## HORTICULTURAL ENTOMOLOGY

# Interactions of Light Intensity, Insecticide Concentration, and Time on the Efficacy of Systemic Insecticides in Suppressing Populations of the Sweetpotato Whitefly (Hemiptera: Aleyrodidae) and the Citrus Mealybug (Hemiptera: Pseudococcidae)

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J. Econ. Entomol. 105(2): 505-517 (2012); DOI: http://dx.doi.org/10.1603/EC11278

ABSTRACT The impact of light intensity on the uptake and persistence of the systemic neonicotinoid insecticides, imidacloprid and dinotefuran, were evaluated in poinsettia (Euphorbia pulcherrima Willd.) and yellow sage (Lantana camara L.). Insecticide residues were measured in leaves sampled from the treated plants at four time intervals after treatment to determine the relationship between insecticide concentration and efficacy against two insect pests; sweetpotato whitefly, Bemisia tabaci Gennadius, and the citrus mealybug, Planococcus citri Risso. The insecticides were evaluated at their respective label rate and at the comparable label rate of the other insecticide under two different light environments: ambient and shade. The uptake of dinotefuran into yellow sage was more rapid at both treatment rates than both rates of imidacloprid, resulting in higher percent mortality of whitefly nymphs (89.8–100) compared with imidacloprid (14.1–89.2) across all 4 wk. Additionally, plants that received both rates of dinotefuran had fewer whitefly pupae (<1.0) at week 4 compared with imidacloprid-treated plants (23.7–25.3). The uptake of dinotefuran into poinsettia plants was also more rapid and resulted in quicker and higher percent mortality of whitefly nymphs (89.5-99.6) compared with imidacloprid (14.1-89.2) across all 4 wk. However, despite efficient uptake, the efficacy of both systemic insecticides was less for citrus mealybug where percent mortality values were <50% among all the treatments across the 4 wk. The use of the two systemic insecticides evaluated in regards to pest management in horticultural cropping systems is discussed.

KEY WORDS greenhouse, floriculture, systemic insecticide, light intensity, pest management

Floricultural crops grown in greenhouses are susceptible to attack by an array of hemipteran insect pests such as aphids, whiteflies, and mealybugs (Brodsgaard and Albajes 1999, Parrella 1999). These insect pests withdraw plant fluids directly from the phloem sieve tubes (food-conducting tissues) using their stylet-like mouthparts, resulting in plant stunting, leaf yellowing, and leaf distortion (Sur and Stork 2003, Jeschke and Nauen 2008). The use of systemic insecticides is an important pest management strategy for suppression of phloem-feeders because systemic activity directly exploits the feeding behavior of this group of insects. Systemic insecticides are commonly applied preventatively to the growing medium as a drench or granule for uptake or absorption through the roots, and then translocated throughout the plant via the vascular system (Bennett 1949, David and Gardiner 1951, Ru-

Systemic insecticides commercially available for use in greenhouses and labeled for growing medium applications include imidacloprid (Marathon: OHP, Inc., Mainland, PA), thiamethoxam (Flagship: Syngenta Crop Protection, Inc., Greensboro, NC), and dinotefuran (Safari: Valent USA Corp., Walnut Creek, CA). These are all neonicotinoid-based insecticides with similar physical and molecular properties, and a mode of action (agonists at the insect nicotinic acetylcholine receptor) analogous to nicotine (Elbert and Overbeck 1990, Tomizawa and Yamamoto 1993, Zhang et al. 2000, Tomizawa and Casida 2003, Kaane et al. 2005, Hollingworth and Treacy 2006). However, the neonicotinoids differ widely in regards to water solubility (Sur and Stork 2003, Jeschke and Nauen 2008, Cloyd and Bethke 2011). For example, dinotefuran is ≈80-fold more soluble in water than imidacloprid, which may affect uptake and translocation in plants (Byrne et al. 2010). Water is essential to mobilize systemic insecticides for uptake through the

dinsky 1959, Norris 1967). During the feeding process, insects imbibe the active ingredient and are killed after ingesting a lethal concentration (Ware and Whitacre 2004, Cloyd 2010).

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roots and distribute them throughout plant tissues via the transpiration stream (Norris 1967).

Light intensity, temperature, and relative humidity directly affect the transpiration rate of plants (Thut 1939, Pallas et al. 1967, Agata et al. 1985), and may also influence absorption and translocation of systemic insecticides within plants (Jeppson 1953, Reynolds 1954). In fact, previous literature has suggested that environmental factors such as light intensity may influence transport of the active ingredient through plant tissues (Wedding 1953, Reynolds 1954, Bennett and Thomas 1954, Bennett 1957). However, it is difficult to evaluate separately the roles of light intensity and temperature on plant transpiration, and thus systemic insecticide uptake and translocation, in a greenhouse environment. These two environmental parameters are closely linked because wavelengths of light energy are converted to heat energy, raising the temperature in greenhouse environments (Grange and Hand 1987). In addition, the amount of moisture in the air directly influences the plant transpiration rate by impacting evaporative demand.

In this study, we measured light intensity, temperature, and relative humidity (RH) to 1) adequately characterize the treatment environments and 2) allow for calculation of vapor pressure deficit (VPD), which is the difference (deficit) between the amount of moisture in the air and how much moisture the air can hold when saturated, and is responsible for establishing the flow of water vapor in a system. The water loss from leaves is governed by the vapor pressure gradient, from the leaf to the air, which mainly depends on the VPD (Grange and Hand 1987). Because water vapor is lost primarily through the stomates on leaf surfaces, it is helpful to understand the extent of stomatal opening so as to evaluate the degree of transpiration (Kirkham 2005, p. 392). As such, we measured stomatal resistance, which ranges from 0.02 s/cm (low plant stress) to 40 s/cm (high plant stress) (Kirkham 2011, p. 149), and is dependent on the VPD.

Competitive enzyme-linked immunosorbent assays (ELISA) specific for the detection of neonicotinoids have been used to monitor the uptake and translocation of these systemic insecticides in plant tissues. This technique has been especially valuable in determining threshold insecticide concentrations necessary to suppress insect pest populations feeding on horticultural crops (Byrne et al. 2005a,b). In this study, ELISA was used to quantify concentrations of imidacloprid and dinotefuran in leaf tissue sampled from plants treated with these systemic insecticides that were maintained under the different light intensities.

The objectives of this study were to 1) evaluate the systemic activity or movement of the two insecticides under two different light intensities and 2) assess their ability to suppress populations of the sweetpotato whitefly, *Bemisia tabaci* Gennadius and the citrus mealybug, *Planococcus citri* Risso on yellow sage, *Lantana camara* L., and poinsettia, *Euphorbia pulcherrima* Willd. ex Klotzsch plants.

## **Materials and Methods**

Three experiments were conducted to assess the systemic activity associated with two neonicotinoidbased insecticides that differ in water solubility, imidaeloprid [Marathon 1% G (granule); OHP, Inc.] and dinotefuran [Safari 20 SG (soluble granule); Valent USA Corp.], in yellow sage (*L. camara*) and poinsettia (E. pulcherrima) plants when exposed to two discrete light environments: ambient and shade. For all experiments, plants were grown in 15.2-cm round, green plastic azalea containers (ITML Horticultural Products, Inc., Brantford, ON; volume of 1325 cm<sup>3</sup>) with Fafard Number 2 growing medium (Conrad Fafard, Inc., Agawam, MA) consisting of 65% Canadian sphagnum peat moss and 35% perlite and vermiculite. Plants were watered as needed, and fertilized, in general, during every irrigation using 200 ppm N from Peter's 20-10-20 (20N-8.3P-8.8K) (Everris, Inc., Marysville, OH).

Four insecticide rates were used in each experiment, all applied to plants in the 15.2-cm containers: the designated label rate of each systemic insecticide (imidacloprid at 0.014 g active ingredient/container and dinotefuran at 0.027 g active ingredient/container) and comparable rates (imidacloprid at 0.027 g active ingredient/container and dinotefuran at 0.014 g active ingredient/container). The comparable rates were used in the study to allow comparisons of the systemic insecticides at equivalent application rates. A distilled water control was also included. Plants were destructively sampled at 1, 2, 3, and 4 wk after application of the systemic insecticide treatments in each experiment.

All treatment combinations were replicated four times, using four replicate ambient and four replicate shade cages. Therefore, 160 plants were used in each experiment [2 light levels  $\times$  (four insecticide rates + 1 distilled water control)  $\times$  four sample dates  $\times$  4 replications with 40 plants sampled per week over a 4-wk period. The light treatment cages consisted of polyvinyl chloride (PVC) square frames  $(1.2 \times 1.2 \times$ 1.2 m) that were covered with either clear 3-ml polyethylene (DuraGreen; DuraGreen Marketing Inc. LLC, Mount Dora, FL) for the ambient treatments, or clear polyethylene plus three layers of 52% heavy white knit shade cloth (PAK Unlimited Inc., Cornelia, GA) for the shade treatments. A HOBO data logger (Onset Computer Corp., Bourne, MA) was placed in each of the eight cages to record light intensity, temperature and RH every 30 min. This information was used to calculate the light intensity index for each experiment, which is the cumulative addition of light intensity measured every 30 min between 7:00 a.m. and 7:00 p.m. for experiments 1 and 2, and 7:00 a.m. and 5:00 p.m. for experiment 3. The VPD, which was explained above, was calculated by using Murray's equation (Murray 1967);

$$VPD = \frac{RH}{100} \!\! \left( \, 0.61078 \, \, exp^{\frac{17.269*TempAir}{237.3+TempAir}} \right) \label{eq:VPD}$$

Stomatal resistance was measured before each weekly sample date using a Decagon SC-1 leaf porometer

(Decagon Devices, Inc., Pullman, WA) on three fully expanded leaves per plant (one each from top, middle, and bottom strata) and results were averaged. Efficacy of both systemic insecticides against the sweetpotato whitefly (*B. tabaci*) and citrus mealybug (*P. citri*) was determined based on percent mortality (number of dead divided by the total number on each plant) sampled 1, 2, 3, and 4 wk after application of the insecticide treatments. Plant growth data associated with fresh and dry weight (dried at 70°C) of whole above-ground plant parts was determined at each sampling date.

Experiment 1: Sweetpotato Whitefly (B. tabaci) and Yellow Sage (*L. camara*). Rooted cuttings of yellow sage 'Pink Caprice' (Buckley Growers, Taylorville, IL) were transplanted on 16 February 2009. Plants were pinched to improve uniformity (so plants were similar in size) and minimize branching on 9 March 2009. Yellow sage plants were infested on 10 March 2009 with whiteflies by placing all plants into a  $1.2 \times 1.2 \times 3.0$ -m cage constructed of PVC pipe frame and covered with antivirus insect screening  $50 \times 25$ (0.2 × 0.8 mm; Green-tek, Edgerton, WI) that contained an active whitefly colony. The whiteflies were originally obtained from Keith Nursery (Marissa, IL) and were identified as sweetpotato whitefly (Byrne et al. 1995). Infested yellow sage plants were moved into the light treatment cages on 25 March 2009 and then the systemic insecticide treatments were applied on 26 March 2009. Stomatal resistance was measured on 1 April (week 1), 8 April (week 2), 15 April (week 3), and 22 April 2009 (week 4). Plants were sampled on 2 April (week 1), 9 April (week 2), 16 April (week 3), and 23 April (week 4), and the number of live and dead whitefly nymphs were counted and recorded per plant as well as the number of live pupae on week 4. In addition, six leaves were collected from each plant on which whiteflies were present. These leaves were used to measure the concentration of the active ingredient of the two systemic insecticides in plant tissues via the ELISA assay.

Experiment 2: Sweetpotato Whitefly (B. tabaci) and Poinsettia (E. pulcherrima). Rooted cuttings of poinsettia 'Infinity Red' (Dummen USA; Hilliard, OH) were transplanted on 9 August 2009 and pinched to 6.0 cm (±1.5 cm), measured from the edge of the container to the top of the foliage canopy, on 25 August 2009 leaving ≈7 nodes per plant. Poinsettia plants were placed on 28 September 2009 in the infestation cage described for experiment 1 that contained the active whitefly colony. Infested poinsettias were moved into the light treatment cages and treated with the systemic insecticides on 6 October 2009 as in experiment 1. Stomatal resistance was measured on 13 October (week 1), 19 October (week 2), 26 October (week 3), and 2 November 2009 (week 4). Plants were sampled on 13 October (week 1), 20 October (week 2), 27 October (week 3), and 2 November (week 4), and the number of live and dead whitefly nymphs were counted and recorded per plant.

Experiment 3: Citrus Mealybug (P. citri) and Poinsettia (E. pulcherrima). Rooted cuttings of poinsettia 'Prestige Red' (Paul Ecke Ranch; Encinitas, CA) were transplanted on 28 August 2009 and pinched to 6.0 cm  $(\pm 1.5 \text{ cm})$ , measured from the edge of the container to the top of the foliage canopy, on 11 September 2009 leaving ≈7 nodes per plant. Poinsettias were moved into the light treatment cages on 16 November 2009 and inoculated with 20 citrus mealybugs (second to third instars) per plant on 18 November 2009 from a laboratory-reared colony (Kansas State University, Manhattan, KS). Stomatal resistance was measured on 24 November (week 1), 3 December (week 2), 10 December (week 3), and 16 December (week 4). Systemic insecticide treatments were applied on 20 November 2009 and plants were sampled on 27 November (week 1), 4 December (week 2), 11 December (week 3), and 18 December (week 4), and the number of live and dead mealybugs (excluding eggs) were counted and recorded per plant.

Insecticide Quantification in Leaf Tissue. The active ingredients, imidacloprid and dinotefuran, were extracted from yellow sage (experiment 1) and poinsettia (experiments 2 and 3) leaves using similar procedures. Leaves were harvested from portions of every plant that contained the respective insect pest (sweetpotato whitefly and citrus mealybug) associated with each experiment. A 1.0 cm<sup>2</sup> cork borer was used to excise disks from leaves sampled from each plant or experimental unit. The leaf disks were inserted into 2-ml microcentrifuge tubes (Fisherbrand; Catalog No. 02-681-321) containing 1.5 ml of methanol (100%). The disks were homogenized using a polypropylene pellet pestle (Kontes, K749520-0000), which was placed on an orbital shaker and then agitated for 12 h at 25°C. There were four replicates for each treatment.

After extraction, the samples were briefly centrifuged to pellet the plant particulate matter. Ten microliter aliquots from each extract were added to 1.5 ml microcentrifuge tubes, dried completely in a TurboVap LV evaporator (Caliper Life Sciences, Hopkinton, MA), and then reconstituted in a 0.05% aqueous solution of Triton X-100 before analysis by ELISA. The reconstituted extracts were used directly for insecticide quantification.

The concentrations of imidacloprid and dinotefuran within the leaf extracts were determined using a competitive ELISA technique. The ELISA kits are available commercially for both imidacloprid (QuantiPlate Kit for Imidacloprid: Catalog No. EP 006; EnviroLogix Inc., Portland, ME) and dinotefuran (SmartAssay Series Dinotefuran Test Kit: Catalog No. 9107001200, Kyoto, Japan) with reported lower sensitivities of 0.2  $\mu$ g imidacloprid per liter and 1.5  $\mu$ g dinotefuran per liter, respectively. The assays were calibrated before use to test for matrix effects associated with leaf tissue homogenates (Byrne et al. 2005a).

Statistical Analysis. Data were analyzed using SAS Systems for Windows, version 9.1 (SAS Institute 2009). To review, there were three separate experiments involving two plant types (yellow sage or poin-

settia) and two insect pests (sweetpotato whitefly or citrus mealybug). Each of the experiments included three factors. There were two systemic insecticides (imidacloprid and dinotefuran), each applied at two levels, resulting in four insecticide-rate combinations. We refer to the four combinations of the systemic insecticides and their specific rates of application as the insecticide-rate factor. There were two additional factors in the experiments, light at two levels (ambient and shade) and time in weeks (1-4). Experimental units were individual potted plants and treatments were randomly allocated in a  $4\times2\times4$  factorial treatment structure with four replications (plants/containers) per treatment.

We used arcsine transformed mortality percentages as a measure of mortality for our response variables associated with each experiment. Data were arcsine-transformed before subject to analysis of variance (ANOVA) to correct for unequal variances and departures from normality. Transformed data were then plotted to verify symmetry and the data were determined to be symmetric. We used a protected Fisher least significant difference (LSD) at the 0.05 significance level for means comparisons, as appropriate, for effects that were found to be statistically significant by ANOVA (SAS Institute 2008).

The main effects of interest were light, rate of application of the systemic insecticides (label and comparable), and concentration of active ingredient, and how these impacted percent mortality of both whiteflies and mealybugs. Across the three experiments, all data associated with whitefly and mealybug percent mortality, in the controls, was 0%, so we focused on determining differences in percent whitefly and mealybug mortality between the four rates of each systemic insecticide (imidacloprid: label and comparable, and dinotefuran: label and comparable). All data presented are nontransformed.

Data associated with the environmental variables of light intensity, day and night temperature, day relative humidity, and VPD were analyzed using SAS Systems for Windows, version 9.1 (SAS Institute 2009) using ANOVA with light (ambient and shade) and week (1–4) as the main effects.

## Results

Mean light intensity, day temperature, night temperature, relative humidity (RH), and VPD for each light treatment (ambient and shade) within each experiment are presented in Table 1. Light intensity in the ambient treatment was at least two-fold higher than the shade environment in each experiment. Day temperature and VPD were higher in the ambient than the shade treatments in experiments 1 and 2, but not experiment 3. Night temperature was only different between the ambient and shade treatments in experiment 3 (Table 1). The reason for this discrepancy in environment for experiment 3 may be associated with the season or months in which the experiment was conducted (mid-November through mid-December). The light intensity index for each

experiment is presented in Fig. 1, which shows a notable difference in the light intensity among the ambient and shade treatments for all three experiments.

Experiment 1: Sweetpotato Whitefly (B. tabaci) and Yellow Sage (L. camara). Insecticide-rate (F =26.52; df = 3, 127;  $P \le 0.0001$ ), week (F = 2.72; df = 3, 127; P = 0.0485), and light (F = 32.84; df = 1, 127;  $P \le$ 0.0001) main effects were all significant regarding the concentration of active ingredients present in leaf tissue. In addition, the insecticide-rate  $\times$  light (F = 5.27; df = 3, 127; P = 0.0021), insecticide-rate × week (F = 8.42); df = 9, 127; P < 0.0001), and insecticide-rate  $\times$  light  $\times$ week (F = 3.23; df = 9, 127; P = 0.0018) interactions were significant. The trends in the data indicated that for both rates of imidacloprid, plants exposed to ambient light generally had higher concentrations of the active ingredient in the leaf tissues (Fig. 2a) compared with plants in shade across all 4 wk (Fig. 2b). Higher concentrations of dinotefuran were present in the leaf tissue of plants exposed to ambient light (Fig. 2a) than shade (Fig. 2b) for both rates (comparable and label). In addition, the label rate was numerically higher than the comparable rate (Fig. 2a,b). Dinotefuran concentrations consistently, regardless of rate and light treatment, were higher in the first 2 wk but declined thereafter (Fig. 2a,b). There were significantly higher concentrations of imidacloprid in the leaf tissue for plants receiving the comparable rate (449.3 ng/leaf disk) than the label rate (267.4 ng/leaf disk) whereas significantly higher concentrations of dinotefuran were present in leaf tissues associated with plants that received the label rate (759.5 ng/leaf disk) compared with plants that received the comparable rate (439.1 ng/leaf disk) as would be expected.

For percent whitefly nymphal mortality, light was not significant (F = 0.54; df = 1, 127; P = 0.4623); however, the main effects of insecticide-rate and week were significant (F = 99.67; df = 3, 127;  $P \le 0.0001$  and F = 26.52; df = 3, 127;  $P \le 0.0001$ , respectively) and the insecticide-rate  $\times$  week interaction was significant (F = 3.25; df = 9, 127; P = 0.017).

Insecticide-rate was significant (F=33.57; df = 3, 31; P<0.0001) for the number of live whitefly pupae on the plants in week 4. There were significantly fewer whitefly pupae on plants treated with both rates of dinotefuran (label =  $0.62\pm3.6$  and comparable =  $0.0\pm3.6$ , respectively) than plants treated with both rates of imidaeloprid (label =  $23.7\pm3.6$  and comparable =  $25.3\pm3.6$ , respectively).

There was an increasing trend over time for percent whitefly nymphal mortality for each imidacloprid application rate across the 4-wk with the highest percent morality being 77.9% at week 4 whereas for both dinotefuran rates percent whitefly nymphal mortality was >89% (89.5–100%) for all 4 wk (Fig. 3). This suggests that, in general, dinotefuran was more effective than imidacloprid, regardless of the rate used. Although the comparable rate of imidacloprid was higher than the label rate, this did not translate into a substantial increase in percent whitefly nymphal mortality except for week 1 (Fig. 3).

For stomatal resistance, light was the only main effect that was significant (F = 53.10; df = 1, 127;  $P \le$ 

Table 1. Environmental parameters estimated including light intensity [ $\mu$ mol/m<sup>2</sup>/s of PAR for 400–700 nm], day temp (°C), night temp (°C), day RH, and VPD (kPa) under both the ambient and shade treatments across the 4-wk for all three experiments (n = 4)

Week	Light intensity <sup>a</sup>	Day temp <sup>b</sup>	Night temp <sup>c</sup>	Day RH <sup>b</sup>	$\mathrm{VPD}^d$
Experiment 1: Sweetpotato whitefly					
B-biotype and yellow sage					
Ambient light					
1 (3/25–4/2)	258	25.1	21.0	31.7	2.19
2 (4/3-4/9)	282	25.7	21.4	27.7	2.39
3 (4/10-4/16)	273	26.0	21.5	32.9	2.25
4 (4/17–4/23)	333	28.4	22.1	34.6	2.54
Shade					
1 (3/25-4/2)	106	23.7	21.3	34.1	1.93
2 (4/3–4/9)	107	23.6	21.5	30.0	2.03
3 (4/10-4/16)	106	23.7	21.6	36.8	1.85
4 (4/17-4/23)	166	26.2	22.3	38.9	2.08
Light	$P \le 0.0001$	$P \le 0.0001$	P = 0.3413	$P \le 0.0001$	$P \le 0.0001$
Week	$P \le 0.0001$	$P \le 0.0001$	P = 0.0008	$P \le 0.0001$	P = 0.0274
Experiment 2: Sweetpotato whitefly					
B-biotype and poinsettia					
Ambient light					
1 (10/7–10/13)	158	23.9	19.4	47.9	1.55
2 (10/14–10/20)	235	25.4	19.6	44.8	1.79
3 (10/21–10/27)	197	24.1	23.6	44.4	1.67
4 (10/28–11/2)	195	22.8	18.4	46.9	1.48
Shade	100		10.1	10.0	1.10
1 (10/7–10/13)	65	23.5	19.9	49.4	1.46
2 (10/14–10/20)	100	24.5	20.1	46.7	1.64
3 (10/21–10/27)	86	23.4	19.8	46.1	1.55
4 (10/28–11/2)	89	22.0	14.7	48.6	1.36
Light	$P \le 0.0001$	P = 0.0040	P = 0.6351	P = 0.0017	P = 0.0032
Week	$P \le 0.0001$	$P \le 0.0001$	P = 0.2874	$P \le 0.0001$	$P \le 0.0001$
Experiment 3: Citrus mealybug and	1 = 0.0001	1 — 0.0001	1 0.2011	1 — 0.0001	1 — 0.0001
poinsettia					
Ambient light					
1 (11/20–11/27)	175	23.1	17.8	39.0	1.73
2 (11/28–12/4)	157	22.9	17.6	33.6	1.85
3 (12/5–12/11)	161	21.4	17.0	33.7	1.69
4 (12/12–12/18)	182	21.6	18.2	30.6	1.79
Shade	102	21.0	10.2	50.0	1.10
1 (11/20–11/27)	65	22.5	18.2	40.5	1.62
2 (11/28–12/4)	78	22.2	18.1	35.0	1.74
3 (12/5–12/11)	68	21.4	18.0	33.9	1.69
4 (12/12–12/11)	67	21.4	18.9	31.5	1.76
4 (12/12-12/18) Light	$P \le 0.0001$	P = 0.1936	P = 0.0015	P = 0.0378	P = 0.0936
Week	P = 0.9413	P = 0.1930 P = 0.0009	P = 0.0046	$P \le 0.0001$	P = 0.6788
** CCN	1 - 0.9415	1 - 0.0009	1 - 0.0040	1 = 0.0001	1 - 0.0700

<sup>&</sup>quot;To exclude periods of darkness from the light intensity averages, only readings >15 lumens/ft² were included. To convert these units to  $\mu$ mol/m²/s of PAR (Photosynthetically Active Radiation) for 400–700 nm, light intensity measured in lumens/m² was multiplied by 0.20 (Thimijan and Heins 1983).

0.0001); shade had a significantly higher mean resistance than ambient light (4.86  $\pm$  0.19 s/cm vs. 2.90  $\pm$  0.19 s/cm, respectively).

Experiment 2: Sweetpotato Whitefly (*B. tabaci*) and Poinsettia (*E. pulcherrima*). Insecticide-rate (F = 85.15; df = 3, 126;  $P \le 0.0001$ ), week (F = 7.03; df = 3, 126; P = 0.0003), and light (F = 27.53; df = 1, 126;  $P \le 0.0001$ ) main effects were all significant regarding the concentration of active ingredients present in the leaf tissue. In addition, the insecticide-rate  $\times$  light interaction was significant (F = 7.74; df = 3, 126; P = 0.0001).

The trends in the data indicated that for both rates of imidacloprid (comparable and label), plants exposed to ambient light generally had higher concentrations of the active ingredient in the leaf tissues compared with plants in shade across all 4 wk (Fig. 4).

Higher concentrations of dinotefuran were present in the leaf tissue of plants exposed to ambient light than shade (Fig. 4) for both rates (comparable and label). In addition, the label rate was numerically higher than the comparable rate (Fig. 4). There were significantly higher concentrations of imidacloprid in the leaf tissue of plants receiving the comparable rate (192.4 ng/leaf disk) than the label rate (101.6 ng/leaf disk) whereas significantly higher concentrations of dinotefuran were present in leaf tissues associated with plants that received the label rate (740.2 ng/leaf disk) compared with plants that received the comparable rate (326.6 ng/ leaf disk). In addition, when averaged over all four insecticide-rate combinations, there were significantly higher concentrations of both insecticides in leaf tissue sampled from plants maintained under

<sup>&</sup>lt;sup>b</sup> Day temp and RH were averages of readings measured between 0600 and 1800 h.

<sup>&</sup>lt;sup>c</sup> Night temp was an avg of readings measured between 1830 and 0530 h.

<sup>&</sup>lt;sup>d</sup> Vapor pressure deficit was calculated using Murray's equation (Murray 1967).

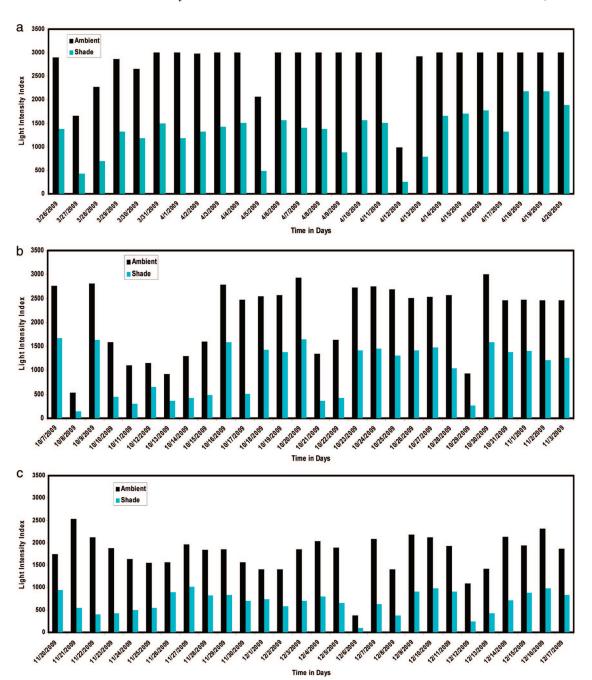


Fig. 1. Light intensity index associated with experiment 1 (a), 2 (b), and 3 (c). Light intensity index is the cumulative addition of measured light intensity every 30 min between 7:00 a.m. and 7:00 p.m. for experiments 1 and 2, and 7:00 a.m. and 5:00 p.m. for experiment 3.

ambient light (420 ng/leaf disk) compared with those under shade (259.6 ng/leaf disk).

For percent whitefly nymphal mortality, light was not significant (F = 0.12; df = 1, 127; P = 0.7298), but the main effects of insecticide-rate and week were significant (F = 78.79; df = 3, 127;  $P \le 0.0001$  and F = 33.07; df = 3, 127;  $P \le 0.0001$ , respectively) and the insecticide-rate  $\times$  week interaction was significant

 $(F=5.16; \mathrm{df}=9, 127; P\leq 0.0001)$ . Percent whitefly nymphal mortality was significantly higher for both the comparable (93.5) and label rate (97.2) of dinotefuran than the imidacloprid comparable (66.2) and label rate (56.7).

There was an increasing trend over time associated with percent whitefly nymphal mortality for each imidacloprid application rate across the 4 wk

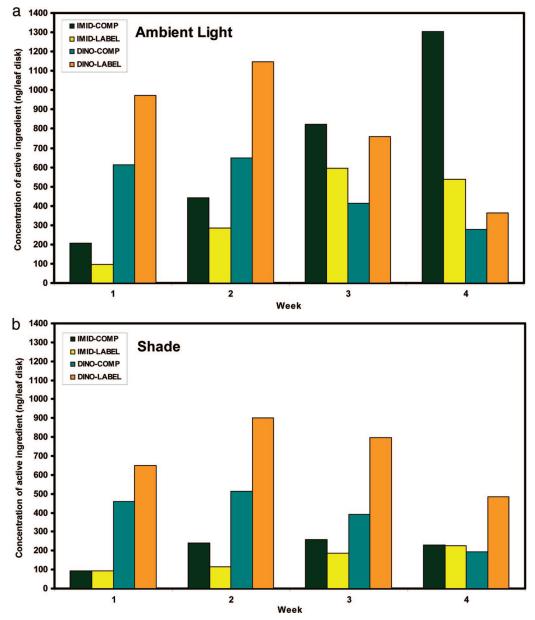


Fig. 2. Mean concentration (ng/leaf disk) of systemic insecticide active ingredient (imidacloprid and dinotefuran when applied at the comparable and label rate) in plant (L. camara) tissues associated with the ambient (a) and shade (b) light treatment for weeks 1 through 4 for experiment 1. Insecticide-rate: Comp = comparable rate (imidacloprid at 0.027 g active ingredient/container and dinotefuran at 0.014 g active ingredient/container), Label = label rate (imidacloprid at 0.014 g active ingredient/container). Statistics: insecticide-rate  $\times$  light  $\times$  week (F = 3.23; df = 9, 127; P = 0.0018).

with the highest percent morality being 78% at week 4 whereas for both the dinotefuran rates percent whitefly nymphal mortality was >89% (89.5–99%) for all 4 wk (Fig. 5). These results corroborate findings from experiment 1, which also showed that dinotefuran was more effective than imidacloprid, particularly at the label rate. Although the comparable rate of imidacloprid was higher than the label rate, this did not result in a substantial increase in

percent whitefly nymphal mortality except for week 1 (Fig. 5).

For stomatal resistance, light and week were the significant main effects (F=45.57; df = 1, 126;  $P\leq0.0001$  and F=14.93; df = 3, 126;  $P\leq0.0001$ , respectively), and the light  $\times$  week interaction was significant (F=8.33; df = 3, 126;  $P\leq0.0001$ ), where shade had significantly higher mean resistance values than ambient light across all 4 wk (Table 2).

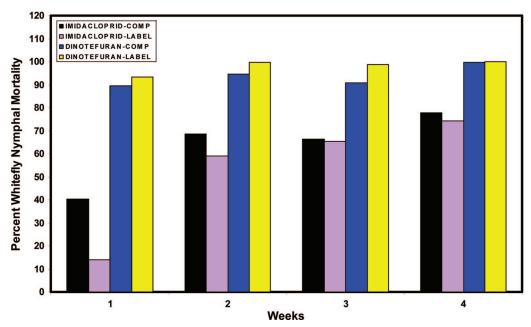


Fig. 3. Mean ( $\pm$ SEM) percent mortality of sweetpotato whitefly, *B. tabaci* nymphs associated with each systemic insecticide-rate (imidacloprid and dinotefuran: comparable and label rate) and week (1 through 4) for experiment 1. Insecticide-rate: Comp = comparable rate (imidacloprid at 0.027 g active ingredient/container and dinotefuran at 0.014 g active ingredient/container), Label = label rate (imidacloprid at 0.014 g active ingredient/container and dinotefuran at 0.027 g active ingredient/container). Statistics: insecticide-rate  $\times$  week (F = 3.25; df = 9, 127; P = 0.017).

Experiment 3: Citrus Mealybug (*P. citri*) and Poinsettia (*E. pulcherrima*). Insecticide-rate (F = 103.75; df = 3, 127;  $P \le 0.0001$ ) and week (F = 15.60; df = 3,

127;  $P \le 0.0001$ ) main effects were both significant regarding the concentration of active ingredients present in leaf tissues. In addition, the insecticide-rate  $\times$ 

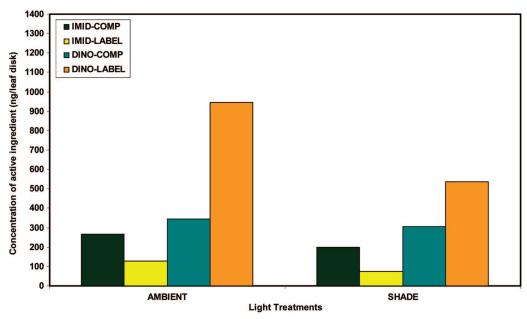


Fig. 4. Mean concentration (ng/leaf disk) of systemic insecticide active ingredient (imidacloprid and dinotefuran when applied at the comparable and label rate) in plant ( $E.\ pulcherrima$ ) tissues associated with the ambient light and shade treatments for experiment 2. Leaf samples were taken once per week over a 4-wk period after treatments had been applied. Insecticide-rate: Comp = comparable rate (imidacloprid at 0.027 g active ingredient/container and dinotefuran at 0.014 g active ingredient/container), Label = label rate (imidacloprid at 0.014 g active ingredient/container and dinotefuran at 0.027 g active ingredient/container). Statistics: insecticide-rate  $\times$  light (F = 7.74; df = 3, 126; F = 0.0001).

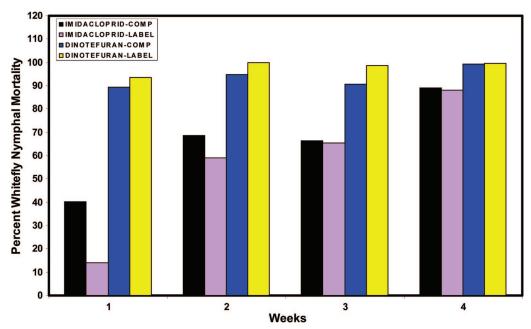


Fig. 5. Mean ( $\pm$ SEM) percent mortality of sweetpotato whitefly, *B. tabaci* nymphs associated with each systemic insecticide-rate (imidacloprid and dinotefuran: comparable and label rate) and week (1 through 4) for experiment 2. Insecticide-rate: Comp = comparable rate (imidacloprid at 0.027 g active ingredient/container and dinotefuran at 0.014 g active ingredient/container), Label = label rate (imidacloprid at 0.014 g active ingredient/container and dinotefuran at 0.027 g active ingredient/container). Statistics: insecticide-rate  $\times$  week (F = 5.16; df = 9, 127;  $P \le 0.0001$ ).

week interaction was significant (F = 2.57; df = 9, 127; P = 0.0107). For imidaeloprid, the mean ( $\pm$ SEM) concentration (ng/leaf disk) of active ingredient associated with the comparable rate (234.3  $\pm$  39.6) was significantly higher than the label rate (145.3  $\pm$  39.6) whereas for dinotefuran, the concentration of active ingredient in the leaf tissues for plants that received the label rate (989.7  $\pm$  39.6) was significantly higher than the comparable rate (736.1  $\pm$  39.6). In regards to the concentration of active ingredient across the 4 wk, the label rate of dinotefuran was numerically higher than the other rates evaluated (Fig. 6). Dinotefuran concentrations associated with the comparable and label rates were highest in weeks 2 (943.0  $\pm$ 79.2 and 1111.6  $\pm$  79.2) and 3 (928.2  $\pm$  79.2 and  $1138.8 \pm 79.2$ ) but declined thereafter (Fig. 6).

For percent mealybug mortality, only the main effect of week was significant (F = 26.26; df = 3, 127;  $P \le 0.0001$ ). Percent mealybug mortality for the compa-

Table 2. Stomatal resistance (s/cm) values (means  $\pm$  SEM) associated with the light treatments (ambient and shade) across the 4 wk for experiment 2

Week	Ambient	Shade	
1	$8.57 \pm 0.94a^a$	$10.29 \pm 0.91$ b	
2	$5.54 \pm 0.91a$	$6.91 \pm 0.91b$	
3	$5.55 \pm 0.91a$	$8.37 \pm 0.91b$	
4	$5.86 \pm 0.91a$	$16.74 \pm 0.91b$	
Mean	6.38	10.5	

<sup>&</sup>lt;sup>a</sup> Means within a row followed by a different letter are significantly different based on an analysis of variance (ANOVA) with a  $P \le 0.05$ .

rable (40.6  $\pm$  3.3) and label (34.8  $\pm$  3.3) rates of imidacloprid and comparable (43.8  $\pm$  3.3) and label (45.3  $\pm$  3.3) rates of dinotefuran were all <50%. This suggests that none of the insecticides, regardless of the rates used, provided satisfactory control of citrus mealybugs feeding on poinsettia.

For stomatal resistance, light and week were the only main effects that were significant (F = 4.69; df = 1,126; P = 0.0329 and F = 17.09; df = 3,126;  $P \le 0.0001$ , respectively); shade had a significantly higher mean resistance value than that for ambient light (19.28  $\pm$  1.19 s/cm vs. 15.27  $\pm$  1.19 s/cm, respectively).

#### Discussion

This study showed that light intensity can adversely affect the uptake of systemic neonicotinoid insecticides, thereby, indirectly influencing their efficacy in controlling populations of the sweetpotato whitefly and citrus mealybug. Light intensity and temperature are likely confounded in impacting plant growth and insect development (Niesenbaum and Kluger 2006). Therefore, we measured light intensity, temperature, relative humidity, and calculated VPD to determine if any of these environmental parameters influenced the movement of the systemic insecticide active ingredients within the plants.

In experiments 1 and 2, VPD was lower in the shade compared with the ambient light treatment but VPD was not different between the light treatments in experiment 3 (Table 1). The lower VPD likely re-

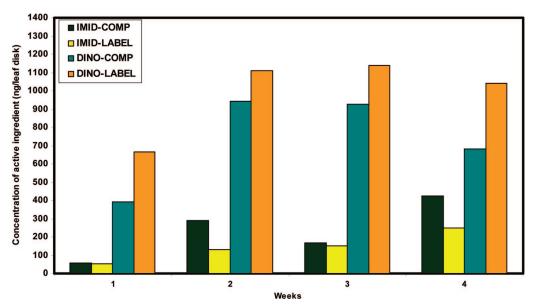


Fig. 6. Mean concentration (ng/leaf disk) of systemic insecticide active ingredient (imidacloprid and dinotefuran when applied at the comparable and label rate) in plant ( $E.\ pulcherrima$ ) tissues associated with weeks 1 through 4 for experiment 3. Insecticide-rate: Comp = comparable rate (imidacloprid at 0.027 g active ingredient/container and dinotefuran at 0.014 g active ingredient/container), Label = label rate (imidacloprid at 0.0.014 g active ingredient/container and dinotefuran at 0.027 g active ingredient/container). Statistics: insecticide-rate  $\times$  week (F = 2.57; df = 9, 127; P = 0.0107).

duced plant transpiration in the shade compared with plants in the ambient light treatment thus influencing translocation of the systemic insecticides into and through plant tissues. Regarding stomatal resistance, shade had a significantly higher mean resistance value than that for ambient light in all three experiments. This result is expected because a higher stomatal resistance indicates that the stomates are more tightly closed, which means that plants are transpiring less.

Although dinotefuran uptake was also influenced by light intensity, the label application rates still provided sufficient active ingredient to compensate for the reduced uptake under low light conditions. In addition to the higher concentrations of dinotefuran at current label rates, the effectiveness of dinotefuran in controlling whiteflies in our study may also be attributed to its greater intrinsic toxicity and more effective uptake from the growing medium than imidacloprid. An increase in the application rate compensated for the lower rate of imidacloprid uptake at low light intensity and provided more effective control of whiteflies.

Imidacloprid and dinotefuran represent extremes with respect to water solubility among the neonicotinoids, and these differences may impact the rate of uptake of these systemic insecticides (Byrne et al. 2007). Furthermore, the dinotefuran treatments (comparable and label rates) in experiment 1 resulted in the development of very few adults based on the number of empty pupal cases ( $\leq$ 0.62) thus reducing the population of whitefly nymphs in the next generation. Because dinotefuran (39 g/L) is more soluble in water than imidacloprid (0.51 g/L) (Wakita et al. 2005) it may be more readily available in the growing medium for root uptake and translocation throughout

plant parts (Castle et al. 2005). Moreover, the delay or lag period from application to the advent of full systemic translocation within plants may also affect the ability of the systemic insecticide to prevent the development of future generations (Castle et al. 2005). Insecticides with higher water solubilities tend to be more mobile within the plant. Therefore, the low solubility of imidacloprid may impede movement throughout plant tissues (Hale and Shorey 1965, Tomlin 1994), particularly in woody plants as woody plant stems may inhibit the concentration of active ingredient that reaches and accumulates within plant leaves although this may vary depending on plant type, developmental stage, and the particular systemic insecticide (Cowles 2010). Both yellow sage and poinsettia develop woody tissue (Liberty Hyde Bailey Hortorium 1976), particularly later in the production cycle, which may delay the movement of imidacloprid throughout the plant. This may account for the lower mortality of both the sweetpotato whitefly and citrus mealybug during the course of the study.

Byrne et al. (2010) demonstrated that dinotefuran drench applications could provide multi-generational control of whitefly populations because of the rapid activity of dinotefuran. This is primarily because of the high mortality provided within 1 to 2 wk, which then reduces the number of individuals in future generations. In fact, in our study, even the comparable rate of imidacloprid (that was an increase of >50% in the concentration compared with the standard rate) did not provide higher levels of mortality than the label rate. This suggests that imidacloprid must be applied before the presence of whiteflies and when plants are herbaceous whereas dinotefuran can be applied later

in the production cycle; however, both were applied later on in the poinsettia production cycle in experiment 3, during bract expression, and this could account for the lack of efficacy against the citrus mealybug. As such, both systemic insecticides may need to be applied before this stage of plant development. The recommended label rates for dinote furan are four-fold higher than imidacloprid, which means that more active ingredient is available for plant uptake. In addition, dinotefuran is more toxic to whiteflies than imidacloprid (Prabhaker et al. 2005). The fact that we obtained sufficient mortality of whitefly nymphs in experiments 1 and 2 with the comparable rate indicates that using a rate of dinotefuran, lower than the recommended label rate, may be just as effective against whiteflies.

The lack of effective control against citrus mealvbugs may be associated with the feeding behavior of this insect as they tend to congregate on plant stems (R.A.C., unpublished data), which may allow them to avoid ingesting lethal concentrations of the active ingredient. In addition, it is possible that the systemic insecticides are primarily located within the xylem of stems where the main transport within the plant occurs (Sur and Stork 2003). It is possible that because the leaves are the sink for the insecticide, once the insecticide reaches the sink, then there is movement from the xylem to the phloem, thereby affecting phloem-feeders more effectively. Essentially, the phloemfeeders may avoid higher doses because the movement of the insecticide between the xylem and phloem is not as pronounced compared with when the insecticide resides at the sink area. However, additional research to explain this observation is war-

Furthermore, it is possible that the citrus mealybug is less susceptible to systemic insecticides than whiteflies, which may be associated with their feeding behavior that involves differences in the number and length of time of intracellular punctures, intervals between the first phloem-ingestion periods and stylet motility during the phloem searching process (Calatayud et al. 1994). This could impact the ability of systemic insecticides to control citrus mealybugs. In addition, treatments were applied when poinsettia plants were beginning to unroll bracts. Bract or flower formation may influence stomatal resistance or transpiration rates (Burrows and Milthorpe 1976) although this depends on plant type (Longstreth and Kramer 1980). However, if the plants were transpiring less during flowering, this would have negatively affected the movement or translocation of the systemic insecticides within the plant tissues, which was observed in experiment 3. As such, reduced transpiration would lead to less absorption of the active ingredient by the root system, thus less active ingredient being translocated throughout the plant resulting in lower mortality against citrus mealybugs. This is supported by the differences in stomatal resistance between experiments 2 and 3 where in experiment 2, stomatal resistance for both ambient and shade was 6.2 s/cm and 10.5 s/cm, respectively, whereas in experiment 3,

stomatal resistance was 15.2 s/cm for ambient and 19.2 s/cm for shade.

Plant developmental stage and type may affect uptake and movement of systemic insecticides. As such, any differences in the concentration of active ingredient may be associated with the plant type as both yellow sage and poinsettia are C<sub>3</sub> plants (Webster et al. 1975) but yellow sage may have a lower transpiration rate than poinsettia, which could result in less active ingredient accumulating in the leaf tissues. In fact, it has been reported that the transpiration rate of poinsettia is 0.00000465 g/cm<sup>2</sup>/s compared with 0.000002083 g/cm<sup>2</sup>/s for yellow sage (Schuch et al. 1995, Thut 1939). Additionally, yellow sage has been shown to have a lower transpiration rate compared with other ornamental species under certain environmental conditions (Starman and Lombardini 2006). In our study, however, the season in which the experiments were conducted seemed to over-ride plant type effect on transpiration. In experiment 1, yellow sage had a much lower stomatal resistance (2.9 s/cm in ambient and 4.9 s/cm in shade) compared with poinsettia in experiment 2 (6.4 s/cm in ambient and 10.5 s/cm in shade). The higher stomatal resistance associated with poinsettia in experiment 2 suggests that less transpiration was occurring, which may have reduced uptake, translocation, and thus efficacy of the systemic insecticide treatments.

In summary, our study has demonstrated that light intensity may impact the uptake and translocation of systemic insecticides, and indirectly influence mortality of insect pests based on the effect of light intensity on plant transpiration. This research also raises interesting questions on the potential impact of season, stage of plant development, and timing of applications on translocation and efficacy. The results from our study indicate that greenhouse producers should take into consideration a multitude of interacting factors that may influence the efficacy of systemic insecticide applications.

## Acknowledgments

We gratefully acknowledge technical support provided by Jessica Goebel, Zachary Starr, Paul Jemison, Jesse Ostrander and Will Fox, and M. B. Kirkham for sharing her leaf porometer and expertise regarding stomatal resistance. Rooted cuttings of poinsettia were donated by Dummen USA (Hilliard, OH) and the Paul Ecke Ranch (Encinitas, CA). Growing medium was donated by Conrad (Fafard, Inc., Agawam, MA). Finally, we want to acknowledge James R. Nechols (Kansas State University, Manhattan, KS) for providing a review of an initial draft of the manuscript. This research was funded in part by a grant from the K-State integrated pest management (IPM) Mini-Grants Program (41531-0-1600) and the College of Agriculture Honors Program, Kansas State University (Manhattan, KS).

## References Cited

Agata, W., S. Hakoyama, and Y. Kawamitsu. 1985. Influence of light intensity, temperature and humidity on photo-

- synthesis and transpiration of Sasa nipponica and Arundinaria pygmaea. Bot. Mag. Tokyo 98: 125–135.
- Bennett, S. H. 1957. The behavior of systemic insecticides applied to plants. Annu. Rev. Entomol. 2: 279–296.
- Bennett, S. H. 1949. Preliminary experiments with systemic insecticides. Ann. Appl. Biol. 36: 160–163.
- Bennett, S. H., and W.D.E. Thomas. 1954. The absorption, translocation and breakdown of schradan applied to leaves, using <sup>32</sup>P-labelled material. II. Evaporation and absorption. Ann. Appl. Biol. 41: 484–500.
- Brodsgaard, H. F., and R. Albajes. 1999. Insect and mite pests, pp. 48–69. In J. C. van Lenteren and Y. Elad (eds.), Integrated pest and disease management in greenhouse crops. Klumer Academic Publishers, Dordrecht, The Netherlands.
- Burrows, F. J., and F. L. Milthorpe. 1976. Stomatal conductance in the control of gas exchange, pp. 103–152. In T. Kozlowski (ed.), Water deficits and plant growth, vol. 4. Academic, New York.
- Byrne, F. J., R. D. Oetting, J. A. Bethke, C. Green, and J. Chamberlin. 2010. Understanding the dynamics of neonicotinoid activity in the management of *Bemisia tabaci* whiteflies on poinsettias. Crop Prot. 29: 260–266.
- Byrne, F. J., N. C. Toscano, A. A. Urena, and J. G. Morse. 2007. Toxicity of systemic neonicotinoid insecticides to avocado thrips in nursery avocado trees. Pest Manag. Sci. 63: 860–866.
- Byrne, F. J., S. J. Castle, J. L. Bi, and N. C. Toscano. 2005a. Application of competitive enzyme-linked immunosorbent assay for the quantification of imidacloprid titers in xylem fluid extracted from grapevines. J. Econ. Entomol. 98: 182–187.
- Byrne, F. J., N. C. Toscano, A. A. Urena, and J. G. Morse. 2005b. Quantification of imidaeloprid toxicity to avocado thrips, Scirothrips perseae Nakahara (Thysanoptera: Thripidae), using a combined bioassay and ELISA approach. Pest Manag. Sci. 61: 754–758.
- Byrne, F. J., I. D. Bedford, A. L. Devonshire, and P. G. Markham. 1995. Esterase variation and squash silverleaf induction in B-type Bemisia tabaci (Gennadius). Bull. Entomol. Res. 86: 175–179.
- Calatayud, P. A., Y. Rahbe, W. F. Tjallingii, M. Tertuliano, and B. Le Rü. 1994. Electrically recorded feeding behaviour of cassava mealybug on host and non-host plants. Entomol. Exp. Appl. 72: 219–232.
- Castle, S. J., F. J. Byrne, J. L. Bi, and N. C. Toscano. 2005. Spatial and temporal distribution of imidacloprid and thiamethoxam in citrus and impact on *Homalodisca co*agulata populations. Pest Manag. Sci. 61: 75–84.
- Cloyd, R. A. 2010. Systemic insecticides 101. Am. Nursery 210: 18–21.
- Cloyd, R. A., and J. A. Bethke. 2011. Impact of neonicotinoid insecticides on natural enemies in greenhouse and interiorscape environments. Pest Manag. Sci. 67: 3–9.
- Cowles, R. S. 2010. Optimizing a basal bark spray of dinotefuran to manage armored scales (Hemiptera: Diaspididae) in Christmas tree plantations. J. Econ. Entomol. 103: 1735–1743.
- David, W.A.L., and O. C. Gardiner. 1951. Investigations on the systemic insecticidal action of sodium fluoracetate and of three phosphorus compounds on *Aphis fabae* Scop. Ann. Appl. Biol. 38: 91–110.
- Elbert A., and H. Overbeck. 1990. Imidacloprid, a novel systemic nitromethylene analogue insecticide for crop protection, pp. 21–28. Proceedings of the Brighton Crop Protection Conference.—Pests and Diseases, BCPC, Alton, Hants, United Kingdom.

- Grange, R. I., and D. W. Hand. 1987. A review of the effects of atmospheric humidity on the growth of horticultural crops. J. Hort. Sci. 62: 125–234.
- Hale, R. L., and H. H. Shorey. 1965. Systemic insecticides for the control of western flower thrips on bulb onions. J. Econ. Entomol. 58: 793–794.
- Hollingworth, R. M., and M. F. Treacy. 2006. Classification and properties of commercial insecticides and acaricides, pp. 36–67. In J. N. All and M. F. Treacy (eds.), Use of management of insecticides, acaricides, and transgenic crops. The American Phytopathological Society, St. Paul, MN.
- Jeppson, L. R. 1953. Systemic insecticides: entomological aspects of systemic insecticides. Agric. Food Chem. 1: 830-832.
- Jeschke, P., and R. Nauen. 2008. Neonicotinoids: from zero to hero in insecticide chemistry. Pest Manag. Sci. 64: 1084–1098.
- Kanne, D. B., R. A. Dick, M. Tomizawa, and J. E. Casida. 2005. Neonicotinoid nitroguanidine insecticide metabolites: Synthesis and nicotinic receptor potency of guanidines, aminoguanidines, and their derivatives. Chem. Res. Toxicol. 18: 1479–1484.
- Kirkham, M. B. 2005. Principles of soil and plant water relations. Elsevier, Amsterdam, The Netherlands.
- Kirkham, M. B. 2011. Elevated carbon dioxide: impacts on soil and plant water relations. CRC, Boca Raton, FL.
- Liberty Hyde Bailey Hortorium. 1976. Hortus Third. Macmillan Publishing Company, New York.
- Longstreth, D. J., and P. J. Kramer. 1980. Water relations during flower induction and anthesis. Bot. Gaz. 141: 69-
- Murray, F. W. 1967. On the computation of saturated vapor pressure. J. Appl. Meteorol. 6: 203–204.
- Niesenbaum, R. A., and E. C. Kluger. 2006. When studying the effects of light on herbivory, should one consider temperature? The case of *Epimecis hortaria F*. (Lepidoptera: Geometridae) feeding on *Lindera benzoin L*. (Lauraceae). Environ. Entomol. 35: 600–606.
- Norris, D. M. 1967. Systemic insecticides in trees. Annu. Rev. Entomol. 12: 127–148.
- Pallas, J. E. Jr., B. E. Michel, and D. G. Harris. 1967. Photosynthesis, transpiration, leaf temperature, and stomatal activity of cotton plants under varying water potentials. Plant Physiol. 42: 76–88.
- Parrella, M. P. 1999. Arthropod fauna, pp. 213–250. In G. Stanhill and H. Z. Enoch (eds.), Greenhouse ecosystems, ecosystems of the world 20. Elsevier Ltd., Amsterdam, The Netherlands.
- Prabhaker, N., S. Castle, T. J. Henneberry, and N. C. Toscano. 2005. Assessment of cross-resistance potential to neonicotinoid insecticides in *Bemisia tabaci* (Hemiptera: Aleyrodidae). Bull. Entomol. Res. 95: 535–543.
- Reynolds, H. T. 1954. Entomological aspects of systemic pesticides. Agric. Chem. 113: 28–31.
- Rudinsky, J. A. 1959. Systemics in the control of forest insects. J. Forest. 57: 284–286.
- SAS Institute. 2008. SAS/STAT 9.1 user's guide. SAS Institute, Cary, NC.
- SAS Institute. 2009. SAS/STAT 9.2 user's guide. SAS Institute. Cary, NC.
- Schuch, U., R. A. Redak, and J. Bethke. 1995. Whole-plant response of six poinsettia cultivars to three fertilizer and two irrigation regimes. J. Am. Soc. Hort. Sci. 121: 69–76.
- Starman, T., and L. Lombardini. 2006. Growth, gas exchange, and chlorophyll fluorescence of four ornamental herbaceous perennials during water deficit conditions. J. Am. Soc. Hort. Sci. 113: 469-475.

- Sur, R., and A. Stork. 2003. Uptake, translocation and metabolism of imidacloprid in plants. Bull. Insectol. 56: 35–40.
- Thimijan, X., and R. Heins. 1983. Photometric, radiometric, and quantum light units of measure: a review of procedures for interconversion. HORTSCIENCE 18: 818–822.
- Thut, H. F. 1939. The relative humidity gradient of stomatal transpiration. Am. J. Bot. 26: 315–319.
- Tomizawa, M., and J. E. Casida. 2003. Selective toxicity of neonicotinoids attributable to specificity of insect and mammalian nicotinic receptors. Annu. Rev. Entomol. 48: 330\_364
- Tomizawa, M., and I. Yamamoto. 1993. Structure-activity relationships of nicotinoids and imidacloprid analogs. J. Pestic. Sci. 18: 91–98.
- Tomlin, C. 1994. The pesticide manual, 10th ed. British Crop Protection Council, Survey, and Royal Society of Chemistry, Cambridge, United Kingdom.

- Wakita, T., N. Yasui, E. Yamada, and D. Kishi. 2005. Development of a novel insecticide, dinotefuran. J. Pestic. Sci. 30: 122–123.
- Ware, G. W., and D. M. Whitacre. 2004. The pesticide book. MeisterPro Information Resources, Willoughby, OH.
- Webster, G. L., W. V. Brown, and B. N. Smith. 1975. Systematics of photosynthetic carbon fixation pathways in Euphorbia. Taxon 24: 27–33.
- Wedding, R. T. 1953. Plant physiological aspects of the use of systemic insecticides. Agric. Food Chem. 1: 832–834.
- Zhang, A., H. Kayser, P. Maienfisch, and J. E. Casida. 2000. Insect nicotinic acetylcholine receptor: conserved neonicotinoid specificity of [<sup>3</sup>H] imidacloprid binding site. J. Neurochem. 75: 1294–1303.

Received 20 August 2011; accepted 25 January 2012.