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REVIEW ARTICLE

An Examination and Critique of Current Methods to Determine Exercise Intensity

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Abstract
Prescribing the frequency, duration, or volume of training is simple as these factors can be altered by manipulating the number of exercise sessions per week, the duration of each session, or the total work performed in a given time frame (e.g., per week). However, prescribing exercise intensity is complex and controversy exists regarding the reliability and validity of the methods used to determine and prescribe intensity. This controversy arises from the absence of an agreed framework for assessing the construct validity of different methods used to determine exercise intensity. In this review, we have evaluated the construct validity of different methods for prescribing exercise intensity based on their ability to provoke homeostatic disturbances (e.g., changes in oxygen uptake kinetics and blood lactate) consistent with the moderate, heavy, and severe domains of exercise. Methods for prescribing exercise intensity include a percentage of anchor measurements, such as maximal oxygen uptake ($\dot{V}O_{2max}$), peak oxygen uptake ($\dot{V}O_{2peak}$), maximum heart rate (HR_{max}), and maximum work rate (i.e., power or velocity—$\dot{W}_{max}$ or $\dot{V}_{max}$, respectively), derived from a graded exercise test (GXT). However, despite their common use, it is apparent that prescribing exercise intensity based on a fixed percentage of these maximal anchors has little merit for eliciting distinct or domain-specific homeostatic perturbations. Some have advocated using submaximal anchors, including the ventilatory threshold (VT), the gas exchange threshold (GET), the respiratory compensation point (RCP), the first and second lactate threshold (LT_1 and LT_2), the maximal lactate steady state (MLSS), critical power (CP), and critical speed (CS). There is some evidence to support the validity of LT_1, GET, and VT to delineate the moderate and heavy domains of exercise. However, there is little evidence to support the validity of most commonly used methods, with exception of CP and CS, to delineate the heavy and severe domains of exercise. As acute responses to exercise are not always predictive of chronic adaptations, training studies are required to verify whether different methods to prescribe exercise will affect adaptations to training. Better ways to prescribe exercise intensity should help sport scientists, researchers, clinicians, and coaches to design more effective training programs to achieve greater improvements in health and athletic performance.

1 Introduction

Exercise is commonly prescribed with the goal of stimulating adaptations that will improve both athletic performance and health [1]. This prescription is usually based on four main principles: frequency, duration, volume, and intensity. Methods for prescribing the frequency, duration, or volume of training are relatively simple as these factors can be altered by manipulating the number of exercise sessions per week, the duration of each session, or the total volume of training performed in a given time frame (e.g., per week). However, there is no consensus regarding which of the many commonly used methods to determine exercise intensity is best. As a consequence, there is controversy regarding the most appropriate methods to normalise exercise intensity between individuals. This likely contributes to sub-optimal exercise prescription, and also complicates the ability to compare the outcomes of different research studies and training programs.

Methods for determining exercise intensity include a percentage of various anchor measurements, such as maximal

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Key Points

There is controversy, and little agreement, about the best approaches to determine exercise intensity.

Some of this controversy arises from the absence of an agreed framework for assessing the construct validity of different methods for determining exercise intensity. In this review, we have evaluated the construct validity of different methods of prescribing intensity based on their ability to provoke homeostatic disturbances (e.g., changes in oxygen uptake kinetics and blood lactate) consistent with the moderate, heavy, and severe domains of exercise.

Prescribing exercise intensity based on a fixed percentage of maximal anchors, such as $\dot{V}O_{2max}$, $\dot{W}_{max}$, $\dot{V}_{max}$, and HR_{max}, has substantial shortcomings as a means for normalising exercise intensity between individuals.

While there is some evidence to support the validity of LT_1, GET, and VT to delineate the moderate and heavy domains of exercise, there is little evidence to support the validity of most commonly used methods, with the exception of CP and CS, to delineate the heavy and severe domains of exercise.

oxygen uptake ($\dot{V}O_{2max}$), peak oxygen uptake ($\dot{V}O_{2peak}$[1]), maximum heart rate (HR_{max}), and maximum work rate (i.e., power or velocity) (i.e., $\dot{W}_{max}$ or $\dot{V}_{max}$, respectively), derived from a graded exercise test (GXT). Submaximal anchor measurements derived from a GXT have also been used to prescribe exercise intensity, including the ventilatory threshold (VT), the gas exchange threshold (GET), the respiratory compensation point (RCP), and the first and second lactate threshold (LT_1 and LT_2) [2, 3]. Other submaximal anchor measurements, such as the maximal lactate steady state (MLSS), critical power (CP), and critical speed (CS) [2, 4, 5], can be derived from a series of constant work rate bouts. The CP and CS metrics can also be derived using a 3-min all-out exercise test (3MT) [6, 7]. Alternative methods to determine intensity are based on the difference between resting and maximal values, such as the HR reserve ($\%HR_R$) and $\dot{V}O_2$ reserve ($\%\dot{V}O_2$). Lastly, the delta (Δ) method uses the percent difference between a maximal anchor (e.g., $\dot{W}_{max}$) and various submaximal anchors (e.g., GET) [8]. Although these common methods to determine exercise intensity are often used interchangeably, there is research challenging this practice [9–13].

Submaximal and maximal anchors have also been used in different models to define different training intensities [15–17]. For example, one model [16, 18] creates five exercise intensity levels (L1–L5) based upon LT_1 and LT_2 derived from a GXT (Fig. 1); these levels can be further characterised by percentages of HR_{max}, blood lactate values, and ratings of perceived exertion (RPE) (Table 1). Another model [17, 19–23], which uses submaximal anchors paired with the retrospective analyses of athlete training distributions, has been used to yield three different training zones (Fig. 2); these zones can be further characterised by percentages of HR_{max} and $\dot{V}O_{2max}$, and blood lactate values (Table 2). There is also a training model based on the domains of exercise (i.e., the moderate, heavy, and severe domains of exercise); these domains are independent of submaximal anchors and characterised by specific oxygen uptake kinetics and blood lactate responses [4, 5, 24–36] (Fig. 3).[2] Submaximal anchors have also been used to define the domains of exercise [37–39], even though the majority of these methods have not been confirmed to elicit domain-specific physiological responses.

Different exercise intensities will provoke specific homeostatic perturbations (e.g., changes in muscle energy turnover, oxygen demand, metabolite accumulation, etc.) [25, 40, 41]. The mechanisms by which these homeostatic perturbations are sensed and then translated into improved function remain unresolved. Nonetheless, one common theory is these homeostatic perturbations in response to exercise will then initiate transcriptional programmes essential to increase the abundance of specific proteins and to ultimately improve cellular function [42–44]. In support of this, there is emerging evidence that in vivo lactate production from glycolysis upregulates genes associated with mitochondrial biogenesis [45, 46]. Thus, while an unresolved question in exercise science is the physiological basis for exercise prescription, an argument can be made for using the attainment of specific homeostatic perturbations as a framework for determining exercise intensity. Within this framework, different methods of determining an apparently equivalent exercise intensity should elicit similar homeostatic disturbances in all participants.

Exercise-induced homeostatic perturbations include systemic responses (e.g., increased $\dot{V}O_2$ and blood lactate concentration), changes in intramuscular substrates and metabolites (e.g., intramuscular phosphocreatine, lactate, and ATP), and mechanical stress [25, 26, 30, 35, 36, 47–56]. Although

[1] $\dot{V}O_{2max}$: refers to the maximal oxygen uptake value from an 8- to 12-min GXT confirmed via a verification exhaustive bout (VEB); $\dot{V}O_{2peak}$: refers to the peak oxygen uptake value from a < 8- or > 12-min GXT or a $\dot{V}O_2$ value not confirmed via a VEB [14].

[2] The extreme domain is a supramaximal domain and defined as an intensity too extreme to permit attainment of $\dot{V}O_{2max}$ prior to fatigue. Methods to appropriately demarcate the extreme domain are beyond the scope of this review and will not be discussed.

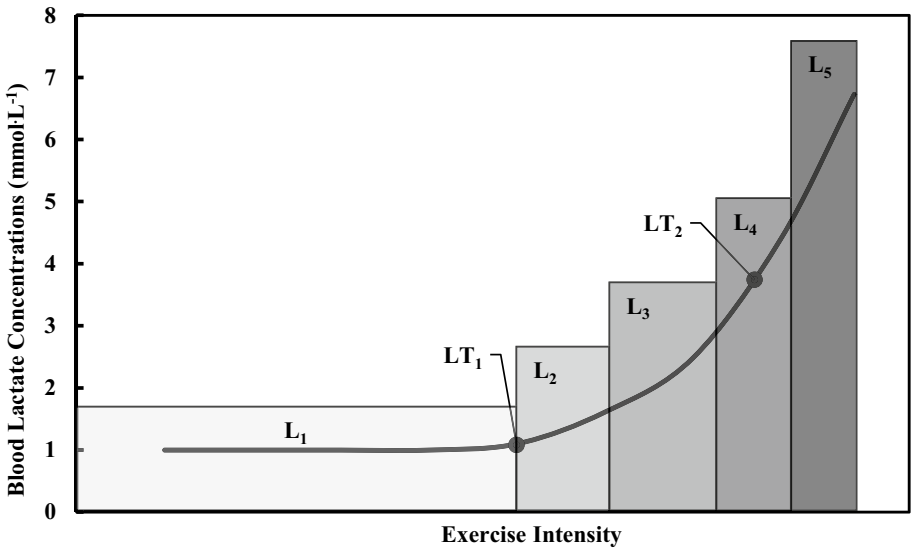


Fig. 1 The five aerobic training levels (L1–L5) based on the first (LT_1) and second lactate threshold (LT_2) derived from a graded exercise test (GXT) [16, 18]. The LT_1 (i.e., lactate threshold 1) represents the rise in blood lactate above baseline. The LT_2 (i.e., lactate threshold 2) represents an acceleration of blood lactate accumulation

Table 1 The five aerobic training levels based on the first and second lactate threshold (LT_1/LT_2) derived from a graded exercise test

Aerobic training zone	L1 (recovery)	L2 (extensive endurance)	L3 (intensive endurance)	L4 (threshold training)	L5 (interval training)
Heart rate (% of HR_{max})	65–75%	75–80%	80–85%	85–92%	> 92%
Blood lactate ($mmol \cdot L^{-1}$)	< 2.0	2.0–2.5	2.5–3.5	3.5–5.0	> 5.0
Rating of perceived exertion (RPE) (6–20)	< 11	11–12	13–14	15–16	17–19
Relative to sub-maximal anchor	< LT_1	LT_1 < LT_2	LT_1 < LT_2	< LT	> LT_2

Each level is characterised by a percent of the maximum heart rate (% of HR_{max}), an absolute blood lactate value, a rating of perceived exertion, and the relationship with a submaximal anchor [26, 48]

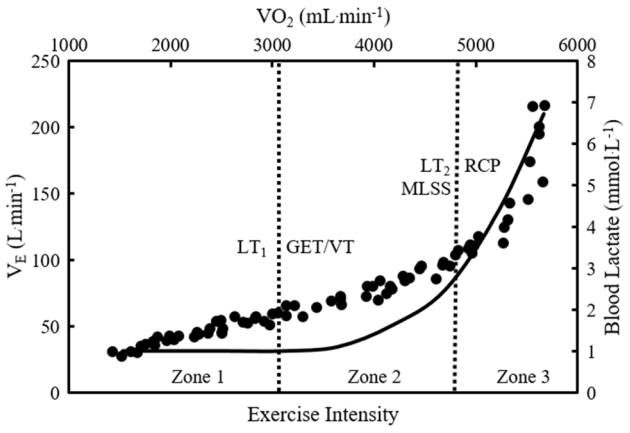


Fig. 2 The training intensity distribution model divides intensity into 3 zones. Zone 1 and zone 2 are demarcated by the first lactate threshold (LT_1), the gas exchange threshold (GET) and/or the ventilatory threshold (VT). Zones 2 and 3 are demarcated by the second lactate threshold (LT_2), the respiratory compensation point (RCP) and/or the maximal lactate steady state (MLSS). The dark circles are pulmonary data points of minute ventilation ($\dot{V}_E$) relative to oxygen uptake ($\dot{V}O_2$). The solid line represents the fitted blood lactate curve [17, 20, 21, 145]

these perturbations are all influenced by exercise intensity [57, 58], many are of limited value to routinely quantify exercise intensity given the lack of research or the invasive nature of the techniques required to obtain some measures (e.g., muscle biopsies). For this reason, less-invasive systemic responses, such as $\dot{V}O_2$ and blood lactate concentration, which are associated with intramuscular changes [25, 48, 49, 59–61], are typically used as indicators of homeostatic perturbations in response to different exercise intensities (Fig. 3).

Although there has been limited research directly assessing the effects of different methods of determining an apparently equivalent exercise intensity on homeostatic disturbances, it is clear that ostensibly similar exercise intensities can result in very different homeostatic perturbations [8, 62–65]. For example, exercise at an intensity of between 60 and 80% of $\dot{V}O_{2max}$ is often referred to as moderate intensity [66, 67]; however, large differences in homeostatic perturbations (e.g., oxygen uptake kinetics and blood lactate responses) [35, 52] have been reported across multiple studies for exercise performed within those percentages of $\dot{V}O_{2max}$ [8, 62–65]. Some investigators have subsequently

Table 2 The training intensity distribution model divides intensity into 3 zones, where zones 1 and 2 are demarcated by the first lactate threshold (LT_1), the gas exchange threshold (GET) and/or the ventilatory threshold (VT) and zones 2 and 3 by the second lactate threshold (LT_2), the respiratory compensation point (RCP) and/or the maximal lactate steady state (MLSS)

Training zone	Zone 1 (low intensity)	Zone 2 (moderate intensity)	Zone 3 (high intensity)
Heart Rate (% of HR_{max})	< 80%	80–90%	> 90%
% $\dot{V}O_{2max}$	65–75%	75–85%	> 85%
Blood lactate ($mmol \cdot L^{-1}$)	< 2.0	2.0–4.0	< 4.0
Relative to sub-maximal anchor	> GET/VT/LT_1	GET/VT/LT_1 < RCP/MLSS/LT_2	> RCP/MLSS/LT_2

Each zone is characterised by a percentage of maximal heart rate (HR_{max}), a percentage of the maximal oxygen uptake ($\dot{V}O_{2max}$), an absolute blood lactate value, and its relationship with submaximal anchors [27–29, 49]

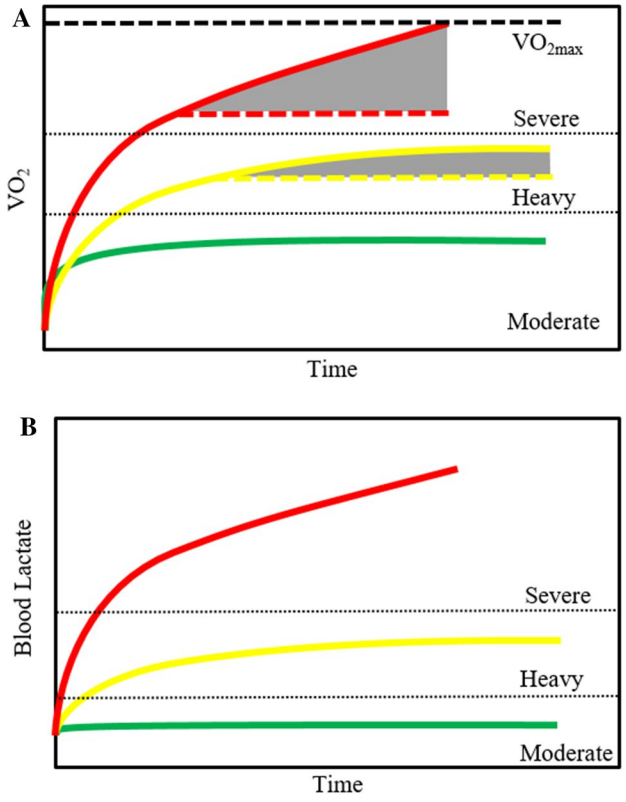


Fig. 3 **a**, **b** $\dot{V}O_2$ and blood lactate responses during constant-load exercise at moderate (green), heavy (yellow), and severe (red) exercise intensity domains [4, 27, 51, 147]. **a** $\dot{V}O_2$ kinetics during moderate exercise are depicted by a mono-exponential uptake response with an attained steady state. In heavy (yellow lines) and severe (red lines) exercise, there is a delayed steady state, whereby there is a slow, exponential rise in $\dot{V}O_2$ above the expected values interpolated from a graded exercise test (dashed lines). The shaded regions indicate the $\dot{V}O_2$ slow component, which represents the additional aerobic energy required, above the expected value, for sustaining a fixed, external load. Heavy exercise is characterised by a smaller slow component with a delayed steady state; severe exercise is characterised by a continual rise in $\dot{V}O_2$ that can eventually evoke attainment of $\dot{V}O_{2max}$. **b** The blood lactate response during moderate exercise remains at baseline given the predominant source of energy is derived via oxidative phosphorylation. The blood lactate concentration during heavy exercise increases above baseline and then attains a steady state. In contrast, the blood lactate during severe exercise continues to rise above baseline with an absence of a steady state

proposed that exercise intensity determined relative to submaximal anchors (e.g., GET, VT, or LT_1) will result in distinct and/or more homogeneous homeostatic perturbations [2, 65]. However, the validity of these anchors to identify exercise intensities that produce distinct and/or homogeneous homeostatic perturbations between individuals has also been called into question [5–7, 24, 39, 68–82].

The domains of exercise (i.e., moderate, heavy and severe) are quantified by their distinct oxygen uptake kinetics and blood lactate response, which are reflective of intramuscular perturbations and can be monitored with relative ease by using a metabolic analyser and venous/capillary blood sampling [2, 52]. During moderate exercise, a plateau of $\dot{V}O_2$ and blood lactate concentrations near baseline levels indicates that ATP production is being met predominantly via oxidative phosphorylation [30, 32, 83, 84], type I muscle fibre recruitment [85], a low rate of muscle glycogen depletion [86], low calcium flux [50], and that muscle lactate and H^+ concentrations will be similar to baseline values [48, 49] (Table 3; Figs. 3 and 4). During heavy exercise, there is an observed 'slow component' of $\dot{V}O_2$, with a delayed steady state, and a rise in blood lactate above baseline with a subsequent plateau, which represents a plateau of intramuscular lactate concentration [30] and a decrease in contractile efficiency attributable to increased cytosolic ATP turnover [28, 87]. Exercise in the heavy domain is also associated with the recruitment of type II muscle fibres [28, 32, 87–90], a moderate rate of glycogen depletion [86] and calcium flux [50], and a decrease and subsequent plateau in muscle pH [25] (Table 4; Figs. 3 and 4). During severe exercise, there is a 'slow component' without a steady state of $\dot{V}O_2$, and a continual increase in blood lactate [35] that is indicative of increased cytosolic ATP turnover (with a continual increase in intramuscular lactate concentrations) [30, 32, 89–91], a greater contribution of phosphocreatine stores to ATP turnover [25, 36], added recruitment of highly fatigable type II muscle fibres [28, 87], rapid rates of muscle glycogen depletion [54, 86], high calcium flux [50], and a continual decrease in muscle pH [25] (Table 5; Figs. 3 and 4).

Table 3 (Moderate). Evidence of the oxygen uptake kinetic response ($\dot{V}O_2$), blood and intramuscular lactate response, muscle fibre recruitment (based on muscle glycogen utilisation), phosphocreatine (PCr) utilisation, intramuscular nicotinamide adenine dinucleotide (NADH) concentration, and substrate utilisation determined via indirect calorimetry and tracer infusion during moderate exercise

References	Exercise intensity	Oxygen uptake kinetics	Blood lactate	Intramuscular lactate	Muscle fibres recruited	PCr utilisation	NADH	Substrate utilisation
Gollnick et al. [90]	31% of $\dot{V}O_{2max}$	–	Baseline	–	Type I	–	–	–
Jorfeldt et al. [38]	29% of $\dot{V}O_{2max}$	–	Baseline	Baseline	–	–	–	–
Vøllestad and Blom [104]	43% of $\dot{V}O_{2max}$	–	Baseline	–	Type I	–	–	–
Sahlin et al. [95]	40% of $\dot{V}O_{2max}$	–	–	Baseline	–	–	Below Baseline	–
Spriet et al. [105]	40% of $\dot{V}O_{2max}$	–	–	Baseline	–	–	Type I and II Fibres Below Baseline	–
Barstow and Moe [106]	35% of $\dot{V}O_{2max}$	$\dot{V}O_2$ plateau	–	–	–	–	–	–
Romijn et al. [107]	25% of $\dot{V}O_{2max}$	–	–	–	–	–	–	FFA, IMTG, Glucose
Barstow et al. [88]	80% of GET	$\dot{V}O_2$ plateau	–	–	–	Plateau	–	–
Rossiter et al. [87]	80% of VT	$\dot{V}O_2$ plateau	–	–	–	Plateau	–	–
Bell et al. [108]	80% of VT	$\dot{V}O_2$ plateau	–	–	–	–	–	–
Simmond et al. 2013	80% of GET	$\dot{V}O_2$ plateau	Baseline	–	–	–	–	–
Black et al. [80]	90% of GET	$\dot{V}O_2$ plateau	Baseline	Baseline	–	Plateau		

$\dot{V}O_2$ *plateau* a plateau in oxygen uptake kinetics without a slow component, *baseline lactate* lactate values not different from baseline values during continuous exercise, *PCr plateau* a plateau in PCr utilisation during continuous exercise following the non-steady state primary component of moderate exercise, *NADH below baseline* low/no cytosolic ATP turnover, and free fatty acid (FFA), intramuscular triglycerides (IMTG), and Glucose (in the plasma) indicate the source of ATP production, $\dot{V}O_{2max}$ maximal oxygen uptake, *GET* gas exchange threshold, *VT* ventilatory threshold, — not measured. Studies included prescribed exercise below the gas exchange/ventilatory threshold or below ~ 45% $\dot{V}O_{2max}$

A potential advantage of using the domains of exercise to determine exercise intensity is that although training status influences exercise tolerance at a given intensity [92], overall muscle glycogen content [93], discrete $\dot{V}O_2$ patterns (e.g., the phase II time constant) [94, 95], and intramuscular lactate oxidation [55, 96], these factors have little effect on $\dot{V}O_2$ and blood lactate kinetic responses during exercise in healthy individuals [97–100]. This suggests that determining exercise intensity based on these distinct and homogeneous homeostatic perturbations (i.e., systemic responses such as oxygen uptake kinetics and blood lactate responses) could be an effective method to normalise exercise intensity between individuals. To date, however, there has been little research assessing this hypothesis.

The aim of this review is to evaluate the construct validity of the most common methods used to determine exercise intensity, and discuss their reliability and validity based on their ability to yield distinct and/or homogeneous homeostatic perturbations. We address protocol designs, criteria for establishing anchors, and the limitations of each method. Discrepancies in the assumed and concurrent validity of the methods used to normalise and determine exercise intensity are also discussed. Lastly, recommendations for determining exercise intensity and future research directions are provided.

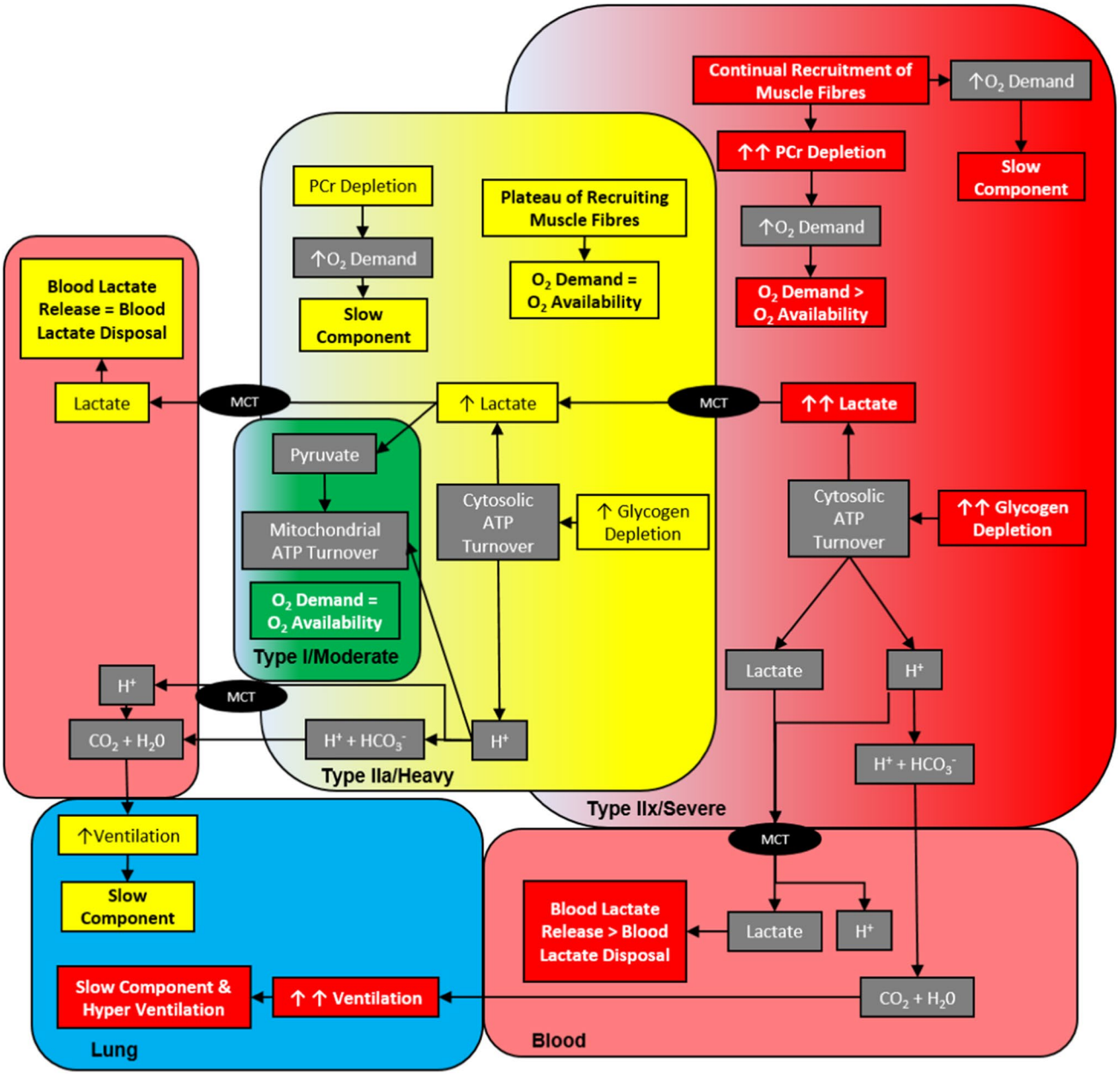


Fig. 4 Schematic illustrating the domain-specific muscle recruitment pattern contributing to the slow component of oxygen uptake, absence of an oxygen uptake plateau, and the blood lactate response. During continuous, moderate exercise, type I muscle fibres are predominantly recruited. ATP is produced solely via mitochondrial ATP turnover (i.e., Krebs Cycle), O_2 demand is equal to O_2 availability, and muscle and blood lactate levels remain at baseline. During continuous, heavy exercise, type I and IIa muscle fibres are recruited, ATP is produced via mitochondrial and cytosolic ATP turnover. The recruitment of less efficient type IIa muscle fibres and an increase in ventilation due to increased non-metabolic CO_2 production results in the delayed steady state of oxygen uptake (i.e., a slow component), and reliance on non-mitochondrial ATP turnover yielding an increase in blood lactate above baseline with an achieved steady state mirroring the oxygen uptake pattern. During continuous, severe exercise, type I, IIa, and IIx muscle fibres are recruited, ATP is produced via mitochondrial, and cytosolic ATP turnover, and via a continual depletion of the phosphocreatine (PCr) stores, which results in the continual increase in O_2 uptake until the cessation of exercise. The recruitment of less efficient type IIa and IIx muscle fibres increases the amplitude of the slow component, and there is a further increase in ventilation (i.e., hyperventilation) due to increased non-metabolic CO_2 production. Lastly, intramuscular lactate production exceeds lactate oxidation, which results in lactate appearance exceeding disappearance. *MCT* monocarboxyl transporter

Table 4 (Heavy). Evidence of the oxygen uptake kinetic response ($\dot{V}O_2$), blood and intramuscular lactate response, muscle fibre recruitment (based on muscle glycogen utilisation), phosphocreatine (PCr) utilisation, intramuscular nicotinamide adenine dinucleotide (NADH) concentration, and substrate utilisation determined via indirect calorimetry and tracer infusion tracer, during heavy exercise

References	Exercise intensity	Oxygen uptake kinetics	Blood lactate	Intramuscular lactate	Muscle fibres recruited	PCr utilisation	NADH	Substrate utilisation
Gollnick et al. [90]	64% of $\dot{V}O_{2max}$	–	Plateau above baseline	–	Type I and II	–	–	–
Jorfeldt et al. [38]	51% and 71% of $\dot{V}O_{2max}$	–	Plateau above baseline	Plateau above baseline	–	–	–	–
Vøllestad and Blom [104]	61% of $\dot{V}O_{2max}$	–	Plateau above baseline	–	Type I and IIa	–	–	–
Sahlin et al. [95]	75% of $\dot{V}O_{2max}$	–	–	Above Baseline	–	–	Above Baseline	–
Spriet et al. [105]	75% of $\dot{V}O_{2max}$	–	–	Above baseline	–	–	Type I and II above baseline	–
Romijn et al., [107]	65% of $\dot{V}O_{2max}$	–	–	–	–	–	–	FFA, IMTG, Glucose, Glycogen
Jones et al. [109]	90% of CP	–	–	–	–	Plateau	–	–
Simmondet al. 2013	Δ40% of GET and $\dot{V}O_{2max}$	$\dot{V}O_2$ slow component and plateau	Plateau above baseline	–	–	–	–	–
Vanhatalo et al. [33]	95% of CP	$\dot{V}O_2$ slow component and plateau	Plateau above baseline	Plateau above baseline	–	Plateau	–	–
Black et al. [80]	~92% of CP	$\dot{V}O_2$ slow component and plateau	Plateau above baseline	Above baseline		Below baseline		

$\dot{V}O_2$ *slow component and plateau* a plateau in oxygen uptake kinetics with an observed slow component, characterised by statistical analysis or via on-transient modelling techniques, *plateau above baseline lactate* lactate values were higher compared to baseline values and stabilised during continuous exercise, *PCr plateau* a plateau in PCr utilisation during continuous exercise following the non-steady state primary and slow component associated with heavy exercise, *NADH above baseline* NADH values above baseline, which is indicative of cytosolic ATP turnover. Sources of ATP production consists of free fatty acids (FFA), intramuscular triglycerides (IMTG), Glucose (from plasma) and muscle Glycogen stores, $\dot{V}O_{2max}$ maximal oxygen uptake, *GET* gas exchange threshold, Δ average work rate of GET and $\dot{V}O_{2max}$, *CP* critical power, not measured. Studies included prescribed exercise just below the critical power or between 46 and 84% of $\dot{V}O_{2max}$

2 Prescribing Exercise Intensity Relative to Maximal Anchors

In both applied and laboratory settings, exercise intensity is often determined based on a percentage of an individual's $\dot{V}O_{2max}$, HR_{max}, $\dot{W}_{max}$, or $\dot{V}_{max}$ [1, 57, 101]. However, in 1978 the first critique of the validity of determining exercise intensity relative to maximal anchors was published [65]. Subsequent research has demonstrated the large variability in metabolic responses (e.g., plasma markers associated with metabolic strain, blood lactate concentration, and oxygen uptake kinetics) when exercise intensity is determined relative to a maximal anchor [62, 64, 98, 102]. Percent maximum prescriptions presume that all participants within a cohort will experience similar homeostatic perturbations to the same relative intensity. While this assumption has shortcomings that have been highlighted by many researchers [62–64, 98, 102, 103], these methods continue to be used to determine exercise intensity. When using these methods to prescribe exercise intensity, authors either extrapolate an associated $\dot{V}O_2$ or HR to a work rate from a GXT or make minor work rate adjustments during exercise to maintain the desired $\dot{V}O_2$ or HR response.

2.1 Maximal Oxygen Uptake

The optimal protocol for establishing $\dot{V}O_{2max}$ is an 8- to 12-min GXT followed by a subsequent VEB [104–106]. The $\dot{V}O_{2max}$ value is deemed valid when the difference between the observed $\dot{V}O_{2max}$ values from the GXT and VEB are within the typical variability of the measurement (i.e., CV = 3%) [6, 14, 104, 105, 107–111]. There is a high test–retest reliability for establishing $\dot{V}O_{2max}$ (CV < 3%) [2]; however, decreasing the GXT slope (increase in work rate

Table 5 (Severe). Evidence of an oxygen uptake kinetic response ($\dot{V}O_2$), blood and intramuscular lactate response, muscle fibre recruitment (based on muscle glycogen utilisation), phosphocreatine (PCr) utilisation for ATP turnover, intramuscular nicotinamide adenine dinucleotide (NADH), and substrate utilisation determined via indirect calorimetry and tracer infusion during severe exercise

References	Exercise intensity	Oxygen uptake kinetics	Blood lactate	Intramuscular lactate	Muscle fibres recruited	PCr utilisation	NADH	Substrate utilisation
Jorfeldt et al. [38]	87% of $\dot{V}O_{2max}$	–	Above baseline and no plateau	No plateau above baseline	–	–	–	–
Vøllestad and Blom [104]	91% of $\dot{V}O_{2max}$	–	Above baseline and no plateau	No plateau above baseline	Type I, IIa, IIax and IIx	–	–	–
Sahlin et al. [95]	100% of $\dot{V}O_{2max}$	–	–	Above baseline	–	–	Above baseline	–
Spriet et al. [105]	100% of $\dot{V}O_{2max}$	–	–	Above baseline	–	–	Type I and II above baseline	–
Poole et al. [43]	105% of CP	$\dot{V}O_2$ slow component and no plateau	Above baseline and no plateau	–	–	–	–	–
Romijn et al. [107]	85% of $\dot{V}O_{2max}$	–	–	–	–	–	–	FFA, IMTG, Glucose, Glycogen
Jones et al. [109]	110% of CP	–	–	–	–	No plateau	–	–
Vanhatalo et al. [33]	105% of CP	$\dot{V}O_2$ slow component and no plateau	Above baseline and no plateau	–	–	No plateau	–	–
Black et al. [80]	105% of CP	$\dot{V}O_2$ slow component and no plateau	Above baseline and no plateau	Above baseline	–	–	–	–

$\dot{V}O_2$ *slow component and no plateau* absence of a plateau in oxygen uptake kinetics with an observed slow component, characterised by statistical analysis ($p > 0.05$) or via on-transient modelling techniques, *no plateau above baseline lactate* lactate values were higher compared to baseline values and continued to rise until the cessation of exercise, *PCr no plateau* no plateau in PCr utilisation during continuous exercise following the non-steady state primary and slow component associated with severe exercise, *NADH above baseline* NADH values above baseline, which is indicative of cytosolic ATP turnover. Sources of ATP production consists of free fatty acids (FFA), intramuscular triglycerides (IMTG), Glucose (from plasma) and muscle Glycogen stores, $\dot{V}O_{2max}$ maximal oxygen uptake, *CP* critical power; — not measured. Studies included prescribed exercise above the critical power or above 85% of $\dot{V}O_{2max}$

relative to time) increases the GXT duration and reduces the reliability [112–115]. Furthermore, decreasing the GXT slope lowers the $\dot{V}O_{2max}$ [14, 116–119] and can cause the agreement between observed $\dot{V}O_2$ values from the GXT and VEB to exceed the error of the measurement [14, 108]. Thus, the validity of the $\dot{V}O_{2max}$ is protocol-dependent and the calculated $\dot{V}O_{2max}$ value is lower and less reliable if the GXT duration exceeds 12 min.

Prescribing exercise intensity as a fixed percentage of $\dot{V}O_{2max}$ requires constant monitoring to verify the desired $\dot{V}O_2$ response is maintained during prolonged exercise [64, 120, 121]. Furthermore, to maintain a constant percentage of $\dot{V}O_{2max}$ when exercise is performed at intensities above moderate requires regular adjustments to the work rate to compensate for the continual increase in $\dot{V}O_2$ (i.e., the slow component) [28, 64]. In lieu of constant monitoring of $\dot{V}O_2$, some researchers extrapolate a work rate from the $\dot{V}O_2$-intensity relationship derived from a GXT. However, this method relies on the assumption of a linear relationship between $\dot{V}O_2$ and work rate [8, 67, 122], whereas this relationship shifts from linear to curvilinear during the latter stages of a GXT [123–125] (Fig. 5). The mean response time (MRT), or time for the pulmonary $\dot{V}O_2$ to reflect the metabolic demand of the working muscle, also increases curvilinearly and becomes more variable [126], which contributes to the increasing departure of the observed $\dot{V}O_2$ response from the assumed linearity. For example, when researchers assigned a constant work rate equivalent to 70% of $\dot{V}O_{2max}$ based on the results of a GXT, four of the nine participants achieved their $\dot{V}O_{2max}$ in under 20 min of exercise

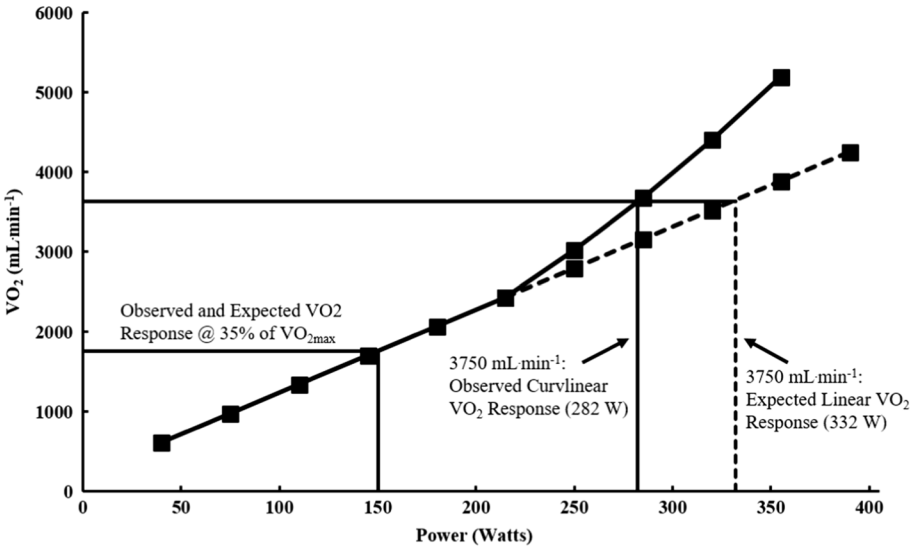


Fig. 5 Expected and observed $\dot{V}O_2$ responses relative to power during a graded exercise test with 3-min stages. During low exercise intensities there is a linear $\dot{V}O_2$-work rate relationship. As exercise intensity increases the relationship between $\dot{V}O_2$ and work rate becomes curvilinear and the observed work rate associated with 70% of $\dot{V}O_{2max}$ (3750 mL·min^{-1} in this example) is lower than that expected for a linear relationship (282 vs. 332 W). Figure based on the data of Zoladz et al. [123]

[8]. Assuming a linear relationship or using a standardised extrapolation technique does not account for the observed curvilinear relationship in the $\dot{V}O_2$-intensity relationship, and it is difficult to achieve a fixed percentage of $\dot{V}O_{2max}$ without constant monitoring/verification.

It is not surprising studies have demonstrated that prescribing exercise intensity as a percentage of $\dot{V}O_{2max}$ (e.g., 60–75% $\dot{V}O_{2max}$) is not a valid method to elicit homogeneous homeostatic perturbations in different individuals. For example, exercise intensity prescribed at 70% of $\dot{V}O_{2max}$ (i.e., an extrapolated work rate corresponding to ~ 70% of $\dot{V}O_{2max}$) resulted in higher concentrations of plasma markers associated with metabolic stress in untrained compared with trained individuals (Fig. 6) [98]. Another study reported a large variability (CV = 52 and 41%, respectively) in the absolute blood lactate observed at 60 and 75% of $\dot{V}O_{2max}$ determined via $\dot{V}O_2$-work rate extrapolation [64]. Furthermore, due to the $\dot{V}O_2$ slow component, modest decreases in work rate were required to maintain $\dot{V}O_2$ at 75% of $\dot{V}O_{2max}$. Another study reported that exercise at 75% of $\dot{V}O_{2max}$ yielded the absence of a lactate steady state [64]. Moreover, higher inter-subject variability for physiological responses and perceptual effort was evident when prescribing exercise intensity relative to $\dot{V}O_{2max}$ compared with prescribing exercise intensity relative to the average work rate associated with the GET and $\dot{W}_{max}$ [8]. A high inter-subject variability was also observed among highly trained cyclists for muscle glycogen utilisation (17–83 mmol·kg^{-1}) and respiratory exchange ratios (0.81–0.97) when cycling at ~ 79% of $\dot{V}O_{2max}$ [121]. Lastly, large ranges of fixed percentages of $\dot{V}O_{2max}$ are associated with the GET (45–74% of $\dot{V}O_{2max}$), the MLSS (69–96% of $\dot{V}O_{2max}$), and CP (60–95% of $\dot{V}O_{2max}$) [9, 127]. Therefore, the evidence does not support the validity of using a fixed percentage of $\dot{V}O_{2max}$ to prescribe exercise intensity to obtain homogeneous homeostatic perturbations or domain-specific physiological perturbations.

2.2 Maximal Work Rate and Peak Treadmill Speed

There is high test–retest reliability for establishing both $\dot{W}_{max}$ and $\dot{V}_{max}$ (CV < 3.0%) [128], but this is constrained to identical GXT protocols. Unfortunately, there is no recommended protocol design for determining either $\dot{W}_{max}$ and $\dot{V}_{max}$ and these values are often reported and compared across studies as though they are independent of protocol design. However, $\dot{W}_{max}$ and $\dot{V}_{max}$ are a function of GXT slope [129, 130] and decreasing the GXT slope results in lower $\dot{W}_{max}$ and $\dot{V}_{max}$ values [14, 117, 131–133]. For example, increasing mean GXT duration from 7 to 30 min (i.e., a slope of 0.83 and 0.14 W·s^{-1}, respectively) resulted in an ~ 108 W decrease in mean $\dot{W}_{max}$ (Fig. 7) [131]. As both $\dot{W}_{max}$ and $\dot{V}_{max}$ are a function of slope, to reasonably compare them between studies and within a study cohort or population the GXT slope must be reported and considered [134].

Prescribing exercise intensity as a percentage of $\dot{W}_{max}$ or $\dot{V}_{max}$ requires only a simple percentage calculation to assign the same relative exercise intensity to participants [98]. However, to our knowledge, no study has compared the individual physiological responses to exercise intensity prescribed at a fixed percentage of $\dot{W}_{max}$ or $\dot{V}_{max}$, determined from the same or different GXT protocols. It is worth noting that the physiological significance of both $\dot{W}_{max}$ and $\dot{V}_{max}$ has been called into question [4, 129, 131]. For example, by simply manipulating the GXT slope a positive or negative training effect can be identified in the absence of an intervention, and controlling for slope in lieu of GXT duration may underestimate the post-intervention value (e.g., $\dot{V}O_{2max}$) as the GXT duration would likely be extended. There is currently no evidence supporting the prescription of

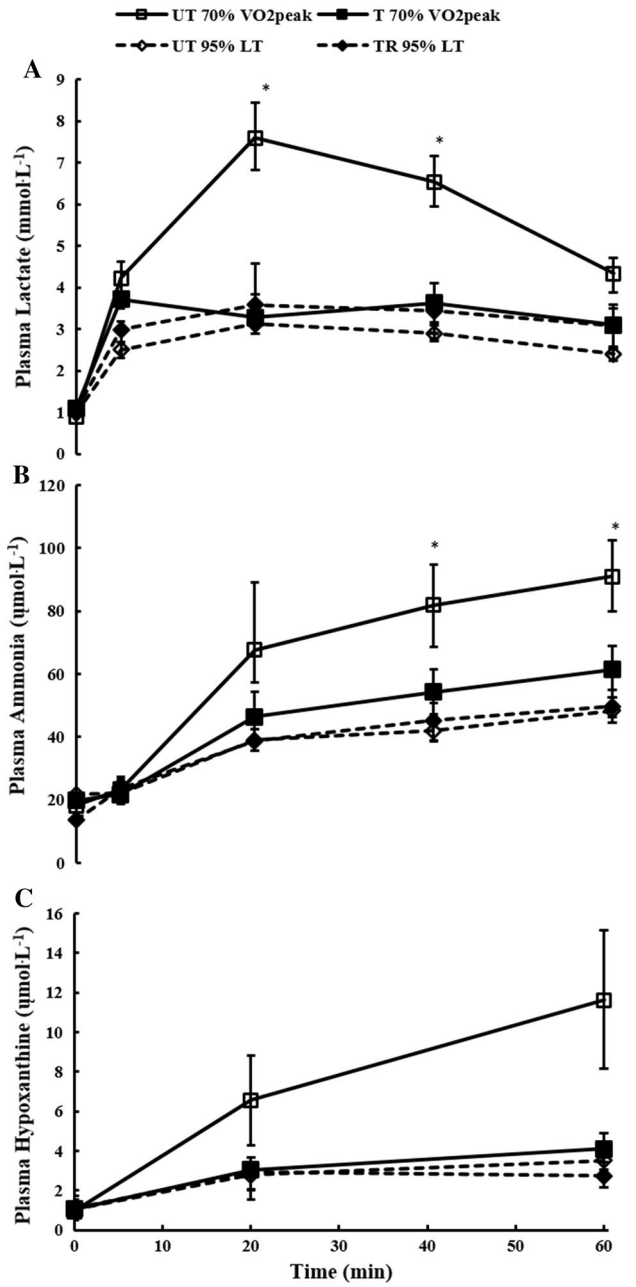


Fig. 6 **a**–**c** Mean ± SD data for **a** Plasma lactate, **b** ammonia, and **c** hypoxanthine values for trained (T) (solid line) and untrained (UT) (dashed line) participants exercising at 70% of $\dot{V}O_{2peak}$ (dark circles) and 95% of the work rate associated with a 1 mmol·L^{-1} increase in blood lactate above baseline (B + 1) (dark triangles). When exercising at 70% of $\dot{V}O_{2peak}$ the plasma lactate (i.e., at 20 and 40 min) and ammonia values (i.e., after 40 and 60 min) were significantly different for the untrained participants compared to all other groups; there were no significant differences for hypoxanthine. * indicates a significant difference from all other trials. Figure based on data from Baldwin et al. [98]

exercise intensity relative to $\dot{W}_{max}$ or $\dot{V}_{max}$ as a valid method for yielding a distinct and/or homogeneous homeostatic perturbations.

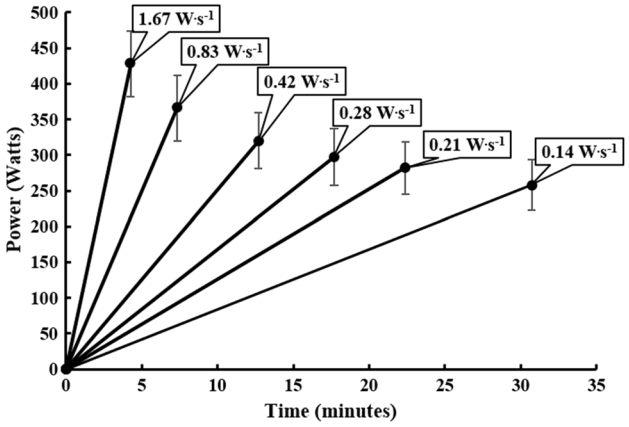


Fig. 7 The relationship between maximum work rate ($\dot{W}_{max}$) and graded exercise test (GXT) duration. $\dot{W}_{max}$ derived from a GXT is a function of the slope (increase in work rate relative to time) ($\dot{W}s^{-1}$). Dark circles represent mean and the error bars the standard deviation. Call outs are the average slope of the graded exercise test. Based on the data of Adami et al. [131]

2.3 Maximum Heart Rate

The HR_{max} is typically determined during a laboratory GXT, which occurs in conjunction with the measurement of $\dot{V}O_{2max}$, and has a high test–retest reliability (CV = 0.9–3.2%) [2, 112–115, 135–137]. The HR_{max} does not appear to be influenced by the GXT protocol [138, 139]; however, higher values are observed during field testing (> 4 beats·min^{-1}) [140–142]. As with $\dot{V}O_{2max}$, prescribing exercise intensity as a percentage of HR_{max} requires constant monitoring of HR or extrapolation of the HR–work rate relationship. Similar to $\dot{V}O_2$ during heavy and severe exercise, there is an observed HR slow component [143]; thus, extrapolation of a HR is subject to similar limitations observed when extrapolating $\dot{V}O_2$ from incremental data. Notwithstanding the known limitations of prescribing exercise intensity as a percentage of HR_{max}, given its simplicity, it remains a staple for prescribing exercise intensity.

Despite its common usage, only one study has investigated the validity of using a percentage of HR_{max} to normalise exercise intensity [65]. When participants exercised at 60, 70, and 80% of HR_{max}, one of the 31 participants was above their VT (as defined by Wasserman et al. [144].) when exercising at 70% of HR_{max}, whereas 17 of the 31 participants were above the VT while exercising at 80% of HR_{max}. Furthermore, large ranges of fixed percentages of HR_{max} are associated with the GET (60–90% of HR_{max}) and MLSS (75–97% of HR_{max}) [127]. These findings suggest prescribing exercise relative to fixed percentages of HR_{max} is not be a valid method to achieve distinct or homogeneous homeostatic perturbations.

2.4 Conclusion: Prescribing Exercise Intensity Relative to Maximal Anchors

Although there is a high test–retest reliability for $\dot{V}O_{2max}$, $\dot{W}_{max}$, $\dot{V}_{max}$, and HR_{max}, based on the laboratory and field evidence, prescribing exercise intensity as a fixed percentage of these maximal anchors has substantial shortcomings as a means for normalising exercise intensity between individuals. There is a large variability in the physiological responses at a fixed percentage of $\dot{V}O_{2max}$ and this response becomes even more variable as the percentage of $\dot{V}O_{2max}$ increases [64, 121]. The GXT protocol influences the determination of $\dot{V}O_{2max}$, $\dot{W}_{max}$, and $\dot{V}_{max}$; furthermore, the GXT slope modulates both the $\dot{V}O_2$- and the HR-work rate relationship. Thus, extrapolating a work rate from a GXT fails to account for the curvilinear relationship between $\dot{V}O_2$/HR and work rate (Fig. 5). Studies that prescribe exercise intensity using these methods typically do not specify if they extrapolate the work rate or make minor work rate adjustments to maintain the desired $\dot{V}O_2$ and HR response. If employing the extrapolation technique, we recommend describing the intensity as a percentage of $\dot{W}_{max}$ or $\dot{V}_{max}$ as it would be erroneous to describe the prescribed intensity as a percentage of $\dot{V}O_{2max}$ or HR_{max} without monitoring the oxygen uptake or heart rate response. Exercise intensity prescribed relative to the maximal anchors results in an indistinct and heterogeneous homeostatic perturbation, and fixed percentage cannot be used as a valid proxy for submaximal anchors. Given these limitations, it is not recommended to prescribe exercise intensity relative to the maximal anchors as a means to elicit distinct or homogeneous homeostatic perturbations.

3 Prescribing Exercise Relative to Submaximal Anchors

Recent reviews have advocated the use of submaximal anchors in lieu of maximal anchors, including the LT_1, LT_2, GET, VT, RCP, MLSS, CP, and CS, to prescribe intensity [2, 134]. These methods rely upon expired air and blood lactate responses, or the assumed depletion of the usable anaerobic capacity, to be established. These anchors can also be used to establish training levels/zones, whereby each method is used as a reference point for demarcating different training levels/zones. For example, LT_1, LT_2, and $\dot{W}_{max}$ derived from a GXT have been used to establish five aerobic training levels (i.e., L1–L5), which can also be characterised by $\%HR_{max}$, absolute blood lactate concentrations, and ratings of perceived exertion (Fig. 1 and Table 1) [16, 18]. The GET, VT, LT_1, LT_2, RCP, and MLSS anchors have been used in training intensity distribution models to describe three exercise intensity zones, where each zone has been characterised by $\%HR_{max}$, $\%\dot{V}O_{2max}$, and absolute blood lactate values (Fig. 2 and Table 2) [17, 19–21, 145]. The overarching shortcoming of these models is they are constrained by their delineating anchors regardless of whether the anchor truly represents a shift in the metabolic state of the working muscle. For example, any LT_1 method could, by definition, be a valid delineator of L1 and L2 or zone 1 and zone 2, regardless of its validity to yield a distinct or homogeneous homeostatic perturbation. Based on the domains of exercise, a LT_1 method that overestimates the boundary between moderate and heavy exercise would not be valid approach to determine exercise intensity; however, the level/zone models do not make such a distinction.

The domains of exercise differ from the training zones/levels in that they are not defined by $\%HR_{max}$ or $\%\dot{V}O_{2max}$, or absolute blood lactate concentrations, nor submaximal anchors, but rather their distinct homeostatic perturbations (i.e., $\dot{V}O_2$ kinetic and blood lactate response) (Tables 4 and 5; Figs. 3 and 4). The moderate domain is typically characterised by a plateau of $\dot{V}O_2$ and blood lactate concentrations near baseline levels, the heavy domain by an observed 'slow component' of $\dot{V}O_2$ with a delayed steady state and a rise in blood lactate above baseline with a subsequent plateau, and the severe domain by a 'slow component' without a steady state of $\dot{V}O_2$ and a continual increase in blood lactate [24, 28, 35, 51, 52, 146, 147]. Nonetheless, even though the domains are not defined by sub-maximal anchors, these anchors are often used to demarcate or estimate the boundaries between the domains. The validity of using submaximal anchors to determine the domains of exercise can be established by determining if exercise relative to these anchors produces distinct and homogeneous homeostatic perturbations (i.e., domain-specific) regardless of an individual's fitness level. Therefore, in this review, we have assessed the validity of using submaximal anchors to prescribe exercise intensity based on their ability to yield domain specific homeostatic perturbations.

3.1 Submaximal Anchors Based on Blood Lactate Measurements

Prescribing exercise intensity based on blood lactate measurements appears to be a favoured method to normalise exercise intensity compared with fixed percentages of $\dot{V}O_{2max}$ [98]. The blood lactate values measured during a GXT are used to calculate LT_1 and LT_2, and to define training levels/zones [148] (Figs. 1 and 2; Tables 1 and 2). The LT_1 (sometimes referred to as the aerobic threshold) derived from a GXT represents the rise in blood lactate above baseline, and is often assumed to also demarcate the moderate and heavy domains of exercise. In contrast, LT_2 (sometimes referred to as the anaerobic threshold) represents the acceleration of blood lactate and purportedly demarcates the heavy and

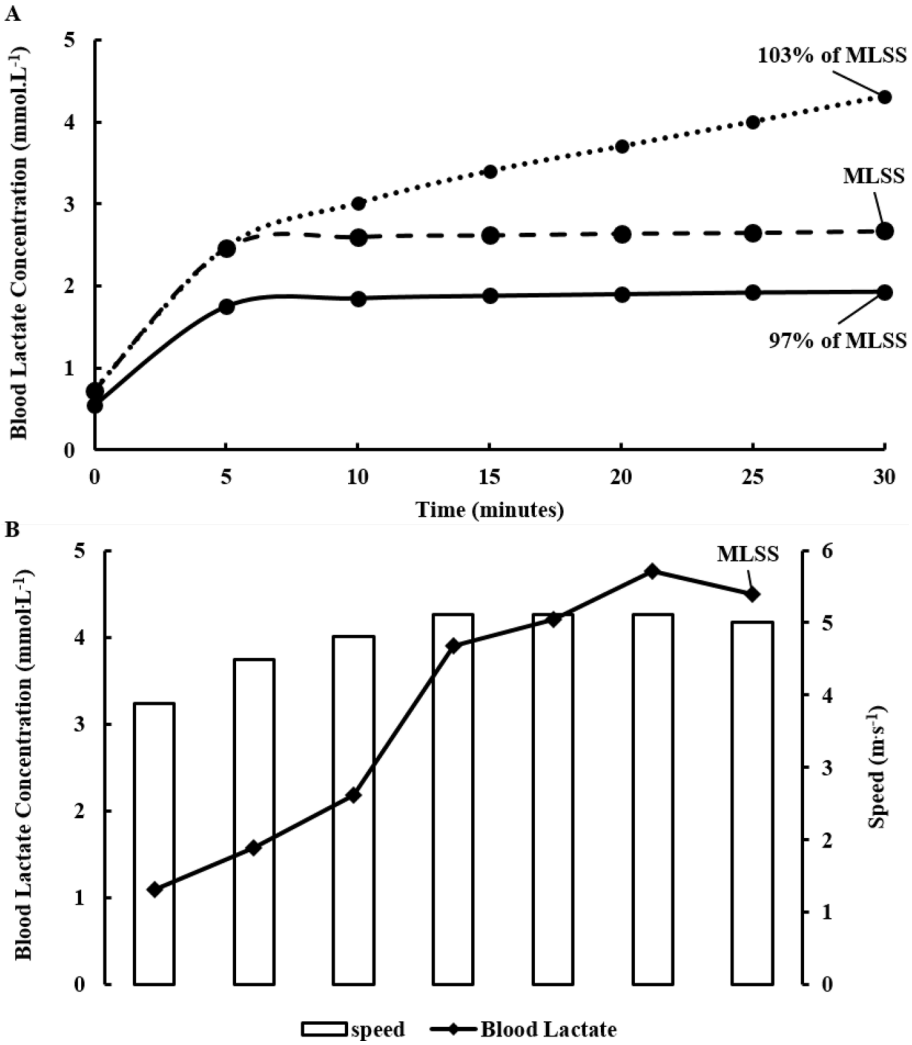


Fig. 8 **a** Representative blood lactate response to exercise performed at 97, 100 and 103% of the MLSS (established using the traditional criteria). Blood lactate increased 0.7, 0.8, and 1.3 mmol·L^{-1}from 10 to 30 min at 97, 100, and 103% of the MLSS, respectively. **b** Representative blood lactate response using criteria developed by Hering et al. [149]. The threshold criteria was achieved where blood lactate increased ≥ 0.5 mmol.L^{-1} and was ≥ 4.0 mmol.L^{-1} without a change in speed, as described by the "Lactate Threshold 2" criterion illustrated in Fig. 1; Ref. [149]. Speed was then decreased by 0.1 m·s^{-1} to confirm the MLSS

severe domains of exercise [148]. Alternatively, blood lactate values measured during a series of constant work rate bouts [5], or single exercise bouts with real-time work rate adjustments [149], are used to establish the MLSS. The MLSS represents the highest intensity where blood lactate appearance and disappearance is in equilibrium and has also been used to demarcate the heavy and severe domains of exercise [150] (Fig. 8a, b).

3.2 Lactate Thresholds

There is no overall consensus regarding the GXT protocol design to establish the LT. A stage length of at least 3 min has been recommended [119], but stage lengths from 1 to 10 min have been used [14, 151]. A customised approach for individualised GXT design has been proposed to ensure a homogeneous GXT duration [3, 14]. There are at least 30 different methods that can be used to calculate the LT, and the calculated work rate at each LT in the same individual can vary ~ 30% depending on the method chosen [148] (Fig. 9). Furthermore, the work rate associated with a specific LT method is influenced by the GXT protocol [14, 132]. Thus, the validity of any lactate threshold to identify the boundary between the domains of exercise will depend on the GXT protocol and the LT calculation method.

The test–retest reliability for select LT methods has been investigated, including a visual inspection point (CV = 51.6%), the D_{max} (CV = 3.8–10.3%), the onset of blood lactate accumulation (OBLA) of 4.0 mmol·L^{-1} (CV = 3.1–8.2%), and baseline plus 0.5, 1.0, and 1.5 mmol·L^{-1} (CV = 1.2–3.7, 3.4–12.6, and 3.1–3.4%, respectively) [151–153]. The reliability of many accepted LT methods has yet to be confirmed, even though these methods are often used for prescribing exercise intensity and for delineating the exercise domains.

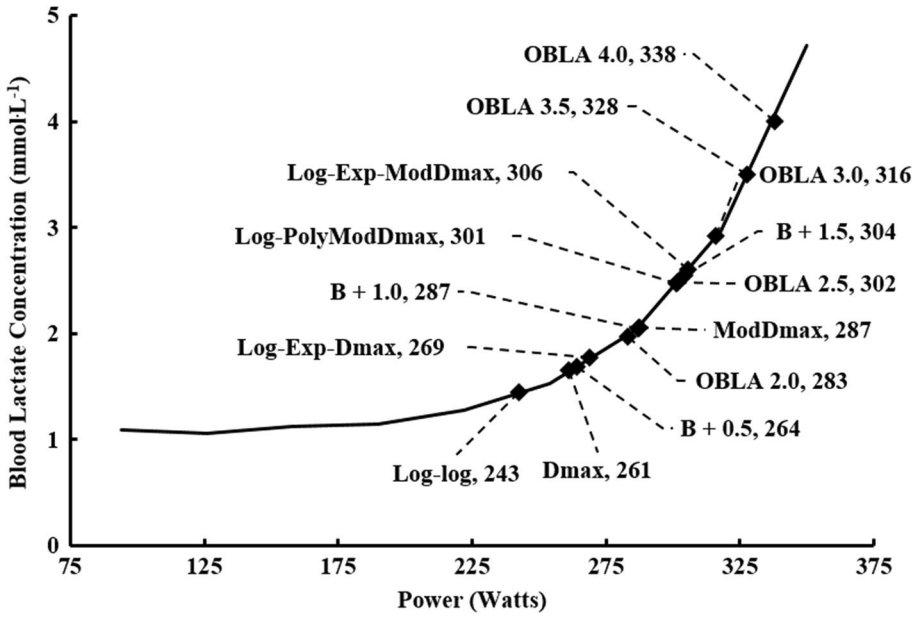


Fig. 9 Representative lactate threshold (LT) curve with the LT calculated using 14 different methods (LT values in Watts appear beside each method). *Log–log* power at the intersection of two linear lines with the lowest residual sum of squares, *log* using the log–log method as the point of the initial data point when calculating the D_{max} or modified D_{max}, *poly* modified D_{max} method calculated using a third order polynomial regression equation, *exp* modified D_{max} method calculated using a constant plus exponential regression equation, *OBLA* onset of blood lactate accumulation; B + lactate value (i.e., 0.5, 1.0, or 1.5 mmol.L^{-1}) = the absolute intensity where blood lactate increases by the indicated value above baseline. Figure based on data from Jamnick et al. [14]

3.2.1 LT_1

The LT_1 has been used to determine the boundary between L1 and L2 [16, 18], and zones 1 and 2 [17, 21, 154], and is assumed to demarcate the moderate and heavy exercise domains [4, 146]. The methods typically associated with determining LT_1 are the visual inspection point, the log–log LT, or an increase in blood lactate of 0.5 mmol·L^{-1} above baseline; these methods are all highly correlated (ICC ~ 0.98) [155]. Despite these correlations, the visual inspection point is unreliable, the baseline + 0.5 mmol·L^{-1} has favourable reliability, and the reliability of the log–log LT is uncertain [151, 153, 155]. The log–log LT and baseline + 0.5 mmol·L^{-1} method are least influenced by GXT protocol design [14] and should be assessed for their validity to delineate the moderate and heavy domains of exercise. There is no research directly investigating the validity of any estimate of LT_1 to delineate the domains of exercise.

Despite the lack of relevant research, prescribing exercise intensity relative to an LT method appears to yield a more homogeneous homeostatic perturbation compared to exercise intensity prescribed as a percentage of $\dot{V}O_{2max}$ [98]. Exercise intensity prescribed at 95% of the work rate associated with an increase of 1 mmol·L^{-1} above baseline (baseline + 1.0 mmol·L^{-1}) yielded more homogenous homeostatic perturbations than exercise at 70% of $\dot{V}O_{2max}$ [98] (Fig. 6). This study demonstrates that this LT method is superior compared to fixed percentages of $\dot{V}O_{2max}$ to yield a homogeneous homeostatic perturbation. Nonetheless, there is no evidence supporting the efficacy of baseline + 1 mmol·L^{-1} as a valid method to delineate any of the domains exercise [14, 151].

3.2.2 LT_2

The LT_2 is often accepted as a valid threshold to demarcate training levels/zones, as well as the heavy and severe exercise domains. Although there is no research directly investigating the validity of LT_2 to make these delineations, its legitimacy has been assessed via concurrent validity with the MLSS (discussed in more detail in Sect. 3.3). There are at least 30 methods to calculate the LT_2 and only select methods have been suggested to provide a valid estimate of the MLSS [12, 14, 73, 151, 153, 156–159]. It is worth noting, however, that the authors advised caution regarding these outcomes as they: were specific to the testing procedures, have not been reproduced, or lacked a comprehensive statistical analysis. Other studies concluded that the LT_2 could not validly estimate the MLSS [12, 14, 73, 151, 153, 156–159]. Furthermore, the validity of LT_2 is often accepted via a single statistical value (e.g., r or p value) or in comparison to other selected methods. Although a high correlation ($r > 0.90$) is often used to establish validity, it is not a sufficient statistical procedure alone to establish validity [160].

Instead, it has been recommended that validity should be based on a combination of statistical procedures (e.g., Pearson product moment correlation, Lin's concordance correlation coefficient, effect size difference, intraclass correlation coefficient, coefficient of variation) or, ideally, via the use of a priori criteria (e.g., the bias and precision of a method should be less than the standard error of the criterion method to establish concurrent validity) [161].

Of the more than 30 LT_2 methods, one (i.e., baseline + 1.5 $mmol \cdot L^{-1}$) has been reproduced as a valid estimation of the MLSS based on 2 studies that recruited trained cyclists and employed a GXT with 3-min stages [14, 158]. However, this result could not be confirmed with any other GXT stage length [14, 151, 162]. The D_{max} [71] and Modified D_{max} [72] methods are curve-fitting LT models that, despite no evidence to support the validity of the original methods to identify the MLSS, or to delineate the heavy and severe domains, remain staple LT methods. The D_{max} and Modified D_{max} methods are influenced by stage length [14], starting intensity [75], regression model employed [74], and the final lactate value [163]. Despite these limitations, a recent study demonstrated that a Modified D_{max} method using the log–log LT as the initial lactate value from a 4-min stage GXT appears to yield high agreement with the MLSS [14]; however, the reproducibility of these results remains uncertain. Fixed blood lactate concentrations (e.g., 2.0 and 4.0 $mmol \cdot L^{-1}$) are also commonly accepted methods that have been proposed to delineate the moderate/heavy and heavy/severe domains, respectively [77, 164]. It is worth noting, however, the original authors cautioned against the use of fixed blood lactate concentrations to estimate the MLSS [73] as there is often a broad range of blood lactate concentrations when exercise is performed at the domain boundaries [35]. The validity of other LT_2 methods is yet to be established [14]. Furthermore, the assessment of validity should be based on domain-specific homeostatic perturbations rather than agreement with another submaximal anchor.

3.3 Maximal Lactate Steady State

The original protocol to establish the MLSS requires a series of 30-min constant work rate bouts, where the rise in blood lactate is < 1.0 $mmol \cdot L^{-1}$ from the 10th to the 30th min (Fig. 8a), or a single visit exercise bout requiring a rise in blood lactate above steady state with modest work rate adjustments [149] (Fig. 8b). The 30-min exercise bouts and single visit protocol have a CV of 3.0 and 1.9%, respectively [149, 165]. The criterion of the MLSS during the 30-min exercise bouts relies on blood lactate kinetics and a time limit [5, 13, 14]. In contrast, the single-visit protocol relies on rapid accumulation of lactate resulting from modest changes in workload [149]. The MLSS is reliable and can be established with two criteria (i.e., 30-min and single visit); however, it is uncertain if these criteria are equivalent (i.e., result in a similar work rate and $\dot{V}O_2$) or whether either method produces a valid anchor to delineate the heavy and severe domains of exercise.

The MLSS derived from a series of 30-min exercise bouts purportedly corresponds to the maximal metabolic steady state [10], and it is assumed exercise performed above the MLSS yields a homeostatic perturbation consistent with the severe exercise domain (i.e., no $\dot{V}O_2$ or blood lactate plateau). Although exercise performed above the MLSS yields blood lactate values above the steady-state criterion, a $\dot{V}O_2$ steady state has been reported [37, 39] that occasionally precludes attainment of $\dot{V}O_{2max}$ [10, 11]. These responses are more consistent with the heavy domain of exercise, which has led to criticism of this criterion [166]; specifically, researchers have criticised the arbitrary blood lactate steady-state criteria (i.e., a change of < 1.0 $mmol \cdot L^{-1}$ from the 10th to the 30th min) and a 30-min time limit [13]. An arbitrary time limit to determine any submaximal anchor or index should be avoided as the time to fatigue at the maximal metabolic steady state varies considerably [13, 37, 167–169]. Furthermore, a steady state for blood lactate can be achieved beyond 30 min for exercise intensities that might otherwise be concluded to be above the MLSS [170]. Although an accepted submaximal anchor for determining a physiological steady state, the 30-min MLSS typically underestimates (~4%) another recommended anchor to delineate the heavy and severe exercise domains (i.e., CP/CS) (see Jones et al. [13] for further critique).

The recent publication of a single-visit MLSS protocol [149], which requires real-time work rate adjustments based upon blood lactate responses, appears to be a promising alternative to the accepted MLSS test. Establishing the single visit MLSS requires a stage above the MLSS; the purpose of this stage is to verify the MLSS by eliciting a rapid accumulation of lactate relative to a modest increase in work rate [149]. This response is indicative of enhanced motor unit activity and the inability to solely meet ATP demands via oxidative phosphorylation [149, 171, 172]. Although the verification stage provides evidence of non-steady-state exercise, the validity of the single visit MLSS to delineate the heavy and severe domains of exercise needs to be confirmed.

3.4 Submaximal Anchors Based Upon Expired Air

Submaximal anchors based upon expired air (i.e., GET, VT and RCP) detect disproportionate changes in ventilation and non-metabolic CO_2 production relative to VO_2 or work rate [69, 70, 144]. These anchors are assumed to be indicative of the shift in metabolic rate and substrate utilisation within the working muscle, and to demarcate the domains of exercise

[38, 39]. Prescribing exercise relative to either the VT or RCP has been reported to yield a more consistent aerobic adaptation than HR-based exercise intensity prescription over a 12-week period [173, 174]. Exercise prescribed relative to both the VT and RCP also attenuated individual variation in training responses compared to HR-based exercise intensity prescription [173, 174]. The likely explanation is the better ability of the VT to normalise exercise intensity compared to HR [65]. However, there is limited evidence that these anchors are valid for yielding domain-specific homeostatic perturbations.

3.4.1 Ventilatory/Gas Exchange Threshold

Both the GET and VT occur at an intensity similar to LT_1, as their mechanistic basis is closely tied [69, 151, 175–180]. The GET and VT are both determined by a non-invasive method that indirectly measures a disproportionate increase in non-metabolic CO_2 production, a consequence of H^+ accumulation and increased cytosolic ATP turnover [32, 68–70, 181, 182]. The GET is determined as an intensity that elicits an increase from steady state to an excess production of CO_2 (Fig. 10a) [3, 69]. The VT is determined as the first inflection point in $\dot{V}_E$ (Fig. 10b), as a systematic increase in $V_E/\dot{V}O_2$ (Fig. 10c) [182] and $P_{ET}O_2$ (Fig. 10d), and the point where $P_{ET}CO_2$ begins to plateau (Fig. 10e) [183]. A high test–retest reliability of both the GET and the VT has been established (CV = 2.0–3.5%) [151, 153]. The GET and VT are influenced by GXT slope and dependent on a standardised technique (i.e., a MRT of 60 s) to extrapolate the corresponding $\dot{V}O_2$ to an associated work rate. Specifically, longer duration GXTs yield a lower $\dot{V}O_2$ and work rate associated with either the GET or VT when a MRT of 60 s is employed [125, 133, 179]. As these anchors are influenced by GXT slope [133, 179, 184], the optimum GXT duration to determine both the GET and VT is 8–12 min [3, 185]. Furthermore, identifying these anchors is dependent on the method chosen and it appears there is superior confidence using a computer vs. manual technique [186]. Use of only one of the aforementioned GET or VT methods results in poor reliability compared to a combination of GET and VT methods [176]. Therefore, it is recommended to use computerised methods to establish the GET and VT and to use a combination of the available GET and VT methods to maximise reliability.

There is some evidence to support the validity of both the GET and VT in normalising exercise intensity and yielding domain-specific homeostatic perturbations. Exercise performed below the GET or VT yields a $\dot{V}O_2$ plateau, and blood and intramuscular lactate concentrations that remain at baseline. In contrast, exercise above the GET or VT results in a $\dot{V}O_2$ 'slow component', a plateau of blood lactate above baseline, and an increase in intramuscular lactate above resting levels/values [52, 56, 147]. It is worth noting, however, that these homeostatic responses were measured distant from the GET and VT (i.e., 80, 90 and 120% of the GET or VT). To confirm the validity of either the GET or VT future research should employ a customised GXT protocol, incorporate multiple GET or VT criteria (Fig. 10), and measure the on-transient oxygen uptake kinetics in response to constant work load exercise performed at the limits of agreement of the GET or VT (e.g., ± reliability of the GET or VT).

3.4.2 Respiratory Compensation Point

The RCP, also referred to as the second ventilatory threshold (VT_2), is a non-invasive marker caused by hyperventilation consequent to an increase in H^+ accumulation that indicates a concomitant increase in blood lactate and H^+ greater than the rate of disposal [70, 181, 187]. The optimum GXT duration to establish the RCP is 8–12 min [3], and it is characterised by a second breakpoint in $\dot{V}_E$ (Fig. 11a), a clear break point in $\dot{V}_E/\dot{V}CO_2$ (Fig. 11b), and the point where $P_{ET}CO_2$ begins to fall after an apparent steady state (Fig. 11c) [68–70]. Similar to both the GET and VT, this anchor is also influenced by the GXT slope [179, 188–190] and typically determined using a standardised 60-s MRT extrapolation technique [125, 126]. This standardisation does not account for the increase in MRT with increased exercise intensity [125, 126, 191]. This increase in the MRT is attributed to the slow component during heavy/severe exercise, which conflates the gain in $\dot{V}O_2$ relative to work rate [39, 51, 184, 189] even when adjusting for the MRT of $\dot{V}O_2$ [125, 184]. This sequence leads to a disassociation between the work rate derived from a GXT and the $\dot{V}O_2$ elicited during a constant work rate exercise bout [39, 190]. Specifically, when performing constant work rate exercise at the RCP derived from a GXT, the $\dot{V}O_2$ would be higher than the RCP $\dot{V}O_2$ observed during the GXT. Despite this uncoupling, the RCP $\dot{V}O_2$ is independent of GXT slope [188, 190]. It is worth noting, however, that recent publications have derived techniques to account for the nonlinearity of the $\dot{V}O_2$–work rate relationship during a GXT [126, 192] and should be employed in lieu of the standardised approaches. A high test–retest reliability of the RCP has been established (CV = 1.9–2.1%) [151, 153]. Similar to the GET and VT, we recommend the use of a combination of available computerised methods to establish the RCP and to maximise reliability.

To our knowledge, no research has directly confirmed the validity of the RCP to yield domain-specific homeostatic perturbations. However, the validity has been implied based on concurrent validity with CP and/or the MLSS [38, 39, 190], where the agreement between the $\dot{V}O_2$ associated with each exercise intensity was based on null hypothesis testing.

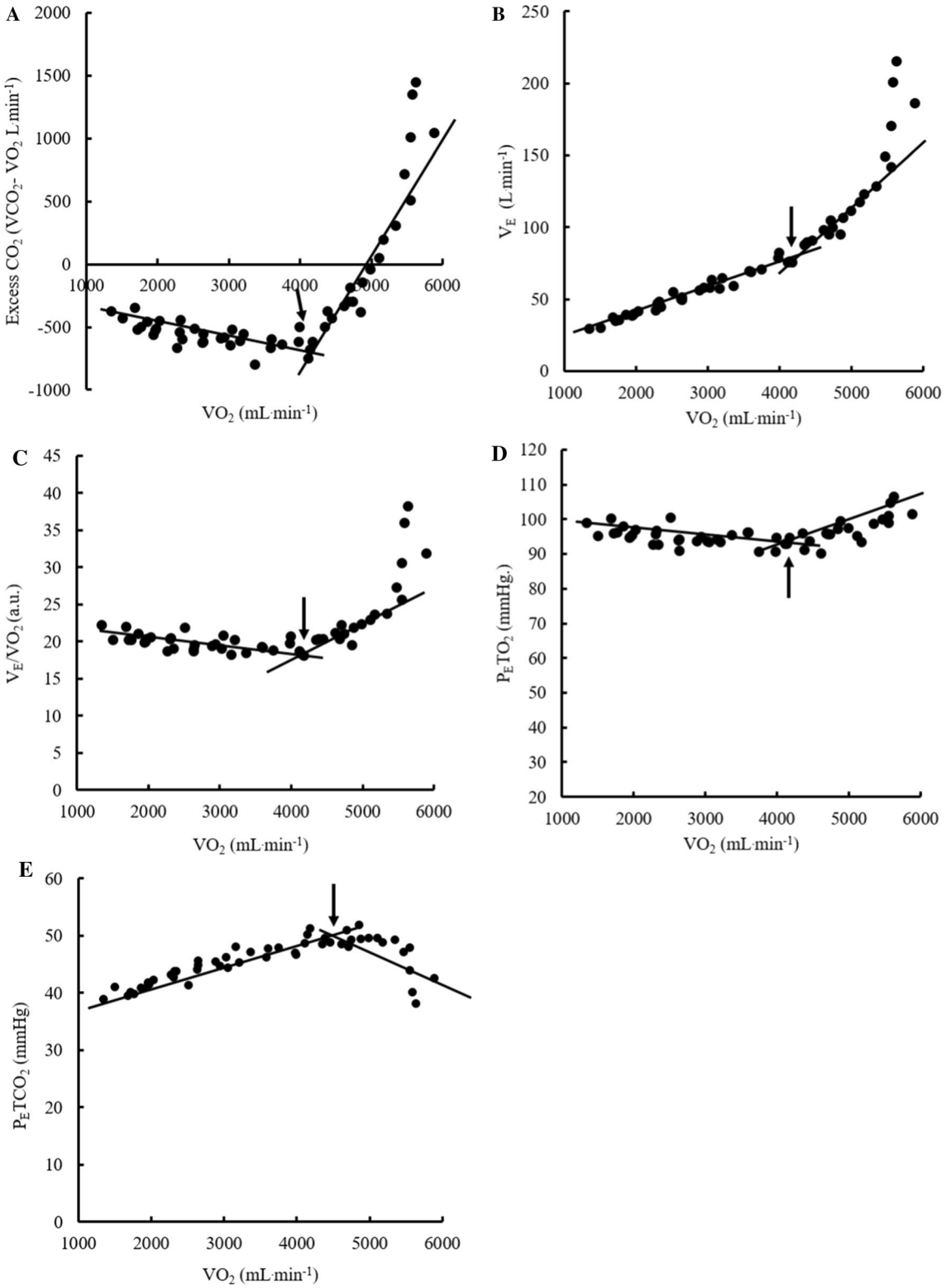


Fig. 10 **a-e** Representative expired air and blood lactate data from an incremental/graded exercise test illustrating the method(s) to determine the gas exchange threshold (GET) and the ventilatory threshold (VT). **a** Disproportionate increase in non-metabolic [excess $CO_2 = (\dot{V}CO_2 - \dot{V}O_2)$] CO_2, **b** the first break point in ventilation ($\dot{V}_E$) production relative to $\dot{V}O_2$ consumption ($\dot{V}O_2$), **c** systemic increase in $\dot{V}_E/\dot{V}O_2$, **d** systemic increase in pressure of end tidal oxygen consumption ($P_{ET}O_2$), and **e** plateau in pressure of end tidal carbon dioxide expiration ($P_{ET}O_2$) following an increase

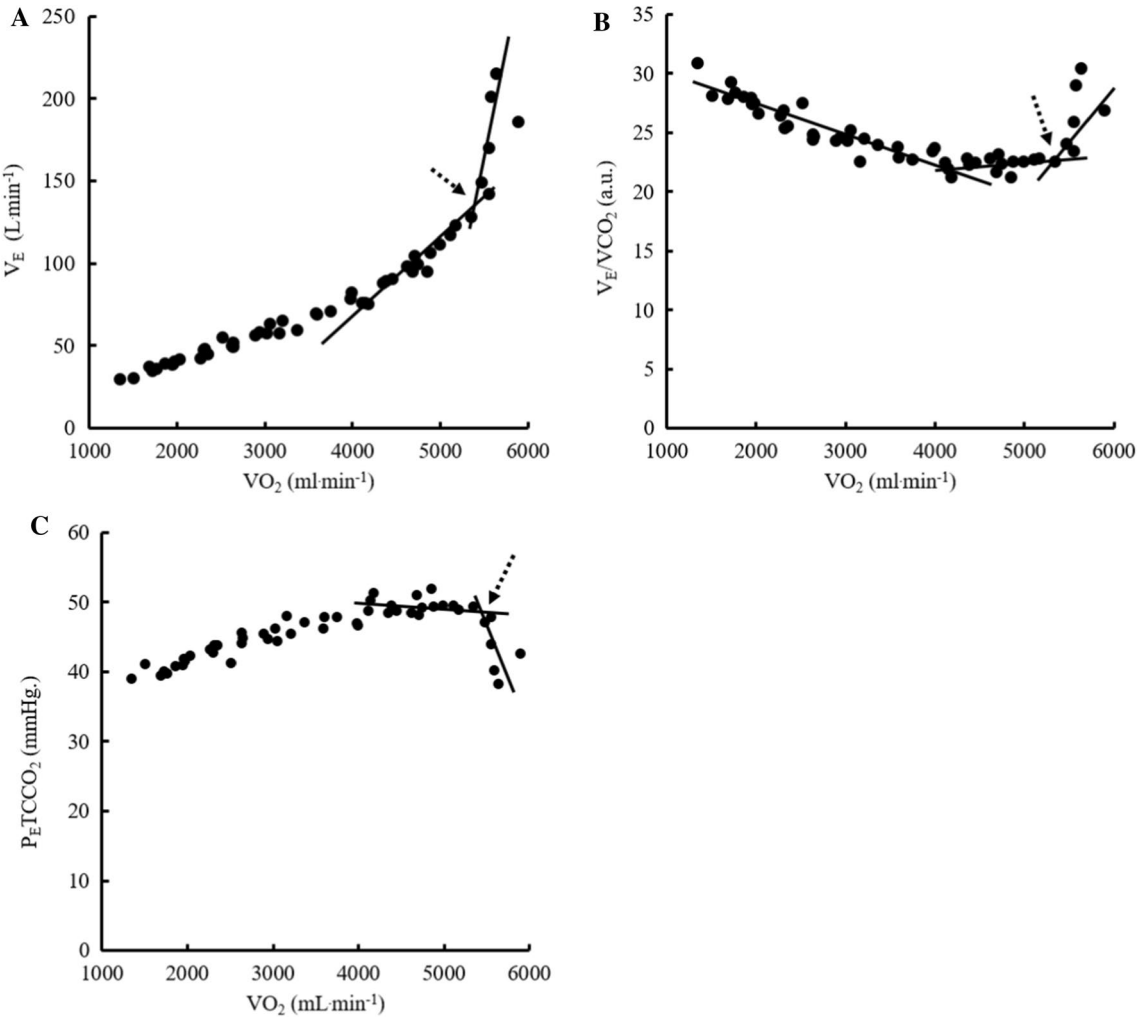


Fig. 11 **a-c** Representative expired air and blood lactate data from an incremental/graded exercise test illustrating the method(s) to determine the respiratory compensation point (RCP). **a** The second break point in ventilation ($\dot{V}_E$), **b** a breakpoint in $\dot{V}_E$ relative to CO_2 expiration ($\dot{V}_E/\dot{V}CO_2$) following a plateau), and **c** second breakpoint in pressure of end tidal carbon dioxide expiration ($P_{ET}O_2$) following a plateau

The statistical rigour of confirming equivalence should go beyond null hypothesis testing (e.g., via the use of a priori criteria, such that the bias and precision of a method is less than the standard error of the criterion method) [161, 193]. However, given the disassociation between work rates derived from a GXT and the $\dot{V}O_2$ derived from constant work rate exercise, some investigators advocate that the RCP cannot be used as a surrogate for either the MLSS or CP [184, 193, 194]. Moreover, although a GXT slope of 0.083 W·s^{-1} yielded high agreement in work rate between the RCP and CP in recreational men, it is worth noting this slope would yield a 40-min GXT duration for a participant with a $\dot{W}_{max}$ of 200 W [190]. Although there is no research refuting the efficacy of the RCP as a valid method to yield domain-specific homeostatic perturbations, it is unlikely to be an appropriate method largely due to the influence of the curvilinear $\dot{V}O_2$–work rate relationship. Lastly, the evidence indicates this method should be avoided as a surrogate for the MLSS or CP—unless employing long duration GXTs to derive the RCP.

3.5 Critical Power/Speed

Both the CP and CS represent the highest intensity without a progressive loss of homeostasis [4] and are often regarded as valid determinants of the boundary between heavy and severe exercise [4, 13, 24, 25, 35, 195]. Establishing the CP or CS requires either a series of exercise bouts (traditional

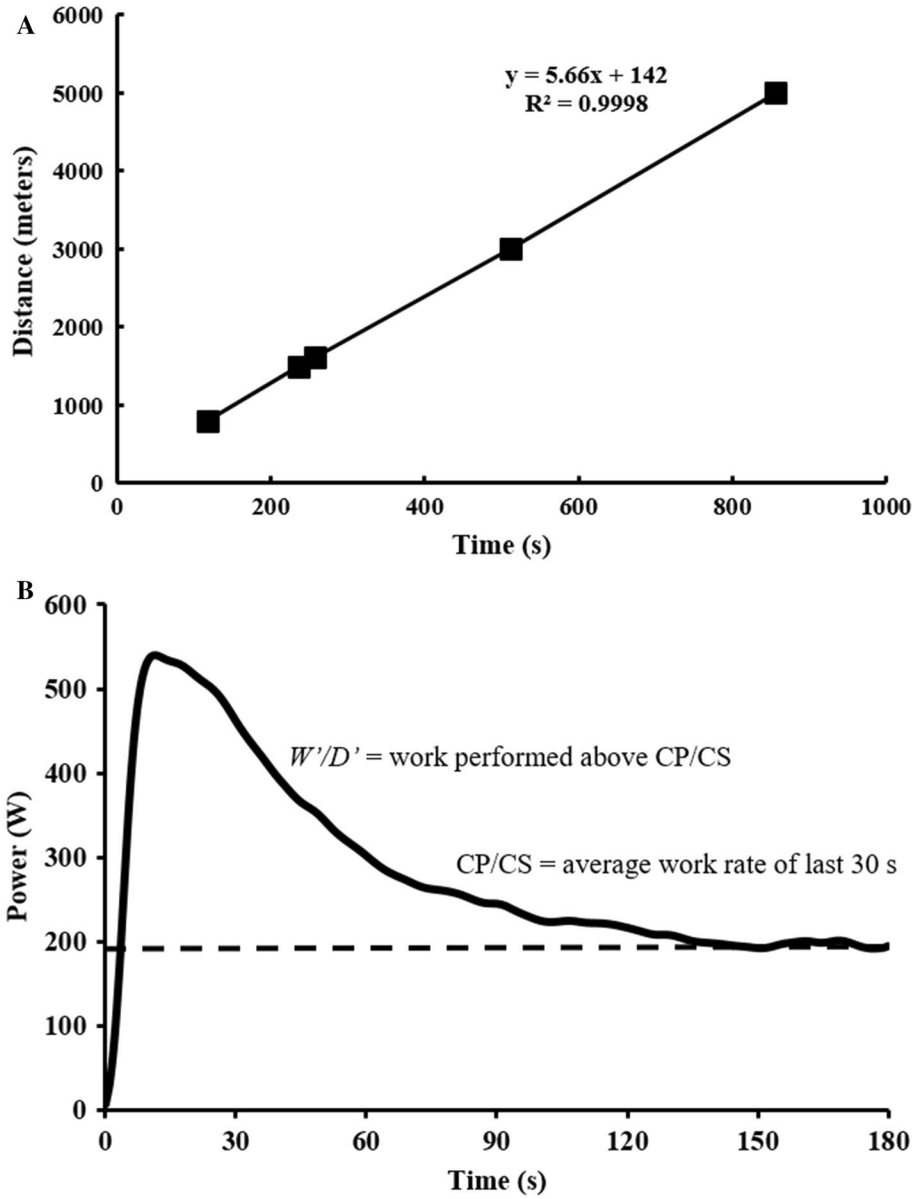


Fig. 12 **a** Linear distance-time model from race performances (800 m, 1500 m, mile, 3000 m and 5000 m) used to calculate critical speed (slope; 5.7 $m \cdot s^{-1}$) and the curvature constant (intercept; *D'* = 142 m). Times retrieved from IAAF.com; athlete code: 14,564,446. **b** Work rate profile from a 3-min, all-out test (3MT). The dashed line represents CP/CS—the average work rate from the last 30 s of the 3MT. *W'/D'* represents the work performed above CP/CS—the average work rate of the first 150 s—CP/CS)

method) above the expected CP or CS, or a single-effort 3MT, to calculate the CP or CS and the curvature constant (*W'* or *D')* [6, 7, 35, 78–82] (Fig. 12).

Establishing CP or CS using the traditional method is subject to restrictions, where the CP and CS are influenced by the duration of the bouts [196–199], the mathematical model employed [200], and the type of bout (i.e., time trial vs. exhaustive constant work rate bout) [201, 202] (see Muniz-Pumares [203] for further review). At least two exercise bouts are required to establish CP or CS via the traditional method, and three are recommended to establish the goodness of fit (r^2) of the regression equation and the standard error of estimate of the CP or CS and *W' or D'* (Fig. 12A). The recommended duration of the exhaustive exercise bouts is 2–15 minutes [4, 35, 196, 204], with at least a 5-min difference between the shortest and longest trials [205–207]. The number of trials chosen does not appear to influence CP [208, 209]. By manipulating the duration of running time trials (i.e., 2, 5, and 10 min. vs. 3, 7, and 12 min), there were marked differences in CS and D' (i.e., ~ 0.12 $m \cdot s^{-1}$ and ~ 20.3 m respectively) [210]. The mathematical model chosen may also influence CP and CS, where the linear and nonlinear models yield higher and lower estimations of CP and CS, respectively [110, 200, 203, 206, 211–216]. Despite recommendations for multiple mathematical models [200], there is no consensus on best practice to establish the CP or CS. Time trials may yield higher CP and CS values compared to exhaustive constant work rate bouts [207, 217, 218],

as well as superior test–retest variability [202]. There is a high reliability (CV = 2.4—6.5%) [210, 219, 220], but it is recommended that familiarisation trials be implemented to increase reliability [203]. The traditional method is subject to many restrictions that influence the establishment of the CP or CS and the *W'* or *D'*; furthermore, there is no current established protocol to confirm the identified parameters.

The 3MT requires a single-visit, all-out effort test, where the average work rate computed from the last 30 s is the CP or CS [6, 7] (Fig. 12b). There is a high test–retest reliability of the CP and CS metrics derived from the 3MT (CV = 1.2–6.7%) [78, 221, 222]. The overarching methodological variable of concern pertaining to the cycling 3MT is the prescribed resistance. While the original 3MT protocol required a GXT to determine the prescribed resistance [7, 78], researchers have since optimised the procedure by prescribing the resistance without a GXT (i.e., a single-visit test) [223], individualised resistance based on fitness level, body mass index, sex, and age [111], and added an exhaustive bout at 10% above CP to verify the observed CP by eliciting $\dot{V}O_{2max}$ [110]. Recently, the 3MT has been criticised [203, 224] for overestimating CP derived via the traditional method—particularly in elite athletes. The validity of the CP or CS derived from a 3MT is typically confirmed by its agreement (i.e., concurrent validity) with the traditional method. In lieu of agreement, validity should be established by determining if exercise performed above and below the derived CP or CS yields systemic responses (e.g., $\dot{V}O_2$ kinetics and blood lactate responses) consistent with the heavy and severe domains of exercise (i.e., construct validity) [25, 225].

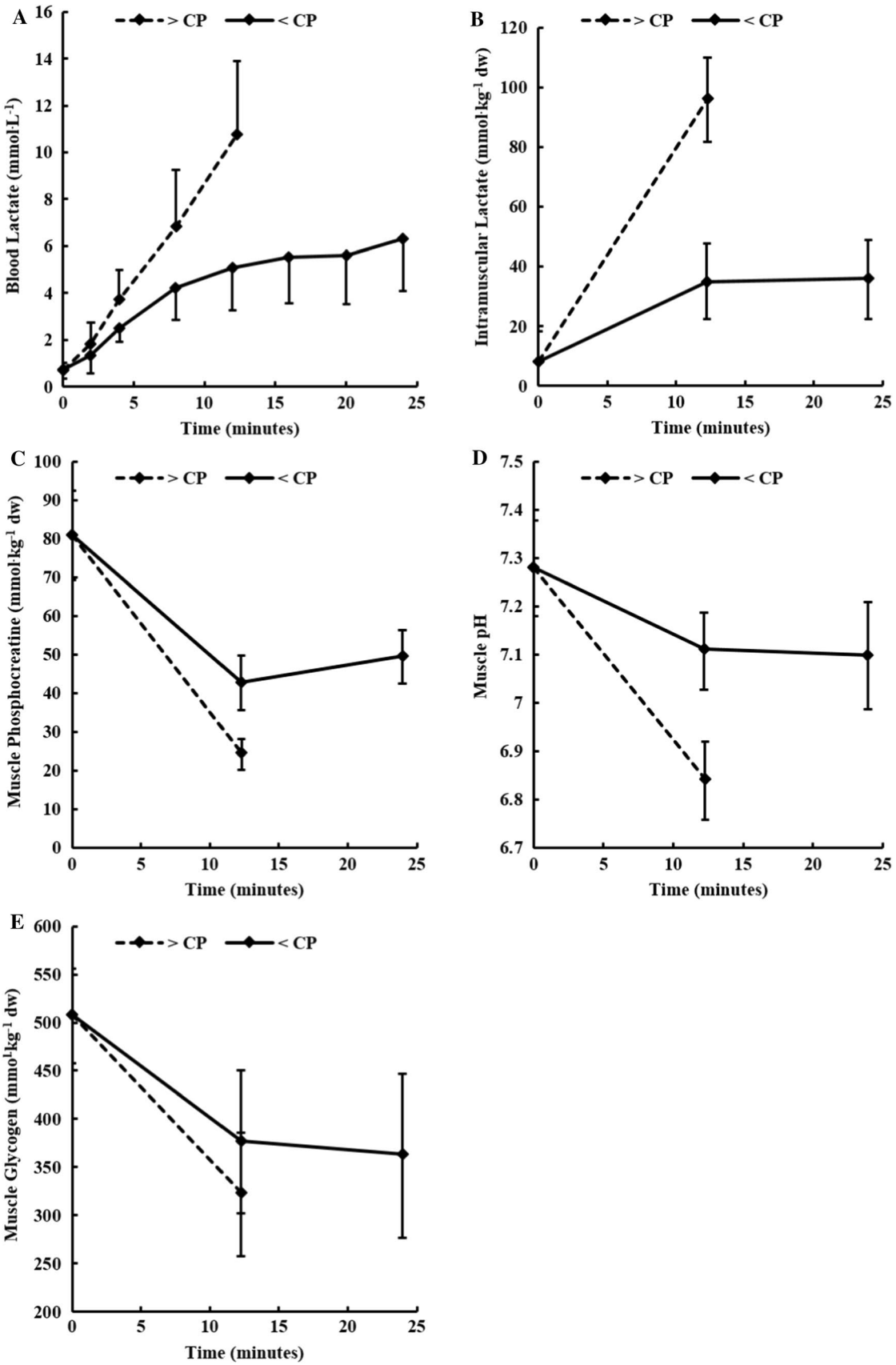
There is evidence to support the validity of either method to delineate the heavy and severe domains of exercise. In the late 1980′s, the first study assessed the homeostatic responses at and above CP (+ 5% of CP) derived via the traditional method [35] and confirmed the validity of CP to establish the boundary between heavy and severe exercise. These results have since been confirmed or reproduced several times [11, 25, 36, 56, 168, 205, 226, 227]. A recent study has strengthened the case for CP as the delineator between heavy and severe exercise. Exercise was performed at an intensity < CP [− 7.6% of CP (− 26 W)], which resulted in the stabilisation of intramuscular lactate, PCr, glycogen, and pH, blood lactate concentrations above baseline, and a $\dot{V}O_2$ slow 'component' with a plateau (Fig. 13) [25]. In contrast, exercise performed at an intensity > CP [+ 7.6% of CP (+ 26 W)] disturbed homeostatic control, and evoked a $\dot{V}O_2$ slow 'component.' Although these data support the validity of the CP to delineate the heavy and severe domains of exercise, the on-transient $\dot{V}O_2$ responses were not modelled. Moreover, a similar pattern would likely be observed when prescribing exercise intensity ~ 26 W above and below other submaximal anchors. Future research should address the inconsistencies in the methodology required to determine CP and CS, and validate the derived CP and CS with exercise bouts above and below (e.g., at the limits of agreement) while monitoring systemic responses.

3.6 Conclusion: Prescribing Exercise Relative to Submaximal Anchors

Submaximal anchors are commonly used by scientists and researchers to determine exercise intensity, and to differentiate training levels/zones or domains of exercise. The homeostatic response to exercise performed in proximity to a submaximal anchor should be used to establish the validity of the various submaximal anchors to determine exercise intensity. However, it appears there is little evidence to support the validity of most commonly used methods. More research is needed to validate the GET, the VT, and LT_1 for delineating the moderate and heavy domains of exercise. The LT_2 is based on the agreement with the MLSS, is influenced by GXT protocol design [55], and should be used with caution as a method to prescribe exercise intensity. In some instances, exercise above the 30-min MLSS results in an apparent steady state; therefore, it appears to underestimate the boundary between heavy and severe domains. Future research should address the validity of the single visit MLSS to yield domain-specific homeostatic perturbations. The CP and CS yield the strongest evidence to demarcate the heavy and severe domains of exercise (i.e., exercise above and below the CP or CS results in domain-specific homeostatic perturbations); however, these results need to be confirmed via on-transient $\dot{V}O_2$ kinetics. Lastly, we recommend that the systemic responses of any submaximal anchor be assessed against domain-specific homeostatic perturbations.

4 Prescribing Exercise Intensity Relative to a Maximal and Submaximal/Resting Values

Alternative methods to elicit a homogeneous response have been recommended based on the average work rates between a maximal and submaximal anchor, or the reserve or difference between the maximal anchor and its corresponding resting value [52, 66, 228–230]. Although these methods have been recommended based on the notion they better normalise exercise intensity [8], there has been little research assessing this hypothesis.


A
> CP
< CP
Blood Lactate (mmol·L-1)
Time (minutes)
B
> CP
< CP
Intramuscular Lactate (mmol·kg-1 dw)
Time (minutes)
C
> CP
< CP
Muscle Phosphocreatine (mmol·kg-1 dw)
Time (minutes)
D
> CP
< CP
Muscle pH
Time (minutes)
E
> CP
< CP
Muscle Glycogen (mmol·kg-1 dw)
Time (minutes)

◂**Fig. 13** **a–e** Mean ± SD blood lactate and muscle metabolite responses to exercise performed at intensities 7.6% above and below critical power (CP). **a** Mean blood lactate concentrations, **b** intramuscular lactate concentrations, **c** intramuscular phosphocreatine concentration, **d** intramuscular pH, and **e** intramuscular glycogen concentrations. Data based on Vanhatalo et al. [25]

4.1 Average Work Rate of Maximal and Submaximal Anchor "Delta"

There is no established protocol for establishing percent difference between a maximal (e.g., $\dot{W}_{max}$ or $\dot{V}_{max}$) and a submaximal anchor (i.e., GET, VT, or LT) from a GXT (also termed delta percent or Δ%). To our knowledge, there is also no research directly investigating the reliability of the physiological responses when exercising at intensities established using the delta method. Despite this shortcoming, the delta method has been used extensively [8, 51, 52, 66, 147, 228, 231–240] and recommended for normalising exercise intensity [8].

The ability of the delta method to normalise exercise intensity has been compared to fixed percentages of $\dot{V}O_{2max}$ [8]. In this study, exercise sessions were performed at 50, 70, and 90% of $\dot{V}O_{2max}$, at 60% of the GET, and at Δ40% and Δ80% of the difference between the GET and $\dot{V}O_{2max}$. The delta method resulted in less inter-subject variability for $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$, end-exercise heart rate, and changes in blood lactate from baseline, than exercise at fixed percentages of $\dot{V}O_{2max}$. A limitation of this study was basing validity on variables with little physiological relevance. For example, the end-exercise heart rate (CV 16–18%), or the difference between rest and end exercise blood lactate (CV 11–52%), are unreliable measures [165, 241, 242] and there is no evidence supporting these variables as a suitable means to characterise exercise intensity. Although the $\dot{V}O_2$ response expressed as a percentage of $\dot{V}O_{2max}$ was less variable across the delta intensities when compared with fixed percentages of $\dot{V}O_{2max}$, $\%\dot{V}O_{2max}$ has limited utility in normalising exercise intensity and does not differentiate between the domains of exercise at intensities above the GET (i.e., the heavy and severe domains)—a concern raised by the authors [8].

4.2 Oxygen Uptake and Heart Rate Reserve

$\dot{V}O_{2R}$ and HR_R are methods used to prescribe exercise intensity based on the difference between maximum (i.e., HR_{max}, $\dot{V}O_{2max}$) and resting values (i.e., $\dot{V}O_{2rest}$ and HR_{rest}) [229, 230]. Again, no research has directly investigated the reliability of determining exercise intensities via HR_R or $\dot{V}O_{2R}$; nonetheless, the reliability of these parameters is likely related to the reliability of measuring $\dot{V}O_{2max}$ [6, 14, 105, 107–111], $\dot{V}O_{2rest}$ [243], HR_{max} [2, 112–115, 135–137], and HR_{rest} [2, 244]. The overarching shortcoming of this method is the assumed linear relationship between $\dot{V}O_2$ or HR and work rate [138, 245]; however, the gain in both $\dot{V}O_2$ and HR relative to work rate increases in a curvilinear manner resulting in greater observed $\dot{V}O_2$ and HR values than predicted [246].

Only one study has investigated the relationship between the LT_1 (i.e., initial rise in blood lactate of greater than 0.2 $mmol \cdot L^{-1}$ above baseline) and percent of HR_R [247]. Extrapolation of the HR-work rate relationship from the GXT indicated that at 85% of HR_R, 20 of the 31 participants would be exercising above their LT_1. Similar to the $\%\dot{V}O_{2max}$ and $\%HR_{max}$, this outcome suggests that HR_R cannot be used to delineate the levels/zones/domains of exercise. Clearly, these methods are subject to the limitations of $\dot{V}O_{2max}$ and HR_{max} and values derived from percentages of $\dot{V}O_{2max}$, HR_{max}, $\dot{V}O_{2R}$, or HR_R have limited utility to normalise exercise intensity between individuals.

4.3 Conclusion: Prescribing Exercise Intensity Relative to a Maximal and Submaximal/Resting Value

Use of the delta method to determine exercise intensity yields more homogeneous physiological responses than methods based on fixed percentages of $\dot{V}O_{2max}$ [8]. However, the use of unreliable physiological variables (e.g., absolute blood lactate concentration and HR) raises concerns about the efficacy of this method to normalise exercise intensity. Exercise intensity determined as a fixed percentage of HR_R yields a heterogeneous homeostatic perturbation relative to LT_1. Based on the current evidence, the reserve methods cannot be recommended as valid methods to normalise exercise intensity as they do not yield domain-specific homeostatic perturbations.

5 Conclusions

Exercise intensity is a critical parameter for exercise prescription, and a large variety of methods have been employed to normalise exercise intensity for use in sports, exercise, clinical, and research settings. Despite common use, it is apparent that prescribing exercise intensity based on a fixed percentage of maximal anchors, such as $\dot{V}O_{2max}$, W_{max}, $\dot{V}_{max}$, and HR_{max}, has little merit for eliciting distinct or domain-specific homeostatic perturbations. In lieu of maximal anchors, some have advocated the use of submaximal anchors, including the LT_1, LT_2, GET, VT, RCP, MLSS, CP, and CS, to prescribe exercise intensity. There is evidence to

support the validity of LT_1, GET, and VT to delineate the moderate and heavy domains of exercise; however, there is little consensus regarding the methodology required to establish these submaximal anchors. Given the curvilinear relationship between $\dot{V}O_2$ and work rate, the RCP does not appear to be a viable option to elicit domain-specific homeostatic perturbations. There is evidence to support the validity of CP and CS to demarcate the heavy and severe domains of exercise; however, future research should address the systemic responses to exercise performed just above and below the established CP or CS. While the 30-min MLSS is often deemed an acceptable method for identifying the boundary between heavy and severe exercise, there is empirical evidence to the contrary and future research should investigate the efficacy of the single-visit MLSS to yield this boundary. The various delta methods yield more homogenous physiological responses than fixed percentages of maximal anchors, but do not yield domain-specific homeostatic perturbations. Thus, there is little evidence to support the validity of most commonly used methods to identify exercise intensities associated with distinct and homogeneous homeostatic perturbations (e.g., the $\dot{V}O_2$ kinetics and lactate responses associated with the various domains of exercise).

In this review, we have evaluated different methods of prescribing an apparently equivalent exercise intensity based on their ability to provoke similar homeostatic disturbances in participants. However, a key, unresolved question in exercise science is the physiological basis for the effects of different intensities on the adaptive response to exercise. More research is required to determine the key signals that are altered by different exercise intensities and sensed by the body to initiate the adaptive response to exercise. More training studies are also required to better understand chronic adaptations to different exercise intensity prescriptions. Better ways to prescribe exercise intensity will help sport scientists, researchers, clinicians, and coaches to design more effective training programs to achieve greater improvements in health and athletic performance.

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Compliance with Ethical Standards

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Conflict of interest Dr. Nicholas Jamnick, Prof Robert W. Pettitt, Dr. Cesare Granata, Prof. David Pyne, and Prof. David Bishop declare that they have no conflict of interest.

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