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Role of the monocarboxylate transporter MCT1 in the uptake of lactate during active recovery

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Abstract

Purpose: We assessed the role of monocarboxylate transporter 1 (MCT1) on lactate clearance during an active recovery after high intensity exercise, by comparing genetic groups based on the T1470A (rs1049434) MCT1 polymorphism, whose influence on lactate transport has been proven. Methods: Sixteen young male elite field hockey players participated in this study. All of them completed two 400 m maximal run tests performed on different days, followed by 40 min of active or passive recovery. Lactate samples were measured immediately after the tests, and at min 10, 20, 30 and 40 of the recoveries. Blood lactate decreases were calculated for each 10 min period. Participants were distributed into three groups according to the T1470A polymorphism (TT, TA and AA). Results: TT group had a lower blood lactate decrease than AA group during the 10-20 min period of the active recovery ($p=0.018$). This period had the highest blood lactate for the whole sample, significantly differing from the other periods ($p \leq 0.003$). During the passive recovery, lactate declines were constant except for the 0-10 min period ($p \leq 0.003$), suggesting that liver uptake is similar in all the genetic groups, and that the difference seen during the active recovery is mainly due to muscle lactate uptake. Conclusions: These differences according to the polymorphic variant T1470A suggest that MCT1 ~~plays a central role in taking up lactate from the plasma to the muscle~~ affects the plasma lactate decrease during a crucial period of active recovery, where the maximal lactate amount is cleared (i.e. 10-20 min period).

Keywords: Monocarboxylate transporters, lactate clearance, active recovery.

Abbreviations: DNA: Deoxyribonucleic acid; MCT: Monocarboxylate transporter; PCR: Polymerase chain reaction; SNP: Single-nucleotide polymorphism.

INTRODUCTION

During exercise, oxidation by muscle and heart and gluconeogenesis by the liver are the main fates for lactate (Bergman et al. 1999). Particularly, the oxidative skeletal muscle contracting at submaximal intensity is the principal consumer (Gladden 2004). This fact is reflected during low or moderate-intensity active recovery, which has been proven to be efficient; increasing the blood lactate clearance after high intensity exercise, especially during the first 20 min. (Baker and King 1991; Baldari et al. 2004; Menzies et al. 2010; Micklewright et al. 2006). Most of the membrane lactate transport (in symport with a proton) occurs via a monocarboxylate transporter (MCT), with the MCT1 being the predominant isoform in muscles (Fishbein et al. 2002; Pilegaard et al. 1999). The relevance of MCT1 after high intensity exercise has already been reported by previous studies, which found an association between MCT1 content and blood lactate removal or blood lactate concentration (Green et al. 2002; Thomas et al. 2005). However, these investigations used a passive recovery protocol, with a lower requirement over MCT1 because resting muscles take less lactate due to a lower metabolic rate (Brooks 2009; Gladden 2008; Miller et al. 2002). On the contrary, it would be more relevant to investigate MCT1 and lactate clearance during active recovery, in which lactate uptake by the muscles is increased (Miller et al. 2002). Moreover, this type of recovery is a common situation during some training protocols, such as high intensity interval training protocols, and during some sports, such as intermittent team sports (Macutkiewicz and Sunderland 2011). Therefore, MCT1 is expected to have a relative importance during active recovery after high intensity exercise. However, we do not know if its implication is constant over the recovery or if it is more pronounced in a particular phase.

The T1470A (rs1049434) polymorphism in the *MCT1* gene (*SLC16A1*) is a single-nucleotide polymorphism (SNP) (Lean and Lee 2009; Merezhinskaya et al. 2000) that has been related with sports performance and lactate transport (Ben-Zaken et al. 2015; Cupeiro et al. 2010; Cupeiro et al. 2012; Fedotovskaya et al. 2014; Sawczuk et al. 2015), suggesting an impaired lactate transport in men carrying the T allele. This reduced lactate transport associated with the *SLC16A1* 1470T allele has recently been confirmed by Sasaki et al., who found a lower lactate uptake in oocytes expressing the wild type protein (i.e. the T allele) (Sasaki et al. 2015). Thus, by comparing blood lactate concentrations of the different genotype groups (TT, TA, AA) we aimed to assess the role of the MCT1 in blood lactate clearance during an active recovery, proposing that a meaningful involvement of MCT1 would be reflected on different lactate levels among genetic groups.

We hypothesize that the main involvement of MCT1 occurs during the period of maximal lactate clearance of the active recovery, that is, the first 20 min~~ute~~-period. In our study we investigated the role of the MCT1 on lactate removal dynamics during an active recovery, using the T1470A (rs1049434) MCT1 genetic variant as a determinant of different functionality. Regarding the genotype comparison of *MCT1* isoforms, we hypothesize that the AA group has a greater blood lactate clearance than the TT and TA groups during the active recovery, while the three groups will have the same blood lactate decrease during the passive recovery, reflecting the role of MCT1 on lactate uptake by the muscle.

METHODS

Participants

Sixteen healthy male field hockey players (age 21.7 ± 2.7 years, height 1.74 ± 0.06 m, body mass 70.0 ± 6.3 kg) of the same team competing in the highest Spanish national level league participated in this study. We tested this sample because it allowed an extensive control of diet and training. During the two years prior to the data collection, these two factors were standardized and controlled. Furthermore, all of the subjects were of the same Spanish (Caucasian) ancestry for at least three generations. All principles outlined in the Declaration of Helsinki were strictly followed. The design and performance of the research study was described in the research protocol at the beginning of the study. All participants signed an informed consent which includes: 1) the goal of the study; 2) a statement for the unique use of the samples for the current study; and 3) explicit anonymity about the final genetic result. The study was presented and approved by an Academic Review board in the Department of Medical Sciences at the University of Cantabria.

Experimental procedure

Participants performed two 400 m maximal run tests on a standard 400 m track, each test followed by 40 min of an active or passive recovery, in the first and second day, respectively. The active recovery involved 40 min of running at a self-regulated intensity, which has proved to be suitable for lactate clearance (Bonen and Belcastro 1976; Menzies et al. 2010). Subsequent analysis revealed that this self-regulated intensity corresponded to 65%-75% of their age-predicted maximal heart rate (Tanaka et al. 2001). The passive recovery consisted of sitting for 40 min. The participants were told to avoid any kind of exercise 24 h before

the tests, which were separated by at least one recovery day. The experimental protocol was double blinded in the sense that neither the evaluators nor the subjects knew the genotype during the study.

Lactate, heart rate and anthropometric measurements

For measuring capillary lactate concentrations, blood samples from the fingertip were obtained at rest before the 400 m run tests, immediately after finishing them and four times during the recoveries: at min 10, 20, 30 and 40. These samples were analyzed immediately after they were drawn using the Accusport portable blood lactate analyzer (Boehringer Mannheim, Manheim, Germany), which has been found to be valid and reliable (Bishop 2001; Pinnington and Dawson 2001). Furthermore, only one device was used for all the measurements in order to avoid potential error due to the use of different analyzers (Bishop 2001; Pinnington and Dawson 2001).

Heart rate was recorded during all the trials and recoveries using a HR monitor (Polar Electro, Kempele, Finland). Anthropometric measurements included height and body mass using a balance with height attachment (Model 713, SECA, Hamburg, Germany).

Genotype assessment

For the genetic analysis genomic DNA was extracted from peripheral blood using a QIAamp DNA Blood Mini kit (Qiagen, Hilden, Germany). Genomic DNAs from the participants were analyzed by polymerase chain reaction (PCR) amplification of a fragment containing the T1470A polymorphism of the MCT1 gene (rs1049434, exon 5) and following direct sequencing. According with Merezhinskaya et al. (Merezhinskaya et al. 2000) primers used for amplification

were as follows: sense primer 5'-ACA CAT ACT GGG CAT GTG GC-3' (1455–1474); antisense primer 5'-AAA TCC CAT CAA TGA ACA ACT GGT ATG ATT TCC AC-3' (1807–1841). PCR reaction was made in a total volume of 50 µL containing: 3 µL genomic DNA, 1.5 mM MgCl2, 0.2 mM dNTP mix, 0.4 µM primer, 4% dimethyl sulfoxide (SIGMA, Sant Louis, MO, USA) and 1U Taq polymerase (BioTaqPolimerase, Bio- Line, London, UK), using a GeneAmp® PCR System 2400 thermal cycler (Perkin Elmer, Applied Biosystems Division, Foster City, CA, USA). The amplification consisted of initial denaturation (94 ºC, 5 min); 35 cycles consisting of denaturation (94 ºC, 1 min), annealing (55 ºC, 1 min), and extension (72 ºC, 1 min); and final extension (72 ºC, 10 min). PCR products were electrophoresed in 1.5% agarose gel to verify successful amplification of the 387 bp fragments. Prior to sequencing, the PCR products were purified using QIAquick PCR Purification Kit (Qiagen, Hilden, Germany). The sequencing reactions were carried out using dRhodamine Terminator Cycle Sequencing Kit (Applied Biosystems, Foster City, CA, USA) and analyzed on the automated ABI Prism 310 Genetic Analyzer (Applied Biosystems). Gene sequence of exon 5 was obtained from GeneBank (Accession: NM 003051).

Data Analysis

To analyze blood lactate clearance, we divided the 40 min recoveries into four phases of 10 min. Lactate removal during each 10 min period was calculated by subtracting the blood lactate concentration at the beginning of the phase from the blood lactate ~~concentration~~ measured at the end of the phase. This calculation reflected the blood lactate decrease in each defined period.

The statistical analysis was performed using the Statistical Package for the Social Sciences software 21.0 (SPSS INC., Chicago, IL, USA) and the level of

significance was set at 0.05. To determine the normal distribution of the variables we used a Shapiro-Wilk test and the Chi-square test was conducted to evaluate the Hardy-Weinberg equilibrium. Given the sample size and the variables distribution, non-parametric tests were used.

To assure both 400 m tests (active recovery test and passive recovery test) were equally executed, in terms of performance and maximal effort and were different in terms of recovery, a Wilcoxon test with Holm adjustment for multiple comparisons was carried out with the entire sample and within each genotype group. The variables for this analysis were time to complete the trials, heart rate values and lactate concentrations measured throughout the tests, as well as the lactate reductions during each 10 min phase of the recovery. Furthermore, Friedman tests followed by Wilcoxon tests were used to compare the lactate removed during the different 10 min phases within each recovery. On the other hand, a Kruskal-Wallis test was used to compare anthropometric parameters and age across genotypes, and to guarantee the equal performance of the tests among genetic groups. Finally, we used a Friedman test to analyze differences in lactate clearance phases across genetic groups. Data are presented as mean and standard deviation (SD).

RESULTS

We did not experience any failure in the sample collection, DNA acquisition or genotyping procedures. Genotype frequencies were 4 (25.0%), 6 (37.5%), and 6 (37.5%) for the TT, TA and AA genotypes respectively, being in accordance with the Hardy–Weinberg equilibrium (χ^2=0.907, p=0.341).

The Wilcoxon test comparing the two 400 m maximal run tests showed no differences in time trial, maximal heart rate reached, basal lactate and lactate concentration immediately after the test (Table 1). These parameters were also similar among genetic groups, as well as the intensity of the active recovery expressed by the percentage of their age-predicted maximal heart rate (data not shown). We observed differences between active and passive recoveries in all the lactate measurements (Table 1) and in all 10 min intervals ($p \leq 0.007$), except for the 20-30 min period. Within the passive recovery the rate of lactate removal was constant over the 40 min except for the first 10 min ($p \leq 0.003$), since blood lactate did not decrease during this period. On the other hand, during the active recovery the highest decrease of blood lactate appeared in the 10-20 min period (5.6 ± 1.1 mM/L). This decrease significantly differed from the others ($p \leq 0.003$), whereas the lactate reduction for the 0-10 min period (1.6 ± 2.3mM/L) was similar to those in the 20-30 (2.0 ± 1.3mM/L) and 30-40 min periods (0.4 ± 0.5mM/L).

Table 1 about here

No differences were observed for anthropometric values or age across genotypes (Table 2). Comparison of lactate concentrations among groups revealed no differences, but the analysis of the lactate decreases reported a difference between TT and AA groups in the 10-20 min period (Figure 1). During this phase (i.e. the one with the maximum decrease of blood lactate) AA group exhibited a higher lactate decrease than TT group. On the other hand, no differences were observed for lactate concentrations or lactate decreases during the passive recovery.

Table 2 about here

Figure 1 about here

DISCUSSION

Our main finding was the greater lactate reduction observed in the AA group compared to TT group during the 10-20 min period of the active recovery, which had the highest lactate clearance compared to the rest of the recovery periods (active or passive). These results suggest a higher participation of MCT1 during this period, reflecting the key role of MCT1 on high lactate transport rates.

The allele frequencies observed in our sample were similar to those seen in previous studies with non-sedentary subjects (Ben-Zaken et al. 2015; Cupeiro et al. 2010; Cupeiro et al. 2012; Sawczuk et al. 2015). An allelic frequency of 30%-35% for the T allele is commonly observed, especially in non-athletic populations (Ben-Zaken et al. 2015; Merezhinskaya et al. 2000; Lean and Lee 2009; Sawczuk et al. 2015). However, within athletic samples, the frequencies vary widely, depending on the sport they perform (Fedotovskaya et al. 2014; González-Haro et al. 2015; Sawczuk et al. 2015). These differences could be due to a sport specialization (Ben-Zaken et al. 2015; Fedotovskaya et al. 2014; Sawczuk et al. 2015) or to an adaptive process of natural selection in populations and the neutral processes of genetic drift in populations of different origin. Therefore, further studies are necessary to conclude this observation.

We observed a difference in blood lactate removal between genotypes according to the T1470A MCT1 polymorphism, which alters lactate movement through this transporter (Sasaki et al. 2015), only during the active recovery. Lactate uptake by different organs involves both the MCT and the less efficient process of diffusion (important at concentration >2 mM/L), thus it must be discussed if the encountered differences could be due to the uptake by other organs, such as the liver, expressing other MCT isoforms (Gladden 2008). Lactate removal by the liver accounts for approximately 30% of total removal in resting humans (Gladden, 2008) and no significant changes are reported after 40 min of low-intensity exercise (Ahlborg, 1974). Consequently, we can assume that the role of liver in active and passive recovery is similar. Therefore, the lack of differences during the passive recovery among genetic groups implies that liver function is similar in all the three groups, and that the difference we found is not due to liver function but to muscle lactate uptake (i.e. the only condition that varies between active and passive recoveries). Furthermore, this difference was only observed from minute 10 to 20 of the recovery, suggesting that the transporter is especially relevant during that period, where previous studies (Baker and King 1991; Micklewright et al. 2006), as well as our results, locate the maximum rate of blood lactate clearance. These results, alongside the fact that MCT1 is the predominant isoform in oxidative skeletal fibers (Fishbein et al. 2002) and in cardiac muscle (Gladden 2008), suggest that this transporter plays a central role in taking up lactate from the plasma to the muscle exhibiting differences according to the polymorphic variant T1470A. On the other hand, we did not find differences in the other recovery periods (i.e. min 0-10, 20-30 and 30-40) maybe because the reported blood lactate concentrations did not reach a minimal threshold (approximately 10 mM/L) to force the MCT1 polymorphic isoform, as

previously hypothesized (Cupeiro et al. 2010; Cupeiro et al. 2012). Furthermore, the first 10 min of the recovery, where lactate concentrations still remain high, seems to be an adjustment phase where the blood lactate concentration depends on multiple factors, including blood redistribution, buffer capacity or lactate efflux from glycolytic fibers (Oyono-Enguelle et al. 1990).

Although these results should be taken with caution, since we did not evaluate other factors influencing lactate removal (e.g. like mitochondrial oxidative capacity or fiber type composition), our data suggests a key role of MCT1 in lactate transport; especially during active recovery, when muscles are exercising at low intensity and type I fibers are the most involved. Under these conditions, MCT1 efficiency should be crucial, since a higher lactate transport would increment the chance to use this metabolite as a substrate and an elevated proton efflux from the muscle could prevent fatigue due to the proton gradient from lactate producers to lactate consumers. Furthermore, we tried to limit the effect of other factors on lactate removal selecting a very homogeneous sample in terms of age, body composition and physical capacity, and controlling and standardizing their training regime over two years.

Our data reflect~~s~~ significant differences in blood lactate clearance on subjects with different efficiencies of MCT1 (i.e. AA and TT groups), observing a more effective lactate take up in the AA genotype group. Trainers should consider this fact for designing personalized recovery timings, both during training and competition. Raising the efficiency of MCT1 must be a molecular target, in order to modulate lactate and proton transport during recovery or low/moderate intensity phases of competition; and therefore, not only improving energy availability but also intracellular acid-base homeostasis. Further studies to extend

the knowledge about the role of MCT1 during recovery are needed, especially for analyzing the involvement of the transporter in women, varying the intensity of the recovery, during intermittent exercise or high intensity interval training.

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DISCLOSURES

The authors have no conflicts of interest to disclose.

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Figure 1. Rates of lactate clearance during the active recovery by genotype groups. * Significant difference between TT and AA, p=0.018.

Table 1. Performance variables in both 400 m maximal run tests and lactate concentrations during active and passive recoveries

	Active recovery test	Passive recovery test	p
Time (s)	65.32 (4.13)	65.22 (4.05)	0.925
Heart Rate at the end of the 400 m run (bpm)	1865.87 (87.86)	1843.71 (87.67)	0.454
Basal blood lactate (mM/L)	1.4 (0.4)	1.4 (0.3)	0.975
Blood lactate at the end of the 400 m run (mM/L)	11.2 (2.5)	11.0 (2.2)	0.552
Blood lactate 10 min after the end of the 400 m run (mM/L)	9.6 (2.0)	12.5 (1.7)	0.005
Blood lactate 20 min after the end of the 400 m run (mM/L)	4.0 (1.7)	9.7 (1.6)	<0.001
Blood lactate 30 min after the end of the 400 m run (mM/L)	2.1 (0.7)	6.7 (2.4)	<0.001
Blood lactate 40 min after the end of the 400 m run (mM/L)	1.7 (0.5)	4.9 (1.4)	<0.001

Values are presented as mean (SD).

Table 2. Descriptive variables for the genotype groups.

	TT (n=4)	TA (n=6)	AA (n=6)	p
Age (years)	21.5 (4.0)	20.8 (1.2)	22.7 (3.0)	0.612
Height (cm)	173.5 (3.7)	175.4 (8.2)	173.7 (5.2)	0.963
Weight (kg)	69.0 (2.0)	72.0 (9.9)	68.7 (3.1)	0.998

Values are presented as mean (SD). There were no statistical differences among genetic groups.

Figure

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