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CROWN RUST RESISTANCE IN HYBRID AVENA (*AVENA STERILIS* AND *AVENA SATIVA)*

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ABSTRACT

Oat production is usually menaced by the presence of overwhelming pathogens worldwide. Oat crown rust disease causes significant yield losses in cultivated oat. Characterization of the genetic components underlying disease resistance is highly relevant for resistance breeding programs. Interspecific crossing was made between the resistant ecotype, spontaneous oats (*Avena sterilis)* and a susceptible cultivated oat (*Avena sativa*). Resistance to crown rust was assessed using the area under the disease progress curve (AUDPC) for each individual plant. Parents were not only contrasting by their reaction to the phytopathogen, but also by their kernel morphology. In spontaneous oat flowers, the lemma is easily dislodged and showed single long twisted geniculate awn, whereas cultivated lines were having opposite characters. Kernel phenotype analysis among F_2, F_3 and BC_1S_1 plants showed that each awn development pattern could be encoded by one semi-dominant factor. Recessive homozygote genotype meant awnless, While, heterozygosis genotype showed an intermediate phenotype. Diversity in awn length and awn number in F_2 and F_3 kernels could be explained by all the possible recombination's of the two factors. Analysis of resistant and susceptible ratio in F_2 and F_3 hybrids using Chi-squared ($\chi 2$) test showed that resistance to oat crown rust of JT_0 seemed to be oligogenic and may be encoded by three dominant genes with epistatic effects. Whereas, JT_5 resistance trait could be encoded by a single dominant gene. Combination of these genes in a local oat cultivar could be an efficient way to reduce losses caused by crown rust disease.

Key words: *Avena sterilis*, *Avena sativa*, crown rust, Awn development, kernel.

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INTRODUCTION

Avena sativa, *Avena byzantina*, *Avena strigosa*, and *Avena abyssinica* are the major cultivated oat species out of 26 species of genus *Avena*. Actually, a loss of genetic diversity was observed in cultivated and semi-cultivated oat species. Eleven cultivated oat lines were carefully chosen for further evaluation to foliar disease resistance and morpho-agronomic features. Line Av.95 showed highly agronomical traits but exhibited a susceptible reaction to oat crown rust (*Puccinia coronata* f. sp. *avenae*) (Hammami *et al*., 2008). Oat crown rust reduces global oat production and decreases the economic value of the grain. It affects grain yield, kernel weight, groat percentage, and straw production (Nazareno *et al*., 2018). In Tunisia, damage to oats caused by fungal diseases, especially *P. coronata* f. sp. *avenae* was assessed as 10 to 30% on plants envisioned for silage production, and as 30 and 70% on oats envisioned for seed production (Hammami *et al*., 2010). Control of these diseases has been through the use of host resistance genes (Simons, 1985), but in Tunisia, frequent changes in pathogen virulence provides a continuing threat to oat production, since this fungus has its sexual cycle on the both hosts: *Avena* sp. and *Rhamnus lycioïdes* (Hemmami *et al*., 2006) which are present in Tunisia. Resistance genes could be found in wild oats that have co-evolved with the pathogen. Wild oat species are a valuable source of new genes useful for the improvement of cultivated oat (Maja *et al*., 2016). Several oat crown rust resistance genes, have been found in wild oat species and have been used in plant breeding (Gnanesh *et al*., 2014). A screening of a local collection of *A. sterilis* to artificial and natural crown rust infection, for many years, led us to identify two resistant ecotypes : JT_0 and JT_5 (Hammami *et al*., 2007). An interspecific crossing between JT_0 and JT_5 with the susceptible line "Av.95" was attempted to characterize the genetic basis of resistance to oat crown rust, and begin to use these genes in oat breeding. In addition to studying the genetic basis of resistance in JT_0 and JT_5, the genetic basis of flower morphology; essentially awns, was studied in the hybrid lines. Thus, a genetic analysis of the inheritance of kernel morphology was added to the genetic analysis of the resistance harbored in the selected oat ecotypes.

MATERIALS AND METHODS

Both oat ecotypes JT_0 and JT_5, (*Avena sterilis*, 2n=42) that showed a high resistance level to oat crown rust during tests of natural and artificial infection, were used in a crossbreeding program with the susceptible

cultivar Av.95 (*Avena sativa*, 2n=42). Seeds from a single plant (Av.95, JT_0 and JT_5) were sown at 2 seeds per pot and 5 pots per line. Five different sowing dates were used, 15 days between each date, to ensure the overlap of the male and female plants flowering time. Potted plants were grown in a glasshouse. This same test was also carried out in the field with 5 lines for each parent at a rate of about 30 seeds per line. The susceptible variety Av.95 was used as the female parent and the selected oat ecotypes as the male parents.

Only one plant of the hybrid F_1 was used to generate the F_2, F_3 and the BC_1 (Av.95 used as a female and the F1 as the male). Likewise, only one plant BC_1 was used to produce BC_1S_1 seeds by self-fertilization. Seeds from each generation were sown in honeycomb plates, then seedlings were replanted in field to be exposed to the natural crown rust infection. A randomized complete block with three replicates was used. Each repeat consisted of around twenty seedlings of the parental lines (Av.95, JT_0 and JT_5), and about forty seedlings of the hybrid lines. Data of kernel features were noted after the harvest. Three parameters were considered: Awn number (Awnless, 1 awn or 2 awns), awn length and kernel dehiscence.

Disease scoring began as soon as the first appearance of crown rust symptoms on leaves. The ratings were based on the number of uredinia per leaf. From this parameter, the area under the disease progress curve (AUDPC) values, was calculated as:

$$\mathrm{AUDPC} = 0.5 \sum (t_{i+1} - t_i)(y_{i+1} + y_i)$$

where the day of the *i*th reading is indicated by t*i* , with *i*= 1 for the first reading, *i* = 2 for the second, etc., and where y*i* is the mean number of uredinia per leaf at the *i*th reading (Hammami *et al*., 2007). The frequencies of susceptible and resistant plants were noted. The resistance criterion is based on the fact that the disease level is low and do not reach the economic threshold.

The ratio of resistant to susceptible lines (R:S) was subjected to the $\chi 2$ test for goodness of fit (Cabral and Park 2016). Several hypotheses were modeled but only five were considered. The expected frequency of resistant to susceptible lines (R:S) was tested from 3:1, 9:7, 15:1 and 15:17. Each of these situations could be achieved when: (i) there is only a single dominant gene for resistance, and the proportion of R:S and the possible genotypes would be 3R (1AA, 2 Aa):1S (aa). (ii) 2 dominant genes either of which gives resistance resulting in the proportion of R:S and the possible genotypes would be 9R (AABB, 2AABb, 2AaBB and 4AaBb):7S (AAbb, aaBB, 2Aabb, 2aaBb, aabb). (iii) 2 dominant genes independents and without cumulative effect (A>a and B>b), resulting in the proportion of R:S and the possible genotypes 15R (AABB, AAbb, 2AABb, 2AaBB, 2Aabb, 2aaBb, 4AaBb and aaBB):1S (aabb). (iv) 3 dominant and independent genes with at least 4 dominant alleles or 3 in heterozygote state to own the resistance (Av.95 "AAbbcc" and $JT_{0/5}$ "aaBBCC", resulting in the proportion of R:S and the possible genotypes 30R (AABBCC, AAbbCC, aaBBCC, 2AABBCc, 2AABbCC, 2AaBBCC, 4AaBBCc, 4AaBbCC and 8AaBbCc):34S (AAbbcc, aaBBcc, aabbCC, aabbcc, 2Aabbcc, 2AaBBcc, 2aaBbcc, 2AABbcc, 2AAbbCc, 2AabbCC, 2aaBBCc, 2aaBbCC, 4AaBbcc, 4AabbCc and 4aaBbCc). (v) 3 dominant and independent genes with at least 4 dominant alleles to be resistant, resulting in the proportion of R:S and the possible genotypes 30R (AABBCC, AAbbCC, aaBBCC, 2AABBCc, 2AABbCC, 2AaBBCC, 4AaBBCc, 4AaBbCC and 8AaBbCc):34S (AAbbcc, aaBBcc, aabbCC, aabbcc, 2Aabbcc, 2AaBBcc, 2aaBbcc, 2AABbcc, 2AAbbCc, 2AabbCC, 2aaBBCc, 2aaBbCC, 4AaBbcc, 4AabbCc and 4aaBbCc). Chi-squared ($\chi 2$) analyses of the data from F_2 and F_3 progeny were conducted for all crosses; in order to test the goodness-of-fit of observed to expected segregation ratios. The $\chi 2$ statistic was calculated using the formula:$\chi 2 = \sum \frac{(O-E)^2}{E}$, where O and E represent the respective observed and expected frequencies of resistant and susceptible F_2 and F_3 individuals. P-value is the risk of rejecting a hypothesis then it is true. If $p < 0.05$ then the hypothesis is rejected because the difference is significant between the observed and the expected values with a probability more than 95%. Kernel morphology factors, disease scores, $\chi 2$ analyses and illustrations were performed using the "R" program (R version 3.4.2) (Lafaye *et al*., 2014).

RESULTS

Kernel morphology: Only kernel from the crossing between JT_0 and Av.95 were analyzed for Awn number, awn length and kernel dehiscence factors.

Awn number: F_1 kernels were homogenous with one short kernel (data not shown). BC_1S_1 kernel showed globally awnless kernels and a few numbers of kernel with awn. Contrariwise, F_2 and F_3 kernels showed an important number of kernels with awn as much as in JT_0. Moreover, the mean number was nearly the same in F_2, F_3 and JT_0 (Figure 1). BC_1S_1 kernels were mostly awnless, only a few kernels showed one awn (Figure 2, Table 1). In contrast F_2 and F_3 kernel were variable and showed awnless kernels, one and two awned kernels. In F_2, the number of kernels with one awn was greater than the number of the two awned kernels, contrariwise, F_3 exhibited, more kernels with two awns than the one awned kernels. (Figure 1, Table 1).

Awn length: In the different hybrid lines, the shortest awns, from 9 mm to 19 mm, were shown only in kernels with one awn, the number of kernels with short awn was 15, 75 and 63 in F_2, F_3 and BC_1S1 lines, respectively (Table 1). The rest of the one awned F_3 kernels exhibited short awns as well ranging from 20 to 29 mm. in the

spontaneous parental line kernels, there is no awn with the shortest length and only 0.8% of kernels showed the second level of awn length (Table 1). Moreover, kernels with the greatest length, 50 mm to 60 mm, were observed only in JT_0, no kernels from all hybrid lines showed that awn length. The maximum awn length revealed in F_2 kernels and F_3 two awned kernels was between 40 mm and 49 mm (Table 1).

Kernel dehiscence: Kernels of thirty F_2 plants, twenty-two F_3 plants and twenty-six BC_1S_1 plants were analyzed for awn number and dehiscent kernels criteria. Results from all hybrid line and JT_0 ecotype showed that all kernels with two awns were easily dehiscent from its panicle, in contrast, awnless or one awned kernels were strongly attached to its rachis, thus, not dehiscent (Table 2).

Resistance to oat crown rust: The results are presented in Table 3. Obtained seeds from the first crossing between the resistant ecotype JT_0 and the susceptible cultivar Av.95 grown up in field and in pots showed high level of resistance to the natural infection with oat crown rust. Some necrosis and a very tiny uredinia appeared on their leaves (Infection type "1;") (Murphy, 1935), in addition the disease progression was mild, with low AUDPC and pustule number per leaf values (Table 2). Mean pustules number per leaf of the hybrid lines was close to that of the resistant ecotype, but high values of this parameter were more pronounced in F_3 than in F_2 than in BC_1S_1 (Figure 2.A). In the second cross "Av.95XJT_5", BC_1S_1 showed intermediate mean uredenia number per leaf values comparing to the susceptible and the resistant check. In addition, resistance in F_3 generation were more discernable than in F_2 (Figure 2.B).

Parental lines showed considerable variation regarding AUDPC parameter, from 1500 to 3300 for the susceptible check, and from 0 to 1000 for the resistant ecotype. This observance showed the amount of genetic variation comparative to environmental disparity (Figure 3). However, histograms of hybrid lines showed a unimodal distribution for a segregating F_2, F_3 and BC_1S_1 populations. This may lead to conclude that the control of crown rust resistance is complex and probably encoded by many genes of small action. Nevertheless, it is important to note that all histograms were skewed in the right side (Figure 3). This may contribute to a contrasting deduction of dominant major gene action. Thus, it was necessary to move to the qui-squared analysis to answer about the number of genes involving in the resistance to oat crown rust hosted by JT_0.

For the first cross "JT_0 X Av.95", hypotheses 4 and 5 could be retained for the F_2 segregation, since p-values were > 0.05 (Table 4). These two hypotheses could explain also the segregation for crown rust reaction in F_3 generation; p-value was 0.14 for both hypotheses (Table 5). Whereas, the descendants from the second crossing between JT_5 and Av.95 aligns quite with the hypothesis number 1. Resistance to oat crown rust seems to be controlled in JT_5 by a single dominant gene. P-values of this hypothesis were 0.79 and 0.27 for F_2 and F_3 segregation, respectively (Table 4 and 5).

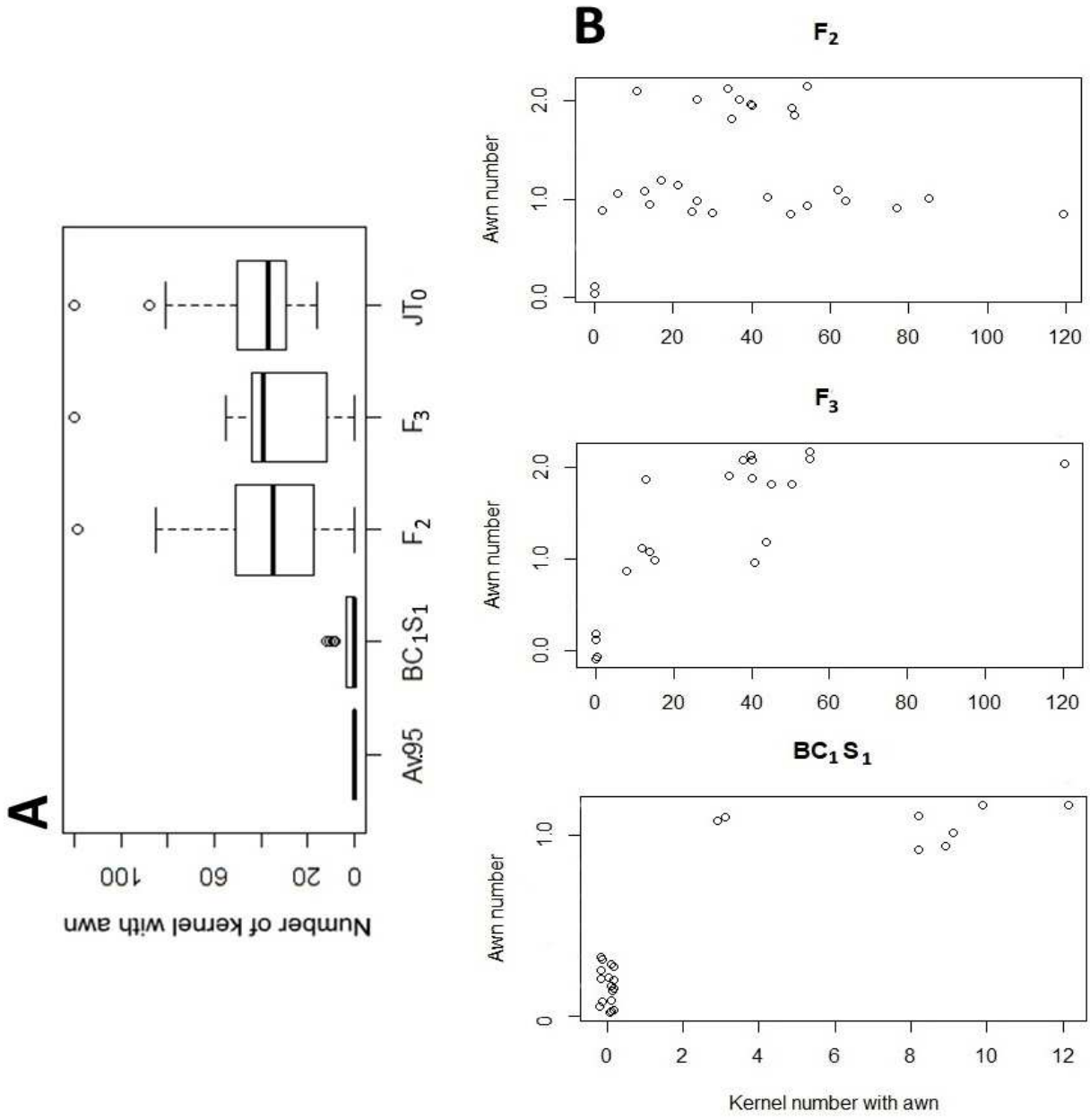

Figure 1. Kernel number with awn: A - number of kernel with awn among parental and hybrid lines. B - Details of kernel number with one or two awns or awnless among F2, F3 and BC1S1 hybrids.

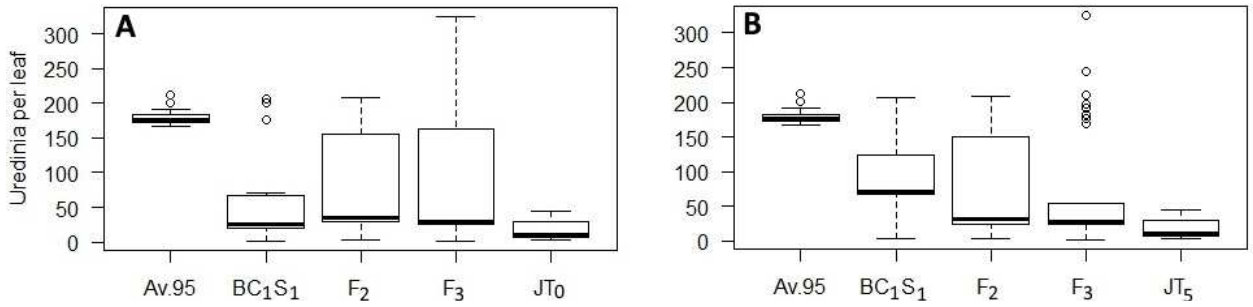

Figure 2. Number of uredinia per leaf shown in the parental and the hybrid lines: A: Av.95XJT0; B: Av.95XJT5.

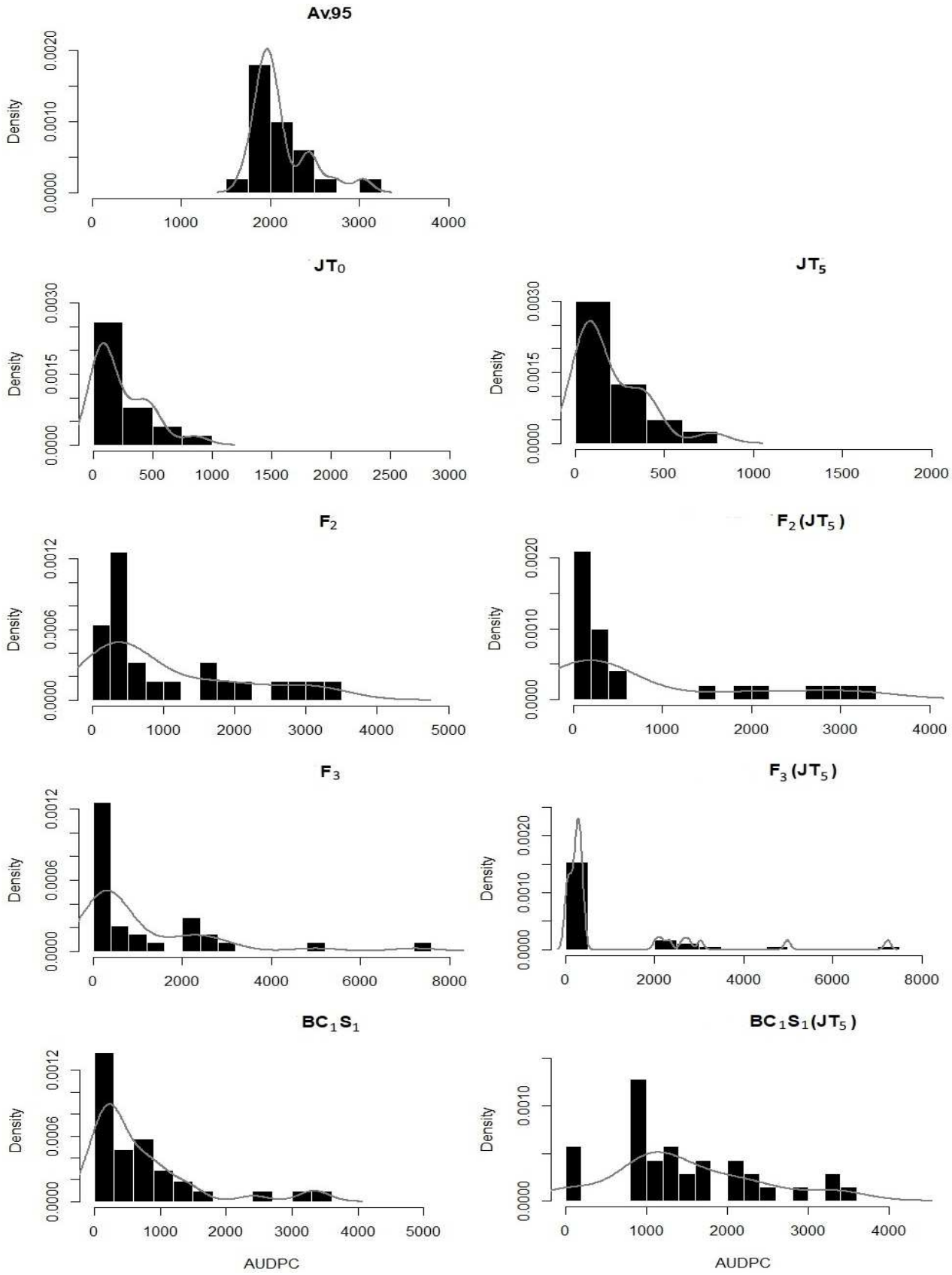

Figure 3. Plants distribution of parental and hybrid lines according to AUDPC values of the both crossing.

Table 1. Awn lengths and number of awnless, one awned and two awned kernels in parental and hybrid lines.

Accessions	Awn length intervals (mm)					Total
	[9,19]	(19,29]	(29,39]	(39,49]	(49,60]	
JT_0	0	11	124	659	546	1340
F_2 (1 awn)	15	376	252	57	0	700
F_2 (2 awns)	0	65	203	111	0	379
F_3 (1 awn)	75	60	0	0	0	135
F_3 (2 awns)	0	133	342	95	0	570
BC_1S_1 (1 awn)	63	0	0	0	0	63
					F_2 (0 awn)	115
					F_3 (0 awn)	285
					BC_1S_1 (0 awn)	1193

Table 2. Kernel morphology data. Awn number and dehiscent kernels are presented by the number of plants.

Accessions	Awn number			Dehiscent kernel		Kernel with awn			Kernel total number
	0	1	2	Yes	no	1	2	Total	
Av.95	30	-	-	-	30	-	-	-	1340
JT_0	-	-	30	30	-	-	1340	1340	1340
F_2	3	17	10	10	20	700	379	1079	1194
F_3	4	6	12	12	10	135	570	705	990
BC_1S_1	18	8	-	-	26	63	-	63	1256

Table 3. Reaction of parental and hybrid lines to the natural infection of oat crown rust.

Oat lines	Reaction	Infection type	Mean AUDPC	Mean pustule number per leaf (last notation)
JT_0	R	1;	232.93	17.51
JT_5	R	1;	200.89	17.48
Av.95	S	4	2119.45	179.41
$F1_{JT0}$	R	1;	186.47	16.21
$F1_{JT5}$	R	1;	176.88	15.78

Table 4. F_2 segregation for crown rust reaction in the crosses JT_0 X Av.95 and JT_5 X Av.95.

Cross	Plants			Expected ratio	X-squared	p-value
	R + M*	S	n			
X1	32	44	76			
X2	56	20	76			
H1	1 dominant gene.			3:1	43.86	3.528e-11
					0.07	0.79
H2	2 dominant genes, independents with complementarily.			9:7	6.17	0.012
					9.38	0.002
H3	2 dominant genes independents and without cumulative effect.			15:1	345.95	< 2.2e-16
					52.22	4.95e-13
H4	3 dominant and independent genes with at least 4 dominant alleles or 3 in heterozygote state to own the resistance.			15:17	0.69432	0.40
					21.93	2.82e-06
H5	3 dominant and independent genes with at least 4 dominant alleles to own the resistance.			15:17	0.69432	0.40
					21.93	2.82e-06

Table 5. F_3 segregation for crown rust reaction in the crosses JT_0 X Av.95 and JT_5 X Av.95.

Cross	Plants			Expected ratio	X-squared	p-value
	R + M*	S	n			
X1	34	72	106			
X2	76	30	106			
H1	1 dominant gene.			2:1	1.2	0.27
H4	3 dominant and independent genes with at least 4 dominant alleles or 3 in heterozygote state to own the resistance.			15:17	2.17	0.14
H5	3 dominant and independent genes with at least 4 dominant alleles to own the resistance.			15:17	2.17	0.14

DISCUSSION

Oats differ from the other commonly cultivated cereals by having an inflorescence in the form of a panicle, while the inflorescences of wheat, barley, and rye are spikes (Bonnett, 1937). Seeds of wild oat especially *Avena sterilis* differ from the grown oat by abscission spikelet separation, lemma hairiness, and development of strong awns (Campbell and Frey, 1972). The hybrid F_1 were homogenous with one short awn and mature kernels strongly linked to the panicle rachis. F_1 kernel morphology data suggest that awn length and awn number traits could be controlled by a semi-dominant factor, but kernel dehiscence trait is encoded by a dominant factor. Furthermore, data set from F_2, F_3 and BC_1S_1 kernels showed that, in the one hand, awn number and awn length traits seem to be inherited as a completely linked as also kernel dehiscence and awn number traits. In the other hand, kernel dehiscence and awn length traits were segregated separately. Previously it was noted that *A. sterilis* seed traits, kernels dehiscence and combined awn, were inherited as an entirely linked set (Campbell and Frey, 1972). In fact, awns are known to show hygroscopic movement that help in the dispersal and the burial of seeds, this movement could induce the dislodging of seeds from spikelets by making substantial twisting (Ladizinsky, 2012). In literature, genetic control of awn development, especially in oat, was distinct. Since the beginning of the 20th century, it was assumed the awn development in oat could be controlled by two dominant factors, one factor for twisted geniculate awns (wild character) and the second factor for the intermediate awns (twisted non-geniculate). The double recessive conditions weak awns or no awns. Another possibility of a third genetic factor that operate in the absence of the first two factors and conditions the awnless kernels (Johnson, 1933). It was also reported that awning is controlled by one principal pair of genes and the heterozygotes showed awns in various intermediate degrees (Surface, 1916). No recent works discussed the oat kernel characters inheritance but for other cereals like barley, wheat (*Triticum aestivum*) and rice (*Oryza sativum*). Several surveys were made as a purpose to characterize, specially, awn development. In rice, it was suggested that awnless in BC_5F_2 population was encoded by one dominant gene (Hu *et al*., 2011). In addition it was stated that long awn in rice was controlled by a single dominant gene that increased cytokinin content in epidermal cells (Gu *et al*., 2015; Hua *et al*., 2015). Recently in wheat, it was indicated that the spikelet phenotypes are encoded by different genetic systems and a recessive major factor controlled the spike architecture (Zhang *et al*., 2017). In barley it was reported that a single gene Lks2 controls awn elongation (Yuo *et al*., 2012). In oats, things may be different. Spikelet on oat is composed of three flowers whose intermediate is sterile. In oat ecotype, lemma of each fertile flower shows single long twisted geniculate awn. Each awn development could be encoded by one factor. Recessive homozygote genotype means awnless, while heterozygosis genotype showed intermediate phenotype and that may explain the intermediate kernel morphology in the hybrid F_1. In our results, BC_1S_1 spikelet were awnless or one awned. Thus, on the basis of this hypothesis, BC_1 seed did not harbor the two dominant alleles, that is why after self-pollination, progenies did not show the ecotype phenotype. Diversity, in awn length and awn number in F_2 and F_3 kernels could be explained by all the possible recombination's of the two factors. Only the spikelets with two awns were easily dislodged from the rachilla. This fact could be explained by two hypotheses. First, is the possibility of no genetic control and the kernels being dislodged cause only of the movement of the twisted and geniculated awns. Alternate possibility is that factors controlling awn length and awn number could control in the same time, kernels dehiscence, but with dominant alleles. Thus, ecotype harboring that genotype showed pedicel weakness after seeds maturity. All these hypotheses needs to be confirmed using molecular and biochemical tools.

Histograms for disease resistance for both crosses showed unimodal distribution for a segregating F_2, F_3 and BC_1S_1 populations (Figure 3). As a first

thought, we could deduct that resistance to oat crown rust may be complex and probably encoded by many genes of small action. Nevertheless, it is worth noted that the distribution is skewed in the right side. This may suggest that a dominant major gene possibly encodes resistance. This supposing that dominant and heterozygote genotype shows individuals with low AUDPC values while the other genotype showed individuals with high AUDPC values. However, polygenic trait could also generate a unimodal distribution with skewed residual error distribution. Looking to the parental distribution, it can be seen that it exhibited a great genetic variation relative to environmental variation. Thus, χ-squared test analysis was necessary. Only five hypotheses were retained from several ones (data not shown). Results suggest that JT_0 and JT_5 did not harbor the same genetic control. In fact, resistance to oat crown rust trait of JT_0 seems to be oligogenic, and may be encoded by three dominant genes with epistasis effects. Whereas, JT_5 resistance trait could be encoded by a single dominant gene (Table 4 and 5). Several surveys proved that north Africa is an important source of resistance to oat crown rust harbored in many oat accessions specially *Avena sterilis* (Loskutov, 2002; Zillinsky and Murphy, 1967). This oat species is known to be the origin of many *Pc*-genes like *Pc39*, *Pc64*, *Pc67*, *Pc68* (Chong *et al*., 2000) and *Pc45* (Gnanesh *et al*., 2014). The potential of *A*. *sterilis* collected from Mediterranean and near east region as a valuable source of resistance to oat crown rust was discovered since the la late 1950's. Since that time, several crosses between resistant oat ecotype and susceptible oat cultivars were made. It was showed that each *A. sterilis* strain harbored a single gene of resistance. Besides, it was reported that these oat ecotypes had one or more genes for field resistance (Bushnell and Roelfs, 1984). A catalog list of 61 genes for oat crown rust resistance was reported since the early 1960s. Most of these genes have been inherited from *A*. *sterils* (Simons, 1979). In this survey, it was hypothesized that JT_0 and JT_5 did not have the same resistance mechanism to crown rust. JT_0 could host an oligogenic resistance but JT_5 may harbored a single gene of resistance. Thus, resistance of JT_0 may be quantitative and that of JT_5 may be qualitative. Quantitative disease resistance was defined as host plant resistance showing a reduction in disease impact instead of the absence of disease infections. This kind of resistance include partial, complex, polygenic, oligogenic, horizontal, field, and lasting. Qualitative disease resistance (monogenous) has not been widely used in breeding programs (St.Clair, 2010). In Tunisia monogenic resistance could easily overcome by local crown rust pathotypes since this fungus is completing his sexual life cycle on the local alternate host *Rhamnus lycioïdes* (Hemmami *et al*., 2006). Differential oat lines known to host single gene for resistance to crown rust were used since 1940. They did not maintain their resistance level for more than 10 years. In USA, it was reported that principal cause of the short lasting of these lines is the abundance and the widespread of the alternate host *Rhamnus cathartica* (Chong *et al*., 2000). In contrast, oligogenic or polygenic resistance would increase the number of genetic changes required for a pathogen to overcome such resistance. Genes of such resistance could interact with the environment and each other, in addition the gene(s) can encode more than one trait (St.Clair, 2010). Combination of diverse genes with different genetic backgrounds leads to particular forms of resistance, the best would be the lasting slow rusting type or partial resistance that allow disease development but without affecting the yield or the crop quality (Akın *et al*., 2016). Both ecotypes are a potential source of resistance to the local crown rust population. Oligogenic resistance of JT_0 may last longer than that of JT_5, but their combination in a local oat cultivar could be an efficient solution to save losses caused by crown rust disease.

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