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Longitudinal Metabolic and Perceptual Responses to an Individualized, Biochemically Guided Interval-Swimming Protocol: A Non-Randomized Controlled Pilot Study

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Abstract

Individualized regulation of training load may help characterize athlete-specific responses during competitive swimming. This controlled longitudinal pilot study evaluated metabolic and perceptual responses to an individualized, biochemically guided interval-swimming protocol. Twelve competitive swimmers were included (experimental group, $n = 5$; control group, $n = 7$) and completed six serial assessments (T0–T5). Group allocation was non-randomized. In the experimental group, work-bout duration was progressively modified using repeated biochemical and perceptual information, whereas the control group followed a predominantly fixed-duration protocol. The principal outcome analyzed in this report was blood lactate measured 1 min after exercise. Post-exercise blood lactate decreased from 11.78 to 7.94 mmol/L in the experimental group and from 12.19 to 11.43 mmol/L in controls. The T0–T5 reduction was greater in the experimental group (−3.84 vs. −0.76 mmol/L), with a between-group difference in change of −3.08 mmol/L (95% CI, −3.72 to −2.45; $p < 0.001$). Ammonia showed a supportive longitudinal difference, whereas uric acid was exploratory because it guided load adjustment. Perceived exertion showed a supportive but borderline change after multiplicity adjustment. Recorded interval pace remained broadly stable while work-bout duration increased substantially in the experimental group; however, no standardized time-trial or competitive-performance outcome was assessed. These preliminary findings support further evaluation of individualized, biochemically guided interval-training protocols in larger, prospectively standardized studies.

Keywords: swimming; blood lactate; training load; individualized training; ammonia; uric acid; rating of perceived exertion

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1. Introduction

Competitive swimming involves repeated exposure to substantial training loads and requires an appropriate balance between physiological stimulus, recovery, and adaptation. Because athletes may respond differently to similar externally prescribed workloads, monitoring internal training load is an important component of individualized training management. In competitive swimming, external load is commonly characterized by training volume, duration, and swimming velocity, whereas internal load may be evaluated

using blood lactate concentration, heart rate, and perceived exertion [1]. In applied competitive swimming environments, training-load monitoring is widely used; an international survey reported that 84% of respondents implemented some form of monitoring, with swimming volume and session rating of perceived exertion among the most frequently collected variables [2].

Blood lactate is one of the most established physiological measures used to assess exercise intensity in swimming [1]. Its response during interval exercise is influenced by exercise intensity, duration, recovery periods, and individual physiological characteristics [1,3,4]. Recent research in competitive swimmers has demonstrated that training sets performed across different intensity domains elicit distinct physiological and blood-lactate responses, supporting interpretation of lactate within the context of the specific training exposure and alongside complementary indicators of internal load [3]. Lactate-threshold testing is also used to characterize individualized swimming intensity, although threshold estimates may vary according to the analytical method applied [4]. In world-class and elite open-water swimmers, swimming velocity at the lactate threshold has been associated with competitive performance, further supporting the practical relevance of lactate-based physiological assessment [5].

However, lactate reflects only one component of the metabolic response to strenuous exercise. High rates of adenosine triphosphate turnover during intense muscular activity are accompanied by changes in adenine nucleotide and purine metabolism [6–8]. Blood ammonia is associated with adenosine monophosphate degradation and responds to increasing exercise intensity [6]. Recent evidence in elite athletes has demonstrated changes in both ammonia and lactate during incremental exercise to exhaustion, indicating that these metabolites may provide complementary information about exercise-related metabolic responses [6]. Purine metabolism is likewise closely related to adenosine triphosphate degradation and resynthesis. Exercise alters circulating purine metabolites, including hypoxanthine, xanthine, and uric acid, and these responses may differ according to training characteristics and training status [7,8]. Accordingly, simultaneous assessment of lactate, ammonia, and purine-related metabolites may provide a broader description of metabolic responses than reliance on a single biochemical variable.

Perceptual measures provide an additional dimension of internal training load. Rating of perceived exertion (RPE) is inexpensive, non-invasive, and suitable for repeated use during training. In elite swimmers, longitudinal assessment has demonstrated good validity and reliability of RPE for monitoring exercise intensity, with associations with heart rate and blood lactate concentration [9]. Its practical relevance is also supported by its frequent use in competitive swimming programs [2]. Integrating perceptual assessment with biochemical monitoring may therefore provide complementary information about athletes' responses to repeated exercise.

Despite the increasing use of internal-load monitoring in swimming, evidence remains limited regarding longitudinal approaches in which repeated biochemical responses are directly incorporated into subsequent adjustment of interval-exercise exposure. Previous research has primarily investigated physiological responses to predefined swimming intensities, lactate-threshold determination, training-load monitoring practices, or associations between physiological indicators and performance [1–5,9]. In particular, evidence regarding the integration of lactate, ammonia, and purine-related responses to guide individualized adjustment of repeated swimming intervals remains limited. This represents an important research gap because similar external swimming workloads may not necessarily produce equivalent internal metabolic responses across athletes.

Accordingly, the aim of this controlled pilot study was to evaluate longitudinal metabolic and perceptual responses to repeated interval swimming under an individ-

ualized, biochemically guided and progressively modified work-bout protocol compared with a predominantly fixed-duration protocol. Blood lactate measured 1 min after exercise was treated as the principal outcome analyzed in the present report, while uric acid, ammonia, resting lactate, lactate increment, RPE, and swimming pace were evaluated as complementary outcomes. We hypothesized that the individualized protocol would be associated with a greater attenuation of the 1-min post-exercise lactate response over time while recorded interval pace remained broadly stable.

2. Materials and Methods

2.1. Study Design and Participants

This controlled longitudinal pilot study was conducted from 9 September to 4 November 2025 at the Central Swimming Pool (Rakhat Fitness Sports Complex), Almaty, Republic of Kazakhstan, in a 25-m swimming pool. All 12 available competitive swimmers aged 16–23 years who met the eligibility criteria were included: five in the experimental group and seven in the control group. No athletes were excluded before or after group formation, no athlete met the protocol-defined non-responder criterion, and all 12 completed the six planned assessments (T0–T5). Thus, the final sample comprised 12 independent athletes with repeated within-athlete measurements across the study period.

Group allocation was non-randomized and was determined by the investigator. Athletes were assigned to the experimental group when their existing training plan could be modified using the individualized biochemical-guidance approach while allowing them to maintain functional reserve rather than train to overt submaximal or maximal fatigue. Athletes whose existing plans were not considered suitable for such modification continued their standard regimen and comprised the control group. The investigators sought to include both relatively higher- and lower-performing competitive swimmers in each group; however, no formal matching based on an objective performance metric was performed. Accordingly, group formation may have reflected pre-existing differences in training status, previous training response, or workload tolerance and could contribute to selection bias or uncontrolled confounding. The sample included one Candidate for Master of Sport, seven Masters of Sport, and four Masters of Sport of International Class. All athletes completed nine training sessions per week, corresponding to approximately 17 h of training per week; however, the detailed content and intensity distribution of training between serial assessments were not quantitatively captured.

Eligibility criteria included a relatively homogeneous age range, more than one year of swimming experience, a competitive qualification of at least First-Class Athlete level, absence of acute illness or injury, and medical clearance for sports participation. Prespecified exclusion criteria were type 1 or type 2 diabetes mellitus; resting capillary blood uric acid above the applicable reference range; non-adherence to requirements concerning vitamin supplementation, diet, or medications that could affect uric acid, ammonia, or lactate; meeting the qualitative biochemical non-responder criterion; and, for the experimental group, unwillingness to train with functional reserve rather than to overt submaximal or maximal fatigue. The protocol allowed these exclusion criteria to be applied after study initiation, but no participant was excluded. A non-responder was operationally defined as an athlete who developed clear physical fatigue—pronounced hyperventilation during or after the swimming distance, marked subjective muscle heaviness, or deterioration of swimming technique attributable to fatigue—without a concomitant increase in blood uric acid or ammonia. One of these clinical/observational fatigue signs was considered sufficient to indicate clear fatigue; Borg RPE and the uric-acid response were used as additional context. No non-responders were observed in the study sample.

Age, body mass index, body fat percentage, muscle mass, swimming experience, and competitive qualification were available for all 12 athletes in the final dataset; therefore, no imputation of baseline characteristics was required. Baseline covariates were not included in the principal mixed-effects model because the independent sample comprised only 12 athletes, and multivariable covariate adjustment was considered likely to produce unstable estimates.

2.2. Interval Swimming Protocol and Biochemical Guidance

At each assessment, athletes completed three freestyle work bouts separated by a 5-min recovery interval. In the experimental group, the target pace was maintained at approximately 29–31 s/50 m, whereas work-bout duration was individually adjusted by the investigator according to repeated metabolic and perceptual responses. The change in capillary blood uric acid concentration served as the principal biochemical guidance signal; blood ammonia, 1-min post-exercise lactate, and perceived exertion assessed using the Borg 0–10 scale were used as additional contextual indicators.

The load-adjustment procedure used protocol-specific operational zones rather than validated physiological cut-offs. A Δ uric acid of approximately 50 μmol/L or less was treated as the target range. Borg RPE scores of 1–2/10 were considered very light, scores of 3–5/10 represented the target range, 6/10 was considered moderately heavy, and scores ≥ 7/10 indicated high perceived fatigue. A Δ uric acid > 50 μmol/L but <102 μmol/L prompted repeat measurement because this range could reflect either a true increase or measurement variability; if confirmed, particularly together with RPE ≥6/10, the response was treated as excessive, and the subsequent work-bout duration was shortened. A Δ uric acid ≥ 102 μmol/L, especially with RPE ≥ 7/10 and overt fatigue signs, was treated as a high-response zone and resulted in a larger reduction in subsequent duration. Conversely, when uric acid and RPE responses were within or below the target range, work-bout duration was generally increased while pace was maintained. Longitudinal lactate was used as an additional modifier: a reduction > 0.5 mmol/L compared with the preceding similar assessment after approximately 2–3 weeks supported continuation or progression, whereas stagnation or an increase > 0.5 mmol/L prompted reconsideration of interval duration; the direction of adjustment was then determined primarily by the uric-acid and RPE profile. The 0.5 mmol/L value was used as a protocol-specific practical change threshold rather than as a validated physiological cut-off. Ammonia was used as a supportive contextual marker and in the non-responder assessment; no fixed numerical ammonia threshold was used for titration. There was no fixed seconds-based increment or decrement rule: the magnitude of change was determined individually by the investigator, with larger reductions applied when uric acid and RPE responses were further above the target range. These operational zones were specific to this pilot protocol and should not be interpreted as validated universal training thresholds.

The control group underwent the same serial exercise-testing schedule under a standard training approach. Work-bout duration remained predominantly fixed and was not individually adjusted on the basis of uric acid, ammonia, lactate, or Borg ratings. The recovery interval between work bouts was 5 min in both groups. Swimming distance was not independently retained as a numeric variable in the available source dataset. For descriptive reporting across T0–T5, approximate group-level swimming distance was therefore calculated from the reported mean work-bout duration and mean recorded pace using distance (m) = duration (s) × 50/pace (s/50 m); total distance across the three work bouts was calculated as three times the estimated distance per work bout. Because these values are derived from rounded group means rather than athlete-level distance records, they are presented as descriptive estimates only and were not used in inferential analyses.

Assessment time was treated as a categorical variable (T0–T5). The six assessments were performed on 9 September (T0), 23 September (T1), 30 September (T2), 14 October (T3), 28 October (T4), and 4 November 2025 (T5), corresponding approximately to study weeks 0, 2, 3, 5, 7, and 8. Time was modeled categorically to avoid imposing an assumed linear temporal trend across the assessment period.

2.3. Physiological and Perceptual Measures

All biochemical measurements used capillary blood obtained by finger prick. Resting/pre-exercise blood lactate was recorded before the session. Post-exercise lactate was sampled immediately after the ammonia measurement scheduled at 1 min post-exercise and therefore at approximately 1 min after completion of the interval exercise series; values were expressed in mmol/L. This standardized, protocol-defined sampling point was applied consistently at all assessments. The retained study records did not document a separate physiological rationale for selecting 1 min, and the sampling point was not intended to identify each athlete's peak post-exercise lactate concentration. The lactate increment was calculated as the post-exercise concentration minus the resting/pre-exercise concentration.

For uric acid, the reference pre-exercise sample was obtained at rest, and the post-exercise sample was obtained 1 h after completion of the main part of the training session. For ammonia, the reference pre-interval sample was obtained immediately before the measured interval after warm-up rather than at complete rest because preliminary study measurements indicated that ammonia could decline during warm-up; the post-exercise sample was obtained 1 min after completion of the interval series. Δ uric acid and Δ ammonia were calculated as the post-exercise value minus the corresponding reference value and were expressed in µmol/L. Perceived exertion was assessed using the Borg 0–10 scale [10]. Information sufficient to adjust the longitudinal analysis for time of day, recent food or caffeine intake, hydration, preceding exercise, recovery/sleep, and menstrual-cycle status was not available; these factors are therefore considered in the limitations.

Blood ammonia was measured using the PocketChem BA PA-4140 blood-ammonia analyzer (ARKRAY, Inc., Kyoto, Japan) with the AMMONIA TEST KIT II (ARKRAY Factory, Inc., Koka, Japan; 50 tests/kit; Ref. 75304); the manufacturer specifies whole-blood measurement over a range of 10–400 N-µg/dL using a single-wavelength reflectance method. Blood uric acid was measured using the EasyTouch GCU ET-301 multifunctional monitoring system (Bioptik Technology, Inc., Zhunan, Taiwan) with EasyTouch Uric Acid Test Strips (Bioptik Technology, Inc., Zhunan, Taiwan; 25 strips/pack); the manufacturer-specified uric-acid measurement range is 179–1190 µmol/L. Blood lactate was measured using the Lactate Plus analyzer (Nova Biomedical, Waltham, MA, USA) with Lactate Plus Lactate Test Strips (Nova Biomedical, Waltham, MA, USA; 25 strips/pack). Lactate Plus uses an electrochemical lactate biosensor with a manufacturer-specified measurement range of 0.3–25.0 mmol/L; reported analytical precision is within-run CV ≤ 5% (SD ≤ 0.2 mmol/L) and day-to-day CV ≤ 7% (SD ≤ 0.3 mmol/L).

Swimming pace was recorded in seconds per 50 m (s/50 m) and was interpreted as the pace maintained during the interval exercise protocol rather than as the outcome of a separate 50-m maximal-performance test.

2.4. Outcomes

For the present report, blood lactate concentration measured 1 min after exercise was treated as the principal outcome analyzed. This was a retrospective analytical designation because the available source documentation did not indicate that lactate had been prospectively specified as a primary endpoint in a registered statistical analysis plan. Accordingly, the manuscript avoids interpreting this variable as a prospectively defined primary end-

point. Complementary continuous outcomes included the change in uric acid (Δ uric acid), change in ammonia (Δ ammonia), resting blood lactate concentration, lactate increment, and recorded interval swimming pace. Borg rating of perceived exertion (RPE) was analyzed as an ordinal supportive outcome. Work-bout duration was considered primarily an intervention-exposure descriptor rather than an efficacy outcome because it was actively titrated as part of the individualized training approach in the experimental group.

2.5. Statistical Analysis

All statistical analyses were performed using R version 4.5.2. Baseline continuous characteristics were summarized according to the Shapiro–Wilk normality assessment: variables were presented as mean ± standard deviation (SD) when $p \geq 0.05$ in both groups and as median [Q1–Q3] when $p < 0.05$ in either group. Categorical variables were summarized as n (%). Exploratory between-group comparisons of baseline characteristics were performed using Welch's t-test with Hedges' g as the effect-size measure when the normality criterion was satisfied in both groups, and the Mann–Whitney U test with Cliff's delta when non-normality was detected. Sex distribution was compared using Fisher's exact test. Rare categories of competitive qualification were described without formal statistical testing. Baseline p-values were considered exploratory and were not interpreted as evidence of group equivalence in this small, non-randomized sample. Borg rating of perceived exertion (RPE) was summarized as median [Q1–Q3] because of its ordinal nature.

The principal longitudinal analysis was performed using a linear mixed-effects model fitted by restricted maximum likelihood (REML), with fixed effects for group, assessment, and the group × assessment interaction, and a random intercept for athlete. The group × assessment interaction constituted the principal inferential test. Degrees of freedom were estimated using the Kenward–Roger approximation. Estimated marginal means with 95% confidence intervals (CIs) were obtained for each group at each assessment. Prespecified pairwise comparisons were adjusted for multiple testing using the Holm method [11,12].

Model diagnostics included inspection of Q–Q plots and standardized residuals, the Shapiro–Wilk test of model residuals, and assessment of random-effects singularity. Because residuals from the principal model deviated from normality, robustness of the lactate finding was evaluated using four sensitivity analyses: (1) an analogous mixed-effects model fitted to log-transformed lactate concentrations; (2) athlete-level comparisons of T0–T5 changes using Welch's t-test and the Wilcoxon rank-sum test; (3) an exact two-sided permutation analysis evaluating all $C(12,5) = 792$ possible assignments of five experimental-group labels; and (4) a leave-one-athlete-out analysis in which the principal model was refitted after sequential exclusion of each athlete. Because group allocation was non-randomized by design, the permutation analysis was interpreted solely as an exploratory sensitivity analysis under an exchangeability assumption and not as a randomization test.

Continuous secondary outcomes were analyzed using analogous linear mixed-effects models. p-values for the group × assessment interactions across secondary outcomes were adjusted using the Holm method as a single multiplicity family. Models exhibiting singular fits or substantial departures from residual normality were interpreted as exploratory or supportive rather than confirmatory. For Borg RPE, emphasis was placed on median [Q1–Q3] summaries and nonparametric analysis of change. The principal R packages used were lme4, lmerTest, and emmeans [13–15].

2.6. Ethical Considerations

The study was conducted in accordance with the Declaration of Helsinki and was reviewed and approved by the Local Ethics Committee of the non-profit educational organization "Kazakhstan–Russian Medical University" (Protocol No. 22, 22 April 2024).

Participation was voluntary, and the confidentiality of participant data was maintained throughout the study. Written informed consent was obtained from all adult participants before participation. For participants under 18 years of age, written informed consent was obtained from a parent or legal guardian, together with the participant's assent.

3. Results

3.1. Participants and Baseline Characteristics

All 12 swimmers completed all six serial assessments, and there were no duplicate athlete × assessment records or missing post-exercise lactate measurements. The experimental group comprised four men and one woman, whereas the control group comprised five men and two women. Age and body-composition measures were available for all 12 athletes. Mean swimming experience was 9.20 ± 1.30 years in the experimental group and 8.86 ± 1.68 years in the control group. Both groups completed nine training sessions per week, corresponding to approximately 17 h of training per week.

Exploratory baseline comparisons did not identify between-group differences at $p < 0.05$; however, the absence of statistically significant differences should not be interpreted as evidence of baseline equivalence. Despite the small sample size, several anthropometric characteristics showed non-negligible between-group effect sizes, including height (Hedges' g = 0.82), body mass (Cliff's delta = 0.66), body mass index (Hedges' g = 1.00), body fat percentage (Hedges' g = 0.70), and muscle mass (Hedges' g = 0.82). Accordingly, potential baseline imbalances were considered descriptively and represent an additional limitation for causal interpretation (Table 1).

Table 1. Baseline characteristics of the study groups.

Characteristic	Control ($n = 7$)	Experimental ($n = 5$)
Participants, *n*	7	5
Men/women, *n*	5/2	4/1
Age, years	18.43 ± 2.30	18.80 ± 2.28
Height, cm	178.07 ± 6.59	184.56 ± 8.30
Body mass, kg	63.70 [60.70–69.45]	79.50 [64.90–80.70]
Body mass index, kg/m^2	19.91 ± 1.57	21.78 ± 1.94
Body fat, %	11.16 ± 3.11	13.58 ± 3.31
Muscle mass, kg	31.66 ± 5.00	35.80 ± 4.17
Swimming experience, years	8.86 ± 1.68	9.20 ± 1.30
Competitive qualification, CMS/MS/MSIC, *n*	1/4/2	0/3/2
Training sessions per week, *n*	9	9
Training volume, h/week	17	17

Data are presented as mean ± SD when the Shapiro–Wilk test yielded $p \geq 0.05$ in both groups and as median [Q1–Q3] when non-normality was detected in either group. Complete baseline data were available for all 12 athletes; no imputation was required. CMS, Candidate for Master of Sport; MS, Master of Sport; MSIC, Master of Sport of International Class. Exploratory baseline comparisons and corresponding effect sizes are presented in Supplementary Table S1 and should not be interpreted as evidence of baseline equivalence.

3.2. Principal Outcome Analyzed: Post-Exercise Lactate

Post-exercise blood lactate trajectories differed between groups, with a group × assessment interaction of $F(5, 50) = 21.07$ ($p < 0.001$). At T0, mean post-exercise lactate concentrations were 12.19 ± 0.76 mmol/L in the control group and 11.78 ± 1.12 mmol/L

in the experimental group, with no statistically significant between-group difference after Holm adjustment ($p = 0.467$). Thereafter, the between-group separation progressively increased across assessments.

As shown in Table 2, post-exercise lactate remained relatively stable in the control group, ranging from 12.21 ± 0.77 mmol/L at T1 to 11.43 ± 0.69 mmol/L at T5. In contrast, the experimental group showed a progressive reduction from 11.78 ± 1.12 mmol/L at T0 to 7.94 ± 1.14 mmol/L at T5. The adjusted between-group difference increased from 0.41 mmol/L (95% CI, −0.76 to 1.57) at T0 to 3.49 mmol/L (95% CI, 2.33–4.65) at T5, with statistically significant between-group contrasts emerging from T1 onward after Holm adjustment (Table 2).

Table 2. Post-exercise blood lactate concentrations across the six assessments.

Assessment	Control, Mean ± SD (mmol/L)	Experimental, Mean ± SD (mmol/L)	Adjusted Between-Group Difference (95% CI), mmol/L	Holm-Adjusted *p*
T0	12.19 ± 0.76	11.78 ± 1.12	0.41 (−0.76 to 1.57)	0.467
T1	12.21 ± 0.77	10.80 ± 1.12	1.41 (0.25 to 2.58)	0.041
T2	11.93 ± 0.81	9.90 ± 1.11	2.03 (0.87 to 3.19)	0.006
T3	11.89 ± 0.93	9.24 ± 1.08	2.65 (1.48 to 3.81)	<0.001
T4	11.57 ± 0.68	8.42 ± 1.14	3.15 (1.99 to 4.31)	<0.001
T5	11.43 ± 0.69	7.94 ± 1.14	3.49 (2.33 to 4.65)	<0.001

Between-group differences were calculated as control minus experimental using estimated marginal means. The global group × assessment interaction was the principal inferential test. Pairwise between-group comparisons were adjusted using the Holm method.

At T5, the adjusted between-group difference (control − experimental) was 3.49 mmol/L (95% CI, 2.33–4.65; Holm-adjusted $p < 0.001$). Relative to T0, the estimated T0–T5 change was −3.84 mmol/L (95% CI, −4.38 to −3.30) in the experimental group and −0.76 mmol/L (95% CI, −1.22 to −0.30) in the control group. Thus, the between-group difference in T0–T5 change was −3.08 mmol/L, indicating a substantially greater longitudinal reduction in post-exercise lactate in the experimental group. Individual and group-level trajectories are presented in Figure 1.

3.3. Athlete-Level and Model-Based Robustness

The mean T0–T5 change in post-exercise lactate was −3.84 mmol/L in the experimental group and −0.76 mmol/L in the control group, corresponding to a between-group difference in change of −3.08 mmol/L (95% CI, −3.72 to −2.45; Welch's *t*-test, $p < 0.001$). The Wilcoxon rank-sum test yielded a consistent result ($p = 0.0057$). An exploratory exact permutation analysis, performed as a sensitivity analysis under the exchangeability assumption, also supported the observed between-group difference ($p = 0.001$) across all 792 possible assignments of five experimental-group labels among the 12 athletes.

The log-transformed mixed-effects model produced a consistent group × assessment interaction ($F = 30.58$, $p < 0.001$). Robustness was further evaluated using a leave-one-athlete-out analysis. After sequentially excluding each of the 12 athletes and refitting the principal model, the group × assessment interaction remained statistically significant in every iteration (all $p < 0.001$). Collectively, these analyses show that the observed lactate association was not dependent on one analytical specification or a single athlete; however, they do not address selection bias or uncontrolled confounding from the non-randomized design. Athlete-level T0–T5 changes in post-exercise blood lactate, together with group-level summaries, are presented in Figure 2.

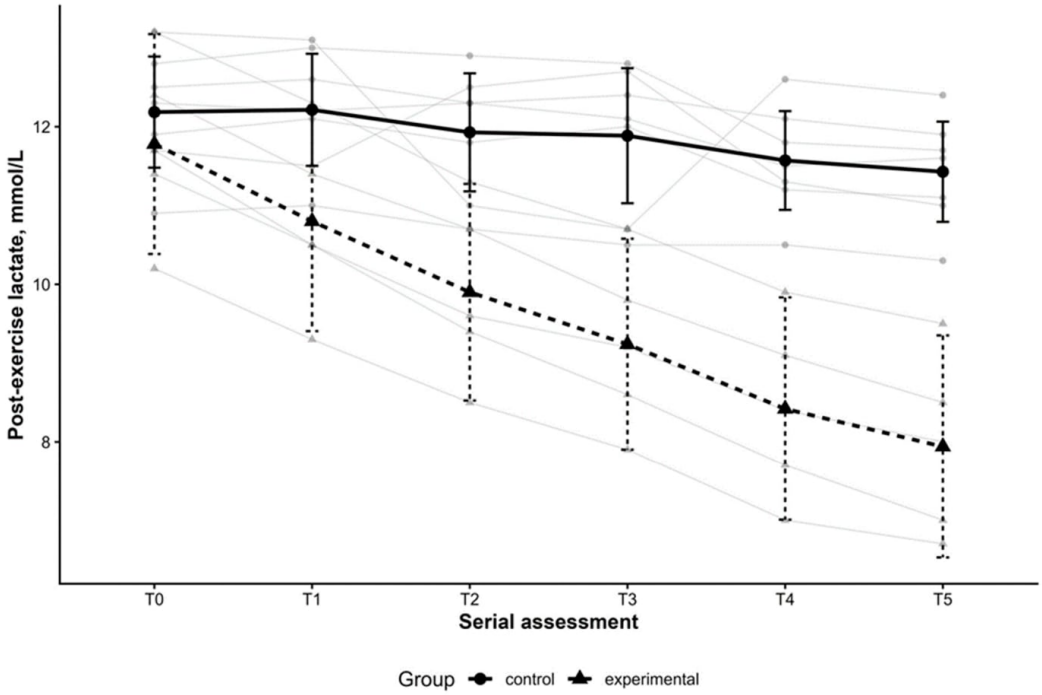

Figure 1. Individual and group-level trajectories of post-exercise blood lactate across T0–T5. Thin lines represent individual swimmers; group means are presented with 95% confidence intervals.

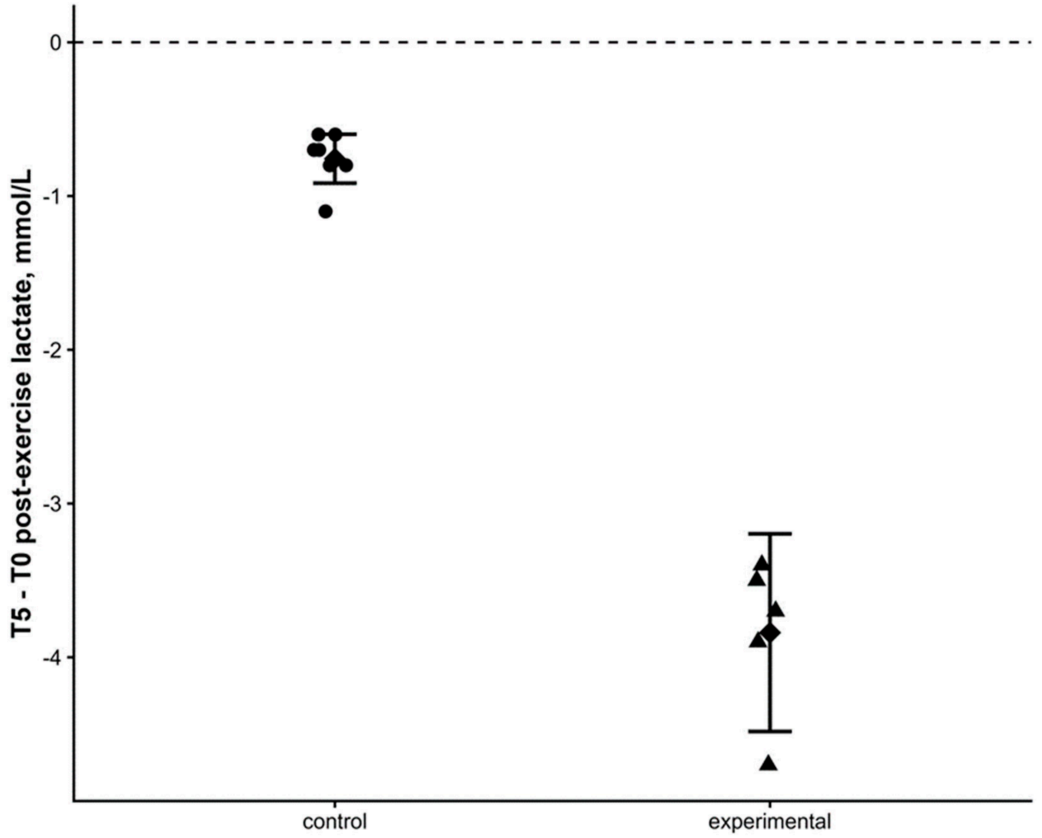

Figure 2. Athlete-level T0–T5 changes in post-exercise blood lactate by group. Individual swimmers are shown as points; group-level summary error bars are superimposed.

3.4. Evolution of Work-Bout Duration and Recorded Pace

Exercise exposure evolved differently between the two groups. In the control group, mean work-bout duration remained 134.29 s at each assessment. In the experimental group, mean work-bout duration was 135.33, 114.00, 127.40, 153.00, 177.20, and 256.67 s at T0, T1, T2, T3, T4, and T5, respectively. Recorded interval pace remained broadly stable across assessments (control, 29.71–30.00 s/50 m; experimental, 30.20 s/50 m). Based on the reported group mean duration and pace, the approximate calculated total swimming distances across the three work bouts at T0–T5 were 674.6, 678.0, 674.6, 678.0, 674.6, and 671.5 m in the control group and 672.2, 566.2, 632.8, 759.9, 880.1, and 1274.9 m in the experimental group, respectively (Table 3). Thus, the experimental protocol involved a progressively modified external exposure that by T5 was substantially longer than the predominantly fixed-duration control protocol, while recorded pace remained similar. Work-bout duration and the derived distance estimates should therefore be interpreted as intervention-exposure characteristics rather than as independent efficacy outcomes.

Table 3. Work-bout duration and calculated swimming-distance estimates across T0–T5.

Assessment	Control Work-Bout Duration, s	Experimental Work-Bout Duration, s	Control Calculated Total Distance, m *	Experimental Calculated Total Distance, m *
T0	134.29 ± 16.18	135.33 ± 19.38	674.6	672.2
T1	134.29 ± 16.18	114.00 ± 16.36	678.0	566.2
T2	134.29 ± 16.18	127.40 ± 15.80	674.6	632.8
T3	134.29 ± 16.18	153.00 ± 16.26	678.0	759.9
T4	134.29 ± 16.18	177.20 ± 22.06	674.6	880.1
T5	134.29 ± 16.18	256.67 ± 12.08	671.5	1274.9

Values are mean ± SD for work-bout duration. * Calculated total distance across three work bouts = 3 × mean work-bout duration (s) × 50/mean recorded pace (s/50 m). Distances are approximate estimates derived from rounded group means; recorded pace values are provided in Supplementary Table S6. Recovery between work bouts was 5 min at every assessment in both groups.

Despite the substantial increase in work-bout duration in the experimental group, recorded interval pace remained essentially stable at 30.20 s/50 m between T0 and T5. Pace was similarly stable in the control group, changing only from 29.86 ± 1.57 s/50 m at T0 to 30.00 ± 1.41 s/50 m at T5. Consistent with these descriptive patterns, the group × assessment interaction for recorded interval pace was not statistically significant ($F(5, 50) = 0.49$; Holm-adjusted $p = 0.786$). Thus, the attenuated 1-min post-exercise lactate response in the experimental group occurred alongside progressively longer work bouts without evidence of systematic slowing during the study protocol (Figure 3). Because work-bout duration was deliberately titrated as part of the intervention, changes in duration should be interpreted as intervention-exposure characteristics rather than independent efficacy outcomes, and the design cannot separate the effects of biochemical monitoring from the effects of the progressively modified exercise exposure itself.

Exact group-level work-bout duration and recorded pace values for T0–T5 are reported in Supplementary Table S6. Swimming distance itself was not independently retained in the source dataset; the distance values in Table 3 are therefore approximate group-level estimates derived from the reported mean duration and pace and were not used for statistical inference.

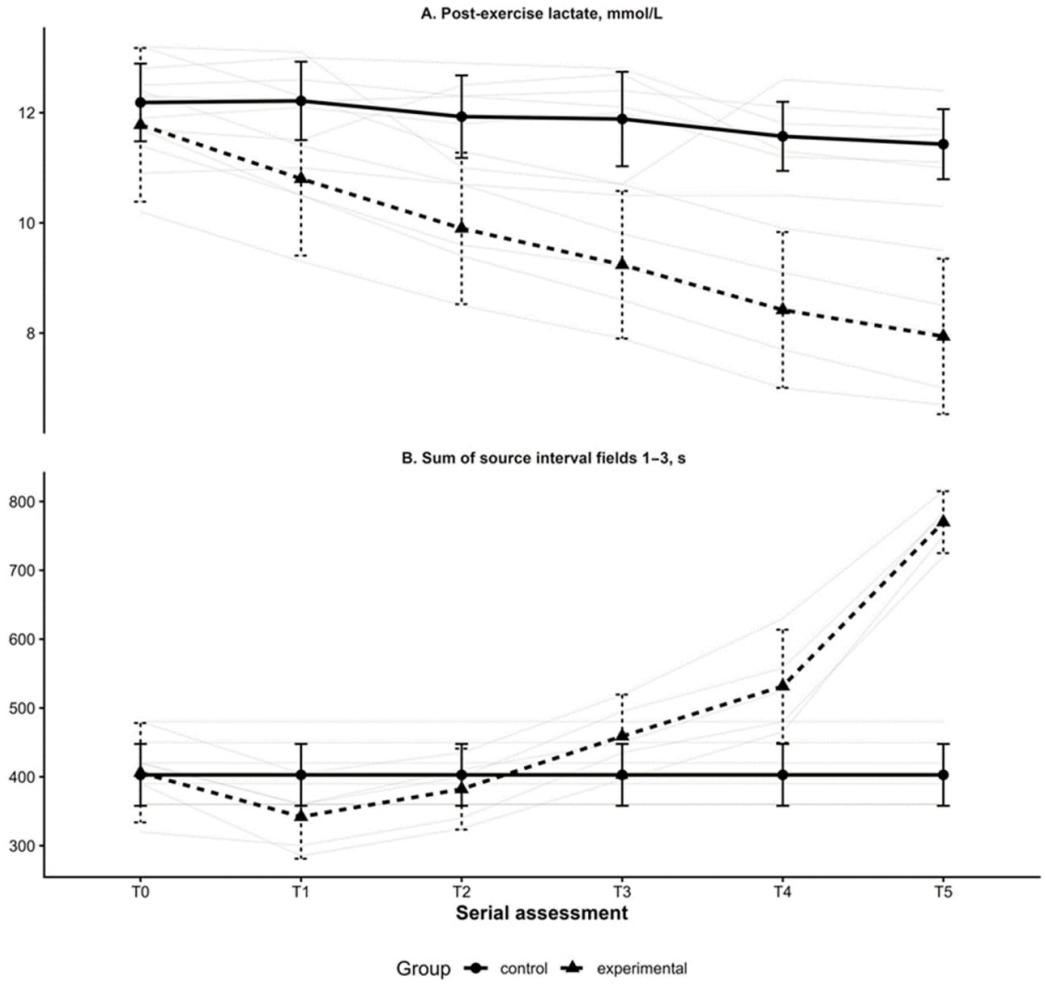

Figure 3. Longitudinal trajectories of post-exercise blood lactate concentration and summed duration of the three work bouts across T0–T5. Separate y-axes are presented in distinct panels to avoid potentially misleading dual-axis scaling.

3.5. Secondary Metabolic and Perceptual Outcomes

Significant group × assessment interactions after Holm adjustment were observed for Δ uric acid, Δ ammonia, and lactate increment (Table 4). In contrast, no evidence of differential longitudinal trajectories was observed for resting lactate or recorded interval pace.

The mixed-effects model for Δ uric acid yielded a singular fit. Moreover, changes in uric acid were used to guide the individual adjustment of work-bout duration in the experimental group. Accordingly, Δ uric acid was interpreted as an exploratory, intervention-guiding biomarker rather than as independent evidence of intervention efficacy. For Δ ammonia, the model was non-singular and the Shapiro–Wilk test of residuals did not indicate a statistically significant departure from normality ($p = 0.075$), making ammonia the most readily interpretable supportive biochemical secondary outcome. Lactate increment also showed a pronounced group × assessment interaction; however, the model residuals deviated from normality, and this finding was therefore interpreted as supportive rather than confirmatory.

Table 4. Continuous secondary group × assessment interaction tests.

Outcome	*F* (df)	*p*	Holm-Adjusted *p*	Singular Fit	Interpretation
Δ uric acid	12.29 (5, 50)	<0.001	<0.001	Yes	Exploratory; intervention-guiding biomarker and singular model
Δ ammonia	8.18 (5, 50)	<0.001	<0.001	No	Supportive secondary biochemical outcome
Resting lactate	1.06 (5, 50)	0.393	0.786	Yes	No evidence of differential longitudinal change
Lactate increment	16.06 (5, 50)	<0.001	<0.001	No	Supportive; residual non-normality
Recorded interval pace, s/50 m	0.49 (5, 50)	0.781	0.786	No	No evidence of differential longitudinal change

Borg RPE was not included in the Gaussian mixed-effects model table because it was treated as an ordinal supportive outcome. Δ indicates the post-exercise value minus the corresponding reference value; for ammonia, the reference sample was obtained after warm-up immediately before the measured interval.

Borg RPE was analyzed as an ordinal supportive outcome rather than within the Gaussian mixed-model framework. The T0–T5 change in Borg RPE was −1.40 points in the experimental group and −0.29 points in the control group. The unadjusted Wilcoxon test yielded $p = 0.014$; after Holm adjustment across secondary change analyses, the corresponding *p*-value was approximately 0.056. Accordingly, the RPE finding was interpreted as supportive and borderline rather than confirmatory.

3.6. Principal Model Diagnostics

The principal random-intercept mixed-effects model did not exhibit a singular fit; however, the residual distribution deviated from normality (Shapiro–Wilk $p < 0.001$), and two standardized residuals had absolute z-scores > 3. Log transformation of post-exercise lactate did not fully normalize the residual distribution, although the group × assessment interaction remained consistent in direction and statistical significance. Accordingly, interpretation of the principal lactate finding was based on convergence across the mixed-effects model and complementary sensitivity analyses, while recognizing that statistical robustness does not remove the design-related risk of selection bias or uncontrolled confounding.

4. Discussion

4.1. Principal Findings

The principal finding of this controlled longitudinal pilot study was a divergence in 1-min post-exercise blood lactate trajectories between swimmers undergoing an individualized, biochemically guided and progressively modified work-bout protocol and those following a predominantly fixed-duration protocol. Both groups showed similar post-exercise lactate concentrations at T0, whereas subsequent assessments demonstrated progressively lower 1-min post-exercise values in the experimental group. By T5, the adjusted between-group difference reached 3.49 mmol/L. The T0–T5 reduction was −3.84 mmol/L in the experimental group compared with −0.76 mmol/L in controls, corresponding to a between-group difference in athlete-level change of −3.08 mmol/L.

The lactate association was consistent across several analytical approaches, including the raw-scale mixed-effects model, log-transformed mixed-effects model, athlete-level Welch and Wilcoxon analyses, exact label-permutation sensitivity analysis, and leave-one-athlete-out refitting. This convergence indicates that the association was not dependent on a single statistical specification or individual swimmer. Nevertheless, statistical consistency cannot correct the non-random allocation, potential selection bias, baseline imbalance, or

uncontrolled confounding; the findings should therefore remain preliminary and should not be interpreted as definitive evidence of a causal intervention effect.

A particularly relevant feature of the findings was that the attenuation of the 1-min post-exercise lactate response occurred while work-bout duration increased substantially in the experimental group. Mean work-bout duration increased from approximately 135 s at T0 to approximately 257 s at T5, whereas recorded interval pace remained close to 30 s/50 m. In contrast, work-bout duration remained predominantly fixed in the control group. Thus, the observed finding is specifically an attenuation of the measured 1-min post-exercise blood lactate concentration during a progressively modified duration-based exposure; it should not be interpreted as direct evidence of reduced metabolic stress, enhanced lactate clearance, or improved metabolic efficiency.

The distinction between external workload and internal physiological response is fundamental to contemporary athlete-monitoring frameworks [1,16]. External load describes the work performed by the athlete, including variables such as duration, distance, and swimming velocity, whereas internal load reflects the individual physiological and perceptual response to that work [1,16]. Contemporary monitoring frameworks therefore support the complementary assessment of internal and external training loads when characterizing athletes' responses to training [1,16].

The present data do not establish the physiological mechanism underlying the lower post-exercise lactate response. Potential explanations could involve differences in lactate production, oxidation, transport, clearance, oxidative metabolism, or the relative metabolic cost of the work performed; however, none of these processes was directly measured. Accordingly, the observed decline in post-exercise lactate should not be interpreted as direct evidence of improved lactate clearance, mitochondrial function, aerobic efficiency, or swimming economy.

4.2. Lactate Response in Relation to Previous Swimming Research

Blood lactate is among the physiological measures used to characterize internal exercise load in competitive swimming, together with measures such as heart rate and perceived exertion [1].

Recent evidence indicates that swimming training sets performed across different intensity domains elicit distinct physiological responses. Nikitakis and Toubekis compared swimming sets across several intensity domains and demonstrated differences in physiological and internal-load responses, supporting the interpretation of lactate within the context of the specific training stimulus rather than in isolation [3]. The present study extends this framework by evaluating repeated within-athlete metabolic responses during a protocol in which work-bout duration was progressively individualized according to biochemical feedback.

Longitudinal evidence also indicates that lactate-related responses can vary across a swimming training cycle. Pelayo et al. followed elite 200-m freestyle swimmers over a 23-week competitive period and evaluated blood lactate recovery in relation to training and performance [17]. Although their study assessed post-exercise lactate recovery rather than the 1-min post-exercise lactate concentration used in the present investigation, it provides relevant longitudinal evidence that lactate-related responses can change across a training period.

Training structure may also influence lactate-related physiological characteristics. Sperlich et al. reported changes in physiological and performance variables following high-intensity interval training in young competitive swimmers [18]. Because their participants were considerably younger and their intervention differed substantially from the present protocol, direct comparison is inappropriate. Nevertheless, their findings demonstrate that

different swimming-training structures may be accompanied by distinct lactate-related and performance responses.

Acute interval studies further demonstrate that blood lactate responses depend on swimming-set characteristics. Terzi et al. evaluated a sprint interval swimming set in relation to the 100-m freestyle event using blood lactate and kinematic variables [19]. Their findings reinforce the importance of interpreting lactate in relation to the intensity and structure of the swimming exercise.

Kabasakalis et al. investigated low-volume sprint interval swimming in master swimmers and demonstrated exercise-induced changes in several blood metabolic biomarkers [20]. Their findings are particularly relevant because they show that interval configuration can influence multiple metabolic responses and that individual biomarkers may exhibit different temporal patterns.

More recent work has examined the relationship between lactate-related metabolic characteristics and sprint swimming performance. Sengoku et al. reported associations between maximal lactate accumulation rate and sprint-performance parameters in male competitive swimmers [21]. However, maximal lactate accumulation rate is methodologically distinct from the 1-min post-exercise lactate concentration measured in the present investigation. Accordingly, the observed decline in post-exercise lactate should not be interpreted as evidence of either increased or decreased glycolytic capacity.

4.3. Swimming Pace and Training Exposure

The maintenance of recorded swimming pace provides context for the training exposure. Despite the substantial increase in work-bout duration in the experimental group, recorded pace remained approximately 30 s/50 m, and no significant group × assessment interaction was identified for pace. Therefore, the progressive attenuation of the 1-min post-exercise lactate response was not accompanied by systematic slowing during the protocol.

However, interval pace should not be considered equivalent to competitive swimming performance. The study did not include a standardized 50-m, 100-m, 200-m, or event-specific time trial as a formal performance outcome. Therefore, the study did not test or verify an improvement in sports performance. The findings demonstrate maintenance of protocol-specific pace during increasing exercise exposure rather than improvement in race performance or proof of enhanced internal metabolic efficiency.

This distinction is consistent with training-monitoring principles emphasizing that internal physiological responses should be interpreted in relation to the external workload performed [1,16]. Changes in a single internal marker should therefore not be interpreted in isolation as evidence of improved sporting performance.

4.4. Perceived Exertion and Multimodal Monitoring

The perceptual findings showed the same general direction as the biochemical outcomes. Borg RPE decreased more between T0 and T5 in the experimental group than in controls. However, after correction for multiple secondary comparisons, the nonparametric result was borderline. Therefore, RPE should be considered a supportive rather than confirmatory outcome.

Psycharakis evaluated RPE longitudinally in elite swimmers and demonstrated its validity and reliability for monitoring exercise intensity, including associations with heart rate and blood lactate [9]. Wallace et al. likewise demonstrated the practical applicability of the session-RPE method for quantifying internal training load in swimmers [22].

Haddad et al. reviewed the validity, ecological usefulness, and factors influencing the session-RPE method and supported its application for internal training-load monitoring while emphasizing that perceptual responses can be affected by several physiological,

psychological, and contextual factors [23]. These considerations support the inclusion of RPE within a multimodal monitoring framework rather than its use as an isolated marker.

Recent competition-based evidence provides additional support for considering physiological and perceptual measures together. López-Hernández et al. evaluated blood lactate, heart rate, RPE, and neuromuscular responses in national-level youth swimmers and found that lactate and RPE were particularly responsive to competition-induced fatigue [24]. This study further illustrates the complementary information provided by physiological and perceptual measures when characterizing swimmers' responses to competitive exercise.

4.5. Ammonia as a Supportive Metabolic Outcome

The ammonia findings provide complementary evidence that the longitudinal metabolic response was not limited to lactate. The experimental group demonstrated a greater attenuation of the ammonia response over time. In contrast to the uric-acid model, the ammonia mixed model was non-singular and showed comparatively satisfactory residual behavior, supporting its interpretation as a supportive biochemical secondary outcome.

Exercise-induced blood ammonia is related to adenine nucleotide metabolism and may increase during high-intensity exercise. Korman et al. evaluated blood ammonia and lactate during incremental exercise to exhaustion in elite athletes and demonstrated that both metabolites responded to increasing exercise demands, although their response patterns were not identical [6].

The parallel attenuation of lactate and ammonia responses in the present study may therefore be compatible with a broader change in exercise-related metabolic response. Nevertheless, this remains a supportive interpretation. The study did not validate ammonia as a training-prescription threshold, nor did it establish the specific metabolic mechanism responsible for the longitudinal ammonia changes.

4.6. Uric Acid and Purine Metabolism

Uric acid requires particularly conservative interpretation because it had a dual role in the experimental protocol. It was measured as a metabolic response but was also the principal biochemical signal used to guide adjustment of subsequent work-bout duration. Consequently, its longitudinal trajectory was partly linked to the intervention algorithm itself and cannot be considered an independent confirmatory outcome.

There is, however, a physiological rationale for examining purine-related metabolites during exercise. Zieliński et al. investigated purine metabolism in sprint- and endurance-trained athletes and demonstrated training-related differences and exercise-induced changes in purine metabolites, including hypoxanthine, xanthine, and uric acid [7]. These findings support the involvement of purine metabolism in exercise-related energetic processes but do not establish uric acid as a validated marker for prescribing swimming workload.

Kabasakalis et al. additionally demonstrated changes in uric acid following sprint interval swimming and showed that its temporal response differed from the more immediate lactate response [20]. This provides biological support for including uric acid in metabolic assessment. However, neither the available literature nor the present study establishes uric acid as a validated threshold for individualized prescription of swimming interval duration.

Furthermore, the uric-acid mixed model in the present analysis was singular. Combined with its direct role in guiding the intervention, this statistical limitation supports its classification as a guidance/exploratory variable rather than independent evidence of efficacy.

4.7. Lactate Increment and Resting Lactate

The pattern of lactate increment provides additional context for interpretation of the principal outcome analyzed. Resting lactate did not demonstrate a differential longitudinal trajectory between groups, whereas the increase from resting to post-exercise lactate became markedly smaller in the experimental group. This pattern indicates that the between-group divergence was observed primarily in the exercise-related measurement rather than in resting lactate; it does not establish a specific mechanism of lactate production or clearance.

This interpretation is consistent with evidence showing that blood-lactate responses depend on exercise intensity, swimming-set configuration, and metabolic demand [3,19,20]. Nevertheless, the present study did not directly measure lactate appearance, transport, oxidation, or clearance. The finding should therefore be described as an attenuated exercise-related lactate response rather than as direct evidence of altered lactate kinetics.

4.8. Practical Implications

From an applied perspective, the findings support further investigation of individualized multimodal monitoring in competitive swimming. Feijen et al. emphasized the importance of selecting monitoring strategies according to the swimmer, training context, practical purpose, and characteristics of the monitoring method [1].

Similarly, broader athlete-monitoring recommendations support consideration of internal responses alongside external training exposure [16]. Halson emphasized the importance of monitoring training load and athlete responses for understanding training-related fatigue while noting that no single marker provides a complete representation of athlete status [25].

The present intervention is compatible with these general principles because it combined biochemical, perceptual, and external training information across repeated assessments. However, the protocol-specific operational zones used here should not yet be considered validated universal training thresholds. The present data do not establish that the uric-acid, lactate, ammonia, or RPE rules used in this pilot study are optimal for other athletes or settings, nor do they demonstrate that any individual marker should independently determine progression or reduction of swimming workload.

Rather, the present findings provide preliminary evidence that repeated individualized metabolic assessment can be incorporated into an interval-swimming protocol and may be associated with a distinct longitudinal internal-response pattern compared with a predominantly fixed-duration approach.

4.9. Strengths and Limitations

Several methodological features strengthen the present investigation. First, all 12 swimmers provided complete post-exercise lactate data across all six assessments, avoiding attrition and missing-data problems for the principal lactate outcome analyzed. Second, repeated observations within athletes were explicitly accounted for using mixed-effects modeling. Third, Kenward–Roger denominator degrees of freedom were used to improve small-sample inference. Fourth, planned contrasts and complementary-outcome analyses incorporated Holm adjustment for multiplicity. Fifth, the lactate association was subjected to several complementary sensitivity analyses, including an analogous log-lactate model, athlete-level Welch and Wilcoxon comparisons, exact label permutation across all 792 possible group-label allocations, and leave-one-athlete-out refitting.

Another strength is that the study simultaneously documented changes in external training exposure and internal metabolic response. The experimental group progressively increased work-bout duration while maintaining a broadly stable interval pace, providing important contextual information for interpreting the attenuation of the measured post-exercise lactate response.

Several limitations should nevertheless be emphasized. First, the sample comprised only 12 independent swimmers, with five athletes in the experimental group and seven in the control group. The repeated-measures design increases the amount of within-athlete information but does not compensate for the very small number of independent participants. This limits precision, statistical power, subgroup analysis, and generalizability and is the principal reason the study should be considered a pilot investigation.

Second, group allocation was non-randomized and was determined pragmatically by the investigator according to whether the athlete's existing plan was considered feasible for modification while preserving functional reserve. This qualitative allocation process may have been related to pre-existing training status, prior training response, or tolerance of workload. Consequently, selection effects and uncontrolled confounding cannot be excluded, and the observed between-group differences cannot be attributed specifically to the biochemical guidance procedure.

Third, although exploratory baseline comparisons did not yield $p < 0.05$, several anthropometric characteristics showed non-negligible between-group effect sizes, including greater body mass and muscle mass in the experimental group. Baseline equivalence therefore cannot be assumed. These differences could have influenced the absolute external work performed and exercise-related metabolite production, distribution, buffering, or clearance independently of the intervention; the direction and magnitude of any such influence cannot be determined from this small dataset.

Fourth, although weekly training frequency and approximate training volume were similar between groups, the content, intensity distribution, and adherence to training outside the serial study assessments were not quantitatively characterized in detail. Longitudinal metabolic changes may therefore partly reflect differences in the broader training process between T0 and T5 rather than the study protocol alone.

Fifth, potentially relevant determinants of metabolic and perceptual responses—including recent nutrition and carbohydrate availability, caffeine intake, hydration, prior exercise, recovery, sleep, time of day, and menstrual-cycle status—were unavailable for adjustment. In addition, lactate was sampled at a fixed 1-min post-exercise time point. Because blood lactate may not peak at 1 min and exercise duration changed considerably across assessments in the experimental group, the fixed sampling time may not represent the same point relative to each athlete's post-exercise lactate peak at T0 and T5.

Sixth, the designation of 1-min post-exercise lactate as the principal outcome analyzed in this report was retrospective rather than prospectively prespecified in a registered statistical analysis plan. Seventh, residuals from the principal raw-scale mixed-effects model departed from normality. Although the finding remained concordant in log-transformed, nonparametric, permutation, and leave-one-athlete-out analyses, these sensitivity analyses address analytical robustness only and cannot remove selection bias, baseline imbalance, or other uncontrolled confounding arising from the non-randomized design.

Eighth, some complementary outcome models were singular or demonstrated departures from residual normality and should therefore remain supportive or exploratory.

Ninth, uric acid was part of the intervention-guidance process, limiting its independence as an outcome and precluding its use as confirmatory evidence of intervention efficacy.

Tenth, Borg RPE was an ordinal supportive outcome, and the multiplicity-adjusted T0–T5 result was borderline rather than clearly significant.

Eleventh, swimming distance was not independently recorded at the athlete level. The distances reported here are approximate group-level estimates derived from rounded mean work-bout duration and pace and should not be interpreted as directly measured external-load data. Finally, recorded pace represented interval pace within the experimental

protocol rather than a standardized competitive-performance outcome. Maintenance of pace therefore supports description of the training exposure but cannot be extrapolated to improved race performance, and the present study did not test whether the individualized protocol improved time-trial or competitive performance.

4.10. Future Research

The present findings justify larger prospective studies evaluating individualized metabolic monitoring in competitive swimmers. Future investigations should use clearly documented random allocation where feasible, prospectively define primary and secondary outcomes, and determine sample size using an a priori calculation based on a prespecified meaningful effect.

Future studies should prospectively prespecify and validate the biochemical-guidance algorithm, including reproducible criteria for increasing, maintaining, or decreasing work-bout duration. The protocol-specific uric-acid, lactate, and RPE decision thresholds used in the present pilot study require independent validation, and analytical reliability, biological variability, and practically meaningful thresholds for each biomarker should be established.

Pre-assessment nutrition, hydration, recent training exposure, recovery, sleep, and sampling time should be standardized or prospectively documented. Future protocols should additionally include formal performance outcomes, such as standardized 50-m, 100-m, 200-m, or event-specific time trials, to determine whether a lower metabolic response during training is associated with meaningful changes in competitive performance.

Integration of biochemical monitoring with heart rate, oxygen-consumption-derived variables, recovery kinetics, perceived exertion, and objective measures of external workload may further clarify the mechanisms underlying the observed responses. Such a multidimensional approach is consistent with contemporary athlete-monitoring principles [1,16,25].

4.11. Overall Interpretation

In summary, the individualized, biochemically guided protocol was associated with a progressive attenuation of the 1-min post-exercise lactate response compared with a predominantly fixed-duration protocol. This occurred while work-bout duration increased substantially and recorded interval pace remained broadly stable. The lactate association was consistent across multiple sensitivity analyses, while ammonia and perceived exertion provided supportive evidence of a broader change in measured internal responses. The design cannot determine whether the observed longitudinal difference arose from biochemical guidance itself, the progressively modified exercise exposure, or their combination.

Nevertheless, the small non-randomized pilot design requires cautious interpretation. Potential baseline imbalance, selection effects, uncontrolled confounding, unmeasured differences in training exposure and recovery conditions, and the fixed 1-min lactate sampling time cannot be excluded as contributors to the observed pattern. Uric acid cannot be considered an independent confirmatory outcome because it was incorporated into intervention guidance. RPE provided only supportive evidence after multiplicity adjustment, and recorded pace was not a formal competitive-performance outcome.

Accordingly, the findings should be regarded as preliminary evidence supporting further prospective evaluation of individualized multimodal metabolic monitoring in competitive swimming rather than as validation of a specific biochemical training-prescription strategy.

5. Conclusions

Across six consecutive assessments, swimmers in the experimental group undergoing an individualized, biochemically guided and progressively modified work-bout protocol

showed a substantially greater attenuation of 1-min post-exercise blood lactate than swimmers following a predominantly fixed-duration control protocol. This pattern occurred while recorded interval pace remained broadly stable and work-bout duration increased substantially in the experimental group. The lactate association was consistent across complementary analytical approaches, but these analyses do not eliminate selection bias or uncontrolled confounding. No standardized time-trial or competitive-performance outcome was assessed, and the study therefore does not demonstrate improved sports performance or establish a mechanism of enhanced lactate clearance or metabolic efficiency.

Given the small sample size, retrospective designation of the principal outcome analyzed in this report, and non-randomized group allocation, these findings should not be interpreted as evidence of a causal intervention effect. Rather, they provide preliminary support for further prospective evaluation of individualized, biochemically guided interval-training protocols with prespecified decision rules, standardized sampling and recovery conditions, objective external-load measures, and formal performance outcomes.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/sports14110479/s1, Supplementary Methods S1: Robustness and Model Diagnostics; Table S1: Exploratory baseline comparisons and effect-size estimates; Table S2: Post-exercise lactate estimated marginal means and between-group contrasts (principal outcome analyzed in this report); Table S3: Within-group contrasts against T0 for post-exercise lactate; Table S4: Athlete-level T0-to-T5 change sensitivity analyses; Table S5: Mixed-model diagnostic summary; Table S6: Work-bout duration, recorded interval pace, and calculated distance estimates across T0–T5; Figure S1: Q–Q plot of residuals from the principal post-exercise lactate mixed-effects model; Figure S2: Residuals versus fitted values for the principal post-exercise lactate mixed-effects model; Figure S3: Borg RPE raw observations and group medians [Q1–Q3]; Figure S4: Descriptive trajectories of secondary metabolic outcomes and recorded interval pace. Separate panels use independent y-scales.

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Informed Consent Statement: Written informed consent was obtained from all adult participants involved in the study. For participants under 18 years of age, written informed consent was obtained from a parent or legal guardian, together with the participant's assent.

Data Availability Statement: The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to ethical approval, participant-confidentiality restrictions, and prior authorization from the Ministry of Healthcare of the Republic of Kazakhstan, which owns the data collected under the Ministry-funded research program.

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