Assessment of DNA-damage in smoker and nonsmoker athletes

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Soheir Korraa*, Zakeya Ahmed Fathy **and Amr F. Sallam***
*Environmental Mutagen Laboratory, Department of Radiation Health, National Center for Radiation Research and Technology, Atomic Energy Authority, Cairo, Egypt.
** Department of Athletics Health Faculty of Athletic Education Al Haram Cairo Egypt.
***Ministry of Youth and Sport Egypt.

ABSTRACT

DNA fragmentation, apoptosis percentage and frequency of micronucleus (FMN) formation in circulating lymphocytes were measured in the blood of 40 non-smoker and 25 smoker athletes compared to age and socio-economic class matching 20 smoker and 20 non-smoker non-athletes as a control. Results showed that DNA-fragmentation and apoptotic lymphocytes were significantly higher among athletes compared to non-athletes and exhibited the highest levels in athlete smokers followed by control smokers. FMN was significantly high among athlete smokers followed by control smokers and there was no significant differences between non-smokers athletes when compared to non-smoker controls. The results of this work reveal the impact of smoking on the health of athletes.

Key words: *apoptosis percentage, athletes, DNA-fragmentation, frequency of micronuclei, smoking.*

INTRODUCTION

Reactive oxygen (ROS) and nitrogen species (RNS) are continuously generated in the biological system and play an important role in a wide variety of physiological and pathological processes, including cancer and emphysema (Halliwell, 1987). Recent evidence suggests that physical exercise augments the generation of ROS and RNS during strenuous physical activity performance, where oxygen consumption becomes increased by 10 to 15-fold over rest to meet energy demands (Clarkson, 1995). The resulting increase in oxygen consumption leads to the generation of ROS and RNS, which if elevated to a level that overwhelms tissue antioxidant defence systems, results in an oxidative stress. The magnitude of this stress depends on the ability of the tissues to detoxify the generated ROS (Sen, 2001), and consequently damaging cellular lipids, proteins and DNA inducing lipid peroxides, protein carbonyls and DNA damage (Wiesman and Halliwell, 1996).

Cigarette smoke induces cancer, emphysema, arteriosclerosis and other diseases after a delay of years (Wiesman and Halliwell, 1996). Cigarette smoke contains trace amounts of ά-particle emitting radioactive element polonium^{210} (Po^{210}), which is a naturally occurring daughter isotope of radium^{226} (Radford and Hunt, 1964, Little *et al.*, 1965). It also contains a large variety of compounds,

including aldehydes, benzopyrenes and coal tar and its pyrrolizidine products, which are oxidants and release free radicals that are capable of initiating or promoting oxidative damage (Church and Pryor, 1985; Reilly *et al.*, 1986). Also, oxidative damage may result from reactive oxygen species generated by the increased and activated phagocytes recruited in the lung due to cigarette smoke (Lehr *et al.*, 1993).

Scope of the study

The aims of the present study were to: (a) evaluate the oxidative damage induced by the increase of consumption of oxygen during exercise in athletes and (b) evaluate the synergism of oxidative damage induced due to dual exercise and cigarette smoke in athletes, compared to age and socio-economic class matching smoker and smoker non-athlete. Markers of DNA-damage were measured in terms of apoptosis percentage and the frequency of micronuclei formation (FMN) in circulating lymphocytes in addition to DNA fragmentation in plasma and total leucocytes.

MATERIALS AND METHODS

Materials (subjects)

Sixty five healthy male athletes, mean age 21 ± 2.4 years (range 18 - 24 years), mean body mass 75.8 ± 10.8 kg (range 65 - 92 kg) and mean of height 175.5 ± 6.72 (range 170 182) participated in the study. They were classified as 40 non-smoker athletes (21.1 ± 2.71) and 25 smoker athletes (20.9 ± 2.14) compared to age and socio-economic class matching 20 smokers (21.4 ± 1.4) and 20 non-smokers non-athletes (19.9 ± 3). All Athletes exercised on a regular basis at Maadi Olympic Centre in Cairo. Both athletes and controls were carrying out their Obligatory Military Service and consequently were expected to receive the same dietary constituents. Each subject in the study was subjected to both clinical and physical examination. Personal data and records for their age (20.55 ± 2.14 vs. 21.8 ± 2.71), smoking habits (20 ± 10 cigarettes / day), heights (175.05 ± 6.72) vs., weights (70.8 ± 10.8 vs. 73.85 ± 9.78), intake of drugs and frequency of training were scored. A sample of twenty ml blood was drawn after the consent of each individual in the study. Lymphocytes were separated using Histopaque lymphocyte separation medium.

Assessment of DNA damage

DNA-damage was assessed in terms of apoptosis and micronuclei formation in circulating lymphocytes. DNA fragmentation was assessed in plasma-rich leukocyte layer just prior to separation of blood components. These measurements were proceeded as follows:

Apoptosis

Lymphocytes were subjected to Histopaque separation, fixed with 70% ethanol for 1 hr and inspected by the fluorescent microscope after being incubated for 15 min. at 37°C with 10-μg RNA-ase and stained with 10-μg propidium iodide (Ioannou and Chen , 1996)

Micronuclei frequency

According to standard method using the cytokinesis block micronucleus assay (Fenech and Morely, 1985), where a duplicate of 1 ml of whole blood was cultured, and proliferation was stimulated by the addition of phytohaemaglunin (5 μg / ml) into 15 ml sterile plastic round bottom tubes containing media 199, 20% fetal calf serum and antibiotic. Cells were incubated for 72 hr with addition of cytochalasin B (3 μg / ml) 48 hr before harvesting. Twenty-four hours after the addition of cytochalasin B, cells were

collected and treated with 0.8% sodium citrate for 3-5 min and then fixed in methanol: acetic acid (5:1). Fixed cells were dropped gently onto clean microscope slides, air dried and stained with 4% Giemsa using standard protocols. Slides were scored at 100x magnification using a Leica Biomed microscope (Leica Lasertechnik GmbH, Heidelberg, Germany). The frequency of micronuclei in circulating lymphocytes was calculated according to standard criteria (Fenech, 1993).

DNA fragmentation

The DNA fragmentation assay was performed as described by Ioannou and Chen (1996). Syringes were allowed to stand in upright position for about two hours and by bending the needle the plasma leukocyte rich layer was aspirated before forming the buffy coat. The plasma leukocyte-rich layer was mixed with cell lyses buffer (0.1% Triton X-100, 5 mM Tris-HCL, pH 8, 20 mM EDTA) and centrifuged. Small fragments of DNA were gradient separated from the precipitate with equal volumes of 2 M NaCl and 5% polyethylene glycol 5000 in ethyl ether cooled and centrifuged. The supernatants were removed and mixed with an equal volume of Hoechst 33258 in phosphate buffered saline (PBS) and incubated for 20 minute at room temperature. Fluorescence was determined at 360 nm excitation and 460 nm emissions. Total DNA was estimated after extracting DNA from 0.5 ml plasma leukocyte-rich layer using Takara GenTLE DNA extraction kit (Takara Biomedicals, USA). DNA standards were prepared at concentrations of 100, 200, 500, 1000, 2000 and 5000 ng/ml. 0.5 ml of a standard or extracted DNA (ng/ml) was mixed with 0.5 ml freshly prepared Hoechst dye reagents and placed in the dark for 10 minutes. Fluorescence was determined at 360 nm excitation and 460 nm.

Statistical methods

Statistical analysis was carried out using the Statistical software for Windows. Data presented as means (SD). Differences among groups were tested using the student t test.

RESULTS

DNA fragmentation percentage

Percentage of DNA fragmentation per total DNA (fig. 3) in combined plasma and leucocytes showed a significant increase among total athletes when compared to controls (mean = 3.8 % $\pm$ 0.25 vs. 1.1 % $\pm$ 0.13, $p < 0.001$). Smoking enhances DNA fragmentation as exhibited by the significant increase in DNA fragmentation percentages of in both smokers groups of athletes and non-athletes compared to non-smoker athletes (mean = 4.8 % $\pm$ 0.41 vs. 1.7% $\pm$ 0.09, $p < 0.001$) and non-athletes (mean = 3.42% $\pm$ 0.23 vs. 0.7% $\pm$ 0.03, $p < 0.001$), respectively. Also, exercise enhances DNA fragmentation as exhibited by the significantly increased levels of DNA fragments in non-smoker athletes compared to non-smoker non-athletes (mean = 1.7% $\pm$ 0.09 vs. 0.7% $\pm$ 0.03, $p < 0.001$). In addition, the mean of DNA-fragmentation of smoker athletes compared to smoker non-athletes was significantly high (mean = 4.8 % $\pm$ 0.41 vs. 1.42 % $\pm$ 0.23, $p < 0.001$).

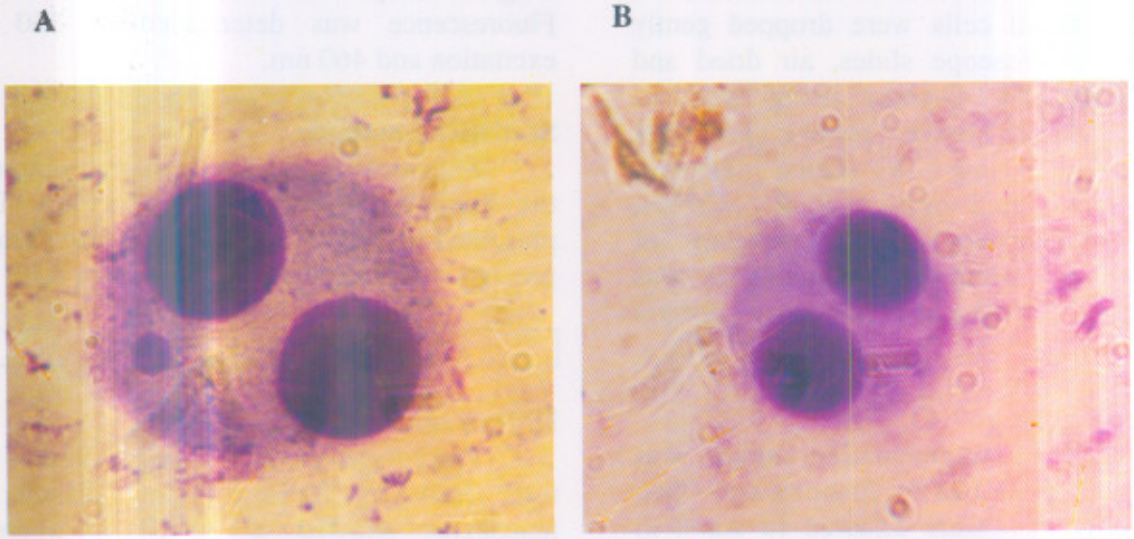

Fig. (1): Cytokinesis blocked binucleated lymphocytes with (A) and without (B) a micronucleus.

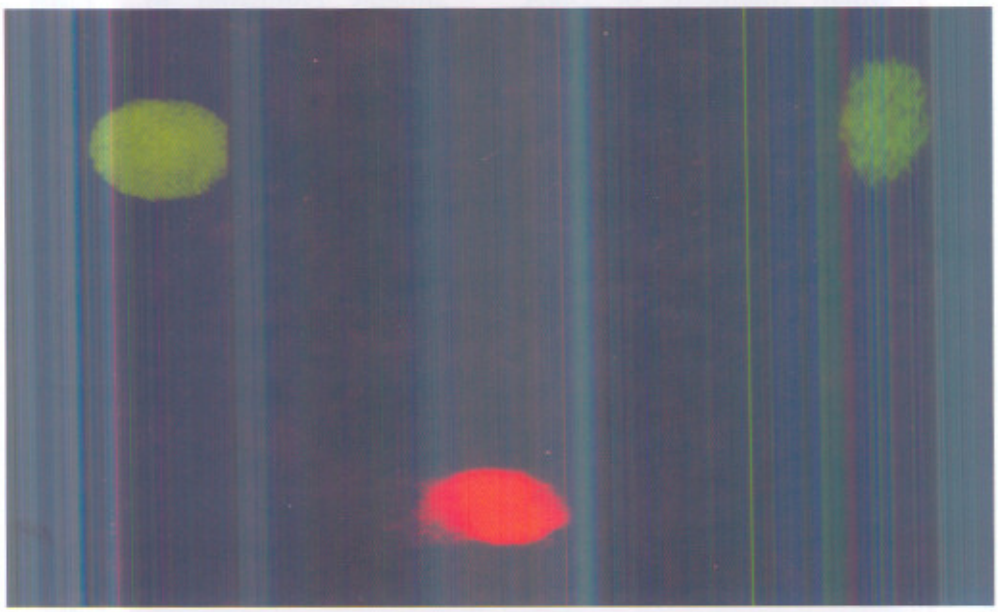

Fig. (2): A double technique stained lymphocytes. Green fluorescing cells are the viable cells stained with fluorecine diacetate. Apoptotic cell are fluorescing red.

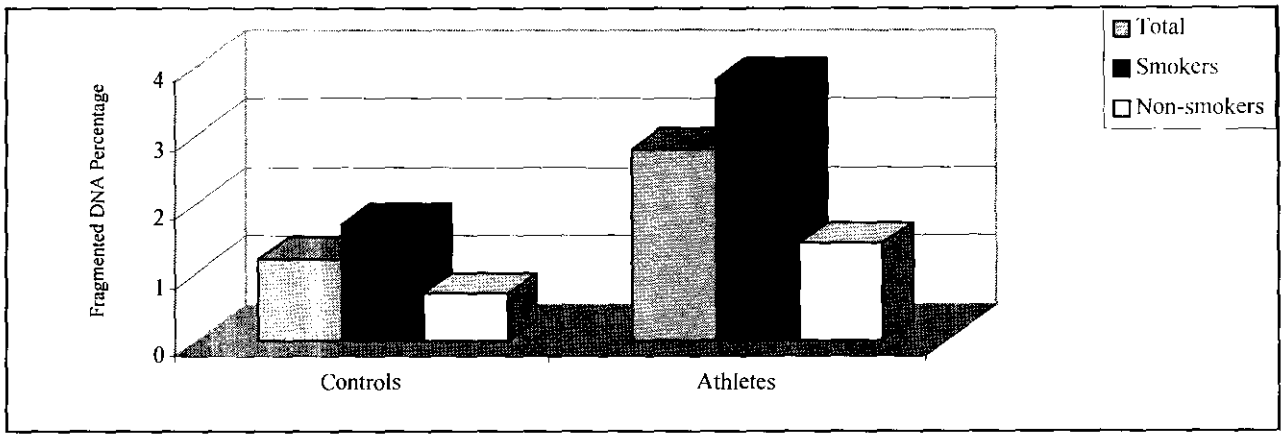

Fig. (3): DNA fragmentation percentages among smoker and non-smoker and total athletes compared to smoker and non-smoker and total non-athletes.

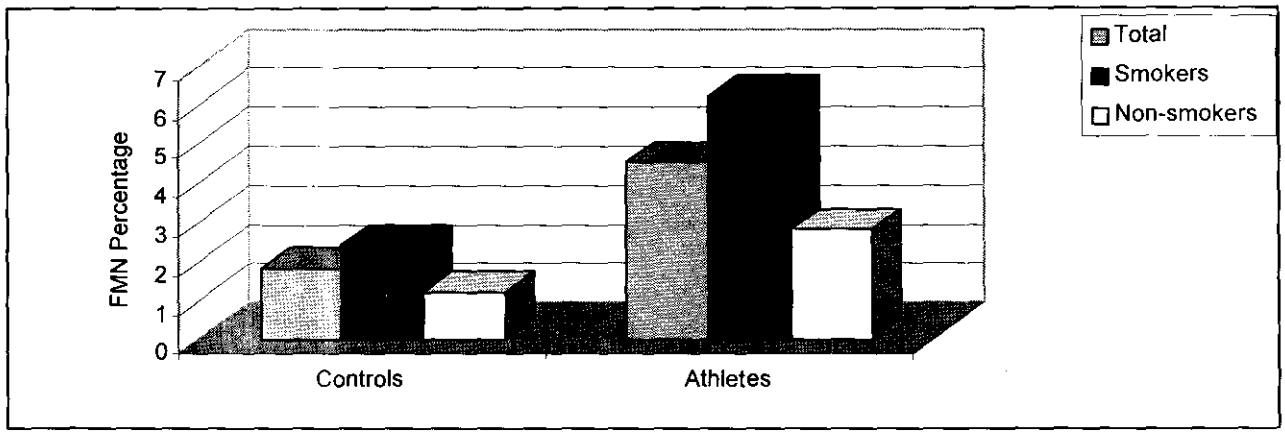

Fig. (4): The frequencies of micronuclei (FMN) among smoker and non-smoker and total athletes compared to smoker and non-smoker and total non-athletes.

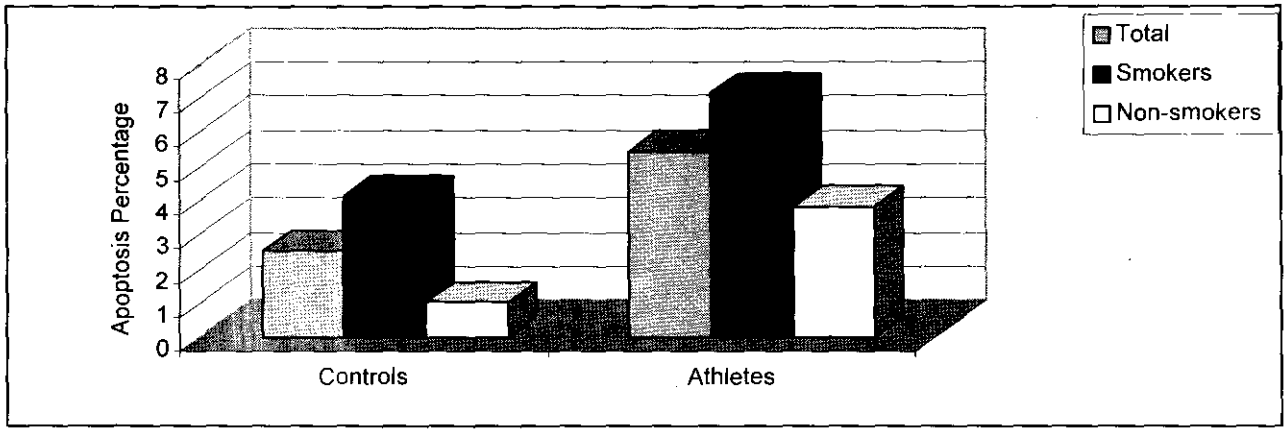

Fig. (5): Apoptosis percentages in circulating lymphocytes among smoker and non-smoker and total athletes compared to smoker and non-smoker and total non-athletes.

Apoptosis in circulating lymphocytes

Apoptosis in circulating lymphocytes showed significant increase among total athletes when compared to total non-athletes (mean = 5.4% ± 1.5 vs. 2.5% ± 1.1, $p < 0.001$). There was significant increase in the percentage of apoptotic cells in circulating lymphocytes of both smoker groups of athletes and non-athletes compared to non-smoker athletes (mean = 7.1% ± 3 vs. 3.8% ± 0.5. , $p < 0.001$) and non-athletes (mean = 4.1% ± 1.2 vs. 1% ± 0.2, $p < 0.001$), respectively. Also exercise enhances apoptosis as exhibited by significantly increased apoptotic percentage of circulating lymphocytes non-smoker athletes compared to non-smoker non-athletes (mean = 7.1% ± 3 vs. 4.1% ± 1.2, $p < 0.05$) and smoker athletes compared to smoker non-athletes (mean = 3.8% ± 0.5 vs. 1% ± 0.2, $p < 0.001$).

Frequency of micronuclei

FMN percentage in circulating lymphocytes showed significant increase among total athletes when compared to total non-athletes (mean = 4.1% ± 1 vs. 2.3% ± 1.1, $p < 0.001$). There was significant increase in FMN in circulating lymphocytes of both smoker group of athletes and non-athletes compared to non-smoker athletes (mean = 6.6% ± 1.8 vs. 1.4% ± 0.3, $p < 0.001$) and non-athletes (mean = 3.4% ± 1.2 vs. 1.2% ± 0.2, $p < 0.001$), respectively. There was no significant difference in FMN in circulating lymphocytes of non-smoker athletes compared to non-smoker non-athletes (mean = 1.4% ± 0.3 vs. 1.2% ± 0.2, $p < 0.001$). Meanwhile, smoker athletes exhibited significantly higher levels of FMN when compared to smoker non-athletes (mean = 6.6% ± 1.8. vs. 3.4% ± 1.2, $p < 0.001$).

DISCUSSION

The present findings show that physical exercise induced oxidative stress due to high oxygen consumption, which appeared to raise apoptosis percentage and DNA fragmentation in plasma and circulating leucocytes. This stress affects muscle tissues as inflammation or injury, which in turn affects lymphoid tissue and blood cells. However, no severe toxicity is detected by exercise as known. In this concern we can say that cigarette smoking and physical exercises combined together, augment apoptosis and induces genetic toxicity to circulating lymphocytes. An adaptive response to exercises as exhibited by an increase in apoptotic percentage in athletes failed to protect the blood against the synergistic effect of oxidative damage induced dually by physical exercises and cigarette smoke.

Over the past several years, a number of studies indicated that acute physical exercise in rats and mice are associated with induction of apoptosis in thymocytes and splenocytes (Concordet and Ferry, 1993; Hoffman-Goetz *et al.*, 1998). Experimental studies on animals demonstrated that acute exercises induce an inappropriate induction of apoptosis in lymphoid tissue (Ferry *et al.*, 1991). It has been suggested that one consequence of physical exercises is muscle inflammation (Hellsten *et al.*, 1997; Smith, 1991; Tibell, 1995) and that the immune system is involved in the muscle-repair process after exercise-induced muscle injury (Tibell, 1995; Niess *et al.*, 1999; Donaldson *et al.*, 1995). Investigation on untrained human subjects after one single exercise resulted in DNA damage in peripheral leukocytes, as measured by single-cell gel electrophoresis (Fehrenbach and Northoff, 2001; Hartmann *et al.*, 1994) and flow cytometer (Hartmann *et al.*, 1995). The flow cytometry documented for the first

time lymphocyte apoptosis in 63% of lymphocytes immediately after exercise and 86.2% after 24 hours exercise. Mediating mechanisms were hypothesized to include exercise-associated oxidative stress (Niess *et al.*, 1996). Thus, the significant increase in apoptosis percentage in circulating lymphocytes observed in the present study, provides further proof to the previous studies, which indicated that exercise induces lymphocyte apoptosis in circulating lymphocytes. The values of apoptotic cell percentages observed in the present study are lower than those previously reported by flow cytometry (Hartmann *et al.*, 1995). This may be due to the fact that the recorded study was carried out after a single exercise performance, but the present study is carried out over athletes, who most probably have had developed an adaptive response to oxidative stress (Niess *et al.*, 1996). It is speculated that exercise-induced apoptosis is a normal regulatory process that serves to remove certain damaged cells without a pronounced inflammatory response, thus ensuring optimal body function. (Phaneuf and Leeuwenburgh, 2001; Mars *et al.*, 1998).

The mechanisms by which cigarette smoke induces genetic toxicity are multiple and well documented. Cigarette smoke is rich in many chemicals that directly forms adducts with DNA, which induces genetic toxicity. Of these, coal tar, aldehydes, pyrrolizidine products and benzopyrenes derivatives are present in the smoke, which induces irritation (Pryor *et al.*, 1998). These compounds have been shown to directly induce apoptosis *in vitro* experiments (Lei *et al.*, 1998). Cigarette smoke, also, directly causes oxidative damage to lung endothelial cells by inducing an inflammatory response and promoting the recruitment of inflammatory-immune cells such as neutrophils (Tsuchiya *et al.*, 1994). Also, the number and function of peripheral blood lymphocytes are decreased due to cigarette smoke as the latter induces the expression of FAS mechanism (Bijil *et al.*, 2001), which is contributed to the fact that cigarette smoke also induces the production of oxygen free radicals whose increase in concentration leads to an induction of programmed cell death to the surrounding cells (Wang *et al.*, 1994; Buttke and Sandstorm, 2000). The latter explains both the significant increase in both apoptosis and FMN observed in smokers whether athletes or non-athletes when compared to non-smokers. Previous results carried out *in vitro* (Vijayalaxmi and Evans, 1982; Stenstrand, 1985) and *in vivo* (Ammenheuser *et al.*, 1997; Surh and Sprent, 1994) support the finding that cigarette smoke is genotoxic as in the present study, which showed increased levels of FMN among smokers in general, compare to non-smokers whether athlete or non-athlete.

Programmed cell death (apoptosis) is a normal physiological process that occurs during embryonic development as well as in the maintenance of tissue homeostasis, including thymocyte selection (Kitamura *et al.*, 1998). Inappropriate induction of apoptosis, however, has broad-ranging pathological implications. For example, it is associated with Alzheimer's disease (Lorenzen *et al.*, 1997), Hodgkin's lymphoma, acquired immune deficiency syndrome and cancer (Wang *et al.*, 1998). Thus, an increase in the elimination of genetically damaged cells by apoptosis represents an adaptive response mechanism that reduces the risk of cancer and other genetically induced diseases from exposure to radiation or other DNA damaging agents (Cregan *et al.*, 1999). It is speculated that exercise-induced apoptosis is a normal regulatory process that serves to remove certain damaged cells without a pronounced inflammatory response, thus ensuring optimal body performance (Cregan *et al.*, 1999).

Accordingly, non-smoker athletes exhibited significantly higher values of DNA fragmentation percentage and apoptotic percentage in circulating lymphocytes but showed no significant differences in FMN, when compared to non-smoker non-athletes. Concurrently, smoker athletes, although they exhibited significantly higher values of DNA fragmentation percentage and apoptotic percentage in circulating lymphocytes when compared to non-smoker athletes and to smoker non-athletes, still. exhibited the significantly highest value of FMN, indicating that cigarette smoke and exercise together exert a synergistic DNA damaging effect, which is beyond the adaptive endogenous antioxidant mechanism and apoptotic mechanisms, which eliminate genetically affected cells in the body system..

It may be concluded that exercise .and cigarette smoke individually induces an oxidative stress that imposes a genetic damage, which is eliminated by an apoptosis induced adaptive response. Exposure to both oxidative stress-inducing agents, results in a genetic damage, that is beyond complete elimination.

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الملخص العربي

قياس الضرر في المادة الوراثية في الرياضيين المدخنين و غير المدخنين

سهير قراعة*، نكية أحمد فتحي و عمرو فاروق سلام*****

*معمل المظفرات البيئية، المركز القومي لبحوث و تكنولوجيا الإشعاع

**قسم علوم الصحة الرياضية. كلية التربية الرياضية، جامعة حلوان

***مديرية الشباب و الرياضة

التكسير في شريط المادة الوراثية و موت الخلية المنظم و معدل تكرار النوية الصغيرة تم قياسه في عدد أربعين رياضي غير مدخنين و خمسة و عشرين رياضي من المدخنين. هذا و قد تمت مقارنة هؤلاء بعدد عشرين مدخن و عشرين غير مدخن من غير الرياضيين متقاربين في السن و الظروف الاجتماعية كمجموعة ضابطة. و قد أظهرت النتائج زيادة معنوية في مستوي التكسير في شريط المادة الوراثية و موت الخلية المنظم في الأفراد الرياضيين عامة بالمقارنة بغير الرياضيين، و أيضا في المدخنين عند مقارنتهم بغير المدخنين عامة حيث سجل أعلي مستوي للتكسير في شريط المادة الوراثية و موت الخلية المنظم في الرياضيين المدخنين و تلاهم في ذلك الأشخاص المدخنين غير الرياضيين. معدل تكرار النوية الصغيرة سجل أعلي ارتفاع ذو دلالة جوهرية في الرياضيين المدخنين و تلاهم في ذلك الأشخاص المدخنين غير الرياضيين و لم تكن هناك فروق جوهرية في معدل النوية الصغيرة بين الرياضيين غير المدخنين بالمقارنة بغير الرياضيين. هذه الدراسة تبرز الآثار السيئة المترتبة عن التدخين علي المادة الوراثية.