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Evaluation of anti-cytogenotoxic potential of *Atriplex portulacoides* crude extract in mice bone marrow.

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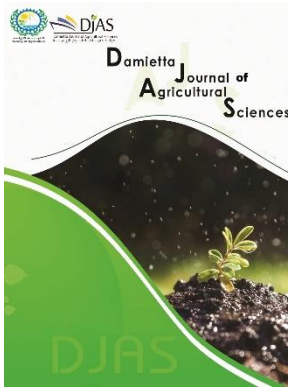

ABSTRACT

Medicinal plants are essential to traditional medical systems because they are abundant in biologically active compounds that support the physiological and biochemical functions of living organisms. Among these actions, anti-cytogenotoxic properties are particularly noteworthy, as they offer protection against cytogenotoxicity caused by unfavorable environmental conditions. Our current study evaluates the anti-cytogenotoxic properties of the crude extract of *A. portulacoides* by assessing the rates of aberrant mitosis in mice bone marrow. Mice were administered 5 mg/kg body weight of *A. portulacoides* crude extract three times a week for two weeks before receiving mitomycin C (MMC). A separate group of mice received MMC alone as a positive control. The findings demonstrated that exposure to MMC significantly increased chromosomal abnormalities compared to the control group. Meanwhile, pre-treatment with *A. portulacoides* crude extract resulted in a significant reduction in the MMC-induced rate of both structural and numerical chromosomal aberrations and increased the mitotic index. Overall, our results suggest that the *A. portulacoides* crude extract could be a protective food supplement and a promising agent to reduce the cytogenotoxicity effects that may result from anticancer drugs. However, further study is necessary to confirm these potential benefits

Key words: Medicinal plants mitomycin C anti-cytogenotoxic

INTRODUCTION

New medicinal preparations to combat a wide range of illnesses, including cancer, Continuously sought. This is due to the fact that current medications frequently have undesirable side effects or eventually lose their effectiveness (**Demirci *et al*., 2010)**. According to epidemiological studies, numerous malignancies depend on both inherited mutator phenotype and multiple mutational etiology. Therefore, looking for mutagenesis inhibitors could be a helpful Strategy for identifying–anticarcinogenic substances. The search for mutagenesis inhibitors may be part of the process of finding novel anti-tumor drugs. One method to focus the search could be to screen for traditional medicinal plants that have been used for centuries to treat a variety of illnesses, including cancer, and that have been effective in treating individuals **(Van Wyk *et al.*, 1997).** As a matter of fact, a single plant can contain a wide variety of phytochemicals, such as alkaloid compounds, diuretics, tannins that act as natural antibiotics, phenolic compounds for antioxidants and many other pharmacological properties, and bitter compounds that stimulate the digestive system. It is also crucial to remember that most traditional medicinal herbs have never undergone the thorough toxicological testing necessary for contemporary pharmaceutical substances. They are frequently thought to be safe due to their lengthy history of traditional use. However, studies have revealed that many plants used in traditional medicine or as food ingredients have mutagenic properties *in vitro* **(Cardoso *et al*., 2006; Déciga-Campos *et al.*, 2007; Mohd-Fuat *et al*., 2007)** or toxic and carcinogenic **(De Sá Ferreira and Ferrão Vargas, 1999)** properties. The genus *Atriplex* is a member of the Chenopodioideae subfamily, specifically the tribe Atripliceae. **(FuentesBazan *et al*., 2012a)** It belongs to the *Amaranthaceae* family, which is the most representative family of halophytes by the North Sea (**Lefèvre and Rivière, 2020).** Because of its molluscicidal, antifungal, antibacterial, antiviral, anti-diabetic, anti-cancer, and antioxidant properties, Atriplex species, including *Atriplex portulacoides*, are potential natural candidates for

potential drug development **(Zanella and Vianello, 2020).** The presence of beneficial Biochemicals, including terpenoids, hydroxyecdysone, flavonoids, and phenolics is linked to these pharmacological effects. These plants are also rich in proteins, flavonoids, amino acids, vitamins A and C, and other nutrients. The efficacy of these properties can be refined with the appropriate use of the compounds derived from these plants.

Or The medicinal potential of these plants can be enhanced by understanding and utilizing their active compounds. Because of its remarkable therapeutic applications, *Atriplex* species are employed as a traditional means of curing illnesses. Therefore, it's crucial to check medicinal herbs for mutagenic potential. **(Ali *et al* ., 2021)**. Clear mutagenic plants should be regarded as potentially dangerous and need more research before their continued usage is advised. However, plants that exhibit clear antimutagenic potential may also be deemed intriguing for medicinal application and demand more thorough studies of their pharmacological characteristics. Because most anticancer drugs (such as the spindle-disturbing substances vinblastine and taxol) are mutagenic, mutagenicity can also be used as an anticancer tool **(Ferguson and Pearson, 1996)**. Short-term mutagenicity assays are effective tools for evaluating the mutagenicity and predicted carcinogenicity of both single and mixed compounds, either in direct applications or under environmental exposure. The chromosomal abnormalities assay in mice has long been used as a reliable and sensitive method to assess the clastogenic effect of mutagenic substances **(Tice *et al*.,1994).** Therefore, this study was designed to investigate the potential protective role of the crude extract of *Atriplex portulacoides* as an anti-agent against the cytogenotoxicity induced by the most anticancer drugs.

Materials and methods

1.Collection and preparation of plant materials

The methods and protocols followed by **Moustafa *et al*., 2014** were used with modifications as follows: Fresh whole plants were collected from Port Said desert in Egypt. The plant materials were dried at ambient temperature for two weeks and stored in a dry place prior to use. The plant was washed well with water, dried at room temperature in the dark, and then ground in an electric grinder to give a coarse powder. Shoot powders (50 g) were extracted by using 1 methylene chloride: 1 methanol as a solvent. The powders were soaked in 175 ml solvent at room temperature for 48h. The plant extracts were collected drop wise and filtered using Whatman No.1 filter paper. The residues were soaked in 150 ml of solvent for 24h and were filtered again. The extract was then dried and finally placed in glass vials and stored at -20º C and the extract was re-suspended in water before testing.

2. Cytogenotoxicity assessment

2.1. Experimental design and treatments

Before the investigation, 15 albino male mice (2n=40) weighing 25±2 grams were obtained from the National Research Center's animal house in Dokki, Cairo, Egypt. They were kept acclimating in preparation for treatments with the Crude extract of *A. portulacoides* (CEA), and Mitomycin C (MMC) was used as positive control. Mitomycin C is one of the most reliable mutagenic materials in Cytogenotoxicity studies due to its consistent ability to cause chromosomal abnormalities and cell death, making it a standard for comparison with other substances **(Li *et al.*, 2009).** Following a two-week acclimatization period for the animals in the laboratory, the mice were then divided into three groups of five animals each, as follows: Group 1(Control): Normal mice, as a negative control. Group 2 (MMC): Received 0.6 mg/kg b.w MMC as positive control 24 hours. Group 3 (CEA-MMC): Crude extract of *A. portulacoides* at the dose of 5 mg/kg b.w 15 days (6 times) was orally given, then received 0.6 mg/kg b.w Mitomycin C for 24 hours Mice were killed and subjected to chromosomal aberration analysis in somatic bone marrow cells.

2.2. Preparation of bone marrow somatic cell chromosomes:

Chromosomes of bone marrow somatic cells were prepared according to **Yosida and Amano, 1965.** At 100x magnification, 50 metaphase spreads per animal were examined under a microscope to score various chromosomal abnormalities, including numerical and structural aberrations

2.3. Estimation of Mitotic Index (MI%)

The mitotic activity of bone marrow (b.m.) cells was assessed using the mitotic index, which is the number of dividing cells per 1000 cells. The mitotic index was computed using the same slides that were prepared for the evaluation of chromosomal abnormalities. The number of dividing cells and the total number of cells were counted by tracking randomly chosen fields on the slides. In each animal, at least 2000 cells were analyzed.

2.4. Statistical Analysis.

All data are expressed as means ± SD. The significance of differences between groups was evaluated using a t-test. Treatment means were

compared using the least significant difference (LSD) method at a 5% probability level ($p<0.05$), using SAS (version 9.1, SAS Institute, Cary, NC, USA). Means sharing the same letter are not significantly different.

RESULTS AND DISCUSSION

Cytogenotoxicity Assessment

The untreated mice had a relatively stable (2n = 40) karyotype (Fig 1) and did not show any polyploidy cells (Table 1). Several chromosomal abnormalities, including numerical and structural aberrations, were observed during the metaphase analysis of bone marrow cells. The numerical aberrations were aneuploidy (<2n: hypoploidy and >2n: hyperploidy) and polyploidy (Fig. 2), while the structural aberrations were deletion and fragmentation (Figs. 3 and 4).

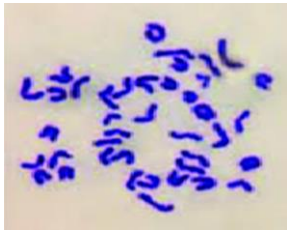

Fig. 1: Normal metaphase spread from the untreated mice bone marrow.

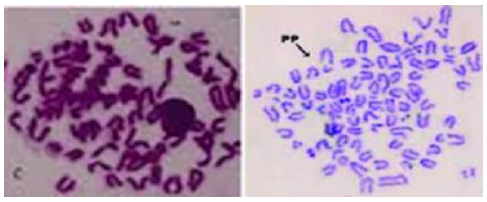

Fig. 2: Metaphase spread from the treated mice bone marrow, displaying polyploidies >80.

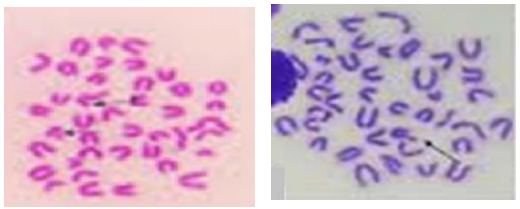

Fig. 3: Metaphase spread from the treated mice bone marrow, displaying deletions.

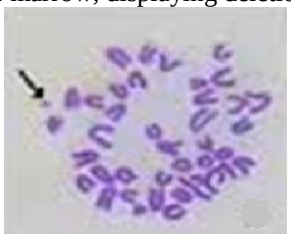

Fig. 4: Metaphase spread from the treated mice bone marrow, displaying fragment.

Table 1: Means and standard deviation of Chromosomal abnormalities and mitotic index in treated mice bone marrow.

Treatments	Structural aberrations			Numerical aberrations				Mitotic activity	
	Dele	Frag	TSA	>2n	<2n	Poly	TNA	DC	MI
Control	0.4±0.55	0.0±0.00	0.4±0.55	0.4±0.89	0.2±0.45	0.0±0.00	0.6±0.89	184±17.0	9.2±0.85
MMC	13.4±5.08	1.0±1.00	14.4±5.32	3.2±1.92	13.8±1.92	0.8±0.84	17.8±3.42	104±33.6	5.2±1.68
CEA-MMC	4.6±3.65	0.0±0.00	4.6±3.65	1.8±1.64	2.0±0.71	0.0±0.00	3.8±1.64	159±20.3	8.0±1.01
LSD 0.05	5.0	0.8	5.1	2.1	1.7	0.7	3.1	34.1	1.7

Dele: deletions; Frag: Fragments; TSA: Total structure aberrations; >2n: hyperploidy; <2n: hypoploidy; Poly: Polyploidy; TNA: Total numerical aberrations; DC: Divided cell; MI: Mitotic index; LSD 0.0: Least Significant Difference at a 5% probability level

Structural and numerical aberrations per 50 metaphase spreads induced by treatment with MMC and CEA-MMC as well as those of the control group are presented for individual animals in Table 1.

Table (1) demonstrates how the MMC and CEA-MMC groups differed in terms of structural and numerical aberrations from the control group. Chromosome abnormalities were significantly higher ($P < 0.05$) in the MMC-exposed group than in the control group. There was a considerable rise in the mean value of overall structural aberrations caused by MMC from (0.4±0.55) in control to (14.4 ±5.32) in MMC. The mean values of the total numerical aberrations caused by MMC considerably increase ($P < 0.05$) from (0.6 ±0.89) in control to (17.8 ± 3.42) in MMC, indicating the high cytogenotoxicity effect of MMC on mice bone marrow cells that was agree with **(Lee *et al*.,2006)** who demonstrated that Mitomycin C (MMC) induces various types of DNA damages that cause significant cytotoxicity to cells.

Wang *et al.*, (2007) demonstrated that because of MMC alkylating activity, it has a clastogenic impact. Accordingly, MMC requires a bioreductive transformation to produce active species that crosslink DNA. Depending on the biotransformation pathway, reactive oxygen species (ROS) may be produced during MMC metabolism (**Gustafson and Pritsos, 1992**). **Steensma *et al.*, (2000**) displayed that biological macromolecules like proteins, lipids, and nucleic acids are indiscriminately damaged when ROS interact with cells and surpass the body's natural antioxidant system.

The most affected type of structural aberrations recorded by MMC was deletions **(Fig.3).** MMC significantly increased the mean deletion values ($P < 0.05$) from (0.4±0.55) in the control group to (13.4±5.08) in MMC, which concurs with **Wanger *et al.*, (1980)** who showed that the primary kind of abnormality that MMC causes in bone marrow cells is deletion. According to **Cohen and Shaw (1964),** chromosome deletions, resulting in shorter chromosomes, can lead to severe consequences like sterility or nonfunctional cells if essential DNA is lost. These deletions can occur in any cell type and organism, potentially causing birth defects. Chromosome breaks, possibly induced by substances like Mitomycin C, are a primary cause of these deletions.

The fragments **(Fig.4)** were also increased, but by a very slight, insignificant, almost non-existent increase **(Table 1)**, it was (0.0±0.00) in control and (1.0±1.00) in MMC-exposed group.

Numerical aberrations include polyploidies **(Fig.2)** and aneuploidies (hypoploidy and hyperploidy). There was a slight increase in both polyploidy and hyperploidy in MMC group, and they were (0.00±0.00, 0.4±0.89) in control group and (0.8±0.84, 3.2±1.92) in MMC-exposed group, respectively. Hypoploidy was the most prevalent form of numerical abnormalities induced by MMC. MMC significantly increased the mean hypoploidy values ($P < 0.05$) from (0.2±0.45) in the control group to (13.8±1.92) in MMC **(Table 1)**. Studies of lymphocyte aneuploidy consistently show a significantly higher number of hypoploid cells than hyperploid cells. This finding is inconsistent with the expected outcome of nondisjunction. While initially considered a technical artifact, the pattern of smaller chromosomes being lost more frequently suggests a biological basis. Two explanations have been proposed: technical issues or chromosome/chromatid loss during cell division, which can result in either two hypoploid cells (when whole chromosomes are lost) or one diploid and one hypoploid cell (when sister chromatids are lost) **(Ford and Correll, 1988).**

The number of dividing cells in relation to total cell population (MI) reflects the mitotic activity of bone marrow cells. The slides prepared for the assessment of chromosomal aberrations are used for calculating the mitotic index. MMC dramatically reduced the mitotic activity of mice bone marrow cells. The mean of the mitotic activity in MMC treated animals decreased to (5.2±1.68) compared to the control, which is (9.2±0.85).

The proportion of mitotic activity values in the MMC-treated groups was lower than in the control group, according to the data. This might be because MMC exposure causes cells to advance from the S (DNA synthesis) phase of the cell cycle to the M (mitosis) phase More slowly. (**Patlolla *et al.*, 2010**). Nonetheless, this disturbance in cell cycle progression is most likely connected to MMC toxicity. It has been suggested that the DNA degradation that follows mitomycin therapy is caused by increased activity of DNA degradative enzymes and increased exposure of DNA to lysosomal action because of nuclear disintegration **(Crooke and Bradner, 1976).**

That is, treatment with MMC increased total structural aberrations and total numerical aberrations and reduced the mitotic index which is proven as MMC Cytogenotoxicity.

Evaluation of Anti-cytogenotoxic potential:

Fig. 5 illustrates the cytogenotoxicity effects of the MMC and pre-treatment with *A. portulacoides* (CEA-MMC) treatments compared with the control in terms of numerical and structural chromosomal aberrations, as well as mitotic index. Pre-treatment with *A.portulacoides* at a dose of 5.0 mg/kg showed that the frequency of chromosomal aberrations was significantly reduced. The pre-treatment group reduced the mean values of total structural aberrations from (14.4 ± 5.32) in the MMC group to (4.6 ± 3.65) in pre-treatment group, and reduced the mean values of the total numerical aberrations from (17.8 ± 3.42) in MMC to (3. 8 ± 1.64) in pre-treatment group**.** This demonstrates that MMC led to an increase in total structural aberrations and total numerical aberrations and that pre-treatment with *A. portulacoides* at a dose of 5.0 mg/kg protects the animals from aberrations that may be caused by MMC therefore significantly reduces aberrations. Pre-treatment with *A. portulacoides* crude extract also increased the mitotic indices significantly from (5.2±1.68) in MMC group to (8.0±1.01) in pre-treatment group, which means that treatment with *A. portulacoides* protects animals from the significant decrease in mitotic indices caused by MMC. This shows the anti-cytogenotoxicity effect of *A. portulacoides* and shows that *A.portulacoides* can lessen the mutagenic effect of MMC.

The ability of medicinal plants to alter the activity of environmental genotoxicants is being increasingly investigated, especially given their widespread use in traditional medicine. Given the established close relationship between carcinogenesis and mutagenesis, research has expanded to include plant extracts and products that can affect the genetic material involved in mutagenesis. This aligns with the findings of **Sarkar *et al.*, (1998)** who demonstrated that natural plant products can modulate the effects of known mutagens on living organisms by various mechanisms including activating the cell's natural defense against mutagens, preventing the formation of new mutagens.

The halophyte *Atriplex portulacoides* exhibits metabolic responses to oxidative and saline stresses and occurs in environments that are subject to seawater flooding. Long-chain lipids, sterols, phenolic compounds, glutathione, and carotenoids are among its constituents. This plant's organic components and micronutrients, including Fe, Zn, Co, and Cu, make it an ideal functional diet that may help combat oxidative stress and inflammation in humans and animals.In fact, a large number of these substances protect people from cancer, heart disease, and aging-related degenerative processes **(Zanella and Vianello, 2020).**

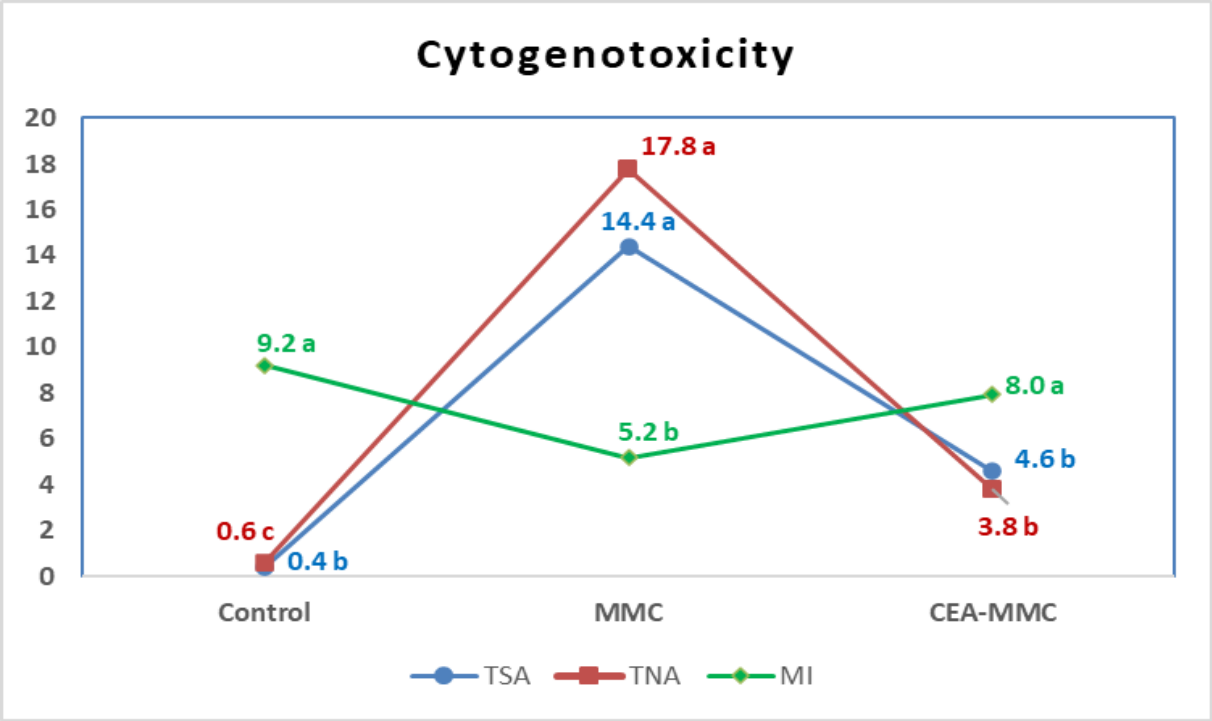

Fig. (5): Effect of Pre-treatment with *A. portulacoides* on Cytogenotoxicity induced by MMC in mice bone marrow cells.
TSA: Total structure aberrations; TNA: Total numerical aberrations; MI: Mitotic index.

Fig.5 shows how MMC increased chromosomal aberrations and decreased the mitotic index. It also shows that the cytogenotoxic effect of MMC is effectively decreased when *A. portulacoides* extract is used as a pre-treatment, which reduces chromosomal aberrations and increases the mitotic index. This may be due to the extract's antioxidant capacity, which affects its ability to interfere with the MMC mutagenic effect.

The findings reveal that the extract from *A. portulacoides* is a potent anti-mutagenic agent that improves and lessens the damage brought on by MMC. Although there is some experimental data on the anti-carcinogenic efficacy of *A. portulacoides* extracts, little is known about their anti-mutagenic activity.

According to the pre-treatment analysis, the extract of *A. portulacoides* may contain several chemicals that have dis-antimutagenic and bio-antimutagenic properties. These findings imply that *A. portulacoides* does not pose a risk to chromosomal integrity when used as a herbal remedy; however, additional molecular research is still required to determine the mechanism of action of these compounds in *A. portulacoides* extract and its anti-mutagenic properties, as well as to test its effects on DNA.

CONCLUSION

In summary, pretreatment with the crude extract of *A. portulacoides* demonstrated significant anti-cytogenotoxic activity against Mitomycin C-induced damage in mice. This was evidenced by a significant reduction in chromosomal aberration rates and a significant increase in the mitotic index, suggesting the potential of the *A. portulacoides* crude extract to reduce the cytogenotoxicity effects of anticancer drugs. However, further study is necessary to confirm these potential benefits.

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CONFLICTS OF INTEREST

The authors declare that they have no conflict of interest.

AUTHORS CONTRIBUTION

The authors developed the concept of the manuscript. All authors checked and confirmed the final revised manuscript.

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الملخص العربى

تقييم القدرة المضادة للسمية الخلوية الوراثية للمستخلص الخام لنبات القطف البحرى في نخاع عظام فئران التجارب

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تعتبر النباتات الطبية ضرورية لأنظمة الطب التقليدي لكونها غنية بالمركبات النشطة بيولوجيًا التي تدعم الوظائف الفسيولوجية والكيميائية الحيوية للكائنات الحية. ومن بين هذه التأثيرات، تبرز بشكل خاص الخصائص المضادة للسمية الخلوية الوراثية ، حيث توفر النباتات الطبية حماية ضد السمية الخلوية الوراثية للخلايا المعرضة للظروف البيئية غير المواتية. دراستنا الحالية هدفها تقييم الخصائص المضادة للسمية الخلوية الوراثية للخلايا للمستخلص الخام لنبات القطف البحرى عن طريق تقييم معدلات الإنقسام الميتوزى الشاذ في نخاع عظام الفئران. حيث أُعطيت فئران التجارب 5 ملجم/كجم من وزن الجسم من المستخلص الخام ثلاث مرات في الأسبوع لمدة أسبوعين قبل تلقي جرعة الميتوميسين سي. كما تلقت مجموعة منفصلة من الفئران جرعة الميتوميسين سي وحده كمجموعة تحكم إيجابية. أوضحت النتائج أن التعرض للميتوميسين سي أدى إلى زيادة معنوية فى معدل التشوهات الكروموسومية مقارنة بمجموعة التحكم غير المعاملة بالميتوميسين. في حين أن المعاملة قبل تلقي جرعة الميتوميسين سي بالمستخلص الخام لنبات القطف البحرى أدت إلى انخفاض معنوى في معدل التشوهات الكروموسومية التركيبية والعددية الناجمة عن الميتوميسين سي كما أدت إلى زيادة معنوية في قيمة دليل الميتوزى. وبشكل عام فإن نتائجنا تشير إلى أن المستخلص الخام لنبات القطف البحرى يمكن أن يكون مكملًا غذائيًا وقائيًا وعاملًا واعداً يقلل من السمية الخلوية الوراثية التي يمكن أن تنتج عن أدوية علاج السرطان. ومع ذلك، لا تزال هناك حاجة إلى مزيد من الدراسات لتأكيد هذه الفوائد المحتملة.

الكلمات المفتاحية : النباتات الطبية – الميتوميسين – السمية الخلوية الجينية