doi.org/10.54141/psbd.1678172

PAMUKKALE JOURNAL OF SPORT SCIENCES
ISSN: 1309-0356 dergipark.org.tr/en/pub/psbd

Pamukkale J Sport Sci, 16(2), 387-397, 2025 Research Article

Metabolic Flexibility Following High-Intensity Interval Exercise in Active Females: Effect of Feeding State

Maryam EBRAHIMI[1*]

[1]*Department of Exercise Physiology, Faculty of Physical Education and Sport Sciences, University of Guilan, Rasht, Iran*

Keywords
CPT-1,
Glucose,
HIIE,
Lactate,
PDK-4.

Article History
Received 17 April 2025
Revised 18 June 2025
Accepted 29 July 2025
Available Online 30 August 2025

[*] **Corresponding Author**:
Maryam Ebrahimi
E-mail Address :
maryam.ebrahimi@guilan.ac.ir

ABSTRACT

Metabolic flexibility is tightly regulated during exercise through the action of metabolites and metabolic enzymes. This randomized parallel-group study aimed to investigate how fed vs fasted state high-intensity interval exercise (HIIE) changes Glucose, lactate, pyruvate dehydrogenase kinase-4 (PDK-4), and carnitine palmitoyltransferase-1 (CPT-1) levels. 23 active females (20.09 ± 1.04 years of age) were selected from eligible volunteers and were randomly assigned into fed and 14-h fast groups. In each group, 6 participants randomly performed 1:4 HIIE (1 min all-out run, 4 min walk, for 20 minutes), and the other participants were seated. After a 5-minute cooldown, blood samples were collected from the brachial vein. Higher lactate levels were similar after HIIE in both feeding states. Fed-HIIE participants showed lower glucose levels, and fast-HIIE participants showed lower PDK-4 and CPT-1 levels than their control counterparts. It seems in the fasted 1:4 HIIE, glucose oxidation is the dominant energy production pathway. Lactate produced during exercise may be used as the precursor of gluconeogenesis in fasting.

INTRODUCTION

Metabolic flexibility is the ability of skeletal muscle to adjust its utilization of substrate pathways to meet metabolic demand and is tightly regulated during nutritional status and exercise (Lovell et al., 2025). In the fed state, glycolysis is the preferred pathway for energy

Citation: Ebrahimi, M. (2025). Metabolic Flexibility Following High-Intensity Interval Exercise in Active Females: Effect of Feeding State. *Pamukkale Journal of Sport Sciences*, 16(2), 387-397. doi.org/10.54141/psbd.1678172

needs. Glucose-induced rise in pyruvate levels exerts an inhibitory effect on pyruvate dehydrogenase kinase (PDK), causing activation of the pyruvate dehydrogenase complex (PDC) and production of acetyl coenzyme A (CoA) that enters the citric acid cycle to undergo oxidative phosphorylation for the generation of ATP (Palmer & Clegg, 2022). Pyruvate dehydrogenase kinase 4 (PDK-4), which is specific to skeletal muscle, decreases PDC activity via phosphorylation and is controlled by nutritional factors and hormones, making its role of great interest in starvation (Jeoung & Harris, 2010). Inactivation of the PDC in a fast state indirectly conserves body protein because it minimizes the need for gluconeogenesis from gluconeogenic amino acids and prevents the complete oxidation of the carbon skeletons of gluconeogenic amino acids. Indeed, survival during starvation depends upon the inactivation of PDC (Jeon et al., 2021).

Increased production of citrate with subsequent export into the cytoplasm leads to increased levels of malonyl-CoA. Malonyl CoA exerts an inhibitory effect on fatty acid β-oxidation by suppressing Carnitine Palmitoyltransferase 1 (CPT-1) and is used for fatty acid synthesis (Palmer & Clegg, 2022). CPT-1 is the rate-limiting step of the mitochondrial beta-oxidation that performs the mitochondrial uptake of long-chain Acyl CoA (Kinase, 2020). Increased fat oxidation following fasting is presumably partly due to the low circulating levels of insulin and high levels of Fatty acids (FAs), which activate fat oxidation and inactivate pyruvate dehydrogenase-a (PDHa), thereby decreasing carbohydrate oxidation (Andersson Hall et al., 2016).

Exercise has proven to be the best physiological stimulus for improving mitochondrial function and provides an ideal challenge to the metabolic environment of skeletal muscle, where fat and carbohydrate (CHO) use within the mitochondria change depending on the stress placed on the body (Lovell et al., 2025). The most common method of assessing the metabolic response to exercise is through indirect calorimetry (Amaro-Gahete et al., 2019). However, tracking metabolic enzymes may help to understand fuel switching in the context of metabolic flexibility. Evidence from acute exercise studies indicates that fasted endurance exercise favorably regulates muscle gene expression of PDK4 and CPT-1 (Aird et al., 2021). This study hypothesized that high-intensity interval exercise (HIIE), as 4 × 30 s all-out cycle sprints against a resistance equivalent to 7.5% BM, interspersed with 4-min active recovery periods in a fast state, could change carbohydrate/fat metabolism and may enhance fat oxidation. One of the proposed mechanisms for the beneficial effects of HIIE on fat oxidation is the regulation of fatty acid movements across the mitochondrial membranes via CPT-1

(Astorino & Schubert, 2018). Therefore, this study aimed to investigate the effects of HIIE in fed and fasted states on these metabolic enzymes. Glucose and lactate levels were also measured to see whether exercise while fasting may alter glucose levels and lactate production.

METHODS

Participants

Upon an announcement, twenty-seven females volunteered to participate in the research. Based on inclusion criteria (18–25-year healthy females, 19-24.9 kg/m2 body mass index, regular concurrent training- three days/week for 6 months at least, no history of metabolic disorders or Anemia, no current musculoskeletal injuries, normal PAR-Q questionnaire, no use of drugs or supplements), twenty-three volunteers were considered eligible. Four volunteers were excluded because of supplement intake (n=2), abnormal PAR-Q (n=1), and out-of-range body mass index (n=1). After explaining the study aims and protocol, all volunteers signed a written informed consent form and were randomly assigned to four groups: control-fast (n=5), control-fed (n=6), HIIE-fast (n=6), and HIIE-fed (n=6) (Table 1).

The research follows the Declaration of Helsinki (1964) and has received approval from the University of Guilan Committee for Ethics in Biomedical Research (IR.GUMS.REC.1398.002) and is registered in the Iranian Registry of Clinical Trials (IRCT20190213042702N1). All participants were fully informed about the research procedure and signed an informed consent form.

Procedures

This randomized trial was designed in four parallel groups. In the first session, the subjects' weight and height were measured using a SECA scale. Body density was calculated from the three-site skinfold measurement (Jackson et al., 1978) using a Lafayette caliper and was used to estimate fat percentage by the Siri equation (Siri, 1956). Basal metabolic rate (BMR) was estimated using the Mifflin equation (Mifflin et al., 1990). Calorie intake was estimated from a 24-hour food recall the day before the trial.

All participants were asked to avoid consuming caffeine 24 hours before the trial and get 8 hours of sleep. The last meal on the night before the trial was similar (spaghetti with meat sauce; containing ~284 kcal: 69% Carbohydrates, 20% protein, and 11% fat), and from

midnight, subjects were just allowed to drink water. On the day of the trial, subjects arrived at the indoor gymnasium at 7:00 am (~55% humidity and ~25-27°C temperature). Participants of the fed groups consumed a similar breakfast (1 boiled egg, 2 slices of whole grain bread, 2 dates, and 200 mL orange juice; containing ~332 kcal: 74% carbohydrate, 18% protein, and 8% fat), and the other subjects remained fasted.

One hour after breakfast, while control groups were seated, participants in exercise groups warmed up for 15 minutes (jogging and static/dynamic stretches) and performed 20 minutes of 1:4 HIIE (1 min run with maximum speed, 4 min walk) (Mahdi Gholizadeh, 2017). Participants were motivated verbally to exert their maximal effort through work intervals. After 5 minutes of active recovery (walking), blood samples were collected from the brachial vein, and serum/plasma samples were transferred to the laboratory for biochemical analysis. Serum CPT-1 (ELISA; ZellBio kit, 0.05 ng/ml sensitivity), serum PDK-4 (ELISA; ZellBio kit, 0.9 ng/ml sensitivity), glucose (Glucose Oxidase; Bionik kit), and lactate (enzymatic calorimetry; Birex fars kit) were measured in a biochemistry laboratory.

Data Analysis

Values are expressed as mean ± standard deviation. The Shapiro-Wilk test was used for the data distribution normality test, and in case of abnormality, data were normalized with the fractional rank/Inverse df method. Two-way ANOVA for non-repeated measures was used to test the hypotheses, and in case of a significant F value, Effect size (η_P^2) and observed power (OP) are reported. Bonferroni pairwise comparisons were applied if the interaction effect was observed to be significant. All data were analyzed by IBM SPSS version 26 software (Chicago, IL, USA) ($P < 0.05$).

RESULTS

This study was designed to investigate the effect of a 20-minute session of HIIE in fed or fasted states on PDK-4, CPT-1, Glucose, and lactate levels in active females.

Glucose and lactate levels

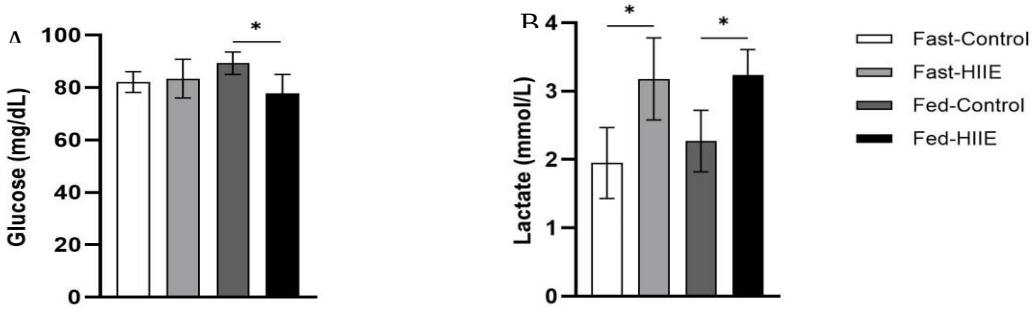
Although the main effect of exercise ($P = 0.068$) and feeding ($P = 0.746$) was not significant, there was a significant interaction effect ($F (1,18) = 6.04$, $P = 0.024$, $\eta_P^2 = 0.251$, $OP = 0.64$). Bonferroni pairwise comparison showed significantly lower glucose levels in the fed-HIIE group compared to fed-controls ($P = 0.006$, $\eta P2 = 0.350$, $OP = 0.837$) (Figure 1, A).

For plasma lactate levels, the effect of nutritional state (F (1,18) = 0.781, P = 0.389) and the interaction effect of feeding state* exercise (F (1,18) = 0.390, P = 0.540) was not statistically different. Meanwhile, the impact of exercise was significantly different (F (1,18) = 26.597, P < 0.001, η_P^2 = 0.596, OP = 0.998), and HIIE increased lactate levels in both nutritional states (Figure 1, B).

Table 1.
Subject's characteristics

Feeding state	**Fast**		**Fed**	
Groups	**Control (n=5)**	**HIIE (n=6)**	**Control (n=6)**	**HIIE (n=6)**
Age (years)	19.60 ± 0.55	20.33 ± 1.37	20.17 ± 0.98	20.17 ± 1.17
Weight (kg)	62.42 ± 7.40	61.25 ± 6.45	58.35 ± 7.22	62.58 ± 2.34
Height (cm)	168.56 ± 5.46	163.30 ± 5.07	164.03 ± 6.11	164.10 ± 4.31
BMI (kg/m^2)	21.90 ± 1.50	22.92 ± 1.38	21.65 ± 1.81	23.25 ± 0.70
Fat (%)	31.62 ± 3.99	30.93 ± 3.57	34.45 ± 2.25	34.90 ± 3.62
BMR (kcal)	1418.70 ± 107.20	1370.46 ± 88.27	1346.88 ± 40.62	1389.63 ± 49.29
Daily calorie intake (kcal)	1449.00 ± 118.84	1324.00 ± 138.84	1323.17 ± 253.80	1444.67 ± 191.44

Figure 1.
Mean ± SD of (A) Glucose and (B) lactate levels in fast-control, fast-HIIE, fed-control, and fed-HIIE groups after an HIIE session. *: Statistically difference at P < 0.05 between control and HIIE groups in same feeding state

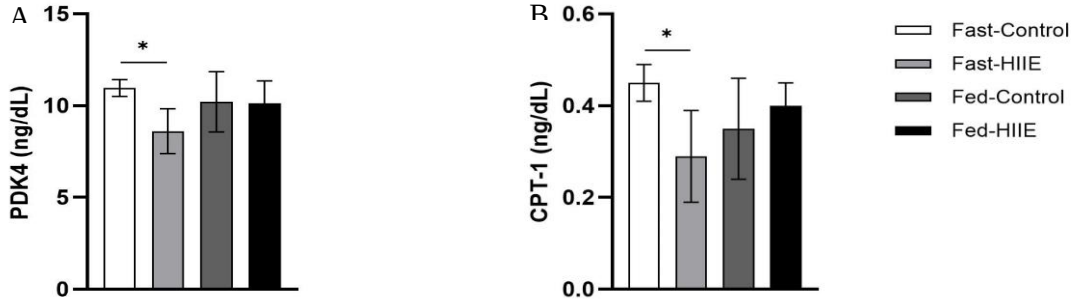


Serum PDK-4 and CPT-1

The main effect of the feeding state for serum PDK-4 was insignificant (F (1,18) = 0.527, P = 0.477). Meanwhile, the main effect of exercise (F (1,18) = 5.138, P = 0.036, η_P^2 = 0.222, OP = 0.573) and the interaction effect of feeding state*exercise (F (1,18) = 4.572, P = 0.046, η_P^2 = 0.203, OP = 0.525) were statistically significant. Pairwise comparisons with the Bonferroni test revealed that mean PDK-4 in the fasted state was significantly lower in the HIIE group compared with the control group (P = 0.008, η_P^2 = 0.331, OP = 0.835) (Figure 2, A).

With CPT-1, the main effects of the feeding state (F (1,19) = 0.000, P = 1.000) and exercise (F (1,19) = 2.507, P = 0.130) were not significant. However, the interaction effect showed a significant F value (F (1,19) = 9.719, P = 0.006, η_P^2 = 0.338, OP = 0.841). Further pairwise comparisons showed that in the fasted but not fed state, serum CPT-1 levels were significantly lower in the HIIE compared with the control group (P = 0.004, η_P^2 = 0.357, OP = 0.869).

Figure 2.
Mean ± SD of (A) PDK-4 and (B) CPT-1 levels in fast-control, fast-HIIE, fed-control, and fed-HIIE groups after an HIIE session. *: Statistically difference at P < 0.05 between control and HIIE groups in same feeding state. Acquired from 2-way



DISCUSSION

This randomized parallel-group study hypothesized that HIIE in fed and fasted states has different effects on glucose, lactate, PDK-4, and CPT-1 levels in active females. Findings reveal that metabolic responses to HIIE differ depending on nutritional status.

Fed-HIIE participants showed the lowest post-exercise glucose levels (76.17 ± 10.32 mg/dL), significantly lower than those of the fed-control group (89.33 ± 4.13 mg/dL). Insulin plays a primary role in the regulation of glucose homeostasis. In the postprandial state, higher levels of insulin suppress the breakdown of triglycerides and inhibit FA metabolism (Kazeminasab et al., 2025). Also, the liver stores glucose as glycogen, which is more dependent on glucose transporter 2 (GLUT2) rather than insulin levels (Dimitriadis et al., 2021). During Exercise, insulin levels typically decrease, as the body is more focused on immediate energy needs under GLUT4 action. Also, exercise enhances the body's sensitivity to insulin. This means that even small amounts of insulin can be more effective in lowering blood glucose levels after a meal (Dimitriadis et al., 2021). This suggests that acute HIIE in the postprandial state may enhance glucose uptake into skeletal muscles, likely mediated by insulin-independent GLUT4 translocation. While insulin levels typically decline during exercise, prior feeding enhances insulin sensitivity, thereby facilitating glucose clearance even with reduced

circulating insulin levels. These findings support the notion that exercise in the fed state amplifies insulin action and promotes efficient glucose utilization.

Glucose can be converted to lactate in the muscles. Most of the lactate (75–80%) is disposed of from the tissue or released and taken up by the working muscle, heart, brain, and liver for gluconeogenesis (Bartoloni et al., 2024). In the current study, plasma lactate levels were higher in both fed (~51%) and fasted (~61%) groups compared with their control counterparts. This indicates that lactate production during HIIE is driven primarily by exercise intensity rather than feeding status. Given the reliance on anaerobic glycolysis during maximal effort intervals, this lactate accumulation is expected. Moreover, lactate serves as a substrate for oxidative tissues and hepatic gluconeogenesis via the Cori cycle, especially in the fasted state. It has been estimated that in the fasted state, approximately 65% of plasma lactate is derived from glucose, while 16–20% of plasma lactate stems from alanine. To a lesser extent, pyruvate may be generated from the catabolism of other amino acids, including serine, threonine, and cysteine (Perriello et al., 1995).

The oxidation of lactate into pyruvate by lactate dehydrogenase (LDH) is the first step to the metabolic removal of lactate. The PDH complex transforms pyruvate into acetyl-coenzyme A (CoA), allowing the entry of acetate into the tricarboxylic acid (TCA) cycle (Adeva-Andany et al., 2014). Phosphorylation of PDC by the PDK inhibits its activity. It has been reported that the expression of the PDK-4 gene is increased in fasting and other conditions associated with the switch from the utilization of glucose to fatty acids as an energy source (Connaughton et al., 2010). The PDK-4 enzyme, known to suppress glucose oxidation, was highest in the fasted-control group (10.36 ± 0.25 ng/dL). However, fasted participants who performed HIIE showed lower PDK-4 levels (~8% decrease), suggesting reduced PDC inhibition and enhanced glucose oxidation. This finding contradicts the common assumption that fasted exercise universally increases fat oxidation; instead, it implies a shift toward carbohydrate utilization to meet the high energy demands of intense interval training.

Interestingly, fasted-control participants showed the highest CPT-1 levels (0.45 ± 0.04 ng/dL), significantly higher (~36%) than their HIIE counterparts. As the product of glycolysis, lactate could provide negative feedback inhibition of glucose disposal. Also, it gives rise to acetyl-CoA and, in turn, malonyl-CoA by the action of acetyl-CoA carboxylase (ACC). Malonyl CoA is a strong inhibitor of CPT-1 (Jeppesen & Kiens, 2012). By inhibiting muscle mitochondrial fatty acid uptake, via malonyl CoA and CPT-1, lactate may control lipid oxidation and overall energy substrate partitioning (Brooks, 2020). The downregulation of

CPT-1 post-exercise may reflect reduced lipid oxidation. One plausible mechanism involves lactate-mediated inhibition of CPT-1 through increased malonyl-CoA concentrations, which suppress fatty acid entry into mitochondria. It is suggested that decreased muscle pH, due to the accumulation of glycolytic by-products, inhibits the transport of long-chain fatty acid into the mitochondria, reducing fatty acid oxidation (Filipovic et al., 2021). These observations suggest that HIIE, even in the fasted state, may transiently suppress fat metabolism in favor of carbohydrate oxidation.

Although fat oxidation is generally expected to increase during fasted exercise, the high-intensity nature of HIIE appears to prioritize rapid ATP production through glycolysis and subsequent glucose oxidation. Lactate produced during high-intensity exercise may also serve as a gluconeogenic precursor, potentially explaining the stable glucose levels observed in fasted participants despite the absence of dietary carbohydrate intake. Moreover, sex hormones may affect fuel metabolism during exercise. For example, estrogen has been shown to reduce lipolysis and plasma catecholamine concentration by either inhibiting secretion or accelerating degradation, and women show higher lipid metabolism and lower carbohydrate utilization after 14-22 hours of fasting, compared with men (Sanchez et al., 2024).

Limitations

This study had limitations, including the lack of baseline (pre-test) sampling. Although control groups were presented in the study, considering individual responses to fasting and HIIE may help in the better interpretation of results. Moreover, phases of the menstrual cycle were not controlled in the present study. Performing tests in the same phase may explain the effect of sex hormones on metabolic flexibility and observed results. Future research should incorporate time-course sampling, measure free fatty acid concentrations, and assess enzyme activity levels with a larger sample size. The use of indirect calorimetry would also help clarify substrate utilization and metabolic flexibility during fasted HIIE.

CONCLUSION

This study demonstrates that acute HIIE in the fasted state favors glucose oxidation over fat oxidation. Despite expectations for increased lipid metabolism during fasted exercise, reduced levels of CPT-1 and PDK-4 in fasted-HIIE participants suggest a metabolic shift toward carbohydrate use, likely due to the high energy demands of HIIE. Lactate production was substantial in both fasted and fed states and may contribute to gluconeogenesis in the

fasted state. These findings highlight the complexity of metabolic regulation during high-intensity exercise and underscore the importance of considering exercise intensity when interpreting substrate utilization in different nutritional states.

PRACTICAL IMPLICATIONS

Coaches and athletes need to consider the dominant fuel of exercise based on nutritional status and the intensity of exercise sessions to induce favorable adaptive metabolic flexibility. HIIE in a fasted state may not provide the expected fat oxidation as assumed and could be a challenging exercise mode for females.

Acknowledgments

This research has received financial support from the University of Guilan under grant number 15p204464.

Authors' Contribution

Not relevant.

Declaration of Conflict of Interest

The authors declare that they have no conflict of interest.

Ethics Statement

This randomized trial was designed in four parallel groups. The research follows the Declaration of Helsinki (1964) and has received approval from the Iran National Committee for Ethics in Biomedical Research (IR.GUMS.REC.1398.002) and is registered in the Iran Registry of Clinical Trials (IRCT20190213042702N1). All participants were fully informed about the research procedure and signed an informed consent form.

REFERENCES

Adeva-Andany, M., López-Ojén, M., Funcasta-Calderón, R., Ameneiros-Rodríguez, E., Donapetry-García, C., Vila-Altesor, M., & Rodríguez-Seijas, J. (2014). Comprehensive review on lactate metabolism in human health. *Mitochondrion*, *17*, 76-100.

Aird, T. P., Farquharson, A. J., Bermingham, K. M., O'Sulllivan, A., Drew, J. E., & Carson, B. P. (2021). Divergent serum metabolomic, skeletal muscle signaling, transcriptomic, and performance adaptations to fasted versus whey protein-fed sprint interval training. *American Journal of Physiology-Endocrinology and Metabolism*, *321*(6), E802-E820.

Amaro-Gahete, F. J., Sanchez-Delgado, G., Jurado-Fasoli, L., De-la-O, A., Castillo, M. J., Helge, J. W., & Ruiz, J. R. (2019). Assessment of maximal fat oxidation during exercise: A systematic review. *Scand J Med Sci Sports*, *29*(7), 910-921.

Andersson Hall, U., Edin, F., Pedersen, A., & Madsen, K. (2016). Whole-body fat oxidation increases more by prior exercise than overnight fasting in elite endurance athletes. *Applied Physiology, Nutrition, and Metabolism*, *41*(4), 430-437.

Astorino, T. A., & Schubert, M. M. (2018). Changes in fat oxidation in response to various regimes of high intensity interval training (HIIT). *European Journal of Applied Physiology*, *118*(1), 51-63.

Bartoloni, B., Mannelli, M., Gamberi, T., & Fiaschi, T. (2024). The Multiple Roles of Lactate in the Skeletal Muscle. *Cells*, *13*(14), 1177.

Brooks, G. A. (2020). Lactate as a fulcrum of metabolism. *Redox biology*, *35*, 101454.

Connaughton, S., Chowdhury, F., Attia, R. R., Song, S., Zhang, Y., Elam, M. B.,…Park, E. A. (2010). Regulation of pyruvate dehydrogenase kinase isoform 4 (PDK4) gene expression by glucocorticoids and insulin. *Molecular and cellular endocrinology*, *315*(1-2), 159-167.

Dimitriadis, G. D., Maratou, E., Kountouri, A., Board, M., & Lambadiari, V. (2021). Regulation of postabsorptive and postprandial glucose metabolism by insulin-dependent and insulin-independent mechanisms: an integrative approach. *Nutrients*, *13*(1), 159.

Filipovic, M., Munten, S., Herzig, K.-H., & Gagnon, D. D. (2021). Maximal fat oxidation: Comparison between treadmill, elliptical and rowing exercises. *J Sports Sci Med*, *20*(1), 170.

Jackson, A. S., Pollock, M. L., & Gettman, L. R. (1978). Intertester reliability of selected skinfold and circumference measurements and percent fat estimates. *Research Quarterly. American Alliance for Health, Physical Education and Recreation*, *49*(4), 546-551.

Jeon, J. H., Thoudam, T., Choi, E. J., Kim, M. J., Harris, R. A., & Lee, I. K. (2021). Loss of metabolic flexibility as a result of overexpression of pyruvate dehydrogenase kinases in muscle, liver and the immune system: Therapeutic targets in metabolic diseases. *Journal of Diabetes Investigation*, *12*(1), 21-31.

Jeoung, N. H., & Harris, R. A. (2010). Role of pyruvate dehydrogenase kinase 4 in regulation of blood glucose levels. *Korean diabetes journal*, *34*(5), 274-283.

Jeppesen, J., & Kiens, B. (2012). Regulation and limitations to fatty acid oxidation during exercise. *The Journal of physiology*, *590*(5), 1059-1068.

Kazeminasab, F., Rafiee, P., Miraghajani, M., Santos, H. O., Symonds, M. E., & Rosenkranz, S. K. (2025). The effects of acute bouts of exercise in fasted vs. fed states on glucose and lipid metabolism in healthy adults: a systematic review and meta-analysis of randomized clinical trials. *Clin Nutr ESPEN*.

Kinase, P. D. (2020). The Effects of Pyruvate Dehydrogenase Kinase 4 (PDK4) Inhibition on Metabolic Flexibility during Endurance Training in Skeletal Muscles of Streptozotocin-induced Diabetic Rats. *Journal of Kerman University of Medical Sciences*, *27*(4), 304-317.

Lovell, D. I., Stuelcken, M., & Eagles, A. (2025). Exercise Testing for Metabolic Flexibility: Time for Protocol Standardization. *Sports Medicine-Open*, *11*(1), 31.

Mahdi Gholizadeh, M. K., Ali Akbarnejad, AliReza Ghardashi Afousi, Sajjad Mohammadyari. (2017). The Effect of Two Types of High Intensity Interval Training (HIIT) on Oxidation of Fat and Carbohydrate in Young Overweight Men. *journal os sport biosciences*, *9*(2), 157-169.

Mifflin, M. D., St Jeor, S. T., Hill, L. A., Scott, B. J., Daugherty, S. A., & Koh, Y. O. (1990). A new predictive equation for resting energy expenditure in healthy individuals. *The American Journal of Clinical Nutrition*, *51*(2), 241-247.

Palmer, B. F., & Clegg, D. J. (2022). Metabolic flexibility and its impact on health outcomes. Mayo Clinic Proceedings,

Perriello, G., Jorde, R., Nurjhan, N., Stumvoll, M., Dailey, G., Jenssen, T.,…Gerich, J. (1995). Estimation of glucose-alanine-lactate-glutamine cycles in postabsorptive humans: role of skeletal muscle. *American Journal of Physiology-Endocrinology and Metabolism*, *269*(3), E443-E450.

Sanchez, B. N., Volek, J. S., Kraemer, W. J., Saenz, C., & Maresh, C. M. (2024). Sex differences in Energy Metabolism: a female-oriented discussion. *Sports Medicine*, *54*(8), 2033-2057.

Siri, W. E. (1956). Body composition from fluid spaces and density: analysis of methods.