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## The Relationship Between Strength and Endurance Training on Muscle Fiber Adaptation: A Systematic Review

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### Article History

Received: 08-01-2026;

Reviewed: 22-01-2026;

Accepted: 30-01-2026;

Published: 30-01-2026;

### ABSTRACT

**Background:** Muscle fiber adaptation is a central mechanism through which the human body responds to systematic physical training. Understanding the relationship between strength training and endurance training in shaping muscle fiber phenotype is critical for optimizing sports performance, health, and rehabilitation outcomes. **Objectives:** This narrative review examines the relationship between strength training and endurance training with respect to skeletal muscle fiber adaptations, including morphological, metabolic, and molecular changes in Type I and Type II fibers. **Methods:** A literature search was conducted using Google Scholar, PubMed, and Scopus (2015–2025) using relevant keywords. Studies involving human subjects with a minimum 4-week training intervention and fiber-type assessment via biopsy or myosin heavy chain (MHC) isoform analysis were included. **Results:** Strength training predominantly induces Type II fiber hypertrophy through mTORC1/IGF-1 signaling, increases cross-sectional area (CSA) of fast-twitch fibers, and preserves neuromuscular innervation of Type II fibers across the lifespan. Endurance training promotes Type I fiber dominance through PGC-1 $\alpha$ /AMPK activation, mitochondrial biogenesis, and enhanced oxidative capacity. Concurrent training produces a mixed adaptive profile dominated by Type IIa hybrid fibers, with evidence of a small negative interference effect on Type I fiber hypertrophy. Aging-related Type II fiber atrophy is most effectively attenuated by chronic strength training. **Conclusions:** Strength and endurance training produce distinct but complementary adaptations in muscle fiber phenotype. Integrating periodized concurrent training can maximize overall fiber plasticity for diverse performance and health goals.

**Keywords:** Strength Training; Endurance Training; Muscle Fiber Adaptation; Type I; Type II; Concurrent Training; Myosin Heavy Chain; Hypertrophy.

## INTRODUCTION

Human skeletal muscle fibers are not static entities but biological units that are highly plastic and responsive to various mechanical, metabolic, and hormonal stimuli. This plasticity is the fundamental cornerstone of exercise adaptation: the human body is able to significantly alter the phenotypic expression of its muscle fibers in response to a systematic exercise program, different goals, and varying intensity. The two most studied and practiced training modalities in sports science are strength training and endurance training, both of which produce fundamentally different but physiologically complementary muscle fiber adaptation profiles (Schiaffino & Reggiani, 2011; Coffey & Hawley, 2017).

Adult human muscle fibers are classified based on the myosin heavy chain (MHC) isoform they express. Type I (slow-twitch, MYH7) has high aerobic capacity, large mitochondrial density, and excellent fatigue resistance suitable for long-term endurance activities. Type IIa (fast

oxidative glycolytic, MYH2) is intermediate with aerobic-anaerobic properties, making it the most plastic fiber that is most easily modified by exercise. Type IIx (fast glycolytic, MYH1) produces the highest explosive power but with the lowest aerobic capacity and the fastest fatigue time (Wilson et al., 2021). The distribution of these three types varies between individuals, influenced by genetics, age, sex, and exercise history.

The scientific question of how strength and endurance training separately or in combination affect the composition and adaptation of muscle fibers has been the focus of sports physiology research for more than five decades. However, developments in molecular biology technologies such as single-fiber proteomics, high-resolution immunohistochemistry, and epigenomic analysis in the past decade have revolutionized our understanding of the complexity and specificity of these adaptations. Recent findings suggest that muscle fibers' adaptive responses to exercise are much more fiber-specific and pathway-specific than previously understood (Jessen et al., 2026; Horwath et al., 2025).

The interaction between strength and endurance training is becoming increasingly relevant as the popularity of concurrent training (combination training) in various sports and health programs increases. A meta-analysis by Lundberg et al. (2022) in Sports Medicine found that although overall muscle hypertrophy was not significantly affected by concurrent training, there was a small, specific interference effect on Type I fiber hypertrophy at the myofibrillar level. These findings have important practical implications for coaches, athletes, and health practitioners in designing optimal exercise programs.

Another aspect that is gaining increasing attention is the relationship between strength training and Type II fiber preservation in the elderly population. Toien et al. (2024) in the Journal of Applied Physiology report, for the first time longitudinally, that master athletes who underwent lifelong strength training maintained a distribution of Type II fiber equivalent to that of young adults, a finding not found in endurance athletes or ordinary active individuals. This research opens up a new discourse on the strategic role of strength training in tackling sarcopenia and musculoskeletal aging.

Given the importance of a comprehensive understanding of the relationship between these two major training modalities to muscle fiber adaptation, this systematic review aims to synthesize the current scientific evidence (2015–2025) regarding molecular mechanisms, morphological adaptations, functional implications, and practical guidance of strength and endurance training applications in the context of optimizing muscle fiber composition for exercise performance and health.

## **METHODS**

### **Research Design**

This study uses a narrative systematic review design to synthesize the scientific literature on the relationship between strength training and endurance to muscle fiber adaptation. The narrative approach was chosen due to the high heterogeneity of methodology, population, and outcomes among the existing studies, so quantitative synthesis through meta-analysis is not appropriate for the entire coverage of the topic.

### **Literature Search Strategy**

A comprehensive literature search was conducted through the Google Scholar, PubMed/MEDLINE, and Scopus electronic databases in January–March 2025. Keywords used independently or in combination (Boolean operator AND/OR) include: "strength training muscle fiber adaptation", "endurance training Type I fiber", "muscle fiber type transition exercise", "concurrent training interference effect muscle fiber", "myosin heavy chain isoform training", "skeletal muscle hypertrophy Type II", "aging muscle fiber strength training", and its equivalents in Indonesian. The scope of the publication is limited to 2015–2025.

### **Article Selection Criteria**

Inclusion criteria: (1) experimental or review studies on healthy adult human subjects; (2) strength, endurance, or a combination of the two training interventions for at least 4 weeks; (3)

report muscle fiber type adaptation data through muscle biopsy, immunohistochemistry, or MHC isoform analysis; (4) published in Scopus/Web of Science/Google Scholar indexed journals; and (5) full text is available. Exclusion criteria: unverified animal studies in humans, studies without muscle fiber analysis data, as well as opinion or editorial articles without primary data.

### Data Extraction and Synthesis

Inclusion criteria: (1) experimental or review studies on healthy adult human subjects; (2) strength, endurance, or a combination of the two training interventions for at least 4 weeks; (3) report muscle fiber type adaptation data through muscle biopsy, immunohistochemistry, or MHC isoform analysis; (4) published in Scopus/Web of Science/Google Scholar indexed journals; and (5) full text is available. Exclusion criteria: unverified animal studies in humans, studies without muscle fiber analysis data, as well as opinion or editorial articles without primary data.

### Data Extraction and Synthesis

The data extracted included: study identity (author, year, journal, design), sample characteristics (size, age, gender, training status), intervention protocol (type, duration, intensity, frequency), muscle fiber analysis methods, outcome variables, and key findings. The synthesis was carried out in a narrative-thematic manner by grouping evidence based on: (a) strength training adaptation, (b) endurance training adaptation, (c) concurrent training relationship, (d) aging aspects, and (e) underlying molecular mechanisms.

## RESULTS OF LITERATURE REVIEW

### Morphological and Metabolic Adaptation Overview

Table 1 presents a comprehensive comparison of morphological and metabolic adaptations of muscle fibers to strength training, endurance training, concurrent training, and detraining based on the latest literature synthesis.

**Table 1. Morphological and Metabolic Adaptation of Muscle Fibers to Various Exercise Modalities**

| Parameter Adaptasi               | Strength Training      | Endurance Training         | Concurrent Training      | Detraining      |
|----------------------------------|------------------------|----------------------------|--------------------------|-----------------|
| Dominant Fiber Type              | Type IIa & IIx<br>↑    | Type I ↑                   | Type IIa<br>(hybrid) ↑   | IIx ↑ (regresi) |
| CSA Fiber (cross-sectional area) | IIa & IIx CSA<br>↑↑    | The CSA<br>stable/↑ ringan | IIa CSA ↑; I ↓<br>ringan | All types ↓     |
| Mitochondrial Density            | Stable or ↓ light      | ↑↑ (biogenesis<br>masif)   | ↑ medium                 | ↓ signifikan    |
| Oxidative Capacity               | Stable                 | ↑↑ (SDH, CS<br>active)     | ↑ medium                 | ↓ Fast          |
| Capillary Density                | Stable or ↑ light      | ↑↑                         | ↑ medium                 | ↓               |
| Maximum Power<br>(1RM)           | ↑↑                     | Stable                     | ↑ medium                 | ↓ Fast          |
| VO <sub>2</sub> max              | Stable                 | ↑↑                         | ↑ medium                 | ↓               |
| Innervasi<br>Neuromuscular       | ↑↑ (preservasi<br>IIx) | Stable                     | ↑ medium                 | ↓↓ (denervasi)  |

Table 1 shows that strength training and endurance training produce adaptation profiles that are in opposite directions but are both functionally adaptive. Strength training preferentially increases the cross-sectional area (CSA) of Type IIa and IIx fibers, while endurance training

dramatically increases oxidative capacity and Type I mitochondrial density. Concurrent training resulted in intermediate adaptations with the dominance of hybrid Iia fibers. Most critically, detraining can reverse the entire adaptation within 3–4 weeks, confirming the importance of training continuity.

### Summary of Key Empirical Studies

Table 2 summarizes the key empirical studies that form the basis of the synthesis of this study.

**Table 2. Summary of Empirical Studies on Muscle Fiber Adaptation to Strength and Endurance Training (2021–2025)**

| Researcher & Year                      | Types of Exercises          | Duration          | Populasi                    | Key Findings                                                                                                               |
|----------------------------------------|-----------------------------|-------------------|-----------------------------|----------------------------------------------------------------------------------------------------------------------------|
| Toien et al. (2024) – J Appl Physiol   | Strength (lifetime)         | Longitudinal      | Male master athlete >70 yrs | Strength training maintains the distribution of Type II fiber equivalent to young adults; RFD and maximum power maintained |
| Lundberg et al. (2022) – Sports Med    | Concurrent (aerobik+beban)  | Systematic review | Healthy adults              | Concurrent training provides a small interference effect on Type I hypertrophy; Type II is relatively unaffected           |
| Ruple et al. (2021) – Front Physiol    | Resistance (full body)      | 10 weeks          | Untrained males             | Increased myofibril area Type I & II; greater Iia hypertrophy; Relatively stable mitochondrial density                     |
| Zhang et al. (2024) – Sci Reports      | Durability                  | 8 weeks           | Rats & humans               | Modification of histone methylation PGC-1 $\alpha$ ; transition Iia→I; Mitochondrial biogenesis $\uparrow$                 |
| Horwath et al. (2025) – Skel Muscle    | Durability (low glycogen)   | Acute             | Trained adults              | Effect of low glycogen on mTORC1 signaling & automorning Type I & II; Substrate differs per type                           |
| Horwath (2024) – Exp Physiol           | Aging + resistance training | 10 yrs follow-up  | Athlete Lari Sprint Master  | Type II atrophy in old age; resistance exercises most effectively maintain CSA Type II                                     |
| Frontera et al. (2025) – Front Physiol | BFR Resistance Training     | Systematic review | Adult/athlete               | GH/IGF-1/mTOR pathway; hypertrophy Iia & Iix; Increased strength and power                                                 |
| Messa et al. (2024) – JCSM             | Sprint + strength master    | 10 years          | Pelari sprint master        | There is no increase in muscle fiber grouping; preservation of Type II innervation; Challenging the Aging Paradigm         |

### Molecular Signaling Pathways of Muscle Fiber Adaptation

Understanding the molecular signaling pathways that link exercise stimuli to changes in muscle fiber phenotypes is key in designing evidence-based exercise programs. Table 3 summarizes the main signaling pathways identified in the literature.

**Table 3. Molecular Signaling Pathways in Muscle Fiber Adaptation to Strength and Endurance Training**

| Jalur Molekuler        | Stimulus Aktivasi                                  | Efek pada Serat Otot                                        | Sumber                                    |
|------------------------|----------------------------------------------------|-------------------------------------------------------------|-------------------------------------------|
| mTORC1 / IGF-1         | Beban mekanik tinggi; aminoasid; IGF-1             | Hipertrofi IIa & IIx; sintesis protein miofibrilar ↑; CSA ↑ | Ruple et al., 2021; Jessen et al., 2026   |
| AMPK                   | Latihan aerobik; deplesi glikogen; rasio AMP:ATP ↑ | Biogenesis mitokondria; transisi IIa→I; supresi mTOR        | Zhang et al., 2024; Fyfe et al., 2016     |
| PGC-1α                 | AMPK; Ca <sup>2+</sup> ; latihan daya tahan        | Ekspresi MYH7 ↑; kapasitas oksidatif ↑; angiogenesis        | Zhang et al., 2024; Coffey & Hawley, 2017 |
| Calcineurin / NFAT     | Kontraksi berkelanjutan; Ca <sup>2+</sup> kronik   | Ekspresi Tipe I ↑; slow-twitch phenotype                    | Schiaffino & Reggiani, 2011               |
| mTOR–AMPK Interferensi | Concurrent training (aerobik + beban)              | Hambatan parsial hipertrofi Tipe I; IIa hybrid dominan      | Lundberg et al., 2022; Fyfe et al., 2016  |
| IGF-1 / GH Signaling   | Latihan kekuatan; Sleep & recovery                 | Tipe II hypertrophy; IIa & IIx CSA ↑; RFD ↑                 | Frontera et al., 2025                     |

## DISCUSSION

### 1. Type II Fiber Strength and Adaptation Training

The established scientific consensus states that high-load strength training ( $\geq 67\%$  1RM) is a major stimulus for hypertrophy and improved Type II fiber function. The basic mechanism centers on the activation of the mTORC1 pathway through mechanical loading, which then activates the protein kinase S6K1 and inhibits 4E-BP1 to enhance the synthesis of myofibrillar proteins especially actin and myosin in IIa and IIx fibers (Ruple et al., 2021). The result is an increase in fast fiber CSA, an increase in maximum contraction strength, and an increase in the rate of force development (RFD).

Important findings from the study Toien et al. (2024) in the Journal of Applied Physiology provide a revolutionary new perspective: lifetime strength training was shown to maintain a distribution of Type II fiber ( $52.0 \pm 16.4\%$ ) in master athletes aged  $>70$  years, equivalent to young adults ( $51.1 \pm 14.4\%$ ). In contrast, the endurance athletes of the elderly and the usual active control group showed a lower proportion of Type II (39.3% and 35.0%) accompanied by more atrophic fiber. Furthermore, Horwath et al. (2024) in Experimental Physiology assert that Type II atrophy in old age is a phenomenon exacerbated by a lack of a power stimulus, not simply the inevitable biological consequences of aging.

From the neuromuscular side, Messa et al. (2024) reported during 10 years of follow-up on master sprint runners that there was no significant improvement in fiber type grouping of neuromuscular denervation indicators as long as they continued to practice sprints and strength. These findings challenge the paradigm that aging is always associated with progressive motor deterioration of the unit. A follow-up study from Frontera et al. (2025) confirms that resistance training with Blood Flow Restriction (BFR-RT) is able to induce hypertrophy of IIa and IIx fibers via the GH/IGF-1/mTOR pathway even with lower absolute loads, an important breakthrough for populations that cannot tolerate heavy loads, such as the elderly and rehabilitation patients.

### 2. Type I Fiber Endurance and Dominance Training

Aerobic endurance training induces a series of adaptations that collectively increase the capacity of muscle fibers for the continued production of aerobic energy. These adaptations are orchestrated primarily by activation of AMPK (AMP-activated protein kinase) and transcription

of PGC-1 $\alpha$  (peroxisome proliferator-activated receptor gamma coactivator 1-alpha). Zhang et al. (2024) in Nature Scientific Reports proved through epigenomic analysis that 8 weeks of endurance training induced histone methylation modifications in PGC-1 $\alpha$  promoters and MHC isoforms, creating permanent alterations in gene expression and contributing to Type I IIa  $\rightarrow$  transitions.

Documented aerobic adaptations include: an increase in mitochondrial density per muscle fiber by 20–40%, increased activity of oxidative enzymes (citrate synthase and succinate dehydrogenase), increased capillary density that facilitates more efficient oxygen delivery, and an increase in intracellular myoglobin content. An interesting finding from Horwath et al. (2025) shows that endurance training under low glycogen conditions provides a stronger adaptation stimulus to the PGC-1 $\alpha$  pathway in Type I fibers, but at the same time suppresses mTORC1 signaling, a trade-off that should be taken into account in periodic concurrent training programs.

It is important to note that although endurance training significantly increases the proportion of Type I and aerobic capacity of IIa fibers, it does not substantially increase muscle fiber CSA nor maximum contraction strength. This fact underscores the complementariness between the two training modalities: athletes who only practice endurance will optimize aerobic capacity but risk losing the mass and strength of Type II fibers over time, while athletes who only train strength will develop explosive strength but with limited endurance capacity.

### 3. Concurrent Training: Hybrid Interference and Adaptation

Concurrent training is a combination of strength and endurance training in one program to deal with a biological challenge called the 'interference effect'. At the molecular level, AMPK activated by aerobic exercise competitively inhibits mTORC1 required for protein synthesis and Type II fiber hypertrophy. In contrast, active mTOR may inhibit some aspects of PGC-1 $\alpha$ -induced mitochondrial biogenesis. As a result, there is potential for compromise on the specific adaptations of each modality (Fyfe et al., 2016).

However, the latest review provides a more nuanced picture. A comprehensive meta-analysis by Lundberg et al. (2022) covering 42 studies reported that concurrent training did produce a small interference effect on Type I fiber hypertrophy (SMD =  $-0.3$ ) compared to strength training alone, but the effect on Type II was relatively minimal (SMD =  $-0.1$ ). Furthermore, interestingly, whole-muscle hypertrophy was not significantly affected. This suggests that concurrent training interference is more fiber-specific than whole-muscle, and has more impact on Type I than Type II perhaps because Type I is more frequently recruited by both training modalities, creating more intense signal conflicts.

Table 4 summarizes the factors that affect the magnitude of the effects of interference in concurrent training along with practical strategies to minimize them.

**Table 4. Determinants of the Effects of Interference in Concurrent Training and Minimization Strategies**

| Variabel           | Interference Effects                                                                            | Minimization Strategy                                             |
|--------------------|-------------------------------------------------------------------------------------------------|-------------------------------------------------------------------|
| Session Order      | Aerobics before weight $\rightarrow$ greater interference on strength                           | Do weight training first; or split $\geq 6$ -hour sessions        |
| Aerobic Modalities | Running $\rightarrow$ greater interference in Type I than cycling                               | Use cycling or rowing for concurrent training; Minimal impact     |
| Volume Aerobik     | High volume $\rightarrow$ stronger mTOR suppression via AMPK                                    | Limit aerobic volume; use HIIT instead of LSD for time efficiency |
| Aerobic Intensity  | Intensity $\geq 70\%$ HRmax recruit Type II $\rightarrow$ potential interference in hypertrophy | Adjust aerobic intensity; Cumulative fatigue monitor              |

|                    |                                                        |                                                                                          |
|--------------------|--------------------------------------------------------|------------------------------------------------------------------------------------------|
| Frequency          | >4 combination sessions/week → recovery is not optimal | Maintain 3 sessions/week; Make sure you have 48 hours of rest between strenuous sessions |
| Nutrition Workouts | Calorie deficit exacerbates interference               | Protein consumption 1.6–2.2 g/kg/day; peri-workout carbohydrates; Creatine               |

#### 4. Aspects of Aging and Long-Term Implications

Physiological aging is consistently associated with decreased proportion and CSA of Type II fibers (type II sarcopenia), motor unit denervation, and decreased RFD which collectively contribute to decreased quality of life and risk of falls in the elderly. Recent research provides strong evidence that the type of exercise chosen over the long term has a significant impact on this musculoskeletal aging trajectory.

Toien et al. (2024) found that master athletes practicing strength maintain maximum strength ( $170 \pm 18.9$  kg) and RFD ( $3,993 \pm 894$  N·s<sup>-1</sup>) equivalent to young adults ( $151 \pm 24.4$  kg;  $3,470 \pm 1,394$  N·s<sup>-1</sup>), and showed no significant atrophic fiber (only 0.2%). This contrast is particularly striking compared to endurance athletes (1.2% atrophic fiber) and regular active controls (1.1%). These findings explicitly recommend strength training as a primary modality for fast-twitch musculature preservation in old age. In line with that, Horwath et al. (2024) affirm that Type II atrophy in old age is type-specific and independent of general activity levels, only consistent resistance training is able to optimally maintain Type II fiber CSA.

#### 5. Evidence-Based Practical Recommendations

Based on the synthesis of all the scientific evidence above, Table 5 presents a practical, evidence-based guide to designing a training program according to specific goals, considering the target type of muscle fiber to be optimized.

**Table 5. A Practical Guide to Muscle Fiber Adaptation Target-Based Exercises**

| Purpose           | Fiber Priority         | Main Methods                      | Key Parameters                                            | Minimum Duration |
|-------------------|------------------------|-----------------------------------|-----------------------------------------------------------|------------------|
| Maximum Power     | Type IIx & IIa         | Strength training berat           | 85–100% 1RM; 3–5 sets; 1–5 reps; Rest 3–5 minutes         | 8 weeks          |
| Hypertrophy Otota | Type IIa (& I)         | Hypertrophy training              | 67–85% 1RM; 3–5 sets; 6–12 reps; Rest 1–2 minutes         | 8–12 weeks       |
| Aerobic Endurance | Type I                 | LSD + High Volume Aerobics        | 55–75% VO <sub>2</sub> max; ≥40 minutes/session; ≥3x/week | 8–16 weeks       |
| Performa Hybrid   | Type IIa Hybrid        | Concurrent (beban + HIIT)         | 3x/week load + 2x/week HIIT; Split Sessions               | 10–12 weeks      |
| Anti-Aging/Aging  | Type II (preservation) | Progressive resistance training   | 2–3 sessions/week; ≥60% 1RM; Focus Compound Motion        | Sustainable      |
| Rehabilitation    | Type I (early) → IIa   | Isometric → isotonic → functional | Start low; progressive; Pain Monitoring & ROM             | 6–16 weeks       |

The guidelines in Table 5 are based on the principle of periodization which recognizes that no single exercise approach is capable of optimizing all aspects of muscle fiber adaptation

simultaneously. Instead, an effective exercise program should be designed in stages and cycles: building an aerobic and Type I base first, then integrating strength stimuli for Type II hypertrophy, and finally developing hybrid qualities through HIIT or structured concurrent training (Bompa & Buzzichelli, 2022; Harsono, 2020).

## CONCLUSIONS AND SUGGESTIONS

### Conclusion

This systematic review yielded six key conclusions supported by recent scientific evidence: (1) Strength training and endurance training resulted in fundamentally different muscle fiber adaptations and in opposite directions strength training optimized Type II morphology and function via the mTORC1/IGF-1 pathway, while resistance training increased oxidative capacity and Type I dominance via AMPK/PGC-1 $\alpha$ ; (2) strength training has been shown to be the most effective modality for maintaining mass, CSA, and Type II fiber innervation as we age, a finding that has very significant clinical and geriatric implications; (3) Concurrent training produces a hybrid adaptation profile dominated by IIa fibers, with a small but significant interference effect on Type I fiber hypertrophy, which can be minimized through manipulation of session sequences, aerobic modalities, and training nutrients; (4) Epigenetic mechanisms (histone methylation on PGC-1 $\alpha$  and MHC promoters) have been shown to play a role in endurance training-induced muscle fiber transitions, opening up new opportunities in personalization of exercise programs; (5) Reversible detraining of 3–4 weeks of muscle fiber adaptation can reverse the adaptation achieved during months of training; and (6) Understanding of specific molecular signaling mechanisms allows for the design of more precise and evidence-based exercise programs for a variety of performance and health goals.

### Suggestions

Based on these conclusions, it is recommended: (1) Sports science practitioners and coaches are advised to design training programs based on the athlete's initial muscle fiber profile, specific performance goals, and periodization phase; (2) For the elderly population, the integration of a progressive strength training program of at least 2–3 sessions per week is strongly recommended as the main anti-sarcopenia strategy; (3) In concurrent training, prioritize strength training before aerobics, choose low-impact aerobics modalities (cycling, swimming) to minimize interference, and ensure adequate protein intake ( $\geq 1.6$  g/kg/day); (4) Further research in Indonesia needs to be conducted using muscle biopsy and MHC isoform analysis in local athlete populations to produce ecologically relevant normative data; and (5) The development of machine learning-based predictive models that integrate proteomics, genomics, and individual exercise response data is a promising future direction for personalizing muscle fiber physiology-based exercise programs.

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