Research Article

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Developing *Drosophila suzukii* management programs for sweet cherry in the western United States

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Abstract

BACKGROUND: The spotted wing drosophila, *Drosophila suzukii* Matsumura (Diptera: Drosophilidae), is a newly introduced pest of sweet cherry on the west coast of North America which produces about 97% of the value of the US sweet cherry crop. *D. suzukii* initially caused considerable economic loss to cherry growers, who were unaware of this new pest. Little control information was available at the time of initial infestation. Pest control studies were initiated to examine the materials, timings and application methods to control *D. suzukii* in three major cherry-producing states (California, Oregon and Washington).

RESULTS: Three classes of registered insecticides, organophosphates, pyrethroids and spinosyns, have demonstrated good topical or residual activity against *D. suzukii*. Neonicotinoids and the systemic organophosphate dimethoate appear to be able to kill eggs or larvae in fruit. Preliminary timing studies indicate that at least two preharvest insecticide sprays are required to obtain control of *D. suzukii* in California cherry orchards. Aerially applied malathion ULV (ultra-low volume) appears to be a viable control tactic for this pest.

CONCLUSION: The results presented here form the basis for developing *D. suzukii* management programs in the western United States. Additional studies are needed to refine management practices for the different growing regions and conventional versus organic production requirements. Cherry growers will likely need to apply broad-spectrum insecticides in a prophylactic manner until treatment thresholds and monitoring methods have been developed and validated.

Keywords: spotted wing drosophila; *Prunus avium*; organophosphate; pyrethroid; spinosyn; neonicotinoid

1 INTRODUCTION

Spotted wing drosophila, *Drosophila suzukii* Matsumura, was detected in the continental United States in 2008. The first detections were in caneberries and strawberries in Santa Cruz County (Bolda MP, private communication, 2011) and were confirmed as *D. suzukii* by the California Department of Agriculture.[1] The first report of damage to sweet cherries was in southern Santa Clara and northern San Benito counties of California in May 2009. Detections were next made in San Joaquin and Stanislaus counties, the major cherry-producing region of California, and there were a number of reports of serious fruit infestations. In the fall of 2009, *D. suzukii* were trapped in the Mid-Columbia region, Oregon's major cherry-producing area, and in a stone fruit production region near Kelowna, British Columbia, Canada.[2] In the spring of 2010, damage was noted in Tulare and Kern counties, the warmest cherry production regions in California. The first detections were made in the primary cherry production region of Washington (east of the Cascade Mountains) in mid-summer of 2010, although *D. suzukii* had been found in backyards and caneberry-growing regions west of the Cascades in 2009.

Several factors made the situation extremely challenging in California in 2009. The primary production regions were well into harvest before a species identification was made.[1] In the absence of this identification, the infestations in caneberries the previous year (in a different county) did not forecast the problem in sweet cherries. California growers had no warning that crop protection measures would be needed, let alone information on effective materials or timings. The main production districts of California are not infested with western cherry fruit fly, *Rhagoletis indifferens* Curran (Diptera: Tephrididae), so growers cease insecticide applications starting about 14 days after bloom; thus, fruit were unprotected during the vulnerable preharvest period.

In contrast, cherry growers in the Pacific Northwest had several months to prepare monitoring and management programs

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because traps deployed in the fall of 2009 detected the presence of *D. suzukii* in the region before the 2010 season. These preparations were made despite the prediction that the semi-arid sage-steppe interior fruit-growing districts of eastern Washington would be unsuitable habitats for *D. suzukii*.[3] Another mitigating factor was that these regions historically provided more or less continuous insecticide coverage for control of *R. indifferens* from the time the cherries turned pale yellow through (and sometimes after) harvest. However, in the past 5 years, Washington and Oregon growers had widely adopted the use of spinosad bait (GF-120 NF; Dow AgroSciences, Indianapolis, IN) for control of *R. indifferans*.[4–6] This material, sprayed in coarse droplets as a bait containing the active ingredient, can be applied by an all-terrain vehicle and an inexpensive sprayer. The dose per hectare of the active ingredient was very low, and, coupled with the method of application, had minimal impact on beneficial arthropods. Neonicotinoid insecticides were also found to be effective in controlling damage from *R. indifferans* and were routinely used preharvest. These compounds were found to be toxic by ingestion[7] and also prevented larval development in fruit in several of *Rhagoletis* spp.[5,8,9] Spinosad bait had largely displaced the use of broad-spectrum organophosphates in the immediate preharvest period, primarily malathion applied by air.[10] The use of post-harvest sprays for sanitation[11] was commonly used, but neonicotinoids had displaced dimethoate because of phytotoxicity concerns with the latter.[4]

The invasion of this new pest species disrupted existing pest management programs, and was extremely problematic for the cherry-growing industries of all regions affected. The species identification, coupled with the common name 'cherry vinegar fly',[1] indicated sweet cherries as a high-risk crop for attack by *D. suzukii*. The three states represented in this study account for 97% of the value of US sweet cherry production[12] with a potentially high crop loss if *D. suzukii* is left uncontrolled.[13] High-value export markets were considered to be at risk because the distribution and quarantine status of the pest was still being determined. Most of the primary literature was in Japanese, and virtually inaccessible to the US cherry industry, limiting access to basic information on life history and control. Regulatory, research and outreach channels were hard pressed to respond to this new and devastating pest.

The research presented in this paper represents the initial attempts of researchers located in the three major sweet-cherry-growing states in the western United States to define the most basic parameters for controlling a new pest, including materials, rates and timings. The emphasis in each region reflects the differences in the pest complex and the historical pesticide use patterns used by growers in that region.

2 MATERIALS AND METHODS

2.1 Laboratory bioassays (Washington)

Several types of bioassay using *D. suzukii* were conducted to determine the efficacy of insecticides registered on sweet cherries, as well as one unregistered material. The adults used in the tests were taken from a *D. suzukii* colony maintained in a controlled temperature room kept at 22 °C. The colony was reared on Drosophila medium (formula 4–24; Carolina Biological Supply Company, Burlington, NC) in 50 mL polystyrene tubes (Genesee Scientific, San Diego, CA). The colony originated in September 2010 from a colony kept in the Mid-Columbia Agricultural Research and Extension Center in Hood River, Oregon.

The insecticides used in these bioassays included spinetoram, spinosad 800 g kg^{-1} (Entrust 80W), spinosad bait 0.24 g L^{-1} (GF-120 NF), tolfenpyrad (unregistered), imidacloprid and a botanical insecticide composed of a blend of ground herbs and spices (EF300) (Table 1). All bioassays included a distilled water check. The

Table 1. Chemical name, trade name, manufacturer and amount of active ingredient of insecticides used for control of *D. suzukii* in studies in California, Oregon and Washington in 2010

Chemical class	Trade name	AI	Formulation	Manufacturer
Botanical	EF300	(Ground herbs)	222 g kg^{-1}	USAgriTech, Inc., Las Vegas, NV
Carbamate	Sevin® XLR	Carbaryl	479 g L^{-1}	Bayer CropScience, Research Triangle Park, NC
Diamide	Cyazypyr® 10SE	Cyantraniliprole	100 g L^{-1}	E.I. du Pont de Nemours & Co., Wilmington, DE
METI[a]	Bexar® 1.25SC	Tolfenpyrad	150 g L^{-1}	Nichino America Inc., Wilmington, DE
Neonicotinoid	Assail® 30SG	Acetamiprid	300 g kg^{-1}	United Phosphorus, Inc., King of Prussia, PA
Neonicotinoid	Assail® 70WP	Acetamiprid	700 g kg^{-1}	Cerexagri-Nisso LLC, King of Prussia, PA
Neonicotinoid	Provado® 1.6F	Imidacloprid	192 g L^{-1}	Bayer CropScience, Research Triangle Park, NC
Neonicotinoid	Actara® 25WDG	Thiamethoxam	250 g kg^{-1}	Syngenta Crop Protection, Inc., Greensboro, NC
Organophosphate	Diazinon® 50WP	Diazinon	500 g kg^{-1}	Makhteshim Agan of N. America, Inc., Raleigh, NC
Organophosphate	Dimethoate 2.67EC	Dimethoate	320 g L^{-1}	Drexel Chemical Co., Memphis, TN
Organophosphate	Malathion® 5EC	Malathion	599 g L^{-1}	Arysta LifeScience North America, LLC, Cary, NC
Organophosphate	Malathion® 8F	Malathion	959 g L^{-1}	Gowan Co., Yuma, AZ
Organophosphate	Fyfanon ULV	Malathion	1186 g L^{-1}	Cheminova, Research Triangle Park, NC
Pyrethroid	Baythroid® XL	Beta-cyfluthrin	120 g L^{-1}	Bayer CropScience, Research Triangle Park, NC
Pyrethroid	Danitol® 2.4EC	Fenpropathrin	288 g L^{-1}	Valent U.S.A Corporation, Walnut Creek, CA
Pyrethroid	Warrior II®	Lambda-cyhalothrin	249 g L^{-1}	Syngenta Crop Protection, Inc., Greensboro, NC
Pyrethroid	Pounce® 25WP	Permethrin	250 g kg^{-1}	FMC Corporation, Philadelphia, PA
Pyrethroid	Mustang®	Zeta-cypermethrin	180 g L^{-1}	FMC Corporation, Philadelphia, PA
Spinosyn	Delegate® 25WG	Spinetoram	250 g kg^{-1}	Dow AgroSciences LLC, Indianapolis, IN
Spinosyn	Entrust® 80WP	Spinosad	800 g kg^{-1}	Dow AgroSciences LLC, Indianapolis, IN
Spinosyn	GF-120® NF	Spinosad	0.24 g L^{-1}	Dow AgroSciences LLC, Indianapolis, IN

[a] Mitochondrial complex I electron transport inhibitors.

bioassay arena was a 100 mL plastic portion cup with a 1 cm hole in the lid covered with micropore tape for ventilation. A 10 mL vial was filled with a 500 mL L^{-1} solution of honey and water, plugged with a 3 cm piece of dental wicking and inverted into a second 1 cm hole in the lid of the arena. In addition to the honey–water solution, 2.5 mL of Drosophila medium was placed in the arena to sustain the adult flies during the course of the bioassay.

Bioassay A tested tolfenpyrad (315 mg L^{-1}) and spinetoram (131 mg L^{-1}) against adult *D. suzukii*. Adults were anesthetized with CO_2, sexed and transferred to a plastic portion cup containing Drosophila medium in groups of 20 males or females (sexes tested separately). The flies were reanesthetized and sprayed along with the arena and food source (topical plus residual) with 2 mL of the appropriate solution in a Potter spray tower (Burkard Scientific, Uxbridge, UK) operated at 44 815 Pa. Each pesticide/sex combination was replicated 5 times. Flies were evaluated for mortality after 48 h. Bioassay B was similar in all respects to the previous one, except that, immediately after the spray was applied, the flies were transferred to a clean arena with food and honey–water (topical only). Bioassay C tested three rates of spinosad (135, 108 and 75 mg L^{-1}) by topical exposure only (as for bioassay B). Males and females were tested separately. Bioassay D tested two rates of EF300 (2800 and 1700 mg L^{-1}) using the same procedures as bioassay C, except that, owing to the large particle size, the pesticide solutions were applied with a piston-type hand sprayer (0.75 mL per arena). Bioassay E compared two rates of spinosad bait (240 and 40 mg L^{-1}) using the same bioassay arenas as described above. One 25 µL droplet (about 7 mm diameter) was applied to the bottom of the arena. Five males and five females were added to the arena, and mortality was evaluated after 48 h. Bioassay F tested fruit protection as evidenced by oviposition punctures. Fresh 'Bing' cherries were treated by dipping either the whole fruit or half the fruit for 5 s into a solution of two rates of spinosad (180 and 135 mg L^{-1}) or imidacloprid (120 mg L^{-1}). The fruit was air dried and then suspended in the plastic portion cup by taping the stem to the lid. Adult flies (three females and two males) were anesthetized and added to the arena. Oviposition punctures were counted after 48 h.

2.2 Field–laboratory bioassays (California)

Three trials were conducted in a commercial 'Bing' cherry orchard in Tracy, California, in 2010. The first trial (applied 12 July) included organophosphate (malathion, diazinon) and spinosyn (spinetoram, spinosad) insecticides; the second trial (applied 10 August) included pyrethroid insecticides (fenpropathrin, zeta-cypermethrin, lambda-cyhalothrin, beta-cyfluthrin, permethrin); the third trial (applied 31 August) included neonicotinoid (acetamiprid, imidacloprid, thiamethoxam) and carbamate (carbaryl) insecticides (Table 1). Treatments were replicated 4 times in a randomized complete block design, with a replicate consisting of an individual tree. There was at least one untreated buffer tree between each replicate. Experimental treatments were applied with a handgun orchard sprayer with a finished spray volume of 1870 L ha^{-1}. An untreated check was included in each trial.

Field-treated leaves were exposed to laboratory-reared adult *D. suzukii*. The laboratory culture was taken originally from an infested raspberry field near Watsonville, California, in the spring of 2009. The colony was maintained on a corn meal, agar, molasses and yeast diet. The flies were reared on diet in a 180 mL polypropylene tube with foam caps (Fisher Scientific, Pittsburgh, PA). The colony was maintained in an environmental cabinet at 23 °C with a 16 : 8 (L : D) photoperiod. Culture tubes were replaced about every 2 weeks.

Treated leaves were collected individually from the field and transported to the laboratory in ice chests. An individual treated leaf was placed in a 3.79 L plastic container. The leaf petiole was placed in a 2 mL microcentrifuge tube (Fisher Scientific, Pittsburgh, PA) containing water to maintain leaf viability. The microcentrifuge tube and leaf were mounted in a sponge base and secured to the center of the plastic container with double-sided tape. The cages contained a small amount of diet (corn meal, yeast, molasses, sugar and agar) and water-soaked sponges in 50 × 9 mm petri dishes (Falcon, Franklin Lakes, NJ) to provide food and moisture. Adult laboratory-reared *D. suzukii* (ten males and ten females) were exposed to treated foliage at 1, 3 and 7 days after treatment (DAT). The adults were introduced into the container through a slit in the organdy top, which was then sealed. The top of the cage was covered with plastic wrap to maintain high relative humidity. After 24 h of exposure, the flies were removed from the containers and mortality was recorded by sex. The experiment was conducted at 23.5 °C in a constant temperature cabinet with a 16 : 8 (L : D) photoperiod.

2.3 Field–laboratory bioassay (Oregon)

A trial was conducted in an experimental 'Bing' cherry orchard at the Mid-Columbia Agricultural Research and Extension Center in Hood River, Oregon, in 2010. At the time the trial was conducted, the orchard was not infested with *D. suzukii*, and thus treated fruit and foliage were tested using laboratory-reared flies. A laboratory colony was started in September 2009 by rearing *D. suzukii* from unsprayed blueberries collected in the Willamette Valley, Oregon. The population was reared on artificial diet (formula 4–24 instant Drosophila medium; Carolina Biological Supply Co., Burlington, NC) confined in 30 × 95 mm capped polystyrene tubes (Genesee Scientific, San Diego, CA). Adult flies were transferred to new diet tubes every 3 weeks to maintain the colony. The colony was kept at 21 °C with a 14 : 10 h L : D photoperiod. Twelve hours before use in bioassays, flies were removed from the culture tubes and held in separate containers. The holding containers were supplied with both plain water and 45% sucrose–water solution dispensed by tubes with dental wicking, but without an oviposition substrate.

The treatments consisted of single applications of various candidate pesticides applied to single-tree replicates (Tables 1 and 5). Treatments were replicated 4 times in a randomized complete block design, with at least one untreated buffer tree between each replicate. Pesticides were applied on 27 July with an airblast orchard sprayer operating at a spray volume of 935 L ha^{-1}. Leaves and fruit were collected from the field 1 DAT and transported to the laboratory in ice chests. Assay arenas were 473 mL plastic containers (Reynolds Del-Pak, Mt Vernon, KY) that had six 4 mm holes punched into both the lid and side of the arena. These holes were covered with a fine nylon mesh attached with glue (No. 2 013 605; Ace Gluesticks, Oak Brook, IL).

For the leaf assays, 3–5 leaves were used to line the bottom and sides of the assay arena (ca 213 cm^2), with the exception of the mesh-covered ventilation holes. Water was supplied during the assay by gluing a 5 cm dental wick to the inside of the lid and then saturating it with water. Fifteen unsexed laboratory-reared adult *D. suzukii* were anesthetized with CO_2 for 3 s and then added to each of the arenas. Mortality was assessed 19 h after exposure.

For the fruit assays, five fruit per replicate were exposed to 15 laboratory-reared adult *D. suzukii*. The fruit were collected individually from the field and transported to the laboratory in ice

chests. The fruit were taped to the bottom of 473 mL ventilated plastic containers provisioned with water as described above. The flies were anesthetized for 3 s and then added to the test arenas. Adult mortality was assessed after 16 and 40 h of exposure. After 4 days of exposure, the flies were removed from the containers and the number of egg respiratory filaments protruding from the fruit surface was recorded. The fruit were held at laboratory temperature (21 °C) with a 16 : 8 (L : D) photoperiod for 2 weeks, and the number of emerged adults was recorded.

2.4 Field–laboratory bioassay (aerial application) (Oregon)

This test was conducted to evaluate whether malathion ULV (ultra-low volume) (Table 1) applied aerially would penetrate mature cherry canopies, thus providing an indication of potential control of *D. suzukii*. This potential was measured in three ways: (1) by evaluating spray coverage in the canopy; (2) by assessing the mortality of caged adults exposed at the time of application; (3) by using field-treated leaves in a laboratory bioassay.

The experiment was conducted in a mature 'Bing' cherry orchard in The Dalles, Oregon. The orchard was planted to 4.2 × 6.4 m (within and between rows) and pruned to 4.3 m tall. Two replicates were 15 years old, and the third replicate was 24 years old. Each replicate block was 90 m wide and 180 m long, with three replicates per treatment (malathion and untreated check).

Before treatment, 5.1 × 7.5 cm single-sided oil-sensitive spray-cards (Gemplers, Madison, Wisconsin) and sentinel *D. suzukii* flies were placed in the test orchards. The spray-cards were stapled to leaves located at the top, middle and bottom of trees, both inside and outside the canopy, and were placed as close to vertical as possible with the sensitive side facing outwards (away from the trunk). Spray cards were also fastened to 10 cm wooden stakes placed in the ground under the tree drip-line or in the drive-row perpendicular to the horizon and the tree row. Eight cards per tree were deployed on four trees per replicate, or 32 cards per replicate (96 per treatment).

Sentinel flies were deployed in two types of arena prior to treatment. The first arena consisted of a nylon screen cage (15 × 15 × 0.6 cm) containing ten unsexed laboratory-reared flies. The second arena was a 1.25 × 7 cm strip of white sticky card (AlphaScents, West Linn, Oregon) with five flies attached by their dorsal surface. Flies were anesthetized with CO_2 for 3 s and then attached to the card with a soft brush. Three cages and three cards were placed in each replicate, in the interior, lower canopy of the tree.

Malathion ULV (1.37 kg AI ha^{-1}) was applied undiluted (0.47 L ha^{-1}) on 27 May (14.4 °C, winds 0.8–2.9 km h^{-1}) by fixed-wing aircraft. One hour after application, leaves were collected from the treated and untreated replicates and kept cool until use. The leaves were used in a bioassay of adult flies as described above (Section 2.3).

2.5 Preharvest field trial (California)

The trial was conducted in a commercial 'Bing' cherry orchard near Gilroy, California. There were five treatments consisting of one, two, three or four applications during the post-bloom period, and an untreated check. The same material and rate were used at each timing (A to D). The application timings (earliest to latest) were: (A) diazinon 2.2 kg ha^{-1} (23 April); (B) zeta-cypermethrin, 28 g ha^{-1} (12 May); (C) fenpropathrin 488 g ha^{-1} (19 May); (D) malathion 2.6 kg ha^{-1} (4 June) (Table 1). The first timing (23 April) coincided with one normally used to control black cherry aphid, *Myzus cerasi* F.; the other timings coincided with the different stages of fruit maturity (12 May, straw; 19 May, pink; 4 June, red). The treatments consisted of additive numbers of spray timings, and thus the first treatment had four applications (A to D), the second had three applications (B to D), the third had two applications (C to D) and the fourth was only a single application (D).

The five treatments were replicated 3 times in a randomized complete block design. Each replicate was 5–7 rows wide by 7–8 trees long, with at least one 'Black Tartarian' pollinizer per replicate. Treatments were applied with an airblast speed sprayer with a spray volume of approximately 2340 L ha^{-1}. All treatments included 3.5 L ha^{-1} of a hydrolyzed corn gluten meal used as a feeding stimulant (Nu-Lure insect bait, 444 g AI kg^{-1}; Miller Chemical & Fertilizer Corporation, Hanover, PA).

A trap was placed in the center of each plot to track seasonal population trends of adult *D. suzukii*. The trap was a 950 mL plastic container with holes drilled near the top and baited with 300 mL of apple cider vinegar (ACV). The traps were deployed on 23 April (the timing of the first application) and monitored weekly until 6 July, about 4 weeks after the beginning of harvest. Trap contents were collected weekly, and the bait was replaced. Adult *D. suzukii* were sexed and counted under magnification.

Fruit infestation was measured weekly by crushing 100 cherries in a brown sugar solution and then extracting the larvae by pouring the contents onto filter paper in a Buchner funnel. Larvae were counted under magnification. Fruit sampling started with the earlier-maturing 'Black Tartarian' (18 May to 1 June) and continued with 'Bing' fruit (9 June to 6 July). Harvest began on 7 June and was completed by 22 June.

2.6 Post-harvest field trial (Washington)

A post-harvest trial was conducted in a commercial 'Sweetheart' cherry orchard in Monitor, Washington. The pesticides tested were dimethoate and imidacloprid (Table 1); these materials were normally used during this period for killing any remaining *R. indifferans* larvae in fruit remaining on the trees after harvest. Treatments were replicated 3 times in a randomized complete block design. Each replicate consisted of nine or ten trees in a single row, with two untreated buffer rows between each treated row. A single application was made on 23 August 2010 with an airblast orchard sprayer operating at a spray volume of 935 L ha^{-1}. The experiment included an untreated check. A sample of fruit (average of 35 per replicate) was taken on 2 September, placed in plastic boxes with ventilated lids and held at 20 °C. Adults emerging from the fruit were collected periodically throughout a 2 week period after removal from the trees, at which time the adult drosophilids were counted and identified to species (*D. suzukii* or other *Drosophila* spp.).

Data Analysis. Bioassay and field data were analyzed using analysis of variance (ANOVA) and mean separation with Fisher's LSD ($\alpha = 0.05$),[14] using the appropriate models for a completely randomized design (laboratory bioassays) or a randomized complete block design (field–laboratory and field experiments). Laboratory bioassay mortality data were transformed arcsine[sqrt(% mortality × 0.01)] before analysis; other data were transformed as necessary to satisfy the assumptions of ANOVA. An additional ANOVA was carried out to determine the effect of sex on mortality in the California and Washington bioassays using a factorial design with main effects of pesticide and sex.

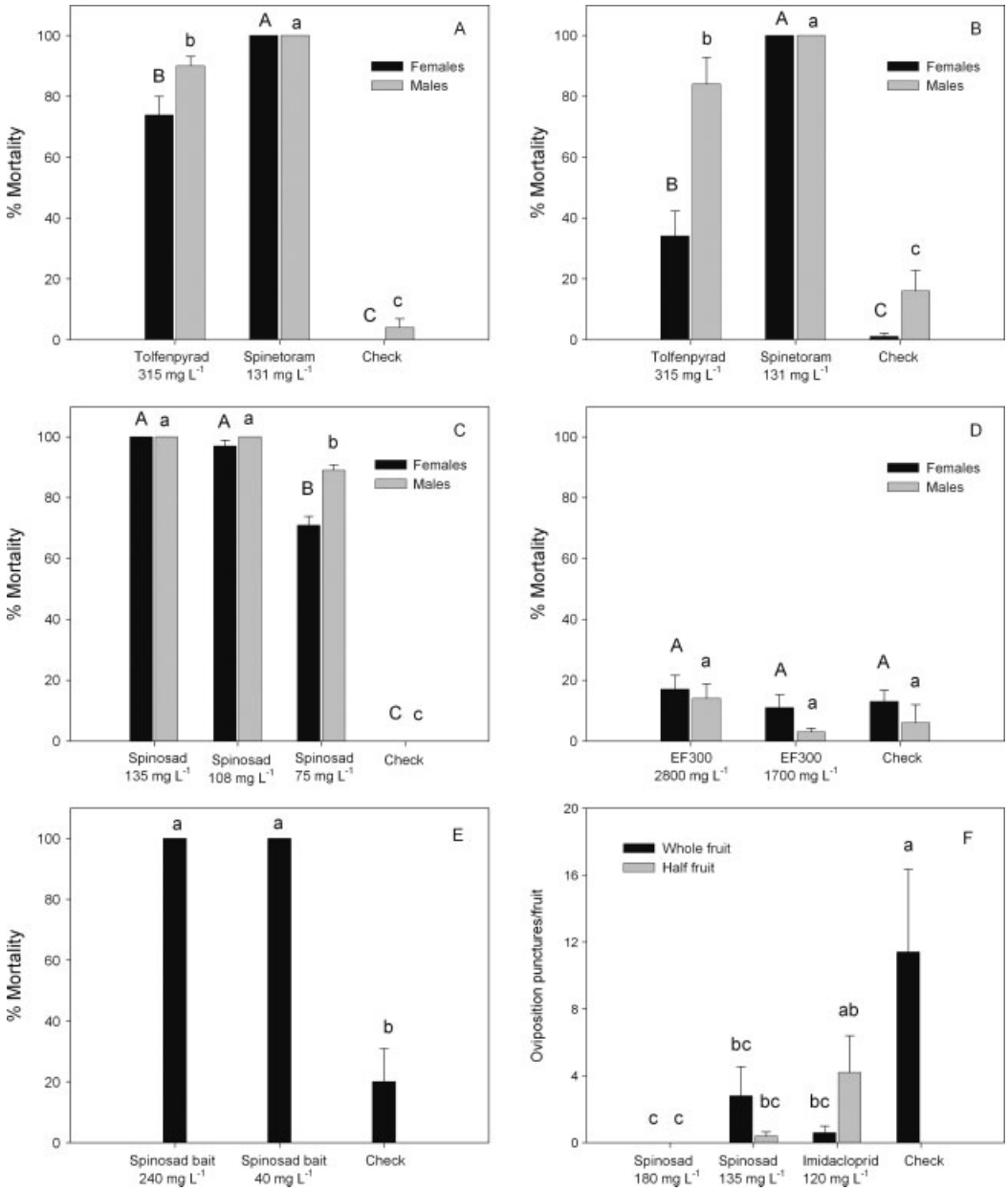

Figure 1. Mean percentage mortality (± SE) of adult *D. suzukii* or oviposition punctures per fruit following exposure to various pesticides in laboratory bioassays. Upper-case letters indicate statistical differences for females; lower-case letters are for males. Bioassay A was topical plus residual; bioassays B to D were topical only; bioassaya E and F were residual only.

3 RESULTS

3.1 Laboratory bioassays (Washington)

Tolfenpyrad and spinetoram (topical plus residual) caused high levels of mortality in *D. suzukii* adults, although mortality was higher in the spinetoram treatments (Fig. 1A). The same pattern occurred with females ($F_{2,12} = 362.42$, $P < 0.001$) as with males ($F_{2,12} = 138.60$, $P < 0.001$). When the adults were exposed topically only (Fig. 1B), spinetoram still caused 100% mortality, but percentage mortality in the tolfenpyrad treatments was lower, about 34% for the females ($F_{2,12} = 167.16, P < 0.001$) and 74% for the males ($F_{2,12} = 44.66, P < 0.001$). Spinosad caused nearly 100% mortality in both males ($F_{3,16} = 2209$, $P < 0.001$) and females ($F_{3,16} = 353, P < 0.001$) at the two higher rates tested (135 mL L^{-1}, 108 mL L^{-1}), but mortality was significantly lower for both sexes at the lowest rate (75 mL L^{-1}) (Fig. 1C). Mortality caused by EF300 was not significantly higher than the untreated check at either rate (males: $F_{2,12} = 2.66, P = 0.11$; females: $F_{2,12} = 0.62, P = 0.55$) (Fig. 1D). Spinosad bait at both rates (240 and 40 mg L^{-1}) caused 100% mortality in the adults (males and females) exposed to a single droplet in the arena ($F_{2,12} = 52.37$, $P < 0.001$) (Fig. 1E). Imidacloprid and spinosad (both rates) caused significant reductions in oviposition punctures in wholly or partially treated fruit in comparison with the untreated check (Fig. 1F), with the exception of half-fruits treated with imidacloprid ($F_{6,28} = 5.21, P = 0.001$).

In bioassays A to D, where males and females were tested separately, males consistently suffered higher mortality that females (A: $F_{1,24} = 7.42, P = 0.012$; B: $F_{1,24} = 24.96, P < 0.001$; C: $F_{1,32} = 17.30, P < 0.001$; D: $F_{1,34} = 4.49, P = 0.04$).

Table 2. Adult *D. suzukii* mortality following exposure to cherry leaves treated on 12 July 2010 with spinosyn and organophosphate insecticides at 1, 3, and 7 days after treatment, Tracy, California[a]

Treatment	g AI ha^{-1}	1 DAT	3 DAT	7 DAT
Spinetoram	123	19.9 (±11.4) c	16.2 (±8.7) bc	3.6 (±2.3) ab
Spinosad	140	21.3 (±9.9) bc	45.3 (±11.8) a	10.1 (±5.3) a
Diazinon	2200	50.6 (±12.3) a	12.8 (±4.4) bc	3.7 (±2.4) ab
Malathion	2600	45.6 (±8.2) ab	25.1 (±8.5) ab	1.3 (±1.3) b
Untreated check	–	7.6 (±4.5) c	1.2 (±1.2) c	0.0 (±0.0) b
	F	4.15	2.93	2.41
	P	0.015	0.049	0.087
	df	4, 12	4, 12	4, 12

[a] Means within columns not followed by the same letter are significantly different according to Fisher's unprotected LSD ($\alpha = 0.05$).

Table 3. Adult *D. suzukii* mortality following exposure to cherry leaves on 10 August 2010 with pyrethroid insecticides at 1, 3 and 7 days after treatment, Tracy, California[a]

Treatment	g AI ha^{-1}	1 DAT	3 DAT	7 DAT
Fenpropathrin	488	48.7 (±6.1) a	19.7 (±8.5) ab	21.1 (±8.2) ab
Zeta-cypermethrin	38	25.6 (±6.3) b	16.6 (±5.7) ab	16.5 (±1.4) ab
Lambda-cyhalothrin	47	32.9 (±4.8) ab	17.8 (±4.7) ab	33.8 (±10.0) a
Beta-cyfluthrin	25	30.5 (±9.4) ab	29.5 (±7.6) a	26.9 (±1.8) ab
Permethrin	224	22.9 (±6.8) b	21.5 (±7.1) a	13.1 (±4.0) b
Untreated check	–	3.6 (±1.2) c	3.8 (±2.4) b	0.0 (±0.0) c
	F	3.76	1.71	6.17
	P	0.013	0.176	0.001
	df	5, 15	5, 15	5, 15

[a] Means within columns not followed by the same letter are significantly different according to Fisher's unprotected LSD ($\alpha = 0.05$).

Table 4. Adult *D. suzukii* mortality following exposure to cherry leaves treated on 31 August 2010 with neonicotinoid and carbamate insecticides at 1, 3 and 7 days after treatment, Tracy, California[a]

Treatment	g AI ha^{-1}	1 DAT	3 DAT	7 DAT
Acetamiprid	168	8.2 (±2.9) a	16.2 (±6.2) a	15.0 (±5.5) a
Imidacloprid	112	10.7 (±9.0) a	15.3 (±7.4) a	8.2 (±2.3) a
Thiamethoxam	96	3.3 (±2.0) a	10.5 (±3.3) a	5.3 (±3.2) a
Carbaryl	2200	1.3 (±1.3) a	10.9 (±5.1) a	9.5 (±5.5) a
Untreated check	–	4.7 (±2.7) a	6.3 (±2.4) a	5.2 (±3.1) a
	F	1.17	0.71	0.30
	P	0.388	0.663	0.941
	df	4, 12	4, 12	4, 12

[a] Means within columns not followed by the same letter are significantly different according to Fisher's unprotected LSD ($\alpha = 0.05$).

3.2 Field–laboratory bioassays (California)

Trial 1 (spinosyns, organophosphates). There was significantly higher mortality of *D. suzukii* adults exposed 1 DAT to foliage treated with diazinon or malathion in comparison with the untreated check (Table 2). At 3 DAT only spinosad and malathion had significantly higher mortality, and at 7 DAT only spinosad caused significantly higher mortality.

Trial 2 (pyrethroids). All pyrethroid insecticides tested caused significantly greater mortality of adult *D. suzukii* exposed to foliage at 1 DAT in comparison with the untreated check (Table 3). On this date, fenpropathrin caused significantly higher mortality than zeta-cypermethrin and permethrin. At 3 DAT only beta-cyfluthrin and permethrin caused significantly higher mortality than the untreated check, but at 7 DAT all treatments again caused significantly higher mortality than the untreated check, with lambda-cyhalothrin causing significantly more mortality than permethrin.

Trial 3 (neonicotinoids, carbaryl). There was no significant increase in mortality relative to the untreated check in any of the treatments at 1, 3 or 7 DAT (Table 4).

Overall, *D. suzukii* males suffered significantly higher levels of mortality in the three bioassays (trial 1: $F_{1,110} = 6.13$, $P = 0.01$; trial 2: $F_{1,132} = 35.60$, $P < 0.001$; trial 3: $F_{1,109} = 14.10$, $P < 0.001$) than females.

Table 5. Mean percentage of *D. suzukii* mortality after 16 h exposure to treated cherry leaves and 16 and 40 h exposure to treated cherry fruit at Hood River, Oregon, 2010[a]

Treatment	g AI ha^{-1}	16 h (leaves)	16 h (fruit)	40 h (fruit)
Malathion[b]	2800	100.0 (±0.0) a	84.2 (±6.9) a	97.5 (±2.5) a
Malathion[c]	2368	100.0 (±0.0) a	56.1 (±15.1) b	76.7 (±13.4) bcd
Lambda-cyhalothrin	36	79.7 (±7.7) bc	82.5 (±5.7) a	92.9 (±4.7) ab
Spinetoram	79	85.3 (±6.0) bc	81.6 (±8.0) a	95.6 (±2.5) ab
Spinosad[d]	140	97.2 (±2.4) ab	95.0 (±6.9) a	100.0 (±0.0) a
Acetamiprid	167	27.9 (±7.9) d	36.0 (±8.2) bcd	57.0 (±17.1) de
Imidacloprid	112	5.1 (±3.4) f	9.6 (±4.7) de	33.8 (±15.7) ef
Carbaryl	1100	63.8 (±12.7) c	42.4 (±9.1) bc	87.4 (±4.9) c
Cyazypyr	99	23.7 (±11.2) de	21.1 (±10.4) cd	68.2 (±10.0) cd
Untreated check	–	9.1 (±5.5) ef	2.5 (±2.5) e	2.5 (±4.3) f
	F	28.4	15.42	14.03
	P	<0.001	<0.001	<0.001
	df	9, 26	9, 27	9, 27

[a] Means followed by the same letter in a column are not significantly different (Fisher's LSD, $P \leq 0.05$).
[b] Malathion 5EC.
[c] Malathion 8F.
[d] Entrust 80W.

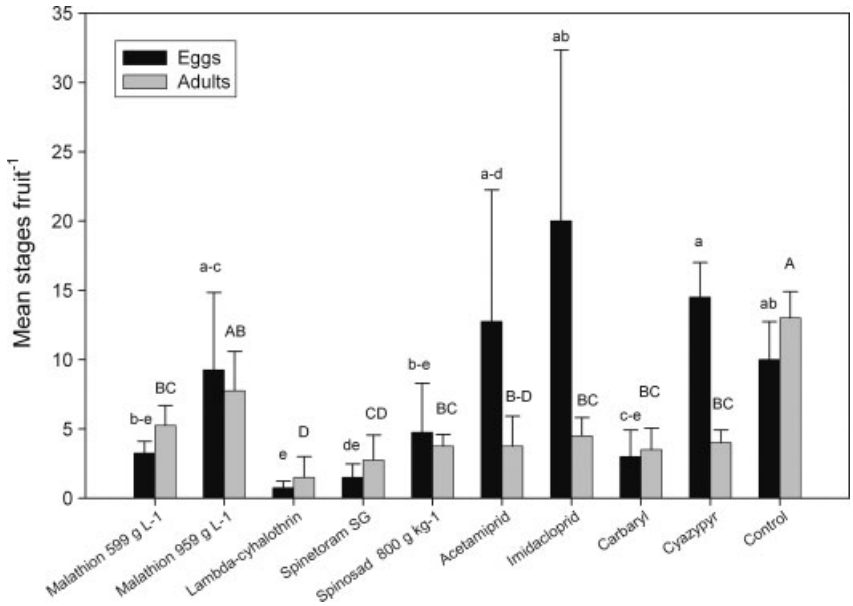

Figure 2. Mean number (± SE) of *D. suzukii* eggs or adults emerging from fruit following exposure to fruit treated with various pesticides, Hood River, 2010. Upper-case letters indicate statistical differences for adults; lowercase letters are for eggs.

3.3 Field–laboratory bioassays (Oregon)

Malathion, spinetoram, spinosad and lambda-cyhalothrin provided good control of adult *D. suzukii* when the flies were exposed to either treated leaves or fruit (Table 5). In the fruit assay, the higher AI rate of malathion (5EC formulation) was more effective than the lower AI rate (8F formulation). Acetamiprid, imidacloprid and cyazypyr did not provide good initial mortality relative to the other insecticides tested. Fly mortality resulting from residual activity of carbaryl on treated leaves was not statistically different from what was observed in the spinetoram or lambda-cyhalothrin treatments, but provided poorer control when exposed to residues on fruit. Mortality of flies exposed to cyazypyr was relatively low at the 16 h assessment but caused intermediate mortality at the 40 h fruit assessment.

Exposure to spinetoram, lambda-cyhalothrin and carbaryl reduced the number of eggs laid in cherries relative to the control ($F_{9,27} = 2.07$, $P = 0.07$) (Fig. 2). The highest egg numbers were found in the imidacloprid, acetamiprid and cyazypyr treatments; however, the number of adults produced in these treatments was not different ($F_{9,27} = 2.72$, $P = 0.02$) from the number produced in treatments with low egg numbers, suggesting reduced adult emergence relative to the number of eggs laid (Fig. 2).

3.4 Field–laboratory bioassays (aerial application)

There were no differences in the average number of ULV droplets (± SEM) cm^{-2} between cards placed outside versus inside the tree canopy (1.4 ± 0.5 and 1.6 ± 0.5 droplets cm^{-2} respectively; $F_{1,84} = 0.48$, $P = 0.49$). In addition, there were no differences in

droplet deposition based on height of the card from ground level (1.7 ± 0.6, 0.9 ± 0.3, 1.2 ± 0.5 and 2.3 ± 0.7 droplets cm^{-2} for cards located on the ground or at the bottom, middle or top of the tree respectively; $F_{3,84} = 0.48$, $P = 0.08$), indicating that droplet deposition was relatively uniform throughout the tree. Higher mortality was observed at 22 h after exposure of flies in cages placed in the malathion ULV test sites than in cages retrieved from untreated trees (71% corrected mortality) ($F_{1,14} = 11.03$, $P = 0.005$). Flies adhered to yellow sticky cards and exposed to malathion ULV also experienced mortality (48% corrected mortality at 36 h after exposure) ($F_{1,8} = 8.01$, $P = 0.02$). There was no difference in mortality of *D. suzukii* exposed to treated or untreated leaves 24 h after exposure (5.6% corrected mortality) ($F_{1,20} = 1.63$, $P = 0.22$), but there was a difference after 50 h of exposure (81.0% corrected mortality) ($F_{1,14} = 29.2$, $P < 0.001$).

3.5 Preharvest field trial (California)

The numbers of *D. suzukii* adults captured in ACV traps rose steeply from 30 April to 7 May and then rapidly decreased to near zero by 1 June (Fig. 3). The adult population remained low for the rest of the study. Thus, a very large *D. suzukii* adult population was active in the orchard in early May. The larval infestation in the treated plots was low from 18 May until 23 June and then increased dramatically. The number of larvae in 100 fruit in the 18 May sample ranged from 4.3 larvae in the untreated check to 0.5 larvae in the three-application treatment regime, but with no significant treatment differences ($F_{4,8} = 1.58$, $P = 0.270$). In the 25 May sample, the untreated check increased to five larvae in 100 fruit and was significantly different from the other treatments, which did not differ significantly ($F_{4,10} = 7.58$, $P = 0.008$). In the 1 June sample, the one-application treatment program had 2.5 larvae in 100 fruit and was significantly greater than all other treatment regimes except for the untreated check ($F_{4,10} = 2.81$, $P = 0.099$). The infestation did not exceed one larva per 100 fruit in the 9 and 15 June samples, with no significant differences among treatments. These low levels occurred about the same time as harvest (7–22 June). Infestation rose markedly in the 23 June sample, with significantly higher levels of infestation (4.7–7.7 larvae) in the one-application regime and untreated check ($F_{4,8} = 5.06$, $P = 0.020$). The difference between the two groups of treatments was even greater in the 28 June ($F_{4,7} = 5.80$, $P = 0.018$) and 6 July samples ($F_{4,8} = 4.86$, $P = 0.022$). Infestation levels were extremely high on the last date (486–542 larvae in the untreated check and single-application treatments, and 10–12 larvae in the two-, three- and four-application regimes), and sampling was terminated owing to rapidly deteriorating fruit condition.

3.6 Post-harvest field trial (Washington)

There was a significant difference among the treatments in mean number of *D. suzukii* produced per treated fruit (Table 6). Fruit treated with dimethoate produced significantly fewer *D. suzukii* adults when compared with the untreated check, but there was no difference between the imidacloprid and the check. Neither treatment reduced the numbers of other *Drosophila* spp. in relation to the check.

Table 6. Adult *D. suzukii* and other *Drosophila* spp. emerging from 'Sweetheart' cherries treated post-harvest, Monitor, Washington, 2010[a]

Treatment	g AI ha^{-1}	Adults fruit^{-1}	
		D. suzukii[b]	Other *Drosophila* spp.[b]
Dimethoate	1497	0.02 (±0.01) b	3.43 (±1.69) a
Imidacloprid	112	0.43 (±0.16) ab	3.22 (±0.74) a
Untreated check	–	1.42 (±0.66) a	5.28 (±1.58) a
	F	6.06	0.40
	P	0.06	0.69
	df	2, 4	2, 4

[a] Means followed by the same letter in a column are not significantly different (Fisher's unprotected LSD, $\alpha = 0.05$).
[b] Data transformed owing to unequal variances {arcsine[sqrt($y \times 0.01$)]}.

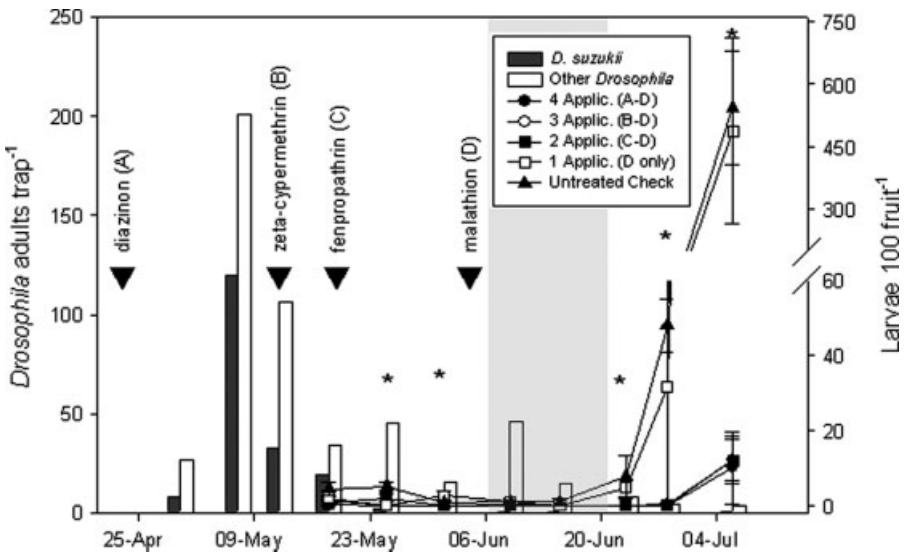

Figure 3. *D. suzukii* larvae 100 fruit^{-1} week^{-1} (± SE) and adults trap^{-1} at Gilroy, California, 2010. Means marked with an asterisk (*) had one or more treatments that were significantly different (Fisher's unprotected LSD); black triangles (▼) indicate spray application timings; shaded box indicates the harvest period.

4 DISCUSSION

In both field and laboratory trials, three classes of registered pesticides emerged as having good topical or residual activity against *D. suzukii*, namely organophosphates, pyrethroids and spinosyns. Laboratory and field trials in small fruit show essentially the same trend.[15] Results with carbaryl (a carbamate) were variable, with very poor control in the laboratory–field bioassays in California, but moderate control when exposed to residues on leaves or fruit in the Oregon trials. This is in contrast to the high levels of mortality found by Bruck *et al.*[15] in topical bioassays. The neonicotinoids tested (acetamprid, imidacloprid and thiamethoxam) caused moderate or low levels of adult mortality when exposed to residues on leaves, but the fruit residue bioassays showed a potentially useful degree of systemic activity for acetamiprid and imidacloprid. The poor adult mortality of acetamiprid and imidacloprid in fruit assays allowed higher levels of oviposition to occur, but with about the same numbers surviving to the adult stage as the other materials tested. A systemic effect has been noted for the neonicotinoids imidacloprid and thiacloprid in tests against *R. indifferans*.[5] This systemic effect was also apparent in the post-harvest field trial in Washington, which provided excellent reduction in survival to the adult stage in the dimethoate treatment, and moderate suppression in the imidacloprid treatment. The results of the California field trial also raised the possibility that preharvest treatments may have a long-term effect on population suppression. The most marked treatment differences in larval infestation occurred well after harvest, and were still evident nearly 7 weeks after the 19 May (fenpropathrin) application common to the three-, four- and five-application treatments. This suggests either that there is a very limited dispersal of adults into treated areas or that the residual or systemic effect is much longer than previously supposed. While the concept needs further investigation, the addition of materials with systemic activity may enhance overall control of this pest.

Two unregistered pesticides also show some potential activity against *D. suzukii*. Cyazypyr performed similarly to the neonicotinoids, in that topical mortality of adults was mediocre but with some indication of systemic activity. This class of pesticides (anthranilic diamides) has been shown to have activity against several species of *Rhagoletis*.[16] Tolfenpyrad had relatively good activity by topical exposure, but residual activity has yet to be determined. Because some of the currently registered pesticides have significant non-target or environmental effects, or are facing increased regulatory restrictions, additional options for control are needed in the future. Having a broad range of modes of action available for this pest will also allow producers to minimize the potential for insecticide resistance.

The widely varying mortalities in bioassays may be largely related to the type of bioassay arena used, as well as the difference between the routes of exposure. The lowest variation generally occurred in the topical laboratory bioassays, where 100% exposure is typical. A relatively minor variation in the set-up of the residual exposure arenas (single leaf in California versus most of the surface area covered with leaves in Oregon) apparently increased the mortality substantially in the latter type. Where untreated surfaces provide refugia from the pesticide residues, sublethal effects such as repellency may play a role. Some of the variability may also be ascribed to using unsexed versus sexed adults. The greater tolerance of female versus male *D. suzukii* in this study was also noted by Bruck *et al.*[15] Given their important role in fruit damage, a conservative approach to bioassays would use only females.

The preharvest field trial indicated that two, three and four sprays provided similar levels of fruit protection; only the treatment including a single preharvest application (malathion) did not provide control. However, it is likely that the first spray timing was too early to affect fruit infestation because the fruit were not yet susceptible to *D. suzukii*.[17] It is expected that the most critical times for sprays are likely to be those applied closer to harvest, although this test was not designed to evaluate all possible numbers, timings and material choices. It should be noted that optimal spray timing and numbers may be different in different growing regions. In California, the *D. suzukii* populations peaked in spring (early May) and were declining as harvest approached in June, whereas populations in Oregon and Washington were non-existent or low in the early part of the season, with the peak populations occurring from mid-August to November (Beers EH and Shearer PW, unpublished), after most of the cherry crop had been harvested.

A second issue illustrated by the California preharvest trial is the relationship between capture of *D. suzukii* in traps and fruit infestation. These appeared to be inversely related; the highest trap captures occurred in spring when fruit infestation levels were low; conversely, the high levels of fruit infestation that occurred in early July coincided with negligible trap captures. The pattern may be unique to California's mild winter climate, but it dictates that, for the moment, trapping data should not be used as an indicator of relative crop risk. In addition, it raises the possibility that traps baited with ACV are less attractive than the natural host, and are an incomplete measure of the seasonal phenology of this species.

Organic cherry growers will face a serious challenge controlling *D. suzukii*. The only organically approved material with a high level of activity is spinosad (Entrust 80W). The current label limits the total amount of active ingredient that can be applied during a growing season, and the length of residual control is not well established under field conditions. Additional materials for *D. suzukii* control in organic systems may be critical for the continued production of these crops.

Adapting the existing cherry insect control program to include *D. suzukii* is a concern for both organic and conventional producers, and the degree of alteration may vary considerably from one region to the next. California growers will have to make several additional pesticide applications during the latter stages of fruit maturity, which they have not had to do in the past. In contrast, the presence of *R. indifferans* in Washington and Oregon cherry-growing regions has necessitated a fairly intensive spray program during the period when this pest is present; this period will largely overlap the period when fruit are susceptible to *D. suzukii*. Growers will need to adjust their choice of insecticides and application methods, and, to a lesser extent, spray timings, to accommodate this new pest. The efficacy of malathion ULV demonstrated in this study is key for preharvest control, because the short preharvest interval provides fruit protection at a critical time. The aerial application method allows rapid coverage of large acreages without the fruit damage or disruption of harvest preparations incurred with ground applications.

Currently, the materials that are effective against *D. suzukii* will also control cherry fruit fly, although the reverse is not always true. For example, the neonicotinoids discussed above are effective against *R. indifferans* but do not provide rapid knockdown of *D. suzukii*. Another example is that of spinosad bait (GF-120). Preliminary trials and grower experience indicated that this material, while highly effective for *Rhagoletis* spp., was not an effective tool for *D. suzukii*, and thus it has been a lower

priority for formal testing. The differences in reproductive biology and behavior of these two species also predict that using this bait formulation is unlikely to be successful (Thistlewood HMA, private communication, 2011), even though the toxicant is clearly effective by topical, residual and ingestion exposure routes. The curtailment of spinosad bait sprays for *Rhagoletis*, with their low environmental impact, rapid application and short preharvest interval, will be a setback to cherry IPM programs.

5 CONCLUSIONS

These experiments provide preliminary data for *D. suzukii* control in sweet cherries, but many key issues have yet to be resolved. Data from laboratory, field–laboratory and field experiments became generally more variable as they approached conditions of typical commercial settings, and the findings under controlled conditions must be tested on a broader scale before firmer conclusions can be drawn. Much about the seasonal pattern of occurrence in the three regions and the relative crop risk needs to be established through further experimentation at all levels. The data presented form the basis for control of *D. suzukii* in the three states, providing cherry producers with a provisional strategy and an array of effective control materials.

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