ABSTRACT

Regular exercising is physically strenuous requiring excessive oxygen uptake which can result into oxidative stress and genetic damage. DNA repair genes and metabolic/antioxidant genotypes can modulate the genetic damage. In the present case-control study (n=400) as a first of its kind, therefore primary genetic and oxidative DNA damage were assessed in peripheral blood leukocytes of players and in healthy controls (doing no strenuous activity) using the Single Cell Gel Electrophoresis (SCGE) and enzymatically-modified assays. Genetic polymorphisms of glutathione S-transferases (GSTT1, GSTM1, GSTP1 A313G- (rs1695)) and superoxide dismutase 2, mitochondrial [SOD2 C47T- (rs4880), SOD2 C399T- (rs1141718)] were examined using multiplex PCR and PCR-RFLP methods for modulation of genetic damage. Lipid levels and oxidative stress biomarkers were assessed spectrophotometrically. The study was conducted after approval from the Institutional Ethics Committee and voluntary written consent of the participants.

Study participants comprised university- and college- level students (aged 18-25y) whose demographic details, anthropometric measurements and sports’ specific details were recorded on a pre-designed questionnaire. Each participant performed the step-bench test to calculate the maximal oxygen uptake (VO2max) for aerobic capacity. The study group matched for age, gender, body mass index, fat-free mass and waist-height-ratio (WHtR) but not for waist circumference (WC), waist-hip-ratio (WHR), non-vegetarian diet, higher juice-intake and aerobic capacities. The sports group included 89% state- and 11% national-level players with average sports age of 6.02±0.22y, 15-30min daily warming-up time and 2-6h/day of regular training and comprised handball, hockey, baseball-softball players and athletes. Handball, hockey and athletics are categorized as high-dynamic, low-static sports whereas baseball and softball are moderate-dynamic, moderate-static sports.

The alkaline SCGE assay in players revealed significantly increased (p<0.000) DNA damage (per cent tail DNA, tail moment, Olive tail moment, Damage Index and Damage Frequency) compared to the values in controls. Male players had more genetic damage than female players, so data on males and females were not pooled and were
analyzed separately. Total oxidized purines and pyrimidines were significantly higher (p<0.000) in players, with males having significantly higher damage than females probably from excessive oxidative stress from uptake of oxygen. Levels of malondialdehyde, total oxidant status, oxidative stress index and total antioxidant capacity also showed significant increase in players while superoxide dismutase levels were similar in both groups. In players, significantly (p=0.000) increased high density lipoprotein (HDL-C), decreased low density lipoprotein (LDL), very low density lipoprotein (VLDL) and lipid ratios (TC/HDL-C, LDL/HDL-C, TG/HDL-C) were observed with more values in male players. On stratification for aerobic capacity, sports age, training duration, obesity, genotypic distribution and population sub-groups there were no differences for genetic damage, oxidative DNA damage, oxidative stress and lipidemia. However, hockey players had highest genetic damage and MDA levels followed by that in runners, baseball-softball and handball players.

Genetic variants were in Hardy-Weinberg equilibrium and distribution of GSTT1, GSTM1, GSTP1 (A313G- rs1695) and SOD2 (C47T- rs4880) and (C399T-rs1141718) genotypes and allelic frequencies were not statistically different between the player and control groups. Genetic damage was elevated in subjects with GSTP1 (A313G) homozygous (GG) variant compared to that in those with AA and AG genotypes in players and in the total participants, and the combinational analysis revealed different results for genetic damage for different combinations.

The predictors of genetic damage were sports age, warm-up time, aerobic capacity, fat-free mass, BMI, WHR, WHtR, mobile phone usage, diet, socioeconomic status, TAC, GSTT1 and SOD2 (C47T).

In conclusion, significantly increased genetic damage, oxidative DNA damage and oxidative stress were observed in sports persons in comparison to the controls probably associated with the strenuous physical activity in players. Male compared to female players had increased genetic damage. Hockey players had the highest genetic damage followed by runners, baseball-softball and handball players. The results imply that genetic polymorphism of GSTP1 can be determining factors for DNA damage provoked by strenuous physical activity in the player group of the present study.