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Screening of Rice Accessions Resistant to Blast in Benin

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Abstract: The objective of this screening is to identify genotypes with effective resistance genes against leaf blast. Two hundred rice accessions were collected in Benin's production areas and screened in upland ecology at Sowe (Glazoue, Benin). The experimental device used is an Alpha lattice 20 x 10 with 3 repetitions. The hierarchical ascending classification (HAC) allowed obtaining three large groups of accessions according to their behavior in relation to the populations of *Magnaporthe oryzae* present on the site: the C1 group composed of resistant genotypes (R), the group C2 composed of genotypes moderately sensitive (MS) and finally the group C3 that consists of sensitive genotypes (S). A highly significant difference was observed between genotypes based on recorded severity scores (five rating stages of disease and the AUDPC or Area Under the Disease Progress Curve). In general, the difference between the phenotypic variable (VP) and the genotypic variable (Vg) is relatively weak for all characters. All the traits studied had recorded high genotypic variation coefficients (GVC) and phenotypic variation coefficients (PVC) (> 20), with the exception of the first level of disease severity (Pyri1), thus justifying the high variability between genotypes with respect to resistance to disease. Heritability rate (H^2%) coupled with high values of expected genetic gain compared to the mean (EGGM), indicated a low environmental influence in disease expression and a prevalence of the effect of additive genes in genetic determinism. The experiment has identified a pool of varieties with good behavior against blast disease that can be used as genetic control material in research and breeding programs in Benin.

Keywords: *Oryza Spp*, Rice Blast, Genetic Control, *Magnaporthe Oryzae*

1. Introduction

In Benin, rice is one of the principal crops selected in the Strategic Plan for Agricultural Development because of its economic and food importance [1]. It is currently the second cereal in terms of consumption after maize [2]. Rice is subject to ever-increasing demand and its production at the national level has increased from 124 975 tons in 2010 to 206 943 tons in 2013 with respectively cultivated areas increasing from 47 058 ha to 68 259 ha. This evolution of rice production does not keep pace with local consumption, thus leading to the non-satisfaction of food needs.

Although domestic production has almost doubled in the past decade after the efforts of the government and his partners, rice consumption needs are covered in 2013 for only 47% [1]. Therefore, the objective of the national rice development strategy is to achieve a production level of 385000 tons of white rice per year by the end of 2018 [3]. To achieve this, more efforts are needed particularly to improve

the productivity and competitiveness of this sector in order to meet national rice requirements and limit imports. In addition, Benin has a significant potential in natural resources for rice production resulting in a high concentration of appropriate lands (75%) in the Center and in the north of the country [4]. Nevertheless, important constraints limit the development of the rice sector in Benin. among the major constrains, we can mention biotic (harmful) and abiotic stresses (drought, scarcity of rainfall), isolation of production areas, lack of adequate credit, lack of equipment, lack of specific quality inputs, lack of markets for the disposal of rice and difficulty of farming operations [5, 6].The impact of these different constraints on production is clearly perceptible. [7] reported that diseases are considered to be the most important causes of decreased quantity and quality of products. Among the fungal diseases of rice, blast remains the most important [8]. It causes estimated losses of more than $ 70 billion a year [9, 10] and represents the largest pathological condition with significant economic impact and a serious threat to food security in the world [11, 12, 7]. Every year it is estimated that rice blast destroy food more than enough to eat for 60 million people and 50% of the rice yield is lost in the field by the occurrence of blast [13]. In the West African sub-region, blast disease is recognized as a major constraint to rice production with 3.2 to 77% yield loss [14]. In the tropics, blast is one of the most threats to rice production and is caused by *Magnaporthe oryzae* [15]. The disease symptoms appear on the aerial parts of the plant. Most infections occur on the leaves, causing diamond-shaped lesions with a gray or white center to appear, which can be all of the leaves and causes of death at any stage of growth and on the panicles, which turn white and die before being filled with grain [16].

Among the control methods, genetic improvement is a method of sustainable management of blast. Efforts have been made to develop and identify resistant varieties to improve the production of small farmers with a substantial reduction in the costs of managing this disease. Several studies evaluated the genetic diversity of rice and demonstrated the resistance of some species of *Oryza sativa* and *Oryza glaberrima* to blast disease [17]. Indeed, [18] evaluated the resistance to blast disease of a few rice varieties and found that about 72% of the varieties tested were susceptible to all strains causing the disease. There are some rice varieties with at least one specific blast resistance gene and some with many blast resistances. However, most of these genes are specific to the kind of the pathogen [19]. There is a need to select the best subject among the local varieties with good resistance potential and stable for each type of rice crop. Thus, the objective of this study was to identify, among the accessions of rice collected in Benin, stable sources of resistance to blast that can be promote among breeders for the development of efficient genotypes.

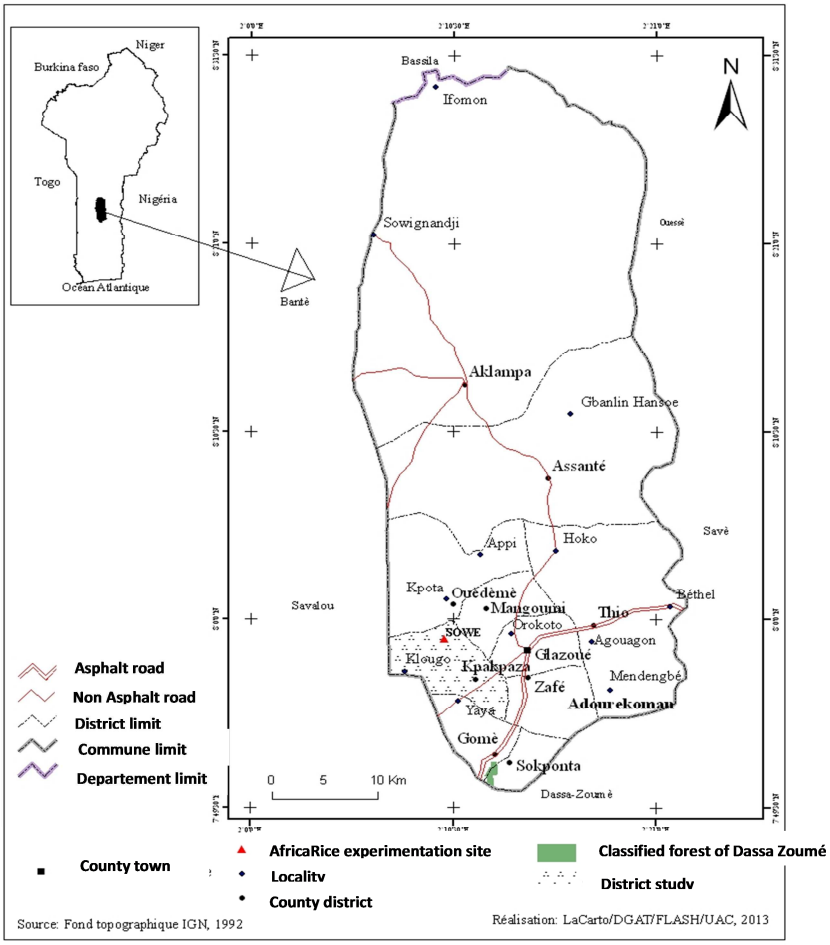


Figure 1. *Location of the experimental site that shelted the tests.*

2. Material and Methods

2.1. Study Area

The study was carried out in 2014 and in 2015 at the bottom of the slope on the experimental site of the Rice Center for Africa. The site is located in Sowe (longitude 7°58'5"N, latitude 2°10'47"E and altitude 160 m located in Glazoue in the Republic of Benin (Figure 1). The average rainfall during the experimental period is 128.41 mm (between June to November) with maximum precipitation recorded during the months of August and September and the minimum during the month of November.

2.2. Plant Material

A total of 200 *Oryza sp* genotypes including 5 controls were used in this study (Table 1). The five controls were composed of two sample resistant to blast disease (Moroberekan and Tetep), and three susceptible to blast disease (Marateli, CO39 and IR BLTA2-Pi).

Table 1. *List of 200 accessions screened for resistance to blast.*

N	Code	Designation	N	Code	Designation
1	V1	BEN 11-136-A-1	102	V102	BEN 11-34
2	V2	BEN 11-104-A	103	V103	BEN 11-34-1
3	V3	BEN 11-104-A-1	104	V104	BEN 11-35-A-1
4	V4	BEN 11-104-B	105	V105	BEN 11-35-A-2
5	V5	BEN 11-105-A-1	106	V106	BEN 11-35-A-3
6	V6	BEN 11-105-B	107	V107	BEN 11-36-A
7	V7	BEN 11-105-C	108	V108	BEN 11-37-A
8	V8	BEN 11-106-A	109	V109	BEN 11-37-B
9	V9	BEN 11-107-B	110	V110	BEN 11-39
10	V10	BEN 11-107-C	111	V111	BEN 11-3-A
11	V11	BEN 11-107-D	112	V112	BEN 11-40-A
12	V12	BEN 11-107-E	113	V113	BEN 11-40-B
13	V13	BEN 11-108-B	114	V114	BEN 11-41-B
14	V14	BEN 11-111-A	115	V115	BEN 11-41-C
15	V15	BEN 11-111-B	116	V116	BEN 11-41-D
16	V16	BEN 11-112-A	117	V117	BEN 11-42-A
17	V17	BEN 11-112-B	118	V118	BEN 11-42-B
18	V18	BEN 11-112-B-1	119	V119	BEN 11-43-A
19	V19	BEN 11-116-B	120	V120	BEN 11-43-B-1
20	V20	BEN 11-119-B	121	V121	BEN 11-43-D
21	V21	BEN 11-12	122	V122	BEN 11-44-A
22	V22	BEN 11-120-B	123	V123	BEN 11-44-C
23	V23	BEN 11-120-C-1	124	V124	BEN 11-45
24	V24	BEN 11-121-A	125	V125	BEN 11-46
25	V25	BEN 11-121-B	126	V126	BEN 11-49
26	V26	BEN 11-121-C	127	V127	BEN 11-4-A
27	V27	BEN 11-122-A	128	V128	BEN 11-50-A
28	V28	BEN 11-122-B	129	V129	BEN 11-50-B
29	V29	BEN 11-123-A	130	V130	BEN 11-50-C
30	V30	BEN 11-123-A-1	131	V131	BEN 11-51-A
31	V31	BEN 11-126-A	132	V132	BEN 11-52
32	V32	BEN 11-126-B	133	V133	BEN 11-53
33	V33	BEN 11-13	134	V134	BEN 11-54
34	V34	BEN 11-131-B	135	V135	BEN 11-55
35	V35	BEN 11-131-C	136	V136	BEN 11-56
36	V36	BEN 11-134	137	V137	BEN 11-57-A
37	V37	BEN 11-135	138	V138	BEN 11-58
38	V38	BEN 11-136-A	139	V139	BEN 11-59-A
39	V39	BEN 11-136-B	140	V140	BEN 11-59-B
40	V40	BEN 11-137-A	141	V141	BEN 11-59-C
41	V41	BEN 11-137-B	142	V142	BEN 11-59-C-1
42	V42	BEN 11-137-C	143	V143	BEN 11-59-D
43	V43	BEN 11-138	144	V144	BEN 11-5-A
44	V44	BEN 11-139-B	145	V145	BEN 11-5-B
45	V45	BEN 11-14	146	V146	BEN 11-60-A
46	V46	BEN 11-142-A	147	V147	BEN 11-60-B
47	V47	BEN 11-146	148	V148	BEN 11-62-A1
48	V48	BEN 11-148	149	V149	BEN 11-62-A2
49	V49	BEN 11-151-B	150	V150	BEN 11-62-B

N	Code	Designation	N	Code	Designation
50	V50	BEN 11-152-B	151	V151	BEN 11-62-B1
51	V51	BEN 11-152-C	152	V152	BEN 11-62-B2
52	V52	BEN 11-154-B	153	V153	BEN 11-62-C1
53	V53	BEN 11-155-B	154	V154	BEN 11-62-D1
54	V54	BEN 11-157-B	155	V155	BEN 11-64
55	V55	BEN 11-158-A	156	V156	BEN 11-68-A
56	V56	BEN 11-159-A	157	V157	BEN 11-69-A
57	V57	BEN 11-159-B	158	V158	BEN 11-69-B
58	V58	BEN 11-16	159	V159	BEN 11-69-C
59	V59	BEN 11-160-A	160	V160	BEN 11-6-A
60	V60	BEN 11-161	161	V161	BEN 11-6-B
61	V61	BEN 11-162-B	162	V162	BEN 11-70-B
62	V62	BEN 11-163-A	163	V163	BEN 11-71-A
63	V63	BEN 11-169-B	164	V164	BEN 11-71-B
64	V64	BEN 11-171	165	V165	BEN 11-71-C
65	V65	BEN 11-172	166	V166	BEN 11-72-A
66	V66	BEN 11-175	167	V167	BEN 11-73-B
67	V67	BEN 11-176-A	168	V168	BEN 11-75-A
68	V68	BEN 11-177-A	169	V169	BEN 11-75-B
69	V69	BEN 11-177-B	170	V170	BEN 11-76-A
70	V70	BEN 11-177-C	171	V171	BEN 11-76-B
71	V71	BEN 11-178-B	172	V172	BEN 11-78-A
72	V72	BEN 11-17-B	173	V173	BEN 11-79
73	V73	BEN 11-181-B	174	V174	BEN 11-7-A
74	V74	BEN 11-182-B	175	V175	BEN 11-7-B
75	V75	BEN 11-183	176	V176	BEN 11-7-C
76	V76	BEN 11-185-B	177	V177	BEN 11-80
77	V77	BEN 11-186	178	V178	BEN 11-80-1
78	V78	BEN 11-190-B	179	V179	BEN 11-82-A
79	V79	BEN 11-190-C	180	V180	BEN 11-82-B
80	V80	BEN 11-2	181	V181	BEN 11-83-A
81	V81	BEN 11-200-A	182	V182	BEN 11-83-B
82	V82	BEN 11-200-B	183	V183	BEN 11-84-A
83	V83	BEN 11-201-B	184	V184	BEN 11-86
84	V84	BEN 11-202-A	185	V185	BEN 11-88-A
85	V85	BEN 11-203-A	186	V186	BEN 11-89-B
86	V86	BEN 11-203-B	187	V187	BEN 11-9
87	V87	BEN 11-203-C	188	V188	BEN 11-90-A
88	V88	BEN 11-204	189	V189	BEN 11-90-B
89	V89	BEN 11-21	190	V190	BEN 11-93-A
90	V90	BEN 11-26-A	191	V191	BEN 11-98-A
91	V91	BEN 11-27-B	192	V192	WAB 32-81
92	V92	BEN 11-29-A	193	V193	WAB 56-104
93	V93	BEN 11-31-B	194	V194	WAB 32-83
94	V94	BEN 11-31-C	195	V195	WAB 638-1
95	V95	BEN 11-32-A	196	V196	IR 64
96	V96	BEN 11-32-B	197	V197	INARIS 88
97	V97	BEN 11-32-C	198	V198	OROU KPEHINIE
98	V98	BEN 11-32-E	199	V199	Moroberekan *
99	V99	BEN 11-32-G	200	V200	Tetep*
100	V100	BEN 11-33-B	201	V201	MARATELI **
101	V101	BEN 11-33-C	202	V202	CO39 **
102	V102	BEN 11-34	203	V203	IR BLTA2-Pi **

* Resistance control.
** Sensibility control.

2.3. Experimental Device and Conduct of the Test

Screening of collected rice accessions was carried out during the period from July to November of 2014 and 2015, corresponding to the long rainy season in the study area. An alpha lattice device has been used with two infesting bands disposed on either side of the elementary plots and perpendicular thereto. This device consisted of incomplete randomized blocks with 200 samples (195 accessions, 3 susceptible and 2 resistant varieties). The elementary plots consisted of three rows of 0.50 m long with 10 cm spacing. Each elementary plot had an area of

0.1 m². The distance between two parcels was 20 cm. The distance between blocks was 1 m and the repetitions 2 m. Each infective band had three lines (4.20 m long and 10 cm spacing) of three susceptible varieties (Maratelli, IRBLTA2-PI, and CO39).

The infestation bands were fertilized at 200 kg/ha of urea at planting to weaken host-parasite relationships. At the 21st and 42nd days after sowing, urea spraying was done at a rate of 100 kg/ha.

For the varieties to be tested, base manure consisting of 200 kg/ ha of N.P.K was applied on the elementary plots at sowing. Nitrogen doses of 200 kg/ ha were applied on the 21st and 42nd day after sowing

2.4. Data Collection on the Outbreak of Blast Disease

The data collected included leaf blast on 12 of the 18 plants in the basic plot. The first scoring was done as soon as the first lesions appeared on the varieties under evaluation and then observations at regular intervals of 7 days until the maximum epidemic level was reached. Scale 1 to 9 of IRRI was used with level 1 corresponding to the absence of symptoms and level 9 equivalent to plants completely attacked and stunted or dead [20].

2.5. Statistical Analysis of the Data

The data was entered and coded in Excel 2003 and the variance analyzes were performed with the XLSTAT-pro 7.5 software. The Student Newman-Keuls (SNK) test compared and ranked the average of the severity scores of varieties at the 5% probability level. Hierarchical ascending classification (HAC) was constructed with ARIS software to determine sensitivity groups or pathotypes. Regarding the calculation of leaf indices, the behavior of varieties was evaluated against the most aggressive race on the site, using the Area Under the Disease Progress Curve (AUDPC). This index represents the cumulative incidence of foliar disease during the observation period. The AUDPC was calculated according to the formula in reference [21]

$$\mathbf{AUDPC} = \sum [(Di+Di+1)/2] (ti+1 - ti).$$

where Di and Di + 1 represent measures of the severity of the disease in percent observed respectively at times ti and ti + 1 with (ti + 1 - ti) representing the time interval between two observations. Variety resistance is meant when the AUDPC has a low value.

For the estimation of the components of the variance, it was obtained by the method of analysis of restricted maximum likelihood (REML) using the software Genstat Edition 15. Through this analysis, the components of the genotypic variance (Vg) and residual variance (Vr) for each variable were obtained. Variance component values were used to estimate phenotypic variance (Vp); genotypic variance coefficient (GVC) and phenotypic variance coefficient (PVC), broad-sense heritability (H^2) and expected genetic gain compared to the mean (EGGM). The estimation of these genetic parameters was made according to the formulas below:

1. Phenotypic variance

$$Vp = Vg + \frac{Vr}{r}$$

Where Vp = phenotypic variance; Vg = genotypic variance; Vr = residual variance and r = number of repetitions. The values of the genotype and residual variances are obtained through the results of the analysis.

2. Genotypic Variance Coefficient (GVC) of and Phenotypic Variance Coefficient (PVC)

$$GCV = \frac{\sqrt{Vg}}{M} 100$$

$$PCV = \frac{\sqrt{Vp}}{M} 100$$

Where GVC = Genotypic Variance Coefficient; PCV = Phenotypic Variance Coefficient; $\sqrt{Vg}$= genotypic standard deviation; $\sqrt{Vp}$ = phenotypic standard deviation and M = overall average of the variable.

3. Heritability H^2.

$$H^2 = \frac{Vg}{Vp} 100$$

Where H^2 = heritability in the broad sense; Vg = genotypic variance; Vp = phenotypic variance. According to Robinson *et al*. (1966), the heritability rate is considered high above 60%, low below 30% and moderate between 30 and 60%.

4. Expected genetic gain (EGG) and expected genetic gain compared to the mean (EGGM)

The expected genetic gain (EGG) and the expected genetic gain compared to the mean (EGGM) were calculated according to the formulas below.

$$GA = H^2 \, i \, \sqrt{Vp}$$

$$GAM = \frac{GA}{M} 100$$

Where H^2 = heritability in the broad sense; i = constant = 2.06; for the selection intensity k = 5%; M = average of the variable.

3. Results

3.1. Analysis of Variance and Ascending Hierarchical Classification

The results of the study shows a diversity of reaction of accessions collected in the different regions of Benin facing the blast. The severity scores for blast fever in the test varied significantly (P <0.0001) between genotypes regardless of the dates of the evaluations (Table 2).

Table 2. *ANOVA of all traits studied.*

Variable	Minimum	Mean	Maximum	F>P
Pyri1	1	1.008 ± 0.04	2	2.194 E-27***
Pyri2	1	1.258 ± 0.15	3	2.224 E-52***
Pyri3	1	1.53 ± 0.25	4	3.338 E-62***
Pyri4	1	1.948 ± 0.42	7	8.228 E-67***
Pyri5	1	2.255 ± 0.46	7	2.902 E-81***
AUDPC	31.5	92.24 ± 32.08	588	6.139 E-62***

*** Highly significative difference at 5 % (P < 0.0001).

The ascending hierarchical classification (AHC) (Figure 2), shows a structure of the genotypes evaluated in three (03) large groups of sensitivity towards the populations of *M. oryzae* present on the sites at the truncation to 0.8 (Figure 2). The C1 group of resistant genotypes (R) is composed of genotypes with severity index between zero and four (0≤index of severity <4) including resistant control genotypes (Moroberekan and Tetep). The C2 group of moderately susceptible genotypes (MS) with severity index between four and six (4≤ indice of severity <6) and the group C3 of susceptible genotypes (S) (6≤index of severity≤9) including control varieties Co39 and Maratelli very susceptible to blast (Table 3). The structuring of the genotypes evaluated allowed to have three distinct groups of susceptibility towards the populations of *M. oryzae*. The C1 group of resistant genotypes (R) is composed of 77% of the genotypes. The C2 group of moderately susceptible genotypes (MS) consists of 9.5% of the genotypes tested against 13.5% for the sensitive genotypes (Figure 3).

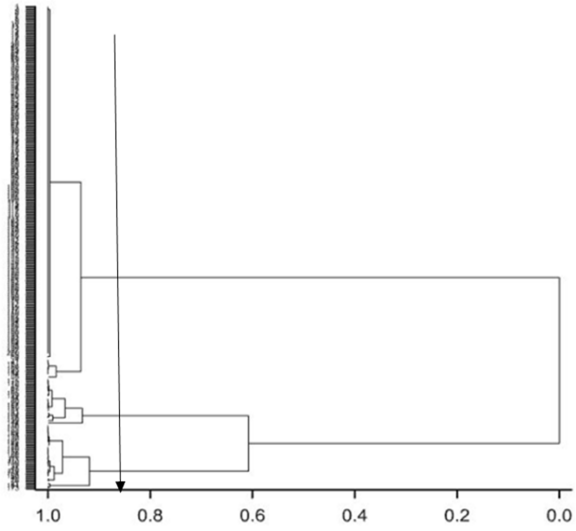


Figure 2. *Dendrogram showing the structuration of genotypes on the basis of their susceptibility to blast.*

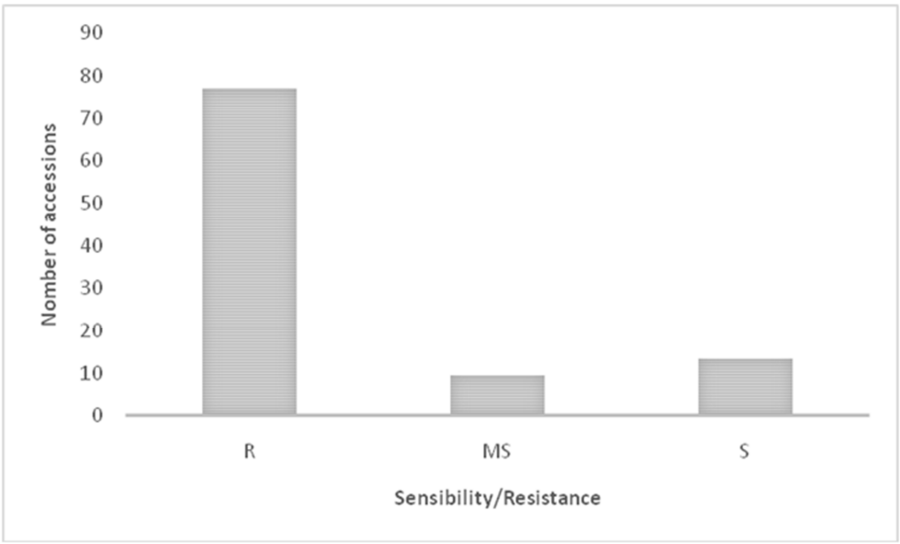


Figure 3. *Proportion of genotypes according to their belonging to a resistance/ sensitivity category.*

Table 3. *Classification of rice genotypes in sensibility groups de in front of foliar attack caused by M. orysea.*

Group of de variety	Classified number of rice variety
Group 1:Resistants genotypes(incidence inferior to 4 % SFM)	1; 3; 5; 6; 7; 8; 9; 10; 11; 12; 13; 14; 15; 17; 18; 19; 20; 22; 23 24; 27; 28; 29; 31; 33; 35; 36; 37; 38; 39; 40; 42; 43; 44; 46; 47; 48; 50; 51; 52; 53; 54; 55; 57; 58; 59; 60; 61; 63; 67; 68; 69; 70; 71; 72; 73; 74; 75; 77; 78; 79; 81; 82; 84; 88; 89; 90; 91; 93; 94; 95; 96; 97; 98; 99; 102; 103; 106; 107; 108; 109; 112; 113; 114; 115; 117; 118; 119; 121; 122; 123; 124; 125; 126; 127; 130; 131; 132; 135; 137; 138; 140; 141; 142; 143; 145; 146; 147; 148; 153; 155; 156; 158; 161; 162; 163; 165; 166; 167; 168; 169; 170; 171; 172; 176; 177; 179; 180; 182; 183; 184; 185; 186; 187; 188; 190; 191; 192; 193; 194; 195; 196; 197; 200; 65; 86; 4; 41; 151; 56; 101; 149; 154; 85

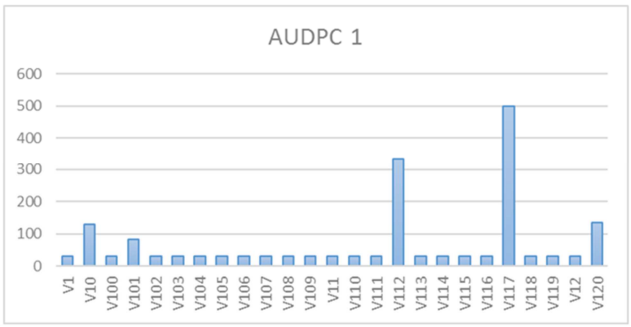
Group of de variety	Classified number of rice variety
Group 2:Resistants genotypes to moderately resistants (from 4 to 15 % SFM)	2; 175; 178; 25; 87; 62; 105; 129; 34; 80; 92; 157; 159; 181; 83; 104; 136; 150; 144
Group 3:Moderately sensitive genotypes at sensitive's (incidence Superior to 15 % SFM)	16; 100; 133; 198; 64; 139; 30; 128; 174; 26; 120; 152; 189; 66; 111; 110; 199; 32; 45; 116; 164; 160; 49; 76; 21; 134; 173

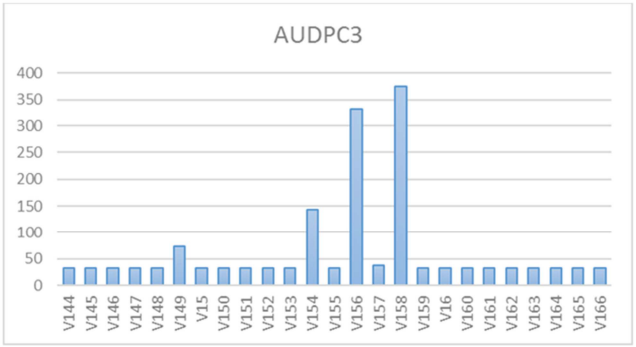
3.2. AUDPC Analysis

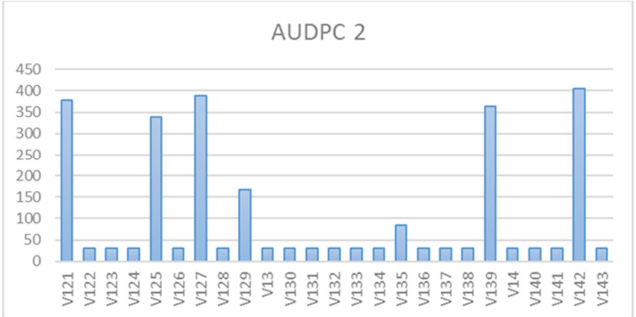
The incidence of the disease is expressed as the Area under the Disease Progress Curve (AUDPC) parameter for each rice genotype (Figure 4). These graphs represent the cumulative AUDPC at different observation dates on the study site. The cumulative AUDPC ranges from 31.5 (V1) to 500 (V117) and is indicative of the overall resistance level of the studied genotypes. The results of the analysis of variance show phenotypic variations based on the resistance to blast disease observed on the genotypes (Table 4). A highly significant effect (P <0.0001) was observed for almost all five disease scoring stages and the AUDPC.

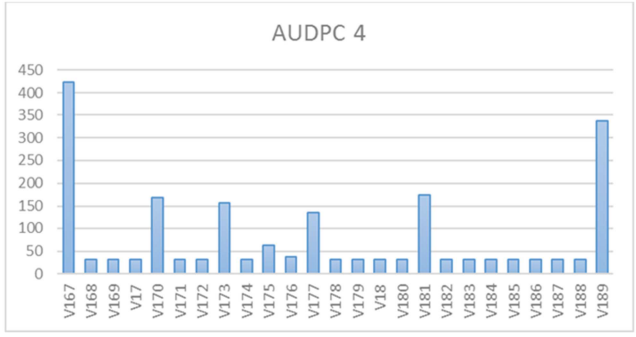
Table 4. *Résultat de l'analyse de variance (ANOVA) des variations phénotypiques observes.*

Treatments	Variables	Ddl	Sum square	Mean square	F value	Pr (>F)
	Repetition	1	0.0025	0.0025	1	1.319
Pyri 1	Genotype	199	2.4775	0.0125	4.980	2.19491E-27
	Residuals	199	0.4975	0.0025		
	Repetition	1	0.4225	0.4225	9.2622	0.00266
Pyri 2	Genotype	199	96.9775	0.4873	10.683	2.22422E-52
	Residuals	199	9.0775	0.0456		
	Repetition	1	0.81	0.81	6.390	0.0122
Pyri 3	Genotype	199	351.64	1.76704	13.9595	3.33851E-62
	Residuals	199	25.19	0.12659		
	Repetition	1	0.3025	0.3025	0.8827	0.3487
Pyri 4	Genotype	199	1075.3975	5.40401	15.7689	8.22892E-67
	Residuals	199	68.1975	0.3427		
	Repetition	1	0.01	0.01	0.02341	0.8785
Pyri 5	Genotype	199	1942.99	9.7638	22.8614	2.90244E-81
	Residuals	199	84.99	0.42709		
	Repetition	1	2166.9025	2166.9025	1.05292	0.3061
AUDPC	Genotype	199	5676804.228	28526.65441	13.8613025	6.13962E-62
	Residuals	199	409543.3475	2058.006771		









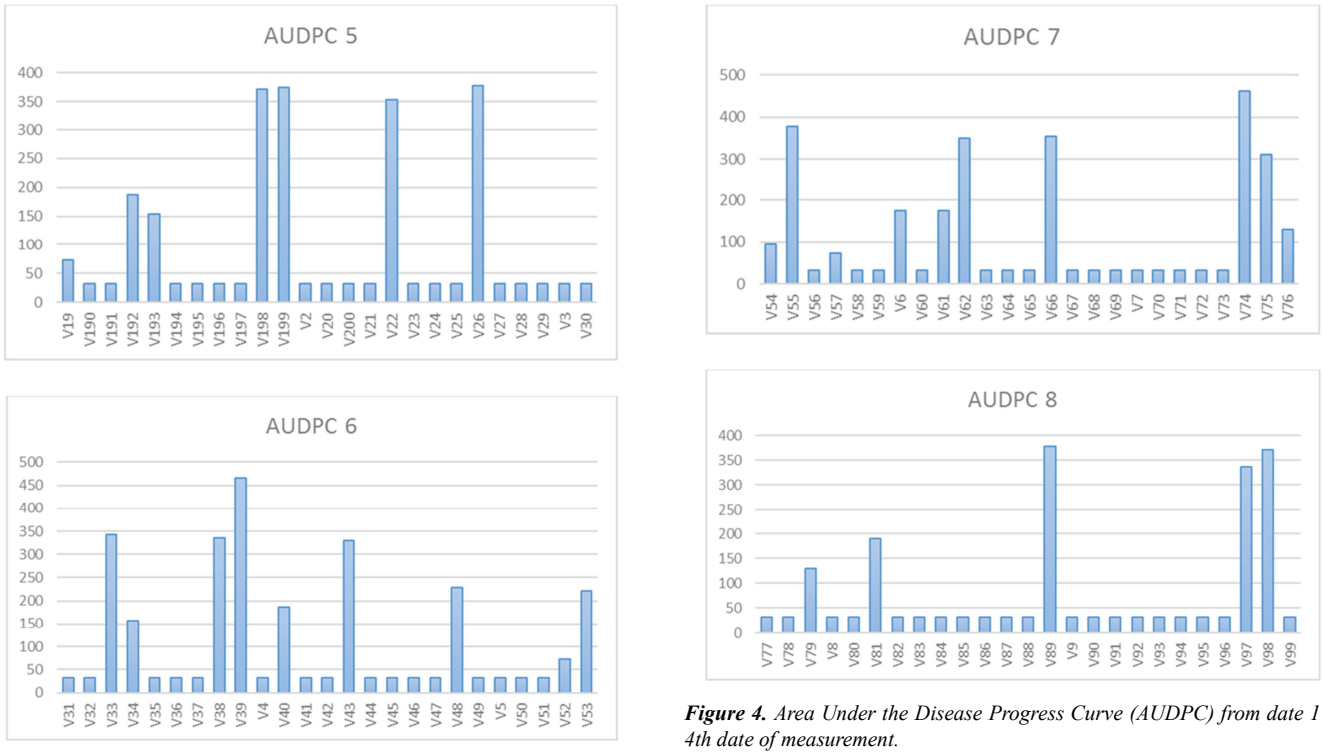


Figure 4. *Area Under the Disease Progress Curve (AUDPC) from date 1 to 4th date of measurement.*

Table 5. *Estimation of genetic measure for blast resistance parameters.*

Variable	Vg	Vp	GVC	PVC	H^2(%)	EGG	EGGM
Pyri1	0.005	0.01	7.00	7.83	79.92	0.13	12.89
Pyri2	0.221	0.24	37.36	39.24	90.64	0.92	73.27
Pyri3	0.820	0.88	59.19	61.44	92.84	1.80	117.49
Pyri4	2.531	2.70	81.67	84.38	93.66	3.17	162.81
Pyri5	4.669	4.88	95.83	97.99	95.63	4.35	193.04
AUDPC	13234.329	14263.33	124.72	129.48	92.79	228.28	247.48

Vg: Genotypic variance; Vp: Phenotypic variance; GVC: Genotypic variance coefficient; PVC: Phenotypic variance coefficient; H^2 %: Heritability; EGG: Expected genetic gain; EGGM: Expected genetic gain mean.

3.3. Estimation of Genetic Variables

The results related to the estimation of genotypic variance (Vg) and phenotypic variance (Vp), genotypic variance coefficient (GVC) and phenotypic variance coefficient (PVC), heritability (H^2%), expected genetic gain (EGG) and the expected genetic gain mean (EGGM) for the five disease and AUDPC scoring stages are generated (Table 5).

Genotypic variance (Vg) and phenotypic variance (Vp): genotypic and phenotypic variances values range from 0.005 to 13234.329 and from 0.01 to 14263.33, respectively, with the observable incidence of blast disease at 14 days after seedling (Pyri1) and leaf area attacked by the disease (AUDPC). Except for the AUDPC, the five disease scoring stages (Pyri1, Pyri2, Pyri3, Pyri4 and Pyri5) showed low genotypic (Vg) and phenotypic variance (Vp) (<10). In general, the differential margin between Vp and Vg is relatively small for all characters.

Genotypic variance coefficient (GVC) and phenotypic variance coefficient (PVC):

The GVC and PVC estimate revealed low magnitude values (<10) for disease severity stages and high magnitude values (> 20) for the AUDPC. The lowest GVC and PVC were recorded by the severity of blast on 14th day after seedling (Pyri1) and the highest AUDPC values of 7 and 7.83 for Pyri1 and 124.72 and 129.48 for the AUDPC. With the exception of the Pyri1 all characters recorded high GVC and PVC (> 20). PVC showed higher values than GCV in general, but with a low differential magnitude.

Heritability (H^2 %): the estimated heritability rate varies from 79.92 to 95.63% obtained respectively with Pyri1 and Pyri5. Thus, all stages of the disease severity including the attacked leaf area (AUDPC) recorded very high heritability.

Expected Genetic Gain Mean (EGGM): the value of heritability alone gives no indication of the importance of the genetic progress that would result from choosing the best individuals, but coupled with genetic gain, this value is more useful. In general, high percentages of genetic gain relative to the mean (> 20) were obtained for the different stages of severity of blast, except for the first scoring (Pyri1) which recorded a moderate EGGM (12.89). The highest EGGM was obtained with the AUDPC (247.48) followed by Pyri5 (193.81), Pyri4 (162.81) and Pyri3 (117.49).

4. Discussion

The present study revealed the existence of a highly significant difference between genotypes for the five disease scoring stages and the affected leaf area (AUDPC), thus indicating the existence of genetic variability between descendants. Thus, it is observed a structuring of the genotypes of the blast collection of Benin in three groups of sensitivity. This structuring shows that there is varietal diversity of rice within the national collection. Some genotypes have genes coding for complete or partial resistance to the microbial strains involve in the disease. In addition, there are genotypes that do not have resistance genes explaining their sensitivity to the pathology. This distribution is similar to the pattern identified among rice genotypes assessed for blast disease in the western Burkina Faso [22]. This structuration can be explained by the diversity of *M. oryzae* strains present on the study area. For example, the susceptibility of a resistant plant is attributed to the high level of genetic variability of pathogenic [14]. Indeed, a study conducted in central and northern Benin in reference[23], it was reported the existence of a multitude of *M oryzae* strains able to dominate approximately 13 genes of resistance (Pi1, Pi7, Pi5, Pikp, Pia, Pita2, Piks Pi3, Pik, Pita, Piz, Pikh and Pikm). Variations observed in genotype behavior could be due to differences in the resistance genes involved in each genotype and the composition of the *M. oryzae* population present on the site. Nuclear and mitochondrial genomes molecular analyses suggest that *M. grisea* pathogen remain in nature as different types of genetically distinct asexually reproducing population [24]. In India, the screening of seventy eight genotypes of rice germplasms against rice blast disease revealed that, none of the genotypes was immune towards *Pyricularia oryzae*. A structuring of the genotypes made it possible to classify them in five categories of sensitivity (Resistant Moderately, Resistant, Moderately susceptible, Susceptible, Highly susceptible) [25]. This difference in classification can be explained by the fact that this study was conducted in a lowland ecology, contrary to our study that took place in an upland ecology. [26]Found that lowland ecologies have more symptoms of blast disease, that is, the incidence of blast disease is higher in this ecology than pluvial or irrigated. From these results obtained, measures can be taken in the use of resistant and moderately resistant genotypes in extension and in varietal improvement programs.

The results obtained by cumulating the AUDPC indicate a continuous variation of the values of this index, reflecting differentiated levels of general resistance within the studied material. The genotypes that can control the progression of the disease are those able to limiting the overall incidence resulting in a low AUDPC. The differentiation of genotypes by this index then confirms the classification based on the severity of the foliar incidence. Based on the AUDPC value, [27] listed Nepal rice genotypes on five categories from resistant to highly susceptible.

The estimation of genetic variability revealed relatively low values of genotypic variance (Vg) and phenotypic variance (Vp) for the studied characters except the AUDPC with however slightly higher Vp values than those of Vg indicating a weak influence of environment on the expression of the disease. The genotypic variance coefficient (GVC) and phenotypic variance coefficient (PVC) make it possible to perceive the total variability present in a character. All the characters studied recorded high GVC and PVC (> 20) except for the first disease severity score (Pyri1), thus justifying the high variability that exists between genotypes with respect to disease resistance. As in case of Vg and Vp, PVC values were higher than those of GVC for all traits studied but with a relatively small difference, indicating a weak influence of the environment in the expression of the disease. These results agree with those obtained by [28] and [29]. Plentiful in the same sense, [30] indicated that high phenotypic variations were composed of high genotypic variations and less environmental variation, indicating the presence of high genetic variability for different traits and less influence of the environment.

The coefficient of variation indicates the extent of total variability present in a trait and does not distinguish between heritable and non heritable portions of variability. Thus, the estimation of the genotypic variance coefficient and heritability, which precisely indicates the expected heritable gain, are of great importance in the selection of parents [31]. High values of heritability (H^2%) were obtained with all resistance parameters to blast, indicating that these parameters would be easily heritable. Similar results were reported in previous studies for the different disease scoring stages and the AUDPC [29, 32]. High heritability indicates the extent of genetic improvement of these traits through selection [30, 33, 34].

A critical analysis of the genetic variability parameters, namely, genotypic variance coefficient (GVC), phenotypic of variance coefficient (PVC), heritability and genetic progress for various economically important traits is a major precondition for any breeders working in crop improvement programs [35]. The values of GVC, PVC and heritability (H^2%) taken alone give no indication of the importance of the genetic progress that would result from the selection of the best individuals, but coupled with the genetic gain compared to the average EGG, this value is more useful [31]. The leaf area affected by blast and all stages of disease severity except the first scoring stage (Pyri1) recorded high values of GVC, PVC. In addition, the heritability rate (H^2%) coupled with high values of expected genetic gain mean (EGGM), indicate a weak influence of the environment in the expression of the disease and a prevalence the effect of additive genes in the genetic determinism of these parameters. Thus, the improvement for rice blast resistance could be made according to conventional breeding methods.

5. Conclusion

The study revealed a difference in the attitude of the genotypes studied with rice blast disease at the Sowé site in

Benin, in a natural condition of infection. As a result, several rice genotypes constituting a pool of varieties resistant to blast have been identified and conserved. The genotype response reflects diversity in the population composition of the pathogen at the site. The observed resistance is of polygenic or quantitative type. Considering these preliminary results, it is desirable to extend the investigations to other sites in Benin with the aim of identifying other resistant genotypes and the population structure of the pathogens in Benin.

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