Anti-genotoxic effect of vitamin E administration in acrylamide intoxicated rat bone marrow cells

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ABSTRACT

This study aims to investigate the effect of oral administration of vitamin E (VE, 300 mg/kg, oral intubation daily for 21 consecutive days) to normal and acrylamide-intoxicated (ACR, 25 mg/kg daily for 21 consecutive days) rats. Bone marrow was used to monitor the changes in micronucleus (MN) incidence and modulating the genotoxic effects of gavaged ACR. Treatment of rats with acrylamide (ACR) significantly elevated the frequency of bone marrow MN. Moreover, VE administration decreased the MN frequency to be near the control values; suggesting the ameliorative effect of VE against ACR toxicity.

Keywords: *Acrylamide; vitamin E; micronucleus test.*

INTRODUCTION

Acrylamide is a white odorless crystalline solid at room temperature with a molecular formula of C3H5No (Grivas *et al.*, 2002). Commercially ACR is produced to be used as a chemical intermediate in the production and synthesis of polyacrylamides (Dorman, 2000; Paulsson *et al.*, 2001; Friedman, 2003 and Nordin *et al.*, 2003). Likewise, ACR used as a control agent in oil-drilling processes, as binders and retention aids in the pulp and paper industry (Paulsson *et al.*, 2001 and Friedman, 2003). On the other hand, ACR is a component of tobacco smoke, which is formed by heating of biological material. Therefore, smoking could potentially be a source of ACR in indoor air (Grivas *et al.*, 2002). Moreover, ACR has been reported to be present in plant material like potatoes, carrots, radish, lettuce, Chinese cabbage, parsley, onions, spinach and rice paddy (Arikawa and Shiga, 1980), in sugar (Schultzova and Tekel, 1996) and olives (Friedman, 2003). Recently, Taubert *et al.* (2004) documented that ACR is formed during the cooking of starchy foods at high temperature. The first evidence demonstrating that ACR induced chromosomal effects was reported by Shiraishi (1978). Also, Dearfield (1988) reported that ACR induced significant increases in aberrations (breaks, fragments, dicentrics, rings and minute chromosomes were scored) and also increased the incidence of aneuploidy. Chromosomal aberrations, sister chromatid exchanges and mitotic disturbances were observed *in vitro* (Adler *et al.*, 1993). Significant increases in micronuclei frequency were also reported in reticulocytes and splenic lymphocytes from mice exposed to ACR at doses of 50mg/kg or more *via* intraperitoneal injection (Kligerman *et al.*, 1991 and Russo *et al.*, 1994). Yang *et al.* (2005) observed micronuclei and chromosomal aberrations at high concentrations of ACR. Likewise, acrylamide increased the

micronucleus frequency in bone marrow cells in mice following doses of ≥ 25 mg/kg (two doses) or a single dose of 50 mg/kg (Adler *et al.*, 1988; Cihak & Vontorkova, 1988; Knaap *et al.*, 1988 and Cihak and Vontorkova, 1990). Acrylamide-DNA adduct formation was saturable, whereas the formation of most glycidamide-DNA adducts was dose dependent. The mutagenicity of ACR in human and mouse cells is based on the capacity of its epoxide metabolite glycidamide to form DNA adducts (Besartinia and Pfeifer, 2004). Moreover, Blasiak *et al.* (2004) suggested that ACR generated reactive oxygen species in lymphocytes.

Vitamin E is a fat-soluble vitamin (Friedrich, 1988). The basic structure of VE is a hydroxylated ring system (chromanol ring) and an isoprenoid side chain. Both the isoprenoid side chain and the aromatic ring have been implicated in the antioxidant function of the vitamin (Tapan and John, 1996). Vitamin E can protect critical cellular structures against damage from both free radicals (such as peroxy radical, hydroxyl radical, and superoxide) and from oxidation products such as (malondialdehyde and hydroxynonenal), which also have deleterious effects (Tappel, 1973 and Erin *et al.*, 1984).

Vitamin E breaks the chain of free radical formation by reacting with the free radicals and converts them to a non-harmful form (Friedrich, 1988; Packer, 1994 and Tapan and John, 1996). Vitamin E, as an important antioxidant, plays a role in inhibition of mutagen formation, and repair of membranes and DNA. Therefore, it has been suggested that vitamin E may be useful in cancer prevention (Newmark and Mergens, 1981; Bright-See and Newmark, 1983 and London *et al.*, 1985).

The aim of this study was to investigate the effect of vitamin E on the bone marrow cells of rat.

MATERIALS AND METHODS

Handling of animals

The experimental animals used in this study were adult male albino rats *(Rattus norvegicus)* weighing 100-140 g. They were housed under normal environmental conditions of temperature and humidity. Animals were kept under the normal light-dark rhythm. Food and water were provided *ad libitum.*

Animals grouping

1- Negative control group (-ve C, 12 animals)

Animals of this group received a daily oral administration of corn oil for 21 consecutive days.

2- Acrylamide group (ACR, 12 animals)

Animals of this group received a daily oral administration of acrylamide (25 mg/kg b.wt.) for 21 consecutive days (Dixit *et al.*, 1981). ACR was purchased from ICN Biomedicals Inc. in the form of white powder. Acrylamide was dissolved in distilled water.

3- Vitamin E group (VE, 12 animals)

Animals of this group received a daily oral administration of vitamin E (300 mg/kg b.wt.) as described by El-Nahas *et al.* (1993) for 21 consecutive days. Vitamin E was purchased from Sigma-Aldrich in the form of α-tocopherol and was dissolved in corn oil.

4- Acrylamide and vitamin E group (ACR and VE, 12 animals)

Animals of this group received a daily oral administration of ACR (25 mg/kg b.wt.) for 21consecutive days. One hour later, these animals received an oral administration of VE (300 mg/kg b.wt.).

5- Oil group (solvent of VE, 12 animals)

Animals of this group received a daily oral administration of oil for 21 consecutive days.

6- Positive control group (4 animals)

Animals of this group received a single oral administration of cyclophosphamide (CP) (20 mg/kg b.wt.) obtained from Batrex

Oncology GmbH, 60314 Frankfurt, Germany as described by Gollapudi and McFadden (1995).Animals of all groups; control, ACR, VE, ACR and VE, oil and the positive control were decapitated after 7, 14, and 21 days.

Tissue samples

Bone marrow smears were produced by removing the adherent muscle from one femur from each animal, cutting off the proximal end and extracting the bone marrow in fetal calf serum. The bone marrow-lump was gently aspirated by flushing the cells into the fetal calf serum in the form of fine suspension. Slides were stained with May-Gruenwald / Giemsa solution for 20min. (Gimmler-Luz *et al.,* 1999), and then they were rinsed in distilled water and left air-dry. Finally, slides were mounted with DPX. Microscopic analysis was done by scoring 1000 polychromatic erythrocytes (PCEs) in addition to the number of normochromatic erythrocytes (NCEs). The frequencies of both micronucleated polychromatic erythrocytes (MNPCEs) and micronucleated normochromatic erythrocytes (MNNCEs) were determined.

Statistical analysis of the data

The statistical analysis of the cytogenetical data was carried out by using one way analysis of variance (ANOVA) followed by Duncan's test (1955). The statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 10. Regression analysis and correlation coefficients were performed using Microsoft excel.

RESULTS AND DISCUSSION

The changes induced in the frequencies of micronucleated polychromatic and normochromatic erythrocytes (MNPCEs and MNNCEs) and in the percent of polychromatic erythrocytes (%PCEs) in bone marrow cells of the rats treated orally with acrylamide (ACR) alone (25 mg / kg b.w.) and in combination with vitamin E (VE) (300 mg / kg b.w.) for 7, 14 and 21 days were presented in Tables (1, 2 and 3), respectively.

Statistically increase (at $P< 0.01$) in the frequencies of MNPCEs was observed after administration of ACR alone when compared to that of the –ve control group at the three treatment periods used (7, 14 and 21 days).

On the other side, coadministration of ACR and VE induced significant reduction ($P < 0.01$) in the frequencies of MNPCEs in the different groups when compared with their corresponding ACR groups. However, the frequency of MNPCEs in the group coadministrated with ACR and VE for 21 days was still significantly higher than that of the –ve control group.

Insignificant changes in the frequencies of MNNCEs were observed in the different treated groups. Also, each of VE and its solvent (oil) did not induce significant changes in MNPCEs or MNNCEs.

Regression analysis indicated a strong positive correlation between MNPCEs/PCEs and days of treatment with ACR alone (R^2 =0.9959) or in combination with VE (R^2 =0.9494) (Figs. 1 and 2). A moderate positive correlation (R^2 =0.6202) was observed between MNNCEs/1000NCEs and days of administration of ACR alone (Fig. 1).

The ratio between PCEs and NCEs was expressed as % PCEs. A significant increase in % PCEs was recorded in all groups treated for 7 days when compared with that of the –ve control group (Table 1).

Table (1): Frequency of micronucleated bone marrow cells of rats after different treatments for 7 days.

Treatment	Dose (mg/kg)	Number of examined PCEs	MNPCEs/1000 PCEs	MNNCEs/1000 NCEs	%PCEs
- ve control	-	4000	0.25	0.00	67.80
ACR	25	4000	5.25^{a**}	1.20	81.40^{a**}
VE	300	4000	1.25	0.00	79.00^{d**}
ACR + VE	25+300	4000	1.25^{b**}	1.55	$75.30^{a**b*c\ d}$
VE Solvent (Oil)	-	4000	1.00	0.70	71.70^{a*}
+ ve control (CP,24 h.)	20	4000	7.75^{a**}	0.00	74.40^{a**}

PCEs: Polychromatic erythrocytes, MNPCEs: Micronucleated polychromatic erythrocytes.
MNNCEs: Micronucleated normochromatic erythrocytes, ACR: Acrylamide, VE: Vitamin E.
CP: Cyclophosphamide, a: Change from –ve control group, b: Change from ACR group, c: Change from VE group, d: Change from oil group, *: Statistically significant at P<0.01, **: Statistically significant at P<0.001.

Table (2): Frequency of micronucleated bone marrow cells of rats after different treatments for 14 days.

Treatment	Dose (mg/kg)	Number of examined PCEs	MNPCEs/1000 PCEs	MNNCEs/1000 NCEs	%PCEs
- ve control	-	4000	0.75	0.96	65.90
ACR	25	4000	6.50^{a**}	6.70	89.50^{a**}
VE	300	4000	0.25	0.38	62.20^{d**}
ACR + VE	25+300	4000	2.00^{b**}	0.00	$75.20^{a**b**c**}$
VE Solvent (Oil)	-	4000	0.75^{b**}	0.00	73.70^{a**}
+ ve control (CP,24 h.)	20	4000	7.75^{a**}	0.00	74.40^{a**}

PCEs: Polychromatic erythrocytes, MNPCEs: Micronucleated polychromatic erythrocytes.
MNNCEs: Micronucleated normochromatic erythrocytes, ACR: Acrylamide, VE: Vitamin E.
CP: Cyclophosphamide, a: Change from –ve control group, b: Change from ACR group, c: Change from VE group, d: Change from oil group, *: Statistically significant at P<0.01, **: Statistically significant at P<0.001.

Table (3): Frequency of micronucleated bone marrow cells of rats after different treatments for 21days.

Treatment	Dose (mg/kg)	Number of examined PCEs	MNPCEs/1000 PCEs	MNNCEs/1000 NCEs	%PCEs
- ve control	-	4000	0.50	0.00	64.90
ACR	25	4000	7.50^{a**}	5.87	78.60^{a**}
VE	300	4000	1.00	0.36	59.70^{a**d**}
ACR + VE	25+300	4000	$3.75^{a***b**c*}$	2.63	$68.30^{ab**c**d**}$
VE Solvent (Oil)	-	4000	0.75	0.13	78.90^{a**}
+ ve control (CP, 24 h)	20	4000	7.75^{a**}	0.00	74.40^{a**}

PCEs: Polychromatic erythrocytes, MNPCEs: Micronucleated polychromatic erythrocytes.
MNNCEs: Micronucleated normochromatic erythrocytes, ACR: Acrylamide, VE: Vitamin E.
CP: Cyclophosphamide, a: Change from –ve control group, b: Change from ACR group, c: Change from VE group, d: Change from oil group, *: Statistically significant at P<0.01, **: Statistically significant at P<0.001.

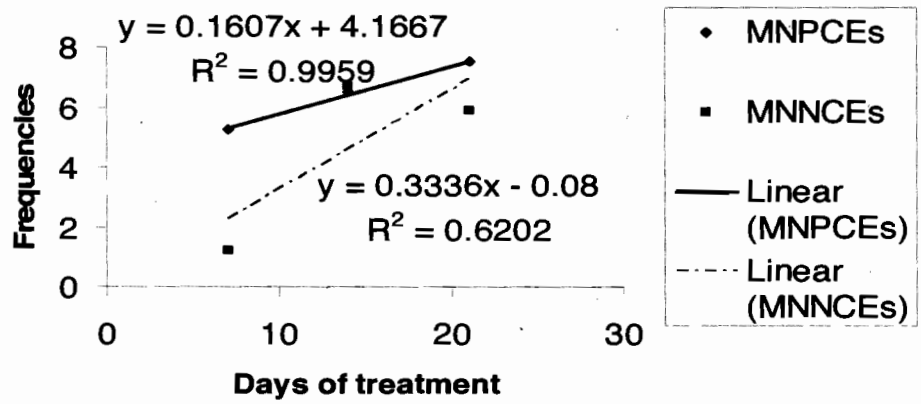

Fig. (1): Regression analysis of the relation between MNPCEs and MNNCEs frequencies and davs of treatment with ACR alone.

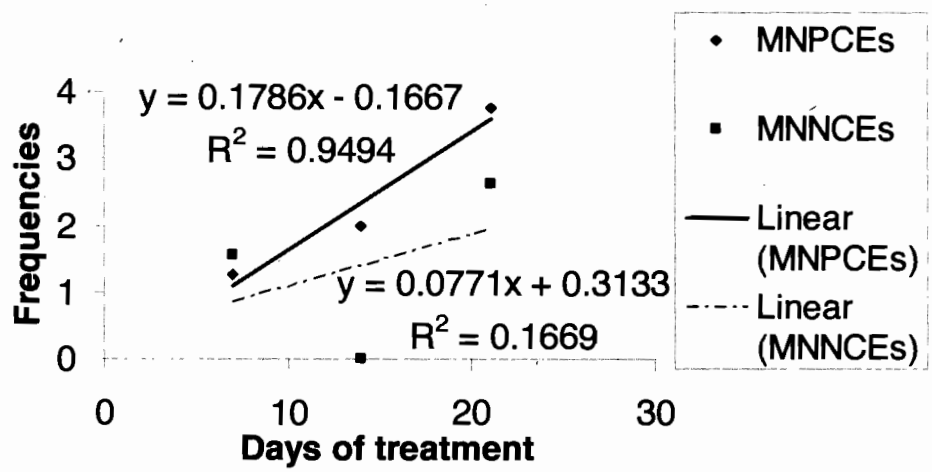

Fig. (2): Regression analysis of the relation between MNPCEs and MNNCEs frequencies and days of combined treatment with ACR and VE.

Also, Table (2) shows increased % PCEs in all groups treated for 14 days except the group treated with VE alone in which the % PCEs did not change from the –ve control level. The % PCEs increased significantly ($P < 0.01$) in the group treated with ACR alone for 21 days and decreased and reached the control level after combined treatment with ACR and VE (Table 3).In general, VE alone induced a significant reduction ($P < 0.01$) in % PCEs, while treatment with oil alone increased significantly ($P < 0.01$) the % PCEs.

The present study was planned to investigate the effect of oral administration of vitamin E on bone marrow of normal and intoxicated adult male albino rats. Since acrylamide was documented as mutagenic to somatic and germ cells of experimental

animals, thus the main objective of this study was to estimate the relative cytogenetic efficiency of ACR in rats; this was performed by studying the MN-induction as end-point reflecting chromosome damage *in vivo* as well as, the estimation the ameliorative effect of vitamin E on the bone marrow toxicity.

The present study demonstrated that acrylamide administration to male albino rats for 21 consecutive days induced a sharp and marked significant elevation of the bone marrow PCEs percentage (78.60 %) as well as the frequency of MNPCEs (7.50 ‰) *versus* the negative control values, while the frequency of MNNCEs (5.87‰) was non-significant when compared with the negative control group. These results are in agreement with (Adler *et al.*, 1988; Cihak and Vontorkova, 1988; Knaap *et al.*, 1988; Cihak & Vontorkova, 1990; EU, 2002; SCF, 2002 and Abramsson-Zetterberg, 2003).Adler *et al.* (1988) stated that ACR treatment had no effect on erythrocyte maturation or survival. The micronucleus frequency in bone marrow cells of mice treatment with 100 mg/kg of ACR was highest after 24 hr. Cihak & Vontorkova (1988) reported that single dose (100 mg/kg) of ACR increased the incidence of mice bone marrow MNPCEs significantly only at 24 hrs. Moreover, mice received (2x25, 2x50 and 2x100 mg/kg i.p.) showed an increment of the bone marrow micronuclei which was a dose dependent.Adler *et al.* (1988) attributed the positive micronucleus data probably to segregation and breakage of chromosome. They added that acrylamide has been reported to react in three different ways: first, it can undergo radical-mediated polymerization (Dearfield *et al.*, 1995). This process is best attained an aerobically (Dearfield *et al.* 1995 and Friedman, 2003). Second, as an α, β-unsaturated carbonyl compound, ACR can function as a "Michael acceptor" and undergo addition to thiol, hydroxyl or amino groups (Dearfield *et al.*, 1995). The thiol addition largely represents a detoxification pathway by yielding primarily acrylamide-glutathione conjugates as urinary excretable end products. However, it can also result in alkaylation of proteins, thereby, possibly causing epigenetic effects (Dearfield *et al.* 1995 and Friedman, 2003). Acrylamide has also been shown to bind to DNA in a similar "Michael type" addition process, however, with low reactivity (Dearfield *et al.*, 1995). Third, acrylamide can be metabolized to an epoxide derivative, glycidamide by cytochrome P-450 (Sumner *et at.*, 1999); being readily reactive toward DNA and other macromolecules (Dearfield *et al.*, 1995). Dearfield *et al.* (1995) suggested that acrylamide principal biological activity is due to adduction with proteins and alkaylation of DNA. The authors supposed that glycidamide form N-7-adduction of guanine which is the predominant adduct in rodent DNA *in vivo*, hence the DNA activity by glycidamide plays a prominent role for gene mutations.

Oral administration of vitamin E to normal rats showed a highly significant increment of PCEs percentage at the beginning of the experiment, while continuous administration of VE for 21 consecutive days induced a highly significant reduction of the percentage of PCEs *versus* oil group. The frequency of MN was non-significant as compared to oil group. In addition, the vitamin E administration after ACR-intoxication induced a sharp and marked significant reduction of the percentage of PCEs at all time intervals *versus* ACR-intoxicated group. Therefore, the ameliorative effect of VE as an antioxidant on ACR-administrated rats is clearly observed as VE seems to inhibit the increase of PCEs percentage as a result of ACR administration in rat bone marrow. These results are in agreement with Zamorano-Ponce *et al.* (2006) who reported that the pretreatment with an antioxidant such as *Aloysia triphylla* infusion

prior to ACR injection significantly reduces the capacity of ACR to induce genetic damage.

Vitamin E is an example of a phenolic antioxidant which donate a hydrogen atom from a hydroxyl (-OH) group on the ring structure to free radicals which then become unreactive. On donating the hydrogen atom, the phenolic compound itself becomes a relatively unreactive free radical because the unpaired electron on the oxygen atom is usually delocalised into the aromatic ring structure thereby increasing its stability (Scott, 1997), thereby, vitamin E can protect the cellular structures from the damage of free radicals and oxidation products (Tappel, 1973).

Moreover, vitamin E plays a role in the inhibition of mutagen formation, repair of membranes and DNA (Newmark and Mergens, 1981; Bright-See and Newmark, 1983 and London *et al.*, 1985). Goncharova and Kuzhir (1989) proposed that antioxidants may reduce clastogen and mutation induction chemically in at least three ways: (1) competition with the nucleophilic sites on DNA for an electrophilic clastogen or mutagen; (2) inhibition of pro-mutagen bio-activation by blocking the oxidation process and (3) reaction with electrophilic metabolites of a pro-mutagen.

Likewise, Blasiak *et al.* (2004) estimated the ameliorative effect of VE and other antioxidants on the induced toxicity by ACR on human lymphocytes, the authors implied that ACR generates reactive oxygen species as well as it induced oxidative DNA damage which may be reduced by the pretreatment of VE and other antioxidants.

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الملخص العربي

التأثير المضاد للسمية الوراثية للمعاملة بفيتامين "هـ" في خلايا نخاع عظام الجرذان المسممة بالأكريلاميد

أماني عبد المنعم تهامي وأميرة أنور بيومي

قسم علم الحيوان والحشرات-كلية العلوم-جامعة حلوان-مصر

تهدف هذه الدراسة إلى تقدير تأثير إعطاء فيتامين "هـ" عن طريق الفم (٣٠٠ مجم/كجم يوميا لمدة ٢١ يوماً متتالياً). استخدمت خلايا نخاع العظام لتتبع التغيرات في معدل الأنوية الدقيقة وتقدير التأثير السمي الوراثي للمعاملة بالأكريلاميد عن طريق الفم. أدت معاملة الجرذان بالأكريلاميد إلى ارتفاع ذي دلالة في معدل الأنوية الدقيقة في خلايا نخاع العظام. أيضاً أدت المعاملة بفيتامين "هـ" إلى خفض معدل الأنوية الدقيقة ليصبح حول القيم الطبيعية، مما يشير إلى التأثير المحسن لسمية الأكريلاميد بواسطة فيتامين "هـ".