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Autor(es): Barcellos, Luiz Alexandre Medrado de; Rezende, Barbara Maximino; Gonçalves, William Antônio; Costa, Walyson Coelho; Coimbra, Cândido Celso; Silva, Albená Nunes; Pinho, Vanessa

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THE ROLE OF INFLAMMATION IN PHYSICAL TRAINING INDUCED SKELETAL MUSCLE REMODELING: THE REACTIVE OXYGEN SPECIES (ROS) PARTICIPATION

Luiz Alexandre Medrado de Barcellos¹; Barbara Maximino Rezende¹; William Antônio Gonçalves¹; Walyson Coelho Costa¹; Cândido Celso Coimbra¹; Albená Nunes Silva²; Vanessa Pinho¹

KEYWORDS: Exercise, physical training, inflammation, ROS production.

Exercise is known by some types of activity including sports and conditioning associated with planned, structured and repetitive movement of skeletal muscle and energy expenditure. Exercise is represented by a potential homeostasis disruption by muscle activity and exercise training has been known to bring about multiple benefits to human health and sport performance, and their maintenance and/or improvement. However, it has been shown that physical exercise induces skeletal muscle damage leading to low-grade local inflammatory response. The inflammation response in skeletal muscle after exercise is still poorly known. Our hypothesis is that inflammatory response may contribute to remodeling and performance improvement of skeletal muscle. Previous data from our group showed that fatiguing exercise might cause ROS dependent neutrophil accumulation in quadriceps muscle. However, the role of neutrophil accumulation and ROS production to skeletal muscle remodeling following chronic exercise are still unknown. Thus, the aim of this study is to evaluate the role of exercise induced inflammatory response in the remodeling of skeletal muscle tissue.

METHODS

C57 mice or Gp91phox deficient were accustomed to a treadmill exercise. After that, the maximal speed capacity was measured by a progressive running until fatigue test (PFT).

¹ Universidade Federal de Minas Gerais.

² Universidade Federal de Ouro Preto.

Email: albenanunes@hotmail.com; barcelloslam@gmail.com

After 3 days, the animals were submitted to a continuous running until fatigue test (CFT) to evaluate the maximal running capacity. Following that, the animals were divided in 4 groups non-trained wild type (nt-wt) and Gp91phox^{-/-} (nt-ko) and trained wild-type (t-wt) and Gp91phox^{-/-} (t-ko), that latter performed 4 weeks of training which included five exercise sessions per week, where the exercise intensity and duration were manipulated based on CFT. 3 days after the end of the training, a second CFT was carried out, which was followed by intravital microscopy of the rectus femoris muscle analysis 18 hours later.

RESULTS

Here, the physical training was able to increase the animals exercise workload. Interestingly, the trained gp91phox knockout workload was also higher than the trained wild-type workload. Our results also show that the number of rolling cells in the muscle vascular endothelium in trained wild-type mice was higher than non-trained 18 hours after CFT. However, there was no significant difference when compared the gp91phox knockout trained and non-trained groups.

CONCLUSION

These data suggest that the leucocyte-endothelium interaction induced by exercise is workload dependent, but this relation is disrupted by ROS production deficiency.