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Genetic analysis of bacterial wilt resistance in chilli (*Capsicum annuum* L.)

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ABSTRACT

Bacterial wilt, caused by Ralstonia solanacearum, is a major concern for chilli production, leading to substantial yield losses. As a highly destructive soil-borne pathogen, R. solanacearum poses a significant threat to hot pepper (Capsicum annuum L.) cultivation worldwide. In India cultivated pepper fields, the relentless spread of R. solanacearum is exacerbated by global warming, posing a serious threat to crop productivity. The most sustainable strategy to combat bacterial wilt lies in the development of resistant pepper varieties. However, resistance to bacterial wilt is quantitatively inherited and varies depending on specific R. solanacearum isolates. Thus, this study aimed to assess the inheritance pattern of bacterial wilt resistance by analyzing an F_2 population of 157 plants along with their parental varieties, B-HP-143 and B-HP-144. In addition, disease reactions in 100 recombinant inbred lines (RILs) from the F_5 population were screened to evaluate disease susceptibility. The severity and progression of bacterial wilt were quantified using the area under the disease progress curve (AUDPC). The analysis of bacterial wilt resistance confirmed a polygenic inheritance pattern in the F_2 population. Several RILs such as, 84, 101, 106, 149, 155, 196, 210, 220, 232, 242, 283, 301, 307, 315, 324, 333, 336, 340, and 342 along with the resistant parent B-HP-143, exhibited complete resistance to bacterial wilt with no signs of infection. In contrast, B-HP-144 displayed a 70% incidence of bacterial wilt. The calculated AUDPC value for the F_2 generation was 545.54, while B-HP-143 and B-HP-144 had values of 0.00 and 735, respectively. These results highlight B-HP-143 as a promising source of high resistance. The incorporation of resistant RILs into breeding programs can greatly enhance the development of bacterial wilt-resistant hot pepper varieties. The findings of this study provide a crucial foundation for integrating bacterial wilt resistance into elite commercial hot pepper genotypes, contributing to future crop improvement efforts.

Keywords: *AUDPC, bacterial wilt, Chilli, inheritance, Polygenic, RIL and screening*

Introduction

Chilli (*Capsicum annuum* L.), commonly known as pepper, is a globally significant vegetable crop and a member of the Solanaceae family. Notably, Asia accounts for approximately 68% of its total production [1]. Chilli is also referred to as hot pepper and has become an essential component of diverse culinary and agricultural systems worldwide. [2]. Chilli belongs to genus *Capsicum* which includes the species *C. chinense, C. baccatum, C. frutescens, C. pubescens* and *C. annuum*. Of these five species, *C. annuum* is the most important one because it is cultivated throughout tropical and temperate area in the world and it is the most versatile of the five species [3]. In contrast, the other four species are cultivated in limited regions of the world or only in tropical areas and they are mainly used as condiments. Chilli have adapted remarkably well to Indian agro-climatic conditions, making India a recognized secondary center of origin [4].

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As an often cross-pollinated, diploid crop with a chromosome number of 2n = 2x = 24, chilli exhibits extensive genetic diversity. Botanically classified as berries, chilli fruits are categorized based on characteristics such as pungency, color, shape, and usage. When mature red pepper pods are dried and ground, they become one of the most widely consumed condiments globally. Due to their pungency, spicy flavor, vibrant color, and distinctive aroma, chillies are an indispensable ingredient in cuisines worldwide. Additionally, biochemical compounds such as carotenoids, capsanthin, and capsorubin serve as natural colorants, while capsaicinoids have significant applications in the food, medicinal, and pharmaceutical industries. In India, major chilli-producing states include Andhra Pradesh, Karnataka, Maharashtra, Odisha, Tamil Nadu, Madhya Pradesh, West Bengal, and Rajasthan. The country cultivates chilli across approximately 363,000 hectares, yielding a production of 4,027,000 metric tons with an average productivity of 11.09 MT/ha [5]. In Odisha, this is cultivated in the area of 65.50 (000 ha) with a production of 64.50 (000) MT [6]. Chilli thrives in warm, humid tropical and subtropical regions. However, its cultivation is often constrained by various biotic and abiotic factors, leading to reduced growth, fruit set, yield, and quality.

In major chilli-growing areas, continuous cultivation in both open fields and polyhouses has intensified the impact of soil-borne pathogens. Among these, *R. solanacearum* is a major threat, leading to severe yield losses due to bacterial wilt. This soil-borne pathogen significantly constrains chilli production, making disease management and resistance breeding critical for sustainable cultivation. The disease is particularly severe in warm or hot weather conditions, especially during the flowering stage. Wilting severity, often indicated by bacterial ooze, serves as a key diagnostic factor for this disease. For effective resistance breeding, understanding the inheritance pattern of disease resistance is crucial. Therefore, this study was conducted to investigate the genetic basis of bacterial wilt resistance in chilli.

Materials and Methods

The present investigation was conducted at the Vegetable farm, Odisha University of Agriculture and Technology, Bhubaneswar, India, from 2017 to 2020. The experimental site is situated at 20°15′ N latitude, 85°53′ E longitude, and an altitude of 25.5 meters above mean sea level. The study was carried out in highly acidic red laterite soil (pH 4.4) with a medium organic carbon content. The soil had available nitrogen (N), phosphorus (P), and potassium (K) levels of 296, 3.92, and 157 kg/ha, respectively. Given its highly acidic soil and hot, humid tropical climate favorable for bacterial wilt incidence coastal Odisha is considered a hotspot for screening chilli varieties for bacterial wilt resistance in India. The experiment involved 157 F_2 plants, 100 recombinant inbred lines (RILs) and two parental lines (B-HP-143 and B-HP-144) arranged in a randomized complete block design (RCBD) with two replications. The crop was cultivated following standard agronomic practices, with a planting spacing of 60 × 45 cm. Among the parental lines, B-HP-143 exhibited high resistance to bacterial wilt.

a. Genetic analysis (F_2 population) and Screening (F_5 population) for bacterial wilt resistance

To investigate the inheritance of bacterial wilt resistance, the F_2 population, along with the parental lines, was artificially inoculated with *R. solanacearum* and subsequently planted in a sick plot during the rainy season of 2017–18. Screening of RILs from the F_5 population against *R. solanacearum* was conducted during the rainy season of 2019–20. The inheritance pattern of bacterial wilt resistance was analyzed using the Chi-square test to determine the segregation ratio and genetic basis of resistance.

b. Isolation, culturing, maintaining of pathogen

In this study, the *R. solanacearum* pathogen was isolated and cultured from bacterial wilt-infected chilli plants collected from the CHES, Bhubaneswar field (Fig. 1A and B). The bacterial culture was grown on TZC (Triphenyl Tetrazolium Chloride) medium using the spreading method [8]. Within one to two days, distinct bacterial colonies with a creamy white appearance and a pointed pink center were observed. For further propagation, sub-culturing was performed using the streaking method to maintain pure cultures of *R. solanacearum*.

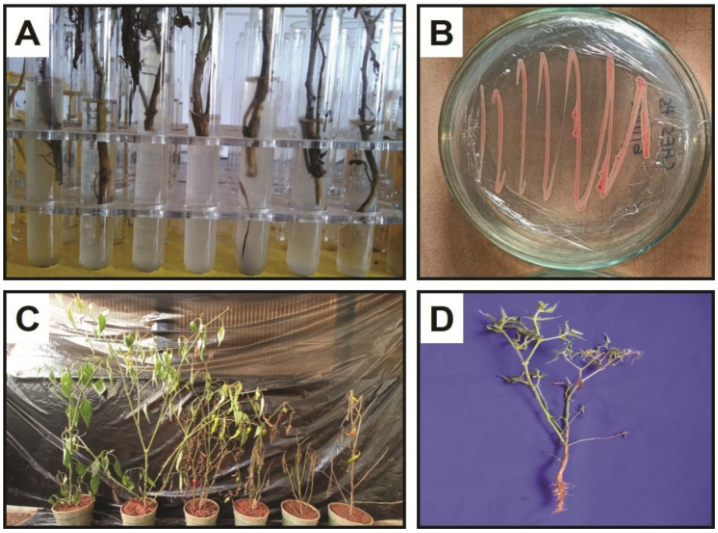

Fig. 1. Isolation, screening, and scoring of *R. solanacearum* and bacterial wilt in chilli **A.** Individual plant wilting confirmed using the ooze test. **B.** *R. solanacearum* pathogen isolated and cultured from infected plants. **C.** Bacterial wilt scoring conducted 35 days post-inoculation. **D.** Chilli plants exhibiting bacterial wilt symptoms.

a. Chemical composition TZC agar medium

Chemical	g/lit
Casein hydrolysate	1.00 g
Peptone	10.00 g
Glucose	5.00 g
Agar	17.00 g
2,3,5- Triphenyl Tetrazolium Chloride	0.05 g
Distilled water	1000 ml

For the preparation of 150 mL of nutrient broth solution, 2 g of nutrient broth powder was dissolved in 150 mL of distilled water and then autoclaved. After sterilization, 5 mL of 1% TTC (Triphenyl Tetrazolium Chloride) solution was added for staining. Pure *Ralstonia solanacearum* colonies from subcultures were inoculated into the broth using a sterile loop and incubated in a shaking incubator at 28°C with a rotation speed of 150 rpm. After 24 hours of incubation, the bacterial culture was centrifuged at 4000 rpm for 15 minutes at 4°C. The resulting white pellet was resuspended in the required volume of distilled water. The bacterial concentration was adjusted to 10^7 CFU/mL using a spectrophotometer, maintaining an optical density (OD) of 0.3 at A_{650} [9].

d. Artificial inoculation of plants

Before transplanting into sick plots, the root systems of one-month-old seedlings were carefully trimmed using a sharp, sterilized knife to create primary injuries, facilitating pathogen entry into the plant vascular system. The wounded seedlings were then dipped in a bacterial suspension (10^7 CFU/mL) for one to two minutes before being transplanted into the sick plot [10].

e. Bacterial wilt scoring

Plants were monitored for bacterial wilt symptoms at three-day intervals, and the percentage disease incidence (PDI) was recorded. To distinguish bacterial wilt from other fungal wilts, individual plant wilting was confirmed using the ooze test. Final disease scoring was conducted 35 days post-inoculation (Fig. 1C and D), and PDI was calculated accordingly [11].

$$\text{Bacterial wilt PDI (\%)} = \frac{\text{Number of dead plants due to bacterial wilt disease}}{\text{Total number of plants transplanted}} \times 100$$

The individual recombinant inbred line was categorized from 0-5 scale [12].

f. Bacterial wilt scoring

Sl. No.	Percent Disease incidence (%)	Score	Disease reaction
1	0	0	Highly resistant (HR)
2	0.01-20.00%	1	Resistant (R)
3	21.00-40.00%	2	Moderately resistant (MR)
4	41.00-60.00%	3	Moderately susceptible (MS)
5	61.00-80.00%	4	Susceptible (S)
6	More than 80%	5	Highly susceptible (HS)

The area under the disease progress curve (AUDPC) was calculated based on the survival percentage recorded at 20, 23, 26, 29, 32, 35, and 38 days post-inoculation (DPI) [13], with observations taken at three-day intervals.

$$AUDPC = \sum_{I=1}^{n-1} [\{\frac{Xi+Xi+1}{2}\} \times \{ti + 1 - ti\}]$$

Where, n: total number of observations; Xi: Survival percentage at i^{th} observation; X_i+1 = survival percentage at $i + 1^{st}$ observation; $t_i+1 - t_i$: number of days between subsequent observations.

RESULTS

a. Disease incidence and progression of bacterial wilt (area under the disease progress curve or AUDPC)

The parental lines (B-HP-143 and B-HP-144), along with the F_2 and F_5 generations, exhibited bacterial wilt symptoms 20 days after planting. In the F_2 and F_5 generations, disease progression continued until day 38. The progression of bacterial wilt in the F_2 generation is illustrated in Fig. 2.

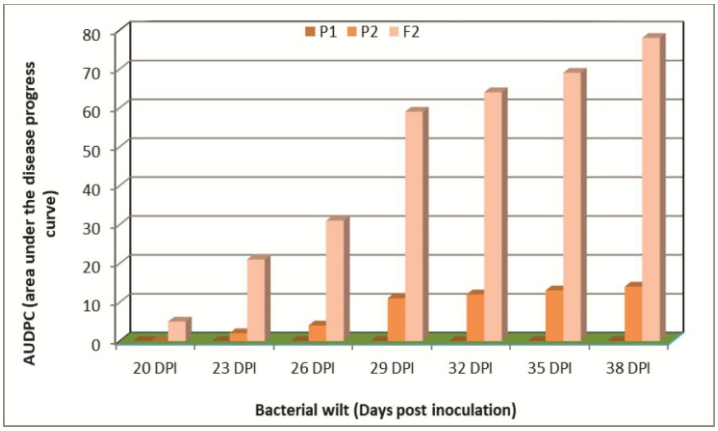

Fig. 2: Area under disease progression curve (AUDPC) of bacterial wilt (parents and F_2 population)

The AUDPC values varied significantly across generations, with notable differences between highly resistant and moderately susceptible lines (F_5 generation, Table 2).

In this study, the highly resistant parent B-HP-143 recorded an AUDPC value of 0, whereas the susceptible parent B-HP-144 exhibited a value of 735. Additionally, significant variation in AUDPC values was observed among different RILs (Table 2).

b. Inheritance Pattern of bacterial wilt disease resistance

The disease response of the F_2 population to bacterial wilt is summarized in Table 1. The resistant parent, B-HP-143, exhibited 100% survivability, whereas the susceptible parent, B-HP-144, showed a survival rate of 30%. The F_2 population had an average survival percentage of 50.3%. This study confirmed that B-HP-143 is highly resistant, while B-HP-144 is susceptible to bacterial wilt. To determine the inheritance pattern of resistance, a chi-square test was performed to assess the goodness of fit. Phenotypic evaluation of the F_2 population revealed that 78 out of 157 plants were susceptible, while 79 survived. Initially, a monogenic inheritance model was tested, but the calculated F-value exceeded the tabulated F-value, leading to the rejection of this hypothesis. Subsequently, a digenic inheritance model with epistatic interaction in a 9:7 ratio was examined, but it was also rejected due to a high F-calculated value. Based on these results, it was concluded that bacterial wilt resistance follows a polygenic inheritance pattern.

Table 1. Chi square test for inheritance pattern of bacterial wilt resistance in chilli: for monogenic inheritance

Sl. No.	Class	Observed Value (O)	Expected Value (E)	(O-E)	$(O-E)^2$	$(O-E)^2/E$
1	Resistant	79	117.75	-38.75	1501.56	12.75
2	Susceptible	78	39.25	38.75	1501.56	38.26
						$\chi^2 = 51.01$

*Table value of chi square at d.f. 1 = 3.81. No other ratio fits on the inheritance pattern of bacterial wilt in chilli. So, it is concluded that the trait may be governed by polygenes.

c. Screening of recombinant inbred lines for resistance against bacterial wilt

A total of 100 recombinant inbred lines (RILs) and two parental lines were evaluated for resistance against bacterial wilt. The disease response of these planting materials to Ralstonia solanacearum is summarized in Table 2. The results indicated a range of disease reactions, from highly resistant, resistant, and moderately resistant to moderately susceptible. Notably, none of the RILs were classified as susceptible or highly susceptible. Among the different RILs, RIL numbers 84, 101, 106, 149, 155, 196, 210, 220, 232, 242, 283, 301, 307, 315, 324, 333, 336, 340, and 342, along with the resistant parent B-HP-143, exhibited high resistance (HR) with 0.0% bacterial wilt infection (Table 2). In contrast, the susceptible parent B-HP-144 showed a 70% infection rate. Additionally, out of the 100 RILs and two parents, RIL numbers 40, 65, 118, 120, 125, 162, 241, 275, 305, and 310 were classified as moderately susceptible, with the highest observed bacterial wilt infection rate of 50.0% (Table 2).

Table 2: Screening of chilli RILs and parents for bacterial wiltresistance

S.L No.	RIL Numbers and parents	Bacterial wilt incidence (%)*	Disease reaction	AUDPC
1	10	20.0 (28.7)	Resistant (R)	75
2	12	20.0 (28.7)	Resistant (R)	75
3	15	30.0 (28.7)	Moderately Resistant (MR)	255
4	16	10.0 (28.7)	Resistant (R)	15
5	23	30.0 (28.7)	Moderately Resistant (MR)	210
6	29	30.0 (39.0)	Moderately Resistant (MR)	165
7	39	30.0 (39.0)	Moderately Resistant (MR)	135
8	40	50.0 (39.0)	Moderately Susceptible (MS)	435
9	41	30.0 (28.7)	Moderately Resistant (MR)	240
10	42	10.0 (0.0)	Resistant (R)	15
11	44	10.0 (0.0)	Resistant (R)	45
12	45	10.0 (28.7)	Resistant (R)	45
13	46	30.0 (28.7)	Moderately Resistant (MR)	225
14	52	10.0 (28.7)	Resistant (R)	15
15	59	10.0 (0.0)	Resistant (R)	45
16	65	50.0 (48.1)	Moderately Susceptible (MS)	360
17	66	30.0 (28.7)	Moderately Resistant (MR)	255
18	84	0.0 (0.0)	Highly Resistant (HR)	0
19	91	20.0 (39.0)	Resistant (R)	105
20	98	30.0 (39.0)	Moderately Resistant (MR)	195
21	99	30.0 (39.0)	Moderately Resistant (MR)	150
22	100	10.0 (0.0)	Resistant (R)	15
23	101	0.0 (0.0)	Highly Resistant (HR)	0
24	103	30.0 (39.0)	Moderately Resistant (MR)	300
25	104	10.0 (28.7)	Resistant (R)	30
26	105	10.0 (28.7)	Resistant (R)	45
27	106	0.0 (0.0)	Highly Resistant (HR)	0
28	110	20.0 (28.7)	Resistant (R)	75
29	118	50.0 (39.0)	Moderately Susceptible (MS)	300
30	119	10.0 (0.0)	Resistant (R)	15
31	120	50.0 (48.1)	Moderately Susceptible (MS)	510
32	121	10.0 (28.7)	Resistant (R)	45
33	123	30.0 (39.0)	Moderately Resistant (MR)	165
34	125	50.0 (48.1)	Moderately Susceptible (MS)	510
35	134	30.0 (39.0)	Moderately Resistant (MR)	135
36	149	0.0 (0.0)	Highly Resistant (HR)	0
37	150	10.0 (28.7)	Resistant (R)	15
38	153	10.0 (28.7)	Resistant (R)	45
39	154	30.0 (39.0)	Moderately Resistant (MR)	315
40	155	0.0 (0.0)	Highly Resistant (HR)	0
41	158	10.0 (28.7)	Resistant (R)	45
42	162	50.0 (48.1)	Moderately Susceptible (MS)	420
43	163	30.0 (39.0)	Moderately Resistant (MR)	195
44	173	30.0 (28.7)	Moderately Resistant (MR)	150
45	174	20.0 (28.7)	Resistant (R)	90
46	185	20.0 (28.7)	Resistant (R)	75

S.L No.	RIL Numbers and parents	Bacterial wilt incidence (%)*	Disease reaction	AUDPC
47	196	0.0 (0.0)	Highly Resistant (HR)	0
48	200	10.0 (0.0)	Resistant (R)	15
49	202	10.0 (28.7)	Resistant (R)	45
50	209	10.0 (0.0)	Resistant (R)	45
51	210	0.0 (0.0)	Highly Resistant (HR)	0
52	220	0.0 (0.0)	Highly Resistant (HR)	0
53	230	30.0 (39.0)	Moderately Resistant (MR)	165
54	232	0.0 (0.0)	Highly Resistant (HR)	0
55	233	30.0 (28.7)	Moderately Resistant (MR)	105
56	241	50.0 (39.0)	Moderately Susceptible (MS)	300
57	242	0.0 (0.0)	Highly Resistant (HR)	0
58	243	10.0 (28.7)	Resistant (R)	15
59	247	10.0 (0.0)	Resistant (R)	45
60	252	30.0 (39.0)	Moderately Resistant (MR)	195

61	253	10.0 (0.0)	Resistant (R)	15
62	255	30.0 (39.0)	Moderately Resistant (MR)	135
63	259	20.0 (28.7)	Resistant (R)	75
64	268	10.0 (28.7)	Resistant (R)	15
65	271	30.0 (28.7)	Moderately Resistant (MR)	225
66	275	50.0 (48.1)	Moderately Susceptible (MS)	375
67	276	10.0 (0.0)	Resistant (R)	75
68	283	0.0 (0.0)	Highly Resistant (HR)	0
69	284	10.0 (28.7)	Resistant (R)	15
70	285	20.0 (39.0)	Resistant (R)	90
71	287	20.0 (28.7)	Resistant (R)	90
72	288	20.0 (28.7)	Resistant (R)	75
73	293	20.0 (39.0)	Resistant (R)	75
74	297	10.0 (28.7)	Resistant (R)	45
75	301	0.0 (0.0)	Highly Resistant (HR)	0
76	303	10.0 (28.7)	Resistant (R)	15
77	304	20.0 (0.0)	Resistant (R)	60
78	305	50.0 (48.1)	Moderately Susceptible (MS)	300
79	306	30.0 (28.7)	Moderately Resistant (MR)	225
80	307	0.0 (0.0)	Highly Resistant (HR)	0
81	308	10.0 (28.7)	Resistant (R)	15
82	310	30.0 (28.7)	Moderately Resistant (MR)	135
83	313	10.0 (0.0)	Resistant (R)	15
84	314	10.0 (0.0)	Resistant (R)	15
85	315	0.0 (0.0)	Highly Resistant (HR)	0
86	316	10.0 (0.0)	Resistant (R)	15
87	318	30.0 (39.0)	Moderately Resistant (MR)	135
88	319	30.0 (28.7)	Moderately Resistant (MR)	135
89	324	0.0 (0.0)	Highly Resistant (HR)	0
90	328	10.0 (0.0)	Resistant (R)	45
91	333	0.0 (0.0)	Highly Resistant (HR)	0
92	335	10.0 (28.7)	Resistant (R)	15
93	336	0.0 (0.0)	Highly Resistant (HR)	0
94	340	0.0 (0.0)	Highly Resistant (HR)	0
95	342	0.0 (0.0)	Highly Resistant (HR)	0
96	344	30.0 (28.7)	Moderately Resistant (MR)	360
97	347	40.0 (28.7)	Moderately Resistant (MR)	435
98	348	10.0 (28.7)	Resistant (R)	15
99	349	10.0 (28.7)	Resistant (R)	45
100	355	10.0 (28.7)	Resistant (R)	15
101	B-HP-143	0.0 (0.0)	Highly Resistant (HR)	0
102	B-HP-144	70.0 (52.8)	Susceptible (S)	735
	S. E m. (±)	1.8		
	CD (p≤ 0.05)	5.2		
	CV (%)	12.1		

**value represents angular value for bacterial wilt incidence*

DISCUSSION

a. Genetic analysis of F_2 and AUDPC of F_2 and F_5 for bacterial wilt resistance

The inheritance pattern of bacterial wilt resistance is influenced by multiple factors, including environmental conditions, pathogen race, strain, and biovar. Understanding inheritance patterns for specific diseases is crucial for crop improvement programs. Analysis of the F_2 segregation pattern confirmed that resistance to bacterial wilt in the studied breeding material follows a polygenic inheritance model, as both monogenic and digenic inheritance hypotheses were rejected. These findings align with previous reports [14, 15].

Disease development and severity were assessed using AUDPC values [16], which provide insights into genotype-specific resistance expression over time and across locations. Since AUDPC considers multiple assessments and does not rely on data transformations, it offers a more precise phenotypic evaluation. Moreover, it is simple to calculate [13] and effectively tracks disease progression throughout the infection period [17]. A higher AUDPC value indicates greater disease susceptibility, whereas a lower value signifies stronger resistance [18]. In this study, the AUDPC value for the F_2 population was 545.54, while the resistant parent B-HP-143 recorded 0.00, and the susceptible parent B-HP-144 exhibited 735. These results confirm that B-HP-143 serves as a strong resistance source. In the F_5 generation, AUDPC values varied among different RILs, reflecting differences in disease progression. Notably, RIL numbers 120 and 125 recorded an AUDPC of 510.0, indicating high disease spread during the wilting period. In contrast, RIL numbers 84, 101, 106, 149, 155, 196, 210, 220, 232, 242, 283, 301, 307, 315, 324, 333, 336, 340, and 342 exhibited an AUDPC of 0.0, demonstrating complete resistance. These findings are consistent with earlier reports [16].

b. Screening of recombinant inbred lines (F_5 population) for resistance against bacterial wilt

Among the different RILs, RIL numbers 84, 101, 106, 149, 155, 196, 210, 220, 232, 242, 283, 301, 307, 315, 324, 333, 336, 340, and 342, along with the resistant parent B-HP-143, exhibited high resistance to bacterial wilt, with an infection rate of 0.0%. In contrast, the susceptible parent B-HP-144 recorded a 70% infection rate. RIL numbers 40, 65, 118, 120, 125, 162, 241, 275, 305, and 310 were classified as moderately susceptible (MS), with the highest bacterial wilt infection rate reaching 50.0%. The results from this experiment revealed a spectrum of disease reactions ranging from highly resistant, resistant, and moderately resistant to moderately susceptible within the experimental population (Table 2). Notably, no RILs exhibited susceptible or highly susceptible reactions. Variation in disease response among different RILs and parental lines suggests the involvement of multiple genes and gene interactions in resistance. Additionally, environmental factors may have influenced bacterial wilt infection levels in the plants.

Conclusion

This study revealed that bacterial wilt resistance follows a polygenic inheritance pattern. Disease progression and severity, measured as the AUDPC, were recorded at 545.54 for the F_2 population, while parent lines B-HP-143 and B-HP-144 showed AUDPC values of 0.00 and 735, respectively. These findings confirm that B-HP-143 serves as a strong source of bacterial wilt resistance. Furthermore, RILs 84, 101, 106, 149, 155, 196, 210, 220, 232, 242, 283, 301, 307, 315, 324, 333, 336, 340, and 342 exhibited an AUDPC value of 0.00, demonstrating high resistance with no bacterial wilt infection. These highly resistant and resistant lines hold significant potential for biotechnological advancements, serving as valuable genetic resources for mapping populations, rootstock breeding, and the development of bacterial wilt-resistant varieties through targeted breeding and crop improvement strategies.

Future scope of the study

The use of bacterial wilt-resistant RILs has the potential to significantly enhance various breeding strategies aimed at improving chili crop quality. The inheritance analysis conducted in this study provides a crucial foundation for effectively incorporating bacterial wilt resistance into elite commercial chili genotypes. This incorporation is essential for strengthening future efforts to improve quality, boost productivity, and develop wilt-resistant chili varieties.

Conflict of interest- Not applicable

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