Preface

Reproductive health is exquisitely sensitive to environmental contaminants and toxicants. Evidence from animal experiments, wildlife studies, and reports of occupational and accidental human exposures indicate that both natural and chemical contaminants can adversely affect reproduction. Both public and scientific communities raised their voice against alarming levels of contaminants in the environment. In view of this, studies are warranted to explore the possible toxicities of environmental contaminants. Environmental contaminants that mimic or interfere with the natural ligand, thereby antagonizing the function of natural ligand comes under endocrine disruptors. Environmental contaminants of different origin act differently at dissimilar points including hypothalamo-pituitary-testicular axis thereby resulting in disorders like infertility and other reproductive malformations, which include retention of nipples, abnormal sexual behavior, birth defects, decreased spermatogenesis, reduced gonad weights. The mechanisms by which these toxicants affect reproductive health of individual are utmost important for scientific fraternity working in areas of reproductive toxicology and biology.

Environmental contaminants are not only chemicals that accidentally or deliberately enter the environment, often, but not always, as a result of human activities. These may include chemicals produced by natural contaminants like fungi e.g. Mycotoxins and chemicals that have been manufactured for industrial use. These contaminants enter easily into food chains, thereby causing deleterious effects. While a general assessment of the impact of these contaminants on public health is difficult to make, it can be argued that direct measures are in place to curb the impact of man-made contaminants. Obligatory approval for synthetic compounds entering the food chain, such as pesticides and preservatives include their toxicological assessment, and guidelines for the application. But these safeguards cannot be applied to naturally occurring toxins. In this case, only indirect measures like good manufacturing practices, soil treatment, the use of resistant varieties and fungicide application are the only way to curb these toxins entering into the food chains.
Natural toxins in food are plant secondary metabolites, bacterial toxins, phycotoxins and mycotoxins. Among them mycotoxins are secondary metabolites produced by fungi causing acute and chronic toxic effects to humans and animals. The most important fungal genera producing mycotoxins that are found in food products are *Aspergillus*, *Fusarium*, *Alternaria* and *Penicillium*. Aflatoxins are the most prevalent mycotoxins produced by *Aspergillus flavus* and *Aspergillus parasiticus*. In developing countries, more than 5 billion people are at risk of chronic exposure to naturally occurring aflatoxins (AfB1, AfB2, AfG1 and AfG2) through contaminated foods. Aflatoxin poisoning can produce recurrent serious health effects which include carcinogenesis, mutagenesis, growth retardation and immune suppression. Amongst aflatoxins, AfB1 is most potent and posing serious health problems to humans as well as animals.

Mammalian reproductive system presents multiple targets for chemical injury and various end points for detecting dysfunction. ‘Reproductive toxicity’ is defined as dysfunction in reproductive processes of organisms, induced by chemicals. The toxic effects of natural and man-made chemicals on the human reproductive system have become a major health concern. Chemicals which induce germ cell damage and sterility appear to be on the increase. A number of chemicals with adverse structures and functions have been shown to exert dramatic effects, especially on hormone synthesis and function.

From earlier studies it is clear that AfB1 is a potent reproductive toxicant. However, the mechanism by which it acts as reproductive toxicant is still not clear. Therefore, the present study is aimed to elucidate the mechanism behind AfB1 mediated reproductive toxicity. It is clear from previous studies that AfB1 acts as mutagen, growth retardant, carcinogen and immune-suppressant. It is well known that the developmental stage of embryo to neonate is very sensitive to endocrine disrupting chemicals and receptive to epigenetical changes. Keeping this in view, the present study also includes the effect of transplacental exposure to AfB1 on adult reproduction in rats. In the present study, developmental landmarks, tissue indices, sperm analysis, steroidogenic enzyme activities, reproductive hormone levels in serum, histology of testis, and fertility parameters are considered as reproductive end points.
The inferences drawn in the present study are based on gravimetric, histologic, biochemical and fertility parameters. The dissertation presents a humble effort by the researcher towards a better understanding of the effect of AfB1 for further studies on male reproductive health. The researcher assumes the responsibility for any deficiencies presented in the text, which could be due to oversight, and earnestly request condonation. Knowledge gained from this study will provide critical information about the toxicities of exposure to AfB1.