



## Biochemical, Haematology Changes by Imidacloprid

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### ABSTRACT

*Indiscriminate usage of pesticides in agriculture is leading to contamination of environment and natural resources, and thereby producing an adverse impact on animal and human health. Imidacloprid is a neonicotinoid insecticide and classified under toxicity class II/III agents by United States Environmental Protection Agency (USEPA, 1994). It is an extensively used for crop protection in the world wide from the last decade due to its low soil persistence and high insecticidal activity at low application rate (Chao and Casida, 1997). Recently imidacloprid has raised concern because of its ability to cause egg shell thinning, reduced egg production and hatching time, which are considered as signs of possible endocrine disrupters (Matsuda et al., 2001). Experimental administration of imidacloprid through various routes in animals produces marked alterations in serum biochemical and haematological parameters.*

**Keywords :** imidacloprid, haematology, serumbiochemistry

### INTRODUCTION

Imidacloprid, a neonicotinoid insecticide, is extensively used in agriculture for control of the sucking insects and coleopteran beetles (Cox, 2001) and also used as foliar treatment for soil and for seed dressing (Felsot, 2001). In Veterinary Medicine, it is used as flea control agent on dogs and cats (Hutchinson et al., 2001). It is one of the fastest sold insecticide across the world because of its high selective toxicity in insects. It acts on nervous system by blocking post synaptic acetylcholine receptors (Tomizawa et al., 2005). Its selective toxicity results from its high affinity to insect's nicotinic acetylcholine receptors compared to mammals (Tomizawa and Casida, 2003). A case of acute poisoning was reported in human following ingestion of a pesticide formulation containing 10% imidacloprid (Wu et al., 2001) and two fatal intoxication cases have been reported recently (Proenca et al., 2005). Since imidacloprid is now being considered as a replacement for other existing pesticides, therefore the relative risk and benefits of this insecticide must be compared to the existing pesticides. In this article we had tried to review few important toxic effects caused by imidacloprid.

### Effect of imidacloprid on biochemical Parameters

In a chronic toxicity study, Wistar rats were administered with imidacloprid at different concentrations (100, 300, 900 and 1800 ppm) for 2 years. There was no inhibition of cholinesterase in brain, plasma or erythrocytes at any level. At 1800 ppm serum alkaline phosphatase, creatinine kinase, aspartate amino transferase levels were elevated with a reduction in plasma cholesterol (USEPA 1998).

In a sub chronic oral toxicity study, Wistar rats of both sexes were provided with imidacloprid at concentrations of 150, 600 and 2400 ppm for a period of 13 weeks. The biochemical changes included elevated levels of alanine amino transferase, serum alkaline phosphatase with slight increase in blood clotting time (USEPA 1998).

Barinderjit et al. (2006) reported that oral administration of imidacloprid at 1 mg/kg b. wt for 21 days in cow calves resulted in elevation of plasma ALT, AKP, without any significant effect on plasma AST, acid phosphatase, cholinesterase enzymes, serum total protein, blood urea nitrogen (BUN), plasma creati-

nine, blood glucose and plasma cholesterol levels.

Siddiqui et al. (2007) noticed that birds fed with imidacloprid orally at 1-2 mg/kg b. wt daily for 28 days showed a non significant decline in total protein and a significant reduction in total albumins which is suggestive of immunosuppression.

Bhardwaj et al. (2010) observed that oral administration of imidacloprid in female rats at the rate of 5, 10 and 20 mg/kg b. wt for 90 days resulted in elevation of ALT, AST, glucose, BUN and decreased acetyl choline esterase in serum and brain.

Kammon et al. (2010) reported that oral administration of imidacloprid at the rate of 139 mg/kg b. wt through oral gavage in chicken resulted increased levels of AKP, ALT, AST and plasma glucose.

Sridhar (2010) recorded a natural toxicity of imidacloprid in buffaloes and observed slight increase in AST and ALT levels whereas there was no change in serum creatinine and BUN.

Balani et al. (2011) evaluated the toxic effects of oral administration of imidacloprid in male white leg horn chicken at 1.25, 1.67 and 2.5mg/kg b. wt for 28 days and noticed an increase in ALT, no change in AST, serum total protein, total globulins, total albumin and serum creatinine levels.

Mohany et al. (2012) reported that oral administration of imidacloprid at 0.21mg/kg b. wt for 28 days in male albino rats resulted in elevation of AST, ALT, ALP and MDA levels.

Soujanya et al. (2013) reported that oral administration of imidacloprid at the rate of 80 mg/kg b. wt for 28 days in male albino rats produced a significant increase in alanine transaminase and aspartate transaminase, decrease in total protein levels whereas co-treatment with vitamin C significantly reversed the imidacloprid induced changes.

Soujanya et al. (2013) reported that oral administration of imidacloprid at the rate of 80 mg/kg b. wt for 28 days in male rats resulted in nephrotoxicity which was evident from significantly increased serum creatinine levels in imidacloprid treated rats whereas co-treatment with vitamin C brought mild to moder-

ate improvement in creatinine levels.

#### Effect of imidacloprid on hematological parameters

Ammar et al., (2003) reported a significant increase in the total leukocytes count due to administration of imidacloprid at 0.1 and 0.25 LD<sub>50</sub> dose in male albino rats.

Bhardwaj et al. (2010) documented an insignificant change in hematological parameters (RBC, WBC, Hb, HCT, MCV and DLC) in female rats by oral administration of imidacloprid at the rate of 5, 10, 20 mg/kg b. wt for 90 days in their experiment.

Sridhar (2010) also reported an insignificant change in hematological parameters (Hb, PCV, TEC, TLC and DLC) in natural cases of imidacloprid toxicity in buffaloes.

Balani et al. (2011) conducted an experiment in male white leg horn chicks for 28 days by administering imidacloprid at the rate of 1.25, 1.67, 2.5 mg/kg b. wt and observed an insignificant change in haematological parameters (Hb, PCV

and TEC) except decreased TLC counts at 2.5 mg/kg body weight.

Mohany et al. (2012) reported that oral administration of imidacloprid at 0.21 mg/kg b. wt for 28 days in male albino rats produced a significant increase in total leucocytes count.

Soujanya et al. (2012) evaluated that oral administration of imidacloprid at the rate of 80 mg/kg b. wt for 28 days in male albino rats produced a significant decrease in TEC, Hb, PCV, MCV, MCH and MCHC and a significant increase in TLC. Whereas co-administration of vitamin-C brought mild to moderate improvement in all these parameters.

#### CONCLUSION

Imidacloprid exposure leads to marked alterations in serum biochemistry and haematological parameters. So imidacloprid was found to be a potent toxic agent. To prevent adverse effects of imidacloprid, it should be used in limited dose for crop protection and as insecticides in dogs and cats.

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