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ORIGINAL ARTICLE

Dextrose 10% drink is superior to sodium-dextrose drink in increasing blood glucose and sprint speed in soccer players: A double-blinded randomized crossover trial study

Supériorité d'une boisson à 10 % de dextrose par rapport à une boisson de dextrose plus sodium pour augmenter glycémie et vitesse de sprint chez des footballeurs : étude croisée randomisée en double aveugle

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KEYWORDS
Ergogenic effect;
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VO_2max;
Dextrose

Summary
Objectives. — This study aimed to examine the effect of dextrose drink (D) on blood glucose, VO_2max, and sprint speed compared with Sodium-Dextrose drink.

Equipment and Methods: We conducted a double-blinded crossover study of 22 young male (19 ± 1.1 years) soccer players, consumed either Dextrose drink or Sodium-Dextrose drink. We compared pre- and post-data of Blood Glucose, VO_2max, and sprint speed between the dextrose and sodium-dextrose drinks groups. The primary outcome was differences in ΔBlood Glucose between D and Sodium-D group. Secondary outcomes were differences in ΔVO_2max and Δsprint speed between D and Sodium-D group.

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Results. — The mean BG level was higher in D compared with Sodium-D group [136 ± 22.9 mg/dl vs. 118 ± 21.5 mg/dl] ($P = 0.009$). Mean differences of ΔBG of D (mean before and mean after) compared with ΔBG Sodium-D (mean before and mean after) is 16.8 mg/dl ($P = 0.001$). The mean of sprint speed is faster in the D group [15.2 ± 1.25 sec vs. 15.9 ± 1.61 sec] ($P = 0.019$), but we found no significant differences in VO_2max between both groups [42.1 ± 3.44 ml/min/kg vs. 42.3 ± 3.98 ml/min/kg] ($P = 0.834$).
Conclusion. — The present study demonstrated that dextrose drink without added sodium has a large influence on increasing blood glucose level and sprint speed.

MOTS CLÉS
Effet ergogénique ;
Aide ergogénique ;
Footballeurs masculins ;
Glycémie ;
Sprint vitesse ;
VO_2max ;
Dextrose

Résumé
Objectifs. — Cette étude visait à examiner l'effet d'une boisson à 10 % de dextrose sur la glycémie, la VO_2max et la vitesse de sprint par rapport à une boisson Sodium-Dextrose.
Équipement et méthodes: Nous avons mené une étude croisée en double insu sur 22 jeunes footballeurs de sexe masculin (19 ± 1,1 ans), consommant une boisson au Dextrose ou une boisson Sodium-Dextrose. Nous avons comparé les données pré- et post- sur la glycémie, la VO_2max et la vitesse de sprint entre les groupes de boissons Dextrose et Sodium-Dextrose. Le critère de jugement principal était les différences de glycémie Δ entre les groupes Dextrose et Sodium-Dextrose. Les critères de jugement secondaires étaient des différences de vitesse ΔVO_2max et Δsprint entre les groupes Dextrose et Sodium-Dextrose.
Résultats. — Le niveau moyen de glycémie était plus élevé dans D par rapport au groupe Sodium Dextrose [136 ± 22,9 mg/dl vs. 118 ± 21,5 mg/dl] ($p = 0{,}009$). Les différences moyennes de ΔBG de Dextrose 10 % (moyenne avant et moyenne après) par rapport à ΔBG Sodium-D (moyenne avant et moyenne après) sont de 16,8 mg/dl ($p = 0{,}001$). La vitesse moyenne de sprint est plus rapide dans le groupe D [15,2 ± 1,25 s vs. 15,9 ± 1,61 s] ($p = 0{,}019$), mais nous n'avons trouvé aucune différence significative de VO_2max entre les deux groupes [42,1 ± 3,44 ml/min/43 kg vs. 42,3 ± 3,98 ml/min/kg] ($p = 0{,}834$).
Conclusion. — La présente étude a démontré que la boisson de dextrose sans sodium ajouté a un effet important sur l'augmentation du taux de glycémie et de la vitesse de sprint.

1. Introduction

Carbohydrate (CHO) ingestion has been known to improve skilled and cognitive performance, and rapid reduction in Blood Glucose (BG) could harmfully affect the skilled actions in a soccer match [1]. BG decreased sharply with the onset of soccer [2] due to the augmentation of glucose uptake via translocation of glucose transporters type 4 (GLUT4) to the active site of muscle membrane [3].

Two groups of glucose transporters are responsible for facilitating glucose passage into and out of the cells: the sodium-glucose co-transporters (SGLTs) and the facilitative glucose transporters (GLUTs) [4]. SGLT1 and GLUT5 are localized in the brush border membrane (BBM) of enterocytes; GLUT2 in the basolateral membrane (BLM) at low luminal dextrose concentration, but in the BBM plus BLM at high dextrose concentrations. Hence, dextrose absorption at low luminal concentration is mediated via SGLT1 in the BBM and GLUT2 in the BLM, but in high concentration, it is also enhanced by GLUT2 in the BBM [5].

Enterocyte has a limited capacity to absorb glucose[6]. Exercise studies have shown that CHO's delivery is limited by the transport capacity of SGLT-1 [7]. High intestinal luminal glucose concentrations ≥ 15 mM, or more modest loads, induces expression of GLUT-2 in BBM [8,9]. Glucose absorption can be amplified by BBM GLUT-2 when the enterocyte and submucosal glucose concentration are lower than in the lumen [10]. The time required to reach steady-state glucose accumulation within the enterocytes in vitro following exposure is ≤ 2 min [11,12] and within 5 to 10 min [13], and a soccer player must achieve it as soon as possible. A lower concentration of luminal glucose than the submucosal has been reported [14], and it will hinder apical GLUT-2 for passive glucose absorption rather than assisted net absorption [10,15].

Outfield soccer players covered 9-12 km of distance during a match; 12% are sprinting [16]. Hence, soccer players require rapid energy turnover from both aerobic and anaerobic metabolisms [17]. The ergogenic effects of CHO ingestion were associated with the maintenance of glycogen levels because of a 30—50 times increment of muscle glucose uptake in exercising muscle [18]. As a result, CHO's availability was shown to influence soccer performance by enhancing the capacity to perform the intermittent activity and may become a limiting performance factor when it is depleted [19].

Sports drinks are designed to replenish CHO, electrolytes, and fluids to the body, which required fast absorption in the BBM [20]. Generally, sports drinks contain either CHO and or sodium-CHO, but to know which one is better in terms of ergogenic effect (BG concentration, VO_2 max, and sprint speed), there is still no trial that has been established. Therefore, we designed a randomized, double-blind trial in male soccer players to evaluate the effect of dextrose

drink with no sodium versus the dextrose drink with 20 mM sodium in blood glucose, VO_2max, and sprint speed. The primary outcome was a mean difference in BG level between Dextrose 10% (D) and Sodium-Dextrose 10% (Na-D) intervention. Secondary outcomes were mean differences in VO_2 max and sprint speed. We hypothesized that D supplementation gives a superior effect than Na-D supplementation in blood glucose, VO_2max, and sprint speed.

2. Methods

2.1. Study design and procedure

This study was a randomized crossover (double-blind) design. Participants and researchers were blinded from the administered fluid type. Participants were randomly assigned to receive either dextrose (D) solution or sodium dextrose (Na-D) solution. Randomization took place immediately after the participants had been confirmed to be eligible to enter the study. The drink's randomization code was not made available to anyone involved in conducting or evaluating the study and was released after the blind review and the freezing of the final database. After randomizing, the study was divided into three phases, as shown in Fig. 1. Assessment of health status, dietary intake, body composition measurement, baseline blood glucose, baseline VO_2 max, and baseline sprint speed was evaluated in phase I. We measured the blood glucose before the participants drink the dextrose or sodium dextrose supplementation in phase II. We asked the participants to sprint before received the solution, then we recorded the time and calculated the VO_2max. After that, we gave one type of solution (dextrose or sodium dextrose). In order to have a comparison of the post supplementation effect, the participants repeated the previously mentioned procedures in phase II. Phase III was similar to phase II except for the nutritional supplementation. The participants could receive either dextrose or sodium dextrose in phase II and the opposite alternative in phase III. This trial was conducted at UNM Banta-Bantaeng, Makassar, Indonesia, from April 2019 to May 2019.

2.2. Supplement preparation

The supplements consisted of dextrose (D) or sodium dextrose (Na-D). Glucose is a monosaccharide, which acts as an essential energy source through aerobic or anaerobic metabolism. Dextrose or D-glucose is an aldohexose stereoisomer of glucose. It is the most commonly occurring isomer of glucose in nature. Our pharmacist used 15 g of dextrose anhydrous to make a 150 ml dextrose 10%, with pH 5.27. Another 150 ml Sodium dextrose solution consisted of 10% dextrose, added with 20 mM of sodium. The material is taken from Japan (Otsuka Holdings Co., Ltd.), and Akademis hospital's licensed pharmacist supervised the entire mixing process.

2.3. Participants

Sample sizes for this research are twenty one, and it was calculated by equation $n = [\{(Z\dot{\alpha} + Z_\beta)\ X\ Sd\}/d]^2$, with $Z\dot{\alpha} = 1.96$; $Z_\beta = 0.842$; $Sd = 28.2$; $d = 10$. Random allocation was performed using a random electronic generator via https://www.random.org/lists/. Our pharmacist staffs were responsible for generating the random allocation sequence, enrolled participants, and assigned participants to interventions. After the assignment of interventions, all of the participants and the researchers were blinded. A total of 50 male outfield academy soccer players were screened for the study. Thirty-two participants entered into the inclusion criteria. All were healthy and free of injury in the three months preceding the study. Three participants refused to join, and 6 were dropped out. Twenty-two participants participated (age, 19.6 ± 1.1 years; height, 165.7 ± 5.25 cm; weight, 52.5 ± 8.47 kg; body fat, 12.5 ± 4.68%; muscle mass, 82 ± 4.7%; BMR, 1346 ± 147.0 kcal) in the study and were successfully analyzed until the end. Before enrolling in the study, each participants' physical examination and health history were taken. Eligibility criteria were healthy men soccer players between 18 and 23 years of age, who exercised on average between 8 and 12 h per week the last month before inclusion. All participants were within the last meal four hours before the test. Exclusion criteria were the use of amylase supplement, suffering from fever and diarrhea, using laxative agents within 24 h, consuming CHO absorption inhibitors, caffeine, creatinine, beta-alanine, sodium bicarbonate supplement within 24 h, mean arterial pressure <65 mmHg, knee or muscle injuries, history of diabetes mellitus and heart disease, going through the ketogenic diet program, history of gastrointestinal surgery, and total body fat percentages > 30%. Participants who were currently taking any other dietary supplement, sports drink, or functional food intended to enhance performance or muscle mass or had taken any of these in the previous week were also excluded.

The study was approved by the Faculty of Medicine Hasanuddin University Research Ethics Committee with reference number 214/UN4.6.4.5.31/PP36/2019. Written informed consent was obtained from all participants before inclusion. The study was registered at ClinicalTrials.gov, with reference number NCT04206579.

2.4. Intervention and procedures

The participants were instructed to abstain from exercise 24 h before the testing in phase I, II, and III, and they arrived at the field track by car, motorcycle, or by public transportation. They were recommended to maintain the usual eating pattern 48 h before the test and had the last meal 4 hours before phase I. Besides, they were told to refrain from coffee, and sports drink 48 h before the test.

Dietary intake was collected using two days of a food recall. BG was measured from capillary blood glucose from the fingertip and immediately analyzed (Aviva; Accucheck, Roche Diagnostics, Indiana, USA), blood pressure was measured using an aneroid sphygmomanometer (R1 shock-proof; Riester, Jungingen, Germany), heart rate was measured with wrist band pulse monitor (Bluetooth 4.0 wireless sport heart rate monitor WP290; Egoman, Shenzen, China), body weight, muscle, fat, water, metabolic rate was measured using the body composition analyzer (BC-545 N; Tanita,

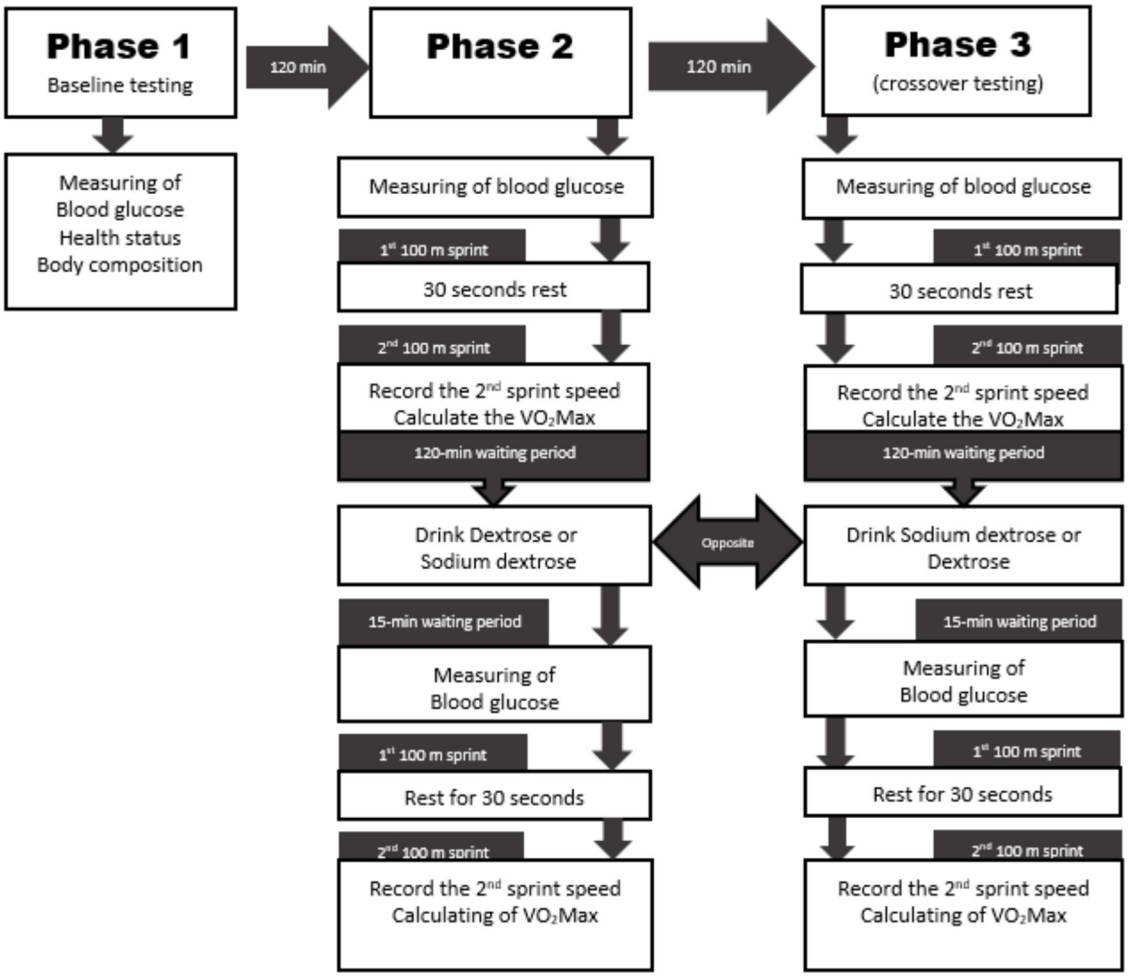

Figure 1 Flowchart of the study.

Tokyo, Japan), body height was measured using a stadiometer (HR-200, Tanita, Tokyo, Japan), sprint speed was measured using a digital stopwatch (sec23589 sec23589P1; Seiko, Tokyo, Japan).

Participants were instructed to refrain from strenuous physical activity in the 2 days preceding trial sessions and recorded all food consumed in the 2 days before the trial. Food records subsequently were analyzed using professional German nutrition software (EBISpro, Nutrisurvey 2007). On arrival at the field, pre supplementation capillary blood samples were collected, and then all players run for 2×100 m and calculated the VO_2max using Uth-Sorenen-Overgaard-Pedersen formula, and sprint speed was recorded. After doing the baseline measurement, each player waited for 15 minutes to consume either dextrose or sodium dextrose solution and then waited for 15 minutes to have another subsequent capillary blood sample measurement. After that, the players run for 2×100 m, recorded the VO_2max and sprint speed. The players remained in a rested state for 120 minutes as a crossover washed-out period and then did the same protocol with a different solution.

2.5. Statistics

Descriptive statistics were used to characterize the participants (mean, standard deviation [SD]), with a 95% confidence interval, and significance was accepted at $P < 0.05$. Data were checked for normality, as indicated by the Shapiro—Wilk test. Paired samples *t*-tests were used for comparison before and after the condition in blood sugar, VO_2max, and sprint speed. All reported *P*-values were based on two-sided tests. Exact values of $P < 0.05$ were considered statistically significant. The data were analyzed using IBM SPSS Statistics 25 for windows (SPSS Inc., Chicago, Illinois, USA). The sample size was selected based on a priori power calculation. Accordingly, with a type I error of 0.05, power analysis revealed that for a 2-tailed paired data Student *t*-test, a sample size of 21 individuals is adequate to achieve a power of > 80%. To interpret the magnitude of the effect, Cohen's d effect sizes ($\pm$ 95% confidence limits) were estimated using a purpose-built spreadsheet, with effect size thresholds set at < 0.20, > 0.50, and > 0.80 for small, moderate, large, effects respectively.

Table 1 Baseline Characteristics of the study participants ($n = 22$).

Characteristic	Mean ± SD
Age (years)	19.6 ± 1.12
Height (cm)	165.7 ± 5.25
Weight (kg)	52.7 ± 8.47
Systolic blood pressure (mmHg)	109 ± 5.4
Diastolic blood pressure (mmHg)	79 ± 9.5
Body fat (%)	12.5 ± 4.68
Muscle mass (%)	82 ± 4.7
Bone mass (kg)	2.5 ± 0.31
BMR (kcal)	1346 ± 147.0
Visceral fat level	2.9 ± 1.0
Total body water (%)	60.7 ± 4.91
Blood Glucose (mg/dl)	93 ± 2.5
VO2 max (ml/min/kg)	41.0 ± 3.21
Sprint speed (sec)	16.13 ± 1.333
Heart rate (times/minute)	60 ± 4.1
Protein Intake (g)[a]	98.4 ± 16.23
Carbohydrate Intake(g)[a]	327.4 ± 41.28
Fat intake (g)[a]	48.1 ± 14.68
Energy intake (kcal)[a]	2137 ± 295.8

Data are presented as mean ± standard deviation (SD) unless otherwise stated.
[a] Average of 48 h food recall.

3. Results

The baseline characteristic for all 22 participants was shown in Table 1. All of the variables are within a normal distribution. All participants ($n = 22$) completed the test without any injuries, and none of the participants in both groups reported any side effects. Due to the little differences in the muscle (82 ± 4.7%) and total body water percentages (60.7 ± 4.91%) of all participants, we used similar dextrose and sodium dextrose doses.

The effect of dextrose or sodium dextrose supplementation on blood glucose, VO_2max, and sprint are presented in Figs. 2–4, respectively. The mean of blood glucose before and after dextrose supplementation is 91 ± 11.6 mg/dl and 136 ± 22.9 mg/dl. The mean of blood glucose before and after sodium dextrose supplementation is 91 ± 9.1 mg/dl and 118 ± 21.5 mg/dl. Dextrose supplementation had a higher blood glucose level than sodium dextrose (136 mg/dl versus 118 mg/dl). This data was also supported by BG's mean differences before dextrose supplementation compared with before sodium dextrose was 0.4 ± 5.86 mg/dl, $p = 0.747$ (95% CI −2.19, 3.01), which means their differences were not statistically significant. The data between the dextrose and sodium dextrose group was shown in Tables 2 and 3. Another corroboration of the data was found that BG's mean differences after dextrose supplementation compared with after sodium dextrose was 17 ± 28.2 mg/dl, $p = 0.009$ (95% CI 4.7–29.8). Cohen's d effect's size = 0.60. However, to sum it all, the primary endpoint is to know the mean differences of ΔBG in the dextrose group (before and after supplementation) compared with a ΔBG sodium dextrose group (before and after supplementation) as shown in

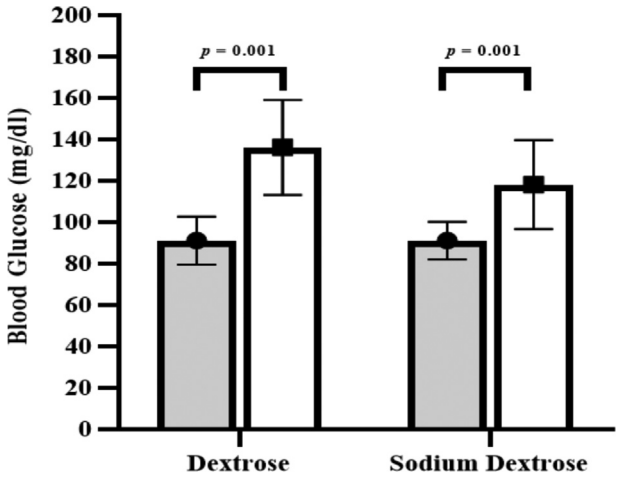

Figure 2 Blood glucose differences. Blood glucose differences before and after dextrose supplementation ($P = 0.001$). Blood glucose differences before and after sodium dextrose supplementation ($P = 0.001$). Shading bar: blood glucose before supplementation; white bar: blood glucose after supplementation

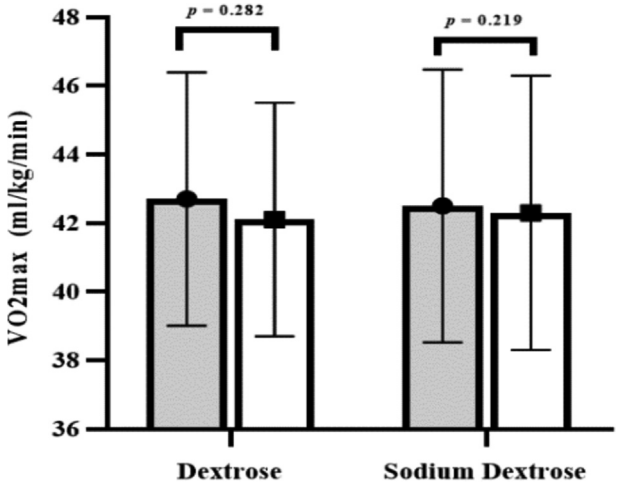

Figure 3 VO_2max differences. VO_2max differences before and after dextrose supplementation ($P = 0.282$). VO_2max differences before and after sodium dextrose supplementation ($P = 0.219$). Shading bar: VO_2max before supplementation; white bar: VO_2max after supplementation

Table 3, which has the P-value of 0.011 and effect's sizes 0.6.

The mean of VO_2max before and after dextrose supplementation is 42.7 ± 3.69 ml/min/kg and 42.1 ± 3.44 ml/min/kg. The mean of VO_2max before and after sodium dextrose supplementation are 42.5 ± 3.98 ml/min/kg and 42.3 ± 3.99 ml/min/kg. The mean differences of VO_2max before dextrose supplementation compared with before sodium dextrose was 0.2 ± 4.57 ml/min/kg, $p = 0.810$ (95% CI −1.79, 2.26). The mean differences of VO_2max after dextrose supplementation compared with after sodium dextrose was −0.16 ± 3.65 ml/min/kg, $p = 0.834$ (95% CI −1.78, 1.45). Cohen's d effect's size = 0.04. The secondary endpoint is to know the mean differences of the ΔVO_2max dextrose

Table 2 Before and after administration of dextrose and sodium dextrose.

	Dextrose				Sodium dextrose			
	n = 22				*n* = 22			
	Before	After			Before	After		
	Mean ± SD	Mean ± SD	*P*-value	Cohen's d effect sizes	Mean ± SD	Mean ± SD	*P*-value	Cohen's d effect sizes
BG (mg/dl)	91.4 ± 11.62	136.0 ± 22.91	0.001	3.02	91.0 ± 9.18	118.7 ± 21.53	0.001	1.29
Sprint (sec)	14.69 ± 1.330	15.26 ± 1.258	0.012	0.59	14.61 ± 1.468	15.90 ± 1.612	0.001	1.23
VO_2max	42.7 ± 3.69	42.1 ± 3.44	0.282	0.23	42.5 ± 3.98	42.3 ± 3.99	0.219	0.27
HR (bpm)[a]	148 ± 3.04	149 ± 3.26	0.204	0.27	148 ± 2.78	148 ± 3.41	0.590	0.11

Data are presented as mean values ± standard deviations (SD). The *P*-value represents the significance level of the difference between the before dextrose or sodium dextrose and after the dextrose or sodium dextrose supplementation. Cohen's d represents the effect sizes of the p-value. BG, blood glucose; VO2max, maximal oxygen uptake; HR, heart rate; BPM, beat per minute.

[a] Measured at the end of the sprint sessions.

Table 3 Differences of blood glucose, sprint and VO_2max before and after dextrose or sodium dextrose supplementation.

	Dextrose	Sodium Dextrose	Diff. Dextrose versus Sodium Dextrose			
	n = 22	*n* = 22				
	Mean $_{diff}$ ± SD	Mean $_{diff}$ ± SD	Mean $_{diff}$	95% CI	*P*-value	Cohen's d effect sizes
BG (mg/dl)	−44.5 ± 14.76	−27.7 ± 21.33	16.8	4.27, 29.44	0.011	0.6
Sprint (sec)	−0.57 ± 0.966	−1.28 ± 1.035	−0.71	−1.24, −0.18	0.011	0.6
VO_2max	0.5 ± 2.46	0.1 ± 0.65	−0.40	−1.64, 0,83	0.507	0.1

Data are presented as mean values, standard deviations (SD), 95% confidence interval (CI), and *P*-value. Diff. dextrose versus sodium dextrose: differences between before and after supplementation of dextrose versus sodium dextrose. BG: blood glucose; VO2max: maximal oxygen uptake.

group (before and after supplementation) compared with a ΔVO_2max sodium dextrose group (before and after supplementation), as shown in Table 3, which has a *P*-value of 0.507 and effect's sizes 0.1, as shown in Table 3.

Before and after dextrose supplementation, the mean sprint speed is 14.6 ± 1.33 sec and 15.2 ± 1.25 sec. The mean sprint speed before and after sodium dextrose supplementation is 14.6 ± 1.46 sec and 15.9 ± 1.61 sec. The mean differences of sprint speed before dextrose supplementation compared with before sodium dextrose were 0.07 ± 0.724 sec, $p = 0.640$ (95% CI −0.24, 0.39). The mean differences of sprint speed after dextrose supplementation compared with after sodium dextrose was −0.64 ± 1.184 sec, $p = 0.019$ (95% CI −1.16, −0.11). Cohen's d effect's size = 0.55. The mean sprint speed is faster in a dextrose supplementation group than the sodium dextrose group (15.2 sec versus 15.9 sec). The secondary endpoint of this study supported this value. The mean differences of Δsprint speed in the dextrose group (before and after supplementation) compared with the Δsprint speed in sodium dextrose group (before and after supplementation) as shown in Table 3, which has a *P*-value of 0.011 and effect's sizes 0.6, as shown in Table 3.

4. Discussion

The mean differences of BG after dextrose versus after sodium dextrose group were 136 ± 22.9 mg/dl and 118 ± 21.5 mg/dl. This positive finding may suggest the mechanism that solely dextrose supplementation via a GLUT-2 was able to transport CHO into the plasma faster than SGLT-1. CHO can be moved through the BBM via a GLUT-2 pathway [15], which may occur if the CHO supplementation is given without sodium as a co-transporter, evading the SGLT-1 pathway [21].

The total CHO concentration in sports drinks is 4—8%, and the sodium content is 10—25 mM [22]. In this study, we were

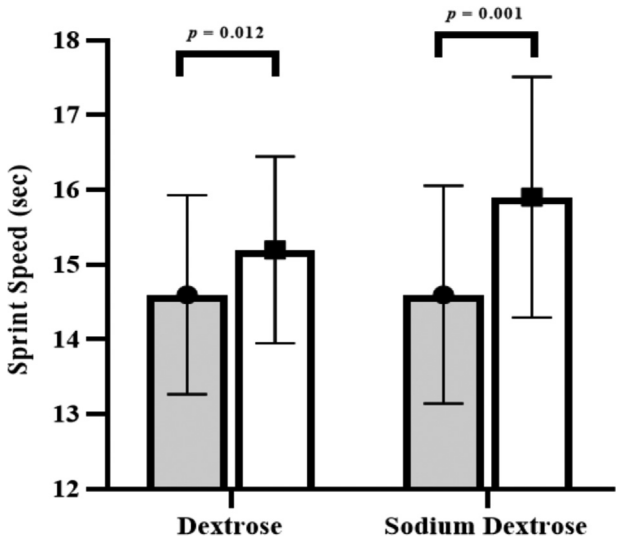

Figure 4 Sprint differences. Sprint differences before and after dextrose supplementation ($P = 0.012$).
Sprint differences before and after sodium dextrose supplementation ($P = 0.001$).
Shading bar: sprint speed before supplementation; white bar: sprint speed after supplementation

using a higher CHO concentration of 10% with 20 mM sodium. Our purpose with using the higher CHO concentration was to induced the GLUT2 absorption pathway in BBM. The dextrose group has a higher mean compared with the sodium dextrose group, and this condition may indicate that at a higher CHO concentration, it will trigger absorption through the GLUT 2 pathway because GLUT2 is rapidly and transiently recruited to the brush border membrane of the enterocyte, leading to a three-fold enhancement of glucose transport [23]. It is also known that the apical GLUT-2 absorption component is greater than the SGLT-1 at high luminal glucose concentrations [24,25].

Traditionally, SGLT-1 is the main route of CHO absorption in the villous BBM, in which absorption of glucose is coupled with the presence of sodium through the Na/K-ATPase activity [26,27], but GLUT-2, another transporter, can also be recruited to the BBM when high concentrations of glucose are present [28]. Expression of BBM GLUT-2 by the high intestinal luminal glucose concentrations is stimulated by enterocyte AMP kinase (AMPK), triggered by the opening of Cav 1.3 Ca^{2+} channels following SGLT-1-dependent depolarization of the apical membrane potential [6].

Apical GLUT-2 is tightly regulated by long, and short-term CHO supply and by local and endocrine hormones, disease (diabetes mellitus), cellular energy level, and the regulation occurs through intracellular signalling pathways [29]. Thus, apical GLUT-2 can act as the main pathway for sugar absorption. Apical GLUT-2 in the disease model acts as a guard mechanism by preventing high concentrations of CHO escape into the large intestine [30].

At ingestion rates over 60—70 g of CHO per h, exogenous CHO oxidation peaks around 50-60 g/h, but at 180 g/h, did not increase exogenous CHO oxidation rates much above 60 g/h [26]. Because this limitation was not caused by gastric emptying, liver glycogen storage, and muscle glucose uptake, it was deduced that absorption had to be limiting [28]. When fructose was ingested in addition to larger amounts of glucose, CHO oxidation rates were elevated above 60 g/h [7], suggested that glucose transport across the epithelial cell was the limiting factor and that the maximal transport capacity of SGLT-1 was reached [31].

Evidence shows that the diffusion part is mediated by the translocation of transient GLUT-2 from the intracellular enterocyte vesicles into the apical membrane, especially after high luminal glucose, allowing bulk absorption of glucose [32,33]. The apical GLUT-2 pathway from luminal sugar uptake could be inserted within minutes into the apical of enterocytes in response to high glucose levels during food assimilation [8]. As in physiologic condition, after a meal, the glucose level in the gut lumen rises, which could saturate the SGLT-1. The GLUT-2 pathway in the apical membrane then facilitates CHO diffusion down the concentration gradient. This study used 150 ml of dextrose 10%, equivalent to 15 grams of dextrose.

The slower mean increment of BG in the administration of sodium dextrose solution can be partly explained by the fact that SGLT-1 is a rate-limiting pathway for enterocyte glucose absorption. SGLT-1 uses a transmembrane sodium gradient to carry out the cellular uptake [34]. Such a situation may occur slower, considering the condition of soccer players is not in the actual match condition [35], suggesting lesser sodium loss through sweat perspiration that may result in a weaker sodium transmembrane gradient [36,37]. One main rule when using sports drinks that contain dextrose and sodium is that they should not be diluted. This would change the concentrations of CHO and sodium. Both will affect the speed at which the drink empties from the stomach, so the whole energy and fluid delivery mechanism will be delayed. The benefits of using sports drinks are proven only for adults involved in rigorous exercise and doing training in the hot condition and high humidity [20].

Soccer is an intermittent, high-intensity sport. Carbohydrates in the formed BG and muscle glycogen are the major substrate to replenish the adenosine triphosphate (ATP) in the contracting skeletal muscles during high and moderate-intensity work-out [38]. ATP is the form of the primary energy system, but it is only stored in small amounts (8 mmol/kg wet weight of muscle), and the phosphocreatine (PCr) system will resynthesize the ATP again from adenosine diphosphate [39]. The stored amounts of ATP and PCr are used up within 10 seconds of an intense exercise or an explosive movement like a sprint because of more than a 1,000-fold increase in ATP demand [39]. In order to deplete the ATP-PCr system, we designed the 2×100 m of a sprint (the first 100 m is to reduce the ATP-PCr system) with a 30 sec of resting time in between and followed the second 100 m (because to recover the PCr stores in muscle, it may take time around from 5 minutes) [39]. Once the storage is depleted, the body will use the BG in the glycolytic system to maintain a continuous ATP supply. As a metabolic fuel, BG, and muscle glycogen used to drop to levels that may damage cognitive function [40]. After dextrose supplementation, skeletal muscle is responsible for taking up 70—90% of the blood glucose [41]. Hence, soccer players are often advised to consume the CHO solution during competitions to supply muscle glycogen reservations and maintain BG concentrations during the match [42] [43]. Consumption of

CHO is recommended to maintain endogenous energy stores and prevent excessive physical and cognitive performance decline throughout the match [42].

Consumption of CHO has been proven to improve the performance index in playing soccer, where the reduction in BG levels can negatively influence the skilled actions involved [44]. Extended CHO release from a low glycemic index food also supports the benefits to fuel the bodily glucose reserve for endurance athletes [45]. Considering the performance benefits of CHO supplementation, which can reduce glycogen ''depletion'' levels throughout the game, it is recommended that soccer players consume 30—60 g/h of exogenous CHO supplementation during competitions to maintain the BG level and glycogen reserves [46]. However, the superior effect on the rate of increase in BG of pure ergogenic dextrose supplementation compared with sodium dextrose has not yet been known, therefore started from that point, this study was conducted.

In soccer players, an increase in BG levels can be observed from 75 min onwards when a 10% CHO-electrolyte solution is consumed before and during soccer matches, compared to a placebo-fluid-electrolyte control [47]. 6—7% of CHO solutions have succeeded in increasing the sprint speed [43]. Glycogen breakdown declines with a simultaneous elevation in extra-muscular BG levels to maintain normal BG during the match [19]. CHO supplementation improved intermittent running, soccer skill performance, the distance covered during the game and was correlated with the maintenance of glycogen levels compared to a placebo beverage [19]. In our trial, the faster sprint speed in the dextrose group might be explained by the effect of the faster increment in BG mean in the dextrose group, which served as a readily available substrate to fuel the muscle glycogen. CHO concentration in the sports drinks range between 4—8%, and the sodium content is between 10—25 mM [22], but in our trial, we used a higher CHO concentration reached 10% with 20 mM sodium. This fact may explain that it will trigger absorption through the GLUT 2 BBM pathway at higher CHO concentrations. Half-time for apical GLUT 2 membrane insertion is around 3.5 min (hence we used a 15 minutes period before tested the effect, but still below 20 minutes since it will provoke the insulin release) and correlated with activation of the protein kinase C βII isoform, which mark the physiological importance of fast response to increasing BG levels [48]. The transient presence of GLUT 2 in the apical enterocyte has been proved using antibodies to epitopes within N-terminal regions [49]. Cell immunofluorescence imaging's location in the apical membrane has been confirmed using extracellular loop antibodies [50]. GLUT 2 can transport glucose and has a very high Kt (transport constant) for glucose, indicate that apical GLUT 2 has the role for the diffusive component of glucose absorption [5], which at high glucose levels, the GLUT2 component accounted for ± 75% of the absorption [8]. When the glucose concentration is higher in the intestinal gut lumen than in plasma, rapid trafficking of GLUT2 to the BBM accounts for the glucose absorption [51]. With BG level rising faster in the dextrose group, it will provide the added benefit of a faster sprint speed, as the muscle can readily uptake the surge elevation of BG. One of the possible underlying mechanisms was the increased availability of substrates, which can be used as a rapid fuel for muscle contraction [52], because during the exercise, the amount of muscle glucose uptake increases, which it is leading to a decrease in blood glucose levels [53].

VO_2max differences between groups given dextrose compared with sodium dextrose were not significant. Soccer is a predominantly intermittent aerobic sport interspersed with periods of high-intensity anaerobic, which may perform different exercise intensities and use the aerobic and anaerobic energy systems. The total duration of active play in soccer is ± 95 min, suggested the main energy source during the match is supplied through aerobic glycolysis. The average VO_2max during match play was reported to be around 75—80% of maximum oxygen uptake [54]. The mean and peak heart rate of players was estimated to be around 85 and 98%, respectively [55]. Nonetheless, VO_2max values of soccer players were shown to vary noticeably, with current high-level soccer players are suggested to acquire maximal oxygen uptakes between 60-70 ml/kg/min, with an estimated minimum of 65 ml/kg/min in elite soccer players [19].

People with high fitness (as measured by VO_2max) are more physically active in their daily life than those with low fitness. VO_2max is a modifiable variable, and it can be increased through exercise. High-intensity interval resting training was found to improve VO_2max more effectively than moderate continuous sessions [56]. We found no differences between the two groups because it has been known that VO_2max is not only influenced by BG levels but also by other factors, such as cardiorespiratory fitness [57]. However, to determine if a dose-response relationship exists between exercise intensity and training-induced increases in VO_2max, a result from meta-analysis suggests that exercise training intensity does not affect the elevation of training-induced VO_2max. However, similar adaptations can be achieved in low training doses at higher exercise intensities than higher training doses of lower intensity [58]. VO_2max is a measurement of the maximum capacity to utilize oxygen in multilevel exercise tests and acts as a measure of soccer players' cardiorespiratory fitness system. The cardiovascular and thermoregulatory systems are first affected by low hydration, especially when exercise is performed in high temperature or high humidity conditions [20], but in this experiment design, the players were not performed in the heat environment. Nevertheless, our study's limitation is that the research was not performed under a real soccer match.

5. Conclusions

Our study indicated that solely dextrose supplementation effectively supports blood sugar and sprint speed in soccer players. A solution of 150 ml dextrose 10% elevated blood glucose faster than sodium dextrose solution. Based on our results, we conclude that concerning the beginning of the soccer match, a 10% of dextrose solution ingested 15 minutes before the match could elevate BG faster and may have a good impact on sprint velocity, as at the beginning of a match, to kick off and dominate it. These findings suggest a future suggestion to re-evaluate the energy drink, which nearly always has a mixture of CHO and electrolyte.

Disclosure of interest

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The authors declare that they have no competing of interest.

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