

Influence Of *Acacia nilotica* On Arsenic Induced Genotoxicity In Male and Female Mice

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Abstract

For centuries, plants have been used in traditional medicine and there has been recent interest in the chemopreventive properties of compounds derived from plants. In the present study, we investigated the effects of extracts of *Acacia nilotica* leaves on the genotoxicity of arsenic .

Arsenic contamination in groundwater a global human health hazard .There is no effective remedial action of chronic arsenicosis. However, a well-nourished diet can modulate the onset of adverse health effects and delayed the effect of arsenic in drinking water. In the present work, genotoxic effects were induced by sodium arsenate through oral administration, and the protective effect of *Acacia nilotica* was studied. Chromosomal aberrations were more pronounced in sodium arsenate treated mice, while supplementation of *Acacia nilotica* with sodium arsenate reduced the incidence of the aberrations. The mean of DNA fragmentation induced by sodium arsenate was highly significant increase. However, the administration of *Acacia nilotica* significantly decreased DNA fragmentation induced by sodium arsenate. The mean number of sperms, were decreased significantly after treatment with sodium arsenate, while administration of *Acacia nilotica* increased the number of sperm in mice treated with sodium arsenate, and also decreased the percentage of sperm abnormalities induced by sodium arsenate.

The outcome of study showed that *Acacia nilotica* has the efficiency to encounter the genotoxic effects induced by arsenic.

Introduction:

For centuries, arsenic has played an important role in science and medicine. Arsenic for its environmental perversiveness has gained unexpected entrance to the human body through food, water and air, thereby poisoning a great threat to public health due to its toxic effect and carcinogenicity. Thus, in modern scenario arsenic is synonymous with "toxic" and is documented as a paradoxical human carcinogen, although its mechanism of induction of neoplasia remains elusive (Mahata *et al.*, 2004).

Arsenic has been found to be genotoxic. It induced sister chromatid exchanges and is clastogenic in a range of in vitro systems using both human and animal derived cells (Bernstam&Nriagu

2000 and Gradecka *et al.*, 2001). Other studies have found that exposure to inorganic arsenic increases the frequency of micronuclei, chromosome aberrations and sister chromatid exchanges both in humans and experimental animals. (Mahata *et al.*, 2003; Mahata *et al.*, 2004 and Avani&Rao 2007). De Chaudhuri *et al.*, (2008) showed that health effects (i.e. peripheral neuropathy, conjunctivitis and respiratory illness) and chromosomal aberrations were significantly higher in the keratotic group compared to individuals with no skin lesions.

Arsenic has also been shown to enhance the clastogenicity and mutagenicity of other DNA damaging agents. Other change in vitro, are related

directly, or indirectly to cell replication (Simeonova and Luster 2000). Gradecka *et al.*, (2001) suggested that arsenic acts through a series of chemical reactions in the cell, interacting strongly with nearby molecules, and changing the structure of cellular components such as DNA. Arsenic has been shown not to affect DNA directly, but to intensify the toxic effects of other physical and chemical agents by inhibiting DNA repair, changing cell redox potential, and altering DNA methylation of cell-cycle control proteins.

Celino *et al.*, (2009) suggested that low doses of arsenic may inhibit spermatogenesis via steroidogenesis suppression, while high doses of arsenic induce oxidative stress-mediated germ cell apoptosis.

Plants are important source of potentially useful structures for the development of new chemotherapeutic agents. The first step towards this goal is the *in vitro* antibacterial activity assay (Tona *et al.*, 1998). Many reports are available on the antiviral, antibacterial, antifungal, antihelminthic, antimolluscal and anti-inflammatory properties of plants (Samy & Ignacimuthu, 2000; Palombo & Semple, 2001; Kumaraswamy *et al.*, 2002; Stepanovic *et al.*, 2003; Bylka *et al.*, 2004; Behera & Misra, 2005; Govindarajan *et al.*, 2006 and Mahesh & Satish 2008). Some of these observations have helped in identifying the active principle responsible for such activities and in the developing drugs for the therapeutic use in human beings.

Acacia nilotica belonging to the Leguminosae family and sub-family Mimosaceae has been subjected to long term clinical trials in folk medicine (El-Tahir *et al.*, 1999). The bark, root, gum, leaves and flowers have found use for skin diseases, diarrhoea, dysentery, cough, diabetes, eczema, wound healing, burning sensation and as an astringent, demulcent, anti-asthmatic. It has been reported that various parts of the plant are rich in tannins (ellagic acid, gallic acid, tannic acid), stearic acid, vitamin-C (ascorbic acid),

carotene, crude protein, crude fiber, arabin, calcium, magnesium and selenium (Marohasy, 1995; Mohanty *et al.*, 1996 and Sotohy *et al.*, 1997).

Acacia nilotica extracts were found in the Ames assay to have potent antimutagenic effects against the direct acting mutagens NPD, sodium azide, and the S9-dependent mutagen 2-AF (Arora *et al.*, 2005). Hédi *et al.*, (2007) indicate that extracts of *Acacia salicina* leaves are a significant source of compounds with antigenotoxic and antioxidant activity (most likely phenolic compounds and sterols), and thus may be useful for chemoprevention.

In this study, we evaluated the genotoxic effects of arsenic in male and female mice and the possible protective effect of *Acacia nilotica* on genotoxic induced by sodium arsenate.

Material and Methods:

Material

Sodium arsenate (Sigma Chemical, USA)
Acacia nilotica extracts

Preparation of *Acacia nilotica* extracts

Acacia nilotica leaves were collected from Upper Egypt. Leaves were air-dried in shade without direct exposure to sun rays and powdered. Leaves were distilled in a round bottom flask using double distilled water (DDW) at 60°C. The procedure was repeated thrice for 12 hrs duration and the leftover residue after the third distillation was filtered and the remaining distilled water was allowed to evaporate. Thereafter, remaining material was dried in oven and collected (Meena *et al.*, 2006).

Animals and Experimental Design:

Forty male and forty female Swiss albino mice (body weight 20-25g) were obtained from the house of experimental animals, the National Research Center, Cairo, Egypt. The animals were kept under the normal healthy laboratory condition and divided into four groups to carry out the experimental study. These groups were as follows:

- 1- Control group.
- 2- *Acacia nilotica* (AN) group, received oral daily 30 mg/kg body weight *Acacia nilotica*.
- 3- Arsenic group, received oral sodium arsenate at a dose of 5.0 mg/kg body weight.
- 4- *Acacia nilotica* and sodium arsenate group, received *Acacia nilotica* at a dose of 30 mg/kg body weight and received sodium arsenate at a dose of 5.0 mg/kg body weight.

Acacia nilotica and sodium arsenate were ingested through a gastric tube. The dose was given daily for 6 weeks.

After 6 weeks of drug administration, the animals were sacrificed and bone marrow was collected. Chromosome preparation from bone marrow cells of both male and female mice was prepared according to the procedure of **Yosida and Amano (1965)**.

DNA fragmentation in liver cells was carried out according to **Burton (1956) and Perandones et al., (1993)**

Sperm abnormality preparation from testis cells was prepared according to the method of **Wyrobek and Bruce (1975)**.

Statistical analysis:

All data were statistically analyzed using the general linear models procedure of the statistical analysis system (**SAS Institute, 1982**). The significance of the differences among treatment groups was determined by Waller–Duncan ratio (**Waller and Duncan, 1969**). All statements of significance were based on probability of $P \leq 0.05$.

Results

Chromosome aberration

In male

In bone marrow cells of mice (control group), the means of aberrant cells were 4.0. The frequency of aberrant cells in the group of animal treated with *Acacia nilotica* only slightly increased when compared to control 5.9. The chromosomal

abnormalities showed an increase in the structure types: gap 1.6, centric fusion 0.5 and end to end association 2.0. However, deletion and break type was the same means, i.e. 1.0 & 0.8 (table 1).

The group of animal treated with sodium arsenate showed a significant increase in the frequency of abnormal cells (11.2). The group of animal treated with *Acacia nilotica* and sodium arsenate, has shown a significant decrease in the number of cell carrying aberrations at a means of 8.7. All types of chromosome aberration exhibited a decrease in number. The number in gaps, deletion, break, centric fusion and end to end association types were 2.0, 2.2, 0.9, 0.4 & 2.8 (table 1).

In female

In mice treated with sodium arsenate, the frequency of chromosome aberration before treatment was 5.4 (control) and after treatment was a high significant increase (11.8). All types of chromosome aberration exhibited increase in number. The number in gaps, deletion, break, centric fusion and end to end association types were 2.8, 3.6, 2.4, 0.4 & 1.8 (table 2).

The effect of *Acacia nilotica* on the frequency of abnormal cells caused increase in the frequency chromosome aberration. It was observed at a level of 6.5. The change in chromosome aberrations showed gaps, deletion, break and end to end association (table 2).

Treatment with *Acacia nilotica* is effective against all types of chromosome aberrations appeared in mice treated with sodium arsenate. There is a significant reduction in the percentage of aberrant cells as well as chromosome aberrations in the treated group compared with arsenic group. The number in gaps, deletion, break and end to end association types were 1.8, 2.3, 1.4 & 0.5 (table 2).

DNA fragmentation

In male

The mean of DNA fragmentation showed nonsignificant increase after treatment with *Acacia nilotica* compared to control group. However the mean of DNA fragmentation induced by sodium arsenate highly significant increased. Administration of *Acacia nilotica* decreased DNA fragmentation induced by sodium arsenate (table 3).

In female

Nonsignificant increased was shown when mice treated with *Acacia nilotica*. The mean of DNA fragmentation induced by sodium arsenate highly significant increased. However, the administration of *Acacia nilotica* significantly decreased DNA fragmentation induced by sodium arsenate (table 4).

Sperm count in male mice

Treatment with *Acacia nilotica* only is effective on the number of sperm in mice at mean of 18.67. However, the mean number

of sperm induced by sodium arsenate highly significant decreased (10.24). Administration of *Acacia nilotica* increased the number of sperm in mice treated with sodium arsenate (15.79) (table 5).

Sperm abnormality

Sodium arsenate induced an increase in the means of abnormal sperm. Such means was found to be highly significant compared with control group. Table (6) illustrates the means of the different types of sperm abnormalities after treatment with arsenic. The dominant abnormalities found were amorph, without hook, banana, big head and coiled tail. The other abnormalities were few in number.

Acacia nilotica highly significant decreased the percentage of sperm abnormalities induced by sodium arsenate. All types of sperm abnormalities exhibited a decrease in number (table 6).

The group of animal treated with *Acacia nilotica* only has shown a significant increase in the means of sperm abnormalities at a means of 38.2 (table 6)

Table 1: Total chromosomal aberration in bone marrow cells of male mice treated with *Acacia nilotica* extract and arsenic (Mean ± S.D)

Groups	Structural chromosomal aberrations					More aberrations in one cell	Total chromosomal aberrations	
	Gap	Deletion	Break and fragment	Centric fusion	End to End association		including gap	excluding gap
Control	1.2 ± 0.83	1.0 ± 0.7	0.8 ± 0.44	0.4 ± 0.2	0.6 ± 0.54	0.4 ± 0.2	4.0 ± 0.7	2.8 ± 0.7
<i>Acacia nilotica</i>	1.6 ± 0.54	1.0 ± 0.0	0.8 ± 0.6	0.5 ± 0.4	2.0 ± 0.7*	0.0	5.9 ± 0.89*	4.3 ± 0.71
Arsenic	2.6 ± 0.5*	2.4 ± 0.89*	1.4 ± 0.54	0.6 ± 0.54	3.2 ± 1.09**	1.0 ± 0.7*	11.2 ± 0.83**	8.6 ± 1.1**
Arsenic + <i>Acacia nilotica</i>	2.0 ± 0.7*	2.2 ± 0.44*	0.9 ± 0.2	0.4 ± 0.2	2.8 ± 0.5**	0.4 ± 0.2	8.7 ± 0.54**	6.7 ± 0.54*

* Significant ** highly significant (P<0.05)

Table 2: Total chromosomal aberration in bone marrow cells of female mice treated with *Acacia nilotica* extract and arsenic (Mean \pm S.D)

Groups	Structural chromosomal aberrations						Total chromosomal aberrations	
	Gap	Deletion	Break and fragment	Centric fusion	End to End association	More aberrations in one cell	including gap	excluding gap
Control	1.6 \pm 0.54	1.4 \pm 0.5	1.4 \pm 0.54	0.0	0.6 \pm 0.54	0.4 \pm 0.2	5.4 \pm 0.45	3.8 \pm 0.5
<i>Acacia nilotica</i>	1.8 \pm 0.4	1.6 \pm 0.54	1.8 \pm 0.44	0.0	0.5 \pm 0.4	0.6 \pm 0.54	6.3 \pm 0.84*	4.5 \pm 0.54
Arsenic	2.8 \pm 0.84*	3.6 \pm 0.9**	2.4 \pm 0.8	0.4 \pm 0.2	1.8 \pm 0.44*	0.8 \pm 0.44	11.8 \pm 0.89**	9.0 \pm 1.5*
Arsenic + <i>Acacia nilotica</i>	1.8 \pm 0.44	2.3 \pm 0.89*	1.4 \pm 0.9	0.0	0.5 \pm 0.4	0.5 \pm 0.4	6.5 \pm 1.1*	4.7 \pm 0.83

* Significant ** highly significant (P<0.05)

Table (3): The effect of arsenic and *Acacia nilotica* extract on DNA fragmentation in male mice

Groups	DNA fragmentation for male Mean \pm S.E
Control	13.98 \pm 0.8
<i>Acacia nilotica</i>	16.93 \pm 0.81
Arsenic	26.26 \pm 0.75*
Arsenic + <i>Acacia nilotica</i>	18. 05 \pm 0. 95

* Significant (P<0.05)

Table (4): The effect of arsenic and *Acacia nilotica* extract on DNA fragmentation in femal mice

Groups	DNA fragmentation for female Mean \pm S.E
Control	18.27 \pm 0.58
<i>Acacia nilotica</i>	23.68 \pm 0.87
Arsenic	29.86 \pm 0. 14 *
Arsenic + <i>Acacia nilotica</i>	24. 06 \pm 1.1 *

* Significant (P<0.05)

Table (5): The effect of arsenic and *Acacia nilotica* extract on epididymal count in male mice

Groups	No.of animals	Sperm count (million/ml)	(%) of abnormal cells
Control	5	24.0218 \pm 1.4794	5.06
<i>Acacia nilotica</i>	5	18.6707 \pm 0.9793*	7.2
Arsenic	5	10.2390 \pm 0.7239* *	13.05
Arsenic + <i>Acacia nilotica</i>	5	15.7860 \pm 1.0649 *	11.1

* Significant * * highly significant (P<0.05)

Table (6): Total sperm abnormality of male mice treated with *Acacia nilotica* extract and arsenic (Mean \pm S.D)

Groups	head abnormalities				Tail abnormal		Total abnormal sperm
	amorphous	Without hook	banana	big	divided	coiled	
control	12.4 \pm 0.54	4.0 \pm 1.2	0.4 \pm 0.2	0.0	0.6 \pm 0.5	8.2 \pm 1.0	25.6 \pm 1.3
<i>Acacia nilotica</i>	17.8 \pm 1.3*	7.8 \pm 1.4*	1.8 \pm 0.8*	1.4 \pm 0.5*	0.4 \pm 0.2	9.0 \pm 1.0	38.2 \pm 1.2*
Arsenic	27.2 \pm 1.4**	15.0 \pm 1.5**	3.2 \pm 1.3**	2.0 \pm 1.0*	0.6 \pm 0.54	27.4 \pm 1.1*	75.4 \pm 0.9**
Arsenic + <i>Acacia nilotica</i>	25.0 \pm 1.2**	5.2 \pm 1.0	1.2 \pm 1.0*	1.2 \pm 0.8*	0.9 \pm 0.6	26.26 \pm 0.82*	59.7 \pm 1.5**

* Significant ** highly significant (P<0.05)

Discussion

Human activities have increased the possibility of exposure to naturally occurring metals causing a greater risk of exposure to toxic levels. The exposure of metals such as arsenic constitutes a major health concern. Arsenite induces DNA damage referred to as genotoxicity in human cells within a pathologically meaningful dose range. Arsenic toxicity is cell specific. Therefore, it is important that target cells be used for investigations (**Hamadeh et al., 2002**).

The genotoxicity of arsenic is due largely to the trivalent arsenicals. In human, arsenic is a chromosomal mutagen. Arsenic appears to have limited ability to induce point mutations. Elevated frequencies of micronuclei, chromosomal aberrations and aneuploidy were detected in the peripheral lymphocytes or urothelial cells, or both, of people exposed to elevated levels of arsenic. In mammal cells, arsenic caused various types of chromosomal mutations and aneuploidy (**IARC, 2004**).

The results of the present study showed that sodium arsenate is a genotoxic agent. This was evidenced by the increase incidence of chromosomal aberrations particularly the gap type in animals receiving arsenic. The importance of recording gaps in assessing the genotoxic effects of compound is controversial. Some authors considered gaps to be a sensitive indicator of chemically induced chromosomal damage, while others considered the scoring of gaps to be highly subjective and, therefore, an unsuitable indicator of genotoxicity (**Hassan, 1997**).

In the present study, sodium arsenate showed a significant increase in the frequency of abnormal cells. Also all types of chromosome aberration exhibited an increase in number. There are several reports on inorganic arsenic increases the frequency of micronuclei, chromosome aberrations and sister chromatid exchanges (**Gebel, 2001 and Waclavicek et al., 2001**). **Mahata et al. (2003)** indicates that enhanced rates of chromosomal aberrations

(CA) and sister chromatid exchanges (SCE) among the residents of North 24 Pargons are indicative of the cytogenetic damage due to long term exposure to arsenic through consumption of contaminated water. **Mahata et al. (2004)** show that sodium arsenite induced a significantly higher percentage of aberrant cells in the lymphocytes of control individuals than in the lymphocytes of both the exposed groups. **De Chaudhuri et al. (2008)** suggests that individuals with keratosis are more susceptible to arsenic-induced health effects and genetic damage and that the arginine variant of p53 can further influence the repair capacity of arsenic-exposed individuals, leading to increased accumulation of chromosomal aberrations. **Singh et al. (2008)** indicate that chromosomal aberrations were more pronounced in arsenic treated mice.

The mean of DNA fragmentation induced by sodium arsenate highly significant increased. However, the administration of *Acacia nilotica* significantly decreased DNA fragmentation induced by sodium arsenate. Arsenic has been shown to induce DNA damage in human cells. Inorganic arsenic inhibits DNA repair. Specifically, a significant increase in comet tail-length at doses 0 to 6.45 mg/kg body weight demonstrated that arsenic trioxide cause DNA damage effectively (**Gebel, 2001 and Waclavicek et al., 2001**). Arsenic was highly genotoxic to the three cell lines. No significant differences in DNA cleavage were observed between acute and chronic exposures. In acute exposure, arsenic genotoxicity was more severe with dendritic cells, while melanocytes were more sensitive to arsenic cytotoxicity. Similarly, chronically exposed dendritic cells showed the maximum genotoxic damage while melanocytes were more sensitive to arsenic cytotoxicity (**Evans et al., 2004**). Chronic arsenic exposure caused significant single-strand DNA damage in lymphocytes as depicted by comet with a tail in arsenic-exposed cells compared with the control cells. **Mishra and Flora (2008)** suggested that arsenic induces differential oxidative stress in brain regions with cortex

followed by hippocampus and causes single-strand DNA damage in lymphocytes. Functional profiling using homozygous deletion mutants provided evidence of the requirement of highly conserved biological processes in the response against both arsenicals including tubulin folding, DNA double-strand break repair, and chromatin modification (**Jo et al., 2009**).

One of the possible mechanisms suggesting arsenic toxicity is the generation of reactive oxygen species (ROS). **Liu et al. (2001)** found that reactive oxygen species, particularly hydroxyl radicals, play an important causal role in the genotoxicity of arsenical compounds in mammalian cells.

The present study indicated that sodium arsenate reduced the number of sperm in mice. Also, sodium arsenate induces an increase the percentage of abnormal sperm and an increase in the different types of sperm abnormalities was observed after treatment with sodium arsenate. **Celino et al. (2009)** suggested that low doses of arsenic may inhibit spermatogenesis via steroidogenesis suppression, while high doses of arsenic induce oxidative stress-mediated germ cell apoptosis. *Acacia nilotica* increased the number of sperm in mice treated with sodium arsenate and highly significant decreased the percentage of sperm abnormalities induced by sodium arsenate. All types of sperm abnormalities exhibited a decrease in number.

In the present study, *Acacia nilotica* was effective against all types of chromosome aberrations appeared in mice treated with sodium arsenate. There was a significant reduction in the percentage of aberrant cells as well as chromosome aberrations in the treated group compared with arsenic group. **Meena et al. (2006)** found that a significant reduction in the frequency of micronuclei was observed in mice treated by oral gavage with the aqueous extracts of *Acacia nilotica*. There is also a significant decrease in total chromosomal aberrations in the form of chromatid breaks, chromosome breaks, centric rings, dicentrics, acentric fragments

and exchange as compared with the group given 7, 12-dimethylbenz (a)anthracene (DMBA).

Acacia nilotica significantly decreased DNA fragmentation induced by sodium arsenate. Antimutagens and anticarcinogens are natural or synthetic substances able to inhibit or to reduce spontaneous or induced DNA alteration. They react directly with mutagens or on the process of their activation (**El Hamss and Idaomar, 2002**).

Acacia nilotica showed a marked anti-mutagenic effect. Furthermore, it was more effective against indirect acting mutagens (**Arora et al., 2003**).

Acacia nilotica (Linn.) gum, flower and leaf extract may play an additional and synergistic antimutagenic role in the presence of tannins (ellagic acid, gallic acid and tannic acid), crude protein, crude fiber, arabin, calcium, magnesium, selenium, stearic acid with three phenolic components (viz. Kaempferol-3-glucoside, isoquercetin, leucocyanidin) Vit.C (ascorbic acid), carotene, alkaloids and saponins (**Sotohy et al., 1997 and Arora et al., 2003**).

Chang et al. (2001) indicate that both bark and heartwood extracts of *Acacia confusa* clearly have strong antioxidant effects. Furthermore, the heartwood extract protected PhiX174 supercoiled DNA against strand scission induced by ultraviolet photolysis of H₂O₂, and it reduced the amounts of intracellular hydrogen peroxide, a reactive oxygen species, when it was co-incubated with human promyelocytic leukemia (HL-60) cells under oxidative stress. **Bouhleb et al. (2008)** showed that *Acacia salicina* extracts decreased the DNA damage caused by H₂O₂.

In conclusion, arsenic was highly genotoxic in both male and female mice, while *Acacia nilotica* reduced the percentage of aberrant cells, decreased DNA fragmentation, increased the number of sperm and decreased the percentage of sperm abnormalities. *Acacia nilotica* protected the cells from the oxidative stress induced by arsenic.

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تأثير خلاصة نبات آكاسيا على السمية الوراثية الناتجة عن الزرنيخ

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يعتبر الزرنيخ من الملوثات البيئية التى تخترق جسم الإنسان من خلال الطعام والماء والهواء بصورة غير متوقعة. وأثبت العديد من الأبحاث أن الزرنيخ يؤدي إلى بعض التشوهات الكروموسومية.

أجرى هذا البحث لدارسة التأثير الواقى لنبات الأكاسيا لتفادى الأثار الضارة على الكروموسومات والحامض النووى الذى أوكسى ريبوزى والحيوانات المنوية التى يسببها الزرنيخ. ولقد أجرى هذا البحث على الفئران البيضاء الذكور والإناث والتي قسمت إلى المجموعات التالية:

- 1- المجموعة الضابطة.
- 2- المجموعة المعاملة بالزرنيخ (بجرعة 5 ملغ /كجم).
- 3- المجموعة المعاملة بنبات الأكاسيا (بجرعة 30 ملغ /كجم).
- 4- المجموعة المعاملة بنبات الأكاسيا(بجرعة 30 ملغ /كجم) و الزرنيخ (بجرعة 5 ملغ /كجم).

وبعد ستة أسابيع تم تشريح عشرة من الفئران فى كل مجموعة (الذكور والإناث) وجمعت العينات من أنسجة الكبد وذلك لتقدير تفتت الحامض النووى الذى أوكسى ريبوزى والخصية لتحضير الحيوانات المنوية كما تم تحضير الكروموسومات من خلايا النخاع العظمى .

تتلخص نتائج هذا البحث فيما يلى :

الفئران المعالجة بالزرنيخ فقط ظهر بها إرتفاع نسبة التشوه الكروموسومى وزيادة نسبة تفتت الحامض النووى الذى أوكسى ريبوزى ونقص عدد الحيوانات المنوية وزيادة نسبة التشوهات بها . أما الفئران المعالجة بنبات الأكاسيا مع الزرنيخ فحدث فيها إنخفاض نسبة التشوه الكروموسومى ونقص نسبة تفتت الحامض النووى الذى أوكسى ريبوزى وزيادة عدد الحيوانات المنوية و إنخفاض التشوهات بها.

وقد أثبتت التجربة مدى فائدة نبات الأكاسيا فى حماية الخلية من الأثار الضارة للزرنيخ.