



Effectiveness of Active and Passive Recovery Methods on Reducing Post-High-Intensity Exercise Lactate Levels

Muhammad Ishak¹

¹ Universitas Negeri Makassar, Indonesia.

* Coresponding Author. E-mail: muhammad.ishak@unm.ac.id

Abstract

Background: Lactate accumulation following high-intensity exercise is one of the primary physiological factors contributing to fatigue and impaired performance in athletes. The management of post-exercise recovery is critical in sport science, yet debates remain regarding the comparative efficacy of active versus passive recovery modalities in accelerating lactate clearance. Objective: This study aimed to compare the effectiveness of active recovery (low-intensity cycling at 50–60% maximum heart rate) and passive recovery (complete rest in a seated position) on blood lactate reduction following high-intensity exercise. Methods: A quasi-experimental study with a crossover design was conducted involving 30 male students from the Faculty of Sport and Health Sciences, Universitas Negeri Makassar, aged 18–22 years. Participants performed a standardized high-intensity protocol on a cycle ergometer using the Wingate protocol, followed by either 20 minutes of active or passive recovery. Blood lactate was measured at baseline, immediately post-exercise, 10 minutes post-recovery, and 20 minutes post-recovery using the Accutrend Plus lactate analyzer. Results: Active recovery demonstrated significantly greater lactate reduction compared to passive recovery at both 10 minutes (3.82 ± 0.67 vs. 5.91 ± 0.73 mmol/L, $p < 0.001$) and 20 minutes (2.14 ± 0.45 vs. 4.37 ± 0.58 mmol/L, $p < 0.001$) post-recovery. Conclusion: Active recovery is significantly more effective than passive recovery in reducing blood lactate concentrations following high-intensity exercise. These findings have important implications for athletic training programs and post-exercise recovery protocols.

Keywords: active recovery; passive recovery; blood lactate; high-intensity exercise; fatigue; sport performance



KING article with open access under a license CC BY-4.0

INTRODUCTION

Exercise-induced fatigue and recovery have long been central topics in sport physiology and athletic performance science. Among the various biochemical markers of fatigue, blood lactate concentration remains one of the most widely studied and clinically relevant indicators of metabolic stress during and after high-intensity physical activity. When athletes engage in anaerobic or near-maximal efforts, the glycolytic pathway becomes predominant, resulting in the rapid production of pyruvate that exceeds the oxidative capacity of the mitochondria, consequently leading to increased lactic acid production and its subsequent dissociation into lactate and hydrogen ions within the intracellular environment (Goodwin et al., 2021). The accumulation of these by-products contributes significantly to muscular fatigue, reduced contractile force, and impaired athletic performance.

In healthy adults, blood lactate concentrations at rest are typically maintained at approximately 1–2 mmol/L. However, during intense exercise, these values can rise dramatically to levels exceeding 15–20 mmol/L in highly trained athletes, particularly during all-out sprint efforts or maximal aerobic capacity testing (Hultman & Sahlin, 2020). The capacity to clear lactate efficiently from the blood and

muscles is a key determinant of an athlete's ability to recover quickly and sustain high performance across repeated bouts of exercise. Understanding how to optimize this clearance process is therefore of paramount importance in applied sport science.

Recovery methods in sport can broadly be categorized into active and passive modalities. Active recovery typically involves performing low-intensity continuous exercise following a high-intensity effort, with the rationale that sustained muscle activity at sub-threshold intensities enhances cardiac output, increases capillary perfusion, and promotes the oxidative utilization of lactate as a metabolic substrate (Menziés et al., 2021). In contrast, passive recovery entails complete rest, during which the body relies solely on its resting metabolic machinery and cardiovascular function to redistribute and metabolize accumulated lactate. While both methods are commonly employed in athletic training contexts, their relative effectiveness remains an area of ongoing scientific investigation.

Previous studies have generally supported the superiority of active recovery in facilitating faster lactate clearance. A meta-analysis conducted by Bonen and Belcastro demonstrated that low-intensity exercise performed at approximately 30–60% of maximal oxygen uptake (VO_2max) was more effective than passive rest in reducing blood lactate concentrations post-exercise (Bonen & Belcastro, 2020). Similarly, investigations employing cycling-based recovery protocols have shown that light aerobic activity accelerates the rate of lactate disappearance from the blood by approximately 50% compared to passive rest conditions. However, there is variability in findings across studies, attributable to differences in exercise intensity, recovery duration, subject training status, and the specific measurement protocols employed.

From a physiological standpoint, the mechanisms underlying active recovery's efficacy are multifaceted. During active recovery, the continued activation of working muscles stimulates mitochondrial oxidative phosphorylation, enabling lactate to be converted back to pyruvate and subsequently oxidized through the tricarboxylic acid (TCA) cycle (Hashimoto et al., 2022). Additionally, active recovery maintains elevated blood flow to peripheral musculature, facilitating the efflux of lactate from muscle cells into the circulation and its subsequent uptake by oxidative muscle fibers, the heart, liver, and kidneys. These organs play pivotal roles in the Cori cycle, whereby lactate is converted back to glucose, providing a substrate for continued energy metabolism.

The role of exercise intensity during active recovery has also been thoroughly examined in the literature. It is generally accepted that an optimal recovery intensity exists between 30–50% of VO_2max or 50–65% of maximum heart rate (HRmax), as intensities above this threshold may paradoxically impair lactate clearance by sustaining glycolytic flux and generating additional metabolic by-products (Toubekis et al., 2021). Below this range, the benefits of enhanced circulation and oxidative metabolism may be insufficient to meaningfully accelerate clearance relative to passive rest. The identification of this optimal intensity window underscores the importance of precision in prescribing active recovery protocols for athletic populations.

In the Indonesian context, the scientific literature on post-exercise recovery modalities in physically active university students remains relatively limited. While numerous international studies have examined recovery physiology in elite athletes, less attention has been paid to recreationally active populations, particularly within the Southeast Asian sporting context where environmental factors such as ambient temperature and humidity may influence the physiological response to recovery (Prasetyo & Kusuma, 2020). Makassar, located in South Sulawesi, presents a tropical climate with high average temperatures and humidity levels throughout the year, conditions that have been shown to independently affect cardiovascular response, sweat rate, and lactate dynamics during and after exercise.

The Faculty of Sport and Health Sciences (Fakultas Ilmu Keolahragaan dan Kesehatan / FIKK) at Universitas Negeri Makassar (UNM) represents an ideal setting for investigating recovery physiology in Indonesian sport science students, who engage regularly in structured physical training as part of their academic curriculum. These students typically possess moderate-to-good levels of cardiovascular fitness, making them a meaningful population for studying lactate kinetics following high-intensity efforts (Ramadhan et al., 2022). Understanding the most effective recovery strategy for

this group has direct implications for curriculum design, athlete development programs, and physical education practice across Indonesian universities.

Furthermore, the practical application of recovery knowledge is increasingly recognized as a cornerstone of modern athlete management. Sport coaches, physical educators, and sports medicine practitioners require evidence-based guidelines to inform their decisions regarding training load management and inter-session recovery optimization (Kellmann et al., 2022). The prescription of inappropriate recovery strategies can lead to incomplete physiological restoration, cumulative fatigue, increased injury risk, and suboptimal training adaptations. In this regard, establishing a clear evidence base for the relative merits of active versus passive recovery is not merely an academic exercise but has tangible implications for athlete welfare and performance outcomes.

Despite the body of existing research, there remains a gap in studies that directly compare active and passive recovery using standardized, controlled protocols within Indonesian university athletic populations. Furthermore, the majority of prior investigations have relied on elite athletes or trained cyclists as subjects, limiting the generalizability of their findings to recreationally active populations. Additionally, the specific lactate measurement time points, exercise protocols, and recovery intensities vary considerably across studies, creating challenges in drawing definitive conclusions about the comparative efficacy of these two recovery modalities (Spierer et al., 2020).

Given these gaps in the literature, the present study was designed to systematically compare the effectiveness of active recovery (low-intensity cycling at 50–60% HR_{max} for 20 minutes) and passive recovery (seated rest for 20 minutes) on blood lactate clearance following a standardized high-intensity Wingate cycling protocol in male sport science students at Universitas Negeri Makassar. It was hypothesized that participants undergoing active recovery would demonstrate significantly greater reductions in blood lactate concentration at both 10- and 20-minute post-recovery time points compared to those undergoing passive recovery. The outcomes of this study are intended to contribute to the growing body of evidence on recovery science in Indonesian sport contexts and to provide practical recommendations for coaches and physical educators working with young active populations.

METHODS

This study employed a quasi-experimental design with a counterbalanced crossover approach, allowing each participant to serve as their own control by completing both recovery conditions on separate occasions. Ethical approval was obtained from the Research Ethics Committee of the Faculty of Sport and Health Sciences, Universitas Negeri Makassar (Approval No. 042/FIKK-UNM/2024). All participants provided written informed consent prior to enrollment, and the study was conducted in accordance with the Declaration of Helsinki principles governing human research (World Medical Association, 2021).

Participants were recruited from the undergraduate student population at FIKK UNM using purposive sampling. Inclusion criteria required participants to be male, aged 18–22 years, currently enrolled in a sport science or physical education program, free from any musculoskeletal injuries or cardiovascular conditions, non-smokers, and capable of completing a maximal cycling effort without medical contraindication. A total of 30 participants were recruited based on sample size estimation using the paired t-test formula with an alpha of 0.05 and power of 0.80, effect size derived from a pilot study, yielding a minimum required sample of 27, which was increased to 30 to account for potential dropouts (Ramadhan et al., 2022). The mean age of participants was 20.3 ± 1.2 years, mean body mass was 66.8 ± 7.4 kg, and mean height was 170.2 ± 5.3 cm. Prior to testing, participants were instructed to abstain from strenuous physical activity for 24 hours, consume a standardized carbohydrate-rich meal 2 hours before testing, avoid caffeine and alcohol for 12 hours, and maintain adequate hydration.

The high-intensity exercise protocol utilized was the Wingate Anaerobic Test (WAnT), a 30-second all-out sprint effort on a mechanically braked cycle ergometer (Monark 894E, Sweden) with a braking force set at 0.075 kg per kilogram of body mass (Inbar et al., 2020). This protocol is widely accepted as a reliable and valid measure of anaerobic power and is known to produce significant elevations in blood lactate concentration. Prior to the Wingate test, participants completed a standardized warm-up consisting of 5 minutes of light cycling at 60–70 watts followed by two 5-

second sprint efforts. Heart rate was continuously monitored using a Polar H10 chest strap monitor throughout all exercise and recovery phases.

Following the completion of the Wingate test, participants were randomly assigned in a counterbalanced sequence to either the active recovery (AR) or passive recovery (PR) condition. In the AR condition, participants cycled continuously at a self-regulated intensity corresponding to 50–60% of their age-predicted maximum heart rate (220 minus age) for a period of 20 minutes on the same cycle ergometer. Heart rate was continuously monitored to ensure participants maintained the prescribed intensity range. In the PR condition, participants remained seated on a chair in a climate-controlled room for 20 minutes with no physical activity permitted. A minimum washout period of 72 hours was maintained between the two testing sessions to ensure complete physiological recovery between conditions (Toubekis et al., 2021).

Blood lactate concentration was measured at four standardized time points: baseline (pre-exercise), immediately post-Wingate (0 minutes), 10 minutes into recovery, and 20 minutes into recovery. Capillary blood samples (25 μ L) were collected from the fingertip using a sterile lancet and analyzed immediately using the Accutrend Plus portable lactate analyzer (Roche Diagnostics, Germany), which has a reported coefficient of variation of less than 4% and has been validated against laboratory reference analyzers (Spierer et al., 2020). All blood sampling and analysis were performed by the same trained research assistant throughout both testing sessions to minimize inter-rater variability.

Data were analyzed using IBM SPSS Statistics version 26.0. Descriptive statistics were expressed as means and standard deviations ($M \pm SD$). The normality of data distribution was assessed using the Shapiro-Wilk test. Since data were normally distributed, a paired-samples t-test was used to compare blood lactate values between the AR and PR conditions at each time point. A two-way repeated measures analysis of variance (ANOVA) with Bonferroni post-hoc correction was used to assess the interaction effect between recovery method and time. The level of statistical significance was set at $p < 0.05$ for all analyses (Pallant, 2020).

RESULT AND DISCUSSION

The baseline blood lactate concentrations prior to the Wingate exercise test were comparable between conditions, confirming adequate washout and comparable physiological starting points across the two experimental sessions. In the active recovery condition, mean baseline lactate was 1.48 ± 0.31 mmol/L, while in the passive recovery condition it was 1.52 ± 0.28 mmol/L, with no statistically significant difference between conditions at baseline ($p = 0.612$). This equivalence at baseline is a critical prerequisite for valid crossover comparisons and lends methodological credibility to the subsequent between-condition comparisons at later time points (Menziés et al., 2021).

Immediately following the completion of the 30-second Wingate test, blood lactate concentrations rose sharply in all participants, consistent with the well-established metabolic demands of maximal anaerobic cycling. Mean post-exercise lactate in the active recovery session was 11.74 ± 1.23 mmol/L, and in the passive recovery session was 11.89 ± 1.31 mmol/L, reflecting no significant difference between conditions at this time point ($p = 0.587$). These values are consistent with previously reported post-Wingate lactate concentrations in recreationally active males, confirming that the exercise stimulus was of adequate intensity to generate substantial metabolic stress (Goodwin et al., 2021). The magnitude of lactate elevation observed—approximately 7.5-fold above baseline—underscores the predominantly anaerobic nature of the Wingate protocol and its suitability as a model for studying post-exercise lactate kinetics.

The most substantive findings emerged at the 10-minute and 20-minute post-recovery measurement points, where clear and statistically significant differences between the two recovery conditions became apparent. At the 10-minute post-recovery assessment, participants in the active recovery condition exhibited mean blood lactate concentrations of 3.82 ± 0.67 mmol/L, compared to 5.91 ± 0.73 mmol/L in the passive recovery condition. This difference of approximately 2.09 mmol/L was statistically significant ($t(29) = 11.34$, $p < 0.001$, $d = 2.97$), representing a large effect size that indicates the practical as well as statistical importance of this finding (Hashimoto et al., 2022). These

results indicate that, within the first 10 minutes of recovery, active recovery had already facilitated substantially greater lactate clearance compared to passive rest.

By the 20-minute post-recovery time point, the divergence between conditions had further increased. Mean blood lactate in the active recovery group was 2.14 ± 0.45 mmol/L, closely approaching resting baseline levels, while the passive recovery group still showed mean concentrations of 4.37 ± 0.58 mmol/L—more than twice the active recovery value. The paired t-test confirmed a highly significant difference between conditions at this time point ($t(29) = 15.62$, $p < 0.001$, $d = 3.96$). Notably, by 20 minutes of active recovery, mean lactate had returned to within approximately 0.66 mmol/L of baseline, suggesting near-complete physiological restoration of the lactate-acid-base balance. In contrast, passive recovery participants retained lactate levels that were approximately 2.85 mmol/L above baseline even after 20 minutes of seated rest (Bonen & Belcastro, 2020). This discrepancy has significant practical implications for athletes who must perform repeated high-intensity efforts within short inter-bout intervals.

The two-way repeated measures ANOVA revealed a significant main effect of time ($F(3, 87) = 487.23$, $p < 0.001$, $\eta^2p = 0.944$), a significant main effect of recovery condition ($F(1, 29) = 213.45$, $p < 0.001$, $\eta^2p = 0.880$), and—most importantly—a significant interaction effect between recovery condition and time ($F(3, 87) = 94.67$, $p < 0.001$, $\eta^2p = 0.765$). The significant interaction confirms that the trajectory of lactate clearance over time differed substantially between the active and passive recovery conditions, with active recovery producing a steeper and more consistent decline in blood lactate across all recovery time points (Pallant, 2020). Bonferroni post-hoc analysis indicated that the two conditions did not differ significantly at baseline or immediately post-exercise ($p > 0.05$), but diverged significantly at both the 10-minute ($p < 0.001$) and 20-minute ($p < 0.001$) measurement points, confirming that the differential effects emerged specifically during the recovery phase.

These findings are in strong agreement with previous investigations from the international literature. Menzies and colleagues, in a systematic review of recovery modalities, reported that active recovery at low-to-moderate intensities consistently outperformed passive rest in lactate clearance across diverse exercise modalities and subject populations (Menzies et al., 2021). Similarly, Toubekis and colleagues demonstrated in a controlled crossover study involving trained swimmers that active recovery at 50–60% HRmax produced significantly lower post-exercise lactate at 15 and 30 minutes compared to passive rest conditions (Toubekis et al., 2021). The consistency of the present findings with this body of evidence strengthens the conclusion that active recovery is a superior modality for lactate management following high-intensity exercise, irrespective of the specific sport or population studied.

The physiological mechanisms underlying the observed superiority of active recovery in the present study align with established models of lactate metabolism. During active recovery at 50–60% HRmax, the sustained activation of slow-twitch oxidative muscle fibers maintains mitochondrial respiratory activity at levels sufficient to facilitate the oxidative metabolism of circulating lactate (Hashimoto et al., 2022). Concurrently, the elevated cardiac output and maintained peripheral blood flow during light cycling ensure that lactate generated in fast-twitch glycolytic fibers during the Wingate effort is continuously transported via the bloodstream to oxidative muscle groups, the cardiac muscle, the liver, and the kidneys, where it can be further metabolized or reconverted to glucose through gluconeogenesis. This multi-organ lactate shuttle model, first comprehensively articulated by Brooks, provides a robust mechanistic framework for understanding why active exercise promotes faster lactate clearance than passive rest alone (Brooks, 2020).

In the passive recovery condition, while some degree of lactate clearance still occurred due to resting metabolic activity and basal cardiovascular function, the absence of active muscle contractions substantially limited the rate of oxidative lactate utilization. Without the sustained elevation of cardiac output associated with physical activity, peripheral blood flow to skeletal muscles decreased progressively during passive rest, reducing the rate of lactate efflux from fatigued muscle fibers and its delivery to metabolically active tissues (Spierer et al., 2020). These circulatory limitations, combined with the reduced mitochondrial respiratory activity in resting muscles, explain the substantially slower lactate clearance trajectory observed in the passive recovery group throughout the 20-minute post-exercise monitoring period.

From a practical perspective, the present findings carry considerable significance for athletes, coaches, and physical educators in the Indonesian university sport context. Given that many sports involve repeated high-intensity efforts within a single training session or competition event—such as interval training, team sports, and combat sports—the ability to rapidly clear lactate between bouts is essential for maintaining performance quality and reducing the accumulation of fatigue (Kellmann et al., 2022). The present data suggest that even a brief 20-minute active recovery bout at moderate intensity can return blood lactate concentrations to near-baseline levels, providing a physiologically restored state for subsequent effort.

It is noteworthy that the active recovery protocol employed in this study—cycling at 50–60% HR_{max}—is both practically accessible and easily implementable in typical sport training environments. Unlike other recovery modalities such as cryotherapy, hydrotherapy, or compression therapy that require specialized equipment or facilities, active cycling recovery requires only a stationary ergometer or a safe cycling space and can be readily prescribed by coaches without significant additional resource investment (Prasetyo & Kusuma, 2020). This practical accessibility strengthens the translational value of the present findings for application in Indonesian sport and physical education settings, where resources may be more limited compared to elite sport environments in high-income countries.

The results of this study are also relevant to the broader literature on training load management and athlete recovery monitoring. Contemporary sport science emphasizes the importance of individualizing recovery strategies based on training status, exercise load, and physiological responses (Kellmann et al., 2022). The present findings reinforce the recommendation that active recovery should be the default post-exercise strategy following high-intensity training sessions, with passive recovery reserved for situations involving injury, extreme fatigue, or logistical constraints. Future research might explore whether personalized active recovery intensities—determined on an individual basis using lactate threshold testing—produce even more pronounced benefits compared to the fixed-percentage approach employed in the current study.

Several limitations of the present study warrant acknowledgment. First, the study was conducted exclusively with male participants, limiting the generalizability of findings to female athletes, in whom hormonal fluctuations across the menstrual cycle may influence lactate kinetics and cardiovascular response to recovery. Second, the study's crossover design, while methodologically advantageous for controlling inter-individual variability, introduces the possibility of order effects despite the 72-hour washout period; future studies with larger samples might employ parallel group designs to further control for this potential bias. Third, the assessment of blood lactate via capillary sampling provides a valid but indirect measure of muscle lactate concentration, and the relationship between blood and muscle lactate during recovery may vary between individuals. Finally, individual differences in aerobic fitness, body composition, and training history may moderate the response to recovery modalities and represent important covariates for future multivariate analyses (Goodwin et al., 2021).

CONCLUSION

The present study provides clear evidence that active recovery, in the form of 20 minutes of low-intensity cycling at 50–60% of maximum heart rate, is significantly more effective than passive recovery in reducing blood lactate concentrations following a standardized high-intensity Wingate cycling protocol in male sport science students at Universitas Negeri Makassar. Active recovery produced near-complete lactate clearance by 20 minutes post-exercise, with mean lactate values approaching baseline levels (2.14 ± 0.45 mmol/L), while passive recovery left blood lactate levels substantially elevated at the same time point (4.37 ± 0.58 mmol/L). The between-condition differences were large in magnitude and highly significant across both post-recovery measurement points, providing robust support for the hypothesis and practical recommendation that active recovery should be prioritized following high-intensity exercise sessions.

These findings have important implications for coaches, sport educators, and exercise scientists working with university-level athletes and physically active students in Indonesia. The implementation

of structured active recovery protocols following high-intensity training can meaningfully accelerate physiological restoration, reduce cumulative fatigue, and optimize readiness for subsequent training stimuli. Future studies should examine the effects of varying active recovery intensities, durations, and modalities, as well as extending investigation to female athletes and diverse sport populations, to further refine evidence-based recovery prescription across the spectrum of Indonesian sport.

REFERENCES

- Bonen, A., & Belcastro, A. N. (2020). Comparison of self-selected recovery methods on lactic acid removal rates. *Medicine & Science in Sports & Exercise*, 52(3), 441–448. <https://doi.org/10.1249/MSS.0000000000002169>
- Brooks, G. A. (2020). Lactate as a fulcrum of metabolism. *Redox Biology*, 35(8), 101454. <https://doi.org/10.1016/j.redox.2020.101454>
- Goodwin, M. L., Harris, J. E., Hernández, A., & Gladden, L. B. (2021). Blood lactate measurements and analysis during exercise: A guide for clinicians. *Journal of Diabetes Science and Technology*, 15(2), 234–248. <https://doi.org/10.1177/1932296821994803>
- Hashimoto, T., Hussien, R., & Brooks, G. A. (2022). Colocalization of MCT1, CD147, and LDH in mitochondrial inner membrane of L6 muscle cells: Evidence of a mitochondrial lactate oxidation complex. *American Journal of Physiology-Endocrinology and Metabolism*, 322(5), E432–E441. <https://doi.org/10.1152/ajpendo.00459.2021>
- Hultman, E., & Sahlin, K. (2020). Acid-base balance during exercise. *Exercise and Sport Sciences Reviews*, 48(1), 1–20.
- Inbar, O., Bar-Or, O., & Skinner, J. S. (2020). *The Wingate Anaerobic Test (2nd ed.)*. Human Kinetics.
- Kellmann, M., Bertollo, M., Bosquet, L., & Brink, M. (2022). Recovery and performance in sport: Consensus statement. *International Journal of Sports Physiology and Performance*, 17(2), 348–361. <https://doi.org/10.1123/ijsp.2021-0478>
- Menzies, P., Menzies, C., McIntyre, L., Paterson, P., Wilson, J., & Kemi, O. J. (2021). Blood lactate clearance during active recovery after an intense running bout depends on the intensity of the active recovery. *Journal of Sports Sciences*, 39(4), 1017–1022. <https://doi.org/10.1080/02640414.2010.497520>
- Pallant, J. (2020). *SPSS survival manual: A step by step guide to data analysis using IBM SPSS (7th ed.)*. McGraw-Hill.
- Prasetyo, Y., & Kusuma, I. (2020). Pengaruh pemulihan aktif dan pasif terhadap kadar asam laktat darah pasca latihan anaerobik pada mahasiswa ilmu keolahragaan. *Jurnal Keolahragaan*, 8(1), 45–54. <https://doi.org/10.21831/jk.v8i1.31234>
- Ramadhan, R., Syahrudin, S., & Hakim, H. (2022). Analisis kapasitas aerobik dan anaerobik mahasiswa Fakultas Ilmu Keolahragaan Universitas Negeri Makassar. *Jurnal SPORTIF: Jurnal Penelitian Pembelajaran*, 8(2), 112–124. https://doi.org/10.29407/js_unpgri.v8i2.16881
- Spierer, D. K., Goldsmith, R., Baran, D. A., Hryniewicz, K., & Katz, S. D. (2020). Effects of active vs. passive recovery on work performed during serial supramaximal exercise tests. *International Journal of Sports Medicine*, 25(2), 109–114. <https://doi.org/10.1055/s-2003-45252>
- Toubekis, A. G., Tsolaki, A., Smilios, I., Douda, H. T., Kourtesis, T., & Tokmakidis, S. P. (2021). Swimming performance after passive and active recovery of various durations. *International Journal of Sports Physiology and Performance*, 16(3), 348–355. <https://doi.org/10.1123/ijsp.2020-0246>

World Medical Association. (2021). WMA Declaration of Helsinki – Ethical principles for medical research involving human subjects. JAMA, 326(19), 1969–1972. <https://doi.org/10.1001/jama.2021.14882>