ABSTRACT

FODERARO, AMANDA MARIE. Investigation of North Carolina Palmer amaranth Response to Very-Long-Chain-Fatty-Acid Inhibiting Herbicides at North Carolina State University. (Under the direction of Dr. Wesley Everman).

Palmer amaranth (*Amaranthus palmeri* S. Watson) is well known now as one of the most widespread, troublesome and economically damaging agronomic weeds in the southeastern U.S. In recent surveys, it has been reported as one of the top 5 most common and troublesome weeds. Its propensity to evolve resistance has only further increased the difficulty in its management. Since the first report of herbicide resistant Palmer amaranth in the late 1980's, it has developed resistance to a total of 9 WSSA modes of action. The more recent reports of resistance to Group 15 herbicides, specifically *S*-metolachlor, in Arkansas and Mississippi have raised concerns regarding the future of Group 15 herbicides and their critical role in effective weed management. Competition with Palmer amaranth is season long and the use of residual herbicides at planting and with postemergence applications to overlap residuals are foundational steps for season-long control. Residual, preemergence herbicides applied directly to the soil, such as VLCFA-inhibitors, need to persist long enough in the soil to provide adequate weed control throughout the duration of the critical weed control period. Understanding what residual herbicides are available and how they can be utilized successfully to ensure season long control is critical when planning an effective weed management program. In North Carolina, Palmer amaranth continues to threaten cropping systems with increased reports of resistance to glyphosate, ALS, PPO and more recently HPPD and PSII. These increases in documented resistance further limits PRE and POST herbicide control options and increases reliance on other modes of action including Group 15/VLCFA-inhibiting herbicides, thus increasing the probability of selecting for additional resistance.

Greenhouse studies were conducted to evaluate the differential response of two Palmer amaranth populations from Wake and Martin County, North Carolina, to five Group 15 herbicides, *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet. One "susceptible" population from Wake County and one "tolerant" population from Martin County were evaluated. The difference in population response to Group 15 herbicides was evaluated by way of dose response and plant back bioassay in the greenhouse.

The results of the dose response study indicated a significant differential response between the two populations to the Group 15 herbicides. R/S ratios were calculated, and the more tolerant Martin County population required 4.1 times more *S*-metolachlor, 5.1 times more acetochlor and 10.5 times more dimethenamid-P to reduce seedling emergence by 50% compared to the more susceptible Wake County population. For the residual efficacy study, the average control demonstrated by the LD_{90} rate determined in the dose response study, was evaluated for each herbicide on each population at 0, 2, 4 and 6 weeks following application. The same "susceptible" population from Wake County and one "tolerant" population from Martin County, were evaluated. The results of the study indicated a significant difference in the overall control of each population. Results also indicated a significant difference between herbicides in the average control of both Palmer amaranth populations over time with pyroxasulfone and *S*-metolachlor averaging higher levels of control over time compared to acetochlor, dimethenamid-P and flufenacet. The estimated I_{50} (weeks until a 50% reduction in control) for *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet was 6.7, 1.2, 1.42, 7 and 2.19 weeks, respectively. The differential responses in the subject Palmer amaranth populations to multiple Group 15 herbicides indicate the potential presence of segregating populations in the Coastal Plain of North Carolina that could be a future concern.

Investigation of North Carolina Palmer amaranth Response to Very-Long-Chain-Fatty-Acid Inhibiting Herbicides

by
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DEDICATION

I would like to dedicate this to my beautiful daughters, Piper and June and my wonderful husband, Mike. Piper and June, you have been my inspiration in ways you will never understand. Mike, thank you for your unwavering love and support over all these years. I could not have done this without you.

BIOGRAPHY

Amanda Foderaro grew up in Millersville, Maryland with her parents Anne and KC and younger brothers Kyle and Kory. She came to North Carolina in 2005 to pursue her undergraduate degree in Biology and Psychology with Minors in Chemistry and Humanities from Greensboro College. It was also there she met her husband, Mike. After graduating and a few years of traveling the world together, Amanda and Mike settled back in Greensboro in 2011. There Amanda started her career at Syngenta where she was introduced into the world of Farming and Agriculture. After working several years in Regulatory at Syngenta, Amanda decided she wanted to take the next step in her education and deepen her knowledge of the Agricultural industry. She started the Masters Program in Crop Science at North Carolina State, January 2018. She also happened to be 12 weeks pregnant with her first daughter, June. In between her full-time job, giving birth to 2 children and experiencing a global pandemic, she managed to complete her course work and conduct her Masters research experiment on site at Syngenta the Spring of 2021 and 2022. She is very excited to have made it to this stage and is extremely grateful to those who have helped along the way.

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There are so many folks that I owe a tremendous amount of gratitude and thanks to. Without my husband and his limitless patience, I would not have made it to the end of this journey. To my children, who I hope will read this someday, you inspire me every day to do better and be better. You are brave; you are smart; you are beautiful; and you can do anything you set your mind to.

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CHAPTER 1: LITERATURE REVIEW

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Palmer amaranth

Amaranthus or amaranth, a genus of summer annual plants native to the southwestern United States, is commonly known as the genus containing some of the world's most common and troublesome weeds. The Amaranthus genus contains nearly 75 species worldwide with an estimated 50 species that are native to the Americas (Steckel 2007; Kigel 1994) and are commonly referred to as "pigweeds". Pigweeds are generally characterized as having extended periods of germination, high relative growth rate, and are known as prolific seed producers (Mitich 1997; Weaver 1984; Bensch 2003; Keeley et al. 1987). Examples of weedy amaranths include redroot pigweed (*Amaranthus retroflexus* L.), smooth pigweed (*Amaranthus hybridus* L.), spiny amaranth (*Amaranthus spinosus* L.), waterhemp (*Amaranthus tuberculatus*) and Palmer amaranth (*Amaranthus palmeri* S. Wats.). Of the pigweed species, Palmer amaranth is arguably the most competitive and aggressive. It is a dioecious, small-seeded, annual broadleaf weed that is described as one of the most widespread, troublesome and economically damaging agronomic weeds in the southeastern U.S (Ward et al. 2013; Chahal et al. 2015; Chandi et al. 2012; Korres et al. 2019; Tekiela and Sbatella 2017).

Palmer amaranth has been shown to grow taller and accumulate greater biomass than other species in the pigweed family (Bertucci et al. 2019; Spaunhorst 2016). It was found to have the highest photosynthetic rate among C_4 plants at 81µmol/m2/s (Ehleringer 1983) which is three to four times the rate of many row crops including C_4 corn and C_3 cotton and soybeans (Gibson 1998; Steckel 2007). In addition, Palmer amaranth leaves are known for having diaheliotropic movement where they orient themselves perpendicular to incoming sunlight allowing the plant to take full advantage of its photosynthetic capacity (Ehleringer 1983). High photosynthetic rates and diaheloptropic movement enable rapid growth. Its growth rate has been reported to be as

much as 2 inches per day under full light (Horak and Loughin 2000). Palmer amaranth has the adaptive ability to increase solute concentrations in the leaves in order to maintain positive turgor and keep stomata open under drought conditions making it highly durable and competitive in high temperatures and dry conditions (Ehleringer 1985; Ward 2013).

Palmer amaranth is well known as a prolific seed producer with a single female plant producing as many as 600,000 seeds when grown in isolation. When grown in competition with crops, a single female Palmer amaranth plant can produce >100,000 seeds (Fine 2020; Keeley et al. 1987). It has also been shown to produce a larger quantity of seed when compared to other pigweed species such as common waterhemp or redroot pigweed, at low weed densities making it highly competitive amongst crops and fellow weed species (Bensch 2003). By nature of its small size, Palmer amaranth seed can easily be transported and disseminated by various measures including wind, irrigation water, animals, contaminated agricultural machinery, contaminated cover crop seed, manure, and contaminated livestock feed sources (Farmer et al. 2017; Norsworthy et al. 2014; Spaunhorst 2016). Further, its extended emergence pattern makes it difficult for preemergence and nonresidual postemergence herbicides to control later-emerging plants making competition with Palmer amaranth season long (Chandi et al. 2012; Korres et al. 2019; Tekiela et al., 2017).

These characteristics have made Palmer amaranth a troublesome weed in important cropping systems including cotton, corn, peanut and soybean (Ward 2013). Its evolution as a troublesome weed began with the first report of Palmer amaranth being outside its original habitat in Virginia in 1915 (Culpepper 2010). By 1995 Palmer amaranth was listed as the most troublesome weed in cotton in North Carolina and South Carolina (Murdock 1995). By 2009, it was ranked as the most troublesome in cotton in 9 southern states and second most troublesome

in soybean (Webster and Nichols 2012). By 2014, Palmer amaranth had become one of the most troublesome and economically important weed species in corn, cotton and soybean in the United States (Webster and Nichols 2012; Beckie 2006; Norsworthy 2014). In the 2019 survey it was reportyed as the number one most common and most troublesome weed amongst broadleaf crops, fruits and vegetables; specifically listed as the number one most troublesome and common weed in cotton, top 2 most troublesome and common in peanuts and top 7 most troublesome and common in soybean (Van Wychen 2019). In the 2020 Weed Science Society of America (WSSA) survey, Palmer amaranth was reported as one of the top 5 most common and most troublesome weed species in corn, sorghum, grass crops, pasture and turf **(**Van Wychen 2020). Limiting its spread is a nationwide challenge. It is estimated that more than 80% of the United States is ideal for the establishment of Palmer amaranth (USDA 2020; PPQ 2020). Yield reduction from Palmer amaranth interference has been reported as high as 91% in corn (*Zea mays* L.) (Chahal et al. 2015; Bensch et al. 2003; Massinga et al. 2001), 68% in soybean (*Glycine max* L. Merr.)(Klingaman and Oliver 1994), 79% in sweet potato (*Ipomoea batatas* L. Lam.) (Basinger et al. 2019), 63% in sorghum (*Sorghum bicolor* L. Moench) (Moore et al. 2004), 92% in cotton (*Gossypium hirsutum* L.)(Chandi et al. 2012), 67% in peanut (*Arachis hypogaea* L.) (Burke et al. 2007) and 77% in dry edible bean (Miranda et al. 2021,2022). Once established, Palmer amaranth is difficult to manage and has the potential to become a threat to the profit of agronomic and horticultural crop productions (Mahoney et al. 2019; Spaunhorst 2016). The widespread adoption of no-tillage systems, reduced reliance on soil-applied residual herbicides and increased herbicide resistance have contributed toward the increased infestation of Palmer amaranth in different cropping systems (Horak 2000; Mayo 1995; Sweat 1998).

Development of herbicide resistance in Palmer amaranth

Palmer amaranth is one of ten Amaranthus species that are dioecious (separate male and female plants). As an obligate outcrosser, the potential for greater genetic diversity is higher helping to facilitate the spread of herbicide resistance (Ward 2013; Steckel 2007, Tranel et al. 2011; Franssen 2001). Weed resistance to herbicides can be conferred either by target site resistance (TSR) and/or non-target site resistance (NTSR) mechanisms. TSR mechanisms largely involve mutation(s) in the target site of action of an herbicide, resulting in an insensitive or less sensitive target protein for the herbicide (Powles 2010; Shyam 2019). In such cases, the TSR is determined by monogenic traits (Délye 2013). In addition, weeds can evolve TSR as a result of overexpression or amplification of the target gene (Sammons 2014).

NTSR mechanisms include reduced herbicide uptake/translocation, increased herbicide detoxification, decreased herbicide activation rates, and/or herbicide sequestration (Devine 1997). Metabolism-based NTSR involves increasing the activity of enzyme complexes such as esterases, cytochrome P450s, glutathione S-transferases (GSTs), and/or UDP-glucosyl transferases (Powles 2010). NTSR is usually governed by many genes (polygenic) and may confer resistance to herbicides with completely different modes of action. (Délye 2013; Preston 2003). Evolution of NTSR via means of herbicide detoxification is a serious threat to weed management, as it can bestow resistance to multiple herbicides, leaving growers with limited herbicide options for weed control as well as granting weeds with potential resistance to herbicides not yet commercially available (Ma et al. 2013). Furthermore, it has been proposed that low herbicide doses result in the evolution of polygenic traits, whereas high herbicide doses may favor monogenic target site-based resistances (Gressel 2011; Neve et al. 2005).

Palmer amaranth has evolved resistance to multiple herbicide modes of action including WSSA Groups 2 (ALS-inhibitors) (Horak and Peterson 1995), 3 (Microtubule-inhibitors), 4

(Auxins), 5 (PSII-inhibitors), 9 (EPSP Synthase-inhibitors), 27 (HPPD-inhibitors) (Parminder 2015; Mahoney 2019), 14 (PPO-inhibitors) (Salas et al. 2016) and more recently Group 15 (VLCFA-inhibitors) have been reported across the country (Brabham 2019; Heap 2019) Table 1. The evolution of herbicide resistant Palmer amaranth, specifically to glyphosate can be attributed to rapid adoption of glyphosate-resistant crops between 1996 and 2006, resulting in an increase in glyphosate reliance and subsequent shift in herbicide use patterns from PRE followed by POST herbicides with multiple modes of action (MOA) to POST only applications of glyphosate (Duke 2015, Givens et al. 2009; Powles 2008). Increased reliance on glyphosate subsequently led to a drastic reduction in herbicide diversity and induction of weed species shifts thus accelerating the evolutionary rate of glyphosate resistant weeds, including Palmer amaranth (Culpepper 2006; Green 2009; Johnson et al. 2009; Kniss 2018; Owen 2008; Owen and Zelaya 2005; Webster and Nichols 2012). With the increase of glyphosate resistant weeds throughout the United States, the use of additional herbicide modes of action has become necessary for effective chemical weed management (Hager et al. 2003; Prince et al. 2012; Riggins and Tranel 2012; Werle et al. 2018).

There are also increasing reports of Palmer amaranth populations having evolved resistance to more than one mode of action (Burgos et al. 2001, Culpepper et al. 2006; Gaeddert et al. 1997; Horak and Peterson 1995; Norsworthy et al. 2008; Sosnoskie et al. 2011; Sprague et al. 1997; Steckel et al. 2008; Wise et al. 2009). Herbicide resistance, particular multiple herbicide resistance, further limits growers' ability to manage and sustainably maintain crop production. The use of synthetic pesticides has been the predominant source of weed control for decades and the continued evolution of resistance puts the continued reliance on this method at risk.

Both TSR and NTSR resistance mechanisms have been found to confer resistance to herbicides in Palmer amaranth. TSR mechanisms involving alterations in the target site of the herbicide such as amino acid substitutions or deletions and increased copy number and/or expression of the target gene have been reported in this species (Shyam 2019; Gaines et al. 2010; Salas et al. 2016; Nakka et al. 2017). Mutations in the gene encoding herbicide-targeted enzymes can reduce herbicide-binding activity leading to resistance. Specifically, in Palmer amaranth single amino acid substitutions, i.e., A122S or A122T, P197S or P197A, T574L, and S653A were reported to confer resistance to ALS-inhibitors in weed species (Küpper et al., 2017; Nakka et al. 2017c; Singh et al. 2019). Palmer amaranth populations resistant to PPO-inhibitor herbicides were found to have the amino acid substitutions R128M/G (also referred as R98), and G399A, as well as a codon (glycine) deletion at the position 210 (Δ210) in *PPX2* gene coding for the target enzyme of PPO-inhibitor herbicides (Salas et al. 2016; Giacomini et al., 2017; Salas-Perez et al. 2017; Varanasi et al. 2017; Rangani et al. 2019). Another commonly identified TSR mechanism in Palmer amaranth is the amplification of the *EPSPS* gene, the molecular target of glyphosate (Gaines et al. 2010; Chahal et al. 2017; Molin et al. 2017; Singh et al. 2018). Furthermore, the amplified *EPSPS* gene copies are present in the form of extrachromosomal circular DNA (eccDNA), with an autonomous replication site, and are randomly inherited during cell division (Koo et al. 2018; Molin et al. 2020). After the first case of glyphosate-resistant Palmer amaranth from Georgia, United States in 2005, it has rapidly spread throughout the United States becoming a serious challenge for agriculture. Apart from *EPSPS* amplification, a mutation in the *EPSPS* gene leading to P102S substitution has also been reported in Palmer amaranth (Kaundun et al. 2019).

NTSR mechanisms do not directly alter the target site but reduce the amount of active herbicide reaching the target site due to either reduced absorption, translocation, or increased metabolism of the herbicide (Shyam 2019). Specifically, in metabolic resistance, the active herbicide is broken down into non-toxic forms before it reaches the target site, thus reducing its efficacy (Shyam 2019). Reduced absorption and translocation imparting glyphosate resistance in a Palmer amaranth population from Argentina have been reported (Palma-Bautista et al., 2019). However, enhanced herbicide detoxification *via* cytochrome P450 monooxygenase (P450s) and glutathione *S*-transferase (GSTs) activity is the most common NTSR mechanism reported in ALS-, PS II-, HPPD-, and PPO-inhibitor-resistant Palmer amaranth (Nakka et al. 2017a, b, c; Varanasi et al. 2018).

The importance of incorporating preemergence herbicides into weed management practices is becoming more critical and more emphasized due to Palmer amaranth becoming more and more difficult to control with traditional postemergence herbicides. In grower fields where glyphosate and ALS-resistant Palmer amaranth are prevalent, the use of residual herbicides at planting and with postemergence applications to overlap residuals are foundational steps for season-long control (Brabham et al. 2019; Culpepper et al. 2009; Norsworthy et al. 2012; Riar et al. 2013). VLCFA-inhibiting (Group 15) and PPO inhibiting herbicides (Group 14) with residual activity are commonly used PRE herbicides (Brabham et al. 2019; NC State Extension 2021). With the increased difficulty in controlling resistant weeds with POST applied herbicides, the use of PRE herbicide, such as Group 15 herbicides, has generally increased. The soybean production area treated with PRE herbicides substantially increased from 2006 through 2017, particularly with *S*-metolachlor (15%) (USDA 2020), indicating higher herbicide mode of action diversity for weed control in soybean cropping systems (Kniss 2018). In 2018, *S*-

metolachlor was applied to 28.3 million acres of corn (USDA NASS). In 2020 Group 15 herbicides accounted for 20.1 million pounds of the active ingredients used on soybean acres (USDA 2020). Group 15 herbicides have also become a common component in PRE tank mixes combined with POST herbicides including Group 5, Group 27, Group 14 and Group 9 herbicides.

The evolution and consequent risk of spread of resistance to PPO and VLCFA inhibiting herbicides is becoming more of a concern with the first confirmation of resistance to a Group 15 herbicide being reported in 2019 (Brabham 2019; Heap 2019).

Group 15 herbicides/Very Long Chain Fatty Acids (VLCFA)

WSSA Group 15 Herbicides or HRAC Group K3s are Long Chain Fatty Acid Inhibitors which are applied preemergence for residual control of annual grasses and some small-seeded broadleaf weeds in a variety of crops (Al-Khatib 2022). Very-Long-Chain-Fatty-Acid (VLCFA)-inhibitors can be grouped into three major chemical families; chloroacetamides (Examples: *S*-metolachlor, acetochlor and dimethenamid-P), pyrazole (Example: pyroxasulfone) and oxyacetamides (Example: flufenacet). Chloroacetanilides or chloroacetamides are represented by the amide derivatives of chloroacetic acid. *S*-metolachlor and acetochlor are 2,6-dialkylanilides in which an alkoxyalkyl group is also found on the aniline nitrogen atom (Székács 2021). Other examples of chloroacetamides include alachlor, dimethachlor, metazachlor (which contains a heterocyclic pyrazolylmethyl group on the aniline nitrogen), and propisochlor (containing a branched alkyl group in its alkoxyalkyl moiety, developed in Hungary) (Székács 2021). The introduction of these compounds ranges from the 1960s to the early 1990s. Dimethenamid, introduced in 1991, contains a dimethylthienylamide group instead of the anilide moiety. Dimethenamids pure *S*-isomer, dimethenamid-P was registered as a separate active ingredient in

2000 (Székács 2021). Pyrazoles are a heterocyclic series that are characterized by a ring structure composed of three carbon atoms and two nitrogen atoms in adjacent positions (National Center for Biotechnology Information 2021). Oxyacetamides are considered an acetamide derivatives, in which the aromatic moiety is attached to the amide nitrogen atom. Flufenacet, introduced in 1995, contains a trifluoromethylthiadiazolyl group as a substituent on the hydroxyl group (Székács 2021).

VLCFA-inhibitors have been an effective herbicide site of action utilized in weed management programs for many years (LeBaron et al. 1988). First discovered by Monsanto Company in 1952, these herbicides are thought to inhibit very long chain fatty acid synthesis (Hamm 1974; Shaner et al. 2014, Böger 2003; Tanetani 2009, Trenkamp 2004). Fatty acids with a carbon chain length of greater than 18 C-atoms are referred to as VLCFA and are critical building blocks for cuticular waxes, storage lipids in seeds, periderm and endodermis components, phosholipds and sphingolipids (Böger 2003; Trenkamp 2004; Matthes 2002). Phospholipids and sphingolipids are required for proper vesicle trafficking and membrane dynamics, especially in dividing cells (Bach et al. 2011; Jenks et al. 2002; Li-Beisson et al. 2010). Cuticular waxes in the leaves and suberin in the roots constitute an important barrier to the environment in the epidermal tissues of seedlings (Busi 2014; Post-Beittenmiller 1996). A plasma membrane lacking VLCFAs will lose its rigidity and permeability associated with leakage and impaired cell division (Böger 2003)

VLCFA are created through the sequential additions of C2 moieties to C18 fatty acids that are derived from fatty acid synthesis in the plastid via “plastidic biosynthesis” (Böger 2003, Eckermann 2003). The elongation process occurs in the endoplasmic reticulum and is mediated by multiple complexes of four enzymes, known as the “elongase system”, that catalyze

sequential condensation, ketoacyl reduction, dehydration and enoyl reductions steps. The reactions proceed with the CoA-activated fatty acid intermediates (outputs from the plastidic biosynthesis), at the end of the process the CoA is split off. The condensation is the starting reaction which is catalyzed the condensing enzyme 3-ketoacyl-CoA synthase (KCS) FAE1 within the elongase enzymatic complex. This is believed to be the target site of VLCFA. Further evidence suggests that different herbicides may differentially inhibit different subsets of condensing enzymes. Many of these compounds are thought to bind the KCS elongase-condensing enzyme via covalent bonding at the conserved cysteine of the reactive site (Böger 2003; Eckermann 2003). A similar enzyme-herbicide binding mechanism is thought to drive the GST-mediated conjugation of VLCFA herbicides with glutathione to confer crop selectivity (Fuerst 1987; Böger 2000; James 1995; Millar 1997).

VLCFA-inhibitor compounds typically affect susceptible weeds prior to emergence but do not inhibit seed germination (Shaner et al. 2014). They are absorbed after germination during internode elongation in emerging seedlings ultimately impacting root and shoot development (Fuerst 1987; Dhillon and Anderson, 1972). Most susceptible grass and broadleaf weeds fail to emerge because the growth of apical meristem and coleoptile are interrupted after germination (Shaner et al. 2014; Cobb and Reade, 2010). Susceptible monocots that do emerge appear twisted and malformed with leaves tightly rolled in the whorl and unable to unroll normally. Broadleaf seedlings may have slightly cupped or crinkled leaves and shortened leaf midribs producing a drawstring effect on the leaf tip. VLCFA-inhibitors do not control emerged weeds (Fuerst 1987).

Of the VLCFA-inhibitors utilized for control of grasses and broadleaf weeds, *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufencet are some of the more commonly used herbicides in the United States (Table 2).

***S*-metolachlor**, which is the resolved isomer of metolachlor, was registered in 1997. New formulations based primarily on the *S*-metolachlor isomer are more active on a gram-for-gram basis than metolachlor formulations composed of a 50:50 a racemic mixture of the R and S isomers (Shaner et al. 2006). It is effective at application rates around 35% lower than original metolachlor. It is formulated for both preemergence and/or postemergence uses in corn, cotton, peanut, pod crops, potato, safflower, grain/forage sorghum, soybean, nursery and landscape plantings and certain turfgrass species as well as a number of minor crops (Environmental Protection Agency 2020). *S*-metolachlor is used for control of yellow nutsedge and many annual grasses (foxtail, barnyard grass, crabgrass, fall panicum, signalgrass, witchgrass and red rice) as well as certain small-seeded broadleaf weeds such as redroot pigweed and Palmer amaranth, carpetweed and Florida pusley (Shaner et al. 2014). Application rates for *S*-metolachlor vary by formulation, crop, application timing, soil type, organic matter content etc. In soybean, rates of Dual Magnum range from 1.33 to 1.67 pints per acre and 1.33 to 2.6 pints in corn. Table 2.

During the most recent five years of available agricultural usage data (2014-2018), an annual average of approximately 53.5 million pounds of *S*-metolachlor were applied to approximately 49.4 million acres of agricultural crops (Kynetec 2019).

Acetochlor, first registered in 1994 is a chloroacetanilide herbicide registered for preplant incorporated and/or preemergence use in field corn, soybeans, cotton, sorghum, peanuts, sweet corn, sugar beets, fallow land, as well as Miscanthus and other non-food perennial bioenergy crops. It provides pre-emergence (to the weed) residual control of most annual

grasses, yellow nutsedge and certain small-seeded broadleaf weeds in soybeans and corn. Acetochlor can be formulated as an emulsifiable concentrate, soluble concentrate, microencapsulation, ready to use solutions, wettable powders, and granular products. Acetochlor can be applied via soil incorporation, aerial, broadcast, band sprayer, ground and chemigation equipment (Environmental Protection Agency 2021; Shaner et al. 2014). Application rates for Acetochlor vary by formulation, crop, application timing, soil type, organic matter content etc. In soybean, rates of Warrant range from 1.25 quarts to 2.0 quarts. In corn, rates of Harness range from 1.5 to 3 pints per acre. Table 2.

According to market research, 45 million pounds of acetochlor were applied to over a total of 35 million acres of crops annually between 2014 and 2018 (Kynetec 2019; Environmental Protection Agency 2021).

Dimethenamid-P, another chloroacetanilide, was first registered by EPA in 1992 and is currently registered for use in dried beans, chickpeas, cotton, corn, fallow lands, garbanzo beans, garlic, hops, horseradish, leeks, lentils, onions, peanuts, potatoes, scallions, shallots, squash, sorghum, soybeans, sugar beets, and grass grown for seed. Dimethenamid-P is also used on major non-agricultural use sites including landscape and grounds maintenance areas, tree plantations, turfgrass areas, golf courses, ornamental gardens, and commercial ornamental production sites. Registered formulation types include emulsifiable concentrates (EC), granules (G), and as a liquid impregnated onto dry bulk fertilizer formulations. Products can be applied using ground, aerial, backpack/handheld sprayers, and soil incorporation equipment, and liquid impregnated dry bulk fertilizer. It can be applied preemergence and/or postemergence for control of many annual grasses such as foxtail, barnyard grass, fall panicum and crabgrass as well as yellow nutsedge and certain annual broadleaf weeds including redroot pigweed and black

nightshade in corn and soybeans. (Environmental Protection Agency 2021; Shaner et al. 2014). Application rates for Dimethenamid-P vary by formulation, crop, application timing, soil type, organic matter content etc. For corn and soybean, rates of Outlook range from 12 to 21 fluid ounces per acre. Table 2.

From 2014-2018, an annual average of 210,000 lbs dimethenamid were applied to about 270,000 acres of corn, dry beans/peas, sorghum, soybeans, and sweet corn (Kynetec 2019; Environmental Protection Agency 2021).

Pyroxasulfone, of the pyrazole class of chemistry was first registered in 2012, with additional crops registered as recently as 2019. It can be formulated as a suspoemulsion (used to combine two active ingredients with very different physical properties into one formulation), suspension concentrate, or as water-dispersible granules. It may be applied preemergence and/or postemergence for control of grasses and small-seeded broadleaves such as Italian ryegrass, barnyardgrass, foxtails, crabgrasses, Palmer amaranth and common waterhemp in corn, wheat and soybean in addition to several minor crops. Applications can be made by air, or by ground as broadcast, banded or spot treatments, or via dry bulk fertilizer impregnation. (Environmental Protection Agency 2021; Shaner et al. 2014). Application rates for Pyroxasulfone vary by formulation, crop, application timing, soil type, organic matter content etc. In soybeans, application rates of Zidua SC range from 3.25-5 fluid ounces per acre and 1.75 to 6.5 fluid ounces per acre in corn. Table 2.

Growers reported pyroxasulfone usage soon after registration in a variety of registered crops for which it is surveyed; however, usage data for pyroxasulfone by growers of individual crops are limited due to the recent approval of the active ingredient. Between 2015 and 2019, on

average approximately 1,000,000 pounds of pyroxasulfone were applied annually to treat 11,000,000 acres of agricultural crops (Kynetec 2019; Environmental Protection Agency 2021).

Flufenacet, an oxyacetamid, is registered in the United State in premixtures only. It may be applied either preplant surface, preplant incorporated and/or preemergence for selective control of most annual grasses and certain small-seeded broadleaf weeds and suppression of nutsedge. It is registered for use in a variety of crops including corn, soybeans, cotton, peanut, wheat, sunflower, and potatoes. (Environmental Protection Agency 2016; Shaner et al. 2014). Application rates for flufenacet vary by formulation, crop, application timing, soil type, organic matter content etc. In soybeans, application rates of Axiom DF, a mixture product containing metribuzin and flufenacet, range from 7-13 fluid ounces per acre and 8-23 fluid ounces per acre in corn. Table 2.

Rates for each herbicide vary by formulation and crop. See Table 2 for comparison of rates for registered products on key crops. In addition, please see Table 3 for comparison of Group 15 Chemical Structure and Properties.

Residual efficacy of VLCFA-inhibitors

Preemergence herbicides applied directly to the soil, such as VLCFA-inhibitors, need to persist long enough in the soil to provide adequate weed control throughout the duration of the critical weed control period. The persistence of a given herbicide in soil may affect both its agronomic properties, such as residual weed control. How long a pesticide persists in a soil is a function of its loss from the soil system and degradation (Hurle and Walker 1980; Monaco 2002; Peter and Weber 1985). Persistence is closely related to the kinetics of degradation, which depends on the molecular structure of a pesticide, and is also influenced by factors such as dose/application rate, temperature, soil water content/rainfall, pH, oxygen level, background

level of the pesticide, soil fertility, tillage and management system and microbial populations (Blume 2004, Gaynor et al. 2000; Poppell et al. 2002; Ma et al. 2004; Shipitalo and Owens 2006). The persistence of pesticides in soils is often indicated by DT_{50}, the time for 50% of initial residues to dissipate. When dissipation of a pesticide in soils can be adequately described by first-order kinetics, DT_{50} is equal to the first-order dissipation half-life, which is the time required for 50% dissipation of residual concentration at any given time (Ma et al. 2004).

Herbicides can be lost from a soil system as a result of volatilization, leaching, surface runoff, loss of soil containing sorbed herbicide and/or herbicide breakdown via chemical decomposition, photodecomposition and/or biological decomposition. (Hance 1980; Kah and Brown 2006; Monaco et al. 2002, Kwon et al. 2004; Torstensson 1980). The predominant loss of VLCFA-inhibitors, specifically, from the soil is due to microbial degradation (Beestman and Deming, 1974; Baran 2004). Zimdahl and Clark (1982) showed that herbicide degradation rates for chloroacetamide herbicides increased as temperature and moisture content increased, which supports the hypothesis that degradation of chloroacetamide herbicides is dominated by microbial degradation (Westra et al. 2014).

There is extensive research on the environmental fate and persistence of pesticides (Gaynor et al. 2000; Poppell et al. 2002; Ma et al. 2004; Shipitalo and Owens 2006; Hance 1980; Kah and Brown 2006; Monaco et al. 2002, Kwon et al. 2004; Torstensson 1980). This paper has focused on the primary systems of degradation for Group 15 herbicides, mainly being microbial degradation, and the characteristics of the subject herbicides that impact these systems.

S-metolachlor: The research around *S*-metolachlor and Metolachlor environmental fate is extensive. Adsorption and desorption are key processes controlling herbicide efficacy and dissipation in soil and influences a compound's environmental fate, persistence in the soil, and

biological activity (Laabs & Amelung 2005). Nonionic herbicides such as *S*-metolachlor can be sorbed to soil particles by Van der Waals forces, hydrophobic partitioning, charge-transfer complexes, ligand exchange and covalent bonding or a combination of these interactions (Berry & Boyd 1985; Dec & Bollag 1997). Sorption of *S*-metolachlor in soil has been positively correlated to organic matter and clay content (Weber et al. 2003; Gannon et al., 2013). A variation in organic matter content from 0.9 to 5.7% increased the sorption coefficient (Kd) of metolachlor by approximately 6-fold (Weber et al., 2003). Another study demonstrated that Kd values of *S*-metolachlor were 1.08 and 9.32 L kg-1 in soils with 1.2 and 4.5% organic matter content, respectively (Gannon et al. 2013). In addition, as organic matter content gradually decreases with depth in the soil profile, an increase on herbicide retention in the topsoil layer is expected (Alletto et al. 2010). This was reported by Bedmar et al. 2011 who found 1.78-fold higher adsorption of *S*-metolachlor in soil surface (0-5 cm) with 4.4% of organic carbon than in subsurface soil (>81 cm) with 0.2% of organic carbon content. Organic matter and clay content also influence desorption process of *S*-metolachlor. In general, metolachlor exhibits hysteresis in soils with high organic matter and clay content (Youbin et al., 2009). This process occurs due to the existence of irreversible binding interaction with the soil constituents or slowly reversible chemical sorption that causes a decrease in the desorbed amount of the herbicide (Zhu & Selim, 2000; Youbin et al., 2009). Metolachlor hysteresis has been demonstrated by Pignatello & Huang (1991), Zhu & Selim (2000) and Youbin et al. (2009). Typically compounds with lower water solubility will have higher sorption coefficient values and will result in higher amounts of herbicide bound to the soil (Bailey 1968). *S*-metolachlor is considered to be moderately soluble in water at 488 mg/L at 20 ° C and adsorbs moderately to soil at 200 mL/g with variability depending on the soil type (Zemolin et al. 2014).

Microbial degradation is considered the major pathway for metolachlor transformation in soil (Accinelli et al., 2001; Ma et al., 2006). Photodegradation contributes to *S*-metolachlor dissipation in soil under prolonged drought periods when the herbicide remains near to the soil surface (Camargo 2007). Metolachlor half-life in soil ranges from 2.5 to 289 days (Sanyal & Kulshrestha, 1999; Wu et al., 2011) according to soil management, edaphic factors and environmental conditions. Of the current chloroacetamide herbicides, *S*-metolachlor appears to be the most persistent (LeBaron et al. 1988; Walker et al. 1985; Zimdahl and Clark 1982). Overall, microbial degradation appears to be the major pathway for *S*-metolachlor dissipation in soil (Zemolin et al. 2014).

Another consideration in assessing a herbicides persistence is solubility. Typically compounds with lower water solubility will have higher sorption coefficient values and will result in higher amounts of herbicide bound to the soil (Bailey 1968). *S*-metolachlor is considered to be moderately soluble in water at 488 mg/L at 20 ° C and adsorbs moderately to soil at 200 mL/g with variability depending on the soil type (Shaner et al. 2014).

Acetochlor: Acetanilides, such as acetochlor, are non-polar, non-ionizable herbicides and are considered moderately to highly soluble in water, moderately persistent in environment, and moderately to highly mobile in soil (Oliveira et al. 2013). Acetochlor itself is considered moderately soluble in water at 233 mg/L at 25 ° C and moderately adsorpent to soil at 170 mL/g (Shaner et al. 2014). There is variability and reported half-life for acetochlor. Acetochlor DT_{50} values have been estimated to be very short (≤10 days) for a number of soils (Mills et al. 2001; Ferri and Vidal 2002; Ma et al. 2004; Baran et al. 2004; Dictor et al. 2008). Other research indicates, the reported half-life (DT_{50}) of acetochlor in the field ranges from 3.4 to 29 days, with a mean value of 12.9 days (Mills et al. 2001, Baran et al. 2004, Newcombe et al.

2005). Jablonkai et al. 2000 reports an average half-life of 90 days. Acetochlor is expected to have low leaching potential in most soils and its mobility correlates well with Kd and OM content. (Breaux 1986; Böger 2000; Jablonkai and Hatzios 1991; Scarponi et al. 1991).

Dimethenamid-P: Dimethenamid-P is considered to be highly soluble in water at 1174 mg/L at 25 °C and moderately adsorps to soil at 155 mL/g. According to the WSSA Herbicide Handbook (Shaner et al. 2014), Dimethenamid-P is reported to have an average half-life of 35-42 days. Pesticide Properties Data Base (PPDB) lists a 7-day half-life in the field. There are various reports throughout literature. Kočárek 2018 investigated the impact of adjuvants on dimethenamid-P behavior in soil and reported that the dimethenamid-P dissipation half-lives ranged from 8.8 days for irrigated treatment without adjuvant to 12.9 days for non-irrigated treatment with adjuvant. Dimethenamid-P dissipation half-life in treatments with adjuvant was significantly longer than was half-life in a treatment without adjuvant. Significantly longer dissipation half-life was observed also in non-irrigated treatments than in irrigated treatments.

Jursík et al. 2020, investigated the effect of different soil and weather conditions on the efficacy and selectivity and dissipation of herbicide on sunflowers. Six sunflower herbicides, including *S*-metolachlor and dimethenamid-P, were tested at two application rates (dimethenamid-P 1 000 and 2 000 g/ha a.i.; *S*-metolachlor 1 150 and 2 300 g/ha a.i.) on three locations (with different soil types) within three years (2015–2017). The results suggest that weather conditions, and especially rainfall, play a key role in herbicides behavior and can have stronger effects than soil properties. Persistence of the tested herbicides was significantly shorter at the 1N rate compared to the 2N rate. Overall, the weakest persistence was shown in case of dimethenamid-P (40% of the applied rate was detected 30 days after application in the 0–5 cm soil layer) (Jursík et al. 2020).

Pyroxasulfone: Pyroxasulfone is considered to have low solubility in water at 3.49 mg/L at 20 °C and moderately adsorps to soil ranging from 57-114 mL/g. It has low leaching potential with less mobility in fine and medium textured soils and more mobility in coarse textured soils. (Tanetani 2009) There is a strong correlation between soil binding, reduced herbicide dissipation, and increased soil organic matter content (Westra et al. 2014). Pyroxasulfone is nonionizable, and therefore soil pH does not affect dissociation. However, soil pH may influence bioactivity due to fewer negative charges on organic and clay surfaces in soils with lower pH (Szmigielski et al. 2014). There is variability in the average reported half-life of pyroxasulfone. According to the WSSA Handbook (Shaner et al. 2014), Pyroxasuflone is not considered to be persistent with a half-life of 16-26 days. Westra et al. 2014 evaluated pyroxasulfone dissipation and mobility in the soil compared to *S*-metolachlor in 2009 and 2010 at two field sites in northern Colorado, on a Nunn fine clay loam, and Olney fine sandy loam soil. Pyroxasulfone dissipation half-life (DT_{50}) values varied from 47 to 134 d. Between years, herbicide DT_{50} values were similar under the Nunn fine clay loam soil. Under the Olney fine sandy loam soil, dissipation in 2009 was minimal under dry soil conditions. In 2010, under the Olney fine sandy loam soil, pyroxasulfone had a half-life of 47 d. Overall, DT_{50} values ranged from 46 to 48 d for pyroxasulfone, compared to previously cited literature that reported DT_{50}s of 8 to 71 d for pyroxasulfone (Mueller and Steckel 2011).

Flufenacet: Flufenacet is considered to have moderate solubility in water at 56 mg/L at 25 °C and moderately adsorps to soil at an average of 355 mL/g depending on soil type. A soil half-life of 34 days has been reported when flufenacet was first presented (Deege et al. 1995) and subsequent research has corroborated. Research indicates that soil moisture has little effect on the dissipation of flufenacet in soil. However, the rate of application and soil type greatly

affected it. At higher rate, the dissipation was slower. It is observed that dissipation of flufenacet is slower in soils having high adsorption capacity (either high OM or clay content) and slower desorption (Gupta et al. 2002). Flufenacet is also reported to strongly adsorb to alluvial soil (Gupta et al. 2001). Under aerobic microbial degradation half-life ranges from 10 to 34 days in various soils. Under anaerobic conditions flufenacet has been reported to have a DT50 up to 240 days. Overall, Flufenacet is expected to have short to moderate persistence and low to moderate mobility in most soil textures. (Shaner et al. 2014).

Please refer to Table 4 for a summary of some of the reported DT_{50}/half-life values throughout the literature.

Comparison Across Group 15/VLCFA: Pyroxasulfone dissipation and mobility in the soil was evaluated and compared to *S*-metolachlor in 2009 and 2010 at two field sites in northern Colorado, on a Nunn fine clay loam, and Olney fine sandy loam soil. Pyroxasulfone dissipation half-life (DT_{50}) values varied from 47 to 134 d, and those of *S*-metolachlor ranged from 39 to 63 d. Between years, herbicide DT_{50} values were similar under the fine clay loam soil. Under the fine sandy loam soil, dissipation in 2009 was minimal under dry soil conditions. In 2010, under the fine sandy loam soil, *S*-metolachlor and pyroxasulfone had half-lives of 39 and 47 d, respectively, but dissipation rates appeared to be influenced by movement of herbicides below 30 cm. Herbicide mobility was dependent on site-year conditions, in all site-years pyroxasulfone moved further downward in the soil profile compared to *S*-metolachlor. (Westra et al. 2014)

Westra et al. 2012 evaluated sorption coefficients for pyroxasulfone and *S*-metolachlor across 25 different soils. There was a high correlation between sorption coefficients and organic matter (OM) for pyroxasulfone and *S*-metolachlor, which has been previously documented (Kozak et al. 1983). In the Westra et al. 2014 study, herbicide mobility was the greatest under

lighter-textured soils compared to other locations that received 57 and 64% more irrigation, which suggests that herbicide mobility was influenced more by soil type than moisture content. Across all site years pyroxasulfone moved further downward in the soil profile compared to S-metolachlor as supported by sorption coefficient values for these two compounds (Westra, 2012). Throughout the duration of the study at one site, the average DT_{50} values ranged from 134 to 104, and 63 to 57 d for pyroxasulfone and *S*-metolachlor, respectively. The calculated half-lives for *S*-metolachlor in the Westra et al. 2014 study were consistent with other results (Braverman et al. 1986; Mersie et al. 2004; Shaner et al. 2006; Walker and Brown 1985; Zimdahl and Clark 1982) that showed ranges of DT_{50}s from 10 to 142 d. Pyroxasulfone DT50s, the values from the Westra et al. 2014 study ranged from 47 to 134, slightly longer than previously cited literature that reported DT_{50}s of 8 to 71 d for pyroxasulfone (Mueller and Steckel 2011).

2017 research investigating the persistence of acetochlor, atrazine and *S*-metolachlor in surface and subsurface horizons, reported that overall, degradation of *S*-metolachlor was low compared with degradation rates obtained with the other herbicides tested (Bedmar 2017). In general, herbicide persistence across all soils and horizons ranged from 15 to 73 d for acetochlor and 82 to 141 d for *S*-metolachlor, which had significantly ($p < 0.01$) greater persistence than acetochlor (Bedmar 2017). Adsorption was considered to be likely the most influential process in determining persistence of acetochlor and *S*-metolachlor in surface and subsurface horizons (Bedmar 2017).

Ribeiro et al. 2021 evaluated the length of soil residual weed control of 11 PRE soybean herbicides in greenhouse bioassays. Palmer amaranth, giant foxtail, radish and cereal rye were used as bioindicaters of herbicide levels in the soil. Bioassay results showed extended soil residual control of Palmer amaranth with Pyroxasulfone. Pyroxasulfone (intercept = 8.9%, slope

= 0.063) was reported as one of the most detrimental PRE herbicides to Palmer amaranth through the soil sampling period. Palmer amaranth grown in soil treated with pyroxasulfone, presented 15%, 41%, and 66% biomass compared with the nontreated control at 100, 500, and 900 Growing Degree Days (GDD), respectively. *S*-metolachlor also resulted in significant Palmer amaranth biomass reduction (≤33% biomass compared with the nontreated control) at 100 GDD. The Palmer amaranth population used in this study was confirmed to be resistant to an ALS-inhibitor herbicide imazethapyr at 70 g ai ha–1 (Oliveira et al. 2020), explaining why ALS-inhibitor herbicides were not as effective. The additional PRE herbicides evaluated were less effective in controlling Palmer amaranth, indicating Pyroxasulfone and/or *S*-metolachlor can be effective PRE herbicide options to control ALS-inhibitor-resistant Palmer amaranth populations in soybeans (Ribeiro et al. 2021).

Persistence across herbicides is also impacted by formulation type. Vasilakoglou et al. 2001 evaluated activity, adsorption, and mobility of emulsifiable concentrate (EC) and microencapsulated (ME) formulations of alachlor and acetochlor as well as of metolachlor, *S*-metolachlor, dimethenamid and flufenacet were studied. Petri-dish bioassay, based on root response of oats (*Avena sativa* L.), was used for their activity in sand and in a silty clay loam soil, and for determination of herbicide concentrations in soil solution (not adsorbed) and in column leachates of the adsorption and mobility studies respectively. Flufenacet and both acetochlor formulations showed the highest activity in both soils and ME-alachlor and metolachlor the lowest; the activity of dimethenamid, EC-alachlor and *S*-metolachlor was intermediate. Activity of both formulations of alachlor and acetochlor decreased with increasing organic matter content, but alachlor activity was reduced more than that of acetochlor. Lower amounts of dimethenamid-P and *S*-metolachlor were adsorbed by soil compared with the other

herbicides and, consequently, greater amounts of these two herbicides were leached through that soil. None of the herbicides tested was detected below 30 cm. Less alachlor and acetochlor were biologically available in soil solution after their application as ME-formulations and, therefore, lower amounts of both ME-alachlor and ME-acetochlor were leached through the soil compared with those applied as EC-formulations. Vasilakoglou et al. 2003 evaluated the persistence of microencapsulated (ME) and emulsifiable concentrate (EC) formulations alachlor and acetochlor, as well as EC formulations of metolachlor, *S*-metolachlor, dimethenamid-P and flufenacet were studied using a bioassay based on root response of oat. Flufenacet was found to be the most persistent of the herbicides, but biologically available residues were not detected at 0- to 10-cm soil depth 50 d after any herbicide treatment.

There are differences between the commercially available VLCFA-inhibitors and their formulations, specifically in the amount of precipitation needed for activation. (Hart et al, 1995, Shrefler and Chandler 1994, Hay et al., 2018). Hay et al. 2018, evaluated residual control of Palmer amaranth (*Amaranthus palmeri* S. Watson) and common waterhemp (*Amaranthus rudis* Sauer) with very-long-chain fatty acid (VLCFA) inhibiting herbicides in the field. Six VLCFA inhibiting herbicides were applied at three different rates (high, middle, and low) based on labeled rate ranges for soybean [*Glycine max* (L.) Merr.]. Percent Palmer amaranth and common waterhemp control was visually estimated at 4 and 8 weeks after treatment (WAT). At one location, pyroxasulfone, *S*-metolachlor, and dimethenamid-P resulted in the greatest Palmer amaranth control at both 4 and 8 WAT. At other locations, pyroxasulfone, *S*-metolachlor, and non-encapsulated acetochlor resulted in the highest Palmer amaranth and common waterhemp control at both 4 and 8 WAT. Pyroxasulfone and *S*-metolachlor were often the most effective herbicides. The high use rate across all herbicides resulted in better control when compared to

the low use rate across all herbicides at all sites and observation times. This research demonstrates the value of utilizing VLCFA inhibiting herbicides as an effective site of action for residual control of Palmer amaranth and common waterhemp as part of integrated weed management plan. Results also supported that timely rainfall for activation is required to move the VLCFA inhibiting herbicide into soil solution to ultimately reach the target site. Delayed activation can contribute to reduced control (Hay 2018).

In short, there are a large number of factors that may affect the efficacy soil applied herbicides. In the specific case of VLCFA-inhibitors applied preemergence, the length of residual efficacy is critical in maintaining season long control of damaging weeds. While there is a large amount of research looking across the various impacts of soil type, weather conditions, application rate, formulation type etc. there is limited research looking at how residual control of VLCFA-inhibitors may vary across a specific weed species i.e. Palmer amaranth, which is known for its inherent genetic variability and ability to develop resistance to herbicides rapidly. The ability of VLCFA-inhibitors to maintain residual control of Palmer amaranth throughout the entire season is essential in minimizing the development and spread of resistant accessions. The following research was conducted to investigate the differences in residual efficacy of PRE applied Group 15 herbicides in control of North Carolina Palmer amaranth accessions.

Resistance in very long chain fatty acid-inhibitors (VLCFA)

Despite the global use of VLCFA-inhibitors occurring over several decades, the development of resistance has been slow to evolve. It has been suggested that target-site mutations in the condensing enzyme might occur less frequently because of the potential loss of function being lethal for the plant (Böger 2003). Another theory is that these herbicides interact with many primary targets and it would require changes in multiple targets occurring at the same

time in order to deliver resistance with the likelihood of this occurrence being very low (Trenkamp 2004, Tanetani 2009). Nonetheless, resistance to VLCFA-inhibitors, though rare, has evolved and has been confirmed in at least 13 species worldwide, 8 of which are found in the United States (International Herbicide Resistant Weed Database, 2022). The majority of the species are grass species and include flufenacet-resistant blackgrass (*Alopecurus myosuroides* Huds.)(Heap 2019) and Italian ryegrass (*Lolium perenne* subsp. multiflo-rum (Lam.) Husnot] (Liu et al. 2016), pyroxasulfone-resistant wild oat (*Avena fatua* L.) (Mangin et al. 2017), butachlor-resistant barnyardgrass (*Echinochloa crus-galli* L. Beauv.) (Juliano et al. 2010), and chloroacetamide- and pyroxasulfone-resistant rigid ryegrass (*Lolium rigidum* Gaud.) (Burnet et al. 1994; Busi et al.2014).

The evolution of VLCFA-inhibitor resistant weeds began with rigid ryegrass (*Lolium rigidum Gaud*.) identified as resistant for the first time in 1982, wild oat (*Avena fatua L*.) in 1989, Chilean needlegrass (*Nassella neesiana)* in 1992, barnyardgrass (*Echinochloa crus-galli L. P. Beauv*.) in 1993, Annual bluegrass (*Poa Annua*) in 1994, Late watergrass (*Echinochloa pyhllopogon*) in 1998, Serrated tussock grass (*Nasseslla trichotoma*) in 2002, Giant Parramatta grass (*Sporobolus fertilis*) in 2004, Italian ryegrass (*Lolium multiflorum Lam*.) in 2005, and the most recently new species of herbicide resistant grass species being blackgrass (*Alopecurus myosuroides Huds*.) in 2007 (Heap 2022a).

In most cases, resistance was found only in multiple-herbicide–resistant accessions, and resistance was not always the result of selection from VLCFA-inhibiting herbicide use. In these cases, resistance to VLCFA-inhibiting herbicides was hypothesized to be conferred by glutathione *S*-transferases (GST) like the tolerance mechanism found in corn (*Zea mays* L.) and sorghum (*Sorghum bicolor* L.) (Busi et al. 2014; Deng and Hatzios 2002; Dixon et al. 1997). It

was determined that pyroxasulfone was rapidly detoxified within 24 hours in resistant rigid ryegrass plants by GSTs, and they furthermore identified increased GST expression that correlated with resistance (Busi et al. 2018). Literature suggests that grasses have a greater ability to evolve herbicide detoxification mechanisms mediated by enhanced metabolic activity (Délye 2013).

The first resistance in a broadleaf species was confirmed in 2018 in both waterhemp (*Amaranthus tuberculatus* Moq. J.D. Sauer) and Palmer amaranth (*Amaranthus palmeri* S. Watson) (Heap 2022a; Brabham 2019; Strom 2019). In the case of Brabham et al. 2019, GSTs were found to be involved in *S*-metolachlor resistance.

Resistance of palmer amaranth to VLCFA-inhibitors

Up until the confirmation of herbicide resistant waterhemp and Palmer amaranth in 2018, it was generally assumed that the ability for resistance to VLCFA-inhibitors to develop was unlikely or rare. The relatively low level of resistance could be due to naturally less selection pressure compared to postemergence pesticides. This is due to the decreased occurrence of VLCFA-inhibitors being used in isolation i.e. without a postemergence application (often glyphosate). As a result, preemergence herbicides are often used in conjunction with a postemergence herbicide offering. This ultimately results in less selection pressure from preemergence herbicides as compared to postemergence which are more frequently used on their own (Somerville et al. 2017).

As suggested previously, the opportunity for target site mutations in is rare. In 2003, Böger et al. presented a general framework for why that might be the case. First, condensing enzymes like the VLCFA synthase have an active SH-site (Protein docking site) represented by a required cysteine residue in a highly conserved reaction center (Böger, 2003). Cysteine

residues play the crucial role in the final three-dimensional conformation of the protein molecule, and make it stable. The cysteine is necessary for both enzyme activity and inhibitor binding. This binding is assumed to be a covalently irreversible one (Böger 2003, Böger 2000). Second, metabolic detoxification requires the gluthione (GSH) system and the increase of several enzymes to produce more or to regenerate GSH, namely glutathione-S transferase (GST), GSH-reductase, and enzymes for biosynthesis of GSH. The likelihood of multiple enzymatic detoxification processes occurring simultaneously in order to confer resistance is unlikely (Böger 2003; Böger 2000). Third, this detoxification requires certain GST isoforms (Somner et al. 1999; Jepson et al., 1997, Böger 2003) which may not be present or cannot be induced in many weeds, particularly not in dicots (Riechers 2003; Böger 2003). Detoxification by the P450-monooxygenase system may occur. Although P450-mediated modification of several herbicides is well-known, at the time no conclusive evidence had been reported that such metabolic resistance plays an effective role for chloroacetamides. It has been shown that O-demethylation of metolachlor or alachlor in microsomes processed at a very low rate (Moreland et al. 1995). Finally, it has been demonstrated that a replacement of VLCFAs by excess “normal length” fatty acids (C18) leads to high chloroacetamide resistance and little impairment of growth (Couderchet et al. 1995). It is unlikely, however, that a fatty-acid profile change will be allowed for a viable reproductive plant, due to loss of membrane rigidity (Böger, 2003).

In Crittenden County, Arkansas Brabham et al. conducted field and greenhouse studies to assess the susceptibility of Palmer amaranth accessions with suspected resistance to VLCFA-inhibitors. Both field and greenhouse studies confirmed a decrease in control across all tested VLCFA-inhibitors with confirmed resistance in two of the accessions to *S*-metolachlor (Brabham 2019). It was estimated that the two confirmed resistant accessions required two-thirds and 1.5

times the 1X rate of *S*-metolachlor (1,064 g ha^{-1}) to obtain 90% mortality compared to the susceptible populations. For the other VLCFA-inhibitors, acetochlor, dimethenamid-P and pyroxasufone, a 1.5 to 2.1-fold and 2.3-to-3.6-fold increase in herbicide rate was estimated to achieve 50% mortality for the same two accessions (Brabham 2019).

Additional work was done to assess how the accessions may be conferring resistance. Based on the data there is evidence to suggest that GST's may have played a role. It is also believed that enhanced herbicide detoxification via cytochrome P450 monooxygenase (P450s) and glutathione S-transferase (GSTs) activity is the most common NTSR mechanism in multiple-herbicide-resistant Palmer amaranth (Nakka et al., 2017a, b, c; Varanasi et al., 2018).

Similar work was conducted in Champaign County, Illinois on waterhemp populations with confirmed resistance to HPPD, PSII, ALS and PPO chemistry. Dose response work conducted in the greenhouse corroborated what was seen in the field studies, that being reduced control, and ultimately confirmed an increase in survival rates (LD_{50}) of the multiple-resistant populations to *S*-metolachlor, acetochlor, dimethenamid-P and pyroxasulfone as compared to the sensitive accessions. R:S ratios were generated with the resulting LD_{50} values and ranged from 4.5 to 64 for the multiple resistant populations among all four VLCFA-inhibiting herbicides evaluated.

Since the original confirmation of resistance to a Group 15 herbicide by Brabham et al. 2019, several investigations into Palmer amaranth accessions have been made across the mid/south-West. Rangani et al. 2021 reported *S*-metolachlor resistance in five accessions collected in 2014 and 2015 from Crittenden and Woodruff counties in Mississippi. 3 to 29-fold resistance levels were reported based on LD_{50} values of resistant accessions ranging from 88 to 785 g ai ha^{-1}. In 2022, Roma-Burgos et al. reported six accessions (four from Arkansas and two from Mississippi)

as resistant to *S*-metolachlor. Researchers evaluated both parent and progeny accessions with LD_{50} for the parent accessions ranging between 73 and 443 g ai ha^{-1} and the F1 progeny LD_{50} rates ranging from 73 to 577 g ai ha^{-1}. The resistance level was reported as generally greater among the progenies of surviving plants than among the resistant field populations. The resistant field populations required 2.2 to 7.0 times more *S*-metolachlor to reduce emergence 50% while the F1 of survivors needed up to 9.2 times more herbicide to reduce emergence 50% compared to the susceptible standard (Kouame 2022). Refer to Table 5 for a comparison of *S*-metolachlor LD_{50} values reported across studies.

With confirmed VLCFA-inhibitor resistance in two separate broadleaf species is of major significance to the agricultural community. Further research is needed to understand the physiological mechanisms for resistance in these species in hopes to prevent any further spread and keep the remaining available modes of action effective for as long as possible.

Implications for North Carolina Agriculture

Soil applied residual herbicides have been regularly used by North Carolina growers for decades and have always served as a key component in a successful weed management program. The emphasis on their importance has grown recently due to the rapid spread of resistance driven by the high selection pressure of years of POST only herbicide applications in GM crops. Palmer amaranth has evolved nationally into one of the most economically damaging herbicide-resistant weed species in the U.S. (Beckie 2006) and specifically in North Carolina it has been listed as one of the most troublesome weeds as recently as 1995 (Dowler 1995). According to a 2016 report published by the Weed Science Society of America's Weed Loss committee, it is estimated that uncontrolled weeds in North Carolina soybean would reduce yields by 47.4%. This potential yield loss corresponds to an approximately $240 million loss in value (North

Carolina Soybean Production Guide, 2022). With difficult to control Palmer amaranth populations continuing to evolve and spread, North Carolina key cropping systems are at risk.

The average seed production from North Carolina Palmer amaranth populations in competition with corn, cotton, peanut and soybean ranges from 51,000 to 273,000 seeds per plant (Mahoney 2020). Further, North Carolina Palmer amaranth populations continue to demonstrate a growing resistance to glyphosate and ALS inhibitors in key cropping systems (Mahoney 2020; Poirier et al. 2014). Currently in North Carolina there is confirmed weed resistance to 7 WSSA Modes of Actions including groups 1, 2, 3, 5, 9, 14 and 27, across 10+ weed species (Table 6). Palmer amaranth resistance has been confirmed for ALS, glyphosate, HPPD and recently PSII-inhibitors (Heap 2022b). Previous work done by Poirier et. al (2014) and Mahoney (2020) specifically in the North Carolina Coastal Plain area, demonstrated resistance to glyphosate and ALS inhibitors has increased rapidly in just a 5-to-10-year period. In addition, the first documented incidence of resistance to a HPPD inhibitor in NC was recorded in 2019 (Mahoney 2020) and the opportunity for increase is anticipated due to the expected increase in the use of mesotrione and other HPPDs on tolerant cotton and soybean (Mahoney 2020). These increases in documented resistance in North Carolina Palmer amaranth accessions, further limits PRE and POST herbicide control options and increases reliance on other modes of action including, VLCFA-inhibiting herbicides, thus increasing the probability of selecting for additional resistance. This is further supported by the majority of cases where VLCFA resistance has been identified, the accessions were already categorized as having multiple herbicide resistance (Strom 2019). With this increased pressure on selection for resistant species comes increased importance in the successful incorporation of a preemergence residual herbicide in weed management systems.

In 2021, additional screening studies were conducted by Moore et. al. (2021) to further evaluate the sensitivity of Palmer amaranth accessions collected by Mahoney et al. (2020) to atrazine, dicamba, *S*-metolachlor and 2,4-D. Differences in Palmer amaranth survival were observed for *S*-metolachlor and 2,4-D when comparing across accessions (Moore et al. 2021).

The following research was conducted to further investigate the differences observed between the Palmer amaranth accessions in the Moore et al. (2021) study and characterize the susceptibility and residual control of the selected accessions to Very-Long-Chain-Fatty-Acids (VLCFA) herbicides applied preemergence (PRE).

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FIGURES AND TABLES

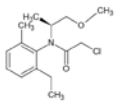
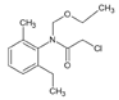
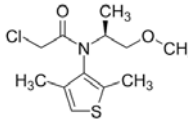
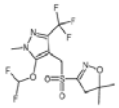
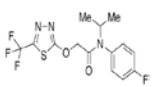
Table 1: List of Mode of Actions associated with Herbicide Resistant Palmer amaranth

WSSA Group (HRAC)	Site of Action	First Year	Crop/Country
2 (B) Ex. imazethapyr	Inhibition of Acetolactate Synthase (ALS)	1993	Alfalfa, Corn (maize), Sorghum, and Soybean/ United States (Kansas)
3 (K1) Ex. Trifluralin	Inhibition of Microtubule Assembly	1989	Cotton, and Soybean/ United States (South Carolina)
4 (O) Ex. 2,4-D	Auxin Mimics	2015	Sorghum/United States (Kansas)
5 (C1/2) Ex. Atrazine	PSII – Serine 264 Binders	1993	Corn and Sorghum/ United States (Texas)
9 (G) Ex. Glyphosate	Inhibition of Enolpyruvyl Shikimate Phosphate Synthase (EPSPS)	1995	Cotton and Soybean/ United States (Georgia)
14 (E) Ex. fomesafen	Inhibition of Protoporphyrinogen Oxidase (PPO)	2011	Soybean/ United States (Arkansas)
15 (K3 N) Ex. *S*-metolachlor	Very Long-Chain Fatty Acid Synthesis inhibitors (VLCFA)	2016	Soybean/ United States (Arkansas)
27 (F2) Ex. mesotrione	Inhibition of Hydroxyphenyl Pyruvate Dioxygenase (HPPD)	2016	Corn/ United States (North Carolina)

Table 2: Comparison of Product Rates for VLCFA-Inhibitor Herbicides

Herbicide	Common Commercial Name(s)	lb ai per gal	Rate
S-metolachlor	Dual Magnum Dual II Magnum	7.62 lb ai / gal	1.33-1.67 pt/A (Soybean)
acetochlor	Harness Warrant	7 lb ai / gal 3 lb ai/gal	2.25 - 2.75 pt/A (Soybean)
dimethenamid-P	Outlook	6 lb ai / gal	14-18 fl oz/A (Soybean)
pyroxasulfone	Zidua ZC	4.17 lb ai / gal	3.25-5 fl oz/A (Soybean)
flufenacet	Sunfire Axiom DF	500 g ai / L	0.48 L/ha (Soybean)

Table 3: Comparison of VLCFA-Inhibitor Chemical Properties[1]

Chemical Pesticide	**CAS No.**	**Chemical Family**	**Molecular Formula**	**Structure**	**Density** (g/cm3)	**Molecular Weight** (g/mol)	**Water Solubility** (mg/L)	**Soil Adsorption (KOC)** (mL/g)
S-metolachlor	87392-12-9	Chloroacetamide	$C_{15}H_{22}ClNO_2$		1.12	283.8	488 (20 °C) (Moderate)	200 (Moderate)
acetochlor	34256-82-1	Chloroacetamide	$C_{14}H_{20}ClNO_2$		1.1±0.1	269.8	223 (25 °C) (Moderate)	170 (Moderate)
dimethenamid-P	163515-14-8	Chloroacetamide	$C_{12}H_{18}ClNO_2S$		1.2±0.1	275.8	1174 (25 °C) (High)	155 (Moderate)
pyroxasulfone	447399-55-5	Pyrazole	$C_{12}H_{14}F_5N_3O_4S$		1.598	391.3	3.49 (20 °C) (Low)	57-114 (Moderate)
flufenacet	142459-58-3	Oxyacetamide	$C_{14}H_{13}F_4N_3O_2S$		1.4±0.1	363.3	56 (25 °C) (Moderate)	344 Average (Moderate)

[1]Chemical Structures and properties obtained from Shaner et al. 2014 and/or Pesticide Properties Data Base (PPDB)

Table 4: Summary of reported DT_{50}/half-life for *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet

Herbicide	Reported Half-life (days)	Reference
S-metolachlor	112-124	Shaner et al. 2014
	10-142	Braverman et al. 1986; Mersie et al. 2004; Shaner et al. 2006; Walker and Brown 1985; Zimdahl and Clark 1982
	82 to 141	Bedmar 2017
	37.9-135.9	Wu et al. 2011
	17.4	Fava et al 2000
	69.3	Coroi et al 2011
	5.7-100.7	Dinelli et al 2000
	50-289	Rice et al. 2002
	6-9.6	Mersie et al 2004
	10-23	Staddon et al 2001
	2.5-18	Sanyal & Kulshrestha 1999
	28-34	Nunes & Vidal 2008
	97-127	Alletto 2013
	23.17-51.8 (Moderately persistent)	Pesticide Properties Data Base
Acetochlor	<10 days	Mills et al. 2001; Ferri and Vidal 2002; Ma et al. 2004; Baran et al. 2004; Dictor et al. 2008
	3.4 to 29 (Avg 12.9)	Mills et al. 2001, Baran et al. 2004, Newcombe et al. 2005
	90	Jablonkai 2000
	15 to 73	Bedmar 2017
	12.1-14 (Non-persistent)	Pesticide Properties Data Base
Dimethenamid-P	35-42	Shaner et al. 2014
	11-15.8 (Non-persistent)	Pesticide Properties Data Base
	8.8-12.9	Kočárek 2018
Pyroxasulfone	16-26	Shaner et al. 2014
	47 to 134	Westra et al. 2014
	8 to 71	Mueller and Steckel 2011
	22 (Non-persistent)	Pesticide Properties Data Base
Flufenacet	10-34 (aerobic) Up to 240 (anaerobic)	Shaner et al. 2014
	9.3-22.5	Gupta 2001
	19.7-39 (Non-Moderately persistent)	Pesticide Properties Data Base

Table 5: Comparison of reported LD_{50} and R:S ratios of *Amaranthus palmeri* and *Amaranthus tuberculatus* populations to *S*-metolachlor.

Study	Accession		LD_{50}[1]	R:S[2]
			g ai^{-1}	
Brabham et al. (2019)	SS	ARE-8	13	1
	SS	ARSE-1	20	1
	R[5]	CW	133	8.1
	R	MAR	156	9.5
Kouame et al. (2022)	SS	SS	63	1
	PR	18CRI-D	289	4.6
	PR	18WOO-B	443	7.0
	PR	17TUN-A	313.0	5.0
	PR	17TUN-D	242.0	3.8
	PR	18WOO-B-F1*	577	9.2
	PR	19WOO-B	520	8.3
Rangani et al. (2021)	SS	SS	27	1
	PR	14MIS-H	207	8
	PR	14CRI-G	418	15
	PR	14MIS-E	467	17
	PR	15CRI-A	593	22
	PR	16WOO-A	785	29
Strom et al. (2019)[6]	SS	ACR	53	1
	SS	WUS	101	1
	R	CHR-M6	1808	23.5
	R	MCR-NH40	3360	43.6

[1]LD_{50}, *S*-metolachlor rate that reduced seedling emergence by 50%
[2]R:S, resistance index, ratio between the LD_{50} value of the *S*-metolachlor-resistant accessions and the LD_{50} value of the susceptible accession
[3]SS, Susceptible standard
[4]PR, Putative resistant
[5]R, Claimed resistant
* progeny
[6] Waterhemp (*Amaranthus tuberculatus*)

Table 6: Herbicide Resistance in North Carolina

Year	Species	MOAs	WSSA	Actives	Situations
1973	*Eleusine indica*	Inhibition of Microtubule Assembly 2	3	trifluralin	Cotton
1980	*Chenopodium album*	PSII inhibitors - Serine 264 Binders	5	atrazine	Corn (maize)
1980	*Amaranthus hybridus (syn: quitensis)*	PSII inhibitors - Serine 264 Binders	5	atrazine	Corn (maize)
1990	*Lolium perenne ssp. multiflorum*	Inhibition of Acetyl CoA Carboxylase	1	diclofop-methyl, sethoxydim	Wheat
1994	*Xanthium strumarium*	Nucleic acid inhibitors	0	MSMA, DSMA	Cotton
1995	*Amaranthus palmeri*	Inhibition of Acetolactate Synthase	2	chlorimuron-ethyl	Soybean
1995	*Poa annua*	PSII inhibitors - Serine 264 Binders	5	simazine	Golf courses, Turf
1999	*Xanthium strumarium*	Inhibition of Acetolactate Synthase	2	imazapyr, pyrithiobac-sodium, chlorimuron-ethyl, primisulfuron-methyl, cloransulam-methyl	Soybean
2003	*Conyza canadensis*	Inhibition of Enolpyruvyl Shikimate Phosphate	9	glyphosate	Cotton

Table 6: (continued)

Year	Species	MOAs	WSSA	Actives	Situations
2005	*Amaranthus palmeri*	Inhibition of Enolpyruvyl Shikimate Phosphate	9	glyphosate	Corn (maize), Cotton, Soybean
2006	*Ambrosia artemisiifolia*	Inhibition of Enolpyruvyl Shikimate Phosphate	9	glyphosate	Cotton
2006	*Ambrosia artemisiifolia*	Inhibition of Acetolactate Synthase	2	diclosulam	Peanut (groundnut)
2007	*Lolium perenne ssp. multiflorum*	Inhibition of Acetolactate Synthase	2	mesosulfuron-methyl	Wheat
2007	*Lolium perenne ssp. multiflorum*	Inhibition of Acetolactate Synthase	2	diclofop-methyl, imazamox, mesosulfuron-methyl, pinoxaden, pyroxsulam	Wheat
2009	*Lolium perenne ssp. multiflorum*	Inhibition of Enolpyruvyl Shikimate Phosphate	9	glyphosate	Corn (maize), Cotton, Soybean
2015	*Ambrosia artemisiifolia*	Inhibition of Acetolactate Synthase Inhibition of Enolpyruvyl Shikimate Phosphate Inhibition of Protoporphyrinogen Oxidase	2 9 14	nicosulfuron, cloransulam-methyl, fomesafen, lactofen, acifluorfen, glyphosate	Corn (maize), Soybean
2015	*Eleusine indica*	Inhibition of Protoporphyrinogen Oxidase	14	oxadiazon	Golf courses

Table 6: (continued)

Year	Species	MOAs	WSSA	Actives	Situations
2016	*Amaranthus palmeri*	Inhibition of Hydroxyphenyl Pyruvate Dioxygenase	27	mesotrione	Corn (maize)
2020	*Amaranthus tuberculatus (=A. rudis)*	Inhibition of Acetolactate Synthase, Inhibition of Enolpyruvyl Shikimate Phosphate, Inhibition of Hydroxyphenyl Pyruvate Dioxygenase, Inhibition of Protoporphyrinogen Oxidase, PSII inhibitors - Serine 264 Binders	2 9 27 14 5	imazethapyr, glyphosate, mesotrione fomesafen, atrazine	Soybean
2020	*Amaranthus retroflexus*	Inhibition of Acetolactate Synthase Inhibition of Protoporphyrinogen Oxidase	2 14	imazamethabenz-methyl, thifensulfuron-methyl, fomesafen, lactofen	Soybean
2022	*Amaranthus palmeri*	PSII inhibitors - Serine 264 Binders	5	atrazine	Corn (maize)

CHAPTER 2: CHARACTERIZATION OF NORTH CAROLINA PALMER AMARANTH (*AMARANTHUS PALMERI* WATSON S.) POPULATIONS TO VLCFA-INHIBITING HERBICIDES

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ABSTRACT

Palmer amaranth is one of the most widespread, troublesome and economically damaging agronomic weeds in the southeastern U.S. In recent surveys, it has been reported as one of the top 5 most common and troublesome weeds. Its propensity to evolve resistance has further complicated management. Since the first report of herbicide resistant Palmer amaranth in the late 1980's, it has developed resistance to a total of 8 WSSA modes of action. The more recent reports of resistance to Group 15 herbicides, specifically *S*-metolachlor, in Arkansas and Mississippi have raised concerns regarding the future of Group 15 herbicides and their critical role in effective weed management. In North Carolina, Palmer amaranth continues to threaten cropping systems with increased reports of resistance to glyphosate, ALS, PPO and more recently HPPD. These increases in documented resistance further limits PRE and POST herbicide control options and increases reliance on other modes of action including Group 15/VLCFA-inhibiting herbicides, thus increasing the probability of selecting for additional resistance. Greenhouse studies were conducted to evaluate the differential response of two Palmer amaranth populations from Wake and Martin County, North Carolina, to five Group 15 herbicides, *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet. One "susceptible" population from Wake County and one "tolerant" population from Martin County, were evaluated. The results of the study indicated a significant differential response between the two populations to the Group 15 herbicides. R/S ratios were calculated, and the more tolerant Martin County population required 4.1 times more *S*-metolachlor, 5.1 times more acetochlor and 10.5 times more dimethenamid-P to reduce seedling emergence by 50% compared to the more susceptible Wake County population.

INTRODUCTION

Palmer amaranth (*Amaranthus palmeri S.*) has become one of the most widespread, troublesome and economically damaging agronomic weeds in the southeastern U.S (Ward et al. 2013; Chahal et al. 2015; Chandi et al. 2012; Korres et al. 2019; Tekiela and Sbatella 2017). Of the pigweed species, Palmer amaranth is arguably the most competitive and aggressive. It was found to have the highest photosynthetic rate among C_4 plants at 81µmol/m2/s (Ehleringer 1983) which is three to four times the rate of many row crops including C_4 corn and C_3 cotton and soybeans ultimately making it a fast growing and highly competitive weed (Gibson 1998; Steckel 2007). In the 2020 Weed Science Society of America (WSSA) survey, Palmer amaranth was reported as one of the top 5 most common and most troublesome weed species in corn, sorghum, grass crops, pasture and turf. In the 2019 survey it was surveyed as the number one most common and most troublesome weed amongst broadleaf crops, fruits and vegetables; specifically listed as the number one most troublesome and common weed in cotton, top 2 most troublesome and common in peanuts and top 7 most troublesome and common in soybean (Van Wychen 2019). Competition with Palmer amaranth is season long and has the ability to reduce yields as much as 91% in corn (Chahal et al. 2015; Bensch et al. 2003; Massinga et al. 2001) and 68% in soybean (Glycine max L. Merr.) (Klingaman and Oliver 1994). Its extended emergence pattern makes it difficult for preemergence and non-residual postemergence herbicides to control later-emerging plants. In addition to Palmer amaranth having a high relative growth rate, as much as 2.5 inches per day, it is a prolific seed producer with a single female plant being able to product >100,000 seeds when competing with crops and as many as 600,000 seeds per plant when growing in isolation (Fine 2020; Keeley et al. 1987).

As a dioecious species (separate male and female plants), there is potential for greater genetic diversity which helps to facilitate the spread of herbicide resistance (Steckel 2007, Tranel et al.

2011). Since the late 1980's, Palmer amaranth has evolved resistance to multiple modes of action including WSSA Groups 2, 3, 4, 5, 9, 14, 27 and more recently Group 15 (Heap 2019). Refer to Table 1 for an overview of the modes of action associated with herbicide resistant Palmer amaranth. In grower fields where glyphosate and ALS-resistant Palmer amaranth are prevalent, the use of residual herbicides at planting and with postemergence applications to overlap residuals are foundational steps for season-long control (Brabham et al. 2019; Culpepper et al. 2009; Norsworthy et al. 2012; Riar et al. 2013). Very-Long-Chain-Fatty-Acid (VLCFA)-inhibiting (Group 15) and PPO inhibiting herbicides (Group 14) with residual activity are commonly used PRE herbicides (Brabham et al. 2019). The evolution and consequent spread of resistance to PPO and VLCFA inhibiting herbicides is becoming increasingly evident with the first confirmation of resistance to a Group 15 herbicide being reported in 2019 (Brabham et al. 2019; Heap 2019).

WSSA Group 15 Herbicides or HRAC Group K3s are Long Chain Fatty Acid Inhibitors which are applied preemergence for residual control of annual grasses and some small-seeded broadleaf weeds in a variety of crops (Al-Khatib 2022). These herbicides are thought to inhibit very long chain fatty acid synthesis (Shaner et al. 2014). Fatty acids with a carbon chain length of greater than 18 (20 to 34 carbons) are referred to as VLCFA and are important building blocks for cuticular waxes and sphingolipids. Sphingolipids are required for proposer vesicle trafficking and membrane dynamics, especially in dividing cells (Bach et al. 2011; Jenks et al. 2002; Li-Beisson et al. 2010; Brabham et al. 2019). These VLCFA-inhibitors compounds interfere with the elongation of C18 fatty acid change (Böger 2003; Hamm 1974) and typically affect susceptible weeds prior to emergence but do not inhibit seed germination (Shaner et al. 2014). VLCFA-inhibitors can be grouped into three major chemical families; chloroacetamides

(Examples: *S*-metolachlor, acetochlor and dimethenamid-P), pyrazole (Example: pyroxasulfone) and oxyacetamids (Example: flufenacet).

In 2018, Brabham et al. conducted field and greenhouse studies to assess the susceptibility of Palmer amaranth populations in Arkansas to VLCFA-inhibitors. Both field and greenhouse studies confirmed a decrease in control across all tested VLCFA-inhibitors with confirmed resistance in two of the populations to *S*-metolachlor (Brabham et al. 2019). The LD_{50} values for the resistant populations were 156 and 133 g ha^{-1}, respectively. Based on these values, the two resistant populations were 9.8 and 8.3 times less responsive to *S*-metolachlor compared to the average LD_{50} value of the susceptible populations at 16 g ha-1. It was estimated that the two confirmed resistant populations required 2/3 and 1.5 times the 1X rate of *S*-metolachlor (1,064 g ha^{-1}) to obtain 90% mortality compared to the susceptible populations. For the other VLCFA-inhibitors, acetochlor, dimethenamid-P and pyroxasulfone, a 1.5 to 2.1 fold and 2.3 to 3.6 fold increase in herbicide rate was estimated to achieve 50% mortality for the same two populations (Brabham 2019).

Since the original confirmation of resistance to a Group 15 herbicide by Brabham et al. 2019, several investigations into Palmer amaranth populations have been made across the mid/south-West. Rangani et al. 2021 reported *S*-metolachlor resistance in five populations collected in 2014 and 2015 from Crittenden and Woodruff counties in Mississippi. 3 to 29-fold resistance levels were reported based on LD_{50} values of resistant populations ranging from 88 to 785 g ai ha^{-1}. In 2022, Roma-Burgos et al. reported six populations (four from Arkansas and two from Mississippi) as resistant to *S*-metolachlor. Researchers evaluated both parent and progeny populations with LD_{50} for the parent populations ranging between 73 and 443 g ai ha^{-1} and the F1 progeny LD_{50} rates ranging from 73 to 577 g ai ha^{-1}. The resistance level was reported as

generally greater among the progenies of surviving plants than among the resistant field populations. The resistant field populations required 2.2 to 7.0 times more *S*-metolachlor to reduce emergence 50% while the F1 of survivors needed up to 9.2 times more herbicide to reduce emergence 50% compared to the susceptible standard (Kouame 2022). Refer to Table 2 for a comparison of *S*-metolachlor LD_{50} values reported across studies.

In North Carolina, the average seed production from Palmer amaranth populations in competition with corn, cotton, peanut and soybean ranges from 51,000 to 273,000 seeds per plant (Mahoney 2020). Further, North Carolina Palmer amaranth populations continue to demonstrate a growing resistance to glyphosate and ALS inhibitors in key cropping systems (Mahoney 2020; Poirier et al. 2014). Previous work done by Poirier et. al (2014) and Mahoney (2020) specifically in the North Carolina Coastal Plain area, demonstrated resistance to glyphosate and ALS inhibitors has increased rapidly in just a 5-to-10-year period. In addition, the first documented incidence of resistance to a HPPD inhibitor in North Carolina was recorded in 2019 (Mahoney 2020). These increases in documented resistance in North Carolina Palmer amaranth populations further limits PRE and POST herbicide control options and increases reliance on other modes of action including, VLCFA-inhibiting herbicides, thus increasing the probability of selecting for additional resistance.

In 2021, additional screening studies were conducted by Moore et. al. (2021) to further evaluate the sensitivity of Palmer amaranth populations collected by Mahoney et al. (2020) to atrazine, dicamba, *S*-metolachlor and 2,4-D. Greenhouse studies were conducted with the seeds collected by Mahoney et al. in 2016. Of the collected populations, 18 survived both *S*-metolachlor and 2,4-D at field use rates in the greenhouse.

The following research was conducted to investigate the response of selected Palmer amaranth populations in the Moore et al. (2021) study to Very-Long-Chain-Fatty-Acids (VLCFA) herbicides applied preemergence (PRE).

MATERIALS AND METHODS

Greenhouse Dose Response Studies

Greenhouse experiments were conducted in the Spring/Summer of 2021 to determine the response of two North Carolina populations of Palmer amaranth to five VLCFA-inhibiting herbicides. Experiments were conducted at the greenhouse facility located on the Syngenta Crop Protection site in Greensboro, NC. The populations used in the study had been originally collected by Mahoney et al. (2020) in the fall of 2016 from soybean fields in Wake and Martin counties of the "Coastal Plain" area of North Carolina (Figure 1). The North Carolina Coastal Plain is considered to be the state's primary row crop producing region (USDA 2018, Mahoney et al. 2020). Moore et al. 2021 conducted additional screening of the Mahoney at al. 2020 populations to assess susceptibility to atrazine, S-metolachlor, dicamba and 2,4-D. The resulting profiles from Mahoney et al. 2020 and Moore et al. 2021 research are outlined in Table 3. The populations, referred to as WAKE and MARTIN for the remainder of this paper, were selected based on the results of the previous screening experiments. WAKE, which had demonstrated some level of resistance to ALS and Glyphosate had no survivors after VLCFA-inhibitor application, was anticipated to be the more "susceptible" population. MARTIN, which had a slightly higher level of resistance to ALS and Glyphosate relative to WAKE was chosen due to the presence of survivors after treatment with a VLCFA-inhibitor and was anticipated to be the more "tolerant" population. Both populations had germination rates >50%.

Dose-response experiments were conducted with the selected populations to determine sensitivity to five VLCFA-inhibitors; *S*-metolachlor (Dual Magnum; Syngenta, Greensboro, NC), acetochlor (Harness; Bayer Crop Science, St Louis, MO), dimethenamid-P (Outlook, BASF, Research Triangle Park, NC), pyroxasulfone (Zidua SC; BASF, Research Triangle Park, NC) and flufenacet (Sunfire; Certis Europe BV, Great Abington, Cambridgeshire). 1X rates were selected based on the currently labeled use rate for soybeans on medium soil type; 1422 g S-metolachlor/ha^{-1} equivalent to 1.33 pt/A of Dual Magnum, 2210 g acetochlor/ha^{-1} equivalent to 2.25 pt/A of Harness, 737 g dimethenamid-P/ha^{-1} equivalent to 15 fl oz of Outlook, 118.9 g pyroxasulfone/ha^{-1} equivalent to 3.25 fl oz of Zidua, and 240 g flufenacet/ha^{-1} equivalent to 0.48 L/ha of Sunfire. The range of herbicide dosages used in the study were 0, 1/64, 1/16, 1/4, 1X and 2X with the exception of flufenacet where the initial two runs were conducted with the aforementioned rate range but the third and fourth set of dose response studies were conducted using a 4X rate instead of the 1/64X rate to ensure 100% control was achieved and attempt to reduce the large amount of variability seen in response to flufenacet across rates in the first and second runs.

Studies were conducted utilizing a completely randomized design with four replications per treatment and two total runs, except for flufenacet, which had four total runs. A replication consisted of 4 in-by 4 in-by 4 in pots filled with Wakeland silt loam field soil, 2% OM, 5.7 pH, CEC 12-15. Prior to applications, pots were filled with soil, tampered and presoaked and then allowed to drain. After pot preparation was complete, an average of 20-30 seeds were scattered over the soil surface, covered with soil and lightly tampered. The pots were lightly watered just prior to applications. Treatments were applied using a research track sprayer equipped with 11015 VP nozzle (Teejet, Wheaton, IL) calibrated to deliver 140 L ha^{-1} at 4.8 km/h. Following

application, pots were watered overhead to activate the herbicides. Pots were watered overhead for the first 10 days of the experiment. After 10 days the pots were moved to trays and watered from below. The study was concluded 21 days after treatment (DAT).

Data Collection and Analysis

21 days after treatment (DAT), live counts and biomass (grams) were recorded. Live counts were also taken at 7 and 14 DAT. For each experimental run, the 21 DAT live counts were converted to percent survival based on the number of seedlings in the non-treated controls as described in Equation 1.

$$y = 100\text{-}((t/n)100)$$

Where y is percent control, t is number of weeds that emerged in treated soil and n is the average emergence of weeds in the respective non-treated soil. Weed control was analyzed with R to test for significant interactions (R Core Team 2020). There were no significant interactions between runs; therefore, data were pooled accordingly ($P>0.05$). There were significant effects of ($P< 0.0001$) rate, population, and herbicide on weed control with significant rate by herbicide interactions ($P<0.05$). See Table 4.

Regression analysis was done with the package drc and mselect in R (Ritz et al. 2015; R Core Team 2020). The model that was selected was based on the Akaike's information criterion (AIC) and p-value for the lack-of-fit test (Ritz 2010). Data for *S*-metolachlor, acetochlor and dimethenamid-P were fitted with a Weibull Type 1, 3-parameter model (Equation 2) while data for pyroxasulfone and flufenacet were fitted with a Weibull Type 2, 3-parameter model (Equation 3). Because previous dose response research had utilized the log-logistic 4 parameter model, i.e. Brabham et al. 2018 and Strom et al. 2019 (Equation 4), the data was also fitted using

this model for general comparison purposes. A summary of R parameter outputs and comparison across models can be found in the enclosed Appendix.

$$Y = c + (d - c)\,(exp\,(-exp\,(b(log\,(x) - log\,(e))))) \quad (2)$$

$$Y = c + (d - c)(1 - exp\,(-exp\,(b(log\,(x) - log\,(e))))) \quad (3)$$

$$Y = C + \frac{D-C}{1+exp\,\{b[log\,(x)\,]-log\,(e)\}} \quad (4)$$

In the equations above, Y is the survival percentage, d is the asymptote at the upper limit, c is the asymptote at the lower limit (fixed at 0 for Weibull models), x is the rate and b is the slope around e, which is the value of x that causes a 50% reduction of Y. (Ritz et al. 2015). The parameter, e, in all models, is the inflection point of the dose-response curve or the point where a change in acceleration in the curve occurs. For symmetric models (i.e. log-logistic four-parameter) this parameter also corresponds to the effective dose resulting in a 50% reduction in the response (LD_{50}). The inflection point will generally be larger than the median effective dose (ED_{50}) for the Weibull Type 1 and smaller for the Weibull Type 2. Estimated LD_{50} and LD_{90} values were calculated using the ED function in R. Parameter outputs can be found in Table 5.

RESULTS AND DISCUSSION

To quantify the susceptibility of the selected Palmer amaranth populations to each herbicide as well as quantify any potential differences between the Wake and Martin County populations, seeds from each population were used to generate dose-response curves in the greenhouse (Figures 2 & 3, Table 7).

Dose Response to S-metolachlor

At 21 DAT, the average survival of the WAKE population at 2X, 1X, 1/4X, 1/16X and 1/64X rates of *S*-metolachlor were 0%, 1%, 30%, 39% and 84%, respectively. For the MARTIN

population, average survival for the same consecutive rates were 2%, 13%, 52%, 71% and 93% (Figures 2A & 3A, Table 7).

At 21 DAT with *S*-metolachlor, the two populations, WAKE and MARTIN, had LD_{50} values of 81 and 336 g ha-1, and LD_{90} values of 698 g $ha^{-1}$and 1768 g ha^{-1} respectively. Based on these values, an estimated 1/16th of the 1X rate (1422 g ai/ha^{-1}) was required to obtain 50% mortality of the WAKE population and 1/4 of the 1X rate was estimated to obtain 50% mortality of the MARTIN population; approximately a 4X rate difference. 1/2 of the 1x (1422 g/ha) rate was the estimated rate required to obtain 90% mortality of the WAKE population and 1 1/4X the 1X rate was estimated to obtain 90% mortality of the MARTIN population; approximately a 2.5X rate difference.

Dose Response to Acetochlor

At 21 DAT the average survival of the WAKE population for the 2X, 1X, 1/4X, 1/16X and 1/64X rates of acetochlor were 0%, 0%, 0%, 14% and 38%, consecutively. For the MARTIN population, average survival for the same consecutive rates were 0%, 0%, 14%, 41% and 71% (Figures 2B & 3B, Table 7).

At 21 days after treatment (DAT) of acetochlor, the two populations, WAKE and MARTIN had LD_{50} values of 19 and 96 g ha^{-1}, and LD_{90} values of 178 g $ha^{-1}$and 660 g ha-1 respectively. Based on these values, an estimated 1/125 of the 1X (2210 g ai/ha^{-1}) rate was the estimated rate required to obtain 50% mortality of the WAKE population and 1/16 of the 1X rate was estimated to obtain 50% mortality of the MARTIN population; approximately a 5X rate difference. 1/16 of the 1x (2210 g ai/ha^{-1}) rate was the estimated rate required to obtain 90% mortality of the WAKE population and 1/2 the 1X rate was estimated to obtain 90% mortality of the MARTIN population; approximately a 4X rate difference. Compared to existing data, there was

approximately a 2X increase in LD_{50} value for acetochlor compared to the Brabham et al. resistant populations. See Table 2 for comparison of LD_{50} rates across studies. This is likely attributed to differences in experimental conditions including soil profile, environmental settings etc. as well as standard variability.

Dose Response to Dimethenamid-P

At 21 DAT the average survival of the WAKE population for the 2X, 1X, 1/4X, 1/16X and 1/64X rates of dimethenamid-P were 0%, 0%, 10%, 17% and 42%, consecutively. For the MARTIN population, average survival for the same consecutive rates were 0%, 4%, 27%, 57% and 71% (Figures 2C & 3C, Table 7).

At 21 days after treatment (DAT) of dimethenamid-P, the two populations, WAKE and MARTIN had LD_{50} values of 6 and 63 g ha^{-1}, and LD_{90} values of 120 g ha^{-1} and 506 g ha^{-1} respectively. Based on these values, an estimated 1/124 of the 1X (737 g ai/ha^{-1}) rate was the estimated rate required to obtain 50% mortality of the WAKE population 1/16 of the 1X rate was estimated to obtain 50% mortality of the MARTIN population; approximately an 11X rate difference. 1/4 of the 1x (737 g ai/ha^{-1}) rate was the estimated rate required to obtain 90% mortality of the WAKE population and 3/4X the 1X rate was estimated to obtain 90% mortality of the MARTIN population; approximately a 4 1/4X rate difference.

Dose Response to Pyroxasulfone

At 21 DAT the average survival of the WAKE population for the 2X, 1X, 1/4X, 1/16X and 1/64X rates of pyroxasulfone were 0%, 0%, 13%, 32%, and 57%, consecutively. For the MARTIN population, average survival for the same consecutive rates were 0%, 5%, 35%, 48%, and 95% (Figures 2D & 3D, Table 7).

At 21 days after treatment (DAT) of pyroxasulfone, the two populations, WAKE and MARTIN had LD_{50} values of 3 and 8 g ha^{-1}, and LD_{90} values of 40 g ha-1and 100 g ha-1 respectively. Based on these values, an estimated 1/45 of the 1X rate (119 g ai/ha^{-1}) was the estimated rate required to obtain 50% mortality of the WAKE population and 1/16 of the 1X rate was estimated to obtain 50% mortality of the MARTIN population; approximately a 3X rate difference. 1/4 of the 1x (119 g ai/ha^{-1}) rate was the estimated rate required to obtain 90% mortality of the WAKE population and the 3/4X rate was estimated to obtain 90% mortality of the MARTIN population; approximately a 2X rate difference.

Dose Response to Flufenacet

At 21 DAT the average survival of the WAKE population for the 4X, 2X, 1X, 1/4X, 1/16X and 1/64X rates of flufenacet were 0%, 16%, 52%, 76%, 72% and 71%, consecutively. For the MARTIN population, average survival for the same consecutive rates were 7%, 52%, 37%, 74%, 61% and 68% (Figures 2E & 3E, Table 7).

At 21 days after treatment (DAT) of flufenacet, the two populations, WAKE and MARTIN had LD_{50} values of 302 and 207 g ha^{-1}, and LD_{90} values of 633 g ha^{-1} and 312,517 g ha^{-1} respectively. Based on these values, an estimated 1 1/4X rate was the estimated rate required to obtain 50% mortality of the WAKE population and 3/4 the 1X rate (240 g ai/ha^{-1}) was estimated to obtain 50% mortality of the MARTIN population; approximately the 1X rate. 2 3/4X the 1x (240 g/ha^{-1}) rate was the estimated rate required to obtain 90% mortality of the WAKE population; 1300X the 1X rate was estimated to obtain 90% mortality of the MARTIN population; approximately a 494X rate difference. There was no existing dose response work for flufenacet available for comparison at the time of this publication. This is very likely due to the significant amount of variability seen in the dose response of Palmer amaranth to all rates of flufenacet.

Comparison Across Herbicides At 21 days after treatment (DAT), all herbicides, with the exception of flufenacet, required less than the 1X rate to obtain 50% mortality in both populations. Overall, the average control for all herbicides across both populations decreased as the rate decreased, with the exception of flufenacet, which had variable control at all rates. For flufenacet, a 4X rate was required in order to obtain 100% control and the lower rates (1/64, 1/16 and 1/4) provided roughly the same level of control (average of 30%) across both populations. In general, flufenacet had consistent variability in control across all rates. Overall, it was difficult to achieve consistent results with flufenacet even with increased replications. Flufenacet, as a standalone product, is not currently sold in the United States and the results of this study may provide some insight as to why.

There was a significant reduction in the average control of MARTIN compared to WAKE across the lower rates (1/64, 1/16 and 1/4) for all herbicides except for flufenacet. For example, dimethenamid-P and acetochlor demonstrated >50% control of the WAKE population at the lowest (1/64) rate compared to the MARTIN population where control dropped to approximately 30% for both herbicides at the same rate. Pyroxasulfone demonstrated 43% control of the WAKE population at the lowest rate and only 5% control of the MARTIN population at the same rate. *S*-metolachlor had <20% control of both the WAKE and MARTIN populations at the lowest rate.

Acetochlor, dimethenamid-P and pyroxasulfone demonstrated a similar level of control across both populations for each rate. *S*-metolachlor had a slightly reduced level of control across populations and rates, comparatively. For example, 1/4 of the labeled rate of acetochlor demonstrated 100% control (no survival) of the WAKE population. The 1/4 rate of acetochlor demonstrated 86% control of the MARTIN population with 100% control demonstrated at the

1X and 2X rates. Pyroxasulfone and dimethenamid-P both demonstrated 100% control of the WAKE population at 1X and 2X rates. Both pyroxasulfone and dimethenamid-P demonstrated >94% control of the MARTIN population at the 1X rate and 100% control at the 2X rate. *S*-metolachlor however demonstrated survival (1%) of WAKE population at the 1X rate and 13% and 2 % survival of the MARTIN population at the 1X and 2X rates, respectively.

Flufenacet and *S*-metolachlor required more than the 1X rate to obtain 90% mortality in both populations. For flufenacet, the necessity for a higher than labeled rate to offer >90% control is not surprising for the reasons outlined above including variability in control. For *S*-metolachlor, the higher than labeled rate required to provide >90% control is potentially of concern due to the recent reports of *S*-metolachlor resistant Palmer amaranth across the country. It is possible that the reduction in control of *S*-metolachlor compared to the other Group 15 herbicides may be a result of reduced sensitivity of the subject populations to *S*-metolachlor. Alternatively, it may be a result of natural tolerance. While a reduction in sensitivity could be an indication of developing resistance additional research with multiple susceptible populations may be needed to determine if the potential reduced sensitivity of these populations to *S*-metolachlor is associated with evolved resistance.

Generally speaking, *S*-metolachlor demonstrated adequate control, however, when compared to other Group 15 herbicides, the differences in overall herbicide control is more evident. This raises potential concern for Palmer amaranth surviving higher than labeled rates and potential evolution of, or presence of, resistance. This is particularly true for *S*-metolachlor which has had resistance confirmed in multiple populations of Palmer amaranth over the last 4 years. While *S*-metolachlor demonstrated overall less control of the subject Palmer amaranth populations, all of the herbicides (with the exception of flufenacet), demonstrated an increase in the rate required to

reduce emergence of the MARTIN population by 50% compared to the WAKE population. In other words, the "resistance ratios" (R/S ratio) demonstrated a potential shift in the MARTIN population toward Group 15 herbicide resistance.

R/S Ratios

There has been a recent increase in research specific to *S*-metolachlor resistance in Palmer amaranth due to the first case of resistance being confirmed by Brabham et al. in 2019. Prior to this, variation and differential tolerance to herbicides amongst Palmer amaranth populations was expected and understood due to naturally occurring genetic background variation (Radosevich et al. 2007). Differential response to labeled rates of various herbicides is well documented and studied amongst older populations of Palmer amaranth in the Midwest (Salas et al. 2016, Salas-Perez et al. 2018, Singh et al. 2018; Bond et al. 2006; Burgos et al. 2001; Kumar et al. 2020). The more recent research published by Brabham et al. 2019, Rangani et al. 2021 and Kouame et al. 2022 has started investigating these differential responses and the potential for this to understand evolving resistance. The initial investigation into *S*-metolachlor resistance specifically, was due to a number of Palmer amaranth populations showing less than 90% control with the labeled rate of *S*-metolachlor (Brabham et al. 2019).

Herbicide resistance is defined by WSSA as "the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type. In a plant, resistance may be naturally occurring or induced by such techniques as genetic engineering or selection of variants produced by tissue culture or mutagenesis" (WSSA 1998). This definition of resistance is consistent with a prior description (Powles et al. 1997) where herbicide resistance was defined as the inherited ability of a weed population to survive a herbicide application that is normally lethal to the vast majority of individuals of that species (Nandula et al. 2010). Some

have defined a weed population as “resistant” when a herbicide that once controlled the population is no longer effective. Sometimes an arbitrary figure of 20% survival is used (Peltzer 2021). This may lead to the question can resistance be confirmed based on a population's response to the labeled rate of a herbicide or the LD_{50} rate determined by dose response work. Literature indicates that observation of reduced control in the field is typically the starting point for investigation into a potentially resistant weed population. The evolution of R/S ratios as a tool for resistance confirmation has taken root in existing dose response literature. It was the primary method for confirming glyphosate resistance back in the 1990’s and even more recently used to confirm Group 15 herbicide resistance in Palmer amaranth populations (Powles 1998; Bradshaw et al. 1997; Pratley et al. 1999; Brabham et al 2019; Strom et al. 2019; Rangani et al. 2021; Kouame et al. 2022; Hager 2013; Hager 2019).

Brabham et al. 2019 LD_{50} and LD_{90} rates were reported based on greenhouse dose response studies designed to confirm resistance in populations in Crittenden County, Arkansas which had demonstrated reduced control in the field. Two susceptible populations were also analyzed. The two confirmed resistant populations were characterized as 9.8 and 8.3 times less responsive to *S*-metolachlor in comparison to the average LD_{50} value of the susceptible populations at 16 g ai/ha^{-1}. The 1X rate used for each herbicide in the current study varied slightly compared to previous work conducted by Brabham et al. in 2019, however, all rates used in the current study were captured in the rate range utilized in Brabham et al. Overall, the susceptible populations in both studies performed similarly in terms of rate response. Slightly higher rates were required to obtain 50% control in the WAKE population compared to the susceptible populations in the Brabbham et al. 2019 study, however this is likely attributed to differences in soil profile, environmental settings and standard variability. The more difficult to control population,

MARTIN, required higher rates to obtain 50% control as compared to the populations identified as “resistant” in the Brabham et al. study. In the case of *S*-metolachlor approximately 2X the average LD_{50} rate for the Brabham et al. (2019) resistant populations was required to obtain 50% control in the MARTIN population. Again, this may be attributed to differences in experimental conditions.

Kouame et al. (2022) assessed populations across 3 states including Arkansas, Mississippi and Tennessee compared to one susceptible population. Six populations were classified as resistant to *S*-metolachlor with R/S ratios ranging from 5.0 to 9.2. Rangani et al. 2021 investigated *S*-metolachlor resistant populations in Arkansas and reported resistance levels ranging 3 to 29-fold with LD_{50} values of resistance populations ranging between 88 and 785 g ai $ha^{-1.}$ This method has been applied to other species of Amaranthus including waterhemp (*Amaranthus tuberculatus*). Strom et al. 2019 investigated *Amaranthus tuberculatus* populations in Champaign County, Illinois, reporting R/S values that ranged from 7.5 to 63.8 when compared to the susceptible populations.

The current study reports a 4-fold (336.4 g ha^{-1}:81.4 g ha^{-1}) difference in the *S*-metolachlor LD_{50} values for the MARTIN population as compared to the susceptible WAKE population; a 5-fold (95.7 g ha^{-1}: 18.7 g ai ha^{-1}) difference between the LD_{50} values for acetochlor; an 11-fold (62.9 g ai ha^{-1}: 5.9 g ai ha^{-1}) difference in LD_{50} values for dimethenamid-P, and a 2.5-fold (8.2 g ha^{-1}: 2.6 g ha^{-1}) difference in LD_{50} values for pyroxasulfone. Comparatively, Brabham et al. 2019 reported R/S values ranging from 2.3 to 3.6 based on an average LD_{50} value of 13 g acetochlor/ha^{-1} for the susceptible population. The estimated 11-fold (62.9 g ai ha^{-1}: 5.9 g ai ha^{-1}) difference in LD_{50} values for dimethenamid-P in this study is markedly higher than what was reported in Brabham et al. 2019 where dimethenamid-P was reported to have R/S values ranging from 1.5 to 2.1 based

on an average LD_{50} value of 12 g ai/$ha^{-1}$for the susceptible population. For pyroxasulfone, Brabham et al. 2019 reported R/S values ranging from 2.0-2.8 based on an average LD_{50} value of 3 g ai/ha^{-1} for the susceptible population. See Table 6 for a comparison of R/S ratios, LD_{50} and LD_{90} rates across studies.

While the more recent research has focused predominantly on *S*-metolachlor and evolving resistance, data suggests that cross-resistance to other VLCFA-inhibitors is not far behind. Both Brabham et al. 2019 and Strom et al. 2019 report an increase in herbicide rate was required for pyroxasulfone, dimethenamid-P and acetochlor in order to achieve 50% mortality in the resistant populations. The reported difference in LD_{50} estimates in the current study for *S*-metolachlor, acetochlor and dimethenamid-P indicate a clear differential response between populations and potential resistance according to the current method of resistance confirmation. Additional screening and dose response work with additional susceptible populations may be necessary to confirm the putative resistance reported in this study.

CONCLUSIONS

In conclusion, the differential responses in the subject Palmer amaranth populations to multiple Group 15 herbicides indicate the potential presence of segregating populations in the Coastal Plain of North Carolina that could be a future concern. Follow-up screening and dose response work with additional susceptible populations may be necessary to confirm the putative resistance reported in this study. Finally, while residual herbicides such as Group 15s are important for season long control, weed management programs that utilize PRE and POST herbicide applications with overlapping/multiple modes of action continue to be critical in the overall objective to manage and prevent weed resistance.

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TABLES AND FIGURES

Table 1: List of Mode of Actions associated with Herbicide-resistant Palmer amaranth (*Amaranthus palmeri*)

WSSA Group (HRAC)	Site of Action	First Year	Crop/Country
2 (B) *Ex. imazethapyr*	Inhibition of Acetolactate Synthase (ALS)	1993	Alfalfa, Corn (maize), Sorghum, and Soybean/ United States (Kansas)
3 (K1) *Ex. trifluralin*	Inhibition of Microtubule Assembly	1989	Cotton, and Soybean/ United States (South Carolina)
4 (O) *Ex. 2,4-D*	Auxin Mimics	2015	Sorghum/United States (Kansas)
5 (C1/2) *Ex. Atrazine*	PSII – Serine 264 Binders	1993	Corn and Sorghum/ United States (Texas)
9 (G) *Ex. glyphosate*	Inhibition of Enolpyruvyl Shikimate Phosphate Synthase (EPSPS)	1995	Cotton and Soybean/ United States (Georgia)
14 (E) *Ex. fomesafen*	Inhibition of Protoporphyrinogen Oxidase (PPO)	2011	Soybean/ United States (Arkansas)
15 (K3 N) *Ex. S-metolachlor*	Very Long-Chain Fatty Acid Synthesis inhibitors (VLCFA)	2016	Soybean/ United States (Arkansas)
27 (F2) *Ex. mesotrione*	Inhibition of Hydroxyphenyl Pyruvate Dioxygenase (HPPD)	2016	Corn/ United States (North Carolina)

Table 2: Comparison of reported LD50 and R/S ratios of Palmer amaranth (*Amaranthus palmeri)* and Waterhemp (*Amaranthus tuberculatus)* populations to *S*-metolachlor

Study	**Population**		**LD_{50}[1]**	**R/S[2]**
			g ai^{-1}	
Foderaro et al. (2022)	SS[3]	WAKE	81	1
	PR[4]	MARTIN	336	4.1
Brabham et al. (2019)	SS	ARE-8	13	1
	SS	ARSE-1	20	1
	R[5]	CW	133	8.1
	R	MAR	156	9.5
Kouame et al. (2022)	SS	SS	63	1
	PR	18CRI-D	289	4.6
	PR	18WOO-B	443	7.0
	PR	17TUN-A	313.0	5.0
	PR	17TUN-D	242.0	3.8
	PR	18WOO-B-F1*	577	9.2
	PR	19WOO-B	520	8.3
Rangani et al. (2021)	SS	SS	27	1
	PR	14MIS-H	207	8
	PR	14CRI-G	418	15
	PR	14MIS-E	467	17
	PR	15CRI-A	593	22
	PR	16WOO-A	785	29
Strom et al. (2019)[6]	SS	ACR	53	1
	SS	WUS	101	1
	R	CHR-M6	1808	23.5
	R	MCR-NH40	3360	43.6

[1]LD_{50}, *S*-metolachlor rate that reduced seedling emergence by 50%
[2]R/S, resistance index, ratio between the LD_{50} value of the *S*-metolachlor-resistant populations and the LD_{50} value of the susceptible population
[3]SS, Susceptible standard
[4]PR, Putative resistant
[5]R, Claimed resistant
* progeny
[6] Waterhemp (*Amaranthus tuberculatus*)

Table 3: Herbicide Resistance Profiles of Selected Populations, WAKE and MARTIN

Population Name	County	Cropping System	Survival [1]					
			Thifensulfuron	Glyphosate	Fomesafen	Mesotrione	Glufosinate	*S*-metolachlor[2]
WAKE	Wake	Soybean	27%	46%	0%	0%	0%	0%
MARTIN	Martin	Soybean	71%	70%	0%	2%	0%	10-15%

[1] < 90% injury; plants with functional green leaves and apical meristems as reported by Mahoney et al. (2020)
[2] Survival at 800 g/ha rate of *S*-metolachlor as reported by Moore et al. (2021)

Table 4: The F and p-values for Palmer amaranth (*Amaranthus palmeri)* survival (%) 21 days after herbicide application in a greenhouse experiment in Greensboro, NC, USA

	21 DAT	
Effect	F Value	P > F
Run	1.45	0.23
Rate	55.46	<0.0001
Run*Rate	2.93	0.021
Population	30.12	<0.0001
Herbicide	27.16	<0.0001
Rate*Herbicide	2.42	0.0019
Run*Rate*Herbicide	2.49	0.0014

Table 5: Dose-response parameter estimates, LD_{50} and LD_{90} values of WAKE and MARTIN populations to *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet. A Weibull Type 1 three-parameter and Weibull Type 2 three-parameter function were used to generate dose-response curves.[a]

Herbicide	Population	Dose-response parameters estimates						
		b	***d***	***e***	***LD_{50}***	***$\pm SE$***	***LD_{90}***	***$\pm SE$***
S-metolachlor	WAKE	0.559	106.35	156.86	81.41	59.32	697.63	479.49
	MARTIN	0.723	99.78	558.30	336.39	219.72	1768.31	893.81
acetochlor	WAKE	0.532	99.86	37.14	18.65	11.99	178.09	94.08
	MARTIN	0.622	101.02	172.56	95.68	41.49	660.25	271.12
dimethenamid-P	WAKE	0.399	100.43	14.88	5.94	5.87	120.20	100.55
	MARTIN	0.576	96.52	118.92	62.93	41.04	505.97	249.34
pyroxasulfone	WAKE	-0.6914	99.20	1.55	2.63	1.44	40.17	33.44
	MARTIN	-0.7552	104.42	5.06	8.23	3.62	99.66	63.25
flufenacet	WAKE	-2.5426	77.80	261.23	301.74	502.35	632.99	286.35
	MARTIN	-0.2573	92.01	49.76	206.75	503.52	312516.7	134560

[a] Weibull Type 1 three-parameter, $Y = d\,(exp\,(-exp\,(b(log\,(x) - log\,(e)))$, was used to fit *S*-metolachlor, acetochlor and dimethenamid-P data. Weibull Type 2 three-parameter, $= c + (d - c)(1 - exp\,(-exp\,(b(log\,(x) - log\,(e))))$, was used to fit pyroxasulfone and flufenacet data. These models were selected was based on the Akaike's information criterion (AIC) and p-value for the lack-of-fit test (Ritz, 2010).

Table 6: Comparison of current study LD_{50} and LD_{90} rate estimates compared to estimates from Brabham et al. 2019 greenhouse dose response studies

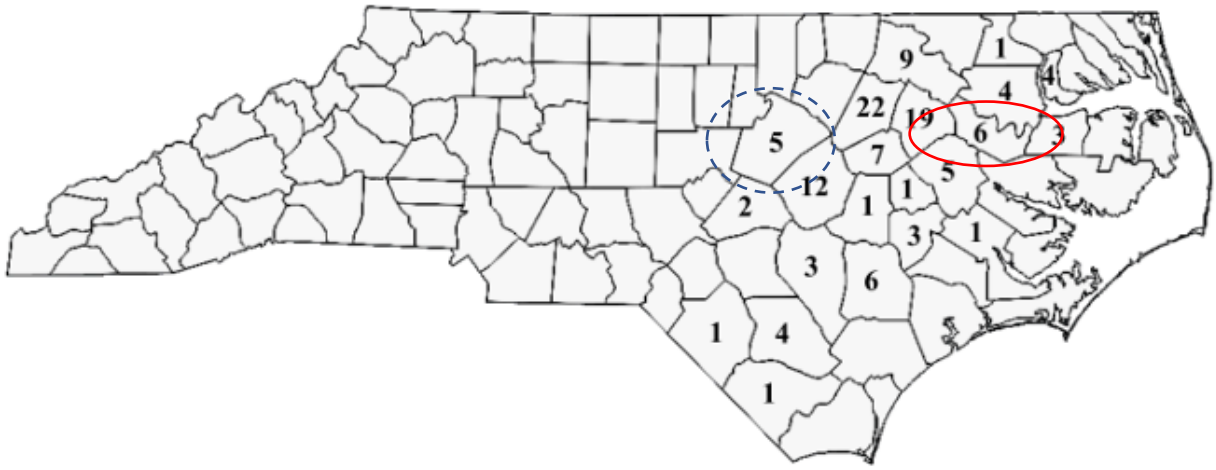
				Brabham et al. (2019)					
	WAKE	**MARTIN**		**ARE-8**	**ARSE-1**	**CW**		**MAR**	
				g ai/ha^{-1}					
***S*-metolachlor**			**R/S Ratio***				**R/S Ratio***		**R/S Ratio***
LD_{50}	81	336	**4.1**	13	20	133	**8.1**	156	**9.5**
LD_{90}	336	1768		112	267	704		704	
acetochlor									
LD_{50}	19	96	**5.1**	10	15	27	**2.2**	45	**3.6**
LD_{90}	178	660		26	60	110		245	
dimethenamid-P									
LD_{50}	6	63	**10.5**	10	13	17	**1.5**	26	**2.3**
LD_{90}	120	506		47	73	53		258	
pyroxasulfone									
LD_{50}	3	8	**2.7**	2	3	5	**2**	7	**2.8**
LD_{90}	40	100		8	13	17		30	
flufenacet				-	-	-	-	-	-
LD_{50}	302	207	**0.7**			-	-	-	-
LD_{90}	633	312517		-	-	-	-	-	-

ARE-8 and ARSE-1: Susceptible controls from Brabham et al. (2019)
CW and MAR: Confirmed resistant populations from Brabham et al. 2019
*R/S Ratio = LD_{50} of susceptible population / LD_{50} of "resistant" population; LD_{50} of susceptible is the average in the case multiple susceptible populations

Table 7: Group 15 Herbicide Dose Response: Average % Survival and Standard Error for *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet on WAKE and MARTIN Palmer amaranth populations

Rate	*S*-metolachlor		acetochlor		dimethenamid		pyroxasulfone		flufenacet	
	% Survival	SE	% Survival	SE	% Survival	SE	% Survival	SE	% Survival	SE
					WAKE					
0	100	0	100	0	100	0	100	0	100	0
0.0156X	84	28.49	38	8.20	42	15.03	57	18.97	71	0.00
0.0625X	39	12.43	14	7.65	17	8.15	32	14.68	72	8.67
0.25X	30	4.96	0	0	10	5.61	13	4.55	76	19.24
1X	1	1.09	0	0	0	0	0	0	52	24.41
2X	0	0	0	0	0	0	0	0	16	10.43
4X	-	-	-	-	-	-	-	-	0	5.42
					MARTIN					
0	100	0	100	0	100	0	100	0	100	0
0.0156X	93	19.61	71	19.25	71	17.63	95	11.09	68	10.57
0.0625X	71	13.72	41	7.55	57	13.57	48	15.29	61	7.50
0.25X	52	9.50	14	5.41	27	8.09	35	7.43	74	9.71
1X	13	4.55	0	0	4	2.82	5	2.24	37	8.55
2X	2	2.08	0	0	0	0	0	0	52	13.16
4X	-	-	-	-	-	-	-	-	7	2.55

Figure 1: North Carolina Counties: Palmer amaranth population collections from Mahoney et al. 2020 and further assessed by Moore et al. 2021

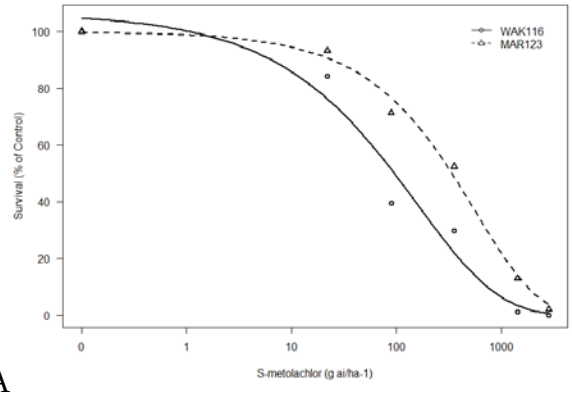


Seed from 120 Palmer amaranth populations were collected from 22 North Carolina counties: 12 from cotton (*Gossypium hirsutum* L.), 31 from peanut (*Arachis hypogaea* L.), 56 from soybean [*Glycine max* (L.) Merr.], and 21 from sweetpotato [*Ipomoea batatas* (L.) Lam.]. Numbers represent the number of populations collected from each county.
The populations used in this research were from Wake and Martin Counties

Wake County

Martin County

Figure 2: The percent survival of WAKE and MARTIN Palmer amaranth populations to increasing rates of (A) *S*-metolachlor, (B) acetochlor (C) dimethenamid-P (D) flufenacet and (E) pyroxasulfone. The regression parameters for each herbicide by population and the rate that causes 50% and 90% mortality/reduction in seedling emergence (LD_{50} and LD_{90}) in each population and herbicide are listed in Table 5.



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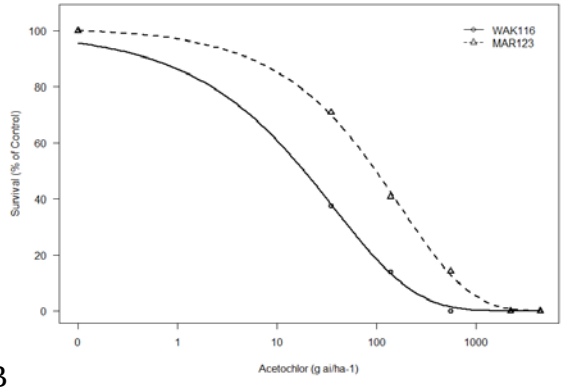

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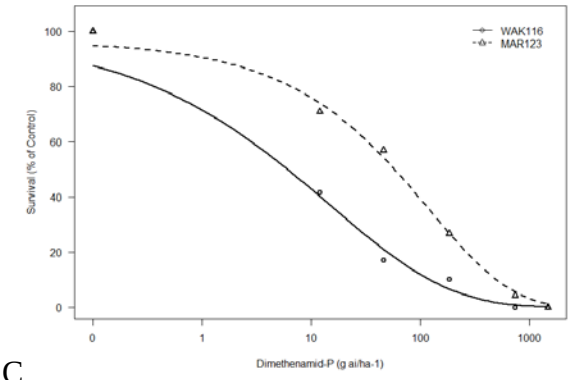

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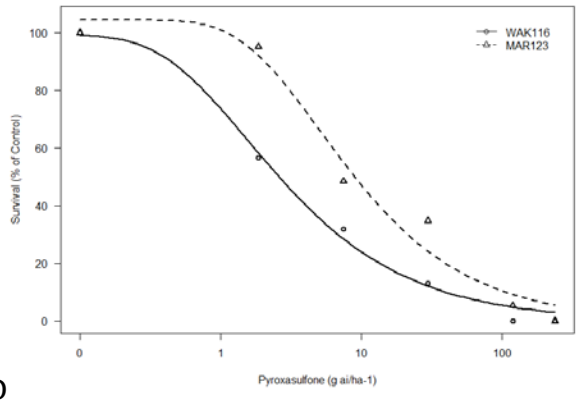

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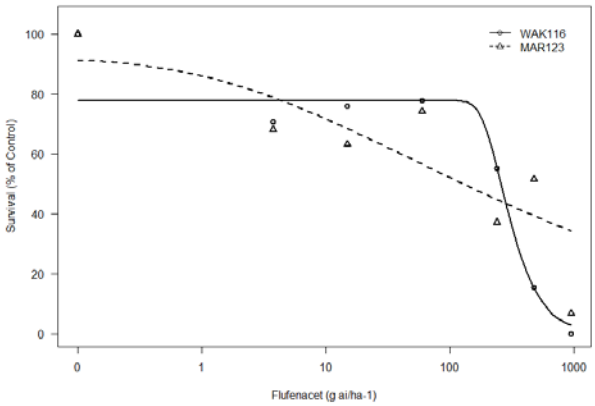

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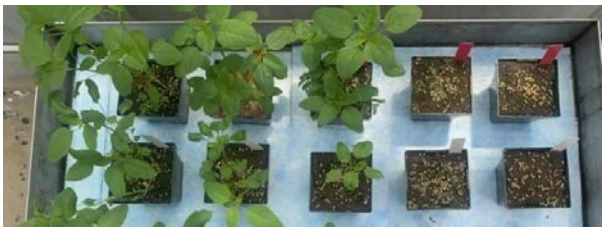
Figure 3: Response of WAKE and MARTIN Palmer amaranth populations to increasing rates of (A) *S*-metolachlor, (B) acetochlor (C) dimethenamid-P (D) flufenacet and (E) pyroxasulfone. MARTIN population on top (red tags), WAKE population on bottom (white tags)

1/64X 1/16X 1/4X 1X 2X

A 

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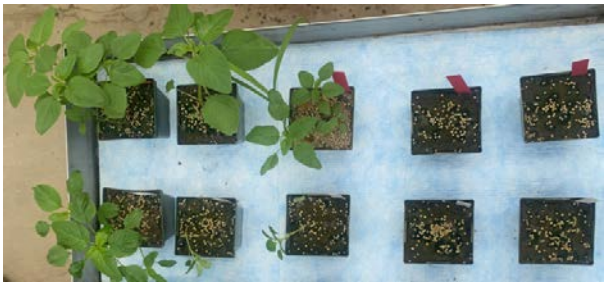
C 

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CHAPTER 3: RESIDUAL EFFICACY OF VLCFA-INHIBITING HERBICIDES IN CONTROL OF TWO NORTH CAROLINA PALMER AMARANTH (*AMARANTHUS PALMERI* WATSON S.) POPULATIONS

Amanda M. Foderaro, Charles W. Cahoon, Katherine M. Jennings, Wesley J. Everman

ABSTRACT

Palmer amaranth is well known now as one of the most widespread, troublesome and economically damaging agronomic weeds in the southeastern U.S. In recent surveys, it has been reported as one of the top 5 most common and troublesome weeds in corn, soybean, cotton and other cropping systems. Its propensity to evolve resistance has only further increased the difficulty of its management. The evolution and consequent spread of resistance to VLCFA inhibiting herbicides is increasingly more of a concern since the first confirmation of Palmer amaranth resistance to a Group 15 herbicide reported in 2018. Competition with Palmer amaranth is season long and the use of residual herbicides at planting and with postemergence applications to overlap residuals are foundational steps for season-long control. Residual, preemergence herbicides applied directly to the soil, such as VLCFA-inhibitors, need to persist long enough in the soil to provide adequate weed control throughout the duration of the critical weed control period. Understanding what residual herbicides are available and how they can be utilized successfully to ensure season long control is important when planning an effective weed management program. Various factors impact how long an herbicide might persist in the soil (i.e. soil type, microbial activity, precipitation etc.) and there is extensive literature assessing the DT_{50}/half-life of Group 15 herbicides under various environmental conditions. Greenhouse studies were conducted to evaluate the residual efficacy of five Group 15 herbicides in control of Palmer amaranth populations from North Carolina. The Group 15 herbicides evaluated were, *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet. The average control demonstrated by the LD_{90} rate (from previous dose response research) was evaluated for each herbicide on each population at 0, 2, 4 and 6 weeks following application. One “susceptible” population from Wake County and one “tolerant” population from Martin County, were evaluated. The results of the study indicated a significant difference in the overall control of each

population. Results also indicated a significant difference between herbicides in the average control of both Palmer amaranth populations over time with pyroxasulfone and *S*-metolachlor averaging higher levels of control over time compared to acetochlor, dimethenamid-P and flufenacet. The estimated I_{50} (weeks until a 50% reduction in control) for *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet was 6.7, 1.2, 1.42, 7 and 2.19 weeks, respectively.

INTRODUCTION

Palmer Amaranth: Palmer amaranth is well known now as one of the most widespread, troublesome and economically damaging agronomic weeds in the southeastern U.S (Ward et al. 2013; Chahal et al. 2015; Chandi et al. 2012; Korres et al. 2019; Tekiela and Sbatella 2017). In the 2020 Weed Science Society of America (WSSA) survey, Palmer amaranth was reported as one of the top 5 most common and most troublesome weed species in corn, sorghum, grass crops, pasture and turf. (Van Wychen, 2020). Competition with Palmer amaranth is season long and has the ability to reduce yields as much as 91% in corn (Chahal et al. 2015; Bensch et al. 2003; Massinga et al. 2001) and 68% in soybean (*Glycine max* L. Merr.) (Klingaman and Oliver 1994). Its extended emergence pattern makes it difficult for preemergence and non-residual postemergence herbicides to control later-emerging plants.

Since the first case of herbicide resistant Palmer amaranth (*Amaranthus palmeri S.*) was confirmed in 1989, the evolution and continued development of resistant Palmer amaranth has become an issue for growers across the world. Since the late 1980's, Palmer amaranth has evolved resistance to multiple modes of action including Weed Science Society of America (WSSA) Groups 2, 3, 4, 5, 9, 14, 27 and more recently Group 15 (Heap 2019) (Table 1). The evolution of herbicide resistant Palmer amaranth, specifically to glyphosate can be attributed to rapid adoption of glyphosate-resistant crops between 1996 and 2006, resulting in an increase in glyphosate reliance and subsequent shift in herbicide use patterns from PRE followed by POST herbicides with multiple modes of action (MOA) to POST only applications of glyphosate (Duke 2015, Givens et al. 2009; Powles 2008). Increased reliance on glyphosate subsequently led to a drastic reduction in herbicide diversity and induction of weed species shifts thus accelerating the evolutionary rate of glyphosate resistant weeds, including Palmer amaranth (Culpepper 2006;

Green 2009; Johnson et al. 2009; Kniss 2018; Owen 2008; Owen and Zelaya 2005; Webster and Nichols 2012). With the increase of glyphosate resistant weeds throughout the United States, the use of additional herbicide modes of action has become necessary for effective chemical weed management (Hager et al. 2003; Prince et al. 2012; Riggins and Tranel 2012; Werle et al. 2018). As a result, use of PRE herbicide, such as Group 15 herbicides, has generally increased. The soybean production area treated with PRE herbicides substantially increased from 2006 through 2017, particularly with *S*-metolachlor (15%) (USDA 2020), indicating higher herbicide mode of action diversity for weed control in soybean cropping systems (Kniss 2018). In 2018, *S*-metolachlor was applied to 28.3 million acres of corn (USDA NASS). In 2020 Group 15 herbicides accounted for 20.1 million pounds of the active ingredients used on soybean acres (USDA 2020). Group 15 herbicides have also become a common component in PRE tank mixes combined with POST herbicides including Group 5, Group 27, Group 14 and Group 9 herbicides.

Palmer Amaranth in North Carolina: In North Carolina, the average seed production from North Carolina Palmer amaranth populations in competition with corn, cotton, peanut and soybean ranges from 51,000 to 273,000 seeds per plant (Mahoney 2020). Further, North Carolina Palmer amaranth populations continue to demonstrate a growing resistance to glyphosate and ALS inhibitors in key cropping systems (Mahoney 2020; Poirier et al. 2014). Previous work done by Poirier et. al (2014) and Mahoney (2020) specifically in the North Carolina Coastal Plain area, demonstrated that resistance to glyphosate and ALS inhibitors has increased rapidly in just a 5-to-10-year period. Palmer amaranth populations collected across North Carolina in 2005 indicated 17 and 18% of the populations survived field use rates of glyphosate (0.75 lb a.e. acre–1) and thifensulfuron-methyl (0.016 lb a.i. acre–1), respectively. Poirier et al. (2014)

reported at least 97% survival when these herbicides were applied to 134 populations collected across North Carolina in 2010 with 93% of populations surviving both herbicides. More recently, Mahoney et al. (2020) surveyed 110 populations collected from the North Carolina Coastal Plain in 2016 and reported that at least 96% of the populations survived glyphosate (0.75 lb acre–1) and thifensulfuron-methyl (0.016 lb acre–1). Fomesafen (0.25 lb acre–1) and mesotrione (0.1 lb a.i. acre–1) controlled these populations 98% and 90%, respectively, while no populations survived glufosinate (0.4 lb acre–1). Since Mahoney et al. (2020) collected the Palmer amaranth populations in 2016, Palmer amaranth resistance to 2,4-D, dicamba, and *S*-metolachlor has been reported in the U.S. (Heap, 2021). In addition, the first documented incidence of resistance to a HPPD inhibitor in North Carolina was recorded in 2019 (Mahoney 2020) and the opportunity for intensification is expected due to the anticipated increase in the use of mesotrione and other HPPDs on tolerant cotton and soybean (Mahoney 2020). These increases in documented resistance in North Carolina Palmer amaranth populations further limits PRE and POST herbicide control options and increases reliance on other modes of action including, Group 15 herbicides, thus increasing the probability of selecting for additional resistance.

Group 15 Herbicides/Very-Long-Chain-Fatty-Acid Inhibitors: WSSA Group 15 Herbicides or HRAC Group K3s are Very Long Chain Fatty Acid Inhibitors (VLCFA) which are applied preemergence for residual control of annual grasses and some small-seeded broadleaf weeds in a variety of crops (Shaner et al. 2014; Al-Khatib 2022). The use of residual herbicides at planting and with postemergence applications to overlap residuals are foundational steps for season-long control (Brabham et al. 2019; Culpepper et al. 2009; Riar et al. 2013). The evolution and consequent spread of resistance to VLCFA inhibiting herbicides is becoming increasingly more

of a concern since the first confirmation of Palmer amaranth resistance to a Group 15 herbicide reported in 2019 (Brabham et al. 2019; Heap 2019).

Group 15 herbicides are thought to inhibit very long chain fatty acid synthesis (Shaner et al. 2014). Fatty acids with a carbon chain length of greater than 18 (20 to 34 carbons) are referred to as VLCFA and are important building blocks for cuticular waxes and sphingolipids. Sphingolipids are required for proposer vesicle trafficking and membrane dynamics, especially in dividing cells (Bach et al. 2011; Jenks et al. 2002; Li-Beisson et al. 2010; Brabham et al. 2019). These VLCFA-inhibitors compounds interfere with the elongation of C18 fatty acid change (Böger 2003; Hamm 1974). They typically affect susceptible weeds prior to emergence but do not inhibit seed germination (Shaner et al. 2014). VLCFA-inhibitors can be grouped into three major chemical families; chloroacetamides (Examples: *S*-metolachlor, acetochlor and dimethenamid-P), pyrazole (Example: pyroxasulfone) and oxyacetamids (Example: flufenacet).

VLCFA Persistence and Residual Efficacy: Preemergence herbicides applied directly to the soil, such as VLCFA-inhibitors, need to persist long enough in the soil to provide adequate weed control throughout the duration of the critical weed control period. The persistence of a given herbicide in soil may affect both its agronomic properties, such as residual weed control. How long a pesticide persists in a soil is a function of its loss from the soil system and degradation (Hurle and Walker 1980; Monaco 2002; Peter and Weber 1985). Persistence is closely related to the kinetics of degradation, which depends on the molecular structure of a pesticide, and is also influenced by factors such as dose/application rate, temperature, soil water content/rainfall, pH, oxygen level, background level of the pesticide, soil fertility, tillage and management system and microbial populations (Blume 2004; Gaynor et al. 2000; Poppell et al. 2002; Ma et al. 2004;

Shipitalo and Owens 2006). The persistence of pesticides in soils is often indicated by DT_{50}, the time for 50% of initial residues to dissipate (Ma et al. 2004).

Herbicides can be lost from a soil system as a result of volatilization, leaching, surface runoff, loss of soil containing sorbed herbicide and/or herbicide breakdown via chemical decomposition, photodecomposition and/or biological decomposition (Hance 1980; Kah and Brown 2006; Monaco et al. 2002; Kwon et al. 2004; Torstensson 1980). The predominant loss of VLCFA-inhibitors, specifically, from the soil is due to microbial degradation (Beestman and Deming 1974; Baran 2004). Zimdahl and Clark (1982) showed that herbicide degradation rates for chloroacetamide herbicides increased as temperature and moisture content increased, which supports the hypothesis that degradation of chloroacetamide herbicides is dominated by microbial degradation (Westra et al. 2014).

Herbicide sorption to soil also influences a compound's environmental fate, persistence in the soil, and biological activity. There have been numerous studies on how chloroacetamide herbicides bind to soil components. For example, soil organic matter is the predominant adsorbent for *S*-metolachlor (Pusino 1992). Typically compounds with lower water solubility will have higher sorption coefficient values and will result in higher amounts of herbicide bound to the soil (Bailey 1968).

There is extensive research on the environmental fate and persistence of pesticides (Gaynor et al. 2000; Poppell et al. 2002; Ma et al. 2004; Shipitalo and Owens 2006; Hance 1980; Kah and Brown 2006; Monaco et al. 2002, Kwon et al. 2004; Torstensson 1980). This paper has focused on the primary systems of degradation for Group 15 herbicides, mainly being microbial degradation, and the characteristics of the subject herbicides that impact these systems.

S-metolachlor: Adsorption and desorption are key processes controlling herbicide efficacy and dissipation in soil and influences a compound's environmental fate, persistence in the soil, and biological activity (Laabs & Amelung 2005). These processes control herbicide availability for runoff, volatilization, leaching, photolysis and microbial degradation processes, affecting its efficacy on weed control and persistence in soil. According to existing literature, *S*-metolachlor exhibits moderate to long persistence in soil with its sorption in soil positively correlated to organic matter and clay content (Zemolin et al. 2014). According to the WSSA Herbicide Handbook (Shaner et al. 2014), *S*-metolachlor is reported to have an average half-life of 112-124 days while the Pesticide Properties Data Base reports 23.17-51.8 days (PPDB 2022). Others have reported Metolachlor having dissipation half-lives (DT_{50}s) that ranged from 10 to 142 d (Braverman et al. 1986; Mersie et al. 2004; Shaner et al. 2006; Walker and Brown 1985; Zimdahl and Clark 1982). Of the current chloroacetamide herbicides, *S*-metolachlor appears to be the most persistent (LeBaron et al. 1988; Walker et al. 1985; Zimdahl and Clark 1982). Overall, microbial degradation appears to be the major pathway for *S*-metolachlor dissipation in soil (Zemolin et al. 2014). *S*-metolachlor is considered to be moderately soluble in water at 488 mg/L at 20 ° C and adsorbs moderately to soil at 200 mL/g with variability depending on the soil type (Shaner et al. 2014).

Acetochlor: Acetanilides, such as acetochlor, are non-polar, non-ionizable herbicides and are considered moderately to highly soluble in water, moderately persistent in environment, and moderately to highly mobile in soil (Oliveira et al. 2013). Acetochlor itself is considered moderately soluble in water at 233 mg/L at 25 ° C and moderately adsorbent to soil at 170 mL/g (Shaner et al. 2014). There is variability in the reported half-life for acetochlor. The Pesticide Properties Data Base reports acetochlor as "non-persistent" with a DT_{50} of 12.1-14 days (PPDB

2022). Others have reported the half-life of acetochlor to be even shorter at <10 days for a number of soils (Mills et al 2001; Ferri and Vidal 2002; Ma et al. 2004; Baran et al. 2004; Dictor et al. 2008). Other research indicates, the reported half-life (DT_{50}) of acetochlor in the field ranges from 3.4 to 29 days, with a mean value of 12.9 days (Mills et al. 2001, Baran et al. 2004, Newcombe et al. 2005). Jablonkai et al. 2000 reports an average half-life of 90 days. Acetochlor is expected to have low leaching potential in most soils and its mobility correlates well with Kd and OM content. (Breaux 1986; Böger 2000; Jablonkai and Hatzios 1991; Scarponi et al. 1991).

Dimethenamid-P: Dimethenamid-P is considered to be highly soluble in water at 1174 mg/L at 25 °C and moderately adsorbs to soil at 155 mL/g. Pesticide Properties Data Base lists dimethenamid-P as "non-persistent" with a DT_{50} of 11-15.8 days (PPDB 2022). According to the WSSA Herbicide Handbook (Shaner 2014), Dimethenamid-P is reported to have an average half-life of 35-42 days. Kočárek 2018 investigated the impact of adjuvants on dimethenamid-P behavior in soil and reported that the dimethenamid-P dissipation half-lives ranged from 8.8 days for irrigated treatment without adjuvant to 12.9 days for non-irrigated treatment with adjuvant. Dimethenamid-P dissipation half-life in treatments with adjuvant was significantly longer than was half-life in a treatment without adjuvant. Significantly longer dissipation half-life was observed also in non-irrigated treatments than in irrigated treatments.

Pyroxasulfone: Pyroxasulfone is considered to have low solubility in water at 3.49 mg/L at 20 °C and moderately adsorbs to soil ranging from 57-114 mL/g. It has low leaching potential with less mobility in fine and medium textured soils and more mobility in coarse textured soils. (Tanetani 2009) There is a strong correlation between soil binding, reduced herbicide dissipation, and increased soil organic matter content (Westra et al. 2014). Pyroxasulfone is nonionizable,

and therefore soil pH does not affect dissociation. However, soil pH may influence bioactivity due to fewer negative charges on organic and clay surfaces in soils with lower pH (Szmigielski et al. 2014). There is variability in the average reported half-life of pyroxasulfone. According to the WSSA Handbook (Shaner et al. 2014) and Pesticide Properties Data Base, Pyroxasuflone is not considered to be persistent with reported DT_{50}'s of 16-26 days and 22 days, respectively. Westra et al. 2014 evaluated pyroxasulfone dissipation and mobility in the soil compared to *S*-metolachlor in 2009 and 2010 at two field sites in northern Colorado, on a fine clay loam, and fine sandy loam soil. Pyroxasulfone dissipation half-life (DT_{50}) values varied from 47 to 134 d. Between years, herbicide DT_{50} values were similar under the fine clay loam soil. Under the fine sandy loam soil, dissipation in 2009 was minimal under dry soil conditions. In 2010, under the fine sandy loam soil, pyroxasulfone had a half-life of 47 d. Overall, DT_{50} values ranged from 46 to 48 d for pyroxasulfone, compared to previously cited literature that reported DT_{50} of 8 to 71 d for pyroxasulfone (Mueller and Steckel 2011).

Flufenacet: Flufenacet is considered to have moderate solubility in water at 56 mg/L at 25 °C and moderately adsorbs to soil at an average of 355 mL/g depending on soil type. A soil half-life of 34 days has been reported when flufenacet was first presented (Deege et al. 1995) Pesticide Properties Data Base lists flufenacet as "Non to Moderately persistent" with a DT50 of 19.7-39 days (PPDB 2022). Research indicates that soil moisture has little effect on the dissipation of flufenacet in soil. However, the rate of application and soil type greatly affected it. At higher rate, the dissipation was slower. It is observed that dissipation of flufenacet is slower in soils having high adsorption capacity (either high OM or clay content) and slower desorption (Gupta et al. 2002). Flufenacet is also reported to strongly adsorb to alluvial soil (Gupta et al. 2001). Under aerobic microbial degradation half-life ranges from 10 to 34 days in various soils.

Under anaerobic conditions flufenacet has been reported to have a DT_{50} up to 240 days. Overall, Flufenacet is expected to have short to moderate persistence and low to moderate mobility in most soil textures. (Pflanzenschutz-Nachrichten 1997).

In short, there are a large number of factors that may affect the efficacy soil applied herbicides. In the specific case of VLCFA-inhibitors applied preemergence, the length of residual efficacy is critical in maintaining season long control of damaging weeds. While there is a large amount of research looking across the various impacts of soil type, weather conditions, application rate, formulation type etc. there is limited research looking at how residual control of VLCFA-inhibitors may vary across a specific weed species i.e. Palmer amaranth, which is known for its inherent genetic variability and ability to develop resistance to herbicides rapidly. The ability of VLCFA-inhibitors to maintain residual control of Palmer amaranth throughout the entire season is essential in minimizing the development and spread of resistant populations. The following research was conducted to investigate the differences in residual efficacy of PRE applied Group 15 herbicides in control of North Carolina Palmer amaranth populations.

MATERIALS AND METHODS

Greenhouse studies were conducted to investigate the difference in residual control of five VLCFA-inhibiting herbicides on two North Carolina Palmer amaranth populations. Experiments were conducted at the greenhouse facility located on the Syngenta Crop Protection site in Greensboro, NC in the Spring 2022.

The studies were arranged in a randomized complete block with a factorial arrangement of treatments and were repeated in time. Factors included: two Palmer amaranth populations (WAKE and MARTIN), six treatments (five herbicide treatments and one non-treated) and four

planting times (0, 2, 4 and 6 weeks after treatment) with 6 replications. The experiment was repeated in time. A replication consisted of 4 in-by 4 in-by 4 in pots filled with a Wakeland silt loam field soil, 2% OM, 5.7 pH, CEC 12-15. The populations used in the study had been originally collected by Mahoney et al. (2020) in the fall of 2016 from soybean fields in Wake and Martin counties of the "Coastal Plain" area of North Carolina. The North Carolina Coastal Plain is considered to be the state's primary row crop producing region (USDA 2018; Mahoney et al. 2020). Moore et al. 2021 conducted additional screening of the Mahoney at al. 2020 populations to assess susceptibility to atrazine, *S*-metolachlor, dicamba and 2,4-D. The resulting profiles from Mahoney et al. 2020 and Moore et al. 2021 research are outlined in Table 2.

The populations, referred to as WAKE and MARTIN for the remainder of this paper, were selected based on the results of the previous screening experiments. WAKE, which had demonstrated some level of resistance to ALS and glyphosate had no survivors after VLCFA-inhibitor application, was anticipated to be the more "susceptible" population. MARTIN, which had a slightly higher level of resistance to ALS and glyphosate relative to WAKE was chosen due to the presence of survivors after treatment with a VLCFA-inhibitor and was anticipated to be the more "tolerant" population. Both populations had germination rates >50%.

The treatment for each herbicide was determined by previous greenhouse dose response work conducted in the Spring of 2021. The dose response data generated in Spring 2021 was fit with a log-logistic 4 parameter model according to existing literature (Brabham et al. 2019; Strom et al. 2019. Estimated LD_{90} values for each population were calculated using the ED function in R (R Core Team 2020). The LD_{90} for the "susceptible" population, WAKE, was utilized in the residual efficacy study. The rate factor for each herbicide in this study are as follows; 1138 g *S*-metolachlor/ha^{-1}, 204 g acetochlor/ha^{-1}, 142 g dimethenamid-P/ha^{-1}, 76 g pyroxasulfone/ha^{-1} and

693 g flufenacet/ha^{-1} respectively. A summary of the log-logistic 4 parameter model, parameter outputs and subsequent use rate for each herbicide is outlined in Table 3.

Prior to applications, pots were filled to the brim with soil, tampered, presoaked, and allowed to drain. The pots were lightly watered just prior to applications. Treatments were applied using a research track sprayer equipped with 11015 VP nozzle (Teejet, Wheaton, IL) calibrated to deliver 140 L ha^{-1} GPA at 4.8 km/h. Following application, pots were placed in the greenhouse and watered over the top to incorporate the herbicides in the soil. Pots were watered over the top daily until 7 days after planting (DAP) when they were moved to trays and watered from the bottom. The greenhouse was maintained at an average temperature of 70-80 degrees Fahrenheit throughout the duration of the study.

For each planting time, seventy-two pots (thirty-six pots per population, twelve pots per treatment) per run were prepped and treated then placed in the greenhouse for the duration of each planting time (6, 4 and 2 weeks). Following the final treatment of the 0 day planting interval pots, approximately 20-30 seeds were scattered over the soil surface of each (all) pots, covered with soil, lightly tampered, and watered over the top. The study was concluded 21 days after planting (DAP) with live counts taken at 7, 14 and 21 DAP.

Data Collection and Analysis

21 days after planting (DAP), live counts were recorded. For each experimental run, live counts were converted to percent control based on the number of seedlings in the non-treated controls as described in Equation 1.

$$y = ((t/n)100) \quad [1]$$

Where y is percent control, t is number of weeds that emerged in treated soil and n is the average emergence of weeds in the respective non-treated soil. Weed control was analyzed with R to test

for significant interactions. There was a slightly significant difference between runs (p= 0.0317). This was attributed to the highly significant main effects and therefore, data were pooled accordingly (P>0.05). There were significant effects of (P< 0.0001) population, time and herbicide on weed control with significant run by herbicide and time by herbicide interactions (P<0.05). (Table 4).

For each herbicide, average weed control for population as affected by time was fit with trend lines using Sigma Plot software (Systat Software, Inc. version 12.5, San Jose, CA 95131) and was modeled using (Equation 1).

(1) $$Y = C + \frac{D-C}{1+exp\{b[log(x)]-log(e)\}}$$

Where Y is weed control achieved at x, c is the asymptote at the lower limit (fixed at 0), b is the slope around e, which is the value of x (time in weeks) where 50% reduction of Y is observed. (Ritz et al. 2015). All r^2 values were >0.9. Time elapsed in weeks until a 50% reduction in weed control was observed (I_{50}) was calculated for each soil and weed species using the respective regression equation to compare weed control over time between herbicides (Seefeldt et al. 1995) (Table 5).

RESULTS AND DISCUSSION

Averaged over herbicides and timings, control of the MARTIN population was less than the control of the WAKE population, at 44.7% and 58.9% respectively (data not shown). No interaction between population and either timing or herbicide was detected. This lack of interaction was due to both populations having a similar response to the herbicides evaluated over time, however the difference in overall control of populations was significant. Since no interaction was observed, only herbicide by timing interactions will be discussed further.

The high variability in the control offered by flufenacet application has been observed in dose response and residual control greenhouse experiments (Foderaro 2022). For the residual control greenhouse study, the average control of flufenacet across all application timings ranged from 36 to 60%. Control of Palmer amaranth with flufenacet never reached greater than 60%, even with the LD_{90} rate at 0 weeks after treatment. There is limited dose response literature available with flufenacet with various levels of success reported (Messelhäuser et al. 2021; Moi et al. 2015; Metcalfe et al. 2017;2018). Moi et al. 2015 reported significant differences in flufenacet ED50 rates between runs in dose response work conducted in 2015 though this was attributed to differences in potting mixture (Moi et al. 2015).

When comparing the response of Palmer amaranth to each herbicide over time, *S*-metolachlor and pyroxasulfone resulted in greater length of control when compared to acetochlor and dimethenamid-P. The average control with *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet at 0 weeks after application was 82%, 64%, 69%, 91%, 48%, respectively (Table 6; Figure 2). As time after application increased, average control for all herbicides decreased with *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet averaging 51%, 11%, 15%, 55% and 36%, respectively, 6 weeks after application (Table 6; Figure 2). The estimated I_{50} (weeks until a 50% reduction in control) for *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet was 6.7, 1.2, 1.42, 7 and 2.19 weeks, respectively. These values were determined by the formula of the best fit model for each herbicide (Table 5).

S-metolachlor and pyroxasulfone demonstrated greater levels of control across all application timings compared to acetochlor, dimethenamid-P and flufenacet. At 6 weeks following application, control of Palmer amaranth with *S*-metolachlor and pyroxasulfone was >50% (Table

6). Palmer amaranth control for acetochlor and dimethenamid-P followed similar trends when averaged over populations (Table 6; Figure 2). Average initial control, at 0 weeks after treatment, was 82% and 81% for acetochlor and dimethenamid-P, respectively and decreased by 40%, 2 weeks after treatment. The 4 and 6 week after treatment control values were very similar for acetochlor and dimethenamid-P with control averaging 27 and 11% and 27 and 15%, respectively. Although *S*-metolachlor and pyroxasulfone provided extended control >50% for the duration of the study, there were subtle differences observed. Overall average control across populations was similar for pyroxasulfone and *S*-metolachlor 73 and 67%, respectively. At 0 week and 2 weeks after treatment, pyroxasulfone control averaged approximately 10% higher than *S*-metolachlor at 91 and 80% and 82 and 69%, respectively. However, at 4 and 6 weeks after treatment pyroxasulfone and *S*-metolachlor averaged similar levels of control at 65 and 55% and 65 and 51%, respectively (Table 6; Figure 2).

The LD_{90} rate utilized for each herbicide was anticipated to demonstrate approximately 90% control of the WAKE population at 0 weeks after application. The results indicated average control of the WAKE population was 93%, 82%, 81%, 95% and 54% for *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufencet, respectively (Table 6; Figure 1). For acetochlor and dimethenamid-P, the overall performance of the LD_{90} rate provides some level of explanation for the significant difference between the labeled rate and the biologically effective rate for each herbicide. In previously conducted dose response greenhouse work, acetochlor and dimethenamid-P demonstrated higher levels of control where the 1X labeled rate and higher were used in addition to the lower tiered rates. The biologically effective rate, represented in these studies by the LD_{90} rate, offered higher initial control of the Palmer amaranth populations around the time of initial application. Control decreased significantly with a control averaging

<50% after 2 weeks for both acetochlor and dimethenamid-P. *S*-metolachlor and pyroxasulfone both had an LD_{90} rate or biologically effective rate that was nearer to the 1X labeled rate (Table 7). For both herbicides, the LD_{90} rate remained biologically effective, offering up >50% control, even after 6 weeks of application with pyroxasulfone having an estimated I_{50} of 7+ weeks.

As previously discussed, pesticide half-life in soil varies significantly depending on dose/application rate, temperature, soil water content, rainfall, soil pH, oxygen level, background level of the pesticide, soil fertility, tillage and management system and microbial populations (Blume 2004, Gaynor et al. 2000; Poppell et al. 2002; Ma et al. 2004; Shipitalo and Owens 2006). There are various reported half-life values in the literature for *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasuflone and flufenacet and a significant body of research exploring many of the aforementioned variables and their impact on persistence and performance of these herbicides. When comparing the I_{50} values determined in this study there are similarities and trends that can be discussed however, the extension of these findings to actual field scenarios is unlikely to be relevant due to the difference in the LD_{90} rates used in the study and the labeled rates that would typically be applied in the field. However, the comparison across herbicides in terms of overall persistence is consistent with what is in the literature. This research indicates that *S*-metolachlor and pyroxasulfone tend to persist longer at a biologically effective rate compared to other VLCFA-inhibitors such as acetochlor, flufenacet and dimethenamid-P. This generally aligns with what others have reported over the years (Westra 2014; Ribeiro et al. 2021; Bedmar 2017)

It is likely that some of these results can be attributed to the rate utilized for each herbicide. The selected rate was based on dose-response data from previous greenhouse experiments where curves were generated in R based on a log-logistic 4 parameter model that has been commonly

used in previous herbicide dose response research (Brabham 2019; Strom 2019; R Core Team 2020). The LD_{90} rate for each herbicide on each population was then estimated using the ED function in R (R Core Team 2020) (Table 3). The LD_{90} rate for WAKE, the susceptible population, was selected for use in the residual efficacy study. In subsequent analysis of the dose response data and other available models, there is an argument to be made that the log-logistic four-parameter model may have overestimated LD_{90} rates for some of the herbicides even though

CONCLUSIONS

While the persistence of labeled rates of Group 15 herbicides is well documented in the literature (Table 8), this study provides some insight into how biologically effective rates of these herbicides may persist. Overall, pyroxasulfone and *S*-metolachlor demonstrated longer periods of residual control compared to acetochlor and dimethenamid-P at comparable initial biologically effective rates. This further emphasizes the importance of using full labeled rates for herbicides such as acetochlor and dimethenamid-P, where labeled rates may be necessary to compensate for reduced residual control (comparatively speaking). Ultimately, environmental conditions are still the primary factors that impact persistence and residual control of pesticides. These study results provide some insight into how long these Group 15 herbicides may persist with consistent environmental conditions, which is not necessarily congruent with real world field conditions. Though this study did demonstrate significant differences in overall control of the two populations, because there were no interactions observed with herbicide or time, it is difficult to say whether or not the differing resistance profiles could be a contributing factor to this difference. Further research with additional populations of Palmer amaranth may be necessary to confirm whether putative resistance in a Palmer amaranth accession may impact the overall residual control these Group 15 herbicides offer. Additional research comparing 1X and LD_{90}

rates of these herbicides may offer additional insight and comparison between biologically effective rates and labeled rates as well as the comparison of these rates on differing soil types. Literature has indicated that the predominant loss of VLCFA-inhibitors, specifically, from the soil is due to microbial degradation (Beestman and Deming 1974; Baran 2004; Zimdahl and Clark 1982) therefore it would of interest to understand how residual efficacy of Group 15 herbicides might be impacted by soil type and varying levels of microbial activity.

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FIGURES AND TABLES

Table 1: List of Mode of Actions associated with Herbicide Resistant Palmer amaranth (*Amaranthus palmeri*)

WSSA Group (HRAC)	Site of Action	First Year	Crop/Country
2 (B) *Ex. imazethapyr*	Inhibition of Acetolactate Synthase (ALS)	1993	Alfalfa, Corn (maize), Sorghum, and Soybean/ United States (Kansas)
3 (K1) *Ex. Trifluralin*	Inhibition of Microtubule Assembly	1989	Cotton, and Soybean/ United States (South Carolina)
4 (O) *Ex. 2,4-D*	Auxin Mimics	2015	Sorghum/United States (Kansas)
5 (C1/2) *Ex. Atrazine*	PSII – Serine 264 Binders	1993	Corn and Sorghum/ United States (Texas)
9 (G) *Ex. glyphosate*	Inhibition of Enolpyruvyl Shikimate Phosphate Synthase (EPSPS)	1995	Cotton and Soybean/ United States (Georgia)
14 (E) *Ex. fomesafen*	Inhibition of Protoporphyrinogen Oxidase (PPO)	2011	Soybean/ United States (Arkansas)
15 (K3 N) *Ex. S-metolachlor*	Very Long-Chain Fatty Acid Synthesis inhibitors (VLCFA)	2016	Soybean/ United States (Arkansas)
27 (F2) *Ex. mesotrione*	Inhibition of Hydroxyphenyl Pyruvate Dioxygenase (HPPD)	2016	Corn/ United States (North Carolina)

Table 2: Profiles of selected Palmer amaranth populations, WAKE and MARTIN

Population Name	County	Cropping System	Survival [1]					
			Thifensulfuron	Glyphosate	Fomesafen	Mesotrione	Glufosinate	*S*-metolachlor[2]
WAKE	Wake	Soybean	27%	46%	0%	0%	0%	0%
MARTIN	Martin	Soybean	71%	70%	0%	2%	0%	10-15%

[1] < 90% injury; plants with functional green leaves and apical meristems as reported by Mahoney et al. (2020)
[2] Survival at 800 g/ha rate of *S*-metolachlor as reported by Moore et al. (2021)

Table 3: Dose-response parameter estimates and LD_{90} values for *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet for control of WAKE and MARTIN populations. A four-parameter log-logistic function was used to estimate the LD_{90} rates[a]

Herbicide	Population	Dose-response parameters estimates				
		b	***c***	***d***	***LD50***	***LD90***
S-metolachlor	WAKE	0.843	-4.857	105.657	84	**1138**
	MARTIN	0.753	-29.131	100.853	596	11018
acetochlor	WAKE	0.9909	-1.5276	99.8988	22	**204**
	MARTIN	0.90673	-4.5222	99.7480	102	1148
dimethenamid-P	WAKE	0.75771	-1.6406	100.179	8	**142**
	MARTIN	0.62967	-20.137	97.8789	102	3329
pyroxasulfone	WAKE	0.69366	-6.2185	99.9216	3	**76**
	MARTIN	0.86584	-5.3365	109.168	10	126
flufenacet	WAKE	3.01917	-4.984	77.8198	335	**693**
	MARTIN	0.9919	-16.108	74.0943	635	5817

[a] Log-Logistic 4 Parameter, $Y = C + D - C/(1 + exp\{b[log(x)] - log(LD50)\}$
Bolded values = rates in g ai/ha^{-1}, used in residual efficacy greenhouse study

Table 4: The F and p-values for Palmer amaranth control (%) 21 Days after Planting (DAP) 0, 2, 4, and 6 weeks after application/treatment in a greenhouse experiment in Greensboro, NC, USA

	21 DAP	
Effect	F Value	P > F
Population	36.99	<0.0001
Time	45.75	<0.0001
Herbicide	43.81	<0.0001
Time*Herbicide	2.64	0.0021
Run	4.65	0.0317
Run*Herbicide	4.11	0.0028

Table 5: I_{50} and regression equation with parameters to model Group 15/VLCFA length of residual control of North Carolina Palmer amaranth populations.

Herbicide	Model[1]	I_{50}	R^2
S-metolachlor	$f = \frac{81.8577}{1+exp(1.2683(log(x)-log(9.5493)))}$	6.7	0.9498
acetochlor	$f = \frac{64.2705}{1+exp(1.5543(log(x)-log(2.7065)))}$	1.2	0.9816
dimethenamid-P	$f = \frac{68.8610}{1+exp(1.5064(log(x)-log(2.7464)))}$	1.42	0.9954
pyroxasulfone	$f = \frac{90.9525}{1+exp(1.3676(log(x)-log(8.1246)))}$	7	0.9963
flufenacet	$f = \frac{50.1919}{1+exp(4.6376(log(x)-log(7.3297)))}$	2.19	0.9353

[1]Log-logistic three-parameter model, $Y = C + \frac{D-C}{1+exp\{b[log(x)]-log(e)\}}$, was used to fit data

Table 6: Group 15/VLCFA LD_{90} rates and average control of North Carolina Palmer amaranth populations over time

			WAKE		MARTIN		WAKE + MARTIN	
Herbicide	**LD_{90} Rate[1] (g ai/ha^{-1})**	**WAT[2]**	**% Control (Average)**	**SE**	**% Control (Average)**	**SE**	**% Control (Average)**	**SE**
S-metolachlor	1138	0	93	2.77	71	9.59	82	7.10
		2	70	8.28	69	7.52	69	5.47
		4	69	6.97	62	6.18	65	4.61
		6	58	9.72	42	7.69	51	6.38
acetochlor	204	0	82	6.08	48	8.68	64	6.37
		2	44	8.91	32	8.66	38	6.19
		4	33	8.14	8	3.25	27	6.15
		6	18	8.10	5	2.12	11	4.32
dimethenamid-P	142	0	81	7.03	57	9.53	69	6.31
		2	41	6.01	42	10.3	42	5.85
		4	24	7.52	30	7.81	27	5.33
		6	10	4.65	19	5.84	15	3.79
pyroxasulfone	76	0	95	2.70	86	3.00	91	2.20
		2	92	3.83	68	10.56	80	6.02
		4	77	5.69	54	11.71	65	6.57
		6	69	7.38	43	9.36	55	6.49
flufenacet	693	0	54	9.85	42	7.85	48	6.30
		2	60	7.83	45	7.56	52	5.54
		4	58	7.88	36	8.36	47	6.05
		6	44	8.70	26	9.49	36	6.54

[1] LD_{90} rate from previous dose response work (Foderaro et al. 2022)
[2] Weeks After Treatment

Table 7: Group 15/VLCFA-inhibiting herbicides, trade names, companies, herbicide families, half-lives, labeled soybean rates, LD_{50}, LD_{90} and I_{50} rates.

Herbicide	Trade Name/ Company/ Location	Herbicide Family	1X Rate[1]	Half-life[2]	LD_{90} Rate[3]	I_{50}
			g ai/ha	Days	g ai/ha	Weeks (Days)
S-metolachlor	Dual Magnum Syngenta Greensboro, NC	Chloroacetamide	1422	23.17-51.8	1138	6.7 (46.9)
acetochlor	Harness Bayer St Louis, MO	Chloroacetamide	2210	12.1-14	204	1.2 (8.4)
dimethenamid-P	Outlook BASF Research Triangle Part, NC	Chloroacetamide	737	11-15.8	142	1.42 (9.94)
pyroxasulfone	Zidua SC BASF Research Triangle Part, NC	Pyrazole	119	22	76	7 (49)
flufenacet	Sunfire Certis Europe BV Great Abington, Cambridgeshire	Oxyacetamide	240	19.7-39	693	2.19 (15.3)

[1] Based on labeled soybean rate for each herbicide and 1X rate used in previously conducted dose response experiment
[2] Half-life values were obtained from the Pesticide Properties Data Base (http://sitem.herts.ac.uk/aeru/ppdb/en/)
[3] From previous greenhouse dose response research with WAKE and MARTIN populations (Foderaro et al. 2022)

Table 8: Summary of reported DT_{50}/half-life for *S*-metolachlor, acetochlor, dimethenamid-P, pyroxasulfone and flufenacet*

Herbicide	Reported Half-life	Reference
S-metolachlor	112-124	Shaner et al. 2014
	10-142	Braverman et al. 1986; Mersie et al. 2004; Shaner et al. 2006; Walker and Brown 1985; Zimdahl and Clark 1982
	82 to 141	Bedmar 2017
	37.9-135.9	Wu et al. 2011
	17.4	Fava et al 2000
	69.3	Coroi et al 2011
	5.7-100.7	Dinelli et al 2000
	50-289	Rice et al. 2002
	6-9.6	Mersie et al 2004
	10-23	Staddon et al 2001
	2.5-18	Sanyal & Kulshrestha 1999
	28-34	Nunes & Vidal 2008
	97-127	Alletto 2013
	23.17-51.8 (Moderately persistent)	Pesticide Properties Data Base
acetochlor	<10 days	Mills et al. 2001; Ferri and Vidal 2002; Ma et al. 2004; Baran et al. 2004; Dictor et al. 2008
	3.4 to 29 (Avg 12.9)	Mills et al. 2001, Baran et al. 2004, Newcombe et al. 2005
	90	Jablonkai 2000
	15 to 73	Bedmar 2017
	12.1-14 (Non-persistent)	Pesticide Properties Data Base
dimethenamid-P	35-42	Shaner et al. 2014
	11-15.8 (Non-persistent)	Pesticide Properties Data Base
	8.8-12.9	Kočárek 2018
pyroxasulfone	16-26	Shaner et al. 2014
	47 to 134	Westra et al. 2014
	8 to 71	Mueller and Steckel 2011
	22 (Non-persistent)	Pesticide Properties Data Base
flufenacet	10-34 (aerobic) Up to 240 (anaerobic)	Shaner et al. 2014
	9.3-22.5	Gupta 2001
	19.7-39 (Non-Moderately persistent)	Pesticide Properties Data Base

*Note this list is not exhaustive of all available literature

Figure 1: Residual control 21 days after planting of preemergence applied Group 15 herbicides on WAKE and MARTIN populations of Palmer amaranth planted 0, 2, 4 and 6 weeks after treatment.

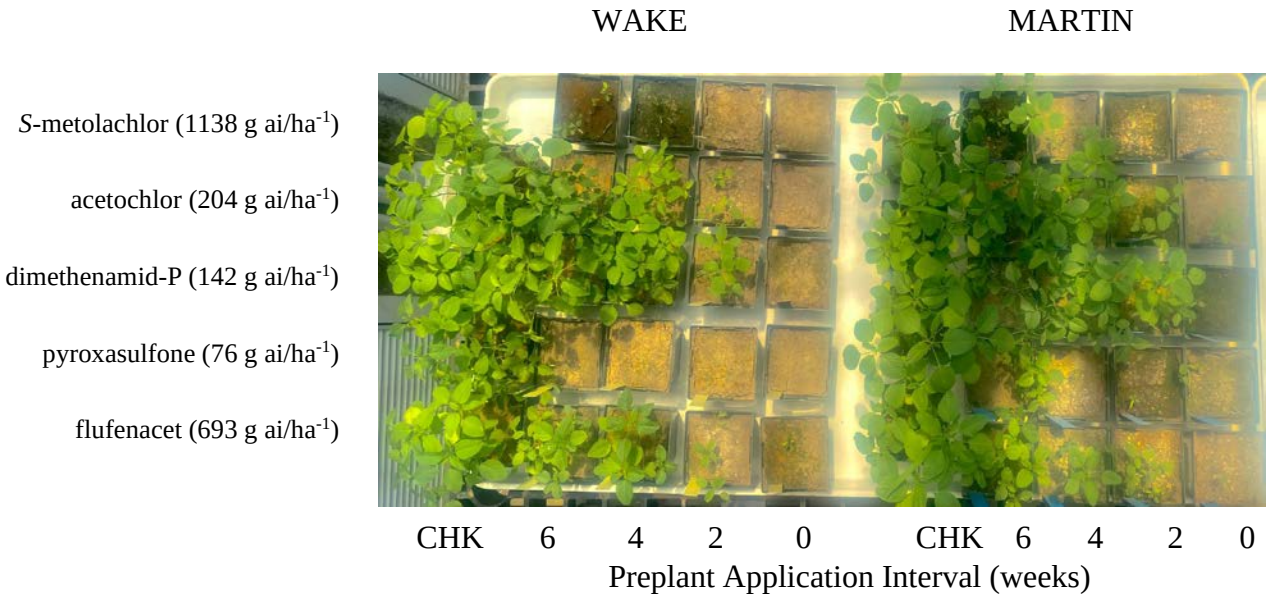
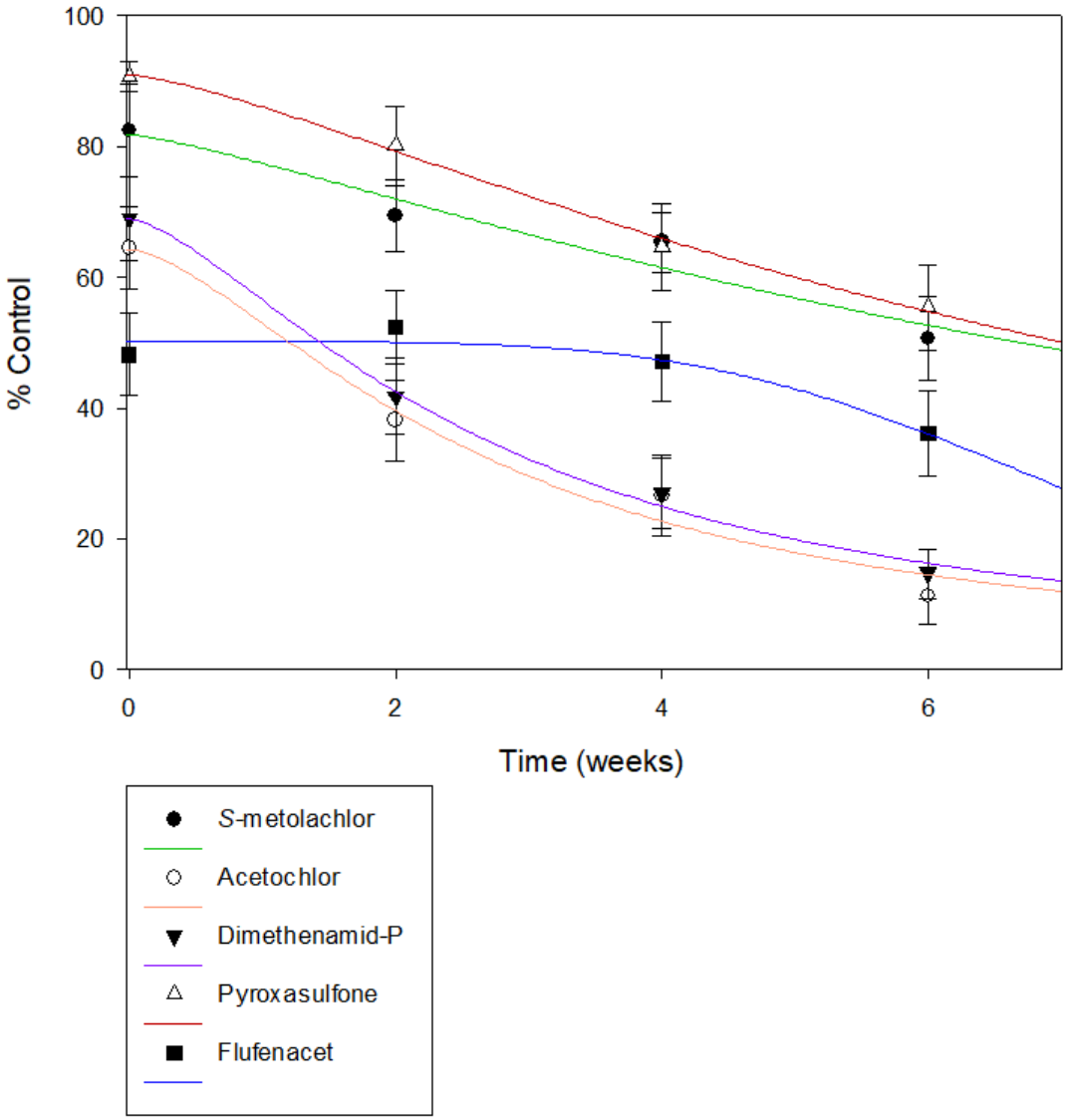


Figure 2: Residual control of Palmer amaranth by Group 15 herbicides



APPENDICES

APPENDIX A: CHAPTER 2

		LD_{50} comparison between models (g ai/ha)									
Herbicide	**Population**	**Log-Logistic (4P)**	**R/S Ratio**	**Weibull Type I (3P)**	**R/S Ratio**	**Weibull Type II (3P)**	**R/S Ratio**	**Log-Logistic (3P)**	**R/S Ratio**	**Weibull Type I (4P)**	**R/S Ratio**
S-metolachlor	WAKE	84		81		71		83		83	
	MARTIN	596	7	336	4	298	4	287	3	394	5
acetochlor	WAKE	22		19		24		22		19	
	MARTIN	102	5	96	5	84	4	89	4	96	5
dimethenamid-P	WAKE	8		6		9		8		6	
	MARTIN	102	13	63	11	92	11	49	6	67	11
pyroxasulfone	WAKE	3		3		3		3		3	
	MARTIN	10	3	10	4	8	3	11	4	10	4
flufenacet	WAKE	335		327		302		156		329	
	MARTIN	635	2	462	1	207	1	135	1	2572	8

		LD_{90} comparison between models (g ai/ha)									
Herbicide	**Population**	**Log-Logistic (4P)**	**Ratio**	**Weibull Type I (3P)**	**Ratio**	**Weibull Type II (3P)**	**Ratio**	**Log-Logistic (3P)**	**Ratio**	**Weibull Type I (4P)**	**Ratio**
S-metolachlor	WAKE	**1138**		698		862		766		732	
	MARTIN	11018	10	1768	3	3199	4	2355	3	2632	4
acetochlor	WAKE	**204**		178		167		170		184	
	MARTIN	1148	6	660	4	938	6	775	5	669	4
dimethenamid-P	WAKE	**142**		120		110		111		115	
	MARTIN	3329	23	506	4	690	6	650	6	655	6
pyroxasulfone	WAKE	**76**		34		40		37		37	
	MARTIN	126	2	74	2	100	2	88	2	72	2
flufenacet	WAKE	**693**		562		633		1847		566	
	MARTIN	5817	8	1951	3	312517	494	55133	30	10647	19

Amount used in residual efficacy study

Amount based on model with best fit (lowest AIC and p-values)

Amount used in residual efficacy compared to model reported in dose response: 1.6X more for S-metolachlor, 26 more grams for acetochlor, 22 more grams for dimethenamid-P, 2X for pyroxasulfone and 131 more grams for flufenacet

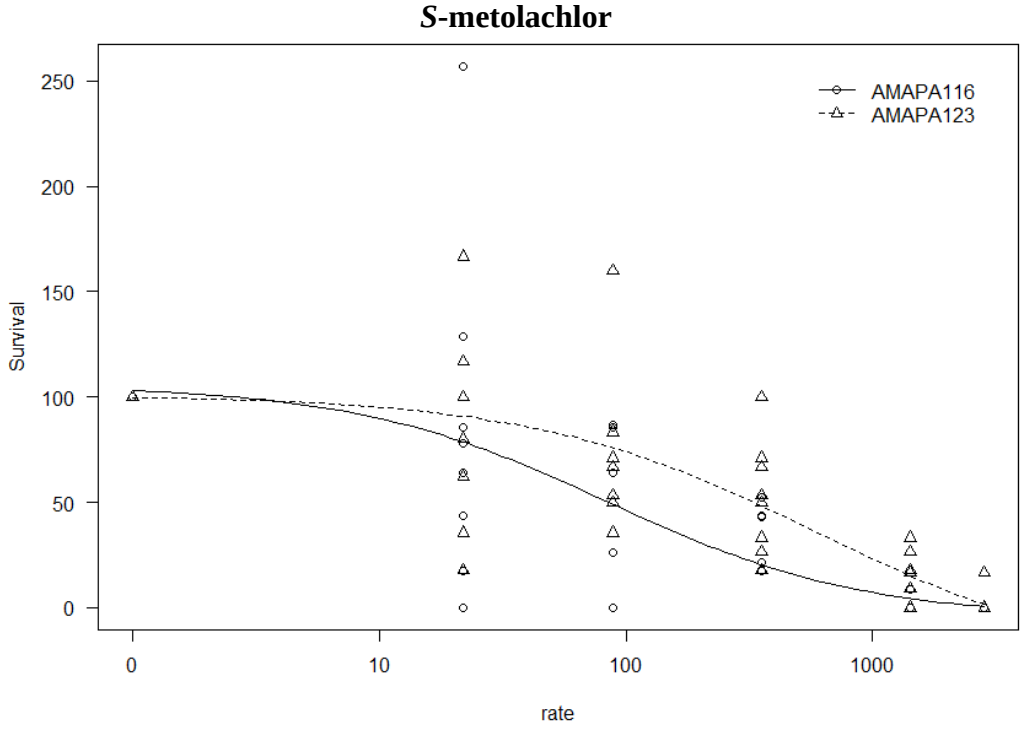
Output from drm and mselect Regression Analysis in R
Dual Magnum (S-metolachlor) Dose Response – 4 Parameter Log-Logistic Model

Model type	Model function (*f*)	Function in *drc*
Generalized log-logistic	$c + \frac{d-c}{(1+\exp(b(\log(x)-\log(e))))^f}$	`llogistic()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = mid-point/LD50

DM <- drm(Survival ~ rate, Population, data = DMAll, fct=LL.4())

> plot(DM, type="all")



Summary of Parameters

> summary(DM)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116   0.84265    0.51146  1.6475    0.1036
b:AMAPA123   0.75325    0.60029  1.2548    0.2134
c:AMAPA116  -4.85709   21.17788 -0.2293    0.8192
c:AMAPA123 -29.13108   71.93623 -0.4050    0.6866
d:AMAPA116 105.65688   22.22146  4.7547 9.230e-06 ***
d:AMAPA123 100.85299   20.97970  4.8072 7.554e-06 ***
e:AMAPA116  83.87069   74.76817  1.1217    0.2655
e:AMAPA123 596.02083  970.06177  0.6144    0.5408
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 35.60109 (76 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(DM, c(10, 50, 90), interval="delta")

```
Estimated effective doses

               Estimate Std. Error      Lower      Upper
e:AMAPA116:50     83.871     74.768    -65.043    232.784
e:AMAPA116:90   1137.741   2379.225  -3600.897   5876.379
e:AMAPA123:50    596.021    970.062  -1336.025   2528.066
e:AMAPA123:90  11017.876  40388.063 -69421.923  91457.675
```

Comparison of 4 Parameter Log Logistic Models to Other Models (Weibull Type 1 &2, Log-Logistic 3P, Cubic, Quadratic and Linear)

mselect(DM, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

```
         logLik       IC Lack of fit  Res var
W1.3  -415.3196 840.6391   0.9736440 1211.330
W2.3  -415.7818 841.5636   0.9317350 1224.735
LL.3  -415.3948 844.7895   0.8880501 1244.616
W2.4  -414.8970 847.7941   0.8400045 1262.321
LL.4  -415.0669 848.1339   0.7872100 1267.438
W1.4  -415.2367 848.4734   0.7331238 1272.571
Cubic -419.4128 848.8256          NA 1335.328
Quad  -421.2745 850.5491          NA 1378.618
Lin   -427.1219 860.2437          NA 1565.231
```

Comparison of Plotted Curves

model.W23 <- drm(Survival ~ rate, Population, data = DMAll, fct=W2.3(fixed=c(NA, 100, NA), names = c("Slope", "Upper Limit", "ED50")))

model.W24 <- drm(Survival ~ rate, Population, data = DMAll, fct=W2.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

model.LL4 <- drm(Survival ~ rate, Population, data = DMAll, fct=LL.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

model.W14 <- drm(Survival ~ rate, Population, data = DMAll, fct=W1.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

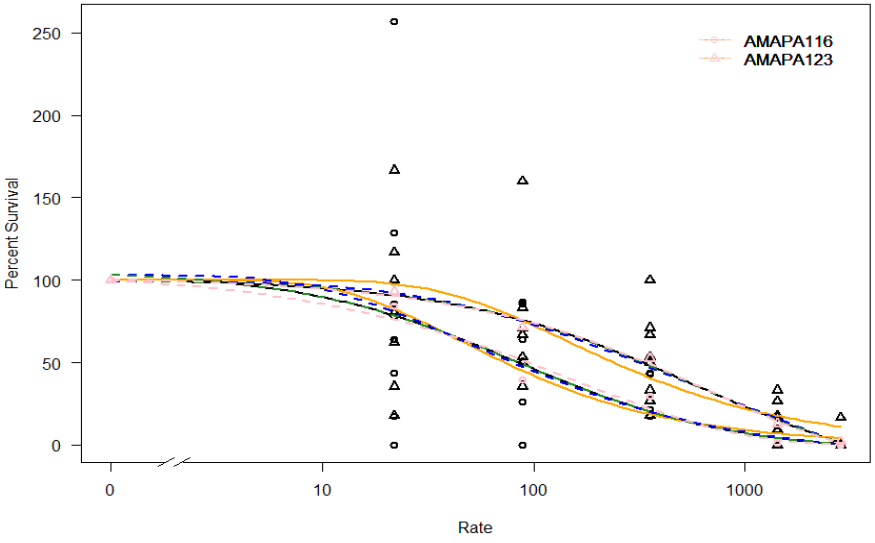
plot(DM, broken = TRUE, xlab="Rate", ylab="Percent Survival", type='all',lty=1, lwd=2)

plot(model.W23, add=TRUE,col="orange",lty=1, lwd=2)

plot(model.W24, add=TRUE,col="blue",lty=2, lwd=2)

plot(model.LL4, add=TRUE,col="forestgreen",lty=2, lwd=2)

plot(model.W14, add=TRUE,col="pink",lty=2, lwd=2)



Model Averaging – ED Values of models with top 3 highest p-values/lowest AIC + Log Logistic 4P

maED(DM,list(W1.3(),W2.3(),LL.3(fixed=c(NA, 100, NA)), W1.4()),c(50, 90),interval="kang")

1. Weibull Type 1 3 Parameter (W1.3())
2. Weibull Type 2 3 Parameter (W2.3())
3. Log Logistic 3 Parameter(LL.3())

AMAPA116 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	83.87069	1137.7413	0.02068252
W1.3	81.41140	697.6301	0.12419443
W2.3	70.62739	861.8353	0.08548715
LL.3	83.01057	765.9591	0.75218278
W1.4	83.00697	731.8696	0.01745312

AMAPA123 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	596.0208	11017.876	0.02068252
W1.3	336.3902	1768.305	0.12419443
W2.3	298.4003	3199.416	0.08548715
LL.3	287.4705	2355.375	0.75218278
W1.4	394.4212	2631.974	0.01745312

Averaged LD50 and LD90 Values across models

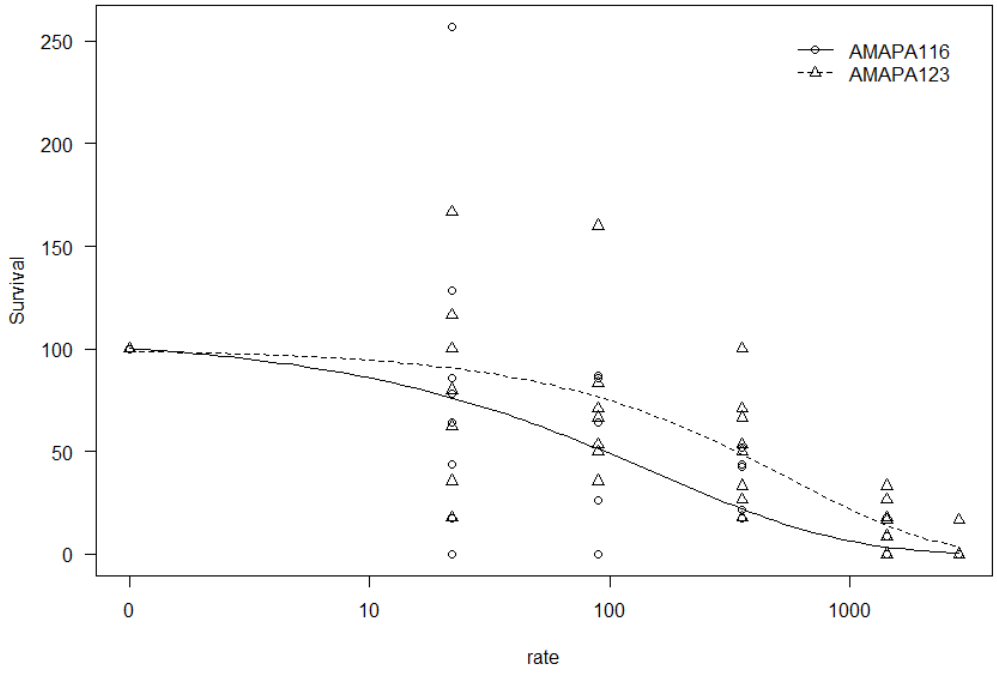
	Estimate	Lower	Upper
e:AMAPA116:50	81.77108	8.844045	154.6981
e:AMAPA116:90	772.76361	-523.315750	2068.8430
e:AMAPA123:50	302.72863	14.290314	591.1669
e:AMAPA123:90	2538.60844	-2272.405037	7349.6219

Dual Magnum (S-metolachlor) Dose Response – Weibull Type 1 3 Parameter Model

Model type	Model function (*f*)	Function in *drc*
Weibull I	$c+(d-c)\exp(-\exp(b(\log(x)-\log(e))))$	`weibull1()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = LD50

DM2<- drm(Survival ~ rate, Population, data = DMAll, fct=W1.3())



Summary of Parameters

summary(DM2)

```
Model fitted: Weibull (type 1) with lower limit at 0 (3 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116   0.55887    0.21209  2.6350   0.01014 *
b:AMAPA123   0.72344    0.33484  2.1606   0.03380 *
d:AMAPA116 106.34843   22.20018  4.7904 7.783e-06 ***
d:AMAPA123  99.78177   18.45576  5.4065 6.800e-07 ***
e:AMAPA116 156.85604   95.68346  1.6393   0.10517
e:AMAPA123 558.30197  278.73563  2.0030   0.04865 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 35.22859 (78 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(DM2, c(50, 90), interval="delta")

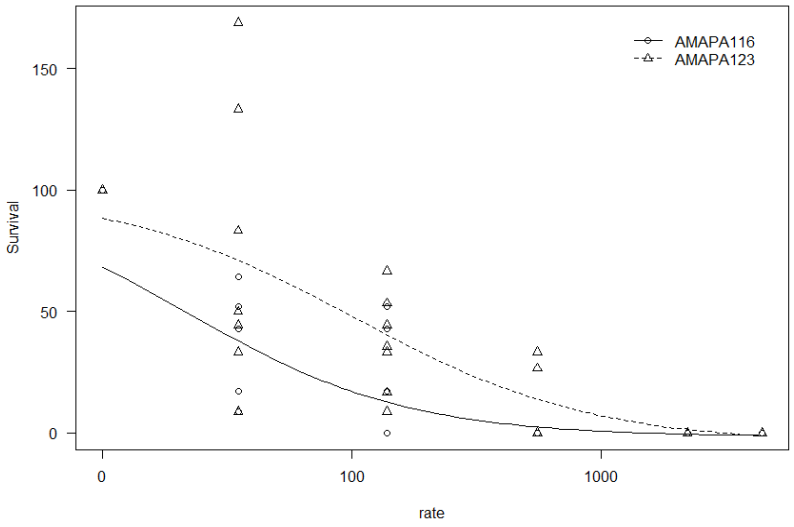
```
Estimated effective doses

              Estimate Std. Error    Lower    Upper
e:AMAPA116:50   81.411     59.321  -36.687  199.510
e:AMAPA116:90  697.630    479.488 -256.958 1652.218
e:AMAPA123:50  336.390    219.719 -101.036  773.817
e:AMAPA123:90 1768.305    893.809  -11.132 3547.743
```

Harness (Acetochlor) Dose Response – 4 Parameter Log-Logistic Model

Model type	Model function (*f*)	Function in *drc*
Generalized log-logistic	$c + \frac{d-c}{(1+\exp(b(\log(x)-\log(e))))^f}$	`llogistic()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = LD50

H <- drm(Survival ~ rate, Population, data = HAll, fct=LL.4())
plot(H, type="all")



Summary of Parameters

summary(H)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116   0.99090    0.52298  1.8947   0.06193 .
b:AMAPA123   0.90673    0.32309  2.8065   0.00636 **
c:AMAPA116  -1.52763    5.94257 -0.2571   0.79782
c:AMAPA123  -4.52221    8.99965 -0.5025   0.61678
d:AMAPA116  99.89886   14.69154  6.7998 2.088e-09 ***
d:AMAPA123  99.74804   14.49395  6.8820 1.464e-09 ***
e:AMAPA116  22.20782   10.87628  2.0419   0.04464 *
e:AMAPA123 101.77527   46.43536  2.1918   0.03146 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 20.75225 (76 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(H, c(50, 90), interval="delta")

```
Estimated effective doses

                Estimate  Std. Error       Lower       Upper
e:AMAPA116:50    22.20782    10.87628     0.54582    43.86981
e:AMAPA116:90   203.94454   199.26976  -192.93559   600.82467
e:AMAPA123:50   101.77527    46.43536     9.29124   194.25931
e:AMAPA123:90  1148.27153  1103.49722 -1049.53367  3346.07672
```

Comparison of 4 Parameter Log Logistic Models to Other Models (Weibull Type 1 &2, Log-Logistic 3P, Cubic, Quadratic and Linear)

mselect(H, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

```
         logLik       IC Lack of fit  Res var
W1.3  -369.6920 749.3841   0.9999978 408.7502
W2.3  -370.3610 750.7220   0.9946911 415.3127
LL.3  -369.9427 753.8853   0.9961785 421.7402
W1.4  -369.6843 757.3685   0.9971184 430.1838
LL.4  -369.7303 757.4607   0.9935673 430.6561
W2.4  -369.8192 757.6383   0.9832670 431.5677
Cubic -388.1343 786.2686          NA 634.0999
Quad  -397.7183 803.4366          NA 786.7998
Lin   -405.2823 816.5646          NA 930.5721
```

Comparison of Plotted Curves

modelH.W23 <- drm(Survival ~ rate, Population, data = HAll, fct=W2.3(fixed=c(NA, 100, NA), names = c("Slope", "Upper Limit", "ED50")))

modelH.W24 <- drm(Survival ~ rate, Population, data = HAll, fct=W2.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

modelH.LL4 <- drm(Survival ~ rate, Population, data = HAll, fct=LL.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

modelH.W14 <- drm(Survival ~ rate, Population, data = HAll, fct=W1.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

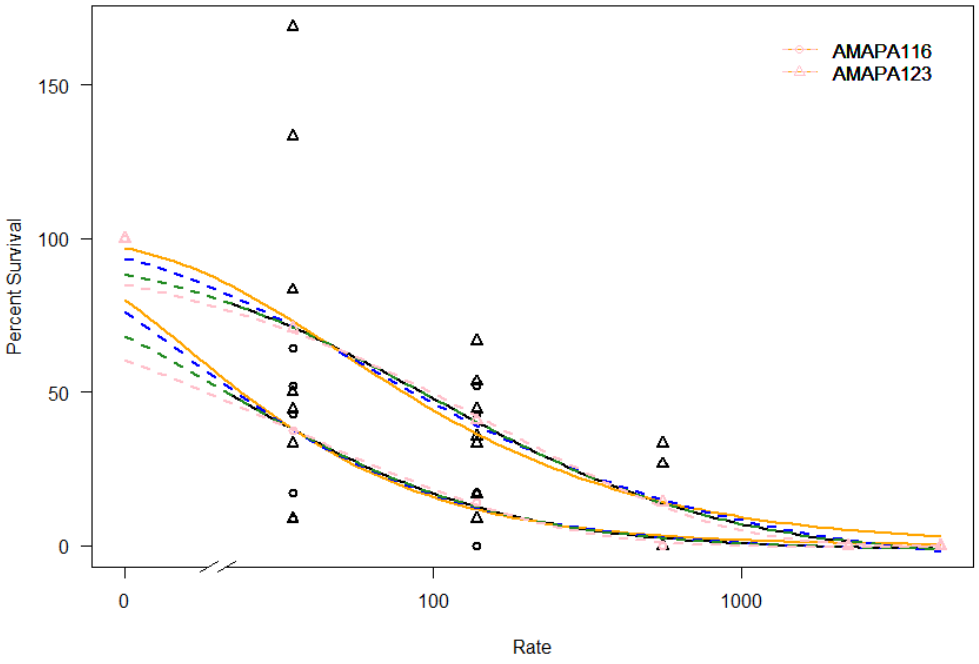
plot(H, broken = TRUE, xlab="Rate", ylab="Percent Survival", type='all',lty=1, lwd=2)

plot(modelH.W23, add=TRUE,col="orange",lty=1, lwd=2)

plot(modelH.W24, add=TRUE,col="blue",lty=2, lwd=2)

plot(modelH.LL4, add=TRUE,col="forestgreen",lty=2, lwd=2)

plot(modelH.W14, add=TRUE,col="pink",lty=2, lwd=2)



Model Averaging – ED Values of models with top 3 highest p-values/lowest AIC + Log Logistic 4P

1. Weibull Type 1 3 Parameter (W1.3())
2. Weibull Type 2 3 Parameter (W2.3())
3. Log Logistic 3 Parameter(LL.3())

maED(H,list(W1.3(),W2.3(),LL.3(fixed=c(NA, 100, NA)), W1.4()),c(50, 90),interval="kang")

AMAPA116 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	22.20782	203.9445	0.01739647
W1.3	18.65138	178.0874	0.13392471
W2.3	23.92373	167.3249	0.06933660
LL.3	22.31609	170.1661	0.76112531
W1.4	18.66146	184.1431	0.01821691

AMAPA123 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	101.77527	1148.2715	0.01739647
W1.3	95.68311	660.2480	0.13392471
W2.3	84.49488	937.5073	0.06933660
LL.3	89.46048	775.2203	0.76112531
W1.4	96.30157	669.0944	0.01821691

Averaged LD50 and LD90 Values across models

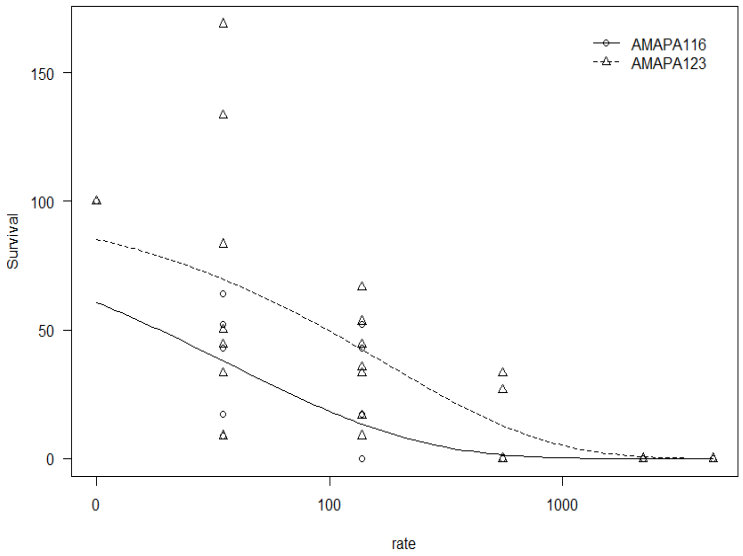
	Estimate	Lower	Upper
e:AMAPA116:50	21.8683	3.599932	40.13667
e:AMAPA116:90	171.8722	-26.170713	369.91504
e:AMAPA123:50	90.2884	42.168041	138.40876
e:AMAPA123:90	775.6316	54.465388	1496.79772

Harness (Acetochlor) Dose Response – Weibull Type 1 3 Parameter Model

Model type	Model function (*f*)	Function in *drc*
Weibull I	$c+(d-c)\exp(-\exp(b(\log(x)-\log(e))))$	`weibull1()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = LD50

H2<- drm(Survival ~ rate, Population, data = HAll, fct=W1.3())
plot(H2, type="all")



Summary of Parameters

summary(H2)

```
Model fitted: Weibull (type 1) with lower limit at 0 (3 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116   0.53207    0.21360  2.4910 0.0148635 *
b:AMAPA123   0.62154    0.17120  3.6304 0.0005039 ***
d:AMAPA116  99.85886   14.48997  6.8916 1.266e-09 ***
d:AMAPA123 101.01948   13.81747  7.3110 2.006e-10 ***
e:AMAPA116  37.14241   16.53779  2.2459 0.0275396 *
e:AMAPA123 172.55731   60.29383  2.8619 0.0054034 **
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 20.47447 (78 degrees of freedom)
```

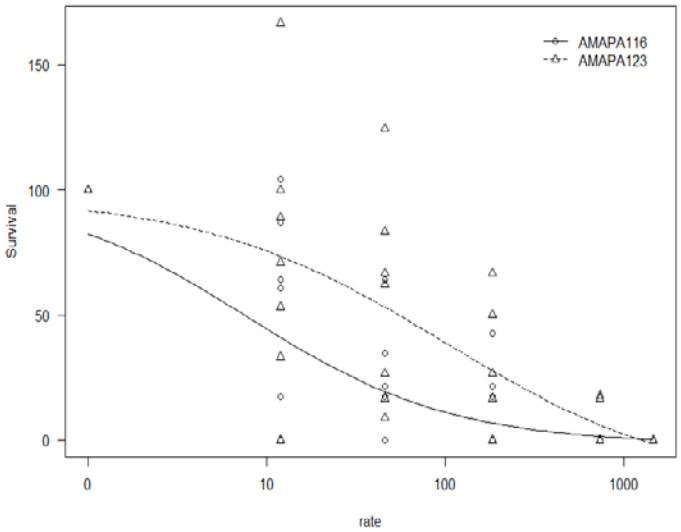
Estimated ED/LD 50 and 90

ED(H2, c(50, 90), interval="delta")

```
Estimated effective doses

                Estimate Std. Error     Lower     Upper
e:AMAPA116:50   18.6514    11.9894   -5.2176   42.5203
e:AMAPA116:90  178.0874    94.0841   -9.2198  365.3945
e:AMAPA123:50   95.6831    41.4941   13.0748  178.2914
e:AMAPA123:90  660.2480   271.1203  120.4891 1200.0070
```

Outlook (Dimethenamid-p) Dose Response – 4 Parameter Log-Logistic Model

O <- drm(Survival ~ rate, Population, data = OAll, fct=LL.4())
plot(O, type="all")



Summary of Parameters

summary(O)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116   0.75771    0.59225  1.2794    0.2047
b:AMAPA123   0.62967    0.38562  1.6329    0.1066
c:AMAPA116  -1.64067   11.53155 -0.1423    0.8872
c:AMAPA123 -20.13731   35.86814 -0.5614    0.5762
d:AMAPA116 100.17895   18.14025  5.5225 4.474e-07 ***
d:AMAPA123  97.87896   19.01081  5.1486 2.001e-06 ***
e:AMAPA116   7.81257    5.63700  1.3859    0.1698
e:AMAPA123 101.57954  115.48260  0.8796    0.3818
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Residual standard error:

 25.74005 (76 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(O, c(50, 90), interval="delta")

```
Estimated effective doses

                 Estimate  Std. Error       Lower       Upper
e:AMAPA116:50      7.8126      5.6370     -3.4145     19.0396
e:AMAPA116:90    141.9589    290.2536   -436.1313    720.0491
e:AMAPA123:50    101.5795    115.4826   -128.4240    331.5831
e:AMAPA123:90   3328.7562   9830.6563 -16250.6927  22908.2052
```

Comparison of 4 Parameter Log Logistic Models to Other Models (Weibull Type 1 &2, Log-Logistic 3P, Cubic, Quadratic and Linear)

mselect(O, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

```
          logLik       IC Lack of fit   Res var
W1.3  -387.8843 785.7686   0.9995632  630.3363
W2.3  -389.1755 788.3510   0.9350252  650.0153
LL.3  -388.3012 790.6023   0.9652278  652.9475
W1.4  -387.8199 793.6398   0.9679199  662.4952
LL.4  -387.8234 793.6468   0.9672800  662.5502
W2.4  -387.9116 793.8232   0.9495289  663.9435
Cubic -400.7553 811.5106          NA  856.3725
Quad  -404.9620 817.9241          NA  934.9041
Lin   -410.8679 827.7358          NA 1062.9358
```

Comparison of Plotted Curves

modelO.W23 <- drm(Survival ~ rate, Population, data = OAll, fct=W2.3(fixed=c(NA, 100, NA), names = c("Slope", "Upper Limit", "ED50")))

modelO.W24 <- drm(Survival ~ rate, Population, data = OAll, fct=W2.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

modelO.LL4 <- drm(Survival ~ rate, Population, data = OAll, fct=LL.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

modelO.W14 <- drm(Survival ~ rate, Population, data = OAll, fct=W1.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

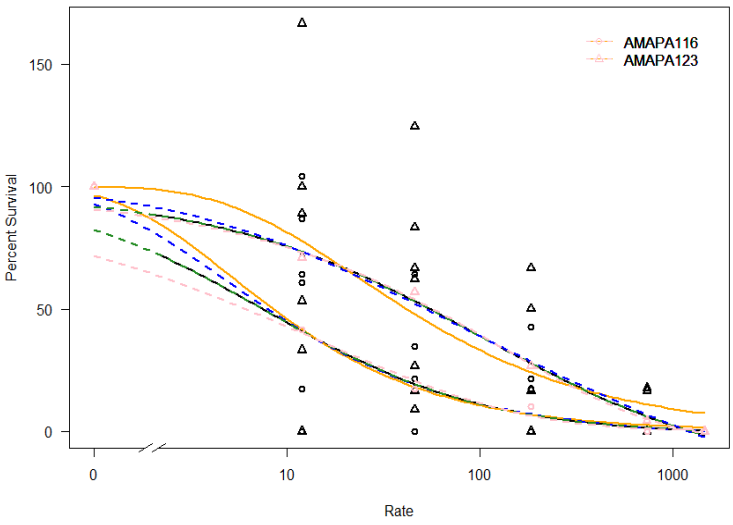
plot(O, broken = TRUE, xlab="Rate", ylab="Percent Survival", type='all',lty=1, lwd=2)

plot(modelO.W23, add=TRUE,col="orange",lty=1, lwd=2)

plot(modelO.W24, add=TRUE,col="blue",lty=2, lwd=2)

plot(modelO.LL4, add=TRUE,col="forestgreen",lty=2, lwd=2)

plot(modelO.W14, add=TRUE,col="pink",lty=2, lwd=2)



Model Averaging – ED Values of models with top 3 highest p-values/lowest AIC + Log Logistic 4P

1. Weibull Type 1 3 Parameter (W1.3())
2. Weibull Type 2 3 Parameter (W2.3())
3. Log Logistic 3 Parameter(LL.3())

maED(O,list(W1.3(),W2.3(),LL.3(fixed=c(NA, 100, NA)), W1.4()),c(50, 90),interval="kang")

AMAPA116 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	7.812570	141.9589	0.02450258
W1.3	5.940655	120.1983	0.17398706
W2.3	8.514028	110.3779	0.05998428
LL.3	7.902726	110.6792	0.71693787
W1.4	5.962490	114.8122	0.02458821

AMAPA123 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	101.57954	3328.7562	0.02450258
W1.3	62.93370	505.9688	0.17398706
W2.3	92.34776	689.5607	0.05998428
LL.3	49.40126	650.3617	0.71693787
W1.4	67.30955	655.4346	0.02458821

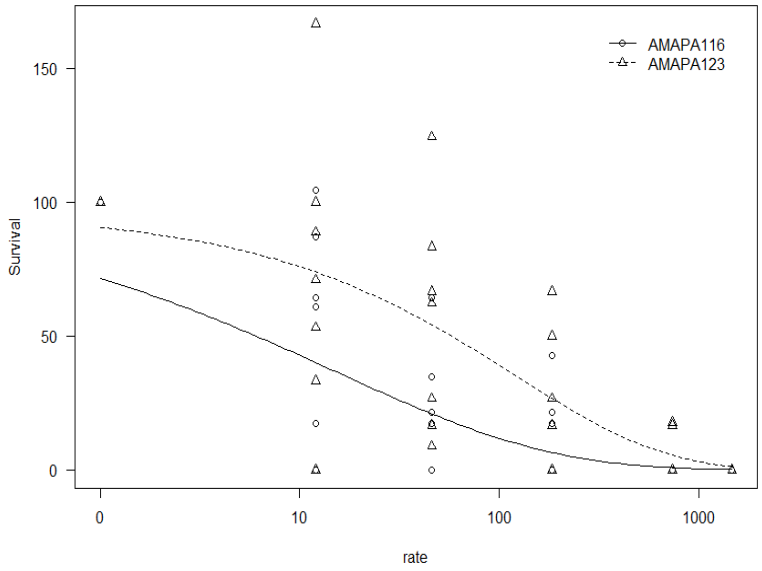
Averaged LD50 and LD90 Values across models

	Estimate	Lower	Upper
e:AMAPA116:50	7.548103	-1.769787	16.86599
e:AMAPA116:90	113.185369	-86.043279	312.41402
e:AMAPA123:50	56.050682	8.268504	103.83286
e:AMAPA123:90	693.342867	-509.279627	1895.96536

Outlook (Dimethenamid-P) Dose Response – Weibull Type 1 3 Parameter Model

Model type	Model function (*f*)	Function in *drc*
Weibull I	$c+(d-c)\exp(-\exp(b(\log(x)-\log(e))))$	`weibull1()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = mid-point/LD50
O2<- drm(Survival ~ rate, Population, data = OAll, fct=W1.3())



Summary of Parameters

summary(O2)

```
Model fitted: Weibull (type 1) with lower limit at 0 (3 parms)

Parameter estimates:

             Estimate Std. Error t-value   p-value
b:AMAPA116   0.39921    0.19064  2.0941  0.039502 *
b:AMAPA123   0.57597    0.20833  2.7647  0.007108 **
d:AMAPA116 100.43020   17.85532  5.6247 2.788e-07 ***
d:AMAPA123  96.52499   17.57639  5.4917 4.808e-07 ***
e:AMAPA116  14.87856   10.04243  1.4816  0.142484
e:AMAPA123 118.91673   59.33970  2.0040  0.048542 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 25.41995 (78 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(O2, c(50, 90), interval="delta")

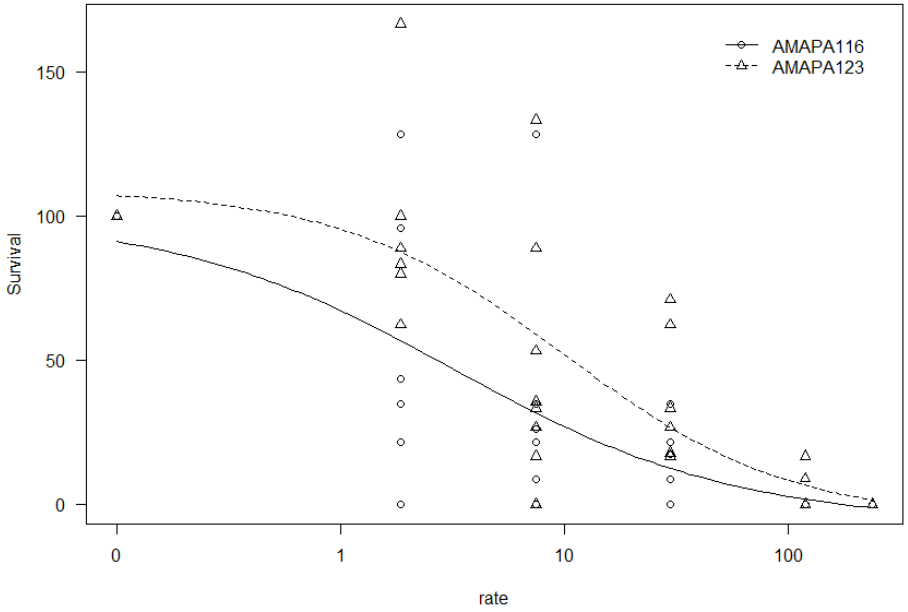
```
Estimated effective doses

               Estimate Std. Error     Lower     Upper
e:AMAPA116:50    5.9407     5.8746   -5.7547   17.6360
e:AMAPA116:90  120.1983   100.5514  -79.9843  320.3808
e:AMAPA123:50   62.9337    41.0393  -18.7693  144.6367
e:AMAPA123:90  505.9688   249.3391    9.5729 1002.3647
```

Zidua (Pyroxasulfone) Dose Response – 4 Parameter Log-Logistic Model

Model type	Model function (*f*)	Function in *drc*
Generalized log-logistic	$c + \frac{d-c}{(1+\exp(b(\log(x)-\log(e))))^f}$	`llogistic()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = mid-point/LD50

```
Z <- drm(Survival ~ rate, Population, data = ZAll, fct=LL.4())
plot(Z, type="all")
```



Summary of Parameters

summary(Z)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116   0.69366    0.40476  1.7138   0.09065 .
b:AMAPA123   0.86584    0.37727  2.2950   0.02449 *
c:AMAPA116  -6.21857   15.94864 -0.3899   0.69769
c:AMAPA123  -5.33659   18.03926 -0.2958   0.76817
d:AMAPA116  99.92169   19.65227  5.0845 2.575e-06 ***
d:AMAPA123 109.16869   16.19362  6.7415 2.684e-09 ***
e:AMAPA116   3.18790    2.49121  1.2797   0.20456
e:AMAPA123   9.94097    6.88271  1.4443   0.15275
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Residual standard error:

 27.82816 (76 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(Z, c(50, 90), interval="delta")

```
Estimated effective doses

                Estimate Std. Error     Lower     Upper
e:AMAPA116:50    3.1879     2.4912   -1.7738    8.1496
e:AMAPA116:90   75.7114   159.9444 -242.8456  394.2683
e:AMAPA123:50    9.9410     6.8827   -3.7671   23.6491
e:AMAPA123:90  125.7542   193.1900 -259.0171  510.5255
```

Comparison of 4 Parameter Log Logistic Models to Other Models (Weibull Type 1 &2, Log-Logistic 3P, Cubic, Quadratic and Linear)

mselect(Z, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

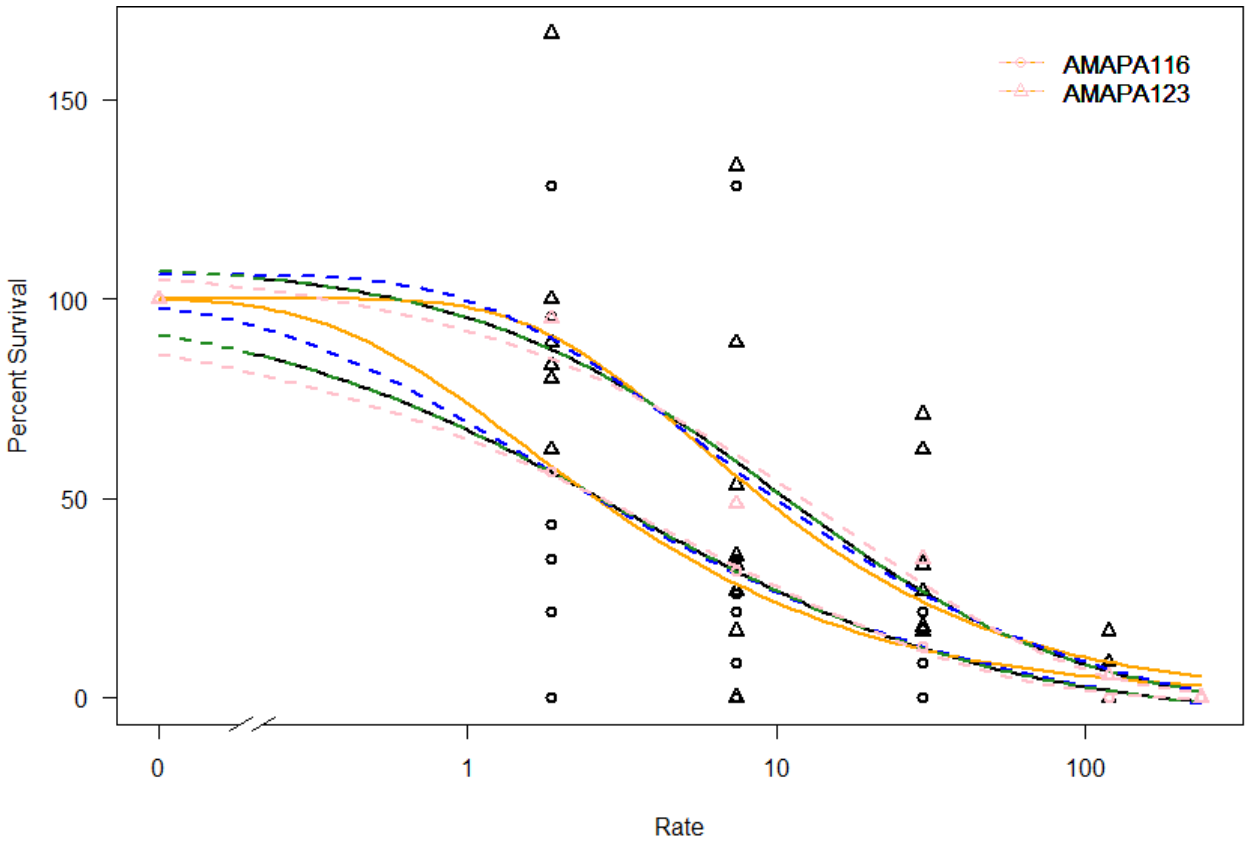
```
         logLik       IC Lack of fit   Res var
W2.3  -394.3895 798.7789   0.9463988  735.9323
W1.3  -394.9377 799.8753   0.8767452  745.6011
LL.3  -394.5571 803.1142   0.8017381  757.8209
W2.4  -393.9545 805.9089   0.7415602  766.6841
LL.4  -394.3754 806.7508   0.6092198  774.4067
W1.4  -394.7247 807.4494   0.5078296  780.8739
Cubic -403.9998 817.9997           NA  925.1509
Quad  -409.1246 826.2492           NA 1032.3078
Lin   -416.4980 838.9961           NA 1215.4144
```

Comparison of Plotted Curves

modelZ.W23 <- drm(Survival ~ rate, Population, data = ZAll, fct=W2.3(fixed=c(NA, 100, NA), names = c("Slope", "Upper Limit", "ED50")))

modelZ.W24 <- drm(Survival ~ rate, Population, data = ZAll, fct=W2.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

```
modelZ.LL4 <-  drm(Survival ~ rate, Population, data = ZAll, fct=LL.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

modelZ.W14 <-  drm(Survival ~ rate, Population, data = ZAll, fct=W1.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))


plot(Z, broken = TRUE, xlab="Rate", ylab="Percent Survival", type='all',lty=1, lwd=2)

plot(modelZ.W23, add=TRUE,col="orange",lty=1, lwd=2)

plot(modelZ.W24, add=TRUE,col="blue",lty=2, lwd=2)

plot(modelZ.LL4, add=TRUE,col="forestgreen",lty=2, lwd=2)

plot(modelZ.W14, add=TRUE,col="pink",lty=2, lwd=2)
```



Model Averaging – ED Values of models with top 3 highest p-values/lowest AIC + Log Logistic 4P

1. Weibull Type 2 3 Parameter (W2.3())
2. Weibull Type 1 3 Parameter (W1.3())
3. Log Logistic 3 Parameter(LL.3())

maED(Z,list(W1.3(),W2.3(),LL.3(fixed=c(NA, 100, NA)), W1.4()),c(50, 90),interval="kang")

AMAPA116 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	3.187897	75.71136	0.01893476
W1.3	2.723570	34.14825	0.09804088
W2.3	2.634104	40.16768	0.14451250
LL.3	2.703851	36.90673	0.72515935
W1.4	2.776318	37.11508	0.01335251

AMAPA123 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	9.940968	125.75423	0.01893476
W1.3	9.820837	73.88042	0.09804088
W2.3	8.227844	99.66169	0.14451250
LL.3	10.610803	87.92826	0.72515935
W1.4	9.720589	72.16007	0.01335251

Averaged LD50 and LD90 Values across models

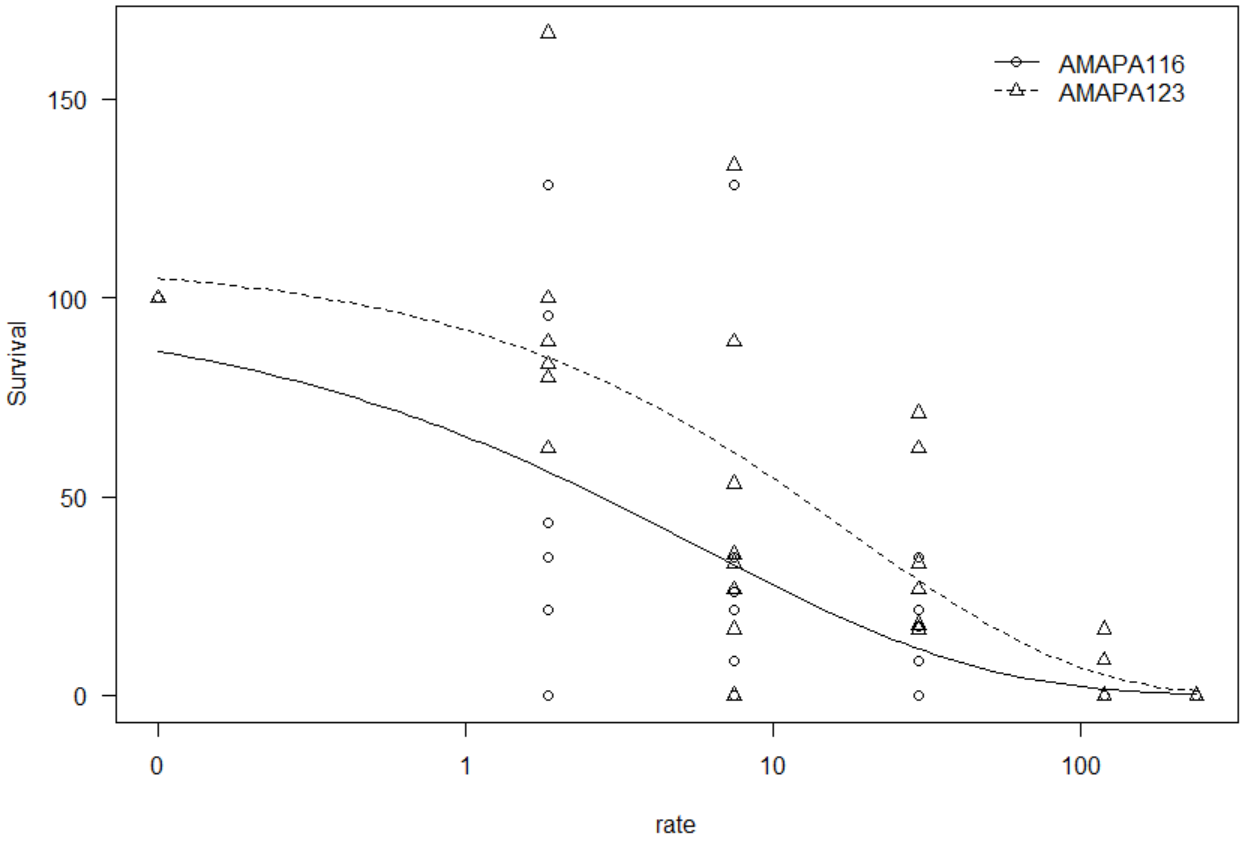
	Estimate	Lower	Upper
e:AMAPA116:50	2.705838	0.2804517	5.131223
e:AMAPA116:90	37.845073	-22.3618552	98.052000
e:AMAPA123:50	10.164417	3.4848494	16.843985
e:AMAPA123:90	88.752308	-17.6211154	195.125731

Zidua (Pyroxasulfone) Dose Response – Weibull Type 1 3 Parameter Model

Model type	Model function (*f*)	Function in *drc*
Weibull I	$c+(d-c)\exp(-\exp(b(\log(x)-\log(e))))$	`weibull1()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = LD50

Z2<- drm(Survival ~ rate, Population, data = ZAll, fct=W1.3())
plot(Z2, type = "all")



Summary of Parameters

summary(Z2)

```
Model fitted: Weibull (type 1) with lower limit at 0 (3 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116   0.47476    0.18027  2.6336 0.0101827 *
b:AMAPA123   0.59494    0.16882  3.5242 0.0007142 ***
d:AMAPA116 100.10491   19.29323  5.1886 1.634e-06 ***
d:AMAPA123 109.84689   16.36116  6.7139 2.745e-09 ***
e:AMAPA116   5.89408    3.46892  1.6991 0.0932839 .
e:AMAPA123  18.18435    7.88126  2.3073 0.0236918 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 27.58561 (78 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(Z2, c(50, 90), interval="delta")

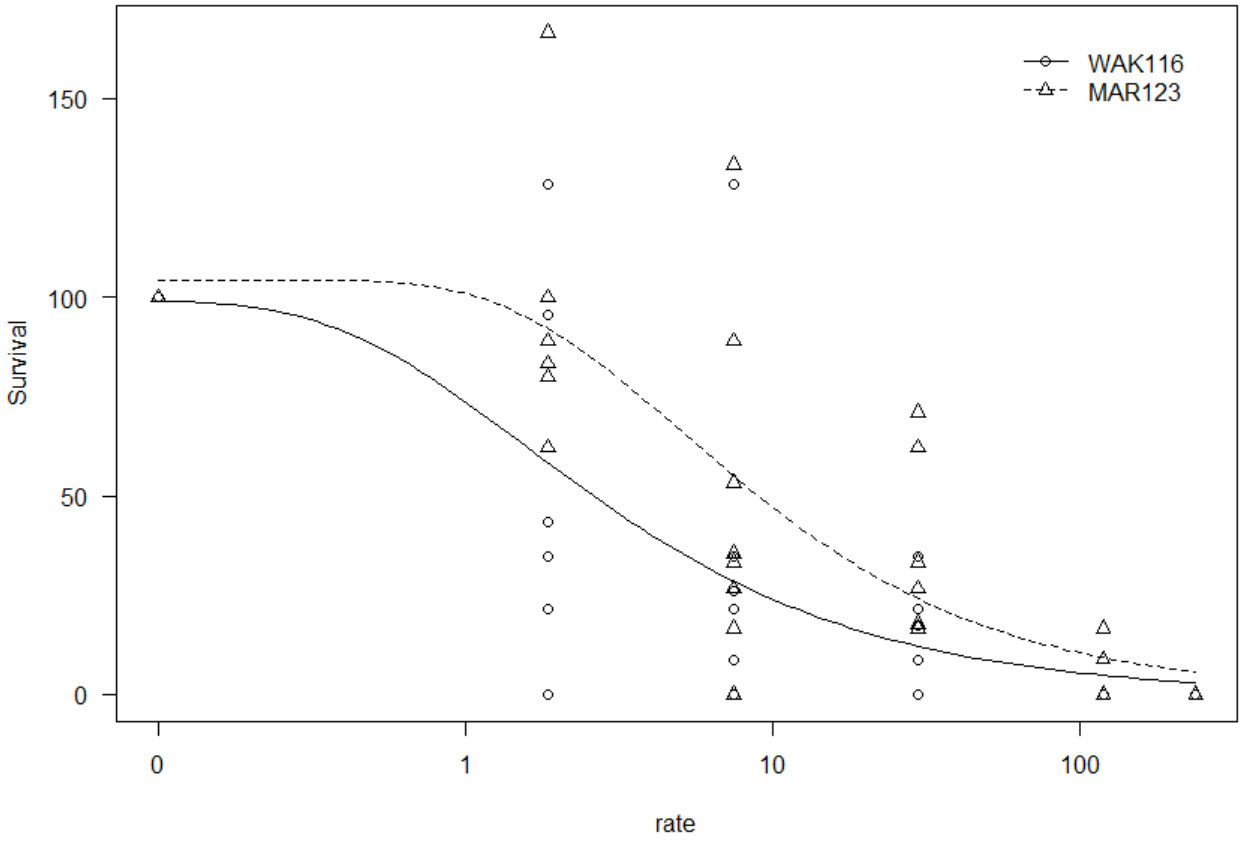
```
Estimated effective doses

                Estimate Std. Error     Lower     Upper
e:AMAPA116:50   2.72357    2.06067  -1.37890   6.82604
e:AMAPA116:90  34.14825   23.47140 -12.57971  80.87621
e:AMAPA123:50   9.82084    5.05828  -0.24942  19.89109
e:AMAPA123:90  73.88042   36.14876   1.91377 145.84708
```

Zidua (Pyroxasulfone) Dose Response – Weibull Type 2 3 Parameter Model

Model type	Model function (*f*)	Function in *drc*
Weibull II	$c+(d-c)(1-\exp(-\exp(b(\log(x)-\log(e)))))$	`weibull2()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = LD50

Z3<- drm(Survival ~ rate, Population, data = ZAll, fct=W2.3())
Plot(Z3, type = "all")



Summary of Parameters

summary(Z3)

```
Model fitted: Weibull (type 2) with lower limit at 0 (3 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116  -0.69144    0.21035 -3.2871  0.001519 **
b:AMAPA123  -0.75528    0.18222 -4.1448 8.568e-05 ***
d:AMAPA116  99.20141   19.63611  5.0520 2.809e-06 ***
d:AMAPA123 104.42413   15.71372  6.6454 3.695e-09 ***
e:AMAPA116   1.55034    0.95526  1.6230  0.108635
e:AMAPA123   5.06449    2.46612  2.0536  0.043363 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 27.45849 (78 degrees of freedom)
```

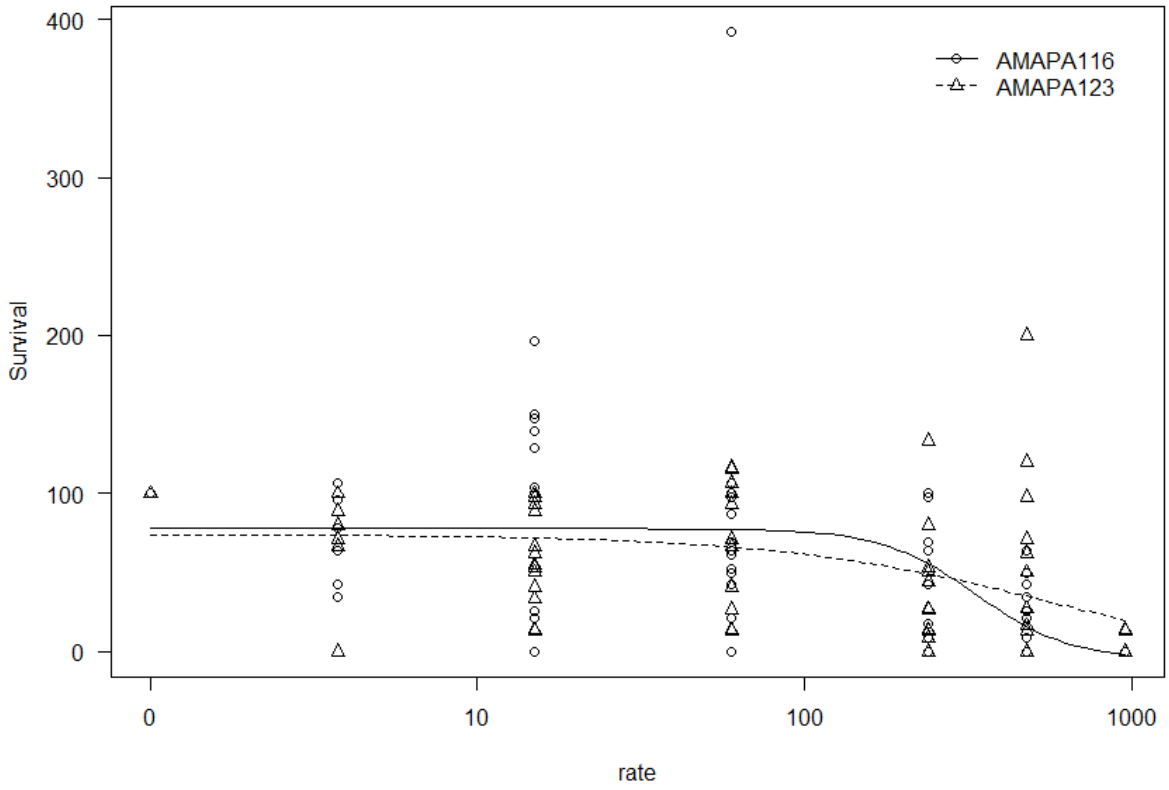
Estimated ED/LD 50 and 90

ED(Z3, c(50, 90), interval="delta")

```
Estimated effective doses

               Estimate Std. Error     Lower     Upper
e:AMAPA116:50   2.63410    1.43564  -0.22404   5.49225
e:AMAPA116:90  40.16768   33.44129 -26.40881 106.74417
e:AMAPA123:50   8.22784    3.62069   1.01961  15.43608
e:AMAPA123:90  99.66169   63.25407 -26.26749 225.59087
```

Sunfire (Flufenacet) Dose Response – 4 Parameter Log-Logistic Model

S<- drm(Survival ~ rate, Population, data = SAll, fct=LL.4())
plot(S, type="all")



Summary of Parameters

summary(S)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

             Estimate Std. Error t-value   p-value
b:AMAPA116    3.01917    2.34346  1.2883  0.199499
b:AMAPA123    0.99190    0.88701  1.1183  0.265144
c:AMAPA116   -4.98400   26.28913 -0.1896  0.849877
c:AMAPA123  -16.10849   77.21835 -0.2086  0.835020
d:AMAPA116   77.81985    7.21049 10.7926 < 2.2e-16 ***
d:AMAPA123   74.09433   12.97792  5.7093 5.424e-08 ***
e:AMAPA116  334.87186  116.24413  2.8808  0.004515 **
e:AMAPA123  634.79519 1036.46822  0.6125  0.541108
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Residual standard error:

 46.58189 (159 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(S, c(50, 90), interval="delta")

```
Estimated effective doses

               Estimate Std. Error     Lower     Upper
e:AMAPA116:50    334.87     116.24    105.29    564.45
e:AMAPA116:90    693.33     571.48   -435.34   1822.00
e:AMAPA123:50    634.80    1036.47  -1412.23   2681.82
e:AMAPA123:90   5816.59   15922.45 -25630.20  37263.37
```

Comparison of 4 Parameter Log Logistic Models to Other Models (Weibull Type 1 &2, Log-Logistic 3P, Cubic, Quadratic and Linear)

mselect(Z, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

```
        logLik       IC Lack of fit   Res var
W2.3  -394.3895 798.7789   0.9463988  735.9323
W1.3  -394.9377 799.8753   0.8767452  745.6011
LL.3  -394.5571 803.1142   0.8017381  757.8209
W2.4  -393.9545 805.9089   0.7415602  766.6841
LL.4  -394.3754 806.7508   0.6092198  774.4067
W1.4  -394.7247 807.4494   0.5078296  780.8739
Cubic -403.9998 817.9997           NA  925.1509
Quad  -409.1246 826.2492           NA 1032.3078
Lin   -416.4980 838.9961           NA 1215.4144
```

Comparison of Plotted Curves

modelS.W23 <- drm(Survival ~ rate, Population, data =SAll, fct=W2.3(fixed=c(NA, 100, NA), names = c("Slope", "Upper Limit", "ED50")))

modelS.W24 <- drm(Survival ~ rate, Population, data = SAll, fct=W2.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

modelS.LL4 <- drm(Survival ~ rate, Population, data = SAll, fct=LL.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

modelS.W14 <- drm(Survival ~ rate, Population, data = SAll, fct=W1.4(names = c("Slope", "Lower Limit", "Upper Limit", "ED50")))

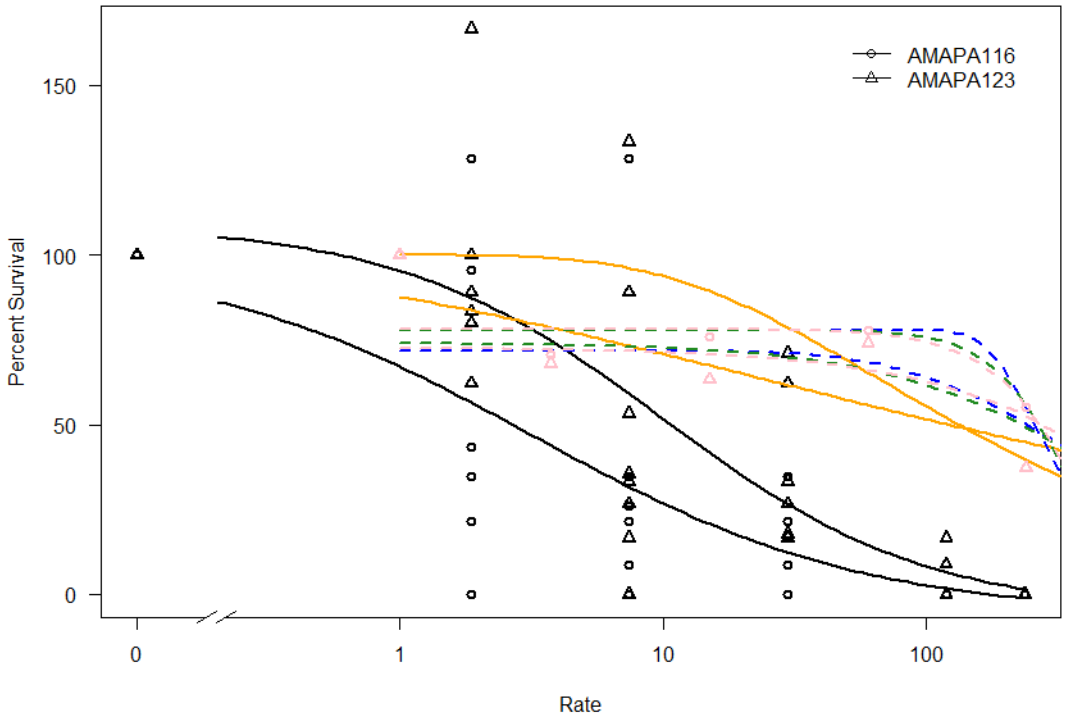
plot(Z, broken = TRUE, xlab="Rate", ylab="Percent Survival", type='all',lty=1, lwd=2)

plot(modelS.W23, add=TRUE,col="orange",lty=1, lwd=2)

plot(modelS.W24, add=TRUE,col="blue",lty=2, lwd=2)

plot(modelS.LL4, add=TRUE,col="forestgreen",lty=2, lwd=2)

plot(modelS.W14, add=TRUE,col="pink",lty=2, lwd=2)



Model Averaging – ED Values of models with top 3 highest p-values/lowest AIC + Log Logistic 4P

1. Weibull Type 2 3 Parameter (W2.3())
2. Weibull Type 1 3 Parameter (W1.3())
3. Log Logistic 3 Parameter(LL.3())

maED(S,list(W1.3(),W2.3(),LL.3(fixed=c(NA, 100, NA)), W1.4()),c(50, 90),interval="kang")

AMAPA116 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	334.8719	693.3291	NaN
W1.3	327.2962	561.9092	NaN
W2.3	301.7364	632.9868	NaN
LL.3	155.8749	1846.8678	NaN
W1.4	329.2095	565.8882	NaN

AMAPA123 LD50 and LD90 Values

	ED50	ED90	Weight
LL.4	634.7952	5816.586	NaN
W1.3	461.6296	1951.317	NaN
W2.3	206.7498	312516.736	NaN
LL.3	134.8202	55133.005	NaN
W1.4	2572.0646	10646.513	NaN

Averaged LD50 and LD90 Values across models

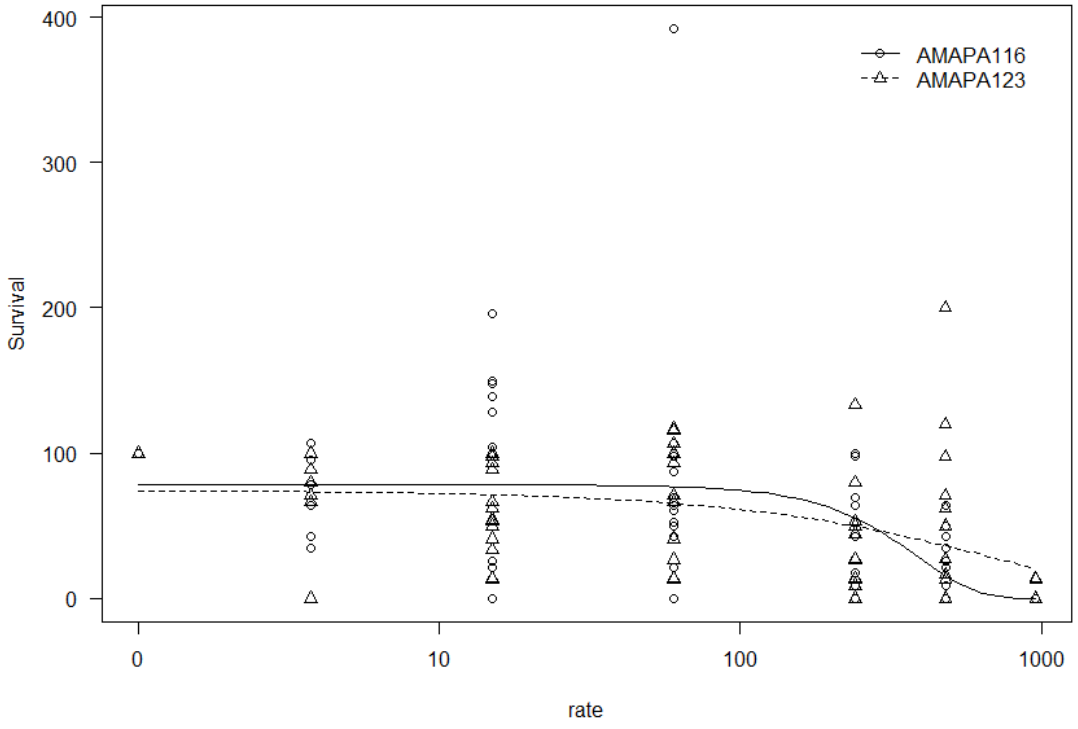
	Estimate	Lower	Upper
e:AMAPA116:50	NaN	NaN	NaN
e:AMAPA116:90	NaN	NaN	NaN
e:AMAPA123:50	NaN	NaN	NaN
e:AMAPA123:90	NaN	NaN	NaN

Sunfire (Flufenacet) Dose Response – Weibull Type 1 3 Parameter Model

Model type	Model function (*f*)	Function in *drc*
Weibull I	$c+(d-c)\exp(-\exp(b(\log(x)-\log(e))))$	`weibull1()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = LD50

S2<- drm(Survival ~ rate, Population, data = SAll, fct=W1.3())



Summary of Parameters

summary(S2)

```
Model fitted: Weibull (type 1) with lower limit at 0 (3 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116   2.22128    1.14952  1.9324   0.05507 .
b:AMAPA123   0.83285    0.86694  0.9607   0.33816
d:AMAPA116  78.26324    7.39151 10.5883 < 2.2e-16 ***
d:AMAPA123  74.36522   14.92439  4.9828 1.604e-06 ***
e:AMAPA116 386.01120   67.86141  5.6882 5.907e-08 ***
e:AMAPA123 716.82777  315.90335  2.2691   0.02459 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 46.27688 (161 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(S2, c(50, 90), interval="delta")

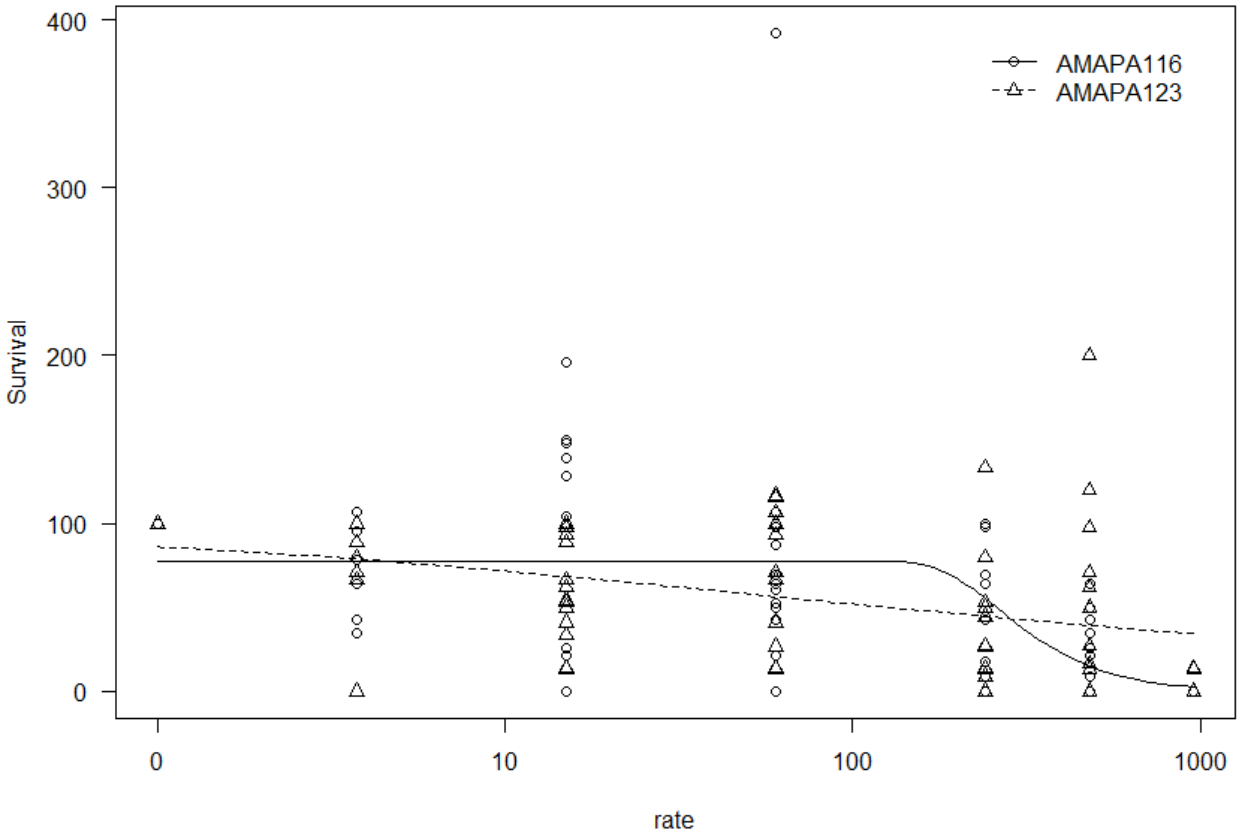
```
Estimated effective doses

               Estimate Std. Error     Lower     Upper
e:AMAPA116:50   327.296     64.404   200.111   454.481
e:AMAPA116:90   561.909    145.958   273.670   850.148
e:AMAPA123:50   461.630    261.908   -55.588   978.847
e:AMAPA123:90  1951.317   2364.109 -2717.344  6619.979
```

Sunfire (Flufenacet) Dose Response – Weibull Type 2 3 Parameter Model

Model type	Model function (*f*)	Function in *drc*
Weibull II	$c+(d-c)(1-\exp(-\exp(b(\log(x)-\log(e)))))$	`weibull2()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = LD50

S3<- drm(Survival ~ rate, Population, data = SAll, fct=W2.3())



Summary of Parameters

summary(S3)

```
Model fitted: Weibull (type 2) with lower limit at 0 (3 parms)

Parameter estimates:

            Estimate Std. Error t-value   p-value
b:AMAPA116  -2.54267    1.30498 -1.9484   0.05310 .
b:AMAPA123  -0.25733    0.20375 -1.2630   0.20843
d:AMAPA116  77.80497    7.00749 11.1031 < 2.2e-16 ***
d:AMAPA123  92.00522   37.12109  2.4785   0.01422 *
e:AMAPA116 261.23199   42.84448  6.0972 7.713e-09 ***
e:AMAPA123  49.75906  167.17520  0.2976   0.76636
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 46.46821 (161 degrees of freedom)
```

Estimated ED/LD 50 and 90

ED(S3, c(50, 90), interval="delta")

```
Estimated effective doses

                 Estimate  Std. Error       Lower       Upper
e:AMAPA116:50  3.0174e+02  5.0235e+01  2.0253e+02  4.0094e+02
e:AMAPA116:90  6.3299e+02  2.8635e+02  6.7498e+01  1.1985e+03
e:AMAPA123:50  2.0675e+02  5.0352e+02 -7.8762e+02  1.2011e+03
e:AMAPA123:90  3.1252e+05  1.3456e+06 -2.3448e+06  2.9698e+06
```

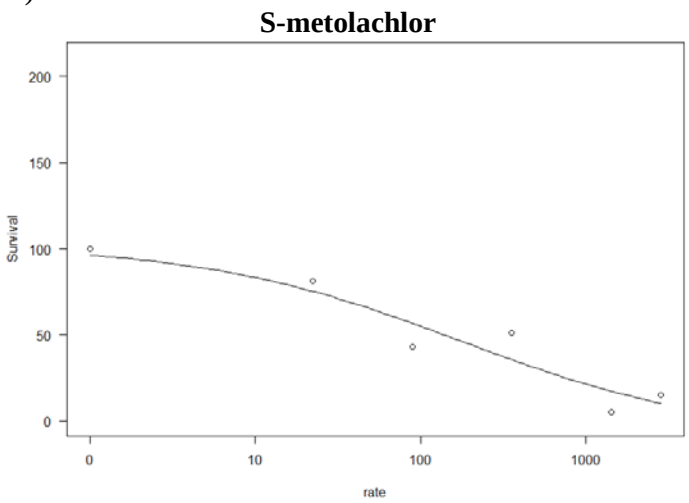
Group 15 Herbicide Redroot Dose Response – 4 Parameter Log-Logistic Model

Model type	Model function (*f*)	Function in *drc*
Generalized log-logistic	$c + \frac{d-c}{(1+\exp(b(\log(x)-\log(e))))^f}$	`llogistic()`

b = slope/steepness of dose-response curve
c = lower asymptotes/limits
d = upper asymptotes/limits
e = mid-point/LD50

Dual Magnum (S-metolachlor) Redroot Dose Response – 4 Parameter Log-Logistic Model

DMRR <- drm(Survival ~ rate, Population, data = DMRR, fct=LL.4())
> plot(DMRR, type="all")



> summary(DMRR)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

              Estimate Std. Error t-value  p-value
b:(Intercept)   0.55744    0.43165  1.2914 0.204353
c:(Intercept)  -8.63818   51.12652 -0.1690 0.866727
d:(Intercept) 102.21839   24.05765  4.2489 0.000134 ***
e:(Intercept) 170.27000  372.60770  0.4570 0.650295
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Residual standard error:

 36.04481 (38 degrees of freedom)
```

ED(DMRR, c(50, 90), interval="delta")

```
Estimated effective doses

               Estimate Std. Error     Lower     Upper
e:AMARE15:50     170.27     372.61   -584.03    924.57
e:AMARE15:90    8769.13   41938.32 -76130.56  93668.81
```

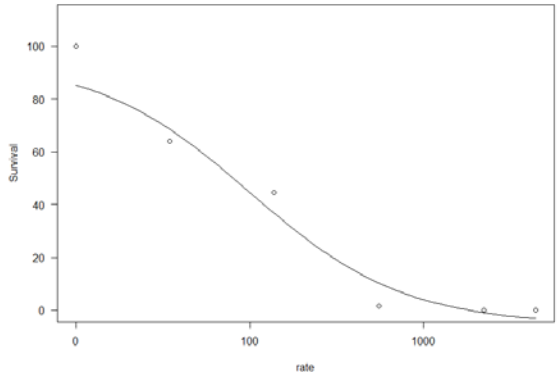
mselect(DMRR, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

```
         logLik       IC Lack of fit  Res var
Quad  -208.7095 425.4189          NA 1306.068
LL.4  -208.0537 426.1074   0.1435675 1299.228
Cubic -208.6527 427.3054          NA 1336.819
Lin   -212.0797 430.1594          NA 1495.099
W1.3          NA       NA          NA       NA
W1.4          NA       NA          NA       NA
W2.3          NA       NA          NA       NA
W2.4          NA       NA          NA       NA
LL.3          NA       NA          NA       NA
```

Harness (Acetochlor) Redroot Dose Response – 4 Parameter Log-Logistic Model

HRR <- drm(Survival ~ rate, Population, data = HRR, fct=LL.4())
> plot(HRR, type="all")

Acetochlor



summary(HRR)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

             Estimate Std. Error t-value   p-value
b:(Intercept)  0.98376    0.35555  2.7669  0.008694 **
c:(Intercept) -5.50450    6.95464 -0.7915  0.433573
d:(Intercept) 94.61203   16.04327  5.8973 7.875e-07 ***
e:(Intercept) 99.81275   46.15391  2.1626  0.036931 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 19.49173 (38 degrees of freedom)
```

ED(HRR, c(50, 90), interval="delta")

```
Estimated effective doses

             Estimate Std. Error    Lower    Upper
e:AMARE15:50   99.813     46.154    6.379  193.246
e:AMARE15:90  931.504    672.780 -430.468 2293.476
```

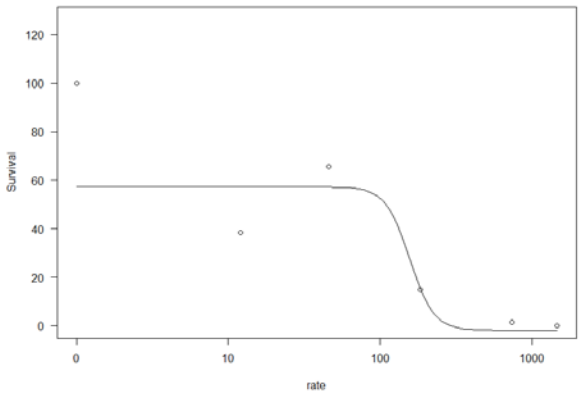
mselect(HRR, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

```
         logLik       IC Lack of fit  Res var
LL.4   -182.2333 374.4665   0.2906496 379.9275
Cubic  -182.6690 375.3381          NA 387.8940
Quad   -194.6496 397.2992          NA 668.6493
Lin    -201.3863 408.7725          NA 898.5071
W1.3          NA       NA          NA       NA
W1.4          NA       NA          NA       NA
W2.3          NA       NA          NA       NA
W2.4          NA       NA          NA       NA
LL.3          NA       NA          NA       NA
```

Outlook (Dimethenamid-P) Redroot Dose Response – 4 Parameter Log-Logistic Model

ORR <- drm(Survival ~ rate, Population, data = ORR, fct=LL.4())
plot(ORR)

Dimethenamid-P



Summary of Parameters

summary(ORR)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

             Estimate Std. Error t-value   p-value
b:(Intercept)   5.4773     9.1408  0.5992  0.552590
c:(Intercept)  -1.9352     7.0146 -0.2759  0.784128
d:(Intercept)  57.2126     6.6298  8.6297 1.736e-10 ***
e:(Intercept) 155.9613    48.4486  3.2191  0.002633 **
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Residual standard error:

 28.06703 (38 degrees of freedom)
```

ED(ORR, c(50, 90), interval="delta")

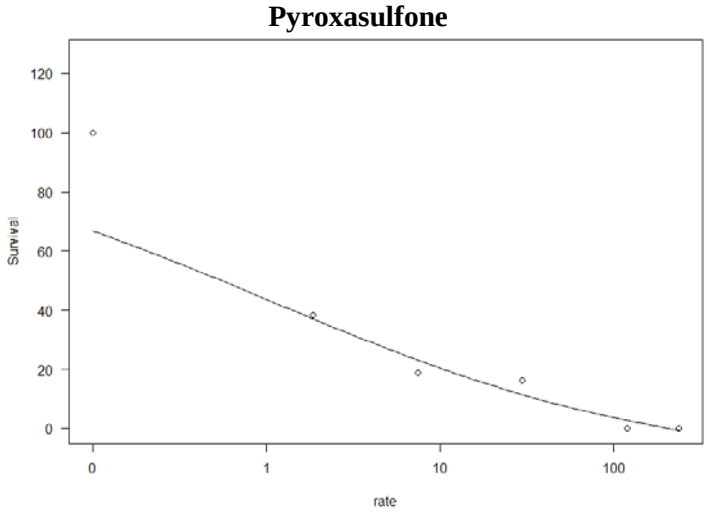
```
Estimated effective doses

             Estimate Std. Error   Lower   Upper
e:AMARE15:50  155.961     48.449  57.882 254.040
e:AMARE15:90  232.936    103.025  24.372 441.499
```

mselect(ORR, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

```
        logLik       IC Lack of fit   Res var
LL.4  -197.5467 405.0934 0.006973376  787.7584
Cubic -198.4074 406.8148          NA  820.7165
Quad  -200.1647 408.3293          NA  869.4685
Lin   -203.7607 413.5213          NA 1006.0644
W1.3          NA       NA          NA       NA
W1.4          NA       NA          NA       NA
W2.3          NA       NA          NA       NA
W2.4          NA       NA          NA       NA
LL.3          NA       NA          NA       NA
```

Zidua SC (Pyroxasulfone) Redroot Dose Response – 4 Parameter Log-Logistic Model

ZRR <- drm(Survival ~ rate, Population, data = ZRR, fct=LL.4())
plot(ZRR)



Summary of Parameters

summary(ZRR)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

              Estimate Std. Error t-value   p-value
b:(Intercept)   0.37479    0.43892  0.8539    0.3985
c:(Intercept) -13.78618   41.93968 -0.3287    0.7442
d:(Intercept) 100.15918   16.01272  6.2550 2.545e-07 ***
e:(Intercept)   1.03949    1.64164  0.6332    0.5304
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Residual standard error:

 22.71341 (38 degrees of freedom)
```

ED(ZRR, c(50, 90), interval="delta")

```
Estimated effective doses

               Estimate Std. Error      Lower      Upper
e:AMARE15:50     1.0395     1.6416    -2.2838     4.3628
e:AMARE15:90   365.4789  2894.9841 -5495.1101  6226.0679
```

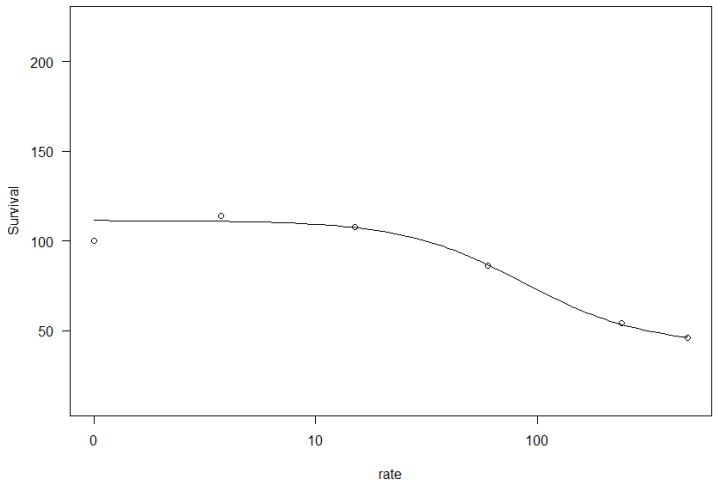
mselect(ZRR, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

```
          logLik       IC Lack of fit  Res var
LL.4   -188.6578 387.3156   0.8666062 515.8988
Cubic  -195.7701 401.5401           NA 723.8542
Quad   -197.0298 402.0597           NA 748.8988
Lin    -199.2550 404.5100           NA 811.7944
W1.3          NA       NA           NA       NA
W1.4          NA       NA           NA       NA
W2.3          NA       NA           NA       NA
W2.4          NA       NA           NA       NA
LL.3          NA       NA           NA       NA
```

Sunfire (Flufenacet) Redroot Dose Response – 4 Parameter Log-Logistic Model

SRR <- drm(Survival ~ rate, Population, data = SRR, fct=LL.4())
plot(SRR)

Flufenacet



summary(SRR)

```
Model fitted: Log-logistic (ED50 as parameter) (4 parms)

Parameter estimates:

             Estimate Std. Error t-value   p-value
b:(Intercept)   1.5910     2.4303  0.6547    0.5166
c:(Intercept)  42.0341    36.7968  1.1423    0.2605
d:(Intercept) 111.4244    14.5742  7.6453 3.351e-09 ***
e:(Intercept)  86.3920   101.6850  0.8496    0.4009
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error:

 47.69989 (38 degrees of freedom)
```

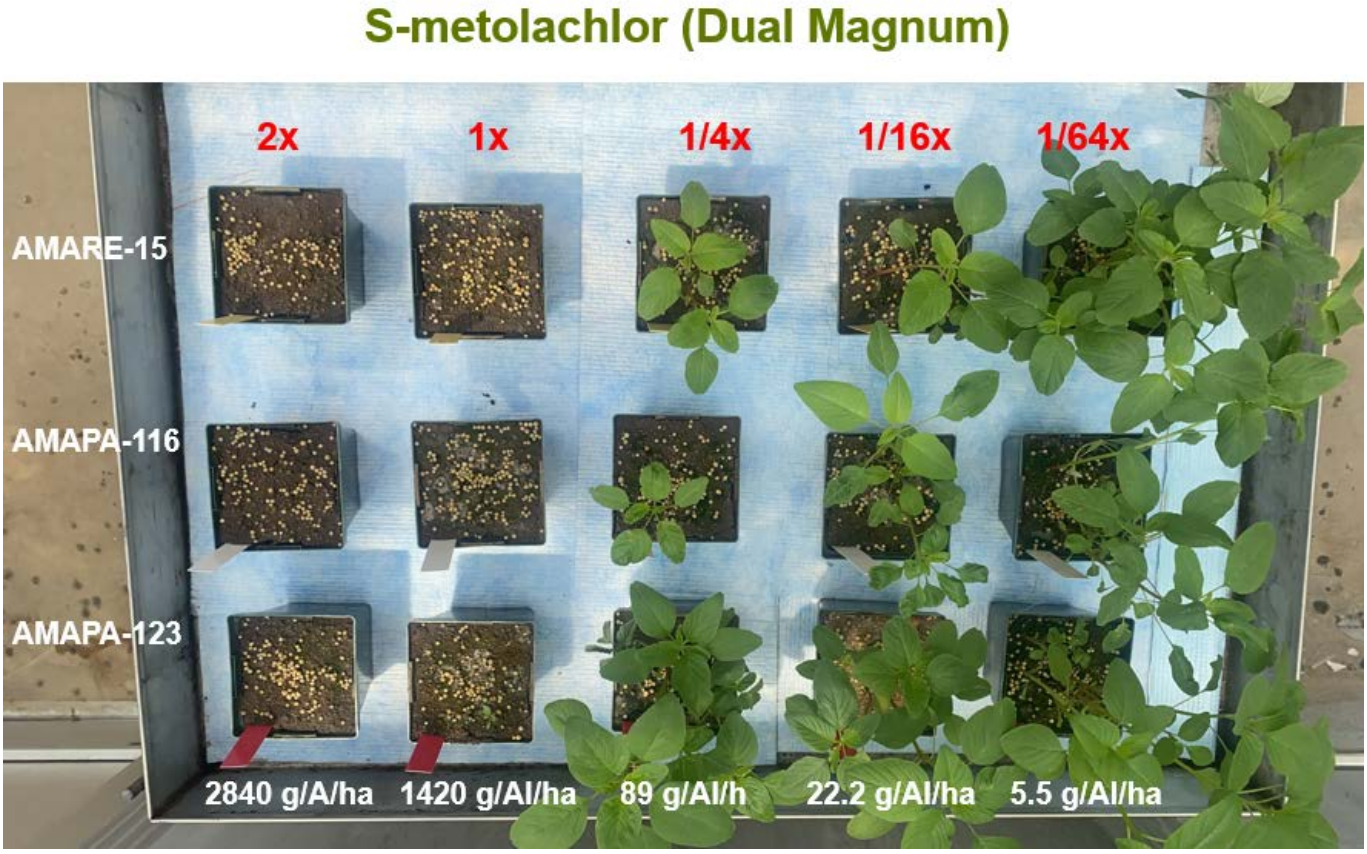
ED(SRR, c(50, 90), interval="delta")

```
Estimated effective doses

               Estimate Std. Error     Lower     Upper
e:AMARE15:50     86.392    101.685  -119.459   292.243
e:AMARE15:90    343.762   1035.591 -1752.683  2440.207
```

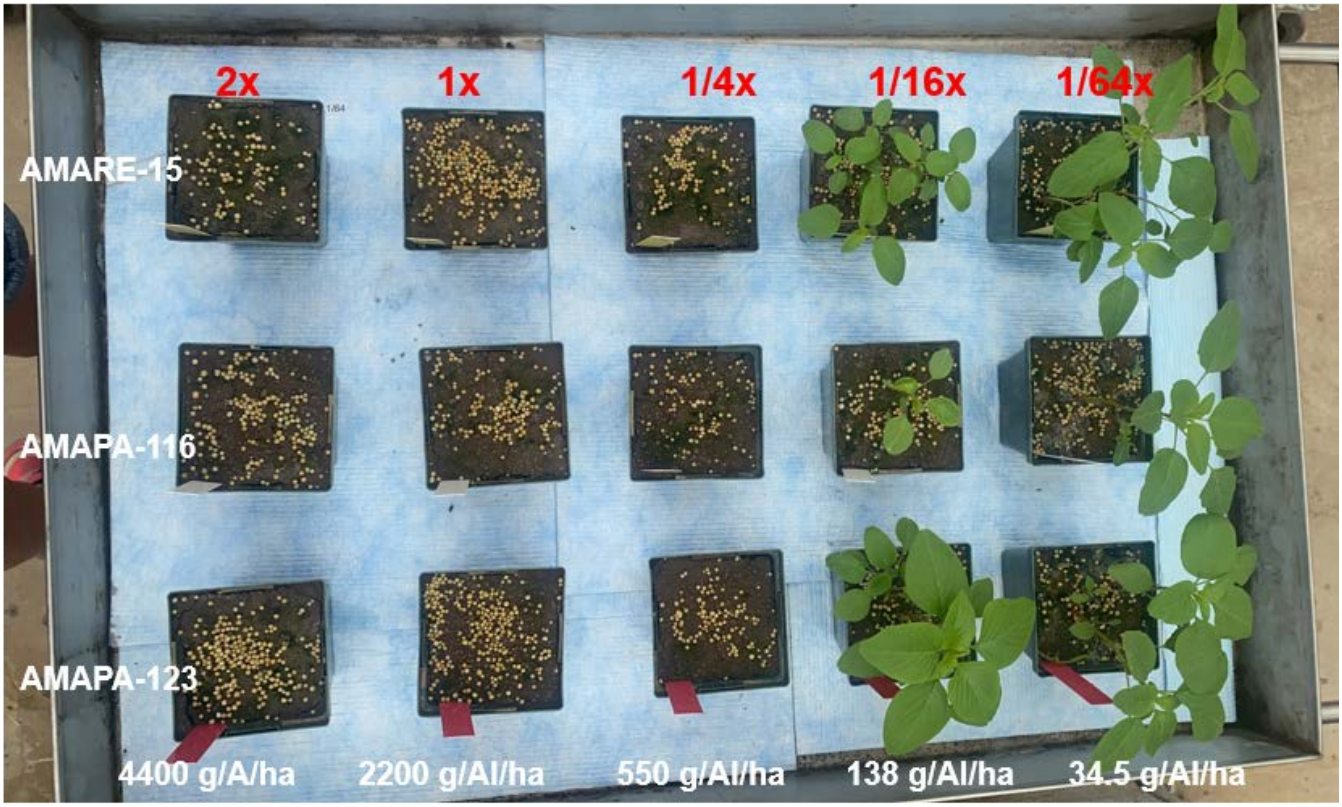
mselect(SRR, fctList = list(W1.3(fixed=c(NA, 100, NA)),W1.4(), W2.3(fixed=c(NA, 100, NA)), W2.4(), LL.3()),linreg=TRUE)

```
         logLik       IC Lack of fit  Res var
Lin   -220.8417 447.6834          NA 2269.204
Quad  -219.8985 447.7971          NA 2225.173
LL.4  -219.8207 449.6414   0.9328628 2275.279
Cubic -219.8453 449.6907          NA 2277.952
W1.3          NA       NA          NA       NA
W1.4          NA       NA          NA       NA
W2.3          NA       NA          NA       NA
W2.4          NA       NA          NA       NA
LL.3          NA       NA          NA       NA
```

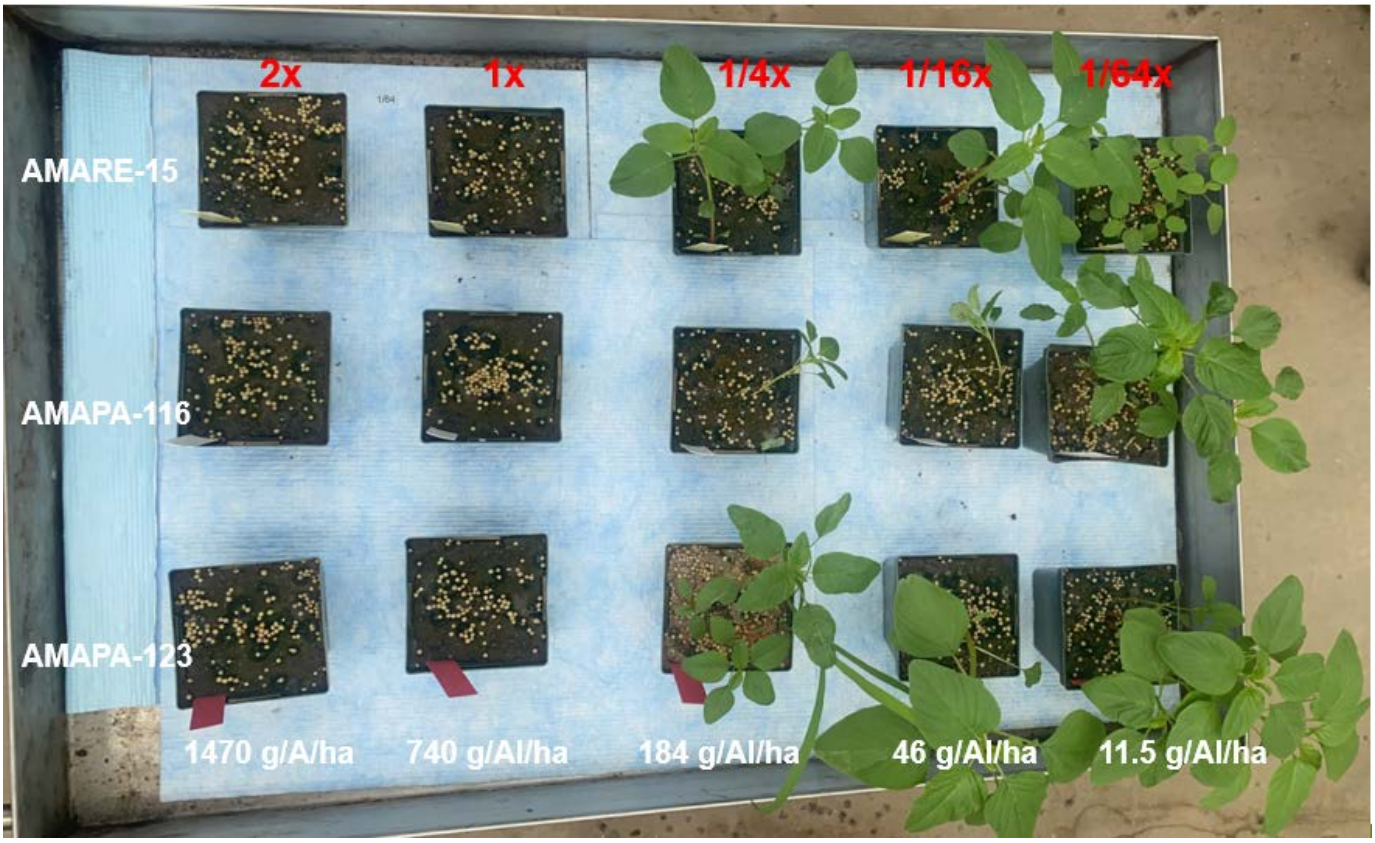
Dose Response with Palmer amaranth (*Amaranthus palmeri)* and Redroot Pigweed (*Amaranthus retroflexus)*



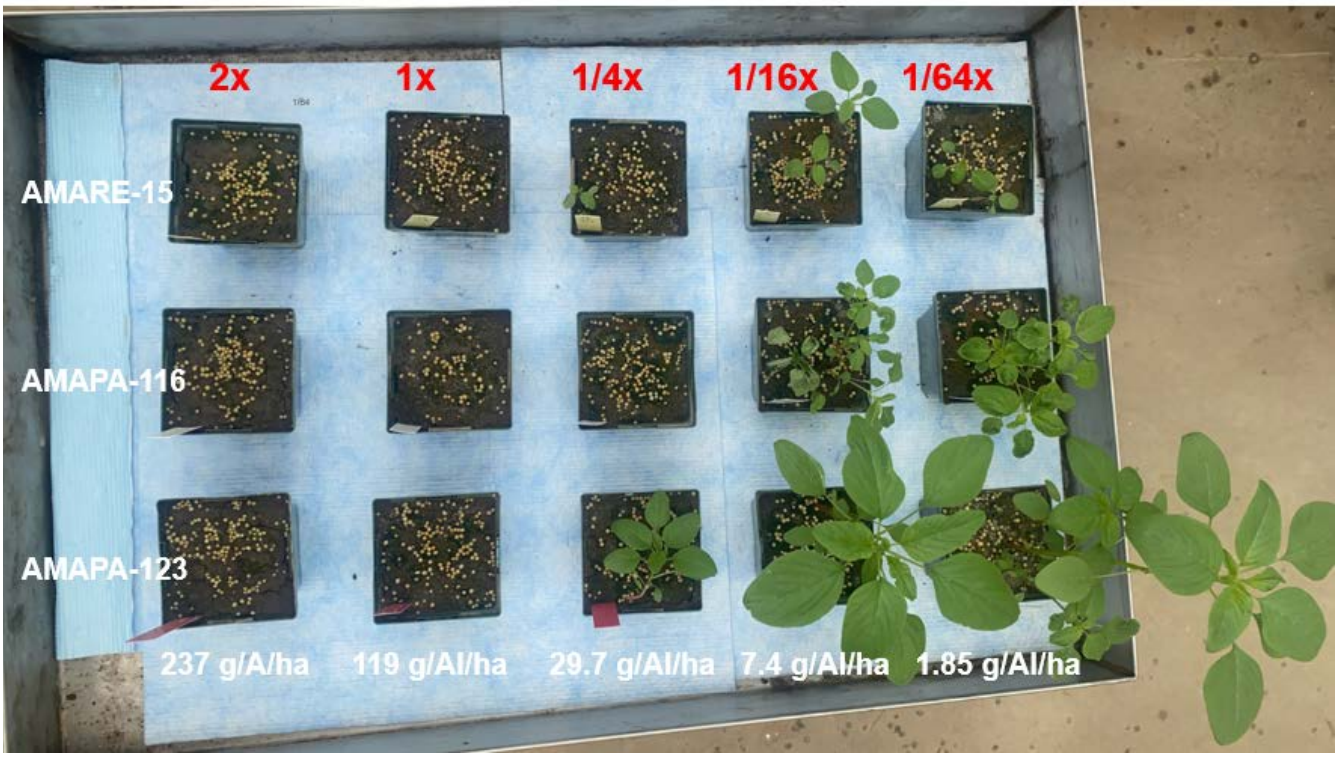
Acetochlor (Harness)



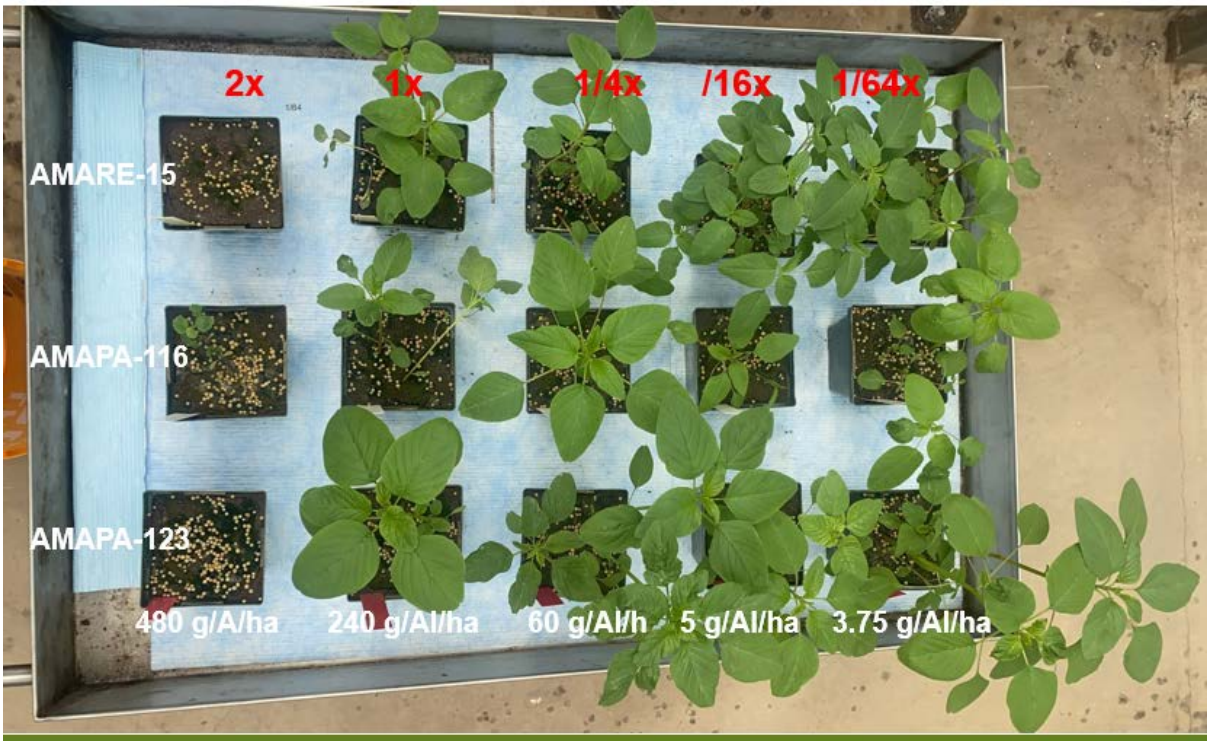
Dimethenamid-P (Outlook)



Pyroxasulfone (Zidua SC)



Flufenacet (Sunfire)



APPENDIX B: CHAPTER 3

Comparison of I_{50} and regression equations used to model Group 15/VLCFA control of North Carolina Palmer amaranth populations over time

Herbicide	Population	Model	I50	R2
S-metolachlor	WAKE	f = y0+a*x+b*x^2 (Quadratic) y0 91.8565 a -10.3681 b 0.8440	7+	0.9252*
	WAKE	f = y0+a*x (Linear) y0 88.4804 a -5.3040	7+	0.8558
	WAKE	Log Logistic 3 Parameter	N/A	Poor fit
	MARTIN	f = y0+a*x+b*x^2 (Quadratic) y0 71.0300 a 1.8686 b -1.1042	5.3	0.9931
	MARTIN	f = y0+a*x (Linear) y0 75.4466 a -4.7563	5.34	0.8471
	MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b))))) Log Logistic 3 Parameter a 70.6894 b 3.8549 x0 6.6364	5.3	0.9972
	WAKE + MARTIN	f = y0+a*x+b*x^2 (Quadratic) y0 81.4627 a -4.3376 b -0.1057	6.3	**0.9628**
	WAKE + MARTIN	f = y0+a*x (Linear) y0 81.8854 a -4.9716	6.4	0.9614
	WAKE + MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b))))) Log Logistic 3 Parameter a 81.8577 b 1.2683 x0 9.5493	6.7	0.9498
Acetochlor	WAKE	f = y0+a*x+b*x^2 (Quadratic) y0 80.7352 a -18.8276 b 1.4349	1.9	0.9761
	WAKE	f = y0+a*x (Linear) y0 74.9955 a -10.2182	2.4	0.9182
	WAKE	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b))))) Log Logistic 3 Parameter a 82.2679 b 1.1616 x0 2.3833	1.6	0.9868**
	MARTIN	f = y0+a*x+b*x^2 (Quadratic) y0 49.5942	-0.03	0.9634

Comparison of I_{50} and regression equations used to model Group 15/VLCFA control of North Carolina Palmer amaranth populations over time

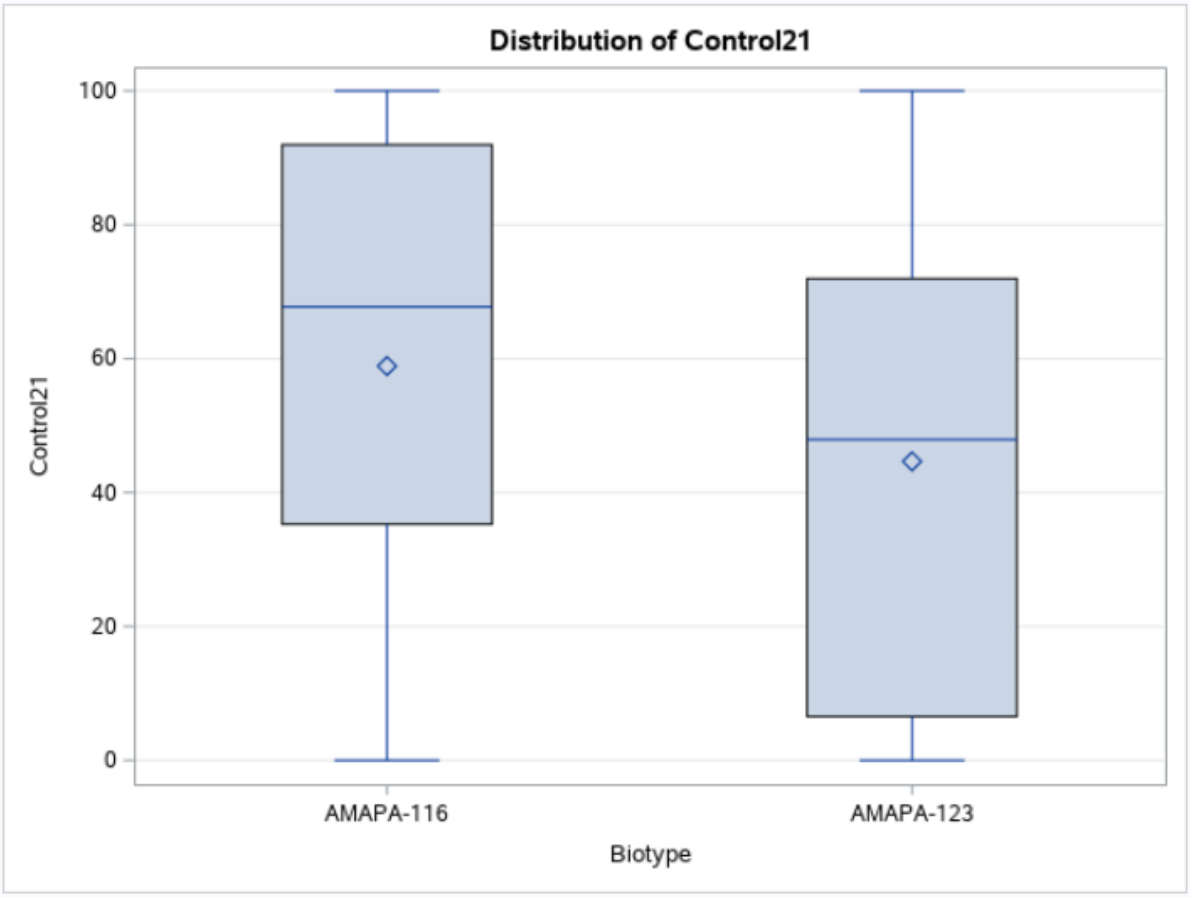
Herbicide	Population	Model	I50	R2
		a -12.4458		
		b 0.7831		
	MARTIN	f = y0+a*x (Linear)	-0.45	0.9329
		y0 46.4616		
		a -7.7470		
	MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) Log Logistic 3 Parameter	0	0.9964
		a 48.1488		
		b 3.1691		
		x0 2.4944		
	WAKE + MARTIN	f = y0+a*x+b*x^2 (Quadratic)	1.15	**0.9886**
		y0 63.5470		
		a -12.6701		
		b 0.6837		
	WAKE + MARTIN	f = y0+a*x (Linear)	-0.44	0.9689
		y0 60.8121		
		a -8.5677		
	WAKE + MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) Log Logistic 3 Parameter	1.2	0.9816
		a 64.2705		
		b 1.5543		
		x0 2.7065		
Dimethenamid-P	WAKE	f = y0+a*x+b*x^2 (Quadratic)	1.6	0.9930
		y0 80.0279		
		a -21.1681		
		b 1.6165		
	WAKE	f = y0+a*x (Linear)	2.04	0.9337
		y0 73.5620		
		a -11.4693		
	WAKE	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) (Log Logistic 3 Parameter)	1.54	0.9945
		a 80.9314		
		b 1.5890		
		x0 2.0977		
	MARTIN	f = y0+a*x+b*x^2 (Quadratic)	0.93	1.0000
		y0 56.8804		
		a -7.6384		
		b 0.2090		
	MARTIN	f = y0+a*x (Linear)	0.93	0.9966
		y0 56.0444		
		a -6.3844		
	MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) (Log Logistic 3 Parameter)	1.18	0.9965
		a 56.7632		
		b 1.6294		
		x0 4.0195		

Comparison of I_{50} and regression equations used to model Group 15/VLCFA control of North Carolina Palmer amaranth populations over time

Herbicide	Population	Model	I50	R2
	WAKE + MARTIN	f = y0+a*x+b*x^2 (Quadratic) y0 68.4632 a -14.4439 b 0.9240	1.39	**0.9969**
	WAKE + MARTIN	f = y0+a*x (Linear) y0 -8.8997 a -6.3844	1.64	0.9637
	WAKE + MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) (Log Logistic 3 Parameter) a 68.8610 b 1.5064 x0 2.7464	1.42	0.9954
Pyroxasulfone	WAKE	f = y0+a*x+b*x^2 (Quadratic) y0 96.3714 a -3.0577 b -0.2829	7+	0.9635
	WAKE	f = y0+a*x (Linear) y0 97.5031 a -4.7553	7+	0.9527
	WAKE	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) (Log Logistic 3 Parameter) a 96.1620 b 1.6347 x0 10.2156	7+	0.9775**
	MARTIN	f = y0+a*x+b*x^2 (Quadratic) y0 86.0122 a -9.6187b0.4151	4.7	1.0000
	MARTIN	f = y0+a*x (Linear) y0 84.3520 a -7.1284	4.82	0.9892
	MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) (Log Logistic 3 Parameter) a 86.0081 b 1.2260 x0 6.1001	4.66	0.9998
	WAKE + MARTIN	f = y0+a*x+b*x^2 (Quadratic) y0 91.2960 a -6.5873 b 0.0863	6.9	0.9918
	WAKE + MARTIN	f = y0+a*x (Linear) y0 90.9506 a -6.0692	6.7	0.9911
	WAKE + MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) (Log Logistic 3 Parameter) a 90.9525 b 1.3676 x0 8.1246	7	**0.9963**
Flufenacet	WAKE	f = y0+a*x+b*x^2 (Quadratic)	-0.66	0.9925**

Comparison of I_{50} and regression equations used to model Group 15/VLCFA control of North Carolina Palmer amaranth populations over time

Herbicide	Population	Model	I50	R2
		y0 54.2281		
		a 5.4742		
		b -1.1900		
	WAKE	f = y0+a*x (Linear)	5.4	0.3769
		y0 58.9880		
		a -1.6656		
	WAKE	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) (Log Logistic 3 Parameter)	5.9	0.9041
		a 57.3216		
		b 43.2598		
		x0 6.1680		
	MARTIN	f = y0+a*x+b*x^2 (Quadratic)	N/A	0.9720
		y0 42.3657		
		a 2.1443		
		b -0.8143		
	MARTIN	f = y0+a*x (Linear)	0	0.7580
		y0 45.6228		
		a -2.7413		
	MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) (Log Logistic 3 Parameter)	N/A	0.9579
		a 43.4955		
		b 3.3273		
		x0 6.7945		
	WAKE + MARTIN	f = y0+a*x+b*x^2 (Quadratic)	0.52	**0.9952**
		y0 48.3368		
		a 3.6297		
		b -0.9522		
	WAKE + MARTIN	f = y0+a*x (Linear)	1.02	0.5966
		y0 52.1458		
		a -2.0838		
	WAKE + MARTIN	f = a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))) (Log Logistic 3 Parameter)	2.19	0.9353
		a 50.1919		
		b 4.6376		
		x0 7.3297		



Level of Biotype	N	Control21	
		Mean	Std Dev
AMAPA-116	237	58.8952766	34.1056530
AMAPA-123	235	44.6582323	33.9587018

S-metolachlor

DM116 Linear

Nonlinear Regression **Monday, August 15, 2022, 11:21:30 AM**

Data Source: SMOC Residual in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9251	0.8558	0.7838	6.8838

	Coefficient	Std. Error	t	P
y0	88.4804	5.7594	15.3628	0.0042
a	-5.3040	1.5393	-3.4458	0.0749

Analysis of Variance:

	DF	SS	MS
Regression	2	21627.3537	10813.6768
Residual	2	94.7730	47.3865
Total	4	21722.1267	5430.5317

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	562.6477	562.6477
Residual	2	94.7730	47.3865
Total	3	657.4207	219.1402

DM123 Linear

Nonlinear Regression **Monday, August 15, 2022, 11:23:03 AM**

Data Source: SMOC Residual in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9204	0.8471	0.7706	6.3910

	Coefficient	Std. Error	t	P
y0	75.4466	5.3471	14.1098	0.0050
a	-4.7563	1.4291	-3.3283	0.0796

Analysis of Variance:

	DF	SS	MS
Regression	2	15423.2286	7711.6143
Residual	2	81.6904	40.8452
Total	4	15504.9190	3876.2298

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	452.4562	452.4562
Residual	2	81.6904	40.8452
Total	3	534.1466	178.0489

DM116 Quadratic

Nonlinear Regression **Monday, August 15, 2022, 11:17:07 AM**

Data Source: SMOC Residual in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9619	0.9252	0.7756	7.0130

	Coefficient	Std. Error	t	P
y0	91.8565	6.8354	13.4383	0.0473
a	-10.3681	5.4886	-1.8890	0.3099
b	0.8440	0.8766	0.9628	0.5121

Analysis of Variance:

	DF	SS	MS
Regression	3	21672.9442	7224.3147
Residual	1	49.1825	49.1825
Total	4	21722.1267	5430.5317

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	608.2383	304.1191
Residual	1	49.1825	49.1825
Total	3	657.4207	219.1402

DM123 Quadratic

Nonlinear Regression **Monday, August 15, 2022, 11:18:37 AM**

Data Source: SMOC Residual in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9966	0.9931	0.9794	1.9143

	Coefficient	Std. Error	t	P
y0	71.0300	1.8658	38.0697	0.0167
a	1.8686	1.4981	1.2473	0.4302
b	-1.1042	0.2393	-4.6144	0.1359

Analysis of Variance:

	DF	SS	MS
Regression	3	15501.2546	5167.0849
Residual	1	3.6644	3.6644
Total	4	15504.9190	3876.2298

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	530.4822	265.2411
Residual	1	3.6644	3.6644
Total	3	534.1466	178.0489

DMBoth Linear

Nonlinear Regression **Monday, August 15, 2022, 3:18:44 PM**

Data Source: SMOC Residual in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9805	0.9614	0.9420	3.1517

	Coefficient	Std. Error	t	P
y0	81.8854	2.6369	31.0534	0.0010
a	-4.9716	0.7047	-7.0544	0.0195

Analysis of Variance:

	DF	SS	MS
Regression	2	18434.5870	9217.2935
Residual	2	19.8668	9.9334
Total	4	18454.4538	4613.6135

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	494.3357	494.3357
Residual	2	19.8668	9.9334
Total	3	514.2025	171.4008

DMBoth Quadratic

Nonlinear Regression **Monday, August 15, 2022, 12:13:45 PM**

Data Source: SMOC Residual in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9812	0.9628	0.8883	4.3763

	Coefficient	Std. Error	t	P
y0	81.4627	4.2655	19.0980	0.0333
a	-4.3376	3.4250	-1.2664	0.4255
b	-0.1057	0.5470	-0.1932	0.8785

Analysis of Variance:

	DF	SS	MS
Regression	3	18435.3016	6145.1005
Residual	1	19.1522	19.1522
Total	4	18454.4538	4613.6135

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	495.0503	247.5251
Residual	1	19.1522	19.1522
Total	3	514.2025	171.4008

DM116 Log Logistic 3 Parameter

Nonlinear Regression **Tuesday, August 16, 2022, 1:40:33 PM**

Data Source: SMOC Residual in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
(NAN)	-12.4019	-39.2056	93.8651

	Coefficient	Std. Error	t	P
a	99.1638508	1174.3242	1.9516E-005	1.0000
b	-0.0192	1883.3028	-1.0214E-005	1.0000
x0	2.4947E-015	7.3072E-007	3.4140E-009	1.0000

Analysis of Variance:

	DF	SS	MS
Regression	3	12911.4618	4303.8206
Residual	1	8810.6648	8810.6648
Total	4	21722.1267	5430.5317

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	-8153.2441	-4076.6221
Residual	1	8810.6648	8810.6648
Total	3	657.4207	219.1402

DM123 Log Logistic 3 Parameter

Nonlinear Regression **Tuesday, August 16, 2022, 1:44:42 PM**

Data Source: SMOC Residual in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9986	0.9972	0.9915	1.2333

	Coefficient	Std. Error	t	P
a	70.6894	0.9644	73.2954	0.0087
b	3.8549	0.4906	7.8568	0.0806
x0	6.6364	0.1684	39.4115	0.0161

Analysis of Variance:

	DF	SS	MS
Regression	3	15503.3981	5167.7994
Residual	1	1.5209	1.5209
Total	4	15504.9190	3876.2298

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	532.6257	266.3129
Residual	1	1.5209	1.5209
Total	3	534.1466	178.0489

DMBoth Log Logistic 3 Parameter

Nonlinear Regression **Tuesday, August 16, 2022, 1:41:48 PM**

Data Source: SMOC Residual in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9746	0.9498	0.8495	5.0796

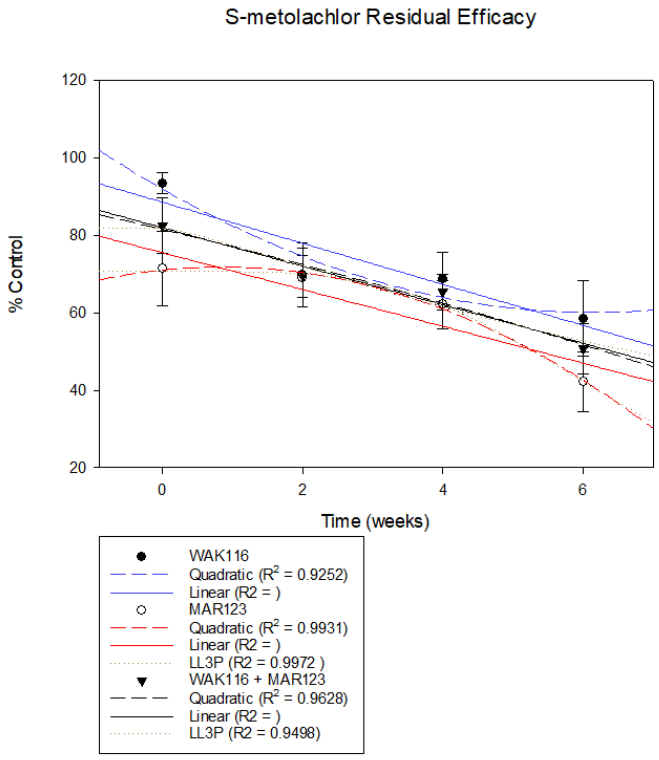
	Coefficient	Std. Error	t	P
a	81.8577	5.0459	16.2225	0.0392
b	1.2683	0.6299	2.0136	0.2934
x0	9.5493	3.4406	2.7754	0.2202

Analysis of Variance:

	DF	SS	MS
Regression	3	18428.6516	6142.8839
Residual	1	25.8022	25.8022
Total	4	18454.4538	4613.6135

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	488.4002	244.2001
Residual	1	25.8022	25.8022
Total	3	514.2025	171.4008



Acetochlor

WAKE Linear

Nonlinear Regression **Monday, August 15, 2022, 11:48:54 AM**

Data Source: Harness in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9582	0.9182	0.8772	9.6468

	Coefficient	Std. Error	t	P
y0	74.9955	8.0711	9.2919	0.0114
a	-10.2182	2.1571	-4.7370	0.0418

Analysis of Variance:

	DF	SS	MS
Regression	2	9952.7226	4976.3613
Residual	2	186.1203	93.0601
Total	4	10138.8428	2534.7107

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	2088.2200	2088.2200
Residual	2	186.1203	93.0601
Total	3	2274.3402	758.1134

MARTIN

Nonlinear Regression - Dynamic Fitting **Monday, August 15, 2022, 11:49:39 AM**

Data Source: Harness in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

Dynamic Fit Options:

Total Number of Fits	200
Maximum Number of Iterations	200

Parameter Ranges for Initial Estimates:

	Minimum	Maximum
y0	-46.4616	139.3849
a	-23.2410	7.7470

Summary of Fit Results:

Converged	100.0%

Results for the Overall Best-Fit Solution:

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9659	0.9329	0.8993	6.5712

	Coefficient	Std. Error	t	P
y0	46.4616	5.4978	8.4509	0.0137
a	-7.7470	1.4694	-5.2724	0.0341

WAKE + MARTIN Linear

Nonlinear Regression **Monday, August 15, 2022, 11:50:20 AM**

Data Source: Harness in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9843	0.9689	0.9533	4.8556

	Coefficient	Std. Error	t	P
y0	60.8121	4.0625	14.9691	0.0044
a	-8.5677	1.0858	-7.8910	0.0157

Analysis of Variance:

	DF	SS	MS
Regression	2	6398.6609	3199.3305
Residual	2	47.1545	23.5772
Total	4	6445.8154	1611.4538

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	1468.1218	1468.1218
Residual	2	47.1545	23.5772
Total	3	1515.2762	505.0921

WAKE Quadratic

Nonlinear Regression **Monday, August 15, 2022, 11:40:29 AM**

Data Source: Harness in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9880	0.9761	0.9283	7.3720

	Coefficient	Std. Error	t	P
y0	80.7352	7.1853	11.2361	0.0565
a	-18.8276	5.7695	-3.2633	0.1893
b	1.4349	0.9215	1.5571	0.3634

Analysis of Variance:

	DF	SS	MS
Regression	3	10084.4964	3361.4988
Residual	1	54.3465	54.3465
Total	4	10138.8428	2534.7107

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	2219.9938	1109.9969
Residual	1	54.3465	54.3465
Total	3	2274.3402	758.1134

MARTIN Quadratic

Nonlinear Regression **Monday, August 15, 2022, 11:42:14 AM**

Data Source: Harness in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9943	0.9886	0.9659	4.1515

	Coefficient	Std. Error	t	P
y0	63.5470	4.0464	15.7047	0.0405
a	-12.6701	3.2491	-3.8996	0.1598
b	0.6837	0.5189	1.3176	0.4133

Analysis of Variance:

	DF	SS	MS
Regression	3	6428.5804	2142.8601
Residual	1	17.2350	17.2350
Total	4	6445.8154	1611.4538

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	1498.0413	749.0206
Residual	1	17.2350	17.2350
Total	3	1515.2762	505.0921

WAKE + MARTIN Quadratic

Nonlinear Regression **Monday, August 15, 2022, 11:41:08 AM**

Data Source: Harness in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9815	0.9634	0.8902	6.8636

	Coefficient	Std. Error	t	P
y0	49.5942	6.6898	7.4134	0.0854
a	-12.4458	5.3716	-2.3170	0.2594
b	0.7831	0.8579	0.9128	0.5290

Analysis of Variance:

	DF	SS	MS
Regression	3	3396.3636	1132.1212
Residual	1	47.1090	47.1090
Total	4	3443.4726	860.8681

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	1239.5723	619.7861
Residual	1	47.1090	47.1090
Total	3	1286.6813	428.8938

WAKE Log Logistic 3 Parameter

Nonlinear Regression **Monday, August 15, 2022, 11:53:58 AM**

Data Source: Harness in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9934	0.9868	0.9605	5.4739

	Coefficient	Std. Error	t	P
a	82.2679	5.4727	15.0324	0.0423
b	1.1616	0.3871	3.0007	0.2048
x0	2.3833	0.5209	4.5754	0.1370

Analysis of Variance:

	DF	SS	MS
Regression	3	10108.8790	3369.6263
Residual	1	29.9639	29.9639
Total	4	10138.8428	2534.7107

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	2244.3764	1122.1882
Residual	1	29.9639	29.9639
Total	3	2274.3402	758.1134

MARTIN Log Logistic 3 Parameter

Nonlinear Regression **Monday, August 15, 2022, 11:54:50 AM**

Data Source: Harness in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9982	0.9964	0.9893	2.1462

	Coefficient	Std. Error	t	P
a	48.1488	2.1444	22.4536	0.0283
b	3.1691	0.4661	6.7995	0.0930
x0	2.4944	0.1554	16.0499	0.0396

Analysis of Variance:

	DF	SS	MS
Regression	3	3438.8663	1146.2888
Residual	1	4.6063	4.6063
Total	4	3443.4726	860.8681

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	1282.0750	641.0375
Residual	1	4.6063	4.6063
Total	3	1286.6813	428.8938

WAKE + MARTIN Log Logistic 3 Parameter

Nonlinear Regression **Monday, August 15, 2022, 12:39:41 PM**

Data Source: Harness in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9908	0.9816	0.9448	5.2810

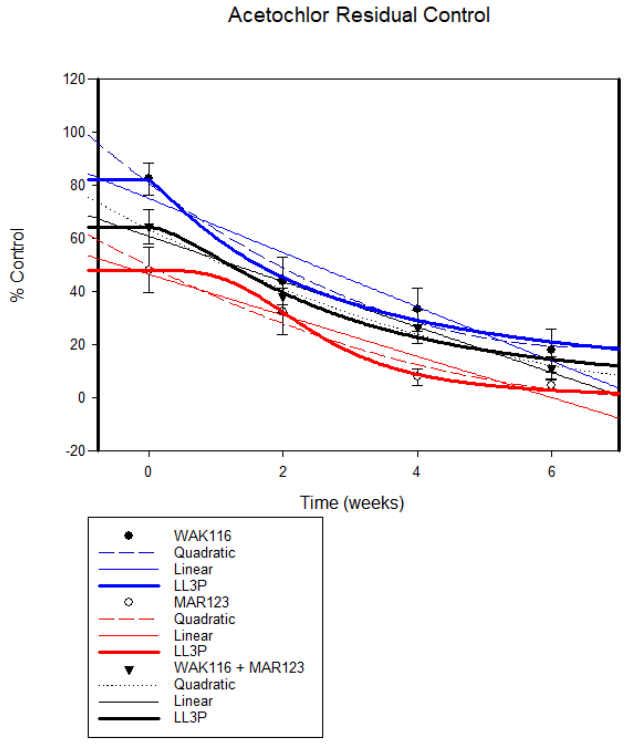
	Coefficient	Std. Error	t	P
a	64.2705	5.2770	12.1794	0.0522
b	1.5543	0.5124	3.0331	0.2027
x0	2.7065	0.5238	5.1669	0.1217

Analysis of Variance:

	DF	SS	MS
Regression	3	6417.9269	2139.3090
Residual	1	27.8885	27.8885
Total	4	6445.8154	1611.4538

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	1487.3877	743.6939
Residual	1	27.8885	27.8885
Total	3	1515.2762	505.0921



Outlook

WAKE Linear

Nonlinear Regression **Monday, August 15, 2022, 2:08:49 PM**

Data Source: Data 3 in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9663	0.9337	0.9005	9.6657

	Coefficient	Std. Error	t	P
y0	73.5620	8.0869	9.0964	0.0119
a	-11.4693	2.1613	-5.3066	0.0337

Analysis of Variance:

	DF	SS	MS
Regression	2	8763.0961	4381.5480
Residual	2	186.8516	93.4258
Total	4	8949.9477	2237.4869

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	2630.8846	2630.8846
Residual	2	186.8516	93.4258
Total	3	2817.7362	939.2454

MARTIN Linear

Nonlinear Regression **Monday, August 15, 2022, 2:10:27 PM**

Data Source: Data 3 in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9983	0.9966	0.9948	1.1869

	Coefficient	Std. Error	t	P
y0	56.0444	0.9931	56.4356	0.0003
a	-6.3844	0.2654	-24.0551	0.0017

Analysis of Variance:

	DF	SS	MS
Regression	2	6259.0442	3129.5221
Residual	2	2.8177	1.4088
Total	4	6261.8619	1565.4655

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	815.2181	815.2181
Residual	2	2.8177	1.4088
Total	3	818.0358	272.6786

WAKE + MARTIN Linear

Nonlinear Regression **Monday, August 15, 2022, 2:11:21 PM**

Data Source: Data 3 in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9817	0.9637	0.9455	5.4636

	Coefficient	Std. Error	t	P
y0	64.7671	4.5712	14.1685	0.0049
a	-8.8997	1.2217	-7.2847	0.0183

Analysis of Variance:

	DF	SS	MS
Regression	2	7380.7594	3690.3797
Residual	2	59.7022	29.8511
Total	4	7440.4617	1860.1154

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	1584.1014	1584.1014
Residual	2	59.7022	29.8511
Total	3	1643.8036	547.9345

WAKE Quadratic

Nonlinear Regression **Monday, August 15, 2022, 2:32:56 PM**

Data Source: Outlook in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9965	0.9930	0.9791	4.4297

	Coefficient	Std. Error	t	P
y0	80.0279	4.3176	18.5354	0.0343
a	-21.1681	3.4668	-6.1059	0.1033
b	1.6165	0.5537	2.9193	0.2101

Analysis of Variance:

	DF	SS	MS
Regression	3	8930.3251	2976.7750
Residual	1	19.6225	19.6225
Total	4	8949.9477	2237.4869

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	2798.1136	1399.0568
Residual	1	19.6225	19.6225
Total	3	2817.7362	939.2454

MARTIN Quadratic

Nonlinear Regression **Monday, August 15, 2022, 2:34:36 PM**

Data Source: Outlook in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
1.0000	1.0000	0.9999	0.1484

	Coefficient	Std. Error	t	P
y0	56.8804	0.1446	393.2775	0.0016
a	-7.6384	0.1161	-65.7733	0.0097
b	0.2090	0.0185	11.2678	0.0564

Analysis of Variance:

	DF	SS	MS
Regression	3	6261.8399	2087.2800
Residual	1	0.0220	0.0220
Total	4	6261.8619	1565.4655

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	818.0138	409.0069
Residual	1	0.0220	0.0220
Total	3	818.0358	272.6786

WAKE + MARTIN Quadratic

Nonlinear Regression **Monday, August 15, 2022, 2:35:51 PM**

Data Source: Outlook in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9985	0.9969	0.9908	2.2486

	Coefficient	Std. Error	t	P
y0	68.4632	2.1917	31.2377	0.0204
a	-14.4439	1.7598	-8.2076	0.0772
b	0.9240	0.2811	3.2875	0.1880

Analysis of Variance:

	DF	SS	MS
Regression	3	7435.4054	2478.4685
Residual	1	5.0563	5.0563
Total	4	7440.4617	1860.1154

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	1638.7473	819.3737
Residual	1	5.0563	5.0563
Total	3	1643.8036	547.9345

WAKE LL3P

Nonlinear Regression **Monday, August 15, 2022, 2:37:27 PM**

Data Source: Outlook in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9973	0.9945	0.9836	3.9295

	Coefficient	Std. Error	t	P
a	80.9314	3.9285	20.6009	0.0309
b	1.5890	0.3311	4.7985	0.1308
x0	2.0977	0.2707	7.7479	0.0817

Analysis of Variance:

	DF	SS	MS
Regression	3	8934.5067	2978.1689
Residual	1	15.4410	15.4410
Total	4	8949.9477	2237.4869

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	2802.2952	1401.1476
Residual	1	15.4410	15.4410
Total	3	2817.7362	939.2454

MARTIN LL3P

Nonlinear Regression **Monday, August 15, 2022, 2:38:19 PM**

Data Source: Outlook in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9983	0.9965	0.9895	1.6911

	Coefficient	Std. Error	t	P
a	56.7632	1.6844	33.6992	0.0189
b	1.6294	0.2030	8.0251	0.0789
x0	4.0195	0.2538	15.8371	0.0401

Analysis of Variance:

	DF	SS	MS
Regression	3	6259.0020	2086.3340
Residual	1	2.8599	2.8599
Total	4	6261.8619	1565.4655

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	815.1760	407.5880
Residual	1	2.8599	2.8599
Total	3	818.0358	272.6786

WAKE + MARTIN LL3P

Nonlinear Regression **Monday, August 15, 2022, 2:49:29 PM**

Data Source: Outlook in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9977	0.9954	0.9863	2.7392

	Coefficient	Std. Error	t	P
a	68.8610	2.7372	25.1575	0.0253
b	1.5064	0.2449	6.1509	0.1026
x0	2.7464	0.2629	10.4462	0.0608

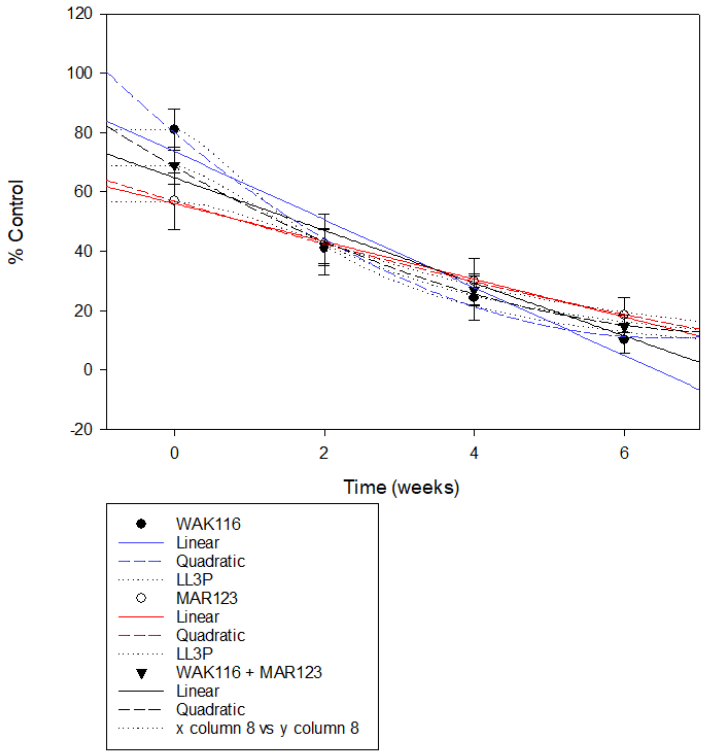
Analysis of Variance:

	DF	SS	MS
Regression	3	7432.9584	2477.6528
Residual	1	7.5032	7.5032
Total	4	7440.4617	1860.1154

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	1636.3004	818.1502
Residual	1	7.5032	7.5032
Total	3	1643.8036	547.9345

Dimethenamid-P Residual Control



Pyroxasulfone

WAKE Linear

Nonlinear Regression **Monday, August 15, 2022, 4:34:17 PM**

Data Source: Data 5 in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9761	0.9527	0.9291	3.3500

	Coefficient	Std. Error	t	P
y0	97.5031	2.8028	34.7878	0.0008
a	-4.7553	0.7491	-6.3482	0.0239

Analysis of Variance:

	DF	SS	MS
Regression	2	28166.0259	14083.0129
Residual	2	22.4447	11.2223
Total	4	28188.4705	7047.1176

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	452.2508	452.2508
Residual	2	22.4447	11.2223
Total	3	474.6955	158.2318

MARTIN Linear

Nonlinear Regression **Monday, August 15, 2022, 4:37:14 PM**

Data Source: Zidua in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9946	0.9892	0.9839	2.3504

	Coefficient	Std. Error	t	P
y0	84.3520	1.9665	42.8944	0.0005
a	-7.1284	0.5256	-13.5631	0.0054

Analysis of Variance:

	DF	SS	MS
Regression	2	16875.5839	8437.7920
Residual	2	11.0489	5.5245
Total	4	16886.6328	4221.6582

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	1016.2699	1016.2699
Residual	2	11.0489	5.5245
Total	3	1027.3189	342.4396

WAKE + MARTIN Linear

Nonlinear Regression **Monday, August 15, 2022, 4:35:15 PM**

Data Source: Data 5 in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9956	0.9911	0.9867	1.8143

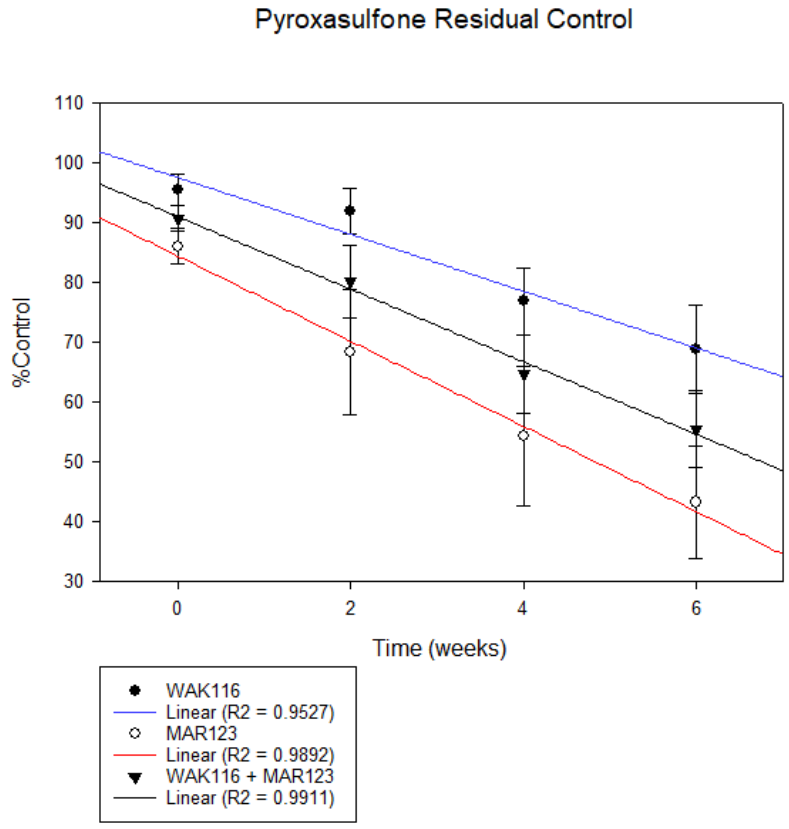
	Coefficient	Std. Error	t	P
y0	90.9506	1.5180	59.9160	0.0003
a	-6.0692	0.4057	-14.9600	0.0044

Analysis of Variance:

	DF	SS	MS
Regression	2	21902.9471	10951.4736
Residual	2	6.5835	3.2918
Total	4	21909.5306	5477.3827

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	736.6971	736.6971
Residual	2	6.5835	3.2918
Total	3	743.2806	247.7602



WAKE Quadratic

Nonlinear Regression **Monday, August 15, 2022, 4:40:07 PM**

Data Source: Zidua in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9816	0.9635	0.8905	4.1620

	Coefficient	Std. Error	t	P
y0	96.3714	4.0566	23.7569	0.0268
a	-3.0577	3.2572	-0.9388	0.5201
b	-0.2829	0.5202	-0.5438	0.6829

Analysis of Variance:

	DF	SS	MS
Regression	3	28171.1487	9390.3829
Residual	1	17.3219	17.3219
Total	4	28188.4705	7047.1176

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	457.3737	228.6868
Residual	1	17.3219	17.3219
Total	3	474.6955	158.2318

MARTIN Quadratic

Nonlinear Regression **Monday, August 15, 2022, 4:41:53 PM**

Data Source: Zidua in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
1.0000	1.0000	0.9999	0.1520

	Coefficient	Std. Error	t	P
y0	86.0122	0.1482	580.5045	0.0011
a	-9.6187	0.1190	-80.8486	0.0079
b	0.4151	0.0190	21.8430	0.0291

Analysis of Variance:

	DF	SS	MS
Regression	3	16886.6097	5628.8699
Residual	1	0.0231	0.0231
Total	4	16886.6328	4221.6582

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	1027.2958	513.6479
Residual	1	0.0231	0.0231
Total	3	1027.3189	342.4396

WAKE + MARTIN Quadratic

Nonlinear Regression **Monday, August 15, 2022, 4:44:14 PM**

Data Source: Zidua in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9959	0.9918	0.9754	2.4711

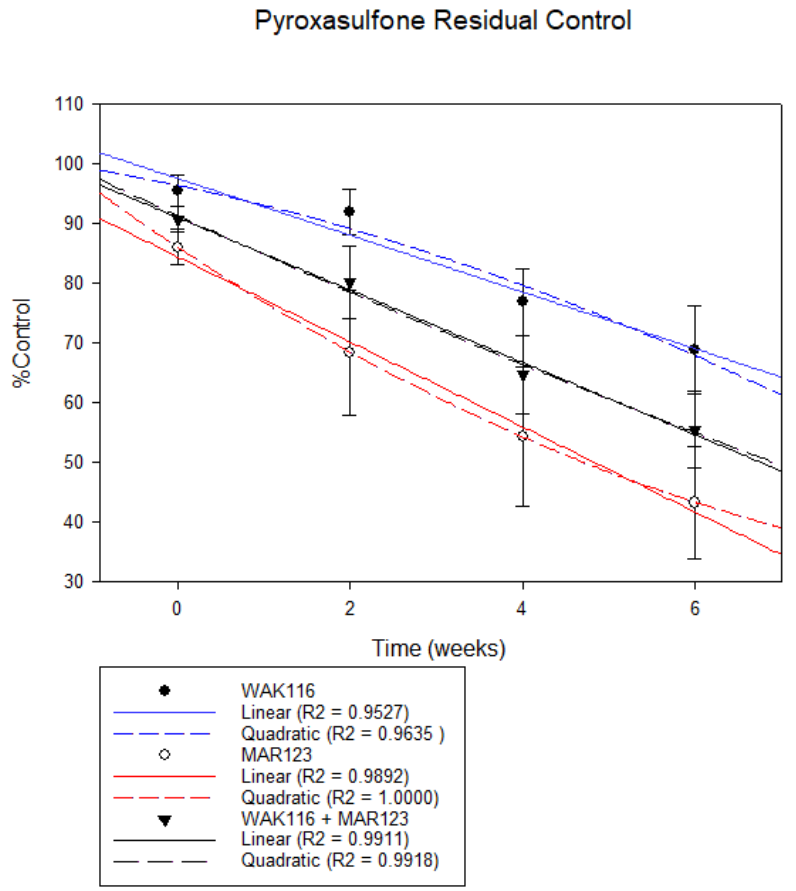
	Coefficient	Std. Error	t	P
y0	91.2960	2.4085	37.9053	0.0168
a	-6.5873	1.9339	-3.4061	0.1818
b	0.0863	0.3089	0.2795	0.8265

Analysis of Variance:

	DF	SS	MS
Regression	3	21903.4243	7301.1414
Residual	1	6.1063	6.1063
Total	4	21909.5306	5477.3827

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	737.1743	368.5871
Residual	1	6.1063	6.1063
Total	3	743.2806	247.7602



WAKE Log Logistic 3 Parameter

Nonlinear Regression **Monday, August 15, 2022, 4:49:18 PM**

Data Source: Zidua in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9887	0.9775	0.9326	3.2665

	Coefficient	Std. Error	t	P
a	96.1620	3.1859	30.1837	0.0211
b	1.6347	0.5447	3.0011	0.2048
x0	10.2156	2.2238	4.5938	0.1365

Analysis of Variance:

	DF	SS	MS
Regression	3	28177.8005	9392.6002
Residual	1	10.6700	10.6700
Total	4	28188.4705	7047.1176

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	464.0255	232.0128
Residual	1	10.6700	10.6700
Total	3	474.6955	158.2318

MARTIN Log Logistic 3 Parameter

Nonlinear Regression - Dynamic Fitting **Monday, August 15, 2022, 4:50:52 PM**

Data Source: Zidua in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

Dynamic Fit Options:

Total Number of Fits	200
Maximum Number of Iterations	200

Parameter Ranges for Initial Estimates:

	Minimum	Maximum
a	0.0000	258.1386
b	-1.0000	3.0000
x0	0.0000	7.5823

Summary of Fit Results:

Converged	96.0%
Singular Solutions	7.5%
Ill-Conditioned Solutions	2.0%
Iterations Exceeding 200	4.0%

Results for the Overall Best-Fit Solution:

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9999	0.9998	0.9993	0.5035

	Coefficient	Std. Error	t	P
a	86.0081	0.5020	171.3148	0.0037
b	1.2260	0.0419	29.2880	0.0217
x0	6.1001	0.1182	51.5953	0.0123

Analysis of Variance:

	DF	SS	MS
Regression	3	16886.3793	5628.7931
Residual	1	0.2535	0.2535
Total	4	16886.6328	4221.6582

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	1027.0654	513.5327
Residual	1	0.2535	0.2535
Total	3	1027.3189	342.4396

WAKE + MARTIN Log Logistic 3 Parameter

Nonlinear Regression **Monday, August 15, 2022, 4:54:35 PM**

Data Source: Zidua in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9981	0.9963	0.9888	1.6652

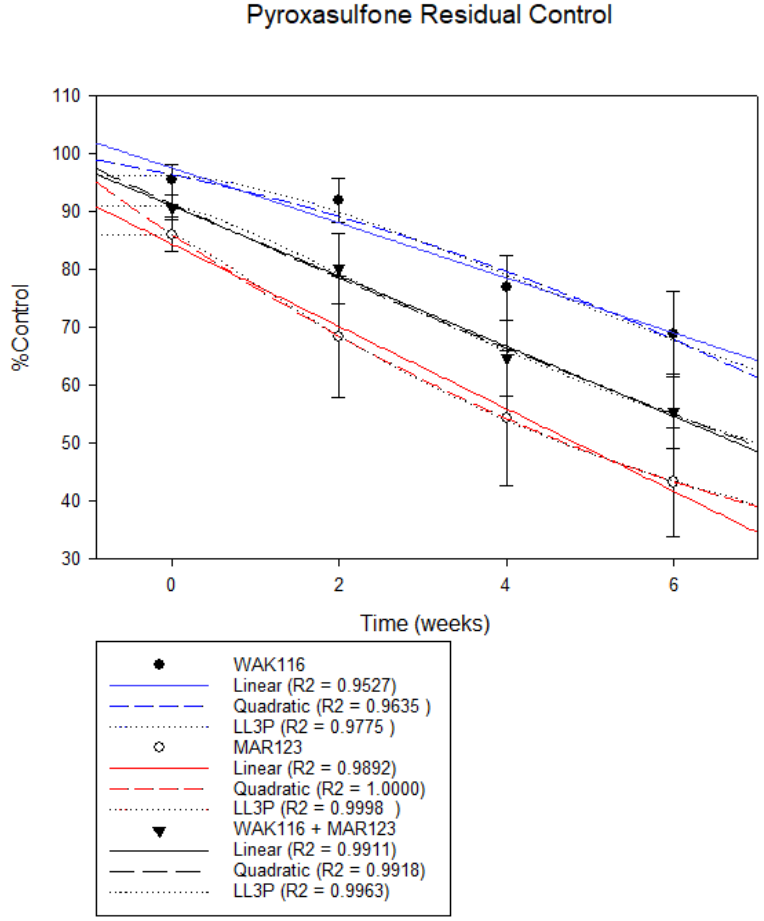
	Coefficient	Std. Error	t	P
a	90.9525	1.6520	55.0551	0.0116
b	1.3676	0.1775	7.7066	0.0821
x0	8.1246	0.6392	12.7109	0.0500

Analysis of Variance:

	DF	SS	MS
Regression	3	21906.7578	7302.2526
Residual	1	2.7729	2.7729
Total	4	21909.5306	5477.3827

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	740.5077	370.2539
Residual	1	2.7729	2.7729
Total	3	743.2806	247.7602



Sunfire

WAKE Linear

Nonlinear Regression **Monday, August 15, 2022, 5:04:30 PM**

Data Source: Data 6 in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.6139	0.3769	0.0654	6.7722

	Coefficient	Std. Error	t	P
y0	58.9880	5.6661	10.4108	0.0091
a	-1.6656	1.5143	-1.0999	0.3861

Analysis of Variance:

	DF	SS	MS
Regression	2	11715.6669	5857.8335
Residual	2	91.7264	45.8632
Total	4	11807.3933	2951.8483

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	55.4847	55.4847
Residual	2	91.7264	45.8632
Total	3	147.2110	49.0703

MARTIN Linear

Nonlinear Regression **Monday, August 15, 2022, 5:06:23 PM**

Data Source: Sunfire in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.8706	0.7580	0.6370	4.8984

	Coefficient	Std. Error	t	P
y0	45.6228	4.0983	11.1320	0.0080
a	-2.7413	1.0953	-2.5028	0.1294

Analysis of Variance:

	DF	SS	MS
Regression	2	5744.9656	2872.4828
Residual	2	47.9894	23.9947
Total	4	5792.9550	1448.2387

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	150.2984	150.2984
Residual	2	47.9894	23.9947
Total	3	198.2878	66.0959

WAKE + MARTIN Linear

Nonlinear Regression **Monday, August 15, 2022, 5:10:05 PM**

Data Source: Sunfire in ResidualEfficacyCurves
Equation: Polynomial, Linear
f = y0+a*x

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.7724	0.5966	0.3949	5.4188

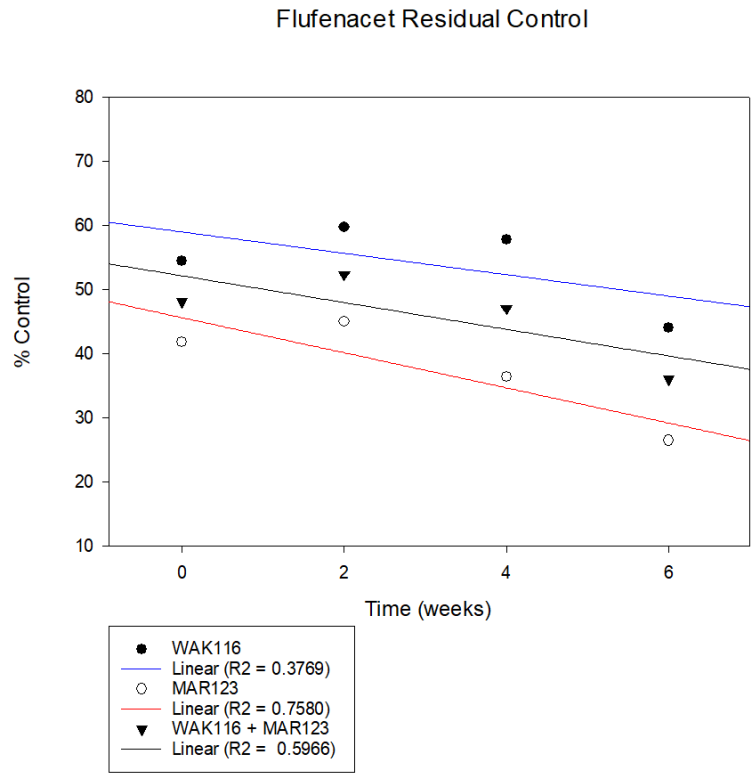
	Coefficient	Std. Error	t	P
y0	52.1458	4.5337	11.5018	0.0075
a	-2.0838	1.2117	-1.7197	0.2276

Analysis of Variance:

	DF	SS	MS
Regression	2	8512.0443	4256.0221
Residual	2	58.7270	29.3635
Total	4	8570.7713	2142.6928

Corrected for the mean of the observations:

	DF	SS	MS
Regression	1	86.8423	86.8423
Residual	2	58.7270	29.3635
Total	3	145.5693	48.5231



WAKE Quadratic

Nonlinear Regression **Monday, August 15, 2022, 5:14:10 PM**

Data Source: Sunfire in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9963	0.9925	0.9776	1.0487

	Coefficient	Std. Error	t	P
y0	54.2281	1.0221	53.0546	0.0120
a	5.4742	0.8207	6.6701	0.0947
b	-1.1900	0.1311	-9.0780	0.0698

Analysis of Variance:

	DF	SS	MS
Regression	3	11806.2936	3935.4312
Residual	1	1.0997	1.0997
Total	4	11807.3933	2951.8483

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	146.1113	73.0557
Residual	1	1.0997	1.0997
Total	3	147.2110	49.0703

MARTIN Quadratic

Nonlinear Regression **Monday, August 15, 2022, 5:15:07 PM**

Data Source: Sunfire in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9859	0.9720	0.9160	2.3570

	Coefficient	Std. Error	t	P
y0	42.3657	2.2973	18.4417	0.0345
a	2.1443	1.8446	1.1625	0.4523
b	-0.8143	0.2946	-2.7638	0.2210

Analysis of Variance:

	DF	SS	MS
Regression	3	5787.3997	1929.1332
Residual	1	5.5552	5.5552
Total	4	5792.9550	1448.2387

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	192.7326	96.3663
Residual	1	5.5552	5.5552
Total	3	198.2878	66.0959

WAKE + MARTIN Quadratic

Nonlinear Regression **Monday, August 15, 2022, 5:15:54 PM**

Data Source: Sunfire in ResidualEfficacyCurves
Equation: Polynomial, Quadratic
f = y0+a*x+b*x^2

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9976	0.9952	0.9857	0.8326

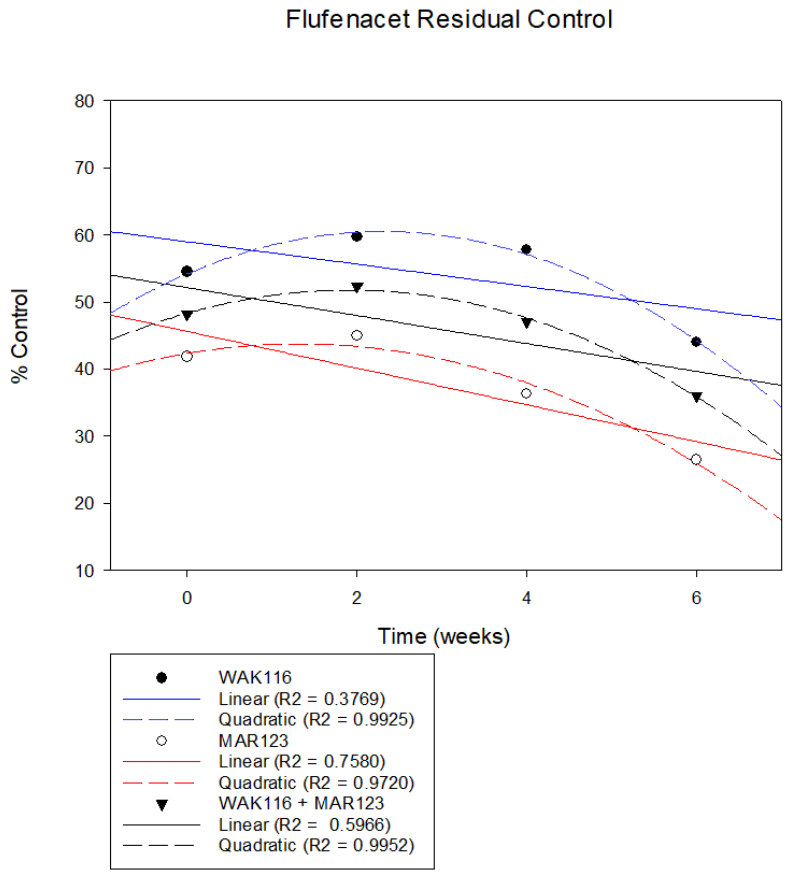
	Coefficient	Std. Error	t	P
y0	48.3368	0.8115	59.5653	0.0107
a	3.6297	0.6516	5.5705	0.1131
b	-0.9522	0.1041	-9.1499	0.0693

Analysis of Variance:

	DF	SS	MS
Regression	3	8570.0781	2856.6927
Residual	1	0.6932	0.6932
Total	4	8570.7713	2142.6928

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	144.8761	72.4381
Residual	1	0.6932	0.6932
Total	3	145.5693	48.5231



WAKE Log Logistic 3 Parameter

Nonlinear Regression **Monday, August 15, 2022, 5:19:37 PM**

Data Source: Sunfire in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9509	0.9041	0.7124	3.7566

	Coefficient	Std. Error	t	P
a	57.3216	2.6561	21.5815	0.0295
b	43.259827910	221.3648	1.5500E-006	1.0000
x0	6.168010	9908.4281	5.6120E-005	1.0000

Analysis of Variance:

	DF	SS	MS
Regression	3	11793.2810	3931.0937
Residual	1	14.1123	14.1123
Total	4	11807.3933	2951.8483

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	133.0988	66.5494
Residual	1	14.1123	14.1123
Total	3	147.2110	49.0703

MARTIN Log Logistic 3 Parameter

Nonlinear Regression **Monday, August 15, 2022, 5:21:33 PM**

Data Source: Sunfire in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9787	0.9579	0.8738	2.8883

	Coefficient	Std. Error	t	P
a	43.4955	2.3658	18.3847	0.0346
b	3.3273	1.6382	2.0311	0.2913
x0	6.7945	0.7879	8.6236	0.0735

Analysis of Variance:

	DF	SS	MS
Regression	3	5784.6126	1928.2042
Residual	1	8.3423	8.3423
Total	4	5792.9550	1448.2387

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	189.9455	94.9727
Residual	1	8.3423	8.3423
Total	3	198.2878	66.0959

WAKE + MARTIN Log Logistic 3 Parameter

Nonlinear Regression **Monday, August 15, 2022, 5:22:52 PM**

Data Source: Sunfire in ResidualEfficacyCurves
Equation: Sigmoidal, Logistic, 3 Parameter
f = if(x<=0, if(b<0,0,a), if(b>0, a/(1+abs(x/x0)^b), a*abs((x/x0))^(abs(b))/(1+(abs(x/x0))^(abs(b)))))

R	Rsqr	Adj Rsqr	Standard Error of Estimate
0.9671	0.9353	0.8058	3.0695

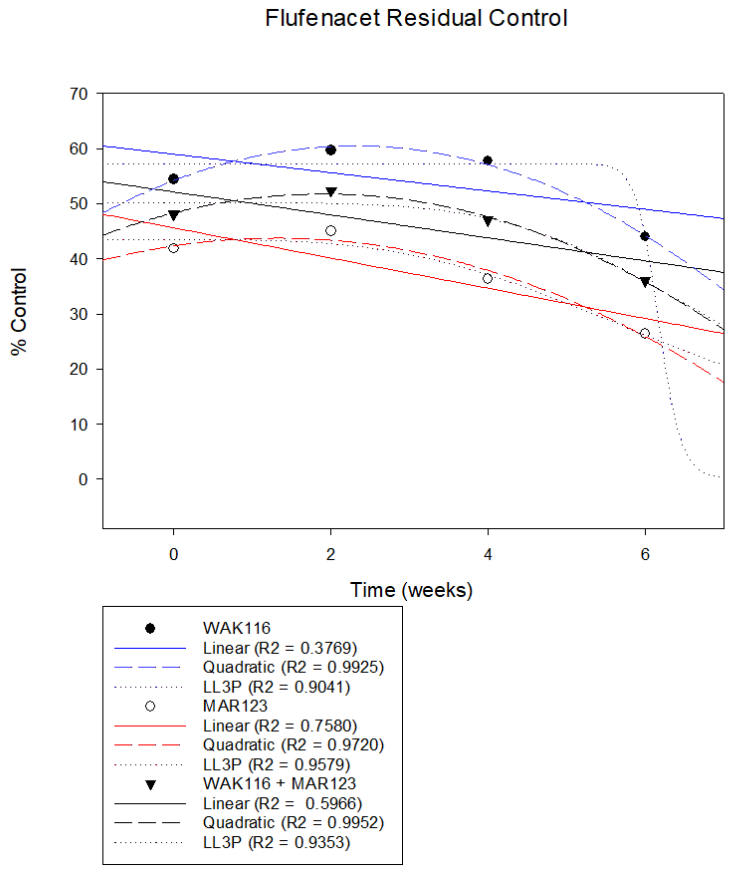
	Coefficient	Std. Error	t	P
a	50.1919	2.2923	21.8957	0.0291
b	4.6376	3.4129	1.3589	0.4039
x0	7.3297	1.1788	6.2177	0.1015

Analysis of Variance:

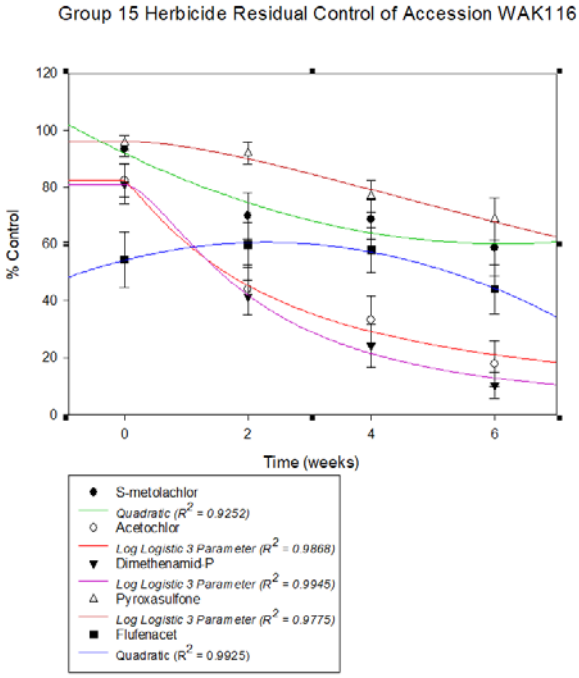
	DF	SS	MS
Regression	3	8561.3495	2853.7832
Residual	1	9.4218	9.4218
Total	4	8570.7713	2142.6928

Corrected for the mean of the observations:

	DF	SS	MS
Regression	2	136.1475	68.0738
Residual	1	9.4218	9.4218
Total	3	145.5693	48.5231



Residual control of WAKE Palmer amaranth by Group 15 herbicides



Residual control of MARTIN Palmer amaranth by Group 15 herbicides