Original Article

Exploring the Effectiveness of Chlorophacinone in Managing Urban Rat Infestation: A Laboratory Study on the Norway Rat, *Rattus norvegicus*, a Common Urban Rat in Malaysia

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Abstract

Background: Rat populations in urban areas must be monitored and controlled, as they can transmit zoonotic diseases and pose a risk to human health. Certain urban rat species may not respond well to some of the rodenticides available on the market. Therefore, this study aimed to assess the efficacy of chlorophacinone in controlling urban rats in a laboratory setting.

Methods: Chlorophacinone, the primary focus of this study, was compared to a commonly used first-generation anticoagulant rodenticide (FGAR) available in the urban market, namely Coumatetralyl. Rat specimens were trapped in Kuantan, Pahang, and the dominant species was identified as *Rattus norvegicus*.

Results: Overall, chlorophacinone demonstrated nearly complete mortality (95%) within an average of eight days post-feeding, followed by coumatetralyl, which exhibited high mortality (85%) within an average of seven days post-feeding. The rats in the chlorophacinone group consumed significantly lower dosages than those in the coumatetralyl group. This was due to variations in the default concentrations of the active ingredient of both treatments. Nevertheless, rats in the chlorophacinone treatment still experienced a higher mortality rate compared to coumatetralyl, despite consuming a lower dosage.

Conclusion: Consequently, this finding suggests that *R. norvegicus* is more susceptible to chlorophacinone than coumatetralyl. This underscores the potential of chlorophacinone as an effective rodenticide for controlling rat infestations in urban areas throughout Malaysia.

Keywords: *Rattus norvegicus*; Rodenticide; First-generation anticoagulant; Chlorophacinone

Introduction

Agricultural activities and urbanization lead to changes in rodent species diversity because of habitat destruction or disturbance (1, 2). Urbanization creates favourable conditions that provide resources and support the proliferation of commensal rodents, such as rats, in human habitation (3, 4). Urban rat infestations damage infrastructure, stored food, and businesses. There were also health concerns for the public because rats could spread microorganism diseases like toxoplasmosis (5), trichinosis (6), hymenolepiasis, giardiasis, amoebiasis, and schistosomiasis (7, 8). Brown rats and black rats are key hosts for *Leptospira interrogans*, the pathogen causing leptospirosis, a significant global public health concern (10, 11).

Anticoagulant rodenticide (AR) is a chronic-acting poison that has been adopted as a default control measure globally to combat rat infestation in agriculture and urban settings (12, 13). The use of ARs has improved rodent control by reducing bait toxicity, providing an

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antidote for accidents, and addressing bait aversion caused by rodents associating conspecific deaths with food ingestion (14). In Malaysia, several ARs are commonly used in both agriculture and urban settings, such as coumatetralyl, diphacinone, brodifacoum, and flocoumafen (15, 16). Chlorophacinone is a first-generation anticoagulant (FGAR) commonly used in agricultural settings and well documented for its competency to control common rodent species in agricultural settings, such as wood rat, *Rattus tiomanicus*, rice field rat, *R. argentiventer*, and house rat, *R. r. diardii* (16‒18). First-generation anticoagulants are typically less harmful and enduring than second-generation anticoagulants, resulting in reduced risk to unintended species from indirect poisoning and accumulation in the environment (19). Besides the report on the toxicant that is widely used in agricultural settings, knowledge of the usage of chlorophacinone in urban settings in Malaysia is scarce since most of the information available is from overseas (20‒23).

The availability of first-generation anticoagulant rodenticides (FGARs) in the Malaysian urban pest control market is limited. Among them, coumatetralyl appears to be the most commonly used FGAR in urban environments. However, the repeated use of a single rodenticide increases the risk of resistance development in rat populations. Such resistance may necessitate the use of more potent rodenticides, which in turn raises the risk of environmental contamination and both primary and secondary poisoning of non-target species. If proven effective against common urban rats, chlorophacinone could offer pest control professionals an alternative FGAR, supporting a more sustainable approach to rodent management in urban settings. Therefore, this study aims to provide essential insights into the efficacy of chlorophacinone against *Rattus norvegicus*, the prevalent urban rat species in Malaysia.

Materials and Methods

Rat Specimens Collection

The wild rat specimens were collected from a wet market lane in Kuantan, which was heavily infested with rats. The dominant species within the vicinity was identified as Norway rat, *R. norvegicus*, by using the key identification provided by Lund (24), which was the common species of urban rats in Malaysia. The rat specimens were collected using a combination of trapping and hunting. Trapping involved using live traps (28 cm (L) X 13 cm (W) X 13 cm (H)) with several food baits such as dried fish, chicken skin, rotten chicken skin, and oil palm fruit. The traps were placed near the rat nests, which were typically burrows, existing holes, water pipeline openings in the building, or along rat paths identified through observation of droppings or faeces. Hunting involved using a fishing net woven to a 35cm diameter round steel frame with a wooden pole of 1 m (Fig. 1A). Both trapping and hunting were conducted at night from 8.00 pm until 12.00 midnight (Fig. 1B).

Laboratory Evaluation of Different Rat Baits on Rat Mortality

The laboratory evaluation was conducted through a no-choice feeding trial. The trial took place at Mammalian Pest Laboratory, PPP Tun Razak, Jerantut, Pahang, Malaysia. All captured wild rats were weighed, sexed, and individually housed in rat cages measuring 28 cm (W)× 36 cm (L)× 16 cm (H) with woodchips provided for bedding and substrate for excretion. Only adult wild rats weighing between 250-350 grams were selected for this trial. Before the feeding trial, all selected rat specimens were acclimatized for two weeks. All rats received a laboratory corn as diet and had access to water *ad libitum* during this period. During this period, all rats were monitored, and any rat showing abnormalities, where those rats which not feeding below their normal range (approximately 8g per day) or general weakness, was removed from the trial and replaced with a healthy specimen. A total of 40 healthy Norway rats (*R. norvegicus*) were randomly divided into two groups of 20 individuals, each consisting of 10 males and 10 females. The randomization was based on a completely randomized design. Each group was assigned treatment as follows: T1 (chlorophacinone 0.005% a.i.) and T2 (coumatetralyl 0.0375% a.i.). An additional ten rats (5 males and 5 females) were allocated as T3 (control group), and so were left untreated. Both treatment baits were commercially produced and purchased directly from the manufacturer.

Each rat in each treatment group received two block baits to meet their average daily consumption of approximately ten percent of their body weight, based on information provided by Smith and Meyer (25), and supported by a preliminary feeding test. *Rattus norvegicus* had a greater body size, with an average of 288.25±15.20 g. The baits were offered to the rats in a single feeding session without any other optional diet, and they had free access to water. Once the rats had consumed the baits, they were maintained on a laboratory diet for a period of 21 days for observation. In the control group, the rats were provided only the laboratory diet (broken maize). Throughout the monitoring period, the rats were observed daily, and any signs of rodenticide toxicosis were recorded through external symptoms such as signs of bleeding and a passive or weak condition. The dead rats were recorded along with the date, and the bodies were weighed. The variables recorded in the trial included rat weight, bait consumption, dosage consumption, mortality rate (%), and time to death. In this study, all data were recorded in a Microsoft Excel spreadsheet, and the variables were analysed accordingly. The lab testing procedure followed the Malaysian Standard MS1256:2010 Household pesticide products–Rat bait–Specification (First Revision).

Results

Table 1 summarizes the results of the no-choice feeding test of chlorophacinone and coumatetralyl against the Norway rat, *R. norvegicus*. Despite each group of rats receiving the same amount of bait, the actual dosage received per body weight was different due to the variation in the default concentration of the formulations as produced by the respective manufacturer. The actual dosage of the toxicant consumed by the rats was 3.5±0.26 mg/kg for the chlorophacinone group, which was much lower than the 13.6±0.55 mg/kg consumed by the rats in the coumatetralyl group.

Despite the lower actual dosage consumed, chlorophacinone bait treatment managed to achieve almost complete control (95%) against *R. norvegicus*, while the coumatetralyl bait had an 85% mortality rate, suggesting the high potency of chlorophacinone despite low dosage consumption by the rats. Regarding the period required to achieve the aforementioned mortality rates, chlorophacinone bait took slightly longer than coumatetralyl. To achieve almost complete control over *R. norvegicus*, the chlorophacinone treatment took an average of 8.02 ±0.37 days, with a range of 3 to 30 days, while coumatetralyl needed an average of 7.12±2.55 days, with a range of 3 to 29 days, to achieve the recorded mortality rate.

A plausible explanation for why chlorophacinone took slightly longer than coumatetralyl, despite its high efficacy against rats, could be the lower concentration of its active ingredient. This may have influenced the speed of its mode of action, delaying its overall effectiveness in the rats' bodies. The wide variability in the number of days to mortality among individuals in the respective treatments was possibly due to physiological or feeding behavioural differences, which affected the level of exposure to the active compound in the bait (26). These differences may have influenced the speed at which the active compound took effect in the systems of different individual rats. It can be interpreted that chlorophacinone, based on the result in this study, despite the lower dosage, resulted in higher mortality compared to coumatetralyl. This suggests that chlorophacinone may be more potent against *R. norvegicus* than is coumatetralyl.

Table 1. Results of the no-choice feeding trial with chlorophacinone and coumatetralyl baits against Norway rats (*Rattus norvegicus*). The results highlight the average bait consumption and mortality of the rats, along with the mean time to death and the range of days to death for each treatment group

Treatment	Average Rat Weight (g)	Average Dosage consumption/rat (mg/kg)	Average % Mortality	Day of death	
				Average	Range
Chlorophacinone 0.005%	288±4.61	3.5±0.26	95±1.55	8.02±0.37	3–30
Coumatetralyl, 0.0375%	274±7.15	13.6±0.55	85±2.90	7.12±2.55	3–29
Control (Untreated)	287±8.65	-	0.00±0.00	-	-

Fig. 1. Hunting activity conducted near a local wet market lane in Kuantan, an area with a high infestation of Norway rats (*Rattus norvegicus*). The method of hunting involved the use of a fishing net, as shown in photo (A), to capture the rats. This activity was conducted at night (photo B), when the rats were most active. In addition to using fishing nets, live traps (28 cm [L]× 13 cm [W]× 13 cm [H]) were also employed in this study; however, they are not depicted in either photo

Discussion

To date, there is very limited information on the effectiveness of chlorophacinone, which focuses on *Rattus norvegicus*. Lund (20) reported that 20 house mice, *Mus musculus*, were fed oat groats mixed with 0.025% chlorophacinone, a concentration higher than that used in our present study, for one day, recording 60% mortality with the lethal dosage in the range of 23–65 mg/kg. This mortality rate was far lower yet associated with a higher lethal dosage than our current finding, which utilized a lower concentration of 0.005 %. Based on the published results, after 10 and 21 days of feeding, chlorophacinone still failed to achieve complete control over *M. musculus*, indicating that the mice were suspected of being more tolerant to chlorophacinone compared to our findings with *R. norvegicus*. The author suggested that a much higher dosage needed to be consumed by the mice, about 58 to 1168 mg/kg, to match the 95% mortality rates observed in the rats in our study, which only required an average consumption of 3.5 mg/kg of the toxicant.

Another set of laboratory trials on the efficacy of chlorophacinone against common rodents in Hawaii, namely the house rat, *Rattus rattus*, and *M. musculus*, was conducted by Pitt et al. (27). The authors reported that only 50% mortality was achieved for both species, *R. rattus* (n= 10) and *M. musculus* (n= 10), in a three-day choice feeding test. Mean bait consumption of *M. musculus* was 8.6 g, and *R. rattus* was 27.5 g. Complete control was only achieved after both species underwent another set of trials with an extended period of multiple feedings over seven days. The mice and the rats had consumed a higher amount of bait at 11.6 g and 69.1 g compared to the first trial. In contrast to Lund (20), *M. musculus* was found to be more susceptible to lower concentrations of chlorophacinone, according to the authors, based on the number of feeding days and mortality rates produced. However, Pitt et al. (27) did not provide dosage-consumption data for this trial, making direct comparisons challenging. Nonetheless, tolerance levels of both rodent species can be inferred through the feeding days and their corresponding mortality rates.

The efficacy of chlorophacinone has also been documented in another species of rat, the wood rat (*R. tiomanicus*), in the latest publication by Ariff Ateed et al. (13). In comparison to our present study, the authors conducted a multiple-feeding test on *R. tiomanicus* (n= 40) using 0.005% chlorophacinone as a treatment for six days. The published results indicated that the treatment did not achieve complete control over the rats; however, it still recorded a high mortality rate of 74.20%. The mean bait consumption was recorded at 5.43 g/day, or when calculated for the extensive six days of feeding, it amounted to approximately 32 to 33 g. However, similar to Pitt et al. (27), the authors did not provide dosage-consumption data for the trial, making direct comparisons impossible. Nevertheless, to infer the tolerance of *R. tiomanicus*, it is noted that the mortality recorded by the authors for *R. tiomanicus* was not as high as that observed for *R. norvegicus* in our study, despite being fed for six consecutive days with the same concentration of chlorophacinone as in the present study. Based on a comparison of the findings regarding the efficacy of chlorophacinone in *R. norvegicus* from our present study with those for other rodent species reported in past literature, such as *M. musculus* (20), *R. rattus* and *M. musculus* (27), and *R. tiomanicus* (13), it is suggested that *R. norvegicus* is more vulnerable to chlorophacinone than the other three species of rodents, making chlorophacinone more effective for controlling *R. norvegicus*.

The higher susceptibility of *R. norvegicus* to chlorophacinone compared to other species may be attributed to differences in LD_{50} values. Specifically, the LD_{50} for chlorophacinone in *R. norvegicus* is lower than in other species such as *M. musculus*. The only direct comparison available is from Ashton et al. (28), who reported an LD_{50} of 0.8 mg a.i./kg-bw (body weight) for *R. norvegicus*, in contrast to 5.95 mg a.i./kg-bw for *M. musculus*. More recent data from the Danish EPA (29) and ECHA (30) indicate LD_{50} values of 3.15 mg/kg (male) and 10.95 mg/kg (female) in *R. norvegicus*. However, the lack of recent LD_{50} data for *M. musculus* and other species complicates direct comparisons. The observed higher susceptibility of *R. norvegicus* to chlorophacinone may also relate to species-specific metabolic capabilities. Supporting this, Witmer et al. (31) demonstrated greater accumulation of chlorophacinone in *R. norvegicus* compared to other species, suggesting a potential deficiency in metabolic processes that detoxify or synthesize the toxicant in *R. norvegicus* relative to other species.

Additionally, a comparative analysis between the efficacy of chlorophacinone and coumatetralyl highlights the differential effects of these two treatments compared to other conventional rodenticides in the market on common rodent pests. As aforementioned in the above paragraph, according to toxicology reports by the Danish EPA (29) and ECHA (30), we can conclude that chlorophacinone possesses a lower LD_{50} value against rats compared to coumatetralyl, which stands at 30 mg/kg (male) and 15 mg/kg (female). This suggests that rats are theoretically more susceptible to chlorophacinone than to coumatetralyl. Consequently, coumatetralyl typically requires higher dosages to effectively control rodent pests, as observed in our current findings. Despite exhibiting higher dosage consumption than chlorophacinone, the mortality produced by coumatetralyl treatment was still lower than that achieved with chlorophacinone.

To support this finding, several past studies have yielded similar results. Notably, the study conducted by Abou El-Khear (32) provides insights into the necessity of higher dosages of coumatetralyl to control common rodent species such as *M. musculus* and *R. rattus*. According to the author's findings, the maximum dosage consumption of 0.0375% coumatetralyl required to achieve complete control over *M. musculus* and *R. rattus* ranged from 10.43 to 10.76 mg/kg and 27.30 to 28.24 mg/kg, respectively. In contrast, in the same trial, both species required much lower dosages of chlorophacinone to achieve the same result as with coumatetralyl. According to the author, the maximum dosage consumption of chlorophacinone recorded for *M. musculus* and *R. rattus* were only 1.43 to 1.49 mg/kg and 3.59 to 3.69 mg/kg, respectively.

However, according to Mikhail and Hasan (33), the maximum dosage required to completely control *R. rattus* was significantly higher, ranging from 72.6 to 80.2 mg/kg, contradicting the findings of Abou El-Khear (32). Additionally, a more recent study by Al-Salahy et al. (34) found that a dosage consumption of 54.54 mg/kg of 0.0375 % coumatetralyl was still insufficient to completely control *R. rattus* in Assiut Governorate, Egypt. This result further contradicts the findings of Abou El-Khear (32), suggesting discrepancies in the dosage requirements for different populations, environments, or periods of time.

Mikhail and Hasan (33) also stated that 50.0 to 55.6 mg/kg of the toxicant with the same concentration was required to completely control *R. norvegicus*, which was significantly higher than the dosage consumption observed in our present study on the same species, at 13.6 mg/kg. This suggests a potential reason why we did not achieve complete control over *R. norvegicus* in the present study. After comparing the past literature with our current findings, it is suggested that *R. norvegicus* is also susceptible to coumatetralyl compared to other common rodent pests such as *M. musculus* and *R. rattus*. However, it is also suggested that coumatetralyl needs to be administered in higher dosages than chlorophacinone to control *R. norvegicus* effectively.

Although comparisons were made between our current findings and those reported in previous studies, direct comparisons remain challenging. This is due to several confounding factors, including differences in the rodent species studied. Different species originate from distinct populations and environments and may possess varying metabolic capacities, all of which can influence the efficacy of chlorophacinone. Variability in the dosages used further complicates the interpretation, as different concentrations may exhibit differing levels of potency. Additionally, discrepancies in experimental design−ranging from single-day feeding protocols to multiple or extended feeding periods−limit the ability to make meaningful comparisons. These differences often reflect varying research objectives among studies.

Despite these limitations, our study acknowledges the constraint of employing a single experimental approach, specifically the feeding test. We recognize the need to incorporate a broader range of methodologies, such as multiple-feeding trials, to more accurately determine the lethal dosages of chlorophacinone and coumatetralyl in *Rattus norvegicus*.

The findings of this study have important implications for urban rodent control strategies in Malaysia. Based on our survey, options for first-generation anticoagulant rodenticides (FGARs) in the Malaysian urban pest control market are currently limited. Coumatetralyl appears to be the most widely used FGAR in urban environments. However, the repeated use of a single rodenticide poses a risk of resistance development within rat populations. Such resistance may necessitate the use of more potent rodenticides, thereby increasing the risk of environmental contamination as well as primary and secondary poisoning of non-target species.

Conclusion

Chlorophacinone, despite being formulated at a lower dosage compared to coumatetralyl, demonstrated a high mortality rate of 95%, indicating both the potency of the treatment and the susceptibility of the Norway rat (*R. norvegicus*), a common urban pest in Malaysia. The high efficacy of chlorophacinone at low dosage makes it not only a potentially cost-effective solution but also a more environmentally friendly option for urban rat control. Incorporating chlorophacinone into urban rat management in Malaysia could reduce reliance on the limited options of first-generation anticoagulant rodenticides (FGARs) or highly toxic rodenticides in general. However, several research directions remain to be explored. These include laboratory studies to determine the optimal effective dosage of chlorophacinone for urban rats in Malaysia, as well as field studies to evaluate its real-world efficacy. Additional gaps that warrant investigation include the post-treatment effects on both target (rats) and non-target animal populations. This includes resistance monitoring and environmental impact assessments to better understand the long-term consequences of chlorophacinone use in urban ecosystems.

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Ethical considerations

The study was conducted based on the procedure set by Malaysian Standard MS1256: 2010 −Household pesticide products −Rat bait −Specification (First Revision)

Conflict of Interest Statement

The authors declare there is no conflict of interest.

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