Convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes (CETS # 123). The results showed that NLRP3$^{-/-}$ mice behaved better than WT mice at the same age in terms of resting time, maximal speed, running distance, and time to exhaustion. In both animal models, administration of melatonin significantly improved all parameters of muscle efficiency, even above the performances measured in 3 months old mice. It is concluded that NLRP3 exerts a type of brake in the motor activity of mice, which may have important consequences when it is activated during inflammaging. Moreover, we also concluded the high efficacy of melatonin to improve skeletal muscle performance. Both findings can have clinical interest in preventing sarcopenia associated to frailty in aged people.
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Aging, sarcopenia, inflammaging, innate immunity, melatonin.

O1-07

NLRP3 INFLAMMASOME AFFECTS MITOCHONDRIAL MORPHOLOGY IN SKELETAL MUSCLE OF MICE

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Pathogens in vertebrates are recognized and eliminated by both innate and adaptive immune systems. Once the former one is activated, it can sense a wide range of pathogens through pattern-recognition receptors (PRRS). Nod-like receptors (NLRs) are a family of PRRs that expressed in cytosol, and recognize intracellular pathogen and danger-associated molecular patterns (PAMPs and DAMPs). Certain NLRs including NLRP3 form large cytoplasmic complexes, termed inflammasomes, which intern activate caspase cascades resulting in proteolytic activation of proinflammatory cytokines. Interestingly, innate immune responses are regulated by mitochondria through reactive oxygen species (ROS)-dependent activation of NLRP3 inflammasomes. To investigate the effect of NLRP3 inflammasome on the morphology and ultrastructure of the gastrocnemius muscle and mitochondria, 3 months-old wild type (WT) and NLRP3 deficient (NLRP3$^{-/-}$) mice, were studied. All experiments were conducted in accordance with the University of Granada's Ethical Committee; the Spanish Protection Guide for Animal Experimentation (R.D. 53/2013), and the European Convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes (CETS # 123). Animals were anaesthetized by intraperitoneal injection ofequithesin, transcardially perfused by trump's fixative, and then the gastrocnemius muscle was dissected and processed for light and transmission electron microscopical (TEM) analysis. Different morphometrical measurements were studied on the acquired digital images. The results showed that gastrocnemius muscle fibers of NLRP3$^{-/-}$ mice had larger cross section area (CSA) than that of the wild type mice at the same age, with the fibers closed to each other and showed less collagenous connective tissue infiltrations in epimysium and endomysium. WT mice revealed few small sized vacuolated mitochondria with damaged cristae, In contrast, NLRP3$^{-/-}$ mice revealed a significant increase in the number of intermyofibrillar (IMF), and CSA, perimeter and diameter of both IMF and subsarcolemmal (SS) mitochondria with numerous cristae, while number of SS mitochondria was significantly reduced. No significant changes in the length of sarcomeres and A-, I-, and H-bands were detected. The results here reported indicate that NLRP3 inflammasome may affect negatively mitochondrial morphology, leading to their dysfunction and damage after its activation by inflammatory signals. These changes may reflect on the efficacy of the skeletal muscle and ultimately loss of its strength.
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CSA, Mitochondria, Muscle fibers, NLRP3 inflammasome.

O1-08

STUDY OF ULTRAVIOLET RADIATION EFFECT ON FIBROBLASTS AND QUERCETIN PROTECTION

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Summary: Most solar adverse effects are due to the action of ultraviolet radiation (UV) on the skin, causing cellular apoptosis and necrosis, skin redness and ocular inflammations. Actual protection against these forms of radiation uses external barriers, such as solar filters or photo blocks, so that achieving effects at a cellular level would multiply this protection without blocking the physiological functions of solar radiation.
Since one of the sources of this cell death originates in free radicals and since quercetin flavonoid is an excellent free radicals scavenger, this research is aimed at quantifying the possible protective effects of flavonoid against the damage produced by ultraviolet radiations.
Methods: The study model is human fibroblast primary cell culture. These model has been used for mechanism of action radiations study and for quercetin treatment and irradiation study.
Oxidative stress and the different cells deaths determinations were performed by image cytometry (ImageStream X, Amnis) with cFDA (Carboxyfluerescein diacetate)/IP(Propidium iodide), HE (Hidroetidine), DHR123(Dihydrorhodamine 123), ADPA (Anthracenedipropionic acid) and Annexin V-IP Kit.
Results: UV DL_{50} (lethal dose 50 %) was determined of 6.97 J/cm^2 and 1.269 J/cm^2 for UVB and UVC respectively with a significant increase of superoxide anion, hydrogen peroxide and oxygen singlet. The most part of the damage was observed in early apoptosis for UVB and late apoptosis for UVC.
Also the results showed how very low doses of quercetin (1–5 μM) proved able to reduce the damage produced by ultraviolet radiation (±60 % UVB and ±25 % UVC), decreasing the mortality, and mostly reducing the apoptosis. Damaging species such as superoxide radical, singlet oxygen and peroxide hydrogen were also reduced (until 90 %).