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Impact of Aryloxyphenoxypropionate Herbicides on *Phalaris minor* in Haryana

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ABSTRACT

Impact of aryloxyphenoxypropionate herbicides used since 1998 to control *Phalaris minor* in Haryana was evaluated during 2008-09 and 2009-10 under screen house conditions. Most of the populations evaluated against fenoxaprop-P-ethyl and clodinafop-propargyl exhibited either a high or a medium level of resistance in 2008-09. Similar results were obtained for clodinafop in 2009-10. Some of the populations were also susceptible to fenoxaprop (120 g/ha) indicating segregation of alleles for the resistant and susceptible traits as homozygous as well as heterozygous individuals in the progeny. Most of the populations were susceptible to phenylpyrazolin derivative- pinoxaden used at 25, 50 and 100 g/ha. However, a fraction of the populations exhibited insensitivity towards pinoxaden without any prior history of exposure. The data are supported by GR_{50} values, ion efflux tests and pigment retention tests. Selection pressure exerted by the aryloxyphenoxypropionate and/or urea herbicides could possibly be responsible for such an effect. Bioassay methods need to be developed for location specific identification of the resistance to herbicides and their early management.

Key words : Clodinafop-propargyl, fenoxaprop-P-ethyl, ion efflux, photosynthetic pigments, *Phalaris minor*, pinoxaden

INTRODUCTION

Phalaris minor Retz. (Little seed canary grass) is an annual weed infesting winter crops across several continents. In India, the weed evolved insensitivity to herbicides like isoproturon [N'(4-isopropyl phenyl) N,N'dimethyl urea] after 10-15 years of continuous use in wheat fields in Haryana and Punjab (Malik and Singh, 1995; Walia *et al*., 1997). Alternative herbicides like fenoxaprop-P-ethyl, clodinafop-propargyl and sulfosulfuron were recommended for its management in 1997-98. Both fenoxapop and clodinafop belong to aryloxyphenoxypropionate (AOPP) category of Group 1 herbicides [acetyl-coenzyme A carboxylase (ACCase) inhibitors]. After 8-10 years of continuous use of these herbicides, complaints regarding their efficacy also started to emerge (Brar *et al*., 2002; Yadav *et al.,* 2002), stressing the need to diversify present weed control methods. With this in view, the efficacy of a newly introduced ACCase herbicide pinoxaden (Hofer *et al*., 2006) belonging to phenyl pyrazolin category of Group 1 of herbicides was planned to be tested against *P. minor* populations in Haryana. Two isoforms of ACCase have been identified in plants, the first is located in chloroplasts and is the site of synthesis for primary fatty acids. Second is present in the cytosol and is involved in the synthesis of long-chain fatty acids. The homomeric ACCase in cytosol and heteromeric ACCase in chloroplasts of dicots is insensitive to aryloxyphenoxypropionate (AOPP), cyclohexandione (CHD) and phenylpropazolin (DEN) category of herbicides (Yu *et al*., 2007). In contrast plastidic homomeric ACCase in almost all grass species is herbicide sensitive and is the basis for selective control of grass weeds by ACCase inhibitor herbicides.

The study, therefore, was aimed at evaluating (i) the impact of ACCase inhibitor herbicides fenoxaprop and clodinafop on *P. minor* populations from Haryana and (ii) evaluating the efficacy of another ACCase inhibitor herbicide pinoxaden on these populations.

MATERIALS AND METHODS

Seeds of 50 populations of *P. minor* were collected from different locations of Haryana in April 2008 and 25 more were collected in April 2009. Seeds were sown in November end during both the years in sandy loam soil in earthen pots (9" dia). Five plants were maintained in four replicates for each biotype. Efficacy

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of fenoxaprop (120 g/ha) and clodinafop (60 g/ha) was tested during both the years. The effect of 25 g/ha pinoxaden was tested during 2008-09 and that of 50 and 100 g/ha was tested during 2009-10. The herbicides were sprayed at the 2-3 leaf stage i. e. 30-35 days after sowing (DAS) with a knapsack sprayer using a flat fan nozzle with a spray volume of 500 ml for spray based on 5 x 2 M area. The plants that remained unsprayed with herbicide served as control. Observations were recorded 30 DAS on weed mortality and dry matter accumulation. The populations that showed mortality in the range of 70-100% were classified as susceptible (S), those that showed mortality in the range of 35-70% were classified as medium susceptible/medium resistant (MS/MR) and those in the range of 0-35% as resistant (R) populations.

In another experiment, five selected populations of *P. minor* viz., HAU- Hisar, Uchana (Karnal), Ambala, Nangla (Hisar) and Chanarthal (Kurukshetra) along with Wheat var. PB 343 were tested for GR_{50} values against the herbicides. Membrane stability index and pigment retention tests were also performed. Seedlings were raised in the same manner as described above. The dose range of the herbicides sprayed was 0, 30, 60, 120 and 240 g/ha fenoxaprop; 0, 7.5, 15.0, 30.0 and 60.0 g/ha for clodinafop and 0, 12.5, 25, 50 and 100 g/ha for pinoxaden. Observations were recorded 30 DAS on weed mortality and biomass accumulation. For plotting dose response curves and calculation of GR_{50} values Microsoft excel computer programme as described in an earlier communication was employed (Dhawan *et al*., 2009).

Membrane stability was assessed by the method of Vanstone and Stobbe (1977). Twenty days after spray of the herbicides, leaf samples were collected from control as well as fenoxaprop (120 g/ha), clodinafop (60 g/ha) and pinoxaden (50 g/ha) sprayed plants. Leaf tissue (100 mg) was taken in 20 ml test tubes containing 10 ml of de-ionized water. These samples were incubated for 24 h at 4°C. The conductance of decanted liquid containing effluxed electrolytes was determined at 25°C with a conductivity meter (TDS meter TCM-15; Toshniwal Mfg. Pvt. Ltd., Ajmer, India) and designated as EC a (Before boiling). Then the samples were subjected to heating at 100°C in a water bath for 10 min. After cooling, the electrical conductivity of the solutions was measured and designated as EC b (after boiling). The electrolyte leakage was expressed by the following formula :

$$\text{Membrane injury (\%)} = \frac{EC\ a}{EC\ b} \times 100$$

Chlorophyll was extracted by the method described by Gunes *et al*. (2007) using dimethyl sulphoxide (DMSO). Twenty-five mg of leaf tissue was placed in a vial containing 3 ml DMSO at room temperature till the tissue became chlorophyll free (12-16 h). The extract was transferred to a graduated tube and absorbance was read at 665, 645 and 454 nm as described by Kaloyereas (1958) on a computer aided spectrophotometer (Systronic India Spectrophotometer 117) running a multiple wave length programme. DMSO was used as blank.

Calculations for different pigments were made according to the formulae (Lichtenthaler, 1987) as given below :

Chlorophyll a (mg/g FW)=(11.75 x A665- 2.35 x A645) x 3/25

Chlorophyll b (mg/g FW)=(18.61 x A645–3.96 x A665) x 3/25

Carotenoid (mg/g FW)=[(1000 x A454)-(2.27 x Chl a)-(81.4 x Chl b) /227] x 3/25

Quantities of these pigments were calculated in mg/g tissue fresh weight.

RESULTS AND DISCUSSION

In 2008-09, 37 populations were found to be highly resistant, 13 biotypes were found to be MR/MS and one population was S to fenoxaprop (120 g/ha) (Table 1). Similarly, 34 populations were found to be R to clodinafop (60 g/ha) and 16 were found to be MR and none was S. Out of these, 22 populations were S to pinoxaden 25 g/ha (½ X dose). In 2009-10, however, 17 out of 26 populations were S fenoxaprop (120 g/ha), seven were MR/MS and two were R. The level of efficacy with clodinafop was different, eight populations were highly resistant, 18 were MS/MR and none was S. Twenty-two populations were S to 50 g/ha pinoxaden (X dose) and four populations were MR/MS. When tested against 100 g/ha of pinoxaden (2X dose) 24 populations were S and two populations were MR/MS (Table 2).

The GR_{50} value for fenoxaprop for population from HAU, Hisar was 28 g/ha, it was 80 g/ha for the population from Ambala, 180 and 200 g/ha for populations from Uchana and Nangla and more than 240 g/ha for population from Chanarthal, Kurukshetra (Table 3). The GR_{50} values for clodinafop did not show the same trend. These were 30 and 35 g/ha in populations

Table 1. Relative efficacy of different herbicides on per cent mortality of *P. minor* biotypes collected from different locations in Haryana in 2008-09

S. No.	Populations	Per cent mortality		
		Fenoxaprop (120 g/ha)	Clodinafop (60 g/ha)	Pinoxaden (25 g/ha)
1.	Sirsa-1	6.6	70.0	20.0
2.	Sirsa-2	0.0	46.6	6.5
3.	Kalka	13.3	60.0	6.5
4.	Palwal	26.6	53.0	33.2
5.	Panchkula	20.0	40.0	26.5
6.	Fatehbad Bhodi	20.0	33.25	6.66
7.	Fatehbad Khanpur	26.6	60.0	53.3
8.	Fatehbad Akanwali	13.3	33.25	33.3
9.	Jind-1	33.3	50.0	60.0
10.	Jind-2	33.3	20.0	60.0
11.	Jind-3	26.6	20.0	40.0
12.	Jind-4	13.3	40.0	53.3
13.	Jind-5	6.66	33.3	6.66
14.	Hisar Umra-1	13.3	13.3	26.6
15.	Hisar HAU-2	13.3	6.66	26.6
16.	Hisar Kirtan-3	13.3	0.0	20.0
17.	Hansi-1	0.0	0.0	26.6
18.	Hansi-2	6.6	6.66	40.0
19.	Hansi-3	20.0	0.0	13.3
20.	Lalodha-1	33.3	20.0	40.0
21.	Lalodha-2	13.3	6.66	0.0
22.	Lalodha-3	13.3	0.0	13.3
23.	Chanarthal-1	20.0	0.0	26.6
24.	Chanarthal-2	0.0	0.0	6.66
25.	Chanarthal-3	0.0	0.0	0.0
26.	Knl-Kuk-4	0.0	26.6	33.3
27.	Knl-Kuk-5	6.66	6.66	33.3
28.	Knl-Kuk-6	13.3	0.0	53.3
29.	Knl-Kuk-7	13.3	0.0	20.0
30.	Ladwa-1	0.0	26.6	20.0
31.	Ladwa-2	26.6	6.66	13.3
32.	Ladwa-2	20.0	0.0	66.6
33.	Ladwa-3	40.0	13.3	20.0
34.	Ladwa-5	33.3	13.3	20.0
35.	Ladwa-6	53.3	20.0	66.6
36.	Karnal-1	33.3	26.6	100.0
37.	Uchana-2	46.6	26.6	100.0
38.	Uchana-3	66.6	33.3	100.0
39.	Ambala	60.0	13.3	100.0
40.	Rambha-5	66.6	13.3	80.0
41.	Asandh-1	80.0	20.0	73.3
42.	Asandh-2	5.3.3	40.0	73.3
43.	Asandh-3	66.6	46.6	86.6
44.	Kaithal-1	53.3	40.0	53.3
45.	Kaithal-2	40.0	40.0	86.6
46.	Indri-1	40.0	53.3	80.0
47.	Indri-2	46.6	70.0	66.6
48.	Indri-3	33.3	26.6	60.0
49.	Indri-4	53.3	40.0	73.3
50.	Gabhipur	40.0	60.0	100.0
	LSD (P=0.05) : Population : 11.3, Herbicides : 3.5, Population x Herbicides : 25			

Table 2. Relative efficacy of different herbicides on per cent mortality of *P. minor* biotypes collected from different locations in Haryana in 2009-10

S. No.	Population	Fenoxaprop (120 g/ha)	Clodinafop (60 g/ha)	Pinoxaden (50 g/ha)	Pinoxaden (100 g/ha)
1.	HAU, Hisar-1	100	55	100	100
2.	Hisar-2 (Gabhipur)	70	55	100	100
3.	Hisar-3 (Vaibhalpur)	75	45	100	100
4.	Hisar-4 (Nangla)	70	40	100	80
5.	Hisar-5 (Lalodha)	35	40	100	100
6.	Hisar-Jandli	55	50	100	100
7.	Fatehabad-1	85	20	100	100
8.	Fatehabad-2 (Dher)	100	30	100	100
9.	Fatehabad-3 (Vaidwala)	60	30	100	100
10.	Rania	70	45	100	100
11.	Dabwali-1 (Badaguda)	75	35	100	100
12.	Dabwali-2 (Jeewan Nagar)	70	35	100	100
13.	Dabwali-3 Sirsa road	85	40	100	100
14.	Dabwali-4 (Mithri)	100	45	100	100
15.	Dabwali-5	80	40	100	100
16.	Kaithal	85	35	85	100
17.	Narwana	55	20	88	90
18.	Jind-1 Rashidan	70	40	75	100
19.	Jind-2 Rashidan	0	10	55	45
20.	Kurukshetra (Chanarthal)	75	25	45	90
21.	Karnal-Uchana	25	25	50	100
22.	Karnal-Sagga	75	35	100	100
23.	Ambala-1 (Bhari)	65	15	55	50
24.	Ambala-2 (Jagtar)	85	35	100	100
25.	Kutail-1	45	35	70	90
	LSD (P=0.05) : Population : 10.5, Herbicides : 4.1, Population x Herbicides : 21.3				

from Ambala and Nangla and 60 g/ha in populations from Uchana and Chanarthal. A variation in GR_{50} values against pinoxaden was also observed. It was in the range of of 10-35 g/ha in populations from HAU, Hisar, Uchana, Ambala and Nangla and more than 120 g/ha in the population from Chanarthal. The resistance factor for populations from Chanarthal was >8.5 for fenoxaprop >2 for clodinafop and >12 for pinoxaden (Table 3).

Ion efflux from leaves after herbicide spray was more relative to unsprayed leaves. It was 2-4 times higher in populations from HAU, Hisar, Uchana, Ambala and Nangla after spray with fenoxaprop (120 g/ha), 1.5 - 4.0 times higher after spray with clodinafop (60 g/ha) and 4-6 times higher after spray with pinoxaden (Table 4). In Chanarthal population the ion efflux after spray with all the three ACCase herbicides was equal to unsprayed control. A similar response was observed with wheat (Table 4).

The decline in Chl a, Chl b and carotenoid contents in HAU, Hisar population after fenoxaprop spray was 86, 96 and 78%, respectively (Fig. 1). The populations from Uchana and Ambala showed a medium level of decline (25-50%) and those from Nangla and Chanarthal showed the least decline (8-25%). After clodinafop spray the decline in Chl a, Chl b and carotenoid contents was 57, 96 and 93%, respectively, in case of HAU, Hisar population. In case of populations from Uchana, Ambala and Nangla the decline in Chl a content was in the range of 3-18%, in Chl b content was in the range of 42-97% and 63-87% in carotenoid content. In population from Chanarthal, the decline was 0, 4 and 13% in Chl a, Chl b and carotenoid contents, respectively. With pinoxaden the decline in Chl a, Chl b and carotenoid contents was 100, 94 and 92% in case of population from HAU, Hisar. It was in the range of 43-97% in populations from Uchana, Ambala and Nangla. In case of populations from Chanarthal it was lower and in the range of 25-29% and was parallel to that in wheat. The populations from Nangla showed relatively lesser decline in all the pigments after fenoxaprop spray and more

Table 3. Effect of fenoxaprop, clodinafop and pinoxaden on GR_{50} value in different biotypes of *Phalaris minor* and wheat

Biotype	GR_{50} value (g/ha)	Regression equation $Y=(a+bx+cx^2)$	R^2	R. F.
		Fenoxaprop		
HAU, Hisar	28	$Y = 159.11 – 0.29x + 10.36x^2$	0.82	1.0
Uchana	180	$Y = 54.63 + 76.89x - 16.96x^2$	0.69	6.4
Ambala	80	$Y = 117.59 - 23.79x + 0.862x^2$	0.78	2.8
Nangla	200	$Y = -9.70 + 130.1x - 24.13x^2$	0.59	7.1
Chanarthal	>240	$Y = 94.8 + 4.742x + 11.143x^2$	0.97	>8.5
Wheat	>240	$Y = 78.16 + 12.20x - 0.914x^2$	0.18	>8.5
		Clodinafop		
HAU, Hisar	60	$Y = 99.16 + 16.84x - 5.65x^2$	0.78	2.0
Uchana	>60	$Y = 106.0 + 9.875x - 4.037x^2$	0.62	>2.0
Ambala	30	$Y = 122.6 - 11.73x - 2.173x^2$	0.88	1.0
Nangla	35	$Y = 115.2 + 9.115x - 6.122x^2$	0.72	1.1
Chanarthal	>60	$Y = 30 + 86x - 10x^2$	0.64	>2.0
Wheat	>60	$Y = 117.6 - 4.457x + 5.142x^2$	0.70	>2.0
		Pinoxaden		
HAU, Hisar	10	$Y = 172.4 - 90.18x + 11.39x^2$	0.94	1.0
Uchana	28	$Y = 148.7 - 42.42x + 2.79x^2$	0.85	2.8
Ambala	28	$Y = 148.7 - 45.031x + 2.79x^2$	0.95	2.8
Nangla	35	$Y = 162.8 - 44.62x + 2.048x^2$	0.71	3.5
Chanarthal	120	$Y = -124 + 275.43x - 48.57x^2$	0.80	12.0
Wheat	>120	$Y = 13.6 + 105.2x - 14x^2$	0.40	>12.0

decline after clodinafop and pinoxaden spray indicating that the population was resistant to fenoxaprop and susceptible to clodinafop and pinoxaden. In contrast, the population from Chanarthal showed similar decline after spray with all the three herbicides indicating resistance against all the three herbicides. The data paralleled with the GR_{50} values of the populations.

The data clearly indicate that the selection pressure exerted by ACCase inhibiting herbicides fenoxaprop and clodinafop has led to the evolution of cross resistance to these AOPP herbicides in *P. minor* populations at most of the locations in Haryana and is in conformity with the earlier findings (Yadav *et al.,* 2002; Chhokar and Sharma*,* 2008; Dhawan *et al*., 2009). A variable response of these populations to fenoxaprop in 2009-10 is, however, intriguing and perhaps has a bearing on the inheritance pattern of the resistant trait. A tetrasomic inheritance has been reported for *P. minor* resulting in higher heterozygosity as compared to the diploid progenitors (Matus and Hucl,1999). Inter- and intra-population diversity has been revealed by RAPD and ISSR markers (McRoberts *et al.,* 2005; Dhawan *et al*., 2009). Kachare *et al*. (2005) indicated the possibilities of involvement of both cytoplasmic and nuclear inheritance for isoproturon resistance in *P. minor*. Similar pattern may be followed for inheritance of the resistance trait against ACCase inhibitors.

Another point that emerged from this investigation was that the populations resistant to fenoxaprop did not necessarily show resistance towards clodinafop. The population from Nangla though exhibited resistance towards fenoxaprop was susceptible to clodinafop. In contrast, the population from Chanarthal was resistant to both the herbicides. This may be due to the fact that the herbicides bind to the same target site. Differences in target site cross resistance could occur as a result of selection of different mutations of ACCase gene in different resistant populations. A range of mutations and their combinations involving different alleles have been reported in *Lolium* populations (Yu *et al*., 2007).

Most of the populations resistant to aryloxyphenoxypropionate herbicides were susceptible to the new herbicide pinoxaden belonging to the phenylpyrazolin category of ACCase inhibitors. However, a few populations from Chanarthal, Ambala, Jind and Uchana showed resistance to this herbicide indicating cross- resistance to this herbicide without any prior exposure. A resistance factor of 3-12 could be observed in the populations tested. A low level of resistance was

Table 4. Effect of fenoxaprop, clodinafop and pinoxaden on ion efflux in different biotypes of *Phalaris minor* and wheat

Population	Control	120 (g/ha)	R. I. C.
		Fenoxaprop	
HAU, Hisar	7.8	35.0	4.4
Uchana	7.9	34.5	4.3
Ambala	8.8	28.3	3.2
Nangla	9.9	23.7	2.3
Chanarthal	13.1	13.2	0.007
Wheat	15.6	15.7	0.006
LSD (P=0.05)	Population	2.9	
	Fenoxaprop	1.4	
	Population x Fenoxaprop	4.1	
		Clodinafop-propargyl	
Population	Control	60 (g/ha)	
HAU, Hisar	7.8	24.2	3.1
Uchana	7.9	32.0	4.0
Ambala	8.8	13.8	1.5
Nangla	9.9	15.6	1.5
Chanarthal	12.8	13.1	0.02
Wheat	15.6	15.6	0.0
LSD (P=0.05)	Population	3.4	
	Clodinafop	1.7	
	Population x Clodinafop	4.8	
		Pinoxaden	
Population	Control	50 (g/ha)	
HAU, Hisar	7.8	35.7	4.5
Uchana	7.9	46.1	5.8
Ambala	8.8	42.0	4.7
Nangla	9.9	50.4	5.0
Chanarthal	13.1	13.2	0.007
Wheat	15.6	15.6	0.0
LSD (P=0.05)	Population	4.8	
	Pinoxaden	2.4	
	Population x Pinoxaden	6.8	

observed by Chhokar and Sharma (2008) also. A cross-resistance against pinoxaden has been reported in other weeds like *Alopecurus myosuroides*, *Lolium temulentum, Lolium perenne* and *Avena fatua* without any prior exposures (Kuk *et al*., 2008; Uludag *et al*., 2008; Ellis *et al*., 2010; Petit *et al*., 2010). A variation in the resistance factor is indicative of the possibility of different modes of resistance evolution to the herbicides. In *A. myosuroides* from France a likelihood of selection for pinoxaden resistance by ACCase inhibitors is suggested. In some other cases the selection by herbicides with other modes of action is also possible. A cautious use of metabolizable herbicides where non-target site based resistance has evolved is proposed. Although resistance against isoproturon in *P. minor* is reported to be metabolic in nature (Singh *et al.,*1998 a, b). Studies on the mode of evolution of resistance to ACCase herbicides in *P. minor* gain relevance since multiple modes of evolution of resistance are possible (De Prado *et al*., 2005).

In view of the heterogeneity observed in the weed and variable results observed with different herbicides, it becomes essential to evolve location specific herbicide management strategies in addition to the agronomic technology involved.

A prior detection of the kind of resistance at a specific location by laboratory assays would perhaps become mandatory for future management.

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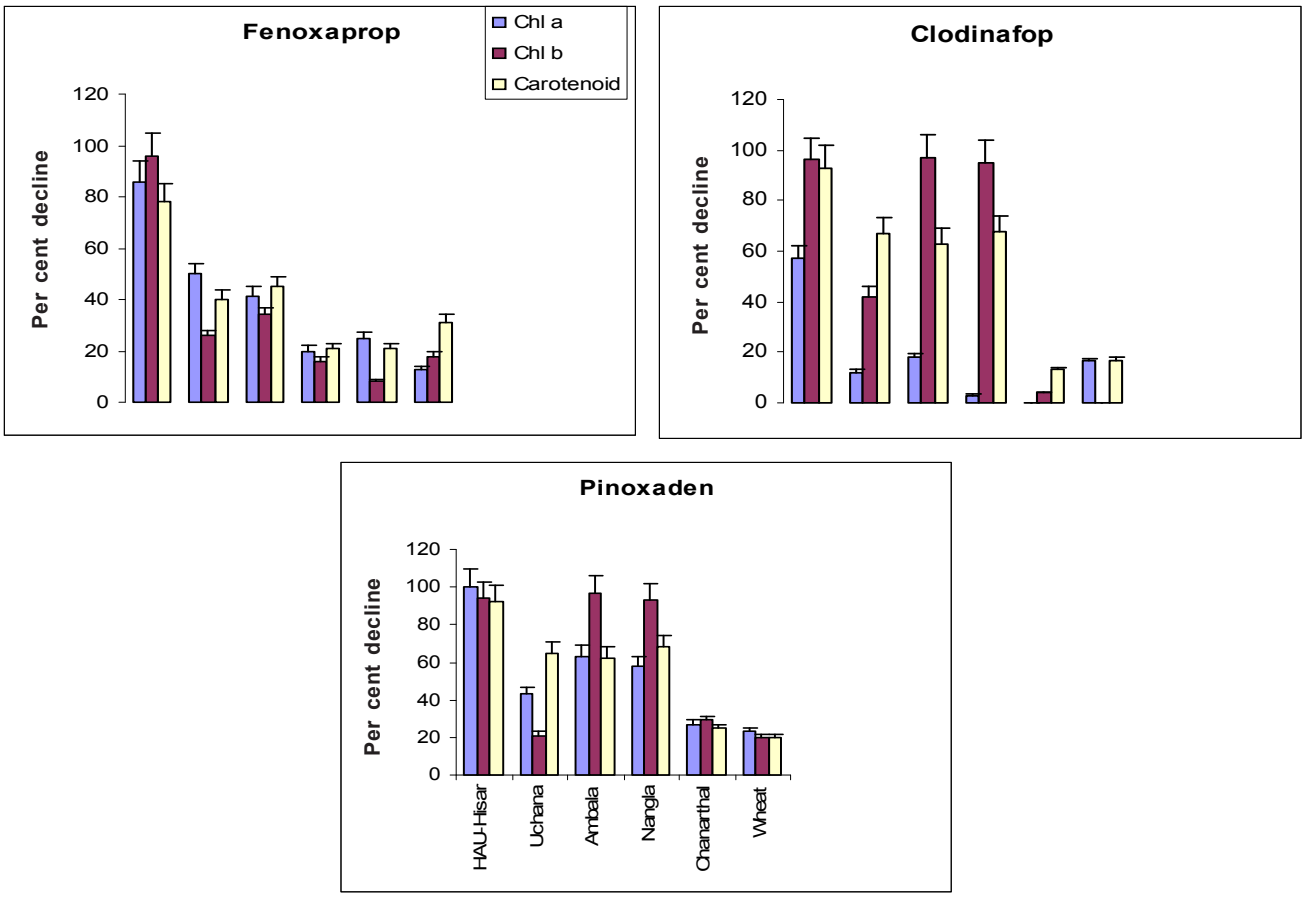

Fig 1. Effect of fenoxaprop 120 g/ha (a), clodinafop 60 g/ha (b) and pinoxaden 50 g/ha on per cent decline in chlorophyll a, chlorophyll b and carotenoid contents in different biotypes of *Phalaris minor* and wheat. Bars represent S. E.

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