1. INTRODUCTION

The cotton leafworm, *Spodoptera littoralis* (Boisd.) was first encountered in Egypt in the field and identified in 1865; about 45 years after cotton cultivation had been introduced in Egypt. Its attack on plants was been observed in 1883 and since then this species has established itself as an injurious insect of great economic importance. It now stands first and foremost among cotton pests in Egypt. In recent years its ravages have been steadily increasing and the losses reflected annually on the cotton crop are sometimes incalculable. Moreover, this insect pest is highly polyphagous infesting about 73 host plants in Egypt including important field crops such as maize, clover and vegetables, and various fruit and ornamental trees. Thus it is a destructive pest throughout the year in Egypt **(Abd El-Aal, 2003).**

Pesticides are toxic chemical compounds designed to control pests, causal organisms of plant disease, weeds and other living organisms that reduce the quantity and quality of crop yields. However their adverse effects have followed in the wake of intensive pesticide use. Pesticides are commonly recorded in water supplies and food chain. The increasing rate of pest resistance to pesticides, loss of biodiversity and particularly human poisoning in developing countries are common **(Mastumura, 1985).**

It was recorded that the Egyptian markets are using large amounts of pesticides between 30-60 thousands metric tons/year.

More than 50% of this quantity ends up in the soil by direct or indirect ways, thus increasing the problem of pollution **(Amr et. al., 1990).**

For many years, (*B. thuringiensis var kurstaki*) was available only for control of lepidoptera, using a highly potent strain. Once it has been solubilised in the insect gut, the protoxin is cleaved by a gut protease to produce an active toxin. This toxin is termed delta-endotoxin. It binds to the midgut epithelial cells, creating pores in the cell membranes and leading to equilibration of ions. As a result, the gut is rapidly immobilised, the epithelial cells lyse, the larva stops feeding, and the gut pH is lowered by equilibration with the blood pH. This lower pH enables the bacterial spores to germinate, and the bacterium can then invade the host, causing a lethal septicaemia. **(Knowles, 1994)**.

Mammalian safety studies were carried out with *Bacillus thuringiensis var kurstaki* orally administered to rats. The clearance and distribution of B. thuringiensis were evaluated. Rats were given a single dose of suspension spores. The bacteria spore culture from faeces, urine, gut content and organ tissues were evaluated at various times. The main way of clearance of B. thuringiensis in rats is through the digestive system. The results confirmed the safety of *Bacillus thuringiensis var kurstaki* to rats **(Tsai et al, 1995)**.

The hazardous effects of benzoylphenylurea insecticides on mammalian tissues are still under investigation and works on it centered mainly on controlling insect production. Administration

of benzoyl-phenylurea resulted in altered enzyme activities of rat liver, renal damage, and reproductive disorders to experimental animals **(Karim, 1998).**

Benzoylphenylurea pesticides act as insect growth regulators or chitin synthesis inhibitors. They act on the larval stages of most insects and inhibit or block the synthesis of chitin. Chitin is the compound that causes the insect's outer covering to become hard, thus interfering with the formation of the insect's shell. The shell is not able to support the insect or withstand the rigors of molting. Typical effects on larvae are the rupture of malformed cuticle or death by starvation. Benzoylphenylurea pesticides are particularly effective when they are applied just prior to insect molting **(Farag. 2001).**

El- Hamaky et. Al. (1990) mentioned that Chlorpyrifos, as organophosphate gave complete control of the pests irrespective of the age of the eggs to which it was applied. The persistence of residues of chlorpyrifos is moderately and had great residual toxicity to larvae.

Organophosphate pesticides work by interfering with the activity of the cholinesterase enzyme, which is necessary for proper nerve function. Without this enzyme, impulses continue to pass down the nerve fiber disrupting the nervous system and ultimately resulting in death by respiratory failure, but do not accumulate in the tissues of humans or animals. Some of the more toxic organophosphate insecticides can present a high risk of irreversible Organophosphate poisoning in humans, from

excessive exposure. This risk is highest to pesticide applicators and non-target animals.

The metabolites of Chlorpyrifos have been widely reported in human tissue. Chlorpyrifos exposure can occur via inhalation of residual air concentrations, dermal or oral exposure from residues on floors and carpets, children toys, food, and dust. The exposure of young rats to chlorpyrifos impairs early nervous system development. Administration of sublethal doses of chlorpyrifos resulted in altered enzyme activities of rat liver, renal damage, and reproductive disorders to experimental animals. After finding that chlorpyrifos is an exposure risk especially to children, **(Lemus and Abd El- Ghany, 2000).**

The present study aims to evaluate the toxicological, biological and biochemical effects of Bactericide (*Bacillus thuringiensis kurstaki)*, Insect growth regulators (Flufenoxuron & Hexaflumuron) and Organophosphorus compounds (Chlorpyrifos) on cotton leaf warm *Spodoptera Littoralis.* Also, the present study aims to evaluate *in vivo* effect of Chlorpyrifos and Flufenoxuron on the biochemical parameters, toxicity, and histopathological changes in tissue specimens of different organs of albino rats.