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ORIGINAL CONTRIBUTION

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Evaluation of non-target effects of OMRI-listed insecticides for management of *Drosophila suzukii* Matsumura in berry crops

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Abstract

The spotted wing drosophila, *Drosophila suzukii* Matsumura, is an invasive pest of many fruit crops throughout North America, South America and Europe. The presence of this destructive pest has led to an increase in the number of insecticide applications. While conventional growers have an arsenal of different insecticides at their disposal, organic growers have a limited selection of effective options and rely heavily on applications of Entrust®, the organic formulation of spinosad. An important part of research is to develop more tools for organic growers and evaluate the effects of insecticides intended to target *D. suzukii* on natural enemies in the system. The effects of six organic pesticides alone and in combination with three adjuvants and two phagostimulants were tested in laboratory bioassays on three common natural enemies in berry production systems including two predators, *Chrysoperla rufilabris* and *Orius insidiosus*, and a parasitoid wasp, *Aphidius colemani*. Under the IOBC toxicity rating scale, spinosad was rated consistently from slightly harmful to harmful across natural enemy species and residue age (the effects of pesticides over time). Sabadilla alkaloids caused mortality to *O. insidiosus* equal to that of spinosad. All tested pesticides were at least slightly harmful to *A. colemani*, and the adjuvant polyether-polymethylsiloxane-copolymer polyether caused mortality that was not significantly different from spinosad. In general, neither the addition of adjuvants nor phagostimulants increased the mortality of the insecticides tested. The exception was polyether-polymethylsiloxane-copolymer polyether, but it is unclear whether it increased the toxicity of the pesticides or was simply toxic itself since it caused high mortality to *A. colemani* when applied alone. Sublethal effects were measured for two predatory species by measuring eggs laid and % egg hatch. Minimal sublethal effects were observed in *C. rufilabris*. In contrast, all tested insecticides caused reduced egg hatch in *O. insidiosus* compared with the control.

KEYWORDS

adjuvant, *Aphidius colemani*, *Chrysoperla rufilabris*, *Orius insidiosus*, phagostimulant, spotted wing drosophila

1 | INTRODUCTION

The spotted wing drosophila (SWD), *Drosophila suzukii* Matsumura (*Diptera*: *Drosophilidae*), is an invasive fruit fly and an economic threat to fruit industries throughout North America (Hauser, 2011; Lee, Bruck, Curry, et al., 2011a; Lee, Bruck, Dreves, et al., 2011b; Walsh et al., 2011), Europe (Calabria, Máca, Bächli, Serra, & Pascual, 2012; Cini, Ioriatti, & Anfora, 2012) and South America (Deprá, Poppe, Schmitz, De Toni, & Valente, 2014). Frequent insecticide applications are made to minimize *D. suzukii* infestations in berry crops (Sial et al., 2019; Van Timmeren & Isaacs, 2013). Extensive use of insecticides throughout the growing season can disrupt natural enemy populations, resulting in reduced fecundity, longevity and development rates of biological control organisms (Croft & Brown, 1975; Desneux, Decourtye, & Delpeuch, 2007). Furthermore, these changes in population dynamics and developmental rates can disrupt phenological synchrony between a natural enemy and its host or prey, resulting in pest resurgence and secondary pest outbreaks (Croft, 1990; Desneux et al., 2007). One strategy to reduce these unwanted effects is to use selective insecticides that control the target organism but have minimal effects on beneficial organisms (Moscardini et al., 2013).

There has been increasing availability of reduced-risk insecticides with more selectivity in fruit crops (Isaacs, Mason, Brewer, Noma, & 'Neal, 2006), especially in organic production (Sial et al., 2019). Reduced-risk insecticides have several advantages over broad-spectrum insecticides including shorter pre-harvest intervals due to their lower mammalian toxicity and greater compatibility with biological control tactics due to their less harmful effects on natural enemies (Atanassov, Shearer, & Hamilton, 2003; Liburd, Arevalo, & Rhodes, 2017; Roubos, Rodriguez-Saona, & Isaacs, 2014a). While many selective reduced-risk insecticides may indicate a low risk to beneficial insects, this is not always the case (Biondi, Desneux, Siscaro, & Zappalà, 2012). For example, Biondi, Zappalà, Stark, and Desneux (2013) demonstrated that spinosad has strong acute toxicity on parasitoids, which die rapidly when exposed to residues, even 10-d old residues, under greenhouse conditions.

Spinosad has been shown to be the most effective organically approved insecticide for *D. suzukii* management (Beers, Steenwyk, Shearer, Coates, & Grant, 2011; Bruck et al., 2011; Sial et al., 2019; Van Timmeren & Isaacs, 2013), and in some cases, the effectiveness of other selective organic insecticides may not be enough to meet the challenges associated with this aggressive pest (Cini et al., 2012). Previous research has indicated that azadirachtin + pyrethrins, *Chromobacterium subtsugae*, and sabadilla alkaloids cause some mortality in *D. suzukii* populations and could be useful in rotations with spinosad. The addition of both adjuvants (Roubos et al., 2019a) and phagostimulants (Roubos et al., 2019b) has been proposed to increase the efficacy of these organic insecticides. Roubos et al. (2019a) found that all three adjuvants tested, which included alcohol ethoxylate, poly-1-p-menthene and polyether-polymethylsiloxane-copolymer polyether (PEPMS), increased the mortality of some insecticides against *D. suzukii* populations and that both alcohol ethoxylate and poly-1-p-menthene caused some mortality when used alone. In contrast, adding sucrose or sucrose + yeast at a concentration of 0.36% to selected organic insecticides did not increase the mortality of *D. suzukii* (Roubos et al., 2019b). Therefore, it is important to determine what effects both the addition of adjuvants and phagostimulants might have on natural enemy populations.

Selective insecticides used in combination with biological control agents may be a more sustainable integrated pest management (IPM) programme than either approach alone (Gentz, Murdoch, & King, 2010). In addition to managing insect pests, combining chemical and biological control tactics are also expected to help minimize selection for resistance (Gentz et al., 2010). Insecticides are necessary as a short-term solution to limit extensive economic damage by *D. suzukii*. However, the development of biological and cultural controls (Rendon et al., 2019) will contribute to the long-term and sustainable management of this invasive pest. Therefore, an accurate evaluation of the potential side effects of insecticides on biological control agents is critical for developing effective IPM strategies (Desneux, Denoyelle, & Kaiser, 2006; Stark, Vargas, & Banks, 2007).

This study evaluated three important biological control agents that are commonly encountered in berry cropping systems and are used extensively in biological control programmes worldwide, including the green lacewing, *Chrysoperla rufilabris* (Burmeister) (Neuroptera: Chrysopidae); the predatory bug, *Orius insidiosus* (Say) (*Hemiptera*: *Anthocoridae*); and the aphid parasitoid, *Aphidius colemani* Viereck (*Hymenoptera*: *Braconidae*). The larvae of *C. rufilabris* are generalist predators that feed on aphids and whiteflies (Dean & Schuster, 1995), which are important pests of many crops. *Orius insidiosus* is an important predator of flower thrips (Liburd et al., 2017) and aphids (Walton & Isaacs, 2011) in blueberries. *Aphidius colemani* is a commercially available aphid parasitoid, one of several parasitoids important in regulating aphid populations in blueberry fields (Walton & Isaacs, 2011).

Although several studies have evaluated the toxicity of conventional and reduced-risk insecticides to these common predators (Barbosa, Oliveira, Barros, Michaud, & Torres, 2018; Garzón, Medina, Amor, Viñuela, & Budia, 2015; Liburd et al., 2017; Moscardini et al., 2013), only few studies have evaluated the non-target effects from organic insecticides. The goal of this present study was to determine the relative risk of different OMRI-listed (organic) insecticides used in organic berry production to natural enemies. Our objectives were to (a) evaluate the relative toxicities of selected insecticides alone, in combination with adjuvants and in combination with phagostimulants to natural enemies, (b) compare residual effects of these insecticides alone, in combination with adjuvants and in combination with phagostimulants and (c) evaluate the sublethal effects of all treatments on predaceous natural enemies.

2 | MATERIALS AND METHODS

2.1 | Insects

Three commercially available natural enemy species were used in the bioassays, including *Aphidius colemani* tested at Michigan

State University, the green lacewing, *Chrysoperla rufilabris* at the University of Georgia and the insidious flower bug, *Orius insidiosus* at the University of Florida. *Aphidius colemani* and *O. insidiosus* were purchased from Koppert Biological Systems, (Howell, MI). *Chrysoperla rufilabris* were purchased from Rincon-Vitova Insectaries, Inc. (Ventura, CA). These species are commonly observed natural enemies in blueberry production systems that represent a range of feeding types. Fresh shipments were used for each residue age (0, 3, and 7 days residuals), and insects were used within 5 days of being received. *Aphidius colemani* were placed in 32 oz plastic containers at 20°C and 75% RH with 16:8 L:D cycle. Each container was provided with a strip of filter paper with a 10% honey solution. *Chrysoperla rufilabris* adults were maintained on a laboratory bench (27°C, 50% RH, 14:10 L:D cycle) in the cardboard tubes in which they were shipped from the supplier. Tubes contained moistened sponges to prevent desiccation of the adults. *Orius insidiosus* was maintained in rearing containers under controlled environment at 25°C, 70% RH, 14:10 L:D. *Orius insidiosus* was provided with deionized water and 10% honey water solution on a diet of *Ephestia kuehniella* Zeller (*Lepidoptera*: *Pyralidae*) eggs. Insects were briefly chilled, to facilitate transfer, before being transferred to test arenas.

2.2 | Laboratory experiment

Insecticides, manufacturers and rates used in this experiment are listed in Table 1. A total of 28 treatments were evaluated in a completely randomized design with five replicates per treatment. Treatments included six OMRI-listed pesticides alone, in combination with an adjuvant and in combination with phagostimulants. The six pesticides used were spinosad, *C. subtsugae*, *Burkholderia* spp., azadirachtin + pyrethrins, hydrogen peroxide + peroxyacetic acid (PAA) and sabadilla alkaloids. The three adjuvants that were tested included polyether-polymethylsiloxane-copolymer polyether (PEPMS), poly-1-p-menthene (P1M) and alcohol ethoxylate (AE). Polyether-polymethylsiloxane-copolymer polyether is a silicone surfactant, P1M is a spreader-sticker-deposition aid and AE is a spreader/penetrant. Each natural enemy species was tested using a single adjuvant, such that PEPMS was tested on *A. colemani*, P1M was tested on *C. rufilabris*, and AE was tested on *O. insidiosus*. The two phagostimulants were sucrose and sucrose + yeast. In addition to these treatments, an untreated control (deionized water) was also included.

Laboratory assays followed the methods described in Roubos, Rodriguez-Saona, Holdcraft, Mason, and Isaacs (2014b). Treatments were applied to the bottom of a 47-mm-diameter plastic Petri dishes (Fisher) using a Potter precision spray tower (Burkard Scientific). Treatment solutions were dispensed at a total volume of 2 ml at 15 psi with an output equivalent to 468 litres/ha (50 gallons/acre) spray volume. Residues were dried under fume hood for 2 hr before releasing any biological control agents. Biological control agents were exposed to fresh (0 day) residues, and residues aged for 3 and 7 days under greenhouse conditions (23 ± 2°C, 70% RH). After residues were aged, adult insects (mix of females and males) were added to each Petri dish (10 *A. colemani*, 5 *C. rufilabris*, or 5 *O. insidiosus*). *Chrysoperla rufilabris* and *O. insidiosus* were examined under 20× magnification (Meiji Techno RZ Stereo Microscope) to determine sex. Sex was determined based on the relative size and shape of the abdomen (Barnard, 1984). A smear of honey water solution (10%) was added to the inside of the lid of each dish as a source of nutrients. A piece of damp cotton dental wick (nonsterile) was also added to each dish to serve as a water source.

Each residue aged for 0, 3 and 7 days after treatment (DAT), mortality was assessed at 24, 48 and 72 hr after the insects were placed in the Petri dishes. The number of dead insects was recorded. Insects were considered dead if they were not moving and did not move when touched with a probe. The 72-hr data for *C. rufilabris* were excluded from the analysis due to high control mortality.

Sublethal effects of each treatment were recorded for the two predatory species, *C. rufilabris* and *O. insidiosus*. Both eggs laid and eggs hatched were recorded as detailed for each species below.

TABLE 1 OMRI-listed insecticide treatments and adjuvants, classes, and rates used in laboratory bioassays

Insecticide trade name	Active ingredient	Manufacturer	Rate[a]
Azera®	Azadirachtin + Pyrethrins	Valent USA Corporation, Walnut Creek, CA	4.10 L/hectare
Entrust® SC	Spinosad	Dow AgroSciences LLC, Indianapolis, IN	0.44 L/hectare
Grandevo®	*Chromobacterium subtsugae*	Marrone Bio Innovations, Davis, CA	3.36 kg/hectare
Jet-Ag®[b]	Hydrogen peroxide + Peroxyacetic acid	Jet Harvest Solutions, Longwood, FL	1.00 L/100 L water
Venerate™ XC	*Burkholderia* spp.	Marrone Bio Innovations, Davis, CA	18.70 L/hectare
Veratran D®	Sabadilla alkaloids	McLaughlin Gormley King Co., Minneapolis, MN	16.80 kg/hectare
Adjuvant trade name	**Active ingredient**	**Manufacturer**	**Rate[a]**
Nu film P®	Poly−1-p-menthene	Miller Chemical & Fertilizer LLC, Hanover, PA	0.44 L/hectare
Oroboost™	Alcohol ethoxylate	Oro Agri Inc., Fresno, CA	1.00 L/100 L water
Leaf life®	Polyether-polymethylsiloxane-copolymer, polyether	Loveland Products Inc., Greeley, CO	0.06 L/ 100 L water

[a]Maximum label rates.

[b]Agricultural sanitizer labelled as a fungicide, bactericide, algaecide.

Sublethal effects were not recorded for *A. colemani* because this would have required providing prey insects and rearing out the next generation of parasitoids, which was beyond the scope of this study.

Over the course of the bioassay, *C. rufilabris* laid eggs in the Petri dishes. Eggs were counted one or two days following the 72-hr mortality check. Dishes were held for an additional 5 days to allow larvae to hatch. For the 7 DAT residues, however, eggs and larvae were counted together 4–6 days following the bioassay. Only one larval count was done because in the absence of an alternative food source the larvae began to eat unhatched eggs and cannibalize other larvae.

Following the 72-hr mortality assessment for each residue age (0, 3 and 7 DAT), *O. insidiosus* survivors were transferred to untreated containers and retained to assess sublethal effects. The number of eggs laid and the number of eggs that hatched were recorded for each replicate.

2.3 | Statistical analysis

Mortality data were arcsine transformed and analysed using repeated measures ANOVA (analysis of variance) (SAS, 2013). Means were separated using LSD (least significant difference) tests. Since a fresh shipment of insects was used for each residue age, each residue age was analysed separately.

The numbers of eggs laid by *O. insidiosus* and eggs per female laid by *C. rufilabris* were analysed using an ANOVA with means separated using LSD tests (SAS, 2013). For *C. rufilabris*, the number of eggs laid per female was square root x + 0.1 transformed to normalize the data. Percent egg hatch was arcsine transformed and analysed in the same way as the number of eggs laid.

For mortality data, the IOBC (International Organization for Biological Control) rating scale was used to describe treatment differences in the results. For laboratory studies, the IOBC toxicity ratings scale is as follows: 1 = harmless (<30% mortality), 2 = slightly harmful (30%–79%), 3 = moderately harmful (80%–99%) and 4 = harmful (>99%) (Sterk et al., 1999).

3 | RESULTS

3.1 | *Aphidius colemani*

For the 0 DAT residues (Figure 1), there were significant differences among treatments ($F = 17.92$, $df = 27, 108$, $p < .0001$) and a significant

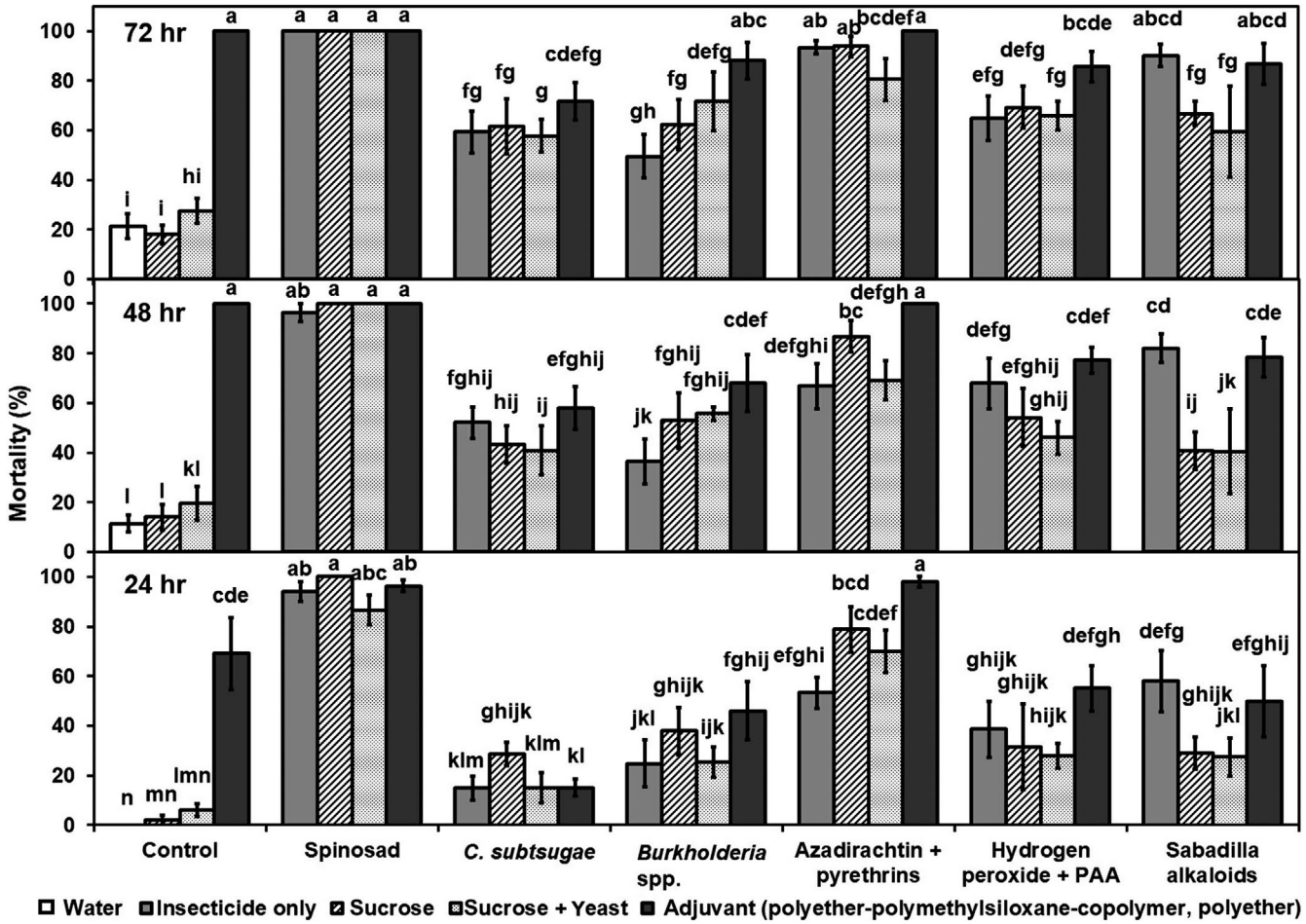

FIGURE 1 Mean percent mortality ± Standard Error (S.E.) of OMRI-listed insecticides on *Aphidius colemani*, after 24, 48 and 72 hr exposure to residue age 0 DAT. Bars with different letter are significantly different ($p < .05$). DAT, days after treatment; PAA, peroxyacetic acid

time*treatment interaction (F = 1.84, df = 54, 216, p = .0023). At 24 hr, most treatments were harmless or slightly harmful. The exceptions were the spinosad treatments and azadirachtin + pyrethrins + PEPMS, which were moderately harmful to harmful. At 48 hr, only water, sucrose and sucrose + yeast remained harmless. Azadirachtin + pyrethrins + sucrose, sabadilla alkaloids and spinosad were moderately harmful, and the other three spinosad treatments, PEPMS, and azadirachtin + pyrethrins + PEPMS were harmful. At 72 hr, spinosad reached the harmful level, while azadirachtin + pyrethrins, azadirachtin + pyrethrins + sucrose + yeast, hydrogen peroxide + PAA + PEPMS and sabadilla alkaloids + PEPMS reached the level of moderately harmful. All other treatments except water, sucrose and sucrose + yeast were slightly harmful.

For the 3 DAT residues (Figure 2), there were significant differences among treatments (F = 17.46, df = 27, 108, p < .0001) and a significant time*treatment interaction (F = 5.83, df = 54, 216, p < .0001). At 24 hr, all treatments were harmless or slightly harmful except for the four spinosad treatments, which were all harmful. At 48 hr, only the water control was harmless. The four spinosad treatments, PEPMS, and azadirachtin + pyrethrins +PEPMS were harmful. Several *C. subtsugae* and azadirachtin + pyrethrins treatments along with sabadilla alkaloids + PEPMS were moderately harmful. At 72 hr, *C. subtsugae* and *C. subtsugae* + PEPMS had reached the level of harmful. The *C. subtsugae* and azadirachtin + pyrethrins treatments that were slightly harmful at 48 hr increased to moderately harmful along with several *Burkholderia* spp. and sabadilla alkaloids treatments.

3.2 | *Chrysoperla rufilabris*

For the 0 DAT residues (Figure 3), there were significant differences among treatments (F = 2.54, df = 27, 108, p = .0004) and a significant time*treatment interaction (F = 2.13, df = 27, 108, p = .0033). All treatments were harmless at 24 hr. At 48 hr, only water, *Burkholderia* spp., *Burkholderia* spp. + sucrose, *Burkholderia* spp. + P1M, sabadilla alkaloids, sabadilla alkaloids + sucrose and sabadilla alkaloids + P1M were harmless. Spinosad + sucrose +yeast and spinosad + P1M were moderately harmful.

For the 3 DAT residues (Figure 4), there were significant differences among treatments (F = 7.28, df = 27, 108, p < .0001) but no significant time*treatment interaction (F = 0.94, df = 27, 108, p = .56). All treatments except spinosad, spinosad + sucrose and spinosad + sucrose +yeast were harmless at 24 hr. The three spinosad treatments were slightly

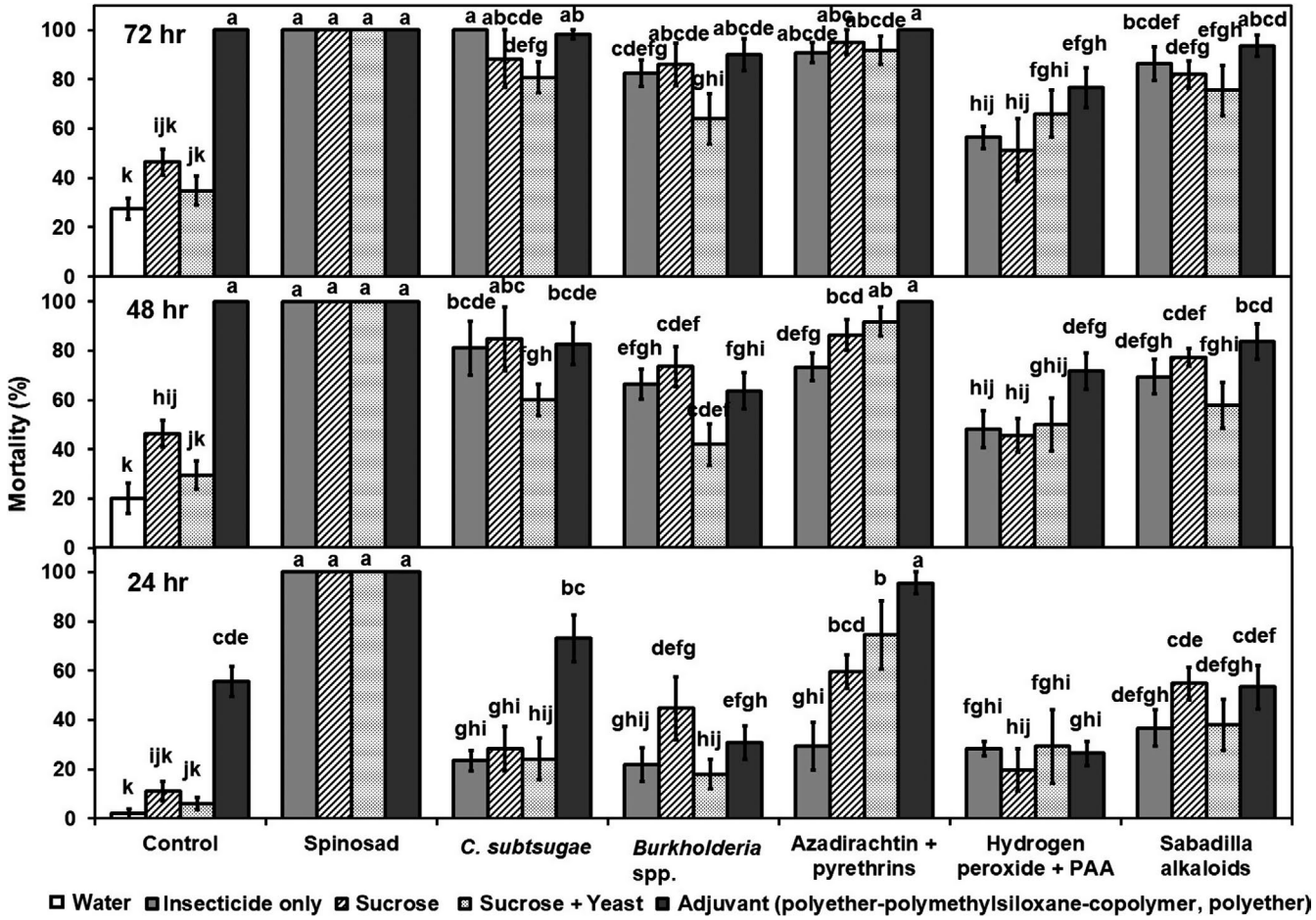

FIGURE 2 Mean percent mortality ± S.E. of OMRI-listed insecticides on *Aphidius colemani*, after 24, 48 and 72 hr exposure to residue age 3 DAT. Bars with different letter are significantly different (p < .05). DAT, days after treatment; PAA, peroxyacetic acid

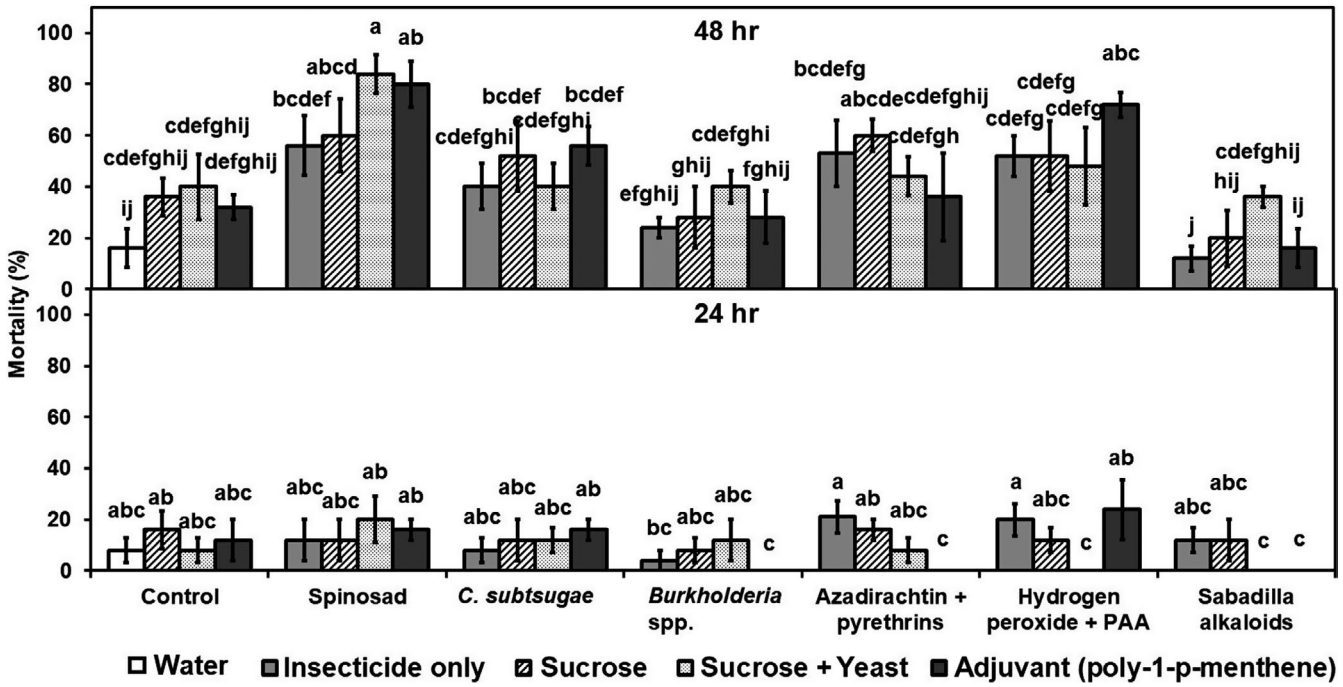

FIGURE 3 Mean percent mortality ± S.E. of OMRI-listed insecticides on *Chrysoperla rufilabris*, after 24 and 48 hr exposure to residue age 0 DAT. Bars with different letters are significantly different ($p < .05$). DAT, days after treatment; PAA, peroxyacetic acid

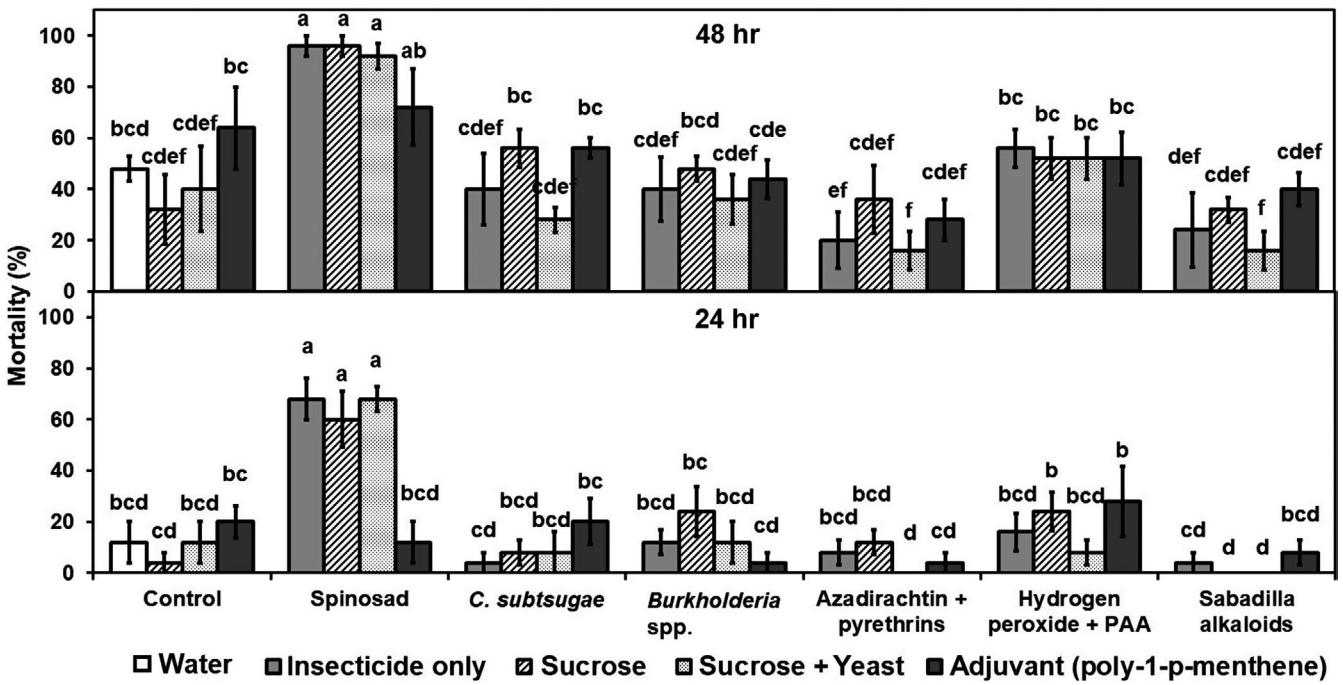

FIGURE 4 Mean percent mortality ± S.E. of OMRI-listed insecticides on *Chrysoperla rufilabris*, after 24 and 48 hr exposure to residue age 3 DAT. Bars with different letters are significantly different ($p < .05$). DAT, days after treatment; PAA, peroxyacetic acid

harmful. At 48 hr, spinosad, spinosad + sucrose and spinosad + sucrose +yeast increased to moderately harmful while only *C. subtsugae* + sucrose +yeast, three of the azadirachtin + pyrethrins treatments and two of the sabadilla alkaloids treatments remained harmless.

For the 7 DAT residues (Figure 5), there were significant differences among treatments ($F = 10.91$, $df = 27, 108$, $p < .0001$) and a significant time*treatment interaction ($F = 2.35$, $df = 27, 108$, $p = .001$). At 24 hr, all treatments except spinosad, spinosad + sucrose and spinosad + sucrose +yeast were harmless. These three spinosad treatments were slightly harmful. At 48 hr, spinosad increased to moderately harmful, the other three spinosad treatments were slightly harmful, and all of the remaining treatments were still harmless.

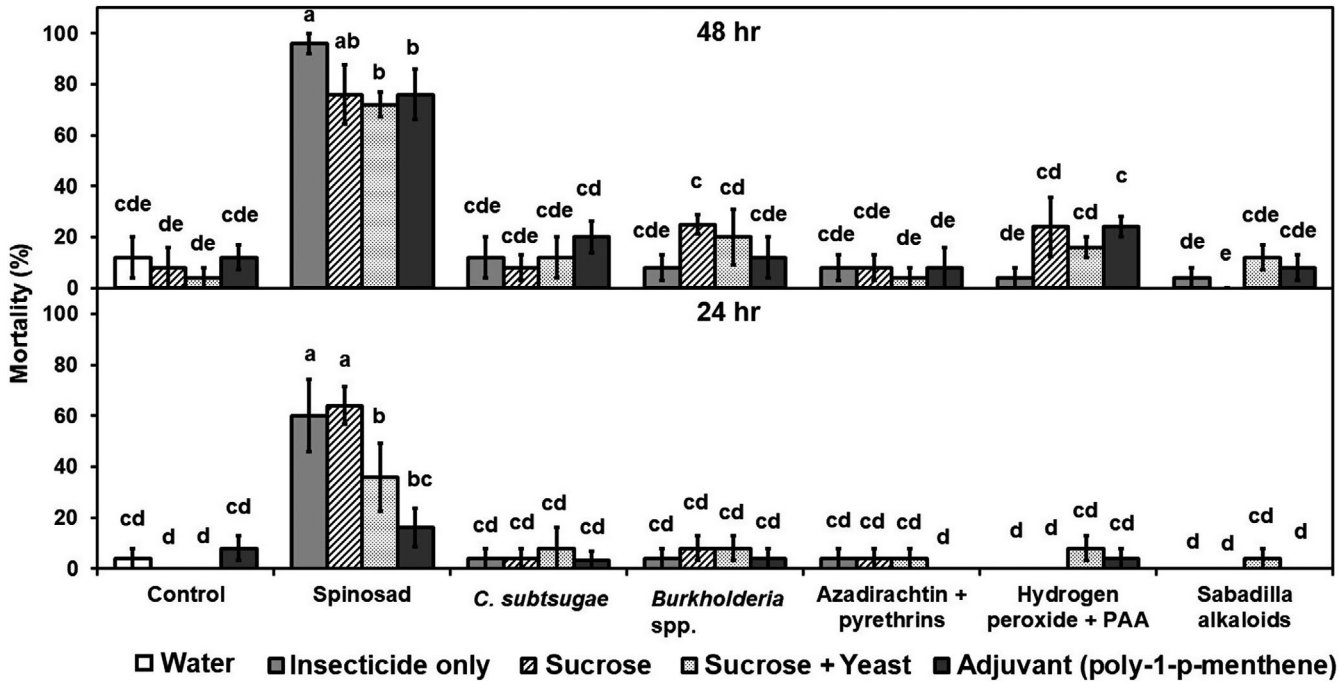

FIGURE 5 Mean percent mortality ± S.E. of OMRI-listed insecticides on *Chrysoperla rufilabris*, after 24 and 48 hr exposure to residue age 7 DAT. Bars with different letters are significantly different ($p < .05$). DAT, days after treatment; PAA, peroxyacetic acid

There were significant differences in eggs laid per *C. rufilabris* female (Table 2) with 0 ($F = 3.09$, $df = 27, 139$, $p < .0001$) and 3 DAT ($F = 2.82$, $df = 27, 139$, $p < .0001$) residues but not 7 DAT ($F = 1.44$, $df = 27, 139$, $p = .1$) residues. With the 0 DAT residues, there were significantly less eggs laid per female in the azadirachtin + pyrethrins, azadirachtin + pyrethrins + sucrose, azadirachtin + pyrethrins + sucrose + yeast and all spinosad treatments compared with the water control. Azadirachtin + pyrethrins +sucrose + yeast had significantly fewer eggs per female compared with the azadirachtin + pyrethrins + P1M treatment. Sabadilla alkaloids had significantly fewer eggs per female compared with sabadilla alkaloids + P1M. With the 3 DAT residues, the number of eggs per female was low and no treatment had significantly fewer eggs per female compared with the control. However, azadirachtin + pyrethrins + sucrose + yeast and hydrogen peroxide + PAA + sucrose + yeast had significantly higher eggs per female compared with the control. *Chromobacterium subtsugae* and *C. subtsugae* + sucrose had significantly fewer eggs per female compared with *C. subtsugae* + P1M and *C. subtsugae* + sucrose + yeast (Table 2).

There were no significant differences in % *C. rufilabris* egg hatch (Table 2) with 0 ($F = 0.81$, $df = 27, 139$, $p = .73$) and 3 DAT ($F = 0.96$, $df = 27, 139$, $p = .53$) residues but there were significant differences with 7 DAT ($F = 12.94$, $df = 27, 139$, $p < .0001$) residues. At 7 DAT, no treatment had a significantly lower % egg hatch compared with the water control. All the azadirachtin + pyrethrins, all the spinosad, all the *C. subtsugae* and all the *Burkholderia* spp. treatments had significantly higher % egg hatch compared with the control.

3.3 | *Orius insidiosus*

For the 0 DAT residues (Figure 6), there were significant differences among treatments ($F = 15.01$, $df = 27, 108$, $p < .0001$) and a significant time*treatment interaction ($F = 2.90$, $df = 54, 216$, $p < .0001$). At 24 hr, all treatments except the spinosad and sabadilla alkaloids treatments were harmless and they remained harmless through 72 hr. The spinosad and sabadilla alkaloid treatments were slightly harmful at 24 hr. By 72 hr, all of the spinosad treatments along with sabadilla alkaloids and sabadilla alkaloids + sucrose had increased to moderately harmful.

For the 3 DAT residues (Figure 7), there were significant differences among treatments ($F = 25.71$, $df = 27, 108$, $p < .0001$) and a significant time*treatment interaction ($F = 4.88$, $df = 54, 216$, $p < .0001$). At 24 hr, all treatments were harmless except spinosad + sucrose and spinosad + sucrose + yeast and all four sabadilla alkaloids treatments which were slightly to moderately harmful. By 72 hr, spinosad, spinosad + sucrose and spinosad + sucrose + yeast had increased to moderately harmful while spinosad + AE was only slightly harmful. Sabadilla alkaloids + sucrose + yeast was harmful at 72 hr while the other three sabadilla alkaloids treatments were moderately harmful.

For the 7 DAT residues (Figure 8), there were significant differences among treatments ($F = 18.36$, $df = 27, 108$, $p < .0001$) and a significant time*treatment interaction ($F = 4.19$, $df = 54, 216$, $p < .0001$). Trends were like 3 DAT residues. By 72 hr, spinosad, spinosad + sucrose and spinosad + sucrose +yeast were moderately harmful while spinosad + AE was harmless. Sabadilla alkaloids and sabadilla alkaloids + sucrose were slightly harmful while the other two sabadilla alkaloids treatments were moderately harmful. All other treatments were harmless.

TABLE 2 Mean ± S.E. numbers of eggs laid and hatchability (%) per female *Chrysoperla rufilabris* exposed to 0, 3 and 7 DAT residue ages of insecticides

	Eggs per female (Mean ± S.E.)			Hatchability % (Mean ± S.E.)		
Treatment	**0 DAT**	**3 DAT**	**7 DAT**	**0 DAT**	**3 DAT**	**7 DAT**
Control	39.0 ± 7.6^{abcd}	2.3 ± 0.6^{bcdefghi}	14.1 ± 2.2^{abc}	16.0 ± 1.6^{abc}	18.7 ± 8.5^{abcd}	0.0 ± 0.0^{e}
Suc	34.9 ± 4.1^{abcde}	1.9 ± 0.8^{efghi}	13.1 ± 1.7^{abcde}	17.2 ± 1.4^{abc}	16.2 ± 12.9^{abcd}	0.8 ± 0.8^{e}
Suc Yst	40.9 ± 4.5^{abc}	4.5 ± 2.0^{abcdefg}	12.7 ± 3.3^{bcde}	15.3 ± 2.7^{abc}	19.2 ± 6.9^{abcd}	0.5 ± 0.5^{e}
P1M	53.1 ± 4.5^{a}	1.8 ± 0.5^{defghi}	8.0 ± 3.0^{cde}	10.8 ± 1.7^{c}	10.2 ± 6.3^{bcd}	0.0 ± 0.0^{e}
Spinosad	21.2 ± 4.3^{efghi}	0.4 ± 0.2^{i}	6.1 ± 2.7^{de}	22.2 ± 4.9^{abc}	10.0 ± 6.1^{bcd}	31.4 ± 8.6^{bcd}
Spinosad + Suc	17.9 ± 5.4^{ghi}	0.7 ± 0.3^{hi}	7.8 ± 2.1^{cde}	14.3 ± 4.0^{bc}	30.0 ± 12.3^{abcd}	19.0 ± 7.5^{bcd}
Spinosad + Suc Yst	19.8 ± 2.8^{efghi}	1.7 ± 0.9^{fghi}	4.4 ± 1.4^{e}	18.4 ± 4.1^{abc}	18.9 ± 7.8^{abcd}	45.6 ± 6.3^{a}
Spinosad + P1M	15.9 ± 3.4^{hi}	2.0 ± 1.1^{defghi}	11.9 ± 2.6^{bcde}	17.7 ± 3.8^{abc}	30.2 ± 7.8^{ab}	24.6 ± 4.4^{bcd}
C. subtsugae	33.9 ± 5.6^{abcdef}	0.9 ± 0.5^{i}	15.3 ± 2.2^{abc}	17.7 ± 3.6^{abc}	6.0 ± 6.0^{d}	14.1 ± 2.9^{d}
C. subtsugae + Suc	40.3 ± 2.1^{abc}	2.4 ± 1.1^{cdefghi}	18.6 ± 4.4^{abc}	19.5 ± 3.5^{abc}	27.7 ± 11.0^{abc}	17.1 ± 4.4^{bcd}
C. subtsugae + Suc Yst	32.3 ± 0.6^{bcdef}	5.9 ± 1.4^{ab}	21.7 ± 4.7^{ab}	22.9 ± 2.8^{ab}	33.7 ± 8.1^{a}	17.5 ± 4.3^{bcd}
C. subtsugae + P1M	32.2 ± 8.0^{bcdefg}	6.2 ± 2.0^{ab}	10.4 ± 2.3^{bcde}	14.2 ± 1.5^{abc}	15.5 ± 7.4^{abcd}	20.6 ± 4.1^{bcd}
Burkholderia spp.	31.0 ± 3.2^{bcdef}	4.1 ± 1.5^{abcdefgh}	13.6 ± 3.6^{abcde}	16.0 ± 1.0^{abc}	20.9 ± 7.7^{abcd}	29.3 ± 9.9^{bc}
Burkholderia spp. + Suc	48.7 ± 7.5^{ab}	5.9 ± 1.5^{ab}	9.7 ± 1.7b^{cde}	13.3 ± 0.8^{abc}	14.8 ± 4.6^{abcd}	28.1 ± 2.6^{b}
Burkholderia spp. + Suc Yst	32.0 ± 4.6^{bcdef}	2.8 ± 1.3^{bcdefghi}	12.5 ± 4.9^{cde}	17.2 ± 1.8^{abc}	23.3 ± 12.5^{abcd}	19.0 ± 3.3^{bcd}
Burkholderia spp. + P1M	34.9 ± 1.7^{abcde}	5.5 ± 2.6^{abcdef}	18.6 ± 7.0^{abc}	15.6 ± 2.7^{abc}	11.6 ± 4.4^{abcd}	16.5 ± 5.2^{d}
Azadirachtin + pyrethrins	21.2 ± 6.3^{fghi}	3.6 ± 1.1^{abcdefgh}	27.0 ± 7.6^{a}	18.1 ± 5.4^{abc}	12.4 ± 2.5^{abcd}	14.2 ± 3.4^{cd}
Azadirachtin + pyrethrins + Suc	21.8 ± 5.8^{efghi}	1.6 ± 0.8^{ghi}	12.5 ± 3.9^{bcde}	30.5 ± 11.5^{a}	14.6 ± 9.9^{abcd}	16.1 ± 6.3^{d}
Azadirachtin + pyrethrins + Suc Yst	15.3 ± 4.9^{i}	6.6 ± 1.3^{a}	21.0 ± 3.6^{ab}	23.4 ± 7.3^{abc}	20.5 ± 7.0^{abcd}	17.0 ± 4.8^{bcd}
Azadirachtin + pyrethrins + P1M	27.8 ± 6.4^{cdefgh}	3.7 ± 1.7^{abcdefgh}	17.0 ± 4.7^{abc}	23.7 ± 1.7^{ab}	21.4 ± 10.6^{abcd}	22.2 ± 1.2^{bcd}
Hydrogen peroxide + PAA	40.9 ± 3.7 ^{abc}	4.1 ± 0.5^{abcde}	16.4 ± 4.6^{abcd}	25.8 ± 1.6^{a}	15.9 ± 6.8^{abcd}	0.4 ± 0.4^{e}
Hydrogen peroxide + PAA + Suc	40.1 ± 3.3^{abc}	5.3 ± 1.2^{abc}	16.6 ± 3.3^{abc}	21.2 ± 3.6^{abc}	11.9 ± 5.5^{abcd}	1.3 ± 1.3^{e}
Hydrogen peroxide + PAA + Suc Yst	33.6 ± 7.7^{bcdefg}	7.5 ± 1.5^{a}	12.3 ± 4.3^{bcde}	15.8 ± 2.9^{abc}	16.6 ± 5.9^{abcd}	1.7 ± 1.7^{e}
Hydrogen peroxide + PAA + P1M	35.7 ± 5.2^{abcde}	4.7 ± 0.7^{abcd}	14.7 ± 4.9^{abcd}	16.0 ± 3.7^{abc}	32.4 ± 4.0^{a}	2.3 ± 1.4^{e}
Sabadilla alkaloids	28.8 ± 8.6^{defghi}	5.9 ± 1.7^{abcd}	10.0 ± 2.5^{bcde}	12.8 ± 3.3^{c}	8.5 ± 3.9^{abcd}	0.0 ± 0.0^{e}
Sabadilla alkaloids + Suc	35.4 ± 4.5^{abcde}	4.9 ± 1.1^{abcd}	12.0 ± 1.8^{abcde}	19.9 ± 2.4^{abc}	34.0 ± 12.1^{ab}	0.0 ± 0.0^{e}
Sabadilla alkaloids + Suc Yst	36.9 ± 4.3^{abcd}	5.1 ± 1.6^{abcd}	9.4 ± 3.9^{cde}	20.1 ± 3.6^{abc}	6.0 ± 4.0^{cd}	0.5 ± 0.5^{e}
Sabadilla alkaloids + P1M	44.6 ± 1.7^{ab}	3.8 ± 0.6^{abcdefg}	16.2 ± 3.6^{abc}	16.1 ± 1.1^{abc}	23.3 ± 11.5^{abcd}	0.0 ± 0.0^{e}

Note: Values with a different letter within columns are significantly different ($p < .05$).

Abbreviations: DAT, days after treatment; P1M, poly-1-p-menthene; PAA, peroxyacetic acid; Suc, sucrose; Yst, yeast.

There were significant differences in eggs laid by female *O. insidiosus* (Table 3) with 0 ($F = 4.92$, $df = 27, 139$, $p < .0001$), 3 DAT ($F = 5.09$, $df = 27, 139$, $p < .0001$) and 7 DAT ($F = 4.60$, $df = 27, 139$, $p < .0001$) residues. For the 0 DAT residues, significantly fewer eggs were laid in all the spinosad, *C. subtsugae* + AE, *Burkholderia* spp. + sucrose, and all the sabadilla alkaloids treatments compared with the water control. There were significantly more eggs laid in the hydrogen peroxide + PAA treatment compared with the hydrogen peroxide + PAA +AE treatment. With the 3 DAT residues, the azadirachtin + pyrethrins +sucrose + yeast, all spinosad, *C. subtsugae*, hydrogen peroxide + PAA + sucrose, *Burkholderia* spp. + AE, and all sabadilla alkaloids treatments had significantly fewer eggs

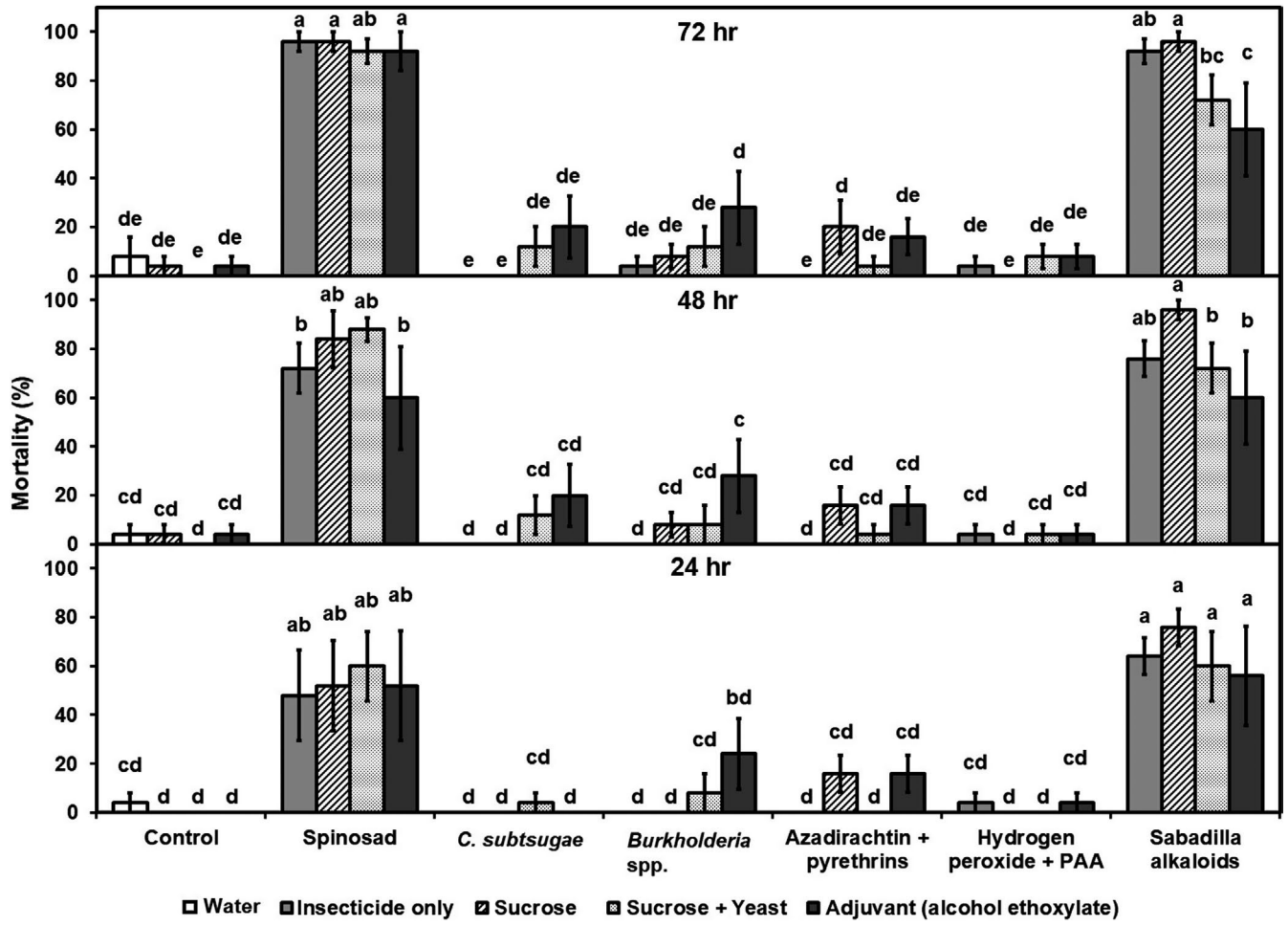

FIGURE 6 Mean percent mortality ± S.E. of OMRI-listed insecticides on *Orius insidiosus*, after 24, 48 and 72 hr exposures to residue age 0 DAT. Bars with different letter are significantly different ($p < .05$). DAT, days after treatment; PAA, peroxyacetic acid

laid compared with the water control. *Burkholderia* spp. + AE had significantly fewer eggs laid compared with the *Burkholderia* spp. treatment. For the 7 DAT residues, the spinosad, spinosad + sucrose, spinosad + sucrose +yeast, *C. subtsugae* + sucrose, and all sabadilla alkaloids treatments had significantly fewer eggs laid compared with the water control. Spinosad + AE had significantly more eggs laid compared with the other spinosad treatments. *C. subtsugae* + sucrose had significantly fewer eggs laid compared with all the other *C. subtsugae* treatments. Hydrogen peroxide + PAA + AE had significantly higher eggs laid compared with hydrogen peroxide + PAA and hydrogen peroxide + PAA +sucrose.

There were significant differences in % egg hatch (Table 3) with 0 ($F = 2.66$, $df = 27, 139$, $p = .0002$), 3 DAT ($F = 3.51$, $df = 27, 139$, $p < .0001$) and 7 DAT ($F = 4.78$, $df = 27, 139$, $p < .0001$) residues. With the 0 DAT residues, all spinosad, *C. subtsugae*, *C. subtsugae* + AE, hydrogen peroxide + PAA + AE, *Burkholderia* spp. + sucrose, *Burkholderia* spp. + sucrose + yeast, sabadilla alkaloids and sabadilla alkaloids + sucrose + yeast had significantly lower % egg hatch than the water control. AE had significantly higher % egg hatch than the water control. *Chromobacterium subtsugae* + sucrose had significantly higher % egg hatch compared with *C. subtsugae* + AE. For the 3 DAT residues, the azadirachtin + pyrethrins + AE, azadirachtin + pyrethrins + sucrose + yeast, all spinosad, hydrogen peroxide + PAA +sucrose and all sabadilla alkaloids treatments had significantly lower % egg hatch compared with the water control. Hydrogen peroxide + PAA had significantly higher % egg hatch compared with hydrogen peroxide + PAA + sucrose. For the 7 DAT residues, all treatments except sucrose and *C. subtsugae* + AE had significantly lower % egg hatch compared with the water control. All the spinosad, the *C. subtsugae* + sucrose, and all the sabadilla alkaloids treatments had significantly lower % egg hatch compared with the AE and sucrose + yeast treatments. *Chromobacterium subtsugae* + AE had significantly higher % egg hatch compared with *C. subtsugae* + sucrose and *C. subtsugae* + sucrose + yeast.

4 | DISCUSSION

Pesticide effects can vary greatly across natural enemy species depending on several factors including the type of natural enemy, life stage exposed, pesticide formulation and sex (Cloyd, 2012; Stark et al., 2007). In our studies, the contact residual activity of insecticides on natural enemies was variable both among natural enemy species and among insecticides.

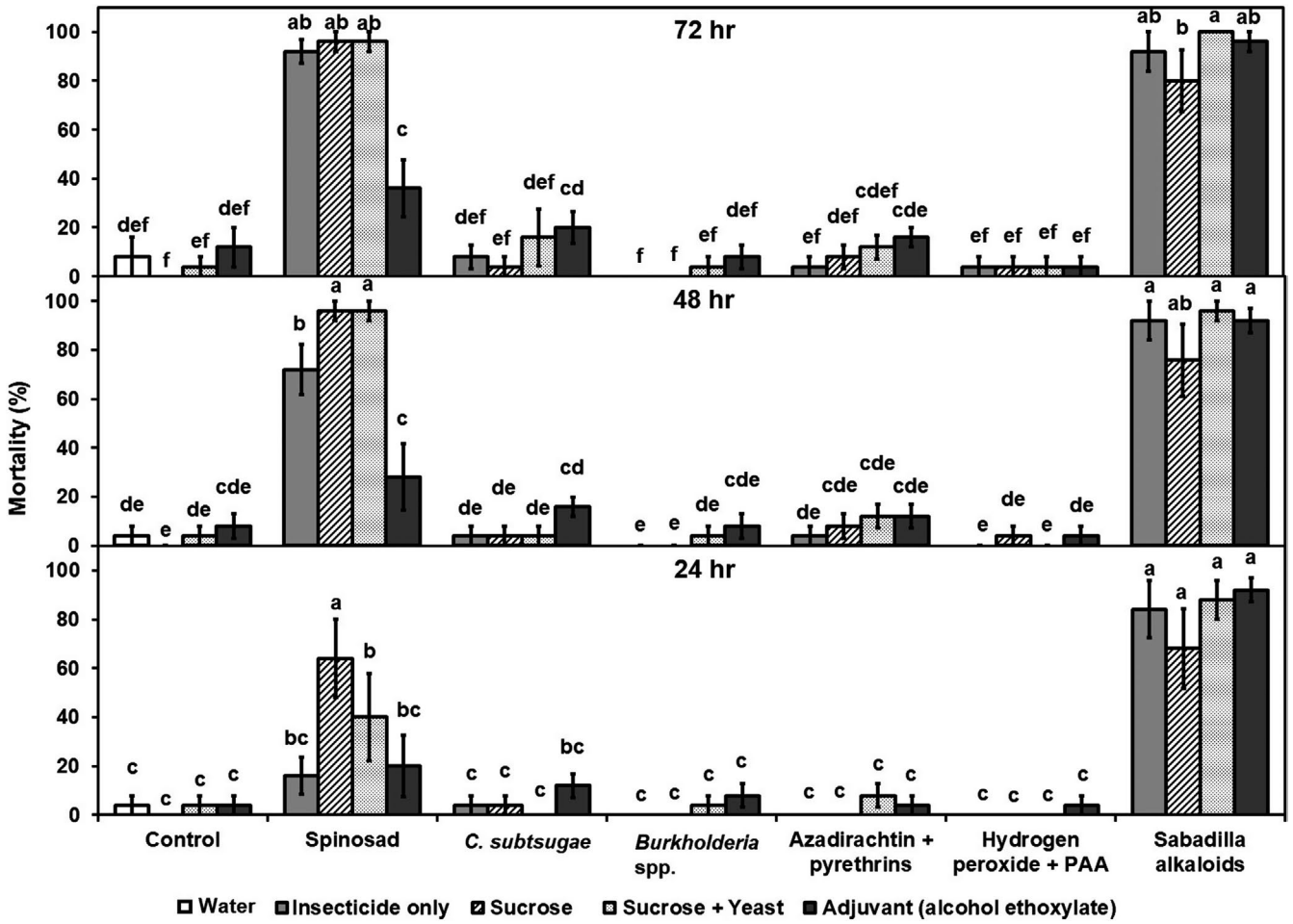

FIGURE 7 Mean percent mortality ± S.E. of OMRI-listed insecticides on *Orius insidiosus*, after 24, 48 and 72 hr exposures to residue age 3 DAT. Bars with different letter are significantly different ($p < .05$). DAT, days after treatment; PAA, peroxyacetic acid

All compounds tested, including PEPMS, were at least slightly harmful to the parasitoid *A. colemani*. Only water, sucrose and sucrose + yeast were harmless. Some studies have shown that hymenopteran parasitoids are more susceptible to spinosad than predatory insects, based on sublethal effects on reproductive capacity and longevity (Desneux et al., 2007; Williams, Valle, & Viñuela, 2003).

Previous studies found several conventional pesticides including organophosphates and carbamates to be highly toxic to *Chrysopa carnea* while pyrethroids were significantly less toxic (Plapp & Bull, 1978; Roubos, Rodriguez-Saona, Holdcraft, et al., 2014b). In our study using insecticides, adjuvants and phagostimulants labelled for organic use, we found similar results. Only spinosad reached moderately harmful status with *C. rufilabris*. For *O. insidiosus*, toxicity was equally low for most of the treatments with only spinosad and sabadilla alkaloids reaching moderately harmful or harmful status.

Novel insecticides with selective modes of action might be compatible with different natural enemies (Gentz et al., 2010). For example, predators that consume the entire prey, such as coccinellid beetles, are potentially exposed to any insecticide that the prey has been in contact with, whereas hemipteran predators like *Orius* spp. may receive less exposure since they do not consume the cuticle and may not, necessarily, consume the gut and its contents (Theiling & Croft, 1988). The results of this study indicate that all tested insecticides except spinosad are compatible with *C. rufilabris*. There is similar compatibility with *O. insidiosus* with the exception of sabadilla alkaloids showing high toxicity in addition to spinosad. In contrast, none of the tested compounds appear to be compatible with *A. colemani*. On the IOBC toxicity rating scale, spinosad was consistently rated from slightly harmful to harmful across natural enemies and residue ages tested in this study. Conversely, Williams et al. (2003) reported predatory species including *O. insidiosus*, *C. rufilabris*, *C. carnea*, *Geocoris punctipes* (Say) and coccinellid species were particularly tolerant to spinosad when exposed in laboratory bioassays. In other studies, spinosad has been found to be toxic towards parasitoids, such as *Bracon nigricans* Szèpligeti (Biondi et al., 2013), *Cotesia plutellae* (Kurdjumov) (Haseeb, Liu, & Jones, 2004), and *Telenomus podisi* Ashmead (Ogburn & Walgenbach, 2019), and predatory insects, such as *Dalotia coriaria* (Kraatz) and *O. insidiosus* (Cloyd & Herrick, 2018; Herrick & Cloyd, 2017). This does not necessarily translate into high field toxicity. Previous work has shown that spinosad caused significantly higher mortality in *O. insidiosus* in petri dish assays compared with treated plants in the field and greenhouse (Studebaker & Kring, 2003). In this case, route of exposure can have a significant effect on

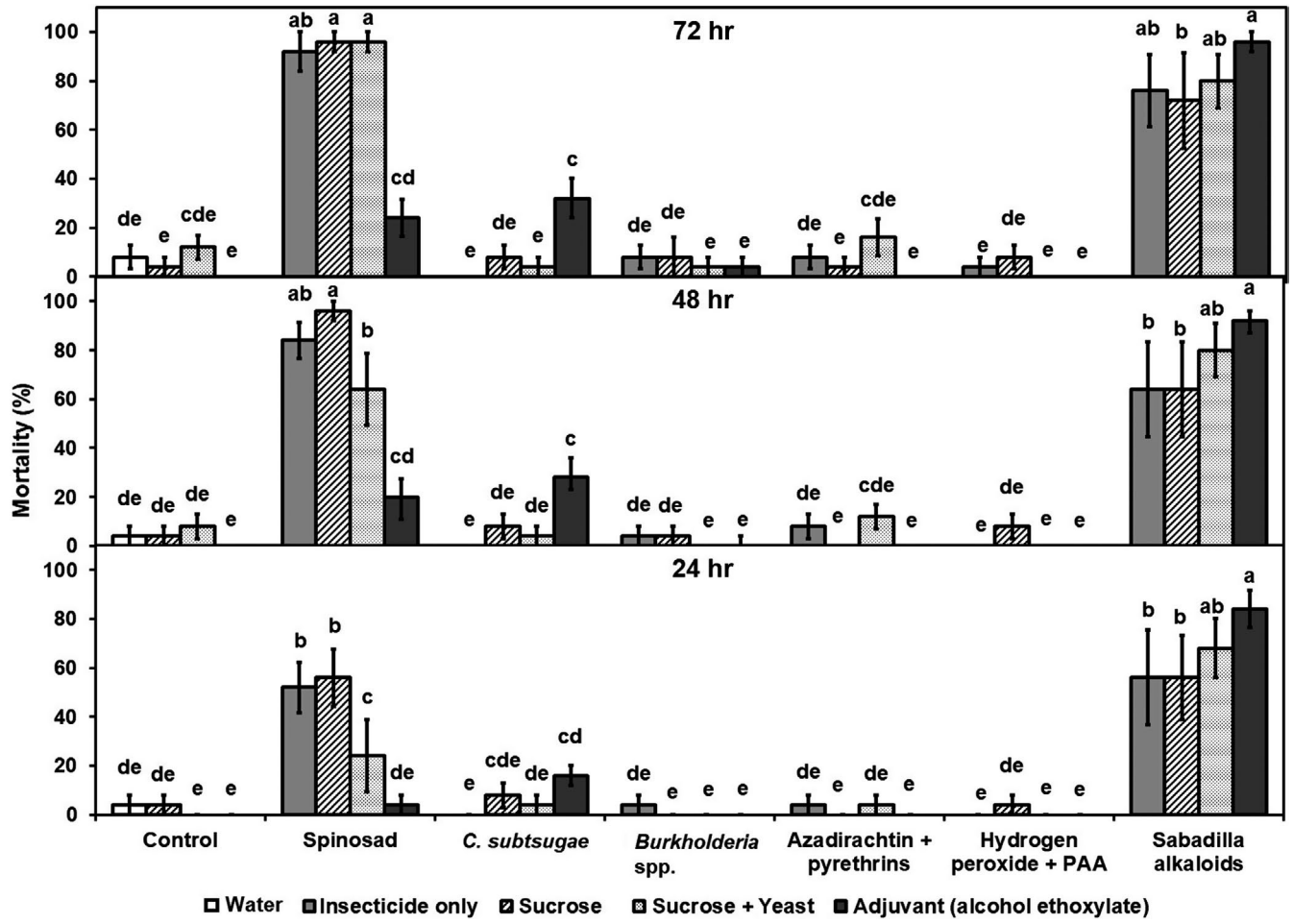

FIGURE 8 Mean percent mortality ± S.E. of OMRI-listed insecticides on *Orius insidiosus*, after 24, 48 and 72 hr exposures to residue age 7 DAT. Bars with different letter are significantly different ($p < .05$). DAT, days after treatment; PAA, peroxyacetic acid

the results. Spinosad residues can degrade quickly, especially under unfavourable environmental conditions such as high temperatures, sunlight and rainfall events (Crouse et al., 2001). Similar discrepancies between assay methods testing azadirachtin-based compounds have been reported in the parasitoid *C. plutellae* (Haseeb et al., 2004). Therefore, it will be important to compare these effects measured through various methodologies (Studebaker & Kring, 2003).

Except for PEPMS, neither the addition of adjuvants nor phagostimulants increased mortality of the tested natural enemies. The addition of PEPMS increased the toxicity of azadirachtin + pyrethrins, *C. subtsugae*, hydrogen peroxide + PAA and *Burkholderia* spp. towards *A. colemani*. Whether this was a synergistic effect or simply due to the high toxicity of PEPMS is unclear. The high mortality of *A. colemani* in the PEPMS treatment was unexpected and highlights the need to test the effects of adjuvants on natural enemies. It is interesting that the addition of AE appeared to decrease the toxicity of spinosad towards *O. insidiosus*. In contrast, the addition of AE did not affect the efficacy of spinosad against *D. suzukii* (Roubos et al., 2019a). However, the addition of PEPMS reduced the efficacy of spinosad against *D. suzukii* (Roubos et al., 2019a), while in this study, the addition of PEPMS did not affect the efficacy of spinosad against *A. colemani*.

There is increasing recognition that sublethal effects observed in laboratory bioassays can translate into effects on pest regulation under field conditions (Biondi et al., 2013; Desneux et al., 2007). Some possible sublethal effects were seen in this study. For *C. rufilabris*, there were fewer eggs laid per female in the azadirachtin + pyrethrins and spinosad treatments with the 0 DAT residues but there was no effect on eggs laid per female with the 3-day residues. There were minimal effects with 7-day residues with only spinosad and spinosad + sucrose + yeast having fewer eggs laid compared with the water control. No treatment had significantly lower % egg hatch compared with the control for any of the residue ages. The reason for the increase or lack of differences in egg hatch among the treatments at 7 DAT compared with the control is unclear and requires further research but could be related to the residual and consequently reduce toxicity of the pesticides. This is consistent with Garzon et al. (2015), who found that only deltamethrin, out of the insecticides they tested, significantly decreased fertility and fecundity of *C. carnea*. The addition of P1M did not negatively affect

TABLE 3 Mean ± S.E. total numbers of eggs laid and hatchability (%) by *Orius insidiosus* exposed to 0, 3 and 7 DAT residue ages of insecticides

	Total number of eggs (Mean ± S.E.)			Hatchability % (Mean ± S.E.)		
Treatment	**0 DAT**	**3 DAT**	**7 DAT**	**0 DAT**	**3 DAT**	**7 DAT**
Control	14.6 ± 1.6^{abc}	13.6 ± 3.0^{abcde}	10.6 ± 1.9^{abcd}	53.6 ± 13.6^{b}	42.8 ± 11.8^{abcde}	63.1 ± 7.1^{a}
Suc	9.0 ± 1.6^{bcde}	14.2 ± 3.2^{abcd}	11.4 ± 2.2^{abcd}	39.7 ± 14.0^{bcdef}	57.2 ± 11.3^{a}	55.4 ± 3.9^{ab}
Suc Yst	10.6 ± 4.2^{bcde}	19.0 ± 4.0^{a}	10.2 ± 2.3^{abcd}	41.6 ± 19.7^{bc}	54.5 ± 6.2^{ab}	40.8 ± 3.4^{bcde}
AE	17.8 ± 7.9^{abc}	9.0 ± 0.9^{abcdefg}	7.0 ± 1.3^{bcde}	76.6 ± 10.2^{a}	45.5 ± 6.4^{abcd}	43.0 ± 7.2^{bcd}
Spinosad	0.6 ± 0.6^{fg}	0.0 ± 0.0^{k}	0.4 ± 0.4^{f}	6.7 ± 6.7^{fg}	0.0 ± 0.0^{h}	0.0 ± 0.0^{i}
Spinosad + Suc	0.0 ± 0.0^{g}	0.0 ± 0.0^{k}	0.2 ± 0.2^{f}	0.0 ± 0.0^{g}	0.0 ± 0.0^{h}	0.0 ± 0.0^{i}
Spinosad + Suc Yst	0.2 ± 0.2^{g}	3.0 ± 3.0^{hijk}	0.0 ± 0.0^{f}	0.0 ± 0.0^{g}	6.7 ± 6.7^{gh}	0.0 ± 0.0^{i}
Spinosad + AE	0.2 ± 0.2^{g}	2.0 ± 2.0^{ijk}	13.6 ± 9.9^{bcde}	0.0 ± 0.0^{g}	4.0 ± 4.0^{gh}	11.0 ± 6.8^{ghi}
C. subtsugae	12.8 ± 4.2^{abcde}	4.8 ± 2.1^{fghij}	16.6 ± 5.9^{abc}	21.1 ± 9.8^{cdefg}	29.8 ± 12.8^{bcdefg}	33.4 ± 3.5^{cdefg}
C. subtsugae + Suc	11.0 ± 3.3^{abcde}	7.8 ± 2.1^{abcdefg}	3.8 ± 2.7^{ef}	48.4 ± 12.3^{bc}	30.7 ± 11.7^{bcdefg}	9.7 ± 6.1^{hi}
C. subtsugae + Suc Yst	15.2 ± 8.6^{abcd}	10.8 ± 1.2^{abcdef}	12.2 ± 4.4^{abcd}	24.5 ± 4.3^{bcdefg}	43.2 ± 9.7^{abcde}	21.4 ± 6.0^{defghi}
C. subtsugae + AE	4.2 ± 1.9^{defg}	12.2 ± 3.8^{abcdef}	14.8 ± 5.0^{abc}	9.7 ± 6.1^{efg}	28.1 ± 8.6^{bcdefgh}	47.2 ± 10.9^{abc}
Burkholderia spp.	8.8 ± 1.5^{bcde}	16.0 ± 2.4^{ab}	16.4 ± 4.4^{abc}	40.1 ± 8.1^{bcdef}	30.7 ± 2.9^{bcdefg}	34.4 ± 10.8^{bcdef}
Burkholderia spp. + Suc	5.2 ± 2.4^{defg}	9.8 ± 2.4^{abcdefg}	10.0 ± 4.9^{cde}	21.3 ± 8.8^{cdefg}	20.4 ± 9.5^{cdefgh}	24.0 ± 14.0^{defgh}
Burkholderia spp. + Suc Yst	6.4 ± 1.1^{bcde}	11.8 ± 5.6^{abcdefg}	4.8 ± 2.4^{def}	18.9 ± 9.9^{cdefg}	16.3 ± 10.4^{efgh}	25.3 ± 13.7^{cdefgh}
Burkholderia spp. + AE	13.6 ± 6.9^{abcde}	3.8 ± 2.3^{ghijk}	23.8 ± 8.9^{a}	37.9 ± 11.2^{bcdef}	36.7 ± 20.0^{abc}	37.2 ± 5.6^{bcdef}
Azadirachtin + pyrethrins	14.2 ± 3.5^{abc}	8.6 ± 5.0^{bcdefgh}	11.0 ± 2.9^{abcd}	25.8 ± 10.7^{bcdefg}	20.7 ± 5.7^{cdefgh}	33.6 ± 11.6^{bcdef}
Azadirachtin + pyrethrins + Suc	21.2 ± 8.1^{ab}	16.2 ± 7.1^{abc}	13.0 ± 1.9^{abc}	25.5 ± 9.6^{bcdefg}	44.2 ± 13.5^{abc}	24.1 ± 7.0^{defgh}
Azadirachtin + pyrethrins + Suc Yst	5.8 ± 2.4^{cdef}	4.6 ± 3.0^{ghijk}	12.6 ± 2.9^{abcd}	30.6 ± 10.9^{bcdefg}	13.8 ± 13.8^{fgh}	28.3 ± 10.0^{cdefgh}
Azadirachtin + pyrethrins + AE	13.6 ± 3.9^{abcd}	7.4 ± 4.3^{defghi}	14.0 ± 3.2^{abc}	42.7 ± 10.2^{bcde}	11.0 ± 7.1^{fgh}	29.1 ± 7.0^{cdefgh}
Hydrogen peroxide + PAA	28.0 ± 9.8^{a}	13.8 ± 4.7^{abcdef}	7.8 ± 3.8^{cde}	26.7 ± 9.9^{bcdefg}	38.0 ± 8.2^{abcde}	19.2 ± 10.1^{efghi}
Hydrogen peroxide + PAA + Suc	21.4 ± 7.6^{ab}	6.6 ± 3.7^{fghi}	9.8 ± 5.0^{cde}	47.4 ± 10.8^{bcd}	4.5 ± 2.8^{gh}	21.7 ± 10.5^{defghi}
Hydrogen peroxide + PAA + Suc Yst	14.8 ± 4.2^{abcd}	7.4 ± 4.3^{efghi}	11.4 ± 3.8^{abcd}	31.9 ± 12.0^{bcdefg}	18.8 ± 7.8^{defgh}	38.5 ± 4.4^{bcde}
Hydrogen peroxide + PAA + AE	9.4 ± 2.7^{bcde}	6.0 ± 2.0^{cdefghi}	20.8 ± 7.0^{ab}	17.5 ± 5.1^{cdefg}	27.5 ± 10.3^{cdefgh}	33.3 ± 7.7^{cdefg}
Sabadilla alkaloids	1.6 ± 1.6^{fg}	0.0 ± 0.0^{k}	3.2 ± 2.3^{ef}	7.5 ± 7.5^{ef}	0.0 ± 0.0^{h}	15.0 ± 10.0^{fghi}
Sabadilla alkaloids + Suc	0.8 ± 0.6^{fg}	4.80 ± 4.8^{hijk}	4.4 ± 4.2^{ef}	20.0 ± 20.0^{bcdefg}	5.8 ± 5.8^{gh}	7.6 ± 7.6^{hi}
Sabadilla alkaloids + Suc Yst	0.6 ± 0.6^{fg}	0.0 ± 0.0^{k}	0.0 ± 0.0^{f}	13.3 ± 13.3^{def}	0.0 ± 0.0^{h}	0.0 ± 0.0^{i}
Sabadilla alkaloids + AE	5.8 ± 3.8^{efg}	0.2 ± 0.2^{jk}	0.2 ± 0.2^{f}	21.5 ± 13.3^{bcdefg}	0.0 ± 0.0^{h}	0.0 ± 0.0^{i}

Note: Values with a different letter within columns are significantly different ($p < .05$).

Abbreviations: AE, alcohol ethoxylate; DAT, days after treatment; PAA, peroxyacetic acid; Suc, sucrose; Yst, yeast.

either eggs laid per female or % egg hatch nor did the addition of phagostimulants.

Sublethal effects of the treatments on *O. insidiosus* were unclear. There were fewer eggs laid in all the spinosad and all the sabadilla alkaloids treatments. This could have been a direct result of the high mortality in those treatments rather than from sublethal effects. Each of the insecticide treatments showed reduced % egg hatch compared with the control. Moscardini et al. (2013) found that four of the seven conventional insecticides they tested caused mortality of *O. insidiosus* eggs. The addition of AE did not negatively affect either number of eggs laid or % egg hatch nor did the addition of phagostimulants.

These findings suggest that growers have options for products that control *D. suzukii* and minimize negative effects on natural enemies. All insecticides except spinosad were harmless or only slightly harmful to *C. rufilabris* adults, and all treatments except spinosad and sabadilla alkaloids were harmless to *O. insidiosus* adults. The parasitoid *A. colemani* was more sensitive to insecticide applications than the predators with all insecticide treatments rated as at least slightly harmful. Since the Petri dish assays used, demonstrate a scenario

of near-constant exposure to the insecticides and other compounds tested, more studies involving semi-field and field experiments are needed to make firm conclusions.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regard to this work.

AUTHORS' CONTRIBUTIONS

OEL, AS and RI conceived the study. NS, CRR, PDF, JS and BAL conducted the experiments. EMR, NS and JS analysed the data and drafted the manuscript. All authors reviewed the MS and approved the manuscript.

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DATA AVAILABILITY STATEMENT

Raw data are accessible as supplementary file: Raw data supplementary material S1.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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