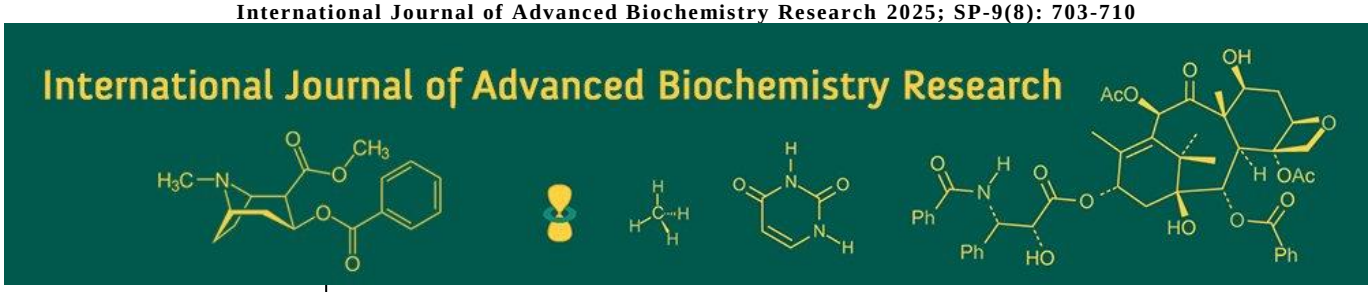

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Pesticide induced histopathological alterations in silkworm, *Bombyx mori* L.

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Abstract
The silkworm, *Bombyx mori* L., is an economically important insect serving as primary producer of silk and supporting the livelihoods of millions involved in sericulture. However, the extensive use of pesticides in agricultural fields often exposes silkworms to harmful residues through contaminated mulberry leaves, air or soil. Such exposure can cause significant physiological, biochemical and structural damage in the silkworm, ultimately affecting silk production and quality. Among the adverse effects, histopathological changes in vital tissues provide critical insights into the toxic impact of insecticides at the cellular and tissue level. This review synthesizes current knowledge on the histopathological alterations in silkworm tissues such as midgut, silk glands and gonadal exposure to different classes of pesticides. The mechanisms underlying tissue damage, including oxidative stress, enzyme inhibition and cellular necrosis are discussed. Understanding these effects is essential for developing sustainable pest management strategies that minimize risks to silkworm health and sericulture productivity.

Keywords: Silkworm, sericulture, pesticides, histopathology, tissue damage

Introduction
Silkworm, *Bombyx mori* L., has been domesticated for silk production for thousands of years and forms backbone of sericulture- a vital agro-based industry in many countries, especially in Asia (Yu *et al*., 2015) [24]. The success of sericulture largely depends on the growth and development of silkworm, which feed exclusively on mulberry leaves (Goldsmith *et al.*, 2002) [5] (Shruthi *et al*., 2024) [17]. However, the widespread and often indiscriminate use of pesticides in agriculture to control pests poses a serious threat to silkworm rearing (Xu *et al*., 2022) [22]. Residues of these chemicals on mulberry leaves or in the environment can inadvertently enter the silkworm body and cause detrimental effects (Zhang *et al*., 2018) [25]. Depending on their chemical nature, mode of action and dosage pesticides can disrupt the normal physiology and metabolism of silkworms leading to reduced growth, poor cocoon quality and increased mortality (Schmutterer, 1990) [15]; Bhosale & Kallapur, 1988) [1]. Among the various parameters used to assess insecticide toxicity, histopathological examination of silkworm tissues offers a direct and sensitive method to detect cellular and tissue-level damage caused by these chemicals (Muhammad *et al.*, 2025) [12]. Key organs such as the fat body, midgut, silk gland, malpighian tubules and gonads are particularly susceptible to pesticide-induced injuries, which can impair vital functions like digestion, detoxification, silk synthesis and reproduction.
This review aims to consolidate the available literature on the effects of insecticides on the histopathology of silkworms. It highlights the nature of tissue damage observed in different organs, explores possible mechanisms underlying these changes and discusses their implications for sericulture sustainability. Such an understanding is crucial for guiding the development of eco-friendly pest management practices that protect both crops and silkworm health.

1. Types of Pesticides Used in Sericulture Areas
Pesticides are substances designed to manage or eliminate organisms that threaten agriculture, food supplies, human health or the environment.Pesticides includes insecticides,

nematicides, fungicides, weedicides, rodenticides that deter, incapacitates, kill or discourage the pest. They are very important and became an integral part of modern agricultural practices and they protect crops from pests, increase agricultural productivity and contribute to maintaining public health standards.

2.1 Pesticides are broadly classified into the following categories

a. Organochlorines (OCs)
- **Examples:** DDT (dichlorodiphenyltrichloroethane), endosulfan, aldrin, dieldrin.
- **Characteristics:** Highly persistent in the environment, lipophilic (accumulate in fat), and long half-lives.
- **Mechanism:** Disrupts nerve impulse transmission by affecting sodium-potassium channels in nerve cells.

b. Organophosphates (OPs)
- **Examples:** Malathion, chlorpyrifos, dimethoate, monocrotophos.
- **Characteristics:** Less persistent than organochlorines but highly toxic to non-target organisms.
- **Mechanism:** Inhibit acetylcholinesterase (AChE), leading to accumulation of acetylcholine and disruption of neural transmission.

c. Carbamates
- **Examples:** Carbaryl, carbofuran, methomyl.
- **Characteristics:** Similar to OPs but with shorter persistence.
- **Mechanism:** Reversible inhibitors of AChE causing neurotoxicity.

d. Synthetic Pyrethroids
- **Examples:** Cypermethrin, deltamethrin, fenvalerate, permethrin.
- **Characteristics:** Synthetic analogues of natural pyrethrins; highly potent and fast-acting.
- **Mechanism:** Affect sodium channels in nerve membranes, causing prolonged depolarization.

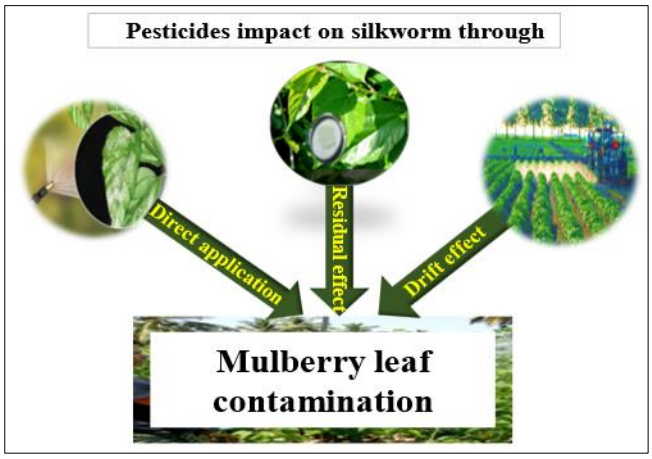
e. Other Classes
- **Neonicotinoids (e.g., imidacloprid):** Act on nicotinic acetylcholine receptors; systemic and persistent.
- **Insect growth regulators (IGRs) (e.g., diflubenzuron):** Disrupt insect development and moulting.

2.2 Pesticide and silkworm, *Bombyx mori* L. – the relevance

Silkworm, *Bombyx mori* L. is a monophagous insect which, solely depends on mulberry for its growth and development and reared for its silk production. It synthesis 70 % of total silk from proteins derived from mulberry leaves. The cocoon productivity and profitability is mainly determined by the quality and quantity of mulberry leaves. This indicates the importance of maintaining quality leaf to obtain high silk production. However, leaf quality is affected by several biotic and abiotic factors, of which pests and diseases play a major role. Therefore, it is rather important to protect mulberry crop from pests and diseases for which several chemicals are being used in the field (Bizhannia *et al*., 2007) [2].

2.3 Use of Pesticides in Agriculture and Possible Exposure of Silkworms

To protect food crops from insect pests and diseases, a wide variety of pesticides are routinely sprayed on fields. Unfortunately, the close proximity of mulberry gardens to these crop fields results in unintended exposure of silkworms to pesticide residues.
- Mulberry leaves harvested from fields adjacent to treated crops often carry spray drift or residual deposits of insecticides.
- Wind and water can transport insecticides from treated fields to mulberry gardens.
- Improper storage of insecticides, accidental spills and equipment contamination can also lead to residues on mulberry foliage.
- Even low levels of these residues, which may be insufficient to kill pests, can harm silkworms due to their extreme sensitivity to chemicals. Chronic or repeated exposure may result in sub-lethal effects, including developmental delays, impaired silk production, and histopathological damage (Li *et al*., 2025) [10].



There are different ways through which the pesticides can contaminate mulberry leaves, which could through direct application, drift effect or residual effect. However, in either of the ways the mulberry leaf gets contaminated with pesticides (Stanely *et al*., 2016)[19]. When pesticides contaminated mulberry leaves fed to silkworms cause disturbance in the normal metabolic activity of the larvae, such as growth and reproduction, cocoon quality, fecundity and hatching. Disruption of key biological functions such as feeding behaviour, metabolism, reproduction and immune responses *etc*. Therefore, Understanding the histopathological impact of pesticides on silkworms is vital for sustainable sericulture practices and safeguarding the economic and ecological balance of silk production systems (Cheng *et al*., 2019) [4].

2.4 Routes of Exposure

Silkworms can be exposed to Pesticides through several routes, including:

a. Ingestion
- The most common route
- Larvae feed on mulberry leaves contaminated with insecticide residues
- Contaminated leaves can carry significant amounts of both surface residues and systemic insecticides absorbed by the plant

b. Contact
- Direct contact with insecticide spray drift settling on silkworm bodies, trays or rearing rooms.

- Contact with residues present on leaves, stems or mulberry fields during leaf collection.

c. Inhalation

- Though less common, silkworms can inhale volatile insecticide vapours or fine aerosols that settle in rearing rooms or mulberry fields.

d. Environmental Contamination

- Soil residues can indirectly contaminate mulberry roots or water sources.
- Airborne drift and rainwater runoff may deposit insecticides in mulberry fields. **(**Hazarika *et al*., 2024) [6]

Importance of Understanding Exposure

Recognizing the types, uses and routes of Pesticide exposure is crucial for assessing the risks to silkworm health. It helps in designing preventive strategies, such as buffer zones between mulberry and crop fields, proper timing of insecticide applications, selection of safer chemicals and regular monitoring of residues in mulberry leaves.

2.5 Impact of pesticides on silkworm growth and development

Symptoms of pesticide poisoning

1. Chain type excreta

2. Swinging of entire silkworm body

3. Incomplete ecdysis

4. Larvae died after moulting

Fig 1: Symptoms of acute poisoning silkworm exposed to the spriotetramat (He *et al*., 2022)

Fig 3: Symptoms of pesticide poisoning in silkworm (Jyothi *et al*., 2019) [8]

a. Larval mortality during spinning stage
b. Dead larvae on mountage with hook shape and shrunken body
c. Ripened but dead larvae
d. Formation of flimsy cocoon
e. Formation of naked pupae and death
f. Larval mortality during rearing

3. Pest control by spraying pesticides and Effect of pesticides on silkworm growth and development

Subramanian *et al*. (2010) [20] conducted an experiment on bioefficacy of insecticides and botanicals *viz.*, dichlorvos 0.076 %, acetamiprid 0.03 %, triazophos 0.04 %, thiomethoxam 0.025 %, neem oil 3 % and pungam oil 3 % against mulberry thrips. The results showed that among the chemicals acetamiprid 0.03 %, thiomethoxam 0.025 % and dichlorvos 0.076 % were most effective in lowering thrips population (0.73, 2.23 and 7.31 per leaf, respectively) at 7 days after spray (DAS) compared to untreated control (62.70 per leaf).

Prabhaker and Castle (2011) [13] evaluated two systemic neonicotinoid insecticides (Imidacloprid 1.58 g and Thiamethoxam 0.55 g) by injecting the formulated materials into the root zone of each mulberry plants and found that the shoots from early August through mid-October showed a substantial reduction in *Maconellicoccus hirsutus* infestation (5-20 bugs / plant) in all treated plants, whereas infestation continued to rise in the untreated trees to peak level (85 bugs / plant) in the late September and by the end of the season, 10 out of 20 imidacloprid treated and 9 out of 20 thiamethoxam treated plants found completely free from mealybug infestation.

The insecticide molecules with unique mode of action were administered to mulberry leaves for management of mealy bugs. The results revealed that among the selected molecules dinotefuran 20 SG @ 0.25 g/l recorded the highest mealybug mortality at both 7 DAS (78.78 %) and 15 DAS (99.44 %). Further, the insecticide treated leaves when fed to silkworms, showed that flonicamid 50 WG @ 0.3 g/l recorded 100 per cent silkworm mortality while no mortality was recorded in all the other treatments. Effective Rate of Rearing (ERR) of 100 per cent were recorded in silkworms fed on mulberry leaves from treatments pymetrozine 50 WG @ 0.3 g/l at 10 DAS, azadirachtin 1% @ 2 ml/l and dichlorvos 76 EC @ 2.63 ml/l at 30 DAS (Yeshika *et al*., 2020) [23].

Sharath *et al.* (2022) [16] investigated the efficacy of a few pesticides against mites and determined their safety to silkworms. Among different molecules used as foliar spray, propargite 57 EC (@ 1.5 ml/L) was found effective against *Polyphagotarsonemus latus* and found safer to silkworm at 16 days after spray as revealed by the economic traits *viz.*, mature larval weight (2.85 g), fifth instar larval duration (184.20 h), cocoon weight (1.31 g), pupal weight (1.1 g), shell weight (0.22 g), shell ratio (16.58 %) and ERR (96.66 %).

A study was conducted by Kalpana *et al*. (2022) [9] to know the effect of a few molecules having both insecticidal and acaricidal properties on the performance of silkworm, *B. mori*. Among six different molecules used as foliar spray, abamectin 1.9 % EC (@ 0.75 ml/L) and diafenthiuron 50 % WP (@ 1 g/L) showed zero mortality, cent per cent larval progression, shortest larval duration, maximum larval weight and ERR percentage at 15 and 20 DAS. They suggested that abamectin 1.9 % EC and diafenthiuron 50 % WP could be used as an alternate molecule to DDVP for managing both thrips and mites in mulberry as they found safer to the silkworms.

4. Pesticide-induced tissue damage in silkworms: Degeneration of midgut

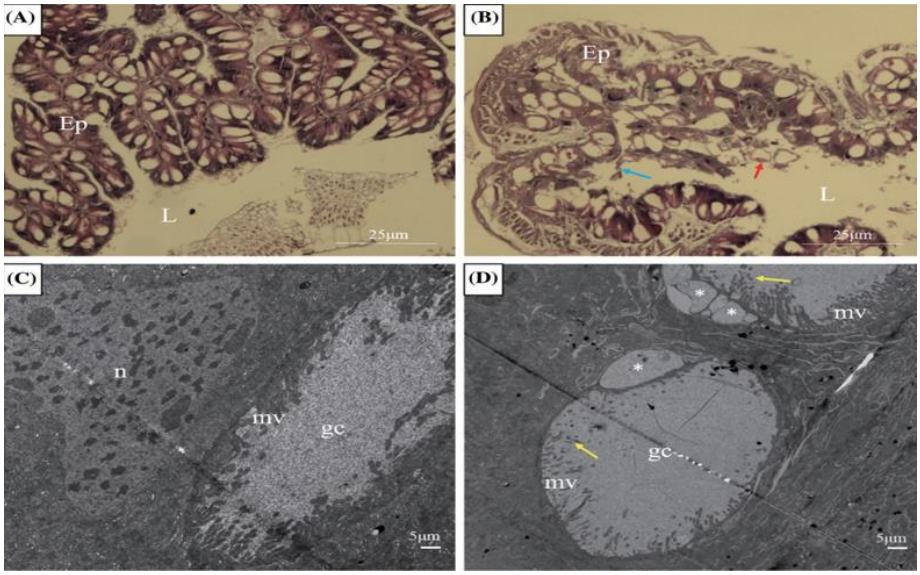


Fig 4: Effects of acetamiprid exposure on midgut histopathology and ultrastructure

Ep: epithelium; L: lumen; n: nucleus; mv: microvilli; gc: goblet cell; Red arrow: cells fell off; Blue arrow: rupture of plasma membrane; Yellow arrows: microvilli fell of; (*): vacuoles on the membrane of goblet cell. A and C- Control group B and D - Acetamiprid exposed group Wang *et al*. (2019) [21] investigated the histopathological and microstructural alterations in the midgut at 48 hours after exposure to acetamiprid. In the control group, the epithelial cells appeared normal, with an orderly alternation of goblet and columnar cells, an intact basal layer, and densely packed microvilli (Fig. 4: A, C). In contrast, the acetamiprid-treated group showed notable morphological

disruptions, including thinning of the basal layer, sparsely distributed microvilli, disintegration of intestinal wall cells, detachment of intracellular microvilli from goblet cells and the presence of vacuoles on the goblet cell membranes (Fig. 4: B, D).

The activities of midgut digestive enzyme *i.e.* amylase, trypsin and lipase recorded lower value compared to the control group at 24, 48, 72 and 96 hours after exposure to acetamiprid. Reduced enzyme activity was due to structural changes and inhibition of normal functioning of midgut resulted due to acetamiprid. Inhibition of these digestive enzymes may results in a metabolic imbalance, growth impairment and mortality of larvae (Wang *et al*., 2019) [21].

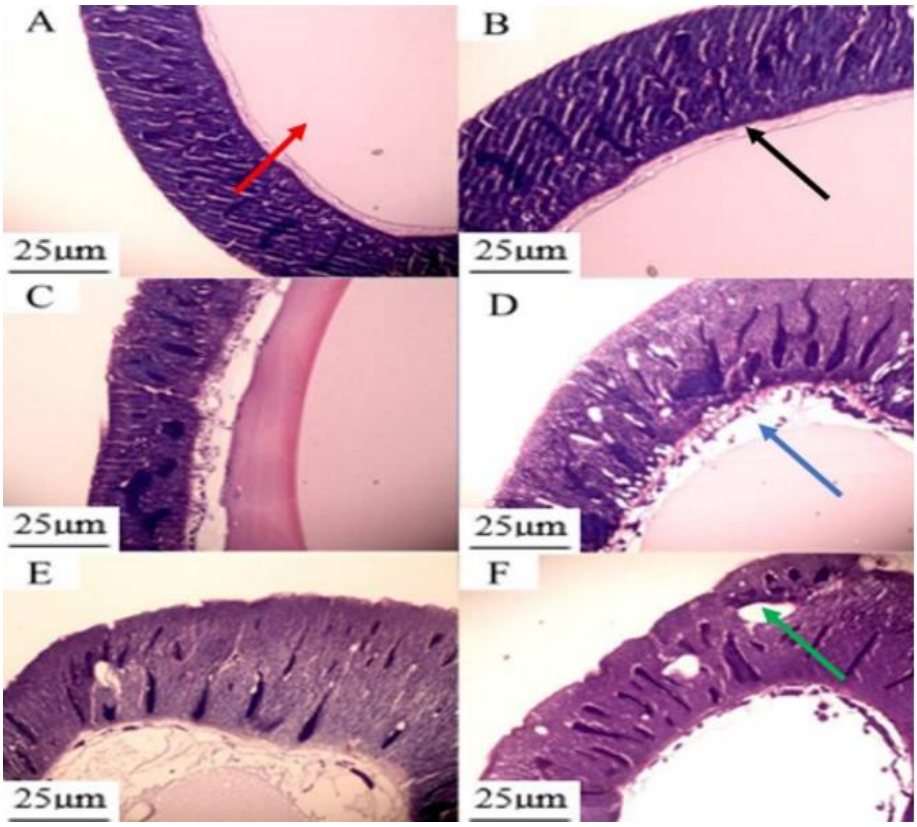


Fig 5: Histological photographs of silk gland tissue in fifth-instar larvae of *B. mori* under phoxim exposure

(A, C, E) Control group at 0, 24, 48 h; (B, D, F) Treatment group after 0, 24, 48 h of phoxim exposure. Black arrow indicates the inner wall of glandular cavity; Red arrow indicates liquid silk fibroin; Blue arrow indicates vacuolization in the lumen; Green arrow indicates damage to the gland membrane

In control group, the silk gland samples displayed a normal structure, characterized by thin walls and a completely filled gland lumen (Fig. 5A, C, E). Conversely, the phoxim-treated silk glands showed pronounced damage. The glands from the phoxim-exposed groups exhibited sparse epithelial cells, crevices in the gland membrane, and noticeable vacuolization within the lumen (Fig. 5B, D, F). At 0 hours of treatment, the silk gland morphology remained intact, showing thin walls and the gland cavity fully packed with protein (Fig. 5B). However, after 24 hours of exposure, vacuolization appeared in the lumen, along with a reduction in silk protein (Fig. 5D). By 48 hours, the gland structure was severely impaired, evident from glandular fissures and vacuoles (Fig. 5F). The extent of damage increased progressively with the duration of phoxim exposure (Cheng *et al*., 2018) [3].

Gonadal disruption

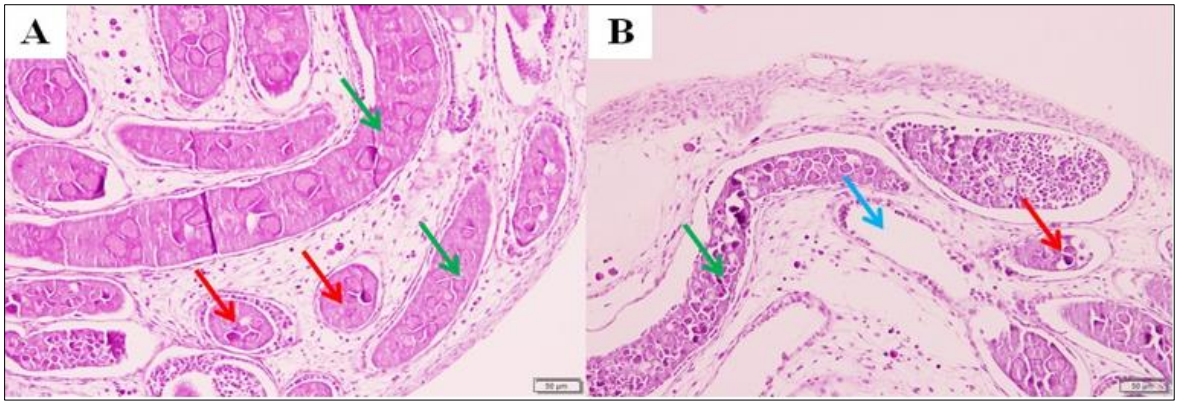


Fig 6: Microscopic images showing the ovarian tissue structure in fifth-instar *Bombyx mori* larvae at 96 hours after exposure to pyriproxyfen

(**a**) Control group and (**b**) 0.001 mg/L of pyriproxyfen treatment group
➡ Oocyte ➡ Oogonia ➡ Vacuoles

Qian *et al*. (2020) [14] investigated the impact of pyriproxyfen on reproductive system of the silkworm. Their findings indicated that silkworms fed with pyriproxyfen exhibited a reduction in the number of oocytes and oogonia in the ovaries, along with the appearance of vacuoles in the ovarian tissue of the treated group. This suggests that pyriproxyfen exposure can disrupt the normal development of ovarian tissue, potentially impairing the reproductive function of silkworms (Fig. 6). Additionally, the expression levels of ovarian developmental genes *Vg*, *Ovo*, *Otu*, *Sxl-S* and *Sxl-L* were significantly lower in the treatment group against the control group, with reduction of 6.08 %, 61.99 %, 83.51 %, 99.31 % and 71.95%, respectively.

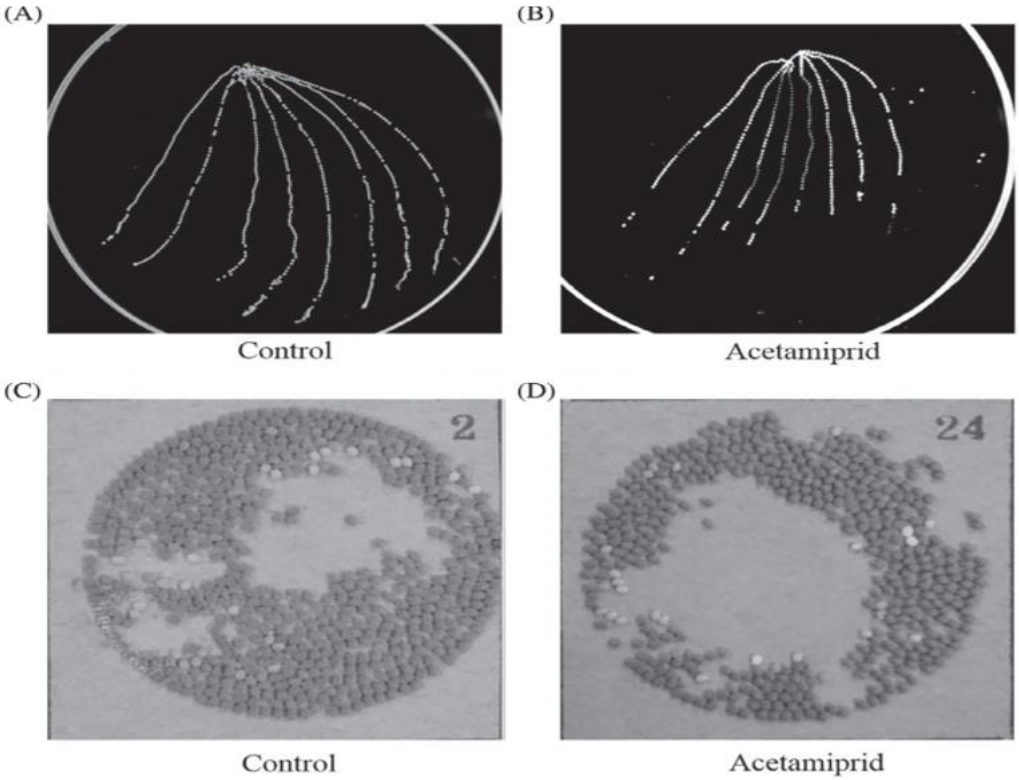


Fig 7: Effects of egg forming mechanism of *B. mori* after acetamiprid exposure. (A, B) The ovipositor of unmated moths (C, D) The oviposition of *B. mori*.

Cheng *et al*. (2019) evaluated the influence of trace levels of acetamiprid on silkworm fecundity by comparing oviduct structure and egg production between treated and control groups. In control, the oviduct was fully developed, containing uniformly sized and neatly arranged eggs in the fallopian tube. In contrast, acetamiprid exposure led to a significant reduction in oviduct length, smaller eggs that were unevenly distributed and minor damage to the lumen can be observed (Fig. 7).

An analysis of female oviposition revealed that the acetamiprid-treated group produced an average of 342 ± 31 eggs, which was 197 fewer than the control group. The weight of individual eggs in the acetamiprid group decreased by 0.524 ± 0.01 mg. The proportion of unfertilized eggs in the acetamiprid group was 8% ± 0.3%, comparable to the control. These findings suggest that even low levels of acetamiprid exposure can impair oviduct development in silkworms, resulting in reduced egg production.

Disruption of silk gland structure

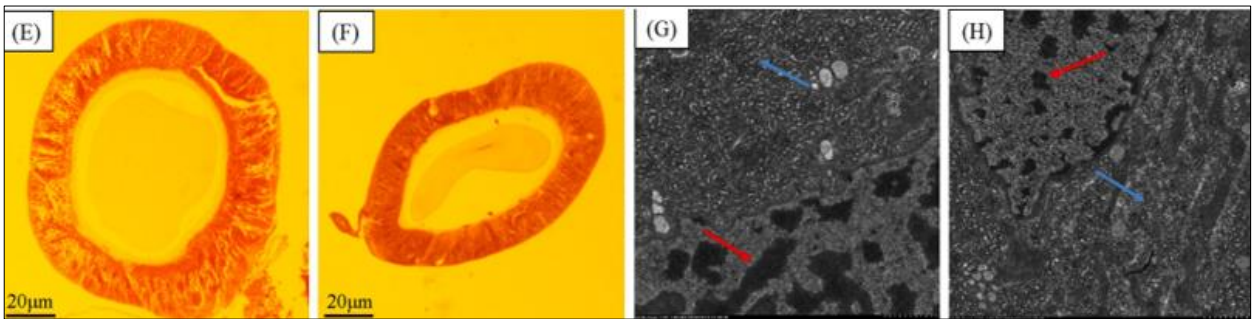


Fig 8: Comparison of PSG tissue at 96 h: (E, F) Histological images of the control (E) and treatment (F) groups; (G, H) Ultrastructural observations of the PSG in the control (G) and treated (H) groups. PSG: Posterior silk gland; Blue arrow: Endoplasmic reticulum; Red arrows: Nuclear bodies.

Lu *et al.* (2020) [11] investigated the effects of acetamiprid on the silk gland of *B. mori*. Histopathological sections showed pronounced vacuolization within the lumen of the PSG in the acetamiprid-treated group (Fig. 8E, F). Furthermore, transmission electron microscopy of PSG ultrastructure revealed that the control group exhibited normal cellular organization, with abundant and evenly distributed endoplasmic reticulum (ER), relatively large nuclear bodies, and intact, prominent nucleoli (Fig. 8G). In contrast, the acetamiprid-treated group displayed uneven chromatin distribution, reduced and fragmented ER, and nucleolar bodies that appeared disintegrated and sparse (Fig. 8H). These findings suggest that acetamiprid induces significant damage to the PSG.

Conclusion

The exposure of silkworm to pesticides poses a serious threat to its health and affect the sustainability of sericulture. Pesticides, through various routes and even at low concentrations disrupt the normal growth, development and productivity of silkworms. Such exposure can lead to physiological, biochemical and structural impairments that ultimately reduce cocoon quality and yield. The severity of the effects depends on the type and amount of pesticide, as well as the mode of exposure. Understanding these adverse impacts is essential for promoting safer agricultural practices, implementing effective preventive measures and adopting eco-friendly pest management strategies that protect both crop productivity and silkworm well-being. Sustainable approaches are crucial to safeguard this economically and culturally significant industry**.** Hence, detailed investigations on the physiological, biochemical and molecular, histopathological impact of plant protection chemicals in silkworm growth is imminent before the pesticide recommendation.

References

1. Bhosale SH, Kallapur VL. Changes in the metabolic fuel reserves of the V instar *Bombyx mori* following endosulfan treatment. Entomon. 1988;10(6):281-283.
2. Bizhannia A, Etebari K, Seidavi A, Mirhosseini SZ. The effect of a pesticide on mulberry silkworm *Bombyx mori* (Lep.: Bombycidae) larvae. In: Proceedings of the British Society of Animal Science. Cambridge University Press; 2007. p. 162.
3. Cheng X, Hu J, Li J, Chen J, Wang H, Mao T, *et al*. The silk gland damage and the transcriptional response to detoxifying enzymes-related genes of *Bombyx mori* under phoxim exposure. Chemosphere. 2018; 209:964-971.
4. Cheng X, Li F, Chen J, Wang H, Mao T, Li J, *et al*. Mechanism of trace acetamiprid-caused reproductive disorders in silkworm, *Bombyx mori*. Pest Manag Sci. 2019;75(10):2672-2681.
5. Goldsmith MR, Shimada T, Abe H. The genetics and genomics of the silkworm, *Bombyx mori*. Annu Rev Entomol. 2002; 50:71-100.
6. Hazarika S, Jekinakatti B, Bharathi BKM, Charitha K, Shruthi GH, Harika KR, *et al*. Impact of Novel Insecticides in Mulberry Ecosystem and Its Residual Effect on Silkworm Growth & Productivity. J Exp Agric Int. 2024; 46(9):37-44.
7. He P, Wei E, Wang R, Wang Q, Zhang Y, Tang X, *et al*. The spirotetramat inhibits growth and reproduction of silkworm by interfering with the fatty acid metabolism. Pestic Biochem Physiol. 2022; 188:105282.
8. Jyothi NB, Prashant NB, Maribashetty VG, Radhakrishna PG. Effect of pesticide residue in soil on silkworm, *Bombyx mori* L-survey analysis. Int J Ind Entomol. 2019;38(2):31-37.
9. Kalpana S, Banuprakash KG, Murali Mohan K, Vinoda KS. Effect of chemicals with insecticidal and acaricidal property on rearing performance of silkworm, *Bombyx mori* L. Multilogic Sci. 2022;12(41):2277-2282.
10. Li T, Hu C, Liu Z, Li Q, Fan Y, Liao P, *et al*. Histopathological and Transcriptional Changes in Silkworm Larval Gonads in Response to Chlorfenapyr Exposure. Insects. 2025;16(6):619-625.
11. Lu Z, Li M, Fang Y, Qu J, Ye W, Dai M, *et al*. The mechanism of damage to the posterior silk gland by trace amounts of acetamiprid in the silkworm, *Bombyx mori* L. Pestic Biochem Physiol. 2020;80(1):104676.
12. Muhammad A, Qian Z, Li Y, Lei X, Iqbal J, Shen X, *et al*. Enhanced bioaccumulation and toxicity of Fenpropathrin by polystyrene nano (micro) plastics in the model insect, silkworm (*Bombyx mori*). J Nanobiotechnol. 2025;23(1):38.
13. Prabhaker N, Castle SJ. Field evaluation of two systemic neonicotinoid insecticides against pink hibiscus mealybug *Maconellicoccus hirsutus* (Green) on mulberry trees. J Pest Sci. 2011; 84:363-371.
14. Qian HY, Zhang X, Zhao GD, Guo HM, Li G, Xu AY. Effects of pyriproxyfen exposure on reproduction and gene expressions in silkworm, *Bombyx mori*. Insects. 2020;11(8):467.
15. Schmutterer H. Properties and potential of natural pesticides from the neem tree, *Azadirachta indica*. Annu Rev Entomol. 1990;35(2):271-297.
16. Sharath M, Narayanaswamy KC, Manjunath Gowda. Efficacy of pesticides against yellow mite in mulberry and its residual toxicity on silkworm. Mysore J Agric Sci. 2022;56(2):315-324.
17. Shruthi GH, Karthik VC, Bharathi BKM, Dodmani BA. Role of Bio-control Agents in Mulberry Pest Management: Effective Strategies and Key Challenges. Int J Environ Clim Change. 2024;14(9):454-460.
18. Sithara H, Sandhya N, Ranjitha Bai H, Hrshitha Mala P. Residual effect of chemical insecticides used against thrips on cocoon and reeling parameters of silkworm, *Bombyx mori* L. Biol Forum Int J. 2024;16(8):236-242.
19. Stanley J, Preetha G. Pesticide toxicity to silkworms: exposure, toxicity and risk assessment methodologies. In: Pesticide Toxicity to Non-target Organisms: Exposure, Toxicity and Risk Assessment Methodologies; 2016: 229-275.
20. Subramanian S, Muthuswami M, Krishnan R, Thangamalar A, Indumathi P. Bioefficacy of botanicals and insecticides against mulberry thrips, *Pseudodentrothrips mori* Niwa. Karnataka J Agric Sci. 2010;23(1):47-50.
21. Wang H, Li F, Qu J, Mao T, Chen J, Li M, *et al*. The mechanism of damage by trace amounts of acetamiprid to the midgut of the silkworm. Environ Toxicol Entomol. 2019;34(9):1043-1051.
22. Xu S, Hao Z, Li Y, Zhou Y, Shao R, Chen R, *et al*. Biochemical toxicity and transcriptome aberration

induced by dinotefuran in *Bombyx mori*. Environ Pollut. 2022;30:119562.
23. Yeshika MP, Banuprakash KG, Murali Mohan K. Field evaluation of novel insecticides against *Maconellicoccus hirsutus* green in mulberry ecosystem and their safety to silkworm, *Bombyx mori* L. J Entomol Zool Stud. 2020;8(4):1067-1072.
24. Yu RX, Wang YH, Hu XQ, Wu SG, Cai LM, Zhao XP. Individual and joint acute toxicities of selected insecticides against *Bombyx mori* (Lepidoptera: Bombycidae). J Entomol Zool Stud. 2015;109:327-333.
25. Zhang MM, Sun YL, Zhao YY, Li EL, Song GZ, Su B, *et al*. Effects of Acetamiprid on growth and development of *Bombyx mori* and Antioxidant enzyme activity of midgut. Sci Seric. 2018; 44:0094-0099.