Ketogenic Diets Are Beneficial for Athletic Performance

TIMOTHY DAVID NOAKES

Cape Peninsula University of Technology, Cape Town, SOUTH AFRICA

An iconic 1967 study proved that a 7-d ketogenic (low-carbohydrate high-fat [LCHF]) diet impairs performance when eaten by athletes habituated to a high-carbohydrate low-fat (HCLF) diet (1).

This suggests I should begin by conceding defeat. Instead, I will present the hidden truth of that study.

That trial was not designed to exclude all possible metabolic mechanisms by which the ketogenic LCHF diet might impair exercise performance. The LCHF diet lowers both starting muscle (1) and liver glycogen concentrations (2); the latter increases the probability that exercise-induced hypoglycemia (EIH) will limit subsequent exercise. That trial (1) produced profound EIH in subjects eating the restricted CHO diets (Fig. 4 in [3]).

Earlier studies (4,5) established that subjects can develop profound fatigue associated with cerebral symptoms of EIH during prolonged exercise. CHO ingested at exhaustion rapidly reversed the fatigue and the cerebral symptoms, allowing exercise to continue (Fig. 3 in [3]).

Christensen and Hansen (5) noted that CHO ingested at exhaustion did not raise the RQ (Figs. 1 and 2 in [5]; Fig. 3 in [3]), so that "… the improved ability to exercise (with glucose ingestion) cannot be explained by the renewed carbohydrate supply to the muscles, because the *beneficial effect is only present if this carbohydrate supply forces the blood sugar level to rise*. The beneficial effect *is thus certainly not a result of the increased carbohydrate depots or a greater percentage of carbohydrate burning in the muscles* (current author's emphasis), but exclusively of the higher blood sugar level and is closely related to the disappearance of the hypoglycemic symptoms. Fatigue must be regarded as a hypoglycemic symptom of cerebral origin" (5, p. 178).

To prove 28 yr later that muscle glycogen depletion alone limited exercise performance, Bergstrom et al. (1) had first to prove that the EIH they measured (Fig. 3 in [3]) had not acted as the true "exercise stopper." But they did not.

Hultman et al. (6) later acknowledged their awareness of this problem: "A low carbohydrate diet … will decrease the liver glycogen store and thus the capacity for glucose production by the liver with risks for blood glucose decreases during exercise" (1).

Subsequent generations of exercise scientists, including notably myself (7), have ignored this original EIH "problem," accepting rather that muscle glycogen provides the obligatory (8–10) fuel for exercise and for which, once depleted, only ingested carbohydrate can effectively substitute.

One set of modern studies (11–14) concluded that Christensen and Hansen were in error, "the decline in plasma glucose that often accompanies prolonged exercise in the fasted state can contribute to the development of fatigue. The inability to continue exercise was related to the lowering of plasma glucose and subsequent fall in carbohydrate oxidation, suggesting that fatigue was due to an inadequate supply or carbohydrate for the working muscle" (12).

However, none of these studies was designed to test Christensen and Hansen's hypothesis. Fitts (15) concludes, "To date, a cellular explanation for an obligatory oxidation of carbohydrates has not been established."

Their premier study (12) found that subjects who ingested placebo developed a progressive EIH (Fig. 1A). CHO ingested at a rate of 100 $g \cdot h^{-1}$ prevented EIH, producing slightly higher rates of whole-body CHO oxidation (Fig. 1B) with progressively lower rates of fat oxidation (Fig. 1C) but without differences in muscle glycogen use (Fig. 1D). This disproved their earlier postulate that "carbohydrate feedings delay fatigue by apparently slowing the rate of muscle glycogen depletion" (16).

Table 1 shows that the ingestion of 300 g CHO (5100 kJ) increased average CHO oxidation rate by 0.20 $g \cdot min^{-1}$ (3.4 $kJ \cdot min^{-1}$), increasing total CHO disappearance by 36.4 g (588 kJ) during 3 h of exercise, accounting for 12% (619 kJ) of the total ingested CHO load. CHO ingestion reduced average fat oxidation rate by 0.10 $g \cdot min^{-1}$ (3.8 $kJ \cdot min^{-1}$) for a total saving of 16.4 g (623 kJ) during 3 h of exercise. After 3 h, rates of CHO oxidation were 0.52 $g \cdot min^{-1}$ (8.8 $kJ \cdot min^{-1}$) lower, whereas rates of fat oxidation were 0.23 $g \cdot min^{-1}$ (8.7 $kJ \cdot min^{-1}$) higher with placebo compared with CHO ingestion.

Address for Correspondence: Timothy David Noakes, M.B.Ch.B., M.D., D.Sc., Ph.D.(hc), 7 Tanja Road, Constantia, 7806, Cape Town, South Africa; E-mail: noakes@iafrica.com

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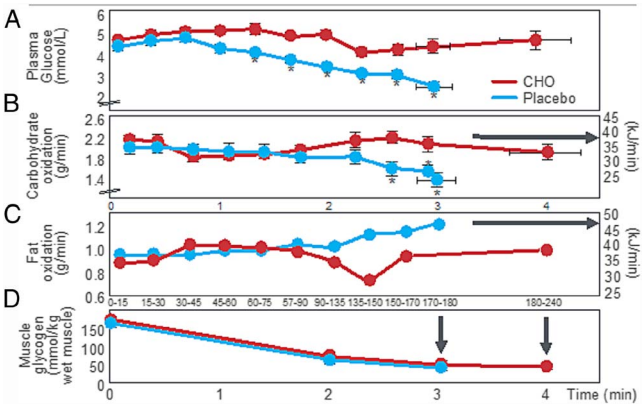

FIGURE 1—BG concentrations (A), rates of CHO (B), and fat (C) oxidation and changes in muscle glycogen concentrations (D) in subjects ingesting either CHO or placebo during 180–240 min of exercise. Mean rates of fat oxidation (g·min^{-1}; kJ·min^{-1}) were calculated from $\dot{V}O_2$, RQ, and rates of CHO oxidation using conventional equations. Energy from fat oxidation exceeded that from CHO oxidation for most of the experiment (*arrows* in panels B and C). Reproduced from Coyle et al. (12) with permission.

The ingestion of 300 g CHO produced an essentially isocaloric substitution of (increased) CHO oxidation for (reduced) fat oxidation, in line with the observation of Fitts (15) that CHO ingestion during exercise produces a small effect on rates of CHO and fat metabolism and which "would not explain the complete exhaustion associated with prolonged exercise (*when CHO is not ingested – current author's addition*)."

These data invite the following questions: How does the oxidation of an additional 36 g CHO during 3 h of exercise, representing 952 kJ or just 8% of the total energy expenditure of 12 463 kJ, allow exercise to be continued for a further hour? How does the substitution of 8.8 kJ·min^{-1} of CHO oxidation for 8.7 kJ·min^{-1} of fat oxidation at the point of exhaustion prevent fatigue and allow exercise to continue for a further hour, especially when this 8.8 kJ·min^{-1} represents only 13% of the total rate of energy expenditure (69.6 kJ·min^{-1}) at that moment? Exactly how does that "obligatory" extra 8.8 kJ of CHO sustain exercise because it is not "sparing" muscle glycogen use? Why is a CHO oxidation rate of 1.90 g·min^{-1} (32.3 kJ·min^{-1}) able to sustain exercise for another 60 min whereas a rate of 1.38 g·min^{-1} (23.5 kJ·min^{-1}) causes incapacitating exhaustion and immediate exercise termination?

Many other studies also report similar and equally small metabolic effects of very high rates of CHO ingestion during prolonged exercise (e.g., 17–21).

The only reasonable conclusion is that Christensen and Hansen were correct: Ingested CHO improves performance by preventing the development of EIH (Fig. 1A), and for which very little CHO is required as the total blood glucose content is only 5 g (22).

Rat studies confirm this finding that EIH is the more important "exercise stopper." Selective inhibition of hepatic gluconeogenesis in rats impairs exercise performance (23), whereas rats with a genetically determined superior capacity for liver glycogen storage have superior endurance because they can maintain higher BG concentrations for longer regardless of the preexercise diet (24). In neither study was muscle glycogen metabolism affected by either intervention despite large differences in exercise performance. By contrast, mice unable to synthesize muscle glycogen due to a lack of muscle glycogen synthase do not have an impaired exercise capacity (25), perhaps because they have normal liver glycogen synthase activity. A greater capacity to increase preexercise muscle glycogen stores also does not enhance exercise performance in some mice (26).

Because muscle glycogen depletion is not the predominant "exercise stopper" (Fig. 1D, *arrows*) and because ingesting CHO during exercise can prevent or reverse the proven "exercise stopper"—EIH (3–5,11–14), it follows that currently there is no biological reason to prescribe a special HCLF preexercise diet to produce a maximum exercise performance (by maximizing preexercise muscle glycogen stores) (3). The ingestion of sufficient CHO to maintain the total BG content at 5 g (22) during exercise is all that is required.

It follows that provided athletes ingest sufficient CHO during exercise, it matters not whether the preexercise diet is high in either fat or CHO. This helps explain why the ketogenic LCHF diet has proven to be as beneficial as is the HCLF diet for athletes interested in peak performances during exercise and training (27,28) at a wide range of exercise intensities (3,29–32).

We have recently established that increased fat oxidation can provide energy at up to 76 kJ·min^{-1} even at very high

TABLE 1. Difference in muscle glycogen disappearance and in total and average rates of CHO and fat oxidation in subjects ingesting either placebo or 100 g CHO·h^{-1} during 180 min of exercise at 71% $\dot{V}O_{2max}$.

	Placebo Ingestion	CHO Ingestion	Difference (CHO vs Placebo Ingestion)
Total muscle glycogen disappearance in 180 min	129 glucose units·kg^{-1}	126 glucose units·kg^{-1}	−3 glucose units·kg^{-1}
Total CHO (g) (kJ) (% total energy) oxidized during 180 min of exercise	326.5 g	361.1 g	+34.6 g
	5551 kJ	6139 kJ	+588 kJ
	44%	49%	+5%
Average rate of CHO oxidation (g·min^{-1}) (kJ·min^{-1}) during exercise	1.81 g·min^{-1}	2.01 g·min^{-1}	+0.20 g·min^{-1}
	30.8 kJ·min^{-1}	34.2 kJ·min^{-1}	+3.4 kJ·min^{-1}
Rate of CHO oxidation (g·min^{-1}) (kJ·min^{-1}) at 180 min	1.38 g·min^{-1}	1.90 g·min^{-1}	+0.52 g·min^{-1}
	23.5 kJ·min^{-1}	32.3 kJ·min^{-1}	+8.8 kJ·min^{-1}
Total fat (g) (kJ) (% total energy) oxidized during 180 min of exercise	181.9 g	165.5 g	−16.4 g
	6912 kJ	6289 kJ	−623 kJ
	55%	50%	−5%
Average rate of fat oxidation (g·min^{-1}) (kJ·min^{-1}) during exercise	1.01 g·min^{-1}	0.91 g·min^{-1}	−0.10 g·min^{-1}
	38.4 kJ·min^{-1}	34.6 kJ·min^{-1}	−3.8 kJ·min^{-1}
Rate of fat oxidation (g·min^{-1}) (kJ·min^{-1}) at 180 min	1.21 g·min^{-1}	0.98 g·min^{-1}	+0.23 g·min^{-1}
	46.0 kJ·min^{-1}	37.3 kJ·min^{-1}	+8.7 kJ·min^{-1}

Values were calculated using data in Figure 1.

exercise intensities (>85% $\dot{V}O_{2max}$) (31,32). This means that fat oxidation alone could have provided 109% of the rate of energy expenditure (69.6 $kJ \cdot min^{-1}$) maintained for 3–4 h in the study of Coyle et al. (12) (Fig. 1), theoretically substituting fat oxidation for any muscle glycogen use.

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