INTRODUCTION

Genotoxicity of chemotherapeutic drugs has been established in several experimental test systems *in vitro* and *in vivo* ***(Sanderson et al., 1996 and Zhong, et al., 2010).***

Many chemotherapy regimens have been shown to cause chromosomal damage in lymphocytes of patients ***(Sorsa et al., 1985 and Walitza, et al., 2007),*** and they are capable of inducing further malignant growth when given to patients at therapeutic dose level ***(IARC, 1990)***.

The main concern of second malignancies of the patients is related to the genotoxic effects of these chemotherapeutic agents.

One of these chemotherapeutic drugs is Mitomycin-C (MMC), one of the most important active chemotherapeutic agents, that has a potent anti-tumor activity against a wide range of human malignancies.

Mitomycin C is an antibiotic derived from *Streptomyces caespitosus* and is generally classified as a DNA alkylating agent. Cellular toxicity after MMC exposure can occur from MMC-induced insults such as the generation of free radicals or DNA monoadducts; however, the most significant effects are

due to the accumulation of covalent DNA interstrand cross-links ***(Danny et al., 2009)***.

The genotoxicity of MMC has been detected by using several methods. MMC has been shown to induce chromosomal breaks and sister chromatid exchanges in bone marrow cells of mice ***(Mahrous et al., 2002).***

MMC also cause many chromosomal damages in both cell types (bone marrow and spermatocytes) and such damages are dose- dependent ***(Noshy and Hassan 2003).***

Concerning germ cells, studies showed that MMC induced chromosomal aberrations ***(Nakagawa and Mori, 2003),*** Micronuclei and DNA damage ***(Hu et al 2005).***

Recently, great attention has been paid to the research of the effects of antioxidants, in particular free radical scavengers, on chemotherapeutic drugs that cause DNA damage ***(Zhang et al., 2010).***

It has been postulated that free radical-mediated reactions are responsible for a wide range of chemotherapy-induced side effects, and antioxidants are able to protect non-malignant cells against damage chemotherapeutic agents ***(Weijl et al., 1997).***

(Li et al., 2009) Inhibited chromosomal aberrations induced by Mitomycin-C by using micrometer powder of selenium-enriched green tea in mice spermatocytes.

Green tea decreases the chromosomal aberrations in bone marrow cells, sperm abnormalities and DNA damage ***(Zowail, et al., 2009).***

Also grape seed oil uses as the have chemopreventive activity in cellular models of cancer ***(kim et al.,2004).*** And also inhibit the side effects of MMC ***(Afzal, and Siddique, 2009).***

Both grape seed oil and green tea present in the same drug (Oxyplex) that is used in this study.