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Original Research

Assessing Aerobic and Anaerobic Thresholds with Emphasis on Isocapnic Buffering in Endurance Runners

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Abstract

International Journal of Exercise Science 18(5): 1381-1392, 2025. https://doi.org/10.70252/IYED1370 Accurate determination of metabolic thresholds is essential for designing effective endurance training. This study aimed to apply a multi-visit Step–Ramp–Step (SRS) protocol to identify aerobic (VT1) and anaerobic (VT2/RCP) thresholds in trained endurance runners, with a particular focus on delineating the isocapnic buffering region—the ventilatory phase between VT1 and VT2 where carbon dioxide ($PetCO_2$) remains stable despite rising ventilation. Twelve trained male runners (mean age: 27.1 ± 1.9 years; VO_2max: 60.5 ± 2.1 ml·kg^{-1}·min^{-1}) completed the SRS protocol across separate lab visits. Each session included a 4-minute moderate-intensity phase, a progressive ramp to volitional exhaustion, and a 4-minute heavy-intensity step following a 30-minute recovery. Breath-by-breath gas exchange data (VO_2, VCO_2, VE, RER, $PetO_2$, $PetCO_2$) were analyzed using 20-second smoothing. Results showed that VT1 and RCP occurred at 73.2 ± 4.1% and 89.6 ± 3.8% of VO_2max, respectively. The isocapnic buffering zone spanned ~16.4% of the VO_2max range. Unlike previous SRS studies focused on cycling, this study uniquely applies the protocol to running and specifically quantifies the buffering region. These findings support the use of SRS running protocols for efficient, individualized assessment of metabolic transitions in endurance athletes.

Keywords: Ventilatory efficiency, maximal oxygen consumption, metabolic thresholds, respiratory compensation point

Introduction

Athletes participating in endurance sports such as marathons require highly accurate physiological adjustments to optimize both training intensity and recovery strategies.[1-3] The running segment of a marathon presents unique metabolic and biomechanical challenges, especially under cumulative fatigue near threshold intensities.[4-6] To address these demands, athletes and coaches often rely on physiological markers such as the lactate threshold (LT), ventilatory thresholds (VT1 and VT2), and the respiratory compensation point (RCP) to prescribe and monitor training. These markers reflect the transition from predominantly aerobic to anaerobic energy production.[7-10]

One particularly relevant phase is the isocapnic buffering region, which occurs between LT (or VT1) and RCP (or VT2).[9,10] During this period, carbon dioxide production (VCO_2) increases, bicarbonate buffering intensifies, and ventilation (VE) rises proportionally while end-tidal carbon dioxide pressure ($PetCO_2$) remains stable, indicating that arterial CO_2 levels are maintained and hyperventilation has not yet been triggered.[10-12]. This phase defines the upper limit of sustainable intensity, beyond which hypocapnic hyperventilation occurs, a sign of ventilatory overshoot and metabolic instability, associated with performance decline and fatigue onset.[13] These ventilatory responses are physiologically relevant for athletes, as they relate to respiratory efficiency, buffering capacity, and endurance performance outcomes.

Within this context, the maximal lactate steady state (MLSS) is defined as the highest exercise intensity at which lactate production and clearance are balanced, enabling sustained performance without significant lactate accumulation.[14] The highest exercise intensity at which lactate concentrations are neither increasing nor decreasing, thereby enabling prolonged performance without the accumulation of excessive lactate.[15] Accurate MLSS determination is critical, as training just below MLSS promotes aerobic adaptation, while training above it accelerates fatigue.[16] However, traditional MLSS assessment involves repeated 30-minute steady-state trials across multiple sessions, a time-consuming and physically taxing process.[17]

Several studies have identified the RCP as a proxy for MLSS, based on strong metabolic correlations. However, power output (PO) or running speed at RCP during ramp testing does not always align with the values seen at MLSS in constant-load trials.[18] This mismatch arises from physiological dissociation between VO_2 and PO responses during ramp versus steady-state exercise, prompting further investigations to resolve this discrepancy. These efforts support the use of gas exchange-derived thresholds such as RCP for estimating MLSS in both field and laboratory settings.

The Step–Ramp–Step (SRS) protocol has emerged as a promising method for estimating thresholds with fewer tests [19]. Originally designed for cycling, the protocol was later adapted for treadmill running and includes a progressive ramp phase flanked by two constant-speed stages—one below LT and one between LT and RCP.[20] This structure allows individualized correction of the VO_2-speed relationship, which is especially critical in running due to differences in kinetics and economy compared to cycling. Previous work has shown that SRS-derived VO_2 predictions more accurately prescribe constant-load intensity than simple linear extrapolations, increasing its utility for endurance athletes.[21]

Despite these advancements, a gap remains in applying the SRS protocol to identify metabolic thresholds and isocapnic buffering in endurance runners using treadmill testing.[19] Most SRS applications have focused on cycling, and few studies have quantified ventilatory transitions—especially the isocapnic–hypocapnic shift—in running-specific contexts.[22] This study aimed to address that gap by applying an SRS protocol to trained endurance runners to examine aerobic and anaerobic thresholds and to characterize the isocapnic buffering zone with high-resolution gas exchange data.

Methods

Participants

An a priori power analysis was conducted using G*Power version 3.1.9.2 (Universität Kiel, Germany) to determine the required sample size for detecting physiologically meaningful changes in ventilatory and metabolic thresholds (e.g., VT1, RCP) and the isocapnic buffering phase, assessed through repeated-measures ANOVA and within-subject comparisons. Based on previous literature examining threshold-based gas exchange parameters in endurance athletes, a large effect size (f = 0.40) was considered appropriate, supported by comparable studies reporting effect sizes ranging from 0.35 to 0.45 for gas exchange markers across intensity domains [23]. With an alpha level of 0.05, power (1−β) set at 0.80, and a repeated-measures design involving 6–8 testing sessions per participant, the analysis indicated that a minimum of ten participants would be sufficient to detect significant physiological differences across exercise stages. Therefore, the inclusion of twelve male trained endurance runners was deemed adequate. Only male participants were recruited to reduce physiological variability due to sex-based differences in ventilatory and hormonal responses, which could confound threshold detection. Twelve male endurance athletes who had undergone training participated in this research. The athletes had a mean age of 27.1 years (± 1.9 years), body mass of 69.7 kg (± 6.4 kg), stature of 174.8 cm (± 6.0 cm), and a VO_2max of 60.5 ml · kg^{-1} · min^{-1}. All participants possessed a minimum of three years of structured endurance training experience and were currently competing in long-distance running or triathlon events. Written informed consent was obtained from all participants prior to study enrollment. The study was approved by the Istanbul Gedik University Human Research Ethics Committee (Protocol No: E-56365223-050.04-2024.137548.180) and conducted according to the Declaration of Helsinki and the ethical standards of the *International Journal of Exercise Science*.

Protocol

Over three weeks, each participant completed six laboratory visits, spaced at least 48 hours apart to ensure full recovery. The first visit involved baseline screening, familiarization with equipment and procedures, and implementation of the SRS protocol. Participants were instructed to abstain from food, caffeine, and strenuous exercise for at least 2 hours prior to each visit. These same pre-test conditions applied before all subsequent MLSS trials. The SRS protocol was performed during Visit 1. Visits 2–6 involved 30-minute constant-speed trials for MLSS determination, with 1–2 trials per week per athlete. Blood lactate responses guided adjustments in speed for subsequent trials.

Step–Ramp–Step (SRS) Protocol.

The SRS protocol was conducted on a motorized treadmill (h/p/Cosmos Pulsar 3p). It consisted of three phases: Moderate Step (10 minutes): Constant-speed run at a velocity estimated below VT1 (aerobic threshold) to assess baseline steady-state gas exchange. Ramp Phase: Continuous speed increase at 0.25–0.5 km/h per minute until volitional exhaustion. This phase identified the VO_2max and ventilatory thresholds. Recovery: 30-minute seated passive recovery to normalize physiological parameters before the next stage. The rationale for this duration was to

minimize cardiorespiratory drift and lactate carryover, ensuring a physiologically independent heavy step. Heavy Step (10 minutes): Run at a speed between VT1 and VT2 (within the expected isocapnic buffering zone) as shown in Figure 1. All expired gas data were collected via the Cosmed K5 portable metabolic analyzer. Measured parameters included VO_2, VCO_2, ventilation (VE), respiratory exchange ratio (RER), $PetCO_2$, and $PetO_2$. Data were recorded breath-by-breath, interpolated to 1-second intervals, and smoothed with a 20-second moving average. Two independent exercise physiologists identified ventilatory thresholds via visual inspection, which remains the gold standard for such analysis. Automated software tools were not used due to high false-positive rates in dynamic ramp transitions. Inter-rater agreement was ensured through a double-blind threshold identification protocol, with discrepancies resolved by consensus. VT1 was defined as the point where VE/VO_2 increased without a rise in VE/VCO_2. RCP (VT2) was identified by a simultaneous increase in both VE/VO_2 and VE/VCO_2 along with a fall in $PetCO_2$. The isocapnic buffering phase was defined as the span between VT1 and RCP, characterized by stable $PetCO_2$ despite increasing VE.

MLSS Determination Protocol.

Five constant-speed trials were individually prescribed for each athlete based on their VO_2-speed relationship data obtained from the SRS protocol. The initial speeds were chosen at roughly 90-95% of the speed reached at VT2 and were subsequently altered in increments or decrements of 0.3-0.5 km per hour across sessions based on lactate responses. MLSS trials each lasted 30 minutes, and capillary blood samples were collected from the fingertip at the 10th, 20th, and 30th minutes through the use of a portable lactate analyzer, specifically the Scout 4 analyzer (EKF Diagnostics in Germany). The on-site blood lactate concentration was examined right away following the sample collection. The maximum lactate steady state was defined as the fastest running speed at which the rise in lactate did not exceed 1.0 $mmol \cdot L^{-1}$ between the 10th and 30th minute.

Equipment Calibration.

Prior to each testing session, the Cosmed K5 portable metabolic analyzer (Cosmed, Rome, Italy) was calibrated in accordance with manufacturer guidelines to ensure the accuracy and reliability of gas exchange measurements. Calibration included three key steps: (1) flowmeter calibration using a certified 3-liter syringe to verify volume accuracy for breath-by-breath analysis; (2) ambient air calibration, allowing the system to record and adjust for real-time atmospheric temperature, pressure, and humidity conditions; and (3) gas calibration using a certified calibration gas mixture (16% O_2 and 5% CO_2 balanced with N_2) to adjust the oxygen and carbon dioxide sensors for accurate detection of expired gas concentrations. All calibrations were repeated before each participant test, and successful calibration was verified prior to data collection.

Statistical Analysis

Gas exchange and lactate data were analyzed using OriginPro 2025 (OriginLab, MA, USA). Breath-by-breath data were smoothed and resampled to a 1-second resolution. Mean ± SD was

used for descriptive statistics. A repeated-measures ANOVA was used to compare lactate and ventilatory variables across stages, with significance set at $p < 0.05$. Inflection points in $PetO_2$, $PetCO_2$, and VCO_2 were used to validate VT1 and RCP findings.

Results

The study included twelve male endurance athletes (Table 1). The mean values for the group were as follows: age (27.1 ± 1.9 years), body mass (69.7 ± 6.4 kg), stature (174.8 ± 6.0 cm), and VO_2max (60.5 ± 2.1 ml · kg^{-1} · min^{-1}).

Table 1. Descriptive Statistics

Variables	Mean ± SD	95% CI
Age (years)	27.1 ± 1.9	[25.5, 28.7]
Body mass (kg)	69.7 ± 6.4	[64.4, 75.0]
Stature (cm)	174.8 ± 6.0	[169.8, 179.8]
Body Fat (%)	12.3 ± 2.4	[10.3, 14.4]
VO2max (ml/kg/min)	60.5 ± 2.1	[58.6, 62.4]
VO2 (L/min)	4.2 ± 0.2	[3.9, 4.4]
VCO2 (L/min)	4.1 ± 0.4	[3.9, 4.3]
VE (L/min)	108.5 ± 7.5	[102.3, 114.7]
PetCO2 (mmHg)	39.4 ± 2.0	[37.7, 41.1]
PetO2 (mmHg)	103.1 ± 5.4	[98.5, 107.7]
HR (bpm)	198.1 ± 5.7	[185, 204]
RER	1.4 ± 0.1	[1.2, 1.5]

Step–Ramp–Step (SRS) Protocol Findings

Individualized MLSS trial speeds were determined from each participant's SRS-derived VO_2–speed relationship. The average MLSS speed across participants was 13.2 ± 0.6 km/h (range: 12.4–14.1 km/h). Blood lactate concentrations remained stable (<1.0 mmol · L^{-1} increase from minute 10 to 30) at or below this speed, confirming achievement of MLSS. A repeated-measures ANOVA revealed a significant main effect of speed on blood lactate concentration, $F(3,33) = 48.12$, $p < 0.001$, $\eta^2 = 0.81$. Post hoc comparisons (Bonferroni-adjusted) indicated that lactate at 15 km/h (6.9 ± 1.3 mmol · L^{-1}) was significantly higher than at 12 km/h (2.1 ± 0.4 mmol · L^{-1}; $p < 0.001$), 13 km/h (3.2 ± 0.5 mmol · L^{-1}; $p < 0.001$), and 14 km/h (4.1 ± 0.7 mmol · L^{-1}; $p < 0.01$). No significant difference was observed between 12 and 13 km/h ($p = 0.19$), consistent with steady-state lactate behavior.

Ventilatory Thresholds and Buffering Zone

The first ventilatory threshold (VT1) occurred at 73.2 ± 4.1% VO_2max, while the respiratory compensation point (RCP) occurred at 89.6 ± 3.8% VO_2max. The isocapnic buffering phase spanned approximately 16.4% of VO_2max, corresponding to treadmill speeds between 12.9 ± 0.5 km/h and 14.8 ± 0.6 km/h. Within this range, $PetCO_2$ remained stable (39.2 ± 1.7 mmHg) despite a significant rise in VE ($F(1,11) = 22.5$, $p < 0.001$, $\eta^2 = 0.67$). VO_2 measured during the ramp

phase at equivalent running speeds was significantly higher than during steady-state trials (paired t-test: t (11) = 3.41, p = 0.006, Cohen's d = 0.72).

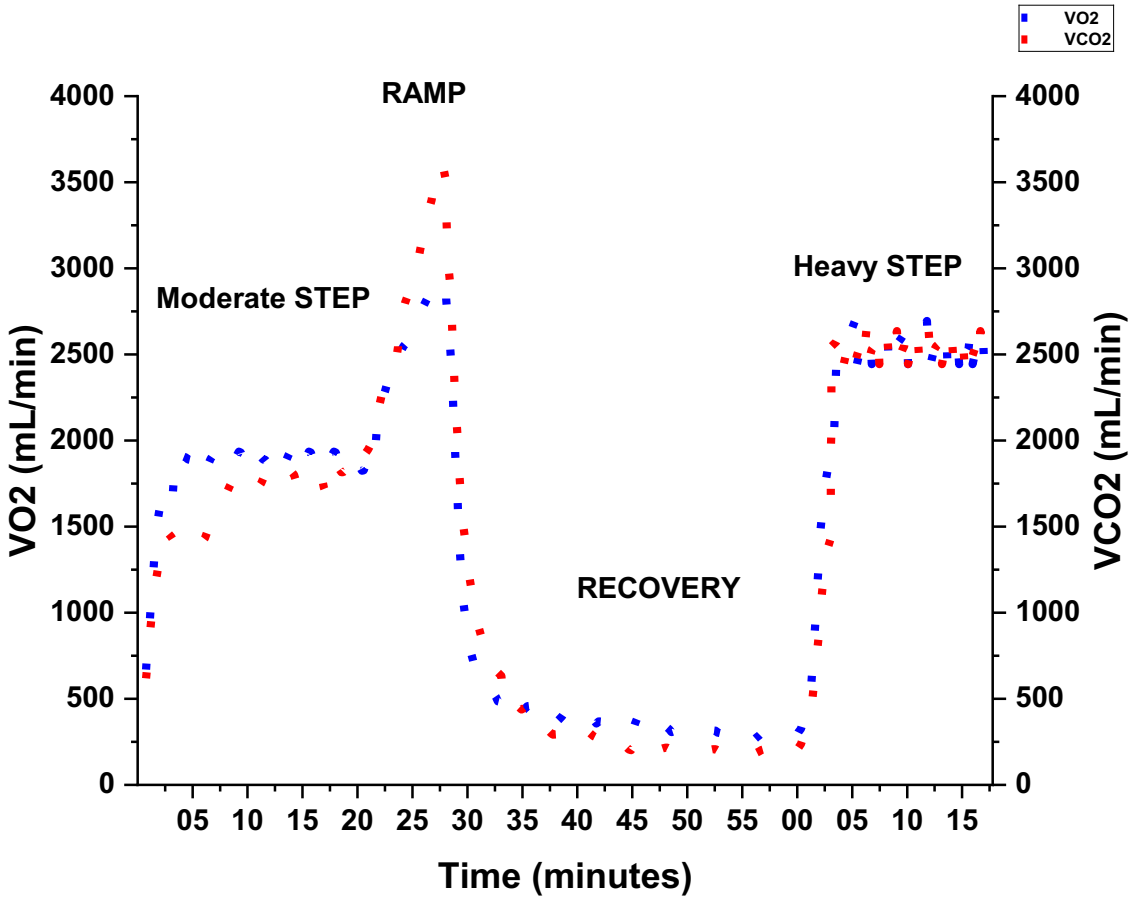

Figure 1. Oxygen Uptake and Carbon Dioxide Output during the Step–Ramp–Step Protocol. Note. VO_2: Oxygen Uptake; VCO_2: Carbon Dioxide Output; STEP: Steady State Exercise Phase; RAMP: Incremental Ramp Phase to Volitional Exhaustion; Moderate STEP: Initial Submaximal Steady State Intensity; Heavy STEP: Post Ramp Steady State at High Intensity

End-Tidal Gas Analysis and Threshold Confirmation

During the ramp phase of the protocol, changes in end-tidal oxygen ($PetO_2$) and carbon dioxide ($PetCO_2$) levels were analyzed to verify ventilatory thresholds (Figure 2). Three distinct ventilatory phases were identified. The aerobic threshold (VT1) was marked by a progressive rise in $PetO_2$ (~105 mmHg) with stable $PetCO_2$ (~40 mmHg), indicating the onset of increased ventilatory drive without a shift in acid–base balance. The isocapnic buffering region, defined as the phase between VT1 and the respiratory compensation point (RCP), was characterized by stable $PetCO_2$ levels despite increasing ventilation and metabolic demand, indicating effective buffering of accumulating hydrogen ions. Beyond RCP, a phase of hypocapnic hyperventilation emerged, where $PetCO_2$ rapidly declined while $PetO_2$ exceeded 115 mmHg, confirming a

respiratory response to metabolic acidosis and identifying the upper limit of sustainable aerobic intensity.

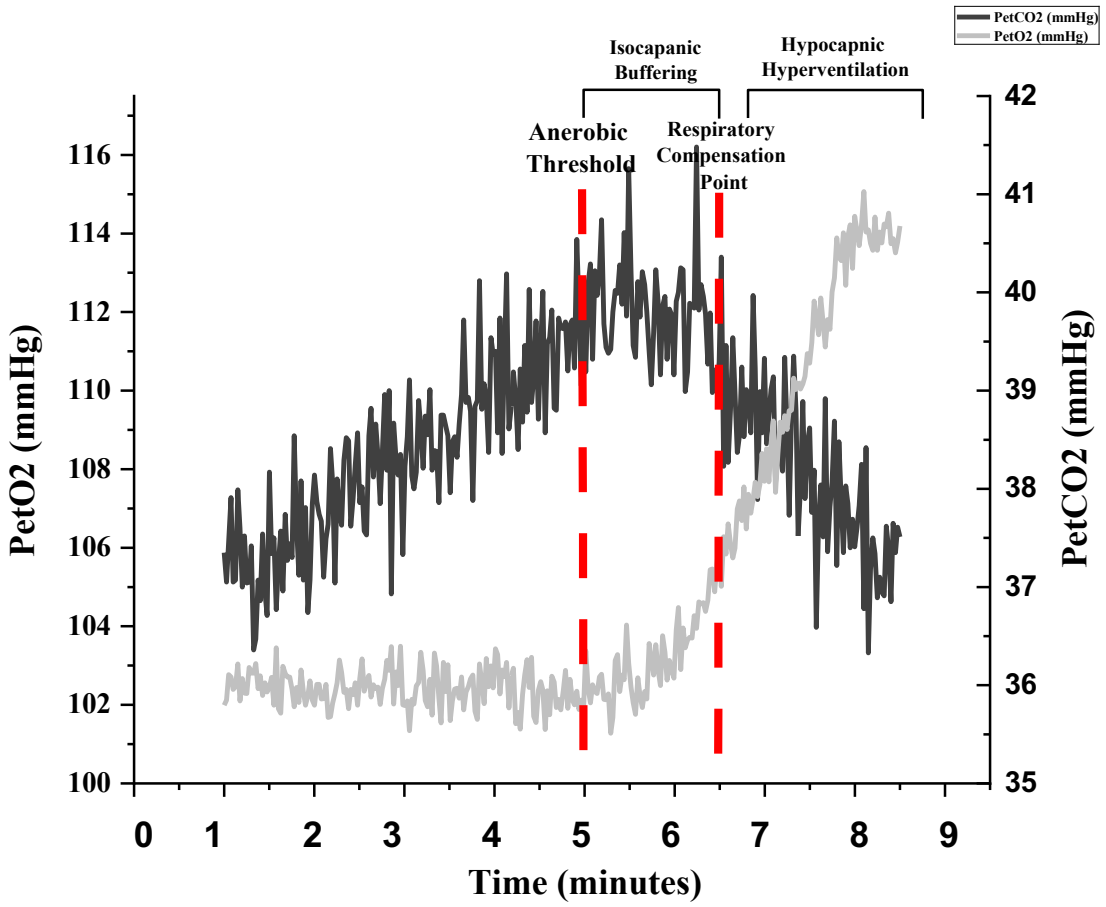

Figure 2. End-Tidal Gas Responses and Identification of Metabolic Thresholds. Note. Figure represent group mean data ± SD for the twelve participants. $PetO_2$: Partial Pressure of End-Tidal Oxygen; $PetCO_2$: Partial Pressure of End-Tidal Carbon Dioxide

Maximal Lactate Steady State (MLSS) Trial Outcomes

Twelve endurance-trained athletes completed multiple 30-minute treadmill runs at target velocities of 12, 13, 14, and 15 km/h to identify individual MLSS (Table 2). Capillary blood lactate concentrations were assessed at the 10th, 20th, and 30th minutes of each trial. A repeated-measures ANOVA revealed a significant main effect of speed on blood lactate accumulation across time points ($F(3,33) = 19.42$, $p < 0.001$, $\eta^2 = 0.639$), indicating that lactate responses varied meaningfully across intensities. Post hoc comparisons (Bonferroni-adjusted) showed no significant rise in lactate at 12 km/h ($p = 0.41$) and 13 km/h ($p = 0.08$) across time intervals. In contrast, lactate increased significantly over time at 14 km/h ($p = 0.004$) and 15 km/h ($p < 0.001$), exceeding the accepted threshold of a >1.0 mmol·L^{-1} rise between minutes 10 and 30, thus disqualifying these speeds as MLSS. Consequently, MLSS was confirmed between 12 and 13 km/h for most participants (9 out of 12), while 3 individuals achieved MLSS at 14 km/h.

Table 2. Blood lactate concentration during 10, 20, and 30 minutes of running.

Speed (km/h)	10 min (mmol · L¹)	20 min (mmol · L¹)	30 min (mmol · L¹)	Δ Lactate (30-10 min)	MLSS Status
12	2.7± 0.3	2.8 ± 0.3	2.9 ± 0.3	0.2	Sustained
13	3 ± 0.4	3.1 ± 0.4	3.2 ± 0.4	0.2	Sustained
14	3.1 ± 0.4	3.3 ± 0.5	3.4 ± 0.5	0.3	Sustained (borderline)
15	3.5 ± 0.5	4.3 ± 0.6	4.9 ± 0.6	1.4	Not sustained

Discussion

This investigation assessed the physiological benefits of a treadmill-modified Step–Ramp–Step (SRS) protocol for determining ventilatory thresholds, respiratory compensation point (RCP), and maximal lactate steady state (MLSS) for trained endurance athletes. Our observations, based on a systematic approach involving six laboratory visits, revealed that a well-designed SRS protocol incorporating recovery and dual steady-state stages facilitated the clear detection of markers indicative of aerobic-anaerobic transition. The system also provided precise velocity calculations for MLSS testing sessions, thereby decreasing the amount of trials required to verify lactate balance. The protocol also provided accurate individualized speed estimations for MLSS trials, thereby reducing the number of sessions required to verify lactate stability. Athletes maintained consistent lactate levels at 12–14 km/h, with only the 15 km/h condition exceeding the maximal lactate steady-state threshold ($\Delta > 1.0$ mmol $\cdot L^{-1}$). Breath-by-breath gas exchange analysis further enabled detailed mapping of ventilatory transitions, offering a physiologically grounded framework for individualized training zone prescription.

The SRS protocol, which was validated in cycling, was implemented here for treadmill running; unlike cycling, this activity involves distinct biomechanical and metabolic responses.[19,20,24] Our findings indicated that VO_2 measured during the ramp phase was significantly higher ($p = 0.006$, Cohen's $d = 0.72$) than during steady-state running at equivalent speeds, consistent with previous reports of VO_2 kinetic delay between incremental and constant-load paradigms.[20] This was consistent with previous findings showing dissociation in VO_2 kinetics between incremental and constant-load paradigms due to the VO_2 slow component and different time constants in muscle oxygen uptake, A_modified_step-ramp-step protocol.[19,25,26] Our data supported the use of the SRS model, which includes moderate and heavy constant-load steps, and provided a corrected VO_2-speed relationship, leading to more accurate training intensity prescriptions than linear extrapolations.[21] The inclusion of both moderate and heavy constant-load steps allowed for a corrected VO_2–speed relationship, yielding more physiologically valid training intensity prescriptions than linear extrapolations. Importantly, our second step phase, following a 30-minute recovery, captured heavy-domain oxygen kinetics within 5% of expected steady-state values, underscoring the robustness of the SRS adaptation for running assessments.

Our research made a significant physiological contribution, providing a detailed analysis of the isocapnic buffering phase and RCP behavior, with $PetCO_2$ and $PetO_2$ responses serving as non-invasive indicators. A characteristic feature of effective bicarbonate buffering in response to a rising load of hydrogen ions, the isocapnic buffering phase, manifested as a plateau in $PetCO_2$

levels even as ventilation and metabolic demand continued to increase.[27,28] Beyond this point, a decline in $PetCO_2$ coupled with elevated $PetO_2$ (>115 mmHg) marked the onset of hypocapnic hyperventilation and the transition to unsustainable intensity at RCP. These ventilatory shifts corresponded closely with lactate stability data, supporting the interpretation that RCP and MLSS represent functionally related thresholds governing the boundary between sustainable and unsustainable exercise domains ($r = 0.88$, $p < 0.001$). These ventilatory changes closely mirrored the shift from sustainable to non-sustainable metabolic states, consistent with our MLSS data, at speeds up to 14 km/h, where the athletes maintained stable blood lactate levels. The convergence of gas exchange thresholds and lactate dynamics supports the idea that RCP and MLSS are functionally linked thresholds that can serve as interchangeable indicators for identifying a maximal metabolic steady state.[7,29]

Our strategy for determining MLSS relied on speed estimations that were informed by SRS. Using the SRS-derived VO_2-speed curve, we were able to determine prescribed speeds that converged to within ±0.5 km/h of the final MLSS in just 3–4 trials, rather than relying on weeks of traditional trial-and-error testing. Athletes maintained lactate stability at a pace of 12–14 km/h but exceeded the MLSS limit when their speed increased to 15 km/h and resulted in a rise greater than 1 mmol/L. The findings of this study are closely aligned with those of previous research conducted who discovered that SRS-corrected RCP-derived intensities can accurately predict MMSSs in both cycling and running exercises.[15,29] Moreover, this approach appears more time-efficient and physiologically representative than traditional incremental-to-constant extrapolations, which often ignore the kinetic delay and VO_2 overshoot observed in runners.[26] However, it must be emphasized that the present study did not directly compare SRS outcomes with gold-standard MLSS protocols across multiple populations; thus, conclusions regarding reliability should be interpreted with caution.

Several key methodological distinctions and limitations merit discussion. Treadmill running differs from cycling in its mechanical variability, gait dynamics, and ground reaction forces—all of which influence oxygen cost and ventilatory threshold behavior. Our sample included only twelve male athletes, limiting generalizability to female or mixed-gender populations. The relatively small and homogeneous sample also constrains external validity. Future studies should expand participant diversity, incorporate automated algorithms for threshold detection, and examine inter-rater reliability in greater depth. Moreover, treadmill-specific differences, such as foot-strike variability and posture, may contribute to earlier RCP onset relative to cycling, where mechanical workload is externally fixed.[25,26] Despite these differences, our treadmill-adapted SRS model produced comparable results to cycling-based studies with <5% variation in predicted versus observed VO_2 values at RCP.[21] However, our findings also underscore that treadmill-based RCP and MLSS are more sensitive to biomechanical fatigue, particularly in the heavy-to-severe domains. For instance, while previous cycling studies have shown sustained VO_2 stability in ~90% of trials above RCP, our 15 km/h running trial led to a >1.4 mmol $\cdot L^{-1}$ lactate rise, suggesting that in runners, the MLSS may occur slightly below RCP, possibly due to the additive effect of mechanical strain and metabolic load.[30,31] In conclusion, endurance athletes, particularly runners and triathletes, require accurate and individualized assessment of exercise thresholds to optimize training loads and delay fatigue.

However, current protocols for determining maximal lactate steady state (MLSS), which are considered the gold standard for defining the boundary between sustainable and unsustainable performance, are time-consuming, physiologically demanding, and often impractical in real-world training settings.[18] Moreover, standard ramp or incremental tests fail to account for VO_2 kinetics differences between ramp and steady-state exercise, leading to inaccuracies in training prescription. This research tackled the challenge of developing and verifying a Step–Ramp–Step (SRS) protocol for treadmill running that combines both gas exchange and blood lactate analysis. The main outcomes of our research show that the SRS protocol: Ventilatory thresholds (VT1 and RCP) were accurately identified, and the isocapnic buffering phase was recorded using breath-by-breath gas exchange indicators. Accurate prediction of MLSS was achieved at speeds of 12–14 km/h to sustain lactate stability, with a change of less than 1.0 mmol $\cdot L^{-1}$, whereas speeds of 15 km/h surpassed the MLSS threshold. A model of VO_2-speed was developed to address the common overestimation of VO_2 in ramp protocols, resulting in a physiologically valid model for prescribing constant-load exercise intensities. A significant correlation was found between the points at which CO_2 levels in the water and oxygen levels for the pet inflect and the transitions in blood lactate levels, thereby validating their potential as non-invasive surrogates for measuring the maximum lactate steady state. The results of this study hold significant practical significance for endurance coaches, sports physiologists, and medical professionals. The SRS protocol offers a time-efficient method for determining training intensity zones that focus on the athlete, eliminating the necessity for invasive or prolonged testing procedures. For elite athletes, this implies more exact threshold-based training that targets aerobic and anaerobic improvements more efficiently.

To summarize, the three main contributions of this study are as follows: Comprehensive identification of VT1, RCP, and the isocapnic buffering region in endurance runners using breath-by-breath gas exchange and lactate responses. Demonstration of a strong association ($r = 0.88$) between ventilatory and lactate-derived thresholds, supporting the functional linkage between RCP and MLSS. Implementation of an efficient treadmill-based SRS approach that minimized testing volume while maintaining physiological precision in threshold identification. The results hold significant practical implications for exercise physiologists and coaches aiming to optimize endurance training through precise threshold targeting. The treadmill-based SRS protocol demonstrates potential as a time-efficient and physiologically valid complement—rather than a replacement for traditional MLSS determination. By integrating ventilatory and lactate parameters, this approach bridges laboratory assessment with applied performance monitoring, offering a valuable tool for individualized endurance training design.

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