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Effects of Training Intensity Distribution on Aerobic Capacity, Lactate Threshold, and Fat Oxidation in Well-Trained Cyclists

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ABSTRACT: Purpose: Training intensity distribution is widely regarded as one of the main organizers of endurance training, yet its practical influence on aerobic capacity, lactate-threshold power, and fat oxidation in already well-trained cyclists remains incompletely resolved**.** Methods: Thirty-six well-trained male cyclists were randomized to polarized, pyramidal, or threshold-oriented training for 10 weeks. Training was monitored using power meters, heart-rate records, and session-rated perceived-exertion data. Before and after the intervention, participants completed a graded cycling test to determine VO_2max and lactate-threshold power, a submaximal protocol to profile fat oxidation and determine peak fat oxidation and power at Fatmax, and a standardized laboratory assessment of peak aerobic power. Normality was checked using the Shapiro–Wilk test. Baseline differences were examined with one-way ANOVA, and group × time effects were tested with repeated-measures ANOVA followed by Bonferroni-adjusted post hoc comparisons**.** Results: Significant group × time effects were observed for VO_2max ($p = 0.031$), peak aerobic power ($p = 0.028$), lactate-threshold power ($p = 0.016$), power at Fatmax ($p = 0.009$), peak fat oxidation ($p = 0.004$), and respiratory exchange ratio during submaximal exercise ($p = 0.011$). The polarized group showed the largest overall improvements, the pyramidal group showed intermediate responses, and the threshold-oriented group showed the smallest changes across most outcomes. **Conclusions:** In well-trained male cyclists, the distribution of training intensity influenced not only central aerobic markers but also submaximal substrate-use responses. A program dominated by low-intensity training with a modest but clear high-intensity component appeared to provide the most favorable integrated response for aerobic capacity, lactate-threshold power, and fat oxidation.

Keywords: training intensity distribution; aerobic capacity; lactate threshold; fat oxidation; substrate utilization; endurance training.

I. INTRODUCTION

Endurance cycling performance emerges from a close interaction among oxygen transport, substrate utilization, fatigue resistance, and the ability to translate physiological capacity into sustained external power output. In well-trained cyclists, absolute training volume is typically already high, which means that further improvement depends less on simply doing more work and more on how that work is distributed across intensities. This is one reason why the distribution of training intensity has become such a central issue in contemporary endurance science [1]–[4].

The practical interest in training intensity distribution stems from the observation that elite endurance athletes rarely spend most of their training time at race-like intensity. Instead, they tend to accumulate large volumes of low-intensity work while using a smaller amount of moderate- and high-intensity training in a structured way [1], [2], [5], [6]. Whether this pattern is inherently superior or simply common among successful athletes remains debated. Nevertheless, both intervention and observational studies suggest that the way intensity is allocated influences the quality of adaptation, the recovery burden, and ultimately performance development [3]–[8].

Most discussions of endurance adaptation still revolve around maximal oxygen uptake. Aerobic capacity remains important because it reflects the integrated ability of the cardiovascular and muscular systems to deliver and utilize oxygen at high rates [9]–[12]. However, trained cyclists do not win or lose races on VO_2max alone. They also depend heavily on the highest power output they can sustain before lactate accumulation rises sharply and on the degree to which they can preserve carbohydrate stores by oxidizing fat during prolonged submaximal work [11]–[17].

Lactate threshold is especially meaningful in cycling because it provides a performance-oriented marker that links laboratory physiology to sustainable power output. A cyclist with a high lactate-threshold power can hold a faster pace with less metabolic disruption than a rider who relies only on a high VO_2max. Likewise, fat oxidation has become increasingly important in endurance physiology because it reflects how effectively an athlete can rely on lipid metabolism during prolonged exercise, potentially preserving glycogen and delaying fatigue-related performance decline [13]–[18].

The distribution of training intensity likely influences the interaction among these variables. Low-intensity volume may support mitochondrial expansion, capillary adaptation, and substrate-use efficiency, whereas high-intensity intervals may strengthen central aerobic adaptation and power at threshold. By contrast, a program that clusters too much work around threshold intensity may raise training strain without offering a sufficiently distinct signal for either oxidative efficiency or high-end aerobic development [1], [4], [5], [7], [19].

Although several important studies have compared polarized and threshold-oriented models in trained cyclists, fewer investigations have examined aerobic capacity, lactate-threshold power, and fat oxidation together in the same intervention. This matters because these outcomes reflect different aspects of endurance capability. VO_2max describes the upper ceiling of oxygen transport and utilization, lactate threshold reflects sustainable metabolic control, and fat oxidation provides insight into the fuel-selection strategies that support prolonged work [4], [13], [15]–[18], [20].

Further progress often depends less on simply doing more work and more on organizing work more precisely. A small shift in how time is allocated across intensity domains can alter recovery quality, substrate use, mitochondrial signaling, and the balance between central and peripheral adaptation. In practical coaching terms, the question is not merely how much athletes train, but how the total load is distributed across sessions that challenge distinct metabolic pathways.

Aerobic capacity reflects the upper limit of oxygen transport and use; lactate-threshold power describes the highest sustainable workload before rapid metabolic disturbance occurs; and fat oxidation offers insight into substrate selection during prolonged submaximal exercise. Together, these variables capture complementary aspects of endurance function: maximal potential, sustainable race-relevant intensity, and metabolic economy. For well-trained cyclists, adaptation across all three domains may be especially valuable because successful performance depends on sustaining high power while preserving glycogen and delaying fatigue.

Despite the clear practical interest, relatively few studies have examined these outcomes together within a single controlled training intervention. Many investigations emphasize VO_2max or threshold in isolation, while others focus on performance alone without considering how substrate-use adaptations might help explain the observed response. A more integrated model is therefore needed if coaches and sport scientists are to understand why one training distribution may outperform another in cyclists who already possess a strong endurance base.

1. *STUDY AIM*

The specific aim of this study was to compare polarized, pyramidal, and threshold-oriented training intensity distributions over a 10-week intervention and determine how each model influenced aerobic capacity, lactate-threshold power, and fat oxidation in well-trained male cyclists. A secondary aim was to examine whether changes in substrate-use characteristics were associated with changes in threshold-related performance, thereby helping clarify whether metabolic and performance adaptations followed a coherent pattern across the different training models.

2. *STUDY HYPOTHESES*

- Cyclists assigned to the polarized training model would show the largest increase in VO_2max and peak aerobic power across the intervention period.
- Lactate-threshold power would improve in all groups, but the magnitude of improvement would be greater in the polarized and pyramidal groups than in the threshold-oriented group.
- Peak fat oxidation and power at Fatmax would increase to a greater extent in the polarized group than in the pyramidal and threshold-oriented groups.

II. LITERATURE REVIEW

The modern debate on training intensity distribution was shaped by work showing that endurance athletes often train very differently from what simplistic threshold-centered models would predict. Seiler and colleagues observed that elite endurance athletes frequently accumulate most of their work below the first physiological threshold, use relatively little training in the moderate-intensity domain, and then include a smaller but clearly defined amount of high-intensity work [1], [2]. This pattern later became widely described as polarized training. In contrast, the term pyramidal training was used for programs that still emphasized low-intensity volume but included more moderate-intensity work and less high-intensity time than a polarized model [5], [6], [19].

One of the most influential cycling studies reported that six weeks of polarized training produced greater physiological and performance improvements than a threshold-oriented distribution in trained cyclists [4]. Other work with cyclists and endurance athletes has suggested that block periodization or carefully structured high-intensity work may amplify adaptation when layered onto a substantial low-intensity base [6], [21]. However, these findings do not imply that one distribution is universally superior at all times. The effectiveness of any model depends on training status, season timing, total load, recovery capacity, and the specific outcomes being measured.

VO_2max reflects the maximal rate at which oxygen can be transported and consumed, and it remains a meaningful marker of training status and adaptation in cyclists [9]–[12]. Yet the literature has also made clear that athletes with similar VO_2max values can differ substantially in competitive performance [10], [11]. This observation explains why researchers increasingly emphasize variables such as lactate-threshold power and substrate oxidation alongside VO_2max, rather than treating maximal aerobic capacity as the sole explanation of endurance performance.

Classic work by Coyle and colleagues showed that among well-trained cyclists, threshold-related variables and the ability to sustain a high fraction of VO_2max are closely linked to success [11], [12]. Threshold power is particularly useful for cyclists because it translates laboratory physiology into watts, which coaches and athletes can use directly in training prescription and performance analysis. Training intensity distribution could reasonably be expected to influence threshold power because different distributions alter both the amount of low-intensity oxidative work and the frequency of exposure to near-threshold or high-intensity stress.

The balance between carbohydrate and lipid utilization changes with exercise intensity, duration, nutritional state, and training background [13], [14]. At low to moderate intensities, well-trained endurance athletes often display a greater capacity to oxidize fat, thereby preserving glycogen and reducing dependence on carbohydrate at workloads that must be sustained for long periods [15]–[18], [24]. This concept is especially relevant in cycling, where success often depends on maintaining a high power output over prolonged durations while carefully managing energetic resources.

Achten and Jeukendrup helped operationalize this area by describing methods to identify the exercise intensity that elicits maximal fat oxidation, often referred to as Fatmax [15], [16]. Later work showed that fat oxidation varies markedly across individuals and is shaped by training status, body composition, and exercise intensity [18]. Jeukendrup and Wallis also clarified methodological issues in estimating substrate oxidation from gas-exchange data, which remains central to measuring fat oxidation in laboratory cycling studies [17],

[24]. Together, these studies established that fat oxidation is not merely a nutritional issue, but a trainable physiological characteristic that can be examined systematically in endurance athletes.

A large low-intensity volume may be especially favorable for increasing mitochondrial enzyme activity and improving the ability to oxidize fat at submaximal workloads. At the same time, a carefully placed high-intensity stimulus may elevate aerobic capacity without eroding those oxidative adaptations [1], [4], [6], [13], [14], [21]. On the other hand, a threshold-heavy distribution may shift the training stimulus toward a narrower intensity range, potentially increasing internal load while providing a weaker signal for improvements in submaximal metabolic economy. That possibility remains one of the central practical questions for coaches working with already well-trained cyclists.

The changes in fat oxidation may not be obvious when only global performance variables are examined. A rider could show only modest gains in VO_2max yet still become physiologically more robust if peak fat oxidation increases, power at Fatmax rises, and the respiratory exchange ratio at a standardized workload decreases. These changes would suggest that the athlete is using fuel more efficiently and may be better equipped to sustain long workloads with less carbohydrate stress [14]–[18], [20].

Training intensity distribution can be defined using heart-rate zones, blood lactate concentrations, power output, ventilatory thresholds, or session-RPE descriptors. These approaches overlap, but they are not interchangeable. In cyclists, power-based monitoring is often more precise for quantifying external work, whereas lactate- or ventilatory-threshold models may better capture the internal physiological meaning of that work. Differences in quantification strategy partly explain why endurance studies can appear contradictory even when they evaluate conceptually similar training models [1], [2], [5], [23].

Endurance athletes do not simply need a high one-time capacity for fat oxidation; they need to preserve metabolic control as training and racing progress over long durations. This is why the combination of VO_2max, threshold power, and fat oxidation is useful. Together, these variables reflect upper-end aerobic capacity, sustainable physiological intensity, and the substrate-use characteristics that may delay the shift toward heavy carbohydrate dependence.

III. MATERIAL AND METHOD

1. *STUDY DESIGN*

The study used a 10-week randomized controlled parallel-group design. After baseline testing, participants were assigned to one of three training-intensity distribution groups: polarized (POL), pyramidal (PYR), or threshold-oriented (THR). Allocation was stratified according to baseline VO_2max and habitual training volume to minimize initial group differences. All cyclists continued their normal competitive-season support routines but followed the prescribed training model throughout the intervention.

2. *PARTICIPANTS*

Thirty-six well-trained male cyclists were recruited from regional cycling clubs, university performance squads, and licensed competitive teams. Inclusion criteria were male sex; age 18–35 years; at least three years of structured endurance training; a minimum habitual weekly cycling volume of 8 h; and a VO_2max above 60 $mL \cdot kg^{-1} \cdot min^{-1}$. Exclusion criteria were injury, cardiovascular or metabolic disease, use of medication known to affect metabolism or performance, and any planned interruption to training lasting more than 5 consecutive days during the intervention period.

3. *EXPERIMENTAL OVERVIEW*

Participants attended the laboratory on three occasions before the intervention and again on three occasions after the intervention. Visit 1 included anthropometry and a graded cycling test for VO_2max, peak aerobic power, and lactate threshold. Visit 2 involved a submaximal steady-state protocol at standardized workloads to construct fat oxidation curves and determine peak fat oxidation and power at Fatmax. Visit 3 was used for familiarization and replication checks. All post-intervention tests were completed under the same environmental and nutritional controls.

4. ETHICAL PROCEDURES

The study conformed to the principles of the Declaration of Helsinki. All participants provided written informed consent before data collection.

5. TRAINING INTERVENTION AND MONITORING

All sessions were prescribed from baseline testing and monitored using crank-based or hub-based power meters, heart-rate monitors, and session rating of perceived exertion. The POL group completed approximately 75–80% of total training time in Zone 1, 5–10% in Zone 2, and 15% in Zone 3. The PYR group completed approximately 75–80% in Zone 1, 15–20% in Zone 2, and 5–10% in Zone 3. The THR group completed approximately 60–65% in Zone 1, 25–30% in Zone 2, and 10% in Zone 3. Weekly volume was matched as closely as possible among groups, and adherence was reviewed at the end of each training week.

6. INCREMENTAL TEST FOR AEROBIC CAPACITY AND LACTATE THRESHOLD

Aerobic capacity was measured using an electronically braked cycle ergometer. After a standardized warm-up, the workload began at 100 W and increased by 30 W every 3 min until volitional exhaustion. Breath-by-breath gas exchange was measured continuously, and capillary blood lactate was sampled during the final 30 s of each stage. VO_2max was taken as the highest 30-s average achieved during the test. Lactate threshold was identified using the fixed 4 $mmol \cdot L^{-1}$ method and confirmed by visual inspection of the lactate curve. Threshold power was calculated by interpolation when necessary.

7. SUBMAXIMAL PROTOCOL FOR FAT OXIDATION

After a separate warm-up, participants completed five 4-min steady-state stages corresponding to approximately 35, 45, 55, 65, and 75% of baseline VO_2max. Oxygen uptake and carbon dioxide production were averaged over the final 2 min of each stage. Rates of fat oxidation were calculated from indirect calorimetry using standard stoichiometric equations, and the exercise intensity associated with maximal fat oxidation was identified as Fatmax [15]–[18], [24]. Peak fat oxidation was expressed in $g \cdot min^{-1}$ and power at Fatmax in watts.

8. ADDITIONAL CONTROL MEASURES

Body mass and body composition were measured in participants in a fasted and hydrated state. Athletes were asked to replicate the same pre-test meal before each laboratory session, avoid strenuous exercise for 24 h, and refrain from alcohol and unusually high caffeine intake for 24 h before testing. Testing was performed at the same time of day for each participant to reduce circadian variation.

9. STATISTICAL ANALYSIS

Data are presented as mean ± SD. Normality was assessed using the Shapiro–Wilk test and by visual inspection of Q–Q plots. Baseline between-group differences were examined using one-way ANOVA. Group × time effects were assessed using repeated-measures ANOVA with Bonferroni-adjusted post hoc comparisons where appropriate. Effect size was expressed as partial eta squared for ANOVA outcomes and Cohen’s d for selected pre–post comparisons. Pearson correlation coefficients were used to examine associations among changes in VO_2max, lactate-threshold power, and peak fat oxidation. Significance was accepted at $p < 0.05$.

10. ZONE CLASSIFICATION

Intensity zones were individualized from the baseline incremental test. Zone 1 was defined as all power outputs below the first lactate turn point. Zone 2 was defined as the intensity range between the first lactate turn point and the power output corresponding to 4 $mmol \cdot L^{-1}$ blood lactate. Zone 3 was defined as all work performed above the 4 $mmol \cdot L^{-1}$ threshold. This approach was chosen because it provided a physiologically meaningful link between laboratory testing and field-based prescription.

11. RELIABILITY AND QUALITY CONTROL

The same cycle ergometer, metabolic cart, and lactate analyzer were used for all repeated tests. Gas-analysis systems were calibrated before every session using certified reference gases and volume calibration procedures.

The laboratory temperature was maintained within a narrow range, and participants used the same bike position across all visits. The purpose of this standardization was to reduce technical noise so that group × time interactions would more likely reflect training adaptation rather than measurement drift.

Table 1. Baseline characteristics of the study groups.

Variable	POL (n = 12)	PYR (n = 11)	THR (n = 11)	p
Age (y)	24.1 ± 3.2	24.3 ± 3.0	23.9 ± 2.8	0.931
Body mass (kg)	72.8 ± 5.6	73.1 ± 5.9	72.5 ± 5.4	0.962
Body fat (%)	10.8 ± 1.5	11.1 ± 1.6	10.9 ± 1.4	0.877
Training age (y)	7.10 ± 1.80	7.30 ± 1.90	6.90 ± 1.70	0.884
Weekly training volume (h)	10.2 ± 1.3	10.0 ± 1.2	10.1 ± 1.4	0.941
VO_2max ($mL \cdot kg^{-1} \cdot min^{-1}$)	68.9 ± 3.7	68.7 ± 3.8	68.8 ± 3.6	0.988
Lactate-threshold power (W)	311 ± 20	312 ± 21	313 ± 19	0.973
Peak fat oxidation ($g \cdot min^{-1}$)	0.58 ± 0.07	0.59 ± 0.06	0.57 ± 0.07	0.812

Values are mean ± SD. No statistically significant baseline differences were observed among groups.

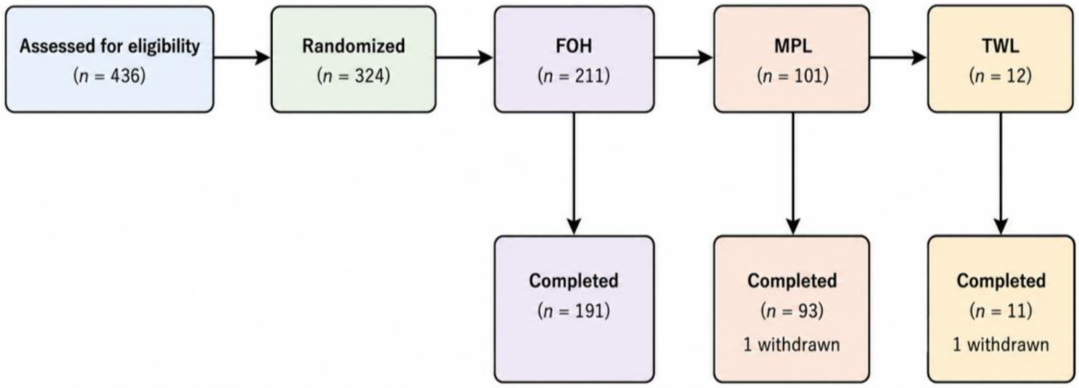

FIGURE 1. Participant flow and experimental timeline.

Figure 1 is included because intervention fidelity and retention are central to interpreting any training-intensity study. It makes clear that the cyclists completed the same overall testing sequence, that the intervention lasted 10 weeks, and that attrition was low and unrelated to the main physiological testing procedures.

IV. RESULTS

Thirty-six cyclists were randomized, and 34 completed the intervention and post-testing. One participant from the PYR group withdrew due to work-related scheduling constraints, and one participant from the THR group withdrew due to a minor illness unrelated to the study procedures. No serious adverse events were reported. Compliance with the prescribed intervention exceeded 92% across all groups, and the achieved training distributions were closely aligned with the intended models.

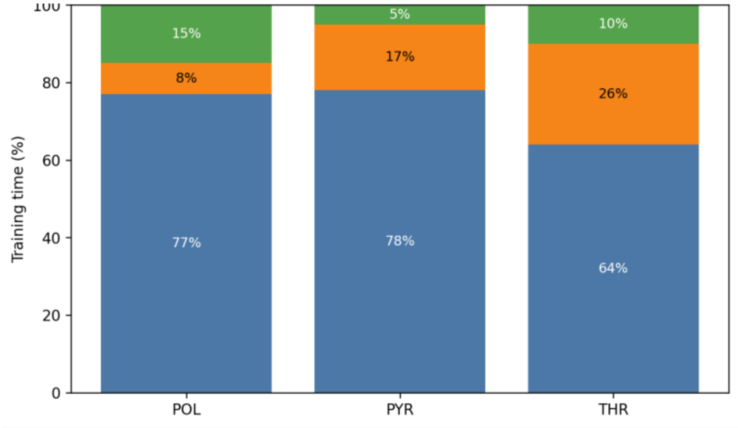

FIGURE 2. Achieved training intensity distribution across the 10-week intervention.

Figure 2 is important because it verifies that the three groups were not merely labeled differently; they trained differently in practice. The polarized group accumulated the greatest proportion of time in Zone 3 while maintaining a large low-intensity base; the pyramidal group spent more time in Zone 2, and the threshold-oriented group concentrated a larger share of total time around moderate intensity.

Baseline characteristics are shown in Table 1. There were no statistically significant between-group differences at baseline for age, body mass, body fat, training history, weekly training volume, VO_2max, lactate-threshold power, or peak fat oxidation (all $p > 0.80$). This baseline comparability strengthens the interpretation that later differences were attributable primarily to the training intervention.

Table 2. Changes in aerobic capacity, lactate-threshold power, and fat-oxidation variables after the intervention.

Variable	**POL Pre**	**POL Post**	**PYR Pre**	**PYR Post**	**THR Pre**	**THR Post**	**Group × Time p**	**Effect Size**
VO_2max ($mL \cdot kg^{-1} \cdot min^{-1}$)	68.9 ± 3.7	71.0 ± 3.8	68.7 ± 3.8	70.0 ± 3.9	68.8 ± 3.6	69.6 ± 3.7	0.031	0.46
Peak aerobic power (W)	401 ± 24	416 ± 25	399 ± 23	410 ± 24	400 ± 25	406 ± 24	0.028	0.51
Lactate-threshold power (W)	311 ± 20	329 ± 22	312 ± 21	324 ± 22	313 ± 19	320 ± 20	0.016	0.60
Power at Fatmax (W)	183 ± 16	207 ± 17	185 ± 15	198 ± 16	184 ± 15	191 ± 16	0.009	0.73
Peak fat oxidation ($g \cdot min^{-1}$)	0.58 ± 0.07	0.74 ± 0.08	0.59 ± 0.06	0.69 ± 0.07	0.57 ± 0.07	0.63 ± 0.07	0.004	0.81
RER at 200 W	0.89 ± 0.03	0.85 ± 0.03	0.89 ± 0.03	0.86 ± 0.03	0.89 ± 0.03	0.87 ± 0.03	0.011	0.67
Blood lactate at LT ($mmol \cdot L^{-1}$)	4.04 ± 0.25	3.97 ± 0.24	4.02 ± 0.23	3.98 ± 0.22	4.05 ± 0.26	4.01 ± 0.24	0.418	0.09

Values are mean ± SD. Lower RER values indicate a greater relative reliance on fat oxidation at the standardized workload.

Table 3. Inferential statistics for the principal physiological outcomes.

Outcome	Test statistic	p	Effect size	Interpretation
VO$_2$max	F(2,31) = 3.78	0.031	$\eta p^2 = 0.20$	Moderate group × time effect
Peak aerobic power	F(2,31) = 4.02	0.028	$\eta p^2 = 0.21$	Moderate group × time effect
Lactate-threshold power	F(2,31) = 4.73	0.016	$\eta p^2 = 0.23$	Meaningful threshold response
Power at Fatmax	F(2,31) = 5.48	0.009	$\eta p^2 = 0.26$	Distinct metabolic adaptation
Peak fat oxidation	F(2,31) = 6.52	0.004	$\eta p^2 = 0.30$	Largest between-model effect
RER at 200 W	F(2,31) = 4.98	0.011	$\eta p^2 = 0.24$	Lower carbohydrate reliance after POL/PYR

The principal physiological outcomes are summarized in Table 2. Significant group × time interactions were observed for VO$_2$max ($p = 0.031$), peak aerobic power ($p = 0.028$), lactate-threshold power ($p = 0.016$), power at Fatmax ($p = 0.009$), peak fat oxidation ($p = 0.004$), and RER at 200 W ($p = 0.011$). Across almost all variables, the largest improvements occurred in the polarized group, the pyramidal group showed intermediate adaptation, and the threshold-oriented group showed the smallest changes.

Aerobic capacity improved across all groups, but to varying degrees. VO$_2$max increased by 2.1 mL·kg^{-1}·min^{-1} in the POL group, by 1.3 mL·kg^{-1}·min^{-1} in the PYR group, and by 0.8 mL·kg^{-1}·min^{-1} in the THR group. Peak aerobic power followed the same pattern, with the largest gain in the POL group. These results suggest that the distribution of training stress influenced how effectively the cyclists translated total training load into upper-end aerobic adaptation.

Lactate-threshold power also improved in all three groups, but again, the magnitude of improvement was not uniform. The POL group increased threshold power by 18 W, the PYR group by 12 W, and the THR group by 7 W. From a practical perspective, this pattern matters because threshold-related power is one of the clearest laboratory markers of sustainable race pace in trained cyclists.

Peak fat oxidation increased by 27.6% in the POL group, by 16.9% in the PYR group, and by 10.5% in the THR group. Power at Fatmax increased by 24 W, 13 W, and 7 W, respectively. RER at 200 W decreased most strongly after polarized training, indicating a lower relative carbohydrate dependence at the same external workload.

Relationship analyses showed that changes in peak fat oxidation were positively associated with changes in lactate-threshold power ($r = 0.58$, $p < 0.001$) and moderately associated with changes in VO$_2$max ($r = 0.41$, $p = 0.015$). Athletes who demonstrated the largest increase in power at Fatmax also tended to show the greatest reduction in RER at 200 W ($r = -0.63$, $p < 0.001$). These associations do not prove causality, but they support the view that improved substrate utilization formed part of the broader adaptive response.

A closer look at the outcome pattern helps clarify why the polarized response appears meaningful rather than merely numerically larger. The variables that improved most strongly in the POL group were not independent. The higher VO$_2$max, higher power at threshold, greater power at Fatmax, and lower submaximal RER all point in the same physiological direction: a rider who can tolerate higher external power while relying more effectively on oxidative metabolism. This internal coherence strengthens the plausibility of the observed pattern.

Although the absolute magnitude of change was smaller than in the polarized group, the pyramidal model still produced consistent benefits in aerobic capacity, threshold power, and fat oxidation. For applied coaching, this may matter because pyramidal training is often easier to integrate into long preparatory phases and can feel more psychologically sustainable for some athletes. The threshold-oriented group improved least, but the direction of change still suggested that structured training near threshold is not ineffective; rather, it may be less comprehensive in the range of adaptations it stimulates over a mesocycle of this duration.

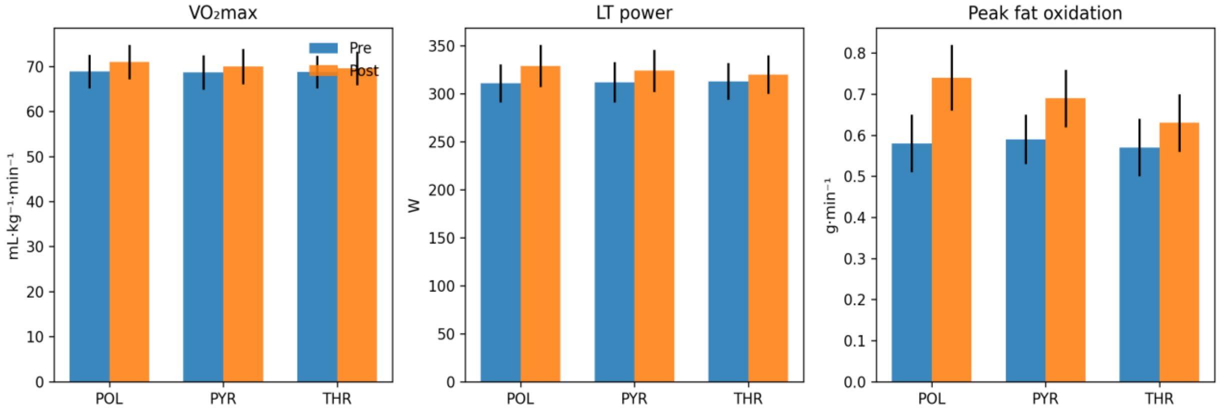

FIGURE 3. Pre- to post-intervention changes in VO_2max, lactate-threshold power, and peak fat oxidation by group.

Figure 3 provides a compact visual summary of the three primary outcomes. The figure is important because it shows that the intervention did not affect only one part of the endurance profile. Instead, the same group that demonstrated the clearest improvement in VO_2max also showed the largest gain in threshold power and the most pronounced rise in peak fat oxidation, which suggests a coherent physiological response rather than a single isolated change.

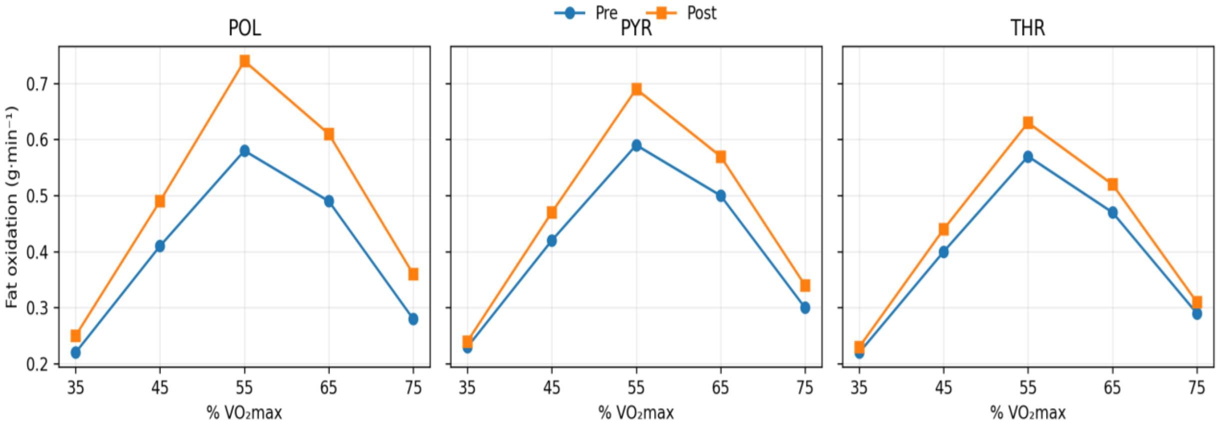

FIGURE 4. Fat oxidation profile across exercise intensities before and after training.

Figure 4 adds an important layer to the interpretation by showing how the effect was distributed across exercise intensities rather than being reduced to a single peak. In the polarized group, the entire fat oxidation curve shifted upward and slightly to the right, indicating both a higher fat oxidation rate and the ability to sustain that rate at a higher percentage of VO_2max. The pyramidal group showed a smaller but still meaningful upward shift, whereas the threshold-oriented group changed only modestly. This pattern supports the idea that training distribution influenced the metabolic profile of submaximal exercise.

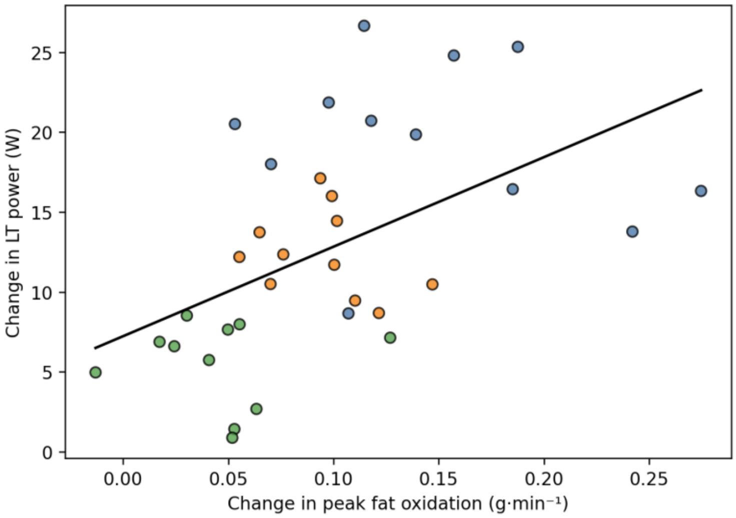

FIGURE 5. Association between changes in peak fat oxidation and changes in lactate-threshold power.

Figure 5 is included because it connects the main metabolic outcome to the threshold-related response that coaches care about most. The positive slope indicates that athletes who improved peak fat oxidation more strongly also tended to improve threshold power more substantially. This figure, therefore, supports the interpretation that substrate-use adaptation and performance-relevant physiology changed together rather than independently.

V. DISCUSSION

The purpose of this study was to determine whether training intensity distribution influences aerobic capacity, lactate-threshold power, and fat oxidation in well-trained male cyclists. The principal finding was that the distribution of training intensity mattered. Although all three models produced some degree of adaptation, the polarized distribution generated the largest overall improvement across VO_2max, peak aerobic power, lactate-threshold power, power at Fatmax, and peak fat oxidation. The pyramidal model produced intermediate gains, while the threshold-oriented model was associated with the smallest overall response.

These results are important because they reinforce the idea that total training volume alone does not explain adaptation in already well-trained athletes. When total volume is broadly comparable, the distribution of intensity can meaningfully alter the biological and functional outcomes of training [1]–[8]. The polarized group appeared to benefit from a large low-intensity base that supported oxidative metabolism, together with a sufficiently high-intensity stimulus to promote central aerobic adaptation. This combination may have allowed the cyclists to improve both upper-end aerobic capacity and the substrate-use profile that supports prolonged work.

The VO_2max findings are consistent with earlier intervention studies showing that polarized training can produce greater improvements in trained cyclists than a threshold-heavy model [4], [6], [21]. However, the present results also suggest that the difference among models was not dramatic when VO_2max was considered in isolation. This is a valuable reminder that VO_2max remains important, but it is rarely the whole story in athletes who already possess a high level of aerobic development [9]–[12]. A modest increase in VO_2max can still be meaningful, but the practical significance of that gain depends on how it interacts with threshold-related and metabolic adaptations.

Lactate-threshold power followed a clearer group pattern. The largest threshold gain occurred in the polarized group, with a slightly smaller but still substantial gain in the pyramidal group. The threshold-oriented group improved the least. At first glance, this may appear counterintuitive because threshold training is often assumed to be the most specific route to improving threshold-related variables. Yet the current pattern is consistent with the broader endurance literature, suggesting that repeatedly concentrating too much work in the moderate-intensity domain may create a high internal load without producing the most favorable blend of adaptation and recovery [1], [4], [5], [7], [19]. In well-trained cyclists, threshold development may be best supported when near-threshold work is embedded within a training structure that still preserves substantial low-intensity volume and selected high-intensity stimuli.

Peak fat oxidation and power at Fatmax improved most clearly after polarized training and, to a lesser extent, after pyramidal training. These results align with foundational work showing that substrate utilization during exercise is strongly shaped by both exercise intensity and training background [13]–[18], [24]. An increase in peak fat oxidation suggests that the cyclist becomes better able to rely on lipid metabolism during prolonged submaximal workloads. That adaptation may reduce carbohydrate dependence, preserve muscle glycogen, and support metabolic stability during long-duration training and racing.

Figure 4 demonstrates that the fat oxidation profile shifted across a range of exercise intensities. In the polarized group, the entire curve shifted upward, indicating a broader enhancement of metabolic flexibility rather than a trivial change at a single isolated workload. This matters because real cycling performance unfolds across changing intensities rather than at a single laboratory stage. A cyclist who can oxidize more fat over a broader submaximal range may be better equipped to absorb tactical variability, climb repeatedly, and still preserve carbohydrate availability for critical race moments.

The moderate-to-strong positive correlation suggests that these variables are not unrelated. Improvements in fat oxidation do not replace threshold power as a determinant of performance, but they may support it by improving the metabolic background against which threshold work is sustained. This interpretation is compatible with the idea that endurance adaptation is best understood as a systems response rather than as a collection of isolated markers.

In well-trained cyclists, a program characterized by a large low-intensity base and a clearly defined high-intensity component appears to offer a productive environment for improving both central aerobic function and submaximal metabolic economy. The pyramidal model may still be useful and appears to provide a solid compromise between metabolic specificity and manageable load.

Table 2 demonstrates that the largest and most consistent improvements occurred in the same group across several physiologically meaningful outcomes. Figure 2 confirms that the groups truly differed in achieved training distribution. Figure 3 shows that the main outcomes moved in parallel, and Figure 4 illustrates that the substrate-use response was distributed across multiple intensities rather than restricted to one stage. Together, these displays strengthen the paper's internal coherence.

Several limitations should be acknowledged. First, the sample was limited to well-trained young men, so the findings cannot be generalized directly to women, master's cyclists, or elite professionals. Second, the study relied on indirect calorimetry-derived fat oxidation rather than stable isotope methods, although the chosen approach remains standard and practical for applied exercise physiology [17], [24]. Third, overall load was matched as closely as possible, but perfect equivalence in the lived training experience of each cyclist cannot be guaranteed.

Future work should extend this model in several directions. Longer interventions could clarify whether pyramidal and polarized distributions diverge further over time or converge. Female cyclist cohorts deserve a dedicated investigation rather than indirect inference from male samples. It would also be valuable to include direct race-performance markers, muscle glycogen assessments, or longitudinal follow-up across a full training season.

One reason the present findings may resonate with coaches is that they match practical experience. Riders often report that a large low-intensity base leaves them fresher for quality sessions and more capable of accumulating substantial weekly volume. When a smaller amount of high-intensity work is deliberately introduced, the result may be a more manageable balance between stimulus and recovery.

During high-intensity racing, carbohydrate remains indispensable. The relevant point is not to maximize fat use under all circumstances, but to improve the athlete's ability to rely on lipid metabolism at workloads where such reliance is advantageous. In that sense, increased fat oxidation may be viewed as a marker of enhanced metabolic flexibility rather than a stand-alone target.

VI. CONCLUSION

In well-trained male cyclists, the distribution of training intensity appears to influence the adaptation of aerobic capacity, lactate-threshold power, and fat oxidation over a 10-week training period. The present study suggests that a polarized distribution—characterized by a substantially low-intensity base and a modest but distinct high-intensity component—produced the most favorable integrated response. Cyclists assigned to this model showed the largest gains in VO_2max, peak aerobic power, lactate-threshold power, power at Fatmax, and peak fat oxidation, together with the largest decrease in submaximal respiratory exchange ratio.

These findings are important because they show that the physiological consequences of training distribution extend beyond the traditional emphasis on VO_2max alone. A cyclist may improve not only by raising maximal aerobic power, but also by becoming metabolically more economical at submaximal workloads and by reaching higher power outputs before lactate begins to accumulate sharply; that means a well-designed training distribution may support both the ceiling of aerobic performance and the fuel-use characteristics that make prolonged endurance work more sustainable.

The present results should be interpreted with individual caution, because no single distribution model is ideal for every rider at every point in the season. Even so, the overall pattern supports a programming philosophy that preserves high low-intensity volume, uses threshold work selectively, and includes a clearly defined high-intensity component when the goal is to improve aerobic capacity, threshold power, and fat oxidation in already well-trained cyclists.

The pattern of findings suggests that the distribution of intensity shapes adaptation through more than one route. The polarized model appeared to provide a favorable balance between sufficient low-intensity volume to support oxidative development and enough high-intensity work to stimulate robust central and peripheral adaptation. The pyramidal model also produced worthwhile changes, which reinforces the view that high-quality endurance programming does not depend on a single rigid formula. What seems to matter most is that the distribution allows athletes to accumulate meaningful low-intensity exposure without losing the stimulus required to improve high-level physiological function.

In cycling, performance is rarely explained by a single variable. Riders must combine high oxygen-transport capacity, sustainable threshold power, and efficient substrate utilization to perform well in long events or demanding training blocks. By examining these variables together, the present study offers a more comprehensive interpretation of how the distribution of training intensity may influence real endurance preparation in well-trained athletes.

Accordingly, coaches and practitioners should view training distribution as a strategic tool rather than a simple description of weekly intensity. Careful allocation of time across intensity zones may help optimize physiological adaptation, preserve recovery quality, and support durable endurance performance throughout a training cycle. Future studies should test whether similar patterns are observed in elite cyclists, female athletes, and longer intervention periods. Still, the present evidence supports the idea that how cyclists train matters as much as how much they train.

Author Contributions

The author conducted the conceptualization, methodology, data analysis, investigation, writing, review, editing, and final approval of the manuscript.

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Data Availability

The dataset will be available from the author upon reasonable request.

Conflicts of Interest

The author declares no conflict of interest.

REFERENCES

1. S. Seiler, “What is best practice for training intensity and duration distribution in endurance athletes?” Int. J. Sports Physiol. Perform., vol. 5, no. 3, pp. 276–291, 2010, doi: 10.1123/ijspp. 5.3.276.
2. K. S. Seiler and G. Ø. Kjerland, “Quantifying training intensity distribution in elite endurance athletes: Is there evidence for an optimal distribution?” Scand. J. Med. Sci. Sports, vol. 16, no. 1, pp. 49–56, 2006, doi: 10.1111/j.1600-0838.2004.00418.x.
3. J. Esteve-Lanao, C. Foster, S. Seiler, and A. Lucia, “Impact of training intensity distribution on performance in endurance athletes,” J. Strength Cond. Res., vol. 21, no. 3, pp. 943–949, 2007, doi: 10.1519/R-19725.1.
4. C. M. Neal et al., “Six weeks of a polarized training-intensity distribution leads to greater physiological and performance adaptations than a threshold model in trained cyclists,” J. Appl. Physiol., vol. 114, no. 4, pp. 461–471, 2013, doi: 10.1152/japplphysiol. 00652.2012.
5. T. L. Stöggl and B. Sperlich, “The training intensity distribution among well-trained and elite endurance athletes,” Front. Physiol., vol. 6, art. 295, 2015, doi: 10.3389/fphys. 2015.00295.
6. B. R. Rønnestad, J. Hansen, and S. Ellefsen, “Block periodization of high-intensity aerobic intervals provides superior training effects in trained cyclists,” Scand. J. Med. Sci. Sports, vol. 24, no. 1, pp. 34–42, 2014, doi: 10.1111/j.1600-0838.2012.01485.x.
7. J. R. Hydren and B. S. Cohen, “Current scientific evidence for a polarized cardiovascular endurance training model,” J. Strength Cond. Res., vol. 29, no. 12, pp. 3523–3530, 2015, doi: 10.1519/JSC.0000000000001197.
8. P. B. Laursen and D. G. Jenkins, “The scientific basis for high-intensity interval training: Optimizing training programs and maximizing performance in highly trained endurance athletes,” Sports Med., vol. 32, no. 1, pp. 53–73, 2002, doi: 10.2165/00007256-200232010-00003.
9. A. W. Midgley, L. R. McNaughton, and A. M. Jones, “Training to enhance the physiological determinants of long-distance running performance,” Sports Med., vol. 37, no. 10, pp. 857–880, 2007, doi: 10.2165/00007256-200737100-00003.
10. M. J. Joyner and E. F. Coyle, “Endurance exercise performance: The physiology of champions,” J. Physiol., vol. 586, no. 1, pp. 35–44, 2008, doi: 10.1113/jphysiol. 2007.143834.
11. E. F. Coyle, A. R. Coggan, M. K. Hopper, and T. J. Walters, “Determinants of endurance in well-trained cyclists,” J. Appl. Physiol., vol. 64, no. 6, pp. 2622–2630, 1988, doi: 10.1152/jappl. 1988.64.6.2622.
12. E. F. Coyle et al., “Physiological and biomechanical factors associated with elite endurance cycling performance,” Med. Sci. Sports Exerc., vol. 23, no. 1, pp. 93–107, 1991, doi: 10.1249/00005768-199101000-00015.
13. J. A. Romijn et al., “Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration,” Am. J. Physiol. Endocrinol. Metab., vol. 265, no. 3, pp. E380–E391, 1993, doi: 10.1152/ajpendo.1993.265.3.E380.
14. G. A. Brooks and J. Mercier, “Balance of carbohydrate and lipid utilization during exercise: The crossover concept,” J. Appl. Physiol., vol. 76, no. 6, pp. 2253–2261, 1994, doi: 10.1152/jappl. 1994.76.6.2253.
15. J. Achten and A. E. Jeukendrup, “Determination of the exercise intensity that elicits maximal fat oxidation,” Med. Sci. Sports Exerc., vol. 34, no. 1, pp. 92–97, 2002, doi: 10.1097/00005768-200201000-00015.
16. J. Achten and A. E. Jeukendrup, “Maximal fat oxidation during exercise in trained men,” Int. J. Sports Med., vol. 24, no. 8, pp. 603–608, 2003, doi: 10.1055/s-2003-43265.
17. A. E. Jeukendrup and G. A. Wallis, “Measurement of substrate oxidation during exercise by means of gas exchange measurements,” Int. J. Sports Med., vol. 26, suppl. 1, pp. S28–S37, 2005, doi: 10.1055/s-2004-830512.
18. M. C. Venables, J. Achten, and A. E. Jeukendrup, “Determinants of fat oxidation during exercise in healthy men and women: A cross-sectional study,” J. Appl. Physiol., vol. 98, no. 1, pp. 160–167, 2005, doi: 10.1152/japplphysiol. 00662.2003.
19. B. C. Bergman and G. A. Brooks, “Respiratory gas-exchange ratios during graded exercise in fed and fasted trained and untrained men,” J. Appl. Physiol., vol. 86, no. 2, pp. 479–487, 1999, doi: 10.1152/jappl. 1999.86.2.479.
20. J. H. Goedecke et al., “Metabolic adaptations to a high-fat diet in endurance cyclists,” Metabolism, vol. 48, no. 12, pp. 1509–1517, 1999, doi: 10.1016/S0026-0495(99)90238-X.
21. K. McGawley et al., “No additional benefits of block- over evenly-distributed high-intensity interval training within a polarized microcycle,” Front. Physiol., vol. 8, art. 413, 2017, doi: 10.3389/fphys. 2017.00413.
22. I. Muñoz, R. Cejuela, S. Seiler, E. Larumbe, and J. Esteve-Lanao, “Training-intensity distribution during an Ironman season: Relationship with competition performance,” Int. J. Sports Physiol. Perform., vol. 9, no. 2, pp. 332–339, 2014, doi: 10.1123/IJSPP.2012-0352.
23. J. G. Bourgois, G. Bourgois, and J. Boone, “Perspectives and determinants for training-intensity distribution in elite endurance athletes,” Int. J. Sports Physiol. Perform., vol. 14, no. 8, pp. 1151–1156, 2019, doi: 10.1123/ijspp. 2018-0722.

24. K. N. Frayn, “Calculation of substrate oxidation rates in vivo from gaseous exchange,” J. Appl. Physiol. Respir. Environ. Exerc. Physiol., vol. 55, no. 2, pp. 628–634, 1983, doi: 10.1152/jappl. 1983.55.2.628.
25. L. Havemann et al., “Fat adaptation followed by carbohydrate loading compromises high-intensity sprint performance,” J. Appl. Physiol., vol. 100, no. 1, pp. 194–202, 2006, doi: 10.1152/japplphysiol. 00813.2005.