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Effects of Computer Use on Human Salivary Oxidant/Antioxidant Status

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Abstract: Computers are widely used in recent years but their effects on human health completely are known. The aim of this study was to investigate possible effects of computer use on human salivary oxidant / antioxidant system. Ten subjects were included in the study. Three saliva samples were taken from the subjects. First was obtained before computer use, second and third samples were obtained two and four hours after they started computer use. Malondialdehyde (MDA) levels and superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GSH-Px) and adenosine deaminase (ADA) enzyme activities were measured in each saliva sample. Malondialdehyde (MDA) level (mean ± standard deviation) was found to increase in the saliva samples obtained after computer use. SOD and ADA activities decreased but, CAT activity increased during this period. Our results show that computer-released radiation causes changes in enzymatic antioxidant defense system and, leads to oxidant stress in saliva samples from subjects. It has been suggested that subjects who use computer intensively should be consumed more antioxidant foods in order to prevent probable oxidation reactions in their bodies.

Key words: Oxidant/Antioxidant Status, Computer Using

INTRODUCTION

Computers are widely used in the office workplace, which provide efficiency, competitive advantages, and the ability to carry out work that would be impossible or less effective without their use. They also provide new methods for managing work and tracking the behavior of employees. Computerized jobs are more sedentary, require more cognitive processing and mental attention, and require less physical expenditure of energy. Yet the production demands of these jobs are often high, with constant work pressure and little decision making possibilities. Many jobs that require heavy daily computer use have been found to be stressful[1,2,3,4,5]. Recently, potential adverse health effects of long-term computer use have been attracted attention. Renewed concerns about radiation, combined with reports of newly-recognized "repetitive stress injuries" such as carpal tunnel syndrome, have led some to call for regulation in the workplace and others to rearrange their offices and computer labs. A lot of people are spending more time doing more tasks with computers and faculty, students and staff at colleges and universities have some of the most computer-intensive work styles in the world[6].

Recent studies indicated that reactive oxygen species (ROS) such as superoxide anion, which is predominantly generated by the mitochondria; hydrogen peroxide produced from superoxide anion by the action of superoxide dismutase , and peroxynitrite, generated by the reaction of superoxide anion with nitric oxide have been implicated in tissue injury. The main ROS that have to be considered are oxygen species ones [7,8]which are scavenged by some enzymes such as SOD, GSH-Px and CAT. When the balance of antioxidants is outweighed by excessively produced ROS as a result of overproduction of oxygen radicals, inactivation of detoxification systems, consumption of antioxidants, and failure to adequately replenish antioxidants in tissue, these endogenous antioxidative defences system are likely to be perturbed. It has been shown in numerous studies that ROS are directly involved in oxidative damage of cellular macromolecules such as lipids, proteins, and nucleic acids in tissues. Malondialdehyde is a breakdown product of the major chain reactions leading to oxidation of polyunsaturated fatty acids and thus serves as a reliable marker of oxidative stress[8,9]. Adenosine deaminase is known as an important enzyme that participates in the degradative pathways of adenosine monophosphate[10]. When ADA fails to catalyze the deamination of adenosine, those compounds that accumulate are readily converted into their respective nucleotides. Deoxyadenosine triphosphate (dATP) is one of the nucleotides generated in this way. In ADA deficiency, deoxyadenosine accumulates intracellularly as dATP which has been recognized as the toxic metabolite in the

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immunodeficiency disease associated with ADA deficiency. This compound is a potent inhibitor of DNA replication because it prevents the synthesis of deoxyribonucleotides from ribonucleotides by interference with ribonucleotide reductase[11].

MATERIALS AND METHODS

Ten subjects were included in the study. Saliva samples were taken from the subjects three times, before they started computer using, and two and four hours subsequently. MDA levels and SOD, CAT and GSH-Px enzyme activities were measured in the samples. Malondialdehyde levels were measured by the thiobarbituric acid reactive substances (TBARS) method[12]. Superoxide dismutase activity was measured as described[13]. In this method, one unit for SOD activity was expressed as the enzyme protein amount causing 50 % inhibition in nitroblue tetrazolium (NBT) reduction rate. CAT activity was determined by measuring decrease of H_2O_2 absorbance at 240 nm as described[14]. GSH-Px activity was measured by following changes in NADPH absorbance at 340 nm as described[15]. In the activity calculations, extinction coefficients of H_2O_2 and NADPH were used for CAT and GSH-Px respectively. ADA activities of the saliva samples were measured colorimetrically by using the method of Giusti[16].

The paried t test was used for the statistical analysis.

RESULTS AND DISCUSSION

Results are given in the table 1, which demonstrate that computer use has caused oxidant stress and peroxidation reactions (Increased MDA levels) in saliva. MDA level (mean ± standard deviation) was found to increase in the saliva samples obtained after computer use. SOD and ADA activities were found to decrease but, CAT activity increase during this period.

Table 1: At initial, 2nd and 4th hours salivary MDA values, CAT, SOD, GSH-Px and ADA activities (Mean ± Standard Deviation):

	Initial	2nd Hour	4th Hour
MDA (nmol/ml)	0.27±0.09	0.56±0.08	1.05±0.06**
CAT (IU/ml)	10.64±1.55	11.82±1.16	17.48 ±3.35**
SOD (U/ml)	8.38±0.85	5.48±1.79*	6.95±1.46**
GSH-Px (IU/ml)	0.33±0.02	0.33±0.02	0.34±0.02
ADA (mIU/ml)	3.41±1.21	1.01±0.01*	0.57±0.01**

* Initial vs. 2nd hour: $p < 0.05$; Paired t test
** Initial vs. 4th hour: $p < 0.05$; Paired t test

DISCUSSION

Computer users suffer from some complaints such as eyestrain, headaches, general malaise, and other visual and musculoskeletal problems[6,17]. It has reported that radiation has emitted from the backs and sides of some terminals. The recent National Institute of Occupational Safety and Health (NIOSH) study is reassuring, but some caution still seems prudent[6]. For at least a decade, concerns have been raised about possible effects of radiation from video display terminals, including cancer and miscarriages. Earlier fears about ionizing radiation, such as X rays, have been laid to rest, since these rays are blocked by modern glass screens. More recent controversy surrounds very low frequency (VLF) and extremely low frequency (ELF) electromagnetic radiation produced by video displays' horizontal and vertical deflection circuits, respectively[6]. Researchers have reported a number of ways that electromagnetic fields can affect biological functions, including changes in hormone levels, alterations in binding of ions to cell membranes, and modification of biochemical processes inside the cell. It is not clear, however, whether these biological effects translate into health effects[6]. Several epidemiological studies have revealed a correlation between VDT use and adverse pregnancy outcomes, whereas other studies found no effect[6,18]. It is pointed that magnetic field strength diminishes rapidly with distance. A final form of radiation, static electric, can cause discomfort by bombarding the user with ions that attract dust particles, leading to eye and skin irritations[6].

Elevated ROS concentrations lead to oxidative stress that causes molecular damage to vital structures and functions. A lot of endogenous factors like inflammatory, exercise, psychological stress and exogenous factors like food, alcohol, cigarette smoke, environmental pollution and radiation cause the susceptibility to oxidative stress[19,20]. Free radicals are very reactive and unstable molecular fragments that have an unpaired electron and they can produce new free radicals by means of chain reactions. This molecules although formed as a result of normal biochemical processes, sometimes they may be damaging and interact with all the macromolecules including lipids, nucleic acids and proteins. There are some mechanisms to neutralize their effects, two of them nutritional and endogenous enzymatic antioxidant defenses that generally hold the production of free radicals and prevent oxidant stress and subsequently tissue damage[20,21]. As a consequence, free radical attack on unsaturated fatty acids of lipid structures leading to lipid peroxidation, and damaging effects on proteins may occur. Lipid peroxidation products e.g.

malondialdehyde has been taken as a biomarker for oxidative stress in biological system[20,22]. This circumstance can lead to 'oxidative stress' i.e. a series of peculiar and potentially damaging biochemical reactions[20,23]. Particularly susceptible to oxidative damage by free radicals are the polyunsaturated fatty acid acyl chains of phospholipids, which lead to lipid peroxidation. Uncontrolled lipid peroxidation is a toxic process resulting in the deterioration of biological membranes[19,20,23]. In a study shown that ROS may generate various lesions in DNA such as base modifications, degradation products of deoxyribose, chain breaks. These various lesions have been characterized and it is possible to quantitate them in the DNA of cells which have been irradiated or treated by free radical generating systems. The biological properties of the bases modified by ROS have been established. For example C8-hydroxyguanine (8-oxoG) is promutagenic since, if present in DNA during replication, it leads to incorporation of dAMP residues, leading to transversion mutation (GC-->TA)[20,24]. The antioxidant enzymes include, SOD, which transform the superoxyde anion into hydrogen peroxyde which in turn will be destroyed by peroxysomal catalase or by various peroxydases and GSH-Px that scavenge hydrogen peroxide and prevent accumulation of the hydroxyl radical[24]. Radiofrequency waves are a very important part of electromagnetic spectrum with respect to their applications and possible health consequences. Epidemiological studies remain inconclusive with regard to the health effects of prolonged exposure to electromagnetic fields [20,25,26,27,28].

The metabolic basis of the immunodeficiency is likely related to the sensitivity of lymphocytes to the accumulation of the ADA substrates, adenosine and 2'-deoxyadenosine. Investigations using ADA-deficient mice have provided compelling evidence to support the hypothesis that T and B cells are sensitive to increased concentrations of 2'-deoxyadenosine that kill cells through mechanisms that involve the accumulation of dATP and the induction of apoptosis. In addition to effects on the developing immune system, ADA-deficient humans exhibit phenotypes in other physiological systems including the renal, neural, skeletal, and pulmonary systems[29].

The data presented here suggest that computer use generates free radicals and causes changes in the antioxidant enzyme activities, which lead to oxidant stress and peroxidation in saliva and possibly in the other parts of the body. Our results also show that computer use causes significant decrease in the ADA activity in the saliva, possibly again through free radical activities, which may result in immune deficiency in the subjects using computer intensively. It has been supposed that nutritional support of antioxidant foods including some vegetables and fruits might be helpful in order to prevent or eliminate toxic potentials of computer radiation in particular in subjects using it more intensively.

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