Toxicological Effects of Imidacloprid WDG 70% on Blood Properties and AChRs of Mice

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ABSTRACT

Imidacloprid (IMI), a widely used neonicotinoid insecticide, has raised concerns due to its potential neurotoxic effects, particularly during early developmental stages. This study investigated the impact of postnatal IMI exposure on hematological parameters and acetylcholinesterase (AChE) activity in mice. Mice were exposed to various concentrations of IMI (0, 10, 20, and 30 mg/kg/day) for 21 days. The results revealed significant alterations in blood cell counts, including a decrease in red blood cells, hemoglobin, and packed cell volume, as well as an increase in white blood cells and neutrophils. Additionally, IMI exposure led to reduced levels of basophils and lymphocytes. Maternal exposure to IMI resulted in adverse reproductive outcomes, such as miscarriages. Further, the study observed significant changes in AChE activity, suggesting potential neurotoxic effects. These findings highlight the detrimental effects of IMI on the hematological system and neuronal function in mice, emphasizing the need for further research to assess the long-term health implications of IMI exposure.

Keywords: Imidacloprid, neurotoxicity, hematological parameters, acetylcholinesterase, mice, environmental pollution.

INTRODUCTION

Imidacloprid (IMI) is a widely used neonicotinoid insecticide, like use in control termites, introduced in the early 1990s, evaluated for its effectiveness against various insect pests in agriculture and beyond (Badgujar et al., 2013). Known for its selective binding to nicotinic acetylcholine receptors (nAChRs) in insects, (Jones, A. K., et al. (2010). IMI stops normal neural functions, ultimately leading to the insect's death (Anderson et al., 2015).

This specificity for insect nAChRs contributes to its relatively low neurotoxicity in mammals, a feature that has made it the most extensively used insecticide worldwide, with global applications reaching approximately 20,000 tons annually by 2010 (Rodríguez-Cabo et al., 2016; Simon-Delso et al., 2015). Several tests concerning toxicity and genotoxic capacity of IMI have been conducted (Sponchiado, G., et al, 2016).

IMI is particularly effective against pests like aphids, whiteflies, and thrips, commonly found in crops such as vegetables, fruits, cotton, and rice. It is also used as a seed treatment, enhancing its appeal in integrated pest management practices (Matsuda et al., 2001, Kiss, A., et al (1991). Even with benefits, the insecticide is linked to oxidative stress, marked by free radical generation, antioxidant system disruption, and lipid peroxidation, leading to tissue damage (El-Gendy et al., 2010; Jin et al, 2011). Studies have shown that IMI main objectives the liver and kidneys, organs highly susceptible to xenobiotics' toxic effects due to their role in metabolism and detoxification (Vohra et al., 2014, Saqer, B. T., et al (2019).

Given IMI's increasing application in agriculture and the potential health risks from consuming pesticide residues in food, investigating its subchronic effects on health is crucial, (Thompson, D. A., et al(2020).This study aims to examine IMI's biochemical impact, potential oxidative stress induction, and histopathological changes in laboratory rats, providing insights into its broader environmental and consumer safety implications,(the environmental risks) (Sardar, A., et al(2023).; Tonietto, B. D., et al(2022), Goulson, D., (2013) .

The effect of imidacloprid (IMI) and its damage to the liver and kidneys: -

Imidacloprid can cause oxidative stress, leading to cell damage and inflammation in the liver, (Duzguner, V.,et al(2012).

Imidacloprid can cause kidney damage, leading to impaired kidney function, (Arfat, Y., et al(2014).

Similar to its effects on the liver, imidacloprid can cause oxidative stress in the kidney, (Taha, M. A., et al(2021).

MATERIALS AND METHODS

Chemicals:

Imidacloprid pesticide (IMI) formulated as a Water Dispersible Granule (WDG) at a 70% concentration.

A primary stock solution of IMI was prepared at a concentration of 25 ppm by dissolving the pesticide in distilled water.

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Four treatment groups were established, with exposure concentrations of 0, 10, 20, and 30 mg/kg/day.

Animals:

Swiss albino mice were used for the study.

Forty adult female mice (30-45 days old) and 20 adult male mice (45 days old) were randomly divided into four groups of 10 females and 5 males each.

Animals were housed under controlled conditions with a 12-hour light/dark cycle, a temperature of 18-20°C, and a relative humidity of 50-55%.

Standard commercial pellet diet and tap water were provided ad libitum.

Experimental Design:

Part 1: Postnatal Exposure

Breeding: Female mice were mated, and pregnant females were selected.

Treatment: Pregnant females were orally gavaged with the respective IMI concentrations from gestational day 6 to 15.

Postnatal Assessment: Postnatal mice were assessed for:

Acetylcholinesterase (AChE) activity

Complete blood count (CBC)

Part 2: Adult Exposure

Treatment: Adult female mice were orally gavaged with the respective IMI concentrations.

Assessment: Adult mice were assessed for:

Acetylcholinesterase (AChE) activity

Complete blood count (CBC)

Objectives:

Investigate the effects of postnatal IMI exposure on AChE activity and hematological parameters in mice.

Determine the effects of adult IMI exposure on AChE activity and hematological parameters in mice.

RESULTS AND DISCUSSION

Fig.1. Shows the following: -- Increase in mortality and abortion: There were cases of mortality and abortion associated with repeated exposure during pregnancy to concentrations of 30 mg/kg/day of imidacloprid, and at rates of 10 and 20 mg/kg/day, compared to the control group, the percentage of abortions increased significantly in pregnant mice, indicating a direct toxic effect on the fetus and mother.

Fig. 1. Effect of different doses of imidacloprid (IMI) pesticide and the negative effects that appear on pregnant female mice

Emergence of effects on the nervous system: The researchers observed the appearance of symptoms of neurotoxicity in the treated females, such as tremors and convulsions, which began to appear early during pregnancy. On the eighth day of pregnancy, during (the third day of treatment).

Weight loss: Treatment with imidacloprid led to a significant decrease in the weight of pregnant females that took doses of 10, 20 and 30 mg/kg/day by 7, 15 and 20% compared to the control group, and this decrease increased with increasing dose.

Interpretation of results:

Mechanism of toxicity: Imidacloprid probably caused these adverse effects by affecting the central nervous system, leading to disturbances in neurotransmission and affecting the functions of vital organs. (Bais, S. S. (2019). Abd-Elhakim, Y. M., et al (2018).

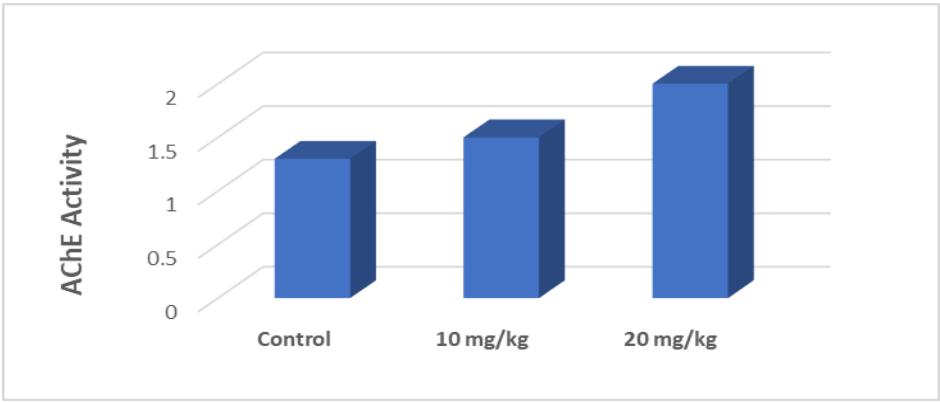

Fig. 2. Effect of Imidacloprid on *Acetylcholinesterase (AChE)* Activity in Mice

Impact on fertility: The increase in miscarriages indicates a negative effect of imidacloprid on fertility, which may be due to a direct effect on the fetus or the uterus. Marutirao, S. A. (2019).

Impact on nutrition: Weight loss may be due to anorexia or disturbances in nutrient absorption due to the toxic effect of imidacloprid. Agatz, A., et al (2013).

Therefore, there is a risk of exposure to imidacloprid, as Fig. 1 confirms. During pregnancy, it can lead to serious adverse effects on the health of the mother and fetus in mice.

Figure 2 illustrates the effect of Imidacloprid (IMI) on acetylcholinesterase (AChE) activity in mice. As the IMI dose increased, AChE activity significantly elevated. The group exposed to 20 mg/kg/day IMI exhibited a 54% increase in AChE activity compared to the control group.

The observed increase in AChE activity in response to IMI exposure is indicative of its potential neurotoxic effects, (Hassanen, E. I., et al (2023), Guerra, L. J., et al (2021). AChE is a crucial enzyme involved in the degradation of acetylcholine, a neurotransmitter essential for nerve impulse transmission, (Thapa, S., et al (2017), Colovic, M. B., et al (2013). Inhibition of AChE leads to the accumulation of acetylcholine, resulting in excessive neural stimulation and subsequent neurotoxicity, (Pope, C., et al (2005), Milatovic, D., et al (2006).

The mechanism underlying the increased AChE activity in response to IMI exposure is not fully understood. However, it is possible that IMI may induce a compensatory response in the nervous system, leading to increased AChE synthesis or decreased AChE degradation. Alternatively, IMI may directly interact with AChE, altering its structure and function, (Yu, R., et al (2011), Tomizawa, M., et al (1999).

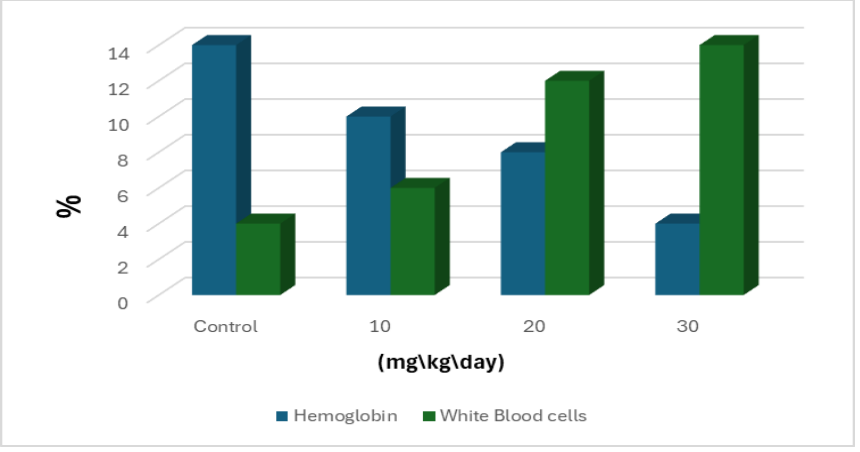

Fig. 3. Impact of Imidacloprid on Hematological Parameters in Mice

Table 1. Impact of Imidacloprid on Maternal Reproductive Parameters in Mice

		Emeduch mg/kg/day			
		Control	**10**	**20**	**30**
Number of mated females		-	10	10	10
Number of dams		9	8	6	0
Body weights (g)	0	25.63±0.1	25.5±0.1	26.00±0.4	26.2±0.3
	6	30.28±0.2	29.77±0.3	31.2±0.6	30.28±0.1
	12	35.46±0.4	26.1±0.2	25.8±0.8	Death & abortions
	15	42.1±0.7	26.3±0.2	25.6±0.7	
	12	45.1±0.4	26.2±0.3	24.1±0.8	

Figure 3 illustrates the effects of imidacloprid exposure on hematological parameters in mice. A significant decrease in hemoglobin levels was observed in all treatment groups compared to the control. Conversely, white blood cell (WBCs) counts were significantly elevated in the 20 and 30 mg/kg/day groups.

The level of hemoglobin decreased in all treatment groups compared to the control. The decrease was more significant in the higher dose groups (20 and 30 mg/kg/day).

The observed decrease in hemoglobin levels suggests that imidacloprid may induce anemia, possibly through mechanisms such as hemolysis or impaired erythropoiesis, (Qadir, S., et al (2014) pointed to Imidacloprid-exposed fish indicating severe anemic condition, (Saqer, B. T., et al (2019) which showed number of red blood cells, hemoglobin levels, and packed cell volume (PCV).

The level of neutrophils decreased in all treatment groups compared to the control. The decrease was most significant in the highest dose group (30 mg/kg/day).

The increase in white blood cell counts, particularly neutrophils, is indicative of an inflammatory response. This may be due to direct toxic effects of imidacloprid on immune cells or secondary to tissue damage caused by oxidative stress, (Mohany, M., et al (2011) he pointed out in his research about, neutrophils and their ability to reach inflammatory sites. (Saqer, B. T., et al (2019), Neutrophils (Neu) were increased in blood of mice given IMI indicative of liver tissue damage.

Table 1. -- show number of Females: Control Group, out of 10 mated females, 9 successfully gave birth, indicating a high reproductive success rate of 90%.

10 mg/kg Group, 8 out of 10 mated females were able to give birth, a success rate of 80%.

20 mg/kg Group, only 6 out of 10 mated females gave birth, lowering the success rate to 60%.

30 mg/kg Group, no successful births were recorded as all pregnancies resulted in death or abortion. This 0% success rate at the highest dose suggests significant reproductive toxicity of IMI at 30 mg/kg/day.

These outcomes suggest a dose-dependent decline in reproductive success, with a dramatic impact at the highest dosage.

-- Maternal Body Weights:

Recorded at four time points (0, 6, 12, and 15 days), to show trends in normal pregnancy progression and potential effects of IMI exposure.

Before IMI, all groups, including the control group, showed similar weights, around 25-26 grams. This proves that there were no significant differences in body weight before IMI.

In day 6, control mice show an increase in weight, reaching 30.28 ± 0.2 g, which is typical as pregnancy advances.

The 10 mg/kg group has a comparable weight of 29.77 ± 0.3 g, suggesting minimal effects at this dosage. In contrast, the 20 mg/kg group shows a notable increase to 31.2 ± 0.6 g, indicating a slight weight gain, but variability might suggest a subtle physiological effect at this dosage. The 30 mg/kg group mirrors the control weight at 30.28 ± 0.1 g, but subsequent data suggest serious adverse effects with this dosage.

In day12, control mice continue to gain weight, reaching 35.46 ± 0.4 g, reflecting normal pregnancy progression.

The 10 mg/kg group shows a marked reduction in weight to 26.1 ± 0.2 g, indicating potential stress or physiological disruption.

The 20 mg/kg group shows further decline to 25.8 ± 0.8 g, and variability is increased, suggesting a greater adverse effect.

The 30 mg/kg group experiences high rates of death and abortion.

In day 15, control mice exhibit continued weight gain, reaching 42.1 ± 0.7 g.

The 10 mg/kg group shows minimal further weight gain 26.3 ± 0.2 g, indicating compromised reproductive development.

The 20 mg/kg group shows a further decline in weight (25.6 ± 0.7 g), continuing the negative trend.

The 30 mg/kg group had no viable pregnancies.

In day 12 (Final), control weights peak at 45.1 ± 0.4 g, while other groups exhibit a marked reduction in body weight, especially at 20 mg/kg (24.1 ± 0.8 g), further highlighting dose-dependent toxicity.

The findings of this study demonstrate the significant adverse effects of imidacloprid on reproductive health, neurotoxicity, and hematological parameters in mice.

In the present study, the effects of (IMI) formulated (WDG) 70% on ACHE and blood properties were investigated. The observed toxic symptoms in the pregnant female mice exposed to 10, 20, and 30 mg/kg/day (IMI) by gavage were muscular tumors and convulsions, these symptoms were referred to the activity of ache enzyme, (Griffith, R. W.,et al(1978),convulsions are encountered in acute toxicity. (Kamrin, M. A. (1997), (Kapoor, U., et al(2011).

Maternal choline esterase activity was markedly increased in the treated group by 20 mg/kg/day. Compared to the control but no significant change in 10mg/kg/day compared to the control.

The level of both Hemoglobin and Neutrophils was decreased by 10, 20mg/kg/day compared to the control.

CONCLUSION

The present study illustrates that Imidacloprid (IMI) has adverse effects on both blood properties and acetylcholine receptors (AChRs) in mice.

Also, the exposure to IMI resulted in severe maternal toxicity such as reduced body weight, increased cholinesterase activity, abortions, and fetal death, as well as, reduced red blood cells, hemoglobin levels, and neutrophils but has a tendency to elevate the level of white blood cells.

AChR disruption: It is supposed that the observed changes of cholinergic toxicity symptoms and cholinesterase activity could be due to IMI action on AChRs.

In conclusion reveals the dangers of exposure to imidacloprid, especially when taken during development. More research is required to come up with effective long-term outcome correlates of IMI exposure as well as to design preventative measures against it.

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الملخص العربي

تأثير سمية إيميداكلوبريد(WDG 70%) على خصائص الدم ومستقبلات الأسيتايل كولين في الفئران

محمد عبدالمنعم خفاجى

يعتبر إيميداكلوبريد مبيدًا حشريًا نيكوتينيًا واسع الاستخدام، وقد أثار قلقًا بسبب آثاره العصبية السامة المحتملة، خاصة خلال مراحل التطور المبكرة. درست هذه الدراسة تأثير التعرض لإيميداكلوبريد بعد الولادة على المعلمات الدموية ونشاط أستيل كولين إستراز (AChE) في الفئران. تم تعريض الفئران لتركيزات مختلفة من إيميداكلوبريد (٠، ١٠، ٢٠، و ٣٠ ملغم/كجم/يوم) لمدة ٢١ يومًا. كشفت النتائج عن تغيرات كبيرة في تعداد خلايا الدم، بما في ذلك انخفاض في خلايا الدم الحمراء والهيموجلوبين وحجم الخلايا المعبأة، بالإضافة إلى زيادة في خلايا الدم البيضاء والنيوتروفيلات. بالإضافة إلى ذلك، أدى التعرض لإيميداكلوبريد إلى انخفاض مستويات الخلايا البدينة والليمفاويات. أدى تعرض الأم للمادة لإيميداكلوبريد إلى نتائج سلبية على التكاثر، مثل الإجهاض. علاوة على ذلك، لوحظت تغيرات كبيرة في نشاط AChE ، مما يشير إلى آثار عصبية سامة محتملة. تسلط هذه النتائج الضوء على الآثار الضارة لإيميداكلوبريد على الجهاز الدموي والوظيفة العصبية في الفئران، مما يؤكد الحاجة إلى مزيد من البحث لتقييم الآثار الصحية طويلة المدى للتعرض لإيميداكلوبريد.

الكلمات المفتاحية: إيميداكلوبريد، سمية عصبية، معلمات دموية، أستيل كولين إستراز، فئران، تلوث بيئي.