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Impact of tank mixing plant hormones with bentazon and mesotrione on sweetpotato injury and weed control

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Abstract

Due to excessive crop damage, currently, there are no selective postemergent (POST) herbicides registered for sweetpotato [*Ipomoea batatas* (L.) Lam] to control broadleaves and nutsedge species. Expansion of bentazon and mesotrione herbicide labels to include sweetpotato would be beneficial for growers. Two experiments were conducted. The first evaluated the dose response of sweetpotato cultivars Beauregard and Covington to bentazon (514, 1,028, and 1,542, g a.i. ha^{-1}) and mesotrione (105, 210, and 315 g a.i. ha^{-1}) when melatonin, 24-epibrassinolide, or ascorbic acid (AsA) were included in the tank mix. The second experiment evaluated the efficiency of bentazon and mesotrione for control of yellow nutsedge (*Cyperus esculentus* L.) and Palmer amaranth (*Amaranthus palmeri* S. Wats.), when different doses of melatonin, 24-epibrassinolide, or AsA were added to each herbicide. In the first experiment, when treated with herbicide alone, Beauregard exhibited injury levels lower than Covington for both herbicides. No injury was observed when plants were treated with plant hormones or AsA alone. At the lowest dose of bentazon and mesotrione, the addition of AsA or plant hormones in the tank mix significantly reduced percentage injury and increased plant tolerance, requiring higher doses of herbicide to cause 10, 20, and 30% injury. In the second experiment, the addition of plant hormones and AsA in the tank mix had no antagonistic effect on herbicide effectiveness, exhibiting similar levels of injuries as herbicides application alone. These results suggest that the use of plant hormones and AsA could improve sweetpotato tolerance to POST applications of bentazon and mesotrione without reducing herbicide effectiveness.

1 | INTRODUCTION

Sweetpotato [*Ipomoea batatas* (L.) Lam] production in the United States has increased by 37.6% from 2012 to 2017, reaching an estimated value of US$654 million in 2018 (USDA, 2019a). Louisiana, California, Mississippi, North Carolina, and South Carolina are largest producers of sweetpotato in the United States (USDA, 2019b). One of the biggest challenges for sweetpotato growers is weed control. Weeds interfere with plant growth and compete for limited water, light, and nutrients (Harrison & Jackson, 2011a). Yellow nutsedge (*Cyperus esculentus* L.) and Palmer amaranth (*Amaranthus palmeri* S. Wats.) can significantly reduce sweetpotato yield. Meyers and Shankle (2015) reported that yellow nutsedge densities of 5 to 90 shoots m^{-2} in planted

Abbreviations: AsA, ascorbic acid; GST, glutathione *S*-transferase; POST, postemergent; ROS, reactive oxygen species; WAT, weeks after treatment.

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sweetpotato resulted in yield losses of 18–96%. Smith et al. (2020) reported that Palmer amaranth, when not controlled, reduced 'Covington' marketable sweetpotato yield by 95% after season-long interference.

In-season weed management for sweetpotato depends on herbicides, between-row cultivation, mowing, and hand removal. However, chemical control is limited to 10 registered herbicides (Kemble et al., 2020). The postemergent (POST) herbicides labeled for sweetpotato are the grass herbicides clethodim, fluazifop, and sethoxydim. Herbicides for POST control of broadleaf weeds are only labeled for between-row application, risking damage to sweetpotato leaves during application. There are no selective POST herbicides registered to control yellow nutsedge and Palmer amaranth in sweetpotato (Webster, 2010). One possible solution to increase POST herbicide options for sweetpotato is to evaluate safener compounds with the potential to reduce nontarget herbicide injury while maintaining effective weed control (Parker, 1983). Hatzios (1991) noted that certain safeners could increase the activity of cytochrome P450s, resulting in greater tolerance to multiple herbicide modes of action through conjugation and metabolism of the herbicide molecule.

Some herbicides can induce the overproduction of reactive oxygen species (ROS) as a secondary effect, leading to oxidative damage in essential tissues (Caverzan et al., 2019). Compounds with the ability to increase cytochrome P450 activity and sequester ROS overproduction have the potential to promote greater stress tolerance in plants. Mandal et al. (2018) noted that melatonin's exogenous application could directly affect genes involved in biotic and abiotic stress response, and melatonin could increase cytochrome P450 activity in watermelon (*Citrullus lanatus* L.). In the same study, powdery mildew (caused by *Podosphaera xanthii*) disease severity significantly decreased when melatonin was applied exogenously. Additionally, Caputo et al. (2020) reported an improvement in sweetpotato tolerance to bentazon due to exogenous melatonin uptake *in vitro*.

Brassinosteroids are plant hormones that improve plant stress tolerance (Mussig & Altmann, 1999; Szekeres et al., 1996). According to Zhou et al. (2015), brassinosteroid-induced stress tolerance is preceded by increased nicotinamide adenine dinucleotide phosphate oxidase and elevated H_2O_2 levels. Brassinosteroids can upregulate cytochrome P450, glutathione S-transferase (GST), hydrolase, and oxidoreductase genes. Another compound that could reduce herbicide injury is ascorbic acid (AsA). Though AsA has no direct involvement in the herbicide detoxification process, it plays an essential role in stress perception and stress signaling responses in plants, having subcellular distribution according to stress source (Zechmann, 2011). Maize (*Zea mays* L.) seeds treated with 0.25 mM AsA showed reduced toxicity when imbibed with glyphosate at 3.6 mg L^{-1} at the seedling stage, with 15% more dry weight than glyphosate alone (Sacała & Roszak, 2018).

Core Ideas

- Plant hormones and ascorbic acid improved herbicide tolerance in sweetpotatoes.
- No antagonistic interaction between plant hormones and herbicides was found in controlling weeds.
- Tank mix of herbicide and plant hormones could enable the use of postemergent herbicides in sweetpotato.

A significant limitation to weed management programs in sweetpotato is the lack of registered POST herbicides for Palmer amaranth and yellow nutsedge control. Expanding POST herbicide options would provide growers with more flexibility in their weed management program. Understanding the interactions between melatonin, 24-epibrassinolide, and AsA with the herbicides bentazon and mesotrione in sweetpotato may lead to improvements in weed management. Hermann et al. (2017) observed 98% yellow nutsedge control when bentazon was applied at 560 g a.i. ha^{-1}. Although Chahal et al. (2019) observed 88% control of Palmer amaranth when mesotrione was applied at 210 g a.i. ha^{-1}.

The main objectives of this study were (a) to characterize the tolerance of 'Beauregard' and 'Covington' to bentazon and mesotrione with and without applications of melatonin, AsA, or 24-epibrassinolide, and (b) to characterize the response of yellow nutsedge and Palmer amaranth to bentazon and mesotrione with and without exogenous applications of melatonin, AsA, or 24-epibrassinolide.

2 | MATERIALS AND METHODS

2.1 | Sweetpotato greenhouse dose response screening

Experiments were conducted in a greenhouse located at the Clemson Coastal Research and Education Center, Charleston, SC (32.794608° lat., −80.068361° long.), using the sweetpotato cultivars Beauregard and Covington. Slips of both cultivars were obtained from New Sprouts Organic Farm (Black Mountain, NC) and transplanted into pots with a volume of 500 cm^3 native soil Yonges loamy sand (fine-loamy, mixed, active, thermic Typic Endoaqualfs), collected from a organic field, cover cropped with italian ryegrass (*Lolium*

multiflorum Lam.) and sunn hemp (*Crotalaria juncea* L.), at the same research station. Treatments consisted of bentazon applied POST at rates of 514, 1,028, and 1,542 g a.i. ha^{-1} (Basagran 4, RedEagle International), or mesotrione at rates of 105, 210, and 315 g a.i. ha^{-1} (Callisto, Syngenta Crop Protection) tank mixed with melatonin (100 g a.i. ha^{-1}, Alfa Aesar), 24-epibrassinolide (100 g a.i. ha^{-1}, MCE Med Chem Express), or AsA (100 g a.i. ha^{-1}, TCI America). The lowest herbicide rates were selected by identifying the most common 1× rate used across different crops. Each potential safener compound was applied without herbicide and a non-treated control was included as well. The experiment was arranged in a randomized complete block design. The experiment was repeated in space in an adjacent greenhouse with the same environmental conditions. All treatments were applied 21 d after transplanting. At time of application, plants measured 10 cm in height and had three to five true leaves. The experiments were conducted from 4 June to 2 July. Plants were maintained 28 d in the greenhouse under natural light at 28 ˚C and 65% relativity humidity and evaluated based on height and visual injury on a 0–100% scale (0% = no injury to 100% = plant death), at 1, 2, and 3 wk after treatment (WAT).

2.2 | Weed antagonism greenhouse screening

Experiments were conducted in a greenhouse located at the Clemson Coastal Research and Education Center, Charleston, SC, to evaluate the effectiveness of bentazon at 514 g a.i. ha^{-1} to control yellow nutsedge and mesotrione at 105 g a.i. ha^{-1} to control Palmer amaranth when different rates of safeners were added in tank mix. To optimize germination, seeds were sown into 500-cm^3 pots containing the same proportion of sand and growing medium (Farfard Growing Mix 3B, Sungro Horticulture). Treatments were applied 2 wk after emergence at the maximum plant height recommended on the herbicide label. At the time of application, Palmer amaranth plants were approximately 5 cm in height with three true leaves, whereas yellow nutsedge plants were 10 cm in height with four true leaves.

Treatments included POST applications of bentazon at 0 and 514 g a.i. ha^{-1}, and mesotrione at 0 and 105 g a.i. ha^{-1}. Herbicides were applied alone or tank mixed with melatonin at 0, 10, 50, 100, and 500 g a.i. ha^{-1}, 24-epibrassinolide at 0, 10, 50, 100, and 500 g a.i. ha^{-1}, or AsA at 0, 10, 50, 100, and 500 g a.i. ha^{-1}, for a total of 26 treatments per weed species. The experiment was arranged in a randomized complete block design with four replications and repeated twice in time. The experiments were conducted from 10 June to 8 July . Plants were maintained 28 d in the greenhouse under natural light at 28 ˚C and 65% relativity humidity, and were evaluated based on height measurements and visual weed control on a 0–100% scale (0% = no injury to 100% = plant death), at 1, 2, and 3 WAT.

2.3 | Treatment preparation and application

Melatonin, 24-epibrassinolide, and AsA were weighed using a Mettler Toledo scale (TLE303E, SNR B705644588), and stirred using an ultrasonic homogenizer (FisherBrand, Model CL-18, Serial no. 2019070469) at 100 W for 5 min for complete solubilization. Treatments were applied using a research track sprayer (Generation 4, Serial no. SB8-270, DeVries Manufacturing), with a water carrier volume of 200 L ha^{-1} through 8002EV8 nozzles (Tee jet; Spraying Systems Company).

2.4 | Data analysis

All data were subjected to ANOVA using mixed model methodology in JMP (version 14, SAS Institute). Herbicide concentration, safener treatment, trial run, and all the interactions between these variables analyzed as a fixed effect and replication was considered random. Trial run did not have an effect on dependent variables, thus trial runs are pooled together. A logistic five-parameter equation was used to determine the herbicide dose response for each cultivar (Gottschalk & Dunn, 2005).

$$y = c + \frac{(d - c)}{\left(1 + \exp\left\{a\left[\log(b) - \log(\text{Herbicide})\right]\right\}^{f}\right)}$$

where y is the herbicide dose necessary to cause the predicted injury, c is the asymptote for low doses, d is the asymptote for high doses, a is the slope parameter, b is the inflection point, and f is the symmetrical power. The upper asymptote is the point of the growth curve representing the maximum of the parameter measured. The lower asymptote is the point of the growth curve representing the minimum of the parameter measured (Paine et al., 2012).

3 | RESULTS

3.1 | Sweetpotato greenhouse dose response screening

The cultivar, herbicide dose, safener, and their interaction had significant effects (Table 1). No differences in plant injury for both cultivars were observed among treatments when the bentazon rate was ≥1,028 g a.i. ha^{-1}. Both cultivars had similar levels of tolerance to bentazon at 514 g a.i. ha^{-1}. When bentazon was applied at 514 g a.i. ha^{-1}, injury to Beauregard was 63% (Figure 1). For Beauregard, tank mixing bentazon at

TABLE 1 Test of the effects of treatment factors and their interactions on injury caused to sweetpotato slips of cultivar Beauregard and Covington by bentazon applications, 3 wk after treatment

Source	df	SS[a]	*F* ratio	*P* > *F*
H. rate[b]	3	308,896.14	463.29	<.0001***
Cultivar	1	8,455.70	12.68	<.0001***
Safener	3	12,868.07	57.90	<.0001***
H. rate × cultivar	3	13,882.22	6.94	<.0001*
H. rate × safener	9	1,772.57	2.65	.0491*
Cultivar × safener	3	9,203.01	13.80	<.0001***
H. rate × cultivar × safener	9	2,548.04	1.27	.2522

[a]SS, sum of squares.
[b]H. rate, herbicide rate.
*Significant at the .05 probability level.
***Significant at the .001 probability level .

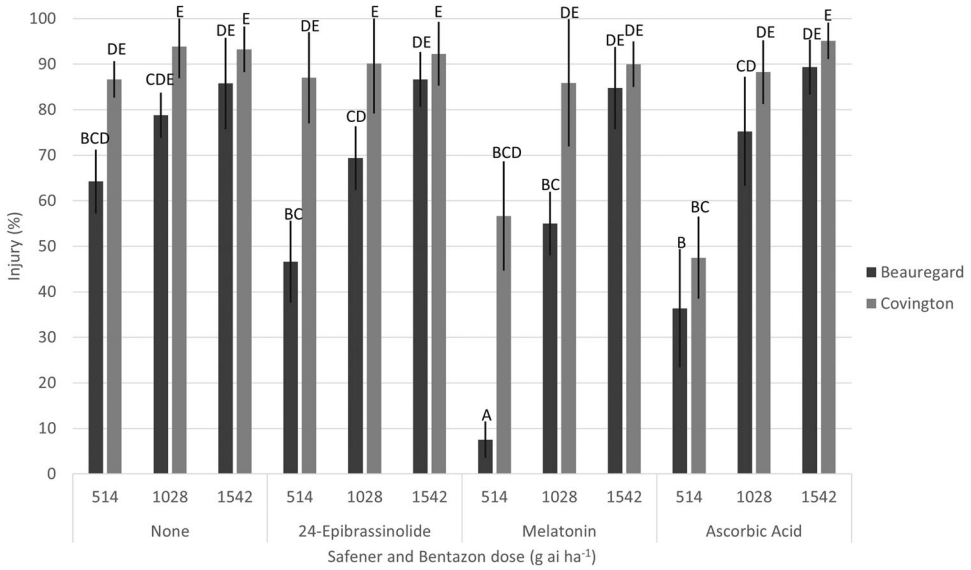


FIGURE 1 Percentage injury at 3 wk after treatment of sweetpotato cultivars Beauregard and Covington, to three doses of bentazon applied alone or in tank mix with melatonin, ascorbic acid, or 24-epibrassinolide. Injury intervals range from 0 to 100% (0% = no injury; 100% = plant death). Values are the averages of four replicates. Means with different letters are significantly different according to Tukey's multiple range tests ($P < .05$). Error bars show the standard error of the mean

514 g a.i. ha^{-1} and melatonin reduced percentage injury by 57%. The lowest rate of bentazon caused 86% injury in Covington, whereas in tank mix with AsA, we observed 38% less injury caused by the same herbicide dose. Applications containing 24-epibrassinolide in tank mix with the herbicide did not reduce percentage injury in either cultivar.

Mesotrione applied at 105 g a.i. ha^{-1} resulted in 78 and 91% injury to Beauregard and Covington, respectively (Figure 2). The cultivar, herbicide dose, safener, and their interaction had significant effects (Table 2). Beauregard plants had higher tolerance to mesotrione than Covington when herbicide was applied at 105 g a.i. ha^{-1}. The percentage injury for treatments containing mesotrione applied at 105 g a.i. ha^{-1} in a tank mix with melatonin and AsA in Beauregard was reduced by 30 and 28%, respectively. For Covington plants, melatonin was the only compound that reduced injury when applied in tank mix with herbicide at 105 g a.i. ha^{-1}, resulting in a 36% less injury at 3 WAT.

Herbicide projections were calculated to determine the dose of bentazon and mesotrione required to cause 10, 20, and 30% injury for each cultivar when applied alone or in tank mix with safeners (Table 3). Tank mixing melatonin, 24-epibrassinolide, or AsA with mesotrione increased Covington herbicide tolerance, requiring a dose six times higher to cause similar damage relative to the herbicide applied alone. However, safeners did not reduce the predicted bentazon dose to cause 10, 20, or 30% injury in Covington. The amount of bentazon required to cause 30% injury was similar when comparing bentazon alone to bentazon + additional compound, and injury above 30% would not be acceptable. The

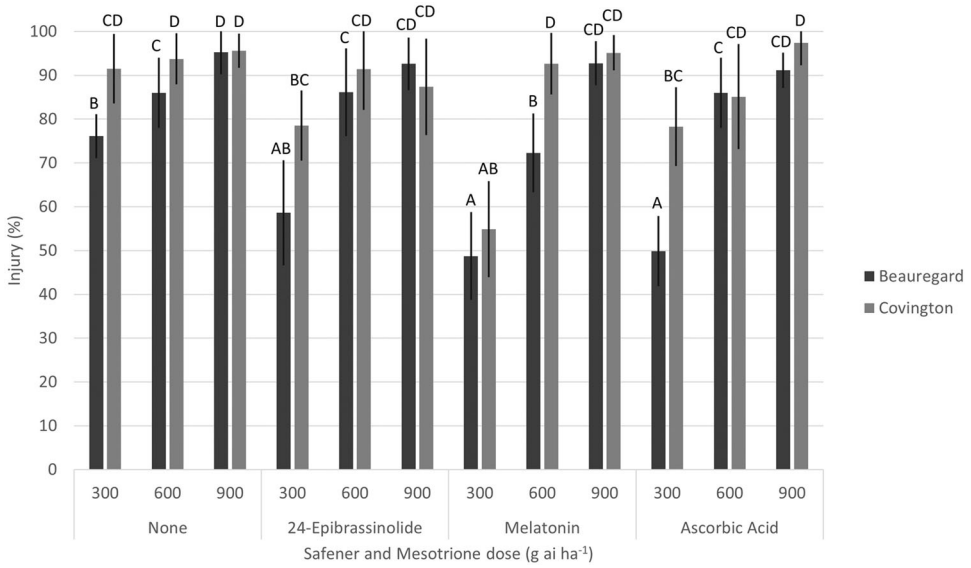


FIGURE 2 Percentage injury at 3 wk after treatment of sweetpotato cultivars Beauregard and Covington, to three doses of mesotrione applied alone or in tank mix with melatonin, ascorbic acid, or 24-epibrassinolide. Injury intervals range from 0 to 100% (0% = no injury; 100% = plant death). Values are the averages of four replicates. Means with different letters are significantly different according to Tukey's multiple range tests ($P < .05$). Error bars show the standard error of the mean

TABLE 2 Test of the effects of treatment factors and their interactions on injury caused to sweetpotato slips of cultivar Beauregard and Covington by Mesotrione applications, 3 wk after treatment

Source	df	SS[a]	*F* ratio	*P* > *F*
H. Rate[b]	3	349,060.61	737.66	<.0001***
Cultivar	1	2,842.22	18.01	<.0001***
Safener	3	3,413.67	7.21	.0001***
H. rate × cultivar	3	3,093.54	6.53	.0003***
H. rate × safener	9	5,784.88	4.07	<.0001***
Cultivar × safener	3	103.11	0.21	.8839
H. rate × cultivar × safener	9	2,162.69	1.52	.2522

[a]SS, sum of squares.
[b]H. rate, herbicide rate.
***Significant at the .001 probability level .

dose of mesotrione necessary to cause 30% injury in Beauregard was 25.18 g a.i. ha^{-1} whereas the application associated with melatonin, 24-epibrassinolide, or AsA increases the dose to 58.24, 51.90, and, 67.66 g a.i. ha^{-1}, respectively. For bentazon, the combination of herbicide and melatonin results in an improvement in plant tolerance, requiring a dose 46% higher to cause 30% injury on Beauregard.

3.2 | Weed antagonism greenhouse screening

No injury was observed in yellow nutsedge plants treated exclusively with melatonin 24-epibrassinolide or AsA compounds. When bentazon was applied at 514 g a.i. ha^{-1} injury observed 3 WAT was 96% (Figure 3). The addition of melatonin, 24-epibrassinolide, or AsA did not result in an antagonist effect, resulting in no significant differences in injury among herbicide treatments with or without safeners. Also, differences were not observed between the multiple ranges of additional compounds associated with bentazon.

A similar response was observed in Palmer amaranth plants. No concentration of melatonin, 24-epibrassinolide, or AsA caused harmful effects on plants at 3 WAT. Mesotrione provide effective control of Palmer amaranth plants, with 96% control when applied alone (Figure 4). No antagonistic effect was observed with addition of melatonin, 24-epibrassinolide, or AsA into tank mix.

TABLE 3 Estimated dose (LD) of bentazon and mesotrione required to cause 10, 20, or 30% visual injury to Beauregard and Covington sweetpotato plants with and without various safeners 3 wk after treatment

Cultivar	Herbicide	Safener	R^2	LD_{10}[a]	LD_{20}[b]	LD_{30}[c]
				g a.i. ha^{-1}		
Beauregard	Bentazon	None	.86	274.22	336.37	379.28
		Melatonin	.90	572.10	719.31	819.51
		24-Epi[d]	.71	140.04	286.40	387.85
		AsA[e]	.81	174.27	350.09	469.42
	Mesotrione	None	.95	6.10	13.24	25.18
		Melatonin	.90	17.24	37.01	59.24
		24-Epi	.90	18.67	35.49	51.90
		AsA	.89	25.90	48.09	67.66
Covington	Bentazon	None	.97	114.93	167.77	203.03
		Melatonin	.82	139.22	206.14	258.44
		24-Epi	.95	127.42	157.93	208.26
		AsA	.86	117.71	170.40	207.91
	Mesotrione	None	.98	4.08	5.09	8.31
		Melatonin	.92	16.09	39.08	54.51
		24-Epi	.85	11.79	35.86	45.04
		AsA	.85	14.53	35.56	47.17

[a]LD10, 10% of injury.
[b]LD20, 20% of injury.
[c]LD30, 30% of injury.
[d]24-Epi, 24-epibrassinolide.
[e]AsA, ascorbic acid.

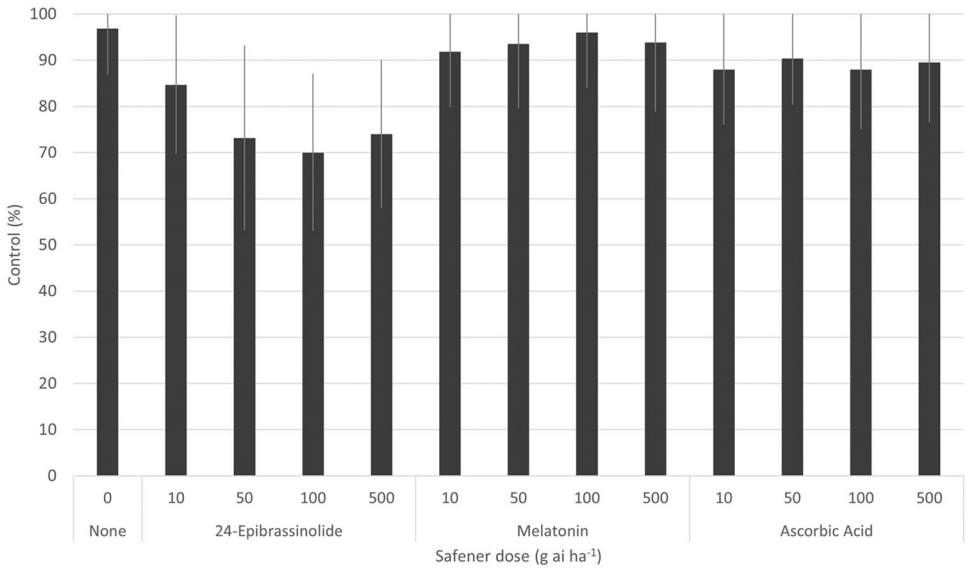


FIGURE 3 Percentage control at 3 wk after treatment of yellow nutsedge to 514 g a.i. ha^{-1} of bentazon applied alone or in tank mix with doses of melatonin, ascorbic acid, or 24-epibrassinolide. Injury intervals range from 0 to 100% (0% = no injury; 100% = plant death). Values are the averages of four replicates. Means with different letters are significantly different according to Tukey's multiple range tests ($P < .05$). Error bars show the standard error of the mean

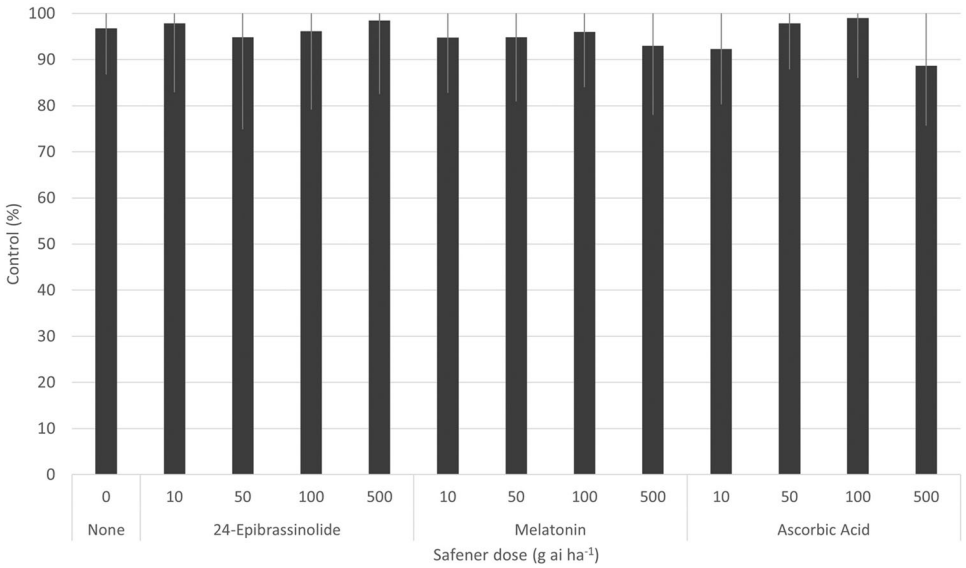


FIGURE 4 Percentage control at 3 wk after treatment of Palmer amaranth to 105 g a.i. ha^{-1} of mesotrione applied alone or in tank mix with doses of melatonin, ascorbic acid, or 24-epibrassinolide. Injury intervals range from 0 to 100% (0% = no injury; 100% = plant death). Values are the averages of four replicates. Means with different letters are significantly different according to Tukey's multiple range tests ($P < .05$). Error bars show the standard error of the mean

4 | DISCUSSION

Bentazon symptoms were observed 3 DAT and included yellowing (chlorosis) of leaf tissue followed by death (necrosis) of the tissue, beginning around the leaf margins and progressing towards the center. Similar symptoms were observed in other susceptible crops (Han & Wang, 2002; Nosratti et al., 2017; Willians & Nelson, 2014). Bentazon is a photosystem II inhibitor that competes with plastoquinone for the binding site on the D1 protein, blocking electron transport from photosystem II. This blockage induces secondary effects in several metabolic pathways, such as ROS production (Macedo et al., 2008; Pospíšil, 2009). When bentazon was applied at 540 g a.i. ha^{-1}, the percentage injury observed in Beauregard and Covington was 64 and 87%, respectively. A similar response was reported by Caputo et al. (2020), where Beauregard had a greater tolerance to bentazon in vitro. The reduction in percentage plant injury when bentazon was tank mixed with melatonin could be associated with an improvement in herbicide tolerance and antioxidant activity. Melatonin is related to improving expression of cytochrome P450 and GST related genes (Arnao & Hernández-Ruiz, 2014; Soares et al., 2016). Additionally, the ability to scavenge radical molecules makes it play a prominent role in the antioxidant defense network of plants, crucial as protectors against oxidative stress (Arnao & Hernández-Ruiz, 2014; Asensi-Fabado & Munné-Bosch, 2010).

Mesotrione applications inhibit the HPPD enzyme (p-hydroxyphenylpyruvate dioxygenase), a precursor to plastoquinone and tocopherols in oxidative degradation of chlorophyll and photosynthetic membranes (Grossmann & Ehrhardt, 2007). Mesotrione is an indirect inhibitor of phytoene desaturase, plastoquinone works as a cofactor for phytoene desaturase, the first step in the carotenoid synthesis pathway. The lack of tocopherols also contributes to plant death by decreasing ROS's buffering capacity (Matringe et al., 2005). Initial stages of symptoms were observed in 5–7 DAT, starting with reduced leaf pigmentation and progressing to total chlorosis of leaves and necrosis. For Covington, the only improvement in herbicide tolerance was observed when melatonin was tank mixed with mesotrione. Conversely, for Beauregard, AsA and melatonin increased plant tolerance to the lowest herbicide dose. However, just melatonin reduced the injury caused by 210 g a.i. ha^{-1} (Figure 2). 24-Epibrassinolide is a member of the brassinosteroids. This class of plant hormone has been related to upregulated pesticide metabolism. Zhou et al. (2015) reported an intrinsic capacity of 24-epibrassinolide applications in co-upregulated genes related to pesticide detoxification, reducing residues of organophosphorus residues, organochlorine, and carbamate pesticides by 30–70% on multiple crops.

Herbicide projections show higher toxicity of mesotrione than bentazon to sweetpotato, requiring lower doses to cause 10, 20, and 30% injury 3 WAT (Table 3). Cultivars had different tolerance responses to herbicides. For both herbicides alone, dose predictions were higher for Beauregard' than Covington. Some compounds did not greatly improve tolerance to mesotrione or bentazon. However, it is expected that herbicides would be more active in the greenhouse than the field, which would mean that these safener

concepts should be included in future field trials. Beauregard has a greater inherent tolerance in metabolizing xenobiotics. Caputo et al. (2020) reported a predicted dose of bentazon 2.5× higher for Beauregard than Covington to cause the same injury level in vitro environment. In a screen of sweetpotato germplasm to clomazone injury conducted by Harrison and Jackson (2011b), Beauregard had ∼10× greater tolerance than sensitive cultivars. Postemergent experiments with halosulfuron demonstrated that Beauregard experienced less storage root injury than Covington (Dittmar et al., 2013), whereas Cutulle et al. (2020) reported that Beauregard was ∼50× more tolerant to *S*-metolachlor, compared with other cultivars.

The use of safeners in a tank mix with herbicide had a positive effect on plant tolerance. In some cases, the injury observed was high even using safeners; however, the dose prediction data could provide important information to design a chemical weed management with low injury to sweetpotato plants. Herbicide response is highly dependent on abiotic factors that can result in decreased cuticle thickness and increased herbicide uptake resulting in more activity in the greenhouse than the field (Varanasi et al., 2016). Thus, the significant decrease in injury observed with some of the treatment combinations relative to mesotrione or bentazon alone is promising. When compared with results reported by Caputo et al. (2020) in vitro, less injury due to bentazon was observed in Beauregard and Covington. Finally, the expected injury due to bentazon in the field might be less, since the plants would be hardier.

Mesotrione and bentazon are herbicides labeled to control Palmer amaranth and yellow nutsedge, so the herbicide treatments were expected to significantly control the weeds. Antagonism is the negative relationship between two or more compounds, resulting in less than the predicted effect of each herbicide applied separately (Merritt et al., 2020). The risk of using non-crop-specific safeners in tank mix is that although it increases the tolerance of nontarget plants, it may reduce the herbicide efficiency on targeted weeds. To minimize that, herbicide applications must follow the technical recommendations to reach full weed control. In these experiments, the treatments were applied when the weeds reached the maximum height recommended on the herbicide label. No reductions in chemical efficiency were observed when safeners were added into tank mixtures with herbicides, reaching the same levels of weed control of herbicide applications alone.

5 | CONCLUSION

In conclusion, this study demonstrated interesting interactions between potential safener compounds and herbicides as it relates to sweetpotato injury. The lower percentage herbicide injury observed in sweetpotato plants when safeners were included in the tank mixture validates those compounds' protective ability. A potential concern when using nonspecific safeners is the antagonist interaction with an herbicide, reducing an herbicides activity to control weeds. This study demonstrated that the labeled herbicide rate applied at maximum plant height effectively controlled the target weeds when either 24-epibrassinolide, AsA, or melatonin were included in the tank-mix. Additional physiological- and biochemical-based research is necessary to gain a better understanding of the interactions between herbicides and melatonin, 24-epibrassinolide, and AsA on plant metabolism. To justify a label expansion, field trials would be necessary to characterize the interaction of mesotrione and bentazon with safeners in commercial settings. Ideally, these trials could generate data to promote label expansion of a much-needed POST herbicide for yellow nutsedge and Palmer amaranth control in sweetpotato.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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