Are very high rates of exogenous carbohydrate ingestion (>90 g/hr) sufficient or indeed necessary to run a sub-2hr marathon? An analysis of the model predictions of Lukasiewicz and colleagues

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Introduction

Lukasiewicz et al. (1) have published a model which predicts that to run a sub-2hr marathon (sub2hrM), prospective male and female runners will need to oxidize ingested (exogenous) carbohydrate (CHO) at respective rates of 93 and 108 g/hr during that race. Their predictions are dependent on the assumptions of their model.

The ability to run at 21.1 km/hr for 2 h requires high rates of (obligatory) CHO oxidation

Their first assumption is that to run at 21.1 km/hr for 2 h requires a high rate of CHO oxidation, intitially from muscle glycogen. But once muscle glycogen concentrations fall below 32% of the starting value, to prevent any slowing of pace thereafter, essentially all the runners' energy must come from the oxidation of ingested (exogenous) CHO with a small contribution from liver glycogen. Table 1 details their core predictions, in particular rates of exogenous CHO oxidation required to run a sub2hrM. These predictions raise a number of interesting points.

Estimations of muscle glycogen use during the first 90−120 min of a sub2hrM attempt in CHO-adapted athletes

Based on the reported range associated with performance decrements, the model predicts that exercise performance deteriorates once the muscle glycogen content falls below 32% of its starting value which, accordingly to the calculated endogenous glycogen stores remaining at the point of slowing, would leave 221 and 160 grams in males and females respectively (Table 1, Row F).

TABLE 1 Modeled rates of total energy expenditure (A); energy from CHO (B, C) and fat oxidation (D); muscle glycogen content at capacity (start of exercise) (E); muscle glycogen use during exercise (F, G); model predicted additional CHO oxidation from blood glucose and exogenous CHO to balance total CHO requirement (H); model predicted CHO contribution from liver glucose disappearance (I); corrected model predicted rates of exogenous CHO contribution to balance total CHO requirement if liver glucose disappearance is 0 g (J); model predicted rates of exogenous CHO oxidation to balance total CHO oxidation (K); model predicted rates of exogenous CHO oxidation required to balance total CHO requirements for runners of different weights (L).

		Male	Female
A	Total energy expenditure (kJ/min) [Calculated from Calculated caloric cost, kcal – Table 2 in Lukasiewicz et al. (1)]	88.3	75.7
B	Model predicted energy from CHO oxidation (g/min) [Table 2 in Lukasiewicz et al. (1)]	5.1	4.4
C	Model predicted total CHO oxidation (g) during 120 min exercise. (Row B × 120)	612	528
D	Model predicted energy from fat oxidation (g/min) [Table 2 in Lukasiewicz et al. (1)].	0.07	0.06
E	Muscle glycogen (g) at capacity (start of exercise) [Table 3 in Lukasiewicz et al. (1)]	690	499
F	Muscle glycogen (g) remaining if exercise terminates (1) when muscle glycogen concentration falls to 32% or (2) to 20% of the starting concentration	1. 221 2. 138 Difference: 83g	1. 160 2. 100 Difference: 60g
G	Total muscle glycogen use (g) if exercise terminates (1) at 32% or (2) at 20% of starting muscle glycogen concentration.	1. 469 2. 552	1. 339 2. 399
H	Model predicted additional CHO requirement from blood glucose and exogenous CHO (g) contribution to balance total CHO requirement according to the authors' model that exercise terminates when muscle glycogen concentration drops (1) below 32% or (2) 20% of the starting concentration (Row C minus Row G)	1. 143 2. 60	1. 189 2. 129
I	Model predicted CHO contribution from liver glucose (g) disappearance [Figures 4A, B in Lukasiewicz et al. (1)]	68	49
J	Corrected model predicted total exogenous CHO oxidation (g) required to balance total CHO requirement if hepatic glucose disappearance is 0 g (not the values listed in Row I) (Row H plus Row I).	1. 211 2. 128	1. 238 2. 178
K	Model predicted exogenous CHO (g/hr) required to balance CHO requirement [Figure 6A in Lukasiewicz et al. (1)].	90	106
L	Model predicted rates of exogenous CHO oxidation (g/min) required to balance total CHO requirements for runners of different weights [From Figure 6C in Lukasiewicz et al. (1)].	70 (50 kg) 82 (54 kg) 105 (58 kg) 120 (62 kg)	90 (42 kg) 103 (46 kg) 114 (50 kg) 128 (54 kg)

Rows A–D: Model predicted CHO and fat oxidation rates. Row E: Muscle glycogen content at capacity (start of exercise). Rows F–G: Contribution of muscle glycogen disappearance to total CHO oxidation if fatigue develops at (1) 32% or (2) 20% of starting muscle glycogen concentration. Row H: Model predicted CHO in addition to that from muscle glycogen oxidation required to balance the total required CHO oxidation under two conditions of muscle glycogen disappearance. The (2) in Rows F–H, and J refers to values if fatigue occurs when muscle glycogen is depleted to 20% of the starting concentration. Row I: Predicted CHO contribution from liver glucose disappearance. From Figures 4A, B in Lukasiewicz et al. (1). Row J: Corrected model predicted rates of exogenous CHO oxidation needed to balance total CHO demand if liver glucose disappearance provides 0g to total CHO requirement. Row K: Model predicted rates of exogenous CHO oxidation required to balance CHO requirement [Figure 6A in Lukasiewicz et al. (1)]. Row L: Model predicted exogenous CHO oxidation rates to balance the total CHO requirement for runners of different ages and either sex. From Figure 6C in Lukasiewicz et al. (1).

The authors also argue that "*running activates only approximately 68% of the total lower limb muscle (sic);*" thus fatigue develops when glycogen depletion occurs in those 68% of all lower limb muscle fibers.

But is it possible to run at >90% VO$_2$max (2) whilst recruiting just 68% of all the quadriceps muscle fibers (as opposed to 68% of all the muscle fibers in the lower limb)? This is relevant because most studies measure exercise-induced changes in muscle glycogen concentrations in that muscle, rather than in all the lower limb muscles. The authors derive their value of 68% from a study (3) of young female physical education students, described as recreational runners with a mean VO$_2$max of 49 ml/kg/min. Yet Sale (4) has calculated that 100% of the quadriceps muscle fibers are activated when cycling at >85%VO$_2$max [Figure 2; page 99 in Sale (4)]. Perhaps the value of 68% is not realistic for world class male and

female athletes competing in the sub2hrM attempt. However, as we show, which ever percentage is chosen, it has little or no influence on the model's predictions.

In the original study (5), on which this model of Lukasiewietz et al. (1) is ultimately based—specifically that there is an obligatory role for CHO oxidation to sustain endurance exercise performance (6)—subjects terminated exercise when their muscle glycogen concentrations were 13%, 10%, and 20% of their starting concentrations following 3 different diets. If subjects in this model terminated exercise with muscle glycogen concentrations reduced to 20% and not 32% of starting concentrations, an additional 83 and 60 g CHO become available for the male and female athletes respectively (Table 1, Row F) leaving a residual 60 g CHO (30 g/hr) in males and 129 g CHO (64.5 g/hr) in females (Lines 2 in Row H,

Table 1) to be provided by liver glucose release and exogenous CHO oxidation.

Estimated CHO contribution from liver glucose disappearance

Panels A and B in the authors' Figure 4 (1) predict that liver glucose disappearance contributes 68 and 49 g CHO to total CHO oxidation in males and females respectively (Table 1, Row I). This is likely overestimated since high rates of CHO ingestion suppress liver glucose disappearance (7–9).

When modified for these two contestable calculations, the predicted rate of exogenous CHO oxidation needed to run a sub2hrM would be 128–211 g CHO/2hr (64–105.5 g CHO/hr) in males and 178–238 CHO/2 hr (80–119 g CHO/hr) in females (Table 1, Row J) compared to rates of 90 and 106 g CHO/hr predicted in the original model (Table 1, Row K). These calculations support the general accuracy of the authors' original conclusions.

Estimated rates of fat oxidation during exercise at >90% VO₂max

The authors' second presumption is that during a sub2hrM, the rate of fat oxidation would be 2–3 kJ/min (0.07 and 0.06 g/min for men and women respectively; Row D; Table 1) providing <3% of the total energy. However, even at exercise intensities >85% VO₂max, some well-trained but recreational athletes oxidized fat at rates >1.5 g/min (57 kJ/min) (10) potentially supplying 65%–75% of the 76–89 kJ/min required for males and female athletes to run a sub2hrM (Table 1, Row A). Other studies report high rates of fat oxidation even at moderate to high exercise intensities (11).

Figure 1 shows the rates of endogenous CHO (muscle glycogen and blood/liver glucose), fat and exogenous CHO oxidation in male (Row A) and female (Row B) athletes running a sub2hrM according to the calculations of Lukasiewicz et al. (1). Increasing the rate of fat oxidation to 0.5 g/min (18 kJ/min) reduces the CHO deficit required from exogenous CHO oxidation to 25 g/hr for males (Row C) and 46 g/min for females (Row D). Increasing the fat oxidation rate to 0.7 g/min in males (Row E) and to 0.9 g/min in females (Row F) removes any requirement for exogenous CHO oxidation to fuel a sub2hrM in either sex.

Thus the model's predictions are critically dependent on the value given to rates of fat oxidation. At present there are few data of rates of fat oxidation in elite athletes exercising at 90% VO₂max or higher. Furthermore, those data that are available, are usually from studies of athletes habituated to high CHO diets which produce submaximal "peak" rates of fat oxidation (11).

Rates of total exogenous CHO oxidation calculated from instantaneous rates of exogenous CHO oxidation during exercise

The third presumption is that ingesting CHO at 90–120 g/min will produce equivalent exogenous CHO rates of 90–120 g/min for

the full duration of the sub2hrM. Figure 2 depicts results from the two laboratory studies (12, 13) that have reported the highest rates of exogenous CHO oxidation during exercise. Figure 2A shows instantaneous exogenous CHO oxidation rates in these studies; Figure 2B the cumulative amounts of CHO ingested and oxidized, including the cumulative amounts remaining unoxidized when these experiments concluded.

The study of Jentjens et al. (12) found that the total amount of exogenous CHO oxidized during 2 h of exercise from the optimum solution of glucose, fructose and sucrose was 137 g or 48% of the total ingested amount of 288 g (Figure 2B) leaving 151 g (52%) unoxidized (Figure 2B). Fifty three and 70% of the ingested CHO from the two other tested solutions remained unoxidized after 2 h exercise (Figure 2B) (12, 13).

These data predict that the maximum amount of exogenous CHO oxidation during a sub2hrM in which subjects ingest CHO even at 144 g/hr, would likely be only 137 g (68.5 g/hr). This is substantially less than the rates of 106 g/hr for females and 90 g/hr for males [Figures 6A, B; Lukasiewicz et al. (1)] that the model predicts are required to complete a sub2hrM. Thus the model overpredicts the available total exogenous CHO oxidation amounts by a minimum of 43.6 g/2hr in males and of 75 g/2hr in females.

Rates of exogenous CHO oxidation fall short of values required to run a sub2hrM

The result is that this model predicts that it is not possible for any athlete to run a sub2hrM relying exclusively on CHO metabolism, even when CHO is ingested at rates of 120–144 g/hr. Rather a reasonable contribution from fat oxidation might make it possible for some athletes to achieve the sub2hrM even whilst ingesting either none or quite small amounts of CHO during the race (Figure 1).

Is there a time penalty associated with drinking frequently when running at 21.2 km/hr during the sub2hrM attempt?

The fourth assumption is that during the sub2hrM attempt, runners can ingest CHO at very high rates without losing time as a result of frequent drinking. With one exception (14), studies of high rates of CHO ingestion during exercise come from tightly controlled laboratory studies of carbohydrate-adapted cyclists (12, 13, 15–24). The presumption is that results from controlled laboratory experiments of cyclists can be extrapolated to runners competing in an uncontrolled real-world environment, frequently disturbed by the presence of other competitors. For example, intra-abdominal pressures are substantially higher during running than during cycling (25, 26), a difference that will likely influence the ability to replace CHO at high rates when running at >90% VO₂max for 2 h.

Rowe et al. (14) reported high rates of CHO ingestion (90 g/hr) and total exogenous CHO oxidation (132 g; 66 g/hr) in runners during 2 h of treadmill running but at much lower exercise intensities than are achieved during the sub2hrM. Subjects ingested
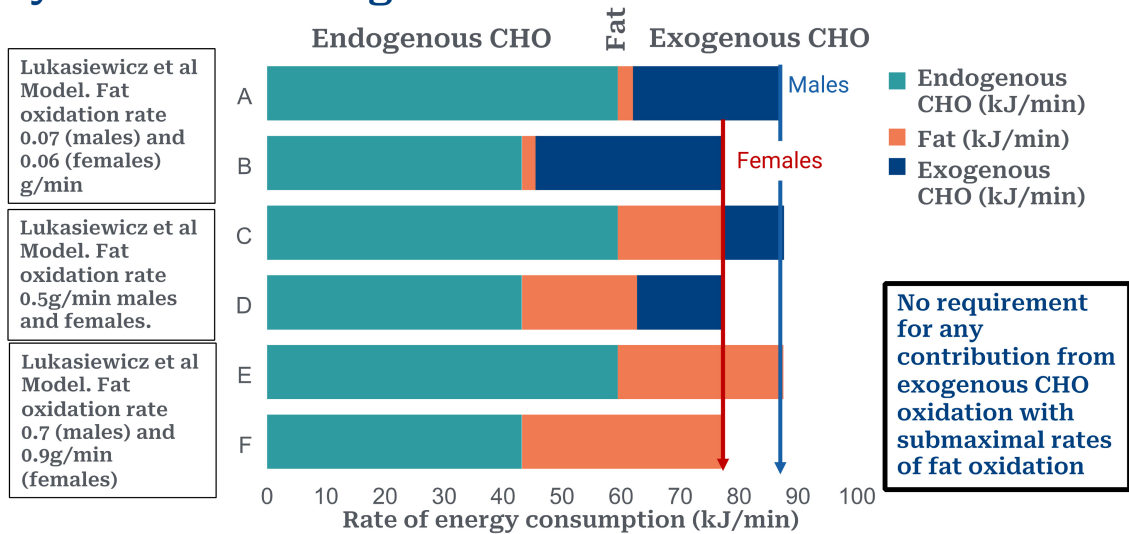
FIGURE 1
Contributions of endogenous (muscle glycogen), fat and exogenous CHO to total energy expenditure in males and females wishing to run a sub2hrM. Rows A and B according to model of Lukasiewicz et al. (1). Rows C and D when rate of fat oxidation is increased to 0.5 g/min; Rows E and F when rate of fat oxidation is increased to 0.7 g/min for males and 0.9 g/min for males. These higher rates of fat oxidation remove the need for any contribution from exogenous CHO oxidation. Liver glucose disappearance likely makes little or no contribution to energy production at such high rates of CHO ingestion (7–9).

fluid at a rate of 400 ml/hr from a 22.5% CHO drink and oxidized 73% of the ingested CHO. Cyclists in the study of King et al. (19) achieved similar rates of exogenous CHO oxidation and CHO ingestion (112.5 g/hr) from a much less concentrated CHO solution (11%) but at a much higher fluid ingestion rate (1 L/hr). Hawley et al. (24) reported similarly high rates of CHO ingestion (120 g/hr) by cyclists who ingested a 15% CHO solution at 800 ml/hr. In that study total exogenous CHO oxidation was 45 g/hr, leaving 150 g unoxidized at the end of exercise.

These scientists seem to have discovered that athletes have substantially greater difficulty ingesting fluid at high rates when running than when cycling, even under laboratory conditions. Additionally the movements of the upper body are quite different in running and cycling. The upper body is essentially static during cycling, but rotates substantially when running, increasing with running speed, contributing to increased running efficiency (27). Repeated drinking whilst attempting to ingest CHO at high rates must potentially impairing running performance. Clearly the possible time-wasting effects of frequent drinking during the sub2hrM need to be quantified.

High rates of CHO ingestion during exercise may have metabolic effects not considered in this model

The final assumption is that ingesting CHO will not produce metabolic effects that could impair running performance. Hawley et al. (24) found that the ingestion of 120 g CHO/hr during exercise

at 70% VO_2max raised blood insulin concentrations, reducing the rate of fat oxidation from ~0.94 to ~0.17 g/min. Total muscle glycogen use during 125 min of exercise was 140 g, 67% greater than in a control group who received enough CHO by infusion to maintain euglycemic blood levels. Similarly the intravenous infusion of 252 g of glucose during 2 h of cycling exercise (126 g/hr) increased total muscle glycogen use by 40 g (28).

Discussion

The authors have gone to extraordinary lengths to develop a model of the rates of exogenous carbohydrate (CHO) ingestion required to run a sub2hrM. We argue that their model contains two potential flaws that might reverse their conclusions.

First, their model requires that fat oxidation makes no significant contribution to energy use during exercise at >90%VO_2max. Whilst this is the accepted doctrine for the past century or so, more recent studies find that athletes chronically adapted to low-CHO diets can achieve high rates of fat oxidation even during exercise at >85% VO_2max (10, 11). We show that rates of fat oxidation of 0.7 g/min in males and 0.9 g/min in females would, according to the predictions of this model, allow athletes of either sex to run a sub2hrM without requiring any additional energy from exogenous CHO oxidation.

Second, the authors assume that high rates of ingested CHO produce equivalent high rates of total exogenous CHO oxidation during exercise. However, studies of high rates of CHO ingestion during exercise show that, at best, only 50% of the ingested CHO is oxidized during the first 2 h of exercise (Figure 2B).
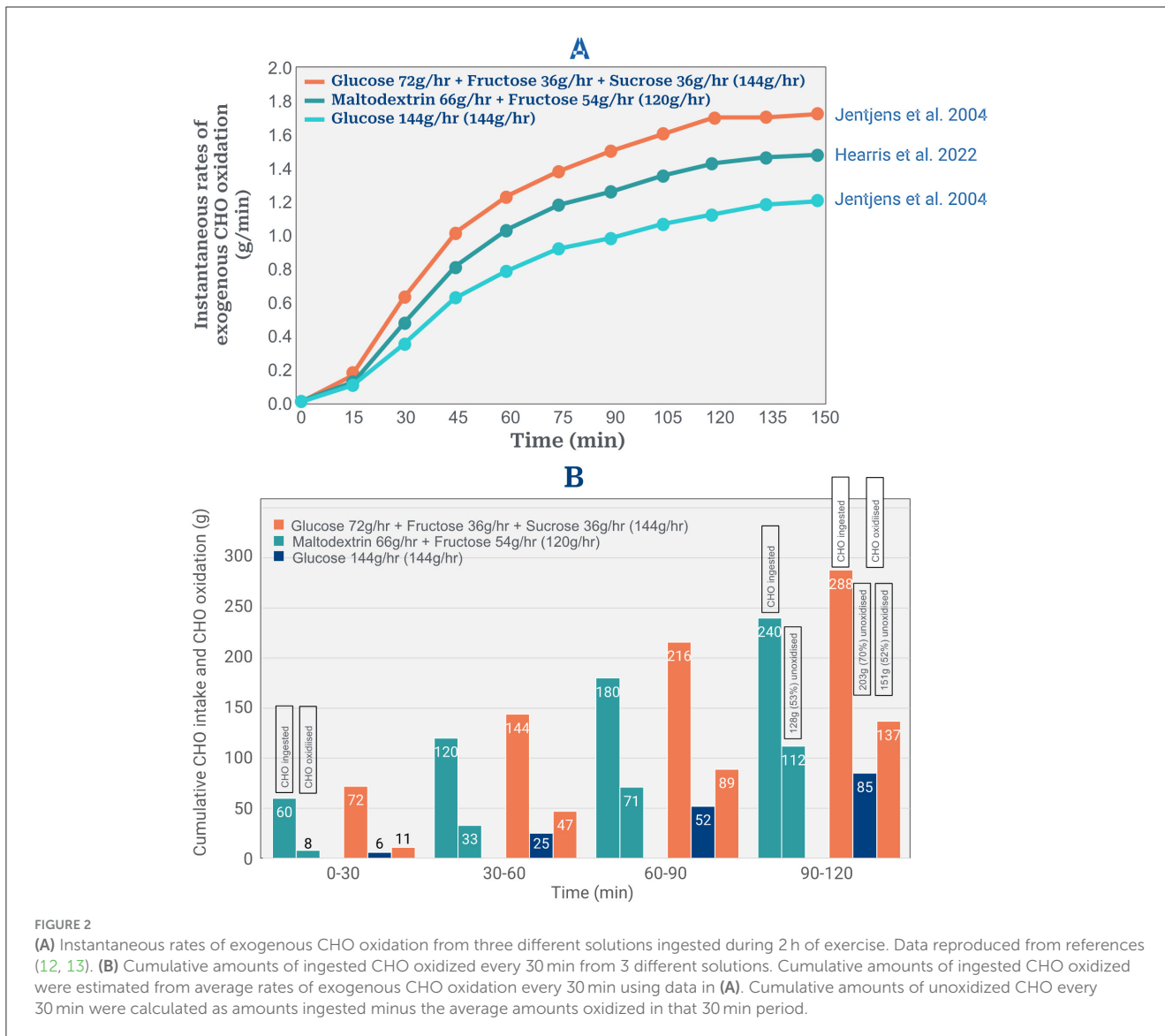
FIGURE 2
(A) Instantaneous rates of exogenous CHO oxidation from three different solutions ingested during 2 h of exercise. Data reproduced from references (12, 13). **(B)** Cumulative amounts of ingested CHO oxidized every 30 min from 3 different solutions. Cumulative amounts of ingested CHO oxidized were estimated from average rates of exogenous CHO oxidation every 30 min using data in **(A)**. Cumulative amounts of unoxidized CHO every 30 min were calculated as amounts ingested minus the average amounts oxidized in that 30 min period.

When this latter correction is made to this model, the prediction must be that it will not be possible for a male or female athlete to run a sub2hrM relying purely on CHO metabolism. But with a reasonable contribution from fat oxidation, the sub2hrM might be possible in some athletes even with little or no contribution from the oxidation of exogenous CHO during exercise.

A limitation of this discussion is that it remains theoretical in nature; therefore, further empirical investigation is necessary to validate and substantiate the proposed concepts. Future experimental research should aim to test these hypotheses and explore their practical implications in greater depth. Additionally, it should be acknowledged that no cellular or tissue-level data measurements were obtained from either group, potentially limiting the direct applicability of our findings. Moreover, our calculations did not account for the contributions of amino acids and ketone bodies, which may have influenced the metabolic outcomes considered.

Author contributions

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Conflict of interest

TN is author of low-carbohydrate nutrition books. TN book royalties are donated to The Noakes Foundation which contributes to the Eat Better South Africa Campaign.

The remaining author declares that the research was conducted in the absence of any commercial or financial

relationships that could be construed as a potential conflict of interest.

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