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# Assessing Aerobic and Anaerobic Exercise as Therapies for Sarcopenia

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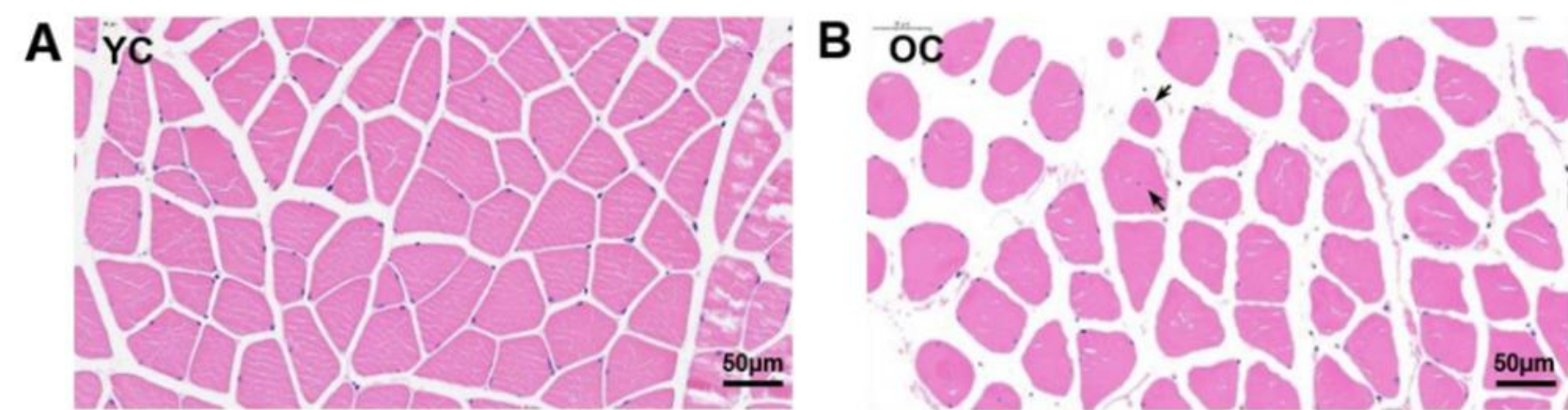
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## INTRODUCTION

Sarcopenia is an age-related disorder characterized by loss of skeletal muscle mass and function. This leads to reduced mobility, functional limitation, and mortality in older adults.<sup>1, 15</sup> Globally the condition impacts 5.7% to 33% of individuals over 65.<sup>19</sup> Various risk factors contribute to progression of the condition. Sarcopenia inhibits quality of life, making its mechanisms and treatments important to assess.



**Figure 1:** HE staining to represent (A) normal healthy muscle tissue and (B) untreated sarcopenic muscle tissue. (Modified from Liang et al., 2021)

### Research Questions:

**Q1:** How do aerobic and anaerobic exercise mitigate sarcopenia?

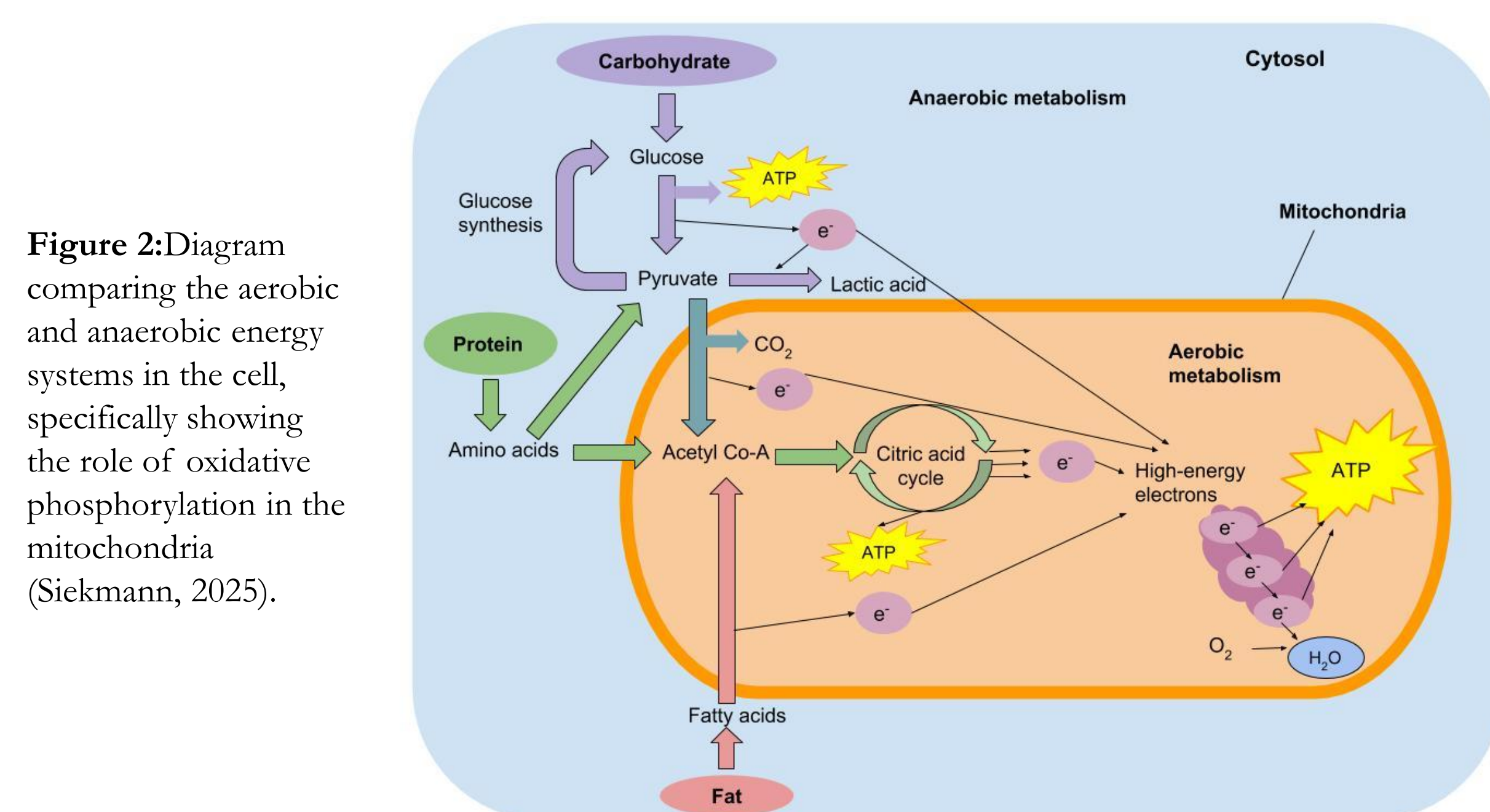
**Q2:** What protocol would provide the optimal intervention?

## MECHANISMS

- Mitochondrial dysfunction: Aging and reduced activity lead to declines in mitochondrial function, impaired metabolic pathways, increased free radical species, and less cellular energy.<sup>8</sup>
- Neuromuscular Junction (NMJ) degradation: Degradation results in reduced muscle-nerve communication which inhibits motor unit integrity and contributes to muscle deterioration.<sup>5, 10</sup>
- Oxidative stress: Oxidative stress increases with mitochondrial dysfunction allows for the generation of free radicals. Oxidative stress has been seen to promote NMJ deterioration and muscle atrophy, highlighting mitochondrial function as a key component of sarcopenia progression.<sup>17</sup>

## EXERCISE TYPES

Aerobic exercises are endurance activities that are oxygen-dependent to allow for oxidative phosphorylation to occur in the mitochondria to produce large amounts of ATP at a slower rate.<sup>3, 16</sup> Anaerobic exercises are high intensity activities where oxygen demand exceeds the supply. This system then is reliant on phosphagens and glycolysis for rapid ATP production, allowing for quick production in a shorter time span.<sup>3, 16</sup>



**Figure 2:** Diagram comparing the aerobic and anaerobic energy systems in the cell, specifically showing the role of oxidative phosphorylation in the mitochondria (Sickmann, 2025).

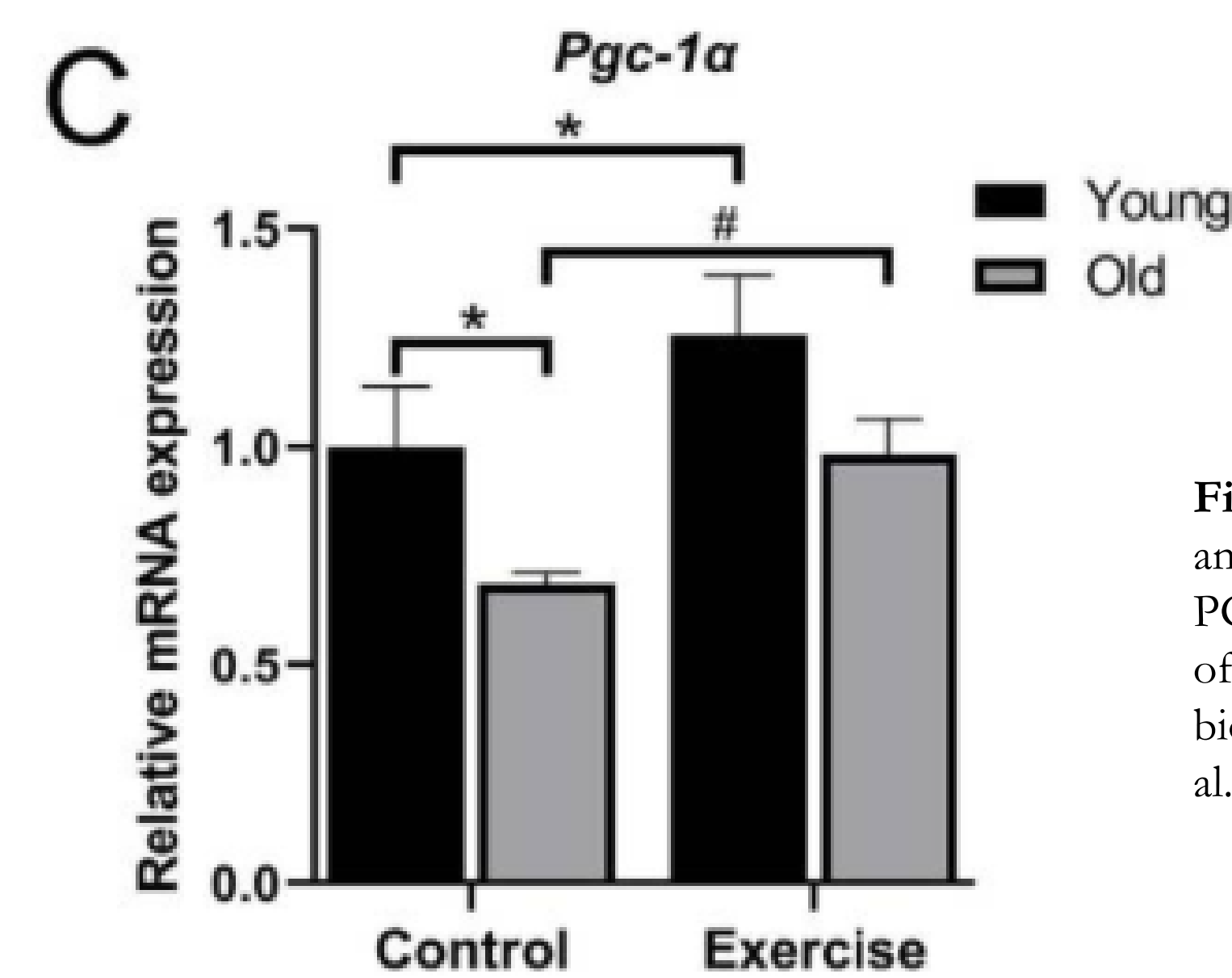
## AEROBIC EXERCISE

### Sesn2 Gene Expression

Aerobic exercise is intertwined with mitochondrial function through oxidative phosphorylation. Increased cardiovascular activity is linked to improved mitochondrial function. The *Sesn2* gene plays a role here. When *Sesn2* is upregulated, the gene supports mitochondrial quality control, protects against oxidative damage, and promotes metabolic health for activation of AMPK.<sup>7</sup> Dysfunctional mitochondria remain in higher amounts when *Sesn2* expression is downregulated.<sup>7</sup> *Sesn2* then functions to reduce inflammation and promote autophagy for mitochondrial and metabolic health.

### AMPK/PGC-1 $\alpha$ Pathway

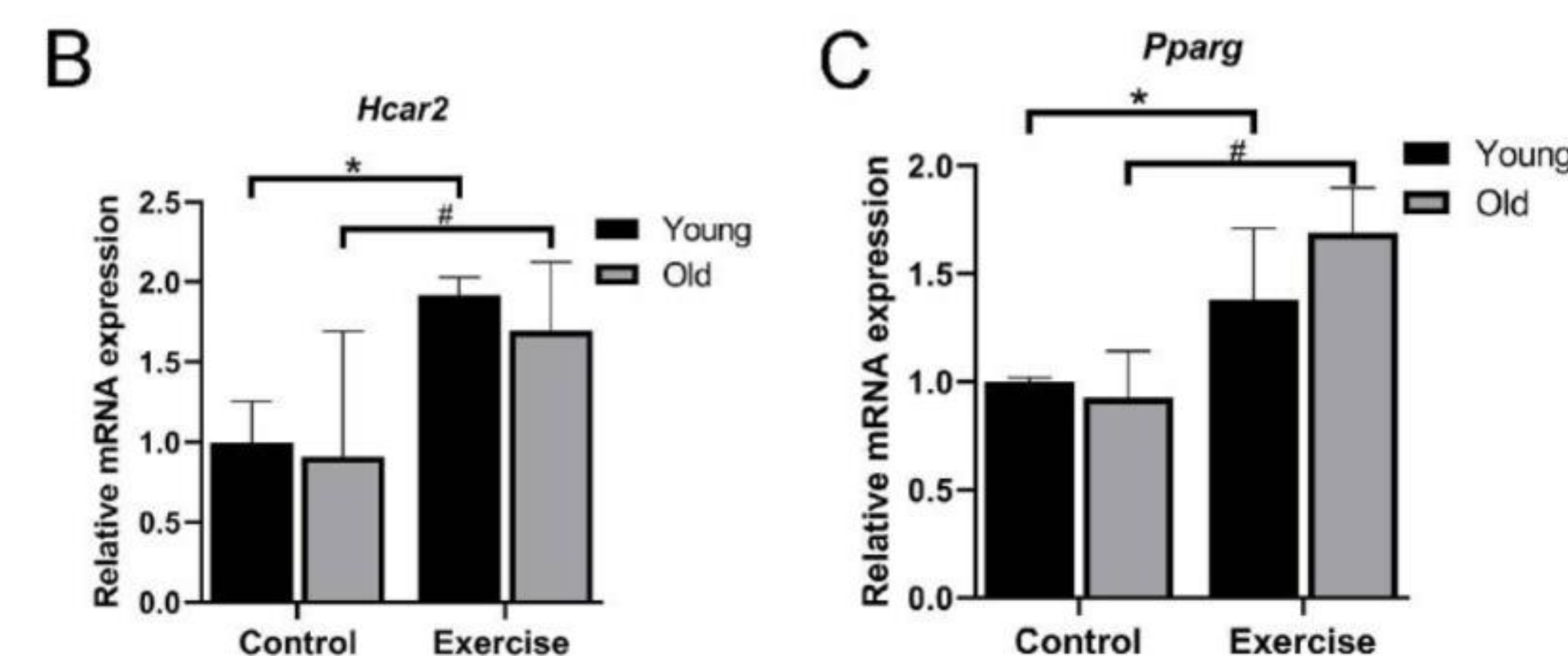
The AMPK/PGC-1 $\alpha$  signaling pathway functions by enhancing mitochondrial function and reducing muscle atrophy. AMPK stimulates PGC-1 $\alpha$  to upregulate mitochondrial synthesis while inhibiting atrophy-related factors like NF- $\kappa$ B, Atrogin-1, and MuRF1.<sup>18, 20</sup> The pathway also suppresses apoptosis to reduce muscle degradation.<sup>6</sup>



**Figure 3:** A comparison in young and aged mice to show the role of PGC-1 $\alpha$  in increasing expression of mRNA for mitochondrial biogenesis (modified from: Zhu et al., 2025).

### $\beta$ -HB/HCAR2-PPARG Pathway

$\beta$ -HB/HCAR2-PPARG pathway functions to mitigate sarcopenia through resistance to oxidative stress. Initiated by  $\beta$ -hydroxybutyrate, this pathway enhances mitochondrial function, reduces oxidative stress, and supports mitochondrial quality control.<sup>20</sup> Exercise-induced activation increases pathway-related mRNA and protein expression, improving muscle mass and function.<sup>20</sup>

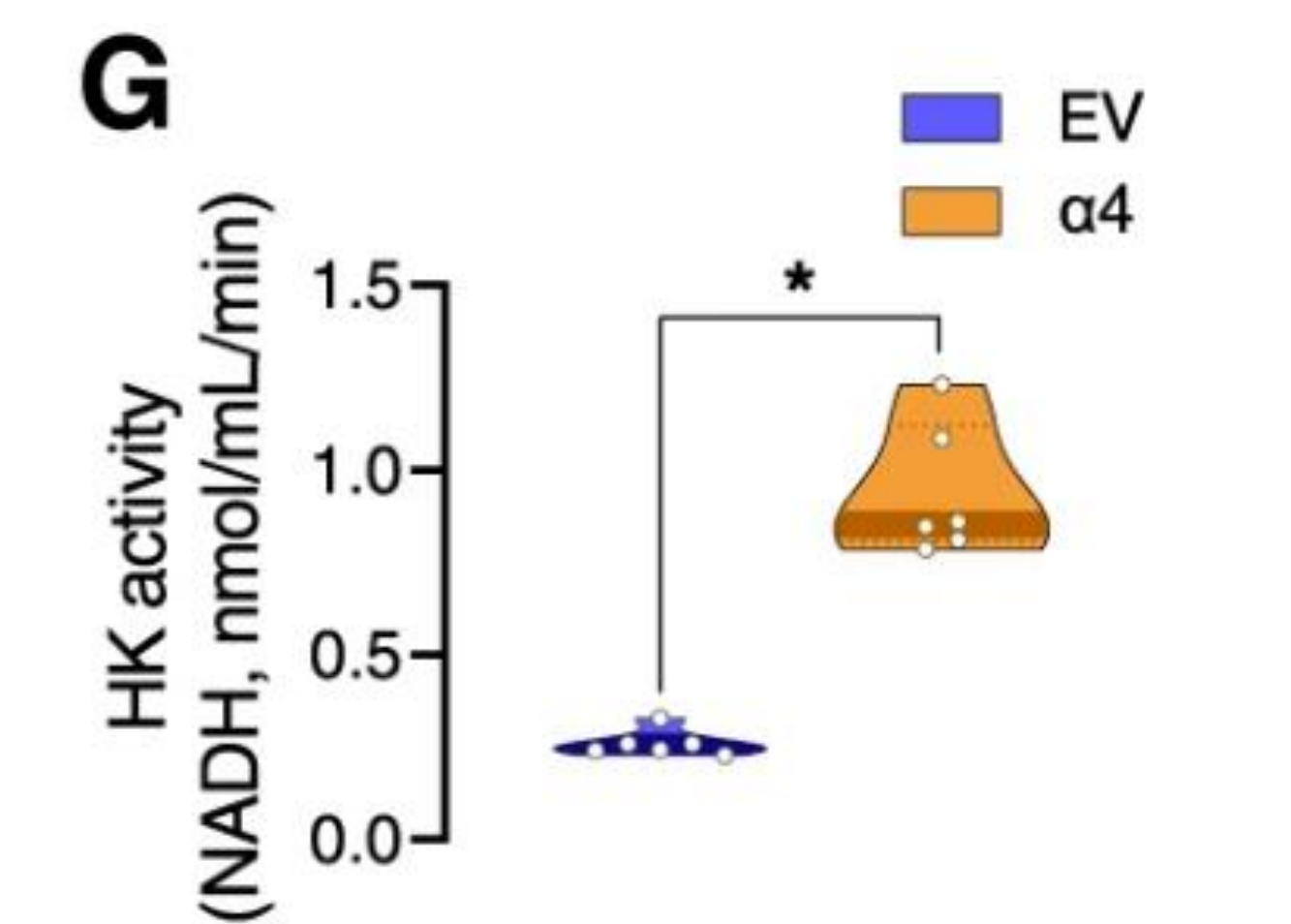


**Figure 4:** A comparison of young and aged mice with sarcopenia to show how aerobic exercise elevates the expression of mRNA associated with an upregulation of the  $\beta$ -HB/HCAR2/PPARG, specifically of HCAR2 and PPARG (modified from: Zhu et al., 2025)

## ANAEROBIC EXERCISE

### PGC-1 $\alpha$ and Metabolic Adaptation

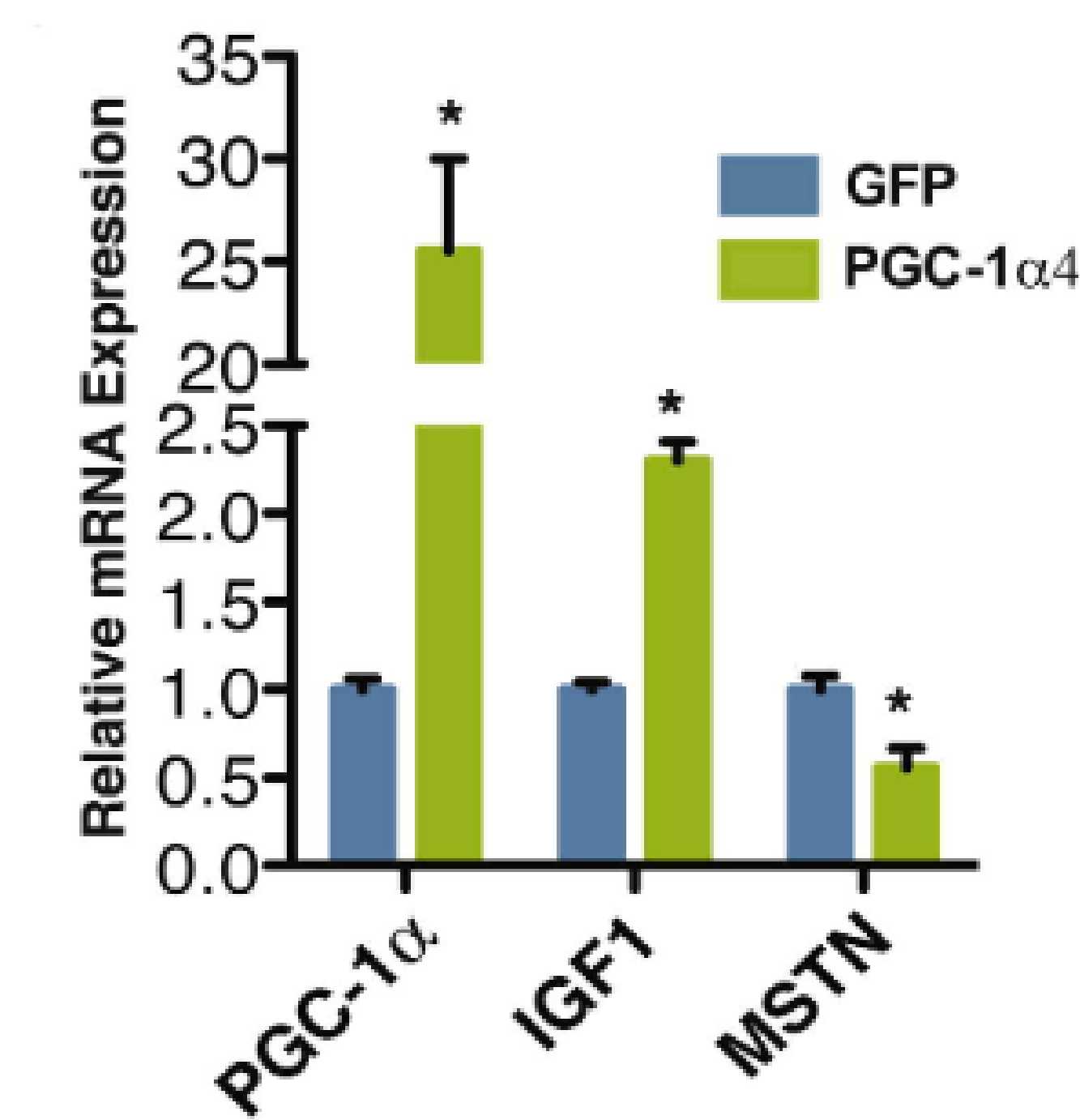
A PGC-1 $\alpha$  isoform PGC-1 $\alpha$ 4 has been found to play a role in resistance exercise. PPAR $\beta$  is stimulated by PGC-1 $\alpha$ 4 to improve anaerobic glycolysis.<sup>4</sup> This results in more effective uptake of glucose and fat oxidation in muscle, allowing for improved muscle function through increased metabolic efficiency and energy usage.



**Figure 5:** The effect of PGC-1 $\alpha$ 4 on increasing hexokinase (HK) activity to show how overexpressed PGC-1 $\alpha$ 4 enhances anaerobic glycolysis compared to an empty vessel control sample (modified from: Koh et al., 2022)

### Muscle Hypertrophy

The PGC-1 $\alpha$ 4 pathway directly counteracts sarcopenia through muscular hypertrophy. PGC-1 $\alpha$ 4 functions by increasing IGF-1 and suppressing myostatin to promote muscle growth and inhibit pathways of muscle loss.<sup>11</sup> Increased intensity levels of activity have been linked to increased IGF-1.<sup>13</sup> This pathway can also be stimulated through the AMPK/PGC-1 $\alpha$  pathway seen in aerobic exercise, and the functionality of the pathway is further amplified when anaerobic exercise is completed following aerobic exercise.<sup>9, 18</sup>



**Figure 6:** The effect of mRNA expression related to the PGC-1 $\alpha$  pathway, IGF-1, and myostatin in the presence of PGC-1 $\alpha$ 4 compared to a GFP control sample, showing that PGC-1 $\alpha$ 4 increases hypertrophy (modified by Ruas et al., 2012)

### NMJ Regeneration

Anaerobic exercise has been seen to beneficially act on NMJ function through enhanced muscle activation, which is linked to NMJ plasticity and maintenance.<sup>2</sup> Increased PGC-1 $\alpha$  with exercise has been shown to promote NMJ remodeling and neuromuscular communication.<sup>2</sup> Increased satellite cells and upregulation in proteins for muscle-nerve communication has also been associated with exercise.<sup>12</sup> These findings indicate that anaerobic exercise functions to improve the NMJ and contribute to regenerative capacities. However, further research is still necessary to determine the full impact.

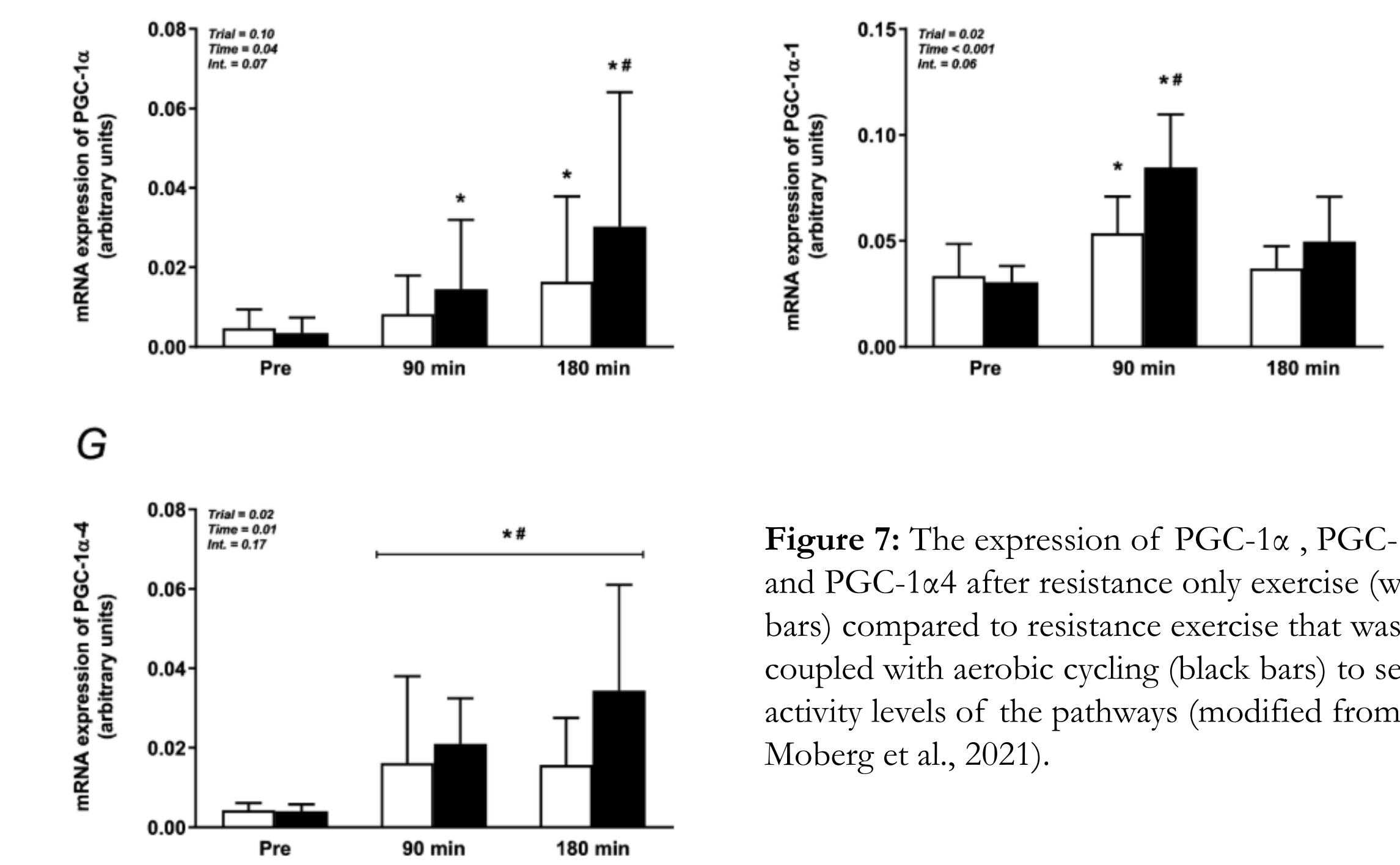
## DISCUSSION

### Q1: How do aerobic and anaerobic exercise mitigate sarcopenia?

Sarcopenia is driven by mitochondrial dysfunction and NMJ instability leading to progressive declines in muscle mass and function. Aerobic exercise functions by improving mitochondrial function and reducing oxidative stress through pathways such as AMPK/PGC-1 $\alpha$ , *Sesn2*, and  $\beta$ -HB/HCAR2-PPARG, while anaerobic exercise promotes hypertrophy via PGC-1 $\alpha$ 4 activation, increasing IGF-1 and suppressing myostatin. Aerobic exercise functions to improve the cellular environment and combat mitochondrial dysfunction and oxidative stress, while anaerobic exercise directly counteracts atrophy through the promotion of hypertrophy and combats NMJ failure.

### Q2: What protocol would provide the optimal intervention?

The mechanisms of aerobic and anaerobic exercise are distinct and complementary, indicating that neither exercise type alone fully addresses sarcopenia. Instead, an approach that utilizes both exercise types would maximize synergistic benefits by enhancing cellular health and restoring muscle mass. Evidence supports this in amplified benefits through the PGC-1 $\alpha$  pathway when a combined approach is involved.<sup>9, 18</sup>



**Figure 7:** The expression of PGC-1 $\alpha$ , PGC-1 $\alpha$ 1, and PGC-1 $\alpha$ 4 after resistance only exercise (white bars) compared to resistance exercise that was coupled with aerobic cycling (black bars) to see the activity levels of the pathways (modified from: Moberg et al., 2021).

## FUTURE RESEARCH DIRECTIONS

Future research should focus on optimizing exercise intensity, dosage, and the ratio of anaerobic to aerobic exercise. Gender-specific protocols should also be studied to maximize therapeutic outcomes. The mechanisms of NMJ regeneration should be further studied.

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