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Caffeine supplementation changes inflammatory biomarkers after exercise

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Abstract

Caffeine is widely consumed by the world population due is a major constituent of beverages like as coffee and tea. Additionally, caffeine supplementation is also taken by many athletes due its ergogenic properties. Thus, several studies demonstrated that caffeine supplementation have significant antioxidant and anti-inflammatory activity. Our study evaluated the caffeine intake associated with swimming exercise in rats. We found that caffeine and exercise training did not change the mitochondrial parameters as compared with control group. However, our results demonstrated that caffeine intake are highly efficacious in modulate inflammation biomarkers.

Keywords: Caffeine; Anti-inflammatory; Antioxidant; Mitochondria

Introduction

Caffeine is a methylxanthine (1,3,7-trimethyxanthine) that is highly consumed by peoplebecause is a key component of beverages such as coffee and tea [1,2].

In addition, caffeine presents *in vitro* antioxidant activity, presenting effectiveness in scavenging mainly hydroxyl radicals [3,4]. *In vivo*, we have proved that caffeine intake has been associated with beneficial antioxidant effects in liver, such as decreased lipid peroxidation and elevated superoxide dismutase and glutathione peroxidase enzymes activity [5]. Recent studies have also been shown that caffeine intake prevent oxidative stress in neurodegenerative diseases and was beneficial effects against inflammatory injury [6-9]. In addition, clinical study demonstrated that coffee consumption decreased significantly the serum concentration of proinflammatory immune mediators [10].

Our study demonstrated that caffeine promote a significant decrease in myeloperoxidase (MPO) activity, indicating that caffeine supplementation act as anti-inflammatory, probably by reducing the neutrophil infiltration and its inflammatory mechanisms activation [11]. Besides, the remarkable decrease in acetylcholinesterase (AChE) activity corroborated to the anti-inflammatory effect, since this inhibition leads to an increase in acetylcholine levels, which is linked to anti-inflammatory functions and suppressed pro-inflammatory cytokines production [12-15]. Interestingly, most of these anti-inflammatory caffeine results are not associated with exercise training, but to an effect of caffeine *per se*.

Furthermore, caffeine supplementation was massive used by athletes during training or competitions [16]. Thus, several studies have been evaluating the mechanism of action of caffeine association with exercise training [17,18].

During the last years a consensus has emerged among sports scientists: ROS and inflammation are determinant to muscle cell regeneration and differentiation/proliferation, and the use of antioxidants and nonsteroidal anti-inflammatory drugs (NSAIDs) may blunt important these important exercise adaptations on young healthy subjects [19,20]. Therefore, drugs that main help on soften oxidative stress and inflammation after an exhaustive exercise should be prefer when comparing to the ones that blunt or completely block these necessary adaptive pathways of skeletal muscle regeneration. Among the natural compounds consumed on a daily basis, creatine (Cr, N-[aminoiminomethyl]-N-methyl glycine) and caffeine consumed by athletes [21,22]. Cr is among the most consumed ergogenic aids by athletes mainly due to its positive effects on fatigue resistance, and improved exercise recovery after an exhaustive exercise [23]. On the same way, caffeine is considered a ergogenic compound due to its antioxidant and antiflammatory role. These effects were proved by our previous study as it is described here [5,11].

Original Study

Our study examined the effect of chronic caffeine intake and exercise training in male Wistar rats. The animals were divided into four groups (control, control/caffeine, exercise, and exercise/caffeine). Animals from Control and Exercise groups received saline, while groups Control/Caffeine and Exercise/Caffeine received caffeine supplementation (6 mg/kg/day) during the training protocol [24]. The exercise chosen was the swimming training, which consists of four weeks of swimming, 50 min per day and five sessions per week [25]. Our data identified that caffeine supplementation, associated or not with exercise training, did not promote any hepatic morphological or functional mitochondrial changes, maintaining a similar mitochondrial membrane potential, swelling, ROS production and mitochondrial complex I activity between groups. Nonetheless, both plasma myeloperoxidase and AChE activities were altered by the chronic caffeine intake (Figure 1). We attribute this decrease in MPO and AChE activity to caffeine anti-inflammatory effect, which is a view shared by other authors [7,26].

Final Considerations

In conclusion, we can observe that even though caffeine antioxidant properties, its intake did not modulate significantly the mitochondrial ROS production. In addition, we also verified that exercise training also did not change the liver mitochondrial parameters, indicating that the rats were well conditioned to swimming training protocol. It should be notice that caffeine intake did not interfered in this training adaptation.

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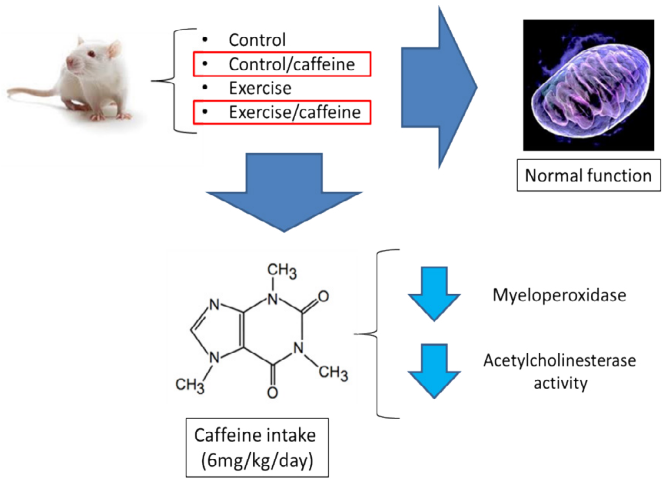

Figure 1: Illustrative diagram of the experimental protocol and main results.

Additionally, it is well known that exercise training produce *per se* an inflammatory process before the adaptation cycle. Thus, our study provided evidences indicating that caffeine supplementation can diminish this inflammatory process decreasing MPO and AChE activity.

Therefore, exercise training and caffeine supplementation may be a viable and effective strategy to diminish the side effects of inflammatory process. If this is a good or a bad effect in medium and long-term for training adaptation and health we cannot be sure right now. The research to find metabolic-related molecules able to modulate the oxidative-inflammatory metabolism but on the same way allow muscle cell differentiation/proliferation is of clinical interest, considering that different muscle disorder patients will benefit from this information.

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