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P-39: Effects of physical exercise training in DNA damage and repair - could the difference be in hOGG1 Ser326Cys polymorphism?

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Acute physical exercise is associated with increased oxygen consumption, which could result in an increased formation of reactive oxygen species (ROS). ROS can react with several organic structures, namely DNA, causing strand breaks and a variety of modified bases in DNA. Physical exercise training seems to decrease the incidence of oxidative stress-associated diseases, and is considered as a key component of a healthy lifestyle. This is a result of exercise-induced adaptation, which has been associated with the possible increase in antioxidant activity and in oxidative damage repair enzymes, leading to an improved physiological function and enhanced resistance to oxidative stress (Radak et al. 2008). Human 8-oxoguanine DNA glycosylase 1 (hOGG1) is involved in the base excision repair (BER) pathway and encodes an enzyme responsible for removing the most common product of oxidative damage in DNA, 8-hydroxyguanine (8-OH-G). The genetic polymorphism of hOGG1 at codon 326 results in a serine (Ser) to cysteine (Cys) amino acid substitution (Ser326Cys). It has been suggested that the carriers of at least one hOGG1Cys variant allele exhibit lower 8-OH-G excision activity than the wild-type (Wilson et al. 2011). The aim of this study was to investigate the possible influence of hOGG1 Ser326Cys polymorphism on DNA damage and repair activity in response to 16 weeks of combined physical exercise training, in thirty healthy Caucasian men. Comet assay was carried out using peripheral blood lymphocytes and enabled the evaluation of DNA damage, both strand breaks and FPG-sensitive sites, and DNA repair activity. Genotypes were determined by PCR-RFLP analysis. The subjects with Ser/Ser genotype were considered as wild-type group (n=20), Ser/Cys and Cys/Cys genotype were analyzed together as mutant group (n=10). Regarding differences between pre and post-training in the wild-type group, the results showed a significant decrease in DNA strand breaks (DNA SBs) ($p=0.002$) and also in FPG-sensitive sites ($p=0.017$). No significant differences were observed in weight ($p=0.389$) and in lipid peroxidation (MDA) ($p=0.102$). A significant increase in total antioxidant capacity (evaluated by ABTS) was observed ($p=0.010$). Regarding mutant group, the results showed a significant decrease in DNA SBs ($p=0.008$) and in weight ($p=0.028$). No significant differences were observed in FPG-sensitive sites ($p=0.916$), in ABTS ($p=0.074$) and in MDA ($p=0.086$). No significant changes in DNA repair activity were observed in both genotype groups. This preliminary study suggests the possibility

of different responses in DNA damage to physical exercise training, considering the hOGG1 Ser326Cys polymorphism.

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