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The role of lipid metabolism in track and field athletes: A comparative analysis of sprinters and long-distance runners

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Abstract
Lipid metabolism plays an important role in determining athletic performance, particularly in relation to different energy system demands of track and field athletes. The comparative aspect of lipid metabolism among sprinters and long-distance runners, two cohorts of athletes with distinct physiological profiles and metabolic demands, is the subject of this narrative review. Sprinters utilize anaerobic energy systems predominantly, with minimal lipid oxidation during exercise, whereas long-distance runners use aerobic metabolism to a great extent, and therefore lipid utilization becomes important for long-term energy production. The review collates recent evidence concerning molecular adaptations, mitochondrial function, enzymatic activity, genetic polymorphisms, and nutritional interventions that influence lipid metabolism in these athletes. In addition, the paper discusses the effects of lipid metabolism on recovery, metabolic health, and performance optimization. Through an analysis of more recent research, this review highlights the value of particular training and diet interventions tailored to enhance lipid metabolic effectiveness based on the specific demands of sprint and endurance running.

Keywords: Lipid metabolism, track and field, sprinters, long-distance runners, fat oxidation, endurance training, anaerobic energy systems, mitochondrial adaptation, athletic performance, sports nutrition, cardiovascular health

Introduction

Athletic performance depends on both rapid energy, which comes mainly from carbohydrates, and sustained endurance, which is largely supported by lipids (Hargreaves and Spriet in 2020) [21]. In track and field, sprinting and long-distance running differ significantly in their energy demands. Sprinters rely on anaerobic pathways such as phosphagen and glycolysis for short, high-intensity efforts (Joyner & Coyle, 2007; Girard *et al*., 2011) [16, 27], while long-distance runners primarily use aerobic metabolism and depend more on fat oxidation, especially when glycogen levels drop (Achten & Jeukendrup, 2003; Van Hall *et al*., 2000) [56].

Lipid metabolism involves fat storage, mobilization, and oxidation to produce ATP, mainly through free fatty acids (FFAs) and intramuscular triglycerides (IMTGs) (Spriet, 2014) [48]. Endurance training improves lipid metabolism by increasing mitochondrial density, oxidative enzymes, and fatty acid transport, which enhances energy efficiency and delays fatigue (Holloszy & Coyle, 1984; Kiens, 2005; Brooks, 2018; Phinney *et al*., 1983) [8, 23, 30, 43].While lipid metabolism contributes little during actual sprints (Ross & Leveritt, 2001; Bangsbo *et al*., 2006) [7, 45], it supports recovery, repeated sprint ability, and adaptations from high-intensity interval training (HIIT), which improves mitochondrial function and fat oxidation (Burgomaster *et al*., 2007; Talanian *et al*., 2006) [9, 50]. Sprinters typically show fewer changes in lipid enzyme activity and muscle fat content (Nikkilä *et al*., 1978; Tamura *et al*., 2008; Nakagawa & Hattori, 2017) [35, 37, 52], yet improved metabolic flexibility benefits all athletes (Goodpaster & Sparks, 2017; San-Millán & Brooks, 2017) [18, 46]. Genetics also play a role. Variants in genes like PPARA, CPT1B, and ACSL1 influence fat oxidation and endurance capacity (Bervejillo & Ferreira, 2019; Maples *et al*., 2015; Zhang & Wang, 2023) [5, 34, 63], contributing to differences in mitochondrial function and fuel use between athletes (Ahmetov *et al*., 2016) [2].Endurance athletes usually have better lipid profiles and cardiovascular health (Durstine *et al*., 2001; Thomas *et al*., 2020) [12], but sprinters can benefit from fat metabolism improvements during recovery and off-season (Maillard *et al*., 2018; Gillen & Gibala, 2014) [15, 33]. Nutritional strategies like periodization, fasted training, and ketogenic diets aim to boost fat oxidation, though their effectiveness remains debated (Impey *et al*., 2018; Burke *et al*., 2011, 2016; Stellingwerff *et al*., 2019) [10, 25, 49].

Lipid metabolism also influences inflammation, recovery, and immune health. Prolonged endurance exercise can increase oxidative stress (Nikolaidis *et al*., 2008; Np *et al*., 2011) [36, 39], while omega-3 supplementation may reduce inflammation and support recovery (Philpott *et al*., 2019; Lewis *et al*., 2016) [42]. Despite substantial progress, several gaps remain in the literature regarding the interplay between lipid metabolism and athletic specialization. The majority of studies focus on endurance athletes, with relatively limited research examining lipid metabolic responses in sprinters, particularly in real-world, elite-level contexts. Furthermore, variations based on sex, age, training background, and hormonal milieu (e.g., estrogen effects on fat metabolism in female athletes) remain underexplored (Tarnopolsky, 2008; Boisseau & Delamarche, 2000) [6, 53].

This review compares lipid metabolism in sprinters and long-distance runners, examining metabolic adaptations, fiber-type differences, genetic influences, and performance implications. It contributes to the development of targeted training and nutritional strategies to enhance athletic performance, metabolic resilience, and long-term health outcomes.

Lipid Metabolism: Mechanisms and Relevance in Athletic Performance

Lipid metabolism involves the formation, degradation, and regulation of lipids required for energy, hormones, and nutrient transport. Lipogenesis produces fatty acids and triglycerides, whereas lipolysis hydrolyzes them to yield energy (Xiu *et al*., 2022) [62]. Lipid oxidation helps generate energy from fatty acids, especially during fasting or exercise. When carbohydrates are scarce, ketogenesis produces ketone bodies as alternative fuel (Xiu *et al*., 2022) [62]. Disruptions in these processes can lead to metabolic disorders, making their regulation crucial for health. Lipid metabolism in humans primarily involves two energy-yielding processes: lipolysis—the breakdown of triglycerides into free fatty acids (FFAs) and glycerol—and beta-oxidation, where FFAs are converted into acetyl-CoA in the mitochondria for ATP production. These processes are regulated by hormonal signals such as insulin, glucagon, catecholamines, and by the energy demands placed on skeletal muscle (van Loon *et al*., 2018) [58]. Lipid metabolism plays a pivotal role in fuelling endurance exercise, with triacylglycerols serving as a major energy source for trained athletes during prolonged physical activity (Horowitz & Klein, 2000) [22]. In endurance athletes, lipid metabolism contributes significantly to energy production, especially during submaximal exercise when glycogen sparing is crucial for sustaining performance (Spriet, 2014) [48]. Studies have demonstrated that endurance training enhances mitochondrial density, oxidative enzyme activity, and the capacity to oxidize FFAs (Holloszy & Coyle, 1984; Philp *et al*., 2012) [23, 41]. This adaptation supports prolonged activity by reducing dependence on carbohydrate stores and delaying the onset of fatigue. In contrast, in sprinting that requires brief bursts of maximal effort (usually lasting <30 seconds), energy is mostly obtained from breakdown of phosphocreatine and anaerobic glycolysis (Ross & Leveritt, 2001) [45]. Lipid metabolism is of lesser importance during sprint performance because of the need for rapid energy and the slow yield of ATP from fat oxidation (Van Hall, 2015) [57]. Nonetheless, it has been evidenced in recent studies that baseline lipid profiles and the ability to adjust between fat and carbohydrate oxidation have the potential to still affect recovery, adaptation to training, and repeated sprint performance (Impey *et al*., 2018; Bartlett *et al*., 2020) [4, 25]. Furthermore, emerging research highlights genetic differences and epigenetic modulation in lipid metabolism between sprint and endurance athletes. Variants in genes such as PPARA, CPT1B, and ACSL1 have been associated with endurance performance and fatty acid utilization efficiency (Stefanetti *et al*., 2018; Lopez-Leon *et al*., 2016) [31]. These molecular insights reinforce the metabolic divergence rooted in athletic specialization.

Comparative Analysis: Sprinters vs. Long-Distance Runners

1. Energy System Utilization

Endurance runners rely mostly on aerobic energy pathways, with lipid metabolism producing as much as 60% of overall energy expenditure during sustained exercise at moderate intensities (Romijn *et al*., 1993) [44]. Fat oxidation efficiency in these runners is supported by increased mitochondrial density, increased capillary density, and elevated expressions of fatty acid transport proteins like FAT/CD36 and CPT1 (Talanian *et al*., 2010; Holloway *et al*., 2008) [24, 51]. Conversely, sprinters depend extensively on the ATP-PCr system and glycolysis, and lipid oxidation is comparatively minor during exercise. Nevertheless, sprint training may affect lipid profiles and metabolic well-being. For example, research has demonstrated that high-intensity interval training (HIIT), a typical element of sprint training, enhances lipid oxidation ability and insulin sensitivity (Gillen & Gibala, 2014; Maillard *et al*., 2018) [15, 33]. This places the focus on the involvement of lipid metabolism not in sprinting itself, but in the larger context of the adaptation and health of athletes.

2. Muscle Fiber Type and Mitochondrial Function:

The fibre composition of sprinters and distance runners is also a testament to their different energy requirements. Sprinters show a higher proportion of Type II (fast-twitch) fibres with high glycolytic potential but reduced mitochondrial density and oxidative ability (Wilson *et al*., 2012) [61]. Endurance athletes, on the other hand, contain more Type I (slow-twitch) fibres that have a high content of mitochondria and are highly competent in burning fat (Gollnick *et al*., 1972) [17]. Mitochondrial adjustments in endurance athletes are enhanced biogenesis, greater coupling efficiency, and augmented expression of lipid oxidation enzymes like β-HAD and citrate synthase (Lundby & Jacobs, 2015) [32]. These adaptations provide fatter oxidation, particularly for sustained efforts. Sprinters, though having lesser basal mitochondrial performance, can yet gain from specificity-trained interventions toward improved mitochondrial function for recovery and metabolic adaptability (Granata *et al*., 2016) [19].

3. Lipid Profile, Inflammation, and Recovery:

There is also a growing interest in how lipid metabolism influences recovery, inflammation, and muscle damage. Long-distance runners tend to have more favorable lipid profiles, including lower triglycerides and higher HDL cholesterol, which are associated with cardiovascular health and recovery capacity (Durstine *et al*., 2001) [12]. Aerobic exercise positively influences HDL quantity and quality, enhancing cholesterol efflux, antioxidant, anti-inflammatory, and vascular functions. The benefits depend on exercise type, intensity, and individual factors, supporting its role in managing lipid profiles and cardiovascular health (Franczyk *et al*., 2023) [14].

These profiles are thought to result from sustained aerobic activity and improved fat metabolism. Sprinters, due to the anaerobic nature of their sport, may experience greater post-exercise inflammatory responses and delayed onset muscle soreness (DOMS) (Howatson & van Someren, 2008). However, training-induced improvements in lipid metabolism and anti-inflammatory pathways, particularly through omega-3 fatty acid supplementation, may help mitigate such effects (Philpott *et al*., 2019) [42]. Moreover, metabolic flexibility—the ability to switch efficiently between lipid and carbohydrate metabolism—has emerged as a critical determinant of performance and recovery in both athlete types (Goodpaster & Sparks, 2017) [18]. While endurance athletes naturally develop this capacity through training, sprinters may require targeted interventions, such as mixed-intensity sessions and dietary modulation, to enhance lipid oxidation during recovery phases.

4. Lipid Metabolism: Sprinters vs. Long-Distance Runners

1.1 Lipoprotein Lipase Activity and Serum Lipids

Long-distance runners exhibit significantly higher lipoprotein lipase (LPL) activity in both skeletal muscle and

adipose tissue compared to controls, which is associated with elevated HDL-cholesterol levels. This adaptive increase in LPL activity is not observed in sprinters, whose serum lipid and lipoprotein concentrations, as well as LPL activities, are similar to those of sedentary controls. The enhanced LPL activity in endurance athletes likely contributes to more efficient lipid metabolism and higher HDL-cholesterol, supporting rapid turnover of triglyceride-rich lipoproteins. In contrast, sprinters do not show these adaptations, reflecting the shorter duration and different metabolic demands of their training (Nikkilä *et al*., 1978) [37].

1.2 **Intramyocellular Lipid (IMCL) Content**

Endurance runners have significantly higher IMCL concentrations in muscles such as the tibialis anterior and medial gastrocnemius compared to sprinters and untrained individuals. These differences are muscle-type specific and reflect cellular adaptations to prolonged endurance training. Sprinters' IMCL levels are comparable to those of untrained subjects, indicating less reliance on intramuscular lipid stores for energy. Additionally, short-term dietary fat loading increases IMCL levels in endurance runners but not in sprinters, further highlighting the greater capacity for lipid storage and utilization in endurance-trained muscles (Tamura *et al*., 2008; Bernús *et al*., 1993; Nakagawa & Hattori, 2017) [3, 35, 52].

1.3 **Gene Expression Related to Lipid Metabolism**

Endurance runners display higher expression of genes involved in lipid metabolism, including PGC1A, adiponectin receptors, and fatty acid transporters, compared to non-athletes. These molecular adaptations are linked to increased IMCL accumulation and enhanced insulin sensitivity, supporting efficient lipid utilization during prolonged exercise. Such gene expression changes are not reported in sprinters, aligning with their lower IMCL content and different metabolic requirements (Kakehi *et al*., 2020) [28].

1.4 **Energy Substrate Utilization**

During high-intensity, short-duration exercise (e.g., 400-m sprints), sprinters rely primarily on glycolysis, as indicated by higher peak blood lactate levels. In contrast, long-distance runners maintain higher speeds in prolonged events (e.g., 3,000-m runs) through greater energy supply from lipid metabolism, as evidenced by higher blood glycerol levels. This demonstrates a fundamental difference in substrate utilization: sprinters depend more on carbohydrates, while endurance runners are adapted for sustained lipid oxidation (Ohkuwa *et al*., 1984) [40].

1.5 **Erythrocyte Membrane Adaptations**

Both sprinters and long-distance runners show increased erythrocyte membrane fluidity compared to sedentary individuals, with the effect being more pronounced in long-distance runners. This is associated with changes in membrane phospholipid composition, particularly an increase in polyunsaturated fatty acids, which may enhance microcirculatory function and oxygen delivery during prolonged exercise (Kamada *et al*., 1993) [29].

Table 1: Comparative Features of Lipid Metabolism in Sprinters and Long-Distance Runners

Feature	Sprinters	Long-Distance Runners
Energy System	Anaerobic (ATP-PCr, glycolysis) (Romijn *et al*., 1993) [44]	Aerobic (oxidative phosphorylation) (Romijn *et al*., 1993) [44]
Lipid Oxidation	Minimal during exercise (Romijn *et al*., 1993) [44]	Significant, especially during prolonged activity (Romijn *et al*., 1993; Maillard *et al*., 2018) [33, 44]
Muscle Fiber Type	Type II (fast-twitch) (Wilson *et al*., 2012) [61]	Type I (slow-twitch) (Wilson *et al*., 2012; Gollnick *et al*., 1972) [61]
IMCL Content	Low, similar to untrained (Tamura *et al*., 2008) [52]	High, especially in oxidative muscles (Tamura *et al*., 2008) [52]
LPL Activity	Similar to controls (Nikkila *et al*., 1978)	Elevated in muscle and adipose tissue (Nikkila *et al*., 1978)
Recovery	Higher DOMS, inflammation (Howatson & van Someren, 2008)	Lower inflammation, better recovery (Howatson & van Someren, 2008)
Gene Expression (Lipid Met.)	Not elevated (Kakehi *et al*., 2020) [28]	Upregulated (e.g., PGC1A, AdipoRs) (Kakehi *et al*., 2020) [28]

The comparative analysis of sprinters and long-distance runners reveals distinct physiological adaptations that align with their respective performance requirements. Sprinters predominantly rely on anaerobic energy systems, utilizing the ATP-PCr system and glycolysis for quick, intense bursts of effort. Conversely, long-distance runners primarily engage aerobic energy pathways, enabling them to sustain prolonged physical activity through efficient oxidative phosphorylation and lipid oxidation (Romijn *et al*., 1993) [44]. Muscle fiber composition is one of the most striking differences between the two groups. Sprinters possess a higher proportion of Type II (fast-twitch) fibers, which are suited for rapid, explosive movements but have a lower capacity for fat oxidation. On the other hand, long-distance runners exhibit a higher proportion of Type I (slow-twitch) fibers, which are rich in mitochondria and are more capable of burning fat for energy, allowing for sustained endurance (Wilson *et al*., 2012; Gollnick *et al*., 1972) [17, 61]. This fiber type specialization is essential for meeting the metabolic demands of each sport.

The table no 1 also highlights the differential adaptations in lipid metabolism. Sprinters demonstrate limited lipid oxidation during exercise, with lower intramyocellular lipid (IMCL) stores, while endurance athletes show significant IMCL accumulation, reflecting the reliance on fat as an energy substrate (Tamura *et al*., 2008) [52]. Additionally, long-distance runners exhibit elevated lipoprotein lipase (LPL) activity, which facilitates efficient lipid metabolism and supports higher HDL-cholesterol levels, beneficial for cardiovascular health (Nikkilä *et al*., 1978) [37].

Recovery strategies and inflammation levels also differ between the two groups. Sprinters often experience greater post-exercise inflammation and delayed onset muscle soreness (DOMS), due to the anaerobic nature of their activity. In contrast, long-distance runners tend to experience lower inflammation, contributing to faster recovery (Howatson & van Someren, 2008). Moreover, gene expression related to lipid metabolism, such as upregulation of PGC1A and adiponectin receptors, is more pronounced in long-distance runners, supporting their capacity for efficient fat utilization (Kakehi *et al*., 2020) [28].

In summary, the metabolic profiles of sprinters and long-distance runners are distinctly tailored to their training regimens and energy demands. These adaptations enable sprinters to excel in high-intensity, short-duration efforts, while long-distance runners are optimized for endurance and fat oxidation during extended physical activity.

4. Implications, Challenges, and Future Directions

Understanding the role of lipid metabolism in athletic specialization has important implications for performance optimization, training prescription, and long-term health. For endurance athletes, strategies to maximize fat oxidation—such as fasted training, periodized carbohydrate intake, and mitochondrial-targeted supplementation—may enhance endurance and spare glycogen stores (Burke *et al*., 2016; Stellingwerff *et al*., 2019) [49]. Sprinters, though less dependent on lipid metabolism during performance, may benefit from improved lipid utilization during recovery and training off-seasons. Enhancing metabolic flexibility through HIIT, resistance training, and tailored nutrition can promote better energy balance, reduce inflammation, and improve overall athletic longevity (Volek *et al*., 2016) [60]. Nevertheless, several challenges remain. Accurately measuring in *vivo* lipid oxidation, individual variability in metabolic adaptation, and the influence of genetics are complex areas that require further exploration. Integrating omics technologies—such as metabolomics and transcriptomics—into athlete monitoring may offer deeper insights into lipid metabolism regulation and individualize training strategies (Nieman *et al*., 2015) [38]. Moreover, future research should address the intersection of sex, age, and hormonal status with lipid metabolism in athletes. Emerging evidence suggests that female athletes may exhibit different lipid oxidation patterns compared to males, influenced by estrogen levels and menstrual cycle phases (Tarnopolsky, 2008; Boisseau & Delamarche, 2000) [6, 53]. Age-related changes in mitochondrial function and fat utilization also warrant consideration in athlete development and aging.

Conclusion

Lipid metabolism plays a vital, though distinct, role in shaping the performance and physiological characteristics of sprinters and endurance runners. These differences arise from the unique energy demands of each sport. Endurance runners primarily rely on fat oxidation to sustain long-duration aerobic exercise. This reliance leads to significant adaptations such as increased mitochondrial density, enhanced lipid utilization efficiency, elevated lipoprotein lipase (LPL) activity, greater intramuscular triglyceride (IMCL) storage, and upregulation of lipid metabolism genes. These changes support efficient and sustained energy production during prolonged exercise. In contrast, sprinters depend mainly on anaerobic energy systems and carbohydrate metabolism to fuel short bursts of high-intensity activity. Although lipid metabolism plays a less direct role in sprint performance, it becomes important for recovery and long-term physiological adaptation. High-intensity training in sprinters can promote metabolic flexibility and modestly improve fat metabolism, though their adaptations related to lipid storage and utilization are generally less pronounced than in endurance athletes. These distinct metabolic profiles reflect the contrasting physiological demands of sprint and endurance training. Comparative insights from recent research highlight the necessity for tailored training and nutritional strategies that optimize lipid metabolism based on an athlete's specialization. Furthermore, genetic predispositions, dietary factors, and individual variability add complexity to the metabolic adaptations seen in athletes. A comprehensive understanding of these mechanisms can inform personalized strategies to enhance performance, support recovery, and promote long-term metabolic health in both sprint and endurance athletes.

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