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Regulatory Role of SIRT1 in Skeletal Muscle Hypertrophy: Molecular Implications

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Abstrak

Latar Belakang: Hipertrofi otot rangka merupakan proses adaptif yang dipengaruhi oleh sinyal molekuler dan kondisi metabolik sel. Sirtuin 1 (SIRT1), enzim deasetilase yang bergantung pada NAD^+, memiliki peran penting dalam regulasi pertumbuhan dan perbaikan otot melalui interaksinya dengan berbagai jalur utama seperti mTOR, PGC-1α, FOXO, serta faktor miogenik. Tujuan: Artikel ini bertujuan untuk merangkum bukti ilmiah terkini mengenai peran SIRT1 dalam hipertrofi otot, termasuk pengaruhnya terhadap efisiensi mitokondria, penurunan stres oksidatif, dan penekanan gen katabolik seperti atrogin-1 dan MuRF1. Metode: Kajian ini dilakukan melalui penelusuran literatur pada basis data PubMed, ScienceDirect, dan Google Scholar dengan fokus pada artikel yang dipublikasikan dalam 10 tahun terakhir. Hasil: Hasil kajian menunjukkan bahwa efek SIRT1 sangat bergantung pada konteks, khususnya kondisi energi sel dan faktor eksternal seperti aktivitas fisik atau restriksi kalori. SIRT1 tidak secara langsung menginduksi hipertrofi, tetapi berperan dalam menciptakan lingkungan seluler yang mendukung pertumbuhan otot jangka panjang. Kesimpulan: Temuan ini menunjukkan potensi besar SIRT1 dalam pengembangan terapi berbasis molekuler untuk mempertahankan atau meningkatkan massa otot, terutama pada populasi lanjut usia atau kondisi patologis tertentu.

Kata kunci: SIRT1, Hipertrofi, Otot Rangka, Molekuler.

Abstract

Background: Skeletal muscle hypertrophy is an adaptive process shaped by molecular signals and metabolic environments. Sirtuin 1 (SIRT1), a NAD^+-dependent deacetylase, is essential for the regulation of skeletal muscle growth and repair via its interaction with critical pathways including mTOR, PGC-1α, FOXO, and myogenic factors. **Aim:** This article encapsulates the existing scientific evidence regarding the role of SIRT1 in muscle hypertrophy, encompassing its impact on mitochondrial efficiency, reduction of oxidative stress, and suppression of catabolic genes such as atrogin-1 and MuRF1. **Method:** This article review was performed utilizing databases including PubMed, ScienceDirect, and Google Scholar, concentrating on articles published within the last decade. **Result:** These findings demonstrate that the effects of SIRT1 are significantly context-dependent, fluctuating with cellular energy levels and external factors such as physical activity or caloric restriction. SIRT1 does not directly induce hypertrophy; instead, it promotes a conducive cellular environment that supports prolonged muscle growth. **Conclusion:** These insights present significant potential for the advancement of molecular-based therapies aimed at preserving or augmenting muscle mass, especially in aging populations or pathological conditions.

Keywords: SIRT1, Hypertrophy, Skeletal Muscle, Molecular

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1. INTRODUCTION

Skeletal muscle hypertrophy is a physiological adaptation resulting from mechanical stimuli, such as resistance training, characterized by an enlargement of muscle fibers due to enhanced protein synthesis and reduced protein degradation[1,2]. This process is crucial for enhancing muscle strength and functional performance while decreasing dependency, particularly among the elderly population [3].

In the last twenty years, scientific inquiry of the molecular mechanisms concerning muscle hypertrophy has intensified, primarily concentrating on the mTOR (Mechanistic Target of Rapamycin) and IGF-1/Akt (Insulin-like Growth Factor-1/Protein Kinase B) pathways, alongside the regulation of transcription factors such as MyoD (Myogenic Differentiation-1) and FOXO (Forkhead Box O) [1,4]. Sirtuin 1 (SIRT1), a NAD^+-dependent deacetylase enzyme, is recognized as a crucial molecular regulator of metabolism, oxidative stress, and skeletal muscle plasticity [5,6].

SIRT1 demonstrates diverse functions in muscle tissue, such as modulating energy homeostasis through PGC-1α (Peroxisome Proliferator-Activated Receptor Gamma Coactivator 1-alpha) [7], inhibiting the atrophy pathway via FOXO deacetylation [8], and engaging with the mTOR pathway implicated in muscle protein synthesis [9]. The modulation of SIRT1 activity by environmental factors, including exercise, caloric restriction, and bioactive compounds, positions it as a potential target for the prevention and treatment of muscle mass disorders [10,11].

The involvement of SIRT1 in muscle metabolism regulation has been extensively researched; however, its function in muscle hypertrophy remains contentious. Certain studies indicate that SIRT1 activation promotes muscle hypertrophy by enhancing metabolic efficiency and reducing oxidative stress, while others propose that excessive SIRT1 activity may impede anabolic signaling [9,12].

This review article seeks to summarize and evaluate the existing scientific evidence regarding the role of SIRT1 in muscle hypertrophy, encompassing its molecular mechanisms, findings from experimental studies, and potential applications in clinical and lifestyle interventions. This methodology is anticipated to yield extensive insights and establish a foundation for the formulation of molecular-based therapies aimed at preserving and augmenting skeletal muscle mass.

2. METHODS

This article synthesizes current scientific evidence on the molecular role of SIRT1 in skeletal muscle hypertrophy. Relevant literature was collected from major biomedical databases including PubMed, ScienceDirect, and Google Scholar. The selection of references was based on their relevance to SIRT1 signaling, skeletal muscle physiology, hypertrophy-related pathways, and experimental or mechanistic findings, using keywords such as "SIRT1 and muscle mass," "SIRT1 AND muscle hypertrophy," “hypertrophy AND molecular mechanisms,” and "SIRT1 AND skeletal muscle.” Priority was given to recent publications (2015–2024) to reflect current advances in molecular research, while seminal earlier studies were included when necessary to support foundational concepts. The authors critically evaluated and integrated findings from original research articles, review papers, and experimental studies to provide a comprehensive and coherent overview of SIRT1-associated molecular mechanisms.

3. RESULT

3.1. Structure and Function of SIRT1

Sirtuin 1 (SIRT1) is the most extensively researched member of the sirtuin family (SIRT1-SIRT7), a collection of proteins that act as NAD^+-dependent deacetylase enzymes. SIRT1 is encoded by the SIRT1 gene, situated on chromosome 10q21.3 in humans. This protein is primarily situated in the cell nucleus, though it can translocate to the cytoplasm based on the cell's physiological requirements [13].

SIRT1 promotes the deacetylation of multiple protein targets, encompassing transcription factors and metabolic regulators such as PGC-1α, Forkhead Box O (FOXO), Nuclear Factor Kappa-light-chain-enhancer of Activated B cells (NF-κB), and p53. The enzymatic function of SIRT1 is contingent upon the presence of NAD^+, rendering it responsive to cellular energy levels and metabolic strain. SIRT1 operates as a "metabolic sensor" that amalgamates nutritional signals, oxidative stress, and physical activity into cellular adaptive responses [5–9].

SIRT1 in skeletal muscle tissue promotes mitochondrial biogenesis, workload adaptation, and defense against oxidative stress. This renders it a crucial molecule for the regulation of muscle growth and function. The activity of SIRT1 is elevated in response to endurance training, caloric restriction, and the administration of activator compounds like resveratrol [2,10].

3.2. Molecular Mechanisms of SIRT1 in Muscle Hypertrophy

3.2.1. SIRT1 and m-TOR Pathway

The mechanistic target of rapamycin (mTOR) pathway is a crucial regulator of skeletal muscle growth and hypertrophy, functioning by enhancing protein synthesis via the activation of the mTORC1 complex. Activation of mTORC1 enhances protein translation via the phosphorylation of critical targets, including S6K1 (Ribosomal Protein S6 Kinase 1) and 4E-BP1 (Eukaryotic Translation Initiation Factor 4E Binding Protein 1). This pathway is significantly affected by nutrient signals, growth factors such as IGF-1, and cellular energy status (Figure 1). In this context, SIRT1 functions as a metabolic sensor that is crucial in regulating activation or inhibition [9].

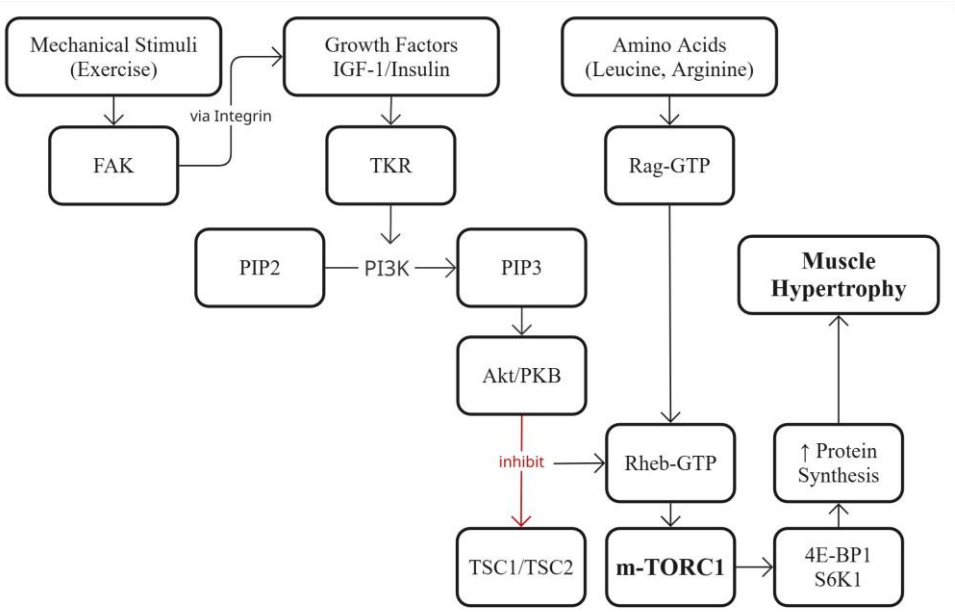

Figure 1. Signaling pathways converge on mTORC1 to promote muscle hypertrophy. Mechanical stimuli (exercise) activate Focal Adhesion Kinase (FAK) and integrin signaling, whereas IGF-1/insulin activates tyrosine kinase receptors (TKR), stimulating the PI3K–Akt pathway. Akt inhibits the TSC1/2 complex, facilitating Rheb-GTP to activate mTORC1. Amino acids simultaneously activate Rag-GTP, promoting the localization of mTORC1 to lysosomes. mTORC1 facilitates protein synthesis through S6K1 (Ribosomal Protein S6 Kinase 1) and 4E-BP1 (Eukaryotic Translation Initiation Factor 4E Binding Protein 1), thereby stimulating muscle hypertrophy.

SIRT1 indirectly inhibits mTORC1 activity via the activation of AMPK (AMP-activated protein kinase). This activation emerges as SIRT1 deacetylates LKB1 (Liver Kinase B1), an upstream kinase essential for activating AMPK. Upon activation, AMPK inhibits mTORC1 via two primary mechanisms: firstly, by activating the TSC2 complex, which serves as an inhibitor of Rheb, a GTPase critical for mTORC1 activation; and secondly, by phosphorylating mTORC1 components like Raptor, thereby diminishing its activity. This leads to a reduction in protein synthesis, consequently inhibiting muscle hypertrophy [14].

Nonetheless, the influence of SIRT1 on mTOR is not invariably detrimental. Under specific physiological

conditions, such as adequate cellular energy levels or the post-exercise recovery phase, SIRT1 indirectly supports mTOR activity. SIRT1 enhances metabolic efficiency, diminishes oxidative stress, and stabilizes the intracellular environment, thereby fostering conditions that activate anabolic pathways such as mTORC1 [9,12]. Mild activation of SIRT1 via exercise or the intake of compounds like resveratrol has demonstrated an increase in muscle mass in certain animal models [15–17].

The relationship between SIRT1 and mTOR is contextual, contingent upon energy status, stimulus type, and activation level [18]. SIRT1 functions as a regulator of energy equilibrium by inhibiting muscle hypertrophy during periods of low energy availability. Conversely, it facilitates metabolic adaptation and muscle anabolism when circumstances allow (Figure 2). A comprehensive comprehension of these dynamics is crucial for formulating molecular strategies that target SIRT1 or mTOR to preserve muscle mass in pathological conditions or aging.

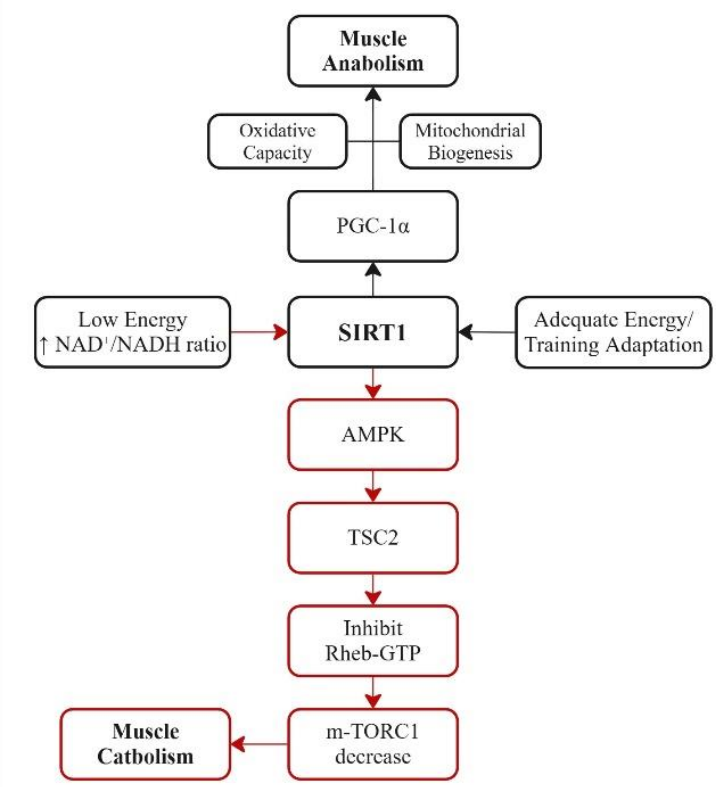

Figure 2. Dual Role of SIRT1 in Regulating Muscle Anabolism and Catabolism. SIRT1 functions as a metabolic switch that regulates muscle mass in response to the cellular energy status. Under low-energy conditions (elevated NAD^+/NADH ratio), SIRT1 activates AMPK, leading to the TSC2-mediated inhibition of Rheb-GTP and reduced mTORC1 activity, resulting in muscle catabolism. In contrast, adequate energy or training adaptation promotes SIRT1-induced PGC-1α activation, enhancing oxidative capacity, and mitochondrial biogenesis, which supports long-term muscle anabolism.

3.2.2. SIRT1 and PGC-1α Pathway

SIRT1 is an enzyme sensitive to cellular energy status. Under low energy conditions such as during exercise or caloric restriction, NAD^+ increases, activating SIRT1 [5,10,11]. In skeletal muscle, SIRT1 mainly targets PGC-1α, a transcriptional coactivator crucial for controlling the expression of genes in the mitochondria [19,20].

The body utilizes classic anabolic signals, such as mTOR, to augment muscle mass in skeletal muscle tissue. It necessitates a robust energy support system for optimal muscle hypertrophy. Consequently, the functions of SIRT1 and PGC-1α are essential.

SIRT1 functions as a guardian of energy balance in cells. When muscles are subjected to metabolic stress, such as resistance training or caloric restriction, the levels of NAD^+ in the cells increase. This increase in NAD^+ levels

triggers the activation of SIRT1 [10,20]. Once activated, SIRT1 deacetylates PGC-1α, a transcriptional coactivator essential for controlling mitochondrial biogenesis. This deacetylation process not only activates PGC-1α but also prolongs the stability and duration of its activity in the cell nucleus [21,22].

Once activated, PGC-1α goes straight to work: it increases the expression of genes responsible for the formation of new mitochondria and the improvement of the function of existing mitochondria. As a result, muscle fibers, especially type I, which are dominant in endurance activities, experience an increase in oxidative capacity and energy production efficiency [21–23].

This pathway is recognized for its role in adaptation to resistance training, and its contribution to muscle hypertrophy is significant. Muscles abundant in mitochondria possess an enhanced capacity to sustain energy provision during protein synthesis. Furthermore, an intracellular milieu devoid of oxidative stress facilitates the stable operation of growth pathways, such as mTORC1 [24,25]. In other words, SIRT1 and PGC-1α create a metabolic foundation that allows the muscles to grow efficiently and durably.

Consequently, while SIRT1 does not directly induce muscle hypertrophy as mTOR does, it facilitates the maintenance of sustained hypertrophy by bolstering the foundational energy mechanisms. When muscles possess adequate energy reserves and a conducive internal environment, growth signals are significantly more efficacious in augmenting muscle mass.

3.2.3. SIRT1 and FOXO-Atrogin-1 Pathway

Once SIRT1 is activated, it targets FOXO transcription factors, specifically FOXO1 and FOXO3a, which naturally translocate into the nucleus when cells are stressed. FOXO associates with the promoters of catabolic genes, including atrogin-1 and MuRF1, within the nucleus. These two genes are components of the ubiquitin-proteasome pathway, a principal mechanism in the breakdown of muscle proteins during muscle atrophy [8,26].

SIRT1 then deacetylates the FOXO regulatory domain, which is a part of FOXO that determines its activity in binding to DNA and turning on target gene expression. This deacetylation causes FOXO to lose its affinity for the promoter of the atrophy gene, and at some stage, can eject it back into the cytoplasm. The expression of atrogin-1 and MuRF1 (Muscle RING-Finger Protein-1) is significantly reduced, thereby inhibiting muscle protein degradation [8,27].

The outcome is a reduction in muscle protein degradation and the preservation of muscle mass from atrophy. This discovery is significant in relation to hypertrophy. Despite mTOR and other growth pathways facilitating protein synthesis, the efficacy of hypertrophy remains contingent upon the degradation rate. SIRT1 contributes to this protective function by inhibiting catabolic pathways at the appropriate moment.

Numerous studies utilizing animal models have demonstrated that activation of SIRT1 by compounds like resveratrol or through exercise significantly reduces the expression of atrogin-1 and MuRF1, concurrently decelerating the loss of muscle mass [28,29]. Consequently, SIRT1 activation not only establishes a more metabolically stable environment but also inhibits muscle degradation pathways, facilitating the effective functioning of mTOR and other anabolic signals.

3.2.4. SIRT1 and Myogenic Factors

When muscles are damaged by intense training, injury, or natural regeneration processes, the body immediately activates satellite cell-muscle stem cells that are inactive under normal conditions, but ready to proliferate when needed [23,30]. Satellite cell activation, proliferation, and differentiation into new muscle fibers are all controlled by myogenic factors, mainly myogenin and MyoD [23,30,31].

SIRT1 is involved in the regulation of this regenerative pathway, particularly during differentiation. Once activated, SIRT1 targets MyoD, a key transcription factor in the early phase of differentiation. SIRT1 deacetylates MyoD and suppresses its transcriptional activity under specific conditions This is not a form of total inhibition

but a form of time regulation. SIRT1 ensures that satellite cells enter the proliferation phase before immediately differentiating into myoblasts. This is important, as the number of available stem cells is not immediately depleted in one regeneration cycle [31–33].

At a later stage, when proliferation is sufficient, SIRT1 permits the full expression of MyoD and activates myogenin, a factor that is more predominant in the terminal phase of differentiation. This interaction allows myoblasts to fuse to form mature myotubules [30,31,33].

With the timely and controlled stimulation of MyoD and myogenin, muscle regeneration was optimized. The damaged muscle tissue was replaced with new, healthy fibers. In the context of weight training, the repeated activation of this process enhances muscle volume by increasing both the quantity and size of fibers, a process functionally termed hypertrophy [33,34].

SIRT1 acts as a regeneration timer, restraining differentiation in the proliferation phase, and releasing its brakes once the environment is ready. When MyoD and myogenin start working, SIRT1 prepares the cell and molecular environment to increase energy efficiency, suppress local inflammation, and protect against oxidative stress during the regeneration process. Therefore, the role of SIRT1 is not only in the early regulatory stage but continues until the integration of myotubes into mature muscle tissue [35,36].

4. DISCUSSION

Although the role of SIRT1 in skeletal muscle physiology is being increasingly investigated, there are still differences in opinion and conflicting findings in the literature, particularly regarding its contribution to the process of muscle hypertrophy.

Several studies have shown that SIRT1 activation favors hypertrophy. Koltai et al. (2017) found that increased SIRT1 expression in overload-induced rat muscle was followed by activation of the Akt/mTOR pathway and a significant increase in muscle mass [12]. Similar findings were reported in studies by Woodman et al. and Bennett et al, which showed that resveratrol supplementation was able to improve muscle mass recovery as well as decrease the levels of inflammatory markers and tissue degeneration [15,16].

In contrast, other studies have reported that SIRT1 activation can inhibit anabolic pathways under certain conditions. Ghosh et al. reported that SIRT1 negatively regulates mTOR activity through AMPK activation, ultimately decreasing muscle protein synthesis. In a study of long-term caloric restriction, Myers et al. showed that increased SIRT1 expression correlated with metabolic maintenance and protection against oxidative stress but did not directly increase muscle protein synthesis or muscle mass [10].

This difference in results is most likely due to differences in the experimental context, among other factors:

1. Stimulus type and intensity (e.g. resistance vs resistance training) [5,6],
2. Duration and dose of SIRT1 activation, whether induced by bioactive compounds or physiological interventions such as exercise or caloric restriction [17,18],
3. Species models used, with mice often showing stronger anabolic responses than humans [25],
4. High cellular energy and redox status influence the direction of activation of the SIRT1 downstream pathway [18].

This suggests that SIRT1 activation cannot be considered a universal approach in hypertrophy induction but is highly dependent on the intensity, duration, and metabolic conditions under which activation occurs. Therefore, further studies are needed to evaluate the dose and time response of SIRT1 activation, as well as how this pathway interacts with other growth signals to maintain muscle mass under physiological and pathological conditions.

5. CONCLUSION

SIRT1's function in skeletal muscle hypertrophy is complex and significantly influenced by context. SIRT1 regulates various molecular pathways as a metabolic sensor and NAD^+-dependent deacetylase. It modulates anabolic signaling via context-specific interaction with the mTOR pathway, enhances mitochondrial biogenesis and energy efficiency through PGC-1α activation, suppresses muscle atrophy by deacetylating FOXO, inhibits proteolytic gene expression, and temporally regulates muscle regeneration through MyoD and myogenin. These mechanisms emphasize that SIRT1 functions not solely as a growth promoter or inhibitor but as an essential coordinator of cellular adaptation, energy homeostasis, and structural remodeling in response to diverse stimuli, including exercise and caloric restriction. Understanding the balance between its activating and inhibitory roles offers promising insights for developing targeted interventions to preserve or enhance muscle mass, particularly in aging or pathological conditions. Future research should seek to elucidate the threshold and duration of SIRT1 activation that maximally facilitates hypertrophy while preserving anabolic signaling..

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