ABSTRACT

BACKGROUND: Aerobic exercise triggers mitochondrial biogenesis, the generation of new mitochondria, through signaling pathways which lead to enhanced expression of PGC-1α. Previous research from our lab has shown that exercise followed by cold recovery enhances the transcription of genes associated with mitochondrial growth and division, however the recovery period was necessary for this response. Perhaps exercising in a colder external environment would induce a greater thermoregulatory response that may lead to altered mitochondrial turnover. These data may help exercise-induced mitochondrial biogenesis response and thus aid in the development of temperature-optimized training protocols to combat mitochondrial dysfunction. PURPOSE: To examine the mRNA expression of PGC-1α and other select genes related to mitochondrial biogenesis after exercise in a cold environmental temperature compared to exercise in room temperature. METHODS: Eleven recreationally trained males cycled at 65% Wpeak for an hour at -2°C and 20°C in a random, counterbalanced order. A muscle biopsy was taken from the vastus lateralis pre-exercise as well as 3-h and 6-h post-exercise for analysis of genes associated with mitochondrial biogenesis (PGC-1α, GABPA, ERRα, NRF1, TFAM, and VEGF). RESULTS: VEGF and PGC-1α increased with exercise and remained elevated compared to pre-exercise a both post-exercise time points (P < 0.05). NRF1 was lower after cycling regardless of temperature (P = 0.005, P < 0.001, respectively). GABPA and ERRα decreased 3-h post-exercise (P < 0.032, P > 0.003, respectively) and were elevated at 6-h post exercise compared to 3-h post-exercise (P < 0.017, P < 0.005, respectively). TFAM expression was increased at 6-h post-exercise compared to both pre-exercise and 3-h post-exercise levels (P < 0.021, P < 0.001, respectively). CONCLUSION: Gene expression related to mitochondrial biogenesis is altered after exercise with no difference between trials at -2°C and 20°C.

INTRODUCTION

• Aerobic exercise triggers mitochondrial biogenesis, the generation of new mitochondria, by ATP depletion and subsequent activation of the AMPK signaling pathway.
• This leads to enhanced expression of PGC-1α, which has been termed the master regulator of mitochondrial biogenesis.
• Previous research from our lab has shown that exercise followed by cold recovery enhances the transcription of genes associated with mitochondrial growth and division, however the recovery period was necessary for this differential response.
• Colder temperatures may lead an alteration in core body temperature and be a stimulus for greater activation of PGC-1α, and thus an increased amount of exercise-induced mitochondrial biogenesis.

METHODS

• 11 recreationally active males between 19 and 45 biked for 60 minutes at 65% VO2peak in a repeated measures, randomized, counterbalanced order at -2°C and 20°C.
• Heart rate, core temperature, skin temperature, and rating of perceived exertion were measured during exercise trials.
• Muscle samples were taken from the vastus lateralis prior to exercise as well as 3-h and 6-h into recovery.
• mRNA analysis of genes associated with mitochondrial biogenesis (PGC-1α, GABPA, ERRα, NRF1, TFAM, and VEGF) were measured using qRT-PCR normalized using 2-ΔΔCT method.
• Differences were analyzed via repeated measures two-way ANOVA (time x trial).
• Variance was determined using Fishers protected least significant difference method. Data indicating a probability of less than 5% for type I error (p < 0.05) will be deemed significant.

RESULTS

Table 1. Subject characteristics. Data are means ± SE (n = 11).

Table 2. Study parameters measured during exercise. Data are means ± SE (n = 11).

CONCLUSIONS

• No significant temperature differences occurred in gene expression related to mitochondrial biogenesis were detected after exercise in temperatures of -2°C and 20°C.
• Select genes related to mitochondrial biogenesis (PGC-1α and VEGF) were amplified after exercise (3h and 6h) while others had a differential response (ERRα, GABPA, and TFAM).
• The development of training protocols and therapies specific to greater induction of mitochondrial biogenesis may help combat mitochondrial dysfunction in humans.

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