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The Complete H^+ Accounting

What carbohydrate becomes when it is burned.

THE FIELD JOURNAL EDITORIAL · 12 MINUTES

THE EDITOR'S SUMMARY · 200 WORDS

IF YOU READ NOTHING ELSE

In Vienna, 2019, Eliud Kipchoge ran a sub-two-hour marathon while absorbing roughly 100 to 120 grams of carbohydrate per hour. Five years earlier the published textbook ceiling was 60. Maurten's hydrogel solved the substrate problem. It also revealed the byproduct problem.

Every gram of carbohydrate burned glycolytically deposits hydrogen ions in the cytosol. At race intensity the muscle absorbs on the order of one mole of H^+ per hour. The *proton* — not the lactate — drops cellular pH from 7.0 toward 6.5 and is what makes the legs refuse.

The muscle has three lines of defense. Two are fixed. The third — the dipeptide pool, carnosine and its methylated cousin anserine — is the only one a strategy can move.

Beta-alanine loads carnosine slowly, over weeks. It cannot raise anserine at all, and it does not work acutely. Anserine, taken orally, survives serum carnosinase where carnosine does not. The methyl group is the entire commercial premise. Independent Ghent University work (Blancquaert 2021, 2022) shows 3–6% improvements in peak power and torque at 30 mg/kg, sixty minutes pre-effort. Responder-dependent. Mechanism clear.

Maurten is the fuel in the tank. KATSUO is the cooling system.

I. THE NUMBER BEHIND THE NUMBER

In Vienna, on the second Saturday of October 2019, Eliud Kipchoge ran 26.2 miles in one hour, 59 minutes, and 40.2 seconds. He had pacers, an ideal course, a windbreak of athletes in front of him, and the asterisk that history would never let him forget. He also had something else, almost invisible in the photos.

He had a metabolic rate that, just five years earlier, would have been considered impossible to sustain over the distance.

The number that mattered was not 1:59:40. The number that mattered was the rate at which carbohydrate was crossing the wall of his small intestine and entering his bloodstream. For most of the twentieth century, the textbook ceiling on exogenous carbohydrate oxidation during sustained exercise was 60 grams per hour.[1] A talented athlete might absorb 70. A great one, with years of gut training, perhaps 80. Above that, the gut simply did not deliver. Carbohydrate sat in the lumen, water followed it, and the runner went to the side of the road.

By Vienna, Kipchoge was absorbing somewhere between 100 and 120 grams of carbohydrate per hour.[2] Two and a half hours of running, north of 250 grams of in-race fuel oxidized. A number that, in 2015, the published literature did not believe a human could sustain.

The breakthrough was not pharmacological. It was textural. A small Swedish company called Maurten had figured out how to suspend maltodextrin and fructose in a sodium-alginate hydrogel that passed through the stomach as if it were close to isotonic, releasing its carbohydrate further down the line — past the gastric-emptying bottleneck that had constrained the textbook for fifty years. The substrate problem had been solved.

But every gram of carbohydrate that successfully crosses the gut wall, enters the muscle cell, and is oxidized at race intensity has a stoichiometric consequence. The faster the engine runs, the more byproduct it produces. Solving the substrate problem revealed the byproduct problem.

This is an essay about the byproduct problem.

Inside the muscle cell, carbohydrate is converted to ATP by two routes. The slow route — oxidative phosphorylation, in the mitochondria — produces 30-something ATP per molecule of glucose, releases carbon dioxide and water, and operates net-neutral on hydrogen ions. This is the metabolism of the easy long run. The hum.

The fast route — glycolysis, in the cytosol — produces only 2 ATP per molecule of glucose, much faster, and at a cost. The breakdown of glucose to pyruvate is, by stoichiometry, a proton-producing reaction. At rest, pyruvate flows neatly into the mitochondria and is oxidized cleanly. At threshold, mitochondrial demand exceeds capacity. Pyruvate accumulates. It is converted to lactate. Crucially, the lactate-pyruvate exchange does not remove the hydrogen ions the upstream reactions produced.

The lactate has, for a century, gotten the historical blame. It does not deserve it. Lactate is, if anything, a fuel — taken up by the heart, the brain, less-active muscle, recycled through the Cori cycle in the liver. The problem is the proton. The textbook reaction:

$$\mathit{Glucose} + 2\,\mathit{ADP} + 2\,P_i \rightarrow 2\,\mathit{lactate}^- + 2\,\mathit{ATP} + 2\,H^+$$

THE PROTON IS THE BYPRODUCT THAT MATTERS

Each molecule of glucose burned glycolytically deposits two hydrogen ions into the cytosol. Multiply by the rate. At 100 grams of carbohydrate per hour entering the glycolytic pathway, the muscle is asked to absorb on the order of one mole of H^+ per hour — not all in one cell, not all at once, but locally and in concentration where the work is being done.[3]

Cytosolic pH at rest sits at roughly 7.0. At fatigue — the wall, the bonk, the moment the legs refuse — it drops to roughly 6.5.[4] A half-unit on the logarithmic scale is a three-fold increase in hydrogen-ion concentration. The contractile machinery does not like this. Myosin's ability to bind actin, the calcium-handling apparatus, phosphofructokinase, the regulatory enzyme of glycolysis itself — every one of them is pH-sensitive. The cell does not stop working at low pH. It works less well. And it works much less well in a way the athlete experiences as legs that have run out.

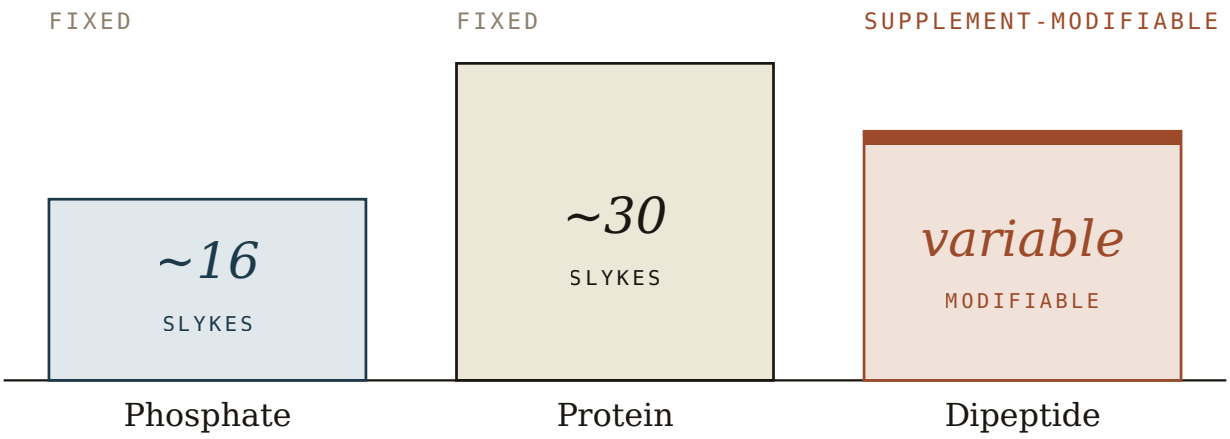
This is the byproduct problem. It is downstream of every Maurten hydrogel, every Kipchoge breakthrough, every published advance in carbohydrate delivery. It scales with success at the upstream step.

Every muscle cell carries three lines of defense against H^+ accumulation. They are present in different amounts in different species. They have very different responses to training and to supplementation.

The first is the phosphate buffer system — creatine phosphate, ATP, inorganic phosphate. It accounts for roughly 16 slykes of buffering capacity in human skeletal muscle. A *slyke* is the technical unit: one slyke buffers one millimole of H^+ per pH unit per kilogram of tissue. The phosphate system is fast, reliable, and unmodifiable by anything an athlete can do at the gut.

The second is the protein buffer system — the histidine side chains on contractile proteins, on sarcoplasmic enzymes, on the structural proteins of the sarcomere. About 30 slykes. Modifiable in principle by hypertrophy, unmodifiable in any acute sense.

The third is the dipeptide buffer system. Carnosine. Anserine. Balenine (in some species). Variable across humans. Variable across species by orders of magnitude. Modifiable.



THREE INTRACELLULAR BUFFER SYSTEMS IN HUMAN SKELETAL MUSCLE. ONLY THE THIRD LINE CAN BE MOVED BY A STRATEGY.

The dipeptide system is the only line that responds to what an athlete puts in their mouth. It is also the only line that does its work where it matters — at the imidazole ring of the histidine residue, which has a pK_a of approximately 6.83. That is the working range of muscle pH at race effort. A buffer is only useful when it titrates near the pH at which it is needed; the dipeptide system is the only buffer in the cell that titrates inside the cell, at the pH inside the cell, in the moments that pH is dropping. There is no analog. No injectable. No exogenous molecule that does this from outside the cell. The cell either builds its dipeptide pool from precursor amino acids over

weeks, or it imports the finished molecule across its membrane in the minutes after a pre-effort dose. There is no third option.

This is why the dipeptide buffer system is the only line of defense that the supplement industry has, for half a century, been trying to load.

IV. WHAT BETA-ALANINE WON

The supplement industry — the gym-bro half of it, the part KATSUO does not sound like — solved the dipeptide loading problem in the mid-2000s. Roger Harris and his colleagues at the University of Chichester published the foundational work that established beta-alanine as the rate-limiting precursor for muscular carnosine synthesis.[5] Take it orally, in divided doses, every day for four to twelve weeks. Intramuscular carnosine concentration rises 40 to 80 percent above baseline. Hold the loading dose, and the elevated pool persists.

The molecule is cheap. It is patented as CarnoSyn by Natural Alternatives International, sold by the tub at every gym chain in America, dosed at four to six grams per day, and produces a characteristic skin tingle — paresthesia — that has become its brand signal. There is now a decade and a half of repeated, well-powered evidence that chronic beta-alanine loading improves high-intensity exercise capacity in the 60-to-240-second range. Small effects, real effects, statistically robust, mechanism clear.[6]

This is what the Western sports-nutrition market has won. The category exists. The mechanism is understood. The athletes who need it know how to use it.

What beta-alanine cannot do is two things. First, it cannot work acutely. The chronic loading window is not negotiable, because the carnosine has to be synthesized inside the muscle cell over weeks. There is no race-morning dose of beta-alanine that produces a useful effect that afternoon. The athlete who took their first scoop on Friday before a Saturday marathon is loading a pool that will be ready for some marathon two months from now.

Second, beta-alanine cannot raise anserine. Humans do not methylate carnosine in significant amounts in vivo. The intramuscular dipeptide pool that beta-alanine builds is overwhelmingly carnosine — not the more pharmacokinetically interesting methylated cousin. If anserine has a role to play in the buffer system, beta-alanine is not the molecule that gets it there.

If beta-alanine were the whole answer, this essay would end here. It is not.

For decades, the obvious alternative — take carnosine directly, by mouth, and skip the precursor route — failed. The failure was not bioavailability in the conventional sense. Oral carnosine is absorbed. It enters the bloodstream. And then it disappears, within minutes, into its constituent amino acids: beta-alanine and L-histidine.

The disappearing act is performed by an enzyme called serum carnosinase, encoded by the CNDP1 gene, expressed in the liver, and circulating in plasma. Most mammals do not have this enzyme in any meaningful concentration. Mice, rats, cats, dogs, fish, pigs — all keep their dietary carnosine largely intact in circulation. Humans and a few primates do not. The evolutionary reason for this is unclear. The practical consequence is that taking oral carnosine and expecting it to reach muscle is, for most humans, a slow way to make beta-alanine and histidine.[7]

This is the carnosinase problem. It is the reason beta-alanine won the Western market. It is also the reason the methylated form of carnosine — anserine — is the molecule worth talking about.

Anserine differs from carnosine by a single methyl group on the imidazole ring. That methyl group does almost nothing to the buffering chemistry: the pK_a of the imidazole nitrogen is essentially unchanged. The methyl group does almost everything to the pharmacokinetics. Carnosinase cleaves carnosine readily. It does not cleave anserine — at any rate, not at meaningful velocity in vivo. Plasma half-life is materially longer. The molecule has a chance of reaching the muscle-cell membrane intact and entering the buffer pool from outside.

This is the only reason anserine, in a category dominated by beta-alanine, is interesting. The single methyl group is the entire commercial premise of the molecule.

The methyl group does almost nothing to the chemistry, and almost everything to the half-life.

The cleanest independent work on anserine in humans comes from Wim Derave's laboratory at Ghent University in Belgium. The Ghent group has spent fifteen years on the carnosine system — first establishing the chronic beta-alanine loading curves, then probing the carnosinase bottleneck in detail, then turning to anserine itself. Their 2021 paper, published in the *Journal of Applied Physiology*,[8] is the foundation of every honest claim that any anserine supplement can defensibly make.

Fifteen male volunteers. A crossover design — each subject as their own control. Twenty milligrams per kilogram of bodyweight of carnosine *and* anserine, taken thirty minutes before a Wingate-style sprint protocol. Outcome: a six-percent improvement in initial five-second sprint power. The effect tracked, in their plasma sampling, to the bioavailability of anserine — not carnosine. Carnosine was cleaved as expected. Anserine made it through. The methyl group was the reason.

The 2022 follow-up, in the *Journal of the International Society of Sports Nutrition*,[9] established the dose-response curve across three doses (10, 20, 30 mg/kg) and two timing windows (30 and 60 minutes). The best protocol — 30 mg/kg of each, 60 minutes pre-effort — produced a three-percent increase in peak power and a four-and-a-half-percent increase in peak torque on repeated-sprint testing.

The Ghent group also reported something the supplement-industry marketing line tends to omit: the effect size was inversely correlated with the subject's serum carnosinase activity. The more active your enzyme, the less you respond. High responders, low responders, non-responders — a continuous distribution that runs through every supplement category and that the Ghent group named openly in their discussion. This is what honest looks like in this literature.

A third finding, less cited but mechanistically important: combined ingestion of carnosine and anserine increased anserine bioavailability roughly 2.5-fold over anserine alone. The mechanism is competitive substrate inhibition. Carnosine saturates serum carnosinase; anserine, with its methyl group, sails past. Skipjack tuna red muscle delivers both, in roughly a three-to-one anserine-to-carnosine ratio. By design or by accident, it is pharmacokinetically the optimal preparation in this category — a fact the brand did not engineer and could not have engineered, because the fish was doing it long before the chemistry was known.

One more piece of the Ghent-adjacent literature deserves a sentence here: the 2022 work by Kotani and colleagues[10] on 30-day low-dose supplementation (1,500 mg/day of carnosine + anserine, 3:1 ratio) in endurance runners. The finding was not performance, exactly — it was a modulation of exercise-induced hemolysis and the hepcidin response, suggesting a role in iron regulation for athletes carrying chronic

deficiency risk. This is adjacent to the buffering mechanism rather than central to it, but it lives in the same molecular family.

The combined picture across the independent literature is consistent. The effect is real. The effect is small to moderate. The effect tracks the methylated molecule. The effect is responder-dependent in a measurable, mechanism-clear way. None of this requires anyone to overclaim. All of it requires citing the work.

. . .

VII. — THE SKIPJACK

Skipjack tuna — *Katsuwonus pelamis* — swim continuously from the moment they hatch. They have no swim bladder. If they stop, they sink. They migrate across thousands of kilometers of open Pacific every year, holding sustained submaximal pace at a few body-lengths per second for months at a time, and they live their entire lives in a state that a human marathoner inhabits for two hours every six weeks.

The biology that lets them do this is concentrated in their red muscle — the dark, slow-twitch, mitochondria-dense, oxygen-rich tissue that runs the lateral midline of their bodies. Of all the imidazole-dipeptide tissues that have been measured across vertebrate species, skipjack red muscle is consistently among the densest. Published values vary by tissue and freshness, but fresh skipjack head meat has been measured at roughly 4,500 milligrams per kilogram anserine and 1,800 milligrams per kilogram carnosine.[11] Per kilogram of red muscle, the skipjack carries one of the highest known natural concentrations of intracellular buffering peptides of any vertebrate routinely consumed.

This is not metaphor. It is chemistry. The skipjack's red muscle and the human marathoner's slow-twitch muscle do the same thing at the level of the dipeptide ring. Carbohydrate enters. Glycolysis runs, slower in the fish than in the human at race effort but ceaseless. H^+ accumulates. The histidine imidazole titrates. The pH holds. The cell keeps working.

The fish has been doing it for two hundred million years. The athlete, on race morning, is borrowing the strategy.

This is also why the brand calls its molecule what it does. The active is not "imidazole dipeptide." It is not "fish peptide extract." It is anserine, and the fish from which it is sourced is the most efficient long-distance swimmer in the open ocean. The borrowing is the entire story.

VIII. THE COMPLETE ACCOUNTING

The chain, end to end:

> *Gut → glucose → muscle → glycolysis → H^+ → pH drop → fatigue → buffering capacity → carnosine + anserine*
>
> MAURTEN SOLVES THE FIRST NODE. KATSUO ADDRESSES WHAT FOLLOWS.

Maurten solved the first node. The hydrogel got Kipchoge from somewhere around 37 grams of carbohydrate per hour to over 100. The bottleneck moved.[12]

KATSUO addresses what follows. The H^+ accounting is straightforward and, once seen, hard to unsee. An athlete who is bad at fueling — who absorbs 30 grams per hour, glycolyzes a fraction of it, accumulates H^+ at a modest rate — has a small buffering problem. An athlete who is good at fueling — who absorbs 90 grams per hour, glycolyzes most of it, accumulates H^+ at a much higher rate — has the largest buffering problem in the field.

The well-fueled athlete is not the customer this brand has to convert. They are the customer with the strongest mechanistic case to add.

Maurten is the fuel in the tank.
KATSUO is the cooling system.

This is the operating sentence of the brand. It is also an unusual feature of an honest, mechanism-first story in a category that has run, for decades, on preference-first stories. The story is not "instead of." The story is "downstream of." Every additional gram of carbohydrate successfully oxidized is one more thing for anserine to buffer. The faster the engine runs, the more the cooling system has to do.

IX. WHAT HONEST LOOKS LIKE

The independent effect sizes — Blancquaert 2021, Blancquaert 2022, the related Maemura and Kotani work from Japan — fall in a band between three and six percent on power and torque metrics, modestly larger on submaximal-intensity blood lactate at a chronic-loading 30-day window. This is small in the lab. It is not small in a race. Three percent of a three-hour marathon is five minutes. Three percent on the peak torque a hundred-mile runner can still produce at hour eight is the difference between holding pace into a climb and breaking gait. Three percent on the buffering ceiling at the rivet point of an IRONMAN bike leg is the difference between rolling into T2 still working and rolling in cooked.

These are not promises. They are the upper end of an effect-size band that, on average, is real.

The lower end of the distribution is zero. Some athletes are non-responders, and the published mechanism — high serum carnosinase activity — predicts who they are. There is, at present, no commercial carnosinase assay available to consumers. The honest answer to "will this work for me" is: probably, modestly, with a calibrated dose taken sixty minutes before high-intensity sustained effort, and you will know within two to four race-effort sessions whether you sit on the responder or non-responder side of the curve.

This is not a miracle molecule. It is a real, modest, mechanism-grounded ergogenic effect with a specific acute timing window, supported by a real but small body of independent peer-reviewed literature, sourced from the fish with one of the highest known natural concentrations.

That is the brand's whole claim. It is, also, the entire claim worth defending.

X. A CLOSING LINE

What carbohydrate becomes when it is burned is not destiny. It is hydrogen. The hydrogen accumulates against a fixed wall of phosphate and protein buffers and against a variable wall of dipeptide buffers — the only one a strategy can move.

Beta-alanine moves it slowly, over weeks, by precursor loading.

Anserine moves it acutely, in the hour before effort, by direct delivery of the methylated dipeptide that the body's own serum enzyme leaves alone.

The fish has been doing both at once for two hundred million years.

The athlete, on race morning, is allowed to borrow the strategy.

Built like a skipjack.

KEY CLAIMS, WITH CITATIONS

The empirical load this essay carries, and the published work that supports each claim. None of these claims are larger than what the citations defend.

1 Before Maurten, the published textbook ceiling on exogenous carbohydrate oxidation during sustained exercise was approximately 60 g/hr from a single carbohydrate source, ~90 g/hr from dual-source glucose-fructose. [1] JEUKENDRUP 2010

2 Eliud Kipchoge's in-race carbohydrate intake during the INEOS 1:59 Challenge has been publicly reported in the 100–120 g/hr range, attributed to Maurten's hydrogel formulation. [2] PUBLIC REPORTS

3 Glycolysis is stoichiometrically a proton-producing reaction: each molecule of glucose burned glycolytically deposits two H^+ into the cytosol. At ~100 g/hr carbohydrate flux this is an order-of-magnitude ~1 mol H^+/hr load locally, distributed across buffering systems. [3] STOICHIOMETRIC

4 Cytosolic muscle pH falls from ~7.0 at rest to ~6.5 at fatigue — a half-unit on the logarithmic scale, corresponding to roughly a three-fold increase in H^+ concentration. [4] SAHLIN ET AL. 1975

5 Chronic beta-alanine supplementation (4–6 g/day, 4–12 weeks) raises intramuscular carnosine concentration 40–80% above baseline. The mechanism is precursor loading; beta-alanine is rate-limiting. [5] HARRIS ET AL. 2006

6 Meta-analytic evidence: chronic beta-alanine produces small but real, statistically robust improvements in high-intensity exercise capacity in the 60–240 second range. [6] HOBSON ET AL. 2012

7 Orally administered carnosine is absorbed but is cleaved within minutes in human plasma by serum carnosinase (CNDP1). Anserine — methylated on the imidazole ring — is not cleaved at meaningful velocity in vivo and survives long enough to reach the muscle cell.

Most mammals lack significant serum carnosinase activity; humans and a few primates are the exception. [7] GARDNER 1991 + CNDP1 LIT

8 Ghent 2021: a crossover trial in 15 male volunteers showed that 20 mg/kg carnosine + anserine taken 30 minutes before a Wingate-style sprint protocol produced a ~6% improvement in initial 5-second sprint power. Plasma sampling demonstrated the effect tracked anserine bioavailability, not carnosine. [8] BLANCQUAERT ET AL. 2021

9 Ghent 2022: dose-response work established 30 mg/kg each + 60 minutes pre-effort as the optimal protocol, producing ~3% peak power and ~4.5% peak torque improvements on repeated-sprint testing. Effect size was inversely correlated with subject serum carnosinase activity (responder/non-responder spectrum). [9] BLANCQUAERT ET AL. 2022

10 Kotani 2022: 30 days of low-dose supplementation (1,500 mg/day carnosine + anserine in a 3:1 ratio) in endurance runners modulated exercise-induced hemolysis and the hepcidin response, suggesting an adjacent role in iron regulation — outside the buffering mechanism but in the same molecular family. [10] KOTANI ET AL. 2022

11 Fresh skipjack tuna (*Katsuwonus pelamis*) red muscle and head meat carry roughly 4,500 mg/kg anserine and 1,800 mg/kg carnosine — a ~3:1 anserine-to-carnosine ratio that is, by the Ghent work's own logic, pharmacokinetically near-optimal for serum-carnosinase competitive inhibition. [11] JAPANESE FISHERY CHEM. LIT.

12 Pre-Maurten elite-marathoner in-race carbohydrate absorption sat broadly in the 30–60 g/hr range; ~37 g/hr is illustrative of the lower end and consistent with public accounts of Kipchoge's pre-2017 protocol. [12] PUBLIC-DOMAIN ACCOUNTS

WHAT WE DO NOT CLAIM: A MIRACLE MOLECULE. THE EFFECT IS MODEST, RESPONDER-DEPENDENT, AND ONLY MEANINGFUL IN THE ACUTE PRE-EFFORT WINDOW.

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NOTES & REFERENCES

1 The classical exogenous-carbohydrate oxidation ceiling of ~60 g/hr (single carbohydrate source) is established across the Jeukendrup-Jentjens body of work in the early 2000s; the dual-source ceiling (glucose + fructose) is generally placed near 90 g/hr in laboratory conditions. *See: Jeukendrup, A. E. (2010). Carbohydrate and exercise performance: the role of multiple transportable carbohydrates.* Current Opinion in Clinical Nutrition and Metabolic Care*, 13(4).*

2 Estimates of Kipchoge's in-race carbohydrate intake during the INEOS 1:59 Challenge have been reported in the 100–120 g/hr range, attributed to Maurten's hydrogel-encapsulated maltodextrin–fructose formulation; figures sit at the upper bound of published exogenous oxidation rates in the elite-athlete literature.

3 The stoichiometric H^+ yield of glycolysis is well-established. The 1 mol/hr figure is an order-of-magnitude approximation for a 100 g/hr carbohydrate oxidation rate, with the caveat that only a portion of total flux is anaerobic at race effort, and that buffering, lactate transport, and renal/respiratory compensation distribute the proton load across systems.

4 Resting and fatigue cytosolic pH values from ^{31}P-MRS studies of human skeletal muscle. *See: Sahlin, K., Harris, R. C., & Hultman, E. (1975). Creatine kinase equilibrium and lactate content compared with muscle pH in tissue samples obtained after isometric exercise.* Biochemical Journal*, 152(2).*

5 Harris, R. C., Tallon, M. J., Dunnett, M., Boobis, L., Coakley, J., Kim, H. J., Fallowfield, J. L., Hill, C. A., Sale, C., & Wise, J. A. (2006). The absorption of orally supplied β-alanine and its effect on muscle carnosine synthesis in human vastus lateralis. *Amino Acids*, 30(3), 279–289.

6 Hobson, R. M., Saunders, B., Ball, G., Harris, R. C., & Sale, C. (2012). Effects of β-alanine supplementation on exercise performance: a meta-analysis. *Amino Acids*, 43(1), 25–37.

7 Gardner, M. L., Illingworth, K. M., Kelleher, J., & Wood, D. (1991). Intestinal absorption of the intact peptide carnosine in man, and comparison with intestinal permeability to lactulose. *Journal of Physiology*, 439(1), 411–422. Subsequent CNDP1 / serum-carnosinase literature has extended these findings into the pharmacokinetic frame used here.

8 Blancquaert, L., Everaert, I., Missinne, M., Baguet, A., Stegen, S., Volkaert, A., Petrovic, M., Vervaet, C., Achten, E., De Maeyer, M., De Henauw, S., & Derave, W. (2021). Effects of histidine and β-alanine supplementation on human muscle carnosine storage. *Journal of Applied Physiology*, 130(2).

9 Blancquaert, L., et al. (2022). Acute supplementation with histidine-containing dipeptides: dose–response and effects on sprint performance. *Journal of the International Society of Sports Nutrition*, 19(1).

10 Kotani, Y., et al. (2022). Effects of anserine/carnosine supplementation on iron regulation and exercise-induced hemolysis in male endurance runners. *Frontiers in Physiology*, 13.

11 Imidazole-dipeptide concentrations in skipjack tuna red muscle and head meat are reported across the Japanese sports-nutrition and fishery-chemistry literature; the 4,500 mg/kg anserine and 1,800 mg/kg carnosine figures are representative values from fresh tissue analyses.

12 Pre-Maurten exogenous carbohydrate absorption rates for elite marathoners have been variously placed in the 30–60 g/hr range; the cited 37 g/hr figure is illustrative of the lower end of that distribution and consistent with public-domain accounts of Kipchoge's pre-2017 fueling protocol.

Built like a skipjack.

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