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INVITED COMMENTARY

Exogenous Lactate Supplementation: A Promising Fueling Strategy to Enhance Endurance Performance

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Purpose: Lactate has historically been mischaracterized as a metabolic byproduct associated with fatigue. The lactate shuttle paradigm describes its role in substrate exchange between glycolytic and oxidative tissues. Recent evidence has highlighted lactate as a key regulator of systemic energy metabolism and cellular signaling. While endogenous lactate production and clearance mechanisms during exercise are well established, the potential of exogenous lactate as an ergogenic fuel remains largely unexplored. Here, we aim to (1) synthesize current knowledge and discuss the mechanistic pathways through which exogenous lactate supplementation may enhance endurance performance in humans and (2) critically appraise the current limitations of high-dose exogenous lactate administration and outline novel formulation strategies, adapted from established sports nutrition practices, that warrant future investigation. ***Conclusion:*** Exogenous lactate supplementation holds promise as a novel fueling strategy to enhance endurance performance. However, human trials to date have failed to demonstrate performance benefits, potentially due to the low doses and bolus administration strategies employed. Future research should therefore focus on developing formulations capable of safely delivering physiologically relevant doses and evaluating their efficacy in well-controlled human studies.

Keywords: lactate shuttle, oral lactate, monocarboxylate transporters, sports nutrition, metabolism

The long-standing view that lactate accumulation in exercising muscle and circulation is responsible for metabolic acidosis and fatigue has been replaced by a more integrative understanding of lactate physiology. Lactate is now recognized as a key intermediary in carbohydrate metabolism, functioning as both a circulating energy substrate and a signaling molecule.[1,2] Beyond serving as a metabolic byproduct, lactate participates in interorgan substrate exchange and contributes to the coordination of whole-body energy metabolism.[3,4] During exercise, whole-body lactate turnover rates can exceed those of glucose on a molar basis.[5,6] However, when expressed in energetic terms, lactate and glucose fluxes are broadly comparable, highlighting lactate as a quantitatively important circulating energy substrate rather than merely a byproduct of glycolysis.[7–9]

From a nutritional perspective, endurance performance is partly constrained by the maximal rate of exogenous carbohydrate oxidation (~1.5–1.8 g/min).[9] This limitation is largely driven by the saturation of intestinal glucose transporters,[10,11] even when athletes consume very high carbohydrate intakes (up to 120 g/h).[12] Consequently, strategies capable of providing additional oxidizable substrates through intestinal transport systems distinct from those used by carbohydrates could theoretically increase total exogenous energy availability during prolonged exercise.

In this context, exogenous lactate supplementation may represent an intriguing possibility. Unlike carbohydrates, lactate is a monocarboxylate and is presumed to be absorbed through monocarboxylate transport systems (ie, apical sodium-coupled monocarboxylate transporter 1 [SMCT1] and/or monocarboxylate transporter 1 [MCT1], and basolateral MCT1) rather than glucose transporters, potentially bypassing one of the principal physiological constraints limiting carbohydrate delivery. Studies have shown that orally ingested lactate can be absorbed and oxidized in humans during exercise,[13,14] suggesting that it may contribute directly to energy metabolism. However, the intestinal transport mechanisms responsible for the absorption of orally ingested lactate in humans remain largely inferred from in vitro and animal studies,[15,16] and the physiological mechanisms through which exogenous lactate may improve endurance performance have not yet been comprehensively evaluated.

Accordingly, this commentary aims to (1) synthesize current knowledge and discuss the mechanistic pathways through which exogenous lactate supplementation may enhance endurance performance in humans and (2) critically appraise the current limitations of high-dose exogenous lactate administration and outline novel formulation strategies, adapted from established sports nutrition practices, that warrant future investigation.

Mechanistic Pathways of Exogenous Lactate: From "Fatigue" Metabolite to Ergogenic Aid

The lactate shuttle theory provides the conceptual foundation for understanding endogenous lactate metabolism. Lactate produced in highly glycolytic fibers (type II) is exported via MCT4 and taken up by oxidative tissues via MCT1.[4] Intracellularly, lactate is oxidized following its conversion to pyruvate by lactate dehydrogenase,

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entering the mitochondrial matrix and fueling the tricarboxylic acid cycle.[17] Almost 20 years ago, the presence of a mitochondrial lactate oxidation complex was demonstrated, supporting direct lactate oxidation within mitochondria in rat cells.[18] Notably, lactate is preferentially utilized by the myocytes and neurons during periods of elevated energy demand, supporting its role as a high-priority oxidative substrate.[19] Moreover, lactate serves as both a major gluconeogenic precursor and a signaling molecule, capable of inducing epigenetic programs linked to mitochondrial biogenesis and metabolic substrate partitioning.[1,20]

While the systemic role of endogenous lactate is well established, mechanistic and functional evidence on exogenous lactate remains limited. Unlike endogenous lactate, which is generated from glycolytic flux and cellular metabolism, exogenous lactate is introduced through oral ingestion or intravenous administration,[21,22] thereby reaching the systemic circulation independently of local glycolytic production.[22] Following oral ingestion, exogenous lactate is thought to be absorbed via MCTs[15,16] and to enter the portal circulation, where it is exposed to hepatic first-pass metabolism. Although circulating lactate is a major gluconeogenic precursor at rest,[5,23] available human tracer studies indicate that lactate ingested during exercise is predominantly oxidized, with little or no prior conversion to glucose.[13] A similar oxidation-dominated fate has been reported following intravenous administration of exogenous lactate.[21]

Lactate exists as 2 stereoisomers (L- and D-lactate), both of which can be produced by microorganisms. However, L-lactate is the predominant form in human metabolism and the primary product of glycolysis, whereas D-lactate is produced only in small amounts by human tissues and is primarily generated by gut microorganisms. Available evidence indicates that L-lactate is well tolerated at physiologically relevant doses (>10–20 g), making it the preferred stereoisomer for exogenous supplementation.[24] In contrast, although D-lactate has attracted increasing interest because of its distinct metabolism and potential biological effects, its safety and toxicity in humans remain incompletely understood.[25]

Within this framework, tracer studies demonstrate that exogenous lactate is rapidly utilized, exhibiting metabolic kinetics and oxidation rates comparable to glucose in humans.[13,14,21] By bypassing upstream glycolytic steps, lactate directly supports mitochondrial oxidative phosphorylation, representing a metabolically efficient substrate for adenosine triphosphate (ATP) production. This has been demonstrated in vitro with extracellular (exogenously supplied) lactate, which enters mitochondria and activates the electron transport chain.[17] As such, lactate could preserve cellular oxidative capacity, particularly under conditions of elevated glycolytic demand. Given the close link between endurance performance,[26] muscle function,[27] and the maintenance of muscle and hepatic glycogen stores,[28] glycogen-sparing strategies are critical. Exogenous lactate may attenuate glycogenolysis by serving as an alternative oxidative substrate to glucose, thereby promoting lactate oxidation and contributing to glycogen sparing, ultimately enhancing exercise capacity.[29,30] However, although glycogen sparing is generally considered beneficial for endurance performance, previous nutritional strategies aimed at achieving this outcome have not consistently translated into ergogenic benefits. For example, exogenous ketone ester ingestion frequently failed to reduce muscle glycogen breakdown or improve high-intensity performance in trained cyclists.[31,32] Therefore, whether lactate-induced glycogen sparing ultimately translates into improved endurance performance remains to be established.

Additionally, endogenous lactate exerts regulatory effects through both receptor-mediated and intracellular pathways. Activation of the hydroxycarboxylic acid receptor 1 (HCAR1/GPR81) inhibits adipose tissue lipolysis, particularly during high-intensity exercise, when preferential carbohydrate utilization over the more oxygen-demanding fat oxidation is metabolically advantageous.[33,34] In parallel, intracellular lactate modulates transcriptional pathways, including PGC-1α signaling, thereby promoting mitochondrial biogenesis.[1] These complementary actions establish lactate not only as an oxidative substrate but also as a signaling molecule "lactormone" that coordinates whole-body energy metabolism. Consequently, exogenous lactate may complement endogenous lactate by acting as both an additional oxidative substrate and a metabolic signal, with its relative contribution likely depending on exercise intensity and metabolic demand, including whether exercise is performed below or above the lactate threshold.

Role of Exogenous Lactate as a Substrate Below the Lactate Threshold: Regulation During Low- to Moderate-Intensity Exercise

Following intestinal absorption and entry into the circulation, it can be hypothesized that oral exogenous lactate may support oxidative metabolism below the lactate threshold by increasing its uptake via MCT1/MCT4 in the skeletal muscle, thereby augmenting mitochondrial substrate availability for ATP resynthesis during low- to moderate-intensity exercise, where oxygen delivery meets demand.[4,35] Lactate availability might facilitate greater reliance on oxidative phosphorylation through lactate stimulation, supporting increased pyruvate entry into mitochondria and promoting acetyl-CoA formation and tricarboxylic acid cycle flux.[17] In turn, this metabolic profile reduces dependence on glycolytic carbohydrate breakdown and contributes to glycogen sparing.[21,30] Although endogenous lactate is associated with reduced systemic lipolysis when accumulated, exogenous lactate direct oxidation in skeletal muscle may therefore promote a relative increase in fat oxidation during low-to-moderate lactatemia (low-to-moderate exercise intensity), alongside improved glycogen storage and metabolic efficiency, as suggested in mice (see Figure 1).[36,37] Notably, the effects of lactate on lipid mobilization appear to be concentration-dependent, with human infusion studies showing little or no suppression of lipolysis or changes in substrate oxidation at physiological lactataemia,[38,39] supporting the possibility that exogenous lactate could serve as an oxidative substrate at low-to-moderate exercise intensities without substantially impairing lipid mobilization. Moreover, as a gluconeogenic precursor, exogenous lactate may enhance Cori cycle activity by increasing hepatic gluconeogenic substrate availability.[4] In the Cori cycle, circulating lactate is taken up by the liver and reconverted into glucose via gluconeogenesis, after which glucose can return to the circulation and be reutilized by skeletal muscle. Therefore, exogenous lactate may contribute to the Cori cycle by supporting hepatic glucose production and promoting glycogen sparing during prolonged low- to moderate-intensity exercise.[40] However, whether orally ingested lactate meaningfully enhances Cori cycle activity remains unknown. Indeed, during exercise, ingested lactate appears to be preferentially oxidized rather than converted into glucose, suggesting that this mechanism may be more relevant at rest or during recovery.[13]

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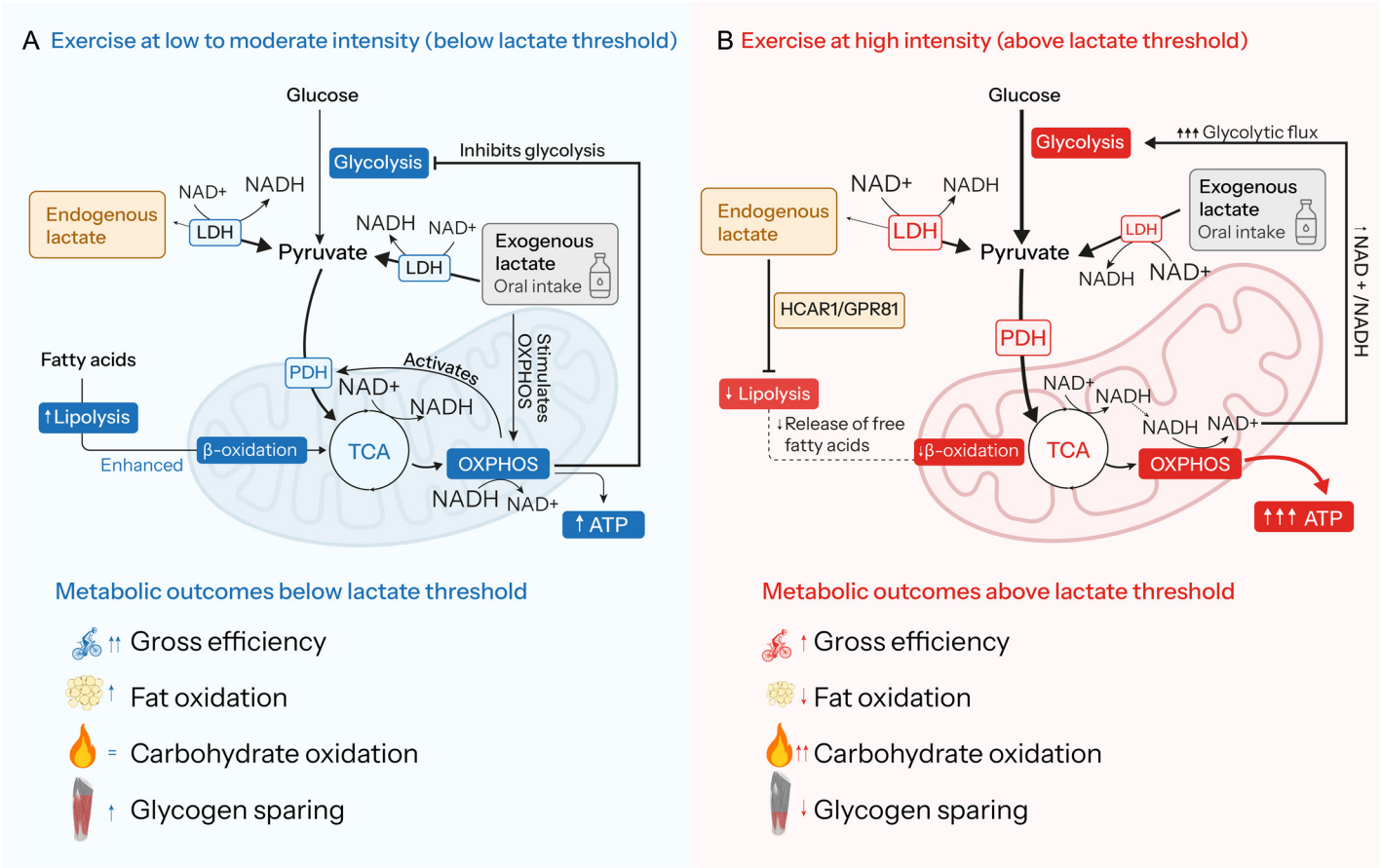


Figure 1 — Potential role of exogenous lactate during exercise performed below and above the lactate threshold. ATP indicates adenosine triphosphate; HCAR1/GPR81, hydroxycarboxylic acid receptor 1/G protein-coupled receptor 81; LDH, lactate dehydrogenase; NAD^+, nicotinamide adenine dinucleotide (oxidized); NADH, nicotinamide adenine dinucleotide (reduced); OXPHOS, oxidative phosphorylation; PDH, pyruvate dehydrogenase; TCA, tricarboxylic acid cycle.

Role of Exogenous Lactate as a Substrate Above the Lactate Threshold: Regulation During High-Intensity Exercise

In contrast to low-intensity conditions, at exercise intensities above the lactate threshold, where oxygen demand approaches or exceeds delivery, carbohydrate oxidation is already the predominant substrate,[34] leaving limited room for further metabolic shift; nonetheless, exogenous lactate may provide an additional oxidative substrate within this already carbohydrate-dominant metabolic profile. Any resulting contribution to ATP production rate or oxygen cost per unit of power relative to fat oxidation remains to be empirically determined.[35]

Mechanistically, these effects may be partly mediated by redox control.[1,4] The interconversion of lactate and pyruvate via lactate dehydrogenase buffers the cytosolic NADH/NAD^+ (nicotinamide adenine dinucleotide [reduced]/nicotinamide adenine dinucleotide [oxidized]) ratio, facilitating sustained glycolytic flux while preserving conditions that allow continued mitochondrial NADH oxidation.[2] By stabilizing redox balance under high flux conditions, exogenous lactate may support the coupling between rapid glycolysis and oxidative phosphorylation, thereby contributing to ATP resynthesis at high intensities.[1,2] Additionally, given the pH sensitivity of key metabolic processes, lactate-associated proton handling may help attenuate local disturbances in pH and preserve enzymatic function.

In parallel, exogenous lactate might theoretically increase intracellular pyruvate availability following its conversion to pyruvate (see Figure 1). Beyond serving as a metabolic substrate, pyruvate directly inhibits pyruvate dehydrogenase kinase, the enzyme responsible for phosphorylating and inactivating pyruvate dehydrogenase (PDH), thereby favoring the active form of the enzyme.[41,42] This covalent regulatory mechanism is distinct from the end-product inhibition exerted by acetyl-CoA and NADH. As PDH activation increases with exercise intensity,[43] lactate-derived pyruvate may further support carbohydrate oxidation by sustaining acetyl-CoA production whenever mitochondrial oxidative capacity is not limiting.

Concurrently, elevated lactate availability contributes to the suppression of fat oxidation. Increased carbohydrate flux elevates acetyl-CoA and malonyl-CoA concentrations, inhibiting carnitine palmitoyltransferase 1 and limiting mitochondrial fatty acid transport.[20] Lactate-mediated signaling and associated redox shifts may further constrain β-oxidation, reducing reliance on fat, a substrate with a higher oxygen cost and slower ATP kinetics.[44]

Collectively, exogenous lactate may support gross efficiency at high intensities by stabilizing redox balance, augmenting PDH-mediated carbohydrate oxidation, and suppressing fat oxidation. This integrated regulation favors substrate utilization patterns that sustain high ATP turnover while minimizing oxygen cost, thereby enhancing the efficiency of high-intensity exercise. Importantly,

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these mechanisms remain largely theoretical and await confirmation in humans during exercise. To date, supporting evidence derives exclusively from animal models. For example, chronic exogenous lactate administration has been reported to increase carbohydrate oxidation during exercise[45] and to improve exercise capacity,[46] while acute administration likewise increased carbohydrate oxidation during exercise.[47] Whether the mechanisms proposed here underlie these observations remains to be established.

Role of Exogenous Lactate as a Substrate During Recovery and Resting Conditions

Beyond endurance exercise, the metabolic and regulatory effects of exogenous lactate may extend to resting conditions, including the recovery period between training sessions, where oxidative metabolism predominates. In mouse models, exogenous lactate supplementation under resting conditions shifts substrate utilization toward greater lipid oxidation and lower carbohydrate utilization, suggesting a modulation of basal metabolic partitioning.[48] This shift may contribute to support glycogen resynthesis in liver and skeletal muscle, suggesting a role for lactate in postexercise metabolic recovery.[30] Additionally, lactate is preferentially oxidized in neurons when available, regardless of whether it is delivered exogenously or produced endogenously, thereby contributing to the sparing of glucose and glycogen stores both in healthy and injured states.[49,50] In murine models of skeletal muscle injury, exogenously administered lactate has been reported to support tissue repair: intraperitoneal sodium lactate enhanced regeneration in glycerol-injured muscle[51] and oral sodium lactate promoted hypertrophy and satellite-cell expansion after cardiotoxin injury.[52] Consistent with this, endogenous endothelial-derived lactate drives proregenerative M2-like macrophage polarization during hindlimb ischemia.[53] Whether these effects translate to humans, or to the doses achievable through oral supplementation, remains to be established. Collectively, these findings indicate that exogenous lactate administration at rest may influence metabolic regulation, recovery processes, and tissue adaptation. Importantly, these effects may not be limited to host tissues alone, but could also involve interactions with the gut microbiota, representing an additional layer of metabolic regulation.

Recent evidence indicates that ^{13}C-labeled lactate can cross the intestinal epithelium and reach the gut lumen, providing a potential substrate for the gut microbiota. Certain bacterial species, such as *Veillonella atypica,* can metabolize lactate and produce short-chain fatty acids, including propionate. Notably, this pathway has been directly linked to endurance capacity, as colonization of mice with *V. atypica* increased treadmill run time to exhaustion by ~13%, an effect reproduced by intraluminal propionate alone.[54] Propionate is transported across enterocytes via MCT1, reaches the systemic circulation, and may subsequently influence skeletal muscle metabolism. Therefore, it may be plausible that a fraction of ingested lactate that escapes proximal absorption may reach the colon, where lactate-utilizing bacteria can convert it into short-chain fatty acids (eg, acetate, propionate) through microbial cross-feeding,[55,56] although direct evidence that ingested lactate reaches the human colon is currently lacking. Given the efficient proximal absorption of ingested lactate, the extent of this luminal interaction at supplement-relevant doses remains to be established. This highlights a previously underappreciated pathway for lactate metabolism that extends beyond classical routes such as the Cori cycle. It is therefore biologically plausible, although presently untested, that targeted delivery of exogenous lactate to the colon could serve as a substrate for specific microbial populations, promoting secondary metabolites that influence host metabolism and potentially contribute to training-associated adaptations or to the efficiency of endogenous substrate utilization during recovery.

Beyond Carbohydrate Limits: Lactate–Carbohydrate Co-Feeding as a Dual-Fuel Strategy for Endurance Performance

To date, substantial evidence demonstrates that high rates of carbohydrate ingestion enhance endurance performance and support postexercise recovery.[34,57] However, these strategies are ultimately constrained by intestinal absorption limits, as glucose and fructose rely on sodium-glucose cotransporter 1 (SGLT1) and glucose transporter 5 (GLUT5), which become saturated at high ingestion rates. In this context, combining carbohydrate-rich feeding strategies with exogenous lactate may help overcome these limitations and further enhance exogenous energy provision. If exogenous lactate can be delivered at sufficiently high rates (eg, ~10–25 g/h), its co-ingestion with carbohydrates during exercise may expand current carbohydrate-based fueling strategies by introducing a complementary substrate. Whereas glucose and fructose depend on SGLT1 and GLUT5 in the enterocytes, lactate is absorbed via MCTs, primarily MCT1, providing a parallel, noncompetitive intestinal uptake pathway (see Figure 2). This may enable an increase in total substrate flux without proportionally increasing gastrointestinal transport competition. Once absorbed, exogenous lactate can be rapidly converted to pyruvate and oxidized in skeletal muscle and other tissues, contributing directly to mitochondrial ATP production while supporting cytosolic redox balance through the lactate–pyruvate equilibrium and substrate regulation. This multipathway fueling paradigm may significantly expand the ceiling of exogenous energy provision, with carbohydrate intake reaching ~90 to 120 g/h, limited by SGLT1 and GLUT5 saturation, and lactate contributing an additional ~10–25 g/h via MCT-mediated transport.[12,60]

Current Human Evidence on Lactate-Derived Supplements and Performance Outcomes

So far, 13 controlled human trials have tested the effects of orally ingested lactate-derived compounds on exercise outcomes. Importantly, these studies have used heterogeneous formulations, doses, and delivery strategies, making direct comparisons difficult and potentially influencing the physiological effects of lactate. Early studies administered a lactate polymer during prolonged submaximal cycling, eliciting responses comparable to an isocaloric glucose polymer but no additional performance benefit.[61,62] Similarly, ingestion of a 2% lactate solution, with or without carbohydrate, did not alter time to exhaustion or peak power.[63] Subsequent studies largely reframed lactate as an extracellular buffer,[64] typically administering single preexercise boluses of sodium, calcium, or magnesium lactate salts (≈19–375 mg/kg) before short, high-intensity exercise. These interventions failed to improve sprint running to exhaustion,[65] 20- or 40-km cycling time trials,[66,67] repeated upper-body Wingate performance either acutely[68] or after 5 days of loading,[69] $\dot{V}O_2$peak or the onset of

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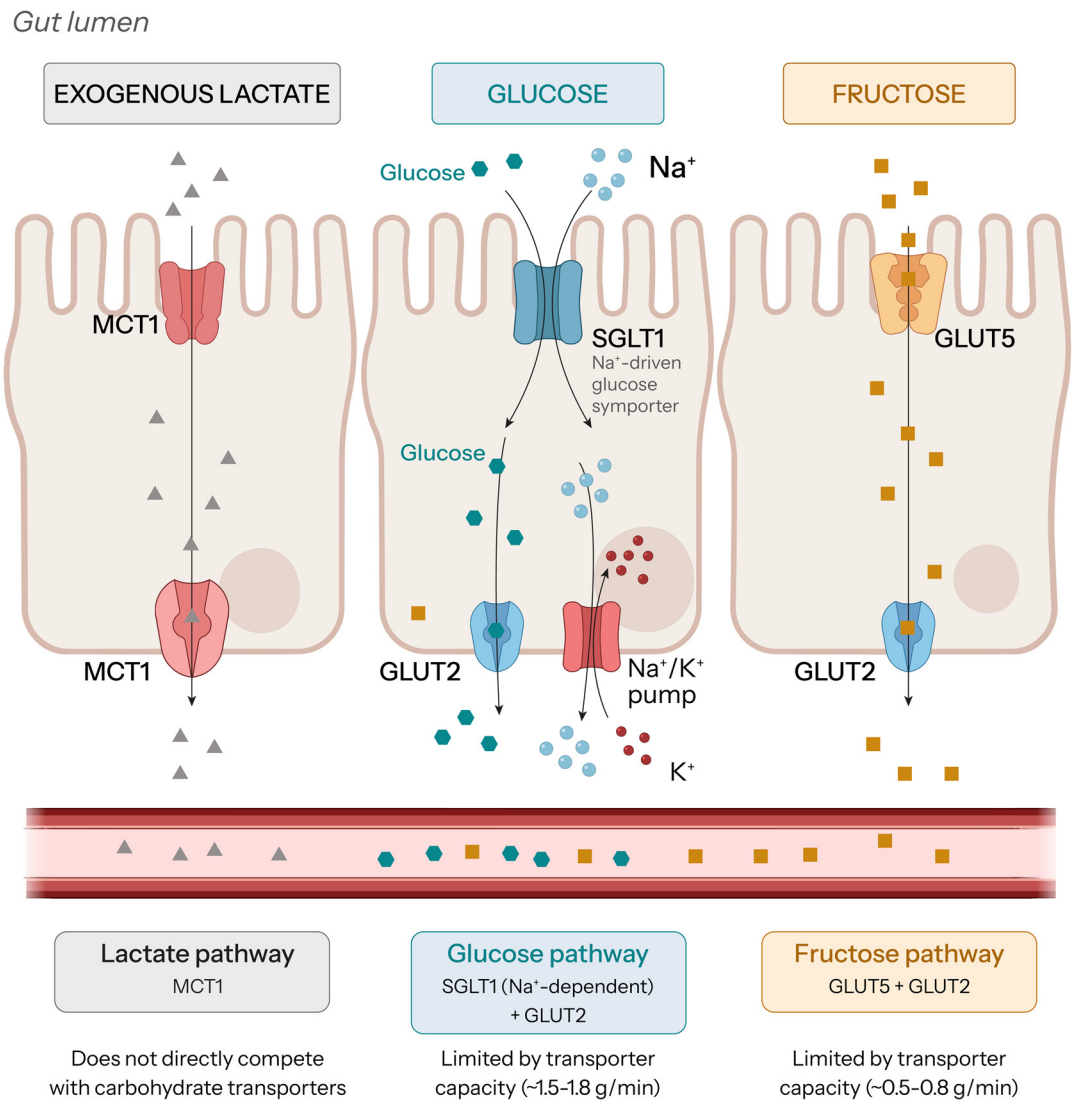


Figure 2 — Intestinal absorption pathways for lactate, glucose, and fructose, representing the dual-fuel model. GLUT2 indicates glucose transporter 2; GLUT5, glucose transporter 5; K^+, potassium ion; MCT1, monocarboxylate transporter 1; Na^+, sodium ion; SGLT1, sodium-glucose cotransporter 1. Based on concepts from Brooks[58] and Jeukendrup[59] with the inclusion of exogenous lactate pathways.

blood lactate accumulation,[70] and repeated 1- and 4-km time trials. In the latter study, perceived exertion was reduced ($P = .012$, $\eta_p^2 = .423$), while significantly increasing flatulence, belching, stomach cramps, stomachache, and bowel urgency.[71] Only 2 studies reported performance benefits of lactate supplementation: a 17% longer time to exhaustion after 120 mg/kg of calcium lactate ingested alone,[72] and a 4% higher mean work rate during a 20-minute time trial after a commercial calcium and magnesium lactate supplement providing ≈1.1 to 1.5 g of lactate 19 mg/kg, with $\dot{V}O_2$peak unchanged.[73] Acute oral lactate ingestion has also been shown to increase resting oxygen consumption without altering physiological responses during exercise.[74]

Collectively, current evidence indicates that lactate-derived supplements, as formulated and dosed to date, do not consistently improve exercise performance. Importantly, however, these studies primarily tested lactate as a buffering agent rather than as a fuel. Lactate was generally administered as a single preexercise bolus of a cation-bound salt, rather than continuously during exercise at higher doses (eg, ~10–25 g/h) to provide a meaningful oxidative contribution. Moreover, most protocols involved short, high-intensity exercise, where lactate production is already high, and no study has directly quantified the oxidation of orally ingested lactate during exercise. Thus, the potential of exogenous lactate as an exercise fuel remains largely untested rather than refuted.

From Physiological Potential to Practical Limitations: Challenges in Exogenous Lactate Delivery

Despite these promising physiological roles, the practical implementation of exogenous lactate supplementation in humans remains challenging, although novel solutions capable of delivering high oral doses on an hourly basis during endurance exercise have been proposed recently. This limitation is largely driven by the constraints of currently available administration methods, including isotope tracer approaches, intravenous infusion, and conventional oral ingestion.[75]

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Previous studies have investigated oral exogenous lactate administration (120–375 mg/kg or 372–1115 mg absolute) using sodium or calcium salts, administered either as a single bolus or through continuous supplementation protocols, across a range of physiological outcomes.[14,67,71,73,74,76] Early studies using lactate tracers employed even lower doses (~500 mg/h), primarily to assess oxidation kinetics rather than ergogenic effects.[14] Overall, these protocols have failed to elicit meaningful physiological or performance outcomes, likely due to the insufficient rates of exogenous lactate delivery.

In contrast, when higher doses of exogenous lactate have been administered, measurable systemic effects have been observed. For instance, the oral administration of 25 g of sodium lactate has been shown to increase blood lactate concentration (>2 mmol/L), as well as modulate growth differential factor 15 and gut-related hormones (ie, lowered ghrelin and increased glucagon-like peptide-1, insulin, and glucagon) in healthy humans at resting conditions.[22] Moreover, these effects led to slower gastric emptying relative to intravenous administration at matched lactatemia, which in the context of the co-supplementation with carbohydrates, could limit lactate delivery and absorption rates. Similarly, studies employing infusion or clamp techniques with intravenous exogenous lactate administration rates ranging from 13 to 24 g/h[21,77,78] and up to 60 g/h[49] have demonstrated increases (>1 mmol/L) in circulating lactate concentrations at rest and metabolic effects, including increased cerebral lactate utilization with reduced glucose utilization, suppressed whole body lipolysis, increased lactate oxidation, reduced glucose production, and glucose sparing.

From a practical perspective, conventional oral bolus-based approaches provide relatively low rates of lactate delivery, well below those likely required to meaningfully contribute to whole-body energy metabolism during prolonged endurance exercise. Based on infusion studies, sustained intakes in the range of ~10 to 25 g/h may be necessary to increase oxidative substrate availability.[21,49] Over prolonged exercise durations (eg, 3–5 h), this would correspond to cumulative lactate intakes of ~30 to 125 g, comparable to established carbohydrate feeding strategies, and may therefore enable physiological effects not observed in previous low-dose studies. For these effects to be translated into exercise practice, such exogenous delivery rates must be achievable through oral formulations that are approved for human use, well tolerated, and capable of delivering sufficient lactate to the systemic circulation. Under the intended conditions of use, tolerability refers to preservation of acid–base balance and the absence of clinically relevant gastrointestinal intolerance. Intravenous administration during exercise is neither feasible nor legally compliant.[75]

Nevertheless, achieving such intakes using conventional salt-based formulations would require excessive cation loads, particularly sodium or calcium, increasing the risk of gastrointestinal distress and electrolyte imbalance.[67] In addition, oral exogenous lactate ingestion may induce systemic alkalinization due to hepatic metabolism and accompanying cations. While mild alkalosis may enhance buffering capacity, excessive alterations in acid–base balance could impair enzymatic function and excitation–contraction coupling.

Taken together, these limitations highlight the need for novel delivery strategies capable of providing high rates of oral exogenous lactate without compromising gastrointestinal tolerance or electrolyte balance. Emerging approaches, including lactate esters, combined salt formulations, and polymer-based formulations, alongside physicochemical technologies such as hydrogel systems and nano- and micro-encapsulation, may help overcome these limitations. For example, ketone ester development has enabled exogenous ketone delivery in high doses (ie, up to 20 g/h),[79] and the co-ingestion of multiple transportable carbohydrates (glucose plus fructose) raises the ceiling of exogenous carbohydrate oxidation beyond that achievable with glucose alone.[10] Crucially, however, advances in delivery have not consistently translated into validated benefits in humans. Despite well-tolerated, high-dose ketone formulations, exogenous ketones frequently failed to enhance, and at times impaired, high-intensity performance.[31,32] Similarly, hydrogel encapsulation has been proposed to improve gastrointestinal tolerability and permit higher carbohydrate intakes during exercise, although controlled trials isolating the hydrogel format from carbohydrate composition have not consistently confirmed benefits in exogenous oxidation or gastrointestinal symptoms.[80–84]

By analogy, advanced lactate delivery systems could enhance systemic lactate availability without excessive ionic load or osmotic stress. Achieving resting circulating lactate in the range ~2 to 4 mmol/L through oral exogenous supplementation could theoretically mimic exercise-induced metabolic states without the associated glycolytic cost, potentially conferring a metabolic advantage for endurance performance. Importantly, such strategies may also have broader applications in clinical and health-related contexts, including metabolic regulation, recovery, and conditions characterized by impaired energy metabolism.

Practical Applications

Current endurance nutritional strategies are largely constrained by the intestinal absorption limits of carbohydrates. Exogenous lactate supplementation may represent a novel complementary fueling strategy by providing an additional oxidative substrate through MCTs, which operate independently of glucose and fructose transport mechanisms. If future formulations can safely deliver physiologically relevant doses (eg, 10–25 g/h), lactate co-ingestion with carbohydrates could increase total exogenous energy availability during prolonged exercise. Beyond its potential role as a fuel, exogenous lactate may also contribute to glycogen sparing, support metabolic flexibility, and facilitate recovery processes. Although current evidence remains largely mechanistic and proof of concept, advances in delivery technologies such as lactate esters, hydrogels, and encapsulation systems may enable practical applications in endurance sports. Future intervention studies are needed to determine whether these theoretical advantages translate into meaningful improvements in exercise performance.

Conclusions

Exogenous lactate supplementation, though still in its infancy, represents a compelling opportunity to support endurance performance through a complementary fueling pathway, provided that delivery rates substantially exceeding those tested to date can be safely achieved. Advances in formulation and a better understanding of exogenous lactate metabolism during exercise will be essential to determine its practical potential. If demonstrated, this approach could reposition lactate from a historically misunderstood metabolic byproduct to a strategic exogenous fuel for endurance exercise.

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