Inheritance of Resistance to Stalk Rot in Cauliflower (*Brassica oleracea* var. *botrytis* L.)

Bhupinder Singh Thakur

Regional Horticultural Research Station, Dr Y S Parmar University of Horticulture and Forestry, Bajaura, Kullu Himachal Pradesh (175 125), India

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Correspondence to

E-mail: bhupindert@gmail.com

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Abstract

Germplasm of late group cauliflower was screened under artificially inoculated conditions for resistance to stalk rot. This screening of the available germplasm revealed that Janavon and RSK 1301 were the genotypes resistant to stalk rot. These resistant genotypes were selected as parents for making crosses with the common high yielding susceptible varieties. Therefore crosses between susceptible PSB1 (commercial variety) and KJ 47 (elite line) and resistant parents (Janavon and RSK 1301) were attempted to work out the inheritance pattern of the disease resistance. All the F_1's of susceptible×resistant parents with mean disease incidence of 60.67 to 71.19% were found to be susceptible to stalk rot disease. As resistance was not shown by any of the F_1's, therefore it was clear that the genes responsible for resistance could not express themselves. The disease reaction recorded in the F_1's revealed that resistance to stalk rot was recessive to susceptibility. The intermediate disease reaction in the F_2 generation and low disease reaction in the back cross generations indicated that resistance to stalk rot is polygenic in nature. Additive effect of the genes was evident from the Generation means analysis revealed pronounced effect of additivity of genes in inheritance of the resistance to stalk rot.

1. Introduction

Cauliflower (*Brassica oleracea* var. *botrytis* L.) is one of the most important cruciferous vegetable crops in India and popularity of the crop has been constantly increasing with the introduction of hybrid varieties, which have wide adaptability and high yield potential as well. Himachal Pradesh is also contributing to the national production through cultivation of off-season crop and seed of the crop as well which is considered as the monopoly of the state. Besides, other cruciferous vegetables are also grown in the state on large scale, due to this reason, some bottlenecks in the form of diseases and insect pests have emerged and become synonymous with these cruciferous crops. The diseases like black rot, stalk rot and curd rot complex are the most severe throughout the state. Stalk rot or white mold (*Sclerotinia sclerotiorum* (Lib) deBarry) damages cauliflower seriously during the critical stages of its growth and development. It has been reported to cause losses up to 90% in seed crop (Sharma, 1979), thus needs utmost attention. The resistance breeding work on this disease in cauliflower is being done but no significant achievement has been made so far. Keeping in view the above facts and the seriousness of the disease, an effort was made to screen out the source of resistance from available genotypes of late cauliflower and to find out the mode of inheritance of resistance.

2. Materials and Methods

In the present studies, 25 genotypes comprising of standard cultivars such as PSB 1 And PSB K 1 and other elite lines (Table 1), were evaluated for resistance against stalk rot during 2005-06 at Research Farm of Regional Horticultural Research Station, Bajaura, Kullu Dr. YS Parmar University of Horticulture and Forestry, Himachal Pradesh. After identification of the sources of resistance different crosses were attempted during the subsequent years involving susceptible and resistant parents. The segregating generation F_2 and backcross generations were also developed. All the generations including parents, F_1's, F_2's and back crosses were evaluated as well as screened during 2006-07, 2007-08 and 2008-09 in a randomized block design for stalk rot resistance, under artificially created epiphytotic conditions in three replications.

2.1. Screening

Screening of the germplasm for stalk rot resistance was done by artificially inoculating the plants with the pathogen. The

Table 1: Incidence of stalk rot and the disease reaction of genotypes

Name of the Lines/ Genotypes	Disease incidence (%)		Reaction
	Laboratory (Seedling stage)	Field (Leaf lesion Basis)	
KJ 38	58.09	56.91	HS
KJ 47	57.28	63.59	HS
KM 1	36.11	33.29	S
Snowball 16	81.50	67.64	HS
Pyramis	18.70	19.21	MR
Monopreco	27.50	22.95	S
Janavon	9.71	7.66	R
Holland Special	72.32	58.10	HS
Himanshu Snow	68.77	62.52	HS
Pusa Himjyoti	88.25	74.16	HS
EC 162587	11.48	10.43	MR
RSK 1301	8.78	9.40	R
BR 2	47.82	42.36	HS
SN 445	75.45	55.82	HS
KT 9	87.25	59.06	HS
KT 25	75.73	56.79	HS
Pusa Synthetic	31.91	35.58	S
Kathmandu Local	55.15	54.21	HS
Janavon S 3	12.87	14.40	MR
KN 81	17.16	12.43	MR
KK 104	24.16	21.37	S
ACC 641	23.55	20.76	MR
Jawahar Moti	84.78	65.92	HS
PSB 1	86.95	72.52	HS
PSBK 1	89.65	67.18	HS

HS=highly susceptible; S=susceptible; MR=Moderately Resistant; R=Resistant

inoculation was done with a week old culture of *Sclerotinia sclerotiorum* multiplied on corn:sand medium (1:1 w/w) as suggested by Dohroo (1988) by mixing the culture in soil around the plant. The quantity of culture mixed was 5^{-10} mg for seedling and 50 mg for mature plant at curd initiation stage. To maintain high soil moisture, irrigations were provided every fourth day. Inoculation was also made on the curds by placing mycelium on it. The disease incidence based on number of leaves affected and later plants showing the disease symptoms were recorded after 20 days of inoculation and subsequently at weekly intervals till the final harvest. The final disease incidence figures were worked out after taking the mean of all the readings and the genotypes were grouped into four categories as resistant (1-10%), moderately resistant (11-20%), susceptible (21-40%) and highly susceptible (>40%). The mean disease incidence of four crosses was analyzed for estimating the gene effects through generation mean analysis.

3. Results and Discussion

Out of the 25 genotypes screened for the disease resistance, majority showed the susceptible reaction (Table 1). The genotypes Janavon, EC 162587 and RSK 1301 were found to be resistant with less than 10% incidence of stalk rot. Janavon S 3, Pyramis and KN 81 with 11-20% disease incidence were moderately resistant, while all remaining genotypes were susceptible to highly susceptible in nature. Janavon and RSK 1301 were used as resistant parents while PSB 1 (Commercial variety) and KJ 47 (elite line) were used as susceptible parents in the crossing programme. The mean disease incidence of the parents, crosses and segregating generations was analyzed for generation means.

All the F_1's of susceptible×resistant parents with mean disease incidence of 60.67 to 71.19% were susceptible. As resistance was not shown by any of the $F_{1's}$, it was clear that the genes responsible for resistance could not express themselves. So owing to non-expression of the resistance genes they can be said to have the recessive ness to the genes responsible for susceptibility. Further perusal of the data (Table 2) shows that interaction phenotype of the disease in F_1 generation although

Table 2: Generation mean analysis for stalk rot resistance

Crosses	Generation Means					
	P_1	P_2	F_1	F_2	B_1	B_2
PSB 1×Janavon	72.52	7.66	71.19 (40.09)	56.41 (55.64)	65.32 (71.85)	49.18 (39.42)
PSB1×RSK 1301	72.52	9.40	64.66 (40.96)	57.45 (52.81)	69.81 (68.59)	45.05 (37.03)
KJ 47×Janavon	63.59	7.66	60.67 (35.63)	51.74 (48.14)	63.75 (62.13)	46.28 (34.16)
KJ 47×RSK 1301	63.59	9.40	58.05 (36.49)	54.63 (47.28)	60.70 (60.89)	45.60 (33.74)
RSK1301×Janavon	9.40	7.66	8.17 (8.53)	10.13 (8.35)	9.03 (8.78)	8.20 (33.74)

Note: Values in the parenthesis are expected values

Table 3: Scaling test for stalk rot resistance

Cross	A	B	C	Joint scaling
PSB 1× Janavon	-13.07*±5.68	19.51±4.30	3.08±6.52	30.48
PSB 1 ×RSK 1301	2.44±6.64	16.03*±6.96	18.56±13.01	6.89
KJ 47× Janavon	3.23±3.59	24.23*±4.83	14.37±9.60	25.70
KJ 47× RSK 1301	-0.27±3.27	23.73*±7.96	29.37*±6.09	32.09
RSK 1301× Janavon	-0.49±1.72	0.69±3.46	7.12±5.77	-

*Significant at 5% level

Table 4: Gene effects for stalk rot resistance

Cross	m	d	h	i	j	l
PSB 1×Janavon	56.41*±0.99	16.14*±2.82	34.45*±7.37	3.35±6.90	-16.28±3.27	-9.79±13.04
PSB1× RSK 1301	57.45*±3.02	24.76*±4.34	23.61*±15.09	0.08±14.90	-6.79±4.65	-18.39±21.72
KJ 47×Janavon	51.74* ±2.14	17.46*±2.54	38.13*±10.19	13.08±9.96	10.49*±2.65	40.55*±13.97
KJ 47×RSK 1301	54.63*±1.44	15.09*±4.21	15.66±4.21	-5.93±10.20	-11.99*±4.28	17.52±17.92
RSK1301×Janavon	10.13±2.38	0.8±1.23	-6.36±4.33	-6.00±7.82	0.07±1.01	4.88±3.42

*Significant at 5% level

on susceptible side but was not complete. Dickson and Petzoldt (1995) also reported resistance to stalk rot or white mold to be recessive in *Brassica oleracea*. The average disease incidence in the F_2 population was less than F_1 and it varied from 51.74% in KJ 47×Janavon to 57.5% in PSB 1×RSK 1301. The expected and observed values in most of the crosses were at par with each other, which indicated that the genes responsible for resistance have their cumulative effect in reducing the disease incidence. Further, the mean disease incidence in F_2 generation of the susceptible×resistant crosses was lower than their respective F_1's and were intermediate to their respective parents and in susceptible range. This type of difference in the mean disease incidence could be attributed to the involvement of more number of genes and partial dominance of the susceptibility operating in the inheritance of disease resistance. These results are in line to those reported by Baswana et al. (1991). Coyne et al. (1977) while working on *Phaseolus vulgaris* L. have also reported low habitability of resistance to white mold.

In the back cross to susceptible parent there was an increase in the disease incidence in all the crosses while the back crosses to the resistant parent showed reduction in the disease incidence. The reduction in disease incidence in the back cross to the resistant parent indicated accumulation of more number of genes which controls the resistance, whereas back crossing to the susceptible parent has decreased their number hence increase in the disease incidence. This indicates clearly towards the additive nature of the genes controlling resistance. Agbo and Wood (1979) and Fuller et al., (1984) also reported that resistance to white mold (*Sclerotinia sclerotiorum*) was quantitatively inherited in beans. The back crosses especially B_2 also showed deviation from the expected mean values thus indicating involvement of other factors or gene effects in the inheritance of resistance. Additive×dominance model was found to be inadequate for all the crosses. On the basis of six-parameter model (Table 2) it can be concluded that additive gene action played an important role in the inheritance of resistance to stalk rot. All the crosses gave significant influence of additive×additive (i) gene effects. Only one cross KJ 47×Janavon gave complementary gene action as both dominance×dominance (l) as well as dominance (h) gene action were positive and significant. Fuller et al. (1984) Baswana et al. (1991) and Dickson and Petzoldt (1995) also reported preponderance of additive gene effects in inheritance of resistance to white mold.

4. Conclusion

These investigations revealed that there are a number of genes involved in controlling the resistance, which act in an additive fashion and have low heritability. Presence of some complementary gene action cannot be ruled out. These results also indicate that rigorous selection may help in bringing improvement for stalk rot resistance, which has earlier been successfully utilized by Dickson and Hunter (1982) in breeding of bean varieties resistant to white mold.

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