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Training Load Control During Training Camp Using Hematological Biomarkers and Digital Training Data

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Abstract

This study examined hematological and cytokine responses during a supervised 10-day triathlon training camp with individualized, lactate-threshold–based intensity prescription in amateur athletes. Eighty-four participants were enrolled, and a biomarker subcohort (n = 59) underwent serial blood sampling at baseline, day 5, and day 10. Training load was managed using individualized heart rate, power, and pace targets, digital training data aggregated in TrainingPeaks, session-RPE, coach observations, and interim complete blood count monitoring. Cytokine profiling was performed post hoc and did not influence real-time training decisions. Despite a twofold increase in cycling session duration on the corresponding sampling days, perceived exertion remained stable. Individual load modifications were made when signs of disproportionate physiological stress were identified. Complete blood count showed moderate increases in leukocytes and neutrophils, with transient lymphocyte redistribution, while all values remained within reference ranges. Cytokine profiling showed reduced CXCL10/IP-10 and interleukin-8 concentrations, alongside increased interleukin-1 receptor antagonist and selected growth-factor– and platelet-associated mediators. Overall, the findings support the feasibility of combining threshold-based prescription, digital training records, and interim hematological monitoring for practical load supervision during intensified triathlon training in heterogeneous amateur athletes.

Keywords

triathlon; cycling; running; training camp; training load; cytokines; immune adaptation

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1 Introduction

Training camps for amateur endurance athletes involve a rapid increase in training volume and frequency compared with habitual training routines. In recreational athletes, heterogeneity in fitness level, training history, recovery capacity, and non-sport stressors may increase the risk of inappropriate load progression and maladaptive fatigue. Therefore, objective intensity prescription and close supervision are essential during intensified training blocks, as training-load monitoring is considered central for understanding fatigue and reducing the risk of maladaptation (Halson, 2014). Recent cycling camp research has also emphasized the value of monitoring internal and external load variables, including session-RPE, training duration, distance, power, and heart rate, across intensified camp microcycles (Martinez-Gonzalez et al., 2024).

Threshold-based exercise prescription, digital training records, perceived exertion, and routine hematological monitoring may provide complementary information for practical load regulation. Session-RPE is an established practical approach for quantifying internal training load and linking athlete perception with session duration (Foster et al., 2001). This study examined hematological and cytokine responses during a supervised 10-day triathlon camp in amateur athletes, with training load managed using individualized lactate-threshold zones, digital training data, coach supervision, and interim complete blood count monitoring.

2 Material and Methods

This study was conducted during a 10-day triathlon training camp. Total training volume ranged from 13.1–15.9 h for cycling and 5.1–6.2 h for running, while swimming volume was standardized at 3 h across groups. Eighty-four amateur athletes participated in the camp. Athletes were allocated separately within each discipline into performance-based training groups: 9 cycling groups (typically 8–10 athletes per group), 5 running groups (typically 14–18 athletes per group), and 6 swimming groups (typically 12–16 athletes per group). These discipline-specific groups were used for training prescription and supervision and should not be interpreted as additive independent statistical subgroups. A biomarker subcohort (n = 59; 11 women; age 31–60 years) underwent serial blood sampling. The subcohort was prospectively selected from athletes aged ≥30 years who consented to serial blood sampling and met eligibility criteria. Participants were not included in biomarker analyses if they joined the camp late, arrived on the sampling day, arrived one day before sampling after crossing more than four time zones, or were younger than 30 years. Inclusion criteria included systematic endurance training for at least 6 months, absence of acute illness or infection during the preceding 3 weeks, and no use of systemic immunosuppressive or anti-inflammatory drugs before sampling.

Before the training block, athletes completed cardiopulmonary exercise testing with breath-by-breath gas analysis and stepwise capillary lactate measurements. Aerobic and anaerobic thresholds were defined at fixed lactate concentrations of 2 and 4 $mmol \cdot L^{-1}$, respectively. Heart rate, cycling power, and running speed at these thresholds, together with $\dot{V}O_2max$, were used to establish individualized training zones and prescribe exercise intensity.

Training was individually prescribed and supervised. Training data, including heart rate, power, pace/speed, distance, duration, elevation gain, session-RPE, athlete comments, and planned-versus-completed sessions, were

uploaded from athletes' personal devices (Garmin, Wahoo, Polar, Suunto, and Coros) and aggregated in TrainingPeaks. These data were reviewed by coaches, physicians, and physiologists to monitor session compliance, training progression, and individual responses throughout the camp. During cycling sessions, a coach rode with the athletes and monitored power, heart-rate compliance, and perceived exertion. During running sessions, pace was controlled, and capillary lactate was measured in selected sessions to verify individualized 2 $mmol \cdot L^{-1}$ reference pace.

Interim complete blood count results, including total leukocytes and leukocyte differential, were reviewed during the camp together with heart rate–power relationships, session-RPE, athlete complaints, and coach observations. When signs of disproportionate physiological stress were identified, load adjustments were made by the physician and physiologists assigned to the athletes. Load adjustments included transfer to a lower-intensity group, reduced intensity within the assigned group, complete rest, or prescription of an individualized running pace. Cytokine profiling was performed post hoc for research purposes and was not used for real-time training decisions.

Venous blood samples were collected at baseline (t0), mid-microcycle (t1; day 5) after a moderate cycling session, and at the end of the microcycle (t2; day 10) after a prolonged cycling session. Samples were obtained within the first 30–60 minutes after exercise. Complete blood count was analyzed using an automated hematology analyzer, and plasma cytokines were quantified using a 39-analyte multiplex immunoassay.

Participants stayed in the same hotel environment and received centrally provided buffet-style meals. Sleep, hydration, and nutrition were not experimentally standardized. Athletes followed a common camp schedule that included regular educational lectures on training load management, recovery, nutrition, and hydration; they were also instructed to maintain regular sleep and hydration practices throughout the camp.

Data are presented as median (interquartile range). Time effects were assessed using repeated-measures models with false discovery rate correction ($q \leq 0.05$). Exploratory analyses were adjusted or stratified by sex, age category, and performance group where applicable, although the study was not powered for definitive subgroup comparisons.

3 Results

During the 10-day training microcycle, perceived exertion remained stable. Borg CR10 scores were 5.0 (3.8–6.0) at day 5 (t1) and 5.5 (4.0–7.0) at day 10 (t2) ($p = 0.14$), despite a twofold increase in training duration from 2.5 to 5.0 hours on the corresponding sampling days.

Individual load modifications were made in athletes showing signs of disproportionate physiological stress based on combined evaluation of TrainingPeaks-derived training data, coach observations, subjective responses, and, where available, complete blood count dynamics. Adjustments included transfer to a lower-intensity group, reduced intensity within the assigned group, complete rest, or individualized running pace prescription.

Complete blood count parameters showed small but statistically robust shifts while remaining within established reference ranges. Total white blood cell count increased from 6.6 (5.7–8.0) at baseline (t0) to 7.9 (6.6–8.6) at t1 and 7.8 (6.6–9.4) at t2 $\times 10^9$/L (FDR-adjusted $q \leq 0.0001$), primarily driven by neutrophils (4.1 [3.2–5.3] to 5.2 [4.3–6.6] $\times 10^9$/L; $q \leq 0.0001$). Lymphocytes demonstrated a transient

decrease at t1 with recovery at t2 (1.7 [1.4–2.0], 1.5 [1.2–1.7], and 1.7 [1.3–2.0] $\times 10^9$/L; $q \leq 0.0001$).

Plasma cytokine profiling showed significant changes in selected inflammatory and regulatory mediators. CXCL10/IP-10 decreased from 251.4 (233.6–287.0) at t0 to 80.9 (27.4–150.3) pg/mL at t2 ($q \leq 0.0001$), representing a 68% reduction. IL-8 declined from 18.7 (15.4–23.0) to 15.0 (12.6–19.1) pg/mL ($q \leq 0.0001$). IL-1RA increased from 5.0 (5.0–19.3) to 16.6 (5.0–31.8) pg/mL ($q \leq 0.05$). TGF-α and sCD40L also increased over time (both $q \leq 0.0001$).

4 Discussion

This study shows that a supervised, threshold-guided triathlon camp model can support increased training exposure in amateur athletes while maintaining stable perceived exertion and a controlled hematological profile. The practical novelty of the model lies in combining laboratory-derived lactate thresholds, digital training records, coach supervision, subjective load markers, and interim complete blood count monitoring. The use of session-RPE and digital training records is consistent with established approaches to internal and external training-load monitoring, where session duration, perceived exertion, heart rate, power, and training history are used to evaluate athlete responses and guide coaching decisions (Foster et al., 2001; Halson, 2014). This approach is also consistent with recent training-camp research in cycling, where internal and external load variables such as session-RPE, training duration, distance, power, and heart rate were used to characterize load variation across camp microcycles (Martinez-Gonzalez et al., 2024).

The stable Borg CR10 scores despite increased cycling session duration suggest that training load progression was tolerated without progressive subjective overload. The moderate leukocytosis with neutrophil predominance, together with transient lymphocyte redistribution and preservation of values within reference ranges, is consistent with expected immunohematological responses to repeated endurance exercise. Exercise-induced leukocytosis and shifts in leukocyte subsets are well-described physiological responses to acute and repeated exercise exposure, reflecting hemodynamic redistribution, neuroendocrine activation, and immune-cell trafficking (Walsh et al., 2011). The absence of clinically concerning hematological changes supports the interpretation that the training block did not produce a maladaptive systemic stress response.

The cytokine findings provide additional post hoc evidence that the training block was not accompanied by escalating systemic inflammatory activation. The reduction in CXCL10/IP-10 was the most pronounced cytokine change. CXCL10 is an interferon-inducible CXCR3 ligand involved in the recruitment of activated T cells and natural killer cells (Metzemaekers et al., 2018). Therefore, the observed reduction may be compatible with attenuation of interferon-related inflammatory signaling during repeated training exposure. However, because no non-training control group was included and resting pre-camp cytokine values were not available outside the post-exercise context, this interpretation should remain cautious.

The decline in IL-8 also supports reduced inflammatory chemokine signaling. IL-8 is a classical chemotactic and inflammatory cytokine involved in neutrophil recruitment (Baggiolini and Clark-Lewis, 1992). At the same time, IL-8 has dual relevance in exercise physiology, because muscular IL-8 is regulated

by muscle contractions and is discussed within the broader context of exercise-induced cytokines and myokine signaling (Nielsen and Pedersen, 2007). Therefore, reduced IL-8 after the camp may reflect lower systemic inflammatory signaling, altered post-exercise kinetics, or adaptation of the acute exercise response rather than a single mechanism.

The increase in IL-1RA is consistent with anti-inflammatory counter-regulation, which aligns with broader models of exercise-induced anti-inflammatory effects (Gleeson et al., 2011). TGF-α and sCD40L should not be interpreted as a single tissue-repair pathway. TGF-α is part of the epidermal growth factor receptor ligand family and is related to growth-factor signaling rather than classical anti-inflammatory signaling (Schneider and Wolf, 2009). In contrast, sCD40L should be interpreted cautiously as a platelet-associated immune mediator. Activated platelets can express CD40L and trigger endothelial inflammatory signaling, supporting its interpretation as a platelet-immune marker rather than a simple repair marker (Henn et al., 1998). Their increase therefore suggests involvement of growth-factor–related and platelet-immune pathways, but mechanistic conclusions require further study.

Overall, these findings suggest that laboratory-derived thresholds, digital training records, and interim complete blood count monitoring can complement each other in the practical supervision of intensified training in amateur athletes. This approach may be especially relevant for amateur populations, where differences in fitness, recovery capacity, training history, and life stress increase the risk of inappropriate load escalation.

5 Limitations

Several limitations should be acknowledged. First, the study did not include a non-individualized or non-training control group; therefore, causal attribution to the supervision model should be made cautiously. Second, although exploratory stratified analyses were performed, the study was not powered for definitive subgroup comparisons by sex, age, or performance group. Third, the biomarker subcohort may be affected by selection bias because participants were included based on age, consent, logistics, arrival timing, and availability of complete sampling. Fourth, nutrition, sleep, and hydration were managed within the practical constraints of a real-world camp but were not experimentally standardized. Finally, cytokine profiling was performed post hoc and should be interpreted as a research outcome rather than a real-time load-adjustment tool.

6 Conclusions

Individualized, threshold-guided supervision provides a practical framework for safe load progression during intensified triathlon training in amateur athletes. This camp-based model may help translate laboratory testing, digital training records, and routine hematological monitoring into applied decisions for heterogeneous recreational endurance populations.

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Conflicts of Interest: The authors declare no conflict of interest.

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