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Effectiveness of three pesticides against carmine spider mite (*Tetranychus cinnabarinus* Boisduval) eggs on tomato in Botswana

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The carmine spider mite (CSM; *Tetranychus cinnabarinus* Bois.) is one of the most destructive pests of vegetables, especially tomatoes. Its management in Botswana has, for years, relied on the use of pesticides. This study evaluated the efficacy of abamectin, methomyl and chlorfenapyr against CSM eggs under laboratory conditions in Botswana. Each treatment was replicated three times. The toxic effect was evaluated in the laboratory bioassay after 24, 48 and 72 h of application of pesticides. This study revealed that chlorfenapyr was relatively more effective since it had lower LD_{50} values than those for abamectin and methomyl. It was further revealed that at recommended rates, 90% mortalities occurred 48 h after application of methomyl and chlorfenapyr, while abamectin did not achieve 90% mortality throughout the study period. This implies that abamectin requires extra dosages to achieve mortalities comparable to those of the other two pesticides. The study has found that chlorfenapyr was the most effective insecticide followed by methomyl and then abamectin when applied on CSM eggs. Further research and field testing is necessary to confirm these laboratory findings.

Key words: Abamectin, methomyl, chlorfenapyr, effectiveness, carmine spider mite, tomato.

INTRODUCTION

The carmine spider mite *Tetranychus cinnabarinus* (Boisduval, 1867) is one of the most destructive pests of crops and vegetables in the world. It has a wide host plant range (Sokeli et al., 2007) including cultivated crops such as tomatoes, pepper, cucurbits, maize, soy, tobacco, cotton, beans, eggplant, and many others (Bu et al., 2015). It occurs mostly where tomatoes are grown causing great economic impact (Mwandila et al., 2013). CSM are a parenchyma cell feeding pest where the adults and nymphs suck sap especially from the mature and older leaves. Heavy infestations cause leaf drop thereby significantly reducing yields, causing severe economic losses (Xu et al., 2014). Its exceptional pest status is due to the diversity and abundance of host plants, its high reproductive potential and its genetic elasticity that leads to a rapid development of resistance to insecticides (He et al., 2009). It has been demonstrated in other parts of the world that CSM quickly develops resistance to many new insecticides employed for its control (Sato et al., 2005; Stumpf and Nauen, 2001).

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Control failures have been reported in several countries for compounds such as organophosphates, dicofol, organotins, hexythiazox, clofentezine, fenpyroximate and abamectin (Kim et al., 2004; Nauen et al., 2001; Sato et al., 2005; Van Leeuwen et al., 2005). This presents a serious threat to its effective management. Unfortunately, in Botswana, control of CSM is heavily dependent on these conventional synthetic pesticides. Abamectin and Chlorfenapyr are among the most widely used pesticides in the control of arthropod pests of vegetables in Botswana (Obopile et al., 2008) while Methomyl has been used worldwide as an acaricide for the control of insects and mite pests of vegetables (Tomlin, 2000).

Abamectin (Mode of Action Group 6 - Glutamate-gated chloride channel (GluCl) allosteric modulator) (IRAC, 2018) is a macrolytic lactone derived from the soil bacterium *Streptomyces avertimilis* (Kim and Goodfellow, 2002). It is more effective as an ingestion toxicant, but also has some contact activity. It acts on the γ-aminobutyric acid (GABA) gated chlorine channels, glutamate-gated chlorine channels leading to paralysis and death of the pest (Sato et al., 2005). It has been shown to be active against both immature and adult spider mites (Ismail et al., 2007). The widespread and frequent use exerts heavy selection pressure on the pest population, which results in the development of pest resistance (Sato et al., 2005). There are several reports of abamectin resistance in spider mites in a number of strains and field populations worldwide (Stumpf and Nauen, 2002). Methomyl, S-methyl (EZ)-N-(methylcarbamoyloxy) thioacetimidate is an oxime carbamate insecticide used as an acaricide to control ticks and spiders. It is also used for foliar treatment of vegetables, fruits and field crops, as well as cotton and commercial ornamentals (Gaete et al., 2013). Methomyl is effective in two ways; contact because it kills target insects upon direct contact and also as a systemic because of its capacity to cause overall systemic poisoning (Aktar et al., 2010) and acts as an acetylcholinesterase inhibitor. Chlorfenapyr, 4-bromo-2-(4-chlorpheny)-1-ethoxymethyl-5 trifluoro methyl-pyrrole-3-carbonitrile, is a broad-spectrum insecticide/acaricide used for control of various species of insects and mites. It is a chitin synthesis inhibitor used against pests in cotton, vegetables, citrus and soy (Tomlin, 2000). It is an *N*-substituted halogenated pyrrole derived from the natural product dioxapyrrolomycin (Hunt and Treacy, 1998). Once the formed metabolite has inhibited oxidative phosphorylation by disrupting the proton gradient across mitochondrial membranes, the ability of cells to produce ATP and ADP is affected, ultimately resulting in cell death and death of the organism (Rahman et al., 2012; Rand, 2004).

The efficacy of these three pesticides has not been evaluated on CSM populations in Botswana despite the fact that they have been used to control the pest for over a decade. Determination of relative effectiveness of pesticides used in the control of CSM is a crucial element for development of suitable and affordable strategies for its effective management. This study evaluated the efficacy of abamectin, methomyl and chlorfenapyr against CSM eggs under laboratory conditions in Botswana.

MATERIALS AND METHODS

The bioassay was conducted at the Botswana University of Agriculture and Natural Resources (BUAN) in Gaborone, Botswana (24°35'29.04" S, 25°56'29.40" E; altitude: 998 m) in the Crop Protection laboratory, at an average temperature of 25 ± 3°C. CSM population used in this study was obtained from a commercial tomato producing farm in Metsimotlhabe just outside Gaborone and identified in the Crop Protection laboratory at BUAN before use in the bioassay. Tomato seedlings (var. Rodade) were raised in the greenhouse in nursery trays and transplanted into small black plastic sleeve pots filled with 1.5 kg loam soil; each pot was 12 cm in diameter and 15 cm in depth. Tomato seedlings were used to rear the CSM and ensure adequate host substrate for oviposition of eggs by adults. The seedlings were watered regularly *ad-lib* to prevent wilting.

Bioassay methods

Three commercially available pesticides, Abamectin (Agromectin®) Arysta LifeScience, Methomyl (Spitfire® 900SP) Bitrad Consulting (PTY) LTD and Chlorfenapyr (Savage 360® SC) registered for use in Botswana, were used in the laboratory bioassay. The method followed the IRAC method 003 (for eggs) (IRAC, 2009). Each pesticide was applied at 5 concentrations separated on a $\log_{10}$ scale, with the recommended rates (0.6 ml/L for abamectin; 0.5 g/L for methomyl; and 0.4 ml/L for chlorfenapyr) included as a check and distilled water was included as a control and as the solvent used to formulate test solutions. Abamectin was applied at 0.4, 0.5, 0.6, 0.7 and 0.8 ml/L; methomyl: 0.3, 0.4, 0.5, 0.6 and 0.7 g/L; chlorfenapyr: 0.2, 0.3, 0.4, 0.5 and 0.6 ml/L water. The six treatments were arranged in a completely randomized design. Leaf discs of 2 cm diameter were cut from chemically untreated tomato leaves using a 2 cm diameter aluminium pipe. Each treatment had nine leaf discs. The test liquids were agitated and each leaf disc was individually dipped in one of the test liquids for 5 s. The surface liquid was allowed to dry from the leaves before placing them in polystyrene cups with a layer of moist cotton wool placed over the base of the cups and tap water added to a point of saturation. A fine brush was used to transfer CSM eggs (not older than 48 h) onto each treated leaf disc. Eggs (10 - 20) were placed on each treated leaf disc. This gave a total of 54 treated leaf discs per bioassay and 162 treated leaf discs all together. The tests were maintained at 25 ± 3°C and at 65 to 90% relative humidity. Each cup had a label which indicated the treatment and its date of application. The bioassay was repeated 3 times.

Assessment of mortality

Each leaf disc was observed daily (24, 48 and 72 h after treatment) under a binocular microscope until complete (or nearly complete) hatching on the control leaf discs (water only) was observed. The number of un-hatched eggs on treated and untreated leaf sections was recorded. Results were expressed as percentage mortality and corrected for untreated mortality using Abbott's formula (Abbott, 1925). Untreated mortality was also recorded.
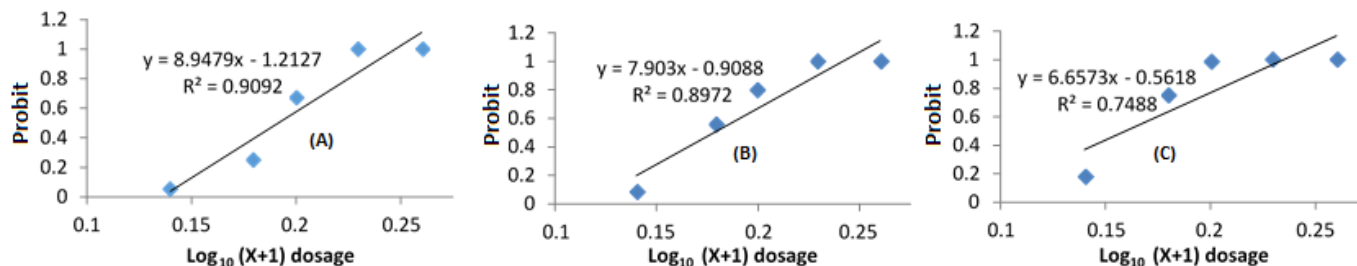
Figure 1. Probit mortality of CSM eggs exposed to different doses of abamectin assessed 24 h(A), 48 h(B) and 72 h(C) following application.
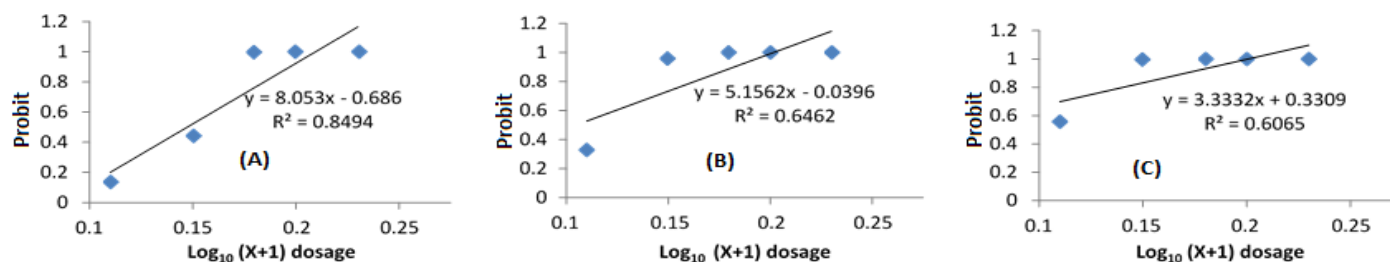

Figure 2. Probit mortality of CSM eggs exposed to different doses of methomyl assessed 24 h(A), 48 h(B) and 72 h(C) following application.

Data analysis

Probit analysis (Finney, 1971) was used to analyse mortality results. The mortality data were transformed to probits while the dosages were transformed to $\log_{10}$ (X+1) before analysis. Data was analysed using $\log_{10}$ of dosage versus probit mortality regression and analysis of variance (ANOVA). LD_{50} and LD_{90} values were estimated from the probit lines. Relative susceptibilities of eggs were compared using LD_{50} values and slopes of probit lines. LD_{90} values were used to compare the mortality from the recommended dosage with the mortalities achieved by the treatments following different durations of exposure to the pesticides. Averages were separated using the Tukey's Honestly significant difference test (Zar, 1984).

RESULTS

CSM egg mortality due to abamectin

Figure 1 (A-C) shows the probit mortality of CSM eggs exposed to different dosages of abamectin assessed at 24 (A), 48 (B) and 72 h (C) after treatment. The figures show transformed dose and mortality data. These figures reveal a positive linear relationship between log dose and probit mortality caused by abamectin (correlation coefficients of 0.91, 0.90 and 0.75, respectively). Figure 1A shows that LD_{50} of 0.55 ml/L and LD_{90} of 0.72 ml/L were achieved 24 h after application. The recommended dose (0.60 ml/L), abamectin showed a probit value of 0.58 (equivalent to 49.60% egg mortality) during this period. Figure 1B indicates that the LD_{50} of abamectin

after 48 h exposure was 0.51 ml/L, while the LD_{90} was 0.69 ml/L. At the recommended dose (0.60 ml/L), abamectin achieved 0.67 on the probit scale, which is equivalent to 54.94% egg mortality. When assessed at 72 h after application, the LD_{50} of abamectin was 0.44 ml/L and the LD_{90} was 0.66 ml/L (Figure 1C). The recommended dosage (0.60 ml/L) of abamectin achieved 0.77 on the probit scale, which is equivalent to 61.34% egg mortality after 72 h exposure.

CSM egg mortality due to methomyl

The probit mortality of CSM eggs exposed to different dosages of methomyl assessed 24 (A), 48 (B) and 72 h (C) after treatment. These figures reveal a positive linear relationship between log dose and probit mortality caused by methomyl (correlation coefficients of 0.85, 0.65 and 0.61, respectively) (Figure 2A-C) during the assessment periods. Figure 2A shows that LD_{50} of 0.40 g/L and LD_{90} of 0.57 g/L were achieved 24 h after application. The recommended dose (0.50 g/L) of the pesticide showed a probit value of 0.76 (equivalent to 60.67% egg mortality) during this exposure period. Figure 2B indicates that the LD_{50} of methomyl at the 48 h assessment period was 0.27 g/L, while the LD_{90} was 0.52 g/L. At the recommended dose, methomyl achieved 0.89 on the probit scale, which is equivalent to 70.63% egg mortality. When assessed at 72 h, the LD_{50} of methomyl was 0.12 g/L and the LD_{90} was 0.48 g/L (Figure 2A-C). The
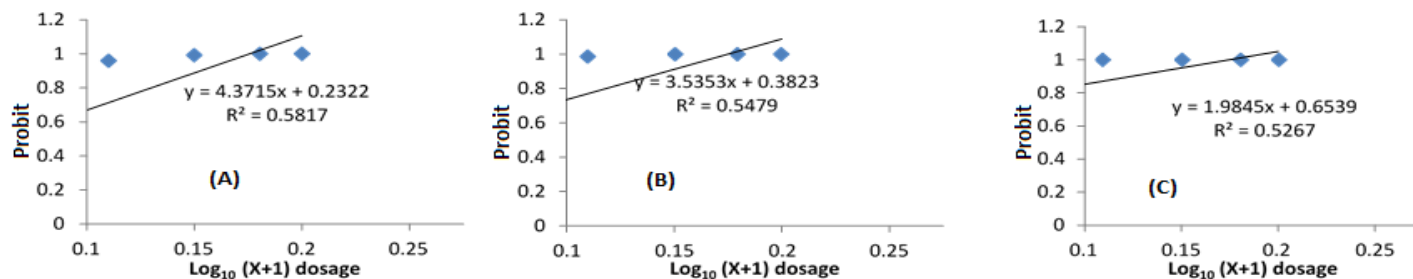
Figure 3. Probit mortality of CSM eggs exposed to different doses of chlorfenapyr assessed 24 h(A), 48 h(B) and 72 h(C) following application.

Table 1. The effect of abamectin concentrations and period of exposure on CSM egg mortality.

Period of application	Means ± SE					
	Control	**0.40 ml/L**	**0.5 ml/L**	**0.6 ml/L**	**0.7 ml/L**	**0.8 ml/L**
24 h	5.00[dB] ± 2.89	13.33[dcB] ± 1.67	30.00[cC] ± 2.89	55.00[bB] ± 7.64	96.67[aA] ± 3.33	100.00[aA] ± 0.00
48 h	11.67[dAB] ± 6.01	16.67[dB] ± 3.33	48.33[cB] ± 1.67	63.33[bAB] ± 3.33	100.00[aA] ± 0.00	100.00[aA] ± 0.00
72 h	18.33[dA] ± 6.67	25.00[dA] ± 2.89	60.00[cA] ± 2.89	83.33[bA] ± 3.33	100.00[aA] ± 0.00	100.00[aA] ± 0.00

**Means followed by the same small letter within a row are not significantly different, P≤0.05 (Tukey's Honestly significant difference test). **Means followed by the same capital letter within a column are not significantly different, P≤0.05 (Tukey's Honestly significant difference test).

recommended dosage achieved 0.93 on the probit scale, which is equivalent to 74.66% egg mortality at the 72 h period.

CSM egg mortality due to chlorfenapyr

Figure 3A to C shows probit mortality of CSM eggs exposed to different dosages of chlorfenapyr assessed 24 (A), 48 (B) and 72 h (C) after treatment. This figure revealed positive linear relationship between log dose and probit mortality caused by chlorfenapyr (correlation coefficients of 0.58, 0.5579 and 0.5367), when treatments were assessed at 24, 48 and 72 after treatment. Figure 3A shows that LD_{50} of 0.15 ml/L and LD_{90} of 0.42 ml/L were achieved 24 h after application. The recommended dose (0.40 ml/L) of the pesticide showed a probit value of 0.89 (equivalent to 70.63% egg mortality) during this assessment period. Figure 3B indicates that the LD_{50} of chlorfenapyr after 48 h exposure was 0.08 ml/L, while the LD_{90} was 0.40 ml/L. At the recommended dose, chlorfenapyr achieved 0.91 on the probit scale, which is equivalent to 72.54% mortality. When assessed at 72 h after application, the LD_{90} was 0.33 ml/l (Figure 3A-C). The recommended dosage achieved 0.95 on the probit scale, which is equivalent to 77.08% egg mortality during the 72 h assessment period.

The effect of abamectin concentrations and period of exposure on CSM egg mortality

Table 1 shows the effect of abamectin concentrations

and period of exposure on CSM egg mortality. The results of this study revealed that concentration and period of exposure interactions were significantly different ($F_{10, 34} = 4.77$; P=0.0003). When comparing the different concentrations at 24 h period of exposure it has been shown that control was significantly different from all the other concentrations except 0.40 ml/L. The recommended rate of 0.6 ml/L only achieved 55% mortality during the 24 h period, which is less than the required 90% mortality. Concentrations of 0.7 and 0.8 ml/L were not significantly different in egg mortality at 24 h period (Table 1). These two concentrations had more than 90% egg mortality and this was significantly higher than the mortality achieved at the recommended rate of 0.6 ml/L. When the concentrations were compared at 48 h period of exposure, control mortality was significantly different from all the other concentrations except 0.40 ml/L. The recommended rate of 0.6 ml/L achieved 63.33% mortality, which was less than the required 90% egg mortality during the 48 h period. Concentrations above the recommended rate of 0.6 ml/L (0.7 and 0.8 ml/L) achieved mortalities of 100% during the 48 h period, and these were not significantly different (Table 1). When assessment was done following 72 h exposure, control mortality (18.33%) was not significantly different from the 25% mortality achieved at 0.40 m/L. The recommended rate of 0.6 ml/L achieved 83.33% CSM egg mortality during the 72 h period, which is less than the required 90% mortality. Concentrations of 0.7 and 0.8 ml/L achieved 100% egg mortality, and these were not significantly different (Table 1).When comparing the concentrations at different periods of exposure it has

Table 2. The effect of methomyl concentrations and period of exposure on CSM egg mortality.

Period of application (h)	Means ± SE					
	Control	0.30 g/L	0.40 g/L	0.50 g/L	0.60 g/L	0.70 g/L
24	$5.00^{dB} \pm 2.89$	$21.67^{cC} \pm 1.67$	$41.67^{bB} \pm 3.33$	$86.67^{aA} \pm 7.26$	$100.00^{aA} \pm 0.00$	$100.00^{aA} \pm 0.00$
48	$15.00^{dAB} \pm 0.00$	$35.00^{cB} \pm 2.89$	$78.33^{bA} \pm 3.33$	$96.67^{aA} \pm 3.33$	$100.00^{aA} \pm 0.00$	$100.00^{aA} \pm 0.00$
72	$20.00^{dA} \pm 2.89$	$48.33^{cA} \pm 1.67$	$86.67^{bA} \pm 2.89$	$96.67^{aA} \pm 3.33$	$100.00^{aA} \pm 0.00$	$100.00^{aA} \pm 0.00$

**Means followed by the same small letter within a row are not significantly different, P≤0.05 (Tukey's Honestly significant difference test). **Means followed by the same capital letter within a column are not significantly different, P≤0.05 (Tukey's Honestly significant difference test).

been shown that mortalities achieved by the control were not significantly different from each other ($F_{10, 34} = 4.77$; P=0.0003). When comparisons were done at the concentration rate of 0.4 ml/L, the mortality of 13.33% achieved at 24 h exposure was not significantly different from mortalities achieved at 48 h (16.67%) and 72 h (25%) (Table 1). When comparing the recommended rate of abamectin (0.6 ml/L) at different periods of exposure, it has been shown that the mortality of 55% achieved at 24 h exposure was not significantly different from mortalities achieved at 48 h (63.33%) and 72 h (83.33%) exposure periods. These were less than the required 90% egg mortality. The concentration of 0.7 ml/L was able to cause 96.67% mortality at the 24 h period which was not significantly different ($F_{10, 34} = 4.77$; P=0.0003) from the 100% egg mortalities achieved at the 48 and 72 h periods of exposure (Table 1).

The effect of methomyl concentrations and period of exposure on CSM egg mortality

Table 2 shows the effect of methomyl concentrations and period of exposure on CSM egg mortality. The results of this study revealed that concentration and period of exposure interactions were significantly different ($F_{10, 34}=14.68$; P=0.0001). When comparing the different concentrations at 24 h period of exposure it has been shown that control mortality (15.00%) was significantly different from all the other concentrations (Table 2). The recommended rate of 0.50 g/L achieved 86.67% egg mortality during the 24 h period, which is not significantly different ($F_{10, 34} = 14.68$; P=0.0001) from the 100% mortalities achieved by 0.60 and 0.70 g/L concentrations. When the concentrations were compared at 48 h period of exposure, control mortality was significantly different from all the other concentrations ($F_{10, 34} = 14.68$; P=0.0001). The recommended rate of 0.50 g/L achieved 96.67% egg mortality, which was not significantly different from the 100% egg mortalities achieved by the 0.6 and 0.7 g/L during the 48 h assessment period (Table 2). When assessment was done following 72 h exposure, control mortality (20.00%) was significantly different from all the other concentrations ($F_{10, 34} = 14.68$; P=0.0001). The recommended rate of 0.50 g/L achieved 96.67%

CSM egg mortality during the 72 h period, which is similar to mortalities achieved by the two higher concentrations of 0.6 and 0.7 g/L. Concentrations of 0.7 and 0.8 ml/L achieved 100% egg mortality, and these were not significantly different (Table 2).

When comparing the concentrations at different periods of exposure it has been shown that mortality achieved by the control at 24 h was not significantly different ($F_{10, 34} = 14.68$; P=0.0001) from the mortality achieved after 48 h (Table 2). However, mortality achieved by the control at 24 h period were not similar to mortality after 72 h ($F_{10, 34} = 14.68$; P=0.0001). When comparisons were done at the concentration rate of 0.30 g/L, the mortality of 21.67% achieved at 24 h exposure was not significantly different from mortalities achieved at 48 h (35.00%) and 72 h (48.33%) (Table 2). When comparing the recommended rate of methomyl (0.50 g/L) at different periods of exposure, it has been shown that the mortality of 86.67% achieved at 24 h exposure was not significantly different from 96.67% mortalities achieved at 48 and 72 h exposure periods. The concentration of 0.60 g/L was able to cause 100% mortality at the 24 h period which was not significantly different ($F_{10, 34} = 14.68$; P=0.0001) from the 100% egg mortalities achieved at the 48 and 72 h periods of exposure (Table 2).

The effect of chlorfenapyr concentrations and period of exposure on CSM egg mortality

Table 3 shows the effect of chlorfenapyr concentrations and period of exposure on CSM egg mortality. The results of this study revealed that concentration and period of exposure interactions were significantly different ($F_{10, 34} = 1.84$; P=0.09). When comparing the different concentrations at 24 h period of exposure it has been shown that control mortality was significantly different from all the other concentrations. The recommended rate of 0.40 ml/L achieved 85.00% egg mortality during the 24 h period, which is not significantly different from the 93.33% mortality achieved at the higher concentration of 0.50 ml/L during the same period. Concentrations of 0.50 and 0.60 ml/L achieved 93.33 and 100% egg mortality during the 24 h assessment period ($F_{10, 34} = 1.84$; P=0.09) (Table 3). When the concentrations were compared at

Table 3. The effect of chlorfenapyr concentrations and period of exposure on CSM egg mortality.

Period of application	Means ± SE					
	Control	0.20 ml/L	0.30 ml/L	0.40 ml/L	0.50 ml/L	0.60 ml/L
24 h	11.67^{dB} ± 1.67	36.67^{cB} ± 3.33	78.33^{bA} ± 3.33	85.00^{abA} ± 5.00	93.33^{abA} ± 6.67	100.00^{aA} ± 0.00
48 h	11.67^{dB} ± 1.67	43.33^{CAB} ± 3.33	83.33^{bA} ± 4.41	93.33^{abA} ± 4.41	100.00^{aA} ± 0.00	100.00^{aA} ± 0.00
72 h	20.00^{cA} ± 2.89	56.67^{bA} ± 3.33	96.67^{aA} ± 3.33	96.67^{aA} ± 3.33	100.00^{aA} ± 0.00	100.00^{aA} ± 0.00

**Means followed by the same small letter within a row are not significantly different, P≤0.05 (Tukey's Honestly significant difference test). **Means followed by the same capital letter within a column are not significantly different, P≤0.05 (Tukey's Honestly significant difference test).

48 h period of exposure, control mortality was significantly different from all the other concentrations. The recommended rate of 0.40 ml/L achieved 93.33% egg mortality, which was not significantly different from the 100% egg mortality achieved by higher concentrations of 0.50 and 0.6 ml/L during the same period (Table 3). When assessment was done following 72 h exposure, control mortality was significantly different from mortalities achieved at all other concentrations. The recommended rate of 0.40 ml/L achieved 96.67% CSM egg mortality during the 72 h period, which is similar to 100% mortalities achieved at higher concentrations of 0.50 and 0.6 ml/L during the same period (Table 3).

When comparing the concentrations at different periods of exposure it has been revealed that mortalities achieved by the control treatment at 24 h were significantly different from mortality achieved at 72 h ($F_{10, 34}$ = 1.84; P=0.09). When comparisons were done at the concentration rate of 0.30 ml/L, the mortality of 78.33% achieved at 24 h exposure was not significantly different from mortalities achieved at 48 h (83.33%) (Table 3).

The concentration of 0.30 ml/L achieved 96.67% mortality at 72 h. When comparing the recommended rate of chlorfenapyr (0.40 ml/L) at different periods of exposure, it has been shown that the mortality of 85.00% achieved at 24 h exposure was not significantly different ($F_{10, 34}$ = 1.84; P=0.09) from mortalities achieved at 48 h (93.33%) and 72 h (96.67%) exposure periods. The concentration of 0.50 ml/L was able to cause 93.33% mortality at the 24 h period which was not significantly different ($F_{10, 34}$ = 1.84; P=0.09) from the 100% egg mortalities achieved at the 48 and 72 h periods of exposure (Table 3).

DISCUSSION

Toxicity of different abamectin dosages to CSM eggs

CSM eggs can only acquire the lethal dose of abamectin through contact with the pesticide material. Even though, generally, abamectin was effective on eggs, its effectiveness was affected by dosage. Higher concentrations, above recommended 0.60 ml/L of abamectin are required to achieve effective control on eggs under laboratory conditions in Botswana. This has been revealed when higher doses than the recommended dose of abamectin were required to achieve 90% (LD$_{90}$= 0.69 ml/L), 96.67 (0.70 ml/L) and 100% (0.80 ml/L) egg mortality in 24 h. In addition, the mortality level of 83.33% was achieved by the recommended dose during the 72 h study period (Figure 1A-C and Table 1). The slopes of the probit lines show that abamectin became more toxic to CSM eggs with each increase in pesticide concentration. Since the recommended dosage of abamectin only achieved 83.33% CSM egg mortality during the assessment period, it suggests that the eggs have lowered sensitivity to the pesticide. The explanation to this loss of sensitivity has to be explored further. Some researchers have stated that abamectin is effective on eggs when the embryo is full-grown, that is when the sickle-shaped mandibles of the newly formed larvae are visible through the chorion of treated eggs (Schuster and Everett, 1983). This implies that abamectin has little or no effect on the early stages of embryo development. Generally, abamectin at the recommended dosage required exposure periods longer than 72 h to cause 90 to 100% CSM egg mortality as demonstrated in this research. There are several reports on the effect of abamectin on eggs of spider mites. Ismail et al. (2007) reported that abamectin had no effect on eggs of the two-spotted spider mite (*Tetranychus urticae* Koch) at different dosages and Massoud et al. (2018) demonstrated that abamectin was highly effective against matured two-spotted spider mite on strawberry.

Toxicity of different methomyl dosages to CSM eggs

Ovicidal activity of methomyl has been demonstrated against eggs of several pest species such as cabbage-looper, *Trichoplusia ni* (Hübner, 1803) (Chalfant et al., 1979), tobacco budworm, *Heliothis virescens* Fabricius 1777 (Pitts and Peters, 1980), and budworm, *Helicoverpa punctiger* (Wallengren, 1860) (Wiate, 1981). However, since it does not have fumigant activity, the eggs have to be covered by the insecticide for it to be effective. Therefore, CSM eggs can only acquire the lethal dose through direct hits or contact with the pesticide material on the leaf surface. The recommended dose of methomyl

was able to cause 86.67% egg mortality in 24 h. In addition, the mortality level of 96.67% caused by the recommended dose (0.50 g/L) during the 48 and 72 h study period, appears to be sufficient to achieve effective control of CSM eggs (Figure 2A-C and Table 2). The slopes of the probit lines showed that methomyl became more toxic to CSM eggs with each increase in pesticide concentration. Similiar observations were recorded by Silva et al. (2018) who stated that methomyl, at the recommended doses, exhibited high toxicity against the eggs of *Neoleucinodes elegantalis* (Guenée, 1854) with higher than 80% of mortality. This insecticide has embryo toxic activity, causing malformations in neonates of *Daphnia obtusa* Kürz (1874) suggesting that it can be used to inhibit reproduction in spider mites therefore reducing outbreaks (Gaete et al., 2013). The high egg mortality achieved with methomyl means that buildup of adult populations from hatching eggs would be reduced, thereby minimizing subsequent damage to the host plants. Therefore, when using methomyl against CSM, the egg stage is a suitable stage to target.

Toxicity of different chlorfenapyr dosages to CSM eggs

Chlorfenapyr is a slow acting insecticide and belongs to the pyrrole group. The suggested mechanism for chlorfenapyr metabolism is conversion of the pro-insecticide chlorfenapyr to toxic form CL30328 by monooxygenases and this toxic form inhibits ATP synthesis in the mitochondria leading to inhibition of oxidative phosphorylation and resulting in the effect on CSM eggs (embryo in eggs) (Verma et al., 2015). The study showed that doses lower than the recommended dosage of chlorfenapyr achieved 90 to 100% egg mortality during the study period (Figure 3A-C and Table 3). This suggests that chlorfenapyr is highly effective against CSM eggs. Ullah and Gotoh (2013) reported that eggs of spider mites, *Tetranychus macfarlanei* and *Tetranychus truncatus* were highly susceptible to chlorfenapyr at LD_{50} level; therefore, the results were expected. CSM egg mortalities were probably due to direct hits or contact with the active ingredient on the leaf surface. When LD_{90}s are used alone to assess the effectiveness of chlorfenapyr, the mortality level caused by the recommended dose during the 72 h study period, appears to be sufficient to achieve effective control. This is in agreement with Amjad et al. (2012) where chlorfenapyr was highly effective against *T. urticae* compared to dicofol, fenpyroximate, azocyclotin, propergite and pyrabiden. However, LD_{90} values alone do not provide sufficient indication of the effectiveness of chlorfenapyr against CSM eggs. The slopes of the probit lines in Figure 3A-C shows that chlorfenapyr became more toxic to CSM eggs with each increase in pesticide concentration. These results are similar to those by Amjad et al. (2012) where mortality of *T. urticae* also

depended on the concentration of chlorfenapyr used. The action of chlorfenapyr against eggs is a desirable property since the buildup of pest populations from hatching eggs would be reduced thereby minimizing subsequent damage to host plants. When using chlorfenapyr against CSM, the egg stage would therefore be a suitable stage to target. This is a desirable property since it affects reproduction of the mites consequently preventing pest outbreaks. Chlorfenapyr has been shown to have little or no toxicity to beneficial insects therefore suitable for use in integrated pest management programs (IPM).

CONCLUSIONS AND RECOMMENDATIONS

In the present study, chlorfenapyr was the best ovicidal followed by methomyl and then abamectin when applied on CSM eggs using minimum amounts of active ingredient. Chlorfenapyr was able to achieve 90% mortality at 24 h whilst methomyl took 48 h at the recommended rates of 0.4 ml/L and 0.5 g/L, respectively. Chlorfenapyr was also able to achieve 90% mortality at a lower dosage than the recommended rate. Therefore, chlorfenapyr offers the most effective and timely control of CSM eggs. More so, since application of chlorfenapyr dosages that were lower than the recommended dose achieved effective control, they can be used without seriously affecting the level of protection of the crop particularly when applications target the egg stage. Abamectin did not achieve 90% mortality during the 72 h of the study period at the recommended rate of 0.6 ml/L. Abamectin was found to achieve effective control at dosages above the recommended rates. Therefore, higher dosages of abamectin would be required to achieve effective control of CSM eggs. It can be recommended that chlorfenapyr be used to target the eggs before they hatch in order to avoid any damage by subsequent damaging stages. According to the results of this research, the three products achieved effective control (90 - 100% mortality) in 24 h; abamectin with 0.72 ml/L, methomyl with 0.57 g/L, and chlorfenapyr with 0.42 ml/L. This means that the three products can be used in a CSM egg management rotation program and thereby avoid the selection of resistant CSM populations by using only one product. In addition, further research and field testing is necessary to confirm these laboratory findings.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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