

Clastogenic and Mutagenic Effect Induced by Some Fast Foods

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ABSTRACT

The present work done on three antipyretic drugs used in Egypt, and these drugs are Ibuprofen, Acetyl salicylate (Acetyl salicylic acid) and Alexopirine, these drugs are tested on white blood cells in human and bone marrow on bones of mice and cells chromosome Mice *Mus musculus* 2n=40 i.e *Vicia faba* and *Allium cepa*.

Many different concentrations are used.

The obtain result showed that different effect had been observed from these three different drugs from small type which represent on sister chromosome exchange and also big type represent on Bridge chromosome, Fragmentation and Replication.

Key words: Clastogenic, Mutagenic, Carcinogenic, Food.

INTRODUCTION

Genotoxic compounds in the diet represent a truly international problem. This problem was recognized in Japan and the United States of America from several years ago by the application of in vitro mutagenicity tests to prepared and unprepared components of our diet. Now new discoveries are appearing every day concerning genotoxic compounds in our food supply from all over the world. It seems likely that naturally occurring genotoxic compounds in the diet as well as genotoxic compounds formed during storage, preparation, and digestion of the food are important for the induction of cancer in humans and of hereditary changes in human germ cells. Other scientific data indicate that dietary constituents have the potential to modify the genotoxic effect of these compounds in humans. These findings indicate new possibilities for the prevention of human diseases of genotoxic origin through relevant changes in the composition of the diet. (Amara *et al.*, 2008).

With this background, and due to the rapid accumulation of new data in the field, it was approved by the Board of the International Association of Environmental Mutagen Societies that one of the satellite meetings of the Fourth International Conference on Environmental Mutagens in Scandinavia 1985 should cover the aspects of mutagens and carcinogens in the diet. However, formation of polycyclic aromatic

hydrocarbons (PAH) occurs during incomplete combustion of organic material. Although the mechanism of this formation is not fully understood, two principal pathways are considered to be involved, pyrolysis and pyro synthesis. At high temperatures organic compounds are initially cracked to smaller molecules, mostly free radicals, which may then recombine to form several relatively stable polycyclic hydrocarbon. Hypothetical schemes for the generation of specific PAH compounds, such as benzo [a] pyrene (BaP), have been suggested (Knudsen, 1986).

PAH are formed in the combustion processes used in the production of curing smoke and for the generation of heat during grilling. The combustion fumes may contain both vapour phase and particle- bound PAH. Contaminants of food during grilling and smoking occurs when the combustion fumes reach the surface of the food. However, since PAH represent an important group of chemical carcinogens, their presence in food and the environment has been studied intensively during the last two decades. Among the various foods studied for PAH, smoked products seem to have been the most popular. Based on epidemiological studies in several countries, a possible connection between an increased incidence of gastric cancer and a high consumption of smoked food, has been suggested (Kundsen, 1986).

In fact, there are two cytogenetic tests in plants that can be performed in *Allium cepa* and *Vicia faba* i.e., the root-tip mitotic and PMC meiotic systems. The former is more widely used and better validated than the latter, especially when chromatid aberrations are used as the indicators of mutagenicity. However, this plant cytogenetic test system is efficient in the preliminary screening of chemical mutagens. Badr *et al.*, (1983), Amara *et al.*, (2008), Badr (1993). It is simple and economical test, and the results can be obtained in 48 hrs. Comparative studies of a given chemical under *Allium*; *Vicia.*, and mammalian system (such as human lymphocytes or mice) are strongly encouraged in order to establish the correlation between the chromosome breaking ability of chemicals in plant and animal system. (Al-Ayoubi, (1998) and Albertini *et al.*, (2001).

Regarding the experimental rodents, mice represent the better animals for genetic toxicological studies.

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The present work aims at disclosing the capability of some fast foods in inducing clastogenic effect assessed by analysis of chromosomes of *Allium cepa* and *Vicia faba* genome, and mutagenic effect assessed by *in vivo* induction of sister chromatid exchanges in mice genome. To achieve such a purpose Corn chips; Potato chips Burger and mice were locally purchased and used.

MATERIALS AND METHODS

Materials:

Allium cepa bulbs and *Vicia faba* were used in this work to assess the possible activity clastogenic, while mice were used to investigate the mutagenic activity by employing *in vivo* induction of sister chromatid exchange SCE technique.

Methods:

Preparation of food materials:

From each type of fast foods six concentrations were freshly prepared. These concentrations were 10; 20, 30, 40, 50 and 100g per liter. Each concentration was prepared as follows:

The proper amount was grinded well in a mortar and mixed with one litre of tap water; shaken overnight on a shaker, centrifuged at 2000 r.p.m. for 15 min., and filtered. The supernatant was saved and immediately used.

Germination of plants:

Allium cepa

Bulbs were allowed to geminate at 25°C using special Jars on tap water until a length of 0.5 cm for adventitious roots had reached, then they were subjected to treatment by transferring them on Jars containing the previously mentioned supernatant for different times of exposures, i.e., 6; 12; and 24 hrs. Mitotic index (MI)

$$MI = \frac{\text{No. divided cells}}{\text{Total examined cells}} \times 100$$

Vicia faba

Vicia faba seeds were soaked in running tap water for 24 hours, transferred to petri dishes on moistened filter paper and allowed to germinate in dark at 25°C. The filter paper inside each dish was moistened with tap water every day. When the primary root reached about 2 cm, the apical meristem was cut in order to stimulate the growing of secondary roots. When secondary roots on each primary one reached about 1 cm in length, seedling were then transferred in petri dishes and predetermined concentration was added. Six concentrations were used. Secondary roots were treated with the proper concentration for 6; 12 and 24 hours, while the control group was on tap water only. After treatment; root-tips of secondary roots and of *Allium cepa* adventitious

roots were cut and transferred to cold glacial acetic acid for one hour and transferred to a fixative solution (Carnoy's solution), and the well-known acetocarmine technique (Darlington and La Cour, 1960) was used to examine the mitotic activity. The frequency of mitotic activity (mitotic index) was calculated as the ratio of the total number of normal dividing cells to the total number of examined cells. The frequency of each type of mitotic aberrations was calculated as the ratio of cells containing this type of aberration to the total number of dividing cells.

5. Sister chromatid exchange technique.

Experimental design

Typically 2-3 month old Swiss albino mice (*Mus musculus*, 2n =40) were locally purchased from the high Institute of Graduate Studies and Research. Four animals per dose were used and analysis of at least 25 cells per animal was carried out. Four selected doses (10,20,30 and 40g/L) of the tested food were tested.

Bromodeoxyuridine tablet preparation

Bromodeoxyuridine tablets were prepared as described Seehy *et. al.*, (1983) as follows:

Bromodeoxyuridine tablets were prepared by using pellet press (Parr instrument co. Moline, III., USA), equipped with a 0.178 in diameter punch and die. Approximately, 55 mg of pure Bromodeoxyuridine powder were weight, placed in the die, and pressed. In order to maintain consistent compaction hardness (and thus the dissolution rate) among tablets, utilization of the same personal and die adjustment when pressing the powder was conducted. BrdU tablets were protected from light and stored in a freezer until usage.

BrdU and extract treatment

The mice were lightly anesthetized by placing it in a closed container with ether until immobile (about 2 min). After removal from the container, each mouse was restrained on its back. A small vial of anesthesia was placed near its nose for use in prolonging the inactive state. The lower lateral region was swabbed with alcohol in order to mat the fur down. Clean scissor or a scalpel was used to make a small (approx. 1 cm) subcutaneous incision. In order to spread open a deeper subcutaneous pocket, forceps were used, and the tablet was inserted. The wound was then closed with 2 –3 outclip sutures taking full care not to break the tablet, and the animal was returned to its cage. Each animal had received 2ml of the extract 8 hr after BrdU treatment. All animals were injected intravenously with 20 µg colcemid (1 mL/mouse, in tail vein) at 19 hr - (following BrdU treatment). Control marrow cells harvested 2 hr later revealed a high fraction of metaphases of optimal sister chromatid differentiation after staining. It was necessary to delay colcemid and

harvest steps for up to several hours when high concentrations were discovered to slow cell cycling and resulted in poor sister chromatid differential staining.

Preparation and implantation of BrdU tablets are given in figures (1a&b).

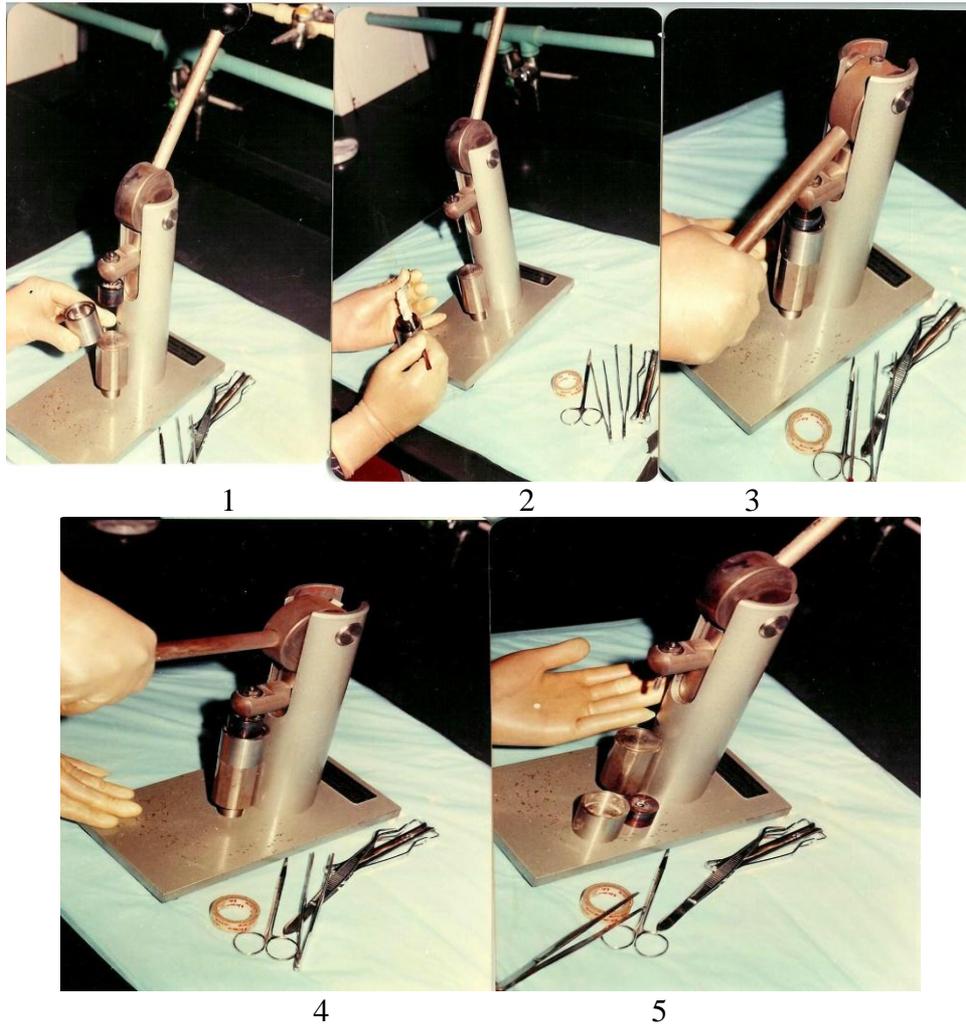


Figure 1a. Preparation of Brdu tablets

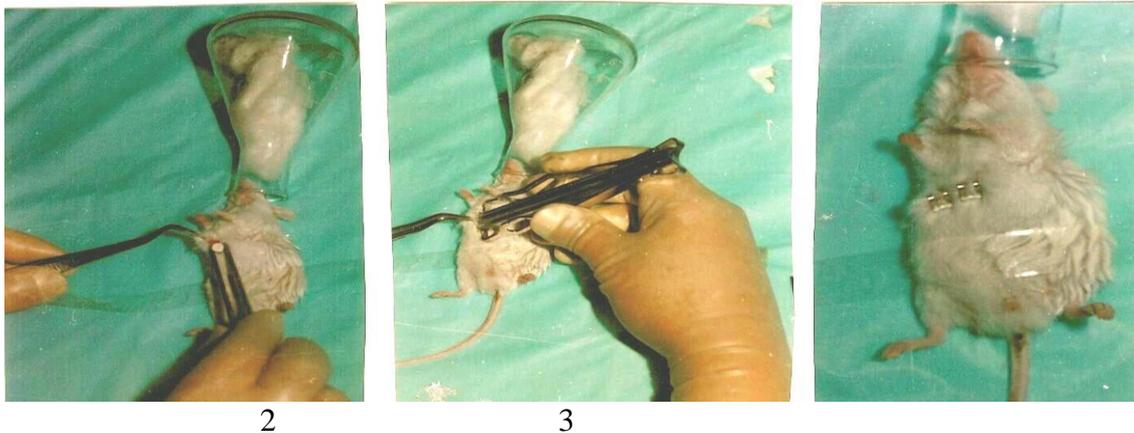


Figure 1-b. Implantation of Bromodeoxyuridine (BrdU) tablets

Marrow cells harvest, slide preparation and staining:

The procedure that described by Perry & Evans 1975 and Seehy *et al.*, 1983 was used.

Screening of slides and analysis

Scanning slides for mitotic spreads was conveniently accomplished with a 25 X magnification objective, and analysis was with a 100X objective. For control of bias, all prepared slides were coded prior to scoring. There are three ways for counting sister chromatid exchange frequencies i.e., (1) from the microscope images of second division cells, (2) the cells may be photographed and SCE frequencies counted from the microscope images. An interstitial exchanged segment was counted to be 2 SCEs. (Seehy *et al.*, 1983).

RESULTS

Mitotic activity

Tables (1-19) illustrate the effect of Potato and Corn chips and of Burger water extract upon cellular activity. The results showed that the water- soluble fraction of these foods displayed cellular toxicity upon *Allium cepa* and *Vicia faba* cells as well.

In *Allium cepa*, mitotic activity ranged from 12.9% to 16.21% in the control group. It decreased to be 4.6; 3.2; and 1% for Potato chips at the heights tested concentration for different times of treatment.

Table (8) shows that, at the highest tested concentration of Burger (100g/L) , no divided cells were obtained, giving an evidence for complete cellular toxicity after 12 hrs treatment time. After 24 hrs treatment time no divided cells (Table,9) at the level of 50 and 100 g/L were observed. Table (18) shows the effect of Burger upon *Vicia faba* cells. At the level of 40; 50; and 100 gm/L, no divided cells were observed, reflecting cellular toxicity of water-soluble fraction of Burger.

The present study showed that all tested water extract foods were effective in causing significant decreases in cell proliferation in *Allium cepa* root cells and *Vicia faba* as well. (Amara *et al.*, 2008).

Chromosomal aberrations:

Fragments, ring chromosomes; stickiness; gaps; and C- Metaphases were observed. They increased with the increasing of the tested concentration as well as with the treatment time that ranged from 6 to 24 hrs. For example, aberrant metaphase in *Allium cepa* (Table, 19) were 2% for the control group. They ranged from 4.2 to 15.4% after 6 hrs treatment time, and from 2.3 to 21.50 after 12 hrs treatment time (Table, 20) , giving a strong evidence that water-soluble fraction of potato chips was shown to be a positive clastogen. Figures (1-10) illustrate the positive effect of the tested foods upon *Allium cepa* and *Vicia faba* genome.

Cytological examination revealed that the percent of total aberrant metaphases after treatment with the extraction of fast foods for 24 hrs. by 20gm/L is given in table 19. Percent of total aberrant metaphases in the control group was found to be 2 % and it decreased to be 18.4 after treatment with corn chips and 22.7 after treatment with potato chips. High percentage of stickiness was observed after treatment with Burger extract (10%). Percent of total aberrant metaphases was found to be 31 % (15~ folds compared that of the with, the negative control).

This result, however, presented strong evidence that the tested fast foods have cellular toxicity and clastogenic effect. (This is in agreement with that reported by Erikson (1981).

Stickiness observed in this work might be resulted from an alteration of the net charge of chromosomal proteins and DNA or both (Seehy *et al.*, 2013).

The present work showed that these fast foods are positive clastogens such a conclusion is in agreement with that reported by El-Sebeay (1999).

As shown in table (21) sister chromatid exchanges were detected and statistically analyzed. Data showed that only one dose (10gm/L) from the three tested extract of fast foods was negative, while three doses i.e., 20,30, and 40g/L were proven to be positive, giving evidence that these fast foods are capable in causing primary DNA damage or mutagenic effect.

Table 1. Mitotic and phase indices in *Allium cepa* root cells after treatment with potato chips extract for 6 hrs

gm/L	*MI	Prophase	Metaphase	Anaphase	Telophase
Control	13.84	4.20	5.01	3.10	1.53
10	11.42	6.00	2.02	2.30	1.10
20	10.66	6.20	2.01	1.8	0.61
30	10.14	7.02	1.86	1.06	0.20
40	10	7.12	1.82	1.02	0.04
50	8.29	7.24	0.23	0.41	0.42
100	3.6	3	-	-	0.6

*Mitotic index

Table 2. Mitotic index and phase indices in *Allium cepa* root cells after treatment with potato chips for 12 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	15.21	5.01	6.00	3.00	1.20
10	10.14	7	2.14	1	0.50
20	10.8	7	1	1	1.8
30	9.01	8	1	0.01	-
40	8.02	7	1.02	-	-
50	7.4	6.8	0.4	0.2	-
100	2.2	2.1	-	-	0.1

Table 3. Mitotic index and phase indices in *Allium cepa* root cells after treatment with potato chips for 24 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	11.9	5.2	4.1	1.4	1.2
10	9.2	6	1.2	1.4	0.6
20	7.62	6.20	1.00	0.40	0.02
30	5	4.1	0.4	0.2	0.3
40	3	2.2	0.2	-	0.6
50	1	1.00	-	-	-
100	0	0	-	-	-

Table 4. Mitotic index and phase indices in *Allium cepa* root cells after treatment with corn chips for 6 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	14.84	4.20	5.01	3.10	2.53
10	14.02	4.12	4.20	2	3.70
20	13.10	4.01	3.88	3.21	2
30	12.00	5.20	2.00	2.5	2.3
40	11.08	6.20	1.7	2.12	1.06
50	10.42	6.50	0.80	1.04	2.08
100	4.11	4.00	-	-	0.11

Table 5. Mitotic index and phase indices in *Allium cepa* root cells after treatment with corn chips for 12 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	15.21	5.01	6.00	3.00	1.20
10	11.1	6	3	1	1.1
20	9.1	6	2.1	0.2	0.8
30	8.2	6	1.2	0.6	0.4
40	5.2	4.2	-	-	1
50	4.3	3.3	-	-	1
100	1.1	1.1	-	-	-

Table 6. Mitotic index and phase indices in *Allium cepa* root cells after treatment with corn chips for 24 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	11.9	5.2	4.1	1.4	1.2
10	10.3	4	3	2	1.3
20	8.4	4	1.4	2	1
30	6.2	4.2	1	1	-
40	3	1	1	0.6	0.4
50	1	0.8	-	-	0.2
100	0.2	0.2	-	-	-

Table 7. Mitotic index and phase indices in *Allium cepa* root cells after treatment with Burger for 6 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	13.84	4.20	5.01	3.10	1.53
10	12.20	5.1	4.1	2	1
20	11.50	4.60	3.20	2.00	1.50
30	10.00	4.80	2.20	1.2	1.8
40	8.2	5.2	1.2	0.6	40
50	7.2	5.4	-	1.2	50
100	5	5.00	-	-	-

Table 8. Mitotic index and phase indices in *Allium cepa* root cells after treatment with Burger for 12 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	15.21	5.01	6.00	3.00	1.20
10	8.4	5.6	1.4	0.60	0.44
20	7.04	4.8	1.2	0.24	0.8
30	5.21	2	1.2	1.01	1
40	3.1	1.1	-	1	1
50	1.1	1.1	-	-	-
100	-	-	-	-	-

Table 9. Mitotic index and phase indices in *Allium cepa* root cells after treatment with Burger for 24 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	11.9	5.2	1	1.4	1.2
10	5.04	3.2	-	1	0.84
20	4.36	2.8	-	0.23	1.30
30	3.12	2.17	-	0.66	0.29
40	1.01	1.01	-	-	-
50	-	-	-	-	-
100	-	-	-	-	-

Table 10. Mitotic and phase indices in *Vicia faba* root cells after treatment with potato chips extract for 6 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	8.12	1.00	4.52	1.20	1.40
10	7.01	2.00	3.00	1.00	1.01
20	6.92	2.10	2.02	1	1.80
30	5.22	3	1	0.4	0.82
40	3.28	3	-	-	0.28
50	1.11	1	-	-	0.11
100	-	-	-	-	-

Table 11. Mitotic and phase indices in *Vicia faba* root cells after treatment with potato chips extract for 12 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	10.20	2.6	5.2	1.4	1.00
10	6.60	2	2.2	1.3	1.1
20	6.2	3	1.2	1	1
30	5.3	3.4	0.6	-	1.3
40	2	2	-	-	-
50	0.02	0.02	-	-	-
100	-	-	-	-	-

Table 12. Mitotic and phase indices in *Vicia faba* root cells after treatment with potato chips extract for 24 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	8.03	1.00	5.00	1.00	1.03
10	3.04	1	1	0.04	1.00
20	2.06	1	1	0.06	-
30	2.01	1.4	-	0.01	0.60
40	1.20	1.20	-	-	-
50	-	-	-	-	-
100	-	-	-	-	-

Table 13. Mitotic and phase indices in *Vicia faba* root cells after treatment with Corn chips extract for 6 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	8.12	1.00	4.52	1.20	1.40
10	8.4	2	3.5	2.4	0.5
20	6.8	2	2	1.2	1.6
30	6.61	3.1	1.2	1.01	1.3
40	5.8	3.8	1	0.8	0.2
50	2.0	2.00	-	-	-
100	1.0	1.00	-	-	-

Table 14. Mitotic and phase indices in *Vicia faba* root cells after treatment with Corn chips extract for 12 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	10.2	2.60	5.20	1.4	1.00
10	7.6	3.00	2.00	1.20	1.40
20	5.90	3.00	1	0.80	1.10
30	5.20	3.20	0.80	-	1.20
40	4.80	3.60	0.40	-	0.80
50	2	1.8	-	-	0.20
100	0	0	-	-	-

Table 15. Mitotic and phase indices in *Vicia faba* root cells after treatment with Corn chips extract for 24 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	8.03	1.00	5.00	1.00	1.03
10	5.06	3.20	1.00	0.60	0.80
20	3.70	2.01	0.80	0.19	0.70
30	3.16	2.00	0.60	0.22	0.34
40	2.01	2.01	-	-	-
50	0	0	-	-	-
100	-	-	-	-	-

Table 16. Mitotic and phase indices in *Vicia faba* root cells after treatment with Burger extract for 6 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	8.12	1.00	4.52	1.20	1.40
10	8.01	2.00	3.00	2.00	1.01
20	7.70	3.00	2.00	1.60	1.10
30	7.84	3.02	1.80	1.00	2.02
40	6.82	4.03	1.06	0.62	1.11
50	5.82	4.00	0.66	1.00	0.16
100	3	3	-	-	-

Table 17. Mitotic and phase indices in *Vicia faba* root cells after treatment with Burger extract for 12 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	10.2	1.60	5.20	1.4	2.00
10	6.17	1.01	3.02	1.04	1.10
20	6.08	1.80	2.00	1.06	1.22
30	5.92	1.89	1.88	1.02	1.13
40	2.22	1.21	1.01	-	-
50	1.00	1.00	-	-	-
100	-	-	-	-	-

Table 18. Mitotic and phase indices in *Vicia faba* root cells after treatment with Burger extract for 24 hrs

gm/L	MI	Prophase	Metaphase	Anaphase	Telophase
Control	8.03	1.00	5.00	1.00	1.03
10	2.16	1.10	0.40	-	0.66
20	2.09	1.00	0.32	0.01	0.76
30	1.17	1.17	-	-	-
40	-	-	-	-	-
50	-	-	-	-	-
100	-	-	-	-	-

Table 19. Chromosomal aberrations induced after treatment of *Allium cepa* roots extraction with fast foods* for 24 hrs

Types	Fragment	Ring chromosome	Stickiness	Gap	C-metaphase	Percent of total aberrant metaphases
Control	-	-	2	-	-	2%
Potato chips	6.2	6.5	8	-	2	22.7
Corn chips	3.4	6	6	1	2	18.4
Burger	8	8	10	2	3	31

*20 gr/L

Table 20. Chromosomal aberrations induced after treatment of *Vicia faba* roots with fast food extraction for 24hrs

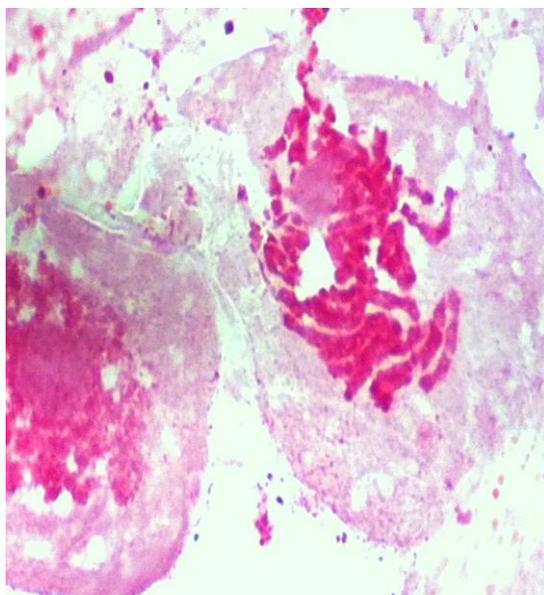
Types	Fragment	Ring chromosome	Stickiness	Gap	C-metaphase	Percent of total aberrant metaphases
Control			2			2
Potato chips	8	7	4	4	2	25
Corn chips	5.5	9.5	6	4	2	26
Burger	11	12	13	2	4	32

*20gm/L

Table 21. Averages \pm S.E of sister chromatid exchanges in mice bone marrow cells after oral treatment with the extract of the tested fast foods

Dose; gm/L	X \pm SE	Range
Control	2.1 \pm 0.1	1-3
Potato chips 10	2.2	1-4
20	3.4*	2-5
30	5.2*	3-6
40	8.4*	4-10
Corn chips 10	2.1	1-4
20	4.2*	2-6
30	4.6*	2-8
40	10.2*	4-12
Burger 10	2.4	2-4
20	4.2*	3-6
30	6.2*	4-8
40	12.4*	7-14

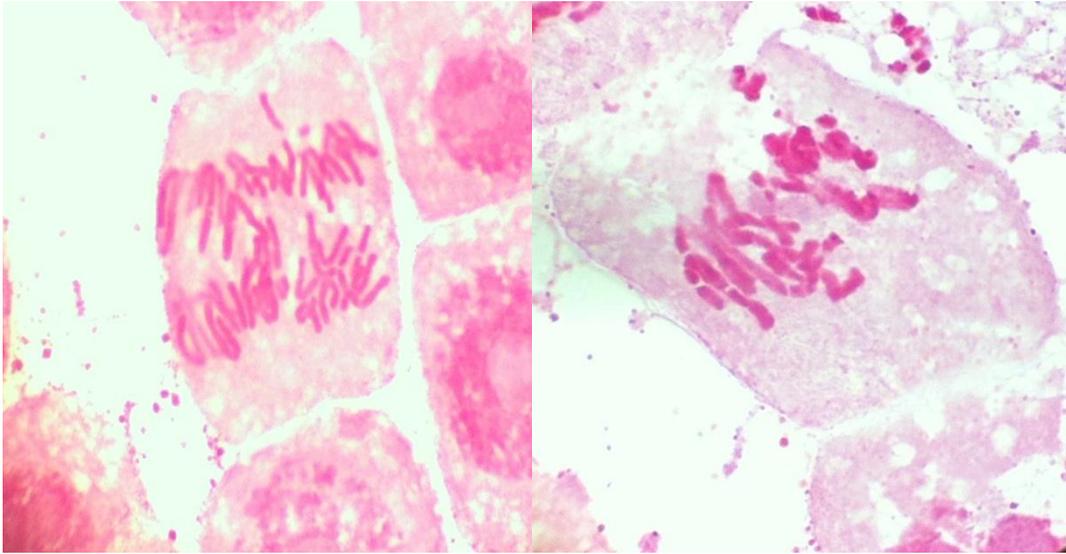
*Significant at 0.05 level of probability.



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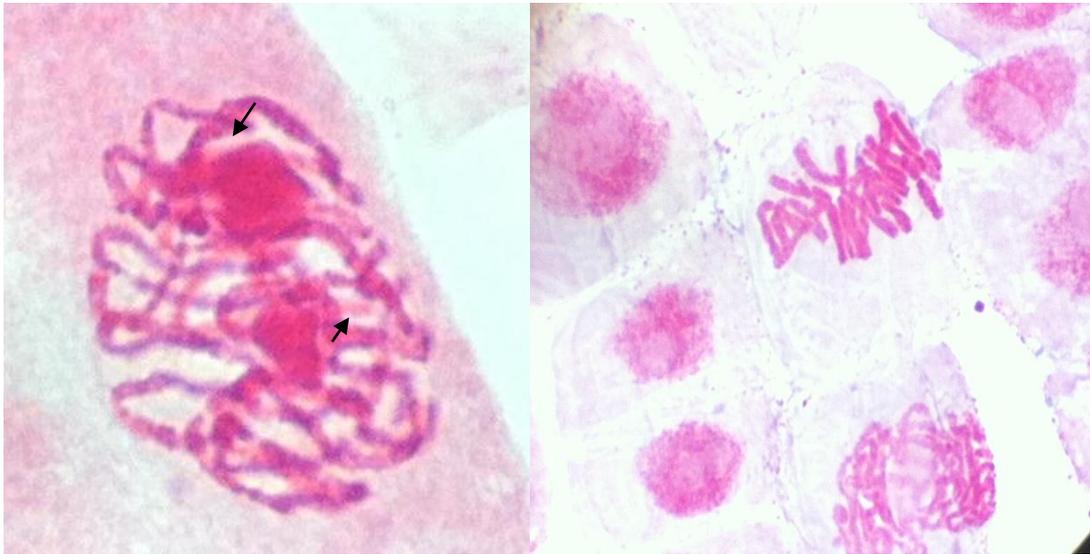


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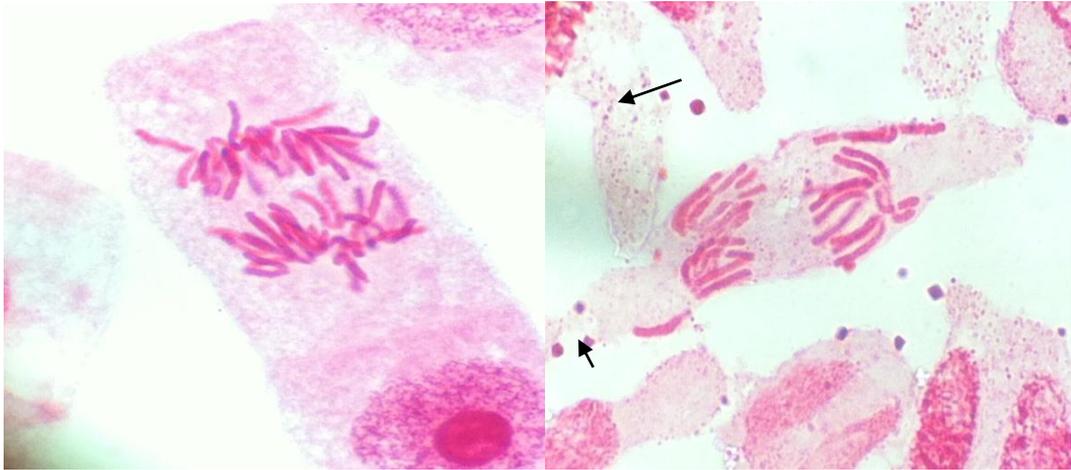
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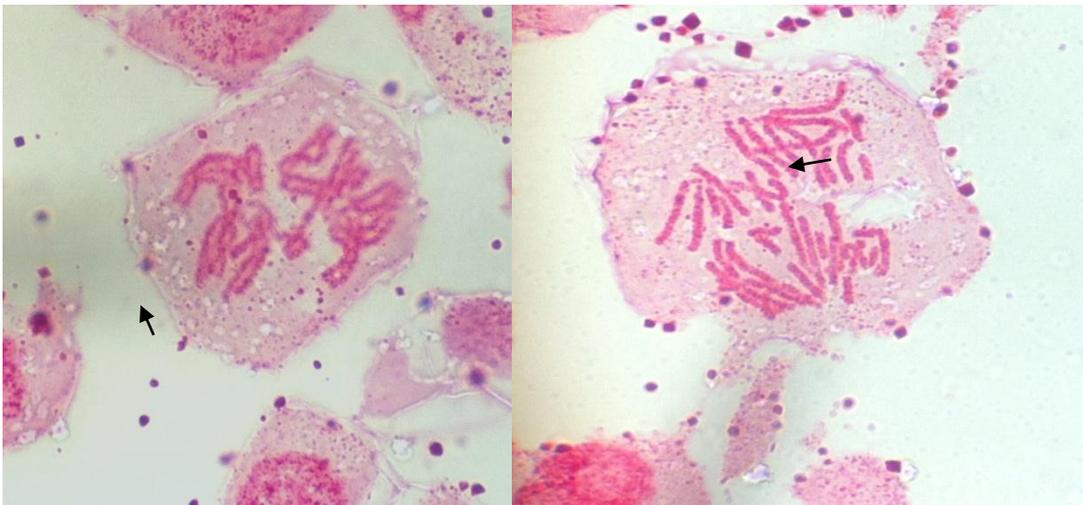
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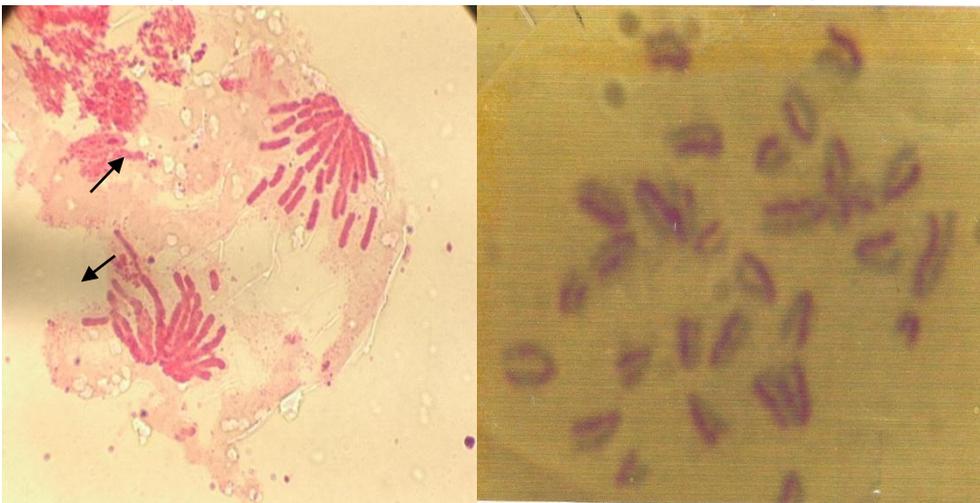
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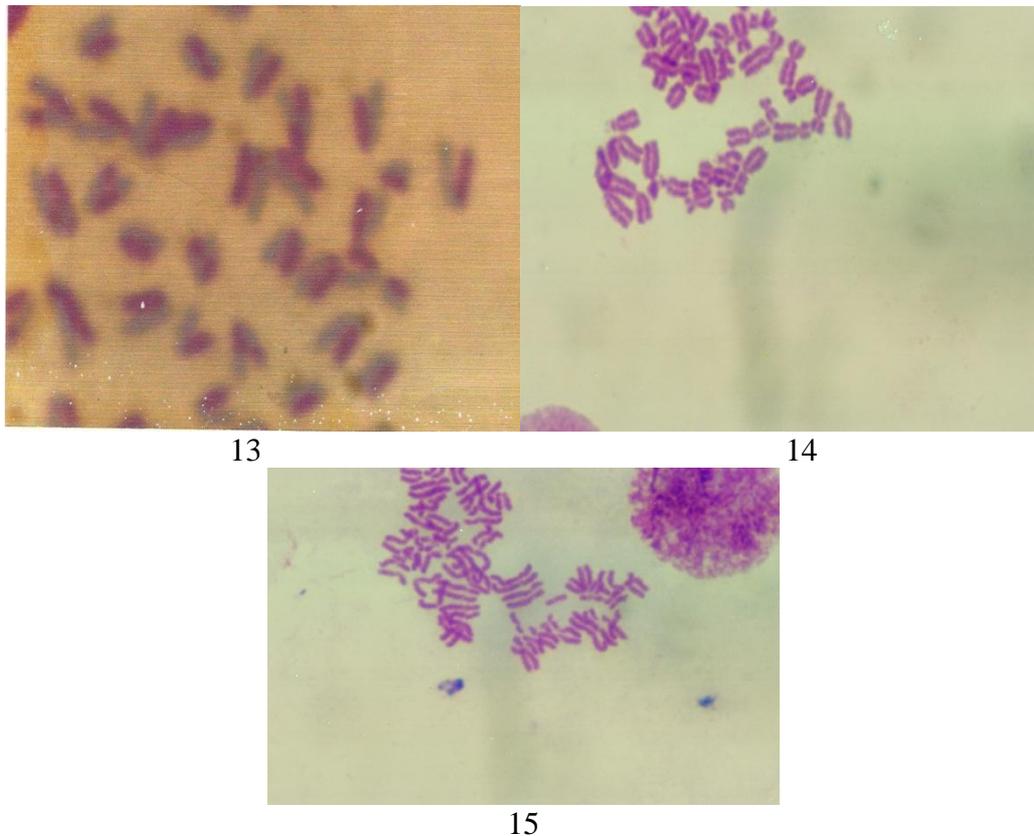


Figure	Description
1	Chromatid deletion <i>A. cepa</i>
2	Fragments <i>V. faba</i>
3	Fragments and Gap <i>V. faba</i>
4	Fragments , Chromatid deletion and Gap <i>A. cepa</i>
5	Chromatid deletion <i>V. faba</i>
6	Binucleolei , <i>V. faba</i>
7	Normal chromosomes <i>A. cepa</i>
8	Chromatid deletion <i>A. cepa</i>
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10	Chromatid deletion <i>A. cepa</i>
11	Fragments and Chromosome deletion, <i>A. cepa</i>
12	Sister chromatid exchange, Burger treatment
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15	Ipo-prophen

DISCUSSION

This work aims at disclosing the possible genotoxic effect (clastogenic and mutagenic as well) induced by some fast foods. The recognition that diet may be one of the most important factors explaining international variation in cancer came when it was appreciated that for many cancers, diet seemed to be the most rational explanation (Higginson and Muir, 1979), coupled with studies at the macro-population level which indicated substantial correlations between incidence or mortality of cancer at various sites and various estimates of population consumption of dietary items (Armstrong and Doll, 1975; Knox, 1977). The explosion of interest that has followed, particularly in the past ten years, has gradually led to recognition that many of the relationships are complex, involving not only factors that increase the risk of cancer, but also protective factors. Further, we do not appear to be dealing with simple expression of carcinogens and mutagens, rather diet seems to act on both the early and late stages of carcinogenesis.

The associations with dietary factors and cancer incidence or mortality studied internationally, through strong, only point to the importance of dietary factors in etiology, they cannot prove causation. For this, it is necessary to conduct studies on diet in individuals, and then to confirm causation and measure to impact of preventive actions, conduct intervention studies in populations. For diet and nutrition, many studies on individuals have been conducted, but so far studies on populations have been reported. The studies on individuals are of two types, case-control and cohort. Case-control studies are based on the disease, looking back to the relevant exposures. Cohort studies are based on the exposure, looking forward to the disease it may induce or prevent. There have been relatively few cohort studies, and most with information available currently were originally planned to concentrate on cardiovascular diseases. Case-control studies of diet and cancer have been more numerous, leading us to evaluate diet largely in relation to individual cancer sites. These vary from those in which diet seems to play a major role, such as stomach and colon and rectum, through those to which diet seems to make a major contribution, such as breast, prostate and many of the hormonally associated cancers, to those with important, well-recognized non-dietary causative factors to which dietary factors contribute to or modify risk, such as lung, bladder and other smoking and/of alcohol-associated cancers (Miller, 1986).

However, anticarcinogens have been reported in other foods and are postulated to mitigate the potentially deleterious effect of the low levels of mutagens and carcinogens that are ingested daily (Ames, 1983;

Wattenberg, 1983). In particular modulator-mediated inhibition of carcinogenesis is of importance given that carcinogenic poly-nuclear arenes and heterocyclic amines are found in some cooked foods. Since case-control epidemiologic investigations of the possible association of beef and other meats with colorectal and breast cancers are at present unclear (Doll and Peto, 1981), one might speculate that the modulator reported herein may under some circumstance act to moderate the effects of low levels of carcinogens that may be present, resulting in epidemiologic findings that are equivocal. Silalahi (2002).

The present investigation showed that the tested fast foods were proven to be clastogenic agents, since they were capable to interfere with spindle fibers, and C-metaphases were observed, giving strong evidence that the water-soluble fraction of potato and corn chips as well as of Burger contained contaminants capable to interact with chromosomes and spindle fibers as well. However they have mutagenic activity as sister chromatid exchanges revealed obtained since positive frequencies of SCEs were observed.

In conclusion clastogenic and mutagenic effect of these fast foods, under study, had been formed.

This positive clastogenic activity of water-soluble fraction of the tested foods may be caused by acrylamide or by heterocyclic amine El-Sebeay (1999) and / or other contaminants. However, this question was not answered at the level of this work.

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الملخص العربي

الأثر الطفرى والتكسير للكروموسومات المستحدث بواسطة بعض الاغذية السريعة

دعاء شوشان ، فتحى قنديل الفقى و محمد عبدالباعث الصيحي

عدة وكانت النتائج ان هذه الادوية لها اثر فعال فى احداث ضرر للمادة الوراثية من النوع الصغير والمتمثل فى تبادل الكروماتيدات الشقيقه وكذلك من النوع الكبير متمثل فى تكسير الكروموسومات والشظايا والتضاعف.

الكلمات المفتاحيه: كلاستوجين - مطفر - مسرطن - غذاء

اجرى هذا البحث على ثلاث عقاقير تستخدم كمضادات التهاب فى جمهورية مصرالعربية وهذه العقاقير هى ايبوبروفين وحمض الاستيل سيلسيلك والالكسوبيرين وتم اختبار هذه العقاقير على خلايا الدم البيضاء فى الانسان وخلايا نخاع العظام من الفيران والكروموسومات خلايا الجذور العرضية لنبات الفول والبصل وتم استخدام تركيزات