Fast Foods Induced Cytogenetic Damage in *Vicia faba* and *Allium cepa* Genomes

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ABSTRACT

The present work was planned to investigate the genotoxic effect of some fast foods upon *Allium cepa* as well as *Vicia faba* genome. Potato and corn chips in addition to Burger were chosen and the water-soluble fraction of these foods were tested using six different concentrations of each food and three different treatment times i.e., 6; 12; and 24 hrs. Cell proliferation was examined by estimating mitotic activity and phase indices. Examination of chromosomal aberrations was carried out. The results obtained indicated that the water-soluble fraction of the tested foods was proven to be a positive clastogen, since significant increases of different types of aberrations were observed. These abarrations were found to increase with the increasing of the tested concentrations and treatment times as well.

INTRODUCTION

Almost at the same time as the first reports appeared regarding the presence of mutagens in beef extracts in broiled and fried meat, and in fish, Maillard reactions were suggested to be involved in their formation (Spingarn and Garvie, 1979; Powrie et al., 1981; Sugimura et al., 1982; Matsushima, 1982; Shibamoto, 1983).

Maillard reactions or nonenzymatic browing reactions are among the most important reactions affecting food quality during storage and heat treatment. The reaction rate is dependent on temperature and water content. In comparatively dry food systems, such as grains, flours, etc., the reaction may take place at temperatures well below 100 C but the rate increases rapidly at temperatures above 100 C, especially since the water content then is usually decreased.

Among the positive effects of Maillard reactions are for instance, development of flavors, texture, and brown pigments, which all contribute to the palatability of cooked foods. Moreover, antioxidative as well as antimicrobial effects of Maillard reactions have been reported (Eriksson, 1981; Waller and Feather, 1983). Among the negative effects are miscoloring and off-flavors during food storage. From a physiological point of view, destruction of protein quality due to Maillard reactions has attracted a great deal of attention. Especially lysine, with its free amino group, reacts readily, with loss of its biological value. Animal studies have demonstrated toxic effects of certain

Maillared reaction products on liver and kidney (Mauron, 1981). However, mediterm studies on laboratory animals fed a diet containing Maillard-reacted foods have not evidenced any toxic effects as long as the diets have been optimal in all essential nutrients (Waller and Feather, 1983). On the other side these feeding experiments were too brief for evaluation of any tumor-inducing effects. This aspect needs more attention when considering the newly reported genotoxic effects of model systems of Maillard reactions (Jagerstad *et al.*, 1986).

Allium cepa and *Vicia faba* display qualities that have long made them a useful subject for cytological study i.e., their large chromosomes, comparatively low chromosome number (2n = 16 & 12) availability and ease of handling. Presently, Allium cepa and *Vicia faba* are being used as a plant test system to assess the mutagenic potentials of environmental chemicals by many investigators (e.g. Badr, 1983; Badr *et al.*, 1983 and Al-Ayoubi, 1998).

In fact, there are two cytogenetic tests 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. 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) are strongly encouraged in order to establish the correlation between the chromosome breaking ability of chemicals in plant and animal system.

The present work aims at disclosing the capability of some fast foods in inducing cytogenetic damage in higher plant chromosomes. To achieve such a purpose Corn chips; Potato chips and Burger were locally purchsed and tested employing *Allium cepa* and *Vicia faba* genome.

Materials and Methods

Materials:

Allium cepa bulbs and *Vicia faba* seeds were used in this work.

Three fast foods i.e., corn chips; potato chips; and prepared Burger were locally purchased and tested.

Methods:

Preparation of food material:

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

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

Germination of plants:

Alium 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.

Vicia faba

Vicia faba 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 treated. 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* adventitions 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 as well. 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.

RESULTS

Mitotic activity

The effect of water- soluble fraction of the tested fast foods upon cell proliferation of Allium cepa as well as of Vicia faba root cells was investigated.

Tables (1-18) illustrate the effect of potato and corn chips and of Burger upon cellular activity. The results showed that the water-soluble fraction of these foods displayed cellular toxicity upon *Allium cepa* and *Vicia faba* 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 heighst tested concentration for different time treatments.

Table (8) shows that, at the heighst tested concentration of Burger (100g/ L), no divided cells were obtained, giving an evidence for complete cellular toxicity after 12hrs 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 g/L, no divided cells were observed, reflecting cellular toxicity of water soluble fraction of Burger.

The present study showed that all tested foods were effective in causing significant decreases in cell proliferation in *Allium cepa* root cells and *Vicia faba* as well.

Chromosomal aberrations:

Tables (19-36) illustrate the effect of the tested foods for their clastogenicity upon *Allium cepa* and *Vicia faba* chromosomes.

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, abarrant metaphases 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) and from 3.5 to 24.40% after 24 hrs treatment time (Table, 21) , 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* genomes.

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

gr/L	* MI	Prophase	Metaphes	Anaphase	Telophase
Control	14.84	4.20	5.01	3.10	2.53
10	12.42	6.00	2.02	2.30	2.10
20	11.66	6.20	2.01	1.8	1.61
30	11.14	7.02	1.86	1.06	1.20
40	11	7.12	1.82	1.02	1.04
50	9.29	7.24	1.23	0.41	0.42
100	4.6	4	-	-	0.6

* Mitotic index.

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	16.21	5.01	6.00	3.00	2.20
10	11.14	7	2.14	1	1
20	11.8	7	1	1	2.8
30	10.01	8	1	1.01	-
40	9.02	8	1.02	-	-
50	8.4	7.8	0.4	0.2	-
100	3.2	3.1	-	-	0.1

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	12.9	5.2	4.1	1.4	2.2
10	10.2	6	1.2	1.4	1.6
20	8.62	6.20	1.00	0.40	1.02
30	6	5.1	0.4	0.2	0.3
40	4	3.2	0.2	-	0.6
50	2	2.00	-	-	-
100	1	1	-	-	-

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

gr/L	MI	Prophase	Metaphes	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.

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	16.21	5.01	6.00	3.00	2.20
10	12.1	6	3	1	2.1
20	10.1	7	2.1	0.2	0.8
30	9.2	7	1.2	0.6	0.4
40	6.2	5.2	-	-	1
50	5.3	4.3	-	-	1
100	2.1	2.1	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	12.9	5.2	4.1	1.4	2.2
10	11.3	4	3	2	2.3
20	9.4	5	1.4	2	1
30	7.2	5.2	1	1	-
40	4	2	1	0.6	0.4
50	2	1.8	-	-	0.2
100	1.2	1.2	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	14.84	4.20	5.01	3.10	2.53
10	13.20	5.1	4.1	2	2
20	12.50	5.60	3.20	2.00	1.50
30	11.00	5.80	2.20	1.2	1.8
40	9.2	6.2	1.2	0.6	1.2
50	8.2	6.4	-	1.2	0.6
100	6	6.00	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	16.21	5.01	6.00	3.00	2.20
10	9.4	5.6	1.4	0.60	1.44
20	8.04	4.8	1.2	1.24	0.8
30	6.21	3	1.2	1.01	1
40	4.1	2.1	-	1	1
50	2.1	2.1	-	-	-
100	-	-	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	12.9	5.2	.1	1.4	2.2
10	6.04	4.2	-	1	0.84
20	5.36	3.8	-	0.23	1.30
30	4.12	3.17	-	0.66	0.29
40	2.01	2.01	-	-	-
50	-	-	-	-	-
100	-	-	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	9.12	2.00	4.52	1.20	1.40
10	8.01	3.00	3.00	1.00	1.01
20	7.92	3.10	2.02	1	1.80
30	6.22	4	1	0.4	0.82
40	4.28	4	-	-	0.28
50	2.11	2	-	-	0.11
100	-	-	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	11.20	2.6	5.2	1.4	2.00
10	7.60	3	2.2	1.3	1.1
20	7.2	4	1.2	1	1
30	6.3	4.4	0.6	-	1.3
40	3	3	-	-	-
50	1.02	1.02	-	-	-
100	-	-	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	9.03	2.00	5.00	1.00	1.03
10	4.04	2	1	0.04	1.00
20	3.06	2	1	0.06	-
30	3.01	2.4	-	0.01	0.60
40	2.20	2.20	-	-	-
50	-	-	-	-	-
100	-	-	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	9.12	2.00	4.52	1.20	1.40
10	9.4	3	3.5	2.4	0.5
20	7.8	3	2	1.2	1.6
30	7.61	4.1	1.2	1.01	1.3
40	6.8	4.8	1	0.8	0.2
50	3.0	3.00	-	-	-
100	2.0	2.00	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	11.2	2.60	5.20	1.4	2.00
10	8.6	4.00	2.00	1.20	1.40
20	6.90	4.00	1	0.80	1.10
30	6.20	4.20	0.80	-	1.20
40	5.80	4.60	0.40	-	0.80
50	3	2.8	-	-	0.20
100	1	1	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	9.03	2.00	5.00	1.00	1.03
10	6.06	4.20	1.00	0.60	0.80
20	4.70	3.01	0.80	0.19	0.70
30	4.16	3.00	0.60	0.22	0.34
40	3.01	3.01	-	-	-
50	1	1	-	-	-
100	-	-	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	9.12	2.00	4.52	1.20	1.40
10	9.01	3.00	3.00	2.00	1.01
20	8.70	4.00	2.00	1.60	1.10
30	8.84	4.02	1.80	1.00	2.02
40	7.82	5.03	1.06	0.62	1.11
50	6.82	5.00	0.66	1.00	0.16
100	4	4	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	11.2	2.60	5.20	1.4	2.00
10	7.17	2.01	3.02	1.04	1.10
20	7.08	2.80	2.00	1.06	1.22
30	6.92	2.89	1.88	1.02	1.13
40	3.22	2.21	1.01	-	-
50	2.00	2.00	-	-	-
100	-	-	-	-	-

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

gr/L	MI	Prophase	Metaphes	Anaphase	Telophase
Control	9.03	2.00	5.00	1.00	1.03
10	3.16	2.10	0.40	-	0.66
20	3.09	2.00	0.32	0.01	0.76
30	2.17	2.17	-	-	-
40	-	-	-	-	-
50	-	-	-	-	-
100	-	-	-	-	-

Table 19. * Chromosomal aberrations induced in *Allium cepa* cells after treatment with *potato chips* for *6 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2	-	-	2
10	-	-	4.2	-	-	4.2
20	-	1	5.3	1	1	8.3
30	1	1	5	1	1	9
40	2	-	5.6	1.2	1	9.6
50	2	1	7	3.4	2	15.4
100	2.3	1	1	-	-	4.3

* Percent

Table 20. * Chromosomal aberrations induced in *Allium cepa* cells after treatment with *potato chips* for *12 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2.3	-	-	2.3
10	1	2	3.1	-	1	7.1
20	2	3.2	4.6	1	1	11.8
30	2.6	2	4.8	1	2	12.4
40	4.5	4	5.2	2	2	17.7
50	4.6	3.2	6.4	2	-	16.20
100	6.2	6.1	7.2	2	-	21.50

* Percent

Table 21. * Chromosomal aberrations induced in *Allium cepa* cells after treatment with *potato chips* for *24 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	1	2.5	-	-	3.50
10	-	2.2	4	-	1.2	7.40
20	2.2	3.2	4	-	2.5	11.90
30	3.3	4.2	6	2.3	2.5	18.30
40	5.2	5.4	8	4.2	-	23.00
50	4.1	6.2	10	4.1	-	24.40
100	7.1	6.5	1	2.5	-	15.10

* Percent

Table 22. *Chromosomal aberrations induced in *Allium cepa* cells after treatment with *corn chips* for *6 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2	-	-	2.00
10	-	-	3	1	-	4.00
20	-	1	3.8	2	1	7.80
30	1	1	4.1	-	1	7.10
40	1	2	4.82	1	1	9.82
50	1	2	6	2.3	1	12.30
100	1.2	2.2	8.2	2	-	13.60

* Percent

Table 23. * Chromosomal aberrations induced in *Allium cepa* cells after treatment with *corn chips* for *12 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2.3	-	-	2.3
10	2	2	4	1	-	9
20	2.2	2	5	2	1	12.2
30	3.5	3	5	3	2	16.5
40	3.5	4	7	2.4	-	16.9
50	4	4	6	4	-	18
100	4	3	6	4	-	17

* Percent

Table 24. * Chromosomal aberrations induced in *Allium cepa* cells after treatment with *corn chips* for *24 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	1.00	2.5	-	-	3.50
10	2	2	4	1	2	11
20	2	3	6	2	2.5	15.5
30	4	4	4	2.5	1.5	16
40	3	4	8	3	4	22
50	3	5	8	2	-	18
100	2	3	5	3	-	13

* Percent

Table 25. * Chromosomal aberrations induced in *Allium cepa* cells after treatment with *Burger* for *6 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2	-	-	2.00
10	-	-	4	-	1	5.00
20	-	1	6	-	2	9.00
30	-	-	6	1.5	2.2	9.70
40	1	1	8	1	2	13.00
50	-	2	6	1	-	9
100	1	2	4	-	-	7

* Percent

Table 26. * Chromosomal aberrations induced in *Allium cepa* cells after treatment with *Burger* for *12 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2.3	-	-	2.3
10	2	-	4	-	1	7
20	4	1	6	1	1	13
30	6	2	6	1	2	17
40	6	3	6	1	-	16
50	8	4	8	1	-	21
100	6	4	4	-	-	14

* Percent

Table 27. * Chromosomal aberrations induced in *Allium cepa* cells after treatment with *Burger* for *24 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	1	2.5	-	-	3.5
10	4	2	4	1	-	11
20	6	2	8	1	2	19
30	6	3	7	2	2	20
40	8	2	6	3	3	22
50	6	1	4	3	-	14
100	4	2	8	2	-	16

* Percent

Table 28. * Chromosomal aberrations induced in *Vicia faba* cells after treatment with *potato chips* for *6 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2	-	-	2
10	1	-	3	4	-	8
20	-	1	3	5	1	10
30	1	-	5	6	1	13
40	1	1	6	5	2	15
50	1	1	7	7	2	18
100	1	1	6	6	2	16

* Percent

Table 29. * Chromosomal aberrations induced in *Vicia faba* cells after treatment with *potato chips* for *12 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	1	-	-	1
10	1	-	4	-	-	5
20	2	1	5	1	-	9
30	2	1	4.5	2.5	-	10
40	3	1	6	3	-	13
50	2	1	6	2	-	11
100	2	2	5	2	-	11

* Percent

Table 30. * Chromosomal aberrations induced in *Vicia faba* cells after treatment with *potato chips* for *24 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2	-	-	2
10	1	2	4	1	-	8
20	4	2	6	2	1	15
30	5	4	6	2	2	19
40	6	3	5	2	2	18
50	8	4	4	3	3	22
100	10	4	6	2	3	25

* Percent

Table 31. * Chromosomal aberrations induced in *Vicia faba* cells after treatment with *corn chips* for *6 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2	-	-	2
10	-	-	4	-	-	4
20	1	-	4	1	-	6
30	1	1	4.5	2	1	9.5
40	2	2	6	2	1	13
50	3	2	6	3	-	14
100	3	4	7	2	-	16

* Percent

Table 32. * Chromosomal aberrations induced in *Vicia faba* cells after treatment with *corn chips* for *12 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	1	-	-	1
10	-	-	1	-	-	1
20	1	1	2.5	-	1	5.5
30	2	2	5.5	1	2	12.5
40	3	3	6	2	3	17
50	2	4	6	3	4	19
100	4	4	4	4	4	20

* Percent

Table 33. * Chromosomal aberrations induced in *vicia faba* cells after treatment with *corn chips* for *24 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2	-	-	2
10	1	1	4	1	-	7
20	2	2	5	2	1	12
30	3	4.5	6	3	2	18.5
40	5	5	7.5	4	2	23.5
50	5	7	8	3	2	25
100	4	7	4	5	2	22

* Percent

Table 34. * Chromosomal aberrations induced in *Vicia faba* cells after treatment with *Burger* for *6 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2	-	-	2
10	-	-	4	-	-	4
20	1	-	4	1	-	6
30	1	1	5	-	-	7
40	2	2	6	-	-	10
50	2	2	8	1	-	13
100	2	1	4	2	-	9

* Percent

Table 35. * Chromosomal aberrations induced in *Vicia faba* cells after treatment with *Burger* for *12 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	1	-	-	1
10	1	2	4.5	-	-	7.5
20	2	2	4	0.5	-	8.5
30	2	1	6	-	1	10
40	2	2	7	2	-	12
50	1	2	7	2	-	12
100	2	4	8	-	-	14

* Percent

Table 36. * Chromosomal aberrations induced in *Vicia faba* cells after treatment with *Burger* for *24 hrs.*

Concentration, gr/L	Fragment	Ring Chromosome	Stickiness	Gap	C-Metaphase	Total abarrant metaphases
Control	-	-	2	-	-	2
10	3	-	4	-	1	8
20	3	4	5	2	2	16
30	4	6	7	2	2	21
40	4	4	8	4	4	24
50	5	5	5	4	3	22
100	4	3	4	4	-	17

* Percent

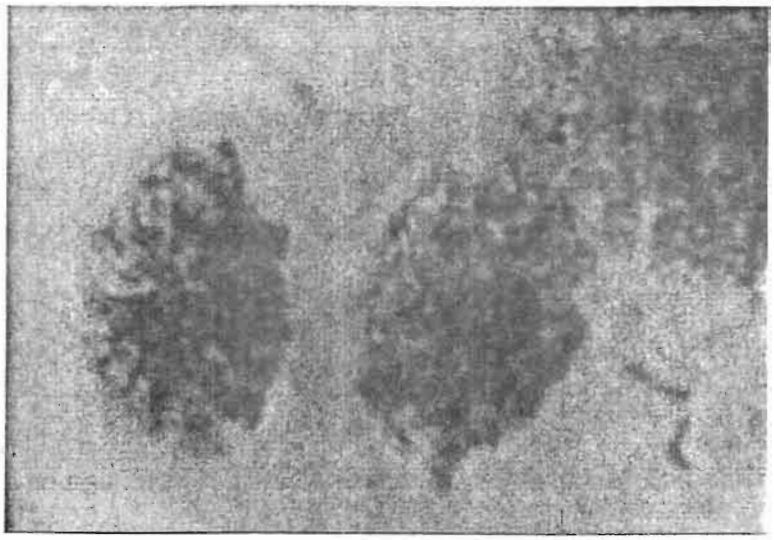

Figure 1. Binucleate cell in *Allium cepa* root-tip after treatment with potato chips.

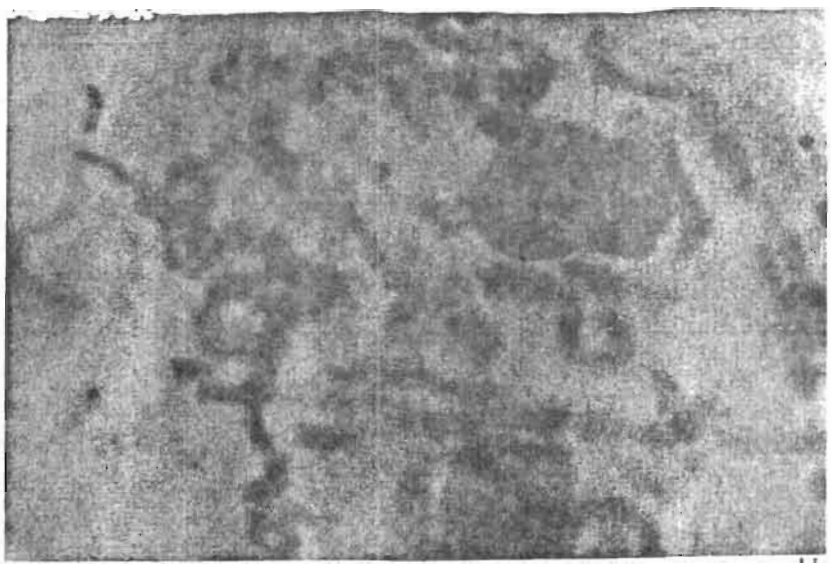

Figure 2. Metaphase stage in *Allium cepa* root-tip after treatment with corn chips showing chromatid gap.

Figure 3. Photomicrograph showing fragment after treatment of *Allium cepa* cells with Burger

Figure 4. Photomicrograph showing C-Metaphase after treatment of *Allium cepa* cells with potato chips.

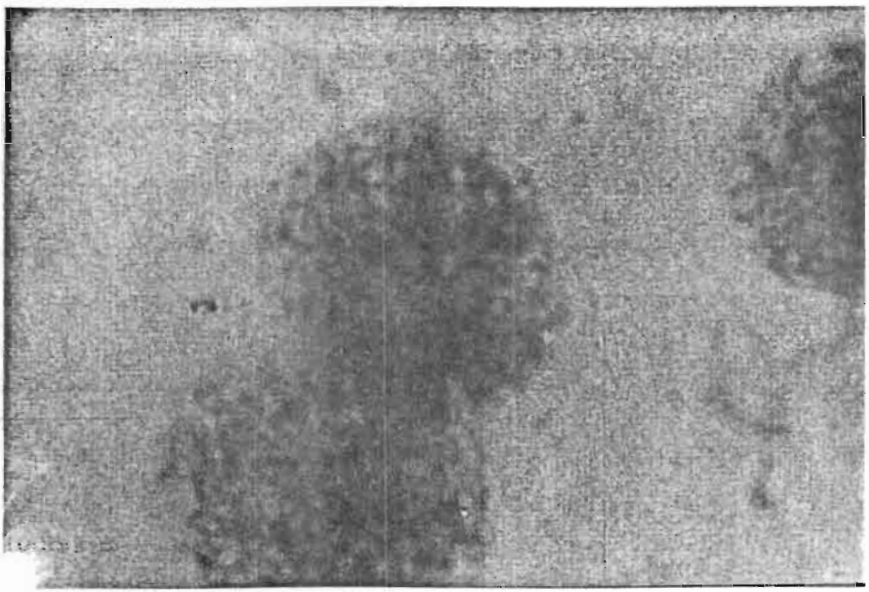

Figure 5. Binucleate cell in *Vicia faba* root-tip after treatment with potato chips.

Figure 6. Metaphase stage in *Vicia faba* root-tip after treatment with corn chips showing chromatid gap.

Figure 7. Photomicrograph showing fragment after treatment of *Vicia faba* cells with Burger

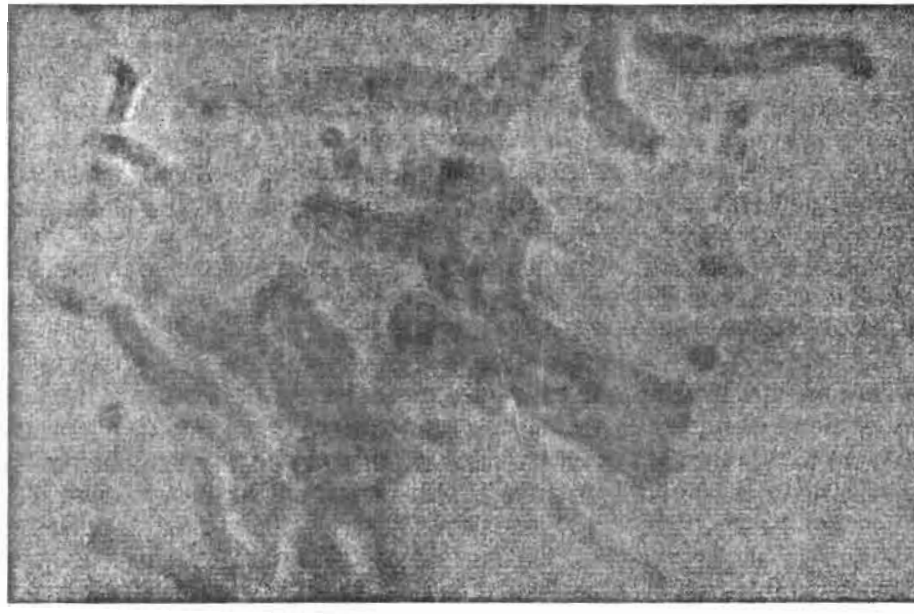

Figure 8. Photomicrograph showing C-Metaphase after treatment of *Vicia faba* cells with potato chips.

DISCUSSION

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 a recognition that many of the relationships are compiex, 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, though 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 the 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 diseases 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 disease. 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 dietar 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 an 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 finding that are equivocal.

The present investigation showed that the tested fast foods were proven to be clastogenic agents, since they were capable to induce significant increases of different types of chromosomal aberrations. On the other hand , the tested foods were found to be capable to interfer with spindle fiber, since C-metaphases were observed, giving a 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.

This positive clastogenic activity of water-soluble fraction of the tested foods may be caused by acrylamide or by heterocyclic amines (Ohgaki et al, 1991) and / or other contaminants. However, this question was not answered at the level of this work :

REFERENCES

Al-Ayoubi, D. (1998). Piroxicam induced clastogenic effect in *Vicia faba* genome. J. Advances in Agric. Res. Alex. Univ; 3 : 100-120.

Ames BN (1983). Dietary carcinognes and anticarcinogens. Science 3 : 24155-24215.

Armstrogn B, and R. Doll (1975). Environmental factors and cancer incidence and mortality in different countries, with special reference to dietary practices. Int J. Cancer 15 : 617-631.

Badr, A. (1983) Cytogenetic activities of atrazine herbicide in root-tips of *Allium cepa* and *Vicia faba*. Mutat. Res., 117: 173-182.

Badr, E.A., M.A. Seehy, and H.G. Shalabi (1983). Sister chromatid exchange detection in plants : A genetic assay of primary DNA damage induced by pesticides . Internat. Cong. Genetics, New Delhi, Abst. Part I : 271.

Darlington, C.D., and L.F. La Cour (1962). The handling of chromosomes. Ed. Georg Allen & Unwin Ltd., London.

Doll R, and R Peto (1981) . The causes of cancer : Quantitative estimates of avoidale risks of cancer in the United States today. JNCI 66 : 1191 – 1308.

Eriksson C (1981). Maillard reactions in food. Prog Food Nutr Sci 5 : 429 – 466.

Higginson J. and CS Muir (1979). Environmental carcinogenesis : misconceptions and limitations to cancer control. J Natl cancer Inst 63 : 1291-1298.

Jagerstad, M., S. Grivas, K. Olsson, A. Laser Reuter-Sward, C. Negishi, and S. Sato (1986). In : Genetic Toxicology of the Diet [Ed. Ib Knudsen] Alan R. Liss Inc. New York.

Knox Eg (1977). Foods and diseases. Br J Soc prev Med 31 : 71 – 80.

Matsushima, I. (1982). Mechanisms of conversion of food components to mutagens and carcinogens. In : MS Arnott, J van Eys, YM wang (eds) : "Molecular Interrelations of Nutrition and Cancer". New York : Raven, PP. 35-42.

Mauron, U. (1981). The Maillard reaction in food; a critical review from the nutritional standpcint. Prog Food Nutr Sci 5 : 5 – 35.

Miller, A. B. (1986). Diet and nutrition in the context of cancer epidemiology. In : Genetic Toxicology of the Diet [Ed. Ib Knudsen] Alan R. Liss Inc. New York.

Ohgaki, H., S. Takayamd, and T. Sugimurd. (1991). Carcinogenicities of heterocyclic amines in cooked food. Mutat. Res. 259 : 399-410.

Powrie, WD., WuCH, MP. Rosin, and HF. Stich. (1981). Clastogenic and mutagenic activities of Maillard reaction model systems. J Food Sci 46 : 1433 – 1438.

Shibamoto, I. (1983). Heterocyclic compounds in browning and browning/ nitrite model systems : occurrence, formation mechanisms, flavor chracteristics and mutagenic activity. In G Charalambous, G Inglett (eds) : "Instrumental Analysis of Foods" (vol I) New York : Academic Press pp. 229 – 278.

Shibamoto, I., O. Hishmura, and S. Mihara. (1980). Heterocyclic compounds found in cooked meats. J Agric Food Chem 28 : 237 – 243.

Spingarn, E., and CT. Garvie. (1979). Formation of mutagers in sugar-ammonia model systems. J Agric Food Chem 27 : 1319-1321.

Sugimura, T., and L. Sato. (1983). Mutagens-carcinogens in food. Cancer Res (Supp1) 43 : 24155-24215.

Sugimura, T., S. Kondo, and H. Takebe. (1982). "Environmental Mutagens and Carcinogens". Tokyo : University of Tokyo Press. PP. 3-20.

Wattenberg, LW. (1963). Inhibition of neoplasia by minor dietary constituents. Cancer Res (Supp1) 43 : 24485-24535.

الملخص العربي

الأغذية السريعة تحدث ضرر وراثى خلوى فى جينوم الفول والبصل

دجانه يحيى الأيوبى

كلية التربية للبنات – وراثة – الرياض – المملكة العربية السعودية

زاد الاهتمام – فى الوقت الحالى – بالصحة العامة للإنسان – والدور الذى تلعبه ملوثات البيئة فى احداث ضرر للمادة الوراثية. ويحتل الغذاء المرتبة الأولى فى اهتمام العلماء ودوره فى احداث ضرر وأمراض وراثية. تم تصميم البحث الحالى لمعرفة الضرر الوراثى الخلوى الذى تحدثه بعض الأطعمة السريعة للمادة الوراثية لنبات البصل والفول – تم اختيار شرائح البطاطس المقرمشة ومقرمشات الذرة علاوة على البرجر كثلاث مصادر مختلفة للطاقة الغذائية المستخدمة فى الوقت الحالى وتم استخلاص الجزء الذائب في الماء وعوملت خلايا القمم النامية فى الجذور العرضية لنبات البصل والجذور الثانوية لنبات الفول لثلاث فترات زمنية مختلفة هى ٦ ، ١٢، ٢٤ ساعة وباستخدام تركيزات مختلفة هى ١٠ ، ٢٠، ٣٠، ٤٠، ٥٠، ١٠٠ جرام لكل لتر ماء. وتم تقدير النشاط الخلوى والتغيرات الكروموسومية الشاذة بأنواعها المختلفة. أظهرت النتائج قدره موجبة لهذه الأغذية على احداث سمية خلوية وإنتاج شذوذ كروكوسومي مثل الفجوات والشظايا واللزوجة الكروموسومية وظهرت بصورة معنوية زادت باستخدام التركيز وزادت بزيادة زمن المعاملة.