J. Phys. Ther. Sci.
26: 825–829, 2014

Original Article

Tai Chi Improves Oxidative Stress Response and DNA Damage/Repair in Young Sedentary Females

Xing-Yu Huang, PhD[1–3], Wichai Eungpinichpong, PhD[1, 3)*], Atit Silsirivanit, PhD[4)], Saowanee Nakmareong, PhD[1)], Xiu-Hua Wu MS[5)]

[1)] *Department of Physical Therapy, Faculty of Associated Medical Sciences, Khon Kaen University: Khon Kaen 4002, Thailand*
[2)] *School of Physical Education, Gannan Normal University, China*
[3)] *Back, Neck and Other Joint Pain Research Group, Khon Kaen University, Thailand*
[4)] *Department of Biochemistry, Faculty of Medicine, Khon Kaen University, Thailand*
[5)] *School of Science and Technology, Gannan Normal University, China*

Abstract. [Purpose] This study was to examine the effects of 12 weeks of Tai Chi (TC) exercise on antioxidant capacity, and DNA damage/repair in young females who did not perform regular physical exercise. [Subjects and Methods] Ten female students from a Chinese university voluntarily participated in this program. All of them practiced the 24-form simplified Tai Chi, 5 times weekly, for 12 weeks. Plasma levels of superoxide dismutase (SOD), glutathione peroxidase (GPx), malondialdehyde (MDA), glutathione (GSH), hydroxyl radical inhibiting capacity (OH·-IC), 8-hydroxy-2'-deoxyguanosine (8-OHdG), and 8-oxoguanine DNA glycosylase (OGG1) were measured at 0, 8, and 12 weeks. Heart rate (HR) was monitored during the last set of the training session at 4, 8, and 12 weeks. [Results] Plasma SOD and OH·-IC levels were increased at 8 and 12 weeks compared to the baseline (0 weeks). Gpx and GSH levels did not change significantly throughout the study period. The plasma MDA level was decreased significantly at 8 weeks but not at 12 weeks compared to the baseline value. While the plasma 8-OHdG level did not change throughout the study period, the plasma OGG1 level was significantly increased at 8 and 12 weeks compared to the baseline value. [Conclusion] TC practice for 12 weeks efficiently improved the oxidative stress response in young females who did not perform regular physical exercise. The TC exercise also increased the DNA repairing capacity.

Key words: Tai Chi, Oxidative stress, DNA damage

(This article was submitted Nov. 8, 2013, and was accepted Jan. 7, 2014)

INTRODUCTION

Oxidative stress refers to an imbalance between free radical [especially reactive oxygen species (ROS)] generation and antioxidant defense[1)]. The antioxidant system in the human body includes enzymatic [e.g. superoxide dismutase (SOD) and glutathione peroxidase (GPx)] and non-enzymatic antioxidants [e.g. glutathione (GSH)] and it plays an important role in the prevention of oxidative stress. Oxidative stress is involved in the pathogenesis of hypertension, atherosclerosis, diabetes, osteoporosis, and cancer[2)]. It is also known to cause and accelerate the aging process[3)]. ROS initiates lipid peroxidation through an attack on polyunsaturated fatty acids, and generates products such as MDA (malondialdehyde)[4)]. ROS also causes oxidative damage to proteins and DNAs[4)]. Among these damages, oxidative DNA damage is the most detrimental one to human health because of its role in the pathogenesis of the various diseases mentioned above. ROS, especially hydroxyl radical (OH•), can cause DNA base changes, strand breaks, damage to tumor-suppressor genes, and enhanced expression of proto-oncogenes[5)]. In addition to the increase of ROS, the decrease of DNA repair capacity is the other reason for the accumulation of oxidative DNA damage in the human body[6)].

Physical exercise is an important factor affecting oxidative stress and oxidative DNA damage, since a sharp increase in oxygen consumption during exercise results in an increase in ROS generation. However, the effect of exercise-induced oxidative damage is variable depending on several factors such as type, mode, duration, and intensity of exercise[7)]. A single bout of exercise induces oxidative stress and DNA damage, whereas regular moderate intensity exercise decreases them[8)]. Under the concept of hormesis, low-to-moderate ROS generation induced by regular moderate intensity exercise is beneficial, since it would up-regulate key antioxidant enzymes[9)]. A previous study demonstrated that long-term high-intensity exercise (75%VO_2max) increased 8-hydroxy-2'-deoxyguanosine (8-OHdG), while regular exercise with moderate-intensity (50%VO_2max) tended to decrease 8-OHdG[10)]. The activity of 8-oxoguanine DNA

*Corresponding author. Wichai Eunpinichpong (E-mail: wiceun@yahoo.com)

glycosylase (OGG1), a crucial enzyme for the repair of 8-oxoguanine lesions, is up-regulated after regular exercise in human and animals[11].

Tai Chi (TC), a traditional Chinese exercise, is classified as a moderate intensity exercise, and its intensity is basically similar among different age and gender groups[12]. Previous studies have reported the numerous tangible benefits of TC practice, such as improvement of balance and prevention of falls, enhancement of flexibility and strength, as well as amelioration of cardio-respiratory and immune function impairments. Studies of the effects of TC exercise on oxidative stress and oxidative DNA damage are limited. TC exercise reduced the degree of oxidative stress in middle-aged women[13], older adults[14], and obese patients with type 2 diabetes[15], and long-term TC practice reduced the degree of DNA damage, measured by the comet assay, in middle-aged[13] and older[16] adults.

However, the effects of TC intervention on oxidative stress and DNA damage have not been investigated in young females who seldom participate in physical exercise. The efficacy of TC exercise on DNA base damage and its repair also remains unclear. Young females are less conscious about physical exercise compared to age-matched males[17]. Although TC exercise is suitable for any population[18], only middle-aged and elderly age groups frequently practice TC for health purposes. TC is becoming popular among Chinese college students since it has been designated as a compulsory sport in the Physical Education curriculum of most colleges. Therefore, the objective of this pilot study was to determine the effects of TC intervention on oxidative stress, DNA damage and repair in young females who did not participate in regular physical exercise.

SUBJECTS AND METHODS

Ten sedentary female students from Gannan Normal University, China, voluntarily participated in this program. They completed inclusion and lifestyle questionnaires. Exclusion criteria were as follows: having experience of TC exercise; having performed physical exercise for more than 1 hr per week in the past 3 months; smoker or regular alcohol drinker; consuming supplements or medical products with antioxidant properties; having a history of cardiovascular and respiratory diseases; having knee joint pain; having a history of lower-extremity fracture within the past 1 year.

The subjects gave their informed consent to participate in this study. They were asked to keep their usual lifestyle including dietary behavior and daily activities, and medication (if any) throughout the study period. At the time of recruitment, their demographic characteristics were as follows: age, 20.40±0.70 yrs old; height, 155.48±6.15 cm; weight, 47.13±6.11 kg; body mass index (BMI), 19.42±1.54 kg/m^2; and resting heart rate, 79.10±12.65 beats/minute.

All of the participants completed the 12-week TC training program. According to this protocol, they practiced the 24-form simplified TC, 5 times (sessions) per week (Monday to Friday), on campus. The first 2 weeks involved familiarization with TC through teaching and learning. In the following 10 weeks, the participants practiced 5 rounds of TC (1 round refers to practice from the 1st movement to the 24th movement of this TC style) accompanied by a classic music for this TC style in each session. An experienced TC instructor led all participants in practice and taught them necessary movement corrections in the intervals between rounds. Thus, each session lasted about 60 min including a 10-minute warm-up for stretching and a 5-minute cool-down for relaxation.

Heart rate (HR) during TC exercise was monitored in the last session of the 5 rounds of the 24-form simplified TC in the last 4th, 8th, and 12th weeks. The exercise duration of five rounds of TC practice lasted for around 30 minutes. HR parameters such as average HR, maximum HR, and minimum HR were recorded using a Polar watch (Suunto t6d Running pack, Finland) and the telemetry HR team system (Suunto team manager, Finland).

Blood samples were collected at baseline, 8, and 12 weeks. In each session of blood collection, 3 mL of whole blood was taken after overnight fasting between 7:30–8:30 a.m. from a forearm vein and placed into a heparinized tube. Plasma was separated by centrifugation at 3,000 rpm for 5 min and kept frozen at −80°C until assayed for 8-OHdG, OGG1, SOD, GPx, MDA, GSH, and hydroxyl radical inhibiting capacity (OH·-IC).

SOD was measured by the hydroxylamine assay using a spectrophotometer (UNICO7200, USA) at 550 nm. GPx was detected by the rate method using a spectrophotometer (UNICO7200) at 412 nm. MDA was assessed by the thiobarbituric acid (TBA) assay using a spectrophotometer (UNICO7200) at 532 nm. GSH was analyzed by colorimetry using a spectrophotometer (UNICO7200) at 420 nm. Hydroxyl radical inhibiting capacity was determined by the Griess colorimetric method using a spectrophotometer (UNICO7200) at 550 nm. The kits for SOD, GPx, MDA, GSH, and OH·-IC measurements were all purchased from Nanjing Jiancheng Bioengineering Institute, China.

Plasma 8-OHdG and OGG1 levels were measured using commercial ELISA (enzyme-linked immunosorbent assay) kits according to the manufacturer's instructions (Santa Cruz Biotechnology, Inc. Dallas, Texas, USA). Briefly, 40 μL of plasma were added to the sample wells. Then, either 10 μL of anti-8-OHdG or anti-OGG1 antibody in combination with 50 μL of streptavidin-biotin-horseradish peroxidase were added to the sample wells in turn. To the standard sample wells, 50 μL of 5 different concentrations of standard samples and 50 μL of streptavidin-biotin-horseradish peroxidase were added. The microtiter plate was then incubated for 1 hour at 37 °C. Then, the plate was washed with wash buffer 5 times. During the procedure of washing, before discarding the buffer, each well was filled with wash buffer for 30 seconds. Color reactions were then conducted for 10 min in the dark at 37 °C after 50 μL of chromogenic agent A and 50 μL of chromogenic agent B were added in turn to each well. Finally, 50 μL of stop solution was added to terminate the reaction and the blue color changed to yellow. Products were then detected with the medical Elisa Analyzer (DG5033A. Huadong Electronics Group Co., Ltd., Shanghai, China) at 450 nm within 15 min, and the concentrations of 8-OHdG and OGG1 in plasma were read

Table 1. Comparison of HR among the different time points

Variables	4 weeks	8 weeks	12 weeks
HRaverage (bmp)	108.3±10.5	110.00±10.7	113.9±18.8
HRmax (bmp)	124.2±12.4	126.9±13.4	132.9±22.5
HRmin (bmp)	88.3±10.9	87.7±8.3	84.8±10.4

HRaverage = mean heart rate, HRmin = minimum heart rate, HRmax = maximum heart rate, bmp = beats per minute

Table 2. Effects of Tai Chi exercise on oxidative stress markers

Variables	baseline	8 weeks	12 weeks
SOD (U/ml)	42.76±3.05	49.81±5.01*	49.05±4.73*
Gpx (U/ml)	130.30±22.88	133.53±18.77	141.60±28.55
MDA (nmol/ml)	2.77±0.41	2.12±0.41*	2.97±0.54
GSH (mgGSH/L)	11.92±5.07	10.48±3.08	9.19±4.56
OH·-IC (U/ml)	351.97±59.02	406.19±34.76*	408.49±49.86*

*p<0.05 compared to baseline, SOD: superoxide dismutase; GPx: glutathione peroxidase; MDA: malondialdehyde; GSH: glutathione; OH·-IC: hydroxyl radical inhibiting capacity

Table 3. Comparison of plasma 8-OHdG and OGG1, and their relationships at different time points

Variables	baseline	8 weeks	12 weeks
8-OHdG (ng/ml)	9.45±2.54	10.93±2.51	10.26±2.34
OGG1 (ng/ml)	6.91±2.05	8.17±1.54*	8.49±1.62*
R (p value)	0.842 (0.003)	0.78 (0.008)	0.545 (0.103)

*p<0.05 compared to baseline, 8-OHdG: 8-hydroxy-2'-deoxyguanosine; OGG1: 8-oxoguanine DNA glycosylase; R: correlation coefficients of plasma 8-OHdG and OGG1 at the same time point

from standard curves.

All data were expressed as the mean ± SD. The differences of plasma and HR monitoring parameters among different time points were determined by repeated measures analysis of variance (ANOVA). Post hoc pair-hoc comparisons (LSD) were performed to test the differences when significance was shown. The correlation between plasma 8-OHdG and OGG1 levels at the same time point was determined using Pearson's correlation coefficient. The Statistical Package for Social Sciences (SPSS) version 17.0 was used for the analysis and values of $p<0.05$ were considered significant.

RESULTS

When the participants were performing 5 rounds of TC, the average HR and maximum HR tended to increase at 4, 8, and 12 weeks, but they did not show significant differences (Table 1). In contrast, the minimum HR tended to decrease during this study period, although the decreases were not statistically significant ($p>0.05$) (Table 1).

The SOD level was significantly increased ($p<0.05$) at 8 and 12 weeks of TC exercise compared to the baseline value (Table 2). OH·-IC was also significantly increased at 8 and 12 weeks. Conversely, the plasma MDA level was significantly decreased at 8 weeks but not at 12 weeks compared to the baseline (Table 2). However, GPx and GSH levels did not alter significantly during the study period.

Although the increases were not statistically significant, the plasma 8-OHdG values in this study showed increases at 8 and 12 weeks compared to the baseline value (Table 3). In contrast, the plasma OGG1 level was significantly increased at 8 and 12 weeks compared to the baseline value (Table 3). Positive correlations between plasma 8-OHdG and OGG1 were found at baseline ($p=0.003$) and 8 weeks ($p=0.008$), but no statistically significant correlation ($p=0.103$) was found at 12 weeks (Table 3).

DISCUSSION

Several empirical studies using different parameters, including heart rate (HR), oxygen consumption (VO_2), and energy expenditure, have shown that TC is an aerobic exercise of moderate intensity[12]. However, in those previous studies, the intensity of TC was measured only when participants practiced one round of TC, from the first movement to the last one of the specific TC style. It is intuitive that several factors such as the individual variances in age, gender, proficiency levels of TC, and also TC style would affect the intensity. In the present study, we monitored the

participants' HR when they practiced 5 rounds of the 24-form simplified TC at 4, 8, and 12 weeks. The results of HR monitoring show that the average HR during TC practice corresponded to 54.3%, 55.1%, and 57.1% of the age-predicted maximum heart rate (=220-age) at 4, 8, and 12 weeks, respectively. These results indicate that the intensity of TC practice of the present protocol was moderate for the young female students.

Although exercise has numerous benefits for human health, it is also often claimed to induce oxidative stress since exercise increases ROS generation. About 2–5% or more of inhaled O_2 is converted into various ROS byproducts[18]. ROS generated during exercise is, however, not always harmful because moderate and gradual exercise helps people to acquire antioxidant defenses[19].

The antioxidant defense system in the human body includes enzymes, such as SOD and GPx, and non-enzymatic antioxidants such as GSH. In the enzymatic system, SOD is the first defense against oxidative stress and is the major defense against $O_2^{-\bullet}$, whereas GPx is responsible for scavenging H_2O_2 and other organic peroxides. GPx requires GSH as a substrate for peroxide decomposition[20]. In the present study, we observed a significant increase in plasma SOD and OH·-IC levels after 8 weeks of TC exercise indicating that 8 weeks of TC exercise following our protocol is sufficient enough to improve the scavenging capacities for $O_2^{-\bullet}$ and OH•. In this study, the plasma MDA level decreased at 8 weeks. Thus, TC exercise can help to reduce the lipid peroxidation products attacked by ROS. In the present study, although the increase was not statistically significant, GPx tended to increase with time of TC practice. Since GPx is efficiently induced by high ROS concentrations[20], our protocol would have generated low concentrations of ROS. Our results are in agreement with those of previous studies concerning the protective effects of TC exercise on oxidative stress in middle-aged and elderly subjects[13, 14].

We propose there are two possible mechanisms of regular moderate-intensity exercise which reduce the markers of oxidative stress. First, based on the theory of hormesis, low-to-moderate oxidants would cause up-regulation of antioxidant enzymes[9]. ROS generated in this type of exercise are not only toxic, but also play an important role in cell signaling and in the regulation of gene expression[9, 19]. In this sense, exercise itself is an antioxidant. Second, the cumulative effects of repeated exercise bouts and exposure to ROS during regular moderate-intensity exercise could be the other mechanism[19]. This type of TC exercise might increase resistance to oxidative stress[21].

In addition to the changes of oxidative stress markers, no significant changes in plasma 8-OHdG were observed throughout this study. In attack by ROS (especially OH•), 8-OHdG is the most ubiquitous product of oxidative DNA base modification which occurs in approximately 1 in 100,000 guanidine residues in a normal human cell[22]. Several studies have demonstrated that exercise increase the plasma 8-OHdG level. For example, the plasma 8-OHdG level was increased by 12 weeks of high-intensity exercise, whereas it tended to decrease after moderate-intensity exercise in young men[10]. 8-OHdG was significantly lower in a moderate endurance exercise group than in a sedentary group[4]. After 10 months of aerobic exercise, the 8-OHdG level was increased significantly compared to the baseline in a group of elderly subjects[23]. Inconsistency among these outcomes may be due to the different age groups, exercise types and modes, as well as the exercise intervention duration.

OGG1 has the specificity to recognize and remove 8-OHdG[24]. Radak et al. reported that marathon running increases the activity of OGG1 in human skeletal muscles[25]. An animal study also showed that 2 months of regular treadmill running significantly upregulated the activity of OGG1 in hepatocyte nuclei of old rats compared to a sedentary old group[11]. Another animal study showed that exercise training increased OGG1 levels/activity in the nucleus of skeletal muscles and specific activity of OGG1 in mitochondrial compartments[26]. Similarly, our present study demonstrated that the plasma OGG1 level significantly increased after TC practice for 8 and 12 weeks. Increase of this enzyme in plasma is likely to be an adaptive response to the increased oxidative damage induced by elevated ROS during TC exercise.

8-OHdG is formed by OH• attack at the C8 position of deoxyguanosine in DNA. The damaged DNA with 8-OHdG may contribute to point mutation during the subsequent replication[27]. With normal body repair mechanisms, 8-OHdG can be cleaved by OGG1 through the way of base excision pathway and normal guanine is added to the site[24]. A few studies have reported a correlation between 8-OHdG and OGG1. For example, Kondo et al. showed a positive association between 8-OHdG levels and OGG1 expression ($r=0.702$, $p<0.05$)[28]. In our study, significant positive correlations between plasma 8-OHdG and OGG1 were observed at baseline ($r=0.824$, $p<0.01$) and 8 weeks ($r=0.78$, $p<0.01$); however, no significant correlation was found at 12 weeks ($r=0.545$, $p>0.05$). These results suggest that the increase of OGG1 activity after 12 weeks of TC exercise exceeds the requirements for immediately excising all 8-OHdG in young females. The enhancement of OGG1 may increase the repair rate[29].

Based on the results of this pilot study, a 12-week TC exercise intervention is effective at increasing the activity of plasma SOD and OGG1 and decreasing plasma MDA concentration in young sedentary females. It suggests that TC exercise helps to alleviate the oxidative stress response and DNA damage in those who have serious levels of oxidative stress and DNA damage, such as older and sick people.

This study had several limitations. First, there was no control group. Second, the sample size was small. Third, the authors only told the participants to keep their daily dietary habits and did not ask them to record their dietary details, especially before each blood sampling. Thus, dietary variation might have affected the data of blood parameters. Nevertheless, this pilot study showed TC intervention can improve the response to oxidative stress and the DNA damage/repair process in female students. Further studies with a longer intervention duration, larger groups, and a control group are needed to confirm our present results. Moreover, the long-term effects of Tai Chi on oxidative stress response

and DNA/repair also need to be studied in the future.

ACKNOWLEDGEMENTS

This study was supported by the Back, Neck and Other Joint Pain Research Group, Khon Kaen University, Thailand. We also thank Professor Yukifumi Nawa for his helping with the modification of this manuscript.

REFERENCES

1) Harman D: Aging: a theory based on free radical and radiation chemistry. J Gerontol, 1956, 11: 298–300. [Medline] [CrossRef]
2) Sakamoto R, Matsubayashi K, Kimura Y, et al.: Comprehensive geriatric assessment of elderly highlanders in Qinghai, China, III: oxidative stress and aging in Tibetan and Han elderly highlanders. Geriatr Gerontol Int, 2009, 9: 352–358. [Medline] [CrossRef]
3) Finkel T, Holbrook NJ: Oxidants, oxidative stress and the biology of ageing. Nature, 2000, 408: 239–247. [Medline] [CrossRef]
4) Radák Z, Kaneko T, Tahara S, et al.: The effect of exercise training on oxidative damage of lipids, proteins, and DNA in rat skeletal muscle: evidence for beneficial outcomes. Free Radic Biol Med, 1999, 27: 69–74. [Medline] [CrossRef]
5) De Bont R, van Larebeke N: Endogenous DNA damage in humans: a review of quantitative data. Mutagenesis, 2004, 19: 169–185. [Medline] [CrossRef]
6) Gil del Valle L: Oxidative stress in aging: theoretical outcomes and clinical evidences in humans. Biomedic Aging Pathol, 2011, 1: 1–7. [CrossRef]
7) Bloomer RJ: Effect of exercise on oxidative stress biomarkers. Adv Clin Chem, 2008, 46: 1–50. [Medline] [CrossRef]
8) Radak Z, Chung HY, Koltai E, et al.: Exercise, oxidative stress and hormesis. Ageing Res Rev, 2008, 7: 34–42. [Medline] [CrossRef]
9) Gomez-Cabrera MC, Domenech E, Viña J: Moderate exercise is an antioxidant: upregulation of antioxidant genes by training. Free Radic Biol Med, 2008, 44: 126–131. [Medline] [CrossRef]
10) Goto C, Higashi Y, Kimura M, et al.: Effect of different intensities of exercise on endothelium-dependent vasodilation in humans: role of endothelium-dependent nitric oxide and oxidative stress. Circulation, 2003, 108: 530–535. [Medline] [CrossRef]
11) Nakamoto H, Kaneko T, Tahara S, et al.: Regular exercise reduces 8-oxodG in the nuclear and mitochondrial DNA and modulates the DNA repair activity in the liver of old rats. Exp Gerontol, 2007, 42: 287–295. [Medline] [CrossRef]
12) Lan C, Chen SY, Lai JS: Relative exercise intensity of Tai Chi Chuan is similar in different ages and gender. Am J Chin Med, 2004, 32: 151–160. [Medline] [CrossRef]
13) Goon JA, Aini AH, Musalmah M, et al.: Effect of Tai Chi exercise on DNA damage, antioxidant enzymes, and oxidative stress in middle-age adults. J Phys Act Health, 2009, 6: 43–54. [Medline]
14) Rosado-Pérez J, Santiago-Osorio E, Ortiz R, et al.: Tai chi diminishes oxidative stress in Mexican older adults. J Nutr Health Aging, 2012, 16: 642–646. [Medline] [CrossRef]
15) Chen SC, Ueng KC, Lee SH, et al.: Effect of t'ai chi exercise on biochemical profiles and oxidative stress indicators in obese patients with type 2 diabetes. J Altern Complement Med, 2010, 16: 1153–1159. [Medline] [CrossRef]
16) Goon JA, Noor Aini AH, Musalmah M, et al.: Long term Tai Chi exercise reduced DNA damage and increased lymphocyte apoptosis and proliferation in older adults. Med J Malaysia, 2008, 63: 319–324. [Medline]
17) Shaohua J, Meiting Q, Ruiguang C, et al.: Research on gender difference of consciousness and behavior of physical exercise among college students in Hebei province. J Beijing Sport Univ, 2006, 29: 42–44 (in Chinese).
18) Bandyopadhyay U, Das D, Banerjee RK: Reactive oxygen species: oxidative damage and pathogenesis. Curr Sci India, 1999, 77: 658–666.
19) Ji LL, Gomez-Cabrera MC, Vina J: Exercise and hormesis: activation of cellular antioxidant signaling pathway. Ann N Y Acad Sci, 2006, 1067: 425–435. [Medline] [CrossRef]
20) Jenkins RR, Goldfarb A: Introduction: oxidant stress, aging, and exercise. Med Sci Sports Exerc, 1993, 25: 210–212. [Medline] [CrossRef]
21) Radak Z, Chung HY, Goto S: Exercise and hormesis: oxidative stress-related adaptation for successful aging. Biogerontology, 2005, 6: 71–75. [Medline] [CrossRef]
22) Grollman AP, Moriya M: Mutagenesis by 8-oxoguanine: an enemy within. Trends Genet, 1993, 9: 246–249. [Medline] [CrossRef]
23) Muñoz ME, Galan AI, Palacios E, et al.: Effect of an antioxidant functional food beverage on exercise-induced oxidative stress: a long-term and large-scale clinical intervention study. Toxicology, 2010, 278: 101–111. [Medline] [CrossRef]
24) Boiteux S, Radicella JP: The human OGG1 gene: structure, functions, and its implication in the process of carcinogenesis. Arch Biochem Biophys, 2000, 377: 1–8. [Medline] [CrossRef]
25) Radák Z, Apor P, Pucsok J, et al.: Marathon running alters the DNA base excision repair in human skeletal muscle. Life Sci, 2003, 72: 1627–1633. [Medline] [CrossRef]
26) Radak Z, Atalay M, Jakus J, et al.: Exercise improves import of 8-oxoguanine DNA glycosylase into the mitochondrial matrix of skeletal muscle and enhances the relative activity. Free Radic Biol Med, 2009, 46: 238–243. [Medline] [CrossRef]
27) Valavanidis A, Vlachogianni T, Fiotakis C: 8-hydroxy-2′-deoxyguanosine (8-OHdG): a critical biomarker of oxidative stress and carcinogenesis. J Environ Sci Health C Environ Carcinog Ecotoxicol Rev, 2009, 27: 120–139. [Medline] [CrossRef]
28) Kondo S, Toyokuni S, Tanaka T, et al.: Overexpression of the hOGG1 gene and high 8-hydroxy-2′-deoxyguanosine (8-OHdG) lyase activity in human colorectal carcinoma: regulation mechanism of the 8-OHdG level in DNA. Clin Cancer Res, 2000, 6: 1394–1400. [Medline]
29) Hollenbach S, Dhénaut A, Eckert I, et al.: Overexpression of Ogg1 in mammalian cells: effects on induced and spontaneous oxidative DNA damage and mutagenesis. Carcinogenesis, 1999, 20: 1863–1868. [Medline] [CrossRef]