CHAPTER 1

Introduction

Many of the food products available in the countryside markets in districts of West Bengal state, India are probably contaminated with metanil yellow (MY), a non-permitted synthetic toxic dye. The amount of MY present in the food products is probably higher than the maximum permissible limit as prescribed by Prevention of Food Adulteration Act of India (PFA, 2008). The exposure of human beings to MY for a longer period of time might produce severe health hazards due to molecular interactions of MY with several biomolecules.

Different toxic effects of MY has been studied by the scientists since last few decades. It has been observed that the amine level of hypothalamus, striatum and brain stem have been significantly affected in rats after the oral administration of MY for a long time (Nagaraja and Desiraju, 1993). Oral administration of MY and orange II showed significant alteration in the level of different biochemical parameters in rats and also produced toxicity in liver (Ramachandani *et al.,* 1992, 1997; Singh and Khanna, 1988). It has been observed that the membrane of the mitochondria, microsome and nucleus have been effected after the administration of the blend of MY and orange II (Ramachandani *et al.,* 1992). In 2002, Gupta et al showed that MY and malachite green promotes the tumor formation during the hepatocarcinogenesis in experimental animals (Gupta *et al.,* 2002). It has been showed that MY and its metabolites p-aminodiphenyl amine has a binding affinity to the serum protein (Raza *et al.,* 1983). Bhunya and Patti showed that oral administration of MY was responsible for the chromosomal aberration in rats (Bhunya and Patti, 1988). It

has been observed that blend of MY and orange II showed mutagenic effect when applied in AHH- human lymphoblast cells (Rastogi *et al.,* 1991). MY has no toxic effect on hematological parameters but altered the cytoarchitectural structure of intestinal villi when applied on rats for a long period of time (Shankar *et al.,* 1992). It has been observed that MY produces toxic effect on fish (Mall and Kishore, 1995; Goel and Gupta, 1995). The glutathione level and lipid peroxidation has been increased in the liver and intestine of rats after the oral administration of MY for 7 days (Ramachandani *et al.,* 1997). It has been reported that oral administration of MY to male albino rats produces significant decrease in absolute and relative weight of testes, activities of lactate dehydrogenase and hyaluronidase (Singh, 1998), testicular damage in gamatogenic elements to arrest spermatogenesis in guinea pigs, rats and mice (Khanna and Das, 1991), degradation in seminiferous tubules, spermatocytes and vacuolation occurs in sertoli cells and also resulted in a remarkable decrease in food intake and body weight gain in both normal protein and low protein groups (Sarkar and Ghosh, 2012). MY also induces histopathological and structural damage at cellular and subcellular organization in stomach, intestine, liver, kidney and alteration in hematopoitic system in rats (Mehrotra *et al.,* 1974; Sarkar and Ghosh, 2010). It has also been reported that oral administration of blend of MY, sunset yellow and tartrazine produces serological changes in swiss albino rats (Saxena and Sharma, 2014).

The possible toxic effects of MY in animal models are still lacking. To examine the probable toxic effects of MY on female reproductive system in human beings explored chronically with MY, the effects of MY on female reproductive system functions have been studied in rat models.

To confirm the presence of MY in some food products as coloring agent, a few samples have been collected from some suspected food products sold in the local market and the amount of MY present in the food sample have been determined prior to experimentally examine the effects of MY on female reproductive system functions in rat models.

The results of the work done have been presented in chapter 4 of this thesis.

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