



⟨Brief Note⟩

Effects of citrate ingestion on blood lactate clearance in collegiate male athletes: A double-blind, randomized, placebo-controlled crossover trial

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Summary Citrate is a common ingredient in soft drinks. Although some studies suggest citrate enhance the post-exercise blood lactate clearance, the effect of its practical dosage has not been demonstrated. This study was designed to determine the effect of citrate ingestion on lactate clearance among male athletes following engagement in anaerobic exercise. A double-blind, randomized, placebo-controlled crossover trial was conducted. Twelve healthy male college athletes ingested either 26 mmol citrate or a placebo drink after a 400-m run. Blood lactate levels were monitored for 120 min after the ingestion. The blood lactate decreased from the post-exercise peak (approximately 15 mmol/L) to levels below 5 mmol/L at 40 min after both citrate and placebo ingestion. Citrate enhanced the reduction of blood lactate to make the concentration significantly lower compared to placebo at 20 min after ingestion ($p < 0.005$). The alteration of post-exercise lactate concentration by citrate could add the significance of citrate in soft drinks.

Key words: Citrate, Anaerobic exercise, Lactate, Recovery, Sports drink

1. Introduction

Citric acid is an abundant organic acid in citrus fruits. It has a pleasant sourness, and it is also used as a refreshing ingredient in soft drinks. In Japan, some citrate-containing foods are registered as “Foods with Functional Claims” bearing claims that “mitigate the temporary fatigue sensation associate with mild exercise” and “mitigate fatigue sensation in daily life.” The classification was supported by findings that the intake

of 2.7 g/day citrate for 7 days reduced fatigue as measured using visual analogue scale versus placebo¹⁻³. However, the 7-day ingestion did not affect physiological and hematological parameters^{2,3}. Therefore, the physiological effects of exogenous citrate ingestion remain indistinct.

A study reported that post-exercise citrate ingestion (0.4 g/kg bodyweight) enhanced blood lactate clearance in healthy male adults⁴. Another study found that a citrate-containing soft drink (mean 0.02 g/kg of citric acid equivalent per bodyweight) significantly

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reduced post-exercise plasma lactate levels than that of the control⁵, the drink, however, contained fructose and polyphenols besides citrate. Thereby, the effects on blood lactate could not be solely attributed to citrate. In addition, there is a preliminary report describing that pre-exercise ingestion of citrate (2.7 g) was reported to enhance the post-exercise blood lactate clearance, although not significant⁶. Considering the doses (as citric acid equivalent per bodyweight) in the previous studies for a person with 60 kg of bodyweight, 0.4 g/kg and 0.02 g/kg correspond to 24 g and 1.2 g, respectively. As citric acid has a strong sour taste, while sodium citrate has a salty taste, single ingestion of over 20 g (as citric acid equivalent) is not easy.

Citrate is widely used as an ingredient for soft drinks and post-exercise citrate ingestion has been shown to enhance blood lactate clearance at high dose. However, the effect at low doses has not been demonstrated. Therefore, this study aimed to determine the effect of low dose of citrate, 5 g as citric acid equivalent, on post-exercise blood lactate clearance.

2. Materials and Methods

Test drink

The test drinks contained either 26 mmol citrate (Citrate) or phosphoric acid and sodium hydrogen carbonate (Placebo). The citrate content was formulated according to the results of preliminary experiments (data not shown). Although Placebo contained sodium hydrogen carbonate, the amount was 0.2 g that was considered too small to affect the systemic buffering system. The drinks, Citrate and Placebo, had the same pH adjusted to 3.2, and the tastes were indistinguishable. Table 1 presents the ingredients of the test beverages.

Each aliquot of the test drinks was added to an aluminum bottle with the same appearance, and the contents were controlled using the ID printed on the container. The test drinks were manufactured and provided by Kowa Co., Ltd (Tokyo, Japan). The order of test drinks for each participant was randomized using Research Randomizer (<https://www.randomizer.org/>) by the principal investigator (YS). The experiment was conducted in a double-blinded

manner, and the key was disclosed by the manufacturer after the data had been recorded.

Table 1 Ingredients of the test drink.

Ingredient	Citrate	Placebo
Energy (kcal)	100	100
Citric acid (g) #	5.0	—
Phosphoric acid (g)	—	0.6
Sodium hydrogen carbonate (g)	—	0.2
Calcium lactate (g)	0.4	0.4
Magnesium sulfate (g)	0.275	0.275
Potassium carbonate (g)	0.2	0.2
Carbohydrates (g) *	25	25
Flavors (g) *	0.45	0.45
Vitamin B1 (g)	0.0015	0.0015
Vitamin B2 (g)	0.0021	0.0021
Vitamin B6 (g)	0.0018	0.0018
Niacin (g)	0.012	0.012
BCAA (g) §	0.05	0.05
Total (mL)	500	500

Citrate (26 mmol) was contained as combination of citric acid and sodium citrate. The amount (gram) expressed as citric acid equivalent.

* Both test drinks contained the same flavors.

§BCAA represents branched chain amino acids (L-leucine: L-isoleucine: L-valine = 2:1:1).

Energy was adjusted by carbohydrates.

Study design

A double-blind, randomized, placebo-controlled crossover design was adopted. The participants attended the same experiments for 2 days, with a washout period of at least 1 day. They refrained from participating in exercises on the previous day of the experiment. On the day of the experiment, they visited our laboratory at 8:00 a.m. after overnight fasting (with their last dinner finished by 9:00 p.m. the previous night).

Participants

Twelve healthy male collegiate athletic throwers (n = 8) and rugby players (n = 4) participated in the study. Male athletes, who were healthy and within the age range of 18–25 years, were included in the study.

The exclusion criteria set were as follows: 1) receipt of a physician-prescribed treatment or drug; 2) current or prior history of a serious cardiovascular

disorder, liver function disorder, renal dysfunction, respiratory disorder, an endocrine disorder, or metabolic disorder; 3) history of chest pain or fainting; 4) allergies related to the test supplement; 5) donation of 200 mL of blood within 1 month prior to the commencement of the study or 400 mL within 3 months; 6) smoking; and 7) deemed unsuitable for this study by a physician.

Consequently, all enrolled twelve participants completed the intervention. Their characteristics (mean \pm SD) were age 19.4 ± 1.1 years, height 176.4 ± 5.8 cm, bodyweight 91.4 ± 16.6 kg, and body mass index 29.2 ± 4.0 kg/m². As Citrate contained 26 mmol citrate, the dose of citrate was 0.29 ± 0.05 mmol/kg bodyweight.

The flow diagram of the crossover trial was presented in Fig. 1. This crossover trial was conducted on June 10–27, 2018, at Juntendo University Sakura Campus.

Study protocol

Participants were informed of the purpose of the experiment, the method of implementation, and the possible risks. All participants provided voluntary, written in-formed consent, to participate in the

experiment.

The participants completed the study protocol, comprising 2 days of experiments with a washout period of more than 2 days. The pre-exercise resting blood lactate concentration was measured and then a 400-m run was performed on a track, without warm-ups. During the run, the pace of the initial 200 m was controlled at 5 min/km using a pacemaker, and the latter 200 m was a full run with maximal effort. After the full effort run, each participant caught his breath and measured his blood lactate concentration by self-puncture of the fingertip using the Lactate Pro 2 LT-1730 system (Arkray, Kyoto, Japan): that took approximately 2 min after exercise. Then, each participant ingested the test drink as hurry as he could. The time to finish drinking was designated as time 0 min. After test drink consumption, participants moved to a quiet room and calmly sat. The blood lactate concentrations were measured at 20, 40, 60, 90, and 120 min after test drink consumption.

Since participants knew high-intensity exercise using a bicycle ergometer, such as the Wingate test, tends to produce gastrointestinal symptoms such as nausea, a 400-m run with a pacemaker in the first 200 m was chosen as the exercise load that increases

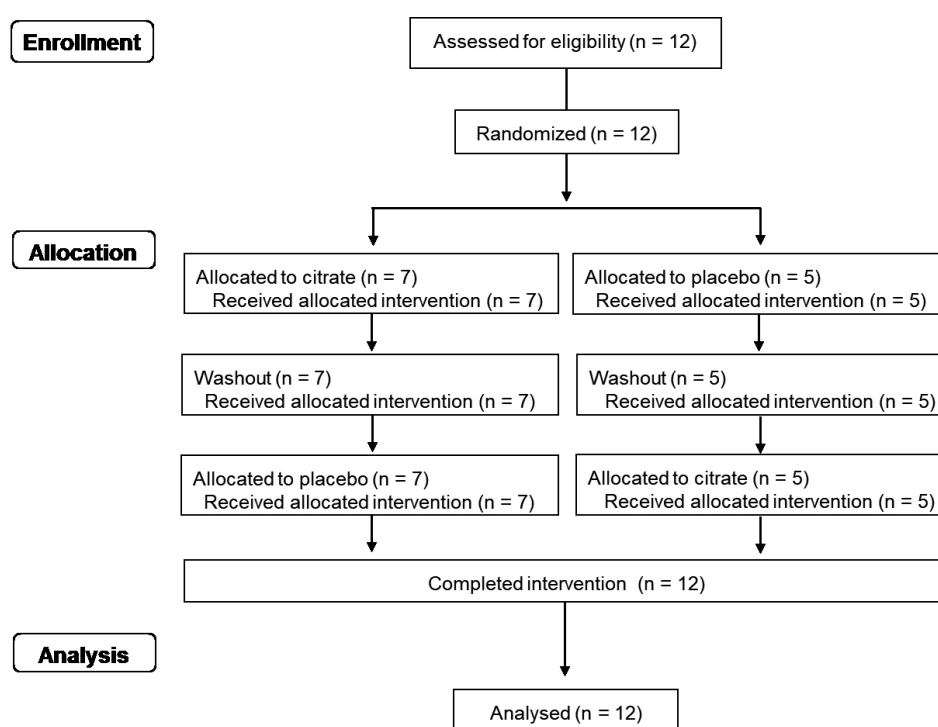


Fig. 1. Flow diagram of the crossover trial.

blood lactate concentration with fewer psychological barriers.

During the study, participants experienced transient hyperlactatemia, however no symptoms was observed attributed to lactate acidosis, e.g. gastric symptoms (nausea, stomach pain, diarrhea), malaise, muscle pain, and overventilation. In addition, any adverse event attributed to test drink was not observed.

Ethical consideration

This study was planned and conducted according to the ethical standards as laid down in the 1964 Declaration of Helsinki and later amendments or comparable ethical standards. The protocol for this study was approved by the Juntendo University Ethics Committee (Approve No: #30-50) and registered with the University Hospital Medical Information Network Clinical Trials Registry (UMIN-CTR ID: UMIN000029578).

Statistical analysis

The results were presented as either mean with standard deviation or estimated marginal mean with standard error. The normality of the data was assessed with the Shapiro–Wilk test. For the blood lactate concentrations, the normal distribution was hypothesized in only pre-exercise, post-exercise, and

60 min after exercise in both Citrate and Placebo groups. Thus, the pre-exercise and post-exercise blood lactate concentrations were compared using Wilcoxon signed-rank test.

The blood lactate concentrations after the test drink ingestion were adjusted by post-exercise peak concentration (peak = 1), and then compared using generalized estimating equation (GEE) of generalized linear model. The models with fixed subject variable (ID), fixed with-in-subject variables including test drinks (Citrate and Placebo), experimental day, and time (20, 40, 60, 90, and 120 min after drink consumption), as well as interactions of all combinations of pairs of within-subject variables, were analyzed. The model including all pairs of with-in-subject variables as interactions was selected according to the quasi-information criterion (smallest).

Then, the area under the curve (AUC) of the standardized blood lactate concentrations were compared using GEE. In the GEE model, the predictive variables were set as follows: ID as a subject variable, test drink (Citrate and Placebo) and experimental day (date) as within-subject variables, and test drink × experimental day as interactions.

SPSS ver. 19 (Japan IBM, Tokyo, Japan) was used for the analysis. Statistical significance was set at $p < 0.05$.

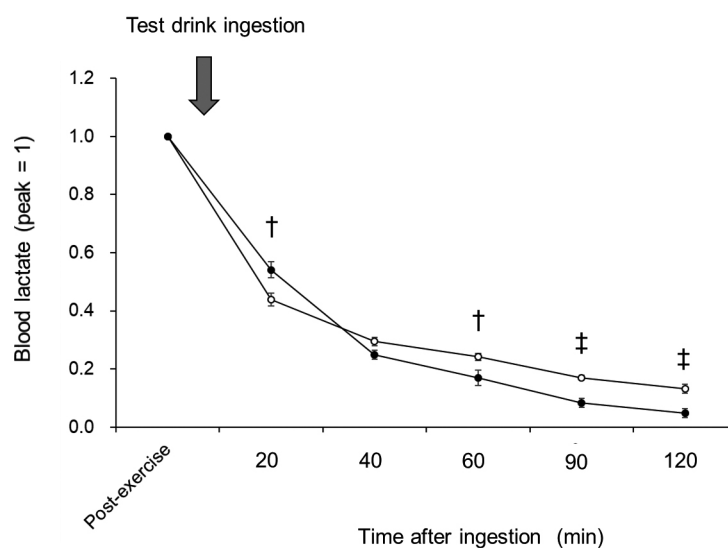


Fig. 2. Kinetics of the blood lactate concentration after the test drink ingestion. Open and closed circles represent Citrate and Placebo, respectively. The symbols and error bars represent the estimated marginal means and standard errors. † $p < 0.005$, ‡ $p < 0.001$

3. Results

The entire measured blood lactate concentrations were shown in Table 2. The pre- and post-exercise blood lactate concentrations were 1.6 ± 0.5 mmol/L (range; 0.9 – 2.6 mmol/L) and 15.0 ± 2.8 mmol/L (range; 9.1 – 19.0 mmol/L), respectively. The mean post-exercise blood lactate concentration was not different between Citrate (15.0 ± 2.4 mmol/L) and Placebo (15.0 ± 3.3 mmol/L), although the pre-exercise level of Placebo (1.8 ± 0.5 mmol/L) was significantly higher than Citrate (1.4 ± 0.3 mmol/L). At 20 min after the test drink ingestion, the mean blood lactate concentration of Citrate (7.1 mmol/L) was smaller than Placebo (8.8 mmol/L), although not significant. Meanwhile, the mean blood lactate concentration of Citrate (1.8 mmol/L) was significantly higher than Placebo (1.4 mmol/L) at 120 min after the test drink ingestion.

As the order of drink ingestion was randomized, 7 participants took Citrate first, and the other 5 participants Placebo first. Between the Citrate-first and Placebo-first groups, there was no significant

difference in background characteristics; age, height, weight, and body mass index.

Meanwhile experimental day can apparently confound the results because the day provide different environment e.g. order of test drink, temperature, humidity, weather. Thereby, the post-exercise lactate reduction was analyzed by distribution free generalized linear model controlled for the experimental day. As mean post-exercise blood lactate concentration was not different between Citrate and Placebo, the blood lactate concentration was standardized by the post-exercise peak concentration (lactate-per-peak; peak = 1).

The lactate-per-peak of Citrate was significantly lower than that of Placebo at 20 min after the test drink ingestion ($p < 0.005$), whereas it significantly higher after 60 min to 120 min (Fig. 2).

The AUC of lactate-per-peak of Citrate during 0 – 60 min after the test drink ingestion was significantly smaller than that of Placebo ($p < 0.001$), while that during 0 – 120 min was indifferent from that of Placebo (Fig. 3).

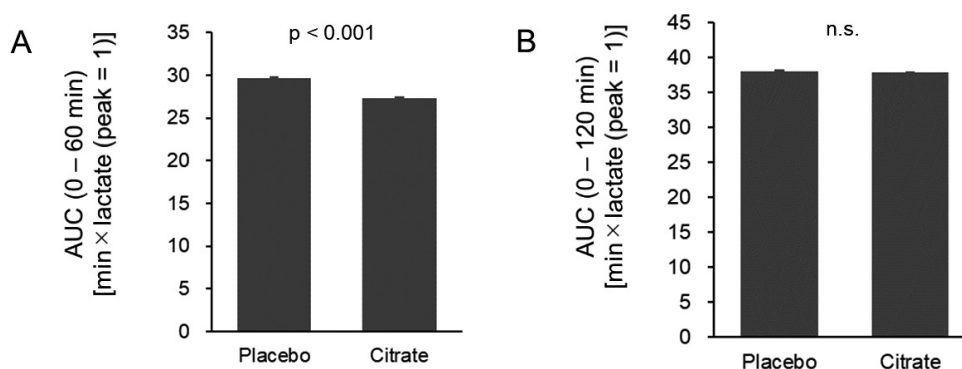


Fig. 3. Area under the curve of the blood lactate concentration after test drink ingestion. The lactate concentration was standardized as post-exercise peak = 1. A. Area under the curve (AUC) between 0 – 60 min. B. AUC between 0 – 120 min. The bars and error bars represent estimated marginal means and standard errors.

Table 2 Each participant's blood lactate kinetics

ID	Drink	Day	Blood lactate (mmol/L)						
			Pre	Post	20 min	40 min	60 min	90 min	120 min
A	Placebo	0610_1	0.9	16.5	15.7	7.4	3.9	2.8	1.1
A	Citrate	0613_2	1.0	14.8	7.4	3.1	2.1	1.8	1.0
B	Placebo	0627_2	1.5	11.3	6.6	4.0	3.0	1.8	1.1
B	Citrate	0613_1	1.4	13.9	5.1	3.6	4.4	2.1	1.4
C	Placebo	0613_1	2.2	15.1	10.4	7.4	2.5	2.2	1.6
C	Citrate	0627_2	1.7	15.8	7.1	5.0	2.8	2.2	1.7
D	Placebo	0613_2	2.6	16.2	13.3	3.4	3.1	2.8	2.6
D	Citrate	0610_1	1.3	17.2	14.9	8.2	6.6	5.3	3.0
E	Placebo	0610_1	2.2	17.6	14.4	4.0	2.8	1.6	1.2
E	Citrate	0613_2	1.2	16.1	4.9	3.5	2.0	1.5	1.9
F	Placebo	0613_2	1.9	18.2	8.1	2.4	4.8	1.9	0.9
F	Citrate	0610_1	1.4	15.9	8.4	6.6	3.9	3.6	2.7
G	Placebo	0627_1	1.4	16.4	5.4	3.3	3.1	1.5	1.4
G	Citrate	0613_2	0.9	14.0	6.2	2.3	2.9	2.1	1.7
H	Placebo	0617_2	1.6	14.2	3.7	2.2	1.7	1.6	1.7
H	Citrate	0610_1	1.4	16.1	4.7	2.6	2.3	1.8	1.7
I	Placebo	0617_2	1.9	9.1	2.7	2.1	1.9	1.2	1.0
I	Citrate	0613_1	1.2	12.8	5.1	2.7	3.0	1.4	1.6
J	Placebo	0610_1	1.5	9.7	5.8	4.0	2.0	1.1	1.3
J	Citrate	0617_2	1.6	9.6	4.0	2.8	2.8	1.9	1.6
K	Placebo	0617_2	1.7	17.2	11.2	5.9	4.6	1.7	1.2
K	Citrate	0610_1	2.2	19.0	10.4	8.0	3.5	3.2	2.2
L	Placebo	0610_1	2.6	18.9	7.9	5.0	3.8	3.1	1.1
L	Citrate	0627_2	1.4	14.2	6.5	5.1	4.4	2.9	1.5
	Placebo	Mean	1.8	15.0	8.8	4.3	3.1	1.9	1.4
		SD	0.5	3.3	4.2	1.8	1.0	0.7	0.5
	Citrate	Mean	1.4	15.0	7.1	4.5	3.4	2.5	1.8
		SD	0.3	2.4	3.1	2.1	1.3	1.1	0.6
	All	Mean	1.6	15.0	7.9	4.4	3.2	2.2	1.6
		SD	0.5	2.8	3.7	1.9	1.1	0.9	0.6
Distribution - Shapiro-Wilk test									
	Placebo	p	0.741	0.091	0.734	0.147	0.601	0.220	0.006 **
	Citrate	p	0.268	0.631	0.021 *	0.040 *	0.072	0.020 *	0.146
	Wilcoxon	p	0.025 *	1.000	0.224	0.969	0.637	0.061	0.005 **

Pre and Post represent pre-exercise and post-exercise, respectively. ID indicates each participant. Day represents the experimental day; the last number indicates which of the two sets of experiments conducted on one day the participant attended. Wilcoxon represents Wilcoxon signed-rank test. * $p < 0.05$, ** $p < 0.01$

4. Discussion

This study examined the influence of citrate ingestion on the post-exercise reduction of blood lactate levels. Male collegiate athletes ingested 26 mmol of citrate (5.0 g as citric acid equivalent; 0.29 ± 0.05 mmol/kg) or Placebo after their blood lactate levels elevated by the 400-m run. The findings indicate that citrate significantly enhanced the reduction of blood lactate levels at 20 min after ingestion.

Miyake et al reported citrate ingestion (0.4 g/kg as citric acid equivalent = 2.1 mmol/kg) enhanced the blood lactate clearance 40 – 60 min after the exercise⁴. Takashino et al reported low dose of citrate (2.7 g) made the post-exercise mean blood lactate concentration at 30 min after the exercise lower than control (no ingestion), while the levels of 45 and 60 min after the exercise were indifferent⁶. A meta-analysis showed that pre-exercise sodium citrate supplementation (0.7 – 2.0 mmol/kg) reduced blood lactate concentrations from the peak (> 15 mmol/L) at 2 min to less than 10 mmol/L at 12 min post-exercise, while the levels at 33 min was higher but statistically indifferent from that of placebo⁷. The results of this study presented citrate ingestion (0.29 mmol/kg) significantly enhanced the post-exercise blood lactate clearance at 20 min after the ingestion, although the levels after 60 – 120 min were significantly higher than that of Placebo. The enhanced clearance within 30 min post-exercise and the lack of the effect over 30 min were in accordance with the low dose experiment⁶ and the meta-analysis⁷. Thereby, citrate seems to enhance the blood lactate clearance within 30 min post-exercise.

As described above, there is a discrepancy of citrate effect on blood lactate levels over 30 min post-exercise. The high dose citrate lowered blood lactate levels over 30 min post-exercise⁴. Low dose experiment showed no effect⁶. Meanwhile this study showed citrate rather suppressed the clearance. The discrepancy could be attributed to the difference of the doses. Also, the participants could have caused the discrepancy. The participants of the high dose experiment were healthy male adults whose blood

lactate levels of pre- and post-exercise were 1 mmol/L and 4 mmol/L, respectively⁴. Those of low dose experiment were female adults without exercise habit with pre- and post-exercise blood lactate levels of 4 mmol/L and 12 mmol/L, respectively⁶. In contrast, the participants of this study were athletic throwers and rugby players who were athletes primarily engaged in anaerobic exercise. Thereby they should have had high amount of type 2 muscle fibers which tend to produce but difficult to eliminate lactate. The pre- and post-exercise blood lactate levels were 1.6 ± 0.5 mmol/L and 15.0 ± 2.8 mmol/L, respectively. The high amount of type 2 fiber may have caused a large difference in blood lactate concentrations pre- and post-exercise, resulting in differences in blood lactate kinetics over 30 min of exercise. The relationship between muscle fiber type and sensitivity to citrate could be an interesting topic for future study.

This study suggested the enhanced blood lactate clearance within 30 min of citrate ingestion. The ingested citrate is absorbed as citrate via sodium-coupled sulfate as well as di- and tri-carboxylate transporter (SLC13A)⁸. Serum citrate concentration increases 15 min after ingestion of citrate⁹. Cytosolic citrate suppresses glycolysis by inhibiting phosphofructokinases (PFK1 and PFK2) and pyruvate kinase indirectly by decreasing the level of fructose-1, 6-bisphosphate¹⁰. Meanwhile, citrate stimulates gluconeogenesis and lipid synthesis; these pathways consume ATP¹⁰. Hence, cytosolic citrate enhances ATP production through the tricarboxylic acid (TCA) cycle and oxidative phosphorylation. This situation promotes the conversion of lactate to pyruvate and oxidizes it in the TCA cycle to enhance lactate clearance. Cytosolic citrate is provided by mitochondria through the citrate carrier protein SLC25A1¹⁰ and from extracellular fluid via plasma membrane citrate transporters¹¹. As discussed above, the rational mechanism in which ingested citrate enhances lactate metabolism can be considered. However, it also seems difficult to discuss that the low dose of citrate (0.29 mmol/kg) affected metabolism as a metabolite. Possibly, citrate or its metabolite could have acted as a messenger. Further

study is warranted to elucidate the mechanism.

The present study has some limitations. Although we found that citrate affects blood lactate level which is an indirect indicator of intramuscular metabolism, the parameters to assess the buffering system, such as pH and HCO_3^- concentration were not measured. Therefore, the effect on the systemic buffering system remains to be elucidated. In addition, only male participants were recruited in this study; the results of this study must also be confirmed in female participants.

In conclusion, citrate is a common ingredient in sports drinks, although the physiological benefit has not been sufficiently elucidated. This study demonstrates that the ingestion of citrate enhances blood lactate clearance. Thus, findings from this study have been able to add new insight into the role of citrate in soft drinks.

Conflicts of interest

This study received funding from Kowa Co., Ltd. (Tokyo, Japan). Authors, K.K., T.M., H.M., and E.M. are employed by Kowa Co. Ltd. They were responsible for the test solutions used in the crossover trial, but they had no role in data collection and analysis, decision to publish, or preparation of the manuscript.

The other authors, Y.T., K.S., and Y.S. declared that they have no competing interests.

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