governing body (NGB) of their sport (Hockey, Football and Gymnastics). Untrained participants were recruited from local schools across South Wales and were not formally engaged in sport training outside of curricular physical education lessons. The final sample consisted of 237 participants encompassing 108 trained (43 girls; age:  $13.5 \pm 2.1$  years) and 129 untrained (51 girls;  $13.8 \pm 1.4$  years) children and adolescents.

### *5.2.1 Experimental Procedures*

All participants were required to attend one session at which they initially had their stature and sitting height measured to the nearest 0.1 cm using a Holtain Stadiometer (Holtain, Crymych, Dyfed, UK) and their body mass measured to the nearest 0.1 kg using electronic scales (Seca 803, Seca, Chino, CA, USA). Maturity status was subsequently estimated using the equations of Mirwald et al. (2002a), with participants deemed pre-pubertal, pubertal and post-pubertal if they were more than one year from, within one year of, or more than one year post peak height velocity (PHV), respectively.



$\dot{V}O_{2\max}$  was assessed using an incremental ramp test to volitional exhaustion on a cycle ergometer (Lode Excalibur Sport, Groningen, Netherlands) which started with a three-minute warm-up at 10 W before increasing by 20 – 25 W·min<sup>-1</sup>, depending on the participant's age. All participants were instructed to maintain a cadence of 60 – 80 revolutions per minute (rpm) throughout the test, with volitional exhaustion defined as when participants could not maintain a cadence above 50 rpm. Inspired and expired air were measured on a breath-by-breath basis throughout the incremental ramp test using a Vyntus metabolic cart (VYAIR medical Ltd, Mettawa, IL, USA). Following five minutes active and ten minutes passive rest, a supramaximal validation bout was performed (Barker et al., 2009). Specifically, participants warmed up for a further three minutes at 10 W before a step-transition to 105% of the peak power achieved during the incremental ramp test. Participants were instructed to maintain a cadence above 50 rpm for as long as possible, with gas exchange measured continuously on a breath-by-breath basis throughout the exercise bout.

Participant's habitual physical activity was subsequently measured for seven consecutive days using a ActiGraph GT3X (ActiGraph, Pensacola, Florida, USA) worn on the right hip, sampling at 100 Hz. Children and adolescents also completed a seven-day log to detail periods when the monitor was removed, waking time and time going to bed, to minimise the misclassification of non-wear time as sedentary time or sleep.

### 5.2.2 Data Analyses

The raw breath-by-breath  $\dot{V}O_2$  data from both the  $\dot{V}O_{2\max}$  and supramaximal bout were averaged into 10-second bins, with  $\dot{V}O_{2\max}$  defined as the highest 10-second moving average during the ramp incremental test. To aid comparisons between sex, maturity and training sub-groups,  $\dot{V}O_{2\max}$  was allometrically scaled to account for body mass differences between participants (Welsman & Armstrong, 2019). The scaling exponent for this study was 0.76 (95%CI: 0.73 – 0.79). Evenson et al. (2008) cut-points were utilised to determine the time spent in each PA intensity which have been shown to be the most reliable for children and adolescents (Trost, 2016; Trost et al., 2011). Sleep time and efficiency was calculated using the algorithms of Sadeh et al. (1994b). Wear-time criteria was set as  $\geq 8$  hours on any three days (Herrmann et al., 2014; Rich et al., 2013). The smallest worthwhile change (SWC) in peak  $\dot{V}O_2$  (l·min<sup>-1</sup>

<sup>1</sup>) and allometrically scaled peak  $\dot{V}O_2$  ( $\text{ml}\cdot\text{kg}^{-b}\cdot\text{min}^{-1}$ ) was calculated for each sex, maturity and training sub-group using the formula  $0.2\cdot\text{group SD}$  (Hopkins, 2000). The SWC was then subsequently presented as a percentage of the group mean to aid comparisons between all sub-groups.

All compositional analyses were conducted in R (<http://cran.r-project.org>) using the compositions package (version 1.40-2) and its dependencies (Chastin et al., 2015). Compositional geometric means were computed to indicate the amount of time spent in each PA behaviour or sleep each day, by expressing each behaviour, after normalisation, as a proportion of the total time (Carson et al., 2016; Chastin et al., 2015). Variance matrices were calculated to provide an indication as to the dispersion and co-dependency of movement behaviours and were calculated by measuring the variance between pair-wise log ratios (Carson et al., 2016; Chastin et al., 2015). Specifically, a ratio tending towards zero indicates high co-dependency, with the numbers further from zero indicating less co-dependency. Sequential linear regression models were created by rotating each of the five behaviours via isometric log ratio (ILR) transformations to examine the relative effect of all movement behaviours on the peak  $\dot{V}O_2$  and allometrically scaled peak  $\dot{V}O_2$  (Carson et al., 2016; Chastin et al., 2015). The first coefficient and its  $p$  value were reported for each rotation to determine whether the individual movement behaviour was associated with the outcome variable relative to the other movement behaviours, and its relative significance. Additionally, the overall model significance ( $p$  value) and  $R^2$  value were reported to gain an insight into the variance explained by the overall movement composition. All movement behaviours were also sequentially mapped against each other, producing ternary heat maps displaying the predicted absolute and allometrically scaled peak  $\dot{V}O_2$  for each sex, training and maturity group. Finally, change matrices were conducted to predict the change in absolute and scaled peak  $\dot{V}O_2$  by systematically reallocating 10 minutes from one movement behaviour to another (Carson et al., 2016; Chastin et al., 2015). All predictive changes were presented as a percentage change relative to the compositional mean, with significant changes identified as any change greater than the SWC (%).

### 5.2.3 Statistical Analyses

All traditional statistical analyses were conducted in SPSS version 26 (IBM, Portsmouth, UK), with significance accepted as  $p < 0.05$ . Between group differences in anthropometric characteristics and absolute and allometrically scaled peak  $\dot{V}O_2$  were assessed using a MANOVA, with post-hoc tests with Bonferroni correction applied to identify the specific location of significant differences as appropriate.

### 5.3 Results

Of the original 237 participants, 61 were excluded for failing to meet the wear-time criteria, therefore 84 trained (40 girls) and 92 untrained (44 girls) children and adolescents were included in the final analyses. There were no significant differences in the anthropometrics of those included and excluded ( $p > 0.05$ ). Post-pubertal adolescents were significantly older, taller, heavier, and more mature than the pubertal or pre-pubertal children ( $p < 0.01$ ), with significant differences in the same parameters also evident between pubertal adolescents and pre-pubertal children ( $p < 0.05$ , Table 1). The trained children and adolescents were taller ( $F_{(1,175)} = 12.7$ ,  $p < 0.01$ ) and had a higher  $\dot{V}O_{2\max}$  ( $\text{l}\cdot\text{min}^{-1}$ ) than their untrained counterparts ( $F_{(1, 175)} = 15.3$ ,  $p < 0.01$ ), which persisted even after allometric scaling ( $F_{(1,175)} = 18.7$ ,  $p < 0.01$ ). Overall, boys had a higher  $\dot{V}O_{2\max}$  and allometrically scaled  $\dot{V}O_{2\max}$  than their female counterparts, irrespective of training and maturity status ( $F_{(1,175)} = 19.7$ ,  $p < 0.01$ ).  $\dot{V}O_{2\max}$  increased with maturity, irrespective of sex and training status ( $F_{(1,175)} = 16.2$ ,  $p < 0.01$ ), but there was no significant difference between any maturity group for allometrically scaled  $\dot{V}O_{2\max}$ . There were no significant training, sex, or maturity interactions for any anthropometric variable or peak  $\dot{V}O_2$ , regardless of how it was expressed.

In the trained participants, the geometric means highlight that the biggest portion of the day was spent in SED (41.2%), followed by sleep (39.2%), with VPA only accounting for 1.6% of the day (Table 2). Similarly, untrained children spent the longest periods of the day in SED (44.1%) and sleep (40.0%), with VPA making up just 1.3% of the day. Trained athletes completed more LPA ( $F_{(1,175)} = 38.1$ ,  $p < 0.01$ ) and VPA ( $F_{(1,175)} = 18.6$ ,  $p < 0.01$ ), but spent significantly less time sleep ( $F_{(1,175)} = 3.8$ ,  $p = 0.05$ ) compared to untrained participants, irrespective of sex or maturity. LPA

and sleep, and SED and LPA, demonstrated the smallest variation and therefore high co-dependency, whereas VPA had the largest pair-wise log ratio variances compared to all other PA behaviours, indicating less co-dependency (Table 3). The ILR model revealed that the composition of PA, SED and sleep significantly predict both  $\dot{V}O_{2\max}$  and allometrically scaled  $\dot{V}O_{2\max}$  (Table 4). Additionally, the overall movement composition explained 48.7% and 37.7% of the variance in peak, and allometrically scaled,  $\dot{V}O_{2\max}$ , respectively. In isolation, when compared against all other PA intensities, only VPA significantly predicted allometrically scaled  $\dot{V}O_{2\max}$  ( $Y_{VPA} = 6.91$ ,  $p < 0.02$ ), with no significant individual associations evident for  $\dot{V}O_{2\max}$ .

VPA was the most influential PA behaviour for absolute, and allometrically scaled,  $\dot{V}O_{2\max}$ , irrespective of training status and sex, but the influence of PA behaviours was less clear in pubertal and post-pubertal adolescents (Figures 1 and 2). VPA continued to dominate the heat maps when expressed against SED and sleep (Appendix 11.2.2 – 11.2.5). Reallocating 10 minutes from any given movement behaviour to any other behaviour had minimal effect on absolute  $\dot{V}O_{2\max}$  in trained children and adolescents, with all changes smaller than the percentage SWC (Appendix 11.2.1). The only exception was in trained pubertal girls in whom displacing 10 minutes of VPA to any other activity behaviour decreased absolute  $\dot{V}O_{2\max}$  by 6.3 – 8.0% (Table 5). In pre-pubertal untrained children, displacing VPA to other movement behaviours decreased  $\dot{V}O_{2\max}$  by 3.2 – 10.5% (Table 6). Additionally,  $\dot{V}O_{2\max}$  was also predicted to increase by 5.2% and 5.8% when LPA was re-allocated to MPA and VPA, respectively, in pre-pubertal untrained girls.

Allometrically scaled  $\dot{V}O_{2\max}$  significantly decreased, irrespective of sex, maturity or training status, when 10 minutes of VPA was reallocated to any other behaviour (Tables 5 and 6). Moreover, allometrically scaled peak  $\dot{V}O_2$  tended to increase when time spent in other movement behaviours was reallocated to VPA. The effect of reallocating time to LPA, SED, or sleep on peak  $\dot{V}O_2$  was negligible, irrespective of how  $\dot{V}O_{2\max}$  was expressed, sex, maturity, or training status.

**Table 5.1** – Participant descriptives

Training Group	Maturity	Sex	Age (years)	Stature (cm)	Body Mass (kg)	BMI (kg·m <sup>-2</sup> )	Maturity Offset (years)	Peak $\dot{V}O_2$ (l·min <sup>-1</sup> )	Scaled Peak $\dot{V}O_2$ (ml·kg <sup>-b</sup> ·min <sup>-1</sup> )
Trained (n = 84)	Pre-Pubertal (n = 34)	Boys (n = 20)	11.8 ± 1.0	146.6 ± 7.5	38.5 ± 7.2	17.8 ± 2.2	-2.27 ± 0.69	2.06 ± 0.37 *	194.1 ± 22.2 *
		Girls (n = 14)	11.4 ± 1.5	151.0 ± 14.2	45.5 ± 13.7	19.4 ± 2.7	-2.37 ± 1.16	1.89 ± 0.46	153.9 ± 23.7
	Pubertal (n = 30)	Boys (n = 14)	14.1 ± 1.1 <sup>a</sup>	167.9 ± 8.9 <sup>a</sup>	53.3 ± 5.8 <sup>a</sup>	18.9 ± 1.3	+0.01 ± 0.43 <sup>a</sup>	2.77 ± 0.43 <sup>*a</sup>	185.7 ± 31.6 *
		Girls (n = 16)	14.4 ± 1.3 <sup>a</sup>	164.6 ± 4.4 <sup>a</sup>	56.4 ± 8.1 <sup>a</sup>	20.7 ± 2.4	+0.02 ± 0.57 <sup>a</sup>	2.16 ± 0.27 <sup>a</sup>	152.5 ± 15.5
	Post-Pubertal (n = 20)	Boys (n = 10)	16.2 ± 1.4 <sup>ab</sup>	178.9 ± 6.9 <sup>ab</sup>	65.2 ± 6.1 <sup>ab</sup>	20.4 ± 1.9	+1.96 ± 0.57 <sup>ab</sup>	3.24 ± 0.71 <sup>*ab</sup>	205.5 ± 34.0 *
		Girls (n = 10)	15.8 ± 1.0 <sup>ab</sup>	165.8 ± 5.7 <sup>ab</sup>	58.9 ± 7.9 <sup>ab</sup>	21.5 ± 3.7	+1.91 ± 0.32 <sup>ab</sup>	2.30 ± 0.45 <sup>ab</sup>	145.7 ± 31.2
Untrained (n = 92)	Pre-Pubertal (n = 22)	Boys (n = 12)	12.3 ± 1.7	151.5 ± 8.1	44.3 ± 10.2 <sup>#</sup>	19.2 ± 3.1 <sup>#</sup>	-1.94 ± 0.94	1.94 ± 0.29 <sup>#*</sup>	142.6 ± 34.7 <sup>#*</sup>
		Girls (n = 10)	12.1 ± 0.7	150.0 ± 10.9	44.9 ± 9.7 <sup>#</sup>	20.0 ± 1.4 <sup>#</sup>	-1.12 ± 0.12	1.35 ± 0.33 <sup>#</sup>	123.9 ± 25.6 <sup>#</sup>
	Pubertal (n = 40)	Boys (n = 26)	14.1 ± 0.9 <sup>a</sup>	164.8 ± 8.2 <sup>a</sup>	57.1 ± 11.2 <sup>#a</sup>	20.9 ± 3.6 <sup>#</sup>	-0.04 ± 0.66 <sup>a</sup>	2.31 ± 0.47 <sup>#*a</sup>	159.5 ± 34.5 <sup>#*</sup>
		Girls (n = 14)	13.1 ± 1.0 <sup>a</sup>	155.8 ± 9.3 <sup>a</sup>	49.4 ± 11.3 <sup>#a</sup>	20.6 ± 3.4 <sup>#</sup>	+0.13 ± 0.38 <sup>a</sup>	1.65 ± 0.20 <sup>#a</sup>	130.9 ± 20.8 <sup>#</sup>
	Post-Pubertal (n = 30)	Boys (n = 10)	15.3 ± 0.32 <sup>ab</sup>	172.0 ± 5.9 <sup>ab</sup>	70.4 ± 14.1 <sup>#ab</sup>	23.7 ± 3.7 <sup>#</sup>	+1.66 ± 0.69 <sup>ab</sup>	2.91 ± 0.62 <sup>#*ab</sup>	166.1 ± 22.6 <sup>#*</sup>
		Girls (n = 20)	14.9 ± 0.7 <sup>ab</sup>	162.3 ± 7.6 <sup>ab</sup>	56.6 ± 10.2 <sup>#ab</sup>	21.6 ± 3.0 <sup>#</sup>	+2.10 ± 0.62 <sup>ab</sup>	1.86 ± 0.38 <sup>#ab</sup>	143.8 ± 34.2 <sup>#</sup>

All values presented as mean ± standard deviation. BMI = Body Mass Index. <sup>#</sup> highlights a significant difference between training groups of the same sex and maturity. <sup>a</sup> significantly different compared to pre-pubertal children, <sup>b</sup> Significantly different compared to pubertal adolescents. <sup>\*</sup>significant difference between boys and girls of the same training and maturity group

**Table 5.2** – Geometric Means for the whole sample

Trained Athletes			
	Overall Mean (minutes·day <sup>-1</sup> )	Geometric Mean (minutes·day <sup>-1</sup> )	% of 24 hours
SED	515.6	594.4	41.2
LPA	186.4	214.9	14.9
MPA	37.4	43.1	3.0
VPA	19.9	22.9	1.6
sleep	489.7	564.5	39.2
Untrained Controls			
SED	517.2 *	634.5 *	44.1 *
LPA	134.4	164.9	11.5
MPA	37.9	46.5	3.2
VPA	15.2 *	18.6 *	1.3 *
sleep	469.7	575.5	40.0

SED = Sedentary Time, LPA = Light Physical Activity, MPA = Moderate Physical Activity, VPA = Vigorous Physical Activity, \*Indicates significant difference between training groups

**Table 5.3** – Pair-wise log ratio variation matrix in the full sample

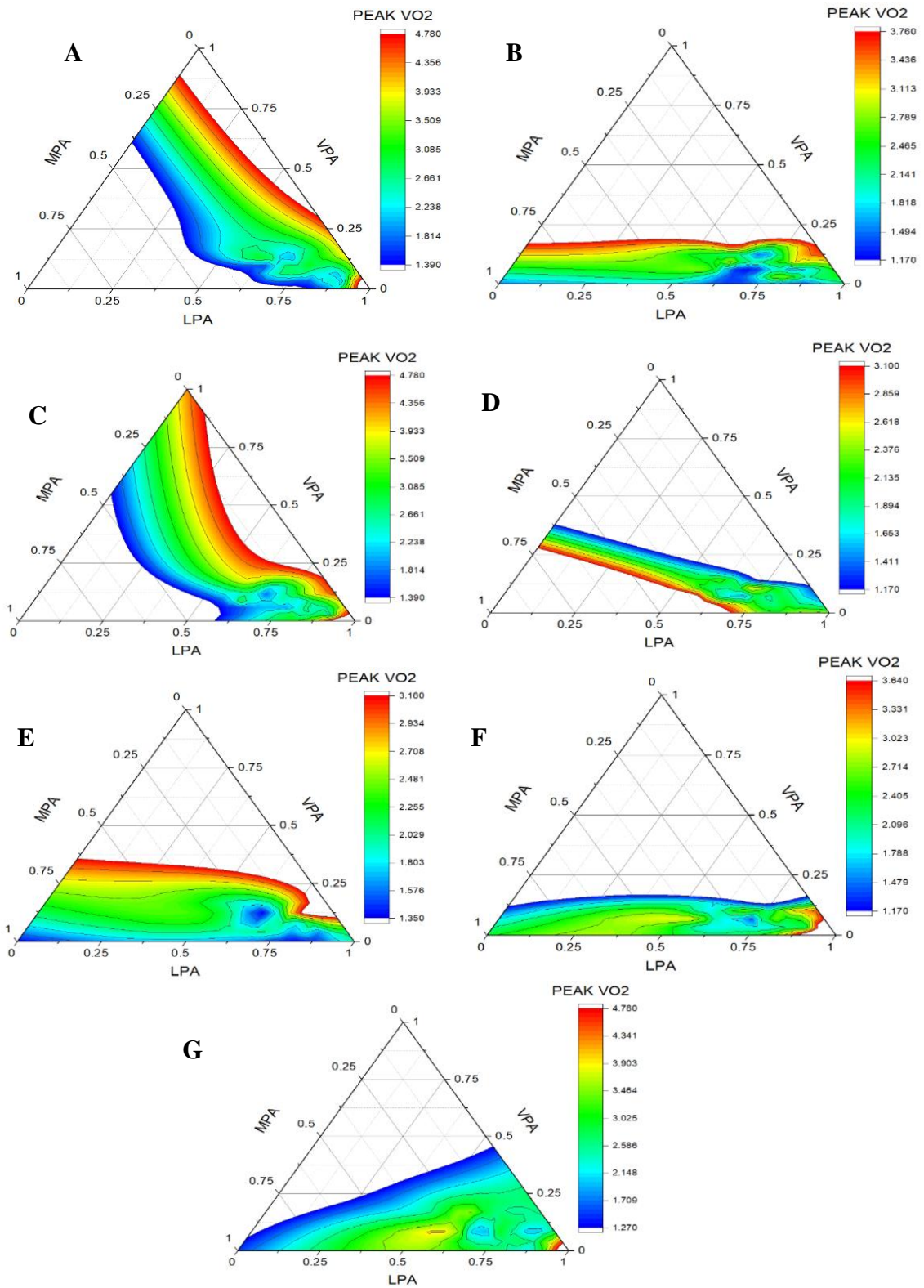
	SED	LPA	MPA	VPA	sleep
SED	-	-0.018	-0.029	-0.053	0.023
LPA	-0.018	-	-0.039	-0.042	-0.016
MPA	-0.029	-0.039	-	-0.020	-0.019
VPA	-0.053	-0.042	0.020	-	-0.045
sleep	0.023	-0.016	-0.019	-0.045	-

SED = Sedentary Time, LPA = Light Physical Activity, MPA = Moderate Physical Activity, VPA = Vigorous Physical Activity,

**Table 5.4** – Model isometric log ratio parameters for  $\dot{V}O_{2max}$  and scaled  $\dot{V}O_{2max}$ 

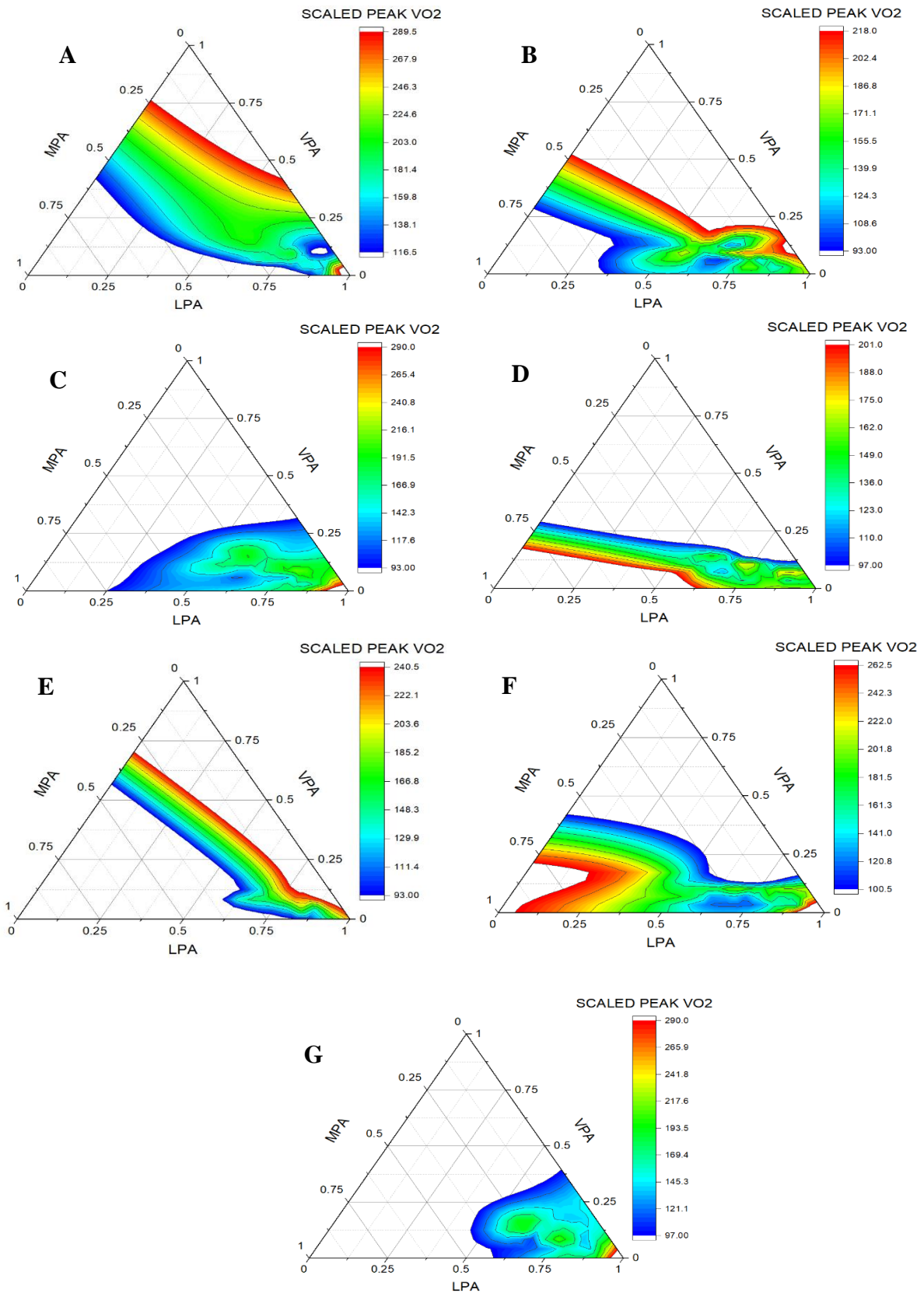
	Model p value	Model R <sup>2</sup>	Y <sub>SED</sub>	p	Y <sub>LPA</sub>	p	Y <sub>MPA</sub>	p	Y <sub>VPA</sub>	p	Y <sub>Sleep</sub>	p
$\dot{V}O_{2max}$ (l·min <sup>-1</sup> )	< 0.001 *	0.486	< 0.001	0.993	-0.070	0.112	0.041	0.483	0.029	0.529	< 0.001	0.997
Scaled $\dot{V}O_{2max}$ (ml·kg <sup>-b</sup> ·min <sup>-1</sup> )	< 0.001 *	0.377	-0.627	0.865	2.390	0.396	-5.606	0.136	6.914	0.019*	1.709	0.668

All models were covaried for training status, sex and maturity. \* indicates a significant predictor of outcome variable.  $\dot{V}O_{2max}$  = Maximal Oxygen Uptake, SED = Sedentary Time, LPA = Light Physical Activity, MPA = Moderate Physical Activity, VPA = Vigorous Physical Activity.



**Figure 5.1** - Ternary heat plots of all physical activity behaviours with expected peak  $\dot{V}O_2$  values for all sub-groups with a) trained athletes; b) untrained controls; c) all boys; d) all girls; e) pre-PHV children; f) circa-PHV adolescents; and g) post-PHV adolescents





**Figure 5.2** – Ternary plots of all physical activity behaviours with expected scaled peak  $\dot{V}O_2$  values for all sub-groups with a) trained athletes; b) untrained controls; c) all boys; d) all girls; e) pre-PHV children; f) circa-PHV adolescents; and g) post-PHV adolescents

**Table 5.5** – Change matrices of reallocating 10 minutes from the behaviour in columns to the behaviour in the rows on  $\dot{V}O_{2max}$  ( $l \cdot min^{-1}$ ) and scaled  $\dot{V}O_{2max}$  ( $ml \cdot kg^{-b} \cdot min^{-1}$ ) in trained children and adolescents, presented as percentage change

$\dot{V}O_{2max}$						Scaled $\dot{V}O_{2max}$					
Pre-Pubertal Boys											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.31	-1.11	-1.59	0.01	SED	-	0.11	1.78	-4.58 *	-0.06
LPA	-0.31	-	-1.42	-1.90	-0.31	LPA	-0.11	-	1.67	-4.69 *	-0.16
MPA	0.87	1.12	-	-0.72	0.87	MPA	-1.39	-1.27	-	-5.97 *	-1.45
VPA	1.01	1.33	-0.10	-	1.01	VPA	2.91 *	3.02 *	4.69 *	-	2.85 *
Sleep	-0.01	0.32	-1.11	-1.59	-	Sleep	0.05	0.17	1.84	-4.52 *	-
Pre-Pubertal Girls											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.49	-1.80	-3.21	0.01	SED	-	0.15	2.49	-7.96 *	-0.06
LPA	-0.46	-	-2.25	-3.67	0.49	LPA	-0.14	-	2.35	-8.10 *	0.12
MPA	1.35	1.84	-	-1.86	1.35	MPA	-1.88	-1.72	-	-9.83 *	-1.94
VPA	1.73	2.21	-0.07	-	1.73	VPA	4.29 *	4.45 *	6.79 *	-	4.23 *
Sleep	-0.01	0.49	-1.80	-3.21	-	Sleep	0.06	0.22	2.56	-7.90 *	-
Pubertal Boys											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.33	-0.87	-1.28	0.01	SED	-	0.15	1.70	-4.47 *	-0.05
LPA	-0.31	-	-1.18	-1.59	-0.31	LPA	-0.13	-	1.56	-4.61 *	-0.19
MPA	0.69	1.02	-	-0.59	0.69	MPA	-1.34	-1.19	-	-5.81 *	-1.39
VPA	0.82	1.15	-0.06	-	0.82	VPA	2.86	3.00	4.56 *	-	2.80
Sleep	-0.01	0.33	-0.87	-1.28	-	Sleep	0.05	0.20	1.75	-4.41 *	-

Pubertal Girls											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.45	-1.79	-7.60 *	0.01	SED	-	0.19	3.24 *	-24.4 *	-0.07
LPA	-0.43	-	-1.34	-8.03 *	-0.43	LPA	-0.18	-	3.46 *	-24.6 *	-0.24
MPA	1.27	1.72	-	-6.33 *	1.27	MPA	-2.28 *	-2.10 *	-	-26.7 *	-2.35 *
VPA	1.98	2.44	0.19	-	1.98	VPA	6.38 *	6.57 *	9.62 *	-	6.31 *
Sleep	-0.01	0.45	-1.80	-7.60 *		Sleep	0.07	0.25	3.30 *	-24.3 *	-
Post-Pubertal Boys											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.32	-0.96	-1.11	0.01	SED	-	0.16	2.03	-4.21 *	-0.05
LPA	-0.30	-	-1.26	-1.41	-0.30	LPA	-0.15	-	1.88	-4.36 *	-0.20
MPA	0.71	1.03	-	0.40	0.72	MPA	-1.51	-1.35	-	-5.72 *	-1.56
VPA	0.71	1.03	-0.25	-	0.71	VPA	2.68	2.84	4.71 *	-	2.63
Sleep	-0.01	0.32	-0.96	-1.12	-	Sleep	0.05	0.21	2.08	-4.16 *	-
Post-Pubertal Girls											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.41	-1.25	-1.89	0.01	SED	-	0.19	2.50	-6.75 *	-0.06
LPA	-0.38	-	-1.88	-2.27	-0.38	LPA	-0.18	-	2.32	-6.93 *	0.23
MPA	0.92	1.32	-	-0.97	0.90	MPA	-1.84	-1.65	-	-8.59 *	-1.90
VPA	1.05	1.46	-2.27	-	1.05	VPA	3.77	3.96	6.27 *	-	3.71
Sleep	-0.01	0.40	-0.97	-1.89	-	Sleep	0.06	0.25	2.48	-6.69 *	-

SED = Sedentary time, LPA = Light Intensity Physical Activity, MPA = Moderate Physical Activity, VPA = Vigorous Physical Activity. All figures presented as percentage change with \* indicating a change above the Smallest Worthwhile Change (%).

**Table 5.6** – Change matrices of reallocating 10 minutes from the behaviour in columns to the behaviour in the rows on  $\dot{V}O_{2max}$  ( $l \cdot min^{-1}$ ) and scaled  $\dot{V}O_{2max}$  ( $ml \cdot kg^{-b} \cdot min^{-1}$ ) in untrained children and adolescents, presented as percentage change

$\dot{V}O_{2max}$						Scaled $\dot{V}O_{2max}$					
Pre-Pubertal Boys											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.63	-1.34	-3.17 *	0.01	SED	-	0.23	2.11	-8.93 *	-0.07
LPA	-0.59	-	-1.93	-3.75 *	-0.59	LPA	-0.22	-	1.90	-9.14 *	-0.28
MPA	1.05	1.69	-	-2.11	1.05	MPA	-1.66	-1.42	-	-10.59 *	-1.72
VPA	1.62	2.25	0.27	-	1.62	VPA	4.58	4.81	6.69 *	-	4.51
Sleep	-0.01	0.63	-1.34	-3.17		Sleep	0.07	0.30	2.18	-9.15 *	-
Pre-Pubertal Girls											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	2.72	-3.93	-8.31 *	0.01	SED	-	0.88	5.17 *	-19.43 *	-0.09
LPA	-2.21	-	-6.14 *	-10.52 *	-2.20	LPA	-0.71	-	4.46 *	-20.13 *	-0.79
MPA	2.48	5.20 *	-	-5.84 *	2.48	MPA	-3.25	-2.36	-	-22.67 *	-3.33
VPA	2.86	5.58 *	-1.07	-	2.86	VPA	6.70 *	7.57 *	11.86 *	-	6.61 *
Sleep	-0.01	2.72	-3.94	-8.31 *		Sleep	0.06	0.96	5.24 *	-19.34 *	-
Pubertal Boys											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.53	-1.12	-3.00	0.01	SED	-	0.27	2.22	-10.64 *	-0.07
LPA	-0.53	-	-1.64	-3.53	-0.53	LPA	-0.25	-	1.97	-10.89 *	-0.32
MPA	0.87	1.44	-	-2.13	0.87	MPA	-1.72	-1.45	-	-12.36 *	-1.79
VPA	1.40	1.97	0.28	-	1.40	VPA	4.96 *	5.23 *	7.18 *	-	4.89 *
Sleep	-0.01	0.57	-1.12	-3.01	-	Sleep	0.07	0.34	2.29	-10.58 *	-

Pubertal Girls											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.77	-1.43	-3.85	0.01	SED	-	0.33	2.58	-12.32 *	-0.08
LPA	-0.71	-	-2.15	-4.57	-0.71	LPA	-0.30	-	2.28	-12.63 *	-0.38
MPA	1.13	1.90	-	-2.72	1.13	MPA	-2.02	-1.68	-	-14.34 *	-2.10
VPA	1.84	2.62	2.72	-	1.84	VPA	5.90 *	6.22 *	4.90 *	-	5.81 *
Sleep	-0.01	0.77	-1.44	-3.86		Sleep	0.08	0.41	2.66	-12.25 *	-
Post-Pubertal Boys											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.62	-1.23	-2.30	0.01	SED	-	0.32	2.67	-8.89 *	-0.06
LPA	-0.56	-	-1.79	-2.86	-0.56	LPA	-0.29	-	2.38	-9.18 *	-0.35
MPA	0.89	1.51	-	-1.41	0.89	MPA	-1.93	-1.60	-	-10.82 *	-1.99
VPA	1.12	1.74	-0.11	-	1.12	VPA	4.34 *	4.66 *	7.00 *	-	4.27 *
Sleep	-0.01	0.62	-1.23	-2.30	-	Sleep	0.06	0.38	2.73 *	-8.83 *	-
Post-Pubertal Girls											
	SED	LPA	MPA	VPA	Sleep		SED	LPA	MPA	VPA	Sleep
SED	-	0.44	-1.54	-3.55	0.01	SED	-	0.02	3.10	-12.70 *	-0.08
LPA	-0.42	-	-1.96	-3.96	-0.41	LPA	-0.19	-	2.91	-12.89 *	-0.27
MPA	1.14	1.57	-	-2.41	1.14	MPA	-2.28	-2.07	-	-14.98 *	-2.36
VPA	1.59	2.02	1.59	-	1.59	VPA	5.70 *	5.91 *	5.68 *	-	5.63 *
Sleep	-0.01	0.44	-1.54	-3.54	-	Sleep	0.08	0.28	3.17	-12.62 *	-

SED = Sedentary time, LPA = Light Physical Activity, MPA = Moderate Physical Activity, VPA = Vigorous Physical Activity. All figures presented as percentage change with \* indicating a change greater than the Smallest Worthwhile Change (%).

#### 5.4 Discussion

This is the first study to examine the effects of changing time spent in various movement behaviours (SED, LPA, MPA, VPA and Sleep), using a five-part compositional analysis, on absolute and allometrically scaled  $\dot{V}O_{2\max}$  in trained and untrained children and adolescents. The main findings of the present study were that allocating time to, and removing time from, VPA significantly increased and decreased allometrically scaled  $\dot{V}O_{2\max}$ , respectively, regardless of sex, training, or maturity status. Additionally, the re-allocation of time to MPA from all movement behaviours in trained pubertal girls significantly decreased allometrically scaled peak  $\dot{V}O_2$ . These findings therefore highlight that intensity of PA may be of paramount importance in determining peak  $\dot{V}O_2$ , especially in girls.

Engaging in 10 minutes more VPA, irrespective of which behaviour it displaces, significantly increases both absolute and allometrically scaled  $\dot{V}O_{2\max}$ , regardless of training status. Moreover, of importance, untrained children were predicted to have a larger magnitude of change for the same 10-minute reallocation, in accord with the review of McNarry & Jones (2014) which concluded that baseline fitness significantly impacts the magnitude of change experienced to a given stimulus. More specifically, Mahon (2008) reported that 52% of the inter-individual variation in participants responses to a training stimuli can be explained by baseline  $\dot{V}O_{2\max}$ . The findings of the current study are, however, discordant with Carson et al. (2016), who reported no significant differences when reallocating time to, or from, any movement behaviour. Such discrepancies may be explained by the use of a proxy measure of  $\dot{V}O_{2\max}$  and not accounting for maturation or training status in the earlier study, which are critical when assessing cardiorespiratory fitness in children and adolescents (Armstrong & Welsman, 2019b, 2020a).

The present study supports the notion that children and adolescents require a vigorous stimulus to significantly improve absolute and scaled  $\dot{V}O_{2\max}$  (Massicotte & Macnab, 1974; McNarry & Jones, 2014). Of concern however, the current findings suggest that children and adolescents may need to increase their time spent in VPA by over 50%. Indeed, the average time spent in VPA in this study was  $17.1 \pm 12.7$  mins $\cdot$ day $^{-1}$ , which is in accord with the levels reported in the millennium cohort study ( $19.9 \pm 10.6$  mins $\cdot$ day $^{-1}$ ). Considering the limited success at increasing PA in the majority of

interventions to date (Love, Adams, & van Sluijs, 2019b; Mannocci et al., 2020), and the small magnitude of increases in VPA reported even in those considered successful (Goode et al., 2017), the current findings highlight the need to drastically change our approach to PA promotion. Indeed, these findings could be speculated to support the contention suggested by many authors that HIIT may represent an important public health intervention tool (Cao et al., 2019; Eddolls et al., 2017; Garcia-Hermoso et al., 2016).

Dencker et al (2006) reported weak, but significant, correlations between VPA and  $\dot{V}O_{2\max}$  ( $r^2 = 0.32$ ) and allometrically scaled peak  $\dot{V}O_2$  ( $r^2 = 0.27$ ). Furthermore, the most recent review of the relationship between PA and  $\dot{V}O_{2\max}$  in youth concluded that, despite decades of research, there was still no overall consensus (Armstrong et al., 2011). These equivocal findings may be related to the reliance on techniques that fail to account for the inter-related and inherently constrained nature of PA behaviours, leading to spurious conclusions (Carson et al., 2016; Chastin et al., 2015; Dumuid et al., 2018a). Moreover, the reliance on ratio scaling  $\dot{V}O_{2\max}$  potentially creates spurious associations (Welsman & Armstrong, 2019). Of note, when  $\dot{V}O_{2\max}$  was allometrically scaled by body mass, the overall PA composition explained ~11% less variance compared to absolute  $\dot{V}O_{2\max}$ . This may be due, at least in part, to physically active children having a higher lean body mass (LBM) than their sedentary counterparts (Bitar et al., 2000; Butte, Puyau, Adolph, Vohra, & Zakeri, 2007), indicating that differences in body composition may also be critical when determining the effect of re-allocating PA. Nevertheless, the PA composition still explained ~37.7% of the variance in allometrically scaled  $\dot{V}O_{2\max}$ , demonstrating the powerful influence of habitual PA on aerobic fitness.

The finding that allocating time to MPA decreased allometrically scaled  $\dot{V}O_{2\max}$  was surprising. These associations could be due, at least in part, due to the misclassification of intensities using population level cut-points which fail to take into account the baseline fitness of the individuals which is critical in determining the intensity of movement (A. Rowlands et al., 2018a; Trost, 2016; Trost et al., 2011). Alternatively, these associations could also be attributable to the fixed time reallocation used within compositional analysis studies to date. More specifically, a 10-minute change in VPA constitutes a ~50% increase in VPA but only a 1.9% increase in SED time. Therefore,

a greater insight into the independent, and interactive, effects of movement behaviours on  $\dot{V}O_{2\max}$  may be gained by investigating the effects of the same percentage change in movement behaviours on  $\dot{V}O_{2\max}$ . Nevertheless, evidence is emerging that the intensity of PA may be critical in improving both performance and health-related parameters in paediatric populations (Carson et al., 2019; Väistö et al., 2019; Whooten et al., 2019) and thus VPA should be encouraged, as opposed to MPA, to engender the greatest long-term health benefits

Compositional analysis techniques have the ability to allow researchers and practitioners to design, and implement, personalised, targeted interventions with the aim of improving specific health outcomes (Chastin et al., 2015). Consequently, the specificity of compositional data analysis could be critical in interventional design and implementation in both trained and untrained children to maximise performance and health (Love et al., 2019a; Love et al., 2019b). Moreover, such analyses could also lead to the development of an ‘optimal’ or ‘idealistic’ composition of PA and enable the determination of the minimum amount of daily MPA and VPA to ascertain the associated health benefits (Chastin et al., 2015). Consequently, compositional analyses have the ability to transform our understanding of PA and each movement inter-relationship with each other, allowing for a greater specificity in national PA guidelines. It must be noted, however, that compositional analysis is not without limitations. Indeed, the linear predictive modelling provides no indication of how long these habitual PA behaviours need to be maintained for in order to achieve the magnitude of change predicted in this study.

Future research should seek to implement targeted interventions informed by compositional analyses, to ascertain the required duration needed to elicit the changes predicted. This is of particular importance as a plethora of research has investigated the influence of different training methodologies on both absolute and allometrically scaled peak  $\dot{V}O_2$ , with their effectiveness being reviewed elsewhere (Baquet et al., 2003; Costigan et al., 2015; Logan et al., 2014; Moro, Bianco, Faigenbaum, & Paoli, 2014). One major issue with the majority of paediatric training studies to date is the lack of accounting for changes in habitual PA levels across the intervention period (Carazo-Vargas & Moncada-Jiménez, 2015; Mahon & Vaccaro, 1989; Massicotte & Macnab, 1974), and this could help explain the equivocal findings of some



intervention types (Becker & Vaccaro, 1983; Carazo-Vargas & Moncada-Jiménez, 2015). Specifically, most interventions elicit an improvement in  $\dot{V}O_{2\max}$  of 5-6% (Baquet et al., 2003) increasing to ~10.4% when allometrically scaled (Leite, Freitas, Campelo, & Maciel, 2016). Thus, if the predicted increases in absolute, scaled,  $\dot{V}O_{2\max}$  can be achieved over 4 – 6 weeks, the typical length of most training interventions (Baquet et al., 2003; Carazo-Vargas & Moncada-Jiménez, 2015; Logan et al., 2014), then it is not possible to delineate whether the improvements in peak  $\dot{V}O_2$  are training-related or stem from increases in habitual PA.

Whilst there are numerous strengths associated with this study, such as the use of a novel five-part compositional analysis approach, allometrically scaling  $\dot{V}O_{2\max}$ , and accounting for training, maturity and sex differences, there are limitations which must be acknowledged. Firstly, a relatively low wear-time criteria was set of any three days with at least eight hours of wear-time; a more stringent wear-time criteria could potentially influence the relationships established between PA metrics and  $\dot{V}O_{2\max}$ . Nevertheless, this wear-time has been validated in a paediatric population (Trost, 2016; Trost et al., 2000) and was used to maximise participant inclusion within the study. Second, the relatively short recording time (7-days) may not reflect habitual PA levels and these results should therefore be interpreted as an estimation of physical activities influence on  $\dot{V}O_{2\max}$ . Additionally, linear predictive models from compositional analyses do not indicate the duration over which the habitual changes need to be maintained in order to observe the associated changes in  $\dot{V}O_{2\max}$ . Moreover, the applicability of cycle derived  $\dot{V}O_{2\max}$  to habitual PA levels is contentious, and therefore future research should endeavour to establish  $\dot{V}O_{2\max}$  using treadmills to maximise specificity, and to establish whether these findings persist. Finally, the physical activity recordings for the trained group did not include their training regimes, and therefore these results should be interpreted as the effect of changing leisure time physical activity patterns on  $\dot{V}O_{2\max}$ . Future research, using compositional analyses, is required to examine the effect of changing training session make-up on  $\dot{V}O_{2\max}$  in youth.

In conclusion, VPA is a significant predictor of allometrically scaled  $\dot{V}O_{2\max}$  in children and adolescents, independent of training, sex and maturity status. Moreover, reallocating time from VPA in pre-pubertal children significantly predicts a reduced

absolute  $\dot{V}O_{2\max}$ , potentially highlighting the importance of promoting VPA in pre-pubertal children. Future research should seek to establish the duration of targeted PA interventions needed to elicit the significant changes predicted from compositional analyses and report the individual levels of MPA and VPA to ascertain the relative importance of VPA for current, and future, health in children and adolescents.

# Chapter 6

The effect of sex, maturity, and training status on maximal sprint performance kinetics

## **Chapter 6 (Study 3) – The effect of sex, maturity, and training status on maximal sprint performance kinetics**

### **6.1 Introduction**

Over-ground sprint running has become a popular method of performance assessment over the past decade (Meyers et al., 2015; Meyers et al., 2017a; Rumpf et al., 2015a; Rumpf et al., 2015b), partly due to the importance of speed in many athletic and sporting activities (Lloyd & Oliver, 2012; Meylan, Cronin, Oliver, & Hughes, 2010). Indeed, over-ground sprinting is commonly used within long-term athlete development (LTAD) programs and talent identification test batteries (Meylan et al., 2010; Unnithan et al., 2012). However, despite this increasingly widespread use, fundamental questions remain to be resolved in terms of the development of speed in youth, especially with regards to the influences of sex and maturity, and their interaction with each other and training status.

The development of speed during adolescence is a non-linear process in boys, with evidence suggesting periods of accelerated development around the age of peak height velocity (PHV; Meyers et al., 2015; Meyers et al., 2017a; Philippaerts et al., 2006; Rumpf et al., 2012). Specifically, in a mixed-longitudinal study involving youth footballers, 30 m sprint time was reported to improve by 0.4 s in the six-months surrounding PHV compared to only 0.2 s following PHV (Philippaerts et al., 2006). Moreover, early maturing boys demonstrate faster 30 m sprint times than their age-matched normal and late maturing counterparts (Rommers et al., 2018), with evidence from non-motorised treadmills suggesting that sprint kinetics (i.e. force and power) only significantly increase from pre- to pubertal maturity statuses, displaying a plateau thereafter (Rumpf et al., 2015b). This period of accelerated development is thought to be mediated by changes in anthropometric variables, increases in muscle size and cross sectional area (CSA) and neuromuscular adaptations, including improved synchronisation of motor units and utilisation of type II muscle fibres (Dotan et al., 2012; Van Praagh, 2000; Van Praagh & Doré, 2002).

Whether similar periods of non-linear development in sprint speed are evident in girls is currently unknown, with little data currently available considering the influence of

growth and maturation, and their interaction, on sprint performance in girls. In one of the only studies to examine sprint development in untrained girls, a plateau in peak velocity ( $V_{\text{peak}}$ ) was observed from 12-13 years compared to 15 years in their male peers (Papaiakovou et al., 2009). However, with no maturity assessment in this study, whether this plateau is attributable to age per se, or rather to concomitant growth and maturation related changes, cannot be elucidated. Indeed, Nagahara et al. (2019) reported a similar plateau in  $V_{\text{peak}}$  at 12.7 years in girls which was attributed to no further increases in step length. No evidence is currently available that considers the development of speed throughout the maturational process or in response to training compared to untrained controls so further inferences regarding the influences of sex and training, and their interaction, are precluded.

In addition to kinematic factors (i.e. stride length/rate), sprint performance is determined by kinetic parameters such as horizontal and vertical force (Morin et al., 2011; Morin et al., 2006; Rossi et al., 2017; Rumpf et al., 2013; Rumpf et al., 2015b; Samozino et al., 2016). However, the evidence exploring the kinetic determinants of sprint performance in paediatric populations has predominately been derived from non-motorised treadmills which limits its ecological validity (Rumpf et al., 2015b; Rumpf et al., 2012). Moreover, the majority of these studies have focused solely on the development of maximal velocity (Meyers et al., 2015; Meyers et al., 2017a; Rumpf et al., 2015b), thereby considering only a small component of sprint performance, or have utilised mean velocity data over a given distance (i.e. 5 meters; Mendez-Villanueva et al., 2010; Papaiakovou et al., 2009). These methodological limitations may be ameliorated by recent advances in radar technology and macroscopic biomechanical modelling techniques which enable velocity, power and force to be calculated near instantaneously across an entire sprint (Samozino et al., 2016; Simperingham et al., 2016). Force-velocity-Power (F-v-P) profiling has been validated against force plate data, demonstrating high reliability in elite adult sprinters (Samozino et al., 2016). Additionally, the combination of radar technology and F-v-P profiling has been deemed highly reliable in both trained and untrained paediatric participants (Runacres et al., 2019a). Consequently, such methods could provide important insights to the kinetic parameters underpinning differences in sprint performance according to sex, maturity and training status.

Therefore, the primary aim of this study was to determine whether the kinetics of sprint performance differ with respect to sex, maturity and training status. The secondary aim was to determine whether the kinetic determinants of sprint performance change with maturational status.

## 6.2 Methods

Trained children and adolescents were recruited through the national governing body for Hockey in Wales and were competing at a national/international level. All of the trained children and adolescents had been training for  $3.0 \pm 1.5$  years and were currently completing  $8 \pm 2$  hours per week of supervised training. Untrained participants were recruited from local schools across South Wales and were required to be involved in no formal exercise training outside of curricular physical education. The final sample consisted of 260 (133 girls) participants, which consisted of 147 (69 girls;  $14.3 \pm 2.1$  years) and 113 (64 girls;  $13.8 \pm 2.7$  years) trained and control youth, respectively. Online parent/guardian consent and a medical pre-screening questionnaire were completed using a custom-built online form (Survey Monkey, Dublin, Ireland). Participants were excluded if their parent/guardian reported they had any known cardiovascular, kidney, metabolic, or any other condition that would have prevented them from completing the study protocol. Written informed assent was obtained from each participant prior to data collection. Ethics approval was granted by the institutional ethics committee, with all procedures conforming to the Declaration of Helsinki.

### *6.2.1 Experimental Procedures*

Standing and sitting stature were measured to the nearest 0.1 cm using a portable stadiometer (Seca 213, Seca, Chino, CA, USA), with body mass measured to the nearest 0.1 kg using electronic scales (Seca 803, Seca, Chino, CA, USA). Subsequently, individual maturity offset was estimated using the predictive equations devised by Mirwald et al. (2002a), with participants classed as pre-pubertal if more than one year away from PHV, pubertal if within a year of PHV, and post-pubertal if more one-year post-PHV.

Prior to the sprint protocol, all participants completed a standardised five-minute warm-up. Specifically, participants completed two minutes of low intensity jogging, followed by running based drills including high knees, heel-flicks, sidesteps and strides over 40 m, before completing two 30 m sprints at 50% and 75% max effort respectively. The warm-up terminated with one maximal 30 m, sprint which acted as a familiarisation trial. Subsequently, participants completed two maximal sprints on an AstroTurf over a distance of 35 m to avoid premature deceleration. Both sprints were conducted from a two-point standing start to minimise vertical displacement during the early phases of the sprint (Mero, Komi, & Gregor, 1992), with participants instructed to start sprinting using auditory cues (i.e. “3....2.....1...GO”). All sprint trials were conducted outside on a surface the participants were comfortable performing on, with a mean temperature and wind speed of  $13.5 \pm 1.9^{\circ}\text{C}$  and  $2.3 \pm 1.0 \text{ m}\cdot\text{s}^{-1}$ , respectively. Where possible, participants ran with the prevailing wind behind them to control the effects this can have on performance (Linthorne, 1994). Velocity was measured throughout both sprint trials using a radar gun (STALKER ATS II, Plano, Texas, USA), mounted on a tripod positioned 10 m behind the start line, in accord with manufacturer instructions. The radar gun recorded velocity at a frequency  $> 46 \text{ Hz}$ , allowing near instantaneous power and force to be modelled throughout the duration of the sprint.

### *6.2.2 Biomechanical Modelling*

The full details of the macroscopic biomechanical model are presented in Samozino et al (2016). However, briefly, prior to data processing, the first 0.3 seconds of the trial were deleted, in line with previous recommendations (Samozino, 2018), following which the raw velocity-time ( $v_h(t)$ ) data were modelled using a mono-exponential curve. Following integration of the  $v_h(t)$  curve, the horizontal displacement ( $x_h(t)$ ) was obtained, with further derivation providing the horizontal acceleration ( $a_h(t)$ ) of the participant’s centre of mass (COM; Samozino, 2018). According to the fundamental laws of dynamics, the horizontal antero-posterior force ( $F_h(t)$ ) was calculated considering aerodynamic drag (Morin et al., 2011; Samozino et al., 2016). Subsequently, power output was determined as the product of force and velocity. All power and force variables were interpolated to 0.1 seconds intervals, with peak power ( $P_{\text{peak}}$ ; W) and peak force ( $F_{\text{peak}}$ ; N) defined as the highest values recorded during the

30 m sprint. Moreover, to allow for the comparison between training, sex, and maturity groups,  $P_{\text{peak}}$  and  $F_{\text{peak}}$  were ratio and allometrically scaled by body mass, using methods reported elsewhere (Nevill et al., 2006). Time to peak power ( $t_{P_{\text{peak}}}$ ; s) was determined as the time from sprint start to  $P_{\text{peak}}$ , with mean power ( $P_{\text{mean}}$ ; W) and force ( $F_{\text{mean}}$ ; N) defined as the average power and force throughout the sprint. Thirty meter sprint time (30mT) was defined as the time elapsed from the start of the sprint until  $x_h(t)$  first exceeded 30 m. Peak velocity ( $V_{\text{peak}}$ ;  $\text{m}\cdot\text{s}^{-1}$ ) was derived from the mono-exponential  $v_h(t)$  curve, with the modelled velocities over the same time period as  $P_{\text{mean}}$  used to determine mean velocity ( $V_{\text{mean}}$ ;  $\text{m}\cdot\text{s}^{-1}$ ). Finally, fatigue rate (FR;  $\text{W}\cdot\text{s}^{-1}$ ) was determined as the average rate of power decline per second from  $P_{\text{peak}}$  until 30mT, with mechanical efficiency index ( $D_{\text{RF}}$ ) represented by the slope of the linear decline of force production with increasing velocity. All variables were calculated for both sprints, but only the fastest sprint (as determined by 30mT) was carried forward for analysis

### 6.2.3 Statistical Analyses

All values are presented as mean  $\pm$  SD unless otherwise stated, with all statistical analyses conducted in SPSS (version 26.0, IBM, Armonk, NY, USA) and significance accepted as  $p < 0.05$ . Multivariate ANOVAs were used to identify significant differences in performance variables between groups and any interaction effects, with Bonferroni corrections to post-hoc tests where appropriate. Cohens  $d$  was also calculated, with effect sizes considered trivial ( $\leq 0.20$ ), moderate (0.21 – 0.60), large (0.61 – 0.80) or very large ( $\geq 0.81$ ).

Hierarchical multiple linear regression was used to ascertain the determinants of 30 m sprint time according to maturity group. Specifically, training status and sex were initially entered into the model given their strong association with sprint performance in children and adolescents (Papaiakovou et al., 2009; Rumpf et al., 2015a; Rumpf et al., 2012). Subsequently, predictor variables were entered to ascertain their independent association with 30mT, with inclusion into the model only accepted if a significant increase in explained variance was observed at the 0.05 level. Collinearity between potential predictors was investigated using the variance inflation factor to determine trivial ( $\text{VIF} = 1$ ), moderate ( $1 < \text{VIF} \leq 5$ ) and high ( $\text{VIF} > 5$ ) collinearity indicating high collinearity (Daoud, 2017). If high co-linearity was found between



variables, the variable explaining the greatest proportion of variance was added to the model (Daoud, 2017). The adequacy of the regression model was determined using the normality of residual values.

### 6.3 Results

With the exception of BMI ( $p > 0.05$ ), all anthropometric variables significantly increased with maturity stage ( $p < 0.01$ ), irrespective of sex or training status (Table 1). Trained children were taller and had a lower BMI than their untrained counterparts ( $p < 0.05$ ). Pre-pubertal hockey players were lighter than their untrained counterparts ( $p < 0.05$ ), whereas pubertal and post-pubertal hockey players were heavier ( $p < 0.05$ ). Boys were significantly taller than girls at all stages of maturity, irrespective of training status ( $p < 0.05$ ), and pre-pubertal and post-pubertal untrained girls were significantly lighter than their male counterparts ( $p < 0.05$ ).

#### *6.3.1 Influence of training status*

As shown in Table 2 and 3, trained youth had a higher  $P_{\text{peak}}$  than their untrained counterparts ( $F_{(1,244)} = 38.8$ ,  $p < 0.01$ ,  $d = 1.05$ ), which persisted even after ratio ( $F_{(1,244)} = 24.6$ ,  $p < 0.01$ ,  $d = 0.78$ ) or allometric scaling ( $F_{(1,244)} = 21.6$ ,  $p < 0.01$ ,  $d = 0.71$ ). Trained youth also had a higher  $F_{\text{peak}}$  ( $F_{(1,244)} = 7.4$ ,  $p < 0.01$ ,  $d = 0.52$ ), although this was ameliorated following ratio and allometric scaling ( $p > 0.05$ ). Trained participants had a higher  $V_{\text{peak}}$  ( $F_{(1,244)} = 131.0$ ,  $p < 0.01$ ,  $d = 1.78$ ),  $V_{\text{mean}}$  ( $F_{(1,244)} = 134.3$ ,  $p < 0.01$ ,  $d = 1.80$ ) and a faster 30mT ( $F_{(1,244)} = 121.0$ ,  $p < 0.01$ ,  $d = 1.71$ ) when compared to their untrained counterparts. Finally, trained children and adolescents had a slower  $t_{P_{\text{peak}}}$ , and a higher  $P_{\text{mean}}$ , relative  $P_{\text{mean}}$  and FR ( $p < 0.05$ ), but there was no significant difference between athletes and controls for  $D_{\text{RF}}$  ( $F_{(1,244)} = 0.95$ ,  $p > 0.05$ ).

#### *6.3.2 Influence of Sex*

Boys produced a significantly higher  $F_{\text{peak}}$  and  $P_{\text{peak}}$  than girls (Table 2 and 3, respectively), which remained after allometrically scaling for body mass (Scaled  $P_{\text{peak}}$ :  $F_{(1,244)} = 14.8$ ,  $p < 0.01$ ,  $d = 0.57$ ; Scaled  $F_{\text{peak}}$ :  $F_{(1,244)} = 32.3$ ,  $p < 0.01$ ,  $d = 0.27$ ). Boys also achieved a higher  $P_{\text{mean}}$  ( $F_{(1,244)} = 33.5$ ,  $p < 0.01$ ,  $d = 0.64$ ), relative  $P_{\text{mean}}$  ( $F_{(1,244)} = 11.0$ ,  $p < 0.01$ ,  $d = 0.51$ ),  $V_{\text{peak}}$  ( $F_{(1,244)} = 14.0$ ,  $p < 0.01$ ,  $d = 0.53$ ),  $V_{\text{mean}}$  ( $F_{(1,249)} = 19.3$ ,  $p < 0.01$ ,  $d = 0.59$ ), a faster 30mT ( $F_{(1,244)} = 13.7$ ,  $p < 0.01$ ,  $d = 0.52$ ) and FR ( $F_{(1,249)} = 22.1$ ,  $p < 0.01$ ,  $d = 0.55$ ) than girls, irrespective of training or maturity status.

However, there were no significant sex differences for  $t_{P_{\text{peak}}}$  ( $F_{(1,244)} = 0.69$ ,  $p > 0.05$ ) or  $D_{\text{RF}}$  ( $F_{(1,244)} = 1.51$ ,  $p > 0.05$ ).

### 6.3.3 Influence of Maturity

As shown in Tables 2 and 3, post-PHV adolescents produced a higher  $P_{\text{peak}}$  and  $F_{\text{peak}}$  than circa-PHV adolescents or pre-PHV children (all  $p < 0.01$ ), with significantly higher values similarly observed for circa-PHV adolescents in comparison to pre-PHV children ( $p < 0.05$ ). However, after ratio and allometric scaling for body mass, no significant differences persisted between any maturity groups ( $p > 0.05$ ). Post-PHV adolescents also had a significantly higher  $V_{\text{peak}}$ , 30mT, and  $D_{\text{RF}}$  than all other maturity groups ( $p < 0.05$ ), with no significant differences evident between pre-PHV and circa-PHV children. There was no significant effect of maturation on any other sprint variable ( $p > 0.05$ ).

### 6.3.4 Interaction of sex, maturity, and training status

There was a significant interaction effect between sex and maturity on  $t_{P_{\text{peak}}}$  ( $F_{(2,244)} = 4.3$ ,  $p < 0.05$ ), relative  $P_{\text{mean}}$  ( $F_{(2,244)} = 3.9$ ,  $p < 0.05$ ),  $V_{\text{peak}}$  ( $F_{(2,244)} = 5.6$ ,  $p < 0.01$ ) and  $F_{\text{peak}}$  ( $F_{(2,244)} = 5.0$ ,  $p < 0.01$ ). Specifically, there was significantly less difference in  $t_{P_{\text{peak}}}$  between post-PHV boys and girls (5%) compared to pre-PHV (14.8%) and circa-PHV (17.0%) boys and girls. Conversely, there was a greater sex difference in relative  $P_{\text{mean}}$  and  $V_{\text{peak}}$  in circa-PHV adolescents (46.9% and 19.8%, respectively) compared to pre-PHV (5.4% and 3.2%) or post-PHV youth (11.6% and 5.6%). A greater sex difference was also evident in  $F_{\text{peak}}$  for pre-PHV children (53.5%) compared that found in circa-PHV (10.6%) or post-PHV adolescents (21.6%).

A significant sex, maturity and training interaction effect was also apparent on  $P_{\text{peak}}$  ( $F_{(2,244)} = 3.8$ ,  $p < 0.05$ ),  $F_{\text{peak}}$  ( $F_{(2,244)} = 5.9$ ,  $p < 0.01$ ), relative  $F_{\text{peak}}$  ( $F_{(2,244)} = 3.1$ ,  $p < 0.05$ ) and scaled  $F_{\text{peak}}$  ( $F_{(2,244)} = 3.3$ ,  $p < 0.05$ ). Specifically, less difference was observed in  $P_{\text{peak}}$  and  $F_{\text{peak}}$  between trained and untrained circa-PHV boys and girls ( $P_{\text{peak}}$ : 26.7%;  $F_{\text{peak}}$ : 28.3%) compared to those found in pre-PHV ( $P_{\text{peak}}$ : 42.3%;  $F_{\text{peak}}$ : 36.1%) or post-PHV youth ( $P_{\text{peak}}$ : 38.0%;  $F_{\text{peak}}$ : 33.7%). Conversely, the biggest differences in relative  $F_{\text{peak}}$  and scaled  $F_{\text{peak}}$  were observed between trained and untrained post-PHV boys and girls (both 24.5%) compared to pre-PHV children

(relative  $F_{\text{peak}}$ : 7.8%; scaled  $F_{\text{peak}}$ : 9.1%) and circa-PHV adolescents (relative  $F_{\text{peak}}$ : 13.3%; scaled  $F_{\text{peak}}$ : 13.9%).

### *6.3.5 Determinants of Sprint Performance*

Model 1 in which only training status and sex were entered explained 33%, 53% and 37% of the variance in 30mT in pre-PHV, circa-PHV and post-PHV children and adolescents, respectively (Table 6.4). Subsequently, scaled  $P_{\text{peak}}$  and  $D_{\text{RF}}$  were found to be significant predictors of performance across all maturity groups, explaining 65% of the variance in 30mT in pre-PHV children which increased to 75% and 80% in circa-PHV and post-PHV adolescents, respectively. No other parameters were found to predict sprint performance in youth.

**Table 6.1** – Anthropometric characteristics for trained and untrained participants

	Hockey Players (n = 147)						Control Participants (n = 113)					
	Pre-PHV		Circa-PHV		Post-PHV		Pre-PHV		Circa-PHV		Post-PHV	
	Boys (n = 17)	Girls (n = 17)	Boys (n = 32)	Girls (n = 15)	Boys (n = 29)	Girls (n = 37)	Boys (n = 22)	Girls (n = 14)	Boys (n = 14)	Girls (n = 34)	Boys (n = 13)	Girls (n = 16)
Age (years)	12.1 ± 0.8	11.2 ± 1.7	14.2 ± 0.8 <sub>a</sub>	13.1 ± 0.8 <sub>a</sub>	16.7 ± 1.4 <sub>ab</sub>	15.8 ± 1.5 <sub>ab</sub>	11.5 ± 0.9	11.4 ± 0.3	14.3 ± 0.8 <sub>a</sub>	13.6 ± 0.3 <sub>a</sub>	16.7 ± 1.0 <sub>ab</sub>	15.3 ± 0.7 <sub>ab</sub>
Stature (m)	1.58 ± 0.07	1.50 ± 0.10* <sup>a</sup>	1.68 ± 0.07	1.58 ± 0.08* <sup>a</sup>	1.74 ± 0.06	1.64 ± 0.07* <sup>a</sup>	1.49 ± 0.08 <sup>#</sup>	1.41 ± 0.06 <sup>#*</sup>	1.57 ± 0.09 <sup>#a</sup>	1.52 ± 0.07 <sup>#*ab</sup>	1.64 ± 0.11 <sup>#ab</sup>	1.73 ± 0.06 <sup>#*ab</sup>
Body Mass (kg)	47.7 ± 7.1	42.5 ± 8.8	55.5 ± 6.8 <sub>a</sub>	51.5 ± 9.3 <sub>a</sub>	63.9 ± 5.2 <sub>a</sub>	58.5 ± 8.9 <sub>a</sub>	52.0 ± 15.3 <sup>#</sup>	36.9 ± 13.5 <sup>#*</sup>	49.3 ± 12.0 <sup>#</sup>	50.9 ± 13.5 <sup>#</sup>	61.3 ± 11.7 <sup>#ab</sup>	50.9 ± 7.0 <sup>#*</sup>
BMI (kg·m <sup>-2</sup> )	19.0 ± 1.5	18.7 ± 2.1	19.7 ± 1.8	20.5 ± 2.4	21.1 ± 2.0	21.7 ± 2.4	23.0 ± 5.3 <sup>#</sup>	18.3 ± 4.6 <sup>#</sup>	20.0 ± 3.7 <sup>#</sup>	22.0 ± 5.9 <sup>#</sup>	22.9 ± 5.3 <sup>#</sup>	20.8 ± 2.4 <sup>#</sup>
Maturity Offset (years)	-1.66 ± 0.45	-1.97 ± 0.85	-0.05 ± 0.54 <sup>a</sup>	+0.30 ± 0.36 <sup>a</sup>	+2.44 ± 0.79 <sup>ab</sup>	+2.17 ± 0.89 <sup>ab</sup>	-2.11 ± 0.75	-2.31 ± 0.77	-0.38 ± 0.54 <sup>a</sup>	-0.16 ± 0.50 <sup>a</sup>	+1.93 ± 0.92 <sup>ab</sup>	+1.45 ± 0.71 <sup>ab</sup>

PHV = Peak Height Velocity, BMI = Body Mass Index; <sup>#</sup> indicate a significant difference between the same maturity and sex between training groups. \* Significant difference between sex between the same maturity and sport group. <sup>a</sup> Significant difference compared to pre-pubertal children of the same sport and sex.

<sup>b</sup> Significant difference compared to pubertal adolescents of the same sport and sex

**Table 6.2** – 30m sprint performance variables in boys

	Hockey Players			Control Participants		
	Pre-PHV	Circa-PHV	Post-PHV	Pre-PHV	Circa-PHV	Post-PHV
$t_{P_{peak}}$ (s)	0.54 ± 0.11	0.55 ± 0.12	0.61 ± 0.13 <sup>a</sup>	0.43 ± 0.13 <sup>b</sup>	0.55 ± 0.16 <sup>b</sup>	0.50 ± 0.19 <sup>ab</sup>
$P_{peak}$ (W)	685.8 ± 119.2	864.7 ± 200.0*	957.4 ± 251.6 <sup>αa</sup>	620.4 ± 142.3 <sup>b</sup>	596.0 ± 202.8 <sup>*b</sup>	822.5 ± 222.6 <sup>*ab</sup>
Relative $P_{peak}$ (W·kg <sup>-1</sup> )	14.3 ± 2.7	15.6 ± 2.9	14.9 ± 3.6	12.6 ± 3.6 <sup>b</sup>	12.4 ± 4.5 <sup>b</sup>	13.8 ± 4.4 <sup>b</sup>
Scaled $P_{peak}$ (W·kg <sup>-b</sup> )	9.3 ± 1.8	9.8 ± 1.8	9.2 ± 2.2	8.1 ± 2.5 <sup>b</sup>	8.0 ± 3.0 <sup>b</sup>	8.6 ± 2.8 <sup>b</sup>
$P_{mean}$ (W)	219.0 ± 47.7	277.2 ± 61.8*	337.6 ± 106.1 <sup>*α</sup>	157.4 ± 53.8 <sup>b</sup>	188.2 ± 64.7 <sup>*b</sup>	229.5 ± 65.5 <sup>*ab</sup>
Relative $P_{mean}$ (W)	4.6 ± 0.9	5.0 ± 0.8	5.2 ± 1.5 *	3.1 ± 0.8 <sup>b</sup>	3.9 ± 1.2 <sup>b</sup>	3.8 ± 1.2 <sup>*b</sup>
$V_{peak}$ (m·s <sup>-1</sup> )	6.60 ± 0.56	6.84 ± 0.42	7.06 ± 0.81 <sup>*α</sup>	5.40 ± 0.75 <sup>b</sup>	6.06 ± 0.94 <sup>b</sup>	6.02 ± 0.88 <sup>*ab</sup>
$V_{mean}$ (m·s <sup>-1</sup> )	5.62 ± 0.36	5.80 ± 0.30	5.87 ± 0.53 <sup>*α</sup>	4.81 ± 0.55 <sup>b</sup>	5.16 ± 0.64 <sup>b</sup>	5.20 ± 0.65 <sup>*ab</sup>
30mT (s)	5.35 ± 0.35	5.19 ± 0.27	5.15 ± 0.47 <sup>*α</sup>	6.31 ± 0.74 <sup>b</sup>	5.92 ± 0.97 <sup>b</sup>	5.86 ± 0.80 <sup>*ab</sup>
$F_{peak}$ (N)	390.6 ± 69.8	467.9 ± 96.2*	502.4 ± 102.4 <sup>*α</sup>	420.6 ± 95.4 <sup>b</sup>	366.1 ± 113.4 <sup>*b</sup>	502.7 ± 139.3 <sup>*ab</sup>
Relative $F_{peak}$ (N·kg <sup>-1</sup> )	8.1 ± 1.2	8.4 ± 1.4	7.8 ± 1.4	8.4 ± 2.0	7.5 ± 2.2	8.3 ± 2.2
Scaled $F_{peak}$ (N·kg <sup>-b</sup> )	8.7 ± 1.2	9.0 ± 1.5	8.3 ± 1.5	8.9 ± 2.1	7.9 ± 2.3	8.8 ± 2.3
FR (W·s <sup>-1</sup> )	137.3 ± 34.7	189.0 ± 86.4*	180.6 ± 73.6*	120.0 ± 41.2 <sup>b</sup>	95.4 ± 52.3 <sup>*b</sup>	159.0 ± 65.6 <sup>*b</sup>
$D_{RF}$ (%·s·m <sup>-1</sup> )	-7.83 ± 1.39	-7.77 ± 1.25	-7.08 ± 1.33 <sup>*α</sup>	-8.50 ± 1.59 <sup>b</sup>	-7.43 ± 1.08 <sup>*b</sup>	-8.10 ± 1.85 <sup>b</sup>

All variables reported as mean ± SD.  $t_{P_{peak}}$  = Time to peak power,  $P_{peak}$  = Peak Power,  $P_{mean}$  = Mean Power,  $V_{peak}$  = Peak Velocity,  $V_{mean}$  = Mean Velocity, 30mT = 30 m Sprint Time,  $F_{peak}$  = Peak Force, FR = Fatigue Rate,  $D_{RF}$  = Mechanical Efficiency Index, PHV = Peak Height Velocity. \* significantly different to pre-pubertal children within the same training group ( $p < 0.05$ ) <sup>α</sup> significantly different to pubertal adolescents within the same training group ( $p < 0.05$ ); <sup>b</sup> significant difference compared to the trained equivalents ( $p < 0.05$ )

**Table 6.3** –30 m sprint performance variables in girls

	Hockey Players			Control Participants		
	Pre-PHV	Circa-PHV	Post-PHV	Pre-PHV	Circa-PHV	Post-PHV
$t_{P_{peak}}$ (s)	0.62 ± 0.11	0.52 ± 0.05	0.59 ± 0.14 <sup>a</sup>	0.48 ± 0.13 <sup>b</sup>	0.44 ± 0.11 <sup>b</sup>	0.61 ± 0.26 <sup>b</sup>
$P_{peak}$ (W)	547.5 ± 179.9	664.7 ± 132.5*	798.6 ± 167.7* <sup>a</sup>	358.3 ± 73.8 <sup>b</sup>	561.6 ± 233.1* <sup>b</sup>	517.1 ± 222.5* <sup>ab</sup>
Relative $P_{peak}$ (W·kg <sup>-1</sup> )	13.2 ± 4.8	13.0 ± 2.1	13.8 ± 2.4	10.5 ± 2.3 <sup>b</sup>	11.1 ± 3.4 <sup>b</sup>	10.1 ± 3.9 <sup>b</sup>
Scaled $P_{peak}$ (W·kg <sup>-b</sup> )	8.6 ± 3.2	8.3 ± 1.4	8.6 ± 1.5	6.9 ± 1.9 <sup>b</sup>	7.1 ± 2.2 <sup>b</sup>	6.4 ± 2.5 <sup>b</sup>
$P_{mean}$ (W)	189.0 ± 56.5	214.3 ± 45.0*	274.9 ± 61.2* <sup>a</sup>	110.8 ± 26.2 <sup>b</sup>	140.3 ± 36.2* <sup>ab</sup>	172.5 ± 39.3* <sup>ab</sup>
Relative $P_{mean}$ (W)	4.6 ± 1.5	4.2 ± 0.87	4.7 ± 0.85*	3.2 ± 0.55 <sup>b</sup>	2.8 ± 0.5 <sup>b</sup>	3.4 ± 0.7* <sup>b</sup>
$V_{peak}$ (m·s <sup>-1</sup> )	6.58 ± 0.62	6.31 ± 0.63	6.68 ± 0.56* <sup>a</sup>	5.51 ± 0.45 <sup>b</sup>	5.16 ± 0.44 <sup>b</sup>	5.70 ± 0.54* <sup>ab</sup>
$V_{mean}$ (m·s <sup>-1</sup> )	5.49 ± 0.50	5.43 ± 0.43	5.62 ± 0.33* <sup>a</sup>	4.79 ± 0.34 <sup>b</sup>	4.61 ± 0.35 <sup>b</sup>	4.84 ± 0.45* <sup>b</sup>
30mT (s)	5.50 ± 0.49	5.55 ± 0.44	5.35 ± 0.31* <sup>a</sup>	6.29 ± 0.45 <sup>b</sup>	6.54 ± 0.52 <sup>b</sup>	6.25 ± 0.62 <sup>b</sup>
$F_{peak}$ (N)	310.8 ± 85.2	393.9 ± 68.7*	447.2 ± 92.1* <sup>a</sup>	240.9 ± 41.6 <sup>b</sup>	395.5 ± 154.4* <sup>b</sup>	434.6 ± 130.3* <sup>ab</sup>
Relative $F_{peak}$ (N·kg <sup>-1</sup> )	7.4 ± 2.0	7.7 ± 0.6	7.7 ± 1.2	7.1 ± 1.3	7.8 ± 2.0	6.5 ± 2.3
Scaled $F_{peak}$ (N·kg <sup>-b</sup> )	7.8 ± 2.1	8.2 ± 0.7	8.2 ± 1.3	7.3 ± 1.7	8.2 ± 2.1	6.9 ± 2.4
FR (W·s <sup>-1</sup> )	102.4 ± 48.7	143.2 ± 40.5*	171.9 ± 47.8*	56.1 ± 19.4 <sup>b</sup>	98.0 ± 48.0* <sup>b</sup>	79.3 ± 48.0* <sup>b</sup>
$D_{RF}$ (%·s·m <sup>-1</sup> )	-7.45 ± 1.37	-8.07 ± 0.65	-7.20 ± 1.35* <sup>a</sup>	-8.04 ± 1.38 <sup>b</sup>	-7.99 ± 1.57 <sup>b</sup>	-6.49 ± 1.57* <sup>ab</sup>

All variables reported as mean ± SD.  $t_{P_{peak}}$  = Time to peak power,  $P_{peak}$  = Peak Power,  $P_{mean}$  = Mean Power,  $V_{peak}$  = Peak Velocity,  $V_{mean}$  = Mean Velocity, 30mT = 30 m Sprint Time,  $F_{peak}$  = Peak Force, FR = Fatigue Rate,  $D_{RF}$  = Mechanical Efficiency Index, PHV = Peak Height Velocity. \* significantly different to pre-pubertal children within the same training group ( $p < 0.05$ ); <sup>a</sup> significantly different to pubertal adolescents within the same training group ( $p < 0.05$ ), <sup>b</sup> significant difference compared to the trained equivalents ( $p < 0.05$ ).

**Table 6.4** – Predictor variables for 30 m time for each maturity group

Group	Predictor Variables	$\beta$	Standard Error	R <sup>2</sup>
Pre-PHV	Training Status	0.55	0.13	0.33 **
	Sex	-0.11	0.10	0.33 **
	Scaled P <sub>peak</sub>	-0.19	0.03	0.60 **
	D <sub>RF</sub>	-0.12	0.04	0.65 **
Circa-PHV	Training Status	0.60	0.09	0.53 **
	Sex	0.19	0.10	0.53 **
	Scaled P <sub>peak</sub>	-0.20	0.02	0.70 **
	D <sub>RF</sub>	-0.14	0.04	0.75 **
Post-PHV	Training Status	0.47	0.07	0.37 **
	Sex	0.06	0.05	0.37 **
	Scaled P <sub>peak</sub>	-0.23	0.02	0.73 **
	D <sub>RF</sub>	-0.13	0.02	0.80 **

PHV = Peak Height Velocity, Scaled P<sub>peak</sub> = Allometrically scaled peak power, D<sub>RF</sub> = Mechanical Efficiency Index. \*\* p < 0.01

#### 6.4 Discussion

This was the first study to investigate the influence of sex, maturity and training status, and their interaction, on the kinetic profile of a maximal sprint utilising radar technology in combination with F-v-P profiling during an ecologically valid sprint. Overall, the findings that boys produced a higher P<sub>peak</sub> and F<sub>peak</sub> than girls even after allometric scaling and irrespective of maturity suggest potential sex-related differences in muscle fibre recruitment. Moreover, given that training and sex account for ~20% more variance in 30mT in pubertal adolescents than pre- and post-PHV children and adolescents. These findings therefore provide evidence that the development of sprint performance is sexually dimorphic which should be considered in the design of training programmes in youth.

A significant interaction between sex, maturity and training status was identified for V<sub>peak</sub>, with a greater difference between trained and untrained circa-PHV participants (19.8%) compared to their pre- and post-PHV counterparts (< 11.0%). This supports the growing body of evidence regarding the non-linear development of sprint performance throughout growth and maturation (Meyers et al., 2015; Meyers et al., 2017a; Moran et al., 2016; Papaiaikovou et al., 2009; Rumpf et al., 2015b; Rumpf et al., 2012), but also indicates the potential potency of training on sprint performance around the time of PHV in boys. Furthermore, this indicates that sprint development

during adolescence may be sexually dimorphic, particularly around PHV, which may be explained, at least in part, by key differences in the hormonal milieu manifest from the onset of puberty. Specifically, close to PHV, boys experience a greater increase in androgenic hormones, including testosterone and growth hormone, than girls, which is associated with increased fat free mass (Farr, Laddu, & Going, 2014; Fellmann & Coudert, 1994a), muscle cross sectional area (Armstrong, 2007; Van Praagh, 2000; Van Praagh & Doré, 2002), and proportion of type II muscle fibres in boys (Van Praagh, 2000; Van Praagh & Doré, 2002). These hormonal changes led to the ‘trigger’ hypothesis being proposed (Katch, 1983) whereby adaptations and performance improvements in response to a training stimuli would be enhanced following the onset of puberty. Whilst the ‘trigger’ hypothesis is largely refuted in relation to cardiorespiratory fitness (Armstrong, 2007; Armstrong & McNarry, 2016; Rowland, 1997), the present study indicates that sprint performance responses to training may be enhanced during puberty in boys.

Despite the non-linear increases in  $V_{\text{peak}}$ , 30mT was only significantly faster in post-PHV adolescents compared to pre-PHV and circa-PHV participants, with no significant differences between pre-PHV and circa-PHV children. These findings are in direct contrast to the Papiakovou et al. (2009) who reported near linear increases in maximum velocity with age. Such discrepancies are likely due to Papiakovou et al. (2009) not accounting for maturity status, with the timing and tempo of maturity varying between individuals, even of the same age, sex and ethnicity (Rogol, 2002; Rogol et al., 2002). Therefore, potential maturational differences between participants within age categories described in Papiakovou et al. (2009) may have produced spurious associations. Nevertheless, the results of the current study are in accord with Meyers et al. (2015) and Rumpf et al. (2015b) who attributed the lack of performance improvements in pubertal boys to ‘adolescent awkwardness’ (Buenen et al., 1998). Adolescent awkwardness is a phenomenon attributed to a period around PHV where adolescents experience a decline or plateau in performance, thought to be reflective of a temporary disruption in motor control (Buenen et al., 1998). Whilst adolescent awkwardness does not affect all adolescents (Lloyd et al., 2015), the present study supports this hypothesis, but also suggests girls may be more susceptible to adolescent awkwardness than boys. More specifically, pubertal girls, irrespective of training



status, had a lower  $V_{\text{peak}}$  and a slower 30mT compared to their pre- or post-PHV counterparts. It is, however, pertinent to note that currently no objective maximal criteria for anaerobic performances are available and it could therefore be postulated that sub-maximal efforts may have been accepted in the pubertal girls, although this seems unlikely given the motivation provided during each sprint, the longer sprint distance to minimise deceleration, and the consistency of performance decline observed in all participants. Nevertheless, future research is warranted to establish maximal sprint criteria and to further elucidate the potential underlying mechanisms for these apparent sex differences.

The lack of differences in 30mT between pre-PHV children and circa-PHV adolescents in the present study could be explained, at least in part, by the lack of significant difference in the technical ability to apply force, indicated by  $D_{\text{RF}}$ . Indeed,  $D_{\text{RF}}$  was only significantly lower in post-pubertal adolescents, irrespective of sex or training status, compared to both pre- and circa-PHV children. A more positive  $D_{\text{RF}}$  indicates a greater ability to maintain a greater horizontal force production at higher sprinting velocities (Morin et al., 2011; Rossi et al., 2017), with  $D_{\text{RF}}$  shown to be more important for sprint performance than total force production in a sample of recreationally active adults (Morin et al., 2011). These results are congruent with the only other study reporting changes in  $D_{\text{RF}}$  in a paediatric population which reported a significant difference in  $D_{\text{RF}}$  between children and adolescents (Rossi et al., 2017). In accord with the present study, these observations were independent of relative  $F_{\text{peak}}$ , and allometrically scaled  $F_{\text{peak}}$ , which remained constant between children and adolescents. Building on the findings of Rossi et al. (2017), the current study shows that maturity-, as well as age-, related, differences in  $D_{\text{RF}}$  may also be evident and explain a significant proportion of variance in sprint performance. Maturity-related differences in  $D_{\text{RF}}$  may be attributable to differences in segmental growth rates in relation to the trunk (Rumpf et al., 2015b). However, given the cross-sectional nature of this study, no conclusions regarding the impact of differing growth rates can be drawn, thus necessitating future research.

Whilst there are strengths associated with the current study, including the large sample size and the quantification of sprint kinetics in field-based settings thus enhancing the ecological validity, there are limitations which must be acknowledged. First, no

spatiotemporal variables (i.e. stride length) were assessed which could have provided greater insight into the kinetic and spatiotemporal interaction on sprint development in youth. Furthermore, whilst all trained participants were part of a LTAD program, they were all involved in the same training regime, precluding inferences regarding the effectiveness of different training methodologies on the kinetic sprint profile. In the absence of no objective criteria of maximal effort, it is possible that some participants produced submaximal efforts, potentially producing spurious associations. However, motivational techniques were used throughout all tests, which, coupled with an extended finish line (35 m), minimised this risk. Additionally, F-v-P profiling does not allow the power and force variables of specific muscles to be elucidated, precluding the development of specific training interventions. Finally, the ecological validity of a single sprint has been questioned, especially in team sports (Mendez-Villanueva et al., 2010; Mujika et al., 2009; Rommers et al., 2018), thus repeated sprint ability may provide greater insights into fatiguability.

In conclusion, this was the first study to examine kinetic changes in sprint development in a large sample of trained and untrained boys and girls, accounting for maturity status. Sprint performance increases may be attributed to increases in power, and an improved technical ability to apply force, irrespective of sex. Moreover, this study provides evidence that girls may be more susceptible to 'adolescent awkwardness' than boys though, as the first study to investigate the kinetic sprint profile in girls, future research is warranted to establish the underlying mechanisms in more detail. Furthermore, studies should seek to establish the kinetic sprint profile over repeated sprints to identify the fatiguing mechanisms in paediatric populations.

# Chapter 7

Understanding the kinetics of  
repeated sprint ability using radar  
technology in national level  
adolescent hockey players

## **Chapter 7 (Study 4) – Understanding the kinetics of repeated sprint ability using radar technology in national level adolescent hockey players**

### **7.1 Introduction**

Repeated sprint ability (RSA), defined as the ability to repeatedly reproduce consistent maximal efforts (Girard et al., 2011), is fundamental to athletic performance in team sports and routinely assessed in long-term athlete development (LTAD) programs and talent identification batteries (Girard et al., 2011; Mendez-Villanueva et al., 2010; Moran et al., 2016; Papaiaikovou et al., 2009). The development of single-sprint performance during childhood and adolescence is thought to be a non-linear process, with accelerated periods of development around the time of peak height velocity (PHV; Mendez-Villanueva et al., 2010; Mujika et al., 2009; Philippaerts et al., 2006; Spencer et al., 2011). However, changes in RSA are less well understood; to elucidate the development of RSA, further consideration of the determinants of RSA is required.

The determinants of RSA have been postulated to be both physiological and biomechanical in nature (Girard et al., 2011; Morin et al., 2011; Morin et al., 2006; Rossi et al., 2017; Rumpf et al., 2013; Rumpf et al., 2015b; Samozino et al., 2016). Indeed, from a biomechanical perspective, sprint performance is suggested to be directly proportional to peak horizontal force ( $F_{\text{peak}}$ ) and peak power ( $P_{\text{peak}}$ ) during the initial acceleration (Morin et al., 2011; Rabita et al., 2015). An athlete's capacity to produce  $F_{\text{peak}}$  whilst running is well described by the force-velocity relationship (Morin et al., 2006; Rossi et al., 2017; Samozino, 2018), which characterises the theoretical limits of the entire neuromuscular system and the theoretical exponents of  $F_{\text{peak}}$ ,  $P_{\text{peak}}$  and maximum velocity ( $V_{\text{max}}$ ; Samozino et al. 2016). These mechanical variables appear to be the primary determinants of single-sprint performance in irrespective of age and maturity ( $r^2$ : 0.98 - 0.99; Rumpf et al., 2015b). However, the relative contribution of performance predictors has been suggested to be dependent on maturity stage (Meyers et al., 2015; Meyers et al., 2017a; Papaiaikovou et al., 2009; Philippaerts et al., 2006), with vertical stiffness, a reflection of the ability to tolerate and overcome gravitational forces, being the greatest predictor of sprint performance

in pubertal adolescents (Rumpf et al., 2013; Rumpf et al., 2015b). This potential role of maturity necessitates the need for a robust, ecologically valid method so that repeated sprint determinants, and development, can be monitored. Indeed, the results of Rumpf et al. (2015a, 2015b) must be interpreted with caution, due to the use of a non-motorised treadmill and derivation of the biomechanical parameters from only the four fastest consecutive steps in Rumpf et al. (2015b), limiting their generalisability to real-world environments.

The mechanisms underpinning  $F_{\text{peak}}$  and  $P_{\text{peak}}$  are multifaceted and represent a complex interaction between neural, anthropometric and morphological factors, and individual muscle properties (Morin et al., 2011; Morin et al., 2006; Rabita et al., 2015). Interestingly, the technical ability to apply force ( $D_{\text{RF}}$ ) has recently been postulated to be more influential than absolute  $F_{\text{peak}}$  (Morin et al., 2011; Rossi et al., 2017), highlighting that technique and skill proficiency may also be of importance. Whilst the influence of  $D_{\text{RF}}$  has been reported in world-class (Rabita et al., 2015) and masters athletes (Slawinski et al., 2017), little is known about its role in sprint performance in youth athletes. Indeed, it was recently reported that single-sprint  $D_{\text{RF}}$  improved with age in a sample of 68 children and adolescents using a novel field-based methodology (Rossi et al., 2017). However, whether  $D_{\text{RF}}$  is equally important for multiple-sprint performance remains largely unknown and thus further research is therefore required to delineate the potential effects of age and maturation and their interaction with training on the determinants of sprint performance.

Rossi et al. (2017) used radar technology, enabling near instantaneous measures of velocity ( $> 46$  Hz), coupled with macroscopic biomechanical modelling, to estimate force and power variables in field-based settings (Samozino, 2018; Samozino et al., 2016). This radar technology has the ability to measure both inbound and outbound velocities (Simperingham et al., 2017; Simperingham et al., 2016), thereby enabling the quantification of between sprint differences. Indeed, such high-resolution quantification of the kinetics underpinning repeated sprints could not only further our understanding of, but also facilitate targeted interventions to improve, RSA during childhood and adolescence (Girard et al., 2011; Samozino et al., 2016; Simperingham et al., 2017). Therefore, the aim of this pilot study was to assess RSA using radar

technology and biomechanical modelling in trained children and adolescents to gain a greater understanding of the underlying kinetics during repeated over-ground sprints.

## 7.2 Methods

Twenty children and adolescents (n=15 girls; Table 1) involved in international age-group hockey tournaments and part of a LTAD program overseen by the national governing body participated. A pre-screening medical questionnaire and informed parent/guardian consent were completed online using a custom-built consent form (Survey Monkey, Dublin, Ireland). Written participant assent was obtained on the day of testing. Ethics approval was obtained from the institutional ethics committee and the study conformed to the Declaration of Helsinki.

### *7.2.1 Experimental Procedures*

Standing and sitting stature were measured to the nearest 0.1 cm using a Seca 213 portable stadiometer (Seca 213, Seca, Chino, CA, USA), with body mass measured to the nearest 0.1 kg using a set of electronic scales (Seca 803, Seca, Chino, CA, USA). Maturity offset was subsequently calculated using the equations of Mirwald et al. (2002a).

Prior to undertaking the repeated-sprint protocol, participants undertook a standardised five-minute low-intensity warm-up, culminating in two 15 m sprints that simultaneously served as familiarisation with the sprint protocol. For the repeated-sprint protocol, participants completed five 20 m shuttle sprints, turning 180° every 20 m. To minimise the potential confounding effects of differences in turning speed between participants and sprints, participants were required to stop for 5 s before accelerating into the next maximal sprint. Sprint times and kinetic variables were subsequently derived from the initial 15 m to minimise the effects of deceleration. Participants started from a two-point standing start to reduce vertical displacement during the early phase of the sprint (Mero et al., 1992) and were instructed to start using auditory cues (i.e. '3.....2.....1.....GO!').

All sprints were completed on outdoor AstroTurf pitches, with the mean air temperature and wind speed being  $15.8 \pm 0.8^{\circ}\text{C}$  and  $1.6 \pm 0.8 \text{ m}\cdot\text{s}^{-1}$ , respectively. Velocity was measured throughout all sprints using a STALKER ATS II radar gun

(STALKER, Plano, Texas, USA) mounted on a tripod positioned 10 m behind the start line, in accord with manufacturer recommendations. The STALKER ATS II has a recording frequency of 46.875 Hz, allowing for near-instantaneous power and force variables to be modelled. The data was first segmented into five sections to represent each repeated sprint and, subsequently, the first reading of each new sprint was assigned time 0, and the first 0.3 s deleted in line with previous recommendations (Samozino, 2018). Using the biomechanical model of Samozino et al. (2016), the following parameters were derived for each sprint: time to peak power ( $t_{P_{peak}}$ ), absolute, relative and scaled peak power ( $P_{peak}$ ), mean power ( $P_{mean}$ ), relative mean power (relative  $P_{mean}$ ), peak and mean velocity ( $V_{peak}$  and  $V_{mean}$ , respectively), 15 m sprint time (15mT), absolute, relative, and allometrically scaled peak force ( $F_{peak}$ ), fatigue rate (FR) and  $D_{RF}$ . Furthermore, to aid comparisons with previous literature, the fatigue index (FI) was calculated using the formula reported elsewhere (Mujika et al., 2009).

### 7.2.2 Statistics

All statistical analyses were conducted in SPSS (Version 26.0, IBM, Armonk, NY, USA), with values presented as mean  $\pm$  SD. Sex differences in anthropometric variables were assessed using an independent samples t-test. A repeated measures ANOVA was used to compare performance parameters between sprints, with Bonferroni corrections applied where necessary. Cohens  $d$  effect sizes were also calculated to determine trivial ( $\leq 0.20$ ), moderate ( $\geq 0.21 - \leq 0.60$ ), large ( $\geq 0.61 - \leq 0.80$ ), and very large ( $\geq 0.81$ ) effects, respectively. Hierarchical linear regressions were used to ascertain the biomechanical determinants of each 15 m sprint repetition. Allometrically scaled  $P_{peak}$  was initially added into the model due to the emerging associations with single-sprint performance in children and adolescents (Rumpf et al., 2015a; Rumpf et al., 2015b). Subsequently, predictor variables were entered into the models to determine the independent association with each repetition's 15mT, with inclusion into the model accepted if a significant increase in explained variance was observed at the 0.05 confidence interval. Collinearity checks were conducted using the variance inflation factor (VIF), with a VIF  $< 1$ , between 1 and 5, and greater than 5 indicating low, moderate and high collinearity, respectively (Daoud, 2017). If high multi-collinearity was found between variables, the variable explaining the greatest

proportion of variance was included in the model (Daoud, 2017). The accuracy and suitability of the model was assessed using the normality of residual values.

To assess overall RSA, the mean percentage decline from the first to last sprint was calculated for each mechanical variable with a subsequent hierarchical stepwise linear regression used to ascertain which variables predicted the decline in performance according to the 15mT. Pearson's correlations were performed to establish the relationship between the relative declines in kinetic variables over the repeated sprints.

### 7.3 Results

There were no sex differences in any anthropometric or repeated sprint parameter ( $p > 0.05$ ), so data from both sexes were pooled for analysis. The repeated measures ANOVA demonstrated a main effect for sprint number, with *post-hoc* analyses demonstrating that, as shown in Figure 1, this was primarily attributable to differences between the first two sprints and all subsequent sprints. More specifically, there was a main effect for  $P_{\text{peak}}$  regardless of how it was expressed, with *post-hoc* tests revealing  $P_{\text{peak}}$  during sprint 1 was significantly higher than sprint 4 ( $d = 0.54 - 0.75$ ; Table 2).  $P_{\text{mean}}$ , relative  $P_{\text{mean}}$ , and  $D_{\text{RF}}$  were significantly higher during sprint 1 compared to all other sprints ( $P_{\text{mean}}$ :  $F_{(4,76)} = 23.7$ ,  $p < 0.01$ , relative  $P_{\text{mean}}$ :  $F_{(4,76)} = 24.7$ ,  $p < 0.01$ ,  $D_{\text{RF}}$ :  $F_{(4,76)} = 2.6$ ,  $p < 0.01$ ), with  $P_{\text{mean}}$  and relative  $P_{\text{mean}}$  also significantly higher during sprint 2 compared to sprint 4 ( $p < 0.01$ ,  $d = 0.45$ ). There was an overall effect sprint number on  $V_{\text{peak}}$  ( $F_{(4,76)} = 29.4$ ,  $p < 0.01$ ),  $V_{\text{mean}}$  ( $F_{(4,76)} = 17.4$ ,  $p < 0.01$ ) and 15mT ( $F_{(4,76)} = 17.1$ ,  $p < 0.01$ ), with *post-hoc* analyses revealing that  $V_{\text{peak}}$ ,  $V_{\text{mean}}$  and 15mT decreased from sprints 1-4, before increasing slightly in sprint 5 (Table 2). However, as shown in Figure 1c, there were no significant differences in  $F_{\text{peak}}$ , irrespective of whether it was expressed in absolute, relative, or allometrically scaled units, over the five sprint repetitions (all  $p > 0.05$ ). Additionally,  $t_{\text{P}_{\text{peak}}}$  and FR did not significantly change with each sprint repetition. The average FI over the five sprint repetitions was  $11.0 \pm 3.9\%$ .

#### *3.1 Biomechanical determinants of repeated sprints*

In model 1 in which only allometrically scaled  $P_{\text{peak}}$  was entered, 74 – 82% of the variance in the 15mT from sprints 1 – 4 was explained, with the explained variance



lower for sprint 5 (62%).  $D_{RF}$  was found to be a significant additional predictor of sprint performance for sprints 1, 2, 3 and 5, increasing the explained variance to 84 – 92%. Mean allometrically scaled  $P_{peak}$  across the five-sprints explained a large proportion of the variance in the mean 15mT (81%), with the inclusion of mean  $D_{RF}$  significantly increasing the explained variance to 90%. The hierarchical linear regression revealed that percentage declines in  $V_{max}$  was the strongest predictor of 15mT (37.9%), followed by  $D_{RF}$  (34.7%) and allometrically scaled  $F_{peak}$  (18.9%). Subsequently, the percentage decline in 15mT was described by:

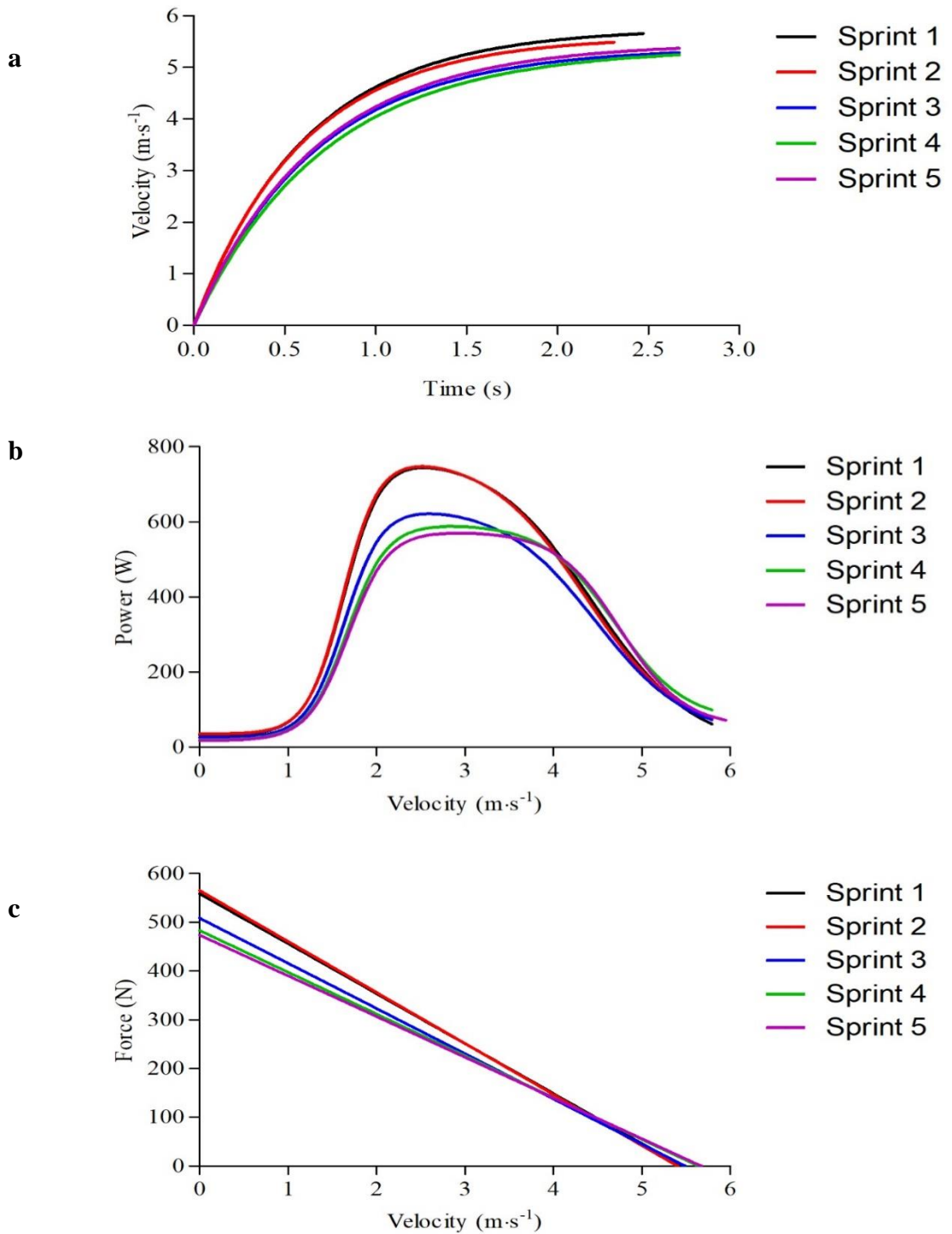
$$\text{Percentage decline in 15mT} = -0.903 + (0.629 * V_{max}) + (0.143 * D_{RF}) + (0.415 * \text{allometrically scaled } F_{peak})$$

A significant correlation was evident between the percentage decline in 15mT and allometrically scaled  $P_{peak}$  ( $R^2 = 0.76$ ,  $p < 0.01$ ) but  $P_{peak}$  was not entered into the regression model due to its high collinearity with  $V_{max}$  and  $F_{peak}$ . Declines in  $F_{peak}$  were more strongly related to declines in  $P_{peak}$  during the repeated sprints ( $R^2 = 0.89$ ,  $p < 0.01$ ) than changes in  $V_{max}$  ( $R^2 = 0.42$ ,  $p < 0.05$ ).

**Table 7.1** – Participant descriptives

	Mean ± SD
Age (years)	14.4 ± 1.0
Height (m)	1.66 ± 0.08
Weight (kg)	58.0 ± 10.4
BMI (kg·m <sup>-2</sup> )	21.1 ± 3.2
Maturity Offset (years)	0.75 ± 0.23

BMI = Body Mass Index



**Figure 7.1** – Post-processed representative traces from the five sprint repetitions for a typical participant showing a) velocity-time profile, b) power-velocity profile and c) the force velocity profile.

**Table 7.2** – Sprint variables from each of the 5 x 15m sprint repetitions across all five sprints

	Sprint 1	Sprint 2	Sprint 3	Sprint 4	Sprint 5	Significant Differences
t <sub>P<sub>peak</sub></sub> (s)	0.56 ± 0.12	0.52 ± 0.11	0.50 ± 0.12	0.50 ± 0.07	0.53 ± 0.11	-
P <sub>peak</sub> (W)	732.0 ± 268.3	647.9 ± 190.8	659.7 ± 238.8	606.2 ± 190.7 <sup>a</sup>	643.3 ± 198.8	1 – 4, <i>d</i> = 0.54
Relative P <sub>peak</sub> (W·kg <sup>-1</sup> )	12.6 ± 3.7	11.2 ± 2.6	11.2 ± 2.8	10.4 ± 2.2 <sup>a</sup>	11.0 ± 2.3	1 – 4, <i>d</i> = 0.72
Scaled P <sub>peak</sub> (W·kg <sup>-b</sup> )	7.9 ± 2.3	7.0 ± 1.7	7.0 ± 1.7	6.5 ± 1.3 <sup>a</sup>	6.9 ± 1.4	1 – 4, <i>d</i> = 0.75
P <sub>mean</sub> (W)	362.1 ± 95.8	318.0 ± 91.4 <sup>a</sup>	308.2 ± 102.0 <sup>a</sup>	287.4 ± 91.4 <sup>a,b</sup>	286.7 ± 77.6 <sup>a</sup>	1 – All other sprints ( <i>d</i> = 0.47 – 0.86) 2 – 4, <i>d</i> = 0.33
Relative P <sub>mean</sub> (W·kg <sup>-1</sup> )	6.3 ± 1.4	5.5 ± 1.4 <sup>a</sup>	5.3 ± 1.3 <sup>a</sup>	5.0 ± 1.2 <sup>a,b</sup>	5.0 ± 1.1 <sup>a</sup>	1 – All other sprints ( <i>d</i> = 0.57 - 1.03) 2 – 4, <i>d</i> = 0.40
V <sub>peak</sub> (m·s <sup>-1</sup> )	5.87 ± 0.51	5.51 ± 0.50 <sup>a</sup>	5.40 ± 0.48 <sup>a,e</sup>	5.30 ± 0.45 <sup>a,b,e</sup>	5.61 ± 0.52 <sup>a</sup>	1 – All other sprints ( <i>d</i> = 0.71 - 1.16) 2 – 4, <i>d</i> = 0.42 4 – 5, <i>d</i> = 0.64
V <sub>mean</sub> (m·s <sup>-1</sup> )	4.40 ± 0.40	4.15 ± 0.30 <sup>a,e</sup>	4.12 ± 0.30 <sup>a,e</sup>	4.05 ± 0.27 <sup>a,e</sup>	4.35 ± 0.35	1 – Sprints 2,3 and 4 ( <i>d</i> = 0.71 - 1.13) 5 – Sprints 2, 3 and 4 ( <i>d</i> = 0.58– 0.96)
T15m (s)	3.43 ± 0.31	3.63 ± 0.29 <sup>a,e</sup>	3.67 ± 0.29 <sup>a,e</sup>	3.73 ± 0.28 <sup>a,e</sup>	3.47 ± 0.29	1 – Sprints 2,3 and 4 ( <i>d</i> = 0.71 – 1.13) 5 – Sprints 2, 3 and 4 ( <i>d</i> = 0.58 – 0.96)
F <sub>peak</sub> (N)	445.2 ± 151.7	421.4 ± 112.1	435.3 ± 139.4	411.9 ± 110.3	417.7 ± 118.5	-
Relative F <sub>peak</sub> (N·kg <sup>-1</sup> )	7.7 ± 2.0	7.3 ± 1.4	7.4 ± 1.5	7.1 ± 1.0	7.2 ± 1.3	-
Scaled F <sub>peak</sub> (N·kg <sup>-b</sup> )	3.6 ± 0.9	3.5 ± 0.7	3.5 ± 0.7	3.4 ± 0.4	3.4 ± 0.6	-
FR (W·s <sup>-1</sup> )	272.6 ± 150.8	245.7 ± 99.4	263.9 ± 133.9	243.2 ± 106.1	211.6 ± 90.2	-
DRF (%·s·m <sup>-1</sup> )	-7.64 ± 1.03	-8.63 ± 1.44 <sup>a</sup>	-8.51 ± 1.49 <sup>a</sup>	-8.93 ± 1.19 <sup>a</sup>	-8.34 ± 1.45 <sup>a</sup>	1 – All other Sprints ( <i>d</i> = 0.40 - 0.78)

All variables presented as mean ± SD. t<sub>P<sub>peak</sub></sub> = Time to Peak Power, P<sub>peak</sub> = Peak power, P<sub>mean</sub> = Average power, V<sub>peak</sub> = Peak Velocity, V<sub>mean</sub> = Average velocity, T15m = 15m sprint time, F<sub>peak</sub> = Peak Force, FR = Fatigue Rate, DRF = Mechanical Efficiency Index. <sup>a</sup> significantly different compared to sprint 1, <sup>b</sup> significantly different compared to sprint 2, <sup>c</sup> significantly different compared to sprint 3, <sup>d</sup> significantly different compared to sprint 4, <sup>e</sup> significantly different compared to sprint 5.

**Table 7.3** – Biomechanical determinants of 15 m sprint time for each repetition.

Sprint Number	Predictor Variables	$\beta$	Standard Error	R <sup>2</sup>
1	Scaled P <sub>peak</sub>	-0.12	0.02	0.78 **
	D <sub>RF</sub>	-0.04	0.02	0.84 *
2	Scaled P <sub>peak</sub>	-0.15	0.02	0.74 **
	D <sub>RF</sub>	-0.08	0.02	0.88 **
3	Scaled P <sub>peak</sub>	-0.15	0.02	0.82 **
	D <sub>RF</sub>	0.07	0.02	0.92 **
4	Scaled P <sub>peak</sub>	-0.19	0.02	0.79 **
5	Scaled P <sub>peak</sub>	-0.16	0.03	0.62 **
	D <sub>RF</sub>	-0.20	0.02	0.84 **

Scaled P<sub>peak</sub> = Allometrically Scaled P<sub>peak</sub>, D<sub>RF</sub> = Mechanical Efficiency Index, \* p < 0.05, \*\* p < 0.01

#### 7.4 Discussion

This study was the first to utilise radar technology in combination with macroscopic biomechanical modelling to gain greater insights into the mechanical properties underpinning repeated-sprint ability in adolescents. The main findings of the current study indicate that declines in allometrically scaled P<sub>peak</sub> are more closely related to declines in F<sub>peak</sub> than peak velocity. Moreover, the similar kinetic determinants revealed that, irrespective of sprint number, the kinetic determinants of single sprints are also key to RSA performance. Finally, D<sub>RF</sub> was found to be a significant predictor of both single- and multiple-sprint kinetics. The results of this study therefore provide important insights for designing and implementing training interventions to improve RSA in youth athletes.

Data from non-motorised treadmills in boys (Rumpf et al., 2015b) and force platforms in girls (Nagahara et al., 2019) indicate that P<sub>peak</sub> and F<sub>peak</sub> are key determinants of single-sprint performance during youth. In contrast, the present study suggests that D<sub>RF</sub>, an indication of mechanical efficiency, is a greater predictor of single-sprint performance than F<sub>peak</sub>. This is in agreement with data in trained youth (Rossi et al., 2017) and adult (Morin et al., 2011) sprinters, which was attributed to F<sub>peak</sub> being the sum of both horizontal and vertical forces, with the latter not significantly influencing performance (Morin et al., 2011; Morin et al., 2006). However, D<sub>RF</sub> represents the linear decline in the ratio of forces (horizontal: vertical) with increasing velocity, and

may therefore be more performance-orientated (Samozino et al., 2016; Slawinski et al., 2017). Of note,  $D_{RF}$  has also been reported to significantly decline in repeated cycling sprints, whereby participant's force-efficiency decreased as pedalling frequency increased (Sanderson, 1991). Whilst it is not possible to exert zero vertical force (and subsequently have a ratio of forces of 100%) as this would preclude running motion (Morin et al., 2011; Morin et al., 2006), future research should seek to establish optimal values that could improve both single and repeated sprints.

Allometrically scaled  $P_{peak}$  and velocity were significantly higher in sprint 1 compared to all other sprints, with the decline in allometrically scaled  $P_{peak}$  reaching significance from sprint 4. In contrast, there were no significant differences between any sprints for  $F_{peak}$ , regardless of how it was expressed. Therefore, it was surprising that declines in  $F_{peak}$  were the primary cause of the reductions in  $P_{peak}$  over the course of the five sprints. Nevertheless, previous research in children has reported that relative  $F_{peak}$  has a very strong relationship with step length and flight length (the distance travelled by the COM from toe-off to touchdown; Lloyd, Meyers, Hughes, Cronin, & Oliver, 2016). Thus, it may be possible that even small changes in relative  $F_{peak}$  may negatively impact upon these crucial kinematic variables, thereby lowering velocity (Lloyd et al., 2016; Meyers et al., 2017b). Of note, allometrically scaled  $F_{peak}$  and  $P_{peak}$  explained significantly more variance in both individual sprint 15mT and the overall decline in 15mT across the five sprint repetitions than absolute or relative values. This may be due to the near-linear relationship observed between  $P_{peak}$ ,  $F_{peak}$  and body mass, irrespective of sex (Doré et al., 2008; Doré et al., 2005), with ratio-scaling consequently penalising heavier, more mature individuals (Nevill et al., 2006; Welsman & Armstrong, 2019). Allometric scaling, unlike conventional ratio scaling, allows the specific exponent of body mass to be calculated, and is currently the most robust statistical method to account for body mass differences in aerobic fitness (Nevill et al., 2006; Welsman & Armstrong, 2019). This study therefore recommends future research utilises allometric scaling techniques when investigating anaerobic performance to provide more meaningful and generalisable results.

In the present study, similar mechanical determinants of single and repeated sprints were evident, therefore suggesting that single- and repeated-sprint ability may be governed by the same mechanical properties. From a physiological perspective,

declines in  $P_{\text{peak}}$  during repeated cycling sprints have primarily been attributed to neuromuscular fatigue, arising from increases in blood lactate concentrations and associated reductions in intramuscular pH (Ratel, Duche, Hennegrave, Van Praagh, & Bedu, 2002; Ratel, Duche, & Williams, 2006a). Such reductions inhibit the ability to recruit type II higher-order muscle fibres, with changes in motor-unit recruitment patterns in the quadriceps, measured using an electromyogram, explaining 97% of the total work during repeated cycling sprints, interspersed with 30 s rest in untrained adults (Girard et al., 2011). Moreover, a reduction in pH may lead to reductions in motor co-ordination (Doré et al., 2005; Ratel, Williams, Oliver, & Armstrong, 2006b), which may also offer an explanation for  $D_{\text{RF}}$  as a predictor of fatigue during repeated sprints. However, although similar fatiguing pathways seem likely (Ratel et al. 2006), whether the same physiological mechanisms are also responsible for the declines in running performances remains to be established.

The trained hockey players in the present study showed a higher decrement in performance, as measured using FI (11.9%), to that reported in highly trained footballers (4.1 – 5.5%; Girard & Farooq, 2012; Mujika et al., 2009), most likely due to differences in the repeated-sprint protocol. Specifically, most repeated-sprint studies incorporate 10 – 30 s of rest between repetitions (Meckel, Machnal, & Eliakim, 2009; Mendez-Villanueva et al., 2010; Mujika et al., 2009; Temfemo et al., 2011), whereas this study utilised a near-continuous protocol. Whilst it could be argued that incorporating rest between sprints is more indicative of team-sport scenarios (Mendez-Villanueva et al., 2010), the inclusion of rest periods facilitates aerobic recovery. Indeed, previous paediatric research found a significant correlation between aerobic capacity and fatigue resistance during repeated-sprint protocols (Mendez-Villanueva et al., 2007). Moreover, Dupont et al. (2005) reported that the magnitude of change in sprint time was negatively correlated with the speed of the pulmonary  $\dot{V}O_2$  kinetics in adults ( $r^2 = 0.80$ ,  $p < 0.01$ ), with faster pulmonary  $\dot{V}O_2$  kinetics postulated to spare intramuscular phosphocreatine for the later sprints and thereby increasing RSA performance (Dupont et al., 2005). Therefore, future research should seek to establish physiological determinants of repeated-sprint performance in children and adolescents, to further explain the declines in mechanical variables during sprint running.

It may be pertinent to note that the last sprint (sprint 5) was faster than sprints 2, 3 and 4, perhaps highlighting that future research should incorporate an increased number of sprint repetitions to induce greater fatigue, as well as the potential role of pacing resulting in sub-maximal sprints (Bishop & Edge, 2006; Impellizzeri et al., 2008; Ratel et al., 2006b). In the absence of criteria to determine a maximal effort, it could be postulated that an element of pacing is involved within all repeated-sprint protocols, irrespective of recovery period. The present study utilised specific strategies to increase and maintain motivation during repeated sprints, including a longer finish line distance (Mendez-Villanueva et al., 2010) and verbal encouragement (Mujika et al., 2009; Philippaerts et al., 2006). However, no studies have examined differences between trials with and without motivational techniques so it is unclear whether these techniques mitigate the role of pacing.

Although there are numerous strengths to this study, certain limitations must be noted. Data collection was curtailed by COVID-19 resulting in a relatively small sample size, thereby limiting generalisability. Furthermore, whilst the macroscopic biomechanical model used provides an overview of the kinetics underpinning sprint performance, muscle-specific inferences cannot be made. Consequently, the specific muscles responsible for the observed power reduction cannot be established, limiting intervention specificity. Finally, all participants within this pilot study were circa-PHV and therefore no inferences can be made thus far about how the determinants of RSA change with respect to PHV.

In conclusion, radar technology in combination with macroscopic biomechanical modelling provides a useful tool for assessing kinetic and kinematic changes in repeated-sprint performance. Future studies should seek to establish the RSA development in untrained children and adolescents, so the trainability of RSA, and any sex, or maturity, differences can be established.

# Chapter 8

Health Consequences of an elite sporting career – long-term detriment or long-term gain? A meta-analysis of 165,000 former athletes



## **Chapter 8 (Study 5) – Health consequences of an elite sporting career – long-term detriment or long-term gain? A meta-analysis of 165,000 former athletes**

### **8.1 Introduction**

The benefits associated with regular exercise for physical and mental health in the general population are well-evidenced, with inactivity strongly correlated with an increased risk of premature mortality (Gremeaux et al., 2012; Lee et al., 1995; Paffenbarger & Lee, 1998; Sharma, Merghani, & Mont, 2015). Indeed, mortality associated with cardiovascular disease (CVD) and cancer, the most prevalent causes of mortality worldwide (Roser & Ritchie, 2019), is exacerbated by physical inactivity (Al-Mallah et al., 2018; Nystoriak & Bhatnagar, 2018) and decreased by regular exercise (Al-Mallah et al., 2018; S Sarna, Kaprio, Kujala, & Koskenvuo, 1997). Specifically, it is suggested that for every 1 unit increase in maximal metabolic equivalent of task (MET) capacity, the likelihood of CVD mortality is reduced by 15% (Al-Mallah et al., 2018). Similarly, cancer incidence and mortality rates were 27% and 37% lower, in the fittest and least fit group, respectively, in a 16-year longitudinal study of Finnish men (Laukkanen et al., 2010). Furthermore, this relationship persisted even after accounting for smoking habits, alcohol intake, waist-to-hip ratio, socioeconomic status and nutritional intake, highlighting the importance of exercise in the prevention of cancer (Laukkanen et al., 2010).

Despite the benefits associated with regular exercise, there is a body of evidence that suggests the exercise-longevity relationship may be ‘J’ shaped, with exercise beyond certain volume and intensity thresholds detrimental to health (M. Armstrong et al., 2015a; Mohlenkamp et al., 2008; O’Keefe, Lavie, & Guazzi, 2015; O’Keefe, O’Keefe, & Lavie, 2018; O’Keefe et al., 2012; Schnohr et al., 2015). Specifically, Mohlenkamp et al. (2008) reported that, over a two-year observational period, recreational German marathon runners had a similar incidence of a cardiovascular (CV) event compared to a population with established coronary heart disease (CHD). Furthermore, the Copenhagen Heart Study reported light and moderate joggers to demonstrate lower mortality hazard ratios (0.22 and 0.66, respectively) compared to strenuous joggers

(HR: 1.97) (Schnohr et al., 2015). Similarly, those who exercised every day in the Million Women study were at an increased risk of a CV event compared to women who had at least one rest day during the week (M. Armstrong et al., 2015a).

Elite athletes typically engage in training at levels far exceeding those reported in epidemiological studies, raising questions as to whether elite athletes are potentially at an elevated risk of premature mortality, CVD and/or cancer (M. Armstrong et al., 2015a; O'Keefe et al., 2018; O'Keefe et al., 2012). Such a concept has received considerable research attention. Indeed, two recent systematic reviews and a meta-analysis investigated the relationship between long-term intensive training, health, and mortality in elite athletes and the general population (Garatachea et al., 2014; Lemez & Baker, 2015; Teramoto & Bungum, 2010b). Taken together, these reviews suggest that elite athletes live longer than the general population and have a lower mortality rate from both CVD and cancer (Garatachea et al., 2014; Lemez & Baker, 2015; Teramoto & Bungum, 2010b). However, these reviews did not stratify by sport type (i.e. aerobic, power, team sports). Consequently, the importance of training types and sporting demands therefore largely remains to be elucidated. For example, in comparison to endurance athletes, power (POW) sport athletes have an increased body mass index (BMI; Agrotou et al., 2013; Benedettini, 2005), which is an independent risk factor for future CVD (Attard et al., 2013). Furthermore, endurance (END) training has been shown to lower several key inflammatory markers (Mikkelsen et al., 2013), which, whilst this remains contentious, could reduce the risk of long-term CVD risk (Mohlenkamp et al., 2008; Rosin, 2017).

Therefore, the aim of this systematic review and meta-analysis was to examine the relationship between chronic intensive exercise training and mortality in former elite athletes, according to sport type, in comparison to their non-elite counterparts.

## 8.2 Methods

### *8.2.1 Data sources, literature search and inclusion criteria*

This systematic review was registered on PROSPERO (registration number: CRD42019130688) and was conducted in accordance with the PRISMA guidelines (Moher et al., 2015; Shamseer et al., 2015). The key words were split into three levels

to search scientific databases and were compromised of the following i) *mortality* or *death* or *longevity*; ii) *elite* or *athletes* or *Olympic*; and iii) *excessive* or *training* or *chronic exercise*. All key words were used in combination and different iterations to capture all results, with the full search terms available in the supplementary material.

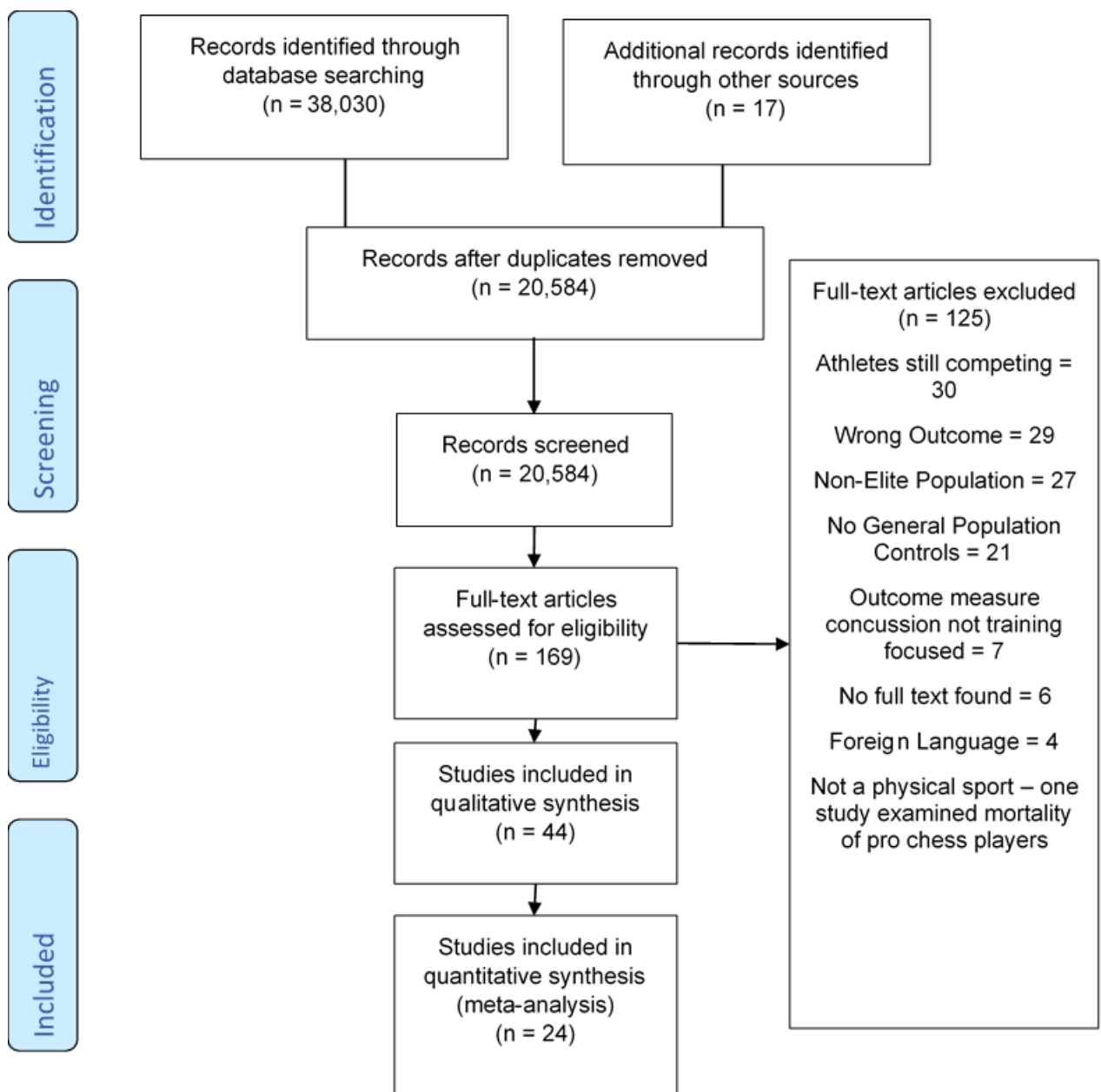
The inclusion criteria for studies in the meta-analysis was: (i) written in the English language; (ii) experimental participants were male or female former athletes of at least national standard, with some information on their sporting history provided; (iii) the study included a general population reference group; (iv) data were reported on mortality, CVD and/or cancer specific mortality in male or female athletes; (v) data were reported as a standardised mortality ratio (SMR), or standardised proportional mortality ratio (SPMR), with 95% confidence limits, or provided sufficient data (observed/expected mortality) to allow either SMR or SPMR to be calculated; and (vi) the studies were of a retrospective, or prospective, methodological design. Any non-peer reviewed grey literature, including conference papers and theses, were excluded. Moreover, any studies that had a follow-up of  $\leq 5$  years were excluded, along with studies which reported the primary outcome of mortality but did not use SMR, or the data was not provided to allow this to be calculated. In the case of any disagreements regarding the inclusion of a study that were not able to be resolved (between AR and MM), KM was consulted which occurred on five occasions.

Studies were searched for, and identified, through scientific databases and by scanning the reference list of identified studies. The search was performed in: Web of Science (1970 – 2019), PubMed (1970 - 2019) and SportDiscus (1970 – 2019). All potentially relevant studies, including reference lists and abstracts, were compiled in Rayyan QCRI software (Ouzzani, Hammady, Fedorowicz, & Elmagarmid, 2016). Two authors (AR and MAM) then screened all identified titles and abstracts to identify studies for full-text review. From an initial search of 38,047 results, 37,878 were excluded. Consequently, 169 were taken forward for full text review of which 43 were finally included within the systematic review; 24 of which were also appropriate for the meta-analysis (Figure 1).

### 8.2.2 Data Extraction

A data table was created extracting the following information: authors and year of publication, number of participants followed, the primary sport of those athletes (if available), how long the athletes were followed for, all-cause mortality SMR, CVD-specific SMR and cancer-specific SMR. When SMR was not directly reported, it was calculated from the reported observed and expected deaths as  $SMR = \text{observed (O) death} / \text{expected (E) death}$  (Garatachea et al., 2014). If the expected number of deaths was not reported from population data, the number in the referent group was used as the expected value and the SPMR defined as:  $(\text{athlete observed death} / \text{number in athlete population}) / (\text{control group death} / \text{number in control group})$ . To calculate 95% confidence intervals (CI) for both methods, the formula:  $SMR \text{ or } SPMR \pm (1.96 * \text{standard error of estimate; SEE})$  defined as:  $\sqrt{(O) / E}$  (Morris & Gardner, 1988) was used. These two metrics are therefore uniform and can be combined to create a pooled SMR. The Newcastle-Ottawa Quality Assessment tool (G. Wells et al., 2019) was used to assess the quality of each study included within the meta-analysis.

Following the overall risk calculations, specific SMR's were calculated, where possible, according to sport. Specifically, in line with other research, END activities were defined as any sport requiring more than 10 minutes of continuous effort (Antero-Jacquemin, Pohar-Perme, Rey, Toussaint, & Latouche, 2018). The END sports in the meta-analysis meeting this criterion were: middle- and long-distance runners, rowers, cross-country skiers, ice skaters and tour de France cyclists. A 'team sport' was defined as any sport in which the performance is predominantly made up of repeated intermittent efforts (Antero-Jacquemin et al., 2018). Team sports identified in this meta-analysis were American footballers, baseball players, footballers, ice hockey players and basketball players. Finally, POW sports were defined as any predominantly anaerobic sport (S. Sarna, Sahi, Koskenvuo, & Kaprio, 1993). The sports in the POW category for this meta-analysis included: boxers, wrestlers, weightlifters, and throwing events in track and field.



**Figure 8.1** – Schematic Flow Diagram of the Systematic Review and Meta-Analysis process

### 8.2.3 Statistics

All meta-analyses statistics were performed using *meta*, *metagen* and *metaforest* packages in R Studio (R Studio v1.2.2019, R Studio, Boston, MA) to calculate the pooled SMR and create the subsequent forest plots. Initially, all SMR and SPMR, values, and their 95% CI's, were logged to determine effect sizes on a natural scale

and then the SEE was calculated for each study. The resulting data was then run through *metagen*, where the pooled SMR was back-transformed to the original SMR scale. The pooled SMR indicates the risk in athletes compared to the general population, with a value of  $< 1$  indicating a lowered risk,  $> 1$  indicating a greater risk and 1 indicating the same risk. Meta-regressions were also run to establish relationships between outcome variables and possible confounding factors using the *metareg* function in R. The pooled SMR was calculated using a random effects model with heterogeneity assessed using the  $I^2$  and  $Q$  statistic. Risk of publication bias assessed using a combination of the Egger's Statistic and funnel plots.

### 8.3 Results

The total number of athletes included within the 24 studies was 165,033, with 139,322 males (84.4%) and 25,711 females (15.6%). There was insufficient data to split the females by sport type, so this was only done for male athletes. Of the male sample, 78,096 (47.3%) were END athletes, 78,689 (47.7%) were team sport athletes, 3,202 (1.9%) were POW sport athletes, and 5,046 (3.1%) of the athletes were Olympians/World Champions where their primary sports could not be established. All included studies were of retrospective methodological design.

The Newcastle-Ottawa scale assesses the methodological quality, and generalisability of an individual study, with higher scores indicating high methodological quality. Of a possible maximum score of 9 on the Newcastle-Ottawa quality score, five, six, nine and four papers scored nine (Baron et al., 2012; Kontro et al., 2018; U. Kujala et al., 2001; Nguyen, Zafonte, Kponee-Shovein, Paganoni, & Weisskopf, 2019; S. Sarna et al., 1993), eight (Antero-Jacquemin et al., 2014; Antero-Jacquemin et al., 2015; Kettunen et al., 2015; Lehman et al., 2012; Mackay et al., 2019; Radonic et al., 2017), seven (Farahmand et al., 2003; Gajda et al., 2018; Gajewski & Poznanska, 2008; Kalist & Peng, 2007; Lincoln et al., 2018; Marijon et al., 2013; Menotti et al., 1990; Schnohr, 1971b; Taioli, 2007) and six (Belli & Vanacore, 2005; Grimsmo, Maehlum, Moelstad, & Arnesen, 2011; van Saase, Noteboom, & Vandenbroucke, 1990; Waterbor, Cole, Delzell, & Andjelkovich, 1988), respectively. Funnel plots were used to assess publication bias (Appendix 11.5.2) with all-cause, CVD, and cancer mortality

demonstrating publication bias, indicated by the wide range of log SMRs reported in the included studies.

Overall, all-cause mortality in male and female athletes was reported in 23 out of 24 studies (164,833 athletes), creating a pooled SMR of 0.67 (95% CI: 0.59 – 0.75;  $p < 0.01$ ), with some evidence of publication bias ( $p < 0.05$ ; Appendix 11.5.2.1) and significant heterogeneity ( $I^2 = 96.9\%$ ;  $Q = 850.7$ ;  $p < 0.01$ ). Sub-group analyses revealed male all-cause mortality was reported in 23 studies (139,122 athletes; 99.7% of all male athletes), creating a pooled SMR of 0.66 (95% CI: 0.58 – 0.74;  $p < 0.01$ ; Figure 2), with no evidence of publication bias ( $p = 0.07$ ) and significant heterogeneity ( $I^2 = 97.0\%$ ;  $Q = 730.3$ ;  $p < 0.01$ ). Female all-cause mortality was reported in four studies (25,711 female athletes; 100%) leading to a pooled SMR of 0.51 (95% CI: 0.40 – 0.65,  $p < 0.01$ ; Figure 3), with no evidence of bias ( $p = 0.41$ ) and no significant heterogeneity ( $I^2 = 45.1\%$ ,  $Q = 5.5$ ,  $p > 0.05$ ). There was insufficient data to calculate a meta-SMR for either CVD or cancer mortality in females, therefore, this was only performed in male athletes.

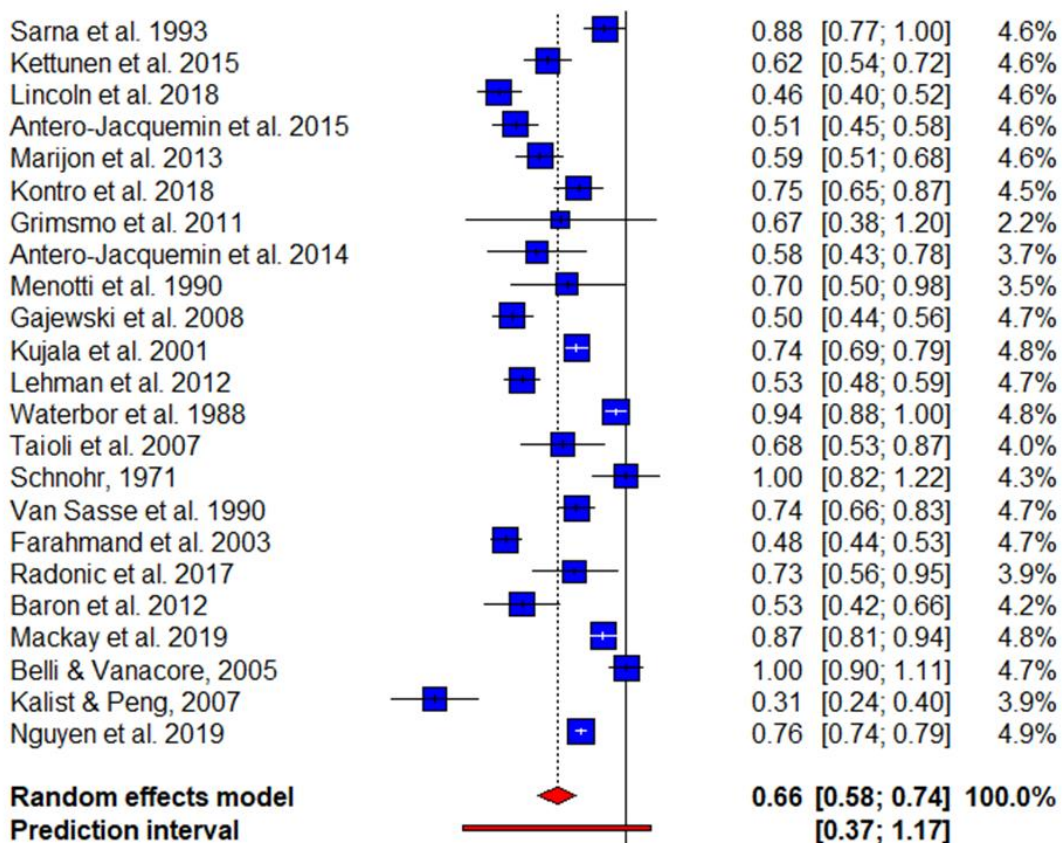
Overall, male CVD mortality was reported in 15 studies (118,288 athletes, 84.8%), demonstrating a pooled SMR of 0.73 (95% CI: 0.62 – 0.85;  $p < 0.01$ , appendix: 11.5.1.1), with no publication bias ( $p = 0.26$ ) and significant heterogeneity ( $I^2 = 81.8\%$ ,  $Q = 82.6$ ,  $p < 0.01$ ; Appendix: 11.5.2.2). Overall cancer mortality was reported in 17 studies (120,782 athletes, 86.7%) with a pooled-SMR of 0.75 (95% CI: 0.63 – 0.89,  $p < 0.05$ , appendix: 11.5.1.2), no evidence of publication bias ( $p = 0.28$ ) and significant heterogeneity ( $I^2 = 88.1\%$ ,  $Q = 143.1$ ,  $p < 0.01$ ; Appendix:11.5.2.3).

Endurance and team sport athlete's all-cause (END:  $I^2 = 98.7\%$ ,  $p < 0.01$ ; Team:  $I^2 = 97.0\%$ ,  $p < 0.01$ ) and CVD mortality (END:  $I^2 = 96.3\%$ ,  $p < 0.01$ ; Team:  $I^2 = 78.0\%$ ,  $p < 0.01$ ) was significantly lower than the general population, however, POW athletes' all-cause ( $I^2 = 77.8\%$ ,  $p > 0.81$ ) and CVD ( $I^2 = 84.9\%$ ,  $p > 0.46$ ) mortality was not significantly different to the general population. For cancer-specific mortality, both team ( $I^2 = 86.2\%$ ,  $p < 0.01$ ) and POW ( $I^2 = 53.3\%$ ,  $p < 0.01$ ) athletes pooled-SMR's were significantly lower than the general population, but endurance athlete cancer mortality was not ( $I^2 = 96.1\%$ ,  $p > 0.11$ ). All of the sub-analyses were heterogeneous ( $p > 0.05$ ), with the exception of cancer mortality for power athletes ( $p < 0.05$ ), with

no evidence of bias except for team sport all-cause mortality (Eggers test  $p < 0.05$ ; Table 2).

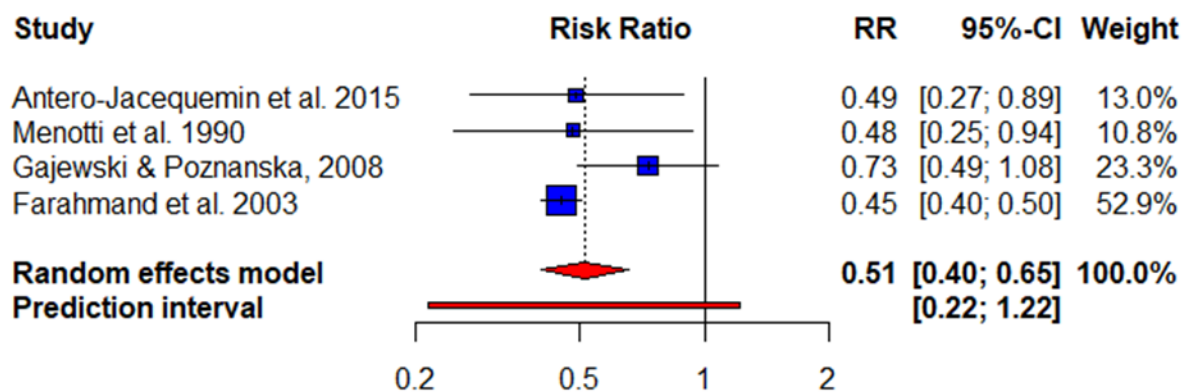
### 8.3.1 Meta-Regression and Sensitivity Analyses

Sensitivity analyses revealed that when i) the four-lowest quality studies were removed, ii) only studies incorporating athletes actively competing after 1945, or iii) only studies published after 2010 were included, the pooled SMR remained similar to the overall SMR (0.64 – 0.68). Indeed, meta-regressions demonstrated no significant interaction with SMR for any of the three data constraints. However, when studies with  $\leq 30$  years follow-up were excluded, the pooled SMR increased to 0.74 (95% CI: 0.65 – 0.84). Moreover, a significant positive association was observed between follow-up length and all-cause ( $\beta = 0.01$ ,  $Z = 2.94$ ,  $p < 0.01$ ) and cancer mortality ( $\beta = 0.01$ ,  $Z = 1.93$ ,  $p > 0.05$ ). However, no significant association was reported between follow-up length and CVD mortality SMR ( $\beta < 0.01$ ,  $Z = 0.90$ ,  $p = 0.36$ ).



**Figure 8.2** – Male all-cause mortality forest plot





**Figure 8.3** – Female all-cause mortality forest plot

**Table 8.1** – Key information about the studies included within the meta-analysis

Author & Year	Number of participants	Average Follow-Up	All-Cause Mortality SMR (95% CI)	CVD Mortality SMR (95% CI)	Cancer Mortality SMR (95% CI)
Sarna et al. (1993)	2,613 Former Finnish Athletes	44.5 years	0.92 (0.84 – 1.01)	0.95 (0.81 – 1.09)	0.96 (0.75 – 1.11)
	1,712 Military Control Participants				
Kettunen et al. (2015)	2,263 Former Finnish Athletes	50 years	0.98 (0.91 – 1.05)	#	0.89 (0.76 – 1.03)
	1,657 Military Control Participants				
Lincoln et al. (2018)	9,778 Former NFL Players	18.5 years	0.46 (0.40 – 0.52) **	0.68 (0.50 – 0.90) **	0.41 (0.26 – 0.62) **
	US Reference Values				
Antero-Jacquemin et al. (2015)	2,403 (601 Female) Former French Olympians	20.3 years - 43.7 years	M – 0.51 (0.45 – 0.59) **	M – 0.55 (0.41 – 0.73) **	M – 0.55 (0.43 – 0.69) **
	French Population Reference values		F – 0.49 (0.26 – 0.85) **	Insufficient data to compute F SMR	Insufficient data to compute F SMR
Marijon et al. (2013)	786 former Tour de France cyclists	32.5 years	0.59 (0.51 – 0.68) **	0.67 (0.50 – 0.88) **	0.56 (0.42 – 0.72) **
	French Population Reference Values				
Kontro et al. (2018)	900 Former Finnish Athletes	77.5 years	1.00 (0.93 – 1.08)	#	1.47 (1.22 – 1.73) **
	900 Brothers of the Finnish Athletes				
Grimsmo et al. (2011)	122 Endurance Skiers	30 years	0.78 (0.50 – 1.05)	Not Reported	Not Reported
	Norwegian Population Reference Values				

Antero-Jacquemin et al. (2014)	203 French Olympic Rowers French Population Reference Values	50 years	0.58 (0.43 – 0.78) **	0.41 (0.16 – 0.84) **	0.59 (0.29 – 1.07)
Menotti et al. (1990)	983 (283 female) former track and field athletes Italian Life Expectancy Tables	18.6 years	Overall – 0.70 (0.59 – 0.82) ** M – 0.73 (0.60 – 0.86) F – 0.48 (0.20 – 0.76)	Not Reported	Not reported
Gajewski & Poznanska, (2008)	2,113 (424 Female) Former Polish Olympic Athletes Polish Population Reference Values	27 years	Overall – 0.51 (0.48 – 0.54) ** M – 0.50 (0.44 – 0.56) ** F – 0.73 (0.48 – 1.05)	Not reported	Not reported
Kujala et al. (2001)	2,009 Former Finnish Athletes Finnish Population Reference Values	47.5 years	0.74 (0.69 – 0.79) **	0.72 (0.64 – 0.82) **	##
Lehman et al. (2012)	3,439 former NFL Players US Population Reference Values	33.5 years	0.53 (0.48 – 0.59) **	0.68 (0.56 – 0.81) **	0.58 (0.46 – 0.72) **
Waterbor et al. (1988)	958 MLB Players US Population Reference Values	59 years	0.94 (0.88 – 1.00)	#	1.05 (0.89 – 1.22)
Taioli, (2007)	5,389 Italian Footballers Italian Population Reference Values	28 years	0.68 (0.52 – 0.86)	0.41 (0.20 – 0.73)	0.31 (0.15 – 0.55)

Schnohr (1971b)	297 former Danish Olympians Danish Population Reference Values	66 years	0.96 (0.79 – 1.12)	0.95 (0.67 – 1.34)	0.94 (0.61 – 1.44)
van Sasse et al. (1990)	2,129 former Dutch endurance skaters Dutch Population Reference Population	32 years	0.76 (0.68 – 0.85)	Not Reported	Not Reported
Farahmand et al. (2003)	73,622 (24,403 female) endurance ski racers Swedish Population Reference Values	5.5 years	Overall – 0.48 (0.46 – 0.51)	Overall – 0.43 (0.35 – 0.51)	Overall – 0.61 (0.52 – 0.71)
			M – 0.49 (0.44 – 0.54) F – 0.45 (0.40 – 0.50)	M – 0.44 (0.36 – 0.54) F – 0.30 (0.11 – 0.50)	M – 0.62 (0.52 – 0.74) F – 0.58 (0.41 – 0.74)
Radonić et al. (2017)	233 Croatian Olympic Medalists Croatian Population Reference Values	35 years	0.73 (0.56 – 0.94)	0.61 (0.38 – 0.93)	0.70 (0.40 – 1.12)
Baron et al. (2012)	3,439 NFL Players US Population Reference Values	34 years	0.53 (0.46 – 0.72)	0.68 (0.56 – 0.81)	0.58 (0.46 – 0.73)
Mackay et al. (2019)	7,676 former professional footballers 23,028 Control Participants	18 years	0.93 (0.91 – 0.95)	#	##
Nguyen et al. (2019)	16,637 former MLB Players US Population Reference Values	36 years	0.76 (0.73 – 0.78)	0.81 (0.77 – 0.85)	0.80 (0.75 – 0.86)
Kalist & Peng (2007)	2,641 former MLB Players	20 years	0.31 (0.23 – 0.39)	Not Reported	Not Reported

	US Population Reference Values				
Belli & Vanacore (2005)	24,000 Italian Footballers Italian Population Reference Values	18 years	1.00 (0.90 – 1.10)	0.83 (0.69 – 1.00)	1.11 (0.97 – 1.28)
Gadja et al. 2008 (2018)	455 deceased polish elite footballers Polish population reference values	-	Not Reported	Under 65 – 1.29 (0.90 – 1.68) Over 65 – 1.17 (0.88 – 1.45)	Under 65 – 0.81 (0.45 – 1.16) Over 65 – 0.94 (0.55 – 1.33)

CI = Confidence Interval; SMR = Standardised Mortality Ratio; NFL = National Football League; MLB = Major League Baseball, M = Male, F = Female. SMR's in bold indicate a significant difference between the athletes and the control population ( $p < 0.05$ ). # Kettunen et al. (2015), Kontro et al. (2018), Mackay et al. (2019) all reported specific SMR values on Ischemic Heart Disease (IHD) respectively, and Waterbor et al. (1988) reported SMR values for Arteriosclerotic Heart Disease therefore they were removed from CVD analyses as overall CVD mortality was assessed. ## Mackay et al. (2019) and Kujala et al. (2001) report SMR's for lung cancer specifically and so they were removed from the overall analysis as overall cancer mortality was assessed.

**Table 8.2** – Sport-specific all-cause, CVD and cancer pooled-SMR's

	Endurance Sports				Team Sports				Power Sports			
	Number of athletes (%)	SMR (95% CI)	Heterogenous (Y/N)	Bias (Y/N)	Number of athletes (%)	SMR (95% CI)	Heterogenous (Y/N)	Bias (Y/N)	Number of athletes (%)	SMR (95% CI)	Heterogenous (Y/N)	Bias (Y/N)
All-Cause Mortality	53,476 (38.4%)	<b>0.65</b> (0.54 – 0.77)	Y	N	78,504 (56.4%)	<b>0.68</b> (0.57 – 0.81)	Y	Y	2,826 (2.0%)	<b>1.04</b> (0.91 – 1.12)	Y	N
CVD Mortality	50,788 (38.4%)	<b>0.63</b> (0.44 – 0.91)	Y	N	65,078 (55.0%)	<b>0.76</b> (0.64 – 0.92)	Y	N	1885 (1.6%)	<b>1.10</b> (0.86 – 1.40)	Y	N
Cancer Mortality	50,511 (38.8%)	<b>0.73</b> (0.50 – 1.07)	Y	N	65,280 (53.3%)	<b>0.73</b> (0.57 – 0.93)	Y	N	1185 (1.5%)	<b>0.51</b> (0.35 – 0.75)	N	N

SMR = Standardised Mortality Ratio, CI = Confidence Interval, Y = Yes, N = No, All SMR's in bold indicate a significant difference compared to the general population ( $p < 0.05$ ). An analysis was deemed heterogeneous based on a combination of the  $I^2$  and Q statistics and if the p value was  $\leq 0.05$  A analysis was deemed not biased if the p value derived from the eggert test was  $\geq 0.05$ .

#### *8.4 Discussion*

This was the first systematic review and meta-analysis to examine sport-specific all-cause mortality in former elite athletes and to consider CVD- and cancer-specific mortality, the two most prevalent diseases worldwide. The key findings from this review are: (i) male and female elite athletes live longer than the general population; (ii) male athletes have a lower incidence of CVD and cancer mortality than the general population; (iii) power sport athletes all-cause and CVD mortality was not significantly different to the general population; (iv) endurance athletes cancer mortality was not significantly different to the general population and (v) increased follow-up length increased the SMR for all-cause and cancer mortality, but not CVD. Furthermore, there is currently insufficient data to allow sport-level comparisons for female athletes.

##### *8.4.1 All-Cause Mortality*

Over recent years, an argument has been made that chronic, intensive exercise may be harmful to health (O'Keefe et al., 2015; O'Keefe et al., 2018; O'Keefe et al., 2012) and lead to a greater chance of premature mortality, or an increased incidence cardiovascular events (Ekelund et al., 2016; Mohlenkamp et al., 2008; Schnohr et al., 2015). However, the current evidence refutes these arguments; male and female athletes had a 31% and 49% lower risk of all-cause mortality than the general population, respectively. This seems to indicate that the female survival advantage (females are expected to live 6 – 8 years longer than males at birth; World Health Organisation, 2019) persists, and is even extended, after a career in elite sport. However, female mortality was only explored in 25,711 athletes, 24,403 (94.9%) of which were identified from a single study (Farahmand et al., 2003), hence there was no significant heterogeneity within the pooled-SMR generated. Therefore, more research in female athletes is needed to confirm the survival benefit in highly active female athletes. Moreover, more research including a follow-up period of  $\geq 30$  years are needed given the positive association between reduced survival estimates and follow-up time.

Given that the standardised mortality ratio was the most common method of reporting the risk of mortality in elite athletes, this method was chosen for the meta-analysis. However, life expectancy and age at death in male athletes has also been explored.

Specifically, Clarke et al. (2012) reported an average 2.8 year survival advantage in a cohort of 15,174 Olympic athletes from nine countries, with a cohort study of 2,814 French Olympians gaining an average of 6.5 years (Antero-Jacquemin et al., 2018). These results are therefore largely in accord with those of the current meta-analysis, as the lowered SMR risk indicates a longer survival in former elite athletes compared to the general population.

Despite the apparent survival benefit of elite athletes, one common and important criticism of the literature is the applicability of comparing former elite athletes to the general population. Elite athletes may be characterised by healthier lifestyles post-retirement than the general population and engage in more leisure-time physical activity (LTPA), both of which predict all-cause mortality (Backmand, Kujala, Sarna, & Kaprio, 2010; Fogelholm, Kaprio, & Sarna, 1994; Gajewski & Poznanska, 2008; Kontro et al., 2018). It is therefore not currently possible to distinguish the influence of intensive training *per se* from overall lifestyle factors. Indeed, it may be worth noting that when Sarna et al. (1993) and Kettunen et al. (2015) used a control group formed of military fit personnel, the SPMR was not significantly different relative to elite athletes (0.92 and 0.98, respectively). Additionally, some studies have only reported survival benefits up to a specific age, rather than across the whole lifespan (Gajda et al., 2018; Mackay et al., 2019; Schnohr, 1971b). Specifically, Schnohr (1971b) found that athletes up to 50 years had a SMR of 0.61, with athletes aged over 50 and 65 years having SMR's of 1.08 and 1.02, respectively. Similarly, former Scottish footballers only had a survival benefit up to the age of 60 years (Mackay et al., 2019), with Polish footballers having a benefit until 75 years (Gajda et al., 2018), after which the mortality was the same or greater than the general population. Conversely, Antero-Jacquemin (2018) reported an increased longevity in French Olympians after 50 years of age, thus, it is unknown why this apparent loss of survival advantage occurs, in some, but not all, athletes. Further work is needed to elucidate the potential mechanisms.

#### 8.4.2 Sport-specific Mortality

Male END athletes had the most favourable all-cause mortality rate and lived significantly longer than the general population (SMR: 0.65). Indeed, Clarke et al. (2012) reported a 13% greater survival benefit for medallists in endurance sports, with



similar benefits reported in marathon runners (+ 4.3 years; Lee-Heidenreich, Lee-Heidenreich, & Myers, 2017), tour de France cyclists (+ 8 years; Sanchis-Gomar, Olaso-Gonzalez, Corella, Gomez-Cabrera, & Vina, 2011) and Olympians involved in endurance sports (+ 6.3 years; Antero-Jacquemin et al., 2018). Endurance athletes have consistently been shown to have favourable mortality compared to the general population, attributed to an increased cardiorespiratory fitness (CRF) and subsequent maintenance of CRF throughout the lifespan. Specifically, every 1 MET increase in maximal capacity reduces the likelihood of all-cause mortality by 15% (Al-Mallah et al., 2018). Furthermore, the difference is unlikely to be explained by genetic factors as it has recently been shown that elite athletes who undertake strenuous aerobic exercise exhibit similar disease-trait-related genotypes to the general population (Ruiz, Moran, Arenas, & Lucia, 2011). Thus, endurance athletes are still predisposed to similar levels of disease to the general population.

Male team sport athletes, the biggest sub-group within the meta-analysis including 78,504 (56.4%) of all male athletes, also demonstrated a favourable all-cause mortality (SMR: 0.68). However, it must be noted that significant bias was evident (Eggers statistic  $p = 0.01$ ) and so these results should be interpreted with caution. This may be explained, at least in part, by two studies in the team sport meta-analysis including athletes competing before 1915 (Schnohr, 1971b; Waterbor et al., 1988). Specifically, sporting practices, training demands, athlete welfare and advances in health care make it difficult to directly compare across such a large time-span and gain reliable results. Nevertheless, a large body of research in North American sports report a survival benefit in former baseballers (+ 4 – 5 years; Abel & Kruger, 2005, 2006a; Saint Onge, Rogers, & Krueger, 2008), American football players (+ 6.1 years; Abel & Kruger, 2006b) and basketballers (+ 4.3 – 5.5 years; Lawler, Lawler, Gibson, & Murray, 2012), but the same was not observed in footballers (- 1.9 years; Kuss, Kluttig, & Greiser, 2011). It should be acknowledged, however, that three of these studies, conducted by Abel and Kruger (2005, 2006a, 2006b), also involved athletes who made their professional debuts before 1940, so the applicability of their findings to a modern population is questionable. Furthermore, Kuss et al. (2011) failed to account for world war deaths, confounding conclusions and potentially explaining the reduced survival incidence reported. Nevertheless, despite these methodological limitations, they

advance our understanding, although the generalisability of their results remains questionable and conclusions must be drawn with caution.

Power sport athlete's all-cause mortality was not significantly different from that of the general population (pooled-SMR: 1.04), however, this analysis was only conducted in two studies with participants totalling 2,826, or 2.0%, of the overall population. Similar patterns are evident in other studies, with male discus throwers (-0.6 years) and 100m runners (-0.9 years) experiencing marginal premature mortality (Lee-Heidenreich et al., 2017). In contrast, Clarke et al. (2012) reported a modest survival benefit in power athletes, albeit of only 5%. Former Olympic male wrestlers have also been reported to live  $13.0 \pm 18.4$  years longer, although this must be interpreted with caution given that the standard deviation spans 0, indicating some have a premature mortality, and the relatively small sample size included within this study ( $n = 341$ ; Keller, 2019). However, contradicting the negative associations of all-cause mortality and power sports, Antero-Jacquemin et al. (2018) reported power athletes gained an average of 7.2 years, suggesting a significantly longer life-span. Given the disparity of results across the literature, and the small statistical power within this meta-analysis, more research is needed to fully elucidate the long-term effects of competing in power sports.

#### *8.4.3 Cardiovascular Disease Mortality*

Overall, the pooled-SMR risk of CVD mortality (0.73 95% CI: 0.62 – 0.85) was significantly lower than the general population. This is not surprising given the long-established relationship between CRF and CVD mortality (Kaminsky et al., 2019). Specifically, men in the highest quintile of fitness, compared to those in the lowest, had a relative risk of 0.22 (0.12 – 0.39) for CVD mortality (Blair et al., 1989), and as little as  $1 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  increase in CRF decreased the risk of CVD mortality by 9% (Laukkanen et al., 2016). Cardiorespiratory fitness is critical for most END and team sports athlete's performance and, consequently, these athletes occupy the top percentile for CRF values and present the lowest risk (SMR: 0.63 and 0.76, respectively). Thus, the superior CRF and consequent lower CVD mortality risk in team and END athletes, is one of the main reasons suggested for the observed increased longevity in END and team athletes (Antero-Jacquemin et al., 2018; Lemez & Baker, 2015), and the lack of protective effect in power athletes (SMR: 1.10).

Four studies (Kettunen et al., 2015; Kontro et al., 2018; Mackay et al., 2019; Waterbor et al., 1988) were not included within the meta-analysis for CVD mortality as they reported a SMR value for the specific CVD of ischemic heart disease (IHD; Kettunen et al., 2015; Kontro et al., 2018; Mackay et al., 2019) or arteriosclerotic heart disease (AHD; Waterbor et al., 1988). Including specific CVD SMR's, as opposed to overall CVD risk, could have induced bias and so the decision was made to remove them. However, the SMR for IHD was not significantly different in former Finnish athletes (SMR: 0.95 95% CI: 0.81 – 1.14; Kettunen et al., 2015; Kontro et al., 2018) or former Scottish footballers (SMR: 0.91, 95% CI: 0.87 – 0.96; Mackay et al., 2019), in relation to the general population. Similarly, AHD mortality risk was not significantly different in 958 former baseball players (SMR: 1.10, 95% CI: 0.99 – 1.22; Waterbor et al., 1988). Furthermore, a recent study in French Olympians reported END athletes are at an increased risk of mortality due to CVD, cumulating in a loss of 1.6 years (Antero-Jacquemin et al., 2018). Taken together, these results indicate that END and team athletes may be protected against some, but not all, CVDs. This may, at least in part, explain the overall protective effect of exercise but the minimal impact on IHD and AHD. However, more research is needed to confirm this hypothesis and to establish whether intensive training lowers the specific risk profile and aetiologies of individual CV diseases.

Power athletes pooled-SMR was not significantly different to that of the general population for CVD (SMR: 1.10), however caution is warranted when interpreting this finding as only 1,885 male athletes (1.2%) from two studies (U. Kujala et al., 2001; S. Sarna et al., 1993) were included. Nevertheless, American football linemen, who share a lot of characteristics with power sport athletes, had a two to three fold increase in CVD mortality compared to counterparts in other positions (Baron et al., 2012; Lincoln et al., 2018). One possible explanation is the increased likelihood of hypertension in power athletes (U. Kujala, Kaprio, Taimela, & Sarna, 1994a; U. Kujala et al., 2001; Laine et al., 2015), a long-established independent CVD risk factor. Additionally, power sport athletes characteristically have a higher BMI and a relationship between playing/competing time BMI and CVD mortality has been observed (Baron et al., 2012). Specifically, American football players who had a playing time BMI of  $\geq 30 \text{ kg}\cdot\text{m}^2$  had twice the risk of CVD mortality (SMRs: 2.02 –

2.07), compared to those with a BMI  $\leq 29.9$  kg·m<sup>2</sup> (Baron et al., 2012). This risk could be further exacerbated as over a 30-year period power athletes reportedly gained an average of 12.8 kg (S Sarna et al., 1997). So, a question remains as to whether playing-time BMI is the primary risk factor of CVD or subsequent weight-gain post-retirement is a greater indicator of CVD mortality in power athletes.

#### *8.4.4 Cancer Mortality*

Cancer mortality (SMR: 0.75) was significantly lower in athletes than the general population. Likewise, elite French athletes had a significantly lower incidence of cancer mortality, gaining an average of 2.3 (1.9 – 2.6) years (Antero-Jacquemin et al., 2018). One possible explanation is that former athletes smoke less, drink less, and engage in more LTPA than the general population (Backmand et al., 2010; Fogelholm et al., 1994; Kontro et al., 2018; Sormunen et al., 2014), all of which significantly contribute to cancer risk and mortality. Indeed, Sourmunen et al. (2014) reported that when LTPA, smoking status, years of smoking and alcohol consumption were accounted for, there was a minimal protective effect on cancer incidence (standardised incidence ratio: 0.89, 95% CI: 0.81 – 0.97). Moreover, Pukkala et al. (2000) reported that elite athletes had a slightly elevated incidence of non-smoking related cancers (SIR: 1.10), with other studies reporting lung cancer mortality was significantly reduced in athletes (U. Kujala et al., 2001; Mackay et al., 2019). This confirms the importance of accounting for lifestyle-related habits when assessing cancer incidence/mortality in this population. This may explain, at least in part, why some populations of footballers (Belli & Vanacore, 2005; Fernandes et al., 2019; Gajda et al., 2018; Mackay et al., 2019), Olympians (Radonic et al., 2017; Schnohr, 1971b) and baseballers (Waterbor et al., 1988) all have similar rates of cancer incidence and mortality to the general population, whilst others demonstrate a reduced risk (Baron et al., 2012; Marijon et al., 2013; Nguyen et al., 2019; Taioli, 2007).

Endurance athletes' risk of cancer mortality was not significantly different to the general population (SMR: 0.73, 95% CI: 0.50 – 1.07). Despite this, END athletes have consistently been found to have favourable longevity compared to the general population and, indeed, other athletes (Antero-Jacquemin et al., 2014; Antero-Jacquemin et al., 2018; Farahmand et al., 2003; Lincoln et al., 2018; Marijon et al., 2013; van Saase et al., 1990). Thus, it is worth considering whether the non-protective

effect on cancer mortality derives from END training, or simply that athletes are living longer and therefore have a greater chance of developing cancer. Whilst distinguishing these factors may be challenging, it deserves consideration given that it could alter the interpretation of the results presented and future study directions. Regardless of their increased longevity, however, END athletes are still at a decreased risk of CVD mortality, suggesting the benefit of training is maintained throughout the life-span.

#### *8.4.5 Limitations and Conclusions*

Whilst there are numerous strengths, there are limitations to this review that require consideration. Specifically, not all the athletes within these studies were elite and of national standard, although they were all considered to be highly trained. Moreover, inferences are not able to be made about the specific training that athletes should undertake as such data was rarely reported. As such, conclusions regarding the long-term effects of participating in specific types of training regimes, such as HIIT, resistance or strength training, are precluded. Furthermore, the small number of studies included within the POW athlete sub-group, and female athletes, potentially limits the generalisability of these results. It is also noteworthy that some sports may have been mis-classified in previous research (for example, Sarna et al. (1993) classified boxing as a power sport), which could have influenced the meta-analysis results. Finally, no inferences can be made as to the relative contribution of lifestyle on overall mortality. Thus, it is hard to distinguish whether any survival benefit observed is because of training, lifestyle choices, or most likely, a combination of both.

The main conclusions from this review are: (i) overall, male and female athletes' all-cause mortality is significantly lower than the general population; (ii) sub-group analyses revealed END and team sport athletes, but not POW athletes, had a reduced all-cause mortality; (iii) POW athletes were at a similar risk of CVD mortality compared to the general population, and; (iv) END athletes cancer mortality was not significantly different to the general population. However, more research is warranted in female and power athletes, with a follow-up of  $\geq 30$  years, to ascertain the long-term benefits/consequences of chronic intensive exercise training in these populations.

# **Chapter 9**

# **Synthesis**

## **Chapter 9 – Synthesis**

The importance of exercise training for health, as well as aerobic and anaerobic athletic performance, is well-recognised. However, fundamental questions remain regarding the influence of sex and maturity on the responses to training during growth and maturation. Importantly, many training studies have failed to appropriately account for habitual physical activity (PA) levels, confounding interpretation of the effectiveness of training interventions, and any potential sex- and maturity-specific differences. Finally, there is also a dearth of research investigating the long-term effects of sport-specific training, which often starts during childhood. Consequently, the aim of this thesis was to explore the effect of sex, maturity and PA on training-related adaptations during childhood and adolescence and to review the potential long-term health implications of such training during youth. The current chapter will synthesise this research, considering the findings of each experimental chapter relative to the other studies and considering their strengths, weaknesses and implications as a whole.

### *9.1 Sex and maturity effects on trainability in youth*

The trained children and adolescents in the current thesis demonstrated a significantly higher absolute and allometrically scaled peak  $\dot{V}O_2$  than their untrained counterparts (Chapters 4 and 5), concordant with the majority of the paediatric literature (Baquet et al., 2003; Danis et al., 2003; Matos & Winsley, 2007; McNarry & Jones, 2014; McNarry et al., 2014b; McNarry et al., 2011c; Nottin et al., 2002; Obert et al., 2003). Furthermore, trained children and adolescents had a significantly faster sprint time compared to their untrained counterparts in **Chapter 6**, suggesting that training has the ability to increase both aerobic and anaerobic performance concomitantly. Whilst it is important to highlight the overlap of some participants between chapters, and the discordance of exercise modalities in **Chapters 4 – 6**, this simultaneous effect of training on aerobic and anaerobic performance outcomes may reflect the type of training the participants were engaged in. Specifically, the majority of participants (230/237) were engaged in a team sport predominantly involving high-intensity intermittent exercise (Cunha et al., 2016; Runacres et al., 2019b; Williams, 2016).

The potential potency of high-intensity exercise interventions to concomitantly improve sprint times and peak  $\dot{V}O_2$  was reported in a recent meta-analysis (Cao et al., 2019), highlighting that the intensity of exercise may be a key determinant of training adaptation. It is therefore interesting to note that team-sport athletes in **Chapter 8** were predicted to demonstrate the greatest reduction in all-cause, cardiovascular disease (CVD) and cancer mortality compared to the general population. Albeit tentatively, this may suggest that intensity, and not volume, of exercise may also be crucial for long-term health, concordant with previous research (Gormley et al., 2008; Lemez & Baker, 2015; Lemez, Wattie, & Baker, 2016; Love et al., 2019a; Schwartz, de Heer, & Bea, 2017; Teramoto & Bungum, 2010a). However, the long-term consequences of sport-specific training in youth is not well understood (Guo, Lou, Zhang, & Song, 2015; Guo, Zhang, Wang, Guo, & Xie, 2013; Väistö et al., 2019; Yang, Telama, Hirvensalo, Viikari, & Riatakari, 2009), and therefore whether the same long-term effects are present throughout the lifespan remains to be established.

The long-term effects of intensive exercise training in youth on adult health remains poorly understood with the literature (Attard et al., 2013; Hasselstrøm et al., 2002; Mika & Fleshner, 2016; Riner & Sellhorst, 2013) but **Chapter 8** highlights key methodological considerations which must be measured, and reported, in any future studies seeking to address this question. More specifically, the consideration of lifestyle factors alongside training history such as socioeconomic status (SES), body mass index (BMI) and alcohol, tobacco, and substance use need to be robustly accounted for to establish the true effect of intensive exercise training in youth and adult health. Whilst the assessment of mortality in former elite athletes is the most appropriate choice in that population (Lemez & Baker, 2015; Teramoto & Bungum, 2010a), it perhaps is not suitable for establishing the effect of childhood training regimes on adult health. Indeed, the likely small number of deaths in young adults would either lead to studies being underpowered to make any firm conclusions, or the recruitment of extremely large populations. Whilst, all-cause, CVD, and cancer mortality could be assessed over the life course (>50 years follow-up) the need to answer this pressing question may require a different approach. Indeed, current health status and CVD risk factors in adults, with careful consideration of lifestyle factors, potentially present a shorter-term exploration of the long-term health effects of



intensive training during childhood. Sub-group analyses could then be performed to explore those who continue to be physically active compared to those who stopped to examine if any protective effect is still evident years after training ceased. Whilst this area is still in its infancy, the potential impact of these results could have wide-reaching impacts for world-wide sporting organisations, public health initiatives, and policy makers to protect the welfare of young athletes.

The present thesis reported similar magnitudes of difference between trained and untrained participants in anaerobic (13.9 – 22.4%; **Chapter 6**) and aerobic (7.5 – 18.7%; **Chapter 4**) performances. However, the development of anaerobic and aerobic parameters were not consistent across the maturation spectrum, with greater training-related differences in post-pubertal adolescents (16.5 - 19.8%) compared to pubertal adolescents (9.2 – 13.5%) and pre-pubertal children (7.5 – 12.9%), irrespective of sex, thereby refuting the maturational threshold hypothesis (Katch, 1983; Rowland, 1997). Indeed, the original maturational threshold hypothesis proposed by Katch (1983) suggested that there may be a window of opportunity surrounding peak height velocity (PHV) during which circulating hormones may engender greater training-related gains than observed pre-puberty. The results of this thesis, in combination with other studies (Buenen et al., 1998; Buenen & Thomis, 2000; Danis et al., 2003; Nagahara et al., 2019; Rumpf et al., 2013; Rumpf et al., 2015b; Weber et al., 1976), may suggest that *if* a maturational threshold is evident in either aerobic or anaerobic parameters it is in the transition from pubertal to post-pubertal adolescence. Given that little research has been conducted in those aged 16 – 18 years, this contention requires further investigation but could have significant implications in the design of future long-term athlete development models.

It is interesting to note the markedly smaller differences between maturity stages in the anaerobic than aerobic responses of the pubertal participants. Specifically, the magnitude of change between pre- and circa-pubertal adolescents for absolute and allometrically scaled peak  $\dot{V}O_2$  was  $19.1 \pm 14.6\%$  and  $13.2 \pm 13.0\%$ , respectively, irrespective of training status (**Chapter 4**). In comparison, 30 m sprint time (30mT) and allometrically scaled peak power ( $P_{\text{peak}}$ ) only increased by  $2.9 \pm 1.3\%$  and  $2.4 \pm 3.0\%$ , respectively, over the same period, indicating that pubertal adolescents may experience declines and/or plateaus in anaerobic parameters during puberty (**Chapter**

6). In accord with previous research, similar magnitudes of change were also observed between pubertal stages in peak force ( $F_{\text{peak}}$ ), which has been widely attributed to ‘adolescent awkwardness’ (Buenen et al., 1998; Buenen & Thomis, 2000; Lloyd et al., 2015; Rumpf et al., 2013; Rumpf et al., 2015b). Potential mechanisms offered for adolescent awkwardness include differing growth rates of the limbs in relation to the trunk (Buenen et al., 1998; Buenen & Thomis, 2000; Rumpf et al., 2015b), impaired sensorimotor and proprioceptive abilities (Quatman-Yates et al., 2012) and declines in habitual PA levels during the mid-teenage years (Bitar et al., 2000; Love et al., 2019a; A. Rowlands & Eston, 2007). Alternatively, it is also possible that rapid increases in muscle accrual (Beneke et al., 2007; Buenen & Thomis, 2000; J. Wells, 2007), a maturation of the glycolytic energy system (Armstrong & Barker, 2012a; Barker, Welsman, Fulford, Welford, & Armstrong, 2010; Doré et al., 2005; Van Praagh, 2000), and possible changes in muscle fibre type distribution (Eriksson, 1972, 1980) occur during puberty, masking the influence of training (Danis et al., 2003; Tattersall & Pyke, 1973; Weber et al., 1976). The development of aerobic and anaerobic performances should be explored simultaneously in future studies by considering the anaerobic: aerobic power ratio as suggested by Ratel et al. (2017). Indeed, similarities and/or divergences in this relationship may indicate an accelerated adaptation, but this was not feasible to calculate in the present thesis due to the discordance between exercise modalities used to determine the aerobic (cycle ergometry, **Chapters 4 and 5**) and anaerobic (sprint running, **Chapters 6 and 7**) performances.

In accord with the notion that training-related differences may be blunted during the pubertal maturity phase, case-controlled, homozygous twin studies have reported no effect of endurance training on peak  $\dot{V}O_2$  around the time of PHV (Danis et al., 2003; Weber et al., 1976). During PHV, both boys and girls experience an influx of hormones, with a testosterone and oestrogen dominance for boys and girls, respectively (Rogol, 2002). Although pubertal adolescents may experience increased levels of androgenic hormones, it is estimated that 98.0 – 99.8% of all testosterone is bound to protein in the blood, thereby facilitating its uptake by muscles and organs, where it is used for various functions related to growth and maturation (Hiort, 2002; Rogol, 2002; Vingren et al., 2010). Consequently, this only leaves 0.2 – 2.0% of

circulating testosterone ‘free’ to initiate an anabolic response to training stimuli (Reed & Meggs, 2017; Vingren et al., 2010), which could explain the blunted training response observed in some (Bitar et al., 2000; Danis et al., 2003; Weber et al., 1976), but not all (Behringer et al., 2010; Kobayashi et al., 1978; Massicotte & Macnab, 1974; Roemmich, Richmond, & Rogol, 2001) studies in pubertal adolescents. Indeed, the lack of ‘free’ testosterone may explain why the training-related difference in peak and allometrically scaled peak  $\dot{V}O_2$  remained relatively constant (within 2.0%) between the pre- and circa-pubertal maturity phase.

Taken together, the results of **Chapters 4** and **6** offer evidence in support of a maturational threshold for specific parameters, but also refute the existence of an enhanced window of trainability during puberty. On the contrary, this thesis presents evidence that anaerobic performance is dampened during puberty with negligible increases in performance (**Chapter 6**). Specifically, a smaller magnitude of training related differences in  $\dot{V}O_{2max}$  were present between the trained and untrained circa-PHV adolescents compared the pre-PHV group (**Chapter 4**). These results therefore question the appropriateness of existing LTAD models that seek to target different parameters of fitness dependent upon maturational stage (Lloyd & Oliver, 2012). More specifically, coaches, practitioners, and sporting organisations need to be cognisant that not all parameters of fitness develop at the same rate during puberty and this should be built into existing models to account for individual variability. Whilst the optimal training methodology remains to be elucidated in order to establish the most favourable performance adaptations in youth (Armstrong & McNarry, 2016), this thesis tentatively suggests that developmentally appropriate training should start in children pre-PHV, with no evidence to suggest they are less trainable than circa-PHV adolescents. Indeed, by delaying certain training types (i.e. resistance training) during early childhood significant performance advances may not be possible. Future research is therefore warranted to re-configure LTAD models based on new and emerging evidence refuting the maturational threshold hypothesis.

The effect of sex on aerobic or anaerobic parameters has received little attention, with the clear majority of the literature that has examined sex differences focussing on pre-pubertal children (McNarry et al., 2015; Winsley et al., 2009) at which point the

physiological sex differences are unlikely to be fully manifest (Rogol, 1994; Rogol et al., 2002). Nevertheless, sex differences have been observed even in pre-pubertal children, with boys reported to have a 6.9 – 21.1% greater absolute (Armstrong et al., 1991b; McNarry et al., 2015; Vinet et al., 2003), and allometrically scaled peak  $\dot{V}O_2$  (McNarry et al., 2015; Obert et al., 2003; Winsley et al., 2009), compared to age- and maturity-matched girls, irrespective of exercise modality. The sex difference in peak  $\dot{V}O_2$  is suggested to be similar across the maturational spectrum (Armstrong & Welsman, 2019b, 2020a; Armstrong & Welsman, 2020c; Armstrong et al., 1991b), a conclusion supported by **Chapter 4** in which reported boys had a greater absolute (23.8%) and allometrically scaled (24.9%) peak  $\dot{V}O_2$ , irrespective of maturity. Furthermore, in agreement with **Chapter 4**, an enhanced peripheral oxygen extraction is proposed to be the main driver behind the sexual dimorphism in peak  $\dot{V}O_2$  (Armstrong & Welsman, 2020a; McNarry et al., 2015; Winsley et al., 2009), with no sex differences in bulk oxygen delivery parameters (cardiac output and stroke volume) once appropriately scaled (Armstrong & Welsman, 2019a; Armstrong & Welsman, 2020a; Nottin et al., 2002; Obert et al., 2003; Vinet et al., 2003; Winsley et al., 2009). The mechanisms underpinning sex-differences in oxygen extraction remain to be fully established but a lesser ability to match oxygen perfusion to demand (Smith et al., 2017) and differences in muscle fibre recruitment patterns during incremental exercise (McNarry et al., 2015), have both been suggested.

One confounding factor in the majority of studies investigating the effect of sex is the mismatching of boys and girls with regard to training load, or a lack of detail regarding prior training history which precludes training load from being established (Armstrong & Welsman, 2019a; Armstrong & Welsman, 2019b; Baxter-Jones et al., 1993; Bitar et al., 2000; Winsley et al., 2009). Therefore, it is worth considering whether the sex differences are truly present or whether the apparent sex differences reflect an inter-participant difference in training load, which could confound interpretations of the effect of sex. For example, Baxter-Jones et al. (1993) reported that boys had a significantly higher peak  $\dot{V}O_2$  at every time-point of a three-year longitudinal study, but the participants were not matched for training volumes. Indeed, interrogation of the data reveals that pre-pubertal boys' training volume was almost twice that of pre-pubertal girls ( $10.6 \pm 4.8$  hours $\cdot$ week $^{-1}$  vs  $5.6 \pm 3.2$  hours $\cdot$ week $^{-1}$ ), with similar training

discrepancies in pubertal and post-pubertal adolescents. Therefore, whether the main effect of sex reported was attributable to physiological sex-differences, or simply a reflection of the increased training load, is not possible to discern. This is of particular concern given the most recent LTAD model is sex-specific and specifies that different parameters of fitness are targeted at certain points throughout maturation (Lloyd & Oliver, 2012). However, these recommendations are based on literature with fundamental methodological flaws and therefore more high quality, robust, research is required in girls to inform LTAD practices and models.

In the present thesis, sub-analyses revealed there was no significant difference in the training volume and/or training history of participants in any experimental chapter, thus discrepancies in training loads are unlikely to explain the greater peak  $\dot{V}O_2$  observed in boys compared to maturity- and training-matched girls (**Chapters 4 and 5**). Interestingly, an interaction between sex and training on peak  $\dot{V}O_2$  was apparent in **Chapter 4**, suggesting that girls may be more responsive to training than maturity-matched boys. More specifically, the difference in absolute peak  $\dot{V}O_2$  between trained and untrained boys was only 0.8% compared to 9.3% in girls, irrespective of maturity and training status. One potential reason for the apparent increased aerobic trainability in girls, regardless of maturity, may be the lower PA levels in untrained girls (**Chapter 5**). Indeed, girls have been consistently shown to have lower habitual PA levels when compared to age- and maturity-matched boys (Bitar et al., 2000; Ekelund et al., 2001; Love et al., 2019a), potentially exaggerating the effect of training on peak  $\dot{V}O_2$ . Similarly, a greater difference in  $P_{\text{peak}}$  was observed between trained and untrained girls (28.5%) compared to boys (18.7%; **Chapter 6**), which may be also be related to differences in vigorous physical activity (VPA). Indeed, VPA is often accrued in short (> 6 s) sporadic bouts (Holman et al., 2011; Wolfe, Lee, & Laurson, 2020) and thus is predominantly anaerobic in nature (Holman et al., 2011; Love et al., 2019a; Väistö et al., 2019). Therefore, it is worth considering whether the lower PA levels in untrained girls (**Chapter 5**) may subsequently result in declines in peak  $\dot{V}O_2$  and anaerobic performances, amplifying the observed training differences between trained and untrained girls. This complex relationship urgently requires further research as it raises questions as to whether current training practices may be less effective in girls,

a conclusion with considerable potential implications for future training programme design and performance.

### *9.2 Experimental Study Strengths*

There are numerous strengths to this thesis. A significant strength was the inclusion of a supramaximal validation bout in **Chapters 4 and 5** for the determination of peak  $\dot{V}O_2$  in all participants (Barker et al., 2009; Poole & Jones, 2017; Schaun, 2017), with secondary criteria having questionable validity (Barker et al., 2009). Indeed, if the secondary criterion of a heart rate  $> 85\%$  age-predicted maximum was used to verify a maximal effort in the current thesis, 20 participants would have been excluded from **Chapters 4 and 5**, with a further 29 and 82 participants excluded on the basis of not achieving an RER  $> 1.1$  or HR  $> 195$  beats $\cdot$ min $^{-1}$ , respectively. This confirms the work of Barker et al. (2009) on the fallacy of utilising secondary criteria in children, thus studies that have relied on these secondary criteria, or failed to incorporate a supramaximal validation bout, must be interpreted with caution. The absence of supramaximal validation bouts in the majority of previous research in children and adolescents may explain, at least in part, why significant questions still persist (Armstrong & McNarry, 2016). Furthermore, the simultaneous determination of haemodynamic and muscle deoxygenation parameters (**Chapter 4**) allowed for sex-, training- and maturity-related differences in the mechanisms underpinning the exercise response to be explored.

**Chapters 6 and 7** successfully implemented a novel combination of field-based methodologies to quantify single (**Chapter 6**) and repeated (**Chapter 7**) sprint kinetics. Indeed, sprint running is highly comparable between populations (Morin et al., 2011; Rossi et al., 2017), and involves simple data collection methods (Simperingham et al., 2017; Simperingham et al., 2016), facilitating large-cohort cross-sectional and longitudinal studies in paediatric populations. The quantification of the underlying kinetics builds upon existing knowledge, enhancing our understanding of sprint-speed development throughout growth and maturation. Moreover, the large sample sizes in **Chapters 4, 5, 6 and 8** compared to similar studies (Cunha et al., 2016; Dencker et al., 2006; Garatachea et al., 2014; McNarry et al.,

2015; Mujika et al., 2009; Philippaerts et al., 2006), and the inclusion of control groups further strengthen the conclusions drawn in this thesis.

### *9.3 Experimental Study Weaknesses*

Although there are many strengths to the current thesis, it is important to acknowledge certain limitations. First, whilst all parameters were allometrically scaled, which is currently considered the most robust statistical method to account for differences in body size (Welsman & Armstrong, 2019), they were scaled by body mass or body surface area which fail to account for differences in body composition. Given the sex- and maturity-dependent changes in body composition during childhood, utilising these parameters may have led to erroneous conclusions and potentially masked important training-related effects. Future research should endeavour to scale peak  $\dot{V}O_2$  and anaerobic power variables by lean body mass given its strong association with performance (Armstrong & Welsman, 2019b; Armstrong & Welsman, 2019c, 2020c; Nevill et al., 2006; Rumpf et al., 2015b) and to gain a greater insight into sex- and maturity-related training adaptations.

Whilst all trained participants in **Chapters 4 to 7** were part of LTAD programs and competed at a national/international level, they were predominantly recruited from Hockey and Football (230/237) which largely involves high intensity, intermittent type exercise (Cunha et al., 2016; Runacres et al., 2019b; Williams, 2016). Therefore, the influence of sex and maturation and their interaction with other training methodologies remains to be elucidated. Indeed, different training types have different characteristics, target different components of fitness, and have different long-term health implications (**Chapter 8**). Therefore, the results of this thesis can only be applied to team sports. Moreover, compared to other similar training studies, the trained children and adolescents included within the current thesis were engaging in lower training volumes (Baxter-Jones et al., 1993; Cunha et al., 2016; McNarry et al., 2014b; Metaxas et al., 2014). This may therefore explain the lack of training effect on the stroke volume response to exercise in **Chapter 4** and consequently the results of this thesis should be interpreted to reflect the effect of moderately trained children and adolescents.

All experimental studies within this thesis were cross-sectional in nature (**Chapters 4, 5, 6 and 7**), only offering a glimpse into the influences of sex, maturity and PA levels on training responses throughout childhood and adolescence. Rigorously designed longitudinal studies including children and adolescents across the maturational spectrum are required to fully establish the sex- and maturity-specific development of aerobic and anaerobic performance to enhance LTAD models and athlete's health and performance. Another common limitation with early training studies is the discordance between training and testing modalities, with suggestions that discrepancies in exercise modalities may create spurious associations and mask physiologically meaningful changes in variables (Armstrong & McNarry, 2016; Armstrong & Welsman, 2020c; Rowland, 1997). It is therefore worth noting that none of the participants were trained cyclists in **Chapters 4 and 5**. Whilst a more representative indication of the absolute training-related effects may have been gained using a treadmill, cycle ergometry was selected to reduce movement artefact, enabling the accurate quantification of haemodynamic and muscle deoxygenation variables during exercise, thereby allowing insights into the mechanisms underlying the sex-specific development of peak  $\dot{V}O_2$  (Barstow, 2019; J Welsman et al., 2005).

Verifying a maximal effort in anaerobic performances is challenging, with no universal objective criteria (Van Praagh, 2000; Van Praagh & Doré, 2002). Nevertheless, motivational techniques were used in both **Chapters 6 and 7** to try and mitigate the risk of the acceptance of sub-maximal performances. Specifically, the finish lines were set longer than the distance over which the velocity-time curve was modelled to minimise deceleration, and verbal encouragement was used throughout each sprint, in accord with previous research (Meyers et al., 2015; Meyers et al., 2017a; Runacres et al., 2019a). However, in the absence of studies comparing trials with, and without, motivational techniques, it is not possible to ascertain whether these strategies are successful in the attainment of a maximal effort in youth. Additionally, a limitation with force-velocity-power profiling, used to estimate kinetic variables in **Chapters 6 and 7**, was the use of an offset derived from block starts (Samozino, 2018; Samozino et al., 2016). Therefore, this offset may not accurately represent the offset needed from two-point sprint starts (Rossi et al., 2017; Samozino, 2018), but was employed in the absence of a standing start equivalent. Future research should seek to



calculate the specific offset required from standing starts to enhance the accuracy and reliability of the associated kinetic parameters.

#### *9.4 Future Research Directions*

The present thesis has highlighted a number of areas that warrant further investigation to better understand the short- and long-term effects of sex, maturity and PA levels on training responses during childhood and adolescence. Evidence regarding the effect of sex on both aerobic and anaerobic trainability is scarce, with a clear male dominance in the literature, much of which is unlikely to be generalisable to their female counterparts. Moreover, when boys and girls have been directly compared, the majority of the literature has focused on pre-pubertal children where the anthropometric and physiological differences between sexes are yet to be fully manifest (Roemmich et al., 2000; Rogol, 1994; Rogol et al., 2002). Indeed, there is no significant sex difference in body composition until the age of 13 years (Roemmich et al., 2001; Rogol et al., 2002), coinciding with PHV in boys and the transition to post-pubertal status in girls. Therefore, purposeful research which compares maturity-matched participants is required to discern the true physiological effect of sex. The physiological significance of sex is of paramount importance to the design, and implementation, of LTAD programs to ensure the optimal performance of young athletes. Additionally, given the potentially different responses to training according to sex reported in **Chapters 4** and **6**, it may be possible that sex influences the long-term effects of intensive training. However, the only research available investigating the influence of training in childhood on health outcomes during adulthood pooled data from boys and girls (Yang et al., 2009), therefore this remains to be established.

A critical element not accounted for in the current thesis was the genetic influence on both aerobic and anaerobic trainability. Indeed, genetics have been estimated to account for up to 60% of the variance in aerobic (Bouchard, Dionne, Simoneau, & Boulay, 1992) and anaerobic performance (Bouchard et al., 1992; Niemi & Majamaa, 2005; Schutte, Nederend, Hudziak, de Geus, & Bartels, 2016). Monozygotic twin studies have the ability to overcome this, allowing for environmental factors (i.e. training) to be studied in more detail (Danis et al., 2003; Weber et al., 1976).

Moreover, studies in monozygotic twins may allow for greater insights into the mechanisms underpinning the development of both aerobic and anaerobic performances which are starting to be elucidated (McNarry et al., 2015; Schutte et al., 2016; Winsley et al., 2009), but remain to be fully established. Given that the myocardium and other areas of the body undergo rapid morphological and structural changes during growth and maturation, chronic intensive exercise training during adolescence could have long-lasting impacts potentially affecting adult health (Farr et al., 2014; J. Wells, 2007). Therefore, by controlling for the genetic influence on long-term health prognosis, it may also be possible to establish the long-term effect of chronic intensive training in childhood. Clarity on this fundamental question may allow for interventions to be implemented, if necessary, to improve the long-term health of elite youth athletes.

The combination of radar technology and macroscopic biomechanical modelling utilised to assess single and repeated sprint ability in **Chapters 6 and 7**, respectively, shows great promise and could enhance our understanding of the kinetic determinants of sprint performance and the mechanisms of fatigue. These methods utilise simple data collection methods whilst retaining ecological validity, enabling large cross-sectional and longitudinal studies. Future research should seek to implement specific training interventions to improve sprint performance, based on an initial kinetic profile. More specifically, the two kinetic determinants of sprint performance found in **Chapter 6**, irrespective of maturity status, were allometrically scaled  $P_{\text{peak}}$  and the efficiency of force application. Therefore, participants identified as power deficient could undertake either resistance or plyometric training, both of which can improve  $P_{\text{peak}}$  in as little as eight weeks if implemented appropriately (Faigenbaum et al., 2009; Girard et al., 2011; Rumpf et al., 2012). Similarly, those with a sub-optimal  $D_{\text{RF}}$  could undergo technique-specific drills to continue effective force application at higher sprinting velocities, enabling continued acceleration or a better maintenance of maximal velocity (Rossi et al., 2017; Samozino et al., 2016). Such methods facilitate a targeted approach and could improve the efficiency of training interventions. Finally, given the high repeatability, radar technology and biomechanical modelling can be used to monitor athlete performances over the course of season, or indeed many years, potentially enhancing talent identification test batteries.

**Chapter 8** highlighted the potential between-sport differences in long-term health outcomes stemming from chronic intensive exercise training, although it may be prudent to note that there is a sparsity of literature that has examined the long-term effects of training in childhood into adulthood. Whilst it could be assumed that the elite athletes included within the studies in the systematic review in **Chapter 8** would have started training intensively during youth to reach the elite levels they did, the study does not allow for the examination of the development of those conditions over time. In one of the only studies reporting the long-term effects of training during childhood, at least three years of sustained sports participation during adolescence was necessary to reduce the development of metabolic syndrome in adulthood (Yang et al., 2009). However, like with many of the early elite athlete studies, the authors did not consider the effects of sport type (Yang et al., 2009), which could have varying long-term health-related effects as demonstrated in **Chapter 8**. Therefore, more research is urgently required to establish the effects of different training types on the long-term health of youth athletes.

### 9.5 Overall Conclusions

In conclusion, this thesis investigated the influence of sex and maturity on the aerobic and anaerobic trainability of children and adolescents. The development of both aerobic and anaerobic parameters were revealed to be sexually dimorphic, supporting the consensus that findings from research conducted in boys cannot be directly applied to girls, with significant differences present even in pre-pubertal children. Of importance, this thesis provides evidence that differences in habitual PA levels significantly influence peak  $\dot{V}O_2$  in children and adolescents, reinforcing the need for future training studies to measure and control for habitual PA levels to determine the true effects of training interventions. Finally, sport-specific differences are apparent in all-cause, CVD and cancer mortality in men, but more research is needed to establish whether these differences also persist in former female athletes.

# **Chapter 10**

## **References**

## **Chapter 10 – References**

- Abbasian, S., Gholamian, S., Attarzadeh, S. R., Khabazan, M. A., & Khodadadi, H. (2011). The validity of between Wingate Test and Running-based Anaerobic Sprint Test (RAST) in young elite basketball players. *International Journal of Health, Education and Computer Science in Sports*, 4(1), 68-70.
- Abel, E., & Kruger, M. (2005). Longevity of major league baseball players. *Research in Sports Medicine*, 13(1), 1-5. doi: 10.1080/15438620590922031
- Abel, E., & Kruger, M. (2006a). The healthy worker effect in major league baseball revisited. *Research in Sports Medicine*, 14(1), 83-87. doi: 10.1080/15438620500528406
- Abel, E., & Kruger, M. (2006b). The healthy worker effect in professional football. *Research in Sports Medicine*, 14(4), 239-243. doi: 10.1080/15438620600985795
- Agrotou, S., Karatzi, K., Papamichael, C., Fatouros, I., Mitrakou, A., Zakopoulos, N., . . . Stamatelopoulos, K. (2013). Effects of Chronic Anaerobic Training on Markers of Sub-Clinical Atherosclerosis. *Hellenic Journal of Cardiology*, 54(3), 178-185.
- Ainsworth, B. E., Haskell, W. L., Whitt, M. C., Irwin, M. L., Swartz, A. M., Strath, S. J., . . . Leon, A. S. (2000). Compendium of Physical Activities: an update of activity codes and MET intensities. *Medicine & Science in Sport and Exercise*, 32, 498 - 516.
- Al-Mallah, M. H., Sakr, S., & Al-Qunaibet, A. (2018). Cardiorespiratory Fitness and Cardiovascular Disease Prevention: An Update. *Current Atherosclerosis Reports*, 20(1) doi: doi: 10.1007/s11883-018-0711-4.
- Anderson, C. S., & Mahon, A. D. (2007). The relationship between ventilatory and lactate thresholds in boys and men. *Research in Sports Medicine*, 15(3), 189-200. doi: 10.1080/15438620701525490
- Antero-Jacquemin, J., Desgorces, F. D., Dor, F., Sedeaud, A., Haida, A., LeVan, P., & Toussaint, J. F. (2014). Row for Your Life: A Century of Mortality Follow-Up of French Olympic Rowers. *Plos One*, 9(11) doi: 10.1371/journal.pone.0113362
- Antero-Jacquemin, J., Pohar-Perme, M., Rey, G., Toussaint, J. F., & Latouche, A. (2018). The heart of the matter: years-saved from cardiovascular and cancer deaths in an elite athlete cohort with over a century of follow-up. *European Journal of Epidemiology*, 33(6), 531-543. doi: 10.1007/s10654-018-0401-0
- Antero-Jacquemin, J., Rey, G., Marc, A., Dor, F., Haida, A., Marck, A., . . . Toussaint, J. F. (2015). Mortality in Female and Male French Olympians: A 1948-2013 Cohort Study. *American Journal of Sports Medicine*, 43(6), 1505-1512. doi: 10.1177/0363546515574691
- Armstrong, M., Green, J., Reeves, G., Beral, V., & Cairns, B. (2015a). Frequent Physical Activity May Not Reduce Vascular Disease Risk as Much as Moderate Activity: Large Prospective Study of Women in the United Kingdom. *Circulation*, 131, 721-729.
- Armstrong, N. (2007). *Paediatric Exercise Physiology*: Churchill Livingstone Elsevier.

- Armstrong, N. (2013). Aerobic fitness and physical activity in children. *Pediatric exercise science*, 25(4) doi: 10.1123/pes.25.4.548
- Armstrong, N. (2015). Aerobic Fitness and Training in Children. *Pediatric Exercise Science*, 27(1), 8-12. doi: 10.1123/pes.2015-0027
- Armstrong, N. (2017). Commentary: Pediatric Aerobic Fitness and Trainability. *Pediatric Exercise Science*, 29(1), 8-13.
- Armstrong, N., & Barker, A. (2011). Endurance Training and Elite Young Athletes. In N. Armstrong & A. McManus (Eds.), *The Elite Young Athlete*. Basel, Karger: Medicine in Sports Science.
- Armstrong, N., & Barker, A. (2012a). New insights in paediatric exercise metabolism. *Journal of Sport and Health Science*, 1(1), 18-26. doi: 10.1016/j.jshs.2011.12.001
- Armstrong, N., Barker, A., & McManus, A. (2015b). Muscle metabolism changes with age and maturation: How do they relate to youth sport performance? *British Journal of Sports Medicine*, 49(13), 860-864. doi: 10.1136/bjsports-2014-094491
- Armstrong, N., & Barker, A. R. (2012b). New insights in paediatric exercise metabolism. *Journal of Sport and Health Science*, 1(1), 18-26. doi: 10.1016/j.jshs.2011.12.001
- Armstrong, N., & McNarry, M. (2016). Aerobic Fitness and Trainability in Healthy Youth: Gaps in Our Knowledge. *Pediatric Exercise Science*, 28(2), 171-177. doi: 10.1123/pes.2015-0251
- Armstrong, N., Tomkinson, G., & Ekelund, U. (2011). Aerobic fitness and its relationship to sport, exercise training and habitual physical activity during youth. *British journal of sports medicine*, 45(11) doi: 10.1136/bjsports-2011-090200
- Armstrong, N., & Welsman, J. (2001). Peak oxygen uptake in relation to growth and maturation in 11- to 17-year-old humans. *European Journal of Applied Physiology*, 85, 546-551.
- Armstrong, N., & Welsman, J. (2019a). Multilevel allometric modelling of maximal stroke volume and peak oxygen uptake in 11-13-year-olds. *European journal of applied physiology*, 119(11-12) doi: 10.1007/s00421-019-04241-3
- Armstrong, N., & Welsman, J. (2019b). Sex-Specific Longitudinal Modeling of Youth Peak Oxygen Uptake. *Pediatric Exercise Science*, 31(2), 204-212. doi: 10.1123/pes.2018-0175
- Armstrong, N., & Welsman, J. (2019c). Sex-Specific Longitudinal Modelling of Short-Term Power in 11- to 18-Year-Olds. *Medicine and Science in Sport & Exercise*, 51(5), 1055.
- Armstrong, N., & Welsman, J. (2019d). Youth cardiorespiratory fitness: evidence, myths and misconceptions *Bull World Health Organ* (Vol. 97, pp. 777-782).
- Armstrong, N., & Welsman, J. (2020a). Multilevel allometric modelling of maximum cardiac output, maximum arteriovenous oxygen difference, and peak oxygen uptake in 11–13-year-olds *European Journal of Applied Physiology* (Vol. 120, pp. 527-537).
- Armstrong, N., & Welsman, J. (2020b). Scientific Rigour in the Assessment and Interpretation of Youth Cardiopulmonary Fitness: A Response to the Paper ‘Normative Reference Values and International Comparisons for the 20-Metre Shuttle Run Test: Analysis of 69,960 Test Results among Chinese Children and Youth. *Journal of Sports Science and Medicine*, 19(3), 627 - 629.

- Armstrong, N., & Welsman, J. (2020c). The Development of Aerobic and Anaerobic Fitness with Reference to Youth Athletes. *Journal of Science in Sport and Exercise* doi: <https://doi.org/10.1007/s42978-020-00070-5>
- Armstrong, N., Williams, J., Balding, J., Gentle, P., & Kirby, B. (1991a). Cardiopulmonary fitness, Physical Activity Patterns and Selected Coronary Risk Factor Variables in 11- to 16-Year Olds. *Paediatric Exercise Science*, 3, 219-228.
- Armstrong, N., Williams, J., Balding, J., Gentle, P., & Kirby, B. (1991b). The peak oxygen uptake of British children with reference to age, sex and sexual maturity. *European Journal of Applied Physiology*, 62, 369-375.
- Armstrong, R., & Laughlin, M. (1984). Exercise blood flow pattern within and among rat muscles after training. *American Journal of Physiology*, 246, 59-68.
- Attard, S. M., Hering, A. H., Howard, A. G., & Gorden-Larsen, P. (2013). Longitudinal trajectories of BMI and cardiovascular disease risk: the national longitudinal study of adolescent health. *Obesity*, 21(11), 2180-2188.
- Backmand, H., Kujala, U., Sarna, S., & Kaprio, J. (2010). Former athletes' health-related lifestyle behaviours and self-rated health in late adulthood. *International Journal of Sports Medicine*, 31(10), 751-758. doi: 10.1055/s-0030-1255109
- Balaban, R., Mootha, V., & Arai, A. (1996). Spectroscopic Determination of Cytochrome C Oxidase Content in Tissues Containing Myoglobin or Hemoglobin. *Analytical biochemistry*, 237(2) doi: 10.1006/abio.1996.0239
- Baquet, G., Berthoin, S., Dupont, G., Blondel, N., Fabre, C., & van Praagh, E. (2002). Effects of High Intensity Intermittent Training on Peak VO<sub>2</sub> in pre-pubertal children. *International Journal of Sports Medicine*, 23, 439-444.
- Baquet, G., Berthonin, S., Gerbeaux, M., & Van Praagh, E. (2001). High-intensity aerobic training during a 10 week one-hour physical education cycle: effects on physical fitness of adolescents aged 11 to 16. *International Journal of Sports Medicine*, 22(4), 295-300.
- Baquet, G., Gamelin, F., Mucci, P., Thevent, D., & van Praagh, E. (2010). Continuous vs interval training in 8- to 11-year old children. *Journal of Strength and Conditioning Research*, 24, 1381-1388.
- Baquet, G., Van Praagh, E., & Berthoin, S. (2003). Endurance training and aerobic fitness in young people. *Sports Medicine*, 33(15), 1127-1143. doi: 10.2165/00007256-200333150-00004
- Barker, A., Day, J., Smith, A., Bond, B., & Williams, C. (2014). The influence of 2 weeks of low-volume high-intensity interval training on health outcomes in adolescent boys. *Journal of Sport Sciences*, 32(8), 757-765.
- Barker, A., Welsman, J., Fulford, J., Welford, D., & Armstrong, N. (2010). Quadriceps muscle energetics during incremental exercise in children and adults. *Medicine & Science in Sport and Exercise*, 42(7), 1303-1313.
- Barker, A., Williams, C., Jones, A., & Armstrong, N. (2009). Establishing maximal oxygen uptake in young people during a ramp cycle test to exhaustion. *British Journal of Sports Medicine*, bjsports63180.
- Baron, S. L., Hein, M. J., Lehman, E., & Gersic, C. M. (2012). Body mass index, playing position, race, and the cardiovascular mortality of retired professional football players. *American Journal of Cardiology*, 109(6), 889-896. doi: 10.1016/j.amjcard.2011.10.050

- Barstow, T. (2019). Understanding Near Infrared Spectroscopy and Its Application to Skeletal Muscle Research. *Journal of applied physiology*, 126(5) doi: 10.1152/jappphysiol.00166.2018
- Barstow, T., Jones, A., Nguyen, P., & Casaburi, R. (1999). Influence of muscle fibre type and fitness on the oxygen uptake/power output slope during incremental exercise in humans. *Experimental Physiology*, 85(1), 109-116.
- Baxter-Jones, A., Goldstein, H., & Helms, P. (1993). The development of aerobic power in young athletes. *Journal of Applied Physiology*, 75(3), 1160-1167.
- Beaver, W. L., Wasserman, K., & Whipp, B. J. (1986). A new method for detecting anaerobic threshold by gas exchange. *Journal of Applied Physiology*, 60(6), 2020-2027.
- Becker, D. M., & Vaccaro, P. (1983). Anaerobic threshold alterations caused by endurance training in young children. *Journal of Sports Medicine and Physical Fitness*, 23, 445-449.
- Behringer, M., von Heede, A., & Mester, J. (2010). Effects of Resistance Training in Children and Adolescents: A Meta-Analysis. *Pediatrics*, 126 doi: DOI: 10.1542/peds.2010-0445
- Belli, S., & Vanacore, N. (2005). Proportionate mortality of Italian soccer players: is amyotrophic lateral sclerosis an occupational disease? *European Journal of Epidemiology*, 20(3), 237-242. doi: 10.1007/s10654-004-6879-7
- Benedettini, M. M. (2005). Analysis of the body mass index (BMI) in athletes over age 18 examined at the Sports Medicine Services of the Republic of San Marino in a 1-year period. *Medicina Dello Sport*, 58(1), 29-35.
- Beneke, R., Hutler, M., & Leithauser, R. M. (2007). Anaerobic performance and metabolism in boys and male adolescents. *European Journal of Applied Physiology*, 101(6), 671-677. doi: 10.1007/s00421-007-0546-0
- Bianco, M., Fabbriatore, C., Sanna, N., Fabiano, C., Palmieri, V., & Zeppilli, P. (2007). Elite athletes: Is survival shortened in boxers? *International Journal of Sports Medicine*, 28(8), 697-702. doi: 10.1055/s-2007-964863
- Bishop, D., & Edge, J. (2006). Determinants of repeated-sprint ability in females matched for single-sprint performance. *European Journal of Applied Physiology*, 97, 373-379.
- Bitar, A., Vernet, J., Coudert, J., & Vermorel, M. (2000). Longitudinal changes in body composition, physical capacities and energy expenditure in boys and girls during the onset of puberty. *European Journal of Nutrition*, 39(4), 157-163. doi: 10.1007/s003940070019
- Blair, S. N., Kohl, H. W., 3rd, Paffenbarger, R. S., Jr., Clark, D. G., Cooper, K. H., & Gibbons, L. W. (1989). Physical fitness and all-cause mortality. A prospective study of healthy men and women. *Jama*, 262(17), 2395-2401. doi: 10.1001/jama.262.17.2395
- Boisseau, N., & Delamarche, P. (2000). Metabolic and hormonal responses to exercise in children and adolescents. *Sports Medicine*, 30(6), 405-422. doi: 10.2165/00007256-200030060-00003
- Boone, J., Koppo, K., Barstow, T., & Bouckaert, J. (2009). Pattern of deoxy Hb plus Mb during ramp cycle exercise: influence of aerobic fitness status. *European Journal of Applied Physiology*, 105(6), 851-859. doi: 10.1007/s00421-008-0969-2



- Bossone, E., Vriza, O., Bodini, B. D., & Rubenfire, M. (2004). Cardiovascular response to exercise in elite ice hockey players. *Canadian Journal of Cardiology*, 20(9), 893-897.
- Bouchard, C., Dionne, F., Simoneau, J., & Boulay, M. (1992). Genetics of Aerobic and Anaerobic Performances. *Exercise and sport science reviews*, 20(1), 27-58.
- Breese, B. C., Saynor, Z. L., Barker, A. R., Armstrong, N., & Williams, C. A. (2019). Relationship between (non)linear phase II pulmonary oxygen uptake kinetics with skeletal muscle oxygenation and age in 11-15 year olds. *Experimental Physiology*, 104(12), 1929-1941. doi: 10.1113/ep087979
- Breese, B. C., Williams, C. A., Barker, A. R., Welsman, J. R., Fawcner, S. G., & Armstrong, N. (2010). Longitudinal changes in the oxygen uptake kinetic response to heavy-intensity exercise in 14- to 16-year-old boys. *Pediatric Exercise Science*, 22(1), 69-80. doi: 10.1123/pes.22.1.69
- Buenen, G. P., Malina, R. M., Van't Hof, M. A., Simons, J., Ostyn, M., Renson, R., & Van Gerven, D. (1998). Adolescent growth and motor performance: A longitudinal study of Belgian boys: Human Kinetics.
- Buenen, G. P., & Thomis, M. (2000). Muscular Strength Development in Children and Adolescents. *Paediatric Exercise Science*, 12, 174-197.
- Butte, N., Puyau, M., Adolph, A., Vohra, F., & Zakeri, I. (2007). Physical Activity in Nonoverweight and Overweight Hispanic Children and Adolescents. *Medicine & Science in Sport & Exercise*, 39(8), 1257-1266.
- Cahill, M., Oliver, J., Cronin, J., Clark, K., Cross, M., & Lloyd, R. (2020). Influence of resisted sled-push training on the sprint force-velocity profile of male high school athletes. *Scandinavian Journal of Medicine & Science in Sports* doi: <https://doi.org/10.1111/sms.13600>
- Campbell, B., & Mbizo, M. (2006). Reproductive maturation, somatic growth and testosterone among Zimbabwe boys. *Annals of Human Biology*, 33(1), 17-25. doi: 10.1080/03014460500424068
- Cao, M., Quan, M., & Zhuang, J. (2019). Effect of High-Intensity Interval Training versus Moderate-Intensity Continuous Training on Cardiorespiratory Fitness in Children and Adolescents: A Meta-Analysis *International Journal of Environmental Research and Public Health* (Vol. 16).
- Carazo-Vargas, P., & Moncada-Jiménez, J. (2015). A meta-analysis on the effects of exercise training on the VO<sub>2</sub> max in children and adolescents. *Retos*, 27, 184-187.
- Carson, V., & Spence, J. (2010). Seasonal variation in physical activity among children and adolescents: a review. *Pediatric exercise science*, 22(1) doi: 10.1123/pes.22.1.81
- Carson, V., Tremblay, M., Chaput, J., & Chastin, S. (2016). Associations Between Sleep Duration, Sedentary Time, Physical Activity, and Health Indicators Among Canadian Children and Youth Using Compositional Analyses. *Applied physiology, nutrition, and metabolism*, 41(6) doi: 10.1139/apnm-2016-0026
- Carson, V., Tremblay, M., Chaput, J., McGregor, D., & Chastin, S. (2019). Compositional analyses of the associations between sedentary time, different intensities of physical activity, and cardiometabolic biomarkers among children and youth from the United States. *PLoS One*, 14(7), e0220009. doi: <https://doi.org/10.1371/journal.pone.0220009>

- Castanheira, J., Valente-Dos-Santos, J., Costa, D., Martinho, D., Fernandes, J., Duarte, J., . . . Coelho-E-Silva, M. (2017). Cardiac remodeling indicators in adolescent athletes. *Revista Da Associacao Medica Brasileira*, 63(5), 427-434. doi: 10.1590/1806-9282.63.05.427
- Catley, M., & Tomkinson, G. (2013). Normative health-related fitness values for children: analysis of 85347 test results on 9–17-year-old Australians since 1985. *British Journal of Sports Medicine*, 47, 98-109.
- Charloux, A., Lonsdorfer-Wolf, E., Richard, R., Lampert, E., Oswald-Mammosser, M., Mettauer, B., . . . Lonsdorfer, J. (2000). A new impedance cardiograph device for the non-invasive evaluation of cardiac output at rest and during exercise: comparison with the "direct" Fick method. *European journal of applied physiology*, 82(4) doi: 10.1007/s004210000226
- Chastin, S., Palarea-Albaladejo, J., Dontje, M., & Skelton, D. (2015). Combined Effects of Time Spent in Physical Activity, Sedentary Behaviors and Sleep on Obesity and Cardio-Metabolic Health Markers: A Novel Compositional Data Analysis Approach. *PloS one*, 10(10) doi: 10.1371/journal.pone.0139984
- Chia, Y. H. M. (2006). Aerobic energy contribution to maximal exercise in children. *Biology of Sport*, 23(2), 117-125.
- Christofaro, D. G. D., Casonatto, J., Polito, M. D., Cardoso, J. R., Fernandes, R., Guariglia, D. A., . . . Oliveira, A. R. d. (2009). Evaluation of the Omron MX3 Plus monitor for blood pressure measurement in adolescents. [OriginalPaper]. *European Journal of Pediatrics*, 168(11), 1349-1354. doi: doi:10.1007/s00431-009-0936-x
- Clarke, P., Walter, S., Hayen, A., Mallon, W., Heijmans, J., & Studdert, D. (2012). Survival of the fittest: retrospective cohort study of the longevity of Olympic medallists in the modern era. *British Medical Journal*, 345 doi: 10.1136/bmj.e8308
- Coleman, L., & Coleman, J. (2002). The measurement of puberty: a review. *Journal of Adolescence*, 25(5), 535-550.
- Coombes, J. S., Law, J., Lancashire, B., & Fasset, R. G. (2015). "Exercise Is Medicine": Curbing the Burden of Chronic Disease and Physical Inactivity. *Asia-Pacific Journal of Public Health*, 27(2), NP600-NP605. doi: 10.1177/1010539513481492
- Costigan, S. A., Eather, N., Plotnikoff, R. C., Taaffe, D. R., & Lubans, D. R. (2015). High-intensity interval training for improving health-related fitness in adolescents: a systematic review and meta-analysis. *British Journal of Sports Medicine*, bjsports - 2014.
- Couto, P. G., Lima, H. M., Soares, R. P., Bertuzzi, R., De-Oliveira, F. R., & Lima-Silva, A. E. (2014). Effect of Fat- and Carbohydrate-Rich Diets on Metabolism and Running Performance in Trained Adolescent Boys. *Journal of Pediatric Gastroenterology and Nutrition*, 59(3), 380-385. doi: 10.1097/mpg.0000000000000427
- Cunha, G., Lorenzi, T., Sapata, K., Lopes, A., Gaya, A., & Oliveira, A. (2011). Effect of biological maturation on maximal oxygen uptake and ventilatory thresholds in soccer players: An allometric approach. *Journal of Sports Sciences*, 29(10), 1029-1039. doi: 10.1080/02640414.2011.570775
- Cunha, G., Vaz, M., Geremia, J., Leites, G., Baptista, R., Lopes, A., & Reischak-Oliveira, A. (2016). Maturity Status Does Not Exert Effects on Aerobic Fitness

- in Soccer Players After Appropriate Normalization for Body Size. *Pediatric Exercise Science*, 28(3), 456-465. doi: 10.1123/pes.2015-0133
- Cunningham, D. A., Paterson, D. H., Blimkie, C. J., & Donner, A. P. (1984). Development of cardiorespiratory function in circumpubertal boys: a longitudinal study. *Journal of Applied Physiology Respir Environ Exerc Physiol*, 56(2), 302-307. doi: 10.1152/jappl.1984.56.2.302
- D'Ascenzi, F., Zorzi, A., Alvino, F., Bonifazi, M., Mondillo, S., & Corrado, D. (2019). Ventricular extrasystoles in young athletes: classification and diagnostic pathway. *Giornale Italiano Di Cardiologia*, 20(4), 229-241. doi: 10.1714/3126.31076
- Danis, A., Kyriazis, Y., & Klissouras, V. (2003). The effect of training in male prepubertal and pubertal monozygotic twins. *European Journal Of Applied Physiology*, 89, 309-318.
- Daoud, J. (2017). Multicollinearity and Regression Analysis. *Journal of Physics: Conference Series*, 949 doi: doi :10.1088/1742-6596/949/1/012009
- Dencker, M., & Andersen, L. (2011). Accelerometer-measured daily physical activity related to aerobic fitness in children and adolescents. *Journal of Sport Sciences*, 29(9), 887-895.
- Dencker, M., Thorsson, O., Karlsson, M., Lindén, C., Eiberg, S., Wollmer, P., & Andersen, L. (2007). Gender differences and determinants of aerobic fitness in children aged 8-11 years. *European journal of applied physiology*, 99(1) doi: 10.1007/s00421-006-0310-x
- Dencker, M., Thorsson, O., Karlsson, M., Lindén, C., Svensson, J., Wollmer, P., & Andersen, L. (2006). Daily physical activity and its relation to aerobic fitness in children aged 8-11 years. *European journal of applied physiology*, 96(5) doi: 10.1007/s00421-005-0117-1
- Doncaster, G., Iga, J., & Unnithan, V. (2018). Assessing Differences in Cardiorespiratory Fitness With Respect to Maturity Status in Highly Trained Youth Soccer Players. *Pediatric Exercise Science*, 30(2), 216-228. doi: 10.1123/pes.2017-0185
- Doré, E., Bedu, M., & Van Praagh, E. (2008). Squat Jump performance during growth in both sexes: comparison with cycling power. *Research Quarterly for Exercise and Sport*, 79(4), 517-524.
- Doré, E., Duché, P., Rouffet, D., Ratel, S., Bedu, M., & Van-Praagh, E. (2003). Measurement error in short-term power testing in young people. *Journal of Sports Sciences*, 21(2), 135-142.
- Doré, E., Martin, R., Ratel, S., Duché, P., Bedu, M., & Van Praagh, E. (2005). Gender differences in peak muscle performance during growth. *International journal of sports medicine*, 26(4), 274-280.
- Dotan, R., Mitchell, C., Cohen, R., Klentrou, P., Gabriel, D., & Falk, B. (2012). Child-Adult Differences in Muscle Activation - A Review. *Pediatric Exercise Science*, 24(1), 2-21.
- Dumith, S., Gigante, D., Domingues, M., & Kohl III, H. (2011). Physical activity change during adolescence: a systematic review and pooled analysis. *International Journal of Epidemiology*, 40(3), 685-698.
- Dumuid, D., Stanford, T., Martin-Fernández, J., Pedišić, Ž., Maher, C., Lewis, L., . . . Olds, T. (2018a). Compositional data analysis for physical activity, sedentary time and sleep research. *Statistical methods in medical research*, 27(12) doi: 10.1177/0962280217710835

- Dumuid, D., Stanford, T., Pedisic, Z., Maher, C., Lewis, L., Martin-Fernandez, J., . . . Olds, T. (2018b). Adiposity and the isothermal substitution of physical activity, sedentary time and sleep among school-aged children: a compositional data analysis approach. *BMC Public Health, 18*(1)
- Dupont, G., Millet, G., Cuinhouya, C., & Berthoin, S. (2005). Relationship between oxygen uptake kinetics and performance in repeated running sprints. *European Journal of Applied Physiology, 95*, 27-34.
- Eddolls, W., McNarry, M., Stratton, G., Winn, C., & Mackintosh, K. (2017). High-Intensity Interval Training Interventions in Children and Adolescents: A Systematic Review. *Sports Medicine, 47*, 2363-2374.
- Eisenmann, J., Katzmarzyk, P., Theriault, G., Song, T., Malina, R., & Bouchard, C. (2000). Cardiac dimensions, physical activity, and submaximal working capacity in youth of the Quebec Family Study. *European Journal of Applied Physiology, 81*, 40-46.
- Ekelund, U., Poortvliet, E., Nilsson, A., Yngve, A., Holmberg, A., & Sjöström, M. (2001). Physical activity in relation to aerobic fitness and body fat in 14- to 15-year-old boys and girls. *European journal of applied physiology, 85*(3-4) doi: 10.1007/s004210100460
- Ekelund, U., Steene-Johannessen, J., Brown, W., Fagerland, M., Owen, N., Powell, K., . . . Lee, I. (2016). Physical activity attenuates the detrimental association of sitting time with mortality: A harmonised meta-analysis of data from more than one million men and women. *The Lancet, 388*(10051), 1302-1310.
- Eriksson, B. (1972). Physical Training, Oxygen Supply and Muscle Metabolism in 11-13-year Old Boys. *Acta physiologica Scandinavica. Supplementum, 384*
- Eriksson, B. (1980). Muscle metabolism in children--a review. *Acta Paediatr Scand Suppl, 283*, 20-28. doi: 10.1111/j.1651-2227.1980.tb15304.x
- Eriksson, B., Gollnick, P., & Saltin, B. (1973). Muscle metabolism and enzyme-activities after training in boys 11-13 years old. *Acta Physiologica Scandinavica, 87*(4), 485-497. doi: 10.1111/j.1748-1716.1973.tb05415.x
- Evenson, K. R., Catellier, D. J., Gill, K. I., Ondrak, K. S., & McMurray, R. G. (2008). Calibration of two objective measures of physical activity for children. *Journal of Sport Sciences, 26*(14), 1557-1565.
- Faigenbaum, A. D., Kraemer, W. J., Blimkie, C. J. R., Jeffreys, I., Micheli, L. J., Nitka, M., & Rowland, T. W. (2009). Youth Resistance Training: Updated Position Statement Paper from the National Strength and Conditioning Association. *Journal of Strength and Conditioning Research, 23*, S60-S79. doi: 10.1519/JSC.0b013e31819df407
- Fairclough, S., Noonan, R., Rowlands, A., van Hees, V., Knowles, Z., & Boddy, L. (2016). Wear compliance and activity in children wearing wrist and hip mounted accelerometers. *Medicine & Science in Sport & Exercise, 48*, 245 - 253.
- Farahmand, B. Y., Ahlbom, A., Ekblom, O., Ekblom, B., Hallmarker, U., Aronson, D., & Brobert, G. P. (2003). Mortality amongst participants in Vasaloppet: a classical long-distance ski race in Sweden. *Journal of International Medicine, 253*(3), 276-283. doi: 10.1046/j.1365-2796.2003.01122.x
- Farr, J. N., Laddu, D. R., & Going, S. B. (2014). Exercise, Hormones, and Skeletal Adaptations During Childhood and Adolescence. *Pediatric Exercise Science, 26*(4), 384-391. doi: 10.1123/pes.2014-0077

- Faude, O., Schnittker, R., Schulte-Zurhasen, R., Muller, F., & Meyer, T. (2013). High intensity interval training vs. high-volume running training during pre-season conditioning in high-level youth football: a cross-over trial. *Journal of Sport Sciences*, 31(13), 1441-1450.
- Fellmann, N., & Coudert, J. (1994a). Physiological Responses To Exercise In Children. *Archives De Pediatrie*, 1(9), 827-840.
- Fenster, J., Freedson, P., Washburn, R., & Ellison, R. (1989). The relationship between peak oxygen uptake and physical activity in 6- to 8 year-old children. *Pediatric Exercise Science*, 1, 127-138.
- Fernandes, G. S., Parekh, S. M., Moses, J., Fuller, C. W., Scammell, B., Batt, M. E., . . . Doherty, M. (2019). Depressive symptoms and the general health of retired professional footballers compared with the general population in the UK: a case-control study. *BMJ Open*, 9(9), e030056. doi: 10.1136/bmjopen-2019-030056
- Fernandez-Santos, J. R., Ruiz, J. R., Cohen, D. D., Gonzalez-Montesinos, J. L., & Castro-Piñero, J. (2015). Reliability and Validity of tests to assess lower-body muscular power in children. *Journal of Strength and Conditioning Research*, 29(8), 2277-2285.
- Ferreira, L., Koga, S., & Barstow, T. (2007). Dynamics of noninvasively estimated microvascular O<sub>2</sub> extraction during ramp exercise. *Journal of applied physiology (Bethesda, Md. : 1985)*, 103(6) doi: 10.1152/jappphysiol.01414.2006
- Fick, A. (1855). Ueber Diffusion. *Annalen der Physik*, 94, 59-86.
- Fick, A. (1870a). *Über die Messung des Blatquantums in den Herzven-trikeln* (Vol. XVI-XVII): Gesellschaft zu Wurzburg.
- Fick, A. (1870b). Ueber die Messung dea Blutquantums in den Herzventrikela. *Verhandlungen der Physikalisch-medizinische Gesellschaft zu Wurzburg*
- Fitzsimons, M., Dawson, B., Ward, D., & Wilkinson, A. (1993). Cycling and running tests of repeated sprint ability. *Australian Journal of Science and Medicine in Sport*, 25(4), 82-87.
- Fogelholm, M., Kaprio, J., & Sarna, S. (1994). Healthy Life-Styles of Former Finnish World-Class Athletes. *Medicine and Science in Sports and Exercise*, 26(2), 224-229. doi: 10.1249/00005768-199402000-00013
- Forbregd, T. R., Aloyseus, M. A., Berg, A., & Greve, G. (2019). Cardiopulmonary Capacity in Children During Exercise Testing: The Differences Between Treadmill and Upright and Supine Cycle Ergometry. *Frontiers in Physiology*, 10 doi: 10.3389/fphys.2019.01440
- Foster, C., Farland, C. V., Guidotti, F., Harbin, M., Roberts, B., Schuette, J., . . . Porcari, J. P. (2015). The Effects of High Intensity Interval Training vs Steady State Training on Aerobic and Anaerobic Capacity. *Journal of Sports Science and Medicine*, 14(4), 747-755.
- Fournier, M., Ricca, A., Taylor, R., Ferguson, R., Montpetit, R., & Chairman, B. (1982). Skeletal muscle adaptation in adolescent boys; sprint and endurance training and detraining. *Medicine & Science in Sport & Exercise*, 14, 453-456.
- Freedson, P. S., Pober, D., & Janz, K. F. (2005). Calibration of accelerometer output for children. *Medicine & Science in Sport & Exercise*, 37, 523 - 530.
- Gajda, A., Smigielski, W., Smigielski, J., Pakos, E., & Drygas, W. (2018). Longevity and cardiovascular mortality of Polish elite football players. *Kardiologia Polska*, 76(12), 1705-1711. doi: 10.5603/KP.a2018.0173

- Gajewski, A. K., & Poznanska, A. (2008). Mortality of top athletes, actors and clergy in Poland: 1924-2000 follow-up study of the long term effect of physical activity. *European Journal of Epidemiology*, 23(5), 335-340. doi: 10.1007/s10654-008-9237-3
- Garatachea, N., Santos-Lozano, A., Sanchis-Gomar, F., Fiuza-Luces, C., Pareja-Galeano, H., Emanuele, E., & Lucia, A. (2014). Elite Athletes Live Longer than the General Population: A Meta-Analysis. *Mayo Foundation for Medical Education and Research*, 1-6.
- Garcia-Hermoso, A., Cerrillo-Urbina, A. J., Herrera-Valenzuela, T., Cristi-Montero, C., Saavedra, J. M., & Martinez-Vizcaino, V. (2016). Is high-intensity interval training more effective on improving cardiometabolic risk and aerobic capacity than other forms of exercise in overweight and obese youth? A meta-analysis. *Obesity Reviews*, 17(6), 531-540. doi: 10.1111/obr.12395
- Girard, O., & Farooq, A. (2012). Effects of Ramadan fasting on repeated sprint ability in young children. *Science & Sports*, 27, 237-240.
- Girard, O., Mendez-Villanueva, A., & Bishop, D. (2011). Repeated-Sprint Ability - Part I Factors Contributing to Fatigue. *Sports Medicine*, 41(8), 673-694.
- Gist, N. H., Fedewa, M. V., Dishman, R. K., & Cureton, K. J. (2013). Sprint Interval Training Effects on Aerobic Capacity: A Systematic Review and Meta-Analysis. *Sports Medicine*, 44(2), 269-279.
- Gonzalez, J. M., Johnson, F. R., Fedoruk, M., Posner, J., & Bowers, L. (2018). Trading Health Risks for Glory: A Reformulation of the Goldman Dilemma. *Sports Medicine*, 48(8), 1963-1969. doi: 10.1007/s40279-018-0881-9
- Goode, A., Hall, K., Batch, B., Huffman, K., Hastings, S., Allen, K., . . . Gierisch, J. (2017). The Impact of Interventions that Integrate Accelerometers on Physical Activity and Weight Loss: A Systematic Review. *Annals of Behavioural Medicine*, 51(1), 79-93.
- Goodman, A., Mackett, R., & Paskins, J. (2011). Activity compensation and activity synergy in British 8-13 year olds. *Preventative Medicine*, 53, 293-298.
- Goran, M., Poehlman, E., & Danforth-Jr, E. (1994). Experimental reliability of the doubly labelled water technique. *American Journal of Physiology*, 266, 510 - 515.
- Gormley, S. E., Swain, D. P., High, R., Spina, R. J., Dowling, E. A., Kotipalli, U. S., & Gandrakota, R. (2008). Effect of intensity of aerobic training on VO2max. *Medicine and Science in Sports and Exercise*, 40(7), 1336-1343. doi: 10.1249/MSS.0b013e318c4839
- Green, M. (2006). From "Sport for All" to Not About "Sport for All": Interrogating Sport Policy Interventions In the United Kingdom. *European Sport Management Quarterly*, 6(3), 217-238.
- Gremeaux, V., Gayda, M., Lepers, R., Sosner, P., Juneau, M., & Nigam, A. (2012). Exercise and Longevity. *Maturitas*, 73(4), 312 - 317.
- Greulich, W. W., & Pyle, S. I. (1959). *Radiograph atlas of skeletal development of the hand and wrist* (Vol. 2nd Edition). California: Stanford University Press.
- Grimsmo, J., Maehlum, S., Moelstad, P., & Arnesen, H. (2011). Mortality and cardiovascular morbidity among long-term endurance male cross country skiers followed for 28-30 years. *Scandinavian Journal of Medicine & Science in Sports*, 21(6), E351-E358. doi: 10.1111/j.1600-0838.2011.01307.x

- Grodjinovsky, A., Inbar, O., Dotan, R., & Bar-Or, O. (1980). Training effect on the anaerobic performance of children as measured by the Wingate Anaerobic test. *Children and exercise IX* (pp. 139-145). University Park Press, Baltimore.
- Guo, J. J., Lou, Y. M., Zhang, X., & Song, Y. Q. (2015). Effect of aerobic exercise training on cardiometabolic risk factors among professional athletes in the heaviest-weight class. *Diabetology & Metabolic Syndrome*, 7 doi: 10.1186/s13098-015-0071-y
- Guo, J. J., Zhang, X., Wang, L., Guo, Y., & Xie, M. H. (2013). Prevalence of Metabolic Syndrome and Its Components among Chinese Professional Athletes of Strength Sports with Different Body Weight Categories. *Plos One*, 8(11) doi: 10.1371/journal.pone.0079758
- Gurley, K., Shang, Y., & Yu, G. (2012). Noninvasive Optical Quantification of Absolute Blood Flow, Blood Oxygenation, and Oxygen Consumption Rate in Exercising Skeletal Muscle. *Journal of biomedical optics*, 17(7) doi: 10.1117/1.JBO.17.7.075010
- Gutin, B., Yin, Z., Humphries, M., & Barbeau, P. (2005). Relations of moderate and vigorous physical activity to fitness and fatness in adolescents. *American Journal of Clinical Nutrition*, 81, 746-750.
- Hallal, P., Anderson, L., Bull, F., Guthold, R., Haskell, W., & Ekelund, U. (2012). Global physical activity levels: surveillance progress, pitfalls, and prospects. *The Lancet*, 380, 247-257.
- Hammami, R., Makhlof, I., Chtara, M., Padulo, J., & Chaouachi, A. (2015). The contribution of vertical explosive strength to sprint performance in children. *Sport Science & Health*, 11, 37-42. doi: 10.1007/s11332-014-0200-2
- Harper, A., Ferreira, L., Lutjemeier, B., Townsend, D., & Barstow, T. (2006). Human Femoral Artery and Estimated Muscle Capillary Blood Flow Kinetics Following the Onset of Exercise. *Experimental physiology*, 91(4) doi: 10.1113/expphysiol.2005.032904
- Hasselstrøm, H., Hansen, S., Froberg, K., & Andersen, L. (2002). Physical fitness and physical activity during adolescence as predictors of cardiovascular disease risk in young adulthood. Danish Youth and Sports Study. An eight-year follow-up study. *International journal of sports medicine*, 23 Suppl 1 doi: 10.1055/s-2002-28458
- Haycock, G., Schwartz, G., & Wisotsky, D. (1978). Geometric method for measuring body surface area: a height-weight formula validated in infants, children and adults. *Journal of Paediatrics*, 93(1), 62-66.
- Hayward, C., Webb, C., & Collins, P. (2001). Effect of sex hormones on cardiac mass. *The Lancet*, 357, 1354-1356.
- He, Q., Horlick, M., Thornton, J., Wang, J., Pierson, R. N., Heshka, S., & Gallagher, D. (2004). Sex-Specific Fat Distribution Is Not Linear Across Pubertal Groups in a Multiethnic Study. *Obesity Research*, 12(4), 725-733.
- Hebestreit, H., Staschen, B., & Hebestreit, A. (2000). Ventilatory threshold: a useful method to determine aerobic fitness in children? *Medicine and Science in Sports and Exercise*, 32(11), 1964-1969. doi: 10.1097/00005768-200011000-00022
- Herrmann, S., Barreira, T., Kang, M., & Ainsworth, B. (2014). Impact of accelerometer wear time on physical activity data: a NHANES semisimulation data approach. doi: 10.1136/bjsports-2012-091410

- Hill, A., & Lupton, H. (1923). Muscular exercise, lactic acid, and the supply and utilisation of oxygen. *Quarterly Journal of Medicine*, *16*, 135-171.
- Hiort, O. (2002). Androgens and puberty. *Best Pract Res Clin Endocrinol Metab*, *16*(1), 31-41. doi: 10.1053/beem.2002.0178
- Holman, R., Carson, V., & Janssen, I. (2011). Does the Fractionalization of Daily Physical Activity (Sporadic vs Bouts) Impact Cardiometabolic Risk Factors in Children and Youth?. *PloS One*, *6*(10), e25733. doi: 10.1371/journal.pone.0025733
- Hopkins, W. (2000). Measures of reliability in sports medicine and science. *Sports Medicine*, *30*, 1-15.
- Hopkins, W., Marshall, S., Batterham, A., & Hanin, J. (2009). Progressive statistics for studies in sports medicine and exercise science. *Medicine & Science in Sport & Exercise*, *41*(1), 3-12.
- Hopkins, W., Schabert, E., & Hawley, J. (2001). Reliability of power in physical performance tests. *Sports Medicine*, *31*, 211-234.
- Hurtig-Wennlof, A., Ruiz, J., Harro, M., & Sjoström, M. (2007). Cardiorespiratory fitness relates more strongly than physical activity to cardiovascular disease risk factors in healthy children and adolescents: the European Youth Heart Study. *European Journal of Preventative Cardiology* doi: <https://doi.org/10.1097/HJR.0b013e32808c67e3>
- Imboden, M. T., Harber, M. P., Whaley, M. H., Finch, W. H., Bishop, D. L., & Kaminsky, L. A. (2018). Cardiorespiratory Fitness and Mortality in Healthy Men and Women. *J Am Coll Cardiol*, *72*(19), 2283-2292. doi: 10.1016/j.jacc.2018.08.2166
- Impellizzeri, F., Rampinini, E., Castagna, C., Bishop, D., Ferrari-Bravo, D., & Tibaudi, A. (2008). Validity of a repeated-sprint test for football. *International Journal of Sports Medicine*, *29*, 889-905.
- Ingle, L., & Tolfrey, K. (2013). The variability of High Intensity Exercise tests in Pre-Pubertal Boys. *International Journal of Sports Medicine*, *34*, 1063-1069.
- Kalist, D. E., & Peng, Y. (2007). Does education matter? Major League Baseball players and longevity. *Death Stud*, *31*(7), 653-670. doi: 10.1080/07481180701405196
- Kaminsky, L. A., Arena, R., Ellingsen, O., Harber, M. P., Myers, J., Ozemek, C., & Ross, R. (2019). Cardiorespiratory fitness and cardiovascular disease - The past, present, and future. *Progress in Cardiovascular Disease*, *62*(2), 86-93. doi: 10.1016/j.pcad.2019.01.002
- Katch, V. L. (1983). Physical conditioning of children. *Journal of adolescent health care*, *3*(4), 241-246.
- Keller, K. (2019). Life Expectancy of Olympic Wrestling Champions in Comparison to the General Population. *Journal of Community Health*, *44*(1), 61-67. doi: 10.1007/s10900-018-0553-6
- Kettunen, J. A., Kujala, U. M., Kaprio, J., Backmand, H., Peltonen, M., Eriksson, J. G., & Sarna, S. (2015). All-cause and disease-specific mortality among male, former elite athletes: an average 50-year follow-up. *British Journal of Sports Medicine*, *49*(13), 893-897. doi: 10.1136/bjsports-2013-093347
- Khambete, N., Brown, B., & Smallwood, R. (2000). Movement artefact rejection in impedance pneumography using six strategically placed electrodes. *Physiological measurement*, *21*(1) doi: 10.1088/0967-3334/21/1/310



- Kinder, J., Lee, K., Thompson, H., Hicks, K., Topp, K., & Madsen, K. (2012). Validation of a hip-worn accelerometer in measuring sleep time in children. *Journal of Paediatric Nursing*, 27(2), 127-133.
- Klijjn, J., & Kloprogge, M. (1974). Movement artefact suppressor during ECG monitoring. *Cardiovascular research*, 8(1) doi: 10.1093/cvr/8.1.149
- Kobayashi, K., Kitamura, K., Miura, M., Sodeyama, H., Murase, Y., Miyashita, M., & Matsui, H. (1978). Aerobic Power as Related to Body Growth and Training in Japanese Boys - Longitudinal Study. *Journal of Applied Physiology*, 44(5), 666-672.
- Kontro, T. K., Sarna, S., Kaprio, J., & Kujala, U. M. (2018). Mortality and health-related habits in 900 Finnish former elite athletes and their brothers. *British Journal of Sports Medicine*, 52(2), 89-95. doi: 10.1136/bjsports-2017-098206
- Kordi, M., Folland, J., Goodall, S., Haralabidis, N., Maden-Wilkinson, T., Sarika Patel, T., . . . Howatson, G. (2020). Mechanical and morphological determinants of peak power output in elite cyclists. *Scandinavian journal of medicine & science in sports*, 30(2) doi: 10.1111/sms.13570
- Kristensen, P. L., Moeller, N. C., Korsholm, L., Kolle, E., Wedderkopp, N., Froberg, K., & Andersen, L. B. (2010). The association between aerobic fitness and physical activity in children and adolescents: the European youth heart study. *European Journal of Applied Physiology*, 110(2), 267-275. doi: 10.1007/s00421-010-1491-x
- Kubicek, W., Karnegis, J., Patterson, R., Witsoe, D., & Mattson, R. (1966). Development and evaluation of an impedance cardiac output system. *Aerospace medicine*, 37(12)
- Kujala, U., Kaprio, J., Taimela, S., & Sarna, S. (1994a). Prevalence of Diabetes, Hypertension, and Ischemic-Heart-Disease in Former Elite Athletes. *Metabolism & Clinical and Experimental*, 43(10), 1255-1260. doi: 10.1016/0026-0495(94)90219-4
- Kujala, U., Tikkanen, H., Sarna, S., Pukkala, E., Kaprio, J., & Koskenvuo, M. (2001). Disease-Specific Mortality Among Elite Athletes. *JAMA*, 285(1), 44.
- Kujala, U. M., Kaprio, J., Taimela, S., & Sarna, S. (1994b). Prevalence of Diabetes, Hypertension, and Ischemic-Heart-Disease in Former Elite Athletes. *Metabolism-Clinical and Experimental*, 43(10), 1255-1260. doi: 10.1016/0026-0495(94)90219-4
- Kuss, O., Kluttig, A., & Greiser, K. H. (2011). Longevity of soccer players: an investigation of all German internationals from 1908 to 2006. *Scandinavian Journal of Medicine & Science in Sports*, 21(6), E260-E265. doi: 10.1111/j.1600-0838.2010.01269.x
- La Mantia, A., Neidert, L., & Kluess, H. (2018). Reliability and Validity of Near-Infrared Spectroscopy Mitochondrial Capacity Measurement in Skeletal Muscle. *Journal of Functional Morphology and Kinesiology* doi: 10.3390/JFMK3020019
- Laine, M. K., Kujala, U. M., Eriksson, J. G., Wasenius, N. S., Kaprio, J., Backmand, H. M., . . . Sarna, S. (2015). Former male elite athletes and risk of hypertension in later life. *Journal of Hypertension*, 33(8), 1549-1554. doi: 10.1097/hjh.0000000000000601
- Latt, E., Maestu, J., Raask, T., Rubin, D., Purge, P., Saar, M., . . . Jurimae, T. (2013). Association of physical activity to cardiovascular fitness and fatness in 12-13

- year old boys in different weight status. *Journal of Public Health*, 21, 231-239. doi: 10.1007/s10389-012-0549-0
- Laukkanen, J., Pukkala, E., Rauramaa, R., Makikallio, T., Toriola, A., & Kurl, S. (2010). Cardiorespiratory fitness, lifestyle factors and cancer risk and mortality in Finnish men. *European Journal of Cancer*, 46, 355 - 363.
- Laukkanen, J., Zaccardi, F., Khan, H., Kurl, S., Jae, S., & Rauramaa, R. (2016). Long-term Change in Cardiorespiratory Fitness and All-Cause Mortality: A Population-Based Follow-up Study. *Mayo Clin Proc*, 91(9), 1183-1188. doi: 10.1016/j.mayocp.2016.05.014
- Laursen, P. B., & Jenkins, D. G. (2002). The Scientific Basis for High-Intensity Interval Training. *Sports Medicine*, 32(1), 53-73.
- Lawler, T., Lawler, F., Gibson, J., & Murray, R. (2012). Does the African-American-white mortality gap persist after playing professional basketball? A 59-year historical cohort study. *Ann Epidemiol*, 22(6), 406-412. doi: 10.1016/j.annepidem.2012.04.009
- Leclair, E., Borel, B., Baquet, G., Berthoin, S., Mucci, P., Thevenet, D., & Reguem, S. (2010). Reproducibility of measurement of muscle deoxygenation in children during exercise. *Pediatric exercise science*, 22(2) doi: 10.1123/pes.22.2.183
- Lee, I. M., Hsieh, C. C., & Paffenbarger Jr, R. S. (1995). Exercise intensity and longevity in men. The Harvard Alumni Health Study. *JAMA*, 273, 1179-1184.
- Lee-Heidenreich, J., Lee-Heidenreich, D., & Myers, J. (2017). Differences in life expectancy between olympic high jumpers, discus throwers, marathon and 100 meter runners. *Bmc Sports Science Medicine and Rehabilitation*, 9 doi: 10.1186/s13102-017-0067-z
- Lehman, E. J., Hein, M. J., Baron, S. L., & Gersic, C. M. (2012). Neurodegenerative causes of death among retired National Football League players. *Neurology*, 79, 1970-1974.
- Leite, S. M., Freitas, J., Campelo, M., & Maciel, M. J. (2016). Electrocardiographic evaluation in athletes: 'Normal' changes in the athlete's heart and benefits and disadvantages of screening. *Revista Portuguesa De Cardiologia*, 35(3), 169-177. doi: 10.1016/j.repc.2015.09.024
- Lemez, S., & Baker, J. (2015). Do Elite Athletes Live Longer? A Systematic Review of Mortality and Longevity in Elite Athletes. *Sports Medicine*, 1(16), 1-14.
- Lemez, S., Wattie, N., & Baker, J. (2016). Early death in active professional athletes: Trends and causes. *Scandinavian Journal of Medicine & Science in Sports*, 26(5), 544-549. doi: 10.1111/sms.12480
- Lincoln, A. E., Vogel, R. A., Allen, T. W., Dunn, R. E., Alexander, K., Kaufman, N. D., & Tucker, A. M. (2018). Risk and Causes of Death among Former National Football League Players (1986-2012). *Medicine & Science in Sport & Exercise*, 50(3), 486-493.
- Linthorne, N. P. (1994). The effect of wind on 100-m sprint times. *Journal of Applied Biomechanics*, 10, 110-131.
- Lloyd, R., Meyers, R., Hughes, M., Cronin, J., & Oliver, J. (2016). The relationship between vertical and horizontal stiffness and maximal running speed in 11 - 15 year old boys. *Journal of Strength and Conditioning Research*, 30, S99.
- Lloyd, R., & Oliver, J. (2012). The Youth Physical Development Model: A New Approach to Long-Term Athletic Development. *Strength & Conditioning Journal*, 34(3), 61-72.

- Lloyd, R., Oliver, J., Radnor, J., Rhodes, B., Faigenbaum, A., & Myer, G. (2015). Relationships between functional movement screen scores, maturation and physical performance in young soccer players. *Journal of Sport Science*, 33(1) doi: 10.1080/02640414.2014.918642
- Loftin, M., Sothorn, M., Warren, B., & Udall, J. (2004). Comparison of VO<sub>2</sub> Peak during treadmill and cycle ergometry in severely overweight youth. *Journal of Sports Science and Medicine*, 3, 254-260.
- Logan, G. R., Harris, N., Duncan, S., & Schofield, G. (2014). A review of adolescent high-intensity interval training. *Sports Medicine*, 44(8), 1071-1085.
- Lollgen, H. (2012). Recent Aspects of ECG Examination in Pre-Participation Examination: How should we Proceed in Young and Older Athletes? *Deutsche Zeitschrift Fur Sportmedizin*, 63(5), 148-151.
- Love, R., Adams, J., Atkin, A., & van Sluijs, E. (2019a). Socioeconomic and ethnic differences in children's vigorous intensity physical activity: a cross-sectional analysis of the UK Millennium Cohort Study *BMJ Open* (Vol. 9).
- Love, R., Adams, J., & van Sluijs, E. (2019b). Are school-based physical activity interventions effective and equitable? A meta-analysis of cluster randomized controlled trials with accelerometer-assessed activity. *Obesity Reviews*, 20(6), 859-870.
- Low, D., Harsley, P., Shaw, M., & Peart, D. (2015). The effect of heavy resistance exercise on repeated sprint performance in youth athletes. *Journal of Sport Science*, 33(10), 1028-1034.
- Luo, S., & Johnston, P. (2010). A review of electrocardiogram filtering. *Journal of electrocardiology*, 43(6) doi: 10.1016/j.jelectrocard.2010.07.007
- Lynch, B. A., Kaufman, T. K., Rajjo, T. I., Mohammed, K., Kumar, S., Murad, M. H., . . . Levine, J. A. (2019). Accuracy of Accelerometers for Measuring Physical Activity and Levels of Sedentary Behaviour in Children: A Systematic Review. *Journal of Primary Care & Community Health*, 10, 1 - 8.
- M, D., O, T., MK, K., C, L., J, S., P, W., & LB, A. (2006). Daily physical activity and its relation to aerobic fitness in children aged 8-11 years. *European journal of applied physiology*, 96(5) doi: 10.1007/s00421-005-0117-1
- Macfarlane, D. J., & Wong, P. (2012). Validity, reliability and stability of the portable Cortex Metamax 3B gas analysis system. *European Journal of Applied Physiology*, 112, 2539-2547.
- Mackay, D. F., Russell, E. R., Stewart, K., MacLean, J. A., Pell, J. P., & Stewart, W. (2019). Neurodegenerative Disease Mortality among Former Professional Soccer Players. *New England Journal of Medicine*, 381(19), 1801-1808. doi: 10.1056/NEJMoa1908483
- Mahon, A. (2008). Aerobic Training. In Armstrong & W. van Mechelen (Eds.), *Paediatric Exercise Science and Medicine* (pp. 513-529). Oxford University Press.
- Mahon, A., & Cheatham, C. (2002). Ventilatory threshold in children: A review. *Pediatric Exercise Science*, 14(1), 16-29.
- Mahon, A., & Vaccaro, P. (1989). Ventilatory threshold and V'O<sub>2</sub> max changes in children following endurance training. *Medicine & Science in Sport & Exercise*, 21, 425-431.
- Malina, R., Beunen, G., Lefevre, J., & Woynarowska, B. (1997). Maturity-associated variation in peak oxygen uptake in active adolescent boys and girls. *Annals of Human Biology*, 24, 19-31.

- Malina, R., & Bouchard, C. (1991). *Growth, Maturation and Physical Activity*. Champaign: IL: Human Kinetics.
- Mandigout, S., Lecoq, A. M., Courteix, D., Guenon, P., & Obert, P. (2000). The effect of gender on response to an aerobic training program in prepubertal children. *Science & Sports, 15*(2), 74-80. doi: 10.1016/s0765-1597(00)88968-6
- Mannocci, A., D'Egidio, V., Backhaus, I., Federici, A., Sinopoli, A., Varela, A., . . . La Torre, G. (2020). Are there effective interventions to increase physical activity in children and young people? An Umbrella Review. *International Journal of Environmental Research and Public Health, 17*(10), 3528.
- Mantia, A. M. L., Neidert, L. E., & Kluess, H. A. (2018). Reliability and Validity of Near-Infrared Spectroscopy Mitochondrial Capacity Measurement in Skeletal Muscle. *Journal of Functional Morphology and Kinesiology* doi: 10.3390/JFMK3020019
- Marijon, E., Tafflet, M., Antero-Jacquemin, J., El Helou, N., Berthelot, G., Celermajer, D. S., . . . Jouven, X. (2013). Mortality of French participants in the Tour de France (1947-2012). *European Heart Journal, 34*(40), 3145-3150. doi: 10.1093/eurheartj/eh347
- Marshall, W., & Tanner, J. (1969). Variations in pattern of pubertal changes in girls. *Archives of Disease in Childhood, 44*(235), 291-303. doi: 10.1136/adc.44.235.291
- Marshall, W., & Tanner, J. (1970). Variations in the pattern of pubertal changes in boys. *Archives of disease in childhood, 45*(239), 13-23.
- Martin, R., Doré, E., Twisk, J., van Praagh, E., Hautier, C., & Bedu, M. (2003). Longitudinal Changes of Maximal Short-Term Peak Power in Girls and Boys during Growth. *Medicine & Science in Sport & Exercise, 36*(3), 498-503.
- Marwood, S., Roche, D., Rowland, T., Garrard, M., & Unnithan, V. B. (2010). Faster pulmonary oxygen uptake kinetics in trained versus untrained male adolescents. *Medicine & Science in Sport & Exercise, 42*(1), 127-134. doi: 10.1249/MSS.0b013e3181af20d0
- Massicotte, D., & Macnab, R. (1974). Cardiorespiratory adaptations to training at specified intensities in children. *Medicine and science in sports, 6*(4)
- Matos, N., & Winsley, R. J. (2007). Trainability of young athletes and overtraining. *Journal of Sports Science and Medicine, 6*(3), 353-367.
- Mattocks, C., Leary, S., Ness, A., Deere, K., Saunders, J., Tilling, K., . . . Riddoch, C. (2007). Calibration of an accelerometer during free-living activities in children. *International Journal of Pediatric Obesity, 2*, 218 - 226.
- McKendry, J., Breen, L., Shad, B. J., & Greig, C. A. (2018). Muscle morphology and performance in master athletes: A systematic review and meta-analyses. *Ageing Research Reviews, 45*, 62-82. doi: 10.1016/j.arr.2018.04.007
- McLellan, G., Arthur, R., & Buchan, D. S. (2018). Wear compliance, sedentary behaviour and activity in free-living children from hip-and wrist-mounted ActiGraph GT3X+ accelerometers. *Journal of Sport Sciences, 36*, 2424 - 2430.
- McManus, A., Armstrong, N., & Williams, C. (1997). Effect of training on the aerobic power and anaerobic performance of pre-pubertal girls. *Acta Paediatrica, 86*, 456-459.
- McManus, A., Cheng, C., Leung, M., Yung, T., & MacFarlane, D. (2005). Improving aerobic power in primary school boys: A comparison of continuous and interval training. *International Journal of Sports Medicine, 26*, 781-786.

- McNarry, M. (2019). Oxygen Uptake Kinetics in Youth: Characteristics, Interpretation, and Application. *Pediatric Exercise Science, 31*(2), 175-183. doi: 10.1123/pes.2018-0177
- McNarry, M., Barker, A., Lloyd, R., Buchheit, M., Williams, C., & Oliver, J. (2014a). The BASES Expert Statement on Trainability during Childhood and Adolescence. *The Sport and Exercise Scientist, 41*, 21-23.
- McNarry, M., Farr, C., Middlebrooke, A., Welford, D., Breese, B., Armstrong, N., & Barker, A. (2015). Aerobic Function and Muscle Deoxygenation Dynamics during Ramp Exercise in Children. *Medicine & Science in Sport & Exercise, 47*(9), 1877-1884. doi: 10.1249/mss.0000000000000609
- McNarry, M., & Jones, A. (2014). The influence of training status on the aerobic and anaerobic responses to exercise in children: A review. *European Journal of Sport Science, 14*(1), 57-68.
- McNarry, M., Mackintosh, K., & Stodefalke, K. (2014b). Longitudinal investigation of training status and cardiopulmonary responses in pre- and early-pubertal children. *European Journal of Applied Physiology, 114*(8), 1573-1580.
- McNarry, M., Welshman, J., & Jones, A. (2011a). Influence of training and maturity status on the cardiopulmonary responses to ramp incremental cycle and upper body exercise in girls. *Journal of Applied Physiology, 110*, 375-381.
- McNarry, M., Welsman, J., & Jones, A. (2011b). Influence of training and maturity status on the cardiopulmonary responses to ramp incremental cycle and upper body exercise in girls. *Journal of Applied Physiology, 110*, 375-381.
- McNarry, M., Welsman, J., & Jones, A. (2011c). Influence of training status and exercise modality on pulmonary O<sub>2</sub> uptake kinetics in pubertal girls. *European Journal of Applied Physiology, 111*(4), 621-631. doi: 10.1007/s00421-010-1681-6
- Meckel, Y., Machnal, O., & Eliakim, A. (2009). Relationship among repeated sprint tests, aerobic fitness, and anaerobic fitness in elite adolescent soccer players. *Journal of Strength and Conditioning Research, 23*(1), 163-169.
- Mendez-Villanueva, A., Buchheit, M., Kuitunen, S., Douglas, A., Peltola, E., & Bourdon, P. (2010). Age-related differences in acceleration, maximal running speed, and repeated sprint-performance in young soccer players. *Journal of sports science, 29*(5), 477-484.
- Mendez-Villanueva, A., Hamer, P., & Bishop, D. (2007). Fatigue responses during repeated sprints matched for initial mechanical output. *Medicine & Science in Sport and Exercise, 39*, 2219-2225.
- Menotti, A., Amici, E., Gambelli, G. C., Milazzotto, F., Bellotti, P., Capocaccia, R., & Giuli, B. (1990). Life Expectancy in Italian Track and Field Athletes. *European Journal of Epidemiology, 6*(3), 257-260.
- Merghani, A., Malhotra, A., & Sharma, S. (2016). The U-shaped relationship between exercise and cardiac morbidity. *Trends in Cardiovascular Medicine, 26*(3), 232-240. doi: 10.1016/j.tcm.2015.06.005
- Mero, A., Jaakkola, L., & Komi, P. (1990). Serum hormones and physical performance capacity in young boy athletes during a 1 year training period. *European Journal of Applied Physiology, 60*, 32-37.
- Mero, A., Komi, P. V., & Gregor, R. J. (1992). Biomechanics of Sprint Running: A Review. *Sports Medicine, 13*(6), 376-392.
- Metaxas, T. I., Mandroukas, A., Vamvakoudis, E., Kotoglou, K., Ekblom, B., & Mandroukas, K. (2014). Muscle Fiber Characteristics, Satellite Cells and

- Soccer Performance in Young Athletes. *Journal of Sports Science and Medicine*, 13(3), 493-501.
- Meyer, T., Lucia, A., Earnest, C. P., & Kindermann, W. (2005). A conceptual framework for performance diagnosis and training prescription from submaximal gas exchange parameters - Theory and application. *International Journal of Sports Medicine*, 26, S38-S48. doi: 10.1055/s-2004-830514
- Meyers, R., Oliver, J., Hughes, M., Cronin, J. B., & Lloyd, R. (2015). Maximal Sprint Speed in Boys of Increasing Maturity. *Pediatric Exercise Science*, 27(1), 85-94. doi: 10.1123/pes.2013-0096
- Meyers, R., Oliver, J., Hughes, M., Lloyd, R., & Cronin, J. (2016). The Influence of Maturation on Sprint Performance in Boys over a 21-month period. *Medicine & Science in Sport & Exercise*, 48(12), 2555-2562. doi: doi: 10.1249/MSS.0000000000001049.
- Meyers, R., Oliver, J., Hughes, M., Lloyd, R., & Cronin, J. (2017a). Influence of Age, Maturity, and Body Size on the Spatiotemporal Determinants of Maximal Sprint Speed in Boys. *Journal of Strength and Conditioning Research*, 31, 1009 - 1016.
- Meyers, R., Oliver, J., Hughes, M., Lloyd, R., & Cronin, J. (2017b). New Insights into the development of maximal sprint speed in male youth. *Journal of Strength and Conditioning*, 39(2), 2-10.
- Meylan, C., Cronin, J., Oliver, J., & Hughes, M. (2010). Talent Identification in Soccer: The Role of Maturity Status on Physical, Physiological and Technical Characteristics. *International Journal of Sports Science & Coaching*, 5(4), 571-592.
- Migueles, J. H., Cadenas-Sanchez, C., Ekelund, U., Nystrom, C. D., Mora-Gonzalez, J., Lof, M., . . . Ortega, F. B. (2017). Accelerometer data collection and processing criteria to assess physical activity and other outcomes: a systematic review and practical considerations. *Sports Medicine*, 47(9), 1821-1845.
- Mika, A., & Fleshner, M. (2016). Early-life exercise may promote lasting brain and metabolic health through gut bacterial metabolites. *Immunology and cell biology*, 94, 151-157.
- Mikkelsen, U. R., Coupe, C., Karlsen, A., Grosset, J. F., Schjerling, P., Mackey, A. L., . . . Kjaer, M. (2013). Life-Long endurance exercise in humans: circulating levels of inflammatory markers and leg-muscle size. *Mechanisms of ageing and development*, 134(11-12), 531-540.
- Milanović, Z., Sporiš, G., & Weston, M. (2015). Effectiveness of high-intensity interval training (HIT) and continuous endurance training for VO2max improvements: a systematic review and meta-analysis of controlled trials. *Sports Medicine*, 45(10), 1469-1481.
- Milicevic, G., Fabecic-Sabadi, V., Rudan, P., Kokos, Z., & Lukanovic, T. (1997). Sex differences in Pubertal Growth of the Heart. *American Journal of Human Biology*, 9, 297-302.
- Mintjens, S., Menting, M. D., Daams, J. G., Poppel, M. N. M. v., Roseboom, T. J., & Gemke, R. J. B. J. (2018). Cardiorespiratory Fitness in Childhood and Adolescence Affects Future Cardiovascular Risk Factors: A Systematic Review of Longitudinal Studies. [ReviewPaper]. *Sports Medicine*, 48(11), 2577-2605. doi: doi:10.1007/s40279-018-0974-5

- Mirwald, R., Bailey, D., Cameron, N., & Rasmussen, R. (1981). Longitudinal comparison of aerobic power in active and inactive boys aged 7 to 17 years. *Annals of Human Biology*, 8(5), 405-414.
- Mirwald, R., Baxter-Jones, A., Bailey, D., & Beunen, G. (2002a). An assessment of maturity from anthropometric measurements. *Medicine and Science in Sport & Exercise Science*, 34(4), 689-694.
- Moher, D., Shamseer, L., Clarke, M., Ghersi, D., Liberati, A., Petticrew, M., . . . Group, P.-P. (2015). Preferred Reporting Items for Systematic Review and Meta-Analysis Protocols (PRISMA-P) 2015 Statement. *Systematic Review*, 4(1), 1-9. doi: doi: 10.1186/2046-4053-4-1
- Mohlenkamp, S., Lehman, N., & Breuckmann, F. (2008). Running the risk of coronary events: Prevalence and prognostic relevance of coronary atherosclerosis in marathon runners. *European Heart Journal*, 29(15), 1903-1910.
- Moran, J., Sandercock, G., Rumpf, M. C., & Parry, D. A. (2016). Variation in Responses to Sprint Training in Male Youth Athletes: A Meta-analysis *International Journal of Sports Medicine*, 38(1), 1-11.
- Morin, J. B. (2018). A Simple Method for Measuring Force, Velocity and Power Capabilities and Mechanical Effectiveness During Sprint Running. In P. Samozino (Ed.), *Biomechanics of Training and Testing: Innovative Concepts and Simple Field Methods*. (pp. 237-269): Springer.
- Morin, J. B., Edouard, P., & Samozino, P. (2011). Technical ability of force application as a determinant factor of sprint performance. *Medicine and Science in Sport & Exercise*, 43(9), 1680-1688.
- Morin, J. B., Jeannin, T., Chevallier, B., & Belli, A. (2006). Spring-mass model characteristics during sprint running: correlation with performance and fatigue-induced changes. *International Journal of Sports Medicine*, 27(1), 158-165.
- Moro, T., Bianco, A., Faigenbaum, A. D., & Paoli, A. (2014). Pediatric Resistance Training: Current Issues and Concerns. *Minerva Pediatrica*, 66(3), 217-227.
- Morris, J. A., & Gardner, M. J. (1988). Calculating confidence intervals for relative risks (odds ratios) and standardised ratios and rates. *British Medical Journal*, 296(6632), 1313-1316.
- Mostafavifar, A. M., Best, T. M., & Myer, G. D. (2012). Early sport specialisation, does it lead to long-term problems? *British Journal of Sports Medicine*, 1-2.
- Moulinier, L., Venet, T., Schiller, N., Kurtz, T., Morris, R., & Sebastian, A. (1991). Measurement of Aortic Blood Flow by Doppler Echocardiography: Day to Day Variability in Normal Subjects and Applicability in Clinical Research. *Journal of the American College of Cardiology*, 17(6) doi: 10.1016/s0735-1097(10)80143-3
- Mountjoy, M. (2008). IOC consensus statement on training the elite child athlete. *Science & Sports*, 23(2), 98-101. doi: 10.1016/j.scispo.2008.02.004
- Mucci, P., Baquet, G., Nourry, C., Deruelle, F., Berthoin, S., & Fabre, C. (2013). Exercise Testing in Children: Comparison in Ventilatory Thresholds Changes with Interval-Training. *Pediatric Pulmonology*, 48(8), 809-816. doi: 10.1002/ppul.22646
- Mujika, I., Spencer, M., Santisteban, J., Gioriena, J. J., & Bishop, D. (2009). Age-related differences in repeated-sprint ability in highly trained youth football players. *Journal of Sport Sciences*, 27(14), 1581-1590.

- Nagahara, R., Haramura, M., Takai, Y., Oliver, J., Wichitaksorn, N., Sommerfield, L., & Cronin, J. (2019). Age-related differences in kinematics and kinetics of sprinting in young females. *Scandinavian Journal of Medicine & Science in Sports* doi: DOI: 10.1111/sms.13397
- Nagai, S., Anzai, D., & Wang, J. (2017). Motion artefact removals for wearable ECG using stationary wavelet transform. *Healthcare technology letters*, 4(4) doi: 10.1049/htl.2016.0100
- Naughton, G., Carlson, J., & Fairweather, I. (1992). Determining the variability of performance on Wingate anaerobic tests in children aged 6 - 12 years old. *International Journal of Sports Medicine*, 13(512-517)
- Nevill, A., Bate, S., & Holder, R. (2006). Modelling physiological and anthropometric variables known to vary with body size and other confounding variables. *American Journal of Physical Anthropology*, 48, 141-153.
- Nevill, A., Holder, R., Baxter-Jones, A., Round, J., & Jones, D. (1998). Modeling developmental changes in strength and aerobic power in children. *Journal of Applied Physiology* (1985), 84(3), 963-970. doi: 10.1152/jappl.1998.84.3.963
- Nguyen, V. T., Zafonte, R. D., Kponee-Shovein, K. Z., Paganoni, S., & Weisskopf, M. G. (2019). All-Cause and Cause-Specific Mortality Among Major League Baseball Players. *JAMA Intern Med* doi: 10.1001/jamainternmed.2019.1218
- Niemi, A. K., & Majamaa, K. (2005). Mitochondrial DNA and ACTN3 genotypes in Finnish elite endurance and sprint athletes. *European Journal of Human Genetics*, 13(8), 965-969. doi: 10.1038/sj.ejhg.5201438
- Nottin, S., Vinet, A., Stecken, F., N'Guyen, L. D., Ounissi, F., Lecoq, A. M., & Obert, P. (2002). Central and peripheral cardiovascular adaptations to exercise in endurance-trained children. *Acta Physiologica Scandinavica*, 175(2), 85-92. doi: 10.1046/j.1365-201X.2002.00975.x
- Nystoriak, M. A., & Bhatnagar, A. (2018). Cardiovascular Effects and Benefits of Exercise. *Frontiers in Cardiovascular Medicine*, 135(5) doi: doi: 10.3389/fcvm.2018.00135
- O'Keefe, J., Lavie, C., & Guazzi, M. (2015). Part 1: Potential Dangers of Extreme Endurance Exercise: How Much Is Too Much? Part 2: Screening of School-Age Athletes. *Progress in Cardiovascular Diseases*, 57(4), 396-405. doi: 10.1016/j.pcad.2014.11.004
- O'Keefe, J., O'Keefe, E., & Lavie, C. (2018). The Goldilocks Zone for Exercise: Not Too Little, Not Too Much. *Missouri Medicine*, 115(2), 98 - 105.
- O'Keefe, J., Patil, H., Lavie, C., Magalski, A., Vogel, R., & McCullough, P. (2012). Potential Adverse Cardiovascular Effects From Excessive Endurance Exercise. *Mayo Clinic Proceedings*, 87(6), 587-595. doi: 10.1016/j.mayocp.2012.04.005
- Obert, P., Cleuziou, C., Candau, R., Courteix, D., Lecoq, A., & Guenon, P. (2000). The slow component of O<sub>2</sub> uptake kinetics during high-intensity exercise in trained and untrained prepubertal children. *International Journal of Sports Medicine*, 21(1), 31-36. doi: 10.1055/s-2000-8856
- Obert, P., Mandigout, S., Vinet, A., Nottin, S., N'Guyen, L., & Lecoq, A. (2005). Relationships between left ventricular morphology, diastolic function and oxygen carrying capacity and maximal oxygen uptake in children. *International journal of sports medicine*, 26(2) doi: 10.1055/s-2004-817857
- Obert, P., Mandigouts, S., Nottin, S., Vinet, A., N'Guyen, L., & Lecoq, A. (2003). Cardiovascular responses to endurance training in children: effect of gender.



- European Journal of Clinical Investigation*, 33(3), 199-208. doi: 10.1046/j.1365-2362.2003.01118.x
- Odman, S., & Oberg, P. A. (1982). Movement-induced potentials in surface electrodes. *Medical & biological engineering & computing*, 20(2) doi: 10.1007/BF02441351
- Oliver, J., Williams, C., & Armstrong, N. (2006). Reliability of a Field and Laboratory Test of Repeated Sprint Ability. *Paediatric Exercise Science*, 18, 339-350
- Ottevaere, C., Huybrechts, I., De Bourdeaudhuij, I., Sjostrom, M., Ruiz, J. R., Ortega, F. B., . . . De Hanauw, S. (2011). Comparison of the IPAQ-A and ActiGraph in relation to VO<sub>2</sub> max among European adolescents: The HELENA study. *Science and Medicine in Sport*, 14, 317 - 324.
- Ouzzani, M., Hammady, H., Fedorowicz, Z., & Elmagarmid, A. (2016). Rayyan - a web and mobile app for systematic reviews. *Systematic Reviews*, 5(210) doi: DOI: 10.1186/s13643-016-0384-4.
- Paffenbarger, R. S., & Lee, I. M. (1998). A natural history of athleticism, health and longevity. *Journal of Sports Sciences*, 16, S31-S45.
- Papaiakovou, G., Giannakos, A., Michailidis, C., Patikas, D., Bassa, E., Kalopisis, V., . . . Kotzamanidis, C. (2009). The effect of chronological age and gender on the development of sprint performance during childhood and puberty. *Journal of Strength & Conditioning Research*, 23(9), 2568-2573. doi: 10.1519/JSC.0b013e3181c0d8ec
- Paterson, D. H., McLellan, T. M., Stella, R. S., & Cunningham, D. A. (1987). Longitudinal study of ventilation threshold and maximal O<sub>2</sub> uptake in athletic boys. *Journal of Applied Physiology* (1985), 62(5), 2051-2057. doi: 10.1152/jappl.1987.62.5.2051
- Pearson, K. (1896). Mathematical contributions to the theory of evolution on a form of spurious correlation which may arise when indices are used in the measurement of organs. *Product of the Royal Society London*, 60, 489-498.
- Pene, P., & Touitou, Y. (2009). Sport and health. *Bulletin De L Academie Nationale De Medecine*, 193(2), 415-429.
- Pfitzinger, P., & Freedson, P. (1997). Blood lactate responses to exercise in children .2. Lactate threshold. *Pediatric Exercise Science*, 9(4), 299-307.
- Philippaerts, R. M., Vaeyens, R., Janssens, M., van Renterghem, B., Matthys, D., Craen, R., . . . Malina, R. M. (2006). The relationship between peak height velocity and physical performance in youth soccer players. *Journal of Sport Science*, 24(3), 221-230.
- Plasqui, G., & Westerterp, K. (2007). Physical activity assessment with accelerometers: an evaluation against doubly labeled water. *Obesity (Silver Spring, Md.)*, 15(10) doi: 10.1038/oby.2007.281
- Pollock, R. D., Duggal, N. A., Lazarus, N. R., Lord, J. M., & Harridge, S. D. R. (2018). Cardiorespiratory fitness not sedentary time or physical activity is associated with cardiometabolic risk in active older adults. *Scandinavian Journal of Medicine & Science in Sports*, 28(6), 1653-1660. doi: 10.1111/sms.13071
- Poole, D., & Jones, A. (2017). Measurement of the maximum oxygen uptake  $\dot{V}O_{2max}$ :  $\dot{V}O_{2peak}$  is no longer acceptable. *Journal of applied physiology (Bethesda, Md. : 1985)*, 122(4) doi: 10.1152/jappphysiol.01063.2016
- Pukkala, E., Kaprio, J., Koskenvuo, M., Kujala, U., & Sarna, S. (2000). Cancer incidence among Finnish world class male athletes. *International Journal of Sports Medicine*, 21(3), 216-220. doi: 10.1055/s-2000-8878

- Puyau, M. R., Adolph, A. L., Vohra, F. A., & Butte, N. F. (2002). Validation and calibration of physical activity monitors in children. *Obesity Research*, *43*, 1360 - 1368.
- Quatman-Yates, C., Quatman, C., Meszaros, A., Paterno, M., & Hewett, T. (2012). A systematic review of sensorimotor function during adolescence: a developmental stage of increased motor awkwardness? *British Journal of Sports Medicine*, *46*, 649-655. doi: doi:10.1136/bjsm.2010.079616
- Rabita, G., Dorel, S., Slawinski, J., Saez-de-Villarreal, E., Courtier, A., Samozino, P., & Morin, J. (2015). Sprint mechanics in world class athletes: a new insight into the limits of human locomotion. *Scandinavian Journal of Medicine in Sport & Exercise*, *26*, 583-594.
- Radonic, V., Kozmar, D., Pocanic, D., Jerkic, H., Bohacek, I., & Letilovic, T. (2017). Mortality and causes of death among Croatian male Olympic medalists. *Croat Med J*, *58*(4), 263-269. doi: 10.3325/cmj.2017.58.263
- Ratel, S., Duche, P., Hennegrave, A., Van Praagh, E., & Bedu, M. (2002). Acid-base balance during repeated cycling sprints in boys and men. [Article]. *Journal of Applied Physiology*, *92*(2), 479-485.
- Ratel, S., Duche, P., & Williams, C. (2006a). Muscle fatigue during high-intensity exercise in children. [Review]. *Sports Medicine*, *36*(12), 1031-1065. doi: 10.2165/00007256-200636120-00004
- Ratel, S., & Martin, V. (2012). Lactic anaerobic exercise in children: The end of a belief? *Science & Sports*, *27*(4), 195-200. doi: 10.1016/j.scispo.2011.08.004
- Ratel, S., Tonson, A., Cozzone, P., & Bendahan, D. (2010). Do oxidative and anaerobic energy production in exercising muscle change throughout growth and maturation? *Journal of Applied Physiology*, *109*(5), 1562-1564. doi: 10.1152/jappphysiol.00167.2010
- Ratel, S., Tonson, A., Le Fur, Y., Cozzone, P., & Bendahan, D. (2008). Comparative analysis of skeletal muscle oxidative capacity in children and adults: a (31)P-MRS study. [Article]. *Applied Physiology Nutrition and Metabolism-Physiologie Appliquee Nutrition Et Metabolisme*, *33*(4), 720-727. doi: 10.1139/H08-039
- Ratel, S., Williams, C., Oliver, J., & Armstrong, N. (2006b). Effects of age and recovery duration on performance during multiple treadmill sprints. [Article]. *International Journal of Sports Medicine*, *27*(1), 1-8. doi: 10.1055/s-2005-837501
- Reed, S., & Meggs, J. (2017). Examining the effect of prenatal testosterone and aggression on sporting choice and sporting longevity. *Personality and Individual Differences*, *116*, 11-15. doi: 10.1016/j.paid.2017.04.022
- Reybrouck, T. M., Weymans, H., Stijns, J., Knops, & Van der Hauwaert, L. (1985). Ventilatory anaerobic threshold in healthy children: age and sex differences. *European Journal of Applied Physiology*, *54*, 278-284.
- Rich, C., Geraci, M., Griffiths, L., Sera, F., Dezateux, C., & Cortina-Borja, M. (2013). Quality Control Methods in Accelerometer Data Processing: Defining Minimum Wear Time. *PloS one*, *8*(6) doi: 10.1371/journal.pone.0067206
- Riner, W., & Sellhorst, S. (2013). Physical activity and exercise in children with chronic health conditions. *Journal of Sport and Health Science*, *2*(1), 12-20.
- Roemmich, J., Clark, P., Mai, V., Berr, S., Weltman, A., Veldhuis, J., & Rogol, A. (1998). Alterations in growth and body composition during puberty: III. Influence of maturation, gender, body composition, fat distribution, aerobic

- fitness, and energy expenditure on nocturnal growth hormone release. *J Clin Endocrinol Metab*, 83(5), 1440-1447. doi: 10.1210/jcem.83.5.4760
- Roemmich, J., Clark, P., Walter, K., Patrie, J., Weltman, A., & Rogol, A. (2000). Pubertal alterations in growth and body composition. V. Energy expenditure, adiposity, and fat distribution. *Am J Physiol Endocrinol Metab*, 279(6), E1426-1436. doi: 10.1152/ajpendo.2000.279.6.E1426
- Roemmich, J., Richmond, E., & Rogol, A. (2001). Consequences of sport training during puberty. *Journal of Endocrinological Investigation*, 24(9), 708-715.
- Rogol, A. (1994). Growth at Puberty - Interaction Of Androgens and Growth-Hormone. *Medicine and Science in Sports and Exercise*, 26(6), 767-770. doi: 10.1249/00005768-199406000-00017
- Rogol, A. (2002). Androgens and puberty. *Molecular & Cellular Endocrinology*, 198(1-2), 25-29. doi: 10.1016/s0303-7207(02)00365-9
- Rogol, A., Roemmich, J., & Clark, P. (2002). Growth at puberty. *J Adolesc Health*, 31(6 Suppl), 192-200. doi: 10.1016/s1054-139x(02)00485-8
- Rommers, N., Mostaert, M., Goossens, L., Vaeyens, R., Witvrouw, W., Lenior, M., & D'Hnodt, E. (2018). Age and maturity related differences in motor coordination among male elite youth soccer players. *Journal of Sports Sciences* doi: DOI:10.1080/02640414.2018.1488454
- Rosdahl, H., Gullstrand, L., Sailer-Eriksson, J., Johansson, P., & Schantz, P. (2010). Evaluation of the Oxycon Mobile metabolic system against the Douglas bad method. *European Journal of Applied Physiology*, 109, 159-171.
- Roser, M., & Ritchie, H. (2019). *Cancer. Our World in Data* Retrieved 03.12.2019, 2019, from <https://ourworldindata.org/cancer>
- Rosin, B. (2017). Is marathon running toxic? An observational study of cardiovascular disease prevalence and longevity in 54 male marathon runners. *Physician and Sportsmedicine*, 45(2), 105-109. doi: 10.1080/00913847.2017.1288545
- Rossi, J., Slotala, R., Samozino, P., Morin, J. B., & Edouard, P. (2017). Sprint acceleration mechanics changes from children to adolescent. *Computer Methods in Biomechanics and Biomedical Engineering*, 20, 181-182.
- Rossiter, H., Kowalchuk, J., & Whipp, B. (1985). A test to establish maximal O2 uptake despite no plateau in the O2 uptake response to ramp incremental exercise. *Journal of Applied Physiology*, 100(3), 764-770.
- Rowland, T. (1997). The "Trigger Hypothesis" for Aerobic Trainability: A 14-Year Follow-Up. *Pediatric Exercise Science*, 9, 1-9.
- Rowland, T., & Boyajian, A. (1995). Aerobic Response to Endurance Exercise Training in Children. *Pediatrics*, 96(4), 654-658.
- Rowland, T., Goff, D., Martel, L., & Ferrone, L. (2000a). Influence of Cardiac Functional Capacity on Gender Differences in Maximal Oxygen Uptake in Children. *Chest*, 117, 629-635.
- Rowland, T., & Green, G. (1988). Physiological responses to treadmill exercise in females: Child-adult differences. *Medicine & Science in Sport & Exercise*, 20, 474-478.
- Rowland, T., Lee, N., & Cunningham, D. (1992). Oxygen Uptake Plateau during Maximal Treadmill Exercise in Children. *Chest*, 101, 485-489.
- Rowland, T., Miller, K., Vanderburgh, P., Goff, D., Martel, L., & Ferrone, L. (2000b). Cardiovascular fitness in premenarcheal girls and young women. *International Journal of Sports Medicine*, 21(2), 117-121.

- Rowland, T., & Obert, P. (2012). Doppler Echocardiography for the Estimation of Cardiac Output with Exercise. [OriginalPaper]. *Sports Medicine*, 32(15), 973-986. doi: doi:10.2165/00007256-200232150-00002
- Rowland, T., Popowski, B., & Ferrone, L. (1997a). Cardiac responses to maximal upright maximal cycle exercise in healthy boys and men. *Medicine & Science in Sport & Exercise*, 29(9), 1146-1151.
- Rowland, T., & Unnithan, V. (2013). Stroke Volume Dynamics During Progressive Exercise in Healthy Adolescents. *Paediatric Exercise Science*, 25, 173-185.
- Rowland, T., Vanderburgh, P., & Cunningham, L. (1997b). Body Size and the Growth of Maximal Aerobic Power in Children: A longitudinal Analysis. *Paediatric Exercise Science*, 9, 262-274.
- Rowlands, A., Edwardson, C., Davies, M., Khunti, K., Harrington, D., & Yates, T. (2018a). Beyond Cut Points: Accelerometer Metrics that Capture the Physical Activity Profile. *Medicine & Science in Sport & Exercise*, 50(6), 1323-1332. doi: 10.1249/mss.0000000000001561
- Rowlands, A., & Eston, R. (2007). The measurement and interpretation of Children's Physical Activity. *Journal of Sports Science & Medicine*, 6(3), 270-276.
- Rowlands, A. V., Edwardson, C. L., Davies, M. J., Khunti, K., Harrington, D. M., & Yates, T. (2018b). Beyond Cut Points: Accelerometer Metrics that Capture the Physical Activity Profile. *Medicine & Science in Sport & Exercise*, 50(6), 1323-1332. doi: 10.1249/mss.0000000000001561
- Ruiz, J. R., Moran, M., Arenas, J., & Lucia, A. (2011). Strenuous endurance exercise improves life expectancy: it's in our genes *Br J Sports Med* (Vol. 45, pp. 159-161). England.
- Rumpf, M., Cronin, J., Mohamad, I., Mohamad, S., Oliver, J., & Hughes, M. (2015a). The effect of resisted sprint training on maximum sprint kinetics and kinematics in youth. *European Journal of Sport Science*, 15(5), 374-381.
- Rumpf, M., Cronin, J., Oliver, J., & Hughes, M. (2013). Vertical and leg stiffness and stretch shortening cycle changes across maturation during maximal sprint running. *Human Movement Science*, 32(4), 668-676.
- Rumpf, M., Cronin, J., Oliver, J., & Hughes, M. (2015b). Kinematics and kinetics of maximum running speed in youth across maturity. *Pediatric Exercise Science*, 27, 277-284.
- Rumpf, M., Cronin, J., Pinder, S., Oliver, J., & Hughes, M. (2012). Effect of Different Training Methods on Running Sprint Times in Male Youth. *Pediatric Exercise Science*, 24, 170-186.
- Runacres, A., Bezodis, N., Mackintosh, K., & McNarry, M. (2019a). The reliability of force-velocity-power profiling during over-ground sprinting in children and adolescents. *Journal of Sport Sciences*, 1 - 7.
- Runacres, A., Mackintosh, K., & McNarry, M. (2019b). The Effect of Constant-Intensity Endurance Training and High-Intensity Interval Training on Aerobic and Anaerobic Parameters in Youth. *Journal of Sports Sciences*, 37(21) doi: 10.1080/02640414.2019.1644890
- Ryan, T., Southern, W., Reynolds, M., & McCully, K. (2013). A Cross-Validation of Near-Infrared Spectroscopy Measurements of Skeletal Muscle Oxidative Capacity With Phosphorus Magnetic Resonance Spectroscopy. *Journal of applied physiology (Bethesda, Md. : 1985)*, 115(12) doi: 10.1152/jappphysiol.00835.2013

- Ratel, S. & Blazevich, V. (2017). Are Prepubertal Children Metabolically Comparable to Well-Trained Adult Endurance Athletes? *Sports medicine (Auckland, N.Z.)*, 47(8) doi: 10.1007/s40279-016-0671-1
- Sabato, T. M., Walch, T. J., & Caine, D. J. (2016). The elite young athlete: strategies to ensure physical and emotional health. *Open Access Journal of Sports Medicine*, 7 doi: 10.2147/OAJSM.S96821
- Sadeh, A., Sharkey, K., & Carskadon, M. (1994a). Activity-based sleep-wake identification: an empirical test of methodological. *Sleep*, 17(3), 201-207. doi: 10.1093/sleep/17.3.201.
- Sadeh, A., Sharkey, K., & Carskadon, M. (1994b). Activity-based sleep-wake identification: an empirical test of methodological issues. *Sleep*, 17(3), 201-207. doi: 10.1093/sleep/17.3.201.
- Saint Onge, J. M., Rogers, R. G., & Krueger, P. M. (2008). Major League Baseball Players' Life Expectancies\*. *Soc Sci Q*, 89(3), 817-830. doi: 10.1111/j.1540-6237.2008.00562.x
- Sako, T., Hamaoka, T., Higuchi, H., Kurosawa, Y., & Katsumura, T. (2001). Validity of NIR Spectroscopy for Quantitatively Measuring Muscle Oxidative Metabolic Rate in Exercise. *Journal of applied physiology (Bethesda, Md. : 1985)*, 90(1) doi: 10.1152/jappl.2001.90.1.338
- Samozino, P. (2018). A Simple Method for Measuring Force Velocity and Power Capabilities and Mechanical Effectiveness During Sprint Running. In J. B. Morin (Ed.), *Biomechanics of Training and Testing: Innovative Concepts and Simple Field Methods* (pp. 237-269): Springer.
- Samozino, P., Rabita, G., Dorel, S., Slawinski, J., Peyrot, N., Saez de Villarreal, E., & Morin, J. B. (2016). A simple method for measuring power, force, velocity properties, and mechanical effectiveness in sprint running. *Scandinavian journal of medicine & science in sports*, 26(6), 648-658.
- Sanchis-Gomar, F., Olaso-Gonzalez, G., Corella, D., Gomez-Cabrera, M. C., & Vina, J. (2011). Increased Average Longevity among the "Tour de France" Cyclists. *International Journal of Sports Medicine*, 32(8), 644-647. doi: 10.1055/s-0031-1271711
- Sanderson, D. (1991). The influence of cadence and power output on the biomechanics of force application during steady state exercise in competitive and recreational cyclists. *Journal of Sport Science*, 9(191-203)
- Sarna, S., Kaprio, J., Kujala, U., & Koskenvuo, M. (1997). Health Status of former elite athletes. The Finnish experience. *Aging Clinical and Experimental Research*, 9, 35-41.
- Sarna, S., Sahi, T., Koskenvuo, M., & Kaprio, J. (1993). Increased life expectancy of world class male-athletes. *Medicine and Science in Sports and Exercise*, 25(2), 237-244.
- Schaun, G. Z. (2017). The Maximal Oxygen Uptake Verification Phase: a Light at the End of the Tunnel? *Sports Med Open* (Vol. 3).
- Schnohr, P. (1971a). Longevity and causes of death in male athletic champions. *Lancet*, 2(7738), 1364-1366. doi: 10.1016/s0140-6736(71)92377-4
- Schnohr, P. (1971b). Longevity and causes of death in male athletic champions. *The Lancet*, 2(7738), 1364-1366.
- Schnohr, P., O'Keefe, J. H., Marott, J. L., Lange, P., & Jensen, G. B. (2015). Dose of Jogging and Long-Term Mortality: The Copenhagen City Heart Study. *Journal of the American College of Cardiology*, 65(5), 411-419.

- Schutte, N., Nederend, I., Hudziak, J., de Geus, E., & Bartels, M. (2016). Differences in Adolescent Physical Fitness: A Multivariate Approach and Meta-analysis. *Behavioural Genetics*, *46*, 217-227.
- Schutz, Y., Weinsier, R., & Hunter, G. (2001). Assessment of Free-Living Physical Activity in Humans: An Overview of Currently available and Proposed New Measures. *Obesity Research*, *9*(6), 368-379.
- Schwartz, A. L., de Heer, H. D., & Bea, J. W. (2017). Initiating Exercise Interventions to Promote Wellness in Cancer Patients and Survivors. *Oncology-New York*, *31*(10), 711-717.
- Scott, J. J., Rowlands, A. V., Cliff, D. V., Morgan, P. J., Plotnikoff, R. C., & Lubans, D. R. (2017). Comparability and feasibility of wrist- and hip-worn accelerometers in free free-living adolescents. *Journal of Science and Medicine in Sport*, *20*, 1101 - 1106.
- Shamseer, L., Moher, D., Clarke, M., Ghersi, D., Liberati, A., Petticrew, M., . . . Group, P.-P. (2015). Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015: elaboration and explanation. *BMJ*, *349*, g7647. doi: <https://doi.org/10.1136/bmj.g7647>
- Sharma, S., Merghani, A., & Mont, L. (2015). Exercise and the heart: the good, the bad, and the ugly. *European Heart Journal*, *36*(23), 1445-+. doi: 10.1093/eurheartj/ehv090
- Siegler, J. C., Rehman, S., Bhumireddy, G. P., Abdula, R., Klem, I., Brenner, S. J., . . . Heitner, J. F. (2011). The Accuracy of the Electrocardiogram during Exercise Stress Test Based on Heart Size *PLoS One* (Vol. 6).
- Simperingham, K., Cronin, J., Pearson, S., & Ross, A. (2017). Reliability of horizontal force-velocity-power profiling during short sprint-running accelerations using radar technology. *Sports Biomechanics*, 1-12.
- Simperingham, K., Cronin, J., & Ross, A. (2016). Advances in Sprint Acceleration Profiling for Field-Based Team-Sport Athletes: Utility, Reliability, Validity and Limitations. *Sports Medicine*, *46*, 1619-1645.
- Slawinski, J., Termoz, N., Rabita, G., Dorel, S., Morin, J., & Samozino, P. (2017). How 100-m event analyses improve our understanding of world-class men's and women's sprint performance. *Scandinavian Journal of Medicine & Science in Sports*, *27*, 45-54.
- Smith, J., Hageman, K., Harms, C., Poole, D., & Musch, T. (2017). Respiratory muscle blood flow during exercise: effect of sex and ovarian cycle. *Journal of Applied Physiology*, *122*, 918-924.
- Sormunen, J., Backmand, H. M., Sarna, S., Kujala, U. M., Kaprio, J., Dyba, T., & Pukkala, E. (2014). Lifetime physical activity and cancer incidence - A cohort study of male former elite athletes in Finland. *Journal of Science and Medicine in Sport*, *17*, 479 - 484.
- Spencer, M., Fitzsimons, M., Dawson, B., Bishop, D., & Goodman, C. (2006). Reliability of a repeated-sprint test for field-hockey. *Journal of Science and Medicine in Sport*, *9*, 181-184.
- Spencer, M., Pyne, D., & Mujika, I. (2011). Fitness determinants of repeated-sprint ability in highly trained youth football players. *Journal of Sports Physiology and Performance*, *6*(4), 497-508.
- Sperlich, B., De Marees, M., Koehler, K., Linville, J., Holmberg, H. C., & Mester, J. (2011). Effects of 5 Weeks of High-Intensity Interval Training vs. Volume

- Training in 14-Year-Old Soccer Players. *Journal of Strength and Conditioning Research*, 25(5), 1271-1278. doi: 10.1519/JSC.0b013e3181d67c38
- Sperlich, B., Zinner, C., Heilemann, I., Kjendlie, P.-K., Holmberg, H.-C., & Mester, J. (2010). High-intensity interval training improves VO<sub>2</sub>peak, maximal lactate accumulation, time trial and competition performance in 9-11 year old swimmers. *European Journal of Applied Physiology*, 110, 1029-1036.
- Sport England.(2019). *Active Lives - Children and Young People Survey Academic Year 2018/19*. Retrieved from <https://www.sportengland.org/news/active-lives-children-and-young-people-survey-academic-year-201819-report-published>
- Sport Wales.(2018). *School Sport Survey*. Retrieved from <https://www.sport.wales/content-vault/school-sports-survey-2018/>
- Spurway, N. C. (1992). Aerobic exercise, anaerobic exercise and the lactate threshold. *British Medical Bulletin*, 48(3), 569-591.
- Stoedefalke, K., Armstrong, N., Kirby, B., & Welsman, J. (2000). Effect of training on peak oxygen uptake and blood lipids in 13- to 14-year old girls. *Acta Paediatrica*, 89, 1290-1294.
- Stojmenović, T., Čurčić, D., Vukašinović-Vesić, M., Andjelković, M., Dikić, N., Kostić-Vučičević, M., . . . Živković, V. (2018). Changes in maximal oxygen uptake during growth and development in girls who actively participate in basketball and non-athletes girls: a longitudinal study. *Vojnosanitetski Pregled*, 75, 481 - 486.
- Stolik, S., Delgado, J., Pérez, A., & Anasagasti, L. (2000). Measurement of the penetration depths of red and near infrared light in human "ex vivo" tissues. *Journal of photochemistry and photobiology. B, Biology*, 57(2-3) doi: 10.1016/s1011-1344(00)00082-8
- Stratton, G., & Williams, C. (2006). Children and fitness testing. In E. Winter, A. Jones, R. Davison, P. Bromley & T. Mercer (Eds.), *Sport and Exercise Physiology Testing Guidelines* (pp. 211-223). Oxon: Routledge.
- Taioli, E. (2007). All Cause Mortality in Male Soccer Players. *European Journal of Public Health*, 17(6), 600-604.
- Tanner, J. (1949). Fallacy of per-weight and per-surface area standards and their relation to spurious correlation. *Journal of Applied Physiology*, 2, 1-15.
- Tanner, J., Whitehouse, R., Cameron, N., Marshall, W., Healy, M., & Goldstein, H. (1983). *Assessment of skeletal maturity and prediction of adult height (TW2) method*. London: Academic Press.
- Tanner, J., Whitehouse, R., Marshall, W., & Carter, B. (1975). Prediction of adult height from height, bone age, and occurrence of menarche, at ages 4 to 16 with allowance for midparent height. *Archives of Disease in Childhood*, 50(1), 14-26. doi: 10.1136/adc.50.1.14
- Tattersall, R. B., & Pyke, D. A. (1973). Growth in diabetic children: studies in identical twins. *The Lancet*, 302(7838), 1105-1109.
- Temfemo, A., Lelard, T., Carling, C., Mandengue, S., Chlif, M., & Ahmaidi, S. (2011). Feasibility and Reliability of a Repeated Sprint Test in Children Age 6 to 8 Years. *Pediatric Exercise Science*, 23, 549-559.
- Teramoto, M., & Bungum, T. J. (2010a). Mortality and longevity of elite athletes. *Journal of Science and Medicine in Sport*, 13(4), 410-416. doi: 10.1016/j.jsams.2009.04.010

- Teramoto, M., & Bungum, T. J. (2010b). Mortality and longevity of elite athletes. *Journal of Science and Medicine in Sport*, *13*, 410-416.
- Thomsen, K., Riis, B., Krabbe, S., & Christiansen, C. (1986). Testosterone regulates the haemoglobin concentration in male puberty. *Acta Paediatrica*, *75*(793-796)
- Till, K., Emmonds, S., & Jones, B. (2019). *Talent Identification*.
- Tolfrey, K., Campbell, I., & Batterham, A. (1998). Aerobic Trainability of Prepubertal Boys and Girls. *Pediatric Exercise Science*, *10*, 248-263.
- Treuth, M. S., Schmitz, K., Catellier, D. J., McMurray, R. G., Murray, D. M., Almeida, M. J., . . . Pate, R. (2004). Defining accelerometer thresholds for Activity Intensities in Adolescent Girls. *Medicine & Science in Sport & Exercise*, *36*, 1259 - 1266.
- Trost, S. (2016). State of the Art Reviews: Measurement of Physical Activity in Children and Adolescents. [review-article]. *American Journal of Lifestyle Medicine* doi: 10.1177\_1559827607301686
- Trost, S., Loprinzi, P., Moore, R., & Pfeiffer, K. (2011). Comparison of Accelerometer Cut-Points for Predicting Activity Intensity in Youth. *Medicine & Science in Sport & Exercise*, *43*, 1360-1368.
- Trost, S., Pate, R., Freedson, P., Sallis, J., & Taylor, W. (2000). Using objective physical activity measures with youth: how many days of monitoring are needed? *Medicine & Science in Sport & Exercise*, *36*, 426 - 431.
- Turley, K., Rogers, D., Harper, K., Kujawa, K., & Wilmore, J. (1995). Maximal Treadmill versus Cycle Ergometry Testing in Children: Differences, Reliability, and Variability of Responses. *7*(1), 60.
- UK Government. (2015). *2010 to 2015 government policy: sports participation. Policy of the 2010 to 2015 Conservative and Liberal Democrat coalition government*. Retrieved 14/03/2017, 2017, from <https://www.gov.uk/government/publications/2010-to-2015-government-policy-sports-participation/2010-to-2015-government-policy-sports-participation>
- Unnithan, V., White, J., Georgiou, A., Iga, J., & Drust, B. (2012). Talent identification in youth soccer. *Journal of Sport Sciences*, *30*(15), 1719-1726.
- Valente dos Santos, J., Coelho e Silva, M., Duarte, J., Figueiredo, A., Liparotti, J., Sherar, L., . . . Malina, R. (2012). Longitudinal Predictors of Aerobic Performance in Adolescent Soccer Players. *Medicina-Lithuania*, *48*(8), 410-416.
- Valente dos Santos, J., Coelho e Silva, M., Tavares, O., Brito, J., Seabra, A., Rebelo, A., . . . Malina, R. (2014). Allometric modelling of peak oxygen uptake in male soccer players of 8 - 18 years of age. *Annals of Human Biology*, 1-9. doi: DOI: 10.3109/03014460.2014.932007
- van Ingen-Schenau, G., Jacobs, R., & de Koning, J. (1991). Can cycle power predict sprint running performance? *European Journal of Applied Physiology and Occupational Physiology*, *63*(3), 255-260.
- Van Praagh, E. (2000). Development of anaerobic function during childhood and adolescence. *Pediatric Exercise Science*, *12*(2), 150-173.
- Van Praagh, E., & Doré, E. (2002). Short-Term Muscle Power During Growth and Maturation. *Sports Medicine*, *32*(11), 701-728.
- van Saase, J. L., Noteboom, W. M., & Vandenbroucke, J. P. (1990). Longevity of men capable of prolonged vigorous physical exercise: a 32 year follow up of 2259



- participants in the Dutch eleven cities ice skating tour. *Bmj*, 301(6766), 1409-1411. doi: 10.1136/bmj.301.6766.1409
- Vanderka, M., & Kampmiller, T. (2013). Kinematics of sprinting in children and youth. *N Stud Athletics*, 28(1), 35-46.
- Venckunas, T., Simonavicius, J., & Marcinkeviciene, J. E. (2016). Cardiac size of high-volume resistance trained female athletes: shaping the body but not the heart. *Physiology International*, 103(1), 105-111. doi: 10.1556/036.103.2016.1.10
- Vinet, A., Mandigout, S., Nottin, S., Nguyen, L., Lecoq, A., Courteix, D., & Obert, P. (2003). Influence of body composition, haemoglobin concentration, and cardiac size and function of gender differences in maximal oxygen uptake in pre-pubertal children. *Chest*, 124, 1494-1499.
- Vingren, J. L., Kraemer, W. J., Ratamess, N. A., Anderson, J. M., Volek, J. S., & Maresh, C. M. (2010). Testosterone Physiology in Resistance Exercise and Training. *Sports Medicine*, 40(12), 1037-1053.
- Väistö, J., Haapala, E., Viitasalo, A., Schnurr, T., Kilpeläinen, T., Karjalainen, P., . . . Lakka, T. (2019). Longitudinal associations of physical activity and sedentary time with cardiometabolic risk factors in children. *Scandinavian journal of medicine & science in sports*, 29(1) doi: 10.1111/sms.13315
- Wang, J., Thornton, J. C., Russell, M., Burastero, S., Heymsfield, S., & Pierson Jr, R. N. (1994). Asians have lower body mass index (BMI) but higher percentage body fat than do whites: comparisons of anthropometric measurements. *The American Journal of Clinical Nutrition*, 60, 23-28.
- Waterbor, J., Cole, P., Delzell, E., & Andjelkovich, D. (1988). The mortality experience of major league baseball players. *The New England Journal of Medicine*, 318, 1278-1280.
- Watt, K. K. O., Hopkins, W. G., & Snow, R. J. (2002). Reliability of performance in repeated sprint cycling tests. *Journal of Science and Medicine in Sport*, 5(4), 354-361.
- Weber, G., Kartodihardjo, W., & Klissouras, V. (1976). Growth and Physical-Training with Reference to Hereditary. *Journal of Applied Physiology*, 40(2), 211-215.
- Wells, G., Shea, B., O'Connell, D., Peterson, J., Welch, V., Losos, M., & Tugwell, P. (2019). *The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses*. Retrieved 05.12.2019, 2019, from [http://www.ohri.ca/programs/clinical\\_epidemiology/oxford.asp](http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp)
- Wells, J. (2007). Sexual dimorphism of body composition. *Best Practice & Research Clinical Endocrinology & Metabolism*, 21(3), 415-430. doi: 10.1016/j.beem.2007.04.007
- Welsman, J., & Armstrong, N. (2019). Interpreting Aerobic Fitness in Youth: The Fallacy of Ratio Scaling. *Pediatric Exercise Science*, 31(2), 184-190. doi: 10.1123/pes.2018-0141
- Welsman, J., Armstrong, N., Chedzoy, S., & Withers, S. (1996). Aerobic training in 10 year old and adult females. *Medicine and Science in Sport and Exercise*, 28(3)
- Welsman, J., Armstrong, N., & Withers, S. (1997). Responses of young girls to two modes of aerobic training. *British Journal of Sports Medicine*, 31, 139-142.
- Welsman, J., Bywater, K., Farr, C., Welford, D., & Armstrong, N. (2005). Reliability of Peak VO(2) and Maximal Cardiac Output Assessed Using Thoracic

- Bioimpedance in Children. *European journal of applied physiology*, 94(3) doi: 10.1007/s00421-004-1300-5
- Whooten, R., Kerem, L., & Stanley, T. (2019). Physical Activity in Adolescents and Children and Relationship to Metabolic Health. *Curr Opin Endocrinol Diabetes Obes*, 26(1), 25-31. doi: 10.1097/med.0000000000000455
- Wiklund, U., Karlsson, M., Ostlund, N., Berglin, L., Lindecrantz, K., Karlsson, S., & Sandsjö, L. (2007). Adaptive spatio-temporal filtering of disturbed ECGs: a multi-channel approach to heartbeat detection in smart clothing. *Medical & biological engineering & computing*, 45(6) doi: 10.1007/s11517-007-0183-0
- Willcocks, R. J., Williams, C. A., Barker, A. R., Fulford, J., & Armstrong, N. (2010). Age- and sex-related differences in muscle phosphocreatine and oxygenation kinetics during high-intensity exercise in adolescents and adults. *NMR Biomed*, 23(6), 569-577. doi: 10.1002/nbm.1495
- Williams, C. (2016). Elite Youth Sports. *Pediatric Exercise Science*, 28(1), 16-18. doi: 10.1123/pes.2016-0006
- Williams, C., Armstrong, N., & Powell, J. (2000). Aerobic responses of prepubertal boys to two modes of training. *British Journal of Sports Medicine*, 34(3), 168-173.
- Winsley, R., Fulford, J., Roberts, A., Welsman, J., & Armstrong, N. (2009). Sex difference in peak oxygen uptake in pre-pubertal children. *Journal of Science and Medicine in Sport*, 12, 647-651.
- Wolfe, A., Lee, J., & Laurson, K. (2020). Socioeconomic status and physical fitness in youth: Findings from the NHANES National Youth Fitness Survey. *Journal of sports sciences*, 38(5) doi: 10.1080/02640414.2020.1713688
- World Health Organisation. (2019). *Female life expectancy. Situation and trends* Retrieved 15.12.19, 2019, from [https://www.who.int/gho/women\\_and\\_health/mortality/situation\\_trends\\_life\\_expectancy/en/](https://www.who.int/gho/women_and_health/mortality/situation_trends_life_expectancy/en/)
- Yang, X., Telama, R., Hirvensalo, M., Viikari, J., & Riatakari, O. (2009). Sustained participation in youth sport decreases metabolic syndrome in adulthood. *International Journal of Obesity*, 33, 1219-1226. doi: 10.1038/ijo.2009.171
- Κανταράς, Π., & Kantaras, P. (2018). The reliability of the VYNTUS CPX.

# **Chapter 11**

## **Appendix**

# Chapter 11 – Appendices

## Chapters 4 and 5

### *Parental Information Sheet*

**PARENT/GUARDIAN INFORMATION SHEET**  
(Version 1.2, Date: 07/02/2019)

**Project Title:**

Long-term athlete monitoring programme

**Contact Details:**

**1. Invitation Paragraph**

Thank you for taking the time to read this information sheet which gives details about our study and hopefully provides you with the information you need to help your son/daughter decide if they want to take part. It is important to say that whether they take part is entirely up to you and your child and that it doesn't matter for other studies if you both decide not to participate.

**2. What is the purpose of the study?**

The purpose of this study is to explore what effect training has on children and adolescents as they grow and mature and whether this effect is greater at any specific age. To answer this question, we need to collect data from the same participants over many years so that we can track the changes in growth and exercise performance.

**3. Why has my child been chosen?**

Your child has been asked if they would like to take part because they are involved within a long-term athlete development program within a sport and are between 8 and 18 years of age. If your son/daughter would like to take part for just one year we would still really appreciate their participation. We will give you both the option each year to see if you would like to come back to the University to undertake these measures again.

**4. What will happen to my child if they take part?**

If they decide to take part they will be asked to attend the Exercise Physiology lab at Swansea University once a year. During this visit, which will last approximately one hour, we will measure their weight, standing height and sitting height. As young people grow and mature at very different rates, we also need to estimate maturity. This is done by entering these three things into an equation which predicts how close they are from growing the fastest. This will allow us to estimate for maturity without the use of any more invasive measures, but it is important to say you do not have to do this, it is completely up to you, and if you do not want your son/ daughter to do this that is completely fine and they can still complete the rest of the study. After this your child will be asked to take a short test of how strong their arms are by measuring their grip strength using a handgrip dynamometer, just like the one in the picture. Each test will last only a matter of seconds, but they will be asked to squeeze a handgrip dynamometer as hard as they can whilst holding it above their head and slowly lowering it down towards their side. They will be asked to do these three times on each arm with a rest in between.

After this, they will then be asked to do an incremental cycling test which starts very easy and gets harder, like pedaling up a hill. The test is stopped when they can't keep going, or they choose to stop the test which they can do whenever they want to. The test lasts approximately 10 minutes. Whilst the final stages of this test are uncomfortable, the discomfort is very short, and they will recover within minutes of completing the test. The exercise is no harder that they will do in training!

During the cycling test they will be asked to:

- Wear a face mask so we can measure the air that they breathe in and out. This mask does not make breathing any harder and they can talk through it and remove it at any time if they feel uncomfortable about wearing it.
- Have 6 small electrodes placed on the upper body so we can see how the heart works during exercise. These electrodes are just like sticky plasters.
- Have a small device stuck to their leg to measure how oxygen is used in the muscles.



After this, they will have a little cool down before we give them a monitor to wear at home. The monitor, shown on the right, will be worn for 7 days continuously, except when they bath, shower or go for a swim and will measure how much physical activity they do. A log will also be provided by the research team which we will ask them to fill in during the week they are wearing the monitor detailing things like: what time they woke up in the morning, what time they went to bed and the quality of sleep. You will be given a pre-paid and signed for envelope so when the monitoring period finishes you can easily return it to us.



You are welcome to come to the University with your child but there will always be two DBS checked adults with them at all times if you cannot attend.

#### **5. What are the possible disadvantages of taking part?**

The exercise involved in this study is hard work, but they will recover quickly when they stop exercising; it is no harder than the exercise they do at training. The physical activity monitor may be uncomfortable during the week they are asked to wear it, but they can remove it at any time they like.

#### **6. What are the possible benefits of taking part?**

You and your child will get to see how they grow and how their fitness changes from year to year! They will also be part of a one-off study that will help us understand the effect training has on young people at different ages.

#### **7. Will their taking part in the study be kept confidential?**

Yes, when your child starts the study we will give them a unique ID code so no one can identify them from their results. All of their personal information will be stored on a password protected computer and only members of the research team will be able to access their information.

#### **8. What if I have any questions?**

If you have any questions, please don't hesitate to contact us on the details provided at the top of this sheet. If you have concerns regarding the study but don't want to talk to us directly, please talk [REDACTED] who is the chair of the research ethics committee where this study was approved



### 11.1.3 Pre-Screening Medical Questionnaire

#### Children Physical Activity Readiness Questionnaire & Health Screening Consent Form

Date:

The purpose of this form is to ensure that we provide every participant with the highest level of care. There are a small number of children or adolescents who may be at risk when participating in an exercise / physical activity session. Completion of this questionnaire is mandatory, and your child cannot participate in any [redacted] exercise session until it has been submitted to a Swansea University researcher.

<b>Childs name:</b>	<b>Childs date of birth:</b>
<b>Parent / Guardian Name:</b>	<b>Current Age of Child:</b>
<b>Mobile Number:</b>	<b>Work Telephone Number:</b>
<b>Email:</b>	

#### Health Questions:

Does your child have, or has he / she ever experienced any of the following? Please tick:

	<b>Yes</b>	<b>No</b>
<b>High or Low blood pressure</b>		
<b>Elevated blood cholesterol</b>		
<b>Diabetes</b>		
<b>Chest pains brought on by physical exertion</b>		
<b>Childhood epilepsy</b>		
<b>Dizziness or fainting</b>		
<b>A bone, joint or muscle problem with arthritis</b>		
<b>Asthma or respiratory problems</b>		
<b>Any sustained injuries or illnesses</b>		
<b>Any allergies</b>		
<b>Is your child taking any medication</b>		
<b>Has your doctor ever advised your child not to exercise</b>		
<b>Is there any reason not mentioned above why any type or physical activity may not be suitable for your child</b>		

If answered 'YES' to any of the above questions, please give full details here:

Is there anything else we should know about your child that has NOT been addressed in the Health questions on this form?

If your child has any known allergies, has the researcher in charge of your session been made aware of medication you are taking and where to find this?

In the absence of a parent/guardian, I understand that my child is responsible for monitoring him or herself throughout the exercises, and should any unexpected symptoms occur, would cease participation and inform the researcher.

In the event that medical clearance must be obtained before my child's participation in an exercise session, I agree to contact the GP to obtain written permission prior to the commencement of the exercise activity, and that the permission is given to the researcher where a copy of the medical advice will be kept on file.

I understand that if my child fails to behave in a manner that is polite and social, he / she could be suspended from that activity.

**Video/Photography Consent**

I understand that occasionally my child may appear in promotional photography/video clips of [REDACTED] and that material may be used by [REDACTED]

Please tick here  if photographs are NOT permitted

By signing this form, I the parent/guardian of the aforementioned child, affirm that I have read this form in its entirety; I have answered the questions accurately and, to the best of my knowledge, will inform Swansea University of any future changes.

**I the parent/guardian of the aforementioned child give permission for him/her to participant in [REDACTED] researchers taking the exercise sessions cannot be liable for any loss of personal injury**

Parent/guardian's signature:

Date:

Please print name:

Researcher(s) signature:

Date:



### 11.1.4 Athlete Participant Information Sheet



#### **PARTICIPANT INFORMATION SHEET (Version 1.1, Date: 07/02/2019)**

**Project Title:**

Long-term athlete monitoring programme

**Contact Details:**



**1. Invitation Paragraph**

Thank you for reading this information sheet which tells you all about our study. It is important to say that whether you take part is up to you and that it doesn't matter if you decide not to.

**2. What is the purpose of the study?**

We are doing this study to see what effect training has on children and adolescents as they grow and whether this effect is greater at any age. To answer this question, we need to collect data from the same people over many years (8 years) so that we can track the changes in both growth and exercise performance. You don't have to say now that you want to come back again next year, you can decide each year if you want to come in to the University.

**3. Why have I been chosen?**

You have been asked if you would like to take part because you do at least two sports sessions per week and are aged between 8 and 18 years of age. If you decide to take part, you can withdraw at any point without any problems. If you just want to take part for one year, that is ok and we would still like you to take part.

**4. What will happen to me if I take part?**

You will be asked to come to Swansea University once a year. During this visit, which will take about one hour, we will measure how much you weigh, how tall you are when you are standing and sitting and the thickness of the skin and fat on your arms, back and stomach. Because young people grow differently, we will also give you 5 standard drawings of children at different ages and ask you to tick which is most like you. This is done on your own, in private and the scores are put in a sealed envelope which only we will open. This is often done in studies which have both children and adolescents taking part but if you do not want to do it, you can still do the rest of the study.

After this, you will be asked to try and squeeze a little device as hard as you can so we can see how strong your arms are. You will start by holding it above your head and then whilst squeezing lower it down to your side. We will do these three times on both arms to see how close you can get between each arm. You will be asked to do an incremental exercise test which starts off easy and gets harder and harder, like pedaling up a hill. The test is stopped when you can't keep going so it's up to you how long it lasts but it is normally



about 10 minutes. The last couple of minutes of this test are hard work but you will be fine again within a couple of minutes of finishing the test!

While you are cycling, we will ask you to:

- Wear a face mask so we can measure the air that you breathe in and out. This mask does not make breathing any harder and you can talk through it and remove it at any time if you feel uncomfortable about wearing it.
- Have 6 small electrodes placed on your upper body so we can see how the heart works during exercise. These electrodes are just like sticky plasters.
- Have a small device stuck to your leg so we can measure how oxygen is used in the muscles.



Finally, after 15 minutes rest, we will ask you to do another short test to check the results of the first test. This will mean cycling for 3 minutes at an easy resistance which will then increase to just above the highest work rate you got to in the test before. We will then ask you to keep cycling for as long as you can! After this, you will have a little cool down before we give you a monitor to wear at home. The monitor is like the one shown in the picture and will be worn for 7 days continuously, except when you bath, shower or go for a swim. You will also be asked to fill in a log sheet where we will ask you to write down some information including: what time you went to bed, what time you woke up, and any time you took the monitor off during the day. After 7-days you will place the accelerometer in the envelope given to you by the researchers which will then send the monitor and log sheet back to us.



#### 5. What are the possible disadvantages of taking part?

The exercise in this study is hard work but you will recover quickly when you stop exercising; it is no harder than the exercise you do at training. Fingertip blood samples may be a little sore when they are done but you will forget they even happened very quickly!

#### 6. What are the possible benefits of taking part?

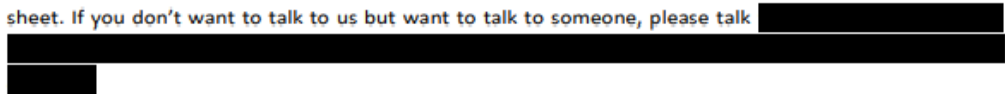
You will get to see how you grow and how your fitness changes from year to year! You will also be part of a one-off study that will really help us understand the effects of training in children and adolescents.

#### 7. Will my taking part in the study be kept confidential?

Yes, when you start the study, we will give you a one-off ID code so no one can identify you from your results. All of your personal information will be stored on a password protected computer and only members of the research team will be able to see your information.

#### 8. What if I have any questions?

If you have any questions, now or in the future, please ring or email us using the details at the top of this sheet. If you don't want to talk to us but want to talk to someone, please talk



## 11.1.5 Control Participants Information Sheet

**PARENT/GUARDIAN INFORMATION SHEET**  
(Version 1.1, Date: 01/06/2018)

**Project Title:**

**Physical Activity, Sedentary Time and Cardiovascular Health**

**Contact Details:**

[Redacted contact details]

### 1. Invitation Paragraph

Thank you for taking the time to read this information sheet which gives details about our study and hopefully provides you with the information you need to help your son/daughter decide if they want to take part. It is important to say that whether they take part is entirely up to you and your child and that it doesn't matter for other studies if you both decide not to participate.

### 2. What is the purpose of the study?

The purpose of this study is to explore what effect physical activity and sedentary behavior has on aspects of health, which can be used to inform future interventions in schools and increase the evidence base in children and adolescents that physical activity is good for us. A secondary aim is to see how children's activity varies over 1 month, and the reasons behind why some weeks are higher / lower than others.

### 3. Why has my child been chosen?

Your child have been asked if they would like to take part because they are aged between 10 and 18 years of age. We want to assess a large variety of children so we can answer this question properly and get a wide spectrum of people involved.

### 4. What will happen to my child if they take part?

If they decide to take part they will be asked to attend the [Redacted] this visit, which will last approximately two hours, we will measure their weight, standing and sitting height. As young people grow and mature at very different rates, we also need to estimate maturity, which can be done by entering these three things into an equation and will give us a value about how fast they are growing. We will then ask your child to lay down on a massage bed where we will take 3 different measures with blood pressure cuffs. Firstly, we will take a resting blood pressure measurement, then perform pulse wave analysis so we can see how the arteries respond to blood pumping through them. We will then use a second partial cuff which goes around the neck to work out pulse wave velocity (to work out how fast their blood is moving). This might feel uncomfortable for them, but it is only held on with Velcro and they can remove this at any time if they feel uncomfortable.



We will then ask your child to have a heart rate recording. This is done using a portable 3-lead ECG machine which will pick up your child's heart rate like the ones shown in the picture. We just want to see how the heart rate responds to different movements including lying down, standing up and slow breathing (like sleep). The whole protocol will last approximately 15 – 20 minutes.

They will then be asked to do an cycling test which starts very easy and gets harder, like pedaling up a hill. The test is stopped when they can't keep going. The test lasts approximately 10 minutes but can stop at any time. Whilst the final stages of this test are hard, they will recover quickly after completing the

test. The exercise is no harder that they will do in P.E in school or at a sports training session they attend!

During these tests they will be asked to:

- Wear a face mask so we can measure the air that they breathe in and out. This mask does not make breathing any harder and they can talk through it and remove it at any time if they feel uncomfortable about wearing it.
- Have 6 small electrodes placed on the upper body so we can see how the heart works during exercise. These electrodes are just like sticky plasters.
- Have a small device stuck to their leg to measure how oxygen is used in the muscles.



After this, they will have a little cool down before we give them a monitor to wear at home. The monitor, shown on the right, will be worn on the wrist like a watch for 7 days continuously as the monitor is waterproof and shock-proof. As your child is wearing this monitor, we will ask them to fill in a log sheet detailing when they woke up in the morning, if and for how long they took the monitor off for, and what time they went to bed. Your child does not have to wear the monitor when they don't want to but we would like them to try and wear it as much as possible, so we can measure how much physical activity they do in 1 month! You are welcome to come to the University with your child but there will always be two DBS checked adults with them at all times if you cannot attend. Also, please note that all procedures carried out are for research purposes only and cannot diagnose any conditions or underlying medical conditions. If we find anything that may be of concern, we will advise you and your son / daughter to visit your GP after the research session.

#### 5. What are the possible disadvantages of taking part?

The exercise involved in this study is hard work, but they will recover quickly when they stop exercising; it is no harder than the exercise they do at training. The activity monitor can also be a little bit uncomfortable, but your child can take it off whenever they want.

#### 6. What are the possible benefits of taking part?

You and your child will get to see how their physical activity level is impacting upon them and get the chance to put some of the knowledge learnt in the classroom into a practical setting and see how scientific research is conducted in the real-world. Also, you will get all of the data back so you will know all of the information that we are gathering.

#### 7. Will their taking part in the study be kept confidential?

Yes, when your child starts the study we will give them a unique ID code so no one can identify them from their results. All of their personal information will be stored on a password protected computer and only members of the research team will be able to access their information.

#### 8. What if I have any questions?

If you have any questions, please don't hesitate to contact us on the details provided at the top of this sheet. If you have concerns regarding the study but don't want to talk to us directly, please talk to [REDACTED]



*Chapter 5 – Additional Material*

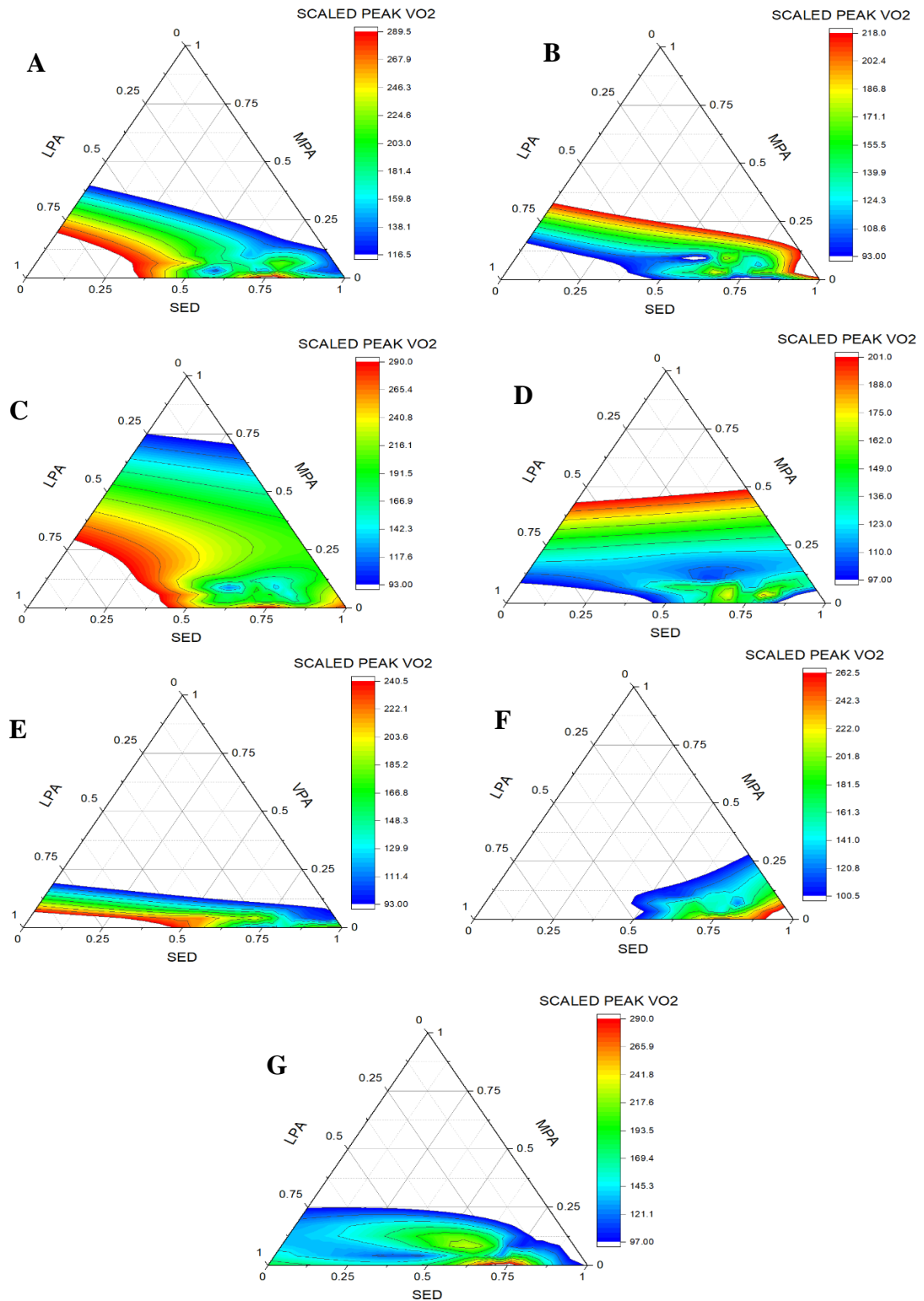
*11.2.1 Smallest worthwhile change table*

**Table 5.7** – Smallest worthwhile change in all sub-groups for peak  $\dot{V}O_2$  ( $l \cdot \text{min}^{-1}$ ) and allometrically scaled peak  $\dot{V}O_2$  ( $\text{ml} \cdot \text{kg}^{-b} \cdot \text{min}^{-1}$ )

Training Group	Maturity	Sex	Peak $\dot{V}O_2$ ( $l \cdot \text{min}^{-1}$ )		Scaled peak $\dot{V}O_2$ ( $\text{ml} \cdot \text{kg}^{-b} \cdot \text{min}^{-1}$ )	
			SWC	SWC (%)	SWC	SWC (%)
Trained	Pre-Pubertal	Boys	0.07	3.6	4.4	2.3
		Girls	0.09	4.9	4.7	3.1
	Pubertal	Boys	0.09	3.1	6.3	3.4
		Girls	0.05	2.5	3.1	2.0
	Post-Pubertal	Boys	0.14	4.4	6.8	3.3
		Girls	0.09	3.9	6.2	4.3
Untrained	Pre-Pubertal	Boys	0.06	3.0	6.9	4.9
		Girls	0.07	4.9	5.1	4.1
	Pubertal	Boys	0.09	4.1	6.9	4.3
		Girls	0.10	5.8	4.2	3.2
	Post-Pubertal	Boys	0.12	4.3	4.5	2.7
		Girls	0.08	4.1	6.8	4.8

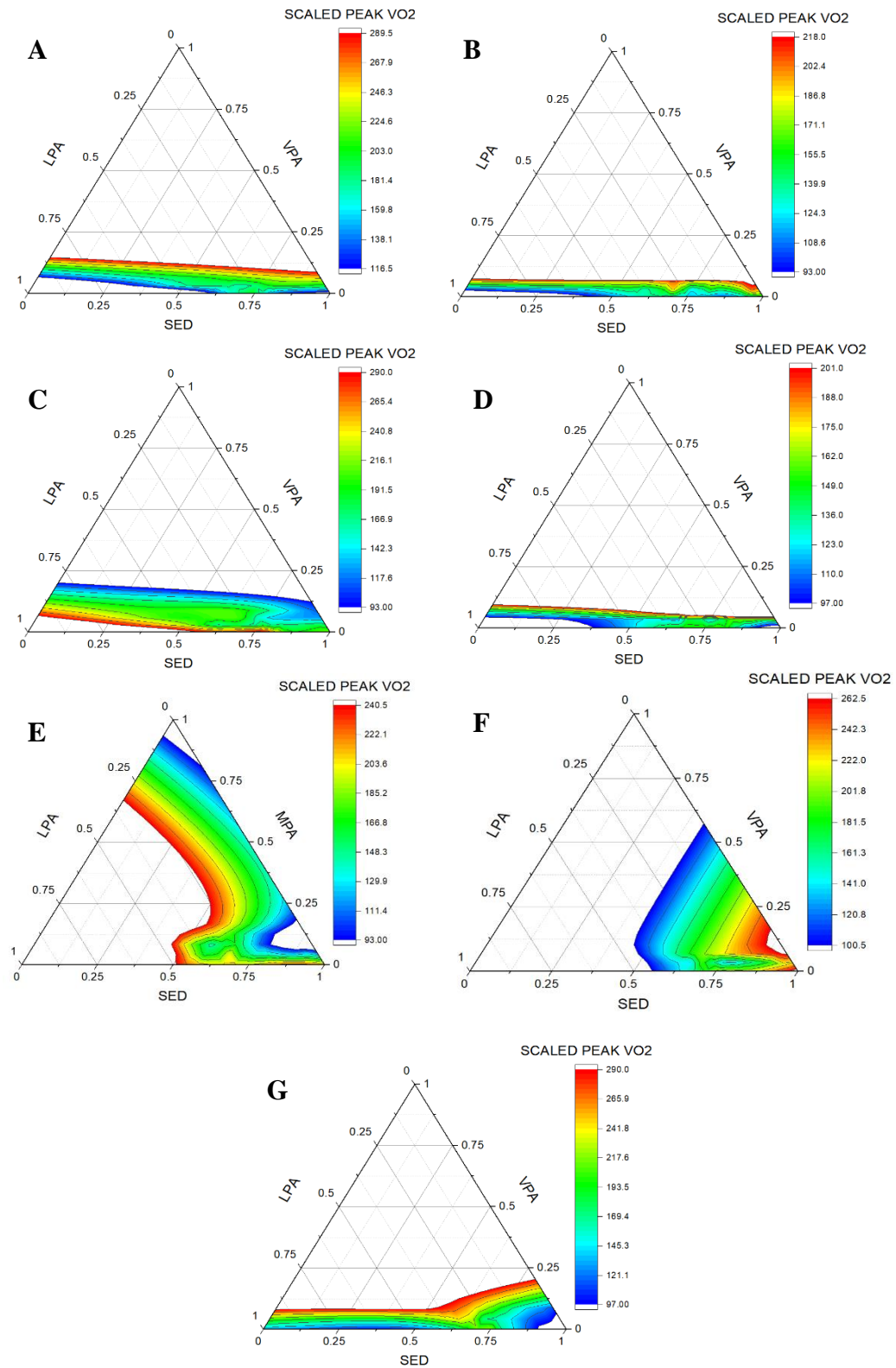
SWC = Smallest Worthwhile change, SWC (%) = Smallest worthwhile change as a percentage of the individual group mean

### 11.2.2. Ternary Plots – SED, LPA, MPA – Scaled $\dot{V}O_{2max}$



**Figure 5.3** - Ternary heat plots of all PA behaviours with expected  $\dot{V}O_{2max}$  values for all sub-groups with a) trained athletes; b) untrained participants; c) all boys; d) all girls; e) pre-pubertal children; f) pubertal adolescents; and g) post-pubertal adolescents

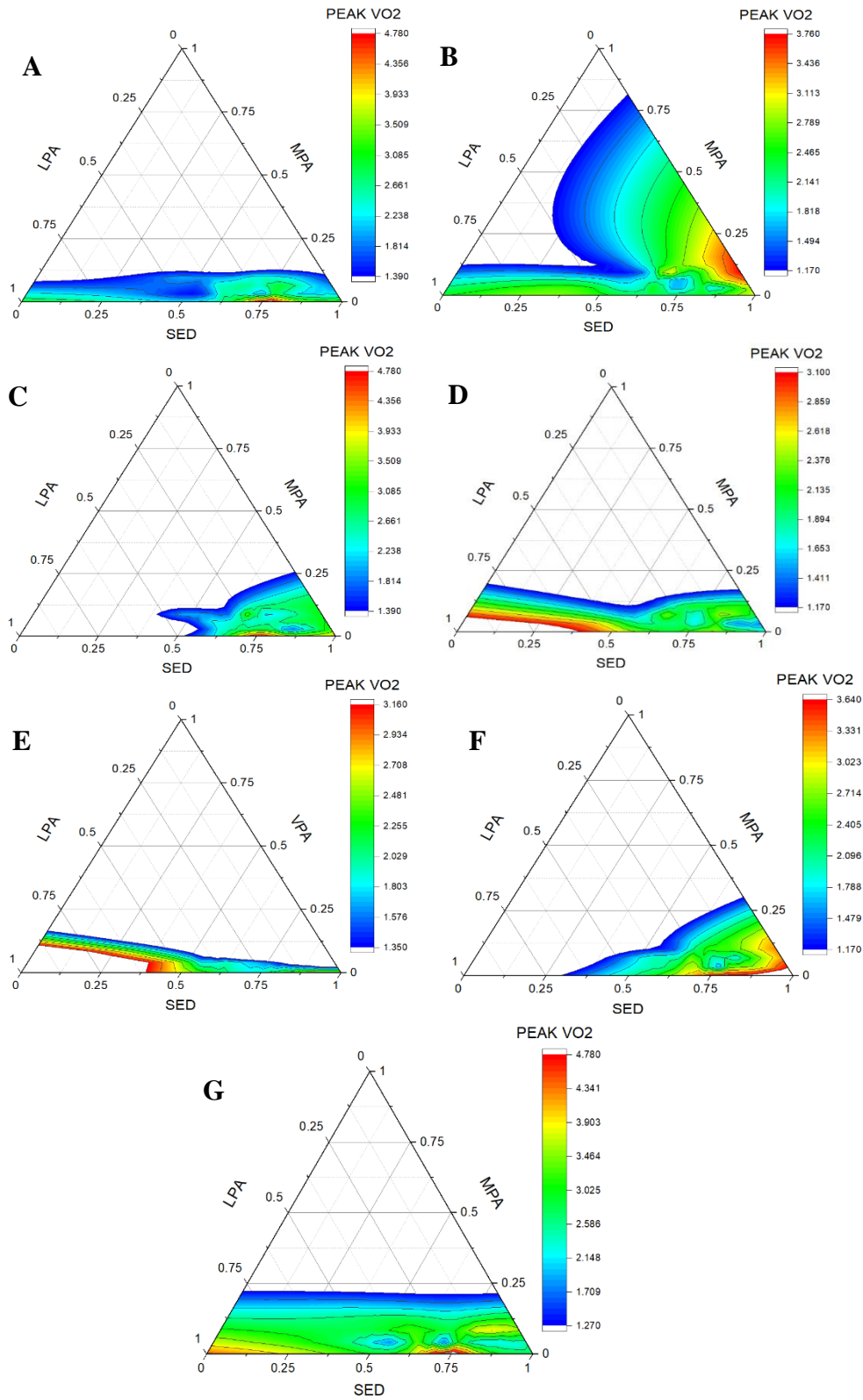
### 11.2.3 Ternary Plots – SED, LPA, VPA – Scaled $\dot{V}O_{2max}$



**Figure 5.4** - Ternary heat plots of all PA behaviours with expected  $\dot{V}O_{2max}$  values for all sub-groups with a) trained athletes; b) untrained participants; c) all boys; d) all girls; e) pre-pubertal children; f) pubertal adolescents; and g) post-pubertal adolescents

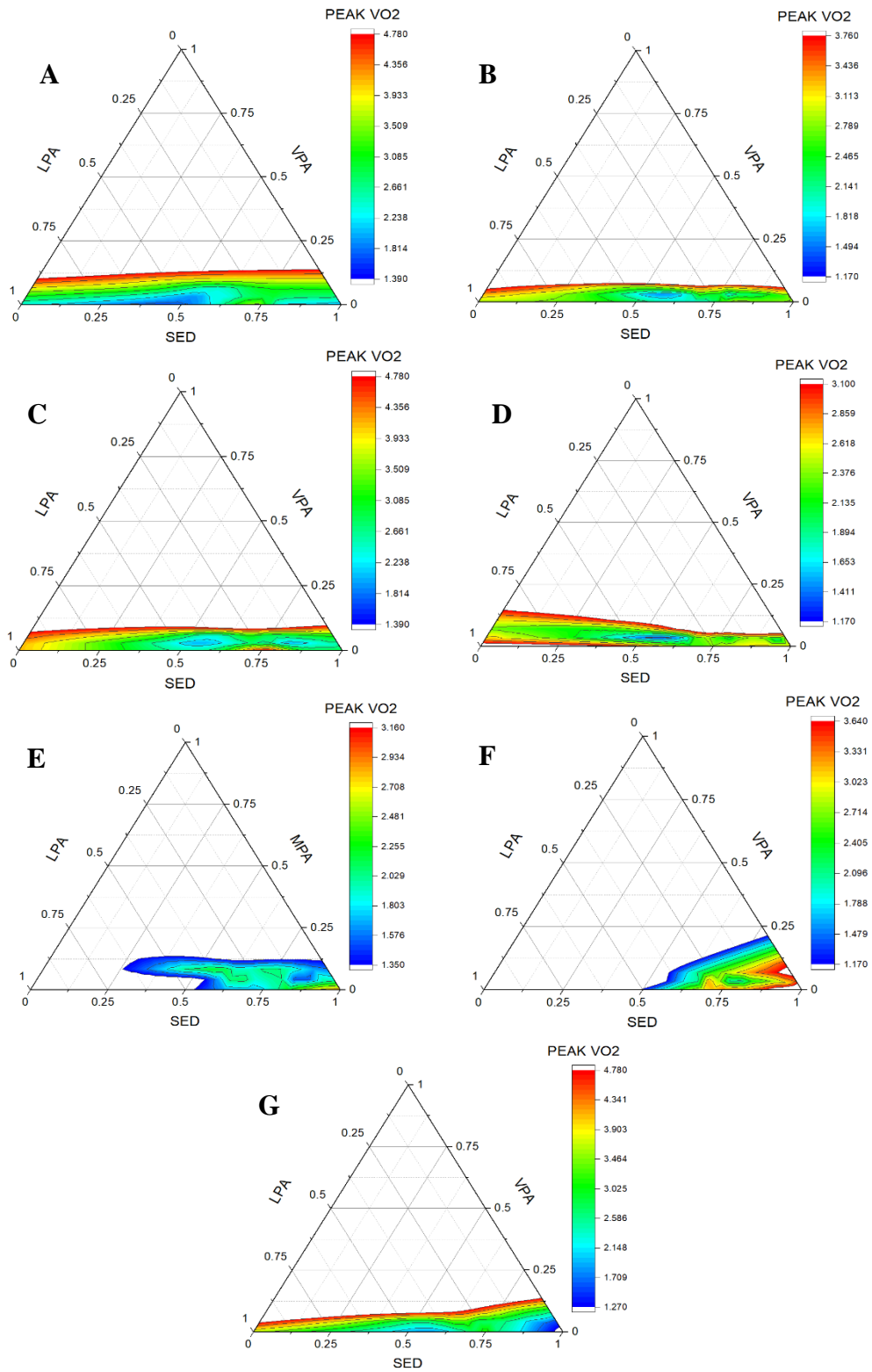


### 11.2.4 Ternary Plots – SED, LPA, MPA – Absolute $\dot{V}O_{2max}$



**Figure 5.5** - Ternary heat plots of all PA behaviours with expected  $\dot{V}O_{2max}$  values for all sub-groups with a) trained athletes; b) untrained participants; c) all boys; d) all girls; e) pre-pubertal children; f) pubertal adolescents; and g) post-pubertal adolescents

### 11.2.5 Ternary Plots – SED, LPA, VPA – Absolute $\dot{V}O_{2max}$



**Figure 5.6** - Ternary heat plots of all PA behaviours with expected  $\dot{V}O_{2max}$  values for all sub-groups with a) trained athletes; b) untrained participants; c) all boys; d) all girls; e) pre-pubertal children; f) pubertal adolescents; and g) post-pubertal adolescents

## 11.3 Chapter 6

### 11.3.1 Parental Information Sheet

**PARENT / GUARDIAN INFORMATION SHEET**  
(Version 1.1, Date: 03/11/2017)

**Project Title:**

Long-Term Athlete Monitoring Program

**Contact Details:**

[Redacted contact details]

**1. Invitation Paragraph**

Thank you for taking the time to read this information sheet which will provide you with all the information you will require to help your son/daughter decide whether they would like to take part. Taking part is completely up to them and it does not matter if your child decides they do not want to participate within this study, it will not impact on any other studies that your child may decide to undertake in the future.

**2. What is the purpose of the study?**

We are doing this study to see the effect of different training methods in children and adolescents to see if one method is better than another as they grow and whether this effect is greater at any age. To answer this question, we need to collect data from children who are doing different types of training, and of different ages to track changes in performance. Even if your child just takes part once, we would still really appreciate your son/daughter's participation within this study.

**3. Why has my child been chosen?**

Your child has been asked if they would like to take part because they are undertaking regular sports training, are performing at a high level, and are between 8 – 18 years of age. If they decide to take part in the study, they can leave the study at any point without any problems. If they ultimately decide they only want to take part in the first session, that is okay we would still really appreciate their participation within the study.

**4. What will happen to my child if they take part?**



If your son/daughter decides to take part, they will be asked to complete a short testing session within a training session. During the session, they will be asked to perform two 30m sprints interspersed with at least 2 minutes of recovery. Whilst your child is performing the sprint trial, their speed will be measured using a STALKER ATS II radar gun (like the one seen in the picture) which will measure their speed at over 46 times a second!! This will give us a detailed insight into how anaerobic performance increases throughout growth.

Alongside 2 sprints we will also take your son/daughter's height, sitting height and weight. This will be used to calculate power and force production during the sprint! It will also be used to see where abouts your child is in their growth spurt to see if changes in sprint performance are influenced by growth and maturation. All testing will take place during a normal training session requiring no extra time commitment or travelling to give your son/daughter the chance to participate within this research.

**5. What are the possible disadvantages of taking part?**

The exercise involved in this study requires your child to work very hard, but they will recover quickly when the exercise tests are completed; it is no harder than what they regularly do during training.

**6. What are the possible benefits of taking part?**

You will be able to see how your child grows, and their fitness improves over the course of their growth and development. You and your child will also get a detailed analysis of their sprint performance so you get to know how fast and powerful they are so you can track their progress over the years!

**7. Will their taking part in the study be kept confidential?**

Yes, when they start the test your child will be given a one-off ID code so no one can identify him/her from any other participant in the study or identify them by their test results. All the information gathered will be saved and stored on a password protected computer which only members of the research team will be able to access.

**8. What if I have any questions?**

If you have any questions, now or in the future, please ring or email us using the details at the top of this sheet. If you don't want to talk to us but feel like you want to talk to someone please contact [REDACTED]

If after reading this information sheet, you wish for your child to participate within the research please click the link below which will take you to an online consent form which you need to fill in **before** your son / daughter can participate within this research.

Consent Form Link: [REDACTED]

11.3.2 Parent/Guardian Consent Form



**PARENT/GUARDIAN CONSENT FORM**  
(Version 1.1, Date: 07/02/2019)

**Project Title:**  
Long-term athlete monitoring programme

**Contact Details:**  
[Redacted]  
[Redacted]  
[Redacted]

- Please initial box**
1. I confirm that I have read and understood the information sheet dated 07/02/2019 (version number 1.1) for the above study and have had the opportunity to ask questions.
  2. I understand that my child's participation is voluntary and that they are free to withdraw at any time, without giving any reason, without their medical care or legal rights being affected.
  3. I understand that sections of any of data obtained may be looked at by responsible individuals from the [Redacted]. I give permission for these individuals to have access to these records.
  4. I understand that data collected on my child may be used in reports and academic publications in anonymous fashion
  5. I declare, to the best of my knowledge, that my child is fit and well and is able to undertake the exercise demands involved within this study
  6. I agree to my child taking part in the above study.

_____	_____	_____
Name of Parent	Date	Signature
_____	_____	_____
Name of Person taking consent	Date	Signature
_____	_____	_____
Researcher	Date	Signature

### 11.3.4 Participant Information Sheet



#### PARTICIPANT INFORMATION SHEET – SPRINT PHASE (Version 1.3, Date: 30/07/2018)

**Project Title:**

Long-Term Athlete Monitoring Programme

**Contact Details:**



**1. Invitation Paragraph**

Thank you for taking the time to read this information sheet which will provide you with all the things you need to know before deciding whether you want to take part. Taking part is up to you and it does not matter if you decide you do not want to.

**2. What is the purpose of the study?**

We are doing this study to see the effect of different training methods in children and adolescents to see if one method is better than another as they grow and whether this effect is greater at any age. To answer this question, we need to collect data from children over a period of many years but you do not have to participate for lots of years if you don't want to! Even if you just do one year we would still really like you to take part!

**3. Why have I been chosen?**

You have been asked if you would like to take part because you are 8 -18 years old and are involved with regular training and perform at a high level, meaning we can observe the effects of training across the ages.

**4. What will happen to me if I take part?**

If you choose to take part, you will be asked to complete a short testing session within a regular training session. During the session you will be asked to complete two 30m sprints. Your speed will be measured using a STALKER ATS II radar gun, which measures your speed over 46 times a second!! In between each sprint you will be given time to rest so that you won't be too tired. During the two sprints do need you to run as fast as you can, but it is no harder to what you usually do in training and you will recover quickly afterwards. We will then ask to take measurements of your height when you are standing and sitting and your weight.

**5. What are the possible disadvantages of taking part?**

The exercise involved in this study requires you to work very hard but you will recover quickly when you stop exercising; it is no harder than what you do in training for your sport.







## 11.4 Chapter 7

### 11.4.1 Parental Information Sheet

**PARENT / GUARDIAN INFORMATION SHEET**  
(Version 1.1, Date: 03/11/2017)

**Project Title:**

Long-Term Athlete Monitoring Program

**Contact Details:**

[REDACTED]  
[REDACTED]  
[REDACTED]

**1. Invitation Paragraph**

Thank you for taking the time to read this information sheet which will provide you with all the information you will require to help your son/daughter decide whether they would like to take part. Taking part is completely up to them and it does not matter if your child decides they do not want to participate within this study, it will not impact on any other studies that your child may decide to undertake in the future.

**2. What is the purpose of the study?**

We are doing this study to see the effect of different training methods in children and adolescents to see if one method is better than another as they grow and whether this effect is greater at any age. To answer this question, we need to collect data from children who are doing different types of training, and of different ages to track changes in performance. Even if your child just takes part once, we would still really appreciate your son/daughter's participation within this study.

**3. Why has my child been chosen?**

Your child has been asked if they would like to take part because they are undertaking regular sports training, are performing at a high level, and are between 8 – 18 years of age. If they decide to take part in the study, they can leave the study at any point without any problems. If they ultimately decide they only want to take part in the first session, that is okay we would still really appreciate their participation within the study.

**4. What will happen to my child if they take part?**



If your son/daughter decides to take part, they will be asked to complete a short testing session within a training session. During the session, they will be asked to perform two 30m sprints interspersed with at least 2 minutes of recovery. Whilst your child is performing the sprint trial, their speed will be measured using a STALKER ATS II radar gun (like the one seen in the picture) which will measure their speed at over 46 times a second!! This will give us a detailed insight into how anaerobic performance increases throughout growth.

Alongside 2 sprints we will also take your son/daughter's height, sitting height and weight. This will be used to calculate power and force production during the sprint! It will also be used to see where abouts your child is in their growth spurt to see if changes in sprint performance are influenced by growth and maturation. All testing will take place during a normal training session requiring no extra time commitment or travelling to give your son/daughter the chance to participate within this research.

**5. What are the possible disadvantages of taking part?**

The exercise involved in this study requires your child to work very hard, but they will recover quickly when the exercise tests are completed; it is no harder than what they regularly do during training.

**6. What are the possible benefits of taking part?**

You will be able to see how your child grows, and their fitness improves over the course of their growth and development. You and your child will also get a detailed analysis of their sprint performance so you get to know how fast and powerful they are so you can track their progress over the years!

**7. Will their taking part in the study be kept confidential?**

Yes, when they start the test your child will be given a one-off ID code so no one can identify him/her from any other participant in the study or identify them by their test results. All the information gathered will be saved and stored on a password protected computer which only members of the research team will be able to access.

**8. What if I have any questions?**

If you have any questions, now or in the future, please ring or email us using the details at the top of this sheet. If you don't want to talk to us but feel like you want to talk to someone please contact [REDACTED]

If after reading this information sheet, you wish for your child to participate within the research please click the link below which will take you to an online consent form which you need to fill in **before** your son / daughter can participate within this research.

Consent Form Link: [REDACTED]

11.4.2 Parent/Guardian Consent Form



**PARENT/GUARDIAN CONSENT FORM**  
(Version 1.1, Date: 07/02/2019)

**Project Title:**  
Long-term athlete monitoring programme

**Contact Details:**  
[Redacted]  
[Redacted]  
[Redacted]

- Please initial box**
1. I confirm that I have read and understood the information sheet dated 07/02/2019 (version number 1.1) for the above study and have had the opportunity to ask questions.
  2. I understand that my child's participation is voluntary and that they are free to withdraw at any time, without giving any reason, without their medical care or legal rights being affected.
  3. I understand that sections of any of data obtained may be looked at by responsible individuals from the [Redacted]. I give permission for these individuals to have access to these records.
  4. I understand that data collected on my child may be used in reports and academic publications in anonymous fashion
  5. I declare, to the best of my knowledge, that my child is fit and well and is able to undertake the exercise demands involved within this study
  6. I agree to my child taking part in the above study.

_____	_____	_____
Name of Parent	Date	Signature
_____	_____	_____
Name of Person taking consent	Date	Signature
_____	_____	_____
Researcher	Date	Signature

### 11.4.3 Participant Information Sheet



**PARTICIPANT INFORMATION SHEET – SPRINT PHASE**  
**(Version 1.3, Date: 30/07/2018)**

**Project Title:**

Long-Term Athlete Monitoring Programme

**Contact Details:**



**1. Invitation Paragraph**

Thank you for taking the time to read this information sheet which will provide you with all the things you need to know before deciding whether you want to take part. Taking part is up to you and it does not matter if you decide you do not want to.

**2. What is the purpose of the study?**

We are doing this study to see the effect of different training methods in children and adolescents to see if one method is better than another as they grow and whether this effect is greater at any age. To answer this question, we need to collect data from children over a period of many years but you do not have to participate for lots of years if you don't want to! Even if you just do one year we would still really like you to take part!

**3. Why have I been chosen?**

You have been asked if you would like to take part because you are 8 -18 years old and are involved with regular training and perform at a high level, meaning we can observe the effects of training across the ages.

**4. What will happen to me if I take part?**

If you choose to take part, you will be asked to complete a short testing session within a regular training session. During the session you will be asked to complete two 30m sprints. Your speed will be measured using a STALKER ATS II radar gun, which measures your speed over 46 times a second!! In between each sprint you will be given time to rest so that you won't be too tired. During the two sprints do need you to run as fast as you can, but it is no harder to what you usually do in training and you will recover quickly afterwards. We will then ask to take measurements of your height when you are standing and sitting and your weight.

**5. What are the possible disadvantages of taking part?**

The exercise involved in this study requires you to work very hard but you will recover quickly when you stop exercising; it is no harder than what you do in training for your sport.

**6. What are the possible benefits of taking part?**

You will also be a part of a study that will really help us understand the effects of different training methods in children and adolescents to see if one is better than another as some people have said. Also, we will be able to see how your speed and power develops as you grow and can also see in lots of detail how fast you are and how quickly you accelerate!!

**7. Will my taking part in the study be kept confidential?**

Yes, when you start the test you will be given a one-off ID code so no one can identify you from another participant in the study or identify you by your results. All your information will be saved and stored on a password protected computer which only members of the research team will be able to access.

**8. What if I have any questions?**

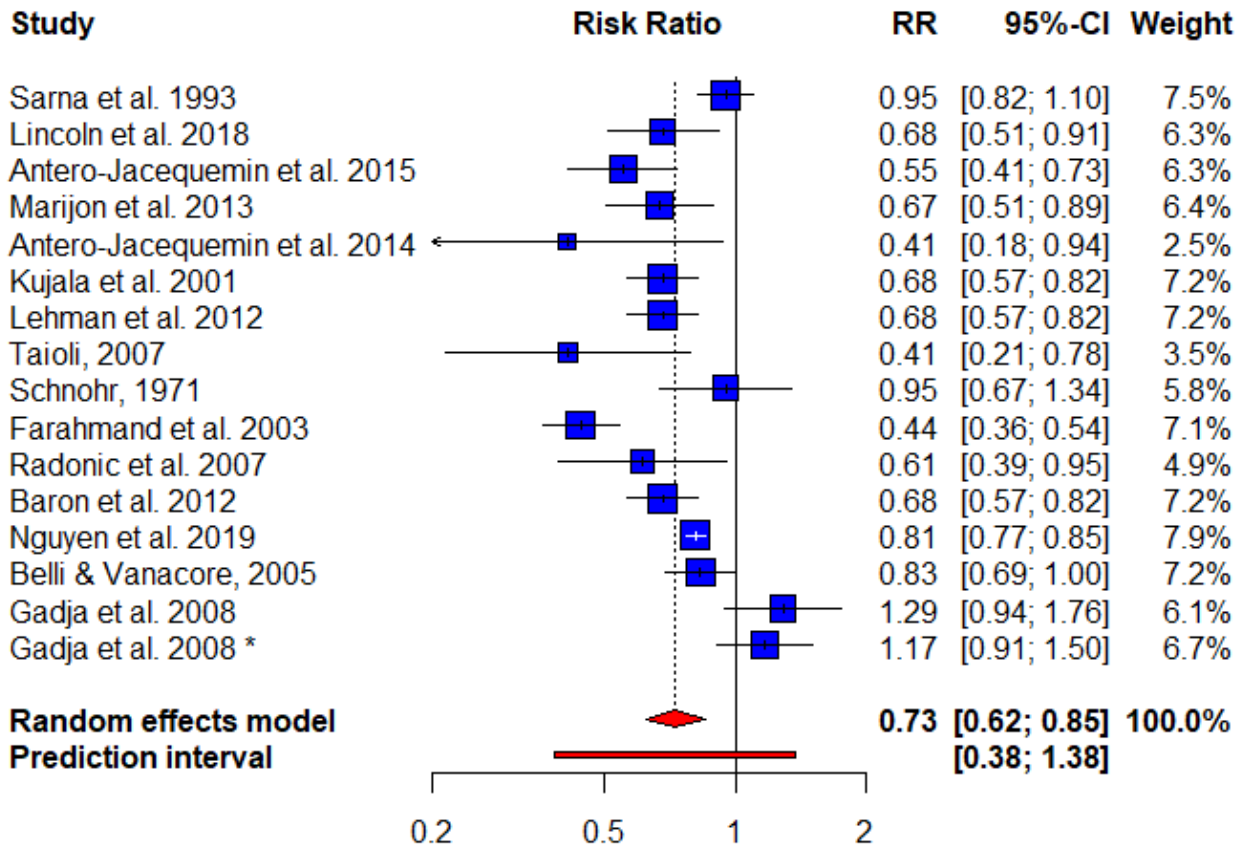
If you have any questions, now or in the future, please ring or email us using the details at the top of this sheet. If you don't want to talk to us but feel like you want to talk to someone please contact [REDACTED]

[REDACTED]

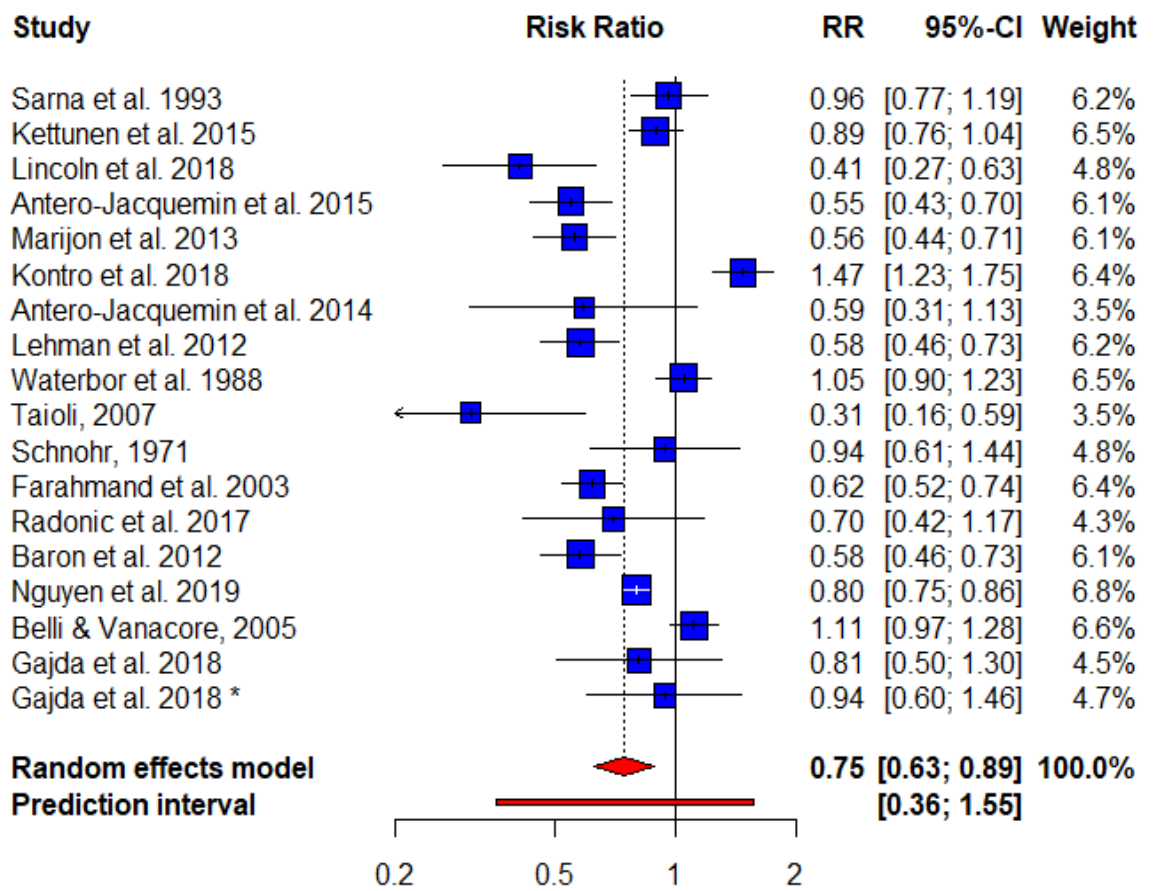


11.5 Chapter 8

11.5.1 Male cardiovascular disease (CVD) mortality forest plot



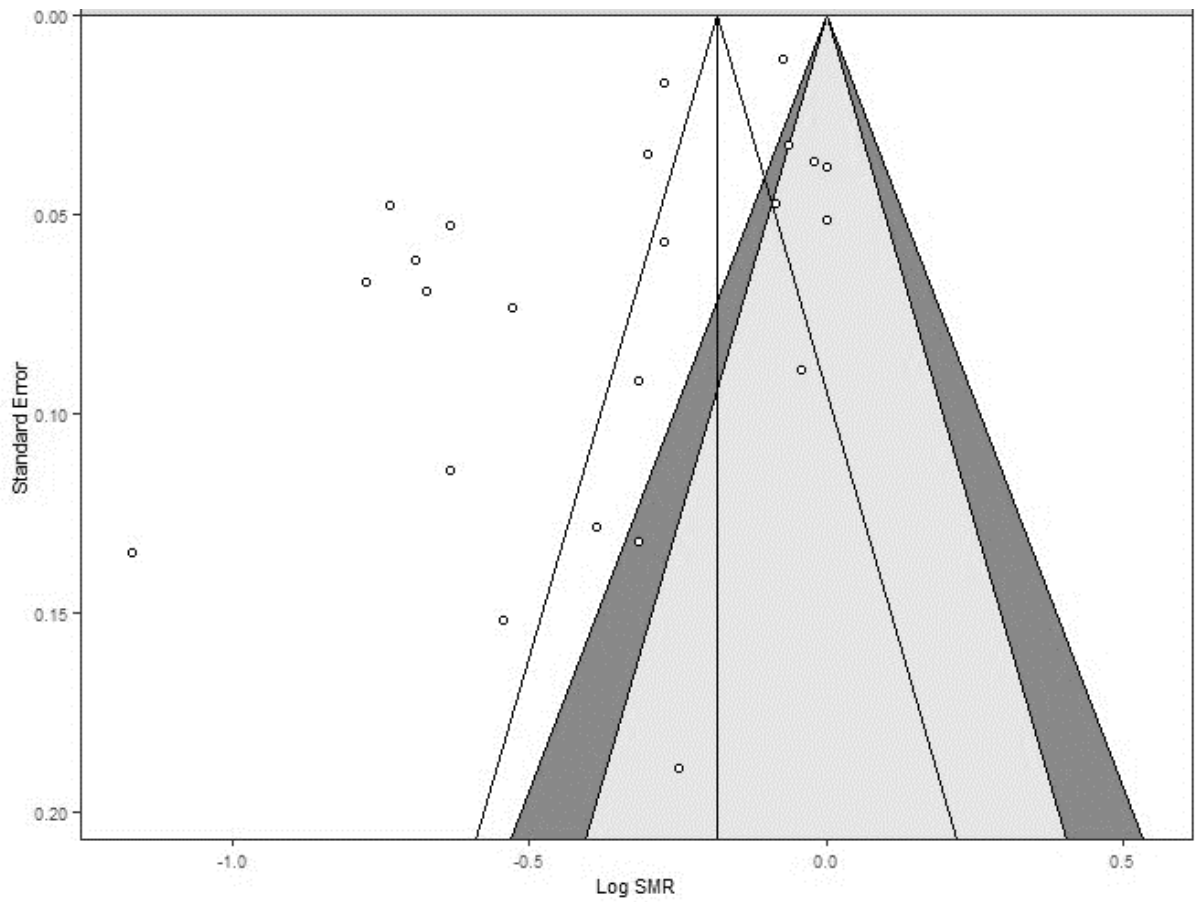
11.5.2 Male cancer mortality forest plot





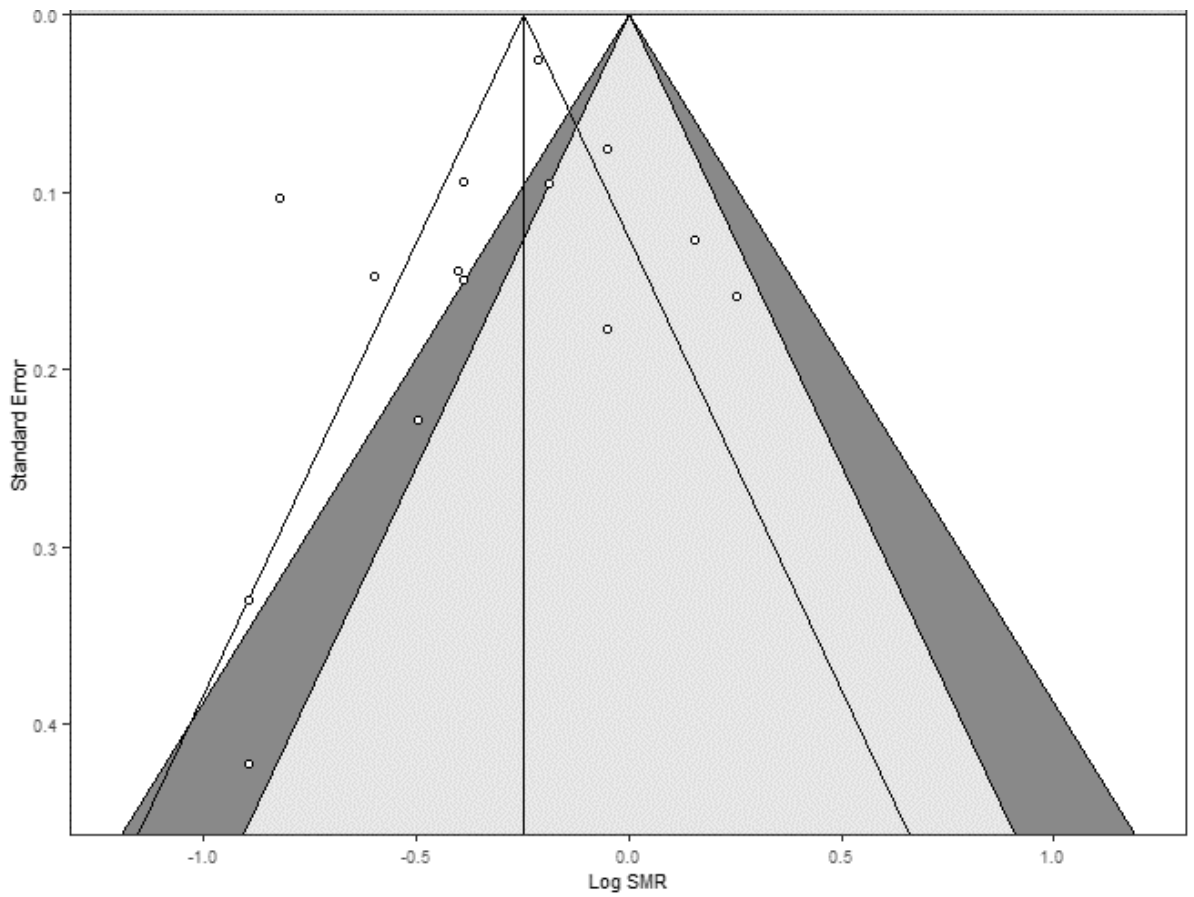
### 11.5.3 Funnel Plots

#### 11.5.3.1 All-Cause Mortality Funnel Plot



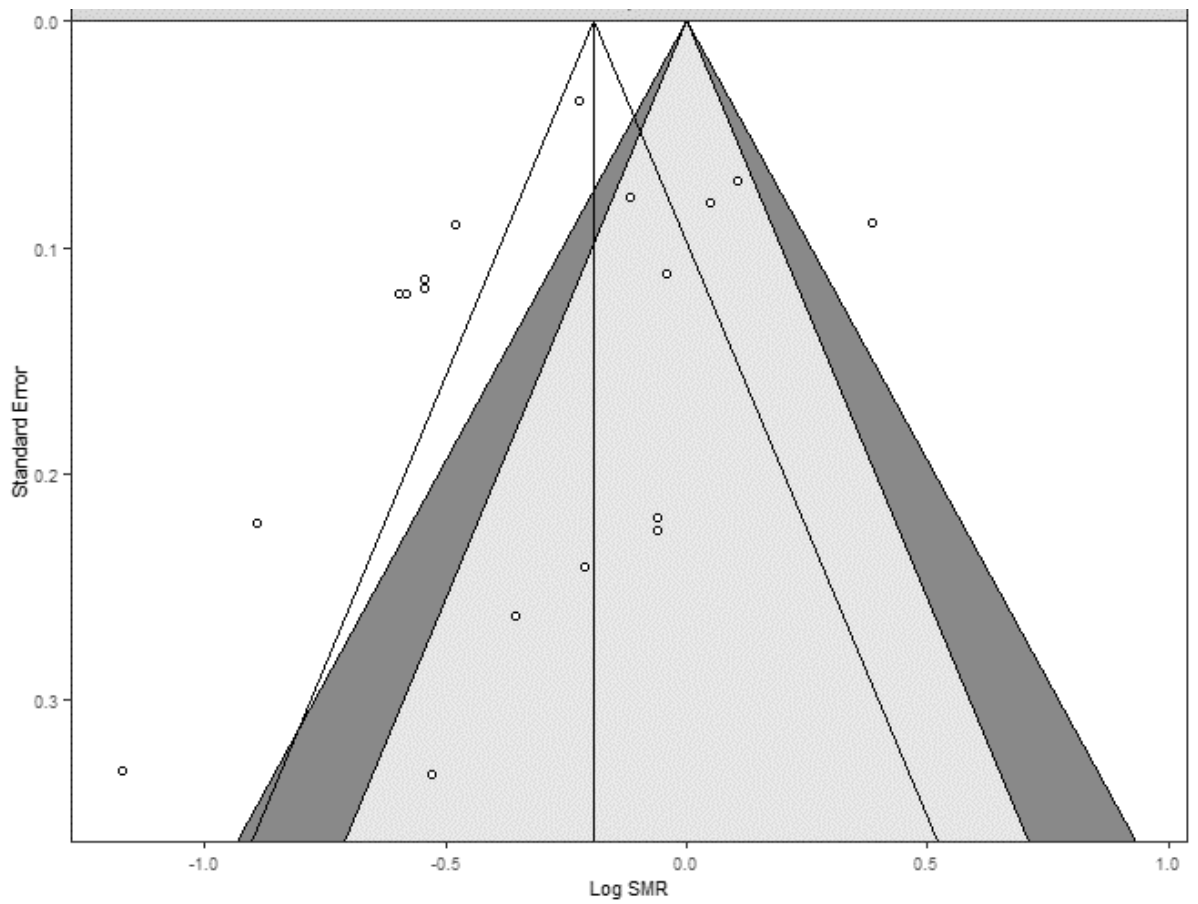
**Figure 8.4** - A funnel plot of the log standard mortality ratio for all-cause mortality versus the logged standard error for all 23 studies included. The light grey and dark grey areas depict significance of  $p < 0.05$  and  $p < 0.01$ , respectively.

11.5.2.2 Cardiovascular Disease Mortality Funnel Plot



**Figure 8.5** - A funnel plot of the log standard mortality ratio for cardiovascular disease mortality versus the logged standard error for all 15 studies included. The light grey and dark grey areas depict significance of  $p < 0.05$  and  $p < 0.01$ , respectively.

### 11.5.2.3 Cancer Mortality Funnel Plot



**Figure 8.6** - A funnel plot of the log standard mortality ratio for cancer mortality versus the logged standard error for all 17 studies included. The light grey and dark grey areas depict significance of  $p < 0.05$  and  $p < 0.01$ , respectively.