INTERNATIONAL JOURNAL of IJSETS SPORT, EXERCISE & TRAINING SCIENCES

e-ISSN 2149-8229 **Volume** 11, **Issue** 3, 246–255, (2025) ***Research Article***

https://dergipark.org.tr/tr/pub/useeabd

Acute and Chronic Lower Limb Ischaemic Preconditioning Increase the Sprint Triathlon Performance in Athletes*

Mehmet Zeki SARI[1], Buğra GENÇTÜRK[2], Aliye GÜNDOĞDU[3], Dilek TOPAL[4], Sabriye ALTIN[5], Begüm ÖZALTUN OĞUL[6], Abdurrahman AKTOP[7], Selma CİVAR YAVUZ[8], Emel ÇETİN ÖZDOĞAN[9], Yaşar Gül ÖZKAYA[10]

Abstract

Aim: This study aimed to investigate the effects of ischemic preconditioning (IPC) on triathlon performance in athletes.
Method: Twenty-four triathletes (8 female, 16 males; mean age: 35.43±1.84 years), with at least 3 years of training and competition experience, participated. Heart rate (HR), blood pressure, rate of perceived exertion (RPE), respiratory frequency, and heart rate variability (HRV) parameters (SDNN, NN50, PNN50, RMSSD, VLF, LF, HF, LF/HF) were measured following rest, sham, and IPC conditions. IPC was applied both acutely (single session) and chronically (7 consecutive days) using a sphygmomanometer cuff inflated to 220 mmHg for 5 minutes, followed by 5 minutes of reperfusion, repeated four times. Sham involved the same protocol with 20 mmHg pressure. Data was analyzed using t-tests.
Results: Triathlon completion time significantly improved after both sham and IPC, but the improvement was greater after IPC ($p<0.05$). Performance enhancement was more pronounced with chronic IPC. Increased cycling performance largely contributed to the improvement in total triathlon time. Post-exercise heart rate was significantly lower following IPC compared to other conditions ($p<0.05$), while no other HRV parameters showed significant differences.
Conclusion: IPC significantly enhanced triathlon performance, especially when applied chronically, likely due to reduced heart rate after exercise. However, its effects on other HRV components were limited.

Key words: *Heart Rate Variability, İschemic Preconditioning, Triathlon.*

Submission Date : 13.06.2025 **Acceptance Date** : 25.09.2025 **Online Publication Date** : 30.09.2025
https://doi.org/10.18826/useeabd.1718989

INTRODUCTION

Ischemic preconditioning (IPC), originally described by Murry et al. in 1986, refers to a phenomenon where brief, repeated episodes of ischemia followed by reperfusion can protect muscle tissues against subsequent ischemic injury. Subsequent investigations have confirmed IPC's cardioprotective role against various cardiac pathologies, including arrhythmias, myocardial infarction, endothelial damage, and microvascular dysfunction (Hausenloy and Yellon, 2008; Veighey and MacAllister, 2012). Previous studies have shown that IPC increases oxygen uptake (Cruz et al. 2015; De Groot et al. 2010b), facilitates the removal of lactate from muscles (Bailey et al. 2012b), and reduces muscle fatigue (Barbosa et al. 2015) which are the parameters that significantly affect athletic performance.IPC appears to confer protection in two distinct phases: an early phase, occurring within minutes post-ischemia and lasting 1–3 hours, and a delayed or late phase that emerges approximately 24 hours after conditioning and may persist for up to 96 hours. While the early phase does not require protein synthesis, the later phase does (Schulz et al., 2001; Carroll and Yellon, 1999). Animal studies indicate that IPC enhances muscle perfusion through the upregulation of ATP-sensitive potassium channels and elevated adenosine levels within skeletal muscle (Riksen et al., 2006). This process facilitates oxygen delivery and potentially improves lactate transport and clearance during physical exertion (Brooks, 2000; Hashimoto and Brooks, 2008).

Additionally, IPC may improve muscle contraction efficiency and strength through improved excitation–contraction coupling (Pang et al., 1995). These changes might collectively enhance

[1] **Corresponding Author:** Akdeniz University, Institute of Health Sciences Department of Movement and Training, Türkiye, Mehmetzekisari8@gmail.com
[2] Akdeniz University, Institute of Health Sciences Department of Movement and Training, Türkiye
[3] Akdeniz University, Institute of Health Sciences Department of Movement and Training, Türkiye
[4] Akdeniz University, Institute of Health Sciences Department of Movement and Training, Türkiye
[5] Akdeniz University, Institute of Health Sciences Department of Movement and Training, Türkiye
[6] Akdeniz University, Institute of Health Sciences Department of Movement and Training, Türkiye
[7] Akdeniz University, Institute of Health Sciences Department of Movement and Training, Türkiye
[8] Akdeniz University Faculty of Sports Sciences, Department of Sports and Health Sciences, Türkiye
[9] Akdeniz University Faculty of Sports Sciences, Department of Sports and Health Sciences, Türkiye
[10] Akdeniz University Faculty of Sports Sciences, Department of Sports and Health Sciences, Türkiye
*University Scientific Research Projects Coordination Unit with project number TYL-2018-3604

mitochondrial capacity and optimize lactate metabolism, which can ultimately support improved athletic performance (Bailey et al., 2012). The standard IPC procedure typically involves three to four cycles of five-minute ischemia followed by reperfusion (De Groot et al., 2010a). As a non-invasive and cost-effective technique, IPC has drawn attention as a potential ergogenic aid for athletes. While some studies suggest IPC improves peak oxygen uptake and performance in cycling (Jean-St-Michel et al., 2011), others have questioned its efficacy in enhancing endurance or high-intensity exercise (Marocolo et al., 2016). Despite a growing body of research on IPC in various sports contexts, its specific effects on triathlon performance and underlying physiological mechanisms remain unclear. This study thus seeks to evaluate whether IPC affects triathlon performance and to assess changes in heart rate variability (HRV) metrics as a potential explanatory factor.

This study aimed to examine the effects of acute and chronic lower limb ischemic preconditioning (IPC) on sprint triathlon performance and selected physiological parameters in trained athletes. It was hypothesized that IPC would significantly reduce total triathlon completion time, particularly by improving cycling and running performance and would lead to favorable changes in heart rate and blood pressure. Additionally, it was expected that chronic IPC would result in greater improvements compared to acute IPC due to cumulative physiological adaptations. Heart rate variability (HRV) indices were also assessed as potential explanatory mechanisms underlying performance changes.

METHOD

Research model

This investigation was structured as a randomized, single-group pre-test – post-test design in accordance with the CONSORT standards. Prior to the commencement of the study, all participants were thoroughly informed about the procedures and risks, and each provided written consent in line with the ethical principles outlined in the Declaration of Helsinki.

Population and sample

The study cohort consisted of 27 athletes (9 females and 18 males), all of whom possessed a minimum of three years of active sports licensure and experience. The participants' average age was 36.66±7.38 years, and their mean body weight was 69.06±13.24 kg.

Data collection tools

Athletes were instructed to arrive at the testing facility following an overnight fast, having maintained their regular diet for 24 hours prior to testing. Body composition measurements such as body weight, % fat, total body water and fat free mass were taken for each participant prior to testing sessions. Body composition was assessed using a TANITA SC 330 ST (Tokyo, Japan) device. The triathlon performance test followed standardized protocols set by the International Triathlon Union and was conducted using a swimming pool (Akdeniz University), an indoor cycling setup (Canyon Aeroad CF Di2), and a treadmill for running.

Performance time was measured as the total duration from the start of the swimming leg to the end of the running segment, including both transitions (T1 and T2). The simulation followed the sprint triathlon format and consisted of a 750-meter swim, a 20-kilometer indoor cycling session, and a 5-kilometer treadmill run. Timing began when the participant entered the water and ended upon completing the final step on the treadmill. Transitions between disciplines were performed under competition-like conditions and included in the total time. A Polar V800 multisport GPS watch was used to collect performance time and physiological data, synchronized with manual stopwatch tracking and video confirmation to ensure accuracy. All sessions were conducted under standardized laboratory conditions using the same equipment for all participants.

Two weeks after the initial assessment, participants underwent both sham and IPC interventions, separated by a two-weeks interval. Additionally, chronic sham and IPC protocols were administered over one-week periods with two-weeks washout phases in between. After each intervention period, triathlon performance and HRV assessments were repeated.

IPC was delivered to both thighs using a sphygmomanometer set to 220 mmHg for 5 minutes of occlusion followed by 5 minutes of reperfusion, repeated over four cycles. In the sham condition, the

same timing protocol was followed, but with a cuff pressure of 20 mmHg. During the pre-test (resting) condition, no cuff pressure was applied (0 mmHg), and participants performed the triathlon simulation without any intervention.

Analysis of Heart Rate Variability

Heart rate variability (HRV) was analyzed using the standard version of Kubios HRV software. RR interval data, initially collected from each participant, were imported into Kubios for processing. The analysis yielded several time- and frequency-domain parameters, including mean heart rate (Mean HR), standard deviation of NN intervals (SDNN), root mean square of successive differences (RMSSD), as well as spectral components such as very low frequency (VLF), low frequency (LF), and high frequency (HF) power.

Data analysis

Statistical analysis of the collected data was conducted using paired t-tests to evaluate differences between pre- and post-intervention results. All values were expressed as mean ± standard deviation. A significance threshold of $p \leq 0.05$ was applied throughout the analyses.

RESULTS

Table 1. The descriptive characteristics of the participants

Variables	X± SD
Age (years)	36,66 ± 7,38
Height (cm)	173,45 ± 10,55
Body weight (kg)	69,06 ± 13,24
Fat (%)	12,36 ± 6,35
Total body water (%)	59,84 ± 3,91
Fat free mass (kg)	61,09 ± 13,09

The body composition results of the partiicipants were presented in Table 1. The descriptive characteristics of the participants are presented in Table 1. The athletes had a mean age of 36.66±7.38 years, an average body weight of 69.06±13.24 kg, and an average height of 173.45±10.55 cm. Their mean body fat percentage was 12.36±6.35%, while the mean fat-free mass was 61.09±13.09 kg. These data confirm that the study group consisted of trained, healthy individuals with low to moderate body fat levels and normal hydration status, supporting the validity of subsequent performance measurements.

Table 2. Heart rate (beats. min^{-1}), systolic and diastolic blood pressure (mmHg) and rate of perceived exertion (RPE) of the participants (mean ± SD)

Variables	Heart rate (min^{-1})	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	RPE
ACUTE	X± SD	X± SD	X± SD	X± SD
PRE- (0 mmHg)	66,60 ± 8,26	120,82 ± 12,73	77,04 ± 9,76	14,36 ± 1,18
SHAM (20 mmHg)	64,45 ± 7,16	113,78 ± 15,49	71,42 ± 10,58	14,61 ± 1,20
IPC (220 mmHg)	62,05 ± 11,14 *	110 ± 13,84 **	65,55 ± 8,75 ***##	14,83 ± 1,07 ***###
CHRONIC				
PRE- (0 mmHg)	61,76 ± 5,87	111,84 ± 7,75 ††	67,38 ± 7,62 †	14,36 ± 1,18
SHAM (20 mmHg)	60,44 ± 7,76	110,22 ± 14,10	64,88 ± 6,75 †	16,51 ± 0,29 **†††
IPC (220 mmHg)	60,77 ± 6,11	113,33 ± 18,02	58,88 ± 12,55 *	17,11 ± 0,60 ***†††

**p<0.05, **p<0.01, ***p<0.001, difference from (acute/chronic) pre-measurement of own group, ##p<0.01, ###p<0.001, difference from (acute/chronic) sham measurement of own group. †p<0.05, ††p<0.01, †††p<0.001, difference from the same measurement of acute application. IPC: Ischaemic preconditioning.*

The heart rate, blood pressure, and RPE (Rate of Perceived Exertion) scores obtained by the individuals participating in the study during acute and chronic applications are presented in Table 2. Acute IPC significantly decreased heart rate ($p=0.029$), systolic blood pressure ($p=0.006$), and diastolic blood pressure ($p<0.001$) compared to baseline. Chronic IPC also led to a significant reduction in diastolic blood pressure ($p<0.046$), although its impact on heart rate was less pronounced. Interestingly, RPE values significantly increased after chronic IPC ($p<0.001$), indicating that although performance improved, participants perceived the effort as more intense. These findings collectively demonstrate enhanced cardiovascular efficiency and altered perceptual response induced by IPC applications.

The heart rate, blood pressure and RPE scores obtained by the individuals participating in the study during acute and chronic applications are presented in Table 2. The mean pulse (min^{-1}) at rest was determined as 66.60±8.26 from acute measurements, and the pulse after IPC application was determined as 62.05±11.14. Accordingly, Acute IPC application significantly reduced heart rate (p=0.029), systolic blood pressure (p=0.006), and diastolic blood pressure (p=0.000) compared to pre-test values. Additionally, RPE scores significantly increased after chronic IPC (p<0.001), indicating a higher perceived exertion during improved performance. Accordingly, the systolic blood pressure at rest was determined as 120.82±12.73 mmHg from acute measurements, and the systolic blood pressure after IPC application was determined as 110.84±7.75 mmHg. Accordingly, it was seen that acute IPC reduced systolic blood pressure (p<0.006). Acute resting systolic blood pressure values were determined as 120.82±12.73 mmHg, and chronic resting value was determined as 111.84±7.75 mmHg. Accordingly, it was seen that chronic resting reduced systolic blood pressure (p<0.004). Among the acute measurements, the mean diastolic blood pressure at rest was determined as 77.04±9.76 mmHg, and the diastolic blood pressure after IPC application was determined as 65.55±8.75 mmHg. Accordingly, it was determined that acute IPC application reduced diastolic blood pressure (p<0.000). At the same time, diastolic blood pressure after sham application was determined as 71.42±10.58. According to these results, it was determined that acute IPC application decreased diastolic blood pressure compared to the data after sham application (p<0.001). On the other hand, diastolic blood pressure value taken at chronic rest was determined as 67.38±7.62 mmHg, and diastolic blood pressure after acute rest was determined as 77.04±9.76 mmHg. Accordingly, it was observed that it decreased diastolic blood pressure after chronic rest (p<0.020). Diastolic blood pressure was determined as 64.88±6.75 mmHg after chronic sham application. Accordingly, it was seen that chronic sham application decreased diastolic blood pressure compared to acute sham application (p<0.040). Diastolic blood pressure was determined as 58.88±12.55 mmHg after chronic IPC application. Accordingly, it was seen that chronic IPC application decreased diastolic blood pressure in its own group (p<0.046).

Table 3. Total triathlon times (sec) of the participants (mean ± SD)

	Variables	Total triathlon score (sec)
ACUTE	PRE (0 mmHg)	6326,304±1161,501
	SHAM (20 mmHg)	5934,409±980,362 **
	IPC (220 mmHg)	5790,5±1008,84***##
CHRONIC	PRE (0 mmHg)	6326,304±1161,501
	SHAM (20 mmHg)	5636,777±1251,997 *
	IPC (220 mmHg)	5118,777±1357,425 **##

**p<0.05, **p<0.01, ***p<0.001, difference from (acute/chronic) pre-measurement of own group, ##p<0.01, difference from (acute/chronic) sham measurement of own group. IPC: Ischaemic preconditioning.*

The total triathlon times (min and sec) obtained by the participants during acute and chronic applications are presented in Table 3. Both acute and chronic IPC significantly reduced total performance time compared to pre-intervention values (p<0,001 and p<0,01, respectively). Sham interventions also led to performance improvements, though to a lesser extent than IPC. The greatest performance enhancement was observed after chronic IPC, resulting in a reduction of approximately 1207 seconds (19%) from baseline. These findings highlight the consistent ergogenic effect of IPC, particularly when applied over multiple days.

Table 4. Swimming, cycling and running scores of the participants (sec) (mean ± SD)

		SWIMMING	BIKING	RUNNING
ACUTE	PRE (0 mmHg)	1075,30 ± 225,06	3954,30± 1027,82	1708,69 ± 272,00
	SHAM (20 mmHg)	1055,77 ± 225,86	2898,90 ± 684,43 ***	1671,72 ± 273,02 *
	IPC (220 mmHg)	1039,23 ± 196,83	2769,28 ± 664,32 ***##	1636,4 ± 283,77 **
CHRONIC	PRE (0 mmHg)	1075,30 ± 225,06	3954,30± 1027,82	1708,69 ± 272,00
	SHAM (20 mmHg)	1075,22 ± 309,71	2699,22 ± 701,87 **†	1626,11 ± 357,82
	IPC (220 mmHg)	1088,11 ± 315,62	2341,33 ± 749,20 **	1524,88 ± 325,92

**p<0.05, **p<0.01, ***p<0.001, difference from (acute/chronic) pre-measurement of own group, ##p<0.01, difference from (acute/chronic) sham measurement of own group, †p<0.05, difference from the same measurement of acute application.*

Table 4 shows the swimming, cycling and running times (sec) obtained by the participants during acute and chronic applications. Table 4 presents the segment-specific performance times in swimming, cycling, and running under different intervention conditions. IPC application significantly improved cycling and running times in both acute and chronic protocols (p <0,01), while swimming performance

remained unchanged across all conditions. The most pronounced improvements were observed in the cycling segment, suggesting that IPC primarily benefits activities involving sustained lower limb effort. These findings reinforce IPC's potential as a modality for enhancing endurance components that are physiologically more demanding.

Table 5. Parameters of heart rate variability of the participants (mean ± SD)

Variables	Pre-Sham	Post-Sham	Pre-IPC	Post-IPC
Heart rate (min^{-1})	61,12563	61,76229	61,21253	57,03335*#
Respiratory frequency (min^{-1})	15,00574	15,30988	15,69628	16,07218056
SDNN	76,0367976	79,3445119	101,9064	84,96605556
NN50	7,702381	8,809524	7,25	10,09722
PNN50	0,12644	0,146512	0,12835	0,178694
RMSSD	81,10613095	81,3757381	105,528875	95,91175
VLF	3227,94175	1183,880714	7550,9614	10196,69568
LF	5198,548	1225,623	16089,19	15111,68
HF	1589,628	602,7346	3870,945	2663,215
LF/HF	2,565738	2,4925595	3,028513	2,986278
Power	10016,11821	3012,238369	27511,09695	27971,5869

**p<0.05, difference from pre-sham measurement of own group, ##p<0.01, difference from pre-IPC measurement.*

The several parameters of HRV are presented in Table 5. Table 5 presents heart rate and HRV parameters obtained before and after sham and IPC interventions. While IPC significantly reduced heart rate ($p<0.05$), no statistically significant changes were observed in other HRV indices such as SDNN, RMSSD, LF, or HF. These findings suggest that IPC's effects on autonomic regulation may be limited or require longer-term adaptations to be detectable via HRV analysis. HRV parameters, including SDNN, RMSSD, LF, HF, and LF/HF ratio, are presented in Table 5. Although heart rate showed a significant decrease after IPC application ($p<0.05$), no significant changes were observed in the LF/HF ratio or other HRV indices across sham and IPC conditions ($p >0.05$). These findings suggest that IPC influenced cardiovascular efficiency as reflected by heart rate, but did not result in measurable changes in sympathovagal balance.

DISCUSSION

This study aimed to investigate the effects of acute and 7-days of ischemic preconditioning (IPC) on triathlon performance in individual athletes. The study results revealed that both acute and chronic IPC applications increased the triathlon performance and caused an improvement in especially biking performance. To our knowledge, this is the first study to examine the effect of IPC on the performance of triathlon athletes.

The IPC protocol used in the study was performed by 4 ischemia and reperfusion cycles applied via a sphygmomanometer connected bilaterally to the lower extremity, which is frequently used in literature. It is suggested that IPC increases exercise performance through neuronal, metabolic and vascular changes (Incognito et al., 2016; Caru et al., 2019). Of these changes, the neuronal pathway includes changes in the spinal cord, autonomic nervous system, and somatosensory system and is mainly activated by adenosine, bradykinin, and opiates (Pell et al., 1998; Schoemaker and van Heijningen, 2000; Liem et al., 2002; Patel et al., 2002; Weinbrenner et al., 2004; Hausenloy and Yellon, 2008; Przyklenk and Whittaker, 2011). It has been demonstrated that these 3 pathways have a protective effect by reducing ischemia-related damage and apoptosis in various organs of the body including the heart, liver, and muscles (Peralta et al., 2001; Giricz et al., 2014). It is known that IPC is used in the clinic to reduce ischemic damage to the heart after cardiac surgery or previous myocardial infarction (Chaturvedi et al. 1998; Yellon and Hausenloy, 2007). IPC is an easy-to-apply, cheap and side-effect-free method, and it has been shown that regional oxygen consumption increases during the bicycle test after IPC application in normal healthy individuals (Groot et al. 2009).

There are many studies in literature investigating the effects of IPC application on athletic performance. In studies investigating the effects of IPC on exercise performance, positive results have been reported regarding prolonging maximal exercise time, increasing maxVO2 or removing lactate from the muscles more rapidly (Incognito et al., 2016; Morocolo et al., 2016), while there are also studies indicating that it does not change exercise performance (Gibson et al., 2013). Jean-St-Michel et al. (2011) showed that the 100 m maximal swimming time was reduced by 0.7 s after acute IPC applied to the upper extremity

in swimmers compared to the group that did not apply IPC. On the other hand, Clevidence et al. (2012) reported that IPC applied before the submaximal bicycle test did not cause a statistically significant difference in aerobic and anaerobic performance parameters. Lalonde and Curnier (2015) showed that there was no statistical difference in the mean anaerobic power, peak power and maximal and average power values obtained per body weight in the Wingate bicycle test performed after IPC applied in the form of 5-minute ischemia and reperfusion cycles compared to the group that did not apply IPC. It is thought that the differences in the results obtained in the above-mentioned studies may be due to differences in the initial performance of the individuals participating in the study, differences in the muscle mass of the individuals or differences in the level of ischemia caused by the applied protocol.

Similar to the findings of the present study, Cruz et al. (2015) reported significant improvements in cycling performance after IPC, attributing these benefits to enhanced oxygen delivery and metabolic efficiency. Additionally, De Groot et al. (2010) demonstrated that repeated IPC application increased time to exhaustion in trained individuals. In contrast, Gibson et al. (2013) and Lalonde and Curnier (2015) found no significant performance benefit following IPC, which may be related to differences in protocol intensity, training level of participants, or the type of exercise modality used. These contradictory findings highlight the need for further research to clarify the potential moderators of IPC efficacy, such as ischemia pressure, occlusion site, training status, and timing of application relative to exercise.

The most obvious physiological mechanism of the effect of IPC on athletic performance parameters is that IPC increases muscle perfusion, oxygen uptake by the muscles, and muscle strength (Paradis-Deschenes et al., 2016). In this study, it was revealed that IPC application significantly shortened the total triathlon time in individuals who had been doing triathlon sports for at least 3 years. This amount was determined as approximately 536 seconds (8.5%) for acute IPC and approximately 1207 seconds (19%) for chronic IPC, and a statistically significant difference was found between the measurements. These results are quite high compared to studies showing that IPC increases the exercise time on the treadmill by approximately 1.5-2% (Bailey et al., 2002). On the other hand, the results of this study were shown to be closer to studies showing an increase in performance in cycling exercise performed using a fixed load after the application of IPC as approximately 15.8% (Cruz et al., 1985) and an increase in performance in cycling exercise performed against a gradually increasing load as approximately 7.9% (Kido et al., 2015). In our study, as a result of examining the effect of IPC on triathlon performance in more detail, it was determined that the performance increase was due to the effect on cycling and running in acute applications and on cycling performance in chronic applications. It was determined that IPC was ineffective on triathlon swimming performance.

When analyzed separately, the performance gains induced by IPC were most prominent in the cycling and running segments, while swimming performance remained relatively unaffected. This may be explained by differences in muscle mass involvement, postural orientation, and cardiovascular demand among triathlon disciplines. Cycling and running engage larger muscle groups of the lower limbs and allow for greater systemic oxygen delivery, which may benefit more directly from IPC-mediated improvements in muscle perfusion and mitochondrial efficiency (Paradis-Deschênes et al., 2016). In contrast, the swimming discipline-typically performed in a horizontal position with reduced gravitational stress-may have elicited less cardiovascular strain, thereby limiting the observable effects of IPC. Additionally, upper body musculature, which is more involved in swimming, was not directly targeted by the lower limb IPC protocol, possibly explaining the lack of performance change in this segment.

An interesting finding of the study is that both the total triathlon duration and the cycling and running duration were shortened after the sham application. This result may be due to the sham application creating an additional source of arousal/motivation in the athletes (Tocco et al., 2015), or it may be due to the desensitization of muscle afferents or subjective factors. On the other hand, in this study, it was determined that the athletes perceived these performances as more difficult, especially after the chronic sham and IPC application, when both the average triathlon performance and the Borg scores of the swimming, cycling and running sections of the triathlon were taken into account. This finding revealed that the athletes performed better under more challenging conditions - at least during the cycling performance. In this case, it would not be wrong to state that further studies are needed to review the

arousal levels, attention and motivation relationships of triathlon athletes, especially in terms of cycling performance. When the pulse and blood pressure parameters obtained in the study were examined, it was determined that acute IPC caused a significant decrease in heart rate, systolic, and diastolic blood pressure values. In addition, this study revealed that the state anxiety scores also decreased after acute IPC. The fact that these changes were not observed after sham applications provides strong evidence that IPC induces a genuine physiological response, rather than a placebo effect. Although these findings suggest increased parasympathetic nervous system activity, one of the limitations of the study is the absence of direct measurements such as HRV spectral analysis or plasma catecholamine levels to confirm this hypothesis. Nevertheless, the observed reduction in heart rate and blood pressure following IPC implies improved cardiovascular efficiency under physiological stress. These results suggest that IPC may enhance not only performance outcomes but also the body's ability to cope with exertional strain, which can be highly beneficial for endurance athletes. Although acute IPC significantly reduced heart rate, no statistically significant differences were observed in HRV parameters such as SDNN, RMSSD, LF, or HF. One possible explanation is that heart rate and HRV reflect different aspects of autonomic regulation. While HR can be immediately influenced by parasympathetic withdrawal or sympathetic inhibition, changes in HRV, particularly in time-domain and frequency-domain indices—require more sustained or pronounced autonomic shifts to become evident (Laborde et al., 2017). Moreover, HRV metrics are sensitive to both internal (hydration status, circadian rhythms, respiration) and external (measurement duration, artifact correction) factors, which may have contributed to variability in our measurements. It is also possible that the sample size limited statistical power to detect subtle changes in HRV, despite physiological relevance. One of the main limitations of this study is the lack of direct measurements of autonomic nervous system activity. Although HRV indices were collected and interpreted, no spectral analysis, baroreflex sensitivity, or biochemical markers (e.g., plasma catecholamines, salivary cortisol) were used to support conclusions about parasympathetic or sympathetic modulation. Furthermore, HRV measurements may be influenced by a variety of external factors such as respiratory patterns, circadian rhythms, and hydration status, which were not strictly controlled. These aspects limit the interpretation of the physiological mechanisms underlying IPC-related changes. These findings suggest that IPC not only improves performance but also modulates physiological strain responses such as heart rate and blood pressure, reflecting enhanced cardiovascular efficiency under stress. On the other hand, it has been shown that acute IPC (first 4 hours) increases regional bradykinin and adenosine secretion, thus stimulating both afferent fibers and increasing autonomic nervous system activation (Loukogeorgakis et al., 2005). Donato et al. (2003) demonstrated that vagal electrostimulation has a cardioprotective effect similar to remote IPC. These results are consistent with the findings regarding changes in pulse and blood pressure obtained in our study.

The limitations of this study should be mentioned. Initial aerobic and anaerobic power values of the athletes were not measured in the study. Also, subjective arousal / motivational factors that may affect the study results were neglected.

CONCLUSION

In conclusion, this study demonstrated that both acute and chronic lower limb ischemic preconditioning (IPC) significantly improved sprint triathlon performance in experienced athletes. Acute IPC reduced total triathlon time by approximately 536 seconds (8.5%), while chronic IPC led to a 1207-second (19%) reduction compared to baseline. These improvements were primarily due to enhanced cycling and running performance, with no significant changes in swimming times. Heart rate and blood pressure decreased significantly after IPC, indicating improved cardiovascular efficiency. However, HRV parameters did not show significant changes, which may suggest that IPC influences cardiovascular strain without significantly altering autonomic balance as measured by HRV indices. IPC, especially when applied repeatedly over one week, may serve as a safe and practical ergogenic aid for endurance athletes aiming to improve race performance.

SUGGESTIONS

From a practical perspective, the findings of this study suggest that IPC can serve as a non-invasive, low-cost, and time-efficient performance enhancement strategy for triathletes and endurance athletes. Acute IPC protocols may be used as part of pre-race warm-up routines to optimize cardiovascular

readiness, while chronic IPC protocols applied for several days leading up to competition may offer additional benefits through physiological adaptation. Coaches and sports scientists may consider integrating IPC into training microcycles, especially during tapering periods, to enhance cycling and running segments without adding physical load. The study provides important clues to trainers working in triathlon sports in creating and implementing training programs. More comprehensive studies need to be planned in the future to elucidate the physiological mechanisms of the results obtained in the study.

Etical Approval and Permission Information

Ethics Committee:	Akdeniz University Faculty of Medicine Clinical Research Ethics Committee
Protocol/Number:	7090450/157

REFERENCES

Bailey, T.G., Jones, H., Gregson, W., Atkinson, G., Cable, N.T., & Thijssen, D.H (2012). Effect of ischemic preconditioning on lactate accumulation and running performance. *Medicine & Science in Sports & Exercise,* 44(11), 2084-2089. https://doi.org/10.1249/mss.0b013e318262cb17

Barbosa TC, Machado AC, Braz ID, Fernandes IA, Vianna LC, Nobrega AC, Silva BM (2015). Remote ischemic preconditioning delays fatigue development during handgrip exercise. *Scandinavian Journal of Medicine & Science in Sports, 25*, 356–364. https://doi.org/10.1111/sms.12229

Brooks, G.A (2000). Intra-and extra-cellular lactate shuttles. *Medicine and science in sports and exercise*, 32(4), 790-799. https://doi.org/10.1097/00005768-200004000-00011

Carroll, R., & Yellon, D.M (1999). Myocardial adaptation to ischaemia–the preconditioning phenomenon. *International Journal Of Cardiology,* 68, S93-S101. https://doi.org/10.1016/S0167-5273(98)00297-6

Caru, M., Levesque, A., Lalonde, F., & Curnier, D. (2019). An overview of ischemic preconditioning in exercise performance: A systematic review. *Journal of sport and health science*, *8*(4), 355-369. https://doi.org/10.1016/j.jshs.2019.01.008

Chaturvedi, R. R., Lincoln, C., Gothard, J. W., Scallan, M. H., White, P. A., Redington, A. N., & Shore, D. F. (1998). Left ventricular dysfunction after open repair of simple congenital heart defects in infants and children: quantitation with the use of a conductance catheter immediately after bypass. *The Journal of Thoracic and Cardiovascular Surgery*, *115*(1), 77-83. https://doi.org/10.1016/S0022-5223(98)70446-5

Clevidence, M. W., Mowery, R. E., & Kushnick, M. R. (2012). The effects of ischemic preconditioning on aerobic and anaerobic variables associated with submaximal cycling performance. *European Journal of Applied Physiology*, *112*, 3649-3654. https://doi.org/10.1007/s00421-012-2345-5

Cooper, C.E., & Brown, G.C (2008). The inhibition of mitochondrial cytochrome oxidase by the gases carbon monoxide, nitric oxide, hydrogen cyanide and hydrogen sulfide: chemical mechanism and physiological significance. *Journal of Bioenergetics and Biomembranes*, 40(5), 533. https://doi.org/10.1007/s10863-008-9166-6

Cruz, R. S. D. O., De Aguiar, R. A., Turnes, T., Pereira, K. L., & Caputo, F. (2015). Effects of ischemic preconditioning on maximal constant-load cycling performance. *Journal of Applied Physiology*, *119*(9), 961-967. https://doi.org/10.1152/japplphysiol.00498.2015

De Groot, P. C., Thijssen, D. H., Sanchez, M., Ellenkamp, R., & Hopman, M. T. (2010). Ischemic preconditioning improves maximal performance in humans. *European journal of applied physiology*, *108*, 141-146. https://doi.org/10.1007/s00421-009-1195-2

Donato, M., Buchholz, B., Rodríguez, M., Pérez, V., Inserte, J., García-Dorado, D., & Gelpi, R. J. (2013). Role of the parasympathetic nervous system in cardioprotection by remote hindlimb ischaemic preconditioning. *Experimental Physiology*, *98*(2), 425-434. https://doi.org/10.1113/expphysiol.2012.066217

Gibson, N., White, J., Neish, M., & Murray, A. (2013). Effect of ischemic preconditioning on land-based sprinting in team-sport athletes. *International Journal of Sports Physiology and Performance*, *8*(6), 671-676.

Giricz, Z., Varga, Z. V., Baranyai, T., Sipos, P., Pálóczi, K., Kittel, Á., ... & Ferdinandy, P. (2014). Cardioprotection by remote ischemic preconditioning of the rat heart is mediated by extracellular

vesicles. *Journal of Molecular and Cellular Cardiology*, *68*, 75-78. https://doi.org/10.1016/j.yjmcc.2014.01.004

Hashimoto, T., & Brooks, G. A. (2008). Mitochondrial lactate oxidation complex and an adaptive role for lactate production. *Medicine & Science in Sports & Exercise*, *40*(3), 486-494. https://doi.org/10.1249/mss.0b013e31815fcb04

Hausenloy, D.J., Yellon, D.M (2008). Remote ischaemic preconditioning: underlying mechanisms and clinical application. *Cardiovascular Research*, 79, 377–86. https://doi.org/10.1093/cvr/cvn114

Incognito, A. V., Burr, J. F., & Millar, P. J. (2016). The effects of ischemic preconditioning on human exercise performance. *Sports Medicine*, *46*, 531-544. https://doi.org/10.1007/s40279-015-0433-5

Jean-St-Michel, E., Manlhiot, C., Li, J., Tropak, M., Michelsen, M. M., Schmidt, M. R., ... & Redington, A. N. (2011). Remote preconditioning improves maximal performance in highly trained athletes. *Medicine and Science in Sports and Exercise*, *43*(7), 1280-1286.https://doi.org/10.1249/mss.0b013e318206845d

Kido, K., Suga, T., Tanaka, D., Honjo, T., Homma, T., Fujita, S., ... & Isaka, T. (2015). Ischemic preconditioning accelerates muscle deoxygenation dynamics and enhances exercise endurance during the work-to-work test. *Physiological Reports*, *3*(5), e12395. https://doi.org/10.14814/phy2.12395

Kilduff, L. P., Finn, C. V., Baker, J. S., Cook, C. J., & West, D. J. (2013). Preconditioning strategies to enhance physical performance on the day of competition. *International Journal of Sports Physiology and Performance*, *8*(6), 677-681.https://doi.org/10.1123/ijspp.8.6.677

Kimura, M., Ueda, K., Goto, C., Jitsuiki, D., Nishioka, K., Umemura, T., ... & Higashi, Y. (2007). Repetition of ischemic preconditioning augments endothelium-dependent vasodilation in humans: role of endothelium-derived nitric oxide and endothelial progenitor cells. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *27*(6), 1403-1410. https://doi.org/10.1161/ATVBAHA.107.143578

Lalonde, F., & Curnier, D.Y (2015). Can anaerobic performance be improved by remote ischemic preconditioning. *The Journal of Strength & Conditioning Research*, 29(1), 80-85. https://doi.org/10.1519/JSC.0000000000000609

Lawson, C. S., & Downey, J. M. (1993). Preconditioning: state of the art myocardial protection. *Cardiovascular Research*, *27*(4), 542-550. https://doi.org/10.1093/cvr/27.4.542

Loukogeorgakis, S. P., Panagiotidou, A. T., Broadhead, M. W., Donald, A., Deanfield, J. E., & MacAllister, R. J. (2005). Remote ischemic preconditioning provides early and late protection against endothelial ischemia-reperfusion injury in humans: Role of the autonomic nervous system. *Journal of the American College of Cardiology*, *46*(3), 450-456. https://www.jacc.org/doi/abs/10.1016/j.jacc.2005.04.044

Marocolo, M., da Mota, G. R., Simim, M. A. M., & Coriolano, H. J. A. (2016). Myths and facts about the effects of ischemic preconditioning on performance. *International Journal of Sports Medicine*, *37*(02), 87-96. https://doi.org/10.1055/s-0035-1564253

Murry, C. E., Jennings, R. B., & Reimer, K. A. (1986). Preconditioning with ischemia: A delay of lethal cell injury in ischemic myocardium. *Circulation*, *74*(5), 1124-1136. https://doi.org/10.1161/01.CIR.74.5.1124

Ozkaya, Y. G., Agar, A., Hacioglu, G. Ö. K. Ç. E., Yargicoglu, P., Abidin, I., & Senturk, U. K. (2003). Training induced alterations of visual evoked potentials are not related to body temperature. *International Journal of Sports Medicine*, *24*(05), 359-362. https://doi.org/10.1055/s-2003-40699

Pang, C. Y., Neligan, P., Xu, H., He, W., Zhong, A., Hopper, R., & Forrest, C. R. (1997). Role of ATP-sensitive K+ channels in ischemic preconditioning of skeletal muscle against infarction. *American Journal of Physiology-Heart and Circulatory Physiology*, *273*(1), H44-H51.https://doi.org/10.1152/ajpheart.1997.273.1.H44

Pang, C. Y., Yang, R. Z., Zhong, A., Xu, N., Boyd, B., & Forrest, C. R. (1995). Acute ischaemic preconditioning protects against skeletal muscle infarction in the pig. *Cardiovascular Research*, *29*(6), 782-788. https://doi.org/10.1016/S0008-6363(96)88613-5

Paradis-Deschênes, P., Joanisse, D. R., & Billaut, F. (2016). Ischemic preconditioning increases muscle perfusion, oxygen uptake, and force in strength-trained athletes. *Applied Physiology, Nutrition, And Metabolism*, *41*(9), 938-944. https://doi.org/10.1139/apnm-2015-0561

Patel, H. H., Moore, J., Hsu, A. K., & Gross, G. J. (2002). Cardioprotection at a distance: mesenteric artery occlusion protects the myocardium via an opioid sensitive mechanism. *Journal of Molecular and Cellular Cardiology*, *34*(10), 1317-1323. https://doi.org/10.1006/jmcc.2002.2072

Pell, T. J., Baxter, G. F., Yellon, D. M., & Drew, G. M. (1998). Renal ischemia preconditions myocardium: role of adenosine receptors and ATP-sensitive potassium channels. *American Journal of Physiology-Heart and Circulatory Physiology*, *275*(5), H1542-H1547. https://doi.org/10.1152/ajpheart.1998.275.5.H1542

Peralta, C., Fernández, L., Panés, J., Prats, N., Sans, M., Piqué, J. M., ... & Roselló-Catafau, J. (2001). Preconditioning protects against systemic disorders associated with hepatic ischemia-reperfusion through blockade of tumor necrosis factor–induced P-selectin up-regulation in the rat. *Hepatology*, *33*(1), 100-113. https://doi.org/10.1053/jhep.2001.20529

Przyklenk, K., & Whittaker, P. (2011). Remote ischemic preconditioning: current knowledge, unresolved questions, and future priorities. *Journal of Cardiovascular Pharmacology and Therapeutics,* 16, 255–9. https://doi.org/10.1177/1074248411409040

Riksen, N.P., Smits, P., & Rongen, G.A. (2006). Ischaemic preconditioning: From molecular characterisation to clinical application-part I. *The Netherlands Journal of Medicine*, 62(10), 353-63.

Schoemaker, R.G., & Van Heijningen, C.L. (2000). Bradykinin mediates cardiac preconditioning at a distance. *Am American Journal of Physiology,* 278, H1571–6. https://doi.org/10.1152/ajpheart.2000.278.5.H1571

Schulz, R., Cohen, M.V., Behrends, M., Downey, J.M., & Heusch, G. (2001). Signal transduction of ischemic preconditioning. *Cardiovascular Research*, 52(2), 181-198. https://doi.org/10.1016/S0008-6363(01)00384-4

Tocco, F., Marongiu, E., Ghiani, G., Sanna, I., Palazzolo, G., Olla, S., ... & Crisafulli, A. (2015). Muscle ischemic preconditioning does not improve performance during self-paced exercise. *International Journal of Sports Medicine*, *36*(01), 9-15. https://doi.org/10.1055/s-0034-1384546

Veighey, K., & MacAllister, R. J. (2012). Clinical applications of remote ischemic preconditioning. *Cardiology Research and Practice*, *2012*(1), 620681. https://doi.org/10.1155/2012/620681

Weinbrenner, C., Schulze, F., Sárváry, L., & Strasser, R. H. (2004). Remote preconditioning by infrarenal aortic occlusion is operative via δ1-opioid receptors and free radicals in vivo in the rat heart. *Cardiovascular Research*, *61*(3), 591-599. https://doi.org/10.1016/j.cardiores.2003.10.008

Yellon, D. M., & Hausenloy, D. J. (2007). Myocardial reperfusion injury. *New England Journal of Medicine*, *357*(11), 1121-1135. https://doi.org/10.1056/NEJMra071667

Ylitalo, K., & Peuhkurinen, K. (2001). Clinical relevance of ischemic preconditioning. *Scandinavian Cardiovascular Journal*, *35*(6), 359-365. https://doi.org/10.1080/14017430152754835

CITATION

Sarı, M.Z., Gençtürk, B., Gündoğdu, A., Topal, D., Altın, S., Özaltun Oğul, B., Aktop, A., Civar Yavuz, S., Çetin Özdoğan, E., & Yaşar Gül Özkaya & Özkaya, Y.G. (2025). Acute and chronic lower limb ischaemic preconditioning increase the sprint triathlon performance in athletes. *International Journal of Sport Exercise and Training Sciences - IJSETS, 11*(3), 246-255. https://doi.org/10.18826/useeabd.1718989