ORIGINAL STUDIES

L-carnitine role in heart rate and blood lactate recovery following high intensity exercise - a double blind study following single doses

Ștefan Adrian Martin ¹, Roxana Maria Martin-Hadmaș ², Cristian Graur ³

¹ Physiology Department, “George Emil Palade” University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Romania
² Community Nutrition and Food Safety Department, “George Emil Palade” University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Romania
³ Human Movement Sciences Department, “George Emil Palade” University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Romania

Abstract

Background. In the absence of conclusive data regarding fatigue mechanisms, an important question refers to which stages can improve athletic performance through L-Carnitine intake.

Aims. Our aim was to study both blood lactate accumulation and recovery alongside changes in heart rate and heart rate recovery while analysing group performances during physical exercise.

Methods. Following one single squat jump test, 16 participants were included in a double blind study by measuring the squat jump performances next to the lactate ([La-]) production and the heart rate response (HR). Lactate accumulation was analysed over four different occasions, starting with the baseline value and the ending test value (post SJT). Two other [La-] measurements took place during a 40 minute passive recovery stage according to the study groups: L-Carnitine and Maltodextrine use.

Results. Following the control study group, during a 20 minute passive recovery stage, [La-] dropped to 7.54 mmol/l (6.2 to 9.8) and further on to 4.6 mmol/l (2.4 to 6.7) during another 20 minute period, reaching 48.71% at recovery. [La-] values changed significantly during the post SJT period, as seen through p=0.0001, Kruskal-Wallis=31.19. Following the placebo study group data, [La-] values changed significantly during the post SJT period (p=0.0001, Kruskal-Wallis=43.85) as seen through a mean drop of 49.61% following the 40 minute post SJT passive recovery period.

Conclusions. One single L-Carnitine dose did not cause changes in both heart rate and blood lactate recovery following the squat jump test.

Keywords: squat jump, L-Carnitine, blood lactate

Introduction

L-Carnitine is an ammonium compound which has various pharmacological approaches (Ferrari & Visioli, 1995) with multiple functional roles, as seen in the literature reports (Gerasimo et al., 2018; Oliveira & Sousa, 2019; Balashov et al., 2020). Of them, the vast majority focus on cell metabolism. L-Carnitine can improve the long-chain fatty acid transport and therefore fat metabolism (Sahlin, 2011). Changes in fat oxidation can also represent the reason for general high intake in both active and non-active participants, regardless of being used as a powder, a liquid compound or being integrated in products with various nutritive contents (Pooyandjoo et al., 2016; Baltazar-Martins et al., 2019).

Based on the literature, L-Carnitine can reduce fatigue during physical exercise (Karlic & Lohninger, 2004). However, by studying the mechanism of action we can observe the inconsistency of the published data (Pooyandjoo et al., 2016). Since numerous studies have shown a lack of effect regarding L-Carnitine role during short high-intensity exercise, it remains to be seen whether L-Carnitine will improve exercise recovery (Ransone & Lefavi, 1997).

Recent published data confirm changes in lactate metabolism due to an increased oxygen consumption.
which can improve pyruvate conversion starting from the lactate onset. Furthermore, Fielding et al. (2018) described a spare of amino acids during physical exercise, which can improve protein synthesis while enhancing fat metabolism and the inactive tissue.

According to Colombani et al., 1996 and Burrus et al., 2018, L-Carnitine intake should take place 2 hours prior to physical exercise to limit lactate accumulation and to promote a drop in the heart rate value during physical exercise (Leelarungrayub et al., 2017). Yet, based on the physical exercise typology, multiple motor units are recruited under metabolism products (Theofilidis et al., 2018), which can increase the fatigue level by reducing maximal voluntary force and/or power output (Taylor & Gandevia, 2008). However, evidence suggests that a reduced blood pH and a drop in the blood flow have limited effects on fatigue (McCully et al., 2004). Considering these general approaches, the results of several papers show that lactate is an important energy fuel during physical exercise by following the monocarboxylate transporter (Kitaoka et al., 2012).

From another point of view, studies refer to L-Carnitine use as a supplement needed to obtain a cardioprotective effect (Wang et al., 2018) due to the oxidative stress which results over physical exercise. According to Wang et al. (2018), L-Carnitine administration can serve against ventricular dysfunction, ischemia-reperfusion injury, cardiac arrhythmia, which may also suggest favorable effects on physically active groups. Such measures are related to a reduction in heart cell oxidative stress (Al-Eisa et al., 2018). However, Broderick et al. (1992) considered that high L-Carnitine intake can affect the heart rate by several energy related actions, whereas in earlier times Greig et al. (1987) proved a lack of changes in the heart rate values due to L-Carnitine intake. Contrary to the existing data, recent papers measured changes in the heart rate values during submaximal physical exercise (Suzuki et al. 1981), while few papers refer to changes in the heart rate during maximal physical exercise (Foster et al., 1999).

In the absence of conclusive data regarding the fatigue mechanisms, an important question refers to which stages can improve athletic performance during L-Carnitine intake, taking into account the changes in lactate, which is considered a functional limiting product during physical exercise (Hsu et al., 2020). Further on, which is the quantity, the product source or the individual characteristics and the physical exercise type that favor such changes.

Our aim was to study blood lactate accumulation and recovery, as well as changes in heart rate and heart rate recovery while analyzing group performances during short high-intensity physical exercise (Martin et al., 2019).

**Material and methods**

**Research protocol**

Following one single squat jump test (SJT) we conducted a double blind study by measuring the squat jump (SJ) performance, as well as lactate ([La]) production and heart rate response (HR). Lactate accumulation was analyzed on four different occasions (n = 4), starting with the baseline value (pre SJT) and the end test value (post SJT). Two other [La] measurements took place during a 40-minute passive recovery stage as further detailed through the study groups which used L-Carnitine and Maltodextrine as control and placebo solutions.

**a) Period and place of the research**

We conducted a double blind study during May - June 2019.

**b) Subjects and groups**

As further shown, the study group consisted of a placebo and a control group. The current methodology brings changes compared to previously published papers (Martin et al., 2019). To create the study group, we obtained the participants’ written informed consent. The approval of the Ethics Committee was recorded for this research methodology, while we assumed and respected the Declaration of Helsinki regarding human experimentation and research protocols.

Sixteen participants (n = 16) were included in the research study out of thirty-five individuals (n = 35) who took part in the inclusion process. One single SJT was performed 15 days before the main performance assessment. This test is further described as a study group inclusion test, conducted without blood lactate, HR analysis or without the administration of any of the above mentioned solutions. During the initial inclusion related test, the individuals had to: (1.) be medically fit, (2.) exclude any clinical-pathological medical conditions which were incompatible with the test procedure and (3.) obtain at least 85% of the best initial SJT performance, in order to be included in the study group.

Nineteen participants (n = 19) were excluded due to the impossibility to achieve the targeted performance (n = 85%).

Sixteen participants (n = 16) complied with the inclusion criteria. The participants took note of and accepted the study methodology. If needed, the participants had a methodology through which they could request and obtain additional information regarding the research procedure, the objective and individual performances.

**c) Applied tests**

The supplement allocation: the placebo and the control study groups

The study group allocation took place by following a double blind methodology. The content was allocated and consumed 65 minutes prior to the SJT, by using 2000 mg vegetable capsules which had no color and no flavor. The allocation was made randomly by a group of researchers who were unaware of the study methodology and the individual test results.

L-Carnitine was used as a test solution and Maltodextrine was used as a placebo solution. The two products had similar characteristics: (1.) white color, (2.) tasteless and (3.) lack of differences in texture. Of the study group, eight athletes (n = 8) were randomly assigned to 3.5 g of L-carnitine, while the rest of the participants (n = 8) were randomly assigned to 3.5 g Maltodextrine.

**Pretest procedure**

Forty-eight hours (48 hours) prior to the test, the physical exercise level was limited to 60 minutes and no more than 75% of VO₂max exercise intensity. Two days (n = 2) prior to testing, all accepted participants came to the lab for general medical and anthropometric measurements. All ergogenic aids were excluded seven days (n = 7) before the test from daily intake.
Cardiac function was evaluated by performing an electrocardiogram for each individual. Blood pressure, including systolic (SBP), diastolic (DBP) and mean blood pressure (MBP), along with the capillary blood lactate value ([La\(^{-}\)]) were part of the general medical evaluations. Further measurements were conducted to obtain complete data over individual anthropometric data. Therefore, the following measurements took place: body height, body weight. The measurements were performed using the ADE height meter (Hamburg, Germany).

**Test procedure: 30 seconds SJT – performance analysis**

One single day (n = 1) was allocated for each participant evaluation. All had the same testing time interval and the same conditions, as further described. The participants arrived at the lab 80 minutes before the test. Sixty-five minutes before the start of the test, the supplement was attributed according to the supplement allocation: the placebo and the control study groups.

The lactate equipment (Nova Biomedical Lactate Plus Meter, USA) was calibrated at the beginning of each testing day with low (0.2 - 1.4 mmol/l) and high (4 - 6 mmol/l) control solutions. One capillary blood sample was taken prior to the warm-up. The rest of the [La\(^{-}\)] samples were taken 60 seconds after the end of the test, while repeating the procedure 20 and 40 minutes after SJT, to assess [La\(^{-}\)] recovery during a passive recovery procedure, as illustrated in Fig. 1.

The testing time was 90 seconds. The testing period was divided into three repetitions (n = 3) of 30 seconds each, referred to as: repetition one (rep 1), repetition 2 (rep 2), and repetition 3 (rep 3). Each repetition of 30 seconds (rep 1-3) was interrupted by a 60-second passive pause (Martin et al., 2019). The measurement was performed using the OptoJump System (Microgate, Bolzano - Italy). The Polar H10 device was used for heart rate (HR) measurement before, during, and after the SJT test. Heart rate data were expressed as beats per minutes (BPM) or % of the theoretical maximum heart rate (HR\(_{\text{max}}\)). During the test, the leading position respected the following indications: the athletes were placed between the two device arms with legs wide apart and hands on the hips.

During the SJT, the data regarding the contact time (Contact\(_{\text{c}}\)), the flight time (Flight\(_{\text{f}}\)), the height of the jump (Jump\(_{\text{h}}\)) and the power output (Power) of each jump (Fig. 2; Martin et al., 2019) were recorded and stored for later analysis.

**d) Statistical processing**

Statistical analysis was performed using the statistical software GraphPad Prism 6.0. Due to the small number of subjects, the evaluation was based on non-parametric tests, as further shown. The Mann-Whitney U test was applied to analyze the difference between two data series, while the Spearman r test was used to analyze the degree of association between two indicators. The Friedman test was applied to analyze the differences between repeated measurements. Descriptive data were illustrated as mean values + SD, median values and minimum to maximum values. The confidence interval was 95%.

**Results**

The study results are presented as comparative data of the two study groups. The main objective was to illustrate the most important results in accordance with the study hypothesis.

**General medical assessment and anthropometric data**

In the study group, the SBP reached 123.9 mmHg (103 to 157 mmHg), while the DBP was 70.4 mmHg (51 to 88 mmHg), with a MBP of 86 mmHg. The average pretest HR was 61 BPM (CV = 15.17%) with a minimum value of 50 BPM and a maximum value of 81 BPM. Within the general evaluation results, we did not identify significant differences between the control and the placebo study groups (p>0.05).

**Fig. 1 – Illustration of the study protocol - including the SJT period, blood lactate analysis and the recovery period.**

**Fig. 2 – Illustration of the squat jump test (SJT) phases (Martin et al., 2019).**
**SJT: SJ performance analysis**

Overall SJ performance is expressed by using the recorded data over the FlightT, the ContactT along with the Power and the JumpH. The Flight\textsubscript{T}, reached a mean value of 0.43 seconds (s) in the control group, as against the placebo study group which reached 0.41 s. Yet, the contact time had a lower value in the control group as against the placebo study group (0.67 vs. 0.71 s), which further on affects the power ratio in favor of the control unlike the placebo study group (17.39 vs. 15.70 W/kg). Other data are illustrated in Table I, where the comparative analysis of the two study groups can be observed.

Differences are seen between the two study groups regarding the power output which affects the Contact\textsubscript{T} and Flight\textsubscript{T}, in the control as against the placebo study group.

**Heart rate response during the SJT**

The HR response was analyzed by transforming real-time HR data (BPM) in % of the HR\textsubscript{max}. During the SJT, the mean HR value reached 76.77% of HR\textsubscript{max} in the control study group unlike the placebo study group which reached 78.75%. Yet, the maximum values were 87.11 and 88.27% of HR\textsubscript{max}, related to the maximum [La\textsubscript{]} value. Progressively, from one rep to another (rep 1-3), the HR value increased in the control study group, reaching a maximum mean of 85% during rep 2, as against the placebo study group which reached a maximum mean value during rep 3 (88.22%), as further illustrated in Fig. 3.

**Heart rate recovery during the SJT**

The HR during the SJT passive recovery periods was different from one period of the test to another. During the post rep 1 period (60 seconds), the control group reached a 12.55% drop in the HR value, unlike 16.74% monitored in the placebo study group. However, a lack of statistical differences was seen between the study groups (p = 0.1540). Similar results were observed during the post rep 2 period (60 seconds), in which the control group had a 15.99% heart rate recovery, unlike 14.45% seen in the placebo study group (p = 0.154). Further on, Fig. 4 illustrates both the final and the initial HR values, which were measured during each SJT rep (Rep 1-3).

![Fig. 3 – Comparative data of the control and the placebo study groups regarding HR\textsubscript{max} values during the SJT: repetition 1-3 (rep 1-3) – Data illustrated as mean values.](image)

**Table I**

| Study group | Control | Placebo | Statically significant difference of the mean value? |
|-------------|---------|---------|-----------------------------------------------------|
| **Data type** | **Mean value (min to max)** | **p value** | **Mann-Whitney U** |
| Flight\textsubscript{T} (s) | 0.43 (0.38 to 0.48) | 0.40 (0.35 to 0.44) | 0.0059 | 151.5 |
| Contact\textsubscript{T} (s) | 0.67 (0.54 to 0.80) | 0.71 (0.64 to 0.84) | 0.0375 | 183.0 |
| Jump\textsubscript{H} (cm) | 23.69 (18.94 to 29.21) | 20.99 (17.52 to 23.94) | 0.0073 | 154.0 |
| Power, W/kg | 17.69 (15.13 to 22.33) | 15.70 (13.46 to 17.83) | 0.0136 | 211.0 |

During the post rep 1 period (60 seconds), the HR was related to the Contact\textsubscript{T} (p = 0.0009, r = -0.785), whereas overall exercise intensity, assessed by using the Flight\textsubscript{T} (p = 0.121, r = 0.648) and the Power value (p = 0.0131, r = 0.642), increased the HR recovery time and therefore individual performance in the control study group. During the post rep 2 period (60 seconds), the HR recovery was dependent on rep 2 performance, as confirmed by measuring the Flight\textsubscript{T} (0.0011, r = -0.774) and the Jump\textsubscript{H} (0.0009, r = -0.785).

In the placebo study group, the first SJT recovery stage was related to rep 1 power output (p = 0.028, r = -0.5167) and also the Flight\textsubscript{T} (p = 0.0001, r = -0.924). Yet, no changes were related to the pre SJT Lactate value (p = 0.194, r = 0.320). The changes in the first SJT passive recovery period were related to both 20 min (p = 0.0142, r = -0.5667) and 40 min (0.0272, r = -0.519) [La\textsubscript{]} recovery. In the same study group, the changes in the HR during the second recovery period were related to the individual performance and the overall exercise intensity, as seen through the power output (p = 0.0110, r = -0.583). A lack of association was seen with the rest of the parameters, including the Contact\textsubscript{T} and the Jump\textsubscript{H} (p>0.05).
SJT: blood lactate - comparative analysis

Following the control study group results, pre SJT [La-] values reached 1.5 mmol/l (0.8 to 2.7 mmol/l, CV = 37.85%), while at the end of the SJT, the athletes had 12.11 mmol/l (9.4 to 14.50 mmol/l, CV = 17.72%). Further on, in the placebo study group, the [La-] was 2.01 mmol/l (1.4 to 3 mmol/l) pre SJT and 11.51 mmol/l (9.90 to 14.30 mmol/l, CV = 12.88%) at the end of the SJT.

Following the control study group results, during a 20-minute passive recovery stage, [La-] dropped to 7.54 mmol/l (6.2 to 9.8) and further on to 4.6 mmol/l (2.4 to 6.7) during another 20-minute period (total of 40 minutes), reaching 48.71% recovery. As a result, [La-] values changed significantly during the post SJT period, as seen through p = 0.0001, Kruskal-Wallis = 31.19. Following the placebo study group data, [La-] values changed significantly during the post SJT period (p = 0.001, Kruskal-Wallis = 43.85), as seen through a mean drop of 49.61% following the 40-minute post SJT passive recovery period. Further on, 7.52 mmol/l (4.40 to 10.20) was the [La-] following a 20-minute passive recovery stage, while over the 40-minute period we monitored 4.05 mmol/l (2.60 to 6.70 mmol/l) [La-], as further detailed in Fig. 5.

The illustration represents a comparative analysis between the two study groups regarding [La-] accumulation.

![Blood lactate accumulation](image)

**Fig. 5** – Blood lactate accumulation in the (a) control group and (b) placebo study group – data illustrated as mean value±SD

In the 20- and 40-minute stages, [La-] recovery was strongly influenced by HR recovery following rep 2 (p = 0.0001, r = 1.00). Similar results were seen in the placebo study group, where [La-] recovery was strongly related to HR recovery during both the first (p = 0.0227, r = 0.533) and the second (p = 0.0001, r = 1.000) SJT passive recovery periods. However, as further seen in the placebo study group results, the [La-] values and the recovery periods show a lack of association between [La-] recovery and individual performance, by following the FlightT (p = 0.839, r = 0.05), the ContactT (p = 0.198, r = 0.318) and the Power output (p = 0.843, r = -0.050), which failed to relate to the recovery time.

Discussion

The current research brings new data to other publications which share the same objectives regarding similar topics (Martin et al., 2019; Kashef et al., 2017). By following the study methodology, one can observe the lack of differences between the study groups, with reference to individual performance (p>0.05), HR changes and HR recovery (p>0.05) along with [La-] recovery (p<0.05). However, small differences can be identified which are most likely due to differences in exercise capacity, as we will further discuss.

**L-Carnitine and SJT performance: general analysis**

The FlightT, the JumpH and the power output had small positive differences in the control group as against the placebo study group, which meant an improved overall performance. Yet, the study group had a high variability.

Similar results were found in the publication of Koozechchain et al. (2018), whereas new data obtained after an adapted methodology show a lack of changes by using L-Carnitine (Martin et al., 2019). As a result, the main differences are probably related to the exercise capacity and individual adaptation over the SJT. Such a hypothesis was brought into discussion due to the lack of significant differences in both the HR response and the [La-] accumulation. According to Lango et al. (2001), carnitine deficiency in the endothelial cells will decrease oxygen supply and blood flow in animals, which therefore can influence muscle strength and physical performance. However, in the current results, the assigned solution does not seem to positively influence performance during the SJT, similarly to Stephens et al. (2018), unlike Erikoglu et al. (2013).

Overall performance during endurance physical activity was improved by using L-Carnitine in a study by Gamze et al. (2013), while less available research reports similar results during short high-intensity physical activities (Heinonen et al., 1996). The main statements refer to a drop in [La-] accumulation due to 3 to 4 g intake. However, the intake period, the training length and the training type along with individual exercise capacity can influence the results.

**L-Carnitine and SJT: heart rate response**

Following the study results, one can observe that the control study group had lower heart rates during the SJT, as against the placebo study group. The result can simply confirm the differences in individual performance during the SJT. However, L-Carnitine-induced changes can be improbable during such a test, whereas improvements in HR recovery are more likely to be seen (Broad et al., 2011). Neither the skeletal muscles nor the heart rate have potential over carnitine production, while an increased carnitine accumulation can be seen due to higher plasma concentration (Longo et al., 2016). Therefore, HR recovery varied between 14–16% in both study groups, with differences not exceeding 1.5%, equivalent to 1–5 BPM.
L-carnitine role in heart rate and blood lactate recovery

Similar changes can be related to the exercise capacity and further on to the neuromuscular response during repetitive SJ and SJT overall performance.

No important differences are observed in the measured heart rate during physical exercise. However, Mostafa et al. (2013) reported a lack of changes in the maximum HR, oxygen consumption and [La-] values (Burrus et al., 2018), whereas Leelarungrayub et al. (2017) reported a drop in both lactate concentration and the heart rate value during physical exercise leading to exhaustion (Orer et al., 2014). The differences can be partially explained by following the physical exercise intensity. According to Flanagan et al. (2010), cellular hypoxia is also associated with carnitine depletion, which can possibly reduce the ATP concentration and negatively affect individual performance. Therefore, the heart rate may be affected by: the exercise capacity, the type of physical exercise, along with age. Of them, exercise capacity could generate the most important changes, seen in the control study group unlike the placebo study group. Yet, no difference seemed to be related to L-Carnitine intake.

However, according to Orer et al. (2014), the importance of L-Carnitine intake after exercise can be taken into discussion over heart rate recovery and overall exhaustion time. Due to the paper of Stuessi et al. (2005), it can be observed that post exercise oxygen consumption does not ensure a drop in the respiratory exchange ratio and therefore it cannot improve fat oxidation until Carnitine content is restored. In the current research, L-Carnitine intake took place before the test, without any reports after physical exercise. 

L-Carnitine and SJT: blood lactate recovery

Differences were observed between the two study groups regarding blood lactate accumulation and therefore the maximum value reached. However, no important differences were seen between the study groups regarding the drop in blood lactate following a 40-minute passive recovery stage. The control study group had an improved lactate recovery during the first 20-minute stage, while at the end of the 40-minute period, the lactate values were similar in both study groups (+0.5% diff%).

Several papers studied lactate recovery using an active, passive or combined method (Gmada et al., 2005; Jenni et al., 2003). The main results suggest that by using a combined method, [La-] recovery is improved, while by using L-Carnitine, muscle protein degradation can be decreased and mitochondrial regulation can be improved (Fielding et al., 2018). Yet, few papers studied passive recovery due to L-Carnitine intake. Within the published papers, the main concern was the participants’ age, referring to the recovery process as being mainly influenced by age (Korhonen et al., 2005). A better recovery process is seen in lower age groups due to a reduced development of the anaerobic capacity. Most papers take into account a 10-12 week supplementation in order to obtain changes in plasma and muscle carnitine content. The same effects are described by Brass (2000) over a considerable period of time due to low bioavailability and therefore an absorption of up to 25%.

According to Orer et al. (2014), acute supplementation can reduce blood lactate accumulation, which can therefore improve lactate recovery, a hypothesis unconfirmed within the 40-minute period. The main changes in lactate are attributed to acetyl-CoA, which allows an adapted flux over the pyruvate dehydrogenase complex which can therefore improve blood lactate accumulation. However, one single ingestion cannot serve as a substrate necessary for important functional changes, due to the product bioavailability, individual exercise capacity, the timing and the physical exercise which cannot influence overall carnitine content.

Conclusions

1. One single L-Carnitine dose did not cause changes in either heart rate or blood lactate recovery following the squat jump test.
2. Since many paper topics refer to L-Carnitine intake during medium - long periods, we should note the lack of response induced by a single serving which took place before the physical exercise.
3. This result strengthens the existing data which refer to the importance of intramuscular carnitine content and the lack of changes with one single dose.

Conflicts of interests

The authors declare no conflict of interest.

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