TRIALLATE EFFICACY TO SUSPECTED HERBICIDE RESISTANT WILD OAT (*AVENA FATUA* L.) IN SPRING WHEAT (*TRITICUM AESTIVUM* L.)

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The Supervisory Committee certifies that this ***disquisition*** complies with North Dakota State University's regulations and meets the accepted standards for the degree of

MASTER OF SCIENCE

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ABSTRACT

Herbicide-resistant weed populations have increased because of continuous use of the same grass herbicides. Several collections of wild oat with resistance to ACCase and ALS herbicides have been documented in North Dakota, leaving limited options for control. Two field experiments were conducted to evaluate the efficacy of triallate to control wild oat and determine spring wheat cultivar tolerance to triallate. Injury was not observed until twice the field rate of triallate was applied. Two greenhouse experiments were conducted to characterize 56 suspected resistant wild oat samples and determine triallate efficacy. Wild oat samples were characterized into three subgroups. Triallate provided at least 92% control of all wild oat samples within each subgroup; therefore, integrating triallate back into North Dakota cropping systems is an option to control wild oat in cereal production.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Wild oat (*Avena fatua* L.) is highly competitive for nutrients and water in cereal crops, especially wheat (*Triticum aestiuvm* L.). Over time, wild oat has evolved closely with cereal crops, making control and management difficult. On average, one wild oat plant per 0.33 m^2 reduced wheat yield by 35% (Saylor 2005). United States growers experience an economic deficit of over $1 billion annually due to wild oat control costs and yield loss (Evans et al. 1991), with more than $150 million lost in North Dakota alone (Miller et al. 1977). Early season management can limit yield loss due to prolonged competition.

The United States Environmental Protection Agency (US-EPA) granted registration of triallate in 1962 in wheat and barley (*Hordeum vulgare* L.) for the control of wild oat (Anonymous 2016b). Since then, the introduction of the post-emergence (POST) Acetyl-CoA carboxylase (ACCase) and Acetolactate synthase (ALS) herbicides has resulted in fewer hectares applied with triallate (McMullen et al. 1985). There has been a lack of interest in triallate usage because double incorporation is required (Fay et al. 1976) and POST herbicides, which can be used in reduced and no-till cropping systems, have become more popular. The early effectiveness of POST herbicides has contributed to wide spread usage, allowing certain wild oat biotypes in North Dakota to become resistant to ACCase and/or ALS herbicides (Holt and Lebaron 1990). Repeated use of herbicides with similar modes of action have resulted in an increase of weeds resistant to those herbicides (Holt 2012). As herbicide-resistant wild oat continues to reduce yield in cereal crops, there is a need to seek new modes of action and other means of control as well as to revisit previously used herbicides. Although most growers are opposed to double incorporation, integrating triallate back into cropping systems is an option in conventional tillage systems to control wild oat in cereal production. The objective of this

research was to determine efficacy of triallate in the spring wheat growers control strategies for wild oat control.

LITERATURE REVIEW

Wheat

World wheat production for the 2015 harvest was estimated to be 727 billion kg, and the United States (U.S.) ranked fifth in wheat production with 56 billion kg of grain (USDA 2016). Six classes of wheat are grown in the U.S.: hard red winter (*Triticum aestivum* L.), hard red spring, hard white spring, soft red winter, soft white spring, and spring durum wheat (*Triticum durum* L.) types. North Dakota produces durum, hard red spring, and hard red winter wheats and is ranked first in the U.S. for wheat production (USWA 2013). Specific wheat varieties are grown for certain food production uses based on different dough characteristics of the processed wheat flour (Vocke 2013). Hard wheat contains higher protein levels, produces more gluten, and is critical for yeast-raised baked goods due to the elastic component of dough which can capture and hold CO_2 (USWA 2013). Soft wheat contains a greater percentage of carbohydrates and less gluten-forming protein and generally is preferred for producing cake and pastry flour (Dexter et al. 2006). Finally, durum spring wheat is used to make pasta products due to density, high protein content, and gluten strength characteristics.

Wheat harvest in the U.S. reached its highest point during 1981, when 76 billion kg of grain were produced (USDA 2016). During the 1980s, wheat-fallow rotations were the dominant cropping rotation in the Great Plains of the U.S., due to the ability in conventional tillage cropping systems to conserve soil water, build up available soil nitogen, increase the ease of seeding, and promote weed control (Johnson and Ali 1982). Fallow reduced weed densities for the subsequent crop, but did not provide an economic return. The introduction of imidazolinone-, glyphosate-, and glufosinate-resistant crops such as canola (*Brassica napus* L.),

corn (*Zea mays* L.), lentil (*Lens culinaris* L.), and soybean (*Glycine max* (L.) Merr) have reduced the amount of wheat grown in North Dakota (USDA 2013).

Wheat is native to southwest Asia and evidence of its first cultivation 10,000 yr ago has been found in the Fertile Crescent region of the Middle East (Gibson and Benson 2002; Lev-Yadun et al. 2000). Over time, wheat has been selectively bred to tolerate alkali soil, disease, drought, and many other conditions (Charmet 2011). Wheat is an erect, annual, self-pollinated grass with a hollow stem that typically reaches 1 m in height (Beckie et al. 2012). Spring wheat is typically planted one to two inches deep with an optimum seeding rate of 370 plants m^{-2} (Mueller 2014). Optimum temperature for germination is approximately 12 C because wheat is a cool season crop (Acevedo et al. 2002).

Seedling leaves twist slightly clock-wise and tillers begin to emerge as early as the three-leaf stage (Herbek and Lee 2009). The number of kernels per head are determined when the plant reaches the jointing stage (Beckie et al. 2012). The plant enters the boot stage once the flag leaf has emerged and the developing head begins to swell inside the sheath. Wheat leaves are flat, narrow, and approximately 20 to 38 cm long and 1 cm wide. Anthesis, or open flowering, occurs when wheat spikes have completely emerged from the sheath (Setter and Carlton 2000). Each spike includes long, slender, spikelets that contain two to four fertile florets (Kirby 2002). Each floret is enclosed by a lemma and palea and awns are present (<1.3 cm long) (Setter and Carlton 2000). Following anthesis, grain filling begins. The endosperm becomes firm, the grain is a golden-yellow color, and kernel moisture decreases to about 30 to 40% at the time of maximum grain weight, or physiological maturity (Kirby 2002). Shortly thereafter, kernel dry weight declines rapidly and once 14% moisture is reached, kernels are ready for harvest and storage (USASK 2016).

Grass Weed Control in Wheat

Grass weeds tend to mimic cereal crops in their emergence timing and growth habits and are therefore difficult to control when the relative time of emergence coincides with planting and emergence of spring-seeded cereals (Ahrens and Her 1991). More than $500 million are expended on herbicides annually in Canada to control wild oat (Evans et al. 1991). In the Great Plains region of the U.S., growers experience an economic deficit of over $1 billion annually due to wild oat control costs and yield loss in annual crop production (Evans et al. 1991).

Several physical, cultural, and chemical approaches are available to producers to effectively manage weeds in cereal crops (Beckie 2006). Physical control options to manage the seed bank include tillage, mowing, hand-picking weeds, and burning windrows shortly after harvest to reduce viable seed left on the soil surface. Cultural control methods include grazing, crop rotation, and planting a fall cover crop to outcompete weeds (Warwick 2014). Increased crop seeding rate would also benefit the competitiveness of wheat to wild oat (Carlson and Hill 1985). Physical and cultural methods can become time consuming and often are less effective when compared to chemical control options. Herbicides with a single site of action are commonly used to control various weeds in wheat in North Dakota (Zollinger et al. 2016).

Herbicides that inhibit Acetyl CoA carboxylase (ACCase) prevent production of membrane lipids and are used to control certain grass weeds in cereal crops (Gonsolus and Curran 1999). The herbicide is absorbed by the foliage and translocated to the meristematic tissue through the phloem, causing newer leaf tissue development to be stunted and prevents cell division and elongation which causes plant to become weak and rot (WSSA and HRAC 2014). Generally, most broadleaf plants are naturally tolerant to ACCase herbicides and certain grasses can also be tolerant due to physiological differences between species (Gonsolus and Curran

1999). Symptoms of susceptible grasses, such as chlorosis and necrosis first develop on newer leaf tissue several days after treatment (DAT). Eventually, the growing point is affected as indicated by the damaged whorl.

Acetolactate synthase-inhibiting (ALS) herbicides affect enzymes in the biosynthetic pathway of the branch-chain amino acids isoleucine, leucine, and valine, resulting in plant deprivation for amino acids, and eventually, causing plant death (Green 2007). ALS herbicides labeled in wheat are available as soil or postemergence (POST) application for grass and broadleaf control (Gonsolous and Curran 2016). Grass leaves develop interveinal chlorosis, red leaf venation, and necrotic leaf margins, which eventually leads to plant death (Wall 1995). Susceptible broadleaf weeds develop chlorosis, spotting, and leaf distortion once the meristem is dysfunctional.

Microtubule inhibitor chemistry affects cell membrane and cell wall construction, inhibiting seedling root growth and these herbicides are labeled for control of many small seeded broadleaf and grasses weeds in cotton (*Gossypium hirsutum* L.), oilseed crops, soybean, and wheat (Vaughn and Lehnen 1991). Microtubule herbicides do not control wild oat as well as grasses such as barnyard grass (*Echinochloa crus-galli* L.), downy brome (*Bromus tectorum* L.), and green (*Setaria viridis* L.) and yellow foxtail (*Setaria pumila* Poir.). The herbicide is absorbed through emerging plant roots, but the herbicide active ingredients have limited translocation via shoots due to their highly lipophilic characteristics (Anthony and Hussey 1999). Symptoms result from the herbicide binding to tubulin, which is required in spindle fiber production and other major microtubule proteins (Vaughn and Lehnen 1991). This prevents the alignment and separation of chromosomes during mitosis preventing cell plates from forming. Most susceptible weeds fail to emerge due to inhibition of coleoptile growth and hypocotyl

extension (Parka and Soper 1977). The base of grass plants and broadleaf hypocotyls may become swollen, brittle, and turn purple and roots appear short and thickened (Vaughn and Lehnen 1991).

Very long-chain fatty acid inhibitors (VLCFA), such as pyroxasulfone, are labeled in wheat to control annual grasses and some broadleaf weeds (Anonymous 2016d). Grasses absorb the herbicide through the meristem and coleoptile after germination, generally causing emergence to fail (Tanetani et al. 2009). However, if the grass does emerge, "buggy-whip" symptoms occur (Gunsolus and Curran1999). This is where young leaves in the whorl continue to grow rapidly, but are unable to emerge from the twisted upper leaves. Broadleaf weeds absorb these herbicides through the root and symptoms include stunting, red leaf, leaf distortion, chlorosis, and necrosis.

Glyphosate, an EPSPS (5-enolpyruvyl- shikimate-3-phosphate) inhibitor, is a foliar-applied, non-selective herbicide used to control perennial and annual weeds (Gonsolous and Curran 2016). Glyphosate is labeled in corn, soybean, small grains, pasture, and noncropland areas as either pre-plant and/or pre-harvest applications. The herbicide is absorbed through the plant cuticle and transport is typically slower than most herbicide translocation (Green 2007). This herbicide leads to depletion of the aromatic amino acids in the shikimate pathway, which are all needed for protein synthesis. EPSPS catalyzes the transfer of the enolpyruvyl moiety of phosphoenolpyruvate (PEP) to the 5-hydroxyl of shikimate-3-phosphate (S3P) (Funke et al. 2006). Deregulation of the shikimate pathway ultimately causes plant death. Plant growth becomes stunted and yellow immature leaves and growing points appear first, followed by chlorotic and necrotic symptoms 10 to 14 DAT (Reddy and Zablotowicz 2003).

Wild Oat

Wild oat has evolved closely to mimic life cycles of grass crops such as wheat, making wild oat control in wheat more difficult compared to other grass weeds (Warwick 2014). Wild oat has invaded 11 million ha of land within the U.S. (Evans et al. 1991). Wild oat is a very troublesome weed in cereals because of the direct competition with crop plants for moisture, light, and nutrients (Carlson and Hill 1985). Competition causes yield reductions between 10 and 60%, depending on the wheat variety, seeding rate, and environmental factors (Beckie et al. 2012).

Wild oat is an erect, annual, self-pollinated grass reaching approximately 1 m in height (Beckie et al. 2012). Wild oat prefers similar cool and moist germination conditions to wheat and is typically found in cropland and other disturbed areas, and prefers cool and moist conditions. Once the minimum temperature for wild oat germination (~10 C) occurs in early spring, a fibrous root system quickly establishes (Beckie et al. 2012). Seedling leaves distinctively twist counter-clockwise and are slightly hairy. Leaf blades are flat and wide, tall and membranous ligules are present, and auricles are absent.

Wild oat inflorescences are open panicles with two to three seeds per spikelet, producing approximately 100 seeds per plant (Sharma and Vanden Born 1978). Flower and seed production occurs from June through August. The lemma have distinctive, bent, twisted awns that coil and uncoil as moisture changes, pushing the seeds down into cracks and crevices in the soil (Muzic 1970). Fecundity, seed shatter, and large, persistent seed banks with variable degrees of primary seed dormancy are key survival traits for wild oat (Beckie et al. 2012). Seeds remain dormant for 2 to 3 yr in disturbed areas; however, seeds can remain viable in the soil for up to 6 yr in undisturbed soil (Sharma and Vanden Born 1978). Depending on

crop competition and abiotic factors, up to 20,000 seeds m^{-2} can be produced in infested areas. Seeds shatter around the parent plant and have the potential to spread further distances via humans, animals, and farm implements.

Herbicide Resistance

Herbicide-resistant weed populations have increased across North Dakota and Minnesota (Durgan 2002). Herbicide resistance is initially caused by a genetic mutation naturally found in a population, and this anomaly allows the weed to survive and reproduce after an herbicide application that is typically lethal to the susceptible population (Green 2007). Resistant phenotypes may build over a period of time due to selection pressure of herbicides (Ditomaso 2000). Selection pressure occurs as susceptible plants are controlled, which allows resistant plants to reproduce and increase the frequency of resistance in the area (Gonsolous 2014). Repetitive herbicide usage increases the selection pressure for a biotype to exhibit resistance, which can increase the possibility of cross or multiple resistance. Cross and multiple-resistances occur through various mechanisms (Beckie and Tardif 2012). Altered target site resistance occurs when small structural changes occur at the biochemical target site to restrict herbicide binding. Enhanced metabolic resistance is capable of deactivating herbicides or enhancing xenobiotic metabolism to protect the plant (Abhilash et al. 2009). Compartmentalization or sequestration occurs when a plant is capable of relocating an herbicide from susceptible sites within the plant to non-effected locations where the herbicide is harmless.

Cross resistance patterns in herbicide resistant weed biotypes depend on the type of resistance mechanism (Beckie and Tardif 2012). Cross resistance is classified as resistance among progeny to more than one herbicide by a single mechanism (Mengistu et al. 2003). The mechanism can be based on target site, such as a mutation, or non-target site, such as altered

metabolism or translocation (Beckie and Tardif 2012). Both enhanced metabolism and reduced translocation prevent phytotoxic levels of herbicide reaching the site of action. Enhanced metabolism is typically responsible for cross resistance across herbicide sites of action, whereas altered target site or translocation generally restricts resistance within the same site of action. Multiple resistance occurs when the progeny is resistant to two or more mechanisms and generally the result of sequential herbicide site of action selection or accumulation of resistant alleles in progeny as a result of pollen flow in outcrossing species (Mengistu et al. 2003; Beckie and Tardif 2012).

The lack of effective alternative herbicide sites of action in many new crops will continue the selection of such biotypes, further complicating weed management (Beckie and Tardif 2012). There is no simple solution to the problem of managing multiple resistant weed populations. Implementing cultural and physical methods and herbicide mixtures and rotations may have the greatest effect to delay resistance (Bekie 2006). In the following narritive, the progression of herbicide production and usage is summarized and how that has resulted in herbicide resistance.

The first thiocarbamate herbicide to be registered in the U.S. was EPTC in 1957 and was used to control grass and broadleaf weeds in several dicotyledons crops (Anonymous 2011; Timmons 2005). Triallate was commercialized in 1962 and was used to control wild oat in wheat and barley (Anonymous; Timmons 2005). Thiocarbamates are applied pre-plant incorporation (PPI) and require incorporation via mechanical or water (Anonymous 2016b). Herbicide uptake occurs through the germinating wild oat coleoptile, and susceptible seedlings often do not emerge (Kern et al. 1996). Resistance is known to occur in areas with more than 15 yr of triallate usage, including Alberta, Manitoba, and Saskatchewan in Canada and Idaho and Montana in the U.S. (Heap 2016c). Resistant coleoptile and shoot cells uptake triallate at a

slower rate, effectively reducing the formation of a triallate concentration gradient across the plasma lemma (Kern et al. 1996). Triallate resistance in plants is conferred by a reduced rate of metabolic activation. For example, triallate uptake and translocation was reduced about 30% in the resistant biotypes when compared to the susceptible.

Trifluralin, a microtubule-inhibiting herbicide, was commercialized in 1963 and labeled to control annual grasses and certain small-seeded broadleaf weeds in cereal crops in northern U.S. and Canada (Anonymous 2016c). The soybean market in southern U.S. relied heavily on microtubule-inhibiting herbicides during the 1970s and 1980s to control a variety of broadleaf and grass weeds (Anthony and Hussey 1999). This over-reliance resulted in herbicide resistance, which was conferred through a mutation in the α-tubulin and is either inherited as a single nuclear gene or as a semi-dominant trait as heterozygous plants have partial resistance to the herbicide (Anthony and Hussey 1999). Six weed species are currently resistant to microtubule-inhibiting herbicides in the U.S., including wild oat (Heap 2016f).

A number of ACCase-inhibiting herbicides were introduced in the 1980s and early 1990s and frequently provided excellent control (90-99%) of wild oat and green foxtail (*Setaria viridis* (L.) P Beauv.) in cereal, oilseed, and pulse crops (Beckie et al. 1999; Beckie et al. 2014). Fenoxaprop was registered in the U.S. in 1987 and quickly became the most widely used ACCase-inhibiting herbicide with over 1.2 billion ha treated annually by the early 1990s (Mengistu et al. 2003). Wild oat resistance to ACCase herbicides was first confirmed in Saskatchewan in 1989, and North Dakota confirmed ACCase-resistant wild oat in 1991 (Heap 2016b). The frequent usage of ACCase herbicides was associated with the occurrence of resistant wild oat (Beckie et al. 2014). Beckie et al. (2014) estimated that one in every nine fields where annual crops were grown (2.4 million ha) in Saskatchewan had ACCase-resistant

wild oat. Fifteen monocot weeds have been identified with resistance to ACCase-inhibiting herbicides in the U.S. (Heap 2016b).

The first ALS-inhibiting herbicide, chlorsulfuron, was commercialized in 1982 and used for broadleaf control (Anonymous 2016a; Tranel and Wright 2002). Imazamethabenz, was the first ALS-inhibiting herbicide commercialized to target wild oat in 1988 (Mengistu et al. 2003). ALS herbicides were desirable and widely used due to crop safety, activity of residue in soil, and wide application windows allowed on labels (Tranel and Wright 2002). Reliance on ALS herbicides for control in the 1990s has resulted in the occurrence of numerous resistant species, including wild oat. Resistance can either occur when the resistant biotype replaces ALS more rapidly than inhibition or have a greater capacity to metabolize the herbicide to a metabolically inactive form when compared to the susceptible biotype (Matthews et al. 1990). The U.S. currently has 19 monocot and 29 dicot weed species resistant to ALS-inhibiting herbicides (Heap 2016c). ACCase and ALS cross-resistant wild oat biotypes were first confirmed in 1994 in Manitoba and 2012 in South Dakota. Multiple resistant wild oat biotypes are also likely to be present in North Dakota and Minnesota.

Glyphosate was commercialized in the U.S. in 1974 and initially labeled as a PRE or post-harvest herbicide (Nandula et al. 2005). The introduction of transgenic glyphosate-resistant soybean in 1996 greatly increased the amount of glyphosate applied because growers favored the ease of flexible applications and the lower cost to control weeds (Green 2007). By 2004, herbicide-resistant canola, corn, cotton, and soybean were grown on approximately 59 million ha (James 2004). The reliance on glyphosate for weed control and limited use of alternative herbicide sites of action increased the selection pressure of glyphosate-resistant weed populations. (Green 2007; Funke et al. 2006). The basis for glyphosate-resistant weed species

has been linked to an altered EPSPS target site, metabolic inactivation, and gene amplification in different species (Dill 2005). The U.S. currently has 10 dicot and 6 monocot weed species resistant to glyphosate (Heap 2016d).

Between 1991 and 2001, POST applications of ACCase, ALS, EPSPS, and glufosinate (glutamine synthase inhibitor) were used for better wild oat control, reducing PRE applications and restricting herbicide and crop rotations. Glufosinate and glyphosate were used in herbicide-resistant crops to control weeds, such as wild oat. Wild oat biotypes have been confirmed to be resistant to ACCase herbicides in both North Dakota and Minnesota and to ALS herbicides in North Dakota (Heap 2016c). ACCase and ALS cross-resistant wild oat restricts available herbicide and crop rotations, leaving triallate as the only herbicide site of action to effectively control resistant biotypes. Responsible crop rotation and herbicide mode of action selection allows triallate as an option for wild oat control in North Dakota and Minnesota.

Triallate

Triallate is a selective thiocarbamate herbicide that inhibits fatty acids and lipid biosynthesis (Shaner et al. 2014). Triallate is sold as Fargo® in either granular of liquid formulatins (Anonymous 2016b). The herbicide is microbially degraded in the soil, has an average half-life of 68 d, and a vapor pressure is 1.1 $x10^{-4}$ mmHg (Shaner et al. 2014). Labeled by the US-EPA for use in cereal crops, triallate provides an average of 80 to 90% control of wild oat (Anonymous 2016b). Triallate requires PPI 8 to 10 cm deep immediately following application to minimize volatilization losses. Losses are substantial when triallate is applied to warm soils and not properly incorporated (Anonymous 2011). The incorporated vapors are able to move small distances in soil, which greatly increases the soil and weed seed volume contacted with critical concentrations of triallate for wild oat control (Miller and Nalewaja 1976).

Above ground wild oat foliage was not effected when exposed to triallate vapors in research trials; therefore, due to the proximity to the weed growing point, contact with tissue below the soil surface is the primary site of triallate vapor action (Miller and Nalewaja 1976). Soil moisture, type, and temperature all influence wild oat control from triallate vapors. Triallate is absorbed through the emerging coleoptile of susceptible wild oat seedings and provides effective control before the first leaf emerges (Banting 1970). Wild oat coleoptiles become discolored, thick, and brittle (Fuerst 1987). Leaves generally do not emerge from the coleoptile; however, if leaves do emerge, "buggy-whip" symptoms typically occur. Some root growth inhibition is present and results in an absent secondary root system.

Triallate provided 18% better wild oat control when applied to tilled soil compared to stubble (Miller and Nalewaja 1980). The field should be tilled twice if stubble is present with either a field cultivator or a chisel plow to provide adequate working conditions and residue burial before the triallate application (Anonymous 2016b). Triallate should be incorporated immediately after application using a harrow or cultivator 5 cm deep at an angle to ensure thorough mixture. Shallow tillage, such as a harrow, is required for incorporation when applied after planting (Miller and Nalewaja 1980).

Objectives

Wild oat has developed resistance to triallate in Idaho and Montana in the U.S., Canada, and other regions of the world (Heap 2016a). Evaluating wild oat response to triallate will validate the utility of this treatment in North Dakota and Minnesota. In addition, modern wheat cultivars have not been evaluated for their phytotoxicity response to triallate. Answers to these two issues will help verify the potential of triallate for future control of wild oat in this region. Wild oat control and wheat cultivar tolerance to several triallate rates were evaluated. Suspected

ACCase and ALS resistant wild oat seed samples were collected in various locations throughout North Dakota and Minnesota. Seed from samples were screened in the greenhouse to characterize response to various herbicide sites of action and determine triallate efficacy. Successful screening of reported difficult-to-control wild oat biotypes and precise evaluation of new cultivar tolerance to triallate will provide useful information to growers for use in their management decisions regarding wild oat control in wheat.

MATERIALS AND METHODS

Field Trials

Wheat response to triallate. Crop injury was evaluated with several rates of triallate using six hard red spring wheat cultivars (Barlow, Faller, Glenn, Prosper, Stingray, and SY Soren) in 2014 near Fargo and Prosper, ND. The Fargo soil was silty clay with 7.2 pH and 6.8% OM (NRCS 2013). Experimental design was an RCBD with a split-block arrangement and four replicates. Factors of cultivar and triallate rate were included in the split-block arrangement, with variety as the whole plot and triallate rates as the sub plot. Experimental units were 2 m long by 2 m wide. After incorporation of herbicide with an S-tine cultivator, wheat cultivars were planted in 2-m-wide strips perpendicular to triallate applications. Studies in 2015 were established at Fargo and Prosper, ND. The hard red spring wheat cultivar Prosper was seeded in each 3 m wide by 9 m long plot and established near Fargo and Prosper, ND. The experiment was an RCBD with four replicates.

Application procedures were the same as previously stated for triallate with rates of 0, 840, 1120, 1680, and 2240 g ha^{-1} in 2014 and additional treatments of 3360 and 4480 g ha^{-1} in 2015. At the two-leaf stage, plant populations (plants m^{-1}) were recorded at two random locations within each treatment and averaged. Estimates of visible crop injury were completed 14 and 35 DAT on a visual scale of 0 to 100% wheat biomass reduction compared to the nontreated control. Weeds were controlled with herbicides appropriate to species present when wheat was in the three-leaf stage. Studies completed in 2014 were not harvested due to the small size of the sub-plots; however, studies in 2015 were harvested as previously stated with a combine.

Data were combined for analysis when the variance of each run was determined similar by comparing mean square error values (within a factor of 10). Data were subjected to analysis of variance in SAS. Experimental run was considered a random effect and herbicide treatment as a fixed effect. Interaction of cultivar and triallate rate was identified if p-value ≤ 0.05 in 2014. Data were combined across environments in 2015.

Soil herbicides for wild oat control in wheat. Field experiments to evaluate efficacy of triallate and other soil herbicides to control wild oat were conducted in areas with natural wild oat infestation near Fargo, ND and Nielsville, MN in 2014 and Fargo and Prosper, ND in 2015. The Nielsville soil was silty clay loam with 8.3 pH (NRCS 2013). The Fargo soil was silty clay with 7.5 pH and 6% organic matter (OM) and the Prosper soil was sitly clay loam with 7.5 pH and 3.5% OM. Each field site experimental design was a randomized, complete-block design (RCBD) with four replicates. Each experimental unit (plot) was 3 m wide by 9 m long, with the center 2 m treated with herbicide for the length of the plot. Treatments included triallate at 560, 840, and 1120 g ai ha^{-1}, flucarbazone, propoxycarbazone, and pyroxasulfone. These PPI and PRE herbicides were included in the experiment to demonstrate the benefit of controlling weeds early in the season, minimizing weed-crop competition. Treatments were applied using a CO_2-pressurized backpack sprayer and boom system with TurboTee 11001 (TeeJet, Spraying Systems Co. 200 W. North Ave, Glendale Heights, IL 60139) nozzle tips at a pressure setting of 276 kPa to deliver 80 L ha^{-1} with the applicator walking approximately 5 km h^{-1}.

Wild oat control is at its greatest when triallate application, incorporation, and planting all occur on the same day (McMullen and Nalewaja 1990). Triallate applications were applied to the soil prior to planting and immediately double incorporated with an S-tine cultivator (11 Series Integral [Light Duty] Field Cultivators. Des Moines Works OMN159448 Issue J7) in

2014, and a rototiller (370 [No. 002800] Deere & Company. Moline, IL 61265) in 2015 with tractor speeds approximately 10 km h^{-1}. The hard red spring wheat cultivar 'Prosper' was seeded at a rate of 1 million seeds ha^{-1} perpendicular to the treatment direction. Pre-emergence treatments were applied to the soil directly after seeding.

Seedling emergence populations were evaluated when the wheat reached the two-leaf stage. Number of plants in 1 m of row were counted at two random locations within each plot. A mid-season application was applied to the experiments to control broadleaf weeds that were competing with the wheat. Wild oat control was visually estimated 14 and 28 DAT on a 0 to 100 scale, where 0 was unaffected (nontreated control) and 100 was plant death. Once wheat reached physiological maturity, 14 m^2 of each plot were harvested with a plot combine (Hege 125B; WINTERSTEIGER Inc. 4705 W. Amelia Earhart Drive, Salt Lake City [USA], UT 84116-2876) with a 2 m wide grain header. Grain was cleaned of chaff, weed seed, and other foreign materials before weight was recorded for yield calculation.

Data were combined for analysis of variances for each year and were determined similar by comparing mean square error values (within a factor of 10). The nontreated and three rates of triallate were run as a regression. The nontreated, standard field rate for triallate, and PRE treatments were subjected to analysis of variance in SAS (Statistical Analysis Software, version 9.4. SAS Institute, Inc., 100 SAS Campus Dr., Cary NC 27513). Experimental run was considered a random effect and herbicide treatment as a fixed effect. Means were separated by Fisher's protected LSD with α=0.05.

Greenhouse Trials

Characterization screening of wild oat collections. Wild oat seed samples were solicited from growers, crop consultants, and extension county agents that extended from Cavalier to

McIntosh County and east and along Highway 2 out to Williams County in North Dakota (Appendix 1A). Samples were also collected in the northwestern counties of Minnesota. These samples were difficult to control with ACCase and/or ALS-inhibiting herbicides based on grower experience. A characterization screening was conducted in the greenhouse to determine response to ACC-ase, ALS, or EPSPS herbicides (Table 1). This study was a complete block design (CBD) consisting of one replicate, eleven treatments, and repeated.

Table 1. Herbicides to characterize 56 wild oat samples from locations in North Dakota and Minnesota collected in 2014.

Treatment	Rate	Site of action	Chemical family
	g ha^{-1}		
Clethodim[a]	70	ACCase[c]	Cyclohexanedione
Clodinafop	560	ACCase	Aryloxyphenoxy propionic acid
Fenoxaprop	93	ACCase	Aryloxyphenoxy propionic acid
Pinoxaden	60	ACCase	Phenylpyrazolin
Flucarbazone[a]	30	ALS[d]	Sulfonylaminocarbonyltriazolinine
Imazamox[a]	35	ALS	Imidazolinone
Pyroxsulam[a]	18	ALS	Triazolopyrimidine
Rimsulfuron[a]	17	ALS	Sulfonylurea
Thiencarbazone	5	ALS	Sulfonylaminocarbonyltriazolinine
Glyphosate[b]	840	EPSPS[e]	Amino Acid Derivative

[a] Treatments include NIS, nonionic surfactant at 0.25% adjuvant.
[b]Treatments include AMS, ammonium sulfate.
[c]ACCase, Acetyl CoA Carboxylase.
[d]ALS, Acetolactate synthase.
[e]EPSPS, 5-enolpyruvyl-shikimate-3-phosphate.

Following collection, seeds were stored at room temperature for one to two months to reduce physiological dormancy before being de-hulled. Seeds were placed in a petri dish lined with Whatman No.9 filter paper (GE Healthcare Amersham Place Little Chalfont, Buckinghamshire, UK), saturated in distilled water, and incubated on damp filter paper in the dark at ~5 C for 24 h before planting. Scarification was required to induce germination, which was accomplished by piercing the dorsal side of the seed with a sterile needle. Seeds were incubated at ~5 C for another 48 h. Peat-based soil mix (Sunshine Mix No. 1. Sun Gro

Horticulture Distribution, Inc., 770 Silver St., Agawam, MA 01001) with wetting agents was placed into pots (TO plastics 450) measuring 10 cm wide by 15 cm long by 5 cm deep and 10 wild oat seeds were placed at a depth of 3 cm. Pots were maintained in the greenhouse at 25 C ± 5 C. Natural light was supplemented with metal halide lights (Phillips Lighting Company, 200 Franklin Square Drive, Somerset, NJ 08873) with an intensity of 200 µmol m^{-2} s^{-1} to maintain a 16 h photoperiod. Watering daily with tap water to the soil surface was necessary to prevent excessive soil drying. Liquid fertilization was mixed using Miracle-Gro® ([24-8-16] Miracle-Gro Products Inc. P.O. Box 267, Marysville, OH 43041) concentrate at a rate of 3 g L^{-1}. Pots were fertilized weekly with this solution to avoid nutrient deficiencies. Commercial formulation of herbicides with required NIS (R-11, a nonionic surfactant, is a blend of alkylphenol ethoxylate, butyl alcohol, and dimethylpolysiloxane. Wilbur Ellis Company 345 California Street, San Fransisco, CA 94104) at 0.25% v/v were applied to treatments (Table 1), using a chamber sprayer (Research Track Sprayer, model number SB8-095. DeVries Manufacturing. Minneapolis, MN.) delivering 93 L ha^{-1} through a 650067 even flat-fan tip (Spraying Systems Co., P.O. Box 7900, Wheaton, IL 60189) once plants reached the two-leaf stage (Beckie et al. 2000). Pots were spatially randomized weekly to minimize microenvironment effects, such as light and temperature differences.

The wild oat herbicides most used in the region, representing products whose wild oat control is attributed to each of three sites of action (ACCase, ALS, and EPSPS) were selected for the experiment. The visible condition of wild oat plants was evaluated at 14 and 28 DAT to determine wild oat control. Data were combined for analysis of variance in SAS. Wild oat biotypes were analyzed separately. Experimental run was considered a random effect and herbicide treatment was a fixed effect. Means were separated by Fisher's protected LSD with

α=0.05. Treatments indicated as controlled provided more than 60% wild oat control ratings 28 DAT.

Wild oat control with PPI and POST herbicides. The characterized wild oat samples were used to determine the efficacy of triallate. A greenhouse experiment was conducted using the experimental design of an RCBD study with three replicates, six treatments, and repeated (Table 2). Similar seed preparation procedures were conducted as previously indicated in the greenhouse characterization study. A non-bleached paper towel was laid on the bottom of each pot (TO plastics 601), measuring 10 cm long by 10 cm wide by 12 cm deep, to prevent soil from leaking through the drainage holes at the bottom. The day before seeding, 3 cm of sifted, nontreated, sandy loam soil (S & S Landscaping Co., Inc. 2777 Fiechtner Dr S Fargo, ND 58103) with 7.7 pH and 3.6% OM was placed in each pot.

Triallate at a rate of 1120 g ha^{-1} was applied through the chamber sprayer to the top of soil placed 5 cm deep in a tray. Soil was then transferred into a concrete mixer (3.5 cu. ft. model 31979. Central Machinery 3491 Mission Oaks Blvd., Camarillo, CA 93011) for 7 min to assure thorough incorporation. Each pot was filled with an additional 3 cm of soil either nontreated or treated with triallate, depending on the prescribed treatment, just before wild oat seeds were seeded. Enough distilled water was added to the soil to establish 80% field capacity of the entire pot volume. Five seeds were placed in each pot and 3 cm of either treated or nontreated soil was added to each pot. Capillary action and gravity drainage allowed water to distribute throughout the entire soil profile.

Table 2. Treatments to evaluate triallate efficacy to 56 wild oat samples collected in North Dakota and Minnesota in 2014.

Treatment	Rate	Timing[a]
	g ai ha^{-1}	
Triallate	1120	PPI
Triallate fb Pinoxaden	1120 fb 60	PPI fb POST
Pinoxaden	60	POST
Triallate fb Theincarbazone	1120 fb 5	PPI fb POST
Thiencarbazone	5	POST

[a]Abbreviations: PPI, pre-plant incorporated; fb, followed by; POST, post-emergence.

Wild oat plants were watered every other day as needed to maintain 80% field capacity. More frequent watering occurred once plants reached the two-leaf stage. The average weight of randomly selected pots was calculated to determine the amount of water required to maintain 80% field capacity. Pots were spatially reorganized on the greenhouse bench area every 7 d in a serpentine order within the replicate to minimize effect of microenvironment. The fertilizer solution previously described in the weed seed characterization study was applied weekly once plants reached the two-leaf stage.

Emergence and triallate efficacy was evaluated 14 DAT. POST applications were applied once nontreated wild oat plants reached the two-leaf stage with a chamber sprayer as previously described. Estimate of biomass reduction for wild oat control was visually evaluated 14 and 28 d after the POST applications. Wild oat biomass above the soil line was harvested and fresh weight was recorded. Samples were then dried at ~51 C before dry weight was recorded to determine treatment effect on wild oat.

Wild oat samples were grouped into three categories: wild oat that survived ALS and both ALS and ACCase applications and ones that were susceptible to both ALS and ACCase herbicides, based on the results from the characterization experiment. Data were combined for analysis when the variances of each run were determined similar by comparing mean square

error values (within a factor of 10). Data were subjected to analysis of variance in SAS. Experimental run was considered a random effect and herbicide treatment as fixed effect. Means were separated by Fisher's protected LSD with α=0.05.

RESULTS AND DISCUSSION

Field Research

Wheat response to triallate. An interaction was not present between cultivar and triallate rate (p value = 0.97); therefore, main effects were not confounded. Cultivars were analyzed across triallate rates, and a cultivar effect occurred (Table 3). Soren and Prosper cultivars had poor emergence of 25 and 28 plants m^{-1}, respectively, when compared to the other cultivars. The highest mean injury occurred with Stingray and Prosper cultivars at 44 and 39% 28 days after treatment (DAT), respectively. Stingray had a maximum injury of 80% at the labeled use rate for triallate (1120 g ha^{-1}), while Prosper was 50% and all other cultivars were 40% or less. Prosper's high injury potential means that if Prosper was deemed tolerant, then most cultivars should also be tolerant. However, there is the possibility a cultivar not included in this research could be less tolerant than Prosper since this experiment demonstrated variable cultivar response. Never the less, Prosper was selected as the single wheat cultivar for further tolerance research in 2015 because of reduced emergence and high injury potential.

Table 3. Wheat cultivar emergence and visible injury response averaged across triallate rates with data combined over Prosper and Fargo, ND, locations in 2014.

		Visible injury 28 DAT[a]	
Cultivars	Emergence[b]	Mean[b]	Maximum[c]
	Plants m^{-1}	----------% ----------	
Barlow	39a	29c	40
Faller	35ab	30c	40
Glenn	38a	30c	35
Prosper	28bc	39b	50
Stingray	39a	44a	80
SY Soren	25c	27c	25
LSD α=0.05	10	5	

[a]Abbreviations: DAT, days after treatment.
[b]Means separated by probability of difference. Means followed by the same letter are not different according to Fisher's protected LSD at α=0.05.
[c]Maximum injury at the triallate field rate of 1120 g ha^{-1}.

General wheat response in this study was a lack of emergence; however wheat seedling stunting also was recorded, especially when triallate rates were applied above 2240 g ha^{-1} (Table 3). Triallate injury to wheat has been reported to occur under cool, wet conditions and high soil temperatures (McMullen and Nalewaja 1990). Wheat phytotoxicity can also occur when seeded into shallow-cultivated, triallate-treated soil or when triallate has been incorporated too deep. These conditions place higher concentrations of triallate at the depth of wheat seedlings and reduce wheat emergence. The wheat coleoptile base remains near the caryopsis because the mesocotyl does not elongate (Carlson and Morrow 1986). Wheat solid stem characteristics may be the cause of triallate-tolerant hard red spring wheat cultivars. Diffusion through solid stem cells would be more difficult for triallate, which decreases the diffusion rate into seedlings (McMullen and Nalewaja 1990).

Prosper was used as the evaluated cultivar in the 2015 experiments because of greater susceptibility to triallate injury demonstrated in 2014. Wheat injury symptoms included stunting and poor emergence, which occurred in streaks consistent with incorporation direction within the treatments. Wheat emergence was reduced 14% when triallate was applied at 2240 g ha^{-1} compared with nontreated soil (Figure 1). Injury in streaks was attributed to inefficient incorporation, especially at higher rates, because of incomplete mixing with the S-tine cultivator operated at suboptimal speed. Injury was much less the second year as a more uniform distribution was with the rototiller occurred when compared to the S-tine cultivator. However, high rates of triallate caused reduction of wheat emergence. Injury greater than 15% did not occur until 2240 g ha^{-1} triallate was applied, which is twice the standard field rate, even though Prosper expressed up to 50% injury with triallate at 1120 g ha^{-1} in 2014 (LSD = 298). Yield also

started to trend downward when triallate at 2240 g ha^{-1} was applied, but was not less than nontreated wheat until treated with triallate at 3360 g ha^{-1}.

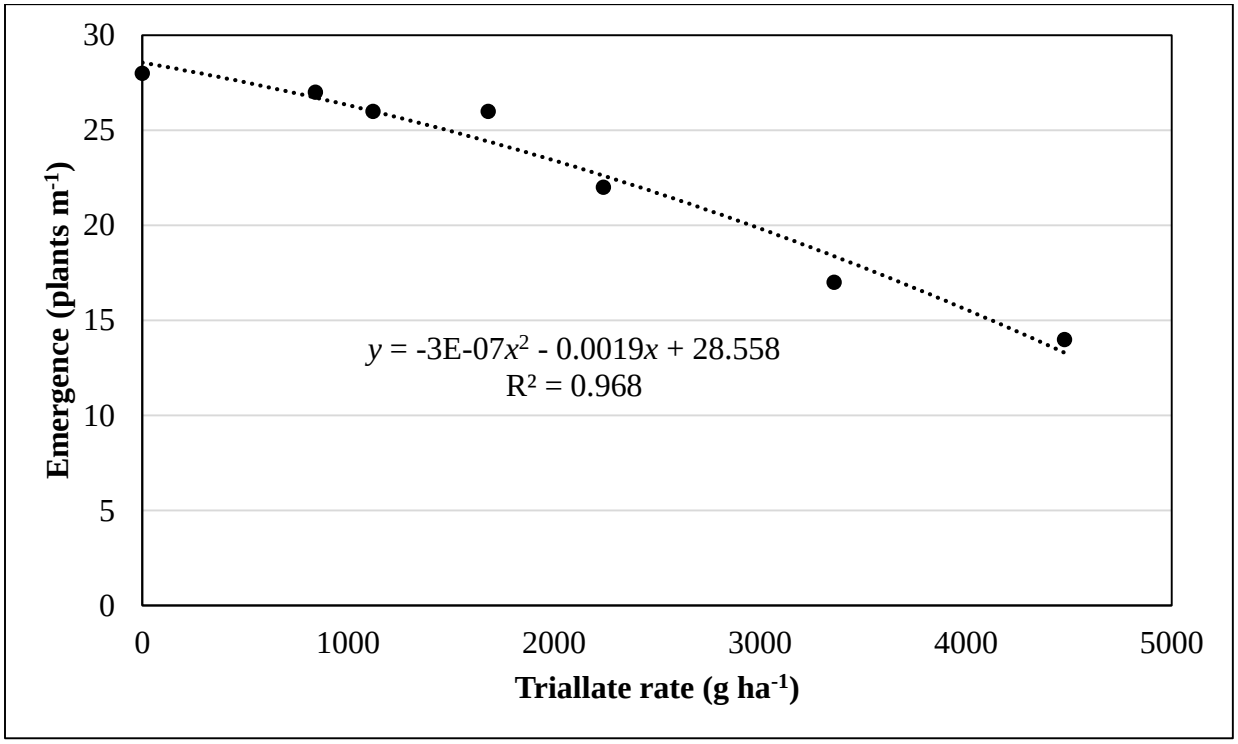

Figure 1. Wheat emergence response to triallate at Prosper and Fargo, ND, combined over locations in 2015.

The labeled rate of triallate in the soil utilized within the experiments was at 1120 g ha^{-1} in wheat (Anonymous 2016b). Higher rates were included in this experiment to evaluate the effect on wheat if application overlap or incorporation issues were to occur. Even though injury was observed at 1120 g ha^{-1} (Figure 2) and decreased plant population occurred at 2240 g ha^{-1} (Figure 1), final grain yield was not affected until 3360 g ha^{-1} (Figure 3), which is three times the labeled field rate. These results confirmed that little to no effect on wheat emergence, visible injury, or yield on newer wheat cultivars should be expected when triallate is applied correctly.

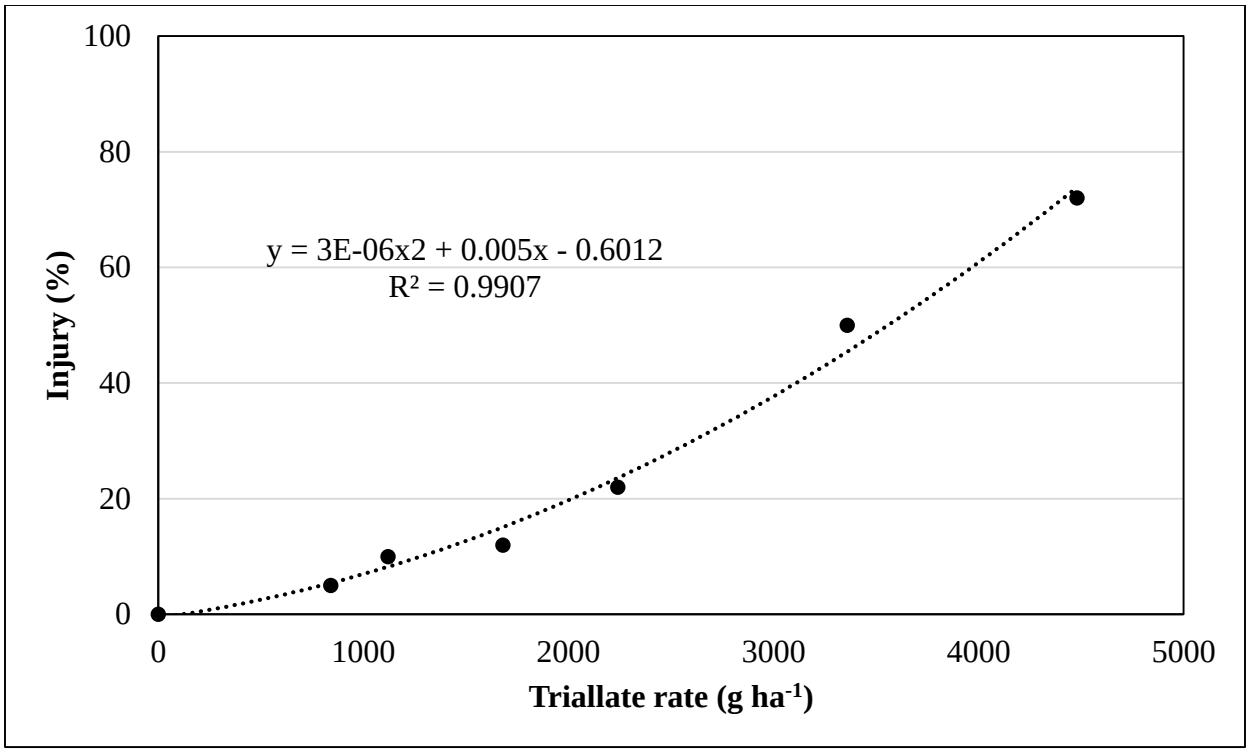

Figure 2. Wheat injury response to triallate at Prosper and Fargo, ND, combined over locations in 2015.

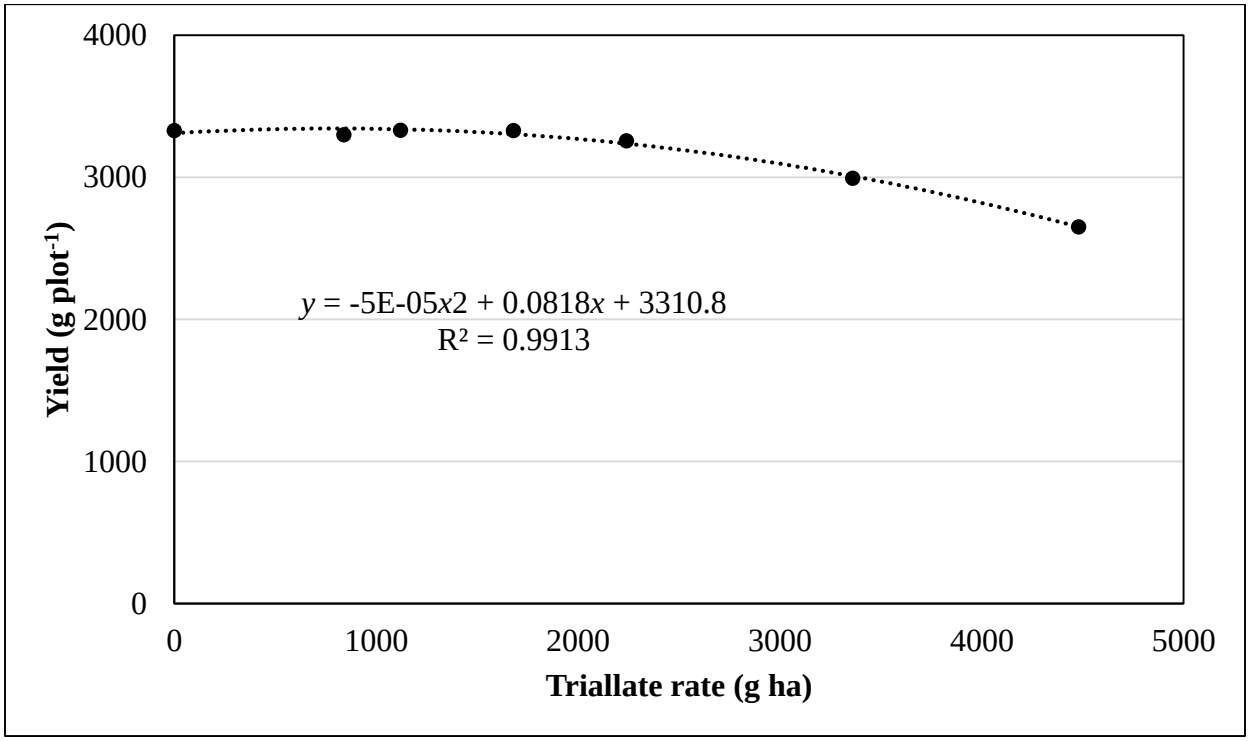

Figure 3. Wheat yield response to triallate at Prosper and Fargo, ND combined over locations in 2015.

PRE herbicides for wild oat control. Triallate at three rates were regressed against wild oat control or Prosper grain yield. Triallate rates included 560, 840, and 1120 g ha^{-1} and were indicated as low, medium, and high, respectively. These rates were included in this experiment to determine which rate had acceptable control levels. The high rate was enough to provide adequate control; however, there could be a benefit from applying a POST herbicide after a PRE. The high rate was also beneficial in resistance management. Below-label herbicide rates can initiate a rapid population response in certain weed biotypes (Norsworthy et al. 2012). Wild oat control increased as the rates of triallate increased from the low to high rate (Figure 4). The linear regression model for triallate rate accounted for 93% of the control response variance.

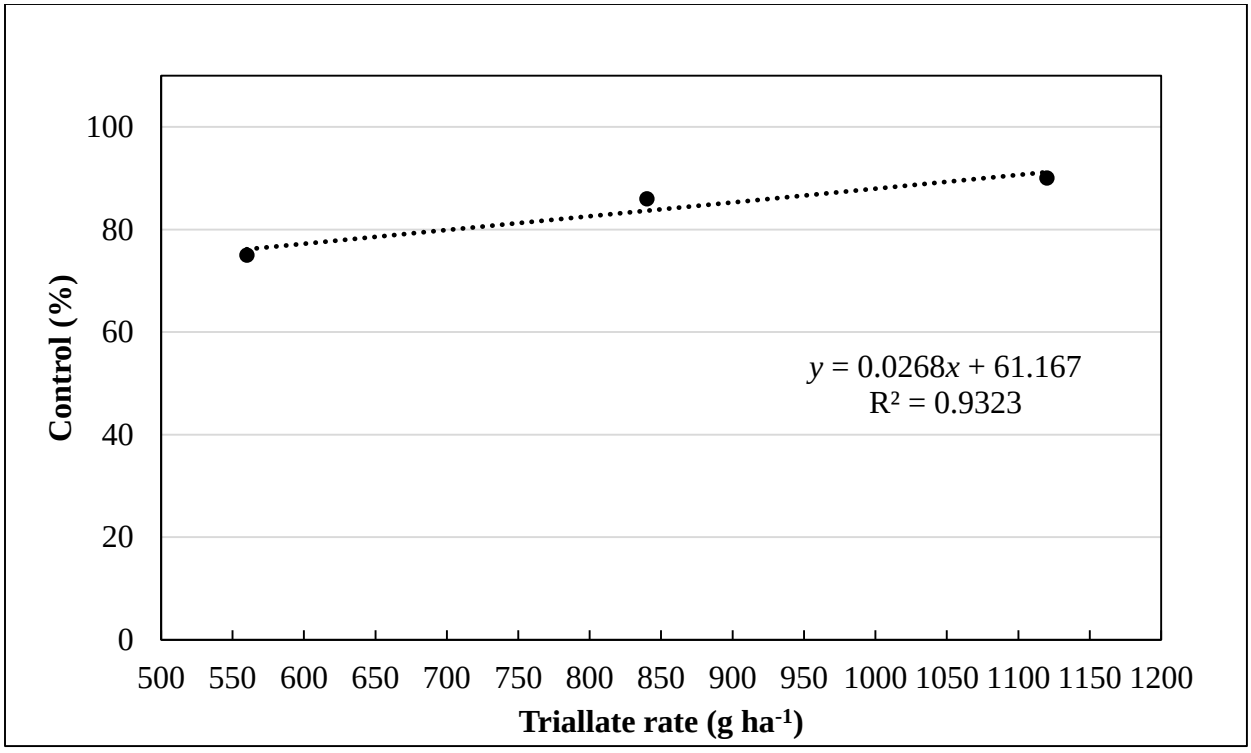

Figure 4. Wild oat control with increasing triallate rates at Fargo, ND and Nielsville, MN, in 2014 and Fargo and Prosper, ND, in 2015 combined over locations and years.

Wheat treated with the medium triallate rate had greater yield numerically when compared to the low and high rates; however, the triallate rate accounted for only 7% of yield response (Figure 5). The low coefficient of determination (r^2) value does not necessarily mean that there is a significant difference between the medium and high triallate rate. While the wheat response trial was conducted in a weed free environment, this experiment was established to evaluate weed control. Yield response was influenced by competition from wild oat that survived an herbicide application or emerged late. Environmental or production factors, such as the incorporation method, could have possibly occurred throughout the multiple locations over the two different years. Wheat injury and yield were not decreased until twice the labeled field rate was applied (Figure 5). Difference in crop safety and yield between the medium and high rates of triallate were small. However, triallate did increase wild oat control 28 DAT at the high rate when compared to the lower rates, even if there was not significance in control.

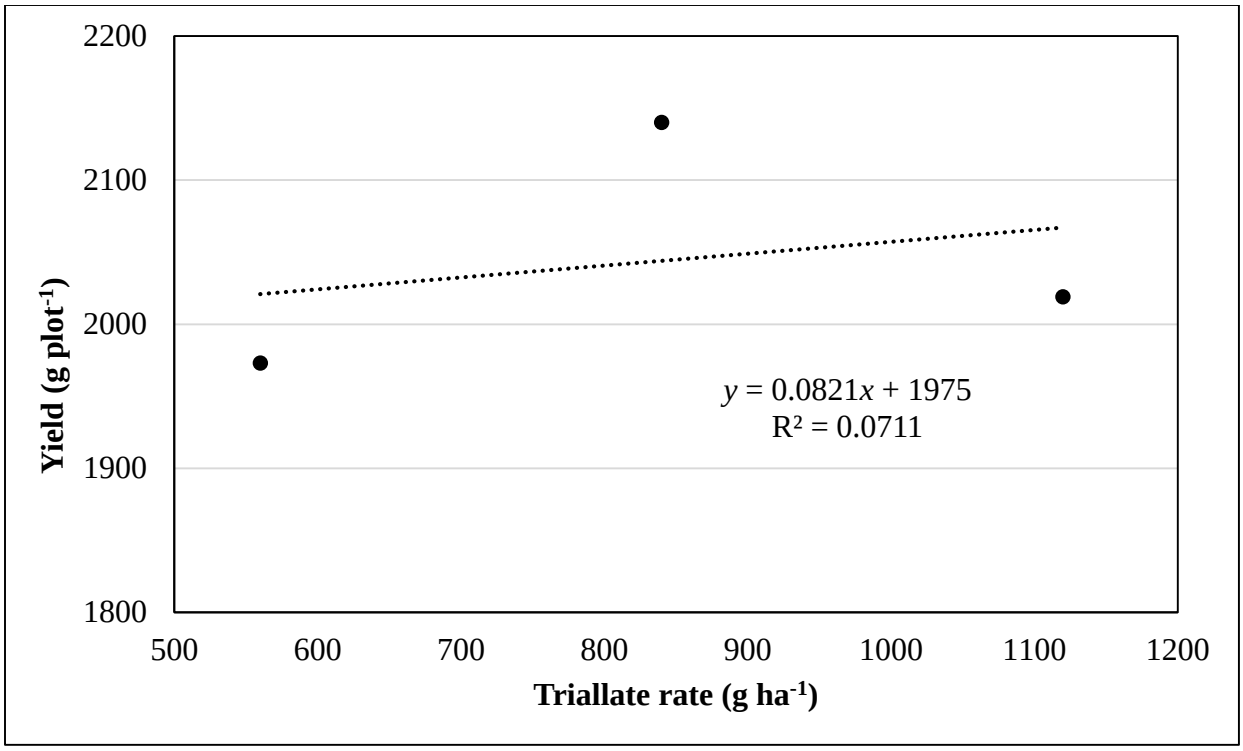

Figure 5. Wheat grain yield harvested following wild oat control with triallate at 560, 840, and 1120 g ai ha^{-1} at Fargo, ND and Nielsville, MN, in 2014 and Fargo and Prosper, ND, in 2015 combined over locations and years.

Triallate provided 90% control of wild oat at 28 DAT (Table 4). However, wild oat control with pyroxasulfone was very similar. Flucarbazone, propoxycarbazone, and pyroxasulfone had similar wild oat control; however, flucarbazone and propoxycarbazone gave poor control numerically. This reduction in wild oat control allowed more weed-crop competition and resulted in less grain for wheat with flucarbazone compared to pyroxasulfone. Plots treated with flucarbazone, propoxycarbazone, and triallate produced similar wheat yield to the nontreated. Triallate yidl was also similar to pyroxasulfone, which was applied at a higher rate than the labeled field rate (119 g ha^{-1}) because the experiment protocol was established on research before the label was approved; therefore, higher visible control ratings and yield were recorded than would be expected under the current label (Anonymous 2016).

Table 4. Visible wild oat control ratings and wheat yield at Nielsville, MN and Fargo, ND in 2014 and Fargo and Prosper, ND in 2015 combined over locations and years.

Treatment	Rate	Visible control[a]	Yield[a]
	g ai ha^{-1}	% of control	g $plot^{-1}$
Triallate	1120.0	90a	2019ab
Flucarbazone	14.7	67b	1809b
Propoxycarbazone	9.8	48b	1765b
Pyroxasulfone	196.0	89ab	2296a
Nontreated	0	0c	1866b
LSD α=0.05		23	289

[a]Means separated by probability of difference. Means followed by the same letter are not different at α=0.05 when using the Fishers protected LSD statistic.

Greenhouse Research

Characterization study. Wild oat samples were characterized for control with herbicides from three sites of action. Results were combined over two years of research. Wild oat health was slightly damaged in 2014 by thrips (*Heliothrips haimorrhoidalis*) that infested the greenhouse. Each wild oat sample was counted as controlled by the treatment if the treatment provided more than 60% wild oat control 28 DAT (Figure 6).

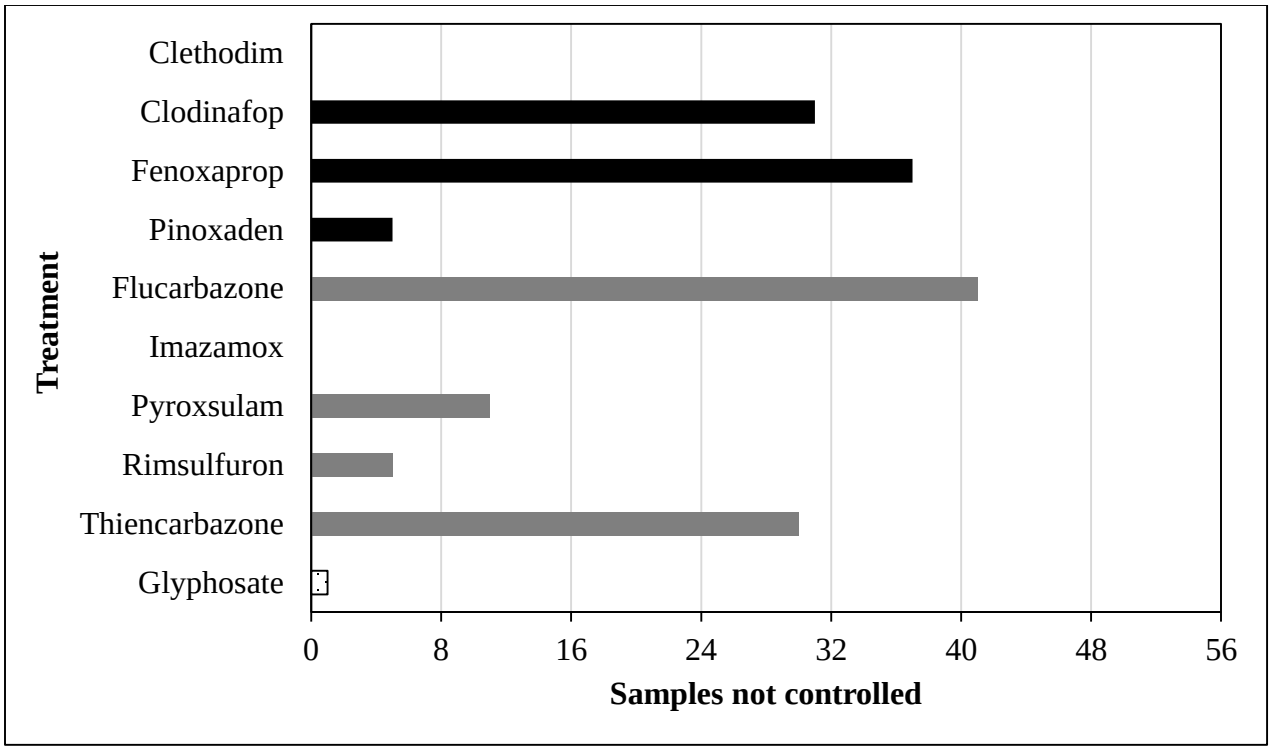

Figure 6. Number of the 56 wild oat biotypes not controlled with given herbicide by more than 60%. Black bars represent ACCase herbicides, grey represent ALS, and white bar represents EPSPS.

ACCase-inhibiting herbicides clodinafop and fenoxaprop controlled fewer wild oat samples than pinoxaden or clethodim (Figure 6). According to Beckie and Tardif (2012), clethodim is the ACCase-inhibiting herbicide at least risk to develop reisitance. Pinoxaden and clethodim were considered stronger wild oat herbicides than clodinafop and fenoxaprop. Their result was confirmed in this experiment with 5 and 0 wild oat samples not controlled by pinoxaden and clethodim, respectively.

Many wild oat samples were not controlled by ALS-inhibiting herbicides, except for imazamox, which controlled 100% of the wild oat samples (Figure 6). According to Beckie and Tardif (2012), imaxamox is a short residual ALS herbicide, which generally reduces selection pressure for resistance. Flucarbazone allowed substantial survival of 41 wild oat samples, more than any other herbicide. Glyphosate generally provided high wild oat control across all

samples; however, glyphosate provided 54% control to one wild oat sample. The wild oat within this sample were not chlorotic or necrotic like all other wild oat samples. Control in other crops is difficult to avoid resistance selection in certain cropping systems with herbicides such as clethodim, glyphosate, imazamox, and rimsulfuron. Due to the reliance on ACCase herbicides, other options are not available in many broadleaf crops grown in North Dakota.

Triallate study. Phytotoxic effects of triallate included brittle and thick coleoptiles followed by discolored and malformed leaf tissue that was also described by McKercher et al. (1975). Leaf tips became necrotic and secondary root systems did not develop, resulting in almost complete wild oat death. Occasionally, the coleoptile sheath would rupture below the tip of the coleoptile shoot causing unintended shoots to emerge. The tip remained trapped, resulting in arched leaf expansion, or buggy-whip effect.

There was an interaction between wild oat samples and herbicide treatments for wild oat control. Samples were analyzed within three characterization classes according to the previous study: survived ALS, survived both ACCase and ALS, and susceptible to both ACCase and ALS herbicides. Treatments that included triallate provided over 90% wild oat control to all three subgroups (Figure 7). Wild oat samples that survived ALS or both ACCase and ALS herbicides were controlled above 92% 14 DAT when treated with triallate.

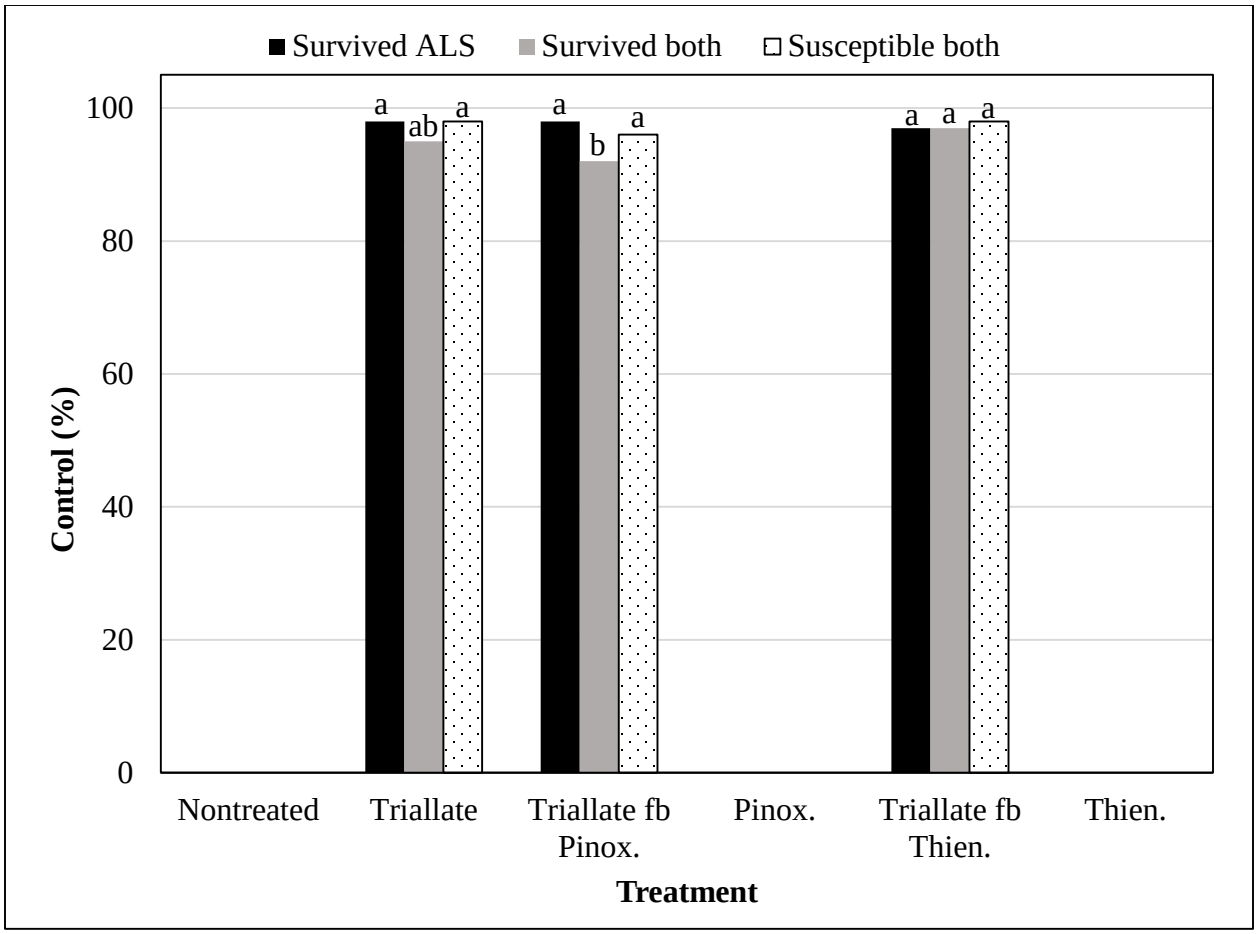

Figure 7. Wild oat control 14 DAT, after seeding, and preplant-incorporation application (but prior to pinoxaden and thiencarbazone application) with triallate for 56 samples in the greenhouse combined over two years. Samples were grouped into three subgroups according to level of susceptibility identified in characterization study to alleviate interaction of factors during analysis. Bars within subgroup with the same letter were similar control according to Fisher's-protected LSD at α=0.05. Abbreviations: fb, followed by; Pinox., Pinoxaden; Thien., Thiencarbazone.

Treatments that included triallate provided greater wild oat control than thiencarbazone, but not for pinoxaden, for wild oat samples that survived ALS and were susceptible to both herbicide groups (Figure 8). Wild oat that survived both ACCase and ALS herbicides were controlled with treatments that include triallate. Triallate control of wild oat tended to increase slightly between the first evaluation at 14 DAT (soil) and 28 DAT (foliar) for wild oat samples within each subgroup (Figures 7 and 8). Pinoxaden alone provided 85% wild oat control, which is numerically less control than treatments including triallate. Wild oat control with

thiencarbazone was lower than all other herbicide treatments. Pinoxaden generally provides excellent (> 90 %) control of wild oat and thiencarbazone provides good to excellent control (80 to 90%) (Zollinger 2016). Between the foliar applied treatments, pinoxaden gave substantially higher wild oat control than thiencarbazone. Wild oat growing points were damaged and leaves became chlorotic and necrotic 14 DAT when treated with pinoxaden. Thiencarbazone, an ALS-inhibiting herbicide, was chosen because it was fairly new and had not been studied extensively for wild oat control. Wild oat generally became stunted and brittle after thiencarbazone application. Necrotic skeletal frames developed in wild oat treated with pinoxaden; however, wild oat treated with thiencarbazone remained green.

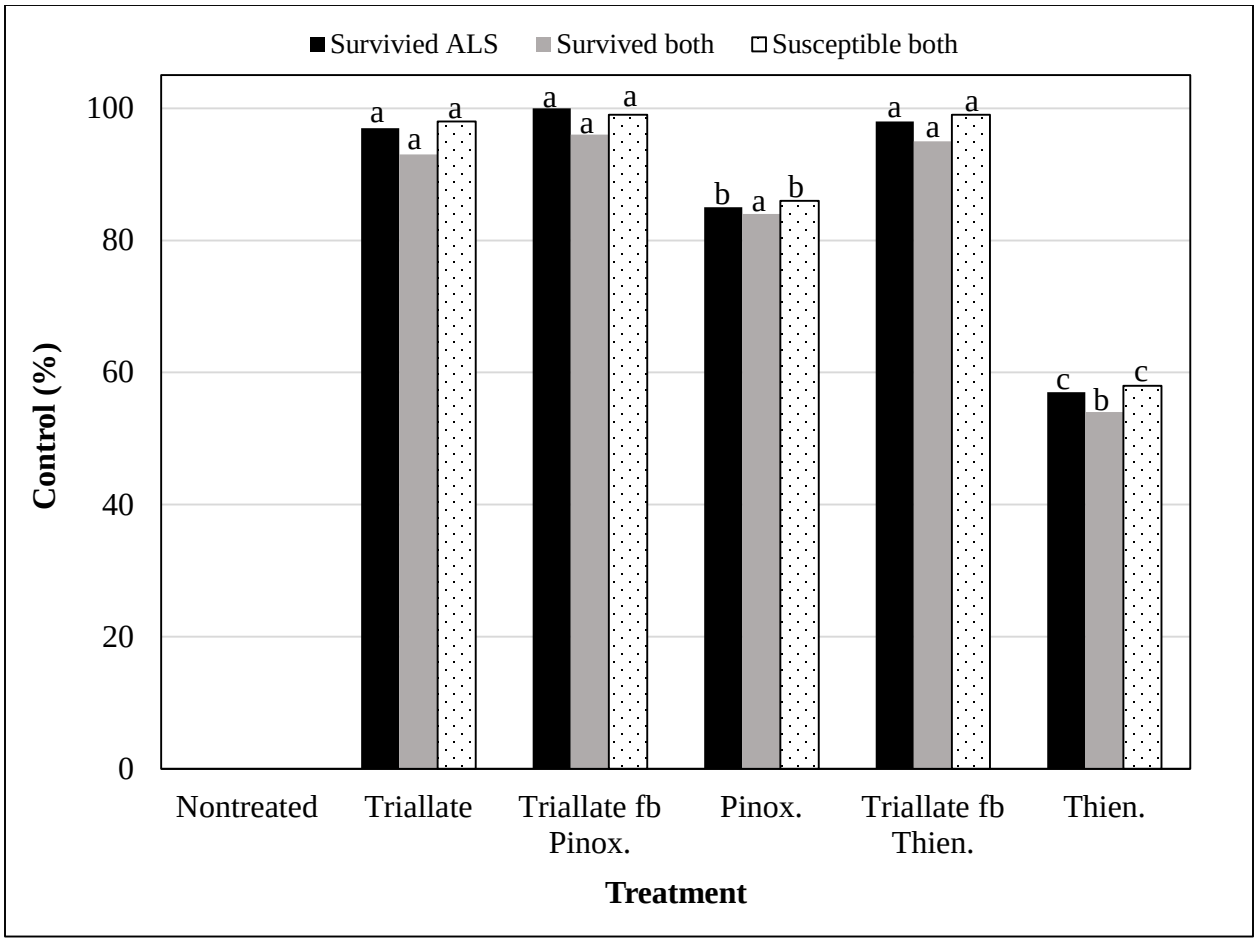

Figure 8. Wild oat control 28 days after post-emergence applications for 56 samples in 2014 and 2015 in Fargo greenhouse combined over both years. All samples grouped into three subgroups according to level of susceptibility from characterization study. Bars within subgroup with the same letter were similar control according to Fishers-protected LSD at α=0.05. Abbreviations: fb, followed by; Pinox., Pinoxaden; Thien., Thiencarbazone.

All biomass ratings for wild oat that survived treatment with ALS were significantly reduced when compared to the nontreated (Figure 9). Pinoxaden and thiencarbazone responded similarly to the triallate alone treatment, but had higher biomass when compared to either combination treatment. Wild oat biomass of plants in subgroups that survived both and were susceptible to both ACCase and ALS herbicides were significantly reduced when compared to the nontreated. However, all treatments that included triallate resulted in the least amount of biomass recorded.

All herbicide treatments significantly decreased wild oat biomass compared with nontreated wild oat (Figure 9). Treatments with pinoxaden or thiencarbazone alone resulted in greater biomass than all other treatments, which was expected. This is because the application occurred at the two-leaf stage where the plant had already established biomass compared to treatments that included triallate, which controlled wild oat during emergence. Certain weed species exhibit extended emergence patterns that continue to emerge following an early spring, non-residual POST application (Tharp and Kells 1999). Including triallate followed by POST treatments demonstrate the benefit of a second application to control possible seedlings that broke out of triallate damage, which would reduce the weed-crop competition. These applications are also important because multiple sites of action are applied to the wild oat, reducing the potential for the development of weed resistance to any of the herbicides.

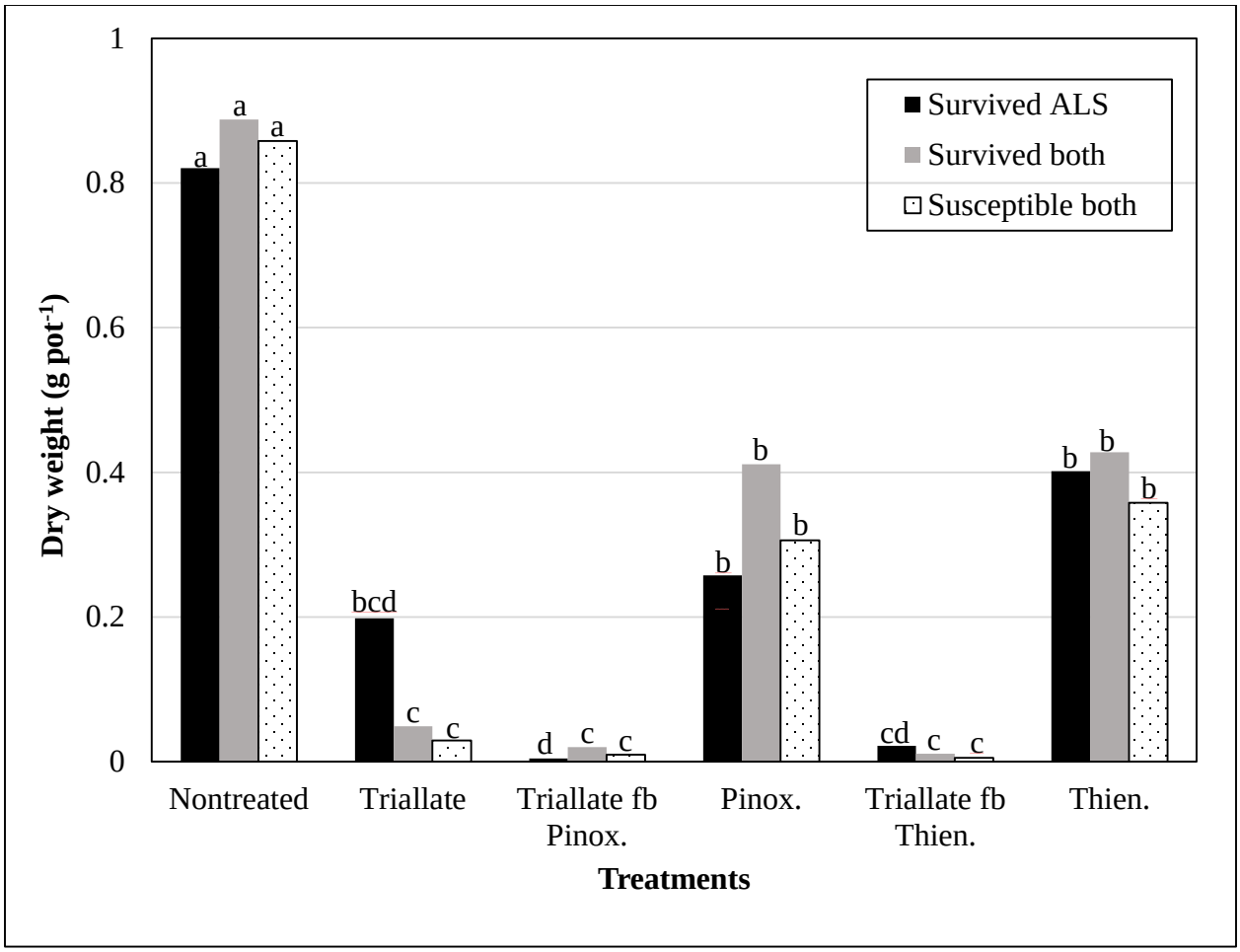

Figure 9. Wild oat dry weight samples collected 28 DAT for 56 samples in 2014 and 2015 in Fargo greenhouse combined over both years. All samples grouped into three subgroups according to level of susceptibility from characterization study. Bars within subgroup with the same letter were similar control according to Fishers-protected LSD at α=0.05. Abbreviations: fb, followed by; Pinox., Pinoxaden; Thien., Thiencarbazone.

Modern wheat cultivars have not been evaluated for their phytotoxicity response to triallate. An interaction between wheat cultivars and triallate rates did not occur in 2014; however, a cultivar effect did occur. The Prosper cultivar resulted in higher injury ratings compared to most cultivars; therefore, Prosper was planted in 2015 and substantial visible injury did not occur until 2240 g ha^{-1} triallate, twice the field rate, was applied. New cultivar tolerance to triallate provided useful information to growers for use in their management decisions regarding wild oat control in wheat. Field studies also indicated that triallate effectively

controlled wild oat, validating potential of triallate for future control of wild oat in North Dakota and Minnesota. Suspected ACCase and ALS resistant wild oat seed samples were solicited from various locations throughout North Dakota and Minnesota. These were screened in the greenhouse and characterized into three groups: survived ALS, survived both ACCase and ALS, and susceptible to both ACCase and ALS herbicides. Wild oat samples with less than 60% control were labeled difficult-to-control. Triallate provided more than 92% control of all wild oat biotypes. The integration of triallate back into North Dakota cropping systems is an option to control wild oat in cereal production.

REFERENCES

Abhilash PC, Jamil S, and Singh N (2009) Transgenic plants for enhanced biodegradation and phytoremediation of organic xenbiotics. Biotech 27:474-488

Acevedo E, Silva P, and Silva H (2002) Wheat growth and physiology. Pages – *in* Curtis BC, Rajaram S, and Macpherson HG ed. Bread wheat. Improvement and Production. Food and Agriculture Organization. Rome: Accessed Press

Ahrens WH and Ehr RJ (1991) Tridiphane enhances wild oat (Avena fatua) control by atrazine-cyanazine mixtures. Weed Tech 5:799-804

Anthony RG and Hussey PJ (1999) Dinitroaniline herbicide resistance and the microtubule cytoskeleton 4:112-116 doi:10.1016/S1360-1385(99)01378-3

Anonymous (2011) EPTAM® herbicide. Gowan Company LLC 10163-283 Gowan Company P.O. Box 5569 Yuma, AZ 85366

Anonymous (2016a) Chlorsulfuron® herbicide. Alligare 81927-43 Alligare, LLC 13 N 8 Street Opelika, AL 36801

Anonymous (2016b) Far-Go® herbicide. Gowan Company LLC 10163-286 070989-IA-001 Gowan Company P.O. Box 5569 Yuma, AZ 85366

Anonymous (2016c) Treflan® herbicide. Dow AgroSciences LLC 23933 CN-23933-005-E Dow AgroSciences Canada Inc. Suite 2100, 450-1 Street SW Calgary, Alberta

Anonymous (2016d) Zidua® herbicide. BASF Corporation 7969-383 BASF Corporation 26 Davis Drive, Research Triangle Park, NC 27709

Banting JD (1970) Effects of diallate and triallate on wild oat and wheat cells. Weed Sci 18:80-84

Beckie HJ (2006) Herbicide-resistant weeds: management tactics and practices. Weed Tech 20:793-814 DOI: http://dx.doi.org/10.1614/WT-05-084R1.1

Beckie HJ, Francis A, Hall LM (2012) The biology of Canadian weeds. 27. *Avena fatua* L. (updated) Can J Plant Sci 92:1329-1357

Beckie HJ and Tardif FJ (2011) Herbicide cross resistance in weeds. Crop Protection 35:15-28 doi:10.1016/j.cropro.2011.12.018

Beckie HJ, Thomas GA, Légére A, Kelner DJ, Van Acker RC, and Meers S (1999) Nature, occurrence, and cost of herbicide-resistant wild oat (Avena fatua) in small-grain production areas. Weed Tech 13:612-625

Carlson HL and Hill JE (1985) Wild oat (Avena fatua) competition with spring wheat: plant density effects Weed Science. Weed Sci 33:176-181 Available at http://www.jstor.org/stable/4044329 Accessed October 14, 2015

Carlson SJ and Morrow LA (1986) Control of wild oat with triallate in spring wheat conservation tillage system Can J Plant Sci 66:181-184

Charmet G (2011) Wheat domestication: lessons for the future CR Biology 334:212-220

Dexter JE, Preston KR, and Woodbeck N (2006) 6 Canadian Wheat. Pages 43-62 *in* Popper L, Schafer W, Freund, W, ed. Future of Flour: A Compendium of Flour Improvement. Agrimedia, Bergen, Dunne, Germany

Dill G (2005) Glyphysate-resistant crops: history, status, and future Pest Manag Sci 62:219-224

Ditomaso JM, Holt JS, Prather TS (2000) Herbicide resistance: definition and management strategies. http://anrcatalog.ucdavis.edu/pdf/8012.pdf. Accessed October 29, 2014

Durgan BR (2002) Wild oat control in small grains. http://appliedweeds.cfans.umn.edu/pubs/02pub02.pdf Accessed: October 29, 2014

Evans RM, Thill DC, Tapia L, Shafii B, and Lish JM (1991) Wild oat (*Avena fatua*) and spring barley (*Hordeum vulgare*) density affect spring barley grain yield. Weed Tech 5:33-39

Fay P, Nalewaja J, and Miller S (1976) Wild oat (*Avena fatus* L.) control with triallate (Far-Go) in wheat. Page 28-31 http://library.ndsu.edu/tools/dspace/load/?file=/repository /bitstream/handle/10365/9740/farm_33_04_07.pdf?sequence=1 Accessed March 20, 2016

Fuerst PE (1987) Understanding the mode of action of the chloroacetamide and thiocarbamate herbicides. Weed Tech 1:270-277

Funke T, Han H, Healy-Fried ML, Fischer M, Schönbrunn E (2006) Molecular basis for the herbicide resistance of Roundup Ready crops. Proc Natl Acad Sci USA 103:10-15

Gibson L and Benson G (2002) Origin, history, and uses of oat (Avena sativa) and wheat (Triticum aestivum). http://agron-www.agron.iastate.edu/ Courses/agron212/Readings /Oat_wheat_history.htm Accessed September 15, 2015.

Green J (2007) Review of glyphosate and Als-inhibiting herbicide crop resistance and resistant weed management Weed Tech 21:547-558

Gunsolus JL (2014) University of Minnesota Herbicide resistant weeds. http://www.extension.umn.edu/agriculture/crops/weed-management/herbicide-resistant-weeds/ Accessed October 30, 2014

Gunsolus JL and Curran W (1999) Herbicide mode of action and injury symptoms Urbana 51: 217-333

Heap I (2016a) Herbicide resistant wild oat globally (*Avena fatua*). The international survey for resistant weeds. http://www.weedscience.org Accessed March 23, 2016

Heap I (2016b) Weeds resistant to ACCase inhibitors (by species and country). The international survey for resistant weeds. http://www.weedscience.org Accessed March 23, 2016

Heap I (2016c) Weeds resistant to ALS inhibitors (by species and country). The international survey for resistant weeds. http://www.weedscience.org Accessed March 23, 2016

Heap I (2016d) Weeds resistant to EPSP synthase inhibitors (by species and country). The international survey for resistant weeds. http://www.weedscience.org Accessed March 23, 2016

Heap I (2016f) Weeds resistant to microtubule inhbitors (by species and country). The international survey for resistant weeds. http://www.weedscience.org Accessed March 23, 2016

Herbek J and Lee C (2009) A comprehensive guide to wheat management in Kentucky. Growth and development. http://www.uky.edu /Ag/GrainCrops /ID125Section2.html Accessed September 15, 2015

Holt JS (2012) The epidemiology of herbicide resistance. Pages 7-8 *in* Proceedings of a National Summit on Strategies to Manage Herbicide-Resistant Weeds. file:///C:/Users/Morgan%20Hanson/Downloads/Chwasty_18730-NAPUSA_Klimat.pdf Accessed September 21, 2015

Holt JS and Lebaron HM (1990) Significance and distribution of herbicide resistance. Weed Tech 4:141-149. http://www.jstor.org/stable/3986861 Accessed: September 18, 2015

James C (2004) Preview: Global status of commercialized biotech/GM crops: Publications, International Service for the Acquisiton of Agri-biotech Applications http://www.isaaa.org/ Accessed March 14, 2016

Johnson R and Ali M (1982) Economics of wheat-fallow cropping systems in western North Dakota Western. J Agri Econ 7:67-78. Accessed March 22, 2016

Kern AJ, Peterson DM, Miller EK, Colliver CC, and Dyer WE (1997) Triallate resistance in *Avena fatua* L. is due to reduced herbicide activation. Pest Biochem and Physiology 56:163-173 doi:10.1006/pest.1996.0070

Kirby E (2002) Botany of the wheat plants. *in* Curtis B, Rajaram S, and Macpherson H, eds. Bread wheat. Improvement and Production. Food and Agriculture Organization, Rome.

Lev-Yadun S, Gopher A, and Abbo S (2000) The cradle of agriculture. http://search.proquest.com/docview/213589665?pq-origsite= gscholar. Accessed September 15, 2015

Matthews JM, Holtum JAM, Lijergren DR, Furness B, and Powless SB (1990) Cross resistance to herbicides in annual ryegrass (*Lolium rigidum*) I. Properties of the herbicide target enzyme acetyl-coenzyme A carboxylase and acetolactate synthase Plant Physiol. 94:1180-1186

McKercher RB, Ashford R, and Morgan RE (1975) Effects of triallate on wild oat grown in a growth chamber. Weed Sci 23:283-285

McMullen MP, Dexter AG, Nalewaja JD, Hamlin W, Davison K (1985) Pesticide use on major crops in North Dakota 1984 agronomy report 3. http://www.ag.ndsu.edu/weeds/survey-pubs/1984%20Pesticide%20use%20in%20ND%20-1%20of%202.pdf Accessed November 5, 2014

McMullen MP and Nalewaja JD (1990) Wheat (*Triticum aestivum*) tolerance to triallate applied spring preplant-incorporated. Weed Tech 4:652-657

Mengistu LW, Messersmith CG, Christoffers MJ (2003) Diversity of herbicide resistance among wild oat sampled 36 yr apart Weed Sci 51:764-773

Miller SD and Nalewaja JD (1980) Wild oat (*Avena fatua*) control with fall and spring applied triallate Weed Sci 28: 417. http://www.jstor.org/stable/4043498?seq=2 Accessed November 5, 2014

Miller SD, Nalewaja JD, and Mulder CE (1977) Difenzoquat (Avenge) a new herbicide for wild oat control. ND Farm Res. 35:19-21

Mueller N (2014) New SDSU research on spring wheat seeding rates iGrow: A service of SDSU extension. http://igrow.org/agronomy/wheat/new-sdsu-research-on-spring-wheat-seeding-rates/. Accessed March 22, 2016

Muzik T (1970) Weed Biology and Control. Page 44

Nandula VK, Reddy KN, Duke SO, and Poston DH (2005) Glyphosate-resistant weeds: current status and future outlook. Outlooks on Pest Management 16:183-187

Norsworthy JK et al. (2012) Reducing the risks of herbicide resistance: best management practices and recommendations. Weed Sci 60:31-62 DOI: http://dx.doi.org/10.1614?WS-D-11-00155.1

[NRCS] Soil Survey Staff, Natural Resources Conservation Service, United States Department of Agriculture (2013) Web Soil Survey. Available online at http://websoilsurvey.nrcs.usda.gov/ Accessed: April 12, 2016

Parka SJ and Soper OF (1977) The physiology and mode of action of dinitroaniline herbicides Weed Sci 25:79-87

Reddy KN and Zablotowicz RM (2003) Glyphosate-resistant soybean response to various salts of glyphosate and glyphosate accumulation in soybean nodules. Weed Sci 51:496-502

Ritz C and Streibig JC (2006) Bioassay for allelochemicals: examples with R.J. Stat. Software. http://jstatsoft.org./ Accessed March 14, 2016

Saylor T (2005) Know thine enemy: weather, weed size can greatly influence grass herbicide applications in small grains. http://www.smallgrains.org/springwh/May05/enemy/enemy.htm Accessed October 21, 2014

Setter T and Carlton G (2000). The structure and development of the cereal plant. Chapter 2. Pages 23-36 *in* Anderson W, Garlinge J, eds. The wheat book, principle and practice. Agriuclture western Australia

Shaner DL (2014) Herbicide Handbook Weed Science Society of America Tenth Edition. 810 E. 10th Street Lawrence, KS 66044-8897

Sharma MP and Vanden Born WH (1977) The biology of Canadian weeds. 27. *Avena fatua* L. Can. J. Plant Sci 58:141-157

Tanetani Y, Kaku K, Kawai K, Fujioka T, Shimizu T (2009) Action mechanism of a novel herbicide, pyroxasulfone. Pesticide biochem and physiology 95:47-55

Timmons FI (2005) A history of weed control in the United States and Canada. Weed Sci 53: 748-761

Tharp BE and Kells JJ (1999) Influence of herbicide application rate, timing, and interrow cultivation on weed control in corn (*Zea mays*) yield in glufosinate-resistant and glyphosate-resistant corn. Weed Sci 13:807-813

Tranel PJ and Wright TR (2002) Resistance of weeds to als-inhibiting herbicides: what have we learned? Weed Sci 50:700-712

Vaughn KC and Lehnen LPJ (1991) Mitotic disruptor herbicides Weed Sci 39:450-457

[USASK] University of Saskatchewan (2016) Harvesting, grain drying, and storage Pages *in* Winter wheat production manual. http://www.usask.ca/agriculture/plantsci/winter cereals/winter-wheat-production-manual/chapter-23.php Accessed April 6, 2016

[USDA] United States Dept. of Agriculture (2013) Wheat: Geographic and climate patterns of U.S. wheat production. http://www.ers.usda.gov/topics/crops/wheat/background.aspx#Geographic Accessed March 14, 2016.

[USDA] IndexMundi (2016) United States wheat production by year. http://www.indexmundi.com/agriculture/?country=us&commodity=wheat&graph=production Accessed on March 23, 2016

[USWA] United States Wheat Associates (2013) Wheat classes http://www.uswheat.org/wheat Classes Accessed April 5, 2016

Vocke G (2013) USDA Economic Research Service-Background http://www.ers.usda.gov/topics/crops/wheat/background.aspx. Accessed March 23, 2016

Wall DA (1995) Response of four annual broadleaf crops to stimulated imazamethabenz spray drift. Can J of Plant Sciences 75:751-757

Warrick BE, Baughman T (2014) Wild oat control in Texas. Available at http://sanangelo.tamu.edu/extension/agronomy/agronomy-publications/wild-oat-control-publication/ Accessed: October 12, 2014

WSSA and HRAC (2014) Summary of herbicide mechanism and action according to weed science society of America and the herbicide resistant action committee classification. Pages 11-15 *in* Herbicide Handbook WSSA edited by Shaner et al. 2014

Zollinger R et al. (2016) North Dakota weed control guide. ND Ag Exp Station, North Dakota State University, Fargo, ND 58005. W-253.

APPENDIX. LOCATIONS OF COLLECTED WILD OAT SEED

Location	County	State
Valley City 1	Barnes	ND
Valley City 2	Barnes	ND
Valley City 3	Barnes	ND
Valley City 4	Barnes	ND
Dazey	Barnes	ND
Fargo	Cass	ND
Calvin 1	Cavalier	ND
Calvin 2	Cavalier	ND
Hannah 1	Cavalier	ND
Hannah 2	Cavalier	ND
Langdon	Cavalier	ND
Milton 1	Cavalier	ND
Milton 2	Cavalier	ND
Sarles 1	Cavalier	ND
Sarles 2	Cavalier	ND
Wales	Cavalier	ND
Reynolds	Grand Forks	ND
Fredonia	McIntosh	ND
Aneta	Nelson	ND
Lakota 1	Nelson	ND
Lakota 2	Nelson	ND
Lakota 3	Nelson	ND
Lakota 4	Nelson	ND
Rugby	Pierce	ND
Jamestown	Stutsman	ND
Fried 1	Stutsman	ND
Fried 2	Stutsman	ND
Cando 1	Towner	ND
Cando 2	Towner	ND
Cando 3	Towner	ND
Dash	Towner	ND
Smith	Towner	ND
Sidney	Towner	ND
Mayville	Traill	ND
Adams	Walsh	ND
Minot	Ward	ND
Tioga	Williams	ND
Stephen	Marshall	MN

Location	County	State
Beltrami	Polk	MN
Crookston 1	Polk	MN
Crookston 2	Polk	MN
Crookston 3	Polk	MN
Crookston 4	Polk	MN
Crookston 5	Polk	MN
Crookston 6	Polk	MN
Crookston 7	Polk	MN
Crookston 8	Polk	MN
Crookston 9	Polk	MN
Crookston 10	Polk	MN
Crookston 11	Polk	MN
Crookston 12	Polk	MN
Crookston 13	Polk	MN
Crookston 14	Polk	MN
Crookston 15	Polk	MN
Erickson	Roseau	MN
Slater	Roseau	MN