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THE PROCESS AND DYNAMICS OF THE SCIENTIFIC PATH

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ПСИХОЛОГІЧНІ МЕХАНІЗМИ ЗАХИСТУ ЯК ЧИННИК СОМАТИЗАЦІЇ СТРЕСОВИХ СТАНІВ У ВІЙСЬКОВОСЛУЖБОВЦІВ
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Rodynskyi Roman
student at the Faculty of Medicine
Dnipro State Medical University, Ukraine

Scientific supervisor: Chernousova Natalia
Associate Professor, Candidate of Biological Sciences,
Department of Biochemistry and Medical Chemistry
Dnipro State Medical University, Ukraine

THE EFFECT OF SARMs ON MITOCHONDRIAL BIOENERGETICS IN SKELETAL MUSCLES

Introduction. Selective androgen receptor modulators (SARMs) are a class of compounds that bind to androgen receptors in a tissue-specific manner, exerting an anabolic effect in muscle tissue without significant androgenic effects on other tissues [1]. Skeletal muscle is the primary site of glucose uptake, fatty acid oxidation, and ATP turnover, accounting for up to 80% of insulin-stimulated glucose disposal in healthy individuals. Mitochondria are the central organelles that orchestrate these processes through oxidative phosphorylation (OXPHOS), the tricarboxylic acid (TCA) cycle, and mitochondrial fatty acid β-oxidation. In conditions such as sarcopenia, cancer cachexia, and type 2 diabetes, a decline in mitochondrial biogenesis, a drop in mitochondrial DNA (mtDNA) copy number, and excessive reactive oxygen species (ROS) production collectively impair muscle energy homeostasis and accelerate atrophy [7]. Because AR signalling is known to influence transcriptional networks that govern mitochondrial function, characterising the impact of SARMs on mitochondrial bioenergetics is of particular scientific and therapeutic importance. So bacsically the aim of this study was to evaluate the effects of SARMs on mitochondrial bioenergetics in skeletal muscle through a systematic analysis of peer-reviewed literature. Scientific materials were collected from authoritative databases and included recent original research articles, preclinical mechanistic studies, and clinical trials.

Restoration of mitochondrial fatty acid β-oxidation and metabolic flexibility. SARMs (such as LGD-4033, MK-2866, and RAD-140) significantly increase the expression of PGC-1α (a key coactivator of mitochondrial biogenesis) by activating androgen receptor-dependent signaling pathways: upon AR activation, downstream phosphorylation of AMP-activated protein kinase (AMPK) promotes the translocation of PGC-1α from the cytoplasm to the nucleus, where it co-activates nuclear respiratory factors 1 and 2 (NRF-1, NRF-2) and the mitochondrial transcription factor A (TFAM). These targets in turn stimulate transcription and

replication of the mitochondrial genome, driving expansion of the mitochondrial network [2]. This directly correlates with an increase in mitochondrial density and a 20-35% rise in the activity of complexes I and III of the respiratory chain compared to the control.

The AR–PGC-1α axis also engages the AMPK/SIRT1 pathway: SIRT1-mediated deacetylation of PGC-1α stabilises its active conformation, while PGC-1α itself induces expression of SIRT3, a mitochondrial deacetylase that activates key enzymes of the TCA cycle and OXPHOS complexes. Loss of AR signalling, conversely, suppresses this cascade; studies on skeletal-muscle-specific AR-knockout mice reveal impaired glycolytic flux, lipid accumulation in the cytoplasm, and a 30% rise in H_2O_2 levels, underscoring the indispensable role of androgen receptor activity in maintaining mitochondrial homeostasis.

Oxidative phosphorylation efficiency and redox balance.

Seahorse analysis (extracellular metabolic flux analysis) demonstrates an increase in basal and maximal oxygen consumption in myotubes treated with MK-2866, indicating improved oxidative phosphorylation [3]. At the same time, a decrease in the production of reactive oxygen species was observed due to increased superoxide dismutase-2 (SOD2) activity. In this way, simultaneously, SARM treatment attenuates mitochondrial ROS emission. This is mediated, in part, by PGC-1α-dependent induction of superoxide dismutase-2 (SOD2) - the primary mitochondrial matrix antioxidant enzyme - as well as the upregulation of SIRT3, which deacetylates and activates SOD2 post-translationally, further amplifying its catalytic efficiency [3]. Reduced superoxide levels protect against mitochondrial DNA oxidation, prevent opening of the mitochondrial permeability transition pore (mPTP), and maintain mitochondrial membrane potential ($\Delta\Psi m$), collectively contributing to the preservation of organelle integrity and function. The net effect is a bioenergetic environment favouring sustained aerobic ATP production rather than anaerobic glycolysis or amino acid catabolism.

In vivo and clinical evidence: sarcopenia models and human data.

In mouse models of sarcopenia, RAD-140 restored mitochondrial membrane potential and increased mitochondrial DNA content, which directly correlated with the restoration of muscle strength [4]. Phase I clinical data provide supporting, though preliminary, evidence in humans. In a randomised, double-blind, placebo-controlled trial involving 76 healthy men, LGD-4033 administered at doses of 0.1, 0.3, and 1.0 mg daily for 21 days produced dose-dependent increases in lean body mass with no clinically significant changes in liver enzymes (AST, ALT), prostate-specific antigen, or haematological parameters [5, 6]. A subsequent case report examining a 21-day course of LGD-4033 (10 mg/day) combined with MK-677 confirmed significant

gains in lean mass alongside a modest, reversible elevation in ALT (from 20 to 61 IU/L) and a reduction in high-density lipoprotein (HDL) cholesterol from 55 to 35 mg/dL, both of which normalised following cessation of SARM use [8]. Phase I clinical data in patients with muscular dystrophy also confirm a trend towards improvement in indirect markers of mitochondrial function, though the effect size and inter-individual variability necessitate larger controlled studies.

Conclusions. SARMs have a significant positive effect on mitochondrial bioenergetics in skeletal muscle by stimulating mitochondrial biogenesis via the AR-PGC-1α axis, increasing the efficiency of oxidative phosphorylation, and reducing oxidative stress. These effects are what make SARMs promising for the treatment of sarcopenia, cachexia, and metabolic disorders. However, large-scale randomized clinical trials are currently needed to confirm their long-term efficacy and safety (recent data suggest possible negative effects of RAD-140 on muscle adaptation in certain models, as well as risks of hepatotoxicity, reduced HDL, and other side effects).

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