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Comparative assessment of mortality and histopathological alterations of deltamethrin and chlorpyrifos on brain and thoracic ganglion of *Sarcophaga ruficornis* (Diptera: Sarcophagidae)

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Abstract
Flesh fly *Sarcophaga ruficornis* causes myiasis and also serves as a vector for mechanical transmission of pathogens (trophozoites, helminth eggs). This study evaluates the histological effects of lethal concentration of deltamethrin and chlorpyrifos insecticide on the nervous system of flesh fly *S. ruficornis* in the laboratory. After being exposed to deltamethrin (0.001% and 0.003%) and chlorpyrifos (0.001% and 0.002%), adult flies mortality was recorded after 24 and 48 hours. To estimate insecticide toxicity, LC_{50} values were calculated using probit analysis. In both insecticide treatments, mortality rose with concentration and span of exposure. Deltamethrin produced mortality ranging from 29–41% at 24 hours and 37–50% at 48 hours, whereas chlorpyrifos caused mortality ranging from 24–38% at 24 hours and 34–48% at 48 hours. After 48 hours, LC_{50} values decreased, signifying that extended contact increases toxicity. Haematoxylin and eosin staining of the brain and thoracic ganglia revealed neurodegeneration, vacuolization, and neuropile degeneration in treated flies, while normal tissue organization was seen in control groups. The outcomes obtained specify that the population of this fly can be controlled by using deltamethrin and chlorpyrifos insecticide.

Keywords: *Sarcophaga ruficornis*, thoracic ganglion, brain, histology, deltamethrin, chlorpyrifos

Introduction
Insects constitute the most diverse and abundant group of organisms on earth, playing crucial roles in ecosystem functioning, including pollination, decomposition, and nutrient cycling.[1] However, certain insect species attain pest status due to their direct impact on human health, livestock, and agriculture, often necessitating targeted control measures. Dipteran flies, particularly members of the family Sarcophagidae (flesh flies), represent this dualism. While they contribute to the decomposition of organic matter, their synanthropic nature brings them into close and often detrimental contact with human settlements and animal husbandry [2, 3].

Sarcophaga ruficornis (Fabricius, 1794) is a widely distributed flesh fly of significant ecological and forensic importance [4]. As a carrion breeder, it plays crucial role in nutrient recycling. Moreover, it is a pioneer colonizer of decomposing remains, making its succession data invaluable in forensic entomology for estimating post-mortem intervals (PMI) [5]. Despite this forensic utility, its biology presents significant challenges in medical and veterinary contexts. The larvae of *S. ruficornis* are facultative agents of myiasis, infesting living tissues of livestock and occasionally humans, causing tissue damage, secondary infections, and economic losses (Zumpt,1965) [6]. Adults act as mechanical vectors for a range of pathogens, including bacteria, viruses, and helminth eggs, as they move between filth, food sources, and animal hosts, posing a threat to public and veterinary health [7]. Thus, the management of *S. ruficornis* populations in peri-domestic and agricultural settings is often essential. Chemical insecticides, including neurotoxic agents such as pyrethroids and organophosphates, remain a cornerstone of integrated pest management (IPM) programs for such dipteran pests (Baron, 1991). Deltamethrin, a sodium channel modulator, and chlorpyrifos, an acetylcholinesterase inhibitor, are two broad-spectrum insecticides with distinct modes of neurotoxic action [8]. However, the efficacy of control and the development of potential resistance are contingent on a thorough understanding of the physiological and morphological impact of these insecticides on the pest's vital systems.

This study investigates the neurotoxic effects of deltamethrin and chlorpyrifos on *S. ruficornis*. The study report adult mortality bioassays and present a histological analysis of the brain and thoracic ganglia to elucidate the structural alterations in the central nervous system following exposure. This combined approach aims to correlate insecticidal efficacy with direct neuroanatomical damage, providing insights that could inform more effective control strategies against this forensically and medically significant fly.

Materials and Methods
1. Insect Rearing
The adult flesh flies were collected from the Aligarh Muslim University campus of North India region. Minced rotten buffalo meat was used as bait for larviposition. Once the flies larviposits, meat was transferred to the cages made of fine wire mesh and plyboard. Cages were transferred to BOD cabinet maintained at a temperature of 27±2° and relative humidity of 60±5%. Larvae, reared on minced buffalo meat, were provided with cotton in their third larval stage to facilitate pupation. Pupae were sorted from cotton manually and kept in separate cages until adult eclosion. Adult flies were reared on rearing media of sugar, honey and milk in the ratio of 1:1:3 soaked in cotton [9, 10, 11].

2. Experimental design
Adult flies were divided into groups of thirty individuals and exposed to two insecticides in two concentrations by oral method: deltamethrin (0.001% & 0.003%) and

chlorpyrifos (0.001% & 0.002%), with each concentration replicated three times alongside a concurrent control group. Mortality was assessed at 24 and 48 hours post-exposure. Brains and thoracic ganglia were dissected from alive and moribund flies under stereo zoom microscope and transferred into cavity blocks.

Mortality percentage was calculated using the following formula:

$$\frac{\text{Flies dead}}{\text{Total number of flies}} \times 100$$

3. Histological preparation

Control and treated tissues were fixed in Bouin's solution for 18 hours, followed by washing in tap and distilled water for 15 minutes. After fixation, dehydration of tissues was done through an ascending alcohol series ranging from 30% to 70% for 15 minutes. Subsequently, tissues were kept in aqueous eosin for staining. Dehydration continued to absolute alcohol for 15 minutes followed by clearing in xylene-alcohol mixture and pure xylene to remove remaining water. Tissues were incubated with a 1:1 xylene-wax mixture for 15 minutes and then pure wax for 2 hours at 62°C before embedding in paper blocks. Blocks were trimmed and sectioned at 5 µm on a rotary microtome (York Scientific industries Delhi India). Ribbons were mounted on albumin-glycerine coated slides. Furthermore, slides were placed on Yorko Slide warming table to remove wrinkles and for drying. Subsequently, slides were de-waxed in xylene twice for 10 minutes. Rehydration of the slides was done via descending alcohol series (100% > 96% > 90% > 80%) and washed with distilled water for 5 minutes. The slides were stained in delafield's haematoxylin for 15 seconds, rinsed in tap water and distilled water for 5 minutes, and counterstained in eosin for 15–25 minutes. Dehydration upto absolute alcohol for 5 minutes at each concentration. The slides were kept in xylene twice for 10 minutes, and were finally mounted with DPX and examined under a compound microscope.

4. LC_{50} Estimation

The sub-lethal concentration (LC_{50}) of the insecticides was calculated from acute toxicity bioassay data obtained at 24 hour and 48 hours for the concentrations. Each observation was tested in triplicate. Mortality was expressed as the proportion of dead individuals relative to the total number of flies.

LC_{50} values were determined using a generalized linear model with a binomial error distribution and probit link function described by the equation:

$$Probit(p) = \beta 0 + \beta 1 log10(C)$$

Where p denotes the proportion of mortality, C represents the insecticide concentration, β0 is the intercept of the regression model, and β1 is the slope of the dose–response relationship. The LC_{50} was calculated as the concentration corresponding to 50% mortality (p = 0.5; probit = 0) using the equation:

$$log10(LC50) = -\beta 1 \beta 0$$

The resulting value was back-transformed to obtain LC_{50} in the original concentration units. All statistical analyses and dose–response curve generation were performed using Python (Statsmodels library).

Results and discussion

1. LC_{50} evaluation

Acute toxicity bioassays revealed a considerable concentration- and time-dependent increase in mortality for both chlorpyrifos and deltamethrin. Both insecticides increased mortality with increasing concentrations at 24 and 48 hours of exposure, indicating a consistent dose-response relationship. Mortality at 24 hours with chlorpyrifos ranged from about 24% at the lowest concentration (0.001) to about 36% at the highest concentration (0.002). Probit analysis assessed the LC_{50} value at 0.0043, which surpassed the tested concentration range, indicating that 50% mortality did not occur within 24 hours. At 48 hours of exposure, mortality increased significantly (34-48%), and the LC_{50} value declined to 0.0023, indicating greater toxicity with prolonged exposure (fig.1). Similarly, deltamethrin exposure resulted in increased mortality with concentration and time. At 24 h, mortality increased from approximately 29% at 0.001 to about 41% at 0.003. It yielded an LC_{50} value of 0.0067 which lay above the highest tested concentration (fig.2). At 48 h, mortality ranged from approximately 37% to nearly 50%, and the LC_{50} value declined to 0.0030 which signifies increased lethality with extended exposure duration.

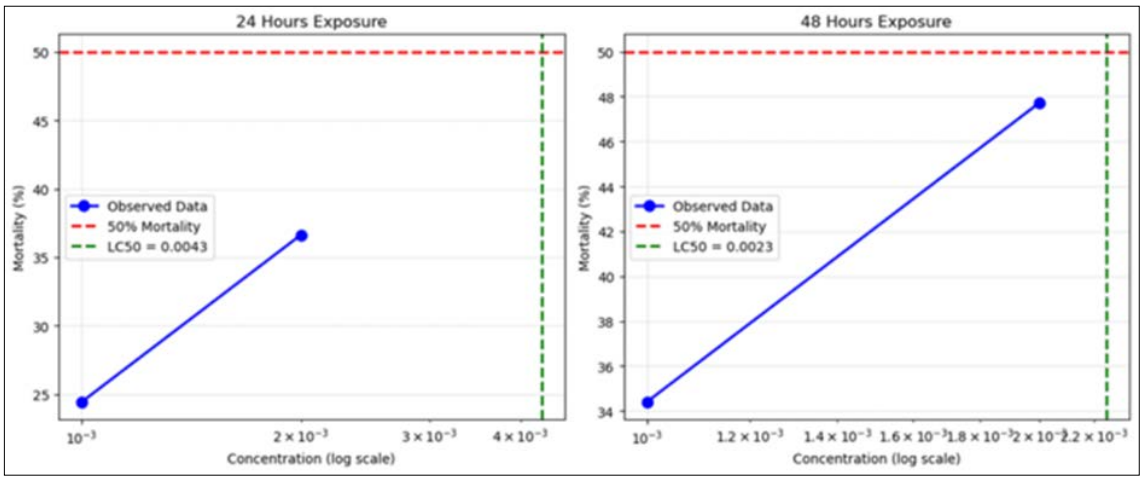


Fig 1: Probit analysis to evaluate LC_{50} value for chlorpyrifos.

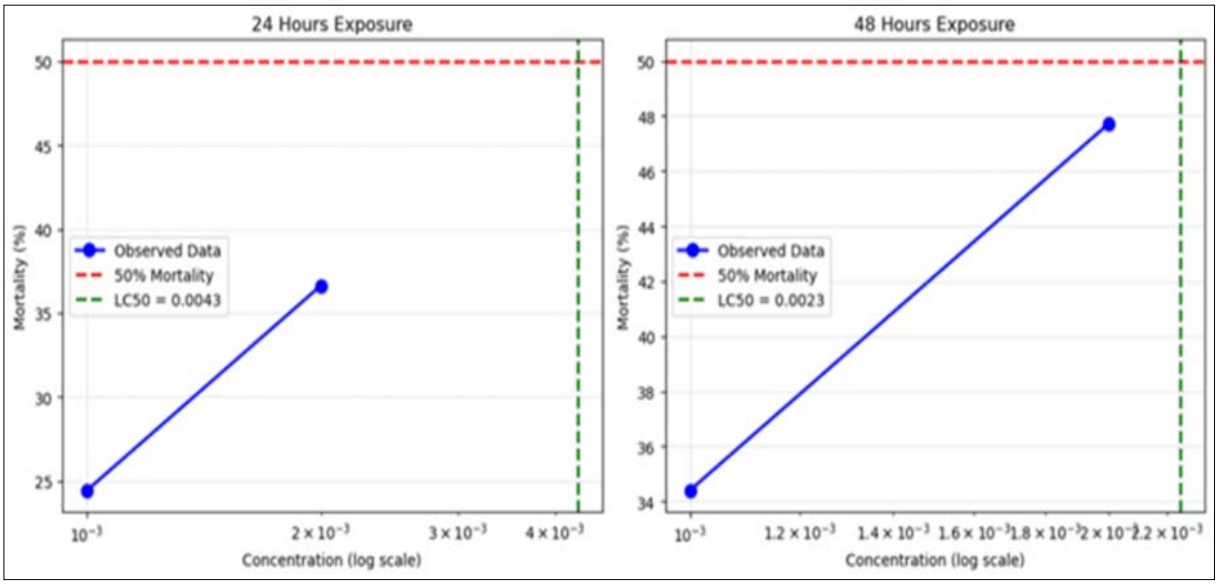
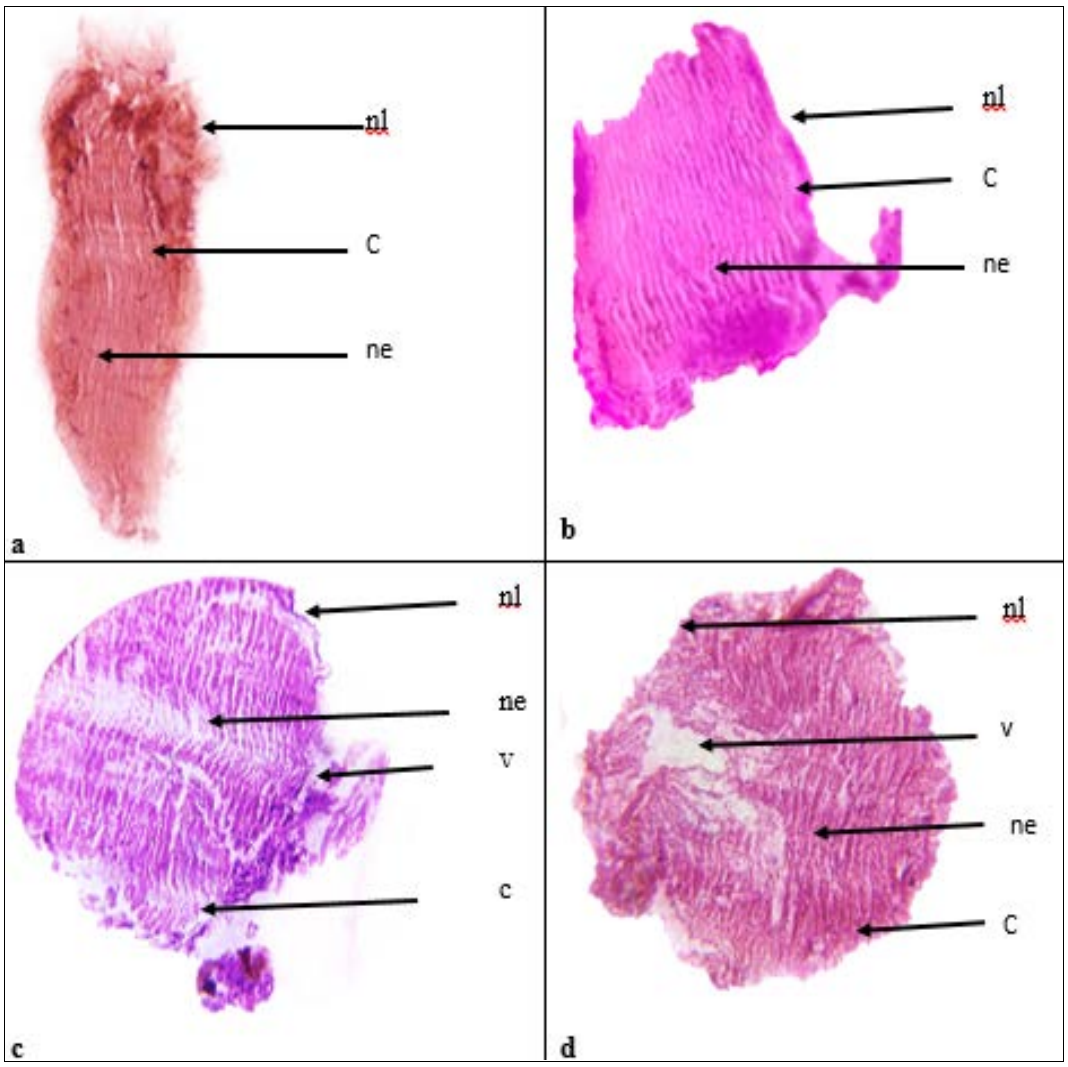


Fig 2: Probit analysis to evaluate LC_{50} value for deltamethrin.

2. Histological observation

The neural lamella is intact in control ganglia (fig.3 a,b). Following 24 hours of treatment, it shows clear rupture, which becomes more extensive upon 48 hours of exposure (fig.3 c to l). Deltamethrin exposure showed stronger vacuolation than chlorpyrifos, which are more severe after 48 hours than after 24 hours. The brain of *S. ruficornis* is divided into distinct regions, with condensed neuropiles in the central region and neuronal cell bodies in the periphery. Protocerebrum, deutocerebrum, optic lobes and compound eyes are evident in the images (fig.4). The suboesophageal commissure lies below the oesophageal ganglion. Unexposed flies do not show significant morphological changes in their thoracic ganglia and brain.



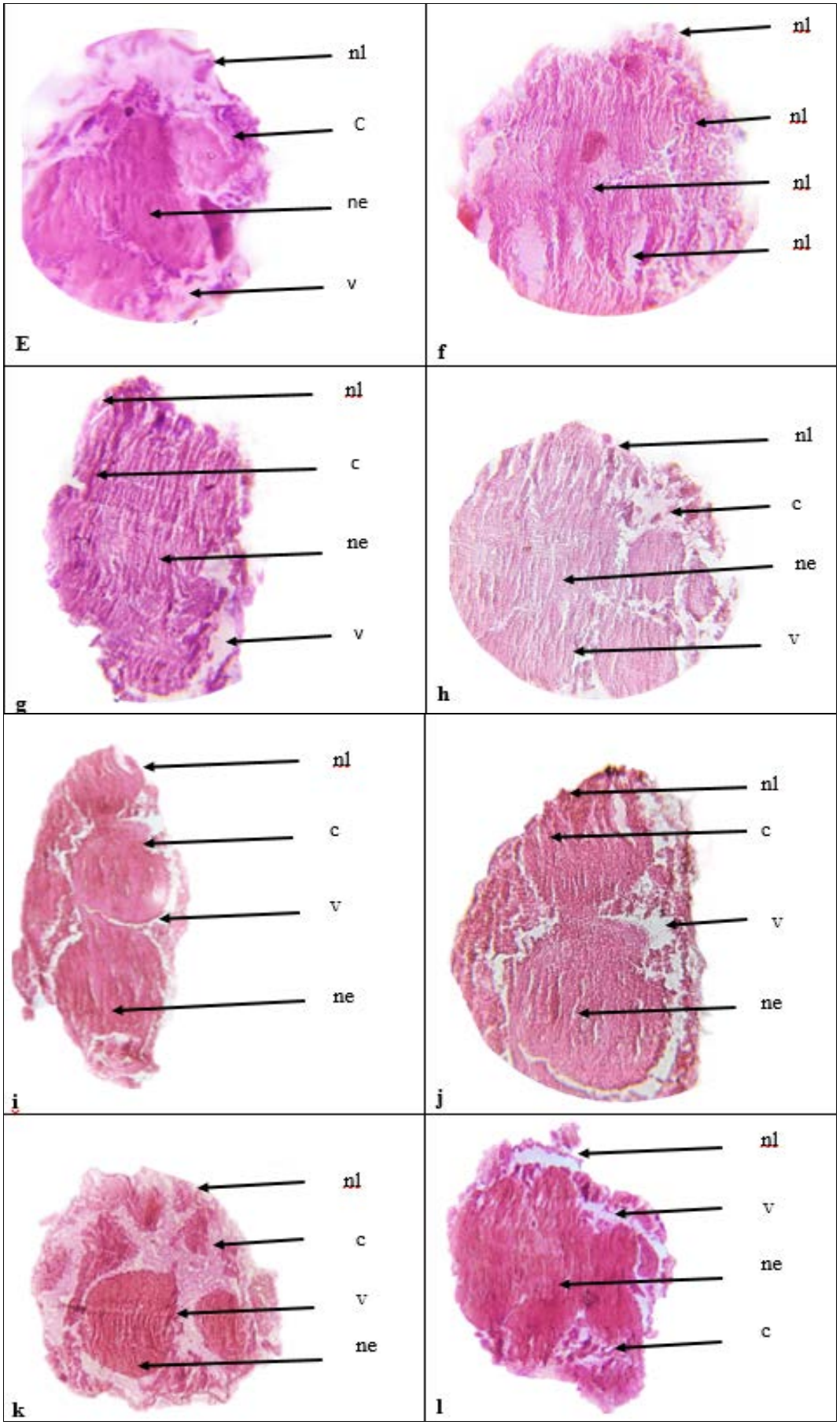
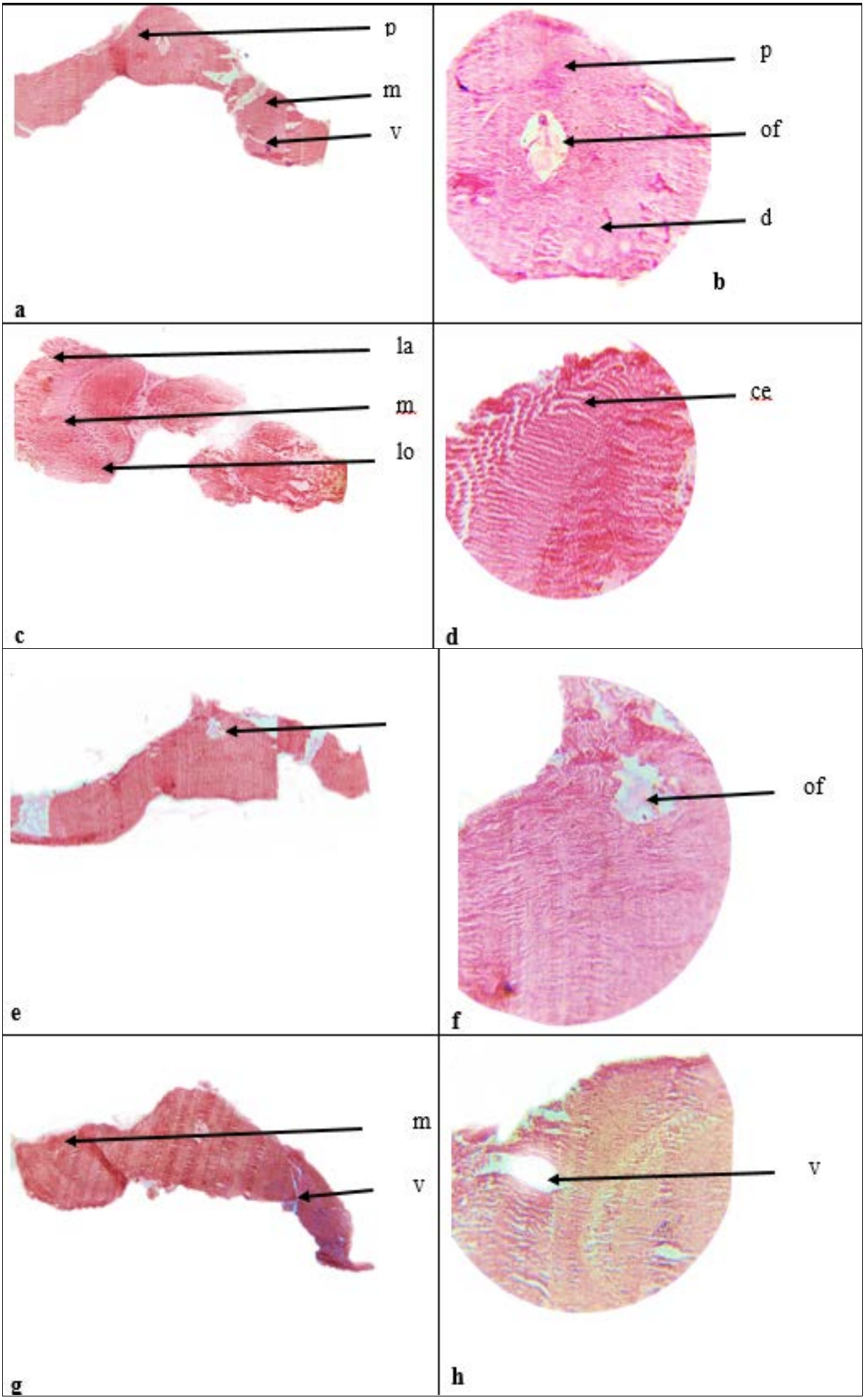


Fig 3: Histological sections of *S. ruficornis* thoracic ganglia stained with hematoxylin and eosin: (a and b) control sections with no exposure to insecticide; (c) 0.001% chlorpyrifos exposure after 24 h; (d) 0.001% chlorpyrifos exposure after 48 h; (e) 0.002% chlorpyrifos exposure after 24 h; (f) 0.002% chlorpyrifos exposure after 48 h; (g) 0.001% deltamethrin exposure after 24 h; (h) 0.001% deltamethrin exposure after 48 h; (i and j) 0.003%deltamethrin exposure after 24 h; (k and l) 0.003% deltamethrin exposure after 48 h. nl; neural lamina, v; vacuoles, ne; neuropile, c; cortex.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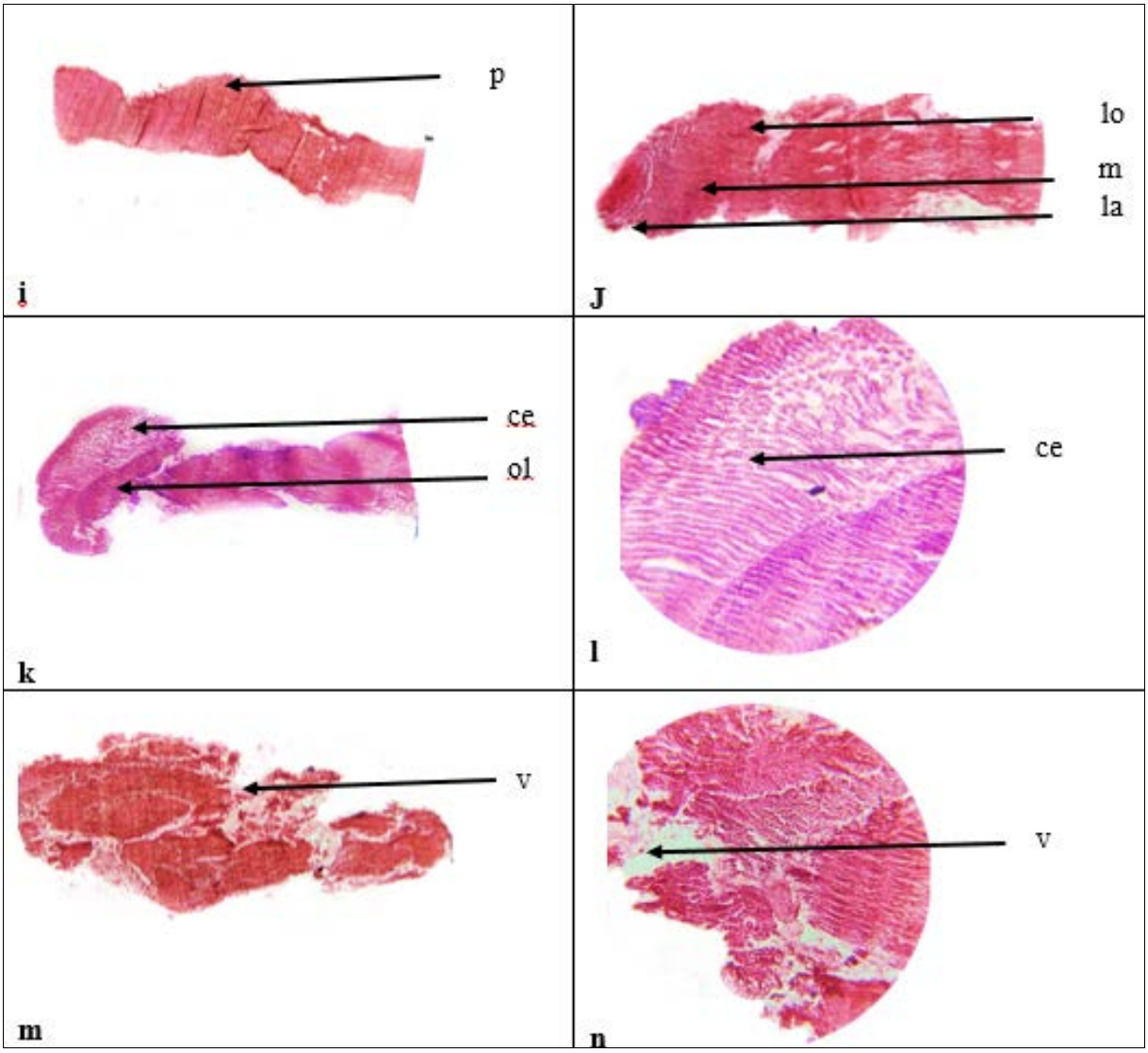

Fig 4: Histological sections of *S. ruficornis* brain stained with hematoxylin and eosin (a and b) 0.001% chlorpyrifos exposure after 24 h; (c and d) 0.001% chlorpyrifos exposure after 48 h; (e and f) 0.002% chlorpyrifos exposure after 24 h; (g and h) 0.002% chlorpyrifos exposure after 48 h; (i) 0.001% deltamethrin exposure after 24 h; (j) 0.001% deltamethrin exposure after 48 h; (k and l) 0.003% deltamethrin exposure after 24 h; (m and n) 0.003% deltamethrin exposure after 48 h. p; protocerebrum, d; deutocerebrum, of; oesophageal foramen, v; vacuoles, ol; olfactory lobe, ce; compound eye, m; medulla, la; lamina, lo; lobula.

Conclusion

In the present study, deltamethrin showed higher toxicity than chlorpyrifos. Sections of the brain and thoracic ganglion revealed notable neural changes, including vacuolated regions, degenerating cells, and a malformed neuropile pattern. Flies showed lack of coordination in thoracic ganglion (wing and leg movement control) at high doses of both insecticides. This results in uncontrolled movements, inability to maintain posture, erratic flight or walking etc. Overall brain and thoracic ganglion structure was preserved in control flies, which included outer chiasma, protocerebrum, lamina, and suboesophageal commissure. The neural lamella is intact in control ganglia.

The mortality concentration response of *Sarcophaga ruficornis* to synthetic insecticides, deltamethrin and chlorpyrifos, and its impact on histology were determined in laboratory. Both insecticides produce dose and time dependant mortality.

Discussion

The study examined the toxicological and histological effects of chlorpyrifos and deltamethrin on *Sarcophaga ruficornis*. After 24 and 48 hours post treatment, mortality was calculated. Probit analysis was used to estimate LC_{50} values. Furthermore, haematoxylin and eosin staining was used for histological analysis of the brain and thoracic ganglia [11].

Flies treated with chlorpyrifos, mortality increased from 24% at 24 hours to 34% at 48 hours at a concentration of 0.001%, while mortality rose from 38% at 24 hours to 48% at 48 hours at a concentration of 0.002%. Likewise, exposure to deltamethrin caused mortality rates of 29% and 41% at 24 hours for concentrations of 0.001% and 0.003%, respectively, which increased to 37% and 50% after 48 hours. These findings propose mortality was higher at 48 hours as compared to 24 hours for both concentrations. Longer exposure period clearly affects the toxic effects as demonstrated by the decline in LC_{50} values.

Long-term introduction may increase insecticidal toxicity due to increased absorption, and accumulation within the insect body. Increased mortality at higher concentrations supports the theory of dose-dependent toxicity.

Deltamethrin produced higher mortality than chlorpyrifos at two different contact periods, especially at higher

concentrations. This suggests that deltamethrin has comparatively stronger insecticidal efficacy [12]. The two compounds different modes of action could account for the differences in toxicity. Chlorpyrifos causes toxicity by inhibiting acetylcholinesterase activity, which causes acetylcholine to build up at synaptic junctions and continuously stimulate the nervous system. Ultimately, this excessive neural excitation causes death and paralysis. On the other hand, the pyrethroid insecticide deltamethrin mainly affects voltage-gated sodium channels in nerve membranes. This prolongs sodium influx and results in hyperexcitation, paralysis, loss of coordination, repetitive nerve firing, and ultimately death [13, 14, 16, 17, 18].

Histological sections of brain and thoracic ganglia of deltamethrin exposure showed stronger vacuolation and neuropile disruption than chlorpyrifos. In particular, the thoracic ganglia are essential for regulating wing movements and locomotion in dipteran insects [12, 13, 20]. Therefore, reduced mobility and motor dysfunction frequently linked to insecticide-induced neurotoxicity may be explained by degeneration of thoracic ganglia. The above study's histological results strongly support the mortality data.

Overall, the present study shows that deltamethrin and chlorpyrifos both have major toxic effects on *Sarcophaga ruficornis*. While LC_{50} values decreased over time, suggesting increased toxicity with extended exposure. Mortality rose with both concentration and exposure duration. The nervous system is a primary target of both insecticides. These results contribute to our understanding of insecticide-induced neurotoxicity in dipteran flies and may provide useful reference evidence for future studies on insect toxicology, pesticide action, and environmental risk assessment.

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Conflict of Interest: Authors declare no conflict of interest.

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