

1. INTRODUCTION

Drug teratogenicity, has been demonstrated experimentally for more than 50 years. However, the clinical implications of the experimental results have been only fully recognized after the discovery of the thalidomide induces embryopathies. The importance of the roles played by drugs in the etiology of birth defect cannot be summarized and this is unlike to genetic or environmental causes. The latter include radiation, viruses, dietary deficiencies, infection and metabolic imbalance.

There are a large number of teratogenic drugs namely antiepileptic, antibiotics, hormones, vitamins, sulfonamides, hypoglycemic drugs, hyperglycemic agents, neuro-drugs, antihistamines and antinauseant drugs, anticoagulants and antitumor drugs.

It is obvious from the literature that the effect of the antiepileptic **phenytoin (PHT)** has been studied on the adult experimental animals. The work done on the newborn, however, is scarce and is essentially concerned with the effect of this drug on the ototoxicity and mortality of the newborn. No other work, as far as known, has been carried out on the effect of **PHT** on mice offspring (mutagenicity).

It is well known that **PHT** diffuses through the placenta with a high level to the fetuses. Therefore, it has been proposed, in the present work to elucidate the effect of this drug on albino mice; *Mus musculus* embryo (newborn). The present investigation comprised; extracting the total DNA genome newborn (normal and drug treated groups).

Digesting the DNA genome by using a specific restriction enzymes, detection of certain gene of normal and drug treated groups by using the polymerase chain reaction; digesting the chosen gene by using several restriction endonuclease enzymes and making, molecular newborn toxicity genotypes according to their total DNA and chosen restriction fragment patterns.