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Genetic Divergence between Wild and Cultivated Pearl Millets (*Pennisetum typhoides*)

II. Characters of Domestication

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With 3 figures and 8 tables

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Abstract

Morphological characters directly involved in the domestication of pearl millet were genetically analysed in two wild × cultivated crosses which previous research had shown to be sources of male sterility. The use of marker genes revealed frequent cases of distorted segregations, always in the direction of an excess of wild characters. This phenomenon was probably a manifestation of pollen competition. No reciprocal effect was observed in the F1's of either cross for biometrical characters. A continuous array of segregants was observed between the extreme wild and cultivated phenotypes with no apparent physiological disturbances. Spikelet shedding and length of the pedicel of the floral involucre appeared to be determined by the same pleiotropic gene system (1 major and 1 minor gene). The diameter of the spike rachis was controlled by a relatively large number of genes (approximately 5 independent genes). A linkage group was found that included genes for esterase, glutamate oxalo-acetate transaminase, foliar limb pubescence and the major gene for the pedicel length of the floral involucre. Globally, the morphological divergence between wild and cultivated forms appeared to involve up to 10 independent genes. The domestication of pearl millet was perceived as a slow, gradual, polydirectional process with multiple origins.

Key words: *Pennisetum typhoides* — distorted segregation — enzyme markers — seed shedding — linkage — biometry

Most recent botanical studies (BRUNKEN 1977, CLAYTON and RENVOIZE 1982) distinguished three interfertile groups in the biological species of pearl millet (*Pennisetum typhoides* = *P. americanum* = *P. glaucum*) corresponding to the wild forms (carrying diverse botanical names, e.g., *P. violaceum*, *P. mollissimum*, etc.), the cultivated forms, and intermediate forms, separated

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by sharp morphological differences and clearly different geographical distributions.

The wild form grows on the one hand in a purely wild state, far from any culture, in precise restricted ecological niches in central and southern parts of the Sahara Desert. It grows on coarse sandy soils in streambeds (e.g., the Tilemsi Valley in Mali) or at the foot of sandstone cliffs (e.g., the Tiguidit Cliff in Niger). It does not seem able to become established over vast areas like *Cenchrus biflorus*, whose seeds have been harvested by nomads for thousands of years without showing any symptom of domestication. The wild form is also abundantly present on the borders of roads, fields, dwellings and wells, always on disturbed sandy soils, from Senegal to Sudan, along the northern border of the pearl millet belt. It is a typical example of the notion of ecological weed developed by HAWKES (1983) according to whom this plant was naturally predisposed to be cultivated. The wild form exhibits little morphological variability: 10 cm long spikes, shedding spikelets on stalks less than 0.3 mm long, seeds 2.5 mm long and 1.3 mm wide, enclosed by long bristles and bracts, the spike rachis 0.7 mm wide, and abundant tillering and branching.

The cultivated form is essentially characterized by the modification of morphological characters that constitute the syndrome of domestication described by HARLAN (1975): Suppression of spikelet shedding, reduction of size of bristles and bracts, less branching and tillering, increase in spike length, spikelet pedicel length, rachis diameter and seed size. But these characters do not always evolve jointly: the 'Zongo' cultivar from Niger has spikes 2 meters long with shedding spikelets. The spikes of cv. 'Tiotandé' are short but have a thick rachis (3.3 mm), a long spikelet stalk (10 mm) and large seeds. Some cultivated millets from India or East Africa have seeds not much larger than wild types. Therefore, it is not possible to morphologically define the cultivated type by a unique character (not even non-shedding).

The intermediate form looks like a hybrid between the wild and cultivated forms. It accompanies the cultivated form in fields with a frequency decreasing from north to south in the West African pearl millet belt.

The considerable morphological diversity of the cultivated form is evidently a consequence of the domestication process. The present study tries to provide information on the genetic mechanisms implied by this process. Are there obstacles to the recombination of wild and cultivated genes? For the main characters of domestication, how many genes are involved, what is their mode of action and what are their relations of linkage?

Estimation of the number of genes controlling these characters provides a measure of the genetic divergence between wild and cultivated forms and also measures the time and labour expended by generations of peasants to develop the current cultivated types; an indirect measure would also be obtained of the effort required by present-day breeders to introgress various wild characters into a given cultivated type.

The degree of association of the genes controlling the different characters is of interest for determining if the domestication syndrome is based upon some particular form of genetic organization. The possible existence of cytoplasmic effects can have a bearing on systematics and plant breeding.

The information available in literature on these questions as pertaining to pearl millet is very scarce. BILQUEZ and LECOMTE (1969) explained shattering by hypothesizing three dominant genes, seed size by about 4 additive genes and stem diameter by about 5 genes. PERNÈS (1983) suggested that pearl millet domestication was a relatively simple and fast process, repeated independently in several places, because characters of domestication are ruled by linked genes, grouped on a small number of chromosomal segments.

Materials and Methods

The material evaluated was the progeny from two crosses between wild and cultivated pearl millets: *P. violaceum* (wild) × 'Tiotandé' (cultivated) and *P. mollissimum* (wild) × J104 (cultivated), which previous research had shown to be sources of male sterility. The parental plants were derived from heterogeneous populations except for J104 which was a pure line. Origins of these four parental samples were given in a previous paper (MARCHAIS and PERNÈS 1984).

Lists of all the F1, F2 and backcross families, with their pedigrees, are given in *Tables 1* and *2*. V and M designate the wild parents and T and J the cultivated parents. Each individual plant is identified by a number, used to designate the pedigree. BCt (v, j, m) is a backcross to T (V, J or M).

Thirteen F2 families (labelled 1 to 13), 10 BCt families (labelled 14 to 23) and 2 BCv families (labelled 24 and 25) from the V × T cross were observed. The subset of families: F2 (12, 13), BCt (20, 21), BCv (24, 25) can reasonably be considered to descend from the same two genetically fixed parents, which had been selfed at least three times. This subset has been used for biometrical analyses.

Twelve F2 families, 4 BCj families, 2 BCm families and 5 selfed progenies from BCj plants were observed from the M × J cross. These families descended from heterogeneous parents and consequently were not analysed biometrically. However a quantitative analysis of heterosis and reciprocal effects was performed on a subset limited to parents and F1's because reciprocal crosses and selfs were carried out with the same two individual wild and cultivated plants.

Parents and all progenies were grown and observed in greenhouses at Bondy (France) in successive years from 1978 to 1982. Each time, the diverse families were disposed in adjacent blocks without any special statistical design.

Three available qualitative characters were observed to check if the segregations were Mendelian:

— the carboxylic esterase E1 gene (E.C.3.1.1.1) described by SANDMEIER et al. (1981);
— Glutamate oxalo-acetate transaminase (E.C.2.6.1.1) coded in pearl millet by at least 2 genes, G.O.T. A and G.O.T. B (TOSTAIN and RIANDEY 1984). Only the G.O.T. A gene which was polymorphic in the M × J cross was studied. The G.O.T. A alleles are codominant and code for dimeric allozymes;
— Presence or absence of pubescence on the foliar limb.

Eight quantitative morphological characters were selected for observation because they were components of the syndrome of domestication described by HARLAN (1975) and because they exhibited a striking difference between the wild and the cultivated parents studied in this paper:

— AB, the degree of shedding of the spikelets

Notation 0 no abscission layer, zero shedding
1 badly shaped abscission layer, zero shedding
2 clear abscission layer, low shedding
3 clear abscission layer, intermediate shedding
4 clear abscission layer, high shedding

— DR, diameter of the rachis of the main spike in 1/10 mm
— LPI, pedicel length of the floral involucre in 1/10 mm measured from the abscission layer (or the rachis) to the base of the involucre
— LS, length of the involucre bristles in 1/10 mm
— AR, length of some bristles extending beyond the involucre in 1/10 mm
— LGLI, length of the upper floret lemma in 1/10 mm
— LG, seed length in 1/10 mm
— DG, seed width in 1/10 mm.

The number of effective factors was estimated using classical procedures described by MATHER and JINKS (1971) with the following assumption: For a given quantitative character of domestication, the wild parent carries all the decreasing (or increasing) genes, whereas the cultivated parent carries all the increasing (or decreasing) genes. This assumption expresses the fact that the wild phenotype occupies one extremity of the range of variation in the whole biological species. If we call P1 (P2) the parent with the increasing (decreasing) genes and VBi the variance of the backcross i ($i = 1, 2$), the mean value of ($P1 - F1$) is $\Sigma (d - h)$. The genetic component of VB1 is $1/4 \Sigma (d - h)^2$. The corresponding expressions with P2 uses ($d + h$) instead of ($d - h$).

So $N(F2) = (P1 - P2)^2 / 8 (VF2 - VF1)$ (classical estimation)

$N(BCi) = (Pi - F1)^2 / 4 (VBi - VF1) \quad i = 1, 2.$

N (F2) and N (BCi) designate the number of genes estimated using the F2 or the backcrosses i. VF1, VF2 and VBi are the variances of their respective generations. The advantage of using a wild parent is that N (BC) is not underestimated [like N (F2) is], in the presence of the effects of dominance.

Results

Non-Mendelian esterase segregations

SANDMEIER et al. (1981) showed that esterase E1 has a dimeric structure coded by a Mendelian gene with about 10 allelic forms. In both crosses (*Tables 1* and 2), frequent cases of non-Mendelian segregations for esterase were found in the F2 and BC generations. When deviations occurred, it was always the wild allele that was in excess. However, in F2's which had non-Mendelian segregations, the percentage of heterozygotes (VT or MJ) was never statistically different from 50%. In the backcrosses, the deviations never occurred when the F1 was used as the female parent. The deviations were found whether the recurrent female parent was wild (*Table 1*, family 24; *Table 2*, family 17) or cultivated (*Table 1*, family 20; *Table 2*, families 13 and 15). Thus, the cytoplasm did not seem to be influencing segregation. The same levels of deviations were observed for seeds (*Table 1*, families 2, 3, 4, 7) and for green plants (*Table 1*, families 1, 10, 24). F1 pollen analysis did not show any particular male sterility (MARCHAIS and PERNÈS 1984).

Thus, we think that the deviations were produced by pollen competition at the level of germinating pollen tubes in stylar tissue, as observed by SARR et al. (1983) in pearl millet and by OTTAVIANO et al. (1982) in maize. The exact mechanism is not yet understood. The following facts are yet to be elucidated:

— The F2 progeny from the F1(V4×T10)6 deviated significantly from a 1 : 2 : 1 ratio (*Table 1*, family 7), but the BC progeny showed a Mendelian segregation (*Table 1*, family 19). Similarly, BC no. 15 (*Table 2*) had a

Tab. 1 P. violaceum x 'Tiotande' cross (V x T). Families, pedigrees, segregations for esterase and limb pubescence. Esterase alleles: V-wild, T-cultivated. Hairy limb (+), glabrous limb (–)

No	Family	Esterase					Limb pubescence			
	F2	VV	VT	TT	Total		+	–	Total	
1	(V4 x T10)1	22	26	4	52	**				
2	(V4 x T10)2	25	41	12	78	*				
3	(V4 x T10)3	26	42	10	78	*				
4	(V4 x T10)4	10	20	9	39					
5	(V4 x T10)5	16	42	20	78					
6	(V4 x T10)6	21	30	1	52	**				
7	(V4 x T10)7	23	32	6	61	**	36	54	90	**
8	[(V4 x V6) 4 x T5.5]1	3	17	6	26		16	28	44	
9	[(V4 x V6) 2 x T5.2]6						10	20	30	
10	[(T5.5 x (V4 x V6)1]2						10	20	30	
11	[(T5.5 x (V4 x V6)1]3						5	24	29	
12	[(V4 x V6) 1.2.1.3 x T5.5.4.3.1]3	13	24	10	47		17	30	47	
13	[T5.5.4.3.1 x (V4 x V6) 1.2.1.3]9	11	23	14	48		13	35	48	
	BCt									
14	[(V4 x V6) 4 x T5.5]1 x T5.5.4		20	22	42					
15	T5.5.6 x [(V4 x V6) 4 x T5.5]1		20	19	39					
16	[(V4 x V6) 2 x T5.2]6 x T5.4		12	13	25					
17	[T5.5 x (V4 x V6) 1] 2 x T5.4		11	5	16					
18	T5.3 x [T5.5 x (V4 x V6) 1]2		19	19	38					
19	T5.2 x (VA x T10)6		15	15	30					
20	T5.5.4.3.1.2 x [(V4 x V6) 1.2.1.3 x T5.5.4.3.1]1		58	36	94	*				
21	[(V4 x V6)1.2.1.3 x T5.5.4.3.1] 2 x T5.5.4.3.1.3		55	45	100					
22	T5.5.4.3.1.5.5.4 x [T5.5.4.3.1 x (V4 x V6) 1.2.1.3]8		24	28	52					
23	T5.5.4.3.1.5.5.1.2 x [(V4 x V6) 1.2.1.3 x T5.5.4.3.1]6		29	23	52					
	BCv									
24	(V4 x V6) 1.2.6.3 x [T5.5.4.3.1 x (V4 x V6) 1.2.1.3]3	42	22		64	*	41	23	64	*
25	[T5.5.4.3.1 x (V4 x V6) 1.2.1.3.] 3 x (V4 x V6) 1.2.6.3	34	40		74		36	38	74	

*(**) significant difference at 5% (1%) level from the 1 : 1 ratio in backcrosses, from the 1VV : 1TT ratio in F2 esterase or from 1 (+) : 3 (–) in F2 limb pubescence

Tab. 2 *P. mollissimum* x J104 cross (M x J). Families, pedigrees, segregations for esterase and limb pubescence. Esterase alleles: M-wild, J cultivated. Hairy limb (+), glabrous limb (–)

No	Family	Esterase					Limb pubescence			
	F2	MM	MJ	JJ	Total		+	–	Total	
1	(M2 x J1)1						10	30	40	
2	(J3 x M11)4						10	30	40	
3	(J3 x M11)1						3	23	26	
4	(J3 x M11)2	26	20	6	52	*				
5	(J3 x M11)3	13	29	10	52		13	36	49	
6	(J3 x M11)6	20	25	7	52	*				
7	(M2 x J1)6	7	13	6	26					
8	(M2 x J1)8	10	26	16	52					
9	(M2 x J1)9	2	18	6	26					
10	(M2 x J1)10	6	14	6	26					
11	(M2 x J1)4	6	18	5	29		8	22	30	
	BCj									
12	(M2 x J1)5 x J2.1		7	11	18					
13	J2.4 x (J3 x M11)1		42	23	65	*				
14	(M2 x J1) 4 x J.2.3.1.2		22	33	55					
15	J2.3.1.6 x (M2 x J1)4		43	13	56	*				
	BCm									
16	(J3 x M11)1 x M 14.5.2.1	28	28		56		26	31	57	
17	M14.5.2.1 x (J3 x M11)1	21	9		30	*	26	4	30	**
	Selfed heterozygous plants from distorted BCj No 15									
18	[J2.3.1.6 x (M2 x J1) 4]1	13	28	11	52					
19	[idem]15	5	15	6	26					
20	[idem]21	8	12	6	26					
21	[idem]28	3	15	8	26					
22	[idem]30	5	17	4	26					

*(**) significant difference at 5% (1%) level from the 1 : 1 ratio in backcrosses, from the 1 MM : 1 JJ ratio in F2 esterase or from 1 (+) : 3 (–) in F2 limb pubescence

distorted ratio but the F2 no. 11 segregation was normal.

— BC no. 23 (*Table 1*) was normal but BC no. 20, practically isogenic, had a significant deviation.

— Progenies from heterozygous plants, chosen from BC no. 15 (*Table 2*) which had a significant deviation, had Mendelian segregations (families 18 to 22).

Pubescence of the foliar limb

The whole set of segregations (presence — absence of pubescence) obtained for families which exhibited Mendelian segration for the esterase gene, gave a good fit to a one gene model (*Tables 1* and *2*). The "absence" allele was completely dominant over the "presence" allele. This result is in agreement with the results of RAO and KODURU (1979).

Table 3 shows all the bivariate segregations noted for esterase and limb pubescence; it is evident that genes for these characters were linked. The recombination rate was estimated over the diverse generations in a cross by the maximum likelihood method (ALLARD 1956). The estimations obtained from

Tab. 3 Recombination rates: esterase-limb pubescence

P. violaceum x 'Tiotande' cross						
Family No	Hairy limb Esterase			Glabrous limb Esterase		
	VV	VT	TT	VV	VT	TT
F2 No 12	12	4	1	1	20	9
F2 No 13	8	5	0	3	18	14
BCv No 24	35	6		7	16	
BCv No 25	28	8		6	32	
	$p1 = 0.178 \pm 0.027$					

P. mollissimum x J104 cross						
Family No	Hairy limb Esterase			Glabrous limb Esterase		
	MM	MJ	JJ	MM	MJ	JJ
F2 No 5	6	5	2	5	23	8
BCm No 16	21	5		7	23	
BCm No 17	20	6		1	3	
	$p2 = 0.247 \pm 0.038$					

Combined estimate $p = 0.201 \pm 0.022$

p1, p2, p: recombination rates

V, M, T, J: esterase alleles of parents

each cross were not statistically different. The combined estimate of the recombination rate between the esterase and pubescence genes was 0.201 ± 0.022.

Glutamate oxalo-acetate transaminase

In the M×J cross, 3 genotypes corresponding to the parents (MM and JJ) and to the F1 (MJ) can be seen in F2 and BC generations with the proportions reported in *Table 4*. G.O.T. allozymes had a monogenic Mendelian segregation in families which had Mendelian segregation for esterase and limb pubescence.

Tab. 4a Recombination rates : G.O.T. — esterase

G.O.T.	MM			MJ			JJ			
Esterase	MM	MJ	JJ	MM	MJ	JJ	MM	MJ	JJ	Total
Family No										
F2 No 5	6	5	2	5	18	5	0	5	3	49
BCj No 14					7	5		4	14	30
BCj No 15					17	8		3	2	30
BCm No 16	22	6		6	21					
BCm No 17	19	7		2	2					

p (G.O.T. — esterase) = 0.288 ± 0.032

Tab. 4b Recombination rates: G.O.T. — limb pubescence

G.O.T.	MM		MJ		JJ		
Pubescence	+	–	+	–	+	–	Total
Family No							
F2 No 11	6	0	2	16	0	5	29
F2 No 5	13	0	0	28	0	8	49
BCm No 16	24	4	2	26			56
BCm No 17	25	1	1	3			30

p (G.O.T. — pubescence) = 0.059 ± 0.018

Tab. 4c Recombination rates: G.O.T. —
Length of involucral pedicel (LPI)

LPI class	0.6 — 2 mm		3 — 4.5 mm		Total
G.O.T.	MJ	JJ	MJ	JJ	
Family No					
16-m-F1 x J	12	2	0	16	30
17-J x m-F1	25	0	0	5	30

p (G.O.T. — LPI) = 0.033 ± 0.022

The segregations are from the mollissimum x J104 cross
p = recombination rate
M, J designate the parental allele
Limb pubescence: hairy (+), glabrous (–)

The same table shows that the G.O.T. gene was linked to the genes controlling esterase and limb pubescence. The respective recombination rates were 0.288 ± 0.032 and 0.059 ± 0.018.

Major gene for pedicel length of the floral involucre (LPI)

Combined frequency distributions for LPI, esterase and G.O.T. regularly exhibited a linkage between short LPI (wild side) and the enzyme genotypes VT or MJ in all the BCt and BCj crosses and, reciprocally, a linkage between long LPI (cultivated side) and the enzyme genotypes TT or JJ (see *Tables 4 c* and *5*). This correlation revealed the existence of an LPI gene linked to the esterase and G.O.T. genes. Furthermore, "non-conforming" enzyme genotypes (VT and MJ in the long LPI class or TT and JJ in the short LPI class) were not localized close to the LPI distribution median but were randomly distributed throughout the range of variation of each class (see *Fig. 1* which illustrates

Tab. 5 Recombination rate: Major gene controlling the length of the involucral pedicel (LPI) and esterase gene

P. violaceum x 'Tiotande' cross						
Family No	Median	LPI ⩽ Median		LPI > Median		Total
	LPI	VT	TT	VT	TT	
BCt – 14	30	17	4	3	18	42
BCt – 15	34	15	5	5	14	39
BCt – 16	10	11	2	2	11	26
BCt – 17	15	7	1	4	4	16
BCt – 18	18	14	5	5	14	38
BCt – 19	24	12	3	3	12	30
BCt – 20	14	18	6	11	13	48
BCt – 21	14	21	3	6	18	48
		p1 = 0.236 ± 0.025				

P. mollissimum x J104 cross						
Family No	Median	LPI ⩽ Median		LPI > Median		Total
	LPI	MJ	JJ	MJ	JJ	
BCj – 12	20	7	0	0	11	18
BCj – 13	20	29	4	4	12	49
BCj – 14	20	8	6	3	13	30
BCj – 15	20	17	8	3	2	30
		p2 = 0.220 ± 0.036				

Combined estimate p (LPI – esterase) = 0.230 ± 0.020

V, T, M, J designate the parental esterase alleles. The distorted segregations from the cross M x J (No 13 and 15) are used because they have two completely disjoint groups situated on either side of the median indicated.

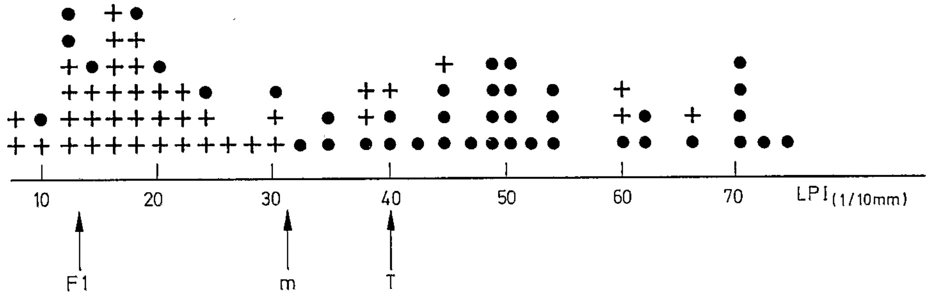

Fig. 1 Frequency distribution of the pedicel length of the floral involucre (LPI) for the pooled backcrosses BCt 14 and 15. Esterase genotypes: + heterozygote VT, • homozygote TT. m = Median; F1 = Superior limit for F1; T = Inferior limit for 'Tiotandé'

the pooled distributions of the reciprocal BCt 14 and 15, chosen because they were grown together and had Mendelian segregations for esterase).

Thus, we can roughly estimate that the median of LPI distributions distinguished two genotypes (Aa and aa) of an LPI gene. This gene can be considered to be a major gene because the variations brought about by other LPI genes remained inside the limits of the two classes determined by the median. Non-conforming enzyme genotypes must have been recombinants rather than misclassified genotypes. This reasonable hypothesis enabled us to estimate the recombination rates between the LPI major gene and the esterase gene (0.23 ± 0.020) and between the LPI and the G.O.T. genes (0.033 ± 0.022). Note that in the case of the G.O.T.-LPI linkage (*Table 4c*) the median was not used because the LPI segregation showed two distinct classes corresponding roughly to F1 and J104, indicative of a single gene. In the other BCt and BCj, many LPI phenotypes occurred that were clearly intermediate between F1 and T or J (see again *Fig. 1*). This indicated the presence of more than one gene in LPI genetic determination.

Figure 2 sums up the recombination rates between the 4 genes (esterase, limb pubescence, G.O.T. and the LPI major gene) which were estimated by combining the results of the two crosses. The overall results are in concordance.

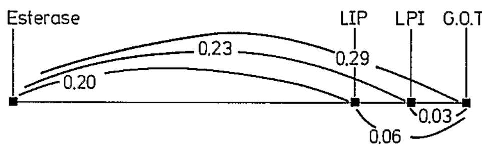

Fig. 2 Distances between esterase, limb-pubescence (LIP), Involucral Pedicel (LPI) and G.O.T. genes. The numbers indicate recombination rates estimated from both crosses

Spikelet shedding

The wild parents, V and M, the F1's and the BCv or BCm exhibited a clear abscission layer and high or moderate spikelet shedding. The cultivated parents (T and J) showed no trace of an abscission layer and zero shedding.

The F2 and BCt or BCj segregated for different degrees of spikelet shedding (on a scale of 0 to 4) depending upon the presence of a well-formed

functional abscission layer and also on other factors such as the thickness of the pedicel. A precise quantitative measurement of spikelet shedding is difficult to achieve.

Mendelian analysis was limited to the presence of an abscission layer. It was possible to distinguish two classes without too much ambiguity:

— Presence of a clear functional abscission layer.
— No abscission layer or presence of a solid callus at the base of the pedicel.

In *Table 6,* the segregations for presence or absence of the abscission layer in the non-distorted families of both crosses are presented. Some simple observations should be emphasized: (1) Most of the segregations did not fit a one-gene model. (2) A constantly high negative correlation existed between spikelet shedding and LPI (from —0.53 to —0.87) (*Table 6*).

Tab. 6 Genetic analysis of the presence/absence of an abscission layer on involucral pedicels

Family	Generation	Correlation AB-LPI	Abscission +	Abscission –	Total	Theoretical segregation	Chi Square
			One-gene model				
V x T No 9	v-F2	-0.79	25	5	30	3 : 1	1.11
V x T No 10	t-F2		25	5	30	3 : 1	1.11
V x T No 17	t-F1 x T	-0.65	7	9	16	1 : 1	0.25
V x T No 18	T x t-F1		15	24	39	1 : 1	2.07
M x J No 11	m-F2	-0.87	25	5	30	3 : 1	1.11
M x J No 14	m-F1 x J	-0.73	11	19	30	1 : 1	2.13
			Cumulative two-gene model				
V x T No 8	v-F2	-0.73	29	13	42	10 : 6	0.34
V x T No 11	t-F2		16	13	29	10 : 6	0.66
V x T No 12	v-F2	-0.60	30	17	47	10 : 6	0.03
V x T No 13	t-F2	-0.69	30	18	48	10 : 6	0.03
V x T No 14	v-F1 x T	-0.65	9	33	42	1 : 3	0.28
V x T No 15	T x v-F1		11	30	41	1 : 3	0.07
V x T No 16	v-F1 x T	-0.65	6	28	34	1 : 3	0.98
V x T No 19	T x v-F1		12	24	36	1 : 3	1.33
V x T No 21	v-F1 x T	-0.53	15	33	48	1 : 3	1.00
M x J No 3	j-F2	-0.78	14	11	25	10 : 6	0.44

\+ Evident functional abscission layer
\- Abscission layer absent or aborted

The second observation was consistent with the fact that the presence of an abscission layer has never been observed by us when the involucral pedicel was much longer than 2 mm. This was true for hundreds of V×T and M×J segregants, as well as for hundreds of cultivated plants of many different cultivars (some cultivars, such as 'Zongo' from Niger, have short pedicels and high shedding). Thus, the association of a long pedicel with shedding seems to be a physiological impossibility.

The simplest explanation is that the major LPI gene (A, a) and the minor LPI gene (B, b) act pleiotropically on spikelet shedding. The following model of two cumulative genes gives theoretical segregations that fit the observations well (*Table 6*):

A— B— }
AA bb } presence of abscission (+)

Aa bb }
aa — } no abscission (—)

which gives in F2: 10 (+) 6 (—) and in BCt or j: 1 (+) 3 (—).
The cumulative action is expressed in the fact that two "strong" A alleles or 1A + 1B can produce a functional abscission layer but 2 "minor" B alleles cannot.

Biometrical analysis

Heterosis and reciprocal effects were evaluated for both crosses in homogeneous families. No significant reciprocal effect was detectable in the F1's of both crosses for all the seven continuous variables DR, LPI, LS, AR, LGLI, LG and DG (*Table 7*), although the existence of cytoplasmic differences between the parents was already demonstrated in a previous paper by phenomenons of male sterility (MARCHAIS and PERNÈS 1984). Heterosis in the direc-

Tab. 7 Biometric characteristics of homogeneous families from the *P. violaceum* x 'Tiotande' cross and the *P. mollissimum* x J 104 cross

V x T cross.	DR	LPI	LS	AR	LGLI	LG	DG
Means							
(V4 x V6) 1.2.1.2.10.	7.6	1.8	77.1	80.0	56.7	22.4	12.3
T5.5.4.3.1.5.5.	32.6	49.1	46.3	17.3	38.5	32.3	22.5
vF1 (see no 12)	11.0	4.8	85.0	79.2	53.1	30.3	19.0
tF1 (see no 13)	11.3	5.0	83.3	90.0	54.9	30.4	19.3
Heterosis							
(F1-V)/(T-V)	0.14**	0.06**	-0.23**	-0.07**	0.15**	0.60*	0.67
Number of genes							
F2	4.7	1.1	0.5	0.2	2.4	2.2	2.6
BC	5.3	1.4	3.6	2.0	4.0	3.7	5.0
M x J cross.							
Means							
M14.5.6.1.	8.7	2.4	98.3	10.0	58.1	21.2	12.9
J.2.3.1.6.	17.9	34.9	44.1	0.0	33.8	26.1	21.9
mF1	15.0	10.3	78.3	6.6	50.0	27.7	17.7
jF1	13.4	8.9	76.7	6.6	50.7	27.3	17.6
Heterosis							
(F1-M)/(J-M)	0.59	0.22**	0.38*	0.34	0.32*	1.26*	0.52

*(**) = heterosis significant at 5 % (1 %) level.

tion of the wild parent was expressed for DR, LPI, LS, AR and LGLI, indicative of the dominance of the corresponding wild genes, but seed size showed a slight dominance of cultivated genes.

As a general rule, F2 and BC generations exhibited continuous variation without transgressive segregants, for all characters. This simple observation corroborates the genetic hypothesis that, in a wild × cultivated cross, the increasing and decreasing alleles are isodirectionally distributed. Furthermore, this continuous array of segregants would imply that the three botanical groups, wild, intermediate and cultivated, are not three biologically distinct entities.

The number of genes was estimated from V×T families only. For the BC estimation, the most variable BC was used: i.e., BCt for DR, LPI, LS, AR and LGLI, and BCv for LG and DG. Although this method of estimation based on variances is not very reliable, in the case of LPI it was in good agreement with the previous analysis and proposed control by 1 major and 1 minor gene (N = 1, 1 or 1, 4). DR would be controlled by about 5 genes. This fits well with the fact that in BCt (20, 21) only 2 plants had a DR value similar to 'Tiotandé' out of a total of 96 plants. It also agrees with the estimation by BILQUEZ and LECOMTE (1969) of the number of genes controlling stem diameter (correlated to DR). For the other characters, it seems that at least two genes are involved.

Association of characters

Principal component analysis was performed on the material used for the biometrical analysis. The main axes of variation were computed for the 95 F2 individuals. The other generations (V, T, F1, BCt and BCv) were included as additional elements in this system of axes.

The F2 correlation matrix with the 1 % level significant correlations underlined is shown in *Table 8*. Two groups of characters are evident: (DR—LPI—LG—DG) and (LS—AR—LGLI). These associations produced a rapid decrease of eigenvalues (40.5 %, 29 %, 10.5 %, 8 %, 6 %, 3.4 %, 2.5 %) in-

Tab. 8 Correlations between biometric characters measured on 95 individuals of F2 families No 12 and 13, from the *P. violaceum* x 'Tiotande' cross

	DR	LPI	LS	AR	LGLI	LG	DG
DR	1,00	<u>0,30</u>	−0,17	0,00	−0,15	<u>0,38</u>	<u>0,55</u>
LPI	<u>0,30</u>	1,00	0,17	0,06	0,02	<u>0,38</u>	<u>0,46</u>
LS	−0,17	0,17	1,00	<u>0,73</u>	<u>0,63</u>	0,20	<u>0,26</u>
AR	0,00	0,06	<u>0,73</u>	1,00	<u>0,57</u>	0,23	<u>0,28</u>
LGLI	−0,15	0,02	<u>0,63</u>	<u>0,57</u>	1,00	0,18	0,21
LG	<u>0,38</u>	<u>0,38</u>	0,20	0,23	0,18	1,00	<u>0,76</u>
DG	<u>0,55</u>	<u>0,46</u>	<u>0,26</u>	<u>0,28</u>	0,21	<u>0,76</u>	1,00

Correlations significant at 1 % level are underlined

dicative of a partly common localization of the genes controlling these diverse characters on the same chromosomal segments. The linear combinations defining the first two components, C1 and C2, also showed the existence of two practically independent groups of characters:

C1 = —0.37DR—0.51LPI—0.68LS—0.68AR—0.58LGLI—0.73LG—0.80DG
C2 = 0.70DR + 0.41LPI—0.61LS—0.53AR—0.60LGLI + 0.40LG + 0.44DG

The second axis represented the gradient from the wild to the cultivated type. Its variance was not significantly different from the variance of axis 1. *Figure 3* shows the 95 % confidence level ellipsoids of the 6 generations on the plane defined by axes 1 and 2. The quasi-independence of the two groups of traits gave almost circular ellipses for F2, BCt and BCv. The intersection of the T and BCt ellipses was very small, which means that the recovery in BCt of full T — like plants was not a frequent event. The means and variances of the C2 component for F1, BCt and T allowed the estimation of the number of genes involved in the C2 segregation. This number amounted to 9.4 genes and measures the global divergence between V and T. The M×J cross was analysed similarly. It showed the same associations of characters and had the same two first axes. The distance (M—J) on axis 2 corresponded to 6.8 genes. J104 would have to be considered to be a little less domesticated than 'Tiotandé' in as much as *P. mollissimum* can be considered to be very close to *P. violaceum* (see their respective means in *Table 7*).

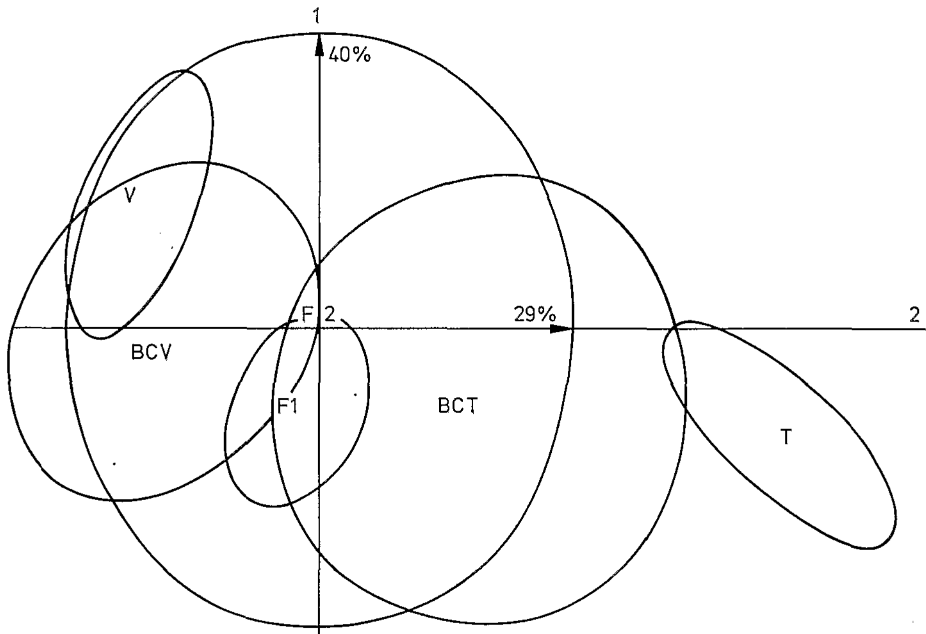

Fig. 3 Principal component analysis. Projection of confidence ellipsoïdes (95 % level) for the six V × T generations onto the plane formed by the first two axes calculated from the F2. The symbol of each generation is indicated at the centre of its corresponding ellipsoïd

Conclusion

The genetic analysis of characters of domestication in pearl millet leads to a better understanding of the nature and the intensity of the genetic modifications brought about by its domestication. The non-Mendelian segregations frequently observed are most probably due to pollen competition, because the phenomenon occurs after pollen shedding and before the germination of seeds. Pollen competition was observed *in vitro* between cultivated millets by SARR et al. (1983). The new fact here is the advantage systematically held by some wild genes over cultivated ones in pollen competition. This competition, associated with the male sterility created by the transfer of some cultivated genotypes into some wild cytoplasms (MARCHAIS and PERNÈS 1984) may be considered to be a slight reproductive barrier between wild and cultivated forms of pearl millet, protecting wild forms against a massive introgression of cultivated genes. The superiority of wild genes in pollen competition and in male fertility restoration (MARCHAIS and PERNÈS 1984) also means that the domestication process would have lead to a general loss of vigour in the cultivated form. Today wild genes for certation could be a great help to breeders if they afford an agronomic advantage for the sporophytic offspring, as has been reported in other plants (MULCAHY et al. 1978, OTTAVIANO et al. 1982). Non-Mendelian segregations also represent a hazard for the plant breeder in managing crosses and selection processes.

Putting aside the problems posed by the barriers of male sterility and pollen competition, the information gained in this study upon the genetics of some characters of domestication affords the following comments:

— A complete array of intermediate phenotypes can be created between the wild and the cultivated types: no discontinuity was observed. Wild genes seem to work with cultivated genes without any particular physiological problems: no monstruous plant was observed. Therefore, wild and cultivated forms must be taken to be members of the same biological species. The breeder can introgress wild germplasm into his cultivated material.

— The number of independent factors involved in the domestication process seems sufficiently high to think that this process was a long and gradual one and today is more or less advanced according to the cultivar studied. Indeed, at Neolithic times, managing about ten independent loci with recessive mutant genes should not have been a simple and rapid operation. In parallel, the high polymorphism created by domestication is easier to understand if we hypothesize many independent domestications with different orientations. That opinion fits well with the idea of a non-center of origin for pearl millet proposed by HARLAN (1971).

Finally, this study adds new information about the genetics of pearl millet. The heredity of spikelet shedding and of the length of the involucral pedicel are explained by a set of two pleiotropic genes. The diameter of the spike rachis involves about 5 genes. A linkage group was found that includes genes for esterase, G.O.T., limb pubescence and length of the involucral

pedicel. This linkage group, frequently subjected to non-Mendelian segregations and involved in the control of spikelet shedding, seems to play a prominent role in the genetics of domestication. Wild forms appear to be a useful tool for the genetic analysis of quantitative morphological characters because they occupy one extremity of the range of variation in the whole species. Our research shows the utility of enzyme markers for detecting the LPI major gene, thereby illustrating the view of TANKSLEY et al. (1982) that enzyme markers can help in the analysis of quantitative characters.

The conclusions presented here were drawn from observations on a limited number of morphological characters in only two crosses. The analysis of other wild/cultivated couples chosen for contrasting morphologies and geographical origins would fill out the information already acquired and permit testing its generality.

Zusammenfassung

Genetische Unterschiede zwischen Wild- und Kultur-Negerhirsen (*Pennisetum typhoides*)

II. Eigenschaften der Domestikation

Morphologische Eigenschaften, die direkt als Folge der Domestikation von Negerhirse auftreten, wurden mittels zweier Kreuzungen von Wild- und Kultur-Negerhirse genetisch analysiert. In früheren Untersuchungen hatten sich diese Kreuzungen als Quellen für männliche Sterilität erwiesen. Mit Hilfe von Markierungsgenen wurden in zahlreichen Fällen abweichende Aufspaltungen festgestellt, die immer einen Überschuß von Wildeigenschaften zeigten. Dieses Phänomen war vermutlich durch Pollen-Konkurrenz bedingt. Es wurden keine reziproken Effekte in den F_1-Generationen beider Kreuzungen für quantitative Merkmale gefunden. Zwischen den Extremen Wild- und Kulturphänotypen wurde eine kontinuierliche Variation beobachtet, wobei keine sichtbaren physiologischen Störungen auftraten. Das Zerfallen der Ährchen und die Länge des Stiels des Hüllkelchs scheinen durch das gleiche pleiotrope Gensystem kontrolliert zu werden (ein Majorgen und ein Minorgen). Der Durchmesser der Ährenspindel wird durch eine relativ große Zahl von Genen kontrolliert (etwa fünf unabhängige Gene). Es wurde eine Kopplungsgruppe gefunden, die Gene für Esterase, Glutamat-Oxalo-acetat-Transaminase, Blattrandbehaarung und das Majorgen für die Stiellänge des Hüllkelchs einschließt. Insgesamt scheinen an der morphologischen Abweichung zwischen Wild- und Kulturformen bis zu zehn unabhängige Gene beteiligt gewesen zu sein. Die Domestikation der Negerhirse wird als ein langsamer, stufenweiser, sich in mehrere Richtungen entwickelnder Prozeß mit vielfachen Ursprüngen verstanden.

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