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Introduction

Through this project, we aimed to determine if repeated heat stress is a viable treatment to reduce or slow muscle atrophy in human muscle subjected to disuse. Specifically, I investigated how repeated heat stress affects the expression of PGC1-a, a gene that is an upstream regulator of mitochondrial biogenesis. This protein promotes muscle growth and normally is triggered by environmental factors including the metabolic needs (energy, temperature, stress, etc.) of the muscle. If it could be increased in the muscle, it could potentially lead to the preservation of muscle strength and function in a variety of disease states. These include cancer, injury, metabolic disease, diabetes, heart problems, intolerance to exercise etc.

Based on previous research (1), we expected that PGC1-a would be decreased during muscle disuse and subsequent atrophy (muscle wasting). We also hypothesized that repeated heat treatments would attenuate these reductions and promote PGC1-a levels, thus counteracting the normal atrophy process.

Methodology

24 young adults (12 male and 12 female) aged 18-39 years were included in this study. 6 males and 6 females were randomized into a heat treatment group (IMM+H) and 6 males and 5 females served as controls (IMM), and received a placebo treatment. The IMM+ H group underwent a 2-hour daily heating of the immobilized leg's vastus lateralis using a short-wave diathermy unit. This heated the muscle about 4 degrees Celsius, mimicking the increase in temperature experienced during exercise. (2) The control group had the diathermy unit placed on their legs, and were not told that it was not turned on. A temperature probe confirmed that they did not experience a change in internal muscle temperature. One subject chose to discontinue the study due to unexpected life events, which was the reason for one less female control. All participants were free of cardiovascular disease and metabolic disease and were recreationally active for at least 3 hours a week. All participants wore a brace that immobilized their left leg in order to induce atrophy for a consecutive 10-day period. The leg that was not immobilized served as a control for circumstantial, food, hormonal, or other variations within this time period. A biopsy was taken from the vastus lateralis muscle of both the immobilized and non-immobilized leg of each participant at the beginning and end of the 10-day immobilization period. Levels of PGC1-a proteins were measured in all biopsy samples via Western Blotting in order to determine changes in PGC1-a within the muscle following this immobilization period.

Results

PGC1a levels decreased in the immobilized control legs significantly. This was expected, and supported our hypothesis (See Figure 1). PGC1a levels also increased significantly in the IMM+H group that underwent diathermy heat treatment daily over the duration of the immobilization. Results were compared

between the immobilized and free-moving legs of each participant, as well as between those treated with a placebo or diathermy-heat treatment.

Discussion

The increase in PGC1a above baseline levels following heat treatment and concurrent immobilization demonstrated the potential for an even greater protective effect of heating against muscle atrophy than originally, we hypothesized. This suggests that short-wave diathermy heating not only prevents the reduction of mitochondrial biogenesis and PGC1a production, but also potentially increases it to higher levels than in comparable, untreated skeletal muscle. Further research will be needed to corroborate this relationship as well as determine the length and types of heating that result in PGC1a and mitochondrial preservation. Including a variety of heating modalities, lengths of treatment, participant characteristics (age, disease-state, activity-level) may also elaborate on these findings and provide a more complete picture of the applicability of heat treatment as a potential muscle-preservation therapy.

Conclusion

This experiment reveals a potential connection between heat-stress and mitochondrial number and function. (Mitochondrial number and function being positively influenced, and managed by PGC1a). By exploring this relationship in a living, human model, greater conclusions can be drawn as to the application of the relationship between short-wave diathermy heating and muscle preservation in humans. These findings could help lead to a therapeutic process that can be applied to patients suffering from atrophy as a result of a wide range of illness. This close examination of the response of PGC1-a in living human muscle tissue illuminates some important discrepancies in research, and provides a more solid understanding of the signaling pathways that lead to the metabolic adaptations and mitochondrial biogenesis seen as a result of heat stress on muscle fibers.

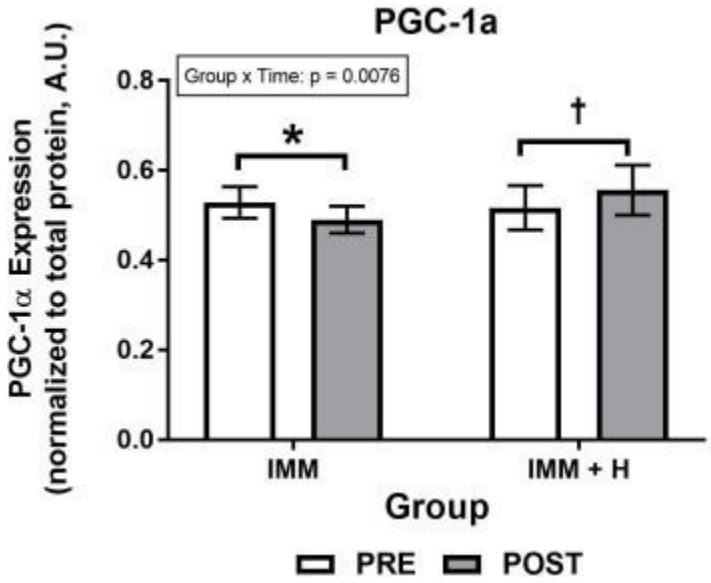

Figure 1 – The change in PGC1-a levels as seen before and after single-leg immobilization in human subjects (IMM) for 10 days with *heat treatment (IMM+H) and without heat treatment (IMM). PGC1a protein levels were obtained from muscle biopsies of the immobilized vastus lateralis, later quantified via the western blot procedure. Values are expressed in arbitrary units normalized to total protein. An average 9.3% increase in PGC1-a was seen following Immobilization plus heat treatment from pre to post biopsy measures (p value was 0.033), and an average 8% decrease in PGC1-a was seen following Immobilization without heat treatment during the same time period (p-value was 0.035).

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