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ORIGINAL ARTICLE

The Relationship of Blood Lactate Level and Swimming Performance with MCT1 after Critical Velocity

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ABSTRACT

Background. The investigation into whether the MCT1 gene in swimming and the inclusion of exercises involving critical velocity in water warm-ups have an effect on performance is ongoing. **Objectives.** This study aims to examine the relationship between swimming performance and blood lactate level with MCT1 after critical velocity. **Methods.** 33 girls and 27 boys were included in the study. Intraoral swab and intraoral buccal swap samples were taken from the participants to be used for MCT1 gene analysis by the expert with transport swab, which can be used in DNA analysis. After DNA isolations, MCT1 gene polymorphisms were performed with Real-time PCR. After resting, the resting blood lactate levels (LArest) were taken from the fingertips with Lactate Scout+. Then, the swimmers performed a water warm-up with critical velocity and were taken to the 4 repetitions of the 50-meter (m) individual medley maximal swimming test. Immediately after the test, the blood lactate levels were taken at the 1st (LA1), 6th (LA6), and 15th (LA15) minute rest periods. The swimming test was recorded with a SJCAM camera. The end time was determined with Kinovea 0.9.5. IBM SPSS 24.0 program was used for data analysis. The relationship between blood lactate and swimming performance vs MCT1 dominant genotype and allele distribution was analyzed by Pearson correlation. The significance level was taken as $p<0.05$. **Results.** No significant differences were found between LArest and performance vs MCT1 ($p>0.05$). A significant correlation was found between LA1, LA6, LA15 values vs MCT1 ($p<0.05$). **Conclusion.** This study confirmed that there is a relationship between the MCT1 dominant genotype and blood lactate levels in swimmers, but this relationship with performance has not been confirmed.

KEYWORDS: ***MCT1, Swimming, Critical Velocity, Performance, Blood Lactate.***

INTRODUCTION

Monocarboxylate (lactate / pyruvate) transporter (MCT1) is located on the short arm of chromosome 1 in the gene family defined as a carrier protein and SLC16, which is known to consist of 14 members in mammals. It causes displacement of aspartic acid and glutamic acid at codon 490 (Glu490Asp). MCT1; proton-coupled carrier proteins that transport monocarboxyl lactic acid, purivic acid, and ketone (1). MCT1 works with LDH and cytochrome oxidase to oxidize lactate in respiratory cells (2). Therefore, it can be said that because lactate is oxidized, it is reduced in the body. Taken together, lactate works extensively in skeletal muscles, heart, brain, kidneys, liver, and other organs (3). Regulation of lactic acid metabolism with exercise is a physiological and biochemical process involving multiple organs and nervous, exercise, circulatory, respiratory, digestive, and endocrine systems (4).

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Lactate during exercise is transported across the plasma membrane via a cell-cell lactate shuttle, facilitated by monocarboxylate transporters (MCTs) that are bound to pH, membrane, and proton, respectively (5). Lactate transporters provide the largest part of muscle H+ removal during high-intensity exercise. In addition, other proteins enhance the transport activity of MCT1 and muscle pH regulation (6). In high-intensity exercise, the increase in skeletal muscle energy level is associated with the accumulation of lactate proton anions and an increased rate of glycolysis. This accumulation is regulated by MCT1 and MCT4 (7). It is stated that MCT1 is associated with athletic performance because it has high activity in oxidative type 1 muscle fibers and provides lactate entry to myocytes in red skeletal muscle for oxidation. We can say that this directly proportional relationship is explained by the decrease in gene expression and muscle inactivation (1, 8, 9). When lactate in fast-twitch fibers is used as an energy source for slow-twitch fibers, lactate moves through the MCT1 protein. When lactate passes into the blood using the MCT4 protein, lactate circulates throughout the body, while lactate moves to the liver via the MCT2 protein and is stored as glycogen during the Cori cycle. In the heart, lactate passes through the blood to the mitochondria and is used as energy (3, 10). A large amount of blood lactate enters the brain via the BBB (blood-brain barrier) via MCT1. Lactate is produced in astrocytes by glycolysis and can be transported out of cells by MCT4 and MCT1 (11). When lactate mechanics is examined in high-intensity studies, some studies have shown that increased MCT protein content (12, 13) and improvements in mitochondrial morphology with stimulation of mitochondrial biogenesis (14, 15) reported that it has a positive effect on metabolism, for example, Larsen et al., Layec et al., obtained contradictory results in their studies (16, 17). In addition, there are studies showing that high blood lactate concentration and related changes in gas exchange kinetics after priming exercises with 20%, 70% and 140% VO_{2max} does not improve exercise performance (18, 19). In the research examined, intercellular lactate mechanics (intracellular input and output of lactate) were reported by decreasing lactate production and increasing MCT1 synthesis in muscle (20). Metabolic alkalosis is known to cause an increase in several lactate/pH-regulating proteins after exercise, and acidosis has been shown to have an unexpected role in reducing exercise-induced mitochondrial respiratory loss in the short term (21). With this information, Lee et al. emphasized that lactate is now used to validate exercise performance, and further studies are needed to evaluate its effect on exercise training (11).

The main concern of this study is whether there is a relationship between performance and lactate vs MCT1 in swimmers. This study was designed because the MCT1 gene in swimmers is under-studied and there is not enough information in the literature to determine its relationship with performance. Critical velocity, which was proved by Neiva et al., (22) that it can be applied in terms of both improving the race performance and delaying the fatigue of the swimmers before the competition, was preferred as water warm-up. In this context, the aim of the study is to examine the relationship between swimming performance and blood lactate level vs MCT1 after critical velocity warm-up.

MATERIALS AND METHODS

Participants. While forming the research group, the desired characteristics were specified: 1) to continue active swimming life in a private club, 2) to do swimming sports for at least 3 years, 3) to be in the youth category at the national level, 4) to be between the ages of 12-15. A power analysis was performed by adhering to the inclusion criteria of the study. Based on these criteria and the ability to detect a change of at least 20% with a significance of 0.05 and a power of 0.95, the required number of participants was 51. To unforeseen circumstances we aimed to recruit 20% more participants than needed; hence, the sample size was increased to n=60. This sample size is consistent with many studies investigating the effects of exercise in humans (23). In line with the number reached by these calculations, 33 girls (age: 13.33±0.92 years; height 161.32±6.41 cm; body mass: 51.42±8.50 kg) and 27 boys (age: 13.19±1.04 years; height 161.63±10.98 cm; body mass: 50.27±10.96 kg) swimmers participated. The research was approved by the Istanbul University Clinical Research Ethics Committee for human research in accordance with the 2013 Declaration of Helsinki. Participants and their families were informed about the research-related goals, practical details, and possible risks and signed a

written informed consent form to participate in the study (Ethics Committee on 05.03.2021-Code:06). All swimmers in the study agreed to participate in the study on a voluntary basis. Since swimmers under the age of 18 are working, their families signed the informed consent forms prepared for the research without being under any influence. This work was funded by the Scientific Research Projects Unit of Haliç University. Project Number: HBAP-III-3.

Procedures. Initially, intraoral epithelial cell samples were taken by the expert for MCT1 gene analysis of swimmers. At this stage, blood samples were not taken from the athletes, instead, an intraoral swab was taken with a medium-free swab that could be used for DNA analysis. This process was carried out by the principal investigator. During the study, intraoral buccal swab samples were taken from the volunteers. Since it is disposable, there was no risk of microbiological contamination from the swab tubes to the volunteers. The swimmers were given anonymous codes representing the individual in the study. Thus, the athlete is protected from possible psychological and educational/sports life situations.

At the end of the process, all samples were collected and carried to the laboratory by the assistant researcher with a bag containing ice packs. DNA isolation of the samples coming to the laboratory was carried out on the same day. After DNA isolations, MCT1 gene polymorphisms (rs1049434) were performed and analyzed by evaluating data obtained from Real-time PCR (Life Technologies, Carlsbad, CA, USA) (24).

Swab samples were used for DNA isolation. Optimization continued until DNA concentrations reached above 50 ng/µl and the measurement at OD 260/280 nm in the range of 1.6-2.0 from all samples were isolated with the same protocol. In addition, with the agarose gel electrophoresis method, the DNAs were exposed to the electrical field in the agarose gel, which shows that isolation is successful and can be seen visually. The DNAs obtained in the desired amount and purity were stored at -20°C.

At the beginning of the study, first of all, primer-probe sets were scanned from the literature, targeting the SNP on the gene determined to have an effect on susceptibility in terms of sportive performance, 1 pair of primers for wild genome which will amplify, as well as 1 advanced set primers to amplify the mutated target for each of these SNPs or back primer designed to meet primer design conditions and requirements. Relevant gene regions in genomic DNA samples were amplified by PCR. For amplification of each of the DNA samples, the PCR conditions are: 2xqPCR buffer (10 mM Tris-HCl, 50 mM KCl, 1.75 mM MgCl2) was mixed with 2.5 mM dNTP, 0.1 unit Taq DNA polymerase and specific primers for the SNP's of gene region (MCT1). A total of 10 µl PCR mix containing 100 pmol/µl and 500 ng DNA from each primer set was prepared for use in Real-Time PCR. Melting peaks were evaluated in order to check whether the PCR products of the desired regions of the MCT1 gene were formed. Accordingly, it was determined whether the desired PCR products were formed in all studies.

After the intraoral epithelial cell samples of the swimmers were taken, sufficient time was given for complete rest, and resting blood lactate levels (LArest) were taken with a Lactate Scout+ (LS, SensLab GmbH, Germany) lactate analyzer. Before the test, the swimmers performed the in-water warm-up which had a total distance of 1200 m and included critical velocity in their own lanes (22). The warm-up protocol applied in Table 1 is given in detail. The swimmers who completed this warm-up were given 2 minutes of passive rest before taking the test. As a test, the swimmers performed the 200-m maximal individually medley broken test (butterfly, backstroke, breaststroke, and freestyle, respectively) in 4 repetitions of 50-m. It was paid attention to reach a total of 200-m medley swimming performance time as a result of the test results, which is up to 105% more than the best times of the swimmers. A rest interval of 1:2 is given for every 50-m. Immediately after the test, the blood lactate levels of the swimmers were taken with the Lactate Scout+ at the 1st (LA1), 6th (LA6), and 15th (LA15) minute rest periods. The swimming test was recorded with a SJCAM external camera and the images were transferred to the Kinovea 0.9.5 video analysis program. With this program, the end time was calculated for each swimmer and 50-m. Figure 1 provides a schematic representation of the study design and test procedures used.

Statistical analysis. IBM SPSS 24.0 (IBM Corp. Released 2016. IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp.) program was used for data analysis in the

research. In the study, the compliance of the data with the normal distribution was determined by the Skewness and Kurtosis values. Skewness and Kurtosis values were found to be between "-2.0" and "+2.0", and it was accepted that the data showed a normal distribution in this direction (25). Genotype distributions were in Hardy–Weinberg equilibrium, being 45% wildtype (A/A), 40% heterozygotes (T/A), and 15% mutated homozygotes (T/T). The relationship between blood lactate and swimming performance vs MCT1 dominant genotype and allele distribution was analyzed by Pearson correlation. The significance level was taken as $p<0.05$.

Table 1. The warm-up protocol

Number	Warm-up task
1	300 m (100 m usual breathing, 100 m breathing in the fifth stroke, 100 m usual breathing)
2	4 x 100 m on 1:50 (2 x [25 m kick + 25 m increased stroke length])
3	8 x 50 m on 1:00 (98– 102% of critical velocity)
4	100 m (easy swim)

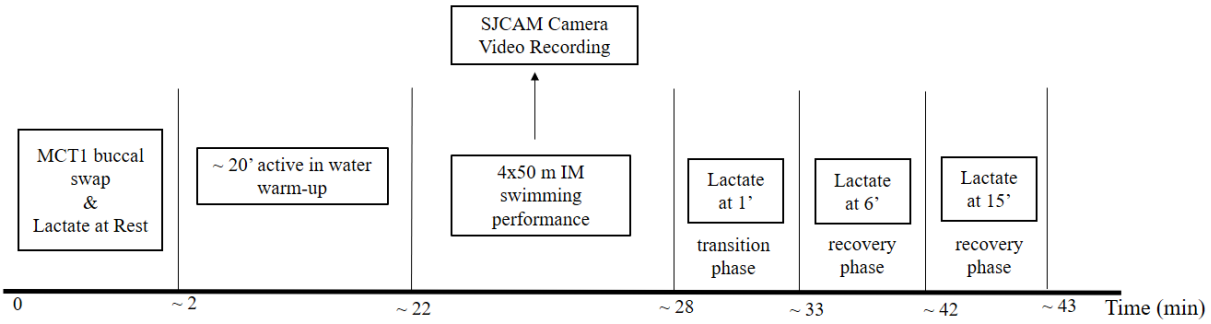

Figure 1. Study design diagram.

RESULTS

The relationships of the lactate values of the swimmers with the mean and standard deviation values according to the MCT1 dominant genotype are given above in table (Table 2) and figure (Figure 2). According to the table and figure, no significant relationship was found between the LArest values of each model and MCT1 ($p>0.05$). However, a significant difference was found between LA1, LA6, and LA15 values and MCT1 ($p<0.05$).

The relationships of the swimmers' performances with the mean and standard deviation values according to the MCT1 dominant genotype are given above in table (Table 2) and figure (Figure 2). As a result of the analysis, it was found that there was no difference in swimming performance according to the MCT1 dominant genotype ($p>0.05$).

DISCUSSION

In recent years, researchers working on athletic performance and genetic polymorphism have been wondering about the relationship between the MCT1 gene and performance and the MCT1 allele frequency distributions of athletes. It is known that MCT1 increases in oxidative muscle fibers during exercise and this situation is associated with lactate carrying capacity. Merezhinskaya et al. reported that individuals with the T allele in the MCT1 gene have lower lactate-carrying capacity than others (1). In another study confirming this result, it was stated that lactate uptake in cells was higher in the A allele (26).

In the literature, it has been observed that the MCT1 rs1049434 polymorphism has been investigated in triathlon, sprint/power, endurance, and runners. In long-distance runners, 4 people were homozygous AA, 8 people were heterozygous AT, and 3 people were homozygous TT; In short-distance runners, 14 people were found to be heterozygous AT and 1 person to be homozygous TT (27). In sprint/power and endurance athletes, the values for TT were 9 and 6, for AT were 15 and 17, and for AA were 7 and 6, respectively (28). While the TT genotype was not found in 10 elite male Turkish Ironman

athletes, 9 individuals had AA genotype and 1 person had AT genotype (29). In this study, 16 of the female swimmers were AA, 11 were AT, and 6 were TT; 11 of the male swimmers had the AA genotype, 13 had the AT genotype, and 3 had the TT genotype. It was found that 45% of the research group was AA, 40% AT, and 15% TT genotype.

Table 2. MCT1 association with the lactate and swimming performance (Mean±SE)

Model		LArest (mmol/L)	p	r	LA1 (mmol/L)	p	r	LA6 (mmol/L)	p	r	LA15 (mmol/L)	p	r
Co-dominant	AA (n=27)	1.15±0.31			7. 20±1.44			6.10±1.35			4.29±1.22		
	AT (n=24)	1.19±0.32	0.194	0.170	8.59±2.50	0.001*	0.470	7.00±1.88	0.001*	0.504	5.33±1.68	0.001*	0.523
	TT (n=9)	1.32±0.38			10.12±1.80			8.91±1.45			6.92±1.74		
A Allel (AA+AT)	AA+AT (n=51)	1.17±0.31	0.194	0.170	7.86±2.11	0.004*	0.369	6.52±1.67	0.001*	0.467	4.78±1.53	0.001*	0.446
	TT (n=9)	1.32±0.38			10.12±1.80			8.91±1.45			6.92±1.74		
T Allel (AT+TT)	AA (n=27)	1.15±0.31	0.353	0.122	7.20±1.44	0.001*	0.410	6.10±1.35	0.002*	0.388	4.29±1.22	0.001*	0.430
	AT+TT (n=33)	1.23±0.34			9.01±2.40			7.52±1.16			5.77±1.81		

Model		Butterfly (s)	p	r	Backstroke (s)	p	r	Breaststroke (s)	p	r	Freestyle (s)	p	r
Co-dominant	AA (n=27)	39.66±4.53			42.56±2.92			47.89±3.87			37.18±2.57		
	AT (n=24)	39.56±4.95	0.313	-0.132	42.18±2.80	0.161	-0.183	47.69±4.38	0.327	-0.129	36.73±2.92	0.326	-0.129
	TT (n=9)	37.55±3.16			40.92±2.43			46.12±3.85			36.24±1.80		
A Allel (AA+AT)	AA+AT (n=51)	39.62±4.69	0.209	-0.165	42.38±2.84	0.152	-0.187	47.79±4.08	0.257	-0.149	36.97±2.72	0.443	-0.101
	TT (n=9)	37.55±3.16			40.92±2.43			46.12±3.85			36.24±1.80		
T Allel (AT+TT)	AA (n=27)	39.66±4.53	0.585	-0.072	42.56±2.92	0.328	-0.128	47.89±3.87	0.552	-0.078	37.18±2.57	0.391	-0.113
	AT+TT (n=33)	39.01±4.58			41.84±2.73			47.26±4.24			36.59±2.64		

*: p<0.05; *AA:* wildtype; *AT:* heterozygotes; *TT:* mutated homozygotes.

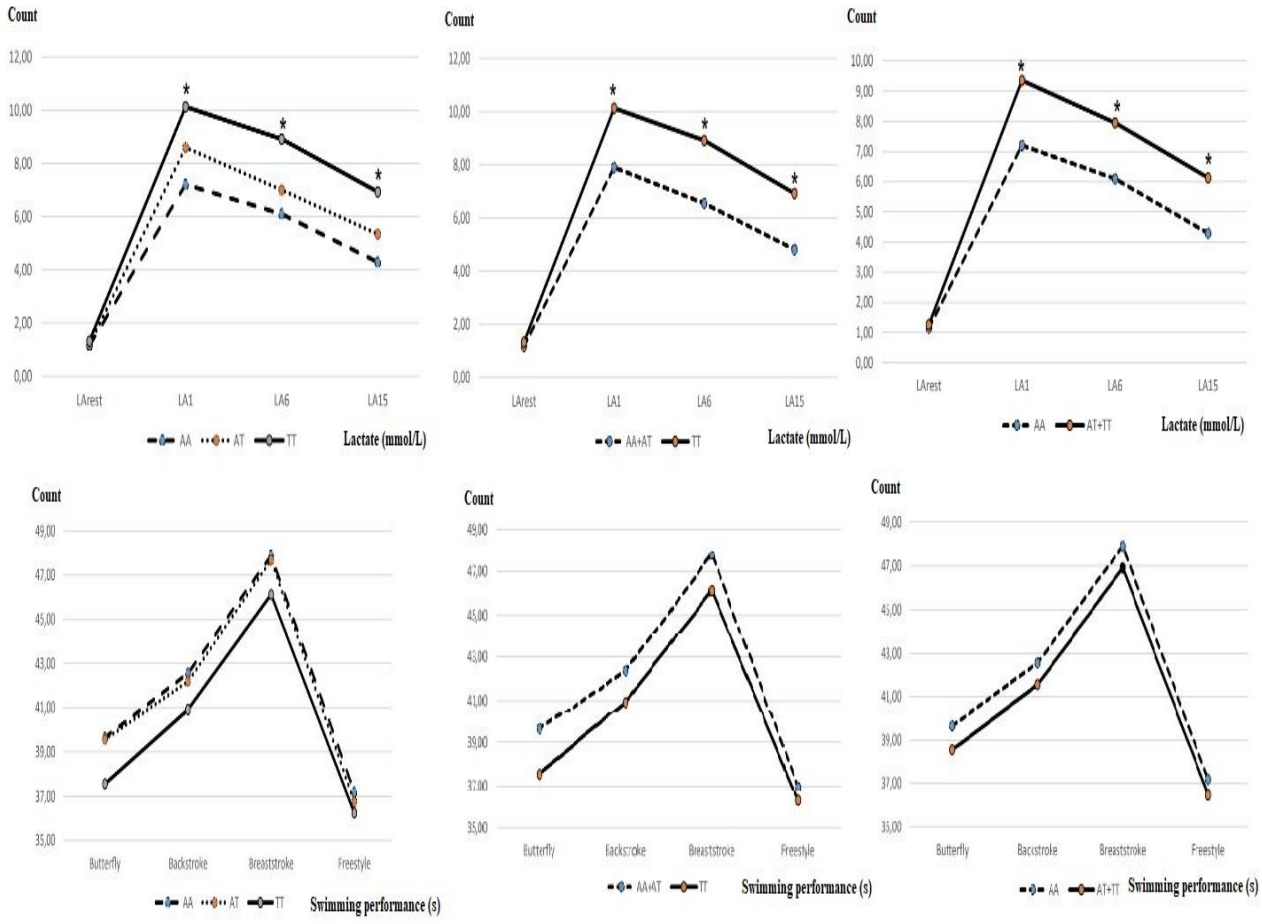

Figure 2. MCT1 association with the lactate and swimming performance.
*: p<0.05; AA: wildtype; AT: heterozygotes; TT: mutated homozygotes.

According to Subak et al., (2017) who cited the compilation study of Ahmetov and Fedotovskaya (2015) on sports genetics in the literature, a study was conducted to determine the positive relationship between MCT1 and athletic performance in endurance and strength athletes, while in two studies it was stated that this relationship was negative or controversial (5, 30). Cupeiro et al.'s study to examine the effect of post-training lactate accumulation of the MCT1 Glu490Asp polymorphism found that those carrying the T allele showed higher lactate accumulation (31). This result was confirmed by an increased fatigue test on the rowing ergometer in a study with Russian rowers, emphasizing the importance of a high A allele in endurance sports (8). However, the same results were not found in Polish and Israeli endurance athletes (32, 33). When endurance and strength athletes were compared, it was reported that the TT genotype of strength athletes was higher (p=0.029). It has been determined that the MCT1 TT genotype is associated with athletic performance in sprint/power athletes (33). Massidda et al. determined the distribution of 39.8% AA genotype, 47.3% AT genotype, and 12.7% TT genotype in elite football players. In addition to this result, they reported that there was a significant relationship between MCT1 and the incidence of muscle injury (p=0.048) and that the incidence of muscle injury was lower in football players with TT genotype compared to football players with AA genotype (p=0.044) (9). In a study of 80 swimmers, the T allele was found to be significantly higher in long-distance swimmers (45%) than long (27%) and middle-distance runners (30%). It has been emphasized that more research is needed to clarify whether this polymorphism shows an advantage in swimming performance (32). In another study, it was reported that there is no significant relationship between personal best times and genotype ratios in sprint/power athletes, but such a relationship exists in endurance athletes. The study indicated that the TT genotype is associated with endurance (28). In support of this study, there was no significant relationship was found between four different style end times and the MCT1 dominant genotype in this study (p>0.05). Although there was no relationship between performance and the MCT1 dominant genotype in the swimmers in the study, further research is needed to discuss whether carrying the A allele can provide an advantage in sprint swimming performance.

In the study of Kikuchi et al., it was aimed to examine the effects of Japanese wrestlers on anaerobic power performance and lactate concentration, and as a result of the study, the AA genotype of MCT1 T1470A polymorphism was detected more in wrestlers than in the control group (p=0.037). It has been reported that there is no statistical difference in MCT1 rs1049434 polymorphism according to national and international grouping (34). The hypothesis of the research conducted with 226 mountaineering athletes from Japan and Poland is that there is a relationship between performance and AA genotype. As a result of the research, contrary to the hypothesis, T allele frequency was found to be higher in Polish mountaineering athletes than in the control group. In addition, it was stated that these results were observed only in the Polish group, and therefore the relationship between genotype and athletic performance may depend on the ethnic background of the population (35). When Cupeiro et al. examined the effect of blood lactate accumulation and MCT1 rs1049434 polymorphism after three different circular training sessions in men and women, the genotype distribution was found to be 27.59% AA, 41.38% AT, and 31.03% TT. It was emphasized that men with the AA genotype of this polymorphism had higher blood lactate levels after training, but the results obtained in women were not the same (36). In Iraqi young handball players, it was found that the athletes with the AA genotype had higher blood lactate levels than the TT genotype (37). 1470T>A (rs1049434), 2197(1414)C>T (rs7169), IVS3-17A>C variants were evaluated in the study examining the performance and blood lactate values of trained road cyclists (n=25) according to MCT1 variants. According to the authors, the most important finding of the study is that wild-type allele carriers 2917(1414)T and IVS3-17C were associated for the first time with higher blood lactate levels at submaximal exercise intensities. It was emphasized that further studies are required to clarify the role of SLC16A1 variants on MCT1 expression as a determining factor of performance in endurance athletes and its relationship to the regulation of other metabolic pathways (38). In this study, a statistically significant positive correlation was

found between the blood lactate values taken in different time periods after the performance of the swimmers and the MCT1 dominant genotype ($p<0.05$). Supporting some studies in the literature, as a result of this study, the blood lactate values of swimmers with TT genotype were found to be higher than those with AA and AT genotypes.

CONCLUSION

In conclusion, this study proved that there is a relationship between the MCT1 dominant genotype and blood lactate levels taken at different times after critical velocity warm-up in swimmers. It was determined that the relationship between swimming performance and MCT1 dominant genotype was not significant. The blood lactate level was found to be higher in swimmers with the T/T genotype. With these results, our research contributes to the effect of MCT1's lactate level taken at different times in swimming. However, more research is needed before we can determine the accuracy of the relationship of MCT1 with performance. In future studies, we need to support the existence of these relationships by working with more sample groups of different ethnic origins.

Although this study did not prove a relationship between swimming performance and MCT1, future studies may discuss this result. While no association was found between short-distance swimming time and MCT1 dominant genotype or allele frequency, an association was found with blood lactate values taken at different times. This result highlights the potential association between the MCT1 gene and physiological parameters in swimmers. It is recommended to investigate whether the MCT1 gene yields similar results for both physiological and performance effects in long-distance swimming.

APPLICABLE REMARKS

- Although the MCT1 dominant genotype does not affect performance in swimmers, it is believed to have a significant impact on blood lactate levels.
- Swimming coaches should be aware of the MCT1 dominant genotype when determining the anaerobic/aerobic threshold levels of swimmers with blood lactate levels.

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AUTHORS' CONTRIBUTIONS

Study concept and design: B. Kistak Altan, I. Odabas. Acquisition of data: B. Kistak Altan, M. T. Hakan, S. U. Zeybek. Analysis and interpretation of data: I. Odabas, B. Kistak Altan, M. T. Hakan, S. U. Zeybek. Drafting the manuscript: I. Odabas, B. Kistak Altan, M. T. Hakan, S. U. Zeybek. Critical revision of the manuscript for important intellectual content: I. Odabas, M. T. Hakan, S. U. Zeybek. Statistical analysis: : I. Odabas, B. Kistak Altan, M. T. Hakan, S. U. Zeybek. Administrative, technical, and material support: I. Odabas, B. Kistak Altan, M. T. Hakan, S. U. Zeybek. Study supervision: I. Odabas, S. U. Zeybek.

CONFLICT OF INTEREST

The research has no conflict of interest.

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