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Fatigue in Final Hour of an Ironman Triathlon with Absence of Carbohydrate Supplementation: A Retrospective Case Study

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Abstract

International Journal of Exercise Science 19(6): 1-11, 2026. This is a retrospective single-case analysis that describes pacing and physiological responses in a highly trained keto-adapted triathlete during an Ironman triathlon using descriptive statistics only. Exercise-induced hypoglycemia (EIH) may be a primary contributor to fatigue and impaired performance during prolonged exercise. As there are only 4-5 grams of glucose circulating in the bloodstream, minimal carbohydrate supplementation may be able to maintain euglycemia during endurance and ultra-endurance competition. The purpose of this case study was to retrospectively examine the use of minimal carbohydrate supplementation (≤10 grams/hour) in a highly trained, keto-adapted triathlete on pace maintenance, average heart rate (AHR), and maximal heart rate (HRmax). Percent change was calculated for the first, middle, and last 30 minutes of each of the three disciplines to assess pacing and physiological measurements. Percent deviation from the overall discipline average was calculated for each measure of interest to evaluate performance stability. Despite minimal deviation in AHR and HRmax during the final 30 minutes of the run (0.71% and 0.14%, respectively), there was an observable decline in pace (13.63% deviation and 23.40% slower pace from first to last 30 minutes) when the athlete stopped ingesting CHO during the last hour, coinciding with the onset of mild symptoms consistent with EIH upon the completion of the triathlon. These findings are observational and suggest the continued ingestion of minimal CHO may have helped maintain euglycemia and potentially preserved pace during the final hour of the race, although fatigue may also have been due to other factors.

Keywords: Ketogenic diet, intra-competition nutrition strategy, metabolic flexibility

Introduction

Historically, muscle glycogen depletion has been thought to be the primary contributor of fatigue during endurance and ultra-endurance events.[1-3] According to this view, muscle glycogen stores must be maximized and maintained prior to and during competition to mitigate fatigue and a subsequent decline in performance. Due to depletion of muscle glycogen stores during prolonged endurance exercise, athletes are often advised to consume large amounts of carbohydrate (CHO), with current guidelines recommending up to 120 grams per hour in order to maintain the exercise intensity,[4-6] although optimal rates vary depending on exercise intensity, athlete size, and environmental factors.[7,8] A growing amount of research suggests, however, that

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fatigue during prolonged exercise may be more closely linked to exercise-induced hypoglycemia (EIH) and inadequate hepatic glucose output than to muscle glycogen depletion.[9,10,12] In order to mitigate the detrimental effects of EIH, euglycemia must be maintained in the bloodstream. Maintaining euglycemia ensures adequate glucose supply to the brain thereby supporting central nervous system (CNS) function–the primary regulator of fatigue.[11,12] Preserving cerebral glucose availability may help sustain the CNS regulation of hepatic glucose output, reinforcing glucose homeostasis and thus may delay the onset of fatigue.[8] Furthermore, the CHO amount required to maintain euglycemia may be far less than current recommendations suggest for hourly CHO consumption during competition. Given that euglycemia requires 4-5 grams of glucose in the bloodstream, small amounts of exogenous CHO may be sufficient to support the blood glucose pool and preserve liver glycogen. Additionally, in athletes adapted to a low-carbohydrate, high-fat (LCHF) diet, enhanced beta oxidation promotes greater use of free fatty acids as fuel, helping preserve liver glycogen during prolonged exercise.[13]

In light of this model of fatigue, recent research shows minimal CHO supplementation (<10 g/hour) may mitigate symptoms of hypoglycemia and improve time to exhaustion (TTE) performance in athletes following LCHF or a high-carbohydrate, low-fat diet (HCLF).[14] This research suggests that minimal supplementation may enhance endurance performance while preserving high rates of fat oxidation, thereby minimizing disruption to underlying substrate metabolism and sparing liver glycogen stores. The mitigation of EIH may lead to increased affect and decreased ratings of perceived exertion (RPE)[15-17] as well as physiological measures of fatigue and hypoglycemia.[14,18] An Ironman triathlon (3.9 km swim, 180.2 km bike, 42.2 km run) requires a sustained effort across multiple disciplines, requiring a well-planned nutritional strategy for athletes over the course of the event. The purpose of this case study, therefore, is to evaluate the physiological effects of minimal CHO ingestion (approximately 4-6 g/30 minutes) during an Ironman triathlon in a well-trained, keto-adapted athlete, with specific attention to heart rate, power output, and pace maintenance over the course of the event, as well as symptoms of EIH upon the completion of the triathlon. As an observational case study without direct metabolic measures, our findings should be interpreted as hypothesis-generating. This descriptive, hypothesis-generating case analysis examines pacing, heart rate, and symptoms consistent with EIH during an Ironman triathlon.

Methods

Participant

A former professional male triathlete (age: 55, height: 184 centimeters, weight: 70 kg, VO_2max: 69 mL/kg/min-1 [measured via indirect calorimetry during a maximal graded exercise test at an external laboratory]) was recruited and interviewed for the study. Written informed consent was obtained for participation and publication, all procedures were conducted in accordance with the Declaration of Helsinki, and this was carried out in accordance with the ethical standards of the *International Journal of Exercise Science*, acknowledging alignment with the ethical policies of IJES.[19] As this was a retrospective single case analysis involving no intervention or identifiable health information, institutional review was not required under local policy. The participant provided written informed consent for the use of his de-identified data and for publication of this case report. The subject has competed in multiple Ironman triathlons over the course of the last 35 years, with 16 years of experience as a professional athlete. He continues to compete in both Ironman and Olympic distance triathlons, training 15-25 hours/week at approximately 80% zone 1 (50-60% HR_{max}), 15% zone 2 (60-70% HR_{max}), and 5% zone 3 (70-80% HR_{max}). For the last 12 years, his diet has consisted of a cyclic LCHF diet. The participant reported consuming approximately 20-25 g/day CHO during ketogenic phases and 50-100 g/day during refeed phases, the participant consumed approximately 30% CHO, 50% fat, and 20% protein. The participant reported no current medications, no recent injury, and no known metabolic disease at time of race completion and data analysis.

Protocol

During a recent Ironman competition, the subject consumed minimal CHO (<10 grams/hour), while recording average heart rate (AHR), maximum heart rate (HR_{max}), wattage while cycling (W), and pace (min/km). The CHO dosage was selected to support the relatively small blood glucose pool, estimated at 4-5 grams, with the participant alternating between consuming 4 or 6 grams of CHO every 30 minutes via GU Roctane Ultra Endurance Energy Gel or a cola containing sugar. This corresponds to an average intake of approximately 10 g/h during cycling (50 g over approximately 5 h) and 8.6 g/h during running overall (30 g over approximately 3.5 h) with 0 g consumed during the final hour of the run (figure 1). Because this is a retrospective single-race case analysis, no randomization or counterbalancing of supplementation conditions was performed. The only exceptions occurred during the swimming portion and the last hour of the running portion, when the subject did not consume any CHO. Heart rate and pace were recorded using a smartwatch (Garmin Forerunner 945, Garmin Ltd, Olathe, Kansas, USA) while power output while cycling was measured with a Quarq power meter (SRAM, Chicago, Illinois, USA). The swim portion lasted approximately 1 hour, the bike approximately 5 hours, and the run approximately 3.5 hours, for a total time of approximately 9.75 hours (accounting for approximately 15 minutes for transitional periods between disciplines). Ambient conditions during the event were approximately 16.1˚C and 53% humidity. Hydration status was not tracked or reported by the participant.

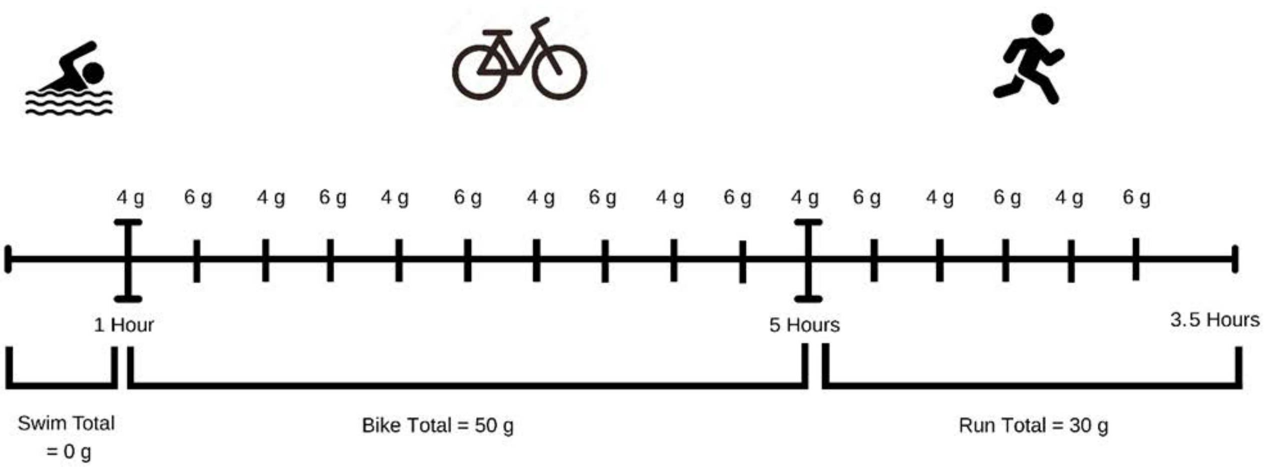

Figure 1. The above figure shows the dosages of CHO consumed at each timepoint. In total the participant consumed 0 g during the swim, 50 g during the bike, and 30 g during the run, for a total of 80 grams.

Lastly, symptoms of EIH (i.e., pounding heart, shivering, hunger, etc.) were evaluated using the Edinburgh Hypoglycemia Symptom Scale.[20] Symptoms were assessed on a scale of 1 to 7 following the completion of the race, with 1 indicating that the participant did not experience the symptom at all and a 7 indicating a significant presence of the symptom. The participant rated these symptoms as felt immediately upon completion of the triathlon, with particular reference to the last hour of the run.

Statistical Analysis

Percent change comparing the analyzed time points was calculated for each physiological measurement (Table 2). This calculation allows for the comparison of changes in measures of interest across the duration of each leg of the triathlon, indicating when the athlete may have begun to experience fatigue, leading to a decline in the ability to maintain pace. All analyses were descriptive; no inferential statistics were performed. Percent change and deviation formulas followed established single-case sport performance methods. Calculations were completed with Microsoft Excel 365. The following formulas were used:

$$\text{Percent change} = \frac{(\text{Segment } 2 - \text{Segment } 1)}{|\text{Segment } 1|} \times 100$$

In addition to percent change, percent deviation from the discipline mean was calculated for AHR, HR_{max}, and pace as well as power output for the cycling portion (Table 3). These enabled comparisons of intra-discipline variability to identify physiological patterns indicative of fatigue or performance preservation under minimal CHO consumption. The following formula was used:

$$Percent\ deviation = \frac{(Segment\ Value - Mean)}{Mean} \times 100$$

Results

Perceived EIH Symptoms

The participant scored 25/126 on the Edinburgh Hypoglycemia Symptom Scale. He reported mild ratings (2/7) for pounding heart, hunger, blurred vision, shivering, anxiety, inability to concentrate, and feeling tearful upon the completion of the race.

Physiological Measurement Averages

Average heart rate during the overall duration of the race was 146.5 beats per minute (bpm). The average pace during the swim was 15:15 min/km, 1:36 min/km during cycling, and 5:26 min/km during the run. Average power output during cycling was 211.70 W (Table 1).

Table 1. The table below shows the AHR, HRmax, pace, and wattage (W) for M1, M2, and M3 of each discipline of the Ironman. M1/M2/M3 represent equal 30-minute segment within each discipline; swim includes M1 and M3 due to a 60-minute duration.

	AHR (bpm)	HR_{max} (bpm)	Pace	Wattage (W)
Swimming				
M1	141.0	155.0	15:12 min/km	
M3	144.0	155.0	15:18 min/km	
Overall Average	142.5	155.0	15:15 min/km	
Cycling				
M1	146.0	156.0	1:43 min/km	211.0
M2	148.0	157.0	1:32 min/km	211.0
M3	141.0	153.0	1:43 min/km	213.0
Overall Average	145.0	155.3	1:36 min/km	211.7
Running				
M1	156.0	160.0	5:00 min/km	
M2	153.0	154.0	5:02 min/km	
M3	154.0	156.0	6:10 min/km	
Overall Average	154.2	157.1	5:26 min/km	

Note: AHR = average heart rate during the segment, HR_{max}= maximal heart rate during segment; M1 = first 30-minutes of discipline, M2 = middle 30-minutes of discipline; M3 = last 30-minutes of discipline.

Carbohydrate Consumption, Percent Change, and Percent Deviation

CHO consumption varied from 0 to 6 grams every 30 minutes, resulting in a total consumption of 80 grams. No CHO was consumed during the swim portion or the last hour of the run. Despite the low CHO intake, both pace and power output were maintained during the cycling segment (Tables 2 and 3) suggesting that ≤ 10 grams/hour of exogenous CHO may have been sufficient to maintain euglycemia during the duration of these disciplines. Pace was preserved during the first two-thirds of the run when CHO was ingested, but the absence of supplementation during the last hour coincided with noticeable fatigue, as demonstrated by the observed decrease in pace, which may have been related to EIH, though other explanations such as general exertion cannot be excluded (Tables 2 and 3).

Table 2 shows the largest percent change in average heart rate (AHR) occurred from M2 to M3 (-4.73%) during the cycling portion, and the largest percent deviation from the leg average during M2 occurred during the cycling portion (Table 3). The smallest percent change occurred from M2 to M3 of the run (0.65%) and the smallest percent deviation during M3 (0.14%). The greatest change in maximal heart rate (HR_{max}) was observed from M1 to M2 during the run (-3.75%) while the largest percent deviation was during M2 of the run (1.98%). The smallest percent change and percent deviation occurred during the swim (0.00%). M1 to M3 during the run had the largest percent change for pace (23.40%), and the largest percent deviation was M3 of the run (13.63%). The smallest percent change occurred during M1 to M2 of the run (0.60%), while the smallest percent deviation occurred during the swim (0.33%). Cycling wattage showed minimal variation across all intervals with the greatest percent change being 0.95% from M1-M3 and M2-M3 and the largest percent deviation of 0.61% during M1 and M2.

Table 2. Percent change in heart rate (AHR, HRmax), pace, and cycling wattage between the first (M1), middle (M2), and last (M3) 30-minute intervals of each discipline. The greatest changes were observed in running pace (M1-M3: +23.4%) while cycling wattage remained stable across all intervals (<1% variation).

Percent Change Comparison	AHR (%)	HR_{max} (%)	Pace (%)	Wattage (%)
Swimming				
M1-M3	2.13	0.00	0.66	
Cycling				
M1-M2	1.37	0.64	-11.04	0.00
M1-M3	-3.42	-1.92	0.00	0.95
M2-M3	-4.73	-2.54	12.41	0.95
Running				
M1-M2	-1.92	-3.75	0.60	
M1-M3	-1.28	-2.50	23.40	
M2-M3	0.65	1.30	22.66	

Note: AHR = average heart rate during the segment, HR_{max} = maximal heart rate during segment; M1 = first 30-minutes of discipline, M2 = middle 30-minutes of discipline; M3 = last 30-minutes of discipline.

Table 3. Percent deviation from the mean for AHR, HR_{max}, pace and cycling wattage during each segment of the race. Swimming yielded the lowest variability across all measurements, while running (particularly M3) showed the greatest deviation from the athlete's average pace.

Percent Deviation	AHR (%)	HR_{max} (%)	Pace (%)	Wattage (%)
Swimming				
M1	1.05	0.00	0.33	
M3	1.05	0.00	0.33	
Cycling				

M1	2.33	0.45	7.50	0.33
M2	3.73	1.09	4.38	0.33
M3	1.17	1.48	7.50	0.61
Running				
M1	1.15	1.84	7.91	
M2	0.80	1.98	7.37	
M3	0.14	0.71	13.63	

Note: AHR = average heart rate during the segment, HR_{max} = maximal heart rate during segment; M1 = first 30-minutes of discipline, M2 = middle 30-minutes of discipline; M3 = last 30-minutes of discipline.

Discussion

The purpose of this case study was to observe how minimal CHO supplementation (≤10 grams/hour) affected physiological markers of fatigue−namely, heart rate and pace−in a fat-adapted triathlete during an Ironman triathlon. The primary finding was that minimal CHO supplementation may have helped mitigate fatigue for 2 out of the 3 disciplines, as changes in physiological measures were minimal (<10%) during both the swimming and cycling legs. During the run, heart rate and pace remained consistent when CHO was ingested. When CHO ceased during the last hour of the race, however, a marked decrease in pace (M1-M3: +23.40%) was observed with the presence of mild EIH symptoms, despite minimal changes in AHR and HR_{max}. These findings suggest that continued or greater CHO intake during the final hour may have helped preserve pace.

This observed reduction in pace with ceased CHO supplementation and without noticeable deviations in heart rate is perhaps the most notable observation. The subject was able to maintain pace during the first two-thirds of the run when CHO was supplemented but lost the ability to maintain pace during the last third without CHO. Although muscle glycogen depletion is traditionally implicated as the primary driver of fatigue during endurance exercise, the mild symptoms on the Edinburgh Scale support the potential presence of EIH, but this observation cannot confirm causality. Furthermore, despite a steep decline in pace during the final hour, AHR (0.14%) and HRmax (0.71%) deviated only minimally from the segment mean, suggesting that the fatigue experienced was unlikely due to cardiovascular fatigue. This pattern is consistent with central fatigue,[11,12] potentially related to inadequate hepatic glucose output.[9,10,21] We speculate that euglycemia may not have been maintained, consistent with the possibility of EIH contributing to fatigue, although alternative explanations (including environmental heat, dehydration, gastrointestinal distress, or musculoskeletal fatigue) remain possible and were not directly measured.[7,8] Collectively, these findings align with the hypothesis that even very low CHO intake may help preserve pacing during prolonged exercise by preventing EIH, but continued CHO intake is required to maintain pace and performance.[21]

The observed decline in pace, despite a stable heart rate, suggests that muscle glycogen depletion was unlikely to be the primary cause of fatigue. Muscle glycogen depletion often results in an increase in catecholamines, which, in turn, increases maximal heart rate.[21,22] As shown in Tables 2 and 3, HR_{max} deviated very minimally from the overall mean HR_{max} during each leg of the triathlon, despite minimal or no CHO consumption. These findings suggest that the fatigue experienced may not have been primarily driven by muscle glycogen depletion, but rather by alternative mechanisms such as impaired hepatic glucose output[9,10,13,21]or reduced central nervous system[11,12] glucose availability.[12,13] Furthermore, the subject's cyclic ketogenic diet may have promoted elevated rates of fat oxidation and ketogenesis, providing alternative fuel substates such as fatty acids and ketone bodies. This may have reduced reliance on hepatic glucose output by partially supplanting cerebral glucose utilization, thereby helping to preserve liver glycogen stores during prolonged exercise.[13,15] These findings suggest that cardiovascular limitations were unlikely and that central nervous system factors, potentially related to impaired cerebral glucose supply, may have contributed to the observed fatigue.

While substantial evidence supports the performance benefits of carbohydrate supplementation across a broad range of intakes (<10 g/h to >90 g/h) in athletes following high-carbohydrate diets,[24-26] emerging research suggests that even low-dose carbohydrate supplementation may also provide performance benefits in athletes adapted to low-carbohydrate, high-fat (LCHF) diets.[14,17,18,26] Webster et al., 2018[18] reported that a fat-adapted athlete performing a 20 km cycling time trial, carbohydrate ingestion at 60 grams/hour improved performance by approximately 1.1% compared to a water-only condition. Similarly, Prins et al., 2024[14] demonstrated that carbohydrate ingestion at 10 grams/hour improved TTE performance by approximately 30% in fat-adapted athletes. These findings suggest that even relatively low doses of CHO may provide meaningful performance benefits in keto-adapted athletes by supporting euglycemia without substantially impairing fat oxidation. This case study builds on that literature by suggesting that even minimal CHO intake (10 g/h) may be sufficient to stabilize physiological performance in some keto-adapted athletes; however, individualized testing with continuous glucose monitoring is recommended.

There are several limitations to this study. First, no direct metabolic markers (blood glucose, ketones, glycogen) were obtained; interpretations remain speculative. This lack of direct metabolic markers limits our ability to determine whether the observed fatigue was due to EIH, glycogen depletion, or other factors. Additionally, environmental and hydration variables were not quantified, and this retrospective single-subject design limits generalizability. As such, the interpretations herein should be considered speculative and hypothesis-generating only. Future research should look to measure these variables using randomized controlled trials. Second, this was a retrospective, single-subject case design, and thus many third variables could affect the results. Future studies should aim to directly measure physiological and metabolic variables in real-time, rather than relying on retrospective, self-reported metrics. Third, the nature of case studies limits the generalizability of findings. Future research should look to confirm these findings with blinded, randomized controlled trials with a significantly larger sample size. Future studies should incorporate measures of perceptual responses, substrate utilization, muscle and liver glycogen content, and enzymatic regulators of metabolism to better distinguish the contributions of glycogen depletion versus EIH in mediating fatigue, and to further clarify the mechanisms by which CHO supplementation influences performance in fat-adapted athletes.

These observational findings suggest that minimal CHO supplementation may help a fat-adapted athlete maintain pacing during an ultra-endurance event, though controlled trials with direct metabolic measures are needed to confirm mechanisms. Furthermore, these findings suggest that even fat-adapted athletes may benefit from CHO supplementation during prolonged exercise. While CHO supplementation is beneficial to fat-adapted athletes, the amount needed to mitigate fatigue may be lower than current CHO supplementation guidelines for endurance and ultra-endurance athletes. However, further research is needed to clarify the mechanisms contributing to fatigue (i.e., glycogen depletion vs. EIH) and to define optimal CHO dosing strategies to support performance.

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