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Lactate Profile Changes in Relation to Training Characteristics in Junior Elite Cyclists

Arne Guellich and Stephen Seiler

Purpose: To compare the intensity distribution during cycling training among elite track cyclists who improved or decreased in ergometer power at 4 mM blood lactate during a 15 wk training period. ***Methods:*** 51 young male German cyclists (17.4 ± 0.5 y; 30 international, 21 national junior finalists) performed cycle ergometer testing at the onset and at the end of a 15 wk basic preparation period, and reported their daily volumes of defined exercise types and intensity categories. Training organization was compared between two subgroups who improved (Responders, n = 17; $\Delta P_{La4} \cdot kg^{-1}$ = +11 ± 4%) or who decreased in ergometer performance (Non-Responders, n = 17; $\Delta P_{La4} \cdot kg^{-1}$ = –7 ± 6%). ***Results:*** Responders and Non-Responders did not differ significantly in the time invested in noncycling specific training or in the total cycling distance performed. They did differ in their cycling intensity distribution. Responders accumulated significantly more distance at low intensity (<2 mM blood lactate) while Non-Responders performed more training at near threshold intensity (3–6 mM). Cycling intensity distribution accounted for approx. 60% of the variance of changes in ergometer performance over time. Performance at t_1 combined with workout intensity distribution explained over 70% of performance variance at t_2. ***Conclusion:*** Variation in lactate profile development is explained to a substantial degree by variation in training intensity distribution in elite cyclists. Training at <2 mM blood lactate appears to play an important role in improving the power output to blood lactate relationship. Excessive training near threshold intensity (3–6 mM blood lactate) may negatively impact lactate threshold development. Further research is required to explain the underlying adaptation mechanisms.

Keywords: endurance athletes, training intensity, blood lactate, training volume

Elite track cyclists are reported to perform a large volume of cycling specific endurance training. For example, Schumacher and Mueller reported that a group of Olympic gold medal winning 4 km pursuit cyclists trained primarily on the road and accumulated cycling volumes of 29 to 34,000 km annually.[1] Debate continues regarding the relative impact of exercise intensity and duration on the physiological

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adaptation process.[2–5] In recent years, a number of descriptive studies have emerged reporting the intensity distribution of well trained endurance athletes in different sports.[1,6–12] Taken together, these studies suggest a common organizational strategy where successful endurance athletes over a range of event durations from 4 min to 4+ h tend to perform about 80% of their training sessions at intensities clearly below the first lactate turn point (<2 mM blood lactate, or 50% to 75% VO_2max). About 20% of training sessions are "high intensity workouts" characterized by a primary portion of the training session being performed as continuous or intermittent bouts at intensities eliciting blood lactate concentrations in the approximately 3 to 10 mM range, or approximately 85% to 98% VO_2max. Potential selective pressures driving training organization toward a common distribution have been introduced and may include specific benefits of longer training duration and/or a strategy to minimize training stress for a given technical or physiological adaptation benefit.[9,11,13,14] However, these arguments remain primarily speculative.

When an athlete trains regularly for weeks or months in preparation for a competitive season, an increase in power at defined blood lactate concentrations (ie, 2 mM and 4 mM) is anticipated. Quantification of the blood lactate-power output relationship is a routine procedure incorporated into the training monitoring of many competitive endurance athletes. However, experience suggests that the magnitude of physiological adaptation observed to a standardized mesocycle of endurance training will vary individually, with some athletes not showing expected physiological development.[15] One of several potential explanatory factors for this individual variation may be variation in the intensity distribution of the performed training.

In the current study we extend previous descriptive observations of training intensity distribution by retrospectively comparing the intensity distribution within specific cycling workouts among (1) high performance junior track cyclists who improved and (2) athletes of the same performance standard who stagnated, or showed deterioration in their cycling power to blood lactate relationship during a training period of 15 wk.

Methods

Study Design

This study employed data from (1) centralized cycling ergometer testing performed at the beginning of the training season (mid November; t_1) and at the end of the Basic Preparation Period (BPP, 15th training week, end of February; t_2) and (2) the complete daily training data provided by members of the men's German junior national track cycling squad. An unchanged scheme of performance assessment and training documentation was employed in German cycling from 1993 until 2002. This standardization enabled the inclusion of complete data sets from 51 members of the junior national squad whose focus was on longer track events. While the central ergometer performance assessment at t_1 and t_2 was obligatory for the squad members, only 26 of the 51 cyclists participated in a 3000 m test on track offered by the NGB at the end of the Specific Preparation Period (25th training week, t_3). The group that chose to perform this 3rd test was highly biased toward the most successful athletes. Therefore this data were not included in the analysis. This study was approved by the German Federal Institute of Sports Science including

the subjects' informed, written consent for their performance and training data to be used for research purposes.

Subjects

All 51 athletes (age 17.4 ± 0.5 y; body mass 70.1 ± 5.6 kg) were members of the national junior development squad at the time of data collection. Of these, 19 won medals at junior world championships, 11 others placed in the top 10 internationally, 13 more were medalists at the national junior championships, and the other 8 were national finalists.

We used the measured change in cycling power per kilogram of body mass at 4 mmol·L^{-1} venous blood lactate ($\Delta P_{La4} \cdot kg^{-1}$) from week 1 to week 15 of the BPP as the criterion measure for comparison of athletes who showed clearly positive physiological responses to training with those athletes who responded poorly. Specifically, the 33rd and 66th percentiles for $\Delta P_{La4} \cdot kg^{-1}$ demarcated two groups of cyclists: "Non-Responders" (n = 17, $\Delta P_{La4} \cdot kg^{-1} = -7 \pm 6\%$, range –18 to 0%) and "Responders" (n = 17; $\Delta P_{La4} \cdot kg^{-1} = +11 \pm 4\%$; range +7 to +20%; group difference $P < .01$). The results of this study are based on comparison of these two subgroups drawn from the initial sample of 51 athletes.

Physiological Testing

An intermittent cycle ergometer test to exhaustion was performed on a Schoberer cycling ergometer at t_1 and t_2 (SRM, Jülich, Germany; 3 min stages, onset at 100 W, 20 W steps). Blood samples were extracted from arterialized blood taken from the earlobe and immediately analyzed (Biosen 5030L; EKF Diagnostik GmbH, Magdeburg, Germany). Cycling power relative to body mass (W·kg^{-1}) at 2 and 4 mmol·L^{-1} venous blood lactate ($P_{La2} \cdot BM^{-1}$; $P_{La4} \cdot BM^{-1}$) was calculated from the blood lactate/cycling ergometer power relationship using dedicated software (Winlactate; Mesics Software for Medical Science GmbH, Münster, Germany). P_{La2} values were not acquired in two athletes because their blood lactate concentration exceeded 2 mmol·L^{-1} already during the initial stage. Incremental increases in load were continued until voluntary exhaustion for the determination of maximal cycling power (P_{max}). In cases where a 3 min stage was only partially completed, P_{max} was calculated as the power of the last completed stage + 20 W • completed time (s)/180. The national cycling governing body did not perform standardized, centralized VO$_2$max testing on junior athletes. Therefore, information regarding the maximal oxygen consumption of these athletes was not available.

Training Monitoring

Before the start of the 15 wk basic preparation period, athletes were briefed regarding the desired training composition by the national coach and provided a reporting scheme. Cycling intensity was categorized as defined by the national cycling federation (see Table 1). Individual heart rate (HR) ranges for the defined intensity categories in training were prescribed based on the stable blood lactate-heart rate relationship determined during the cycling ergometry test performed at t_1. Heart rate was controlled during all cycling sessions via real-time HR-monitoring and downloadable records (Polar, Kempele, Finland). Athletes reported the daily distance

Table 1 Category definitions for specific cycling training intensity as prescribed by the German national governing body in cycling

Training Intensity Category	Distance per session (km)	Cadence (n·min⁻¹)	Heart rate (% HRmax)	Blood lactate (mmol·L⁻¹)
Compensation range (easy ride alone or with friends)	no prescription	no prescription	no prescription	minimal
Basic Endurance range	1 • 80–200 km	90/100	60–75%	≤2
On Bike Strength Training range	1–8 • 1–5 km	50–70	67–73%	2–3
Development range	2–6 • 5–20 km	80–100 / 110–120	70–90%	3–6
Competition (road racing)	80–130 km	80–100 / 110–120	70–90%	3–6
Peak range, Race-Specific Endurance range	2–6 • ≤2 km	115–125 / 120–130	90–100%	>6

First column labels represent direct translations from the national governing body's documents.

Note: Cadence ranges for Basic Endurance, Development, Competition, and Peak range represent prescriptions for individual and for group training. Individual heart rates for targeted intensity ranges based on Lactate-HR-relation during cycling ergometry at t_1. Cadence and heart rate in road races may momentarily exceed the prescribed values.

performed in each intensity category in specific cycling workout and also the time spent in noncycling specific strength training and general athletic training (game play, jogging, gymnastics) in a digital training diary. The scheme for noncycling specific strength training prescribed by the NGB during the BPP was composed by 1 to 2 times per week 5 to 10 • 10 • 50% to 85% 1RM squat (lowering down to 70° knee angle) and 3 to 6 • 10 • 50% to 80% 1RM of each, arm curl and arm press. The training data reported here represents the actual training reported by the athletes for the complete 15 wk of the BPP.

The intensity distribution in specific cycling was compared between Responders and Non-Responders using time-in-zone analysis based on heart rate cut-offs from ergometer testing performed at t_1.

Statistical Analysis

All statistical analyses were performed using SPSS version 16.0. Physical, physiological, and training characteristics are presented as means and standard deviations. Interactions between Group and Time for physical and performance variables were tested using a 2×2 ANOVA with repeated measures for time. Independent Samples T-tests were conducted for group comparisons of specific cycling training volumes at different intensities and Kruskal-Wallis test for group comparisons of time spent in noncycling specific strength training and general athletic training because of their skewed distribution. To study the multivariate contribution of cycling volumes at varying intensities to explaining changes in ergometer performance from t_1 to t_2, we used Multiple Linear Regression Analyses (MLR, backward variable inclusion method; exclusion criterion $P > .10$). A P value of <0.05 was considered statistically significant in all procedures.

Results

Responders and Non-Responders did not differ significantly in age (17.5 ± 0.5 vs 17.2 ± 0.4 y; $P > .05$) or body mass at t_1 (68.1 ± 5.7 vs 71.3 ± 4.8 kg; $P > .05$) or t_2 (68.2 ± 5.4 kg vs 71.4 ± 4.0 kg, respectively; $P > .05$).

Responders performed absolute P_{La2}, P_{La4}, and P_{max} scores of 226 ± 40, 283 ± 35, and 334 ± 34 W, respectively, at t_1 and 259 ± 36, 314 ± 38, and 356 ± 29 W at t_2. The Non-Responders' data were 254 ± 39, 308 ± 34, and 350 ± 30 W at 2 and 4 mmol·L^{-1} blood lactate and at the exhaustion stage at t_1 and 230 ± 30, 284 ± 21, and 335 ± 24 W at t_2. A histogram of the individual changes in P_{La4}·BM^{-1} for all 51 athletes is presented in Figure 1.

The P_{La2}·BM^{-1}, P_{La4}·BM^{-1}, and P_{max}·BM^{-1} values of both groups at t_1 and t_2 are shown in Figure 2. While there were no significant group differences at t_1 ($P > .05$), their differing responses to the training period resulted in significant differences in threshold cycling power and maximal power output at t_2 ($P < .01$). The performance increase of the Responders and the decrease of the Non-Responders from t_1 to t_2 were both significant ($P < .01$) as well as the interaction of Group and Repeated Measure for time ($P < .01$).

Responders and Non-Responders did not differ significantly in the total time invested in noncycling specific training (strength training 19 ± 11, range 3 to 44 vs
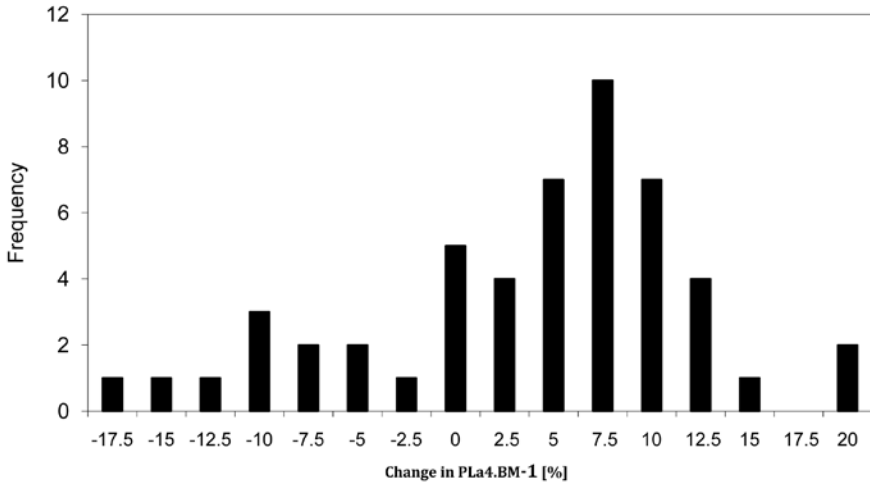
Figure 1 — Frequency distribution of change in P4mM during the 15 wk training period (n = 51).

18 ± 13, range 0 to 38 h; $P > .05$; general athletic training 30 ± 14, range 1 to 59 vs 26 ± 17, range 6 to 48 h; $P > .05$, respectively), or total cycling volume (4073 ± 785 vs 3648 ± 353 km; $P > .05$). The two groups did differ significantly in the intensity distribution of their cycling specific training (Table 2). Responders accumulated significantly more training in the lowest intensity categories (<2 mmol·L^{-1} blood lactate; Compensation, Basic Endurance range). Non-Responders actually accumulated significantly more kilometers at near lactate threshold intensity (3–6 mM blood lactate). Overall, Responders performed 6 ± 3% of their cycling volume at 3 to 6 mM blood lactate compared with 12 ± 3% in Non-Responders ($P < .01$). Neither group trained meaningful amounts at peak, track-specific intensity during the basic preparation period. However, the very small amount the Responders trained at peak cycling intensity was significantly greater than that of Non-Responders ($P < .05$).

Multivariate analyses (MLR) revealed that the independent variables of cycling volumes at different intensities accounted for a considerable amount of variance of the dependent variable of relative change in ergometer performance from t_1 to t_2. Accordingly, ergometer performance at t_1 together with the training organization from t_1 to t_2 accounted for a substantial portion of the variance in ergometer performance at t_2. Based on equations E1 to E6 below, 35% of the variance in P_{max} changes over time, over 60% in performance changes at La4 and La2, and about 70% of the variance in performance at t_2 was explained:

$$\%\Delta P_{max} \cdot BM^{-1} = 0.04 + 0.16 \cdot s_{CR} + 0.26 \cdot s_{SoB} - 0.22 \cdot s_{DR} - 0.27 \cdot s_{Comp} + 0.28 \cdot s_{PR}$$
$$R = 0.67;\ R^2_{adj} = 0.35 \tag{E1}$$

Table 2   Intensity distribution of cycling distance over 15 wk Basic Preparation Period in athletes whose performance improved (Responders; n = 17) and whose performance deteriorated (Non-Responders; n = 17) in $P_{La4} \cdot BM^{-1}$

Intensity Range	Responders Mean ± SD	Non-Responders Mean ± SD	P value
Compensation	216 ± 155	67 ± 53	< 0.01
Basic Endurance	3506 ± 692	3061 ± 321	< 0.05
On Bike Strength training	104 ± 85	77 ± 56	n.s.
Development	50 ± 32	88 ± 39	< 0.01
Competition	195 ± 83	354 ± 112	< 0.01
Peak range, Race-Specific Endurance	3 ± 2	1 ± 1	< 0.01
Σ <2 mmol·L^{-1} blood lactate	3722 ± 724	3128 ± 310	< 0.01
Σ 3–6 mmol·L^{-1} blood lactate	244 ± 103	442 ± 107	< 0.01

Mean values ± standard deviations of the accumulated distances (km) from t_1 until t_2 in each category. n.s. = not significant ($P > .05$).

Note. Σ <2 mmol·L^{-1} blood lactate sums the volume from the intensity categories of "Compensation" and "Basic Endurance" range. Σ 3–6 mmol·L^{-1} blood lactate sums the training kilometers performed in intensity categories "Development" range and Competition.

$$\%\Delta P_{La4} \cdot BM^{-1} = 0.04 + 0.17 \cdot s_{CR} + 0.10 \cdot s_{BER} - 0.18 \cdot s_{SoB} - 0.42 \cdot s_{DR} - 0.32 \cdot s_{Comp} + 0.28 \cdot s_{PR}$$

$$R = 0.84; \; R^2_{adj} = 0.64 \tag{E2}$$

$$\%\Delta P_{La2} \cdot BM^{-1} = 0.14 + 0.13 \cdot s_{BER} + 0.13 \cdot s_{SoB} - 0.55 \cdot s_{DR} - 0.27 \cdot s_{Comp} + 0.29 \cdot s_{PR}$$

$$R = 0.82; \; R^2_{adj} = 0.62 \tag{E3}$$

$$P_{max} \cdot BM^{-1}_{t2} = 3.60 + 0.17 \cdot P_{max} \cdot BM^{-1}_{t1} + 0.28 \cdot s_{CR} + 0.29 \cdot s_{BER} + 0.26 \cdot s_{SoB} - 0.23 \cdot s_{DR} - 0.31 \cdot s_{Comp}$$

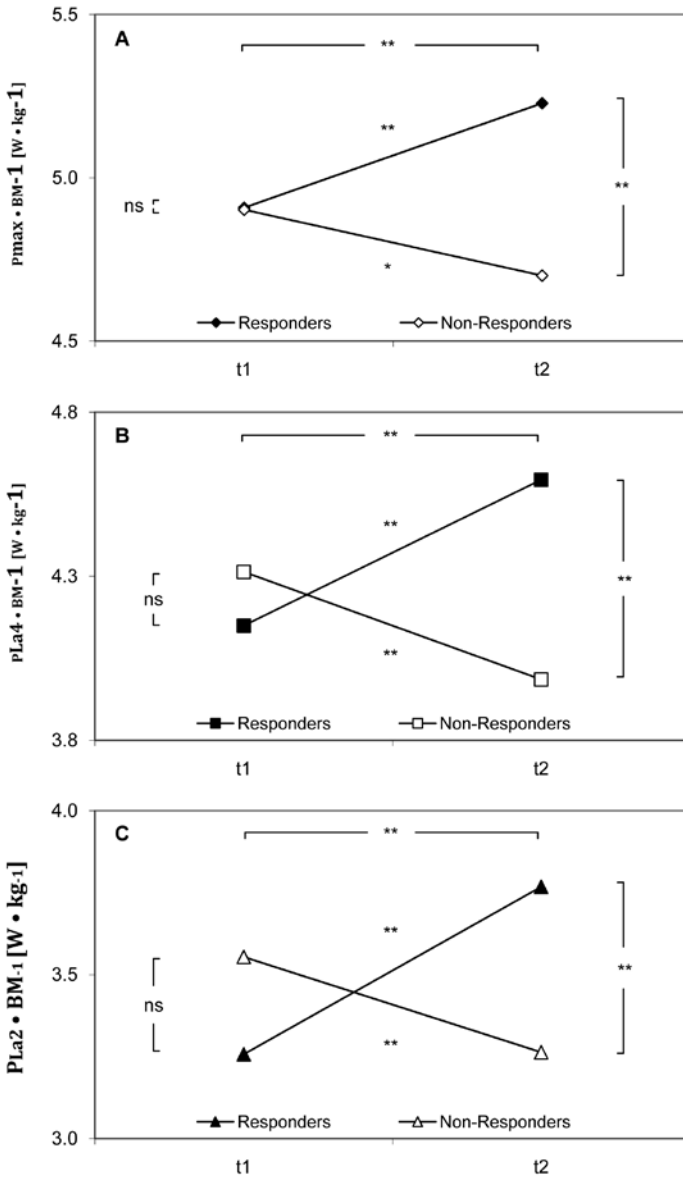$$R = 0.86; \; R^2_{adj} = 0.68 \tag{E4}$$

$$P_{La4} \cdot BM^{-1}_{t2} = 1.56 + 0.46 \cdot P_{La4} \cdot BM^{-1}_{t1} + 0.22 \cdot s_{CR} + 0.29 \cdot s_{BER} - 0.33 \cdot s_{DR} - 0.28 \cdot s_{Comp} + 0.21 \cdot s_{PR}$$

$$R = 0.88; \; R^2_{adj} = 0.72 \tag{E5}$$

$$P_{La2} \cdot BM^{-1}_{t2} = 1.91 + 0.68 \cdot P_{La2} \cdot BM^{-1}_{t1} + 0.27 \cdot s_{SoB} - 0.52 \cdot s_{DR} - 0.33 \cdot s_{Comp} + 0.40 \cdot s_{PR}$$

$$R = 0.90; \; R^2_{adj} = 0.75 \tag{E6}$$

Figure 2 — $P_{max} \cdot BM^{-1}$ (A), $P_{La4} \cdot BM^{-1}$ (B), and $P_{La2} \cdot BM^{-1}$ (C) at t_1 and t_2 in the groups of Responders and Non-Responders. Mean values (standard deviations omitted for clarity). * $P < .05$; ** $P < .01$; ns = not significant ($P > .05$). A: Group difference at t_2: T = –5.65; $P < .01$. Δ_{t2-t1}: Responders +7 ± 6%; T = –4.77; $P < .01$; Non-Responders –4 ± 7%; T = 2.34; $P < .05$. Interaction Group × Time: $F = 22.77$; $P < .01$. B: Group difference at t_2: T = –5.61; $P < .01$. Δ_{t2-t1}: Responders +11 ± 4%; T = 12.86; $P < .01$; Non-Responders –7 ± 6%; T = 4.87; $P < .01$. Interaction Group × Time: $F = 104.14$; $P < .01$. C: Group difference at t_2: T = –3.71; $P < .01$. Δ_{t2-t1}: Responders +19 ± 16%; T = 5.73; $P < .01$; Non-Responders –9 ± 6%; T = 3.75; $P < .01$. Interaction Group × Time: $F = 47.61$; $P < .01$.

where

s_{CR} = Cycling distance at Compensation range intensity (<2 mM blood lactate)

s_{BER} = Cycling distance at Basic Endurance range intensity (≤ 2 mM)

s_{SoB} = Cycling distance performing On Bike Strength training (2–3 mM)

s_{DR} = Cycling distance at Development range intensity (3–6 mM)

s_{Comp} = Cycling distance in Competition (3–6 mM)

s_{PR} = Cycling distance at Peak range (>6 mM)

While the two groups were clearly distinguishable by their change in power at 2 and 4 mM blood lactate during the BPP, increase or decrease in ergometer performance during the early training season were comparably represented among athletes who attained international and national peak success 5 to 6 months later, based on their performance in the national or World championships (Table 3).

Table 3   End-of-season medal success in early season Responders and Non-Responders

	BPP Responders	BPP Non-Responders
International Medal	6	6
International Final (place 4–10)	4	4
National Medal	5	3
National Final (place 4–10)	2	4

Discussion

This study documents several findings that are potentially important in under-standing the endurance training process. First, in 51 well-trained, junior track cyclists followed during a 15 wk basic preparation period, the mean improvement in cycling power output at 4 mM blood lactate (P_{4mM}) was only 3%. However, the variability in training response was large, with the bottom one-third of athletes actually performing poorer 15 wk later (mean change in P_{La4} −7%, P_{max} −4%, and in P_{La2} −8%), compared with the upper one-third, who increased P_{La4} by a mean of 11% (7% improvement in P_{max} and 19% in P_{La2}).

Significant differences between Responders and Non-Responders in the amount of training performed during the 15 wk period were not observed, although total training volume did tend to be higher in Responders. However, significant differences in intensity distribution were seen. Responders to the 15 wk BPP performed more cycling volume at low intensity and less volume at intensities eliciting 3 to 6 mM blood lactate, and an overall higher ratio of cycling volume at low versus high intensity.

Previous descriptive studies have demonstrated that successful endurance athletes in different sports emphasize performing large volumes of continuous

exercise at intensities clearly below the lactate threshold.[1,6–9,12,15] The actual intensity distribution depends somewhat on the method of quantification.[11] When heart rate "time-in-zone" analysis is used, over 90% of training time may be accumulated at an intensity eliciting <2 mM blood lactate.[11,12] When a nominal distribution of training sessions is used based on the primary intensity of the session, about 80% to 85% of training sessions among elite endurance athletes can be characterized as "long slow distance" workouts. The present findings are based on total cycling volume, not allocation of training sessions, but they fall in line with this distribution.

The more surprising, and perhaps counter-intuitive finding is that cyclists performing more volume at intensities eliciting a blood lactate concentration of 3 to 6 mM actually had a poor response to training, when measured as change in the maximal cycling power and the power to blood lactate relationship. Cyclists showing a clear right shift in the blood lactate-cycling power relationship tended to perform more volume at lower intensity. Unfortunately, maximal oxygen consumption changes were not documented in these athletes, so we cannot report whether individual changes in maximal oxygen consumption tracked with changes in threshold powers and P_{max}.

While the present observations may seem at odds with the principle of training specificity and the current popularity of high intensity training for fitness in untrained and recreational exercisers, they are not unique. Esteve-Lanao et al randomized 12 subelite distance runners into two groups.[13] One group trained 81% of their training below the first ventilatory turn point, 12% between VT1 and VT2 and 8% above VT2. The second group performed twice as much "threshold training" (67, 25, and 8% for the same three intensity zones). Total training load was matched between the two groups using TRIMPS. Improvement in a cross-country time trial performed before and after the 5 mo period revealed that the group that had trained more low intensity training and less threshold zone training showed significantly greater race time improvement (–157 ± 13 s vs –122 ± 7 s, P = .03). Ingham et al randomized 18 national class rowers into one of two training groups following a 25 d training free period.[16] One group performed 100% of their training below 75% of VO_2peak (LOW). The other group performed 70% of their training at this intensity, plus 30% of their training halfway between their LT power and their power at VO_2peak. The authors found that rowing ergometer performance gains and VO_2peak increase were statistically equivalent in the two groups. However, rowing power at both 2 and 4 mM blood lactate increased significantly more in the LOW only group (14 vs 5%). The present findings combined with these studies and descriptive reports of the training of elite endurance athletes suggest that comparatively large volumes of "low intensity" training play an important role in optimizing physiological adaptations to endurance training in high-performance sport.

Finally, we observed that variability in response to training during this early phase of preparation for the season failed to predict success at the national or international level 5 to 6 mo later in this sample. In this highly selective group of elite junior athletes, both Responders and Non-Responders to the early training period were equally successful at the end of the season. Given the large time gap between the training period documented here and their final competition, and the relative success-homogeneity of this sample, this is not too surprising. In addition, the data were collected over multiple years, adding the element of variation in the strength of competition from year to year. Non-Responders may have adjusted their

training. Other factors beyond physiological capacity contribute to ultimate success in the peak competition, and the intensity distribution differences seen may have resulted in other positive adaptations in Non-Responders that were not measured.

It is also important to consider the impact of typical measurement error on our interpretation of the data. Day-to-day biological variation, instrumental errors associated with the precision of the SRM power output measurements, and Biosen blood lactate concentration measurements have all contributed to measurement error in the individual threshold power determinations. The typical error for lactate threshold power measurements appears to be in the 3% to 6% range (reference 17 and W.G. Hopkins personal communication), suggesting that small changes in threshold power typical of highly trained athletes could be masked by measurement error. In the current study, we have identified Responders based on the 66th percentile for change in power at 4 mM blood lactate relative to body mass. Non-Responders were identified as those below the 33rd percentile. Individually, the "Positive Responders" as defined here had a measured increase in 4 mM power between 7 and 20%. Based on single subject calculations of the likelihood of a clinically positive, trivial, or negative true change proposed by Hopkins,[18] and assuming a typical error for lactate threshold power determination of ±5%, the likelihood that any one of these athletes identified from testing as positive Responders was in truth a Non-Responder was between 11 and 25%. Similarly, the "Non-Responders" group ranged from –18% to 0% change in threshold power: The likelihood that any one of these athletes was actually a positive Responder with 7% or higher increase in 4 mM power relative to body mass ranged between 10 and 24%. Therefore, we must assume that a small number of the 34 athletes incorporated into the group analysis were incorrectly assigned due to random measurement error. However, we do not think this would change the overall interpretation of the present findings.

In conclusion, we have documented substantial variation in lactate profile response to a presumably standardized period of endurance training in competitive cyclists. We also report that a substantial portion of this variation can be explained by variation in intensity distribution. In already highly trained junior cyclists, the blood lactate-cycling power relationship tends to respond positively to a high volume of training below 2 mM blood lactate. Excessive volumes of training at intensities eliciting 3 to 6 mM blood lactate may have a negative impact on the power/lactate relationship. However, our understanding of the interactions among training intensity distribution, training volume, and physiological adaptation remains observational, not mechanistic.

Acknowledgments

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