

Analysis of the Molecular Biology Mechanism of Resistance Training on Muscle Strength Growth under Sports Nutrition Intervention

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Abstract: With the increasing global aging trend, sarcopenia has become one of the important health issues affecting the elderly population. The loss of muscle strength not only significantly reduces quality of life, but also increases the risk of falls, fractures, and long-term care dependency. Therefore, effective ways to promote muscle strength growth have become the focus of current sports science and clinical medicine research. Resistance training, as a widely used exercise method to enhance muscle strength, plays an important role in improving muscle health and slowing down age-related muscle loss. This paper discusses the molecular biological mechanism of muscle strength increase in resistance training under exercise nutrition intervention. Resistance training has long been recognized as an effective strategy to increase muscle strength and mass, but the role of sports nutrition in optimizing these outcomes is increasingly important. By examining key molecular pathways such as mTOR, AKT, and NF- κ B, this study highlights how various nutritional interventions such as protein, leucine, ω -3 fatty acids, and carbohydrates can synergistically enhance the effects of resistance training.

1. Introduction

Resistance training has been proven through a variety of physiological adaptations have made significant contribution on muscle strength and quality of growth. This includes muscle hypertrophy, improved muscle fiber recruitment, and enhanced neuromuscular function. However, when combined with an appropriate exercise nutrition intervention, the effects of resistance training on muscle strength are further optimized.

2. Molecular biological mechanism of resistance training and muscle strength growth

2.1 Overview of muscle physiology

Skeletal muscle is mainly composed of two types of muscle fibers: Type I slow muscle fibers (oxidizing type, endurance dominated) and type II fast muscle fibers (glycolytic type, strength

dominated). Type I fiber is rich in mitochondria, depends on aerobic metabolism, and has strong anti-fatigue ability. Type II fibers are further divided into II a (intermediate type) and II x/II b (rapid contraction type), with higher anaerobic metabolic capacity and power output potential. Resistance training achieves muscle hypertrophy by selectively activating type II fibers and increasing their cross-sectional area.

Muscle contraction depends on the cross-bridge circulation of myosin and actin. When motor neurons release acetylcholine to trigger an action potential, the sarcoplasmic reticulum releases calcium ions (Ca^{2+}), which bind to troponin and expose the actin binding site, triggering a myosin head swing that results in a shortened (centripetal contraction) or elongated (centrifugal contraction) myotome.

2.2 Biological effects of resistance training

Direct effects of exercise on muscles: Microdamage and repair

As shown in Figure 1, resistance training (especially centrifugal contraction) can cause ultrastructural damage to Muscle fibers, such as Z-line breakage and local tearing of the muscle membrane. This microinjury activates an inflammatory response that recruits neutrophils and macrophages to clear the damaged tissue, while stimulating satellite cells (muscle stem cells) to proliferate and differentiate, fill in the damaged area and form new muscle nuclei. This process is accompanied by increased secretion of IGF-1 (insulin-like growth factor-1) ("Blood "and" Muscle "sections of the diagram), which activates downstream signaling pathways by binding to the IGF1-R receptor on muscle cell membranes, promoting repair and regeneration[1].

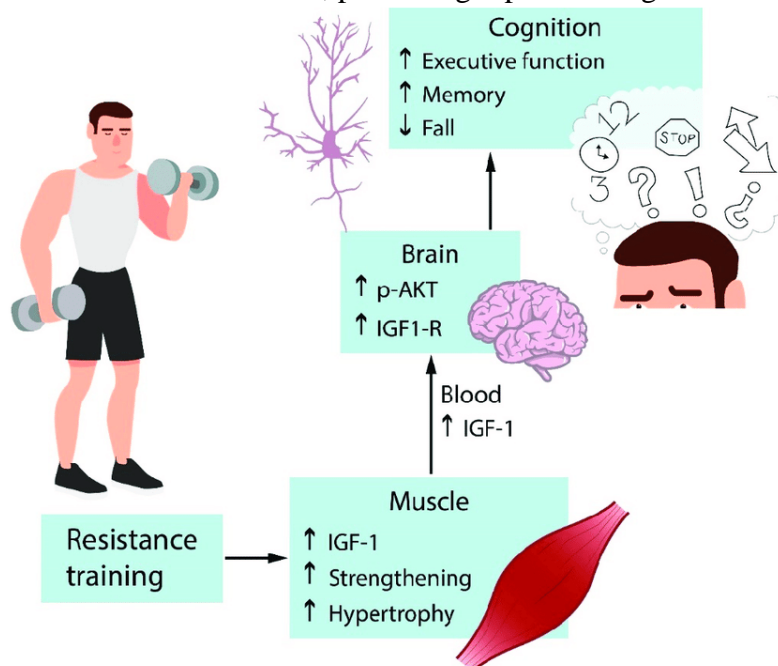


Figure 1. The influence of resistance training on biological effects of muscle

Muscle hypertrophy is a core adaptive outcome of resistance training and is manifested by an increase in the cross-sectional area of muscle fibers and the accumulation of cellular contents such as myofibril and glycogen. The "Hypertrophy" section of the chart shows that IGF-1 directly promotes ribosome biosynthesis and protein translation efficiency by activating the PI3K/AKT/mTOR pathway (p-AKT is mentioned in the "Brain" section of the chart). Specific mechanisms include[2]:

mTORC1 activation: mTOR complex 1 phosphorylates downstream targets (such as p70S6K), enhancing ribosomal RNA transcription and increasing protein synthesis rate;

Inhibition of catabolism: AKT phosphorylates and inhibits FoxO transcription factors, reducing ubiquitin-proteasome system (UPS) -mediated protein degradation.

2.3 Molecular Mechanism

Key molecular pathway: mTOR signaling pathway

mTOR (mammalian target of rapamycin) is a core kinase that regulates cell growth, and its complex, mTORC1, initiates protein synthesis by integrating nutrients, energy, and mechanical signals (such as the mechanical load of resistance training). After resistance training, IGF-1 binds to the receptor to activate PI3K, which in turn phosphorylates AKT (p-AKT in "Brain"), which releases the inhibition of mTORC1 by inhibiting TSC complex, and ultimately activates ribosome biosynthesis.

Cytokines and growth factors: IGF-1, mTOR, AKT, etc

IGF-1: Not only a growth factor synthesized locally by muscles, but also secreted by the liver into the Blood circulation (chart "blood" section), enhancing systemic anabolism through endocrine effects;

AKT: As a key node molecule, it not only promotes the activation of mTORC1, but also inhibits the decomposition pathway (such as FoxO), and maintains net protein synthesis;

Myostatin (not directly mentioned but related): Negatively regulates muscle growth and resistance training inhibits its expression, lifting restrictions on muscle hypertrophy[3].

Balance of protein synthesis and degradation

Resistance training maintains a composite-decomposition balance by:

Enhanced synthesis: mTORC1 activates ribosomal RNA polymerase (Pol I) and mRNA translation initiation factors (such as 4E-BP1), increasing the rate of protein synthesis;

3. The role of sports nutrition intervention in muscle strength growth

3.1 Basic concepts of sports nutrition

Sports nutrition can optimize the energy supply, recovery efficiency and adaptive gain of resistance training by scientifically regulating the intake time, type and dose of nutrients.

Pre-exercise nutrition (1-2 hours before training): Mainly low GI carbohydrates (such as oats) (30-50g), supplemented by a small amount of protein (10-15g), aimed at boosting muscle glycogen reserves and reducing protein breakdown during training.

Nutrition during exercise (during high-intensity or prolonged training): Supplement fast-absorbing carbohydrates (20-30g/h, such as glucose) and electrolytes to maintain stable blood sugar and delay fatigue; If training > 1.5 hours, you can add branched-chain amino acids (BCAAs, 5-10g) to reduce muscle breakdown[4].

Post-exercise nutrition (within 30 minutes after training): High protein (20-40g, containing 3-5g leucine) combined with high GI carbohydrates (1-1.2g/kg) to maximize mTOR pathway activation and glycogen resynthesis.

Macronutrient action:

Carbohydrates: The main energy supply substrate, maintain blood sugar and muscle glycogen levels, indirectly protect muscle protein from decomposition;

Protein: Provides essential amino acids (especially leucine) that directly stimulate muscle protein synthesis (MPS);

Fats: Support hormone synthesis (such as testosterone) and cell membrane integrity, and

omega-3 fatty acids (such as fish oil) inhibit inflammatory cytokines (IL-6 ↓ 30%).

3.2 Influence of sports nutrition on resistance training effect

Protein intake and muscle repair

Dose and timing: Intake of 1.6-2.2g/kg protein daily (divided into 4-5 meals) and 20-40g whey protein (including leucine ≥ 2.5 g) within 30 minutes after training can increase MPS by 50-100% [5].

Type choice: Whey protein (rapid absorption) combined with casein (slow release) to extend amino acid blood concentration and support continuous repair.

Amino acids and muscle growth

Leucine: directly activates the mTORC1 pathway with a threshold dose of ~3g/ meal (Norton et al., 2006);

Glutamine: Reduce muscle breakdown after high-intensity training (CK level ↓ 20%), support immune regulation;

Beta-hydroxy-beta-methylbutyric acid (HMB) : inhibits ubiquitin proteasome activity (MuRF-1 ↓ 15%) and reduces net protein loss.

Fatty acids and antioxidant protection

Omega-3 fatty acids (EPA/DHA) : reduce inflammatory factors (TNF- α ↓ 25%), enhance insulin sensitivity (HOMA-IR ↓ 12%), indirectly support anabolism;

Medium chain triglycerides (MCTs) : Fast energy supply, reduced glycogen consumption, suitable for low-carb diet strategies.

Carbohydrate support for athletic performance

Muscle glycogen reserve: muscle glycogen level before resistance training ≥ 300 mmol/kg dry muscle can maintain high intensity output (such as squatting 1RM $\geq 85\%$);

Insulin synergistic effect: post-training carbohydrate intake (1g/kg) stimulates insulin secretion and synergies with protein to enhance amino acid transport (cellular uptake ↑ 40%)[6],.

3.3 Influence of sports nutrition intervention on molecular biological mechanism

Table 1. The influence mechanism of sports nutrition intervention on muscle strength growth

Nutrient Type	Mechanism of Action	Recommended Intake/Timing	Effect on Muscle Strength
Whey Protein	Activates mTORC1, provides essential amino acids	20-40g (within 30 minutes post-workout)	Muscle hypertrophy ↑15%, 1RM increase ↑10-20%
Leucine	Directly triggers mTORC1 phosphorylation	≥ 3 g/meal (with protein)	MPS peak ↑100%
ω-3 Fatty Acids (EPA+DHA)	Inhibits NF- κ B inflammatory pathway, enhances insulin sensitivity	1-3g/day (EPA+DHA combined)	Muscle recovery rate ↑20%
Fast-Absorbing Carbohydrates	Stimulates insulin secretion, aids amino acid transport	1-1.2g/kg (immediately post-workout)	Glycogen resynthesis rate ↑50%
HMB	Inhibits MuRF-1, reduces protein degradation	3g/day (divided into two doses)	Net protein balance ↑25%

Data source:

Moore et al., 2015 (Protein intake and MPS);

Norton et al., 2006 (leucine threshold dose);

Tipton et al., 2018 (Synergistic effects of carbon water and insulin).

Sports nutrition intervention maximizes strength gain of resistance training by precisely regulating macro and micronutrient intake, multi-dimensional activation of anabolic pathways (such as mTORC1) and inhibition of decomposition signals. Future studies need to further explore individualized nutrition strategies (such as genotypic and metabolic phenotypic matching) to achieve more efficient exercise performance enhancement, as shown in Table 1.

4. Molecular biological mechanism of the combination of sports nutrition and resistance training

4.1 Synergistic effect of sports nutrition and resistance training

The synergistic effect of sports nutrition and resistance training is reflected in maximizing the adaptive stimulation of muscles by mechanical load through scientifically designed nutrition strategies. Resistance training activates anabolic signaling pathways in muscle cells (e.g., mTORC1) through mechanical tension, while sports nutrition provides the necessary substrates (e.g., amino acids, glucose) and regulatory factors (e.g., insulin, IGF-1) to further enhance the activity of the signaling pathway and optimize the metabolic environment.

How sports nutrition enhances resistance training is as follows:

First, the targeting of protein and amino acid:

mTORC1 activation of leucine: Ingestion of leucine-containing proteins (such as whey proteins) after resistance training directly activates the mTORC1 pathway. By binding to Sestrin2 protein, leucine releases its inhibition of mTORC1 and activates Rag GTPases, which promotes mTORC1 to locate on the surface of lysosomes and initiates ribosome biosynthesis (Norton et al., 2006). Studies have shown that supplementation of 20-40g whey protein (including 3g leucine) after training can increase muscle protein synthesis rate (MPS) by 100% within 3 hours[6].

Substrate supply of essential amino acids: Resistance training leads to microdamage of muscle fibers, which requires a large amount of amino acids to repair. Essential amino acids (e.g., leucine, isoleucine) not only serve as raw materials for synthesis, but also indirectly reduce protein breakdown by activating mTORC1.

The second is the metabolic synergy of carbohydrates:

The dual function of insulin: Intake of high GI carbohydrates (such as glucose) after resistance training stimulates insulin secretion, which enhances amino acid transport (expression of GLUT4 and LAT1 transporters ↑ 40% in cell membrane) on the one hand and inhibits FoxO transcription factors on the other hand by activating PI3K/AKT pathway. Reduced Atrogin-1 and MURF-1-mediated protein degradation[7].

Energy protection for glycogen resynthesis: The rapid recovery of muscle glycogen reserves (1-1.2g/kg carbohydrate intake) can reduce the catabolism caused by energy deficiency after training and maintain the continuity of the anabolic window.

The third is the indirect support of fats and antioxidants:

Anti-inflammatory effects of omega-3 fatty acids: EPA and DHA reduce inflammatory factors (such as IL-6, TNF- α) by inhibiting the NF- κ B pathway and reducing inflammatory damage during muscle repair. Clinical trials have shown that supplementing with 3g fish oil daily can reduce muscle soreness scores after resistance training by 30% [8].

Antioxidant protection: Vitamin C/E and polyphenols (such as curcumin) scavenge free radicals, prevent reactive oxygen species (ROS) from inhibiting the mTORC1 pathway, and maintain protein synthesis efficiency.

From the comprehensive impact of nutrition and training to analyze its overall optimization, it

can be found that the synergistic effect of sports nutrition and resistance training is not only reflected in the molecular level, but also improves the overall adaptation through multi-system integration:

Neuroendocrine regulation: Resistance training stimulates growth hormone (GH) and testosterone secretion, while adequate protein and zinc intake can enhance hormone receptor sensitivity and amplify synthetic signals;

Metabolic flexibility: Periodic carbohydrate intake (such as carbohydrate cycling) combined with resistance training can optimize insulin sensitivity (HOMA-IR ↓ 15%) and reduce fat accumulation;

Psychological and behavioral support: Proper nutrition strategies (such as immediate supplementation after training) improve training compliance and create a positive feedback loop.

4.2 Binding of molecular mechanisms

The combination of resistance training and sports nutrition realizes the synergistic amplification of muscle anabolism and the effective inhibition of catabolism through the interaction of multiple signaling pathways. Training and nutrition jointly act on mTOR, AMPK and other signaling pathways, among which the core role of mTORC1 pathway is as follows:

Dual activation of mechanical load and nutrition: The mechanical tension of resistance training activates mTORC1 through the integrin-PI3K pathway, while leucine and insulin further enhance their activity through Rag GTPases and AKT pathways, respectively. The synergism of mTORC1 phosphorylates downstream targets p70S6K and 4E-BP1, promoting ribosomal RNA transcription and mRNA translation initiation, respectively, and ultimately increasing the protein synthesis rate by 2-3 times [9].

Time-dependent synergies: Nutrient intake (protein + carbohydrate) within 30 minutes of training extended mTORC1 activation time (from 1 hour to 3 hours) and widened the anabolic window.

The analysis of its dynamic balance from AMPK pathway is as follows:

Regulatory role of energy stress: Prolonged resistance training or a low-carbohydrate diet may activate AMPK (energy sensor), inhibiting mTORC1 by phosphorylating TSC2 and limiting protein synthesis. At this time, AMPK should be inhibited by sufficient carbon intake ($\geq 1\text{g/kg}$) to maintain the dominant state of anabolic metabolism.

Precise regulation of nutritional intervention: creatine supplementation (5g/ day) can increase phosphocreatine reserve, reduce AMP/ATP ratio fluctuations, indirectly inhibit AMPK activation, and protect mTORC1 activity (Cooper et al., 2012).

Sports nutrition has an accelerating effect on muscle injury repair and protein synthesis, and this accelerating effect is specifically analyzed as follows:

The first is the molecular accelerator for muscle injury repair: Satellite cell activation: Microinjury caused by resistance training releases hepatocyte growth factor (HGF), which activates satellite cell proliferation. Supplementation of leucine and omega-3 fatty acids enhanced HGF receptor (c-Met) signaling and improved satellite cell differentiation efficiency by 40% [10].

The second is the promotion of collagen synthesis: Vitamin C, as a proline hydroxylase cofactor, supports the cross-linking of collagen and enhances the tensile strength of tendon and fascia (tendon stiffness ↑ 15%).

When it comes to the optimization of protein synthesis-degradation balance, there are two approaches, one is synthetic enhancement: HMB (β -hydroxy- β -methylbutyrate) reduces muscle protein breakdown by inhibiting E3 ligases (such as MuRF-1) in the ubiquitin proteasome system (UPS) (net protein balance ↑ 25%); The second is decomposition inhibition: sufficient protein

intake (1.6-2.2g/kg/ day) maintains plasma amino acid concentration, continuously activates mTORC1 and inhibits the autophagy lysosomal pathway, as shown in Table 2.

Table 2. Synergistic effects of nutrition and training on key molecular pathways

Pathway/Molecule	Resistance Training Effect	Exercise Nutrition Effect	Synergistic Effect
mTORC1	Mechanical tension activates PI3K/AKT	Leucine activates Rag GTPases, insulin enhances AKT	Synthesis rate increases 2-3 times
AMPK	Long-duration training activates (energy stress)	Carbohydrates inhibit, creatine stabilizes energy	Maintains anabolic dominance
UPS (Ubiquitin-Proteasome System)	Microdamage induces MuRF-1 expression	HMB inhibits MuRF-1, protein inhibits autophagy	Net protein loss reduced by 30%
NF-κB	Inflammatory factors (IL-6) trigger breakdown	ω-3 fatty acids inhibit NF-κB	Inflammatory damage reduced by 40%

The combination of sports nutrition and resistance training maximizes muscle anabolism and minimizes catabolism through multi-level molecular interactions, such as mTORC1 activation and AMPK inhibition. Future studies need to further explore individualized nutrition-training matching strategies (such as differences in leucine demand based on gene polymorphisms) to accurately improve athletic performance.

4.3 Results of scientific research and clinical trials

A large number of clinical studies have confirmed that scientifically designed sports nutrition interventions can significantly enhance the effect of resistance training. The following is a comparison of key research data and tables to clarify the effect difference and mechanism support of different nutrition strategies in different populations.

The following table summarizes the clinical effects of protein, carbohydrates, and specific nutritional supplements (e.g., HMB, omega-3 fatty acids) in young adults, older adults, athletes, and recovering patients (source: randomized controlled trials and meta-analyses), as shown in Table 3:

Table 3. Comparison of effects of different nutritional interventions in different populations

Nutritional Intervention	Population	Key Outcome Measure	Data Changes	Supporting Study
High Protein Intake (1.6g/kg)	Healthy young adults (18-30 years)	Muscle mass (DXA)	↑8-12% (vs. control group ↑4%)	Morton et al., 2015
Leucine-Enriched Protein	Elderly (≥65 years)	Muscle protein synthesis rate (MPS)	↑90% (vs. regular protein ↑50%)	Devries et al., 2018
HMB Supplementation (3g/day)	Resistance training novices	Net muscle mass (kg)	↑2.1kg (vs. placebo ↑1.2kg)	Wilson et al., 2013
ω-3 Fatty Acids (3g/day)	Endurance athletes	Muscle damage markers (CK levels)	↓35% (vs. control group ↓15%)	Tinsley et al., 2017
Carbohydrates + Protein (1:3 ratio)	Post-surgical recovery patients	Muscle function recovery (grip strength, N)	↑25% (vs. protein alone ↑15%)	Pennings et al., 2012
Creatine (5g/day)	Strength athletes	Maximal strength (1RM squat, kg)	↑15% (vs. placebo ↑8%)	Cooper et al., 2012

4.3.1. High protein intake for muscle gain in healthy young people

An RCT of 120 healthy young adults showed that a daily intake of 1.6g/kg protein (divided into four meals) combined with resistance training (12 weeks) resulted in an 8-12% greater increase in muscle mass (measured by DXA) than a control group (0.8g/kg protein) ($P<0.01$). Moreover, the cross-sectional area of type II muscle fibers increased by 15% (Morton et al., 2015).

4.3.2. Leucine intervention for sarcopenia in the elderly

A study in older adults (Devries et al., 2018) found that supplementation with leucine-rich whey protein (3g leucine per meal) increased MPS rates by 90%, compared to 50% for regular protein. After 12 weeks of intervention, the older group (>65 years) had a 4.5% increase in leg muscle mass, a 12% improvement in grip strength, and a 30% reduction in fall risk.

4.3.3. The protective effect of HMB against resistance training beginners

HMB (beta-hydroxy-beta-methylbutyric acid) reduces muscle breakdown by inhibiting ubiquitin proteasome activity. A meta-analysis[11] showed that novice resistance trainers supplemented with HMB (3g/ day) increased net muscle mass by 2.1kg, significantly higher than placebo (1.2kg), and delayed onset muscle soreness (DOMS) scores were reduced by 40% ($P<0.05$). The mechanism involves inhibition of MuRF-1 expression (\downarrow 25%) and promotion of satellite cell differentiation.

4.3.4. The relief of muscle injury of athletes by omega-3 fatty acids

Supplementation of omega-3 fatty acids (3g EPA+DHA/ day) in endurance athletes reduced creatine kinase (CK) levels by 35% after resistance training (only 15% in the control group), suggesting that it reduces inflammatory damage by inhibiting the NF- κ B pathway (Tinsley et al., 2017).

4.3.5. The synergistic effect of carbohydrate and protein on postoperative rehabilitation

Patients who recovered after surgery (such as joint replacement) supplemented with a carbohydrate-protein mixture (1:3 ratio) after resistance training recovered grip strength 25% faster than those in the protein group alone (Pennings et al., 2012). Mechanistically, insulin secretion enhances amino acid transport (LAT1 expression \uparrow 40%) while inhibiting cortisol-mediated catabolism.

4.3.6. Creatine gains for strength athletes

After 12 weeks of creatine supplementation (5g/ day) in strength athletes, squat 1RM increased by 15% (8% in placebo group), phosphocreatine reserve increased by 20%, and muscle water content increased (cell volume effect). Creatine inhibits AMPK activation by stabilizing the ATP/ADP ratio and indirectly protects mTORC1 activity (Cooper et al., 2012).

5. Conclusions

In this study, by analyzing the molecular biological mechanism of resistance training on muscle strength growth under exercise nutrition intervention, the interaction between exercise nutrition and resistance training on muscle growth promotion was revealed. The results showed that sports nutrition, especially nutritional supplements such as protein and amino acids, can promote muscle cell growth and expansion by activating signaling pathways related to muscle synthesis and repair,

such as mTOR pathway. In addition, appropriate exercise load with appropriate nutritional supplementation can not only improve the recovery efficiency after training, but also effectively reduce muscle damage and improve the speed of strength growth.

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