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# Management of Herbicide-Resistant Wild Oat (*Avena fatua*) Patches

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**Key words:** *Avena fatua*, gene flow, herbicide resistance, integrated weed management, seed bank, seed shed, seed spread, wild oat

## Abstract

A study was conducted at a 64-ha site in western Canada to determine how preventing seed shed from herbicide-resistant wild oat affects patch expansion over a 6-yr period. Seed shed was prevented in two patches and allowed to occur in two patches (untreated controls). Annual patch expansion was determined by seed bank sampling and mapping. All crop management practices were performed by the grower. Area of treated patches increased by 35% over the 6-yr period, whereas untreated patches increased by 330%. Patch expansion was attributed mainly to natural seed dispersal (untreated) or seed movement by equipment at time of seeding (untreated and treated). Extensive seed shed from plants in untreated patches before harvest or control of resistant plants by alternative herbicides minimized seed movement by the combine harvester. Although both treated and untreated patches were relatively stable over time in this cropping system, preventing seed production and shed in herbicide-resistant wild oat patches can markedly slow the rate of patch expansion.

## Introduction

The distribution of weeds is aggregated or patchy (e.g., Marshall 1988; Wiles et al. 1992), and patches are generally stable (Rew and Cussans 1995; Wilson and Brain 1991). Factors that affect the stability of patches include natural seed dispersal (e.g., wind, dehiscence), tillage, combine harvesting, herbicides, pollen-mediated gene flow, seed persistence, and seed predation. Seed movement by natural dispersal or tillage is often less than 1 m (Rew and Cussans 1995, 1997). This type of movement has been termed “phalanx spread”, because the mean movement distance is short and the leading edge or front is small (Lovett-Doust 1981). Phalanx spread is quantifiable and therefore can be incorporated into spatial and population models. In contrast, “guerilla spread” is caused by a small percentage of seeds that move unpredictable and sometimes long distances by uptake and subsequent spread by a combine harvester (Woolcock and Cousens 2000), animals, or transport on machinery (Lovett-Doust 1981). For this type of movement, new daughter patches or colonies can form from the original parent patch (Kenkel and Irwin 1994).

Natural seed dispersal is an important process influencing the aggregated spatial pattern of wild oat (Colliver et al. 1996; Shirliffe et al. 2002; Thornton et al. 1990). An individual plant may produce up to 500 seeds, although commonly 100 to 150 seeds (Sharma and Vanden Born 1978). The majority of wild oat seeds are shed in late summer usually before crop harvest; seed

shed, however, will generally be less in early-swathed crops such as canola (*Brassica napus* L.) or barley (*Hordeum vulgare* L.) (Shirtliffe et al. 2000). Recently shed seeds are dormant. The persistence of this species in the soil seed bank (3 to 6 yr) is attributed to its dormancy characteristics (Sharma and Vanden Born 1978). Seeds are relatively heavy, and are not adapted for long-distance movement; dispersal is generally within 2 to 3 m of the parent plant (Thill and Mallory-Smith 1997). Thus, wild oat seed dispersal is categorized as phalanx spread (Kenkel and Irwin 1994). Although tillage moves wild oat seeds only 2.5 m or less, it affects vertical seed distribution in soil and consequently, seed predation, dormancy, and persistence (Thill and Mallory-Smith 1997). Seeds can move long distances (>150 m) when taken up by a combine harvester and chaff is returned to the ground (Shirtliffe 1999; Shirtliffe et al. 1998). The species is highly selfing, with 0.17% outcrossing for plants within 0.6 m of each other in a dense stand; thus, the contribution of pollen movement to herbicide resistance evolution and spread is insignificant compared with resistant seed production and dispersal (Andrews et al. 1998; Cavan et al. 1998; Murray et al. 2002).

Weed extension personnel advocate field scouting for suspected herbicide-resistant weed patches after in-crop herbicide application. If a patch occupies a small area (< 0.4 ha), then preventing seed production by mowing, tillage, or non-selective herbicides is recommended (Manitoba Weed Supervisors Association 1997). In a small-plot study, Wilson and Cussans (1975) found that viable seed bank density of wild oat decreased by about 90% and seedling density by 75% when seed shed was prevented in the previous year. Shirtliffe (1999) showed that wild oat seed spread was greatly reduced when chaff was collected as it exited the combine and was removed from the field compared with no chaff collection. The effect of management practices on herbicide-resistant weed patch dynamics has not been reported. The objective of the grower-participatory, field-scale study described in this report is to determine how preventing seed shed from herbicide-resistant wild oat affects patch expansion over a 6-yr period.

## **Materials and Methods**

### *Field Site and History*

The 64-ha field is located near Carrot River, Saskatchewan (53.3° N; 103.3° W) in the sub-humid Boreal Transition ecoregion (Acton et al. 1998). The soil at the site is a Tisdale-Arborfield clay loam (Boralfic Haploboroll) with 3.5% organic matter and pH 6.8. The topography is level to undulating (0.5-2% slopes).

The field had been surveyed for post-herbicide weed abundance in 1995 (Thomas et al. 1996), and the grower had completed a management questionnaire. Based on the herbicide-use history, the field was rated a high risk for weed resistance to group 1 [acetyl-CoA carboxylase (ACCase) inhibitor] or group 2 [acetolactate synthase (ALS) inhibitor] herbicides (Mallory-Smith and Retzinger 2003). In 1996, the field was cropped to wheat (*Triticum aestivum* L.) and sprayed with clodinafop-propargyl, a group 1 herbicide. In late summer, the field was surveyed for herbicide-resistant wild oat (Beckie et al. 1999). Two patches of wild oat exhibiting resistance to fenoxaprop-P, sethoxydim (group 1), imazamethabenz (group 2), or flamprop (an arylaminopropionic acid, group 25) were identified.

### *Patch Identification in 1997 and Herbicide Resistance*

The field was re-surveyed for herbicide-resistant wild oat in late summer of 1997. Four patches were identified. The center point of each patch was georeferenced by triangulation (Figure 1). Patches 1 and 2 had been identified in the previous year.

Seeds were collected from each patch and tested for resistance to six group 1 herbicides, imazamethabenz, and flumprop in pot assays in a greenhouse. Environmental conditions were a 20/16 C day/night temperature regime with a 16-h photoperiod supplemented with 230  $\mu\text{mol/m}^2/\text{s}$  illumination. Three aryloxyphenoxypropionate (APP) herbicides and three cyclohexanedione (CHD) herbicides were used in group 1 resistance testing. The three APP herbicides were fenoxaprop-P (formulated without a safener) at 150 g/ha, clodinafop-propargyl at 55 g/ha, and quizalofop at 35 g/ha; the three CHD herbicides were sethoxydim at 110 g/ha, tralkoxydim at 200 g/ha, and clethodim at 45 g/ha. Imazamethabenz was applied at 500 g/ha and flumprop at 350 g/ha. Recommended adjuvants were included in the herbicide spray solutions.

Thirty-six plants were grown in flats measuring 52 by 26 by 5 cm that were filled with a commercial potting mixture amended with a slow-release fertilizer. Plants were treated at the two- to four-leaf stage. Herbicides were applied using a moving-nozzle cabinet sprayer equipped with a flat-fan spray tip (TeeJet 8002VS) calibrated to deliver 200 L/ha of spray solution at 275 kPa in a single pass over the foliage. Plants were visually assessed as herbicide-resistant or herbicide-susceptible at 21 d after treatment. A minimum of 100 seedlings per sample (patch) were screened in each resistance test. Treatments (and untreated controls) were replicated three times and tests were repeated. Known herbicide-resistant and herbicide-susceptible biotypes were included in all tests (Beckie et al. 2000).

### *Crop Management*

All management practices were performed by the grower. Cereal and oilseed crops were grown in the field from 1997 to 2002 (Table 1). Crops were seeded in early to mid-May, except for late May seeding of barley. Glyphosate was applied at 440 g ai/ha about 3 d before seeding. Seedlots were free of wild oat seeds. Crops were direct-seeded into standing stubble with an 18-m wide air seeder equipped with knife openers spaced 23 cm apart. Fertilizer N, P, K, and S were banded at the time of seeding at rates recommended by soil tests. Group 1 herbicides were applied in cereals in 1997 and 1999, glyphosate (group 9) in glyphosate-resistant canola in 1998, flumprop in canary seed (*Phalaris canariensis* L.) in 2000 and 2002, and a group 2 herbicide (1:1 imazamox:imazethapyr mixture) in imidazolinone-resistant canola in 2001. Group 1 herbicides and flumprop were tank-mixed with a broadleaf weed herbicide. Herbicides were applied when most weeds were at the two- to four-leaf stage at recommended rates with a field sprayer equipped with 8001 flat fan spray tips calibrated to deliver a carrier volume of 110 L/ha at 275 kPa. At or near crop maturity, plants were swathed and later threshed with a combine harvester. Weather conditions during the 1997 to 2001 growing seasons were conducive for average to above-average seed yields, except for dry conditions in 2002 that markedly reduced yield of canary seed.

### *Experimental Treatment*

Each summer after herbicide application, the four patches were georeferenced by triangulation using the 1997 coordinates. At reproductive stage of wild oat each year, the perimeter of each visible patch was marked with flags and mapped in relation to the center point using measuring tape and global positioning system (GPS). Panicle density was measured in eight 1-m<sup>2</sup> quadrats per patch. Experimental treatment was the prevention of seed shed in patches 3 and 4 by clipping panicles from wild oat plants and bagging them. The two patches were monitored periodically to ensure no seed was shed. Patches 1 and 2 were untreated controls, in the context of the entire area of the field sprayed each year with a wild oat herbicide. The percentage of empty spikelets in a panicle (i.e., shedded seed) for each of 20 plants in each untreated patch was determined immediately before swathing to estimate the extent of seed shed at the time of harvest.

### *Viable Seed Bank*

The viable fraction of the herbicide-resistant wild oat seed bank complemented mapping in demarkation of patch boundaries each year. Soil was sampled after crop harvest. Two 5-cm-diam by 10-cm-deep soil cores were collected at each grid point spaced 2 m apart. The area sampled included that occupied by the patch plus an outer zone of width equal to the longest dimension of a patch. Soil samples were frozen until the following spring when three growth periods were conducted in a greenhouse with environmental conditions described previously. Soil in flats measuring 52 by 26 by 5 cm was watered twice daily. A liquid N-P-S fertilizer was applied to each flat once during each of the three growth periods. Wild oat seedlings were counted at the two- to three-leaf stage and then sprayed with fenoxaprop-P as described previously. Three wk after spraying, survivors were counted and removed, and the soil was remixed. The sample was returned to the freezer for 3 wk in advance of the second growth period. This procedure was repeated for the final growth period.

### *Data Analysis*

The x and y coordinates with resistant wild oat in the seed bank furthest from a patch center located in 1997 (0 m x-coordinate, 0 m y-coordinate) on each transect were mapped each year. The area occupied by a patch each year was estimated using grid paper with straight lines connecting the coordinates, and the annual change in patch area was calculated. The annual change in area of treated patches 3 and 4 were compared with that of untreated patches 1 and 2. Data were subjected to ANOVA, with each treatment replicated twice (patch as an experimental unit) in a completely randomized design. Data were arcsin transformed before ANOVA, but back-transformed data are presented. Data were also analyzed by nonparametric statistics, using the Kruskal-Wallis test (SAS 1999), to confirm the parametric statistical results. Treatment effect was considered significant at  $P < 0.10$  (ANOVA) or  $P < 0.20$  (Kruskal-Wallis test). These significance levels (alpha) are within the range (0.10 to 0.20) typically used in landscape or field-scale studies because variances are high and uncontrollable; a more rigorous level would greatly increase the probability of a type II error (beta), i.e., failing to detect a true treatment difference (Walley et al. 1996). ANOVA results are only presented because of complete agreement with those of the Kruskal-Wallis test.

## Results and Discussion

### *Herbicide Resistance of Patches*

Wild oat in all four patches in 1997 were resistant to fenoxaprop-P, ranging from 16% of plants in patch 3 to all plants tested in patch 1 (Table 2). Resistant patches are commonly comprised of a mixture of resistant and susceptible plants whose proportion depends on factors such as selection pressure, seed bank longevity and seedling recruitment (Beckie et al. 2002; Davidson et al. 1996). Plants in patches 3 and 4 were not resistant to the other seven herbicides. In contrast, plants in patch 1 were resistant to other group 1 herbicides, including clodinafop-propargyl, quizalofop, sethoxydim, and tralkoxydim, but not to imazamethabenz (group 2) or flamprop (group 25). Plants in various proportions of the population in patch 2 were resistant to the three APP herbicides. Forty-one percent of plants in this patch were resistant to imazamethabenz and 8% were resistant to flamprop. Differences in resistance among wild oat patches within a field to herbicides within a group or across groups, as a consequence of independent evolution through selection, have been documented previously (Andrews et al. 1998; Beckie et al. 2002).

### *Effect of Seed Shed Prevention on Patch Containment*

Untreated patches 1 and 2 expanded in area at an average annual rate of 31 and 38%, respectively (Figure 2 and Table 3). Over the 6-yr period, patch 1 had expanded by 270% and patch 2 by 390%. The rate of patch expansion varied by year. For example, patch 1 expanded by nearly 50% from 1998 to 1999 and again from 1999 to 2000, but only by 4% between 2001 and 2002. Relatively high seed production and extensive seed shed before harvest in 1999 and 2000 (Table 3) would have resulted in significant natural seed dispersal. Good herbicide efficacy and hot, dry conditions in the summer of 2002 resulted in low panicle density and almost complete seed shed. These conditions may have restricted expansion of patch 1, located on a broad upper slope, more than patch 2 located in a lower-slope area with greater soil moisture and having a greater wild oat panicle density. Relatively rapid expansion of patch 2 in 2001 may have been aided by harvesting operations in canola as the majority of wild oat spikelets contained seeds. Although plants in this patch were resistant to imazamethabenz (Table 2), they were not resistant to the imazamox:imazethapyr mixture (data not shown). Susceptibility to this herbicide mixture was evident by the low panicle density in canola (Table 3). There was a tendency of these patches to preferentially expand east-west vs. north-south over the 6-yr period, which is likely attributed to the same seeding and combine harvester direction maintained each year by the grower.

Seed shed prevention markedly reduced the rate of area expansion of patches 3 and 4 (Figure 3 and Table 3). Patch 3 expanded at an average annual rate of 4% and patch 4 at a rate of 8%. After 6 yr, the treated patches had expanded 35% in contrast to average expansion of 330% for untreated patches. A significant treatment effect on area expansion was indicated by ANOVA (Table 3) and the Kruskal-Wallis test ( $P \leq 0.12$ ), except from 2001 to 2002 (Table 3). Lack of difference between expansion of treated and untreated patches from 2001 to 2002 ( $P=0.478$ ) was due to the variability in expansion between the untreated patches. The slow, limited expansion of treated patches over the 6-yr period likely resulted from movement of seed in soil by equipment at the time of direct seeding. Wild oat persistence in the seed bank combined with movement of crop residue at or near the soil surface by the knife openers likely facilitated seed movement.

Tillage equipment has been shown to move wild oat seeds 2.5 m or less (Thill and Mallory-Smith 1997).

The percentage of empty spikelets per wild oat plant immediately before harvest was high in all the cereal crops because plants matured before swathing. In this region, malting barley may be swathed when the crop is completely ripe and combined immediately after (or straight combined) to minimize the risk of seed quality reduction by rain. Canary seed is typically harvested at full maturity because it will dry faster and receive less bird damage if left standing. The straw is wiry and combining is more difficult if the crop is swathed before maturity. In contrast, empty spikelets per plant comprised only about 15 to 25% in canola, which is swathed when about 30 to 40% of seeds in pods on the main raceme have changed color. Swathing at this stage, when plants are relatively green, minimizes seed shatter and optimizes yield. However, high efficacy of herbicides used in herbicide-resistant canola resulted in few wild oat plants (Table 3). Therefore, wild oat seed movement by the combine harvester was deemed to be less important than natural seed dispersal and movement by seeding equipment because of extensive seed shed before cereal harvest and excellent wild oat control by glyphosate and imidazolinone herbicides in canola.

As a consequence of limited seed movement by equipment, untreated patches in this field were relatively stable over the 6-yr period. Other factors that may have favored the stability of these patches include low soil disturbance in this no-tillage system, restricted range of natural seed dispersal, seed predation or mortality of unburied seeds, low outcrossing rate, or lengthy seed bank persistence (Rew and Cussans 1995; Thill and Mallory-Smith 1997; Wilson and Brain 1991). Management tactics can be used to exploit the relative stability of both treated and untreated herbicide-resistant wild oat patches.

#### *Implications for Management*

Patch dynamics of wild oat need to be considered in relation to the cropping system. In this study, crop management practices were not conducive to wild oat seed movement. Crops were seeded with minimal soil disturbance into standing stubble and cereal crops were harvested when wild oat seed shatter was nearly complete. Although most wild oat plants contained seeds in spikelets in herbicide-resistant canola, excellent wild oat control resulted in few plants and consequently little seed return. Seed movement would be expected to be greater in fields with more intensive soil disturbance by tillage or seeding equipment, or when the crop is harvested before the shedding of wild oat seeds. These conditions would favor faster expansion of wild oat patches or result in daughter patches (personal observation). Although rarely adopted, chaff collection would effectively mitigate seed movement by the combine harvester (Shirliffe et al. 1998). Relatively stable patches observed in this study suggests that a single weed map may be used for multiple years to manage herbicide-resistant wild oat to slow the rate of infestation throughout a field.

The grower was able to effectively manage herbicide-resistant wild oat in this field because of available alternative herbicides. Withdrawal from the market of flumetrop and older alternative chemistries and preference for postemergence vs. preemergence herbicide application means that group 1 or 2 herbicides are usually applied to control wild oat in cereals and often in broadleaf crops. Restricted herbicide group options in cereals consequently results in increased frequency

of canola in this cropping system from one in four year (recommended for disease control) to one in three year in this study. Herbicide-resistant canola, introduced in 1995, will continue to be an important tool in managing herbicide-resistant wild oat, especially in the sub-humid region of the Northern Great Plains where the weed is well adapted and incidence of herbicide resistance is greatest.

This study demonstrates that expansion of herbicide-resistant wild oat patches can be severely restricted by preventing seed shed. In a survey of 158 growers in Alberta in 2001 and Manitoba in 2002, about 75% of them in both provinces indicated that they do not control herbicide-resistant weeds in suspected patches before harvest or limit seed spread at harvest (unpublished data). The present adoption of this weed management practice is low despite a majority of growers who indicated that they scout fields after herbicide application to check for uncontrolled weed patches. Nonetheless, the majority of growers who do not manage suspected herbicide-resistant weed patches can still slow their rate of infestation in a field by employing crop management practices that favor patch stability.

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**Table 1.** Crop Management in the 64-ha Field Near Carrot River, Saskatchewan From 1997 to 2002.

	1997	1998	1999	2000	2001	2002
Crop <sup>a</sup>	Barley	Canola	Wheat	Canary seed	Canola	Canary seed
Cultivar	AC Oxbow	Quest	AC Barrie	Keet	46A76	Keet
Seeding date	25 May	8 May	9 May	1 May	14 May	15 May
Seeding rate (kg/ha)	105	8	100	40	8	40
Seeding depth (cm)	4-5	2-3	4-5	4	2-3	4
Seed yield <sup>b</sup> (kg/ha)	4570	2240	4370	1960	1960	390
Grass herbicide	Fenoxaprop-P	Glyphosate <sup>c</sup>	Clodinafop-propargyl <sup>d</sup>	Flamprop	Imazamox: imazethapyr <sup>e</sup>	Flamprop
Herbicide rate (g/ha)	90	440	55	260	30	260

<sup>a</sup>Barley, *Hordeum vulgare* L.; canary seed, *Phalaris canatiensis* L.; canola, *Brassica napus* L.; wheat, *Triticum aestivum* L.

<sup>b</sup>Estimated by the grower.

<sup>c</sup>Isopropylamine salt.

<sup>d</sup>Proposed common name; chemical name: 2-[4-(5-chloro-3-fluoro-2-pyridinyl)oxy]-phenoxy]-2-propynyl ester.

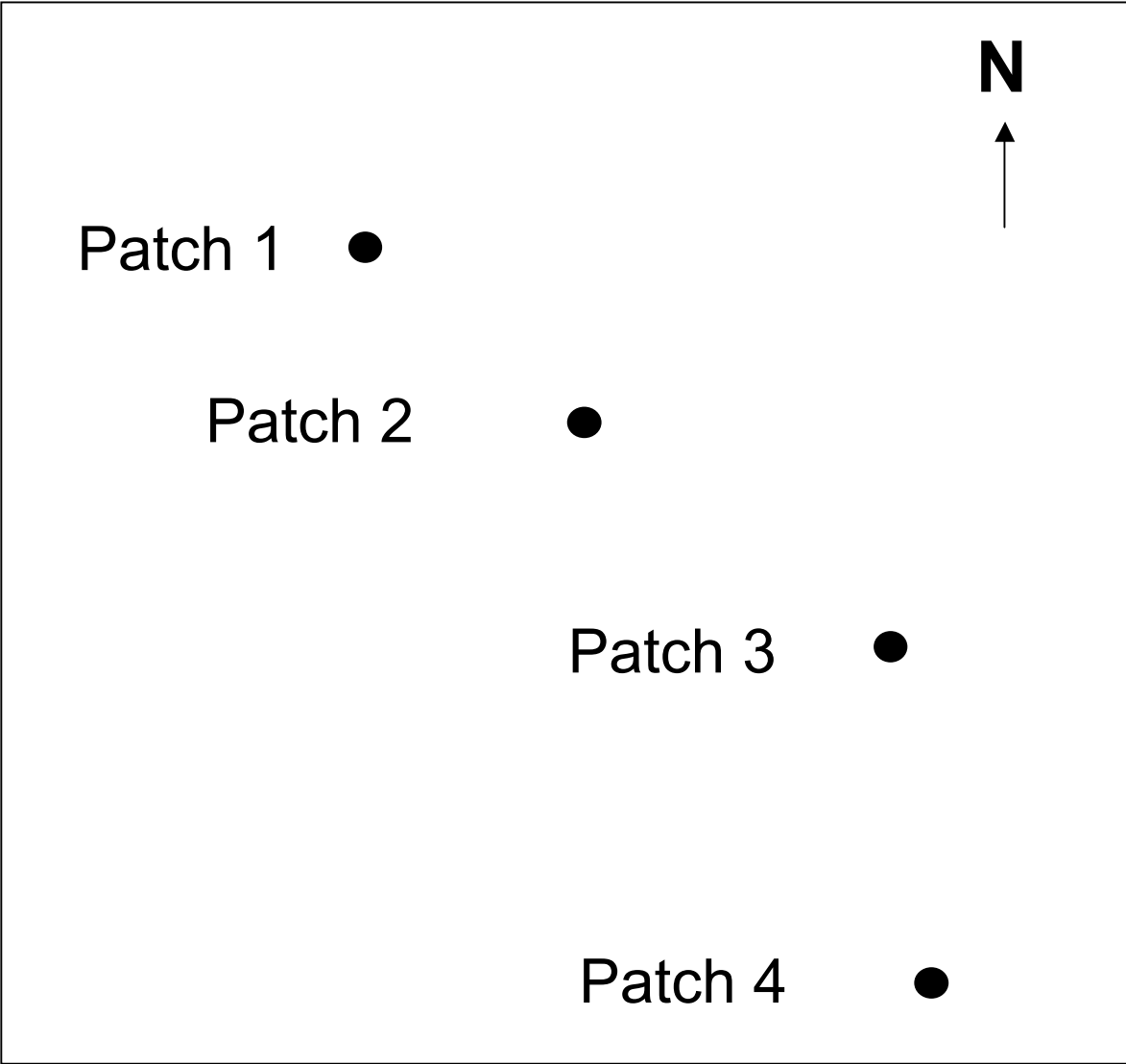
<sup>e</sup>1:1 mixture.

**Table 2.** Herbicide Resistance of Wild Oat Patches in the Field near Carrot River, Saskatchewan in 1997.

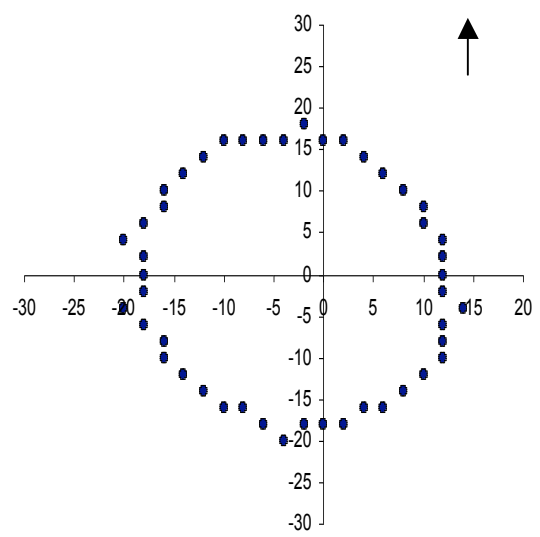
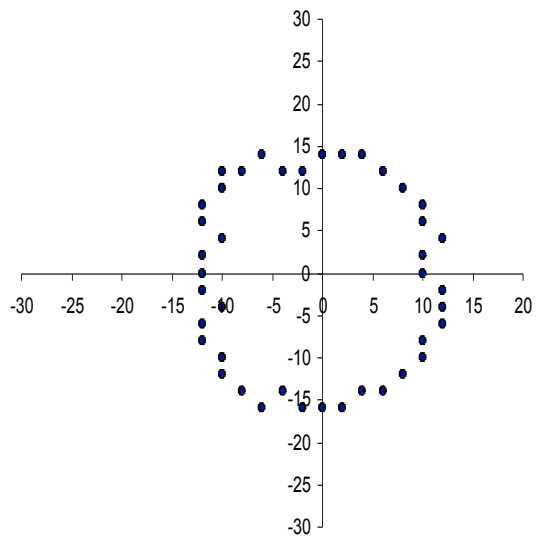
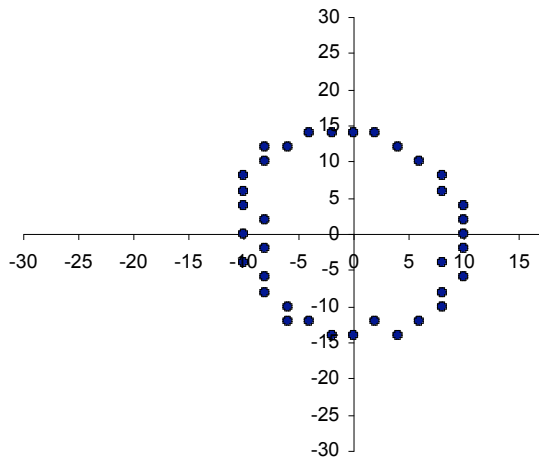
Herbicide	Patch 1	Patch 2	Patch 3	Patch 4
Fenoxaprop-P	100	75	16	25
Clodinafop-propargyl	90	9	0	0
Quizalofop	100	42	0	0
Sethoxydim	83	0	0	0
Tralkoxydim	97	0	0	0
Clethodim	0	0	0	0
Imazamethabenz	0	41	0	0
Flamprop	0	8	0	0

**Table 3.** Mean Wild Oat Panicle Density in Patches Immediately Before Seed Shed Prevention Treatment of Patches 3 and 4, Percentage of Empty Spikelets Per Wild Oat Plant at Crop Harvest, and Calculated Patch Area and Expansion from 1997 to 2002 in the Field Near Carrot River, Saskatchewan (Standard Errors in Parentheses).

	Untreated controls		Treated	
	Patch 1	Patch 2	Patch 3	Patch 4
Mean panicle density (no./m <sup>2</sup> )				
1997	248 (29)	148 (22)	86 (19)	104 (22)
1998	1.4 (0.1)	0.9 (0.1)	1.2 (0.1)	1.2 (0.1)
1999	137 (19)	8.2 (2.0)	0.3 (0.1)	0.2 (0.2)
2000	85 (32)	51 (16)	0.9 (0.2)	0.6 (0.2)
2001	0.2 (0.1)	0.8 (0.1)	0.2 (0.1)	0.2 (0.1)
2002	0.2 (0.1)	6.2 (1.0)	0.4 (0.1)	1.4 (0.1)
Empty spikelets/plant (%)				
1997	98 (1)	99 (1)		
1998	19 (2)	16 (2)		
1999	96 (1)	97 (1)		
2000	94 (1)	98 (1)		
2001	26 (1)	21 (2)		
2002	99 (1)	97 (1)		
Calculated patch area (m <sup>2</sup> )				
1997	440	100	1020	280
1998	580	130	1070	310
1999	840	170	1110	340
2000	1210	220	1170	360
2001	1590	340	1210	380
2002	1630	490	1270	410
Patch expansion (%)				
1997-1998	32	30	4	11
1998-1999	45	31	4	10
1999-2000	44	29	5	6
2000-2001	31	54	3	6
2001-2002	4	44	5	8
1997-2002	270	390	24	46
Patch expansion: treated vs. untreated control (ANOVA: P>F)				
1997-1998	0.017			
1998-1999	0.055			
1999-2000	0.051			
2000-2001	0.080			
2001-2002	0.478			
1997-2002	0.040			



**Figure 1.** Location of four herbicide-resistant wild oat patches in a 64-ha field near Carrot River, Saskatchewan.

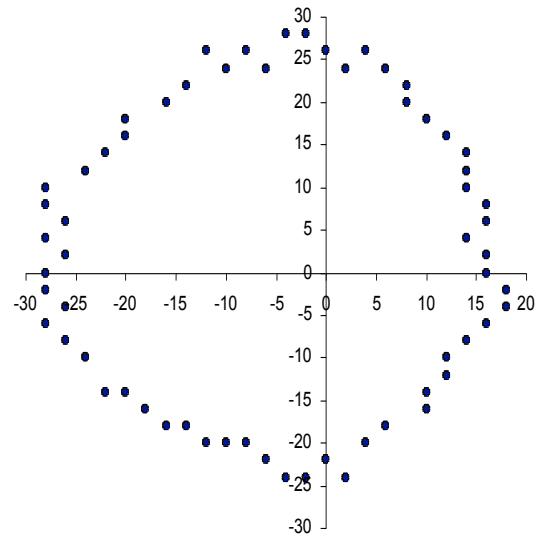
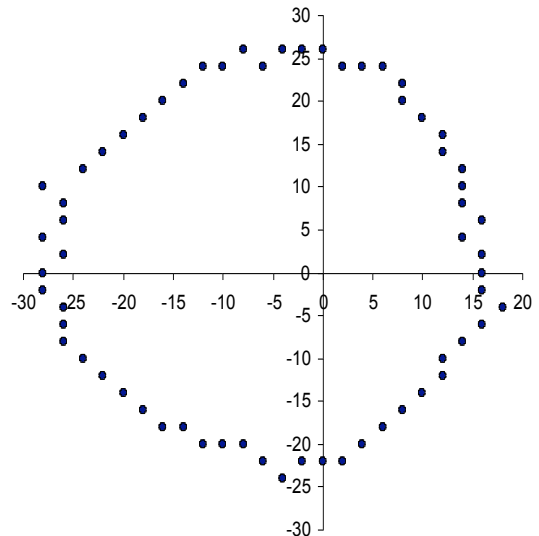
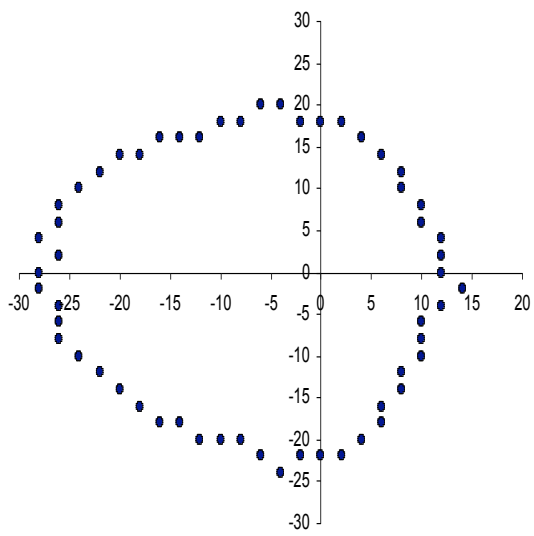


**(A)**

1997

1998

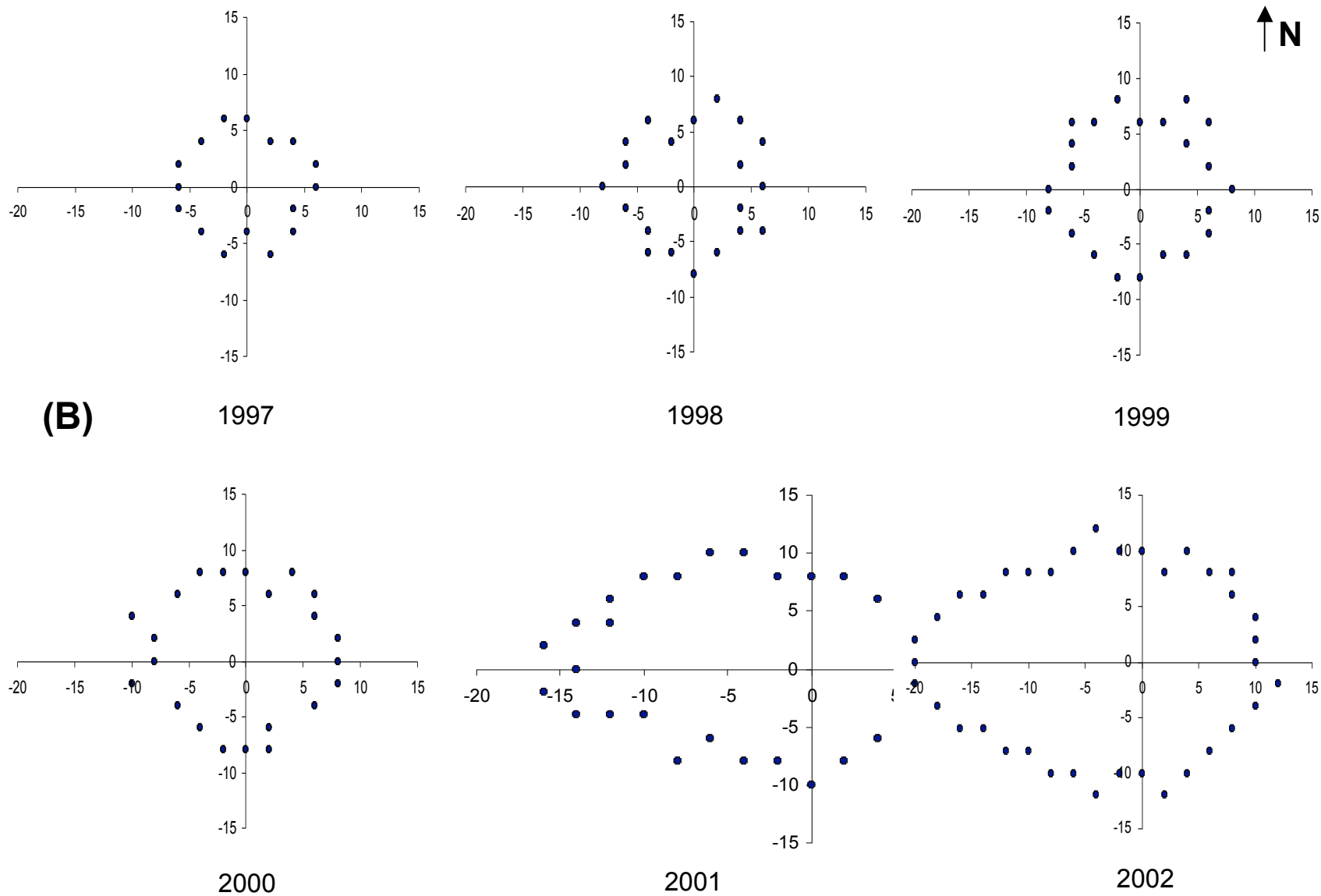
1999



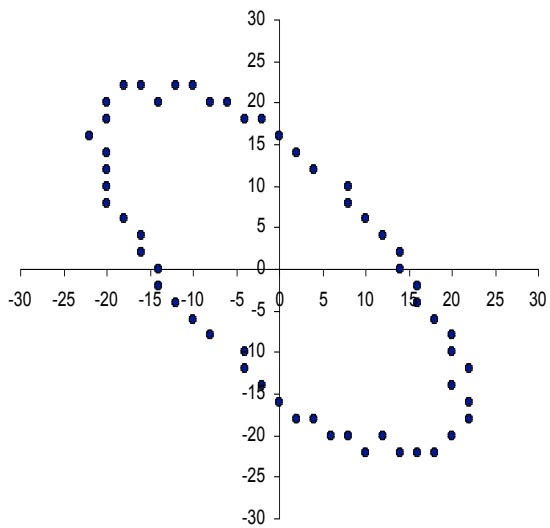
2000

2001

2002

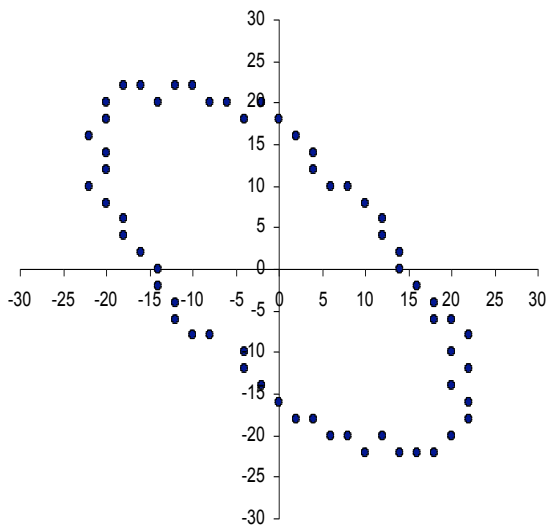


**Figure 2.** Untreated controls: area of wild oat patch 1 (A) and patch 2 (B) from 1997 to 2002.

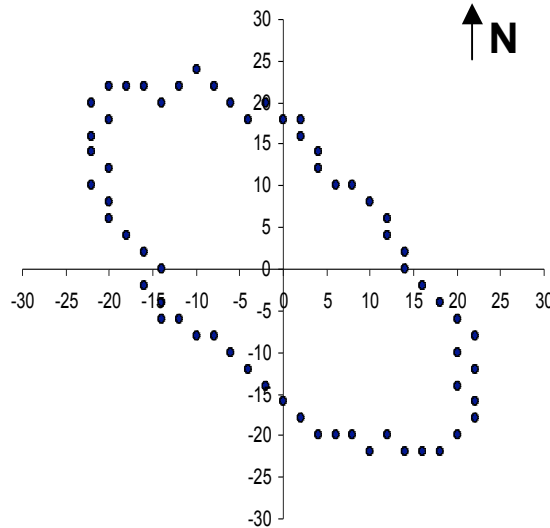


(A)

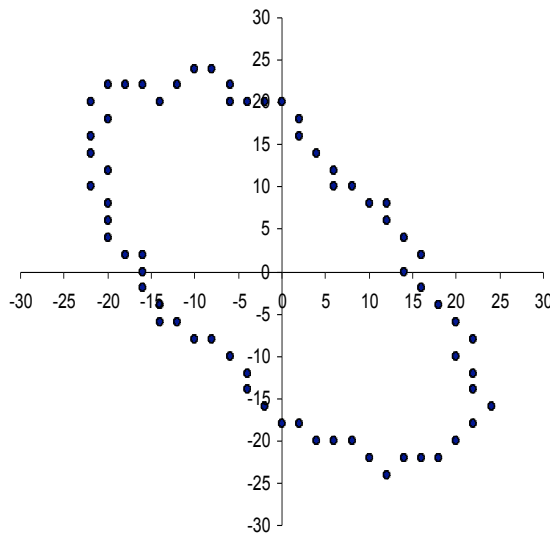
1997



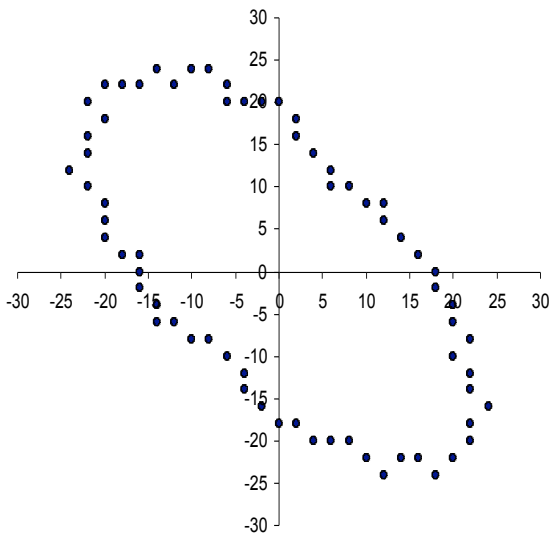
1998



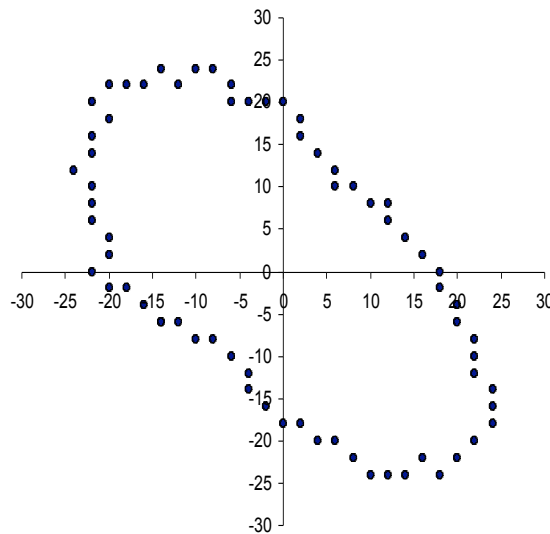
1999



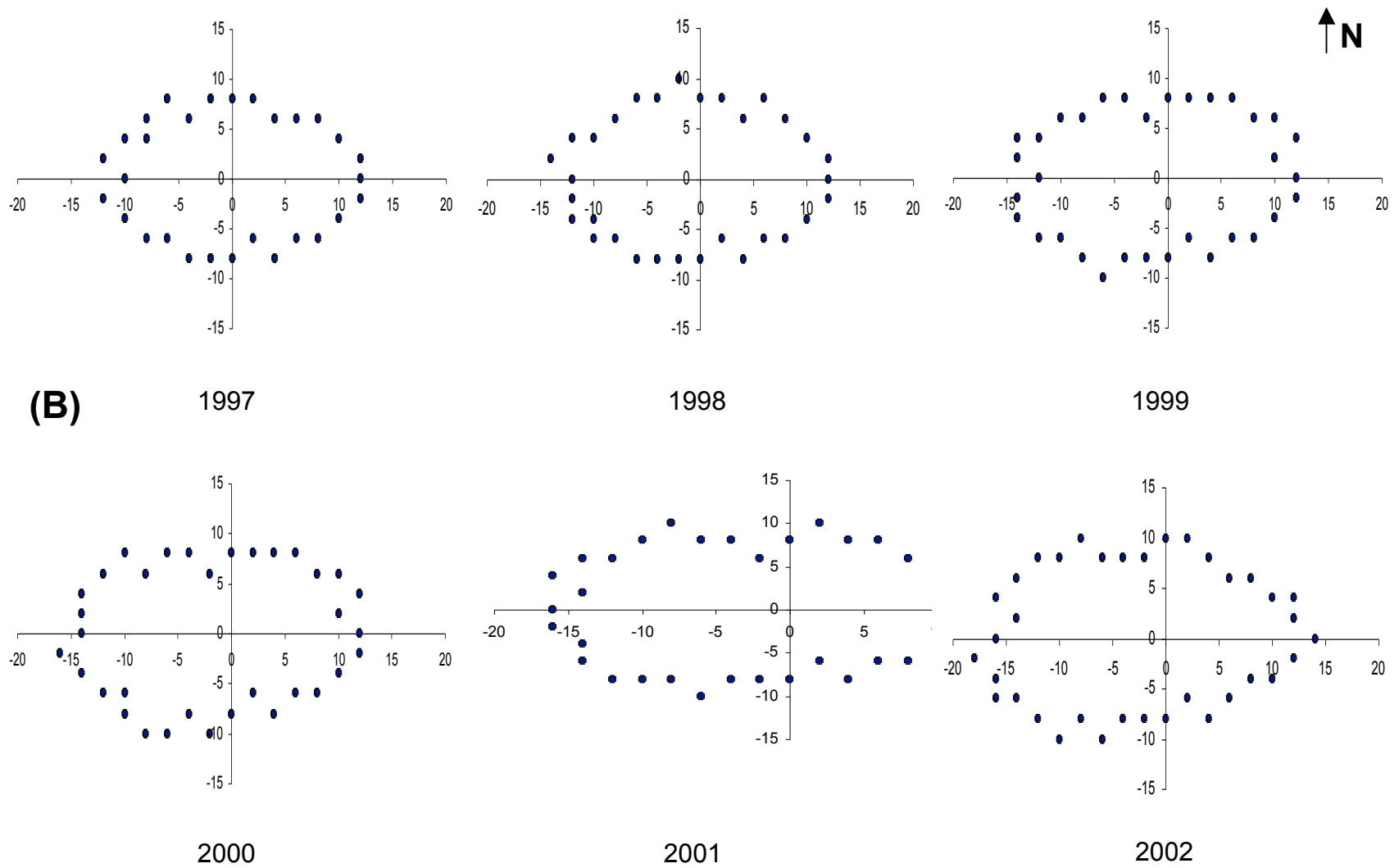
2000



2001



2002



**Figure 3.** Seed shed prevention treatment: area of wild oat patch 3 (A) and patch 4 (B) from 1997 to 2002.