Influence of High vs. Low Carbohydrate Ingestion on Substrate Oxidation Patterns of Males and Females During Running Bouts at the Individual Anaerobic Threshold

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Abstract

Background: To date, it remains unclear how pre-exercise CHO availability modulates the oxidative regulation of substrates when exercise is conducted at the intensity (V\text{IAT}) where the Individual Anaerobic Threshold (IAT) is located. This study aimed in assessing the impact of High CHO (HC) vs. Low CHO (LC) diets (where on the LC day a combination of low CHO diet and a glycogen depleting exercise was implemented) on the oxidative regulation of CHOs and lipids while exercise is conducted at V\text{IAT}.

Methods: 16 recreational runners (m=8; f=8; 28 ± 3 y; 1.76 ± 0.09 m; 72 ± 13 kg; 23 ± 2 kg/m\textsuperscript{2}) performed 3 different running protocols, each allocated on a different day. At day 1, a maximal stepwise incremental test was implemented to assess the IAT and V\text{IAT}. During days 2 and 3, participants ran a constant-pace bout (30 min) at V\text{IAT} that was combined with randomly assigned HC (7g/kg/d) or LC (3g/kg/d) diets for the 24 h before testing. Breath-by-breath gas exchange data was measured continuously and used to determine substrate oxidation. Dietary data and differences in substrate oxidation were analyzed with a paired t-test. A two-way ANOVA tested the diet X gender interaction (α = 0.05).

Results: Overall, the IAT and V\text{IAT} were 2.74 ± 0.39 mmol/l and 11.1 ± 1.4 km/h, respectively. CHO oxidation was 3.45 ± 0.08 and 2.90 ± 0.07 g/min during HC and LC bouts respectively (P < 0.05). Likewise, lipid oxidation was 0.13 ± 0.03 and 0.36 ± 0.03 g/min (P < 0.05). Females had 14% (P < 0.05) and 12% (P > 0.05) greater lipid oxidation compared to males during HC and LC bouts, respectively.

Conclusion: Twenty-four hours of high CHO consumption results in concurrent higher CHO oxidation rates and overall utilization, whereas maintaining a low systemic CHO availability significantly increases the contribution of lipids to the overall energy metabolism. The observed gender differences underline the necessity of individualized dietary planning before exerting at intensities associated with performance exercise. Ultimately, it remains to be established how these findings can be extrapolated to training and competitive situations and with that provide trainers and nutritionists with improved data to derive training prescriptions.
Keywords
Carbohydrate intake; Individual anaerobic threshold; Running; Substrate oxidation

Abbreviations

CHO : Carbohydrate
VO2peak : Peak Oxygen uptake
IAT : Individual Anaerobic Threshold
V1AT : Individual Anaerobic Threshold's intensity
HC : High Carbohydrate
LC : Low Carbohydrate
BMI : Body Max Index
%BF : Percentage Body Fat
VO2max : Maximal oxygen uptake
HRmax : Maximal Heart Rate
VO2 : Oxygen uptake
VCO2 : Carbon dioxide output
RER : Respiratory Exchange Ratio
Med : Medical check
Glyc depl : Glycogen depleting bout

Introduction

Carbohydrate (CHO) and lipids are the main substrates fueling exercise, each having its oxidation patterns regulated by several factors such as intensity and duration of the activity, dietary intake pattern, gender and training status [1-5]. When described as a sole function of exercise intensity, the oxidative metabolism of these two substrates has a clear pattern. At low and moderate intensities, lipid (intramyocellular lipids and plasma free fatty-acids) is the main substrate being oxidized while CHO metabolism (blood glucose and stored muscle glycogen) increases parallel to exercise intensity and predominates at times of high physical exertion [6-8]. Steady-state exercise on the other hand (i.e., an exercise level that can be maintained for a prolonged period of time), normally favors lipid oxidation [8].

Based upon these regulatory mechanisms and depending on individual goals, professional and recreational athletes may be advised to vary their training regimen around different intensities (using and conditioning both aerobic and anaerobic energetic pathways), while aiming to expand endurance capacity, power and performance [9]. Likewise, nutrition has the potential to alter the metabolic regulation of substrates with the intake of CHO in particular, being not only crucial to fuel exercise at intensities above 65% of maximal oxygen uptake (VO2max), but also directly assisting in the post-exercise recovery phase [3,10]. For instance, CHO-loading strategies (7-10 g/kg/d) may increase not only glycogen storage (up to 42% post-prandial) but also its overall usage, which in turn delays fatigue allowing exercise to be prolonged and endurance performance to be improved [3,11-14]. Still, this latter mechanism is somewhat restricted to the male athletic population as females are well known for having a greater reliance on lipid metabolism compared to males [15].

In addition, female athletes have had mixed results when it comes to increasing muscle glycogen storage capacity and/or enhancing endurance exercise performance (i.e., despite CHO-loading equivalent to ~75% of the energy intake during 4-6 days) [7,13,16,17].

Yet, it remains unclear how pre-exercise CHO intake modulates the oxidative regulation of CHO and lipids, when exercise is conducted at the intensity where the Individual Anaerobic Threshold (IAT) is located (V1AT). Namely, a metabolic marker delineating the upper levels of endurance capacity in which a shift in the oxidative regulation of substrates is expected favoring a CHO driven metabolism [18-20]. The IAT represents the upper border where constant load endurance exercise can be sustained, being commonly used to guide athletic training (e.g., when aiming to improve endurance capacity) or in performance diagnostics [19-22]. Exertion at V1AT can be generally sustained for up to 60 minutes, though the average speed of a marathon is only slightly under it [18,19]. Consequently, in order to assist coaches, trainers and nutritionists in their pre-exercise nutritional plans, it is necessary to investigate and understand how pre-exercise nutrition (especially CHO intake) affects the metabolic regulation of substrates as individuals exercise in accordance to such specific biomarkers of performance and exercise capacity [23,24]. Thus, the aim of the present investigation was to assess the impact of High CHO (HC) vs. Low CHO (LC) diets on the oxidative regulation of CHO and lipids while moderately endurance-trained males and females run at V1AT.

Methods

Subjects

Sixteen healthy recreational runners (8 males/8 females) voluntarily took part in this investigational study. The ethics committee of the University of Potsdam approved the study and participants gave their written informed consent after receiving detailed information on the investigational protocol and study aims. To increase the cohort's homogeneity in regards to physical conditioning, subjects, were only included if weekly training was ≥3 hours. Anthropometric characteristics are provided in table 1.

Table 1: Anthropometric data of subjects.

| Subject | Overall (n=16) | Males (n=8) | Females (n=8) | P values |
|---------|---------------|------------|---------------|----------|
| Age (yrs.) | 28 ± 3 | 30 ± 3 | 26 ± 2 | 0.005 |
| Height (m) | 1.76 ± 0.09 | 1.83 ± 0.08 | 1.70 ± 0.03 | 0.001 |
| Weight (kg) | 72 ± 13 | 83 ± 8 | 61 ± 5 | 0.000 |
| BMI (kg/m²) | 23 ± 2 | 24.9 ± 1.1 | 21.2 ± 1.3 | 0.000 |
| %BF | 14.7 ± 3.3 | 14.1 ± 3.5 | 15.3 ± 3.0 | 0.510 |

General design

All examinations were conducted at the Outpatient Clinic from Potsdam University. A full medical check (anamnesis,

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anthropometrics, physical examination, resting ECG) was carried out preceding the first exercise appointment as recommended by the German Federation for Cardiovascular Prevention and Rehabilitation [25]. At day 1, participants performed a baseline running test in which the IAT [26], \( V_{\text{IAT}} \) peak oxygen uptake (VO\(_{2\text{peak}}\)) and maximal Heart Rate (HR\(_{\text{max}}\)) (RS 400, ©Polar Electro, Finland) were determined. On days 2 and 3, a submaximal running test at \( V_{\text{IAT}} \) was carried out on the same treadmill ergometer (H/P/Cosmos Pulsar Graphics 2005®, Germany). A breath-by-breath Metamax 3B system (Cortex Biophysik GmbH, Leipzig, Germany) was used to monitor respiratory data and to determine CHO and lipid oxidation rates via indirect calorimetry (detailed below). For the 2 submaximal runs, HC (7g/kg/d) and LC (3g/kg/d) dietary protocols were prescribed for the 24 hours preceding each test (detailed below). As part of the LC protocol, a glycogen-depleting running bout was additionally performed (60 min at 75% HR\(_{\text{max}}\) in the evening, 12 h prior to the actual submaximal running bout; figure 1 depicts a flowchart of the investigational design). Participants were additionally advised to refrain from any other exercise practices during the 48 hours preceding each submaximal bout.

**Experimental design**

**Baseline test:** Subjects performed a stepwise incremental test until volitional exhaustion. The initial stage (6 km/h), stage increment (2 km/h) and stage duration (3 min) were defined to exhaust subjects in not less than 4 stages [27]. Lactate concentrations were measured in between stages from capillary blood samples taken from the hyperemized earlobe (Biosen S line, EKF diagnostic GmbH, Magdeburg, Germany). The plan was standardized for breakfast, lunch and dinner (plus in between snacks), and consisted of foods typically eaten in Germany. The plan was standardized with no caffeine (with the exception of a standardized morning coffee) alcohol or supplements included, and individually adapted to body mass to achieve CHO aims. As part of the LC protocol, an exercise bout with duration and intensity proven to deplete glycogen stores was implemented [32,33]. This bout combined to the LC diet (which subsequently avoids glycogen recovery or super compensation) [34,35], would then create a metabolic state where low CHO availability can be assumed. The amounts of CHO intake (i.e., 7 vs. 3 g/kg/d) were chosen, as these are common thresholds used in both clinical and scientific settings.

**Gas exchange data analysis & calculations:** Values from respiratory volume and gas concentrations were transmitted directly to the analysis software (Metasoft 3, version 3.9). All tests had the investigated gas exchange parameters viewed with an average time interval of 10 seconds. VO\(_{2\text{peak}}\) was defined as the highest Oxygen uptake (VO\(_2\)) recorded during the baseline test within a period of 30 seconds. For the two submaximal runs, calculations of CHO and lipid oxidation rates were performed using stoichiometric equations in accordance to the non-protein respiratory quotient technique [36].

Lipid oxidation rate (mg/min\(^{-1}\))=-1.7012 VCO\(_2\) + 1.6946 VO\(_2\)

CHO oxidation rate (mg/min\(^{-1}\))=4.585 VCO\(_2\) - 3.2255 VO\(_2\)

This technique provides calculations for substrate oxidation under the assumption that urinary nitrogen excretion is negligible. Markers were set every 5 minutes during the possible 30 minutes of each submaximal exercise bout. Respiratory data as well as CHO and lipid oxidation values were averaged from the last 30 seconds preceding every marker.

**Statistics:** All of the analyzed parameters are descriptively reported as mean and Standard Deviation (±SD). Statistical analysis was performed using a commercial software package SPSS, version 20, IBM, USA and Microsoft Excel 2011. Samples were checked for normality using the Shapiro-Wilk test. Gender differences in anthropometry, baseline parameters and within nutritional protocols were tested with an unpaired t-test. Differences in dietary data, cardiopulmonary parameters as well as differences in substrate oxidation between the trials with different nutritional protocols (including gender comparisons) were computed with a paired t-test. The interaction of the gas-exchange variables between diet and gender was analyzed with a two-way ANOVA for repeated measures (diet x gender). Significance was set at an alpha level of 0.05.
Results

Baseline characteristics: As presented in table 2, the overall values for IAT, \( V_{\text{IAT}} \) and \( HR_{\text{max}} \) were 2.74 ± 0.39 mmol/l, 11.1 ± 1.4 km/h and 194 ± 10 beats/min respectively, with no significant gender differences. \( VO_{2\text{peak}} \) differed significantly between genders with males achieving 50 ± 0 ml/min/kg and females 44 ± 5 ml/min/kg.

|                | Overall | Males | Females | P values |
|----------------|---------|-------|---------|----------|
| \( VO_{2\text{peak}} \) (ml/min/kg) | 47 ± 5  | 50 ± 0 | 44 ± 5  | 0.003    |
| \( HR_{\text{max}} \) (beats/min)   | 194 ± 10 | 193 ± 12 | 195 ± 5  | 0.808    |
| \( V_{\text{IAT}} \) (km/h)          | 11.1 ± 1.4 | 11.4 ± 0.8 | 10.7 ± 1.8 | 0.366    |
| IAT (mmol/l)    | 2.74 ± 0.39 | 2.71 ± 0.43 | 2.77 ± 0.40 | 0.750    |

Table 2: Performance at baseline test.

All values are means±SD; P values reflect gender comparisons only

Dietary intake: Overall CHO intake differed significantly (P = 0.005) between HC (7.1 ± 1.5 g/kg/d) and LC (3.4 ± 0.8 g/kg/d) protocols (Figure 2). Accordingly, the mean deviations from the targeted CHO intake were 2 ± 21% (P = 0.566) and 15 ± 28% (P = 0.001) for HC and LC protocols respectively. As shown in figure 3, during the HC days, intake was 7.0 ± 1.4 g/kg/d for males and 7.2 ± 1.7 g/kg/d for females (P = 0.661), resulting in a deviation of 3.1 ± 0.7 and 3.8 ± 0.9 g/kg/d (P = 0.078), which reflects a target deviation of 3 ± 24% (P = 0.520) and 26 ± 29% (P = 0.003) respectively.

Cardiopulmonary parameters during HC and LC bouts: As shown in figure 4a, with no significant differences between bouts at any of the measured points (P = 0.756 at rest; P = 0.768 at 5 min; P = 0.145 at 10 min; P = 0.067 at 15 min; P = 0.069 at 20 min; P = 0.089 at 25 min; P = 0.079 at 30 min), the overall \( HR \) ranged from 161 ± 11 (at 5 min) to 176 ± 13 beats/min (at 30 min; P = 0.001) during the HC bout, and from 165 ± 12 to 178 ± 11 beats/min (P = 0.007) during the LC bout respectively. Mean \( VO_{2} \) was 38 ± 5 and 39 ± 5 ml/min/kg during HC and LC bouts respectively (P = 0.086 at 5 min; P = 0.060 at 10 min; P = 0.189 at 15 min; P = 0.518 at 20 min; P = 0.059 at 25 min; P = 0.132 at 30 min; figure 4b). The Respiratory Exchange Ratio (RER) was significantly higher during the HC bout at all measure points (P = 0.006 at 5 min; P = 0.000 at 10 min; P = 0.003 at 15 min; P = 0.001 at 20 min; P = 0.007 at 25 min) but the last (P = 0.059 at 30 min; figure 4c).

CHO and lipid oxidation: Relative (%) and absolute (g/min) values for overall CHO and lipid oxidation recorded during the two submaximal runs are presented in figure 5a-d. Substrate oxidation differed significantly between HC and LC bouts at...
minutes 5 to 25 but not at minute 30 (P = 0.000; P = 0.001; P = 0.010; P = 0.003; P = 0.059). CHO oxidation was on average 3.45 ± 0.08 and 2.90 ± 0.07 g/min (P = 0.000) during HC and LC bouts respectively. Likewise, lipid oxidation rates were 0.13 ± 0.03 and 0.36 ± 0.03 g/min (P = 0.000). CHO metabolism accounted for 84 ± 15 and 72 ± 20% (P = 0.000) of the overall oxidized substrates during both HC and LC bouts respectively. Figure 6a-d displays gender differences in the amount of oxidized substrates during the runs and relative to overall substrate use. When comparing CHO and lipid oxidation within each of the two nutritional states, significant gender differences could only be shown during the HC run, and at the measurement times of 5, 10, 25 and 30 minutes (P = 0.002; P = 0.004; P = 0.017; P = 0.006), but inconsistently at minutes 15 and 20 (P = 0.066; P = 0.059). The relative contribution of CHOs to the overall oxidative metabolism was greater in males compared to females (i.e., 90 ± 11% vs. 76 ± 16% (P = 0.033) in the HC run and 77 ± 13% vs. 65 ± 24% (P = 0.059) in the LC run respectively). Consistently, the relative contribution of lipids was higher in females compared to males (i.e., 24 ± 16% vs. 10 ± 11% (P = 0.033) in the HC run and 35 ± 24% vs. 23 ± 13% (P = 0.059) in the LC run respectively). The analysis of interaction effects between nutrition and gender resulted in non-significant findings (P = 0.766).

Discussion

The present study analyzed the oxidative regulation of CHOs and lipids in a group of recreational runners as they performed 2 running bouts with standardized intensity at V_{\text{IAT}}. Participants were well fed with CHOs (HC protocol) or presumably, in a metabolic state of reduced CHO availability (LC protocol) before completing each bout. Baseline results indicate a fairly homogeneous physical conditioning among subjects as no significant gender differences were observed for IAT (mmol/l) or V_{\text{IAT}} (km/h). Throughout the 30 minutes submaximal runs, overall HR increased constantly and equally (~80 to 90% HR_{\text{max}}), with high but steady-state VO_{2} recordings (~80% VO_{2\text{max}}). Yet, as clearly depicted in the overall ventilatory response to exercise (Figure 4c), the applied dietary scheme has influenced the oxidative regulation of substrates, with CHO metabolism prevailing throughout runs. Overall, CHO oxidation was 0.55 g/min (~16%; P = 0.000) greater during the HC compared to the LC run. Conversely, lipid oxidation was 0.23 g/min (~64%; P = 0.000) greater during the LC compared to the HC bout. In relation to the overall energy metabolism, these differences reflect a significant increase of 12% in the oxidative activity of each substrate depending on which dietary protocol had been implemented.

At a gender level, CHO oxidation was also predominant, though females were able to consistently oxidize more lipids than males under both conditions and throughout the entire duration of bouts (i.e., 14 and 12% greater lipid oxidation during HC and LC bouts, respectively). Males on their side had higher CHO oxidation rates computed at both conditions, with highest and significant differences being recorded during the HC bout. It should be noted nonetheless, that during the LC bout lipid oxidation might have even been suppressed in females, as CHO intake was exceeded in 26% (P = 0.003) compared to only a 3% (P = 0.520) extrapolation by males. However, as glycogen itself was not measured, it cannot be completely assured whether the performed pre-exercise protocols had any effect on glycogen concentrations or its subsequent utilization during exercise. Nevertheless, as pointed out by Andrews et al., [13], the significantly higher RER recorded in the HC bout are reflective of a higher rate of CHO metabolism, indicating that

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when CHO is made available through pre-exercise loading, one will also preferentially utilize CHO. In addition, a significantly higher VO₂ capacity from males at both baseline (12%) and during the submaximal bouts (~15%) could partly explain why males consistently oxidized more CHOs [18], even though relative to VO₂peak, exercise was performed at the same intensity by both genders (i.e., 41 ± 2 vs. 35 ± 6 ml/min/kg and 42 ± 2 vs. 36 ± 5 ml/min/kg for males and females during HC and LC bouts respectively).

Physiological explanations to the observed findings suggest that increasing endogenous CHO availability will result in a greater muscle glycogenolysis and/or muscle glucose uptake, thus preventing a decline in blood glucose concentration during subsequent exercise, ultimately favoring a CHO driven metabolism while lipid oxidation is partially inhibited [13,14,37]. Moreover, during constant exercise at V̇O₂peak, anaerobic glycolysis is enhanced (fuelled almost exclusively from plasma glucose entering the muscle fiber via facilitated diffusion and the glucose transporter type 4, or from glucose-phosphate provided through glycogenolysis from muscle glycogen), and provides a constantly increasing portion of the energy yield [19,20]. Still, why females burn more lipids than males even at high exertion levels remains debatable. Plausible explanations in literature imply that a variety of factors such as the distribution and activation of α and β-adrenergic receptors, aerobic capacity but mostly endocrine mediated responses, predispose females to have a greater reliance on lipid oxidation compared to males [13,38,39]. Additionally, glycogen supercompensation occurs to a smaller extent in females compared to males [40], thereby directly affecting its subsequent availability for oxidation.

To our knowledge, the current investigation is the first to analyze how a simple, 24-hour manipulation of CHO intake may affect substrate oxidation during a constant, high-intensity running bout at V̇O₂peak. In this sense, we would like to point out some plausible practical implications to our findings before making appraisals to previous investigations as well as raising a few prospective questions. Coaches, trainers and nutritionists should be aware of the reported oxidative patterns and how those ultimately influence the empying rates of glycogen (or glycogen sparing for that matter, as well as how those may influence high-intensity training and competition performance, which still remain to be established), and therefore, reinforce an individual and gender-based approach to pre-exercise nutrition. For example, as females show a greater reliance on lipid metabolism compared to males, in spite of similar (or greater) systemic CHO loading. In addition, they should be attentive when planning training strategies to the fact, that an identical bout of exercise may result in different metabolic reactions and may thus, cause different metabolic adaptations to training. Unfortunately, as shown by Wissman & Willoughby [41], only a limited amount of studies [40–42] have reported on substrate oxidation while combining CHO-loading strategies and exercising conditions that are similar to the ones applied in the present investigation. Tarnopolsky et al. [42] reported on gender differences in substrate oxidation when CHO intake was increased from 55 to 75% of the total energy intake during 4 days prior to exercise. They showed that when cycling at 75% VO₂peak for 60 minutes, females oxidized significantly more lipids and less CHOs compared to males. However, these findings should be critically interpreted as CHO intake was not prescribed relative to body weight, and consequently males ended up having a higher intake than females (i.e., 8.2 and 6.4 g/kg/d respectively). In one other study, Walker et al. [40] used a CHO-loading strategy consisting of moderate (4.7 g/kg/d) and high (8.2 g/kg/d) intakes of CHO for 7 days before participants (females only) cycled at ~80% VO₂max until volitional exhaustion. Results, which account for the first 75 minutes of exercise, reveal a significant increase of 0.44 g/min (~16%) in CHO oxidation during the high compared to the moderate intake bout. Conversely, lipid oxidation was 0.17 g/min (~40%) greater during the moderate intake bout (P < 0.05). In this particular study, muscle glycogen increased 13% after the high compared to the moderate CHO protocol. Though significant, the magnitude of this supercompensation was still smaller than those previously observed in male athletes [40]. Other investigations, for instance, the so-called “Train Low” studies, have reported on the effects of training in a glycogen-depleted state and found it to be an effective strategy to increase lipid metabolism in athletes [43,44]. However, the benefits of such a protocol remains debatable, as no gains in endurance performance have been consistently observed [24,43]. Moreover, as recently highlighted by Scharhag-Rosenberger [24], such a training strategy may induce a down-regulation in CHO metabolism, which consequently hinders the body’s ability to make use of the potentially spared glycogen stores. Therefore, it would be of interest for prospective studies to investigate the effects of systematic training at V̇O₂peak (e.g., on substrate oxidation activity and adaptability over time, as well as against performance time-trials or bouts until volitional exhaustion) whilst subjects are well fed with CHOs or in a metabolic state of reduced CHO (glycogen) availability.

Lastly, we would like to acknowledge a few limitations of the current study. Female’s menstrual cycle was not controlled. Therefore we cannot account on the eumenorrheic or amenorrheic status of the assessed female participants, as well as whether and how the follicular or luteal phases of their menstrual cycle could have influenced substrate oxidation. Dietary intake was only controlled for the 24 hours prior to each submaximal exercise bout. In addition, due to methodological limitations, glycogen levels were not objectively assessed. Hence, we cannot assure if previous nutrient intake (i.e., outside of the controlled 24 hours) would have resulted in significant additional accumulation CHOs, or whether that could have influenced glycogen concentrations and subsequently substrate utilization. Future studies should in this case control for baseline glycogen levels before nutritional interventions are began and introduce longer dietary control

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periods. Still, our combined protocols of controlled dietary and exercise regimes have certainly brought subjects into the intended acute metabolic states of high and low systemic CHO availability.

Conclusion

The current investigation aimed in providing more evidence and a better understanding on the metabolic regulation of substrates when pre-exercise CHO intake is manipulated and exercise is performed at a level of upper endurance capacity. Our findings suggest that 24 hours of high CHO consumption results in concurrent higher CHO oxidation rates and overall utilization, whereas maintaining a low systemic CHO availability significantly increases the contribution of lipids to the overall energy metabolism. The observed gender differences clearly underline the necessity of individualized dietary planning before exerting at intensities associated with performance exercise (e.g., prolonged exercise at V\textsc{\textsubscript{IAT}}). Ultimately, it remains to be established how these findings can be extrapolated to training and competitive situations and with that provide trainers and nutritionists with improved data to derive training prescriptions.

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