PREVENTIVE EFFECT OF CRANBERRY CONSUMPTION AGAINST DNA DAMAGE AFTER EXHAUSTIVE EXERCISE IN ATHLETE MEN

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ABSTRACT

This study was performed to determine Effect of short-term cranberry consumption on DNA damage after exhaustive exercise in athlete men. Twenty male athletes in a randomized and double-blind design were allocated in two equal supplement and placebo groups (take cranberry soft chews 500mg for 14 days). After supplementation, all participants were participated in Bruce test. The blood samples were taken in three phases (before and after the supplementation and after the exercise). This experiment used single cell gel electrophoresis including modified comet assay (with FPG and ENDO III enzymes) to detect the oxidative DNA damage induced by exhaustive exercise in athlete blood cells, and the product 8-hydroxy- 2-deoxyguanosine (8-OHdG) of oxidation of DNA, also, the level of MDA, BUN and CK in athlete plasma were observed. The normal data (Mean ± SD) were analyzed by repeated measure ANOVA, Tukey and independent t-test (P≤0.05). The results showed that a 14-day cranberry consumption hadn’t significant effect on the damage index of modified comet assay (with FPG and ENDO III enzymes) and the level of 8-OHdG and also the level of MDA, BUN and CK in plasma at rest status (P>0.05). However, exercise-induced decrease of 8-OHdG and the value of comet assay and the plasma level of MDA, BUN and CK in the cranberry group were significantly more in comparison with those in the placebo group (P<0.01). Result of the study indicates that 14-day cranberry consumption can reduce the production of lipid oxidation and oxidative DNA damage during exhaustive exercise.

Keywords: Cranberry, DNA damage, single cell gel electrophoresis (SCGE), exhaustive exercise, Lipid oxidation.

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Introduction

Cranberries contain various sugars like sucrose, glucose, fructose, and sorbitol. Interestingly, apart from the presence of vitamin C in cranberries, a wide variety of vitamins and provitamins have been identified. It is well known that both acute aerobic and anaerobic exercise has the potential to result in increased free radical production, if the increase in reactive oxygen species (ROS) exceed the antioxidant defense system present, thereby resulting in oxidative damage to specific biomolecules and the extent of oxidative damage mostly depend on both intensity and duration.

Oxidative damage of biomolecules such as DNA and lipids has been implicated in the modification of aging and degenerative diseases and reactive oxygen species (ROS) play an important part as mediators of tissue injury and inflammation after exhaustive exercise. Aside from the measurement of 8-OHdG, assessment of DNA damage has also been performed using the single cell gel electrophoresis assay (Comet assay) which detects DNA damage with high sensitivity. It has been experimentally confirmed antioxidant supplements could prevent or reduce the level of DNA damage.

Recently, different studies indicated that natural antioxidants contained in vegetables and fruits (flavonoids, carotenoids) may be useful in prevent-
ing deleterious consequences of oxidative stress\(^5\), so antioxidant supplements are commonly used by athletes as dietary nutrition to counteract the oxidative stress of exercise to increase exercise performance. In this regard, adopting healthier dietary habits to improve body antioxidant defenses must be considered important in the global maintenance of health. Cranberry juice has been long used to prevent infections because of its effect on the adhesion of the bacteria to the host surface\(^6\). A lot has been said about its role in urinary tract infection and other systemic diseases\(^6\), but little is known about its effect as a source of antioxidant during exercise.

A recent study in 2004 found that cranberry was used as a dietary supplement recent study showing a measured increased level of total antioxidant status after cranberry juice consumption\(^7\). Cranberries (Vaccinium macrocarpon) are one of the most important sources of flavonoids, including quercetin and myricetin, which are known to be potent antioxidants\(^8\). Some studies have reported that low-energy cranberry juice (2 cups/day) significantly reduces lipid oxidation and increases plasma antioxidant capacity in women with metabolic syndrome\(^9\). Cranberries are a valuable source of biologically active substances with well-known health beneficial properties. They contain vitamins (C, A, B1 and B2), microelements (potassium, calcium, sodium, phosphorus, magnesium, iron and iodine, pectins, dietary fibre) as well as organic acids belonging to different chemical groups\(^10\).

However, the current studies about the effect of Cranberry on preventing DNA damage after exhaustive exercise are still rarely reported. Whether exhaustive exercise does increase the need for additional antioxidants in the diet is not clear. This experiment used single cell gel electrophoresis including modified comet assay (with FPG and ENDO III enzymes) to detect the oxidative DNA damage induced by exhaustive exercise in athlete blood cells, and the product 8-hydroxy-2-deoxyguanosine (8-OHdG) of oxidation of DNA, also, the protective effect of Cranberry on athlete blood cells were observed, all of which were to provide a reference basis on how to supplement Cranberry in human body. Furthermore, it was aimed to observe the effect of Cranberry to reduce the oxidative stress and whether could it enhance the DNA repair capacity to increase exercise ability.

### Materials and methods

#### Subjects

20 athlete male students majored in physical education of Minnan Normal University (aged 22.16±1.07years, weight 68.25±3.45 kg and height 175.50±2.76 cm, detail see from Table 1), to participate in this research, as the samples were selected voluntarily and with their consent. Subjects were selected according to the following criteria: non-smoking and apparently healthy. Furthermore, all selected subjects were not taking any antioxidant supplements (such as vitamin A, C, or E) 2 month before and during the study; they were asked to follow a rigorously standardized basal diet, avoid of cranberry products. Volunteer subjects randomly replaced in two groups of receiver of cranberry supplements (cranberry soft chews 500mg distributed by Wal-Mart Stores, USA, take one chew daily with a meal for fourteen days) and placebo (dextrose capsule). For control of subjects Nutrition, dietary questionnaire of 24-hour retention was used.

#### Exercise protocol

First day of study, height, weight and percentage body fat was measured in all subjects. Initial blood sample, at baseline, before starting supplementation, were taken from the Antecubital vein from all the participants. Second samples were taken after completion of 14-day period of supplementation and before Bruce test. During the Bruce test, the VO2max and heart rate (HR) were recorded. The VO2max situation was accepted if the oxygen uptake reached a plateau, the plasma lactate concentration was above 8 mmol/l, or the respiratory exchange rate was above 1.15. After the Bruce test, third samples were taken from subjects. At each stage of depletion, Peripheral blood samples were collected at different time points from patients and control individuals, at rest, early in the morning, into two tubes with anticoagulant. One aliquot was used for the comet assay and the other aliquot to obtain blood plasma for the remaining analysis.

All measurements were done in same temperature, humidity, ventilation and lighting. In addition, subjects, 48 hours before the test, don’t do any heavy physical activity, and their meal before the test was similar.

#### Alkaline comet assay

The alkaline comet assay was done as described by Godard et al.\(^11\). Fully-frosted clean
microscope slides were covered with 1% normal melting point (NMP) agarose and allowed to polymerize at room temperature to allow agarose to dry. After solidification, the gel was scraped off from the slide. The slides were further coated with 0.6% NMP agarose. When this layer had solidified a second layer containing the whole blood sample (0.5μL) mixed with 0.5% low melting point (LMP) agarose was placed on the slides. After 10 min of solidification on ice, slides were covered with 0.5% of LMP agarose. An amount of 100 μL of this agarose cell suspension was layered on the top of the second layer. Finally, the fourth layer of 0.5% low melting point (LMP) agarose was added to cover the third layer and allowed to solidify for 10 min at 4°C.

Afterwards the slides were immersed for one hour in ice-cold freshly prepared lysis solution (2.5M NaCl, 100 mM Na2EDTA, 1% Na-sarcosine, 1% 10 mM Tris–HCl, pH 10 with Triton X-100 and 10% DMSO added fresh to lyse cells and allow DNA unfolding). After lysis, slides were placed in the freshly prepared electrophoresis buffer (300 mM NaOH, 1 mM Na2EDTA, pH 13.0) to remove salts. The slides were set in this alkaline buffer for 10 min to allow DNA unwinding and expression to alkal labile sites. Denaturation and electrophoresis was performed at 4°C under dim light at 25 V (300 mA). After electrophoresis, the slides were washed three times at 5 min intervals with buffer (1% Triton X-100, 10% DMSO) to remove excess alkali and detergents. Each slide was stained with ethidium bromide (20 μg/ml) for 10 min and covered with a cover slip. Slides were stored at 4°C in humidified sealed containers until analysis. To prevent additional DNA damage, handling with blood samples and steps included in the preparation of slides for the comet analysis were conducted under yellow light or in the dark. Slides were examined at 100x magnification on an Olympus fluorescence microscope (Olympus Optical Co, Ltd, Tokyo) with excitation at 520 nm green barrier filter. For DNA damage evaluation, 100 cells per sample were analyzed by optical microscopy at 100x magnification. The cells were visually scored by measuring the DNA migration length and the amount of DNA in the tail into five classes, from undamaged -0 to maximally damaged -4, and a damage index (DI) value was calculated for each sample\textsuperscript{(12)}. Damage index, thus, ranged from 0 (completely undamaged: 100 cells × 0) to 400 (with maximum damage: 100 cells × 4).

**Detection of Oxidized Bases**

The comet assay was modified by using specific enzymes to expose oxidative damage. The enzyme formamidopyrimidine DNA glycosylase (FPG) recognizes the common oxidized purine 8-oxo-7,8-dihydroguanine and ring-opened purines (13), whereas endonuclease III (ENDO III) converts oxidized pyrimidines to strand breaks (14). After lysis, the slides were washed for 5 minutes each in enzyme buffer (40 mM HEPES-KOH, 1 MKCl, 5 mM EDTA, 2.5 mg/mL bovine serumalbumin fraction V-BSA, and pH 8.0). The suspension was added to the slide, covered with coverslip, and incubated for 45 (ENDO III) and 30 minutes (FPG) at 37°C. Subsequent steps were the same as in the alkaline version of comet assay.

**Measurement of plasma 8-OHG**

Determination of plasma 8-OHG levels was performed using the method of Farhadi et al\textsuperscript{(15)}. For the measurement of plasma 8-OHG, 1 ml plasma was spiked with 1,000 cpm (14C)-OHG. Plasma protein was precipitated by the addition of an equal volume of acetonitrile, and precipitated protein was separated by centrifugation at 3,000g for 15 min at 4°C. Supernatant was transferred to a new tube and mixed with eight volumes of water. The resulting sample was applied to the preconditioned C18/OH solid-phase extraction column. The solidphase extraction column was washed with 5 ml of 50 mmol/l KH2PO4 buffer (pH 7.5), and then retained. Compounds were eluted with 3 ml of 15% methanol in the same buffer. The elute was loaded into the immunoaffinity column prepared with monoclonal antibody for 8-OHG. Purified 8-OHG was dissolved in 50 ml water and injected into an HPLC device equipped with a Beckman Ultrasphere ODS column (5 cm, 4.6 cm, 25 cm) and an electrochemical detector. The height of the 8-OHG peak and the total radioactivity of the elute were measured. The height of the peak was used to determine the total amount of 8-OHG injected, which was the sum of the plasma 8-OHG and (14C)-OHG added. The amount of 8-OHG was determined by subtracting the amount of (14C)-OHG injected from the total amount of 8-OHG injected. The amount of (14C)-OHG injected was determined from the calibration curve of the peak height of (14C)-OHG. The radioactivity of the elute was used to determine the amount of loss of 8-OHG during the purification procedure using the immunoaffinity column.
Estimation of Plasma MDA, BUN and CK

Aliquots of whole blood were taken immediately for the comet assay analysis. Remaining blood was centrifuged at 2500×g for 10 min; Plasma was then aliquoted to cryotubes for various assays. The process of lipid peroxidation results in the formation of malondialdehyde (MDA). Lipid peroxidation was determined indirectly by measuring MDA formed by reacting with thiobarbituric acid (TBA) to give a red species having a maximum at 532 nm. MDA was determined spectrophotometrically according to the method of the Nanjing Jiancheng Bioengineering Institute (China) with a spectrometer. Plasma creatine kinase (CK) activity and blood urea nitrogen (BUN) were measured for evaluating the cell membrane injury by a model 7170A automatic analyser (Hitachi) with commercial assay reagents (Amresco).

Statistical analysis

The data are presented as means ± SD of independent experiments. They were analyzed using the software of statistical package for the social sciences (SPSS) version 16.0 for Windows. The statistical difference between groups was determined with repeated measure ANOVA followed by Dunnett’s test (two-sided) as multiple comparisons. The minimum level of significance was considered to be P < 0.05.

Results

The general characteristics of the placebo group and cranberry group are shown in Table 1. T-test results showed no significant differences between the variables of height, weight, age, BMI and percentage body fat of two groups’ subjects that indicated to be homogeneous both groups in these variables.

The comet assay results are shown in Table 2. DNA damage is visualized at the individual cell level as an increased migration of genetic material (“comet tail”) from the nucleus (“comet head”). The basal DI in the alkaline comet assay that detects DNA single- and double-strand breaks and alkali labile sites, the results of which showed that the higher this values, the greater the damage that has occurred to the nuclear DNA. The value of DI in the alkaline comet assay did not show significant difference between placebo group and cranberry groups at rest(P>0.05).

<table>
<thead>
<tr>
<th>Table 1: Personal characteristics of Cranberry and placebo groups.</th>
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<tbody>
<tr>
<td><strong>variable</strong></td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
</tr>
<tr>
<td><strong>Height (cm)</strong></td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
</tr>
<tr>
<td><strong>BMI (kg/m²)</strong></td>
</tr>
<tr>
<td><strong>Percentage body fat (%)</strong></td>
</tr>
<tr>
<td><strong>Heart rate (beats per min)</strong></td>
</tr>
<tr>
<td><strong>Blood pressure (mmHg)</strong></td>
</tr>
</tbody>
</table>

After supplementation, the results of DI in the alkaline comet assay still did not have obvious significance compared with the value of baseline for both two groups, at the same time, there was no obvious significance between placebo group and cranberry groups(P>0.05).

<table>
<thead>
<tr>
<th>Table 2: Comet assay results between placebo goup and Cranbeberry goup in different phases.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>variable</strong></td>
</tr>
<tr>
<td><strong>Damage index</strong></td>
</tr>
<tr>
<td><strong>alkaline comet assay</strong></td>
</tr>
<tr>
<td><strong>Damage index</strong></td>
</tr>
<tr>
<td><strong>FPG comet assay</strong></td>
</tr>
<tr>
<td><strong>Damage index</strong></td>
</tr>
<tr>
<td><strong>ENOD III comet assay</strong></td>
</tr>
</tbody>
</table>

However, the value of DI in the alkaline comet assay showed that cranberry and placebo groups have significant difference after exhaustive exercise (bruce test) (P<0.05). Also, the value of DI in modified comet assay (with FPG and ENOD III enzymes) showed significantly higher damage index in placebo group compared with cranberry group after exhaustive exercise(P<0.01), and there was no significant difference between groups at baseline and after supplementation(P>0.05).

As observed in Figure 1, the product 8-hydroxy- 2-deoxyguanosine (8-OHdG) has been measured as an index of exercise induced oxidation of DNA(15), and the level of plasma 8-OHdG of repeated measure ANOVA for any groups showed that significant difference in effect of measurement phases in cranberry group (P<0.05), but, significant
difference showed in placebo group (P≤0.01). The results of Tukey post-hoc test for placebo group showed significant difference in before and after bruce test (P≤0.01).

![Graph showing plasma 8-hydroxy-2-deoxyguanosine in baseline and after supplementation](image)

**Figure 1**: plasma 8-hydroxy-2-deoxyguanosine in baseline and after supplementation and exhaustive exercise of cranberry and placebo groups.

Also, statistically significant differences (P≤0.01) in the level of plasma 8-OHdG were found between placebo group and cranberry groups through independent t-test.

From the table 3, it showed that the level of BUN and CK did not have significant differences between placebo group and cranberry groups (P>0.05) at baseline and after supplementation. However, both the level of BUN and CK increased significantly after exhaustive exercise compared with the value of BUN and CK at baseline and after supplementation (P<0.05).

<table>
<thead>
<tr>
<th>phases</th>
<th>Group</th>
<th>N</th>
<th>BUN (mmol/L)</th>
<th>CK (IU/L)</th>
<th>MDA (nmol/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>baseline</td>
<td>placebo</td>
<td>8</td>
<td>5.51±0.78</td>
<td>213.6±89.62</td>
<td>4.79±0.85</td>
</tr>
<tr>
<td></td>
<td>cranberry</td>
<td>8</td>
<td>5.28±0.95</td>
<td>202.5±72.71</td>
<td>4.86±0.99</td>
</tr>
<tr>
<td>after supplementation</td>
<td>placebo</td>
<td>8</td>
<td>5.62±0.94</td>
<td>206.6±95.76</td>
<td>4.67±1.06</td>
</tr>
<tr>
<td></td>
<td>cranberry</td>
<td>8</td>
<td>5.37±0.75</td>
<td>196.5±72.71</td>
<td>4.82±0.96</td>
</tr>
<tr>
<td>after exhaustive exercise</td>
<td>placebo</td>
<td>8</td>
<td>6.59±1.15</td>
<td>267.6±122.42</td>
<td>7.11±1.06</td>
</tr>
<tr>
<td></td>
<td>cranberry</td>
<td>8</td>
<td>5.37±0.98</td>
<td>207.7±105.23</td>
<td>5.20±0.95</td>
</tr>
</tbody>
</table>

**Table 3**: The changing of bun, CK and MDA on plasma in different group.

All the data was expressed by x±s, *P<0.05, **P<0.01, ***P<0.001. Significant difference between cranberry and placebo groups; †P<0.05, ‡P<0.01, §§P<0.001, Significant difference after supplementation and after exercise protocol.

For the level of MDA, there were no significant differences between groups (P>0.05) at baseline and after supplementation, but, the value of MDA in placebo group increased obviously after exhaustive exercise compared with the value of MDA at baseline and after supplementation (P<0.001), as well as the MDA level in cranberry group significantly increased after exhaustive exercise compared with baseline and supplementation (P<0.05). Also, there were significant difference between placebo group and cranberry groups after exhaustive exercise (P<0.01).

**Discussion**

Cranberry administration has been reported to ameliorate dyslipidemia, hyperglycaemia and oxidative stress in individuals with the metabolic syndrome (16). It is well known that most of athletes have to run to exhaustion to achieve wonderful exercise performance, and it is clear that the consequences develop in the days following severe exercise are soreness and stiffness, which would give the athlete a big trifle to begin a new training (17).

Moreover, during exercise there would be a 10-40-fold increase in oxygen consumption compared to the resting state, specially, endurance exercise may contribute to a two-to three-fold increase in the free radical concentrations of the muscle and the liver (20). Exhaustive physical exercise can increase susceptibility to injury, promote chronic fatigue and overtraining, partially due to the overproduction of reactive oxygen species (ROS), and reactive oxygen species (ROS) cause lipid peroxidation and oxidation of some specific proteins, thus affecting many intra- and intercellular systems (21). DNA damage are physical abnormalities in the DNA, such as single- and double-strand breaks and oxidative DNA damage might result in diseases (20).

Recent epidemiological studies have demonstrated that diets rich in fruits and vegetables are associated with lower incidences of oxidation-linked diseases such as cancer, cardiovascular disease and diabetes. Cranberries are a good source of phenolic compounds including flavonols, anthocyanins and proanthocyanidins (22), and cranberry phenolics-mediated antioxidant enzyme response in oxidatively stressed porcine muscle (23). An extract prepared from cranberry juice powder using a mixture of chloroform and methanol was particularly effective at inhibiting lipid oxidation in mechanically separated turkey (24).

Comet assay has been widely used in various studies to detect the DNA damage connected with kinds of diseases due to its rapid, simple, and sensitive technique for measuring DNA breaks and
repair in single cells\textsuperscript{(21)}. DNA damages can be recognized by enzymes, and, thus, they can be correctly repaired if redundant information, such as the undamaged sequence in the complementary DNA strand or in a homologous chromosome, is available for copying\textsuperscript{(25)}.

Moreover, DNA damaging agents can damage other biomolecules such as proteins, carbohydrates, lipids, and RNA. Generally, DNA damage is the leading basis in the carcinogenic process, however DNA can be repaired, for the body has some repair mechanism after injury, so as to avoid the occurrence of tumor\textsuperscript{(26,27)}. Blood mononuclear cell DNA oxidative damage is found to increase in human body after intense exercise\textsuperscript{(29,30)}. It was also found that the DNA of skeletal muscle cells, lymphocytes and blood cells appeared damage to some extent\textsuperscript{(31)}. In the present study, the SCGE technique was used to detect DNA damage in athlete blood cell, the results showed that the value of damage index in the alkaline comet assay of placebo groups were significantly increased post exhaustive exercise compared with rest status and cranberry group (P < 0.05), suggesting that DNA of athlete blood cells appeared serious injury, this result supported previous reports\textsuperscript{(29,30)}. This also suggests that cranberry supplementation has the ability to protect human body from serious DNA damage.

Most damage to bases in DNA is repaired by the base excision repair pathway. Formamidopyrimidine DNA glycosylase (FPG) is a base excision repair enzyme which recognizes and removes a wide range of oxidized purines from correspondingly damaged DNA\textsuperscript{(32)}. Also, endonuclease III (Endo III) is major repair enzyme for pyrimidine lesions formed by reactive oxygen species\textsuperscript{(32)}.

In this study, the value of the value of DI in modified comet assay (with FPG and ENDO III enzymes) increased severely after exhaustive exercise in placebo group compared to cranberry group (P < 0.01). It was suggested that there was severe oxidative DNA damage produced in athlete blood cell during exhaustive exercise.

Cumulative oxidative DNA damage have a significant effect of the impairment on normal cellular repair mechanisms and one of the main etiological hypotheses linking genomic instability, mutagenesis and tumorigenesis is that of deficient cellular repair mechanisms due to extensive oxidative DNA damage and cellular injury\textsuperscript{(33)}. The resultant damaged bases in DNA may be responsible for mutations that lead to carcinogenesis.

One of the major forms of oxidative DNA damage, 8-hydroxydeoxyguanosine (8-OH-dG) has been proposed as a key biomarker relevant to carcinogenesis\textsuperscript{(15)}. It is usually for our organisms exposed to reactive oxygen species (ROS) produced by environmental hazards, such as radiation and toxic chemicals, and also by physical activities, so it would beneficial for our health, especially for the athlete, to find out the protective supplements to reduce muscle damage.

Antioxidants work together in animal cells against toxic reactive oxygen species\textsuperscript{(28)}. Recent studies have provided novel findings on the health benefits of cranberry consumption in restoring serum cholesterol profiles and reducing side effects of adiposity by suppressing the production of pro-inflammatory cytokines and inhibition of oxygen radical production in adipose tissue\textsuperscript{(34)}.

Another study reported that cranberries reduce lipid accumulation during preadipocyte differentiation by down-regulation of the mRNA level of several genes associated with lipid metabolism, such as fatty acid binding protein (aP2), lipoprotein lipase (LPL), fatty acid synthase (FAS), hormone sensitive lipase (HSL)\textsuperscript{(35)}. It is well accepted that lipid peroxidation was induced by free radicals. The present study also investigates the status of lipid peroxidation. Malondialdehyde (MDA) is the product of lipid peroxidation and its level is a marker of lipid oxidation, also its content can reflect the degree of lipid peroxidation in vivo and indirectly reflect the extent of the damage of cells\textsuperscript{(36)}.

The results of the present experiments showed that plasma MDA activity increased significantly after acute exercise as well as DNA damage in athlete blood cell. The release of cytoplasmic enzymes including creatine kinase (CK), which was considered to be an available parameter for the working muscle injury during exercise, and CK activity was the most specific and sensitive indicator to detect and monitor for the muscle injury\textsuperscript{(37)}. BUN (blood urea nitrogen) is a marker of kidney function, and it is a waste product of cell metabolism.

In this study, the level of CK and BUN increased significantly after exhaustive exercise in placebo group compared with cranberry group. However, the Cranberry supplementation reduce the MDA level and DNA damage, which was suggested that DNA damage was related with tissue lipid peroxidation, and the high intensity exercise caused oxidative damage in the blood and eventually lead to DNA damage of blood cells.
The fact that, in the present study, a significant level of DNA damage was detected after exhaustive running in the athlete probably shows that DNA might be a weak link in a cell’s ability to tolerate oxygen free-radical attack. It is conceivable that the levels of exercise attained in our experiments could be associated with oxidative stress, and perhaps the deleterious effects associated with such stress. It is possible that a depression in the running performance of athlete could be attributed to disruption of the oxidant/antioxidant balance consequently resulting in oxidative stress.

Cranberry as an antioxidant one hand can inhibit the oxidase enzymes system, activate and protect anti-oxidase system, on the other hand, it can directly react with lively free radical, change lipid peroxides into hydroxyl resin, and it is a strong free radical scavenger. The study found that oxidative DNA damage of cranberry group were significantly lower than that of placebo in athlete blood cell after exhaustive exercise, indicating that Cranberry can effectively alleviate DNA damage of blood cell caused by high intensity exercise, the reason may be related to antioxidant effect in vivo on Cranberry.

The results also showed the level of CK, BUN and MDA content in cranberry group were significantly lower than those of placebo group in plasma of athlete, also, the present results indicate that short-term cranberry supplementation significantly decreased 8-hydroxy-2-deoxyguanosine after exhaustive activity in the cranberry group compared to the placebo group, which suggesting the supplement of Cranberry can alleviate the oxidative damage effect on the blood induced by high intensity exercise, which reduce the free radicals attacking the blood nuclei and nuclear genetic material. The possible mechanism for cranberry in reducing oxidative DNA damage is that cranberry with increasing intracellular antioxidants such as bilirubin, uric acid, and serum albumin can enhance the total capacity of antioxidant.

In conclusion, the current data indicated that strenuous exercise induced elevated DNA damage due to lipid peroxidation in organisms. The evidence from the present investigation suggests that exhaustive exercise increased oxidative DNA damage as measured by modified comet assay (with FPG and ENDO III enzymes) and plasma 8-OHdG level and also short-term cranberry supplementation decreased oxidative DNA damage caused by exhaustive exercise.

Cranberry consumption inhibits lipid peroxidation and has free radical scavenging activity, which can be beneficial for the protection against oxidative stress and reduced the DNA damage. So it is beneficial for the athletes to take cranberry to relieve oxidative stress to prevent from more DNA damage.

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