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The Impact of Exercise-Related Metabolites on Metabolic Health

By Moran Liu

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Moran Liu is a senior at Middlesex School in Concord, Massachusetts. She lives in North Billerica and is from Beijing, China. She is passionate about biological sciences, nutrition, and neuroscience, and has conducted research on metabolism and public health. As Editor-in-Chief of her school's science magazine and captain of the varsity volleyball team, she enjoys combining analytical thinking with teamwork and leadership. Moran hopes to pursue studies in biology and public health in college, aiming to bridge scientific research with real-world impact.

ABSTRACT

Muscle health and insulin sensitivity are crucial for metabolic homeostasis and overall well-being. This study investigates the distinct effects of succinic acid (SA) and lactic acid (LA), two key metabolites, on insulin sensitivity, myogenesis, and myofiber dynamics. Our results reveal that moderate concentrations of SA enhance insulin sensitivity by increasing the phosphorylation of key signaling proteins such as AKT and ERK. However, higher concentrations of SA promote myofiber degradation, as evidenced by elevated Myostatin levels, indicating a dual role in metabolic regulation and muscle integrity. In contrast, LA moderately enhances insulin sensitivity at higher concentrations without significantly influencing myogenesis or inducing myofiber degradation, suggesting a more protective role in muscle health. These findings have important implications for dietary and metabolic health. Nutritional sources that influence the levels of SA and LA in the body could differentially impact muscle function and metabolic processes. Moderate SA levels may support insulin sensitivity, whereas excessive SA could impair muscle repair and growth. LA, by maintaining muscle integrity, emerges as a safer metabolite in muscle-related interventions. Our study underscores the importance of balanced nutritional strategies in promoting muscle health, particularly through the regulation of metabolite levels. These insights provide a foundation for future research into dietary and metabolic interventions aimed at enhancing muscle integrity, improving insulin sensitivity, and preventing muscle degeneration associated with aging and metabolic disorders.

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INTRODUCTION

Metabolic Health and Insulin Sensitivity

Metabolic health refers to the body's ability to efficiently regulate and utilize energy, which includes maintaining balanced blood sugar levels, managing cholesterol and triglycerides, regulating blood pressure, and achieving a healthy body weight. Proper metabolic function significantly reduces the risk of chronic conditions such as diabetes, cardiovascular disease, and stroke (Chomiuk et al., 2024).

A critical component of metabolic health is insulin sensitivity, which measures how effectively the body's cells respond to insulin, the hormone responsible for regulating blood sugar levels. When muscle, fat, and liver cells are highly sensitive to insulin, they absorb glucose from the bloodstream for immediate use or storage, maintaining stable blood sugar levels. Conversely, insulin resistance, or reduced insulin sensitivity, disrupts this process, leading to elevated blood sugar levels and a heightened risk of metabolic diseases (Cleveland Clinic, 2024).

Insulin resistance is a major driver of type 2 diabetes (T2D), a chronic condition characterized by persistent hyperglycemia due to impaired insulin action or insufficient insulin production. In the United States, over 84 million adults have prediabetes—a precursor to T2D—emphasizing its widespread prevalence. Insulin resistance disrupts glucose uptake and often requires increased insulin production, creating a cycle that culminates in prediabetes or T2D (Zhao et al., 2023). Preventive strategies, including balanced diets, regular exercise, and healthy weight maintenance, are essential for improving insulin sensitivity and supporting overall metabolic health. Factors contributing to T2D include genetics, obesity, certain medications, aging, and sedentary lifestyles, with obesity-induced inflammation playing a particularly significant role in insulin resistance.

Exercise and Metabolic Health

Regular physical activity is a cornerstone of metabolic health, improving insulin sensitivity, lipid profiles, blood pressure, and body composition. Exercise reduces visceral fat, decreases pro-inflammatory markers, and increases anti-inflammatory cytokines (Chomiuk et al., 2024). These effects lower the risk of metabolic syndrome (MetS) and related diseases such as obesity and T2D by enhancing glucose uptake and mitigating systemic inflammation.

Exercise also triggers the production of myokines—cytokines secreted by muscle tissue—and modulates oxidative stress pathways, which are critical for reducing inflammation and preventing metabolic disorders.

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These physiological adaptations demonstrate the profound link between regular physical activity and improved metabolic health outcomes (Kim & Kim, 2020).

Exercise-Related Metabolites: Lactic Acid and Succinic Acid

Lactic acid, produced during anaerobic respiration, plays a dual role in muscle function and energy metabolism. During exercise, when oxygen is limited, lactate serves as a vital alternative energy source, linking glycolysis with oxidative phosphorylation and gluconeogenesis. Beyond its role in energy production, lactate influences insulin sensitivity, regulates protein function, and modulates gene transcription, contributing to cell proliferation and epigenetic modifications.

However, excessive lactic acid accumulation can impair muscle function by slowing metabolic processes and increasing inflammation, resulting in fatigue and muscle pain (Cleveland Clinic, 2023). Although lactate is often associated with muscle fatigue and acidosis, emerging research highlights its function as a valuable metabolic fuel for skeletal muscles, the heart, and the brain (Lee, 2021). This duality underscores lactate's complex role in energy metabolism and its potential impact on insulin signaling and glucose uptake.

Succinic acid, a key component of the tricarboxylic acid (TCA) cycle, plays an essential role in cellular energy production by supporting ATP generation via oxidative phosphorylation. Recent evidence suggests succinic acid enhances mitochondrial respiration in skeletal muscle, improving oxygen utilization and reducing fatigue (Fernández-Veledo et al., 2024). Additionally, succinic acid promotes the transition of muscle fibers from fast-twitch to slow-twitch types, which are more efficient for endurance activities.

While succinic acid shows promise in improving mitochondrial function and reducing muscle fatigue, its role in insulin sensitivity and glucose regulation remains inconclusive. However, it holds potential as a supplement to enhance muscle energy and function, particularly for individuals unable to engage in regular physical activity (Lattibeaudiere & Alexander-Lindo, 2024).

Muscle Tissue and Metabolic Health

Muscle Tissue Function and Differentiation

Muscle tissue, composed of specialized fibers capable of contraction, is critical for movement, physical performance, and metabolic health. Skeletal muscle, in particular, plays a key role in insulin-mediated glucose uptake via glucose transporter 4 (GLUT4) (Kim & Kim, 2020). A loss of skeletal muscle mass can result in

SCHOLARLY REVIEW

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insulin resistance, increased lipolysis, and elevated free fatty acids, which further impair muscle regeneration and exacerbate metabolic dysfunction.

Muscle tissue differentiation is vital for regeneration and function. Factors such as insulin resistance and chronic inflammation disrupt this process, leading to reduced muscle mass and metabolic disorders. Insulin resistance promotes gluconeogenesis and muscle protein breakdown, while chronic inflammation elevates pro-inflammatory cytokines, compounding muscle dysfunction (Zhang et al., 2019). These disruptions highlight the need for therapeutic strategies aimed at enhancing muscle regeneration and improving metabolic health.

Insulin Sensitivity and Diabetes

Muscle mass and function are closely tied to insulin sensitivity. Sarcopenia, or the loss of muscle mass and strength, is associated with metabolic disorders such as diabetes (Zhao et al., 2023). Insulin resistance in skeletal muscle impairs glucose uptake, increasing the risk of diabetes.

Myokines, secreted by muscle tissue, are critical for metabolic health. Exercise enhances muscle mass and promotes the release of beneficial myokines such as interleukin-6 (IL-6), which improves glucose uptake, fat oxidation, and insulin sensitivity (Hwang et al., 2015). Chronic inflammation associated with sarcopenia, however, exacerbates insulin resistance and metabolic dysfunction. Thus, preserving muscle mass and function is essential for preventing insulin resistance and managing metabolic disorders like diabetes.

METHODS

Cell culture

C2C12 mouse myoblast cells, obtained from the American Type Culture Collection (ATCC), were cultured in Dulbecco's Modified Eagle Medium (DMEM; Corning) supplemented with 10% fetal bovine serum (FBS; Sigma-Aldrich) and 1% penicillin-streptomycin (Gibco). For myoblast differentiation, the 10% FBS was replaced with 5% horse serum (Gibco). The cells were maintained at 37°C in a humidified atmosphere containing 5% CO_2, and the culture medium was refreshed daily.

To investigate the effects of lactate and succinate on myoblast differentiation, C2C12 cells were seeded in 6-well plates, and treatments with the metabolites began on the third day following the initiation of differentiation. For studies examining myofiber degradation, the metabolites were introduced seven days after differentiation induction.

SCHOLARLY REVIEW

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To assess insulin resistance, cells were treated with the metabolites, followed by the addition of 100 nM insulin. Samples were collected at various time points, and downstream protein phosphorylation was measured to evaluate insulin signaling activity.

Western blots

Western blotting was performed to quantify protein levels, following standard protocols. Protein samples were resolved by SDS-PAGE under reducing conditions and then transferred to nitrocellulose membranes (GE Healthcare) using electrophoresis. The membranes were blocked with Odyssey Blocking Buffer (LI-COR) prepared in Tris-Buffered Saline (TBS).

Primary antibodies targeting specific proteins were applied overnight at 4°C, including: Myosin (Sigma-Aldrich, M4276, 1:2000), Myostatin (Proteintech, 19142, 1:2000), Vinculin (Sigma-Aldrich, V9264, 1:5000), Phospho-AKT (S308) (Cell Signaling Technology, 13038, 1:2000), AKT (pan) (Cell Signaling Technology, 4691, 1:2000), Phospho-p44/42 MAPK (Erk1/2) (Thr202/Tyr204) (Cell Signaling Technology, 9101, 1:2000), p44/42 MAPK (Erk1/2) (Cell Signaling Technology, 4696, 1:2000). After primary antibody incubation, membranes were washed three times with TBST and incubated with secondary antibodies—donkey anti-rabbit Alexa Fluor 488 (A-21206; Thermo Fisher Scientific, 1:10,000) or donkey anti-mouse Alexa Fluor 555 (A31570; Thermo Fisher Scientific, 1:10,000)—for 1 hour at room temperature. Fluorescent signals were visualized using the LI-COR Odyssey CLx Imaging System, and data were analyzed for protein quantification.

RESULTS

The Effect of Succinic Acid and Lactic Acid on Insulin Sensitivity

To evaluate the effects of succinic acid (SA) and lactic acid (LA) on insulin sensitivity, C2C12 myoblasts were treated with metabolites at varying concentrations. Insulin (100 nM) was then added to induce signaling, and phosphorylation levels of downstream proteins were measured at 15 and 30 minutes post-treatment.

<u>15-Minute Treatments</u>

Succinic Acid (SA): Phosphorylation levels following SA treatment were comparable to the control group at lower concentrations (SA-1: 100 μM and SA-2: 200 μM). As the concentration increased to SA-5 (500 μM) and SA-10 (1.0 mM), phosphorylation decreased slightly, but at SA-15 (1.5 mM), phosphorylation levels

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rebounded to match those of the control. This pattern indicates a concentration-dependent modulation, with SA-15 demonstrating the most significant insulin response enhancement.

Lactic Acid (LA): LA treatments showed an overall similar trend but with slightly weaker effects compared to SA. At LA-1 (100 μM) and LA-2 (200 μM), phosphorylation was lower than the control. At LA-5 (500 μM), phosphorylation increased noticeably, showing a stronger response compared to lower concentrations. This trend continued at LA-10 (1.0 mM), with phosphorylation peaking at LA-15 (1.5 mM). However, LA-15 also exhibited a slight drop in p-ERK, while p-AKT phosphorylation remained elevated.

30-Minute Treatments

Succinic Acid (SA): At 30 minutes, SA treatments consistently enhanced phosphorylation of insulin signaling markers. SA-1 and SA-2 showed moderate increases in p-ERK and p-AKT compared to the control, while SA-10 and SA-15 exhibited the most robust phosphorylation levels, indicating enhanced insulin sensitivity.

Lactic Acid (LA): LA treatments showed less phosphorylation overall compared to SA. While LA-1 and LA-2 demonstrated lower phosphorylation than the control, LA-5 and LA-10 displayed incremental improvements, with LA-15 achieving the strongest insulin response. However, LA-induced phosphorylation remained weaker than SA across all concentrations

Comparison of SA and LA

Both SA and LA exhibited dose-dependent effects on insulin signaling, with the strongest responses observed at the highest concentrations (SA-15 and LA-15). However, SA consistently outperformed LA, producing stronger and more stable activation of insulin sensitivity markers across both time points. At lower concentrations, SA demonstrated greater phosphorylation enhancement compared to LA, which showed reduced efficacy at these doses.

Overall, the results indicate that SA is more effective than LA in enhancing insulin sensitivity. SA treatments yielded higher phosphorylation levels of key signaling proteins (p-ERK and p-AKT) at nearly all concentrations and time points, highlighting its stronger and more consistent ability to potentiate insulin signaling.

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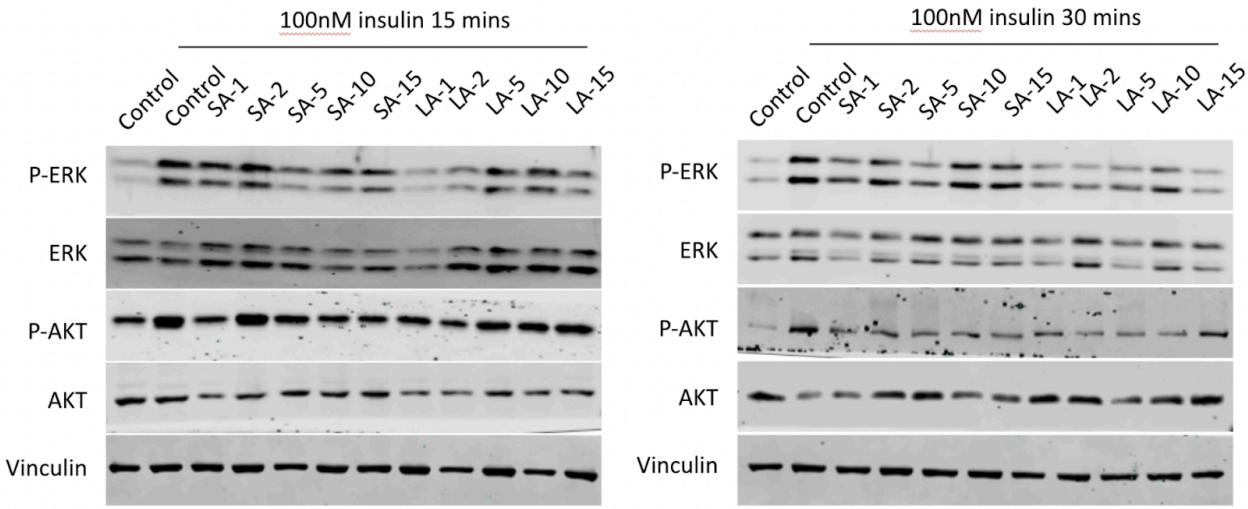

Figure 1. The activity of insulin signaling.

The Effect of Metabolites on Myogenesis

This study examined the effects of succinic acid (SA) and lactic acid (LA) on myogenesis by measuring the expression levels of Myosin and Vinculin, key markers of muscle cell differentiation.

Succinic Acid (SA): SA treatment significantly enhanced myogenesis in a concentration-dependent manner. At lower concentrations (SA-1 and SA-2), there was a noticeable increase in Myosin and Vinculin expression compared to the control, indicating a stimulatory effect on muscle cell differentiation. The expression of Myosin peaked at SA-5, suggesting that this concentration was optimal for promoting myogenesis. However, at higher concentrations (SA-10 and SA-15), the expression of Myosin and Vinculin declined, indicating that excessive amounts of SA might inhibit the differentiation process after reaching its peak efficacy at SA-5.

Lactic Acid (LA): In contrast, LA treatment did not produce significant changes in Myosin or Vinculin expression at any concentration. The levels of these markers remained comparable to the control group, suggesting that LA has no substantial impact on myogenesis or muscle fiber differentiation.

These findings demonstrate that succinic acid plays a significant role in promoting myogenesis, the process by which myoblasts differentiate into myotubes, contributing to muscle fiber growth and repair. The highest expression of Myosin and Vinculin was observed at SA-5, indicating that a moderate concentration of SA is most effective in supporting the differentiation of myoblasts into myotubes and facilitating muscle regeneration.

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Interestingly, higher concentrations of SA (SA-10 and SA-15) resulted in reduced expression of differentiation markers, suggesting a dose-dependent biphasic effect. While moderate levels of SA promote muscle cell differentiation, excessive concentrations may exert inhibitory effects, potentially due to metabolic stress or feedback inhibition mechanisms. This underscores the importance of maintaining an optimal concentration of SA for efficient muscle fiber development.

In contrast, LA did not influence myogenesis under the conditions tested, indicating it is not a significant modulator of muscle cell differentiation or growth. The results highlight succinic acid as a potent enhancer of myogenesis at moderate concentrations, supporting its role in muscle fiber formation and regeneration. Conversely, lactic acid does not appear to contribute to muscle cell differentiation in this model. These findings suggest a potential application of succinic acid in therapies aimed at improving muscle regeneration and repair, provided that its concentration is carefully regulated.

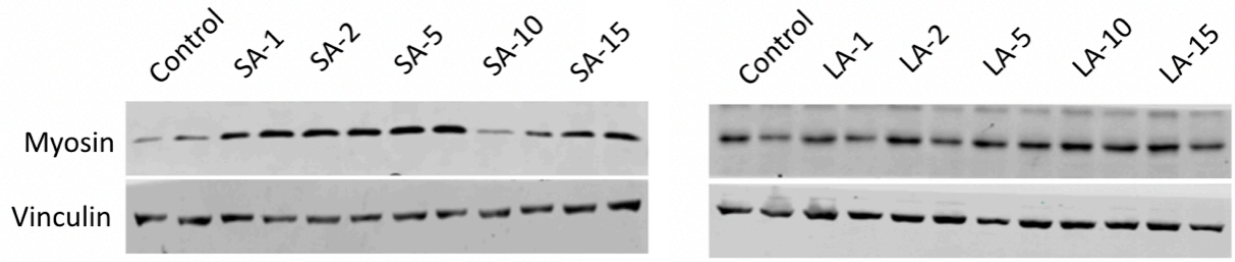

Figure 2. The effect of succinate and lactate treatment on myogenesis.

The Effect of Metabolites on Myofiber Degradation

This study investigated the effects of succinic acid (SA) and lactic acid (LA) on myofiber degradation, as indicated by the expression levels of Myostatin, a key regulator of muscle breakdown.

Succinic Acid (SA): The data suggest that SA promotes myofiber degradation in a concentration-dependent manner. In the Control group, Myostatin expression was minimal, reflecting normal myofiber formation with negligible degradation. At low concentrations (SA-1 and SA-2), there was a slight increase in Myostatin levels compared to the control, indicating the early onset of myofiber degradation. At SA-5, Myostatin expression reached a noticeable peak, suggesting significant promotion of muscle fiber breakdown. This effect became even more pronounced at higher concentrations (SA-10 and SA-15), where Myostatin levels were at their highest. The consistent increase in Myostatin expression with rising SA concentrations suggests a dose-dependent enhancement of myofiber degradation, potentially interfering with muscle regeneration and growth.

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Lactic Acid (LA): In contrast, LA treatment showed no significant effect on Myostatin levels. Across all tested concentrations (LA-1, LA-2, LA-5, and LA-10), Myostatin expression remained similar to the control group, indicating that LA does not influence myofiber degradation under the experimental conditions.

These results demonstrate that succinic acid promotes myofiber degradation in a concentration-dependent manner, with high concentrations (SA-10 and SA-15) strongly enhancing Myostatin expression. While low levels of SA (e.g., SA-1 and SA-2) may not substantially hinder muscle regrowth, higher concentrations appear to exacerbate muscle fiber breakdown, potentially impairing muscle repair and growth. This suggests that excessive exposure to SA could be detrimental to muscle health, particularly in contexts requiring robust muscle regeneration.

Conversely, the lack of significant changes in Myostatin expression with lactic acid treatment implies that LA does not contribute to myofiber degradation. This suggests that LA is less likely to interfere with muscle repair and regeneration processes compared to SA. The findings highlight a stark contrast between succinic acid and lactic acid in their effects on myofiber degradation. While SA promotes the breakdown of muscle fibers at higher concentrations, potentially limiting muscle regeneration, LA does not appear to impact this process. These results suggest that succinic acid must be carefully regulated in contexts involving muscle repair or growth to avoid adverse effects, while lactic acid may be neutral in such scenarios.

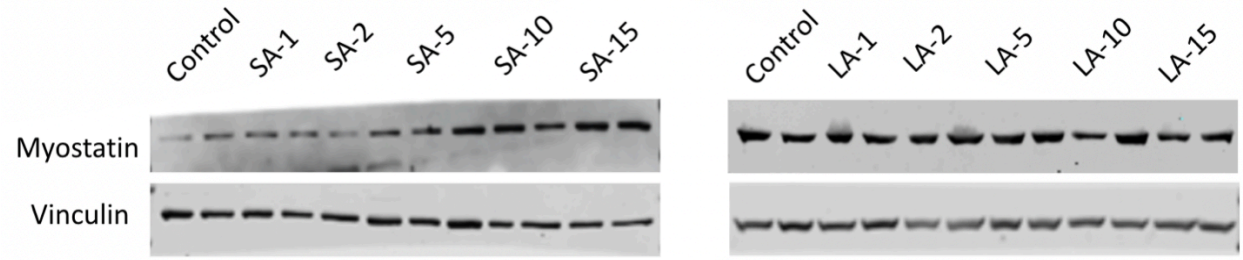

Figure 3. The effect of succinate and lactate on myofiber degradation.

DISCUSSION

Our findings reveal distinct roles for succinic acid (SA) and lactic acid (LA) in regulating myofiber dynamics and insulin sensitivity, with significant implications for muscle health and potential applications in nutrition and daily life.

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Succinic acid displayed dual roles in muscle physiology. On one hand, moderate concentrations of SA enhance insulin sensitivity, as demonstrated by increased phosphorylation of key signaling proteins such as AKT and ERK, which are critical mediators of glucose uptake and metabolic regulation. On the other hand, higher concentrations of SA promoted myofiber degradation by upregulating Myostatin expression, a known inhibitor of muscle growth and repair. This suggests that while SA may support metabolic health at certain levels, excessive accumulation could compromise muscle integrity and function.

In contrast, lactic acid had a more modest effect on insulin sensitivity, with enhancements primarily observed at higher concentrations. Notably, LA did not significantly influence Myostatin levels, suggesting it does not contribute to myofiber degradation. This indicates that LA may have a protective or neutral role in maintaining muscle integrity, even under conditions of metabolic fluctuation. Together, these results highlight the nuanced and context-dependent roles of these metabolites in muscle health.

Succinate and lactate are intermediates in cellular metabolism, and their levels can be influenced by dietary intake and metabolic state. Foods rich in succinate precursors, such as certain fermented products and citric acid cycle intermediates, may increase succinate levels in the body. Similarly, lactate levels can be elevated through the consumption of carbohydrate-rich meals, especially under conditions of intense physical activity or anaerobic metabolism.

These findings underscore the importance of dietary patterns in modulating muscle health. A balanced diet that avoids excessive accumulation of succinate or lactate precursors may help optimize muscle function. For example, moderation in the intake of highly processed foods that may elevate succinate levels could help prevent potential myofiber degradation. Conversely, incorporating nutrient-dense carbohydrates to support lactate production during exercise may enhance metabolic function without compromising muscle integrity.

Muscle health is a critical determinant of overall well-being, particularly as it relates to mobility, metabolic regulation, and resistance to age-related decline. These findings suggest that targeted nutritional interventions could play a pivotal role in maintaining muscle integrity and function. For instance, moderate consumption of succinate-rich foods could be explored as a strategy to enhance insulin sensitivity, potentially benefiting individuals with metabolic disorders such as diabetes. However, caution is warranted regarding excessive succinate accumulation, which may impair muscle repair and growth.

Lactic acid, on the other hand, presents a safer profile in terms of muscle health, as it does not promote myofiber degradation. Encouraging activities or diets that favor lactate production, such as aerobic and anaerobic exercise combined with appropriate nutritional support, could help sustain muscle function and metabolic health.

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Muscle health is essential for maintaining physical independence, preventing chronic diseases, and improving quality of life. Nutritional interventions that balance the levels of metabolites such as succinate and lactate could serve as practical strategies to promote muscle health. This is particularly important as sedentary lifestyles and poor dietary habits become increasingly prevalent. Tailoring dietary recommendations to support optimal metabolite levels while mitigating potential adverse effects can provide a foundation for enhancing muscle function and preventing age-related muscle degeneration.

By elucidating the distinct roles of succinic acid and lactic acid in muscle dynamics and insulin sensitivity, this study highlights the intricate interplay between metabolism, diet, and muscle health. These findings not only enhance our understanding of muscle physiology but also emphasize the potential for nutritional interventions to support muscle integrity and metabolic function in daily life. Further research into the dietary regulation of these metabolites and their broader impacts on muscle and metabolic health will help pave the way for evidence-based nutritional strategies aimed at improving human health.

REFERENCES

Chomiuk, T., Kucharz, E. J., Zareba, L., Kłosiewicz-Latoszek, L., & Wysokiński, A. (2024). Physical activity in metabolic syndrome. *Frontiers in Physiology, 15*, Article 1365761. https://doi.org/10.3389/fphys.2024.1365761

Cleveland Clinic. (2023, June 13). *Lactic acidosis: Symptoms, causes, treatment & what it is.* https://my.clevelandclinic.org/health/diseases/25066-lactic-acidosis

Cleveland Clinic. (2024, November 21). *Insulin resistance: What it is, causes, symptoms & treatment.* https://my.clevelandclinic.org/health/diseases/22206-insulin-resistance

Fernández-Veledo, S., Vendrell, J., & Chacón, M. R. (2024). Type 2 diabetes and succinate: Unmasking an age-old molecule. *Diabetologia, 67*(3), 430–442. https://doi.org/10.1007/s00125-023-06063-7

Hwang, S. Y., Kim, Y. S., Kim, Y. H., Lee, J. S., & Lee, J. H. (2015). Folic acid promotes the myogenic differentiation of C2C12 murine myoblasts through the Akt signaling pathway. *International Journal of Molecular Medicine, 36*(4), 1073–1080. https://doi.org/10.3892/ijmm.2015.2311

Kim, G., & Kim, J. H. (2020). Impact of skeletal muscle mass on metabolic health. *Endocrinology and Metabolism (Seoul, Korea), 35*(1), 1–6. https://doi.org/10.3803/EnM.2020.35.1.1

Lattibeaudiere, K. G., & Alexander-Lindo, R. L. (2024). Oleic acid and succinic acid: A potent nutritional supplement in improving hepatic glycaemic control in type 2 diabetic Sprague-Dawley rats. *Advances in Pharmacological and Pharmaceutical Sciences, 2024*, Article 5556722. https://doi.org/10.1155/2024/5556722

SCHOLARLY REVIEW JOURNAL

E-ISSN: 2996-8380
Published by Leadership & Innovation Lab

WINTER 2025/2026

Lee, T.-Y. (2021). Lactate: A multifunctional signaling molecule. *Yeungnam University Journal of Medicine, 38*(3), 183–193. https://doi.org/10.12701/yujm.2020.00892

Zhang, J., Xu, L., Yang, Y., & Wang, Z. (2019). EPA and DHA inhibit myogenesis and downregulate the expression of muscle-related genes in C2C12 myoblasts. *Genes, 10*(1), Article 64. https://doi.org/10.3390/genes10010064

Zhao, X., Xu, C., Zhang, W., & Li, X. (2023). The crucial role and mechanism of insulin resistance in metabolic disease. *Frontiers in Endocrinology, 14*, Article 1149239. https://doi.org/10.3389/fendo.2023.1149239