Range Management and Agroforestry 45 (2) : 351-356, 2024
ISSN 0971-2070 (Print); 2249-5231 (Online)
https://doi.org/10.59515/rma.2024.v45.i2.22

Short communication

Inheritance studies of *Pyrenophora* leaf spot resistance genes in oat (*Avena sativa* L.) crop

Harshavardan J. Hilli[1, 2*], Rahul Kapoor[1], Harpreet Oberoi[1], Puja Srivastav[1], Ashlesha Singla[1] and Priti Sharma[1]

[1]Punjab Agricultural University, Ludhiana-141004, India
[2]Sanskriti University, Mathura-281401, India
[*]Corresponding author email: harshajh1995@gmail.com

Received: 09th November, 2023 Accepted: 15th November, 2024

Abstract

Oat is a hexaploid cultivated crop known for its usage as a fodder crop mainly, though being a dual purpose crop. Oats are affected by several abiotic and biotic stresses. Among them, foliar diseases like rust, leaf spot, smut, *etc,* are important and have threatened oat production. *Pyrenophora* leaf spot, which is a fungal disease that accounts for 40 to 50% yield losses in India. Keeping this in view, a study was conducted to identify the inheritance pattern of *Pyrenophora* leaf spot resistance genes by the previously developed F_2 population involving resistant and susceptible parents. The population showed a minimum of 22% DSI and a maximum of 83% DSI. The result showed that populations derived from parents were separated into 114 resistant individuals and 26 susceptible individuals, fitting into a gene ratio of (13 resistant: 3 susceptible) which is less than a table value with non-significant deviation observed for the trait studied, indicating observed and expected distributions were not same. Further ratio indicated that inhibitory gene action was involved in governing it.

Keywords: DSI, Gene postulation, Inheritance, Inhibitory action, Oat

Winter cereal crop oats (*Avena sativa* L.), a member of the genus *Avena* and family Graminae, had its origin in the Mediterranean region (Poaceae). It is a crop that is self-pollinated and has little variation (Rana *et al*., 2019). *A. sativa*, *A. nuda* and *A. byzantina* are the only species of the genus *Avena* that are grown for commercial purposes. The cultivated oats have chromosomal number 2n=6x=42 and are allohexaploid. In terms of production, oats come in sixth place after wheat, maize, sorghum, rice, and barley (Hilli *et al*., 2021; 2022). The world's top producer of oats is the European Union, followed by Russia, Canada, the United States of America, and Australia (Kumari *et al*., 2019). Oat grains are now more commonly consumed as human food as a result of their growing significance in breakfast cereal (Boshoff *et al*., 2019; Chaturvedi *et al*., 2016). The estimated total area in India for growing fodder oats is approximately 10.0 lac hectares, with Uttar Pradesh accounting for 34% of the total area, followed by Punjab (20%), Haryana (9%) and Madhya Pradesh (6%). Other states, such as Uttaranchal, Maharashtra, Gujarat and Orissa, share the rest of the area. In Punjab, oat is grown as fodder crops on 1.0 lac hectares (Anonymous, 2020).

With the exception of foliar diseases, oats are relatively less susceptible to pests and diseases (Rosentrater and Evers, 2018). In locations where oats are grown, diseases like crown rust, stem rust, Stagonospora 3 avenae leaf blotch, and *Drechslera avenae* leaf spot are common. This extends into China, Korea, Japan, and Malaysia, as well as all of North and South America, Europe, Russia, Australia, New Zealand, the Indian subcontinent, and parts of Africa (Carmona *et al*., 2004; Filipas *et al*., 1997; Ariyawansa *et al*., 2014; Hetherington *et al*., 2002; Kim *et al*., 1995; Morrissey *et al*., 2000; Martens, 1985; Leonard 2003; Cunfer, 2000). *Drechslera avenae* (teleomorph: an important foliar disease of oats caused by a necrotrophic fungal pathogen) is a member of the class Dothideomycetes, subclass Pleosporomycetidae, order Pleosporales, and family Pleosporaceae (Carmona *et al*., 2004; Ariyawansa *et al*., 2014). The fungus that causes *D. avenae* leaf spots in oats was first identified as *Helminthosporium teres* (var.) avenae-sativae in Italy in 1889 (Briosi and Cavara 1889). Two years later, the same fungus was characterized independently as an oat-specific disease that was not specific to wheat or barley and was given the name of *H. avenae* (Eidam, 1891). Additionally, later on, oats were shown to be infected with *Helminthosporium* in

Holland, Denmark and the United States (Johnson and Brown, 1940; Turner and Millard, 1931). The two fungal species were determined to be identical and given the name *H. avenae* based on the similarities of the fungus (pathogens) isolated from Italy, Holland, Denmark, and the USA (Dennis, 1933). The growth of *D. avenae* and the infection process are similarly best facilitated by cool temperatures and high humidity (Turner and Millard, 1931). Particularly, the disease was mainly recorded in areas where it rained and had chilled weather during planting season (De Tempe, 1964). The mycelium can survive at 14°C for at least 56 days in laboratory settings, but the pathogen can survive at a minimum temperature of 2 to 3°C and an optimal temperature of around 20°C (Dennis, 1933).

In 1984, *D. avenae*, a common oat disease, significantly damaged oat crops in the southern United States, Western Europe, Japan, and India (Meyers, 1985). In addition to being the most serious oat disease in Canada in the 1920s (Drechsler, 1923) and Scotland in the following decade (O'Brien and M'Naughton, 1933), it was also economically significant in Denmark (Turner and Millard, 1931). When leaf spot epidemics hit Germany and the southern United States, losses were estimated to be between 30 and 40% (Gough and McDaniel, 1974). In Finland and Sweden, where the disease is frequently seen as a seed-borne pathogen, yield losses of up to 10% were documented, whereas, in India, the disease was shown to harm oats from the seedling stage to the maturity of the plant. Brazil believed *D. avenae* to be the main oat kernel pathogen despite the rarity of severe infestations. Instances of seed-borne infection often occur at a rate of roughly 14% (Kunovski and Breshkov, 1981), sometimes even at rates of 40 to 70%, but the yield losses were only 3 to 5% (Olofsson, 1976). The present method of effective control through seed treatment was the source of this minimal yield loss. Due to the pathogen's ability to thrive on crop residue, the disease is still prevalent and significant in areas that grow oats, but the application of efficient fungicides could reduce output losses (Lashram, 2019).

Leaf spots initially appear as small, rectangular patches with white centers and a reddish-brown halo (Ellis and Barnes, 1980). Later, they turn dark with occasionally sunken centers, frequently a purplish brown or grey tone, and a reddish-brown edge encircled by a lighter halo that gradually blends into the leaf's typical green color (Turner and Millard, 1931). Additionally, these distinct spots unite to create striped lesions that are laterally constrained by the leaf veins (Chełkowski, 1995). Further, due to the presence of fungal mycelium, which varies in color depending on its density and enzyme activity, the infection can spread to grain sheath, causing the spikelet to droop and kernels to become shriveled, darkened, lightweight, and of poor quality (Lashram, 2019; Clark *et al*., 2008). Due to the propagation of fungus, the infection also causes nodes next to infected leaves to darken, resulting in a symptom known as 'black stem' (Luke *et al*., 1957; Jones and Clifford, 1983). In severe cases, the fungal mycelia are found in the stem cavity of infected stems, which makes them fragile and prone to breaking (Luke *et al*., 1957; Clark *et al*., 2008).

The most cost-effective and ecologically friendly method of disease management in plants is genetic resistance, which is a primary goal in the majority of crop breeding efforts. The categorization of disease resistance is a controversial subject, and there is a wealth of literature discussing the advantages of various forms of resistance (Buschiazzo and. Gemmell, 2006). There is a continuum of resistant phenotypes, from the hypersensitive reaction to a small slowing of the pace at which epidemics spread. There are many combinations of locus number, allele effects, stage of expression, and race specificity throughout the continuum (Browning and Ayanni, 1977). According to reports, dominant and recessive genes regulate how resistance is inherited, and in certain situations, epistasis was also observed (Stefenson *et al*., 1996). Three dominant epistatic genes were observed to be involved in the genetic regulation of spot blotch (Sharma *et al*., 1997; Kapoor *et al*., 2023). Recessive genes that work together in harmony offer resistance to wheat blight. In addition, Sharma *et al.* (1997) demonstrated a quantitative inheritance of spot blotch resistance in wheat, where a single dominant gene controlled resistance. Again, for the breeding of resistant cultivars, knowledge of the chromosomal location of resistance genes is particularly helpful. By applying marker-assisted selection, the discovery of closely related molecular markers and disease-resistance genes promotes the selection and transfer of disease-resistance genes (Weerasena *et al*., 2004; Hilli *et al*., 2024; Kapoor *et al*., 2022). Keeping the above in view, the present study was conducted to identify the inheritance pattern of *Pyrenophora* leaf spot resistance genes by the previously developed F_2 population involving resistant and susceptible parents (Contreras-Govea and Albrecht, 2006).

The study was conducted to identify the resistant and susceptible source for leaf spot disease artificially during 2019-20 *Rabi* season. Further, the parental line, *i.e*., EC/0007662 (resistant) and EC/0131291 (susceptible), were used for the hybridization and development of F2 population. Here F1s produced were sent to off-season Keylong (India) to advance the generation and to get F2 seeds. Further, F2 seeds were planted at the Forage section, Punjab Agricultural University, Ludhiana, to get F2 plants and these were artificially screened through inoculation of fungal spores (Hilli *et al*., 2022). The average rainfall of the region was 700 mm and the weather was favorable during the entire crop growth. Materials were evaluated with a recommended package of practices. The F2 plants were analyzed for disease reaction and data

was scored according to the scale proposed by Chang *et al*. (2014). Also, the inheritance pattern for disease was studied by chi-square analysis. Figures displaying the resistant and susceptible nature of parental lines were recorded (Fig 1) and also the disease severity index and frequency distribution of populations were recorded (Table 1). Further these disease ratings given were put in the below-mentioned formula to calculate the disease severity index (DSI) in percent. Here, the scale was based on a percent of leaves covered with spots (symptoms), which were used for assigning particular ratings to each line and then a formula was used to calculate DSI [DSI (%) = {(Sum of disease ratings)/(Total number of ratings x Maximum grade) x 100}]

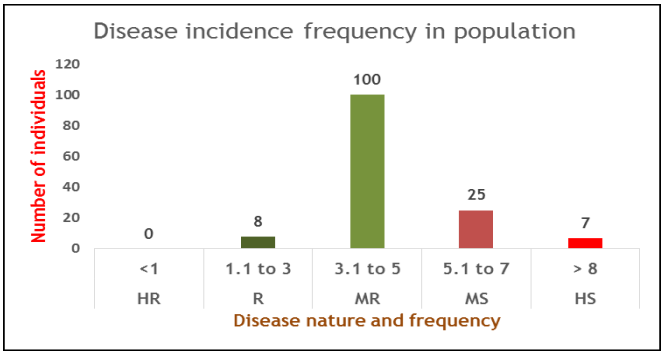

Fig 1: Frequency distribution of the individuals for disease incidence

In the experiment for blight resistance, 140 F2 plants were screened. The details of the resistance and susceptible natures were recorded (Table 2). At 5 and 1% level of significance, the F2 generation of the cross EC/0007662 x EC/0131291 separated into 108 resistant individuals and 32 susceptible individuals, fitting into an inhibitory gene action ratio of 13 resistant: 3 susceptible (Table 2) with chi-square of 0.453 which was less than a table value with non-significant deviation observed for the trait studied indicating observed and expected distributions were not same. While grouping, MR and R were considered as resistant and S and HS as susceptible plants for chisquare analysis. This indicated that one dominant inhibitory gene and one dominant recessive gene controlled resistance. In this instance, the dominant allele of the first gene resulted in resistance, but the recessive allele caused a disease. The first dominant inhibitory gene produced a character, whereas the second dominant inhibitory gene inhibited that character. Characters that were produced by recessive homozygotes of the first gene were formed when both genes were present in dominant form. The recessive allele of the second gene did not have its own character, but when it was present in the dominant form, it inhibited the expression of the first gene. Also, the resistant parent showed 22% DSI at 3 rating and the susceptible showed 78% DSI with 8 rating. Here by seeing the population data, it was assured that some transgressive segregants were present since the population also showed disease reactions beyond the

Table 1. Disease severity index of the F2 population

S.N.	DSI (%)	S.N.	DSI (%)	S.N.	DSI (%)	S.N.	DSI (%)
1	33.33	40	33.33	79	81.48	118	40.74
2	29.63	41	40.74	80	40.74	119	44.44
3	59.26	42	70.37	81	33.33	120	44.44
4	44.44	43	22.22	82	29.63	121	48.15
5	81.48	44	66.67	83	33.33	122	44.44
6	44.44	45	40.74	84	33.33	123	51.85
7	33.33	46	62.96	85	37.04	124	55.56
8	29.63	47	33.33	86	40.74	125	51.85
9	62.96	48	29.63	87	44.44	126	40.74
10	33.33	49	29.63	88	59.26	127	48.15
11	29.63	50	40.74	89	48.15	128	37.04
12	29.63	51	44.44	90	44.44	129	55.56
13	70.37	52	70.37	91	37.04	130	44.44
14	25.93	53	44.44	92	40.74	131	55.56
15	29.63	54	44.44	93	33.33	132	59.26
16	66.67	55	40.74	94	37.04	133	44.44
17	37.04	56	40.74	95	44.44	134	33.33

S.N.	DSI (%)	S.N.	DSI (%)	S.N.	DSI (%)	S.N.	DSI (%)
18	37.04	57	33.33	96	40.74	135	29.63
19	44.44	58	37.04	97	44.44	136	37.04
20	77.78	59	48.15	98	48.15	137	40.74
21	40.74	60	51.85	99	44.44	138	33.33
22	70.37	61	48.15	100	44.44	139	37.04
23	85.19	62	81.48	101	33.33	140	44.44
24	25.93	63	44.44	102	40.74		
25	33.33	64	40.74	103	48.15		
26	33.33	65	59.26	104	33.33		
27	29.63	66	70.37	105	37.04		
28	33.33	67	44.44	106	37.04		
29	37.04	68	55.56	107	37.04		
30	40.74	69	48.15	108	37.04		
31	48.15	70	22.22	109	40.74		
32	85.19	71	22.22	110	44.44		
33	48.15	72	29.63	111	44.44		
34	77.78	73	40.74	112	48.15		
35	44.44	74	33.33	113	40.74		
36	29.63	75	92.59	114	48.15		
37	33.33	76	51.85	115	55.56		
38	74.07	77	44.44	116	48.15		
39	22.22	78	40.74	117	37.04		

Table 2. Inheritance studies in oats for *Pyrenophora* leaf spot

Population	Class	Observed (O_i)	Expected (E_i) 13:3	χ^2 (Chi square 13:3 value)	Gene governing resistance nature
F2	Resistant (MR + R)	108	114	0.315	One recessive and one dominant gene (inhibitory gene action)
	Susceptible (S + HS)	32	26	0.138	
	Total	140		0.453 (1df) ^{ns}	

parental scoring. The current findings were consistent with those of Chen and Line (1992) and Braithwaite *et al.* (1998), who investigated the transmission of several barley stripe rust pathotypes against various F2 populations of barley lines. Their F2 population was separated into 217 resistant individuals and 43 susceptible individuals, resulting in an inhibitory action of 13:3, showing resistance through recessive genes.

The F2 plants of cross EC/0007662 x EC/0131291 developed were screened for disease incidence artificially to study the inheritance pattern of genes. The result showed that lines separated into 108 resistant individuals and 32 susceptible individuals, fitting into an inhibitory gene ratio of 13:3, which was less than a table value, indicating non-significant deviation observed for the trait studied, indicating observed and expected distributions were not the same and the transgressive segregants were noticed since the disease severity by populations showed beyond the parental range.

Acknowledgment

The senior author would like to thank Punjab Agricultural University, Ludhiana for allowing him to conduct the experiment as part of his PhD research work.

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