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African-derived South American Populations: A History of Symmetrical and Asymmetrical Matings According to Sex Revealed by Bi- and Uni-parental Genetic Markers

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ABSTRACT Estimates of African, European, and Amerindian contributions to the gene pool of 11 predominantly African-derived South American populations were obtained using five autosomal and one Y chromosome hypervariable loci, as well as mitochondrial DNA (sequences of the first hypervariable segment of the control region, plus two restriction sites and the presence or absence of the CoII/tRNA^{Lys} intergenic 9-bp deletion). The three latter characteristics are reported here for the first time for 42 individuals living in three Brazilian populations. Thirty-eight sequences were identified in these persons; 17 (45%) could be classified as being of African, 4 (11%) of Amerindian, and 2 (5%) of European origin. Evidence for asymmetrical matings in relation to sex and ethnic group was obtained for nine of the 11 populations. The most consistent finding was the introduction of European genes through males, but the results differ in the several communities, indicating the importance of local factors in such interactions. Am. J. Hum. Biol. 11:551–563, 1999. © 1999 Wiley-Liss, Inc.

The first Africans were introduced into America about four years after the arrival of the European colonizers, and during the colonial slave trade period about nine million were forced to migrate to South America (Mattoso, 1982). Present-day African South American populations are the product of an original African gene pool that has received European and Amerindian genes for about 12 generations. The estimation of the proportion of Amerindian and European genes in rural and urban African-derived South American groups, as measured with classical genetic polymorphisms, has been the subject of considerable investigation due to its anthropological implications (Azevedo, 1980; Franco et al., 1982; Schneider et al., 1987; Bortolini et al., 1992, 1995a, 1995b, 1997a; Castro de Guerra et al., 1993, 1996). For instance, it was possible to quantify the amount of interethnic unions which had occurred in the past and to detect the intro-

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duction of foreign hidden alleles, thus providing details about the history of these populations.

Historical and sociological studies also indicate that at some time during the history of the contemporary South American populations there may have been substantial asymmetric frequencies of unions between individuals of different ethnic origins (Fernandes, 1965; Valle-Silva, 1991; Sans et al., unpublished data). The genetic impact of this phenomenon can, in principle, be accurately evaluated by a comparison of admixture estimates using bi- and uni-parental genetic markers. Previous investigations using this approach with American populations included Mexican Americans (Merriwether et al., 1997), Black North Americans (Hsieh and Sutton, 1992), and an African-derived South American group (Bravi et al., 1997; Sans et al., unpublished data).

In the present study, tri-hybrid admixture estimates are provided for three urban and eight rural African-derived South American populations considering data from mitochondrial DNA, five autosomal and one Y chromosome hypervariable loci. New mtDNA data are presented for 42 individuals from three of these populations. The admixture results were compared with those obtained in earlier investigations of classical genetic polymorphisms in these same groups, and the possibility of differential gene flow according to sex was considered. This report relates to a long-term program which aims at the interpretation of the general factors that underlie the genetic variability of urban and rural South American African-derived communities.

Fig. 1. Geographic location of the populations considered in the present study.

SUBJECTS AND METHODS

The African-derived urban South American populations investigated are (Fig. 1): (1) Porto Alegre, the capital of Brazil's southernmost state, Rio Grande do Sul (30°5′S; 51°10′W); (2) Salvador, the capital of the Brazilian northeastern state of Bahia (12°55′S; 38°29′W); and (3) Ribeirão Preto, located in the north of the state of São Paulo (21°12′S; 48°10′W). The African-derived rural South American populations studied are: (1) Cametá, in the region of the lower Tocantins River, State of Pará, Amazonian region, northern Brazil (2°3′S; 59°55′W); (2) Trombetas, at the margins of the Trombetas and Cuminá rivers, Amazonian region, state of Pará, northern Brazil (1°8′–1°46′S; 55°51′–57°W); (3) Cajueiro, located in the county of Alcantara, state of Maranhão, in northeastern Brazil (2°25′; 44°20′W); (4) Paredão, in the Porto Alegre metropolitan region, state of Rio Grande do Sul, southern Brazil (28°20′S; 50°90′W); and (5) Curiepe, (6) Birongo, (7) Sotillo, and (8) Panaquire, all four of which are situated in northern Venezuela along the north-central coast, state of Miranda (10°–10°50′N; 60°–66°22′W). With the exception of Panaquire, all of the rural groups were derived from descendants of fugitive slaves. Panaquire was founded by European colonizers, but evolved, for economic and historical reasons, mainly as a Black population due to migration of African-derived persons (Castro de Guerra et al., 1997). Additional information about these populations can be obtained in Schneider et al. (1987), Castro de Guerra et al. (1990, 1996, 1997), Bortolini (1991), Bortolini et al. (1992, 1995a, 1997a, 1998), and Zago et al. (1996).

Morphological classification concerning the occurrence of non-African ancestry in the subjects was made taking into consideration skin color and characteristics such as hair type and nose and lip shape. In Ribeirão Preto only those persons who presented no indications of admixture were chosen. In Porto Alegre and Salvador, how-

ever, those classified as admixed (Mulatto) were also included.

Table 1 provides information about the genetic data sets used in this study. The weighted average allele frequencies of each ancestral stock for the hypervariable loci (D1S80, APOB, D4S43; VW-I, F13A1, and DYS19) were estimated using all of the information that could be assembled about them; these are furnished in the Appendix. For some comparisons, results from protein loci obtained in these populations were also used. This information, however, was extracted from already published sources (Bortolini et al., 1995a, 1997a, 1998; Castro de Guerra et al., 1996) without further analyses, and therefore, does not appear in Table 1.

The degree of admixture, using the two sets of hypervariable loci (autosomal: D1S80, APOB, D4S43; VW-I, F13A1, and Y chromosome DYS19), was calculated using the gene identity method (Chakraborty, 1985). The fit of this model can be evaluated in terms of R^2, that is, an expected multiple correlation coefficient of the allele frequencies in a hybrid population and those of the parental populations, under the assumption of a true admixture model (Chakraborty, 1986). The admixture values considering the DYS19 locus were also estimated using the weighted least squares method (Long and Smouse, 1983; Long, 1991a, b). In this case, the fit of the model can be evaluated in terms of the mean square error (MSE), which represents the proportion of allele frequency variation unexplained by the admixture model (Long, 1991b). The calculations were done using the ADMIX 3 and ADMIX programs, kindly made available by Drs. R. Chakraborty and J.C. Long, respectively. According to Chakraborty et al. (1991), the precision of the admixture estimates is directly dependent on the level of frequency differences between populations (δ) considering the marker used. Thus, the δ values were also calculated for each hypervariable locus according to the method described by Shriver et al. (1997).

For the mtDNA data set, the relative maternal contribution of their putative ancestors to the Afro-South American populations was estimated by two methods. First, each parental contribution was considered as being inversely proportional to the average number of nucleotide differences between the mtDNA lineages of the African-

TABLE 1. Genetic data used in this study[1]

Population		Hypervariable loci			Mitochondrial DNA				
Urban	Rural	Autosomal (D1S80, APOB, F13A1, D4S43, VW-I)	Y-Chrom. (DYS19)	Refer.[2]	Sequences (HVS-I)	Restriction enzime Hpa-I (3592)	Restriction enzime Hinf-I (10806)	9-bp-deletion	Refer.[2]
Porto Alegre		+	–	1	+	+	+	+	5,6
Salvador		–	–		+	+	+	+	5,6
Ribeirão Preto		+	+	2	+	+	+	+	7
	Cametá	+	+	3,4	+	+	+	+	7
	Trombetas	+	+	3,4	+	+	+	+	7
	Cajueiro	+	+	3,4	+	+	+	+	7
	Paredão	+	+	3,4	+	+	+	+	5,6
	Curiepe	+	+	3,4	–	–	–	–	
	Birongo	+	+	3,4	–	–	–	–	
	Sotillo	+	+	3,4	–	–	–	–	
	Panaquire	+	+	3,4	–	–	–	–	

[1]+, determined; –, not determined.
[2]References: (1) Data for the D1S80 locus only; Heidrich et al. (1995); (2) Zago et al. (1996); (3) Silva-Jr. et al. (unpublished); (4) Bortolini et al. (1998); (5) Bortolini et al. (1997b); (6) Present study; (7) Unpublished data.

derived populations and their ancestral groups (Pinto et al., 1996). The average number of nucleotide differences between populations was calculated according to Nei and Tajima (1981), while the data for parental groups was obtained for Africans from Graven et al. (1995) and Watson et al. (1997), for Europeans from Calafell et al. (1996), Pinto et al. (1996), and Richards et al. (1996), and for South Amerindians from Santos et al. (1996), Ward et al. (1996), and Bonatto and Salzano (1997). Second, an estimation of admixture was also made using the proportion of continent-specific haplogroups (of African, European, and Amerindian origins) present in the South American communities. The bibliographic sources were the same as those indicated above. For Africans, previous mtDNA studies have revealed that between 60% and 100% of the sub-Saharan African mtDNAs were characterized by a Hpa I site gain at nucleotide (nt) 3592, and they were classified as haplogroups *L1* and *L2* (Chen et al., 1995; Watson et al., 1997). *L1* is also defined by a Hinf I site gain at nt 10806, while *L2* lacks this site, and present a substitution of G to A at nt 16389. Recently, Watson et al. (1997) designated as *L3* the haplogroup lacking the Hpa I site at 3592, and identified sub-haplogroups in *L1* (*L1a, L1b* and *L1i*) and *L3* (*L3a, L3b* and *L3c*) based on substitutions in the first hypervariable segment of the mtDNA control region (HVS-I) relative to the reference sequence (Anderson et al., 1981). In sub-Saharan populations *L1, L2,* and *L3,* with their respective sub-haplogroups, have been observed (Chen et al., 1995; Watson et al., 1997). However, mtDNAs characterized by the Hpa I site at nt 3592 are found at very low frequencies outside Africa, suggesting that only African mtDNAs without this site were carried from Africa by *Homo sapiens sapiens* (Chen et al., 1995). Furthermore, the CoII/tRNA^{Lys} intergenic 9-bp deletion in Africans is associated with at least two sub-haplogroups of *L1* (Soodyall et al., 1996; Watson et al., 1997), while in Amerindian/Asians it occurs with haplogroup *B* (characterized by this deletion, and by T to C transitions at nucleotide positions 16189 and 16217). This mutation has also been described in European lineages, but with a different background (Torroni et al., 1995).

For some of the populations studied, data were not available either on mtDNA or the DYS19 marker. In these cases, the formulae proposed by Hsieh and Sutton (1992) were used to calculate the female and male contributions when just one type of uniparental and biparental markers are available.

For 42 individuals from three populations (Porto Alegre, Salvador, and Paredão), data are presented for the Hpa I (nt 3592) and Hinf (nt 10806) sites, as well as for the 9-bp deletion. DNA was extracted as previously described (Bortolini et al., 1997b), and the entire mtDNA of these samples was amplified in two fragments by PCR using the primer pairs and amplification conditions described in Torroni et al. (1992, 1993). Each PCR product was subsequently digested with the two indicated restriction enzymes. The resulting fragments were separated by 2%–4% agarose gel electrophoresis and visualized by UV fluorescence. Additionally, the samples were searched for the presence or absence of the CoII/tRNA^{Lys} intergenic 9-bp deletion, using the primers and PCR conditions described in Bortolini et al. (1997b).

RESULTS

Table 2 shows the 38 mtDNA sequences present in the 42 individuals defined by HVS-I and the three other mtDNA regions. In the sample as a whole, three sub-haplogroups of *L1* (*L1a, L1b,* and *L1i*) were identified *L1a* (sequences 1, 2, and 3), have been previously found in Blacks from eastern, central, and southern Africa, and in Pygmies, although single examples were also observed in some West African and Middle East populations (Calaffel et al., 1996; Watson et al., 1997; Graven et al., 1995). However, a subset of *L1a,* defined by the 9-bp deletion (sequence 3), may represent a Bantu marker, while *L1b* (sequence 8) could be defined as a specific West African marker, since it was described in people living in this African region only (Watson et al., 1997). Although sequences 4 and 6 present some undescribed substitutions in HVS-I (16184 C→G, 16265 A→C; and 16260 C→T, 16265 A→C, 16286 C→A), they also show other mutations observed earlier in African "isolated lineages," which characterize the sub-haplogroup *L1i* (Watson et al., 1997). According to these authors, "isolated lineages," which are not shared between African populations, could represent relics of a less dramatic and more ancient expansion event across Africa.

TABLE 2. Mitochondrial sequence haplotypes and their distributions in 42 African-derived individuals[1]

No.	Sequence lineages variant sites[2,3]	Hpa I (nt 3592)[4]	Hinf I (nt 10806)[4]	9 bp-del. (CoII/ tRNA^{Lys})[4]	Pop.[5]	Morphol. classif.[6]	Probable haplogroup	Probable origin
1	93C,129A,148T, 168T,172C,187T, 188G,189C, 223T,230G, 278T,293G, 311C,320T	+	+	–	POA	B/M	L1a	African
2	129A,148T,168T, 172C,187T, 188G,189C, 223T,278T,298C, 311C,320T	+	+	–	POA	M	L1a	African
3	148T,172C,187T, 188G,189C, 223T,230G, 311C,320T	+	+	+	POA	M	L1a	African
4	80G,184G,187T, 189C,223T, 265C,294T, 311C,320T	+	+	–	POA	B	L1i	African
5	187T,189C,223T, 265C,278T, 286G,294T, 311C,360T	+	–	–	POA	B	L2	African
6	129A,187T,189C, 223T,260T,265C, 278T,286A,294T, 311C,360T	+	+	–	POA	M	L1i	African
7	51G,114T,187T, 189C,223T,293T, 311C,316G, 355T,362C	–	–	–	POA	M	L3	Undetermined
8	126C,187T,189C, 223T,264T,270T, 278T,293G,311C	+	+	–	POA	B	L1b	African
9	173T,189C,223T	–	–	+	POA	B	?	Undetermined
10	189C,223T,294T	+	–	–	POA	B	L2	African
11	172C,189C,192T, 223T,320T	–	–	–	POA	M	L3	Undetermined
12	172C,189C,223T, 320T	+	–	ND/–	SAL/POA	M/M	L2	African
13	172C,189C,223T, 320T	–	–	–	POA	B	L3	Undetermined
14	172C,189C,209C, 223T	–	–	–	POA	M	L3	Undertermined
15	172C,189C,223T	–	–	–	SAL	B	L3	Undetermined
16	172C,189C,217C	–	–	+	POA	M	B	Amerindian
17	93T,172C,189C, 223T	–	–	–	POA	M	L3	Undetermined
18	51G,172C,223T	–	–	–	PAR	ND	L3	Undetermined
19	126C,209C,223T, 298C,325C,328T	–	–	–	SAL	B	C	Amerindian
20	172C,223T,298C, 325C,327T	–	–	–	PAR	ND	C	Amerindian
21	223T,278T,286T, 294T,309G	+	–	–	POA	M	L2	African
22	223T,298C,325C, 327T,362C	–	–	–	POA	M	C	Amerindian
23	148T,223T,320T	–	–	–	SAL	B	L3	Undetermined
24	223T,265T	–	–	–	POA/PAR	B/ND	L3	Undetermined
25	124C,150T,223T, 278T,360T,362C	–	–	–	POA	B	L3b	Undetermined
26	124C,223T,278T, 311C,362C	–	–	–	POA	M	L3b	Undetermined
27	223T,278T,290T	+	–	–	SAL	B	L2	African

(continued)

TABLE 2. Mitochondrial sequence haplotypes and their distributions in 42 African-derived individuals (Continued)

No.	Sequence lineages variant sites[2,3]	Hpa I (nt 3592)[4]	Hinf I (nt 10806)[4]	9 bp-del. (CoII/ tRNA^{Lys})[4]	Pop.[5]	Morphol. classif.[6]	Probable haplogroup	Probable origin
28	114A,129A,223T, 278T,354T	+	–	–	POA	B	L2	African
29	114A,129A,213A, 223T,278T	+	–	–	POA	M	L2	African
30	129A,223T,294T	+	–	–	POA	B	L2	African
31	129A,223T,278T, 294T	+	–	–	SAL	B	L2	African
32	223T,278T,286T, 294T,301T	+	–	–	SAL	B	L2	African
33	193C,213A,223T, 239T,278T,294T, 330C	+	–	–	PAR	ND	L2	African
34	223T,311C	–	–	–	POA	M	L3a	Undetermined
35	129A,209C,223T, 292T,295T,311C	–	–	–	POA	B	L3a	Undetermined
36	187T,298C,311C	–	–	–	POA	M	L3	Undetermined
37	293G,311C	–	–	–	SAL	M	H	European
38	358A	–	–	–	SAL	B	H	European

[1]ND: Not determined. Note: Sequences no. 1, 12, and 24 occur in two individuals. All the others were detected in one person only.
[2]Described in terms of variant positions, relative to the reference sequence (Anderson et al., 1981), in the HVS-I. Position numbers are given without the prefix 16.
[3]Data compiled from Bortolini et al. (1997b).
[4]+: Presence; –: absence of the restriction site and of the 9-bp deletion.
[5]Key to the population names: POA: Porto Alegre; SAL: Salvador; PAR: Paredão.
[6]Morphological classification: B: Black without signs of admixture; M: Mulatto.

Sequences 5, 10, 12, 21, and 27–34 present mutations that characterize *L2*. Haplogroup *L3,* lacking the Hpa I site at nt 3592, was identified in 16 sequences. Some of these could be classified into subhaplogroups *L3a* (sequences 34 and 35) and *L3b* (25 and 26). In Africa, *L3a* occurs mainly in the east, but this haplogroup is also found in European and Asian mtDNAs. As a matter of fact, sequence types with motif 16223 (T), 16292 (T), and 16295 (T) (which in our study characterized sequence 35) have been observed in European mtDNAs (Richards et al., 1996). Represented by sequences 25 and 26, *L3b,* characterized by the nt 16124 (C), 16223 (T), and 16278 (T) motif, is found in southwestern Africa and it is also widespread in western Africans. On the other hand, sequences 37 and 38 lacked the Hpa I 3592 site but, due to the presence of other mutations, could be identified as belonging to European haplogroup *H* (Torroni et al., 1994).Sequence 16, and sequences 19, 20, and 22 could be assigned to the Amerindian haplogroups *B* and *C,* respectively (Torroni et al., 1993).

Sequence 9, which lacks the Hpa I nt 3592 and Hinf I nt 10806 sites, and presents transitions at nt 16173 (T), 16189 (C), and 16223 (T) in HVS-I, also shows the 9-bp deletion between the COII and tRNA^{Lys} genes. No African, European, or Amerindian mtDNAs with the 9-bp deletion present this combination of markers. This suggests two possibilities. First, this sequence represents an African, European, or Amerindian mtDNA with the 9-bp deletion, that through subsequent mutational episodes created new markers or abolished the pre-existing primary markers in HVS-I. Or second, it is a sequence of undetermined origin that gained the 9-bp deletion in a new mutational event. Considering only the number of mutational occurrences necessary in the two alternatives, it is more probable that the second suggestion is the correct one.

Sequences 31, 32, and 33 present the two substitutions in HVS-I, namely 16223 (T) and 16278 (T) that characterize a supposed additional Amerindian founder haplogroup (Forster et al., 1996; Ribeiro-dos-Santos et al., 1996). However, sequences with this motif have been recently observed in Africans (Watson et al., 1997). Furthermore, the presence of the Hpa I 3592 site in these sequences reinforce the idea that these are of African rather than Amerindian origin. All of the other sequences are non-specific, since they are present in Africa, Europe, or Asia.

In summary, of the 38 sequences, 17 (45%) could be classified as being of African,

TABLE 3. Frequency differences (δ × 100) for five autosomal and one Y hypervariable loci considering the three parental stocks

	Comparison			
Loci	African/European	African/Amerindian	European/Amerindian	Mean per locus
F13A1	74.8	39.9	75.3	63.0
D1S80	56.3	73.1	50.0	59.8
D4S43	63.7	40.4	41.1	48.4
APO B	36.4	58.1	42.2	46.5
VWF-I	21.0	36.4	32.2	29.9
Average	50.4	49.4	48.2	49.5
DYS19	39.9	74.4	63.3	59.2

4 (11%) of Amerindian, and 2 (5%) of European origins. No clear association between these results and the morphological classification of the individuals was evident.

The δ values calculated for the six hypervariable loci are presented in Table 3. F13A1 presents the highest average frequency differences between ethnic groups (63%), while VW-I shows the lowest (30%). Considering all of the autosomal loci and the DYS19 locus, the values obtained (about 50% and 60%, respectively) indicate that these markers should be accurate as population-admixture estimators (Shriver et al., 1997).

Table 4 provides the calculated admixtures using the five autosomal hypervariable loci and relates them to previous estimates obtained with protein markers. The present estimates concerning the African contribution vary from 49% for Cajueiro and Paredão to 73% for Ribeirão Preto; the corresponding range for European admixture is 15% (Birongo) to 46% (Porto Alegre), while for Amerindian ancestry is from zero (Porto Alegre) to 26% (Panaquire). All R^2 are higher than 97%, indicating that an evolutionary model based mainly on admixture provides a reasonable explanation for the genetic structure of these African-derived populations, at least considering these hypervariable loci.

Table 4 also shows the admixture values obtained in earlier studies for the same African-derived South American populations using classical genetic polymorphisms. Two features of this table should be emphasized first, in both the hypervariable and protein sets, the African genetic component predominates with the exception of the Porto Alegre data based on the latter data set. Second, despite some differences in the relative contributions of each ethnic group as measured by the two sets of genetics markers (African component of Paredão, 31% discrepancy; Amerindian fraction of Cajueiro, 25%; European contribution to Birongo, 22%), the average differences for each ethnic group is typically only 10% (African, 11%; European, 10%; Amerindian, 9%).

Admixture estimates considering the two uniparental genetic markers are given in Table 5. Examining first the mtDNA data, the African contribution obtained using the average number of nucleotide differences method (Pinto et al., 1996), vary from 58% (Paredão) to 90% (Salvador); the values for European and Amerindian contributions occur in the range of 5% (Salvador) to 17% (Carmetá), and of 5% (Salvador) to 27% (Paredão), respectively. Admixture estimates based on the counting of specific mtDNA haplogroups is also given in Table 5. Due to uncertainties concerning the origin of some sequences, intervals of possibilities are given. For example, 41% of the sequences from Porto Alegre are of undetermined origin. Thus, the European contribution to the mtDNA gene pool of the hybrid population could be 41% if all of the unclassified sequences had an European origin, or zero if all were of non-European sources. On the other hand, when the results obtained employing the two methods (using the average of the extreme values in method 2) are compared, the largest discrepancy is in the values for Cametá (59% and 40%, 17% and 0%; 24% and 60%, for the African, European, and Amerindian contributions, respectively). The numbers obtained from the average nucleotide differences method may not reflect the real ancestral proportions due to parallel mutational events or reversals (homoplasy). For example, the transition C→T at position 16223 is present in the Amerindian/Asian C haplogroup, and also in the L1 and L2 African haplogroups (Wallace, 1995; Watson et al., 1997).

Table 5 also gives the admixture proportions considering one Y chromosome marker

TABLE 4. *Admixture estimates for 11 African-derived South American populations considering two sets of the biparental genetic markers*[1]

Populations		Genetic markers									
		Autosomal hypervariable loci				Protein loci					
Urban	Rural	African	European	Amerindian	R^2	African	European	Amerindian	R^2/MSE	Method[2]	Refer.[3]
Porto Alegre		53.6 ± 0	46.4 ± 0	0	99.9	41.0 ± 4.7	59.0 ± 4.7	0	88/	1	1
Salvador		ND	ND	ND		58.0 ± 0.6	38.0 ± 0.4	4.0 ± 0.5	99/	1	1
Riberão Preto		73.1 ± 1.6	21.8 ± 1.0	5.1 ± 1.9	99.9	ND	ND	ND			
	Cametá	53.4 ± 8.1	24.2 ± 2.1	22.4 ± 8.2	98.8	48.0 ± 0.7	17.9 ± 0.6	34.1 ± 0.7	/2.7	2	2
	Trombetas	57.6 ± 7.2	32.5 ± 3.2	9.9 ± 6.2	99.8	56.4 ± 12.6	23.8 ± 11.0	19.8 ± 12.5	/3.1	2	2
	Cajueiro	48.8 ± 6.8	26.2 ± 3.9	25.0 ± 7.5	97.2	67.4 ± 15.3	32.6 ± 15.3	0	/2.3	2	3
	Paredão	49.2 ± 1.8	38.0 ± 0.9	12.8 ± 1.2	99.9	80.2 ± 11.0	19.8 ± 12.3	0	/9.7	2	2
	Curiepe	70.4 ± 0.4	23.2 ± 0.4	6.4 ± 0.4	99.9	64.8 ± 13.6	35.2 ± 13.6	0	/4.2	2	3
	Birongo	60.1 ± 2.7	15.3 ± 3.0	24.6 ± 3.1	99.6	45.6 ± 12.7	37.5 ± 18.0	16.9 ± 19.6	/3.4	2	3
	Sotillo	61.0 ± 6.4	25.6 ± 4.3	13.4 ± 8.3	97.8	53.7 ± 13.7	24.7 ± 16.6	21.6 ± 19.1	/3.6	2	3
	Panaquire	54.6 ± 0.7	19.3 ± 0.3	26.1 ± 0.9	99.9	59.0 ± 3.2	15.0 ± 2.0	26.0 ± 2.6	ND	1	4

[1]ND, not determined.
[2]Methods: (1) Gene identity (Chakraborty 1985); (2) Weighted least-squares (Long and Smouse, 1983; Long, 1991 a,b). R^2 and MSE indicate the fit of the data to a strict admixture model.
[3]References: (1) Bortolini et al. (1997a); (2) Bortolini et al. (1995a); (3) Bortolini et al. (1998); (4) Castro de Guerra et al. (1996).

(DYS19). The values obtained with the two methods are similar, showing that the African contribution is the majority in Ribeirão Preto, Trombetas, Cajueiro, and Paredão, while the European ancestry is larger for Cametá, Curiepe, Birongo, Sotillo, and Panaquire. Again, the R^2/MSE statistics indicate a good adjustment of the gene frequency variations in these populations to the postulated tri-hybrid model. However, for Paredão this is not true; 31% to 33% of the DYS19 gene frequencies could be due to drift and/or to sampling error in the estimation of parental frequencies.

Applying the formulae proposed by Hsieh and Sutton (1992), the African female contributions (boxed, Table 5) for the four Venezuelan groups vary from 100% (Curiepe and Birongo) to 44% (Sotillo); the numbers for the Amerindian ancestry diverge in a complementary manner. There is no indication of European female contributions for these populations. On the other hand, for Porto Alegre and Salvador, the European male contribution is majority, while the Amerindian male component is not detectable.

DISCUSSION

Comparison of the admixture components for the four types of markers show different and interesting patterns for each population.

Porto Alegre

The data from biparental markers reveal that the presence of African and European genes is approximately the same in the nuclear gene pool of this population. The results presented in Table 5, however, suggest an asymmetry, with African females and European males furnishing larger contributions than the others for the formation of this population. It is known that during colonial times, the shortage of White women favored a higher rate of unions between White men and Black or Indian women. On the other hand, the presence, in lower frequencies, of European markers in the mtDNA and African markers in the paternal estimate may indicate the recent reversal of the historical trend, with African-derived males looking for European-derived females as mates (Fernandes, 1965; Valle-Silva, 1991). The two Amerindian mtDNA sequences detected, on the other hand, indicate that Amerindian women may have con-

TABLE 5. Estimates of admixture (× 100) for 11 African-derived populations using two uniparental genetic markers

Populations		Genetic markers								
		mtDNA				Y Chromosome (DYS19)				
Urban	Rural	African[1]	European[1]	Amerindian[1]	Method[2]	African[1]	European[1]	Amerindian[1]	R2 MSE	Method[2]
Porto Alegre		75	11	14	(1)	20	80	0	–	–
		52–93	0–41	7	(2)					
Salvador		90	5	5	(1)	55	45	0	–	–
		45–67	22–44	11	(2)					
Ribeirão Preto		82	8	10	(1)	96 ± 4	4 ± 4	0	99	(3)
		60–95	5–40	0	(2)	94 ± 7	2 ± 7	4 ± 3	0.5	(4)
	Cametá	59	17	24	(1)	28 ± 0	54 ± 0	18 ± 0	100	(3)
		40	0	60	(2)	34 ± 8	48 ± 9	20 ± 4	0.5	(4)
	Trombetas	80	7	13	(1)	84 ± 1	13 ± 1	3 ± 0	100	(3)
		50–90	0–40	10	(2)	80 ± 5	8 ± 6	11 ± 3	0.3	(4)
	Cajueiro	59	17	24	(1)	78 ± 22	24 ± 22	0	86	(3)
		50–70	0–20	30	(2)	71 ± 21	27 ± 23	2 ± 7	4	(4)
	Paredão	58	15	27	(1)	65 ± 32	35 ± 32	0	67	(3)
		20–80	0–60	20	(2)	68 ± 64	32 ± 68	0	31	(4)
	Curiepe	100	0	0	–	0	80 ± 3	20 ± 3	99	(3)
						2 ± 50	81 ± 61	17 ± 27	18	(4)
	Birongo	100	0	0	–	0	93 ± 2	7 ± 2	99	(3)
						2 ± 26	93 ± 29	5 ± 10	4	(4)
	Sotillo	44	0	56	–	49 ± 10	51 ± 0	0	92	(3)
						36 ± 23	50 ± 27	14 ± 11	4	(4)
	Panaquire	75	0	25	–	0	62 ± 3	38 ± 3	99	(3)
						0	69 ± 26	31 ± 13	3	(4)

[1]Data about Sub-Saharan African, European and Amerindian populations were compiled from earlier studies (see Subjects and Methods section).
[2]Methods: (1) The ancestral contribution was considered as being inversely proportional to the average number of nucleotide substitutions between the African-derived populations and each parental group (Pinto et al., 1996); (2) The admixture proportions were obtained by direct observation of specific-ethnic mtDNA haplogroups; (3) Gene identity (Chakraborty, 1985); (4) Weighted least squares (Long and Smouse, 1983; Long, 1991a, b).
Within boxes: The proportion (%) of male and female contributions to Porto Alegre, Salvador, Curiepe, Birongo, Sotillo and Panaquire calculated according to Hsieh and Sutton (1992).

tributed in a small scale to the gene pool of this population.

Salvador

Whereas the Amerindian contribution seems to be exclusively due to females, the African and European estimates suggest a similar participation of males and females.

Ribeirão Preto

This sample is different from the others because only Blacks without morphological signs of admixture were included. No indications of asymmetrical mating were found.

Cametá

The large Amerindian component in the gene pool of this population is expected, since it is located in the Amazonian region, where the significant influence of Brazilian Indians is well recognized (Santos and Guerreiro, 1995). There is, however, a high difference (36%) in the estimates of this contribution when the two methods based on the mtDNA data are considered. The European fraction seems to have been mainly furnished by males.

Trombetas

No evidence for asymmetrical matings in relation to sex and ethnic groups could be found.

Cajueiro

The results are somewhat inconclusive due to the large difference (25%) in relation to the Amerindian contribution provided by the two types of biparental sets of systems. But, there are indications that most of this contribution should have derived from females.

Paredão

The Amerindian contribution seems to be due exclusively to females. This can represent remote admixture, since the last reference about Indian presence in this region is reported to the nineteenth century (Bortolini, 1991).

Curiepe and Birongo

Important asymmetrical matings seem to have occurred in these localities. The African presence in the gene pool of these populations seems to be due to an exclusive contribution of African females, while the European and Amerindian genes would have been introduced exclusively from males. Historical information indicates the existence of prejudice and prohibitions of matings between Amerindian women and African-derived men (Sagnes, 1955; Bastide, 1974), corroborating these findings.

Sotillo

Amerindian genes seem to have been introduced predominantly through female lineages, and European genes exclusively through males.

Panaquire

The African contribution seems to have derived exclusively from females, while the European fraction could be exclusively furnished by males.

In summary, evidence for asymmetrical matings in relation to sex and ethnic group for nine of 11 populations considered has been obtained. The exceptions are Ribeirão Preto and Trombetas. The most consistent finding is the introduction of European genes through males. This is generally explained by a combination of male aggressiveness associated with the privileged position of European men, especially in colonial times, in South America. But, the results differ when diverse populations are considered, pointing to the importance of local factors in such situations.

LITERATURE CITED

Anderson S, Bankier AT, Barrel BG, De Brujin MHL, Coulson AR, Drouin J, Eperon IC, Nierlich DP, Roe BA, Sanger F, Schreiner PH, Smith AJH, Staden R, Young IG. 1981. Sequence and organization of the human mitochondrial genome. Nature 290:457–474.

Azevedo E. 1980. Subgroup studies of Black admixture within a mixed population of Bahia, Brazil. Ann Hum Genet 44:55–60.

Bastide R. 1974. As América Negras. São Paulo: Editora da Universidade de São Paulo.

Boerwinkle E, Xiong W, Fourest E, Chan L. 1989. Rapid typing of tandemly repeat hypervariable loci by the polymerase chain reaction: Application to the apolipoprotein B3′ hypervariable region. Proc Natl Acad Sci USA 86:212–216.

Bonatto SL, Salzano FM. 1997. Diversity and age of the four major mtDNA haplogroups, and their implications for the peopling of the New World. Am J Hum Genet 61:1413–1423.

Bortolini MC. 1991. Estudos genéticos em comunidades negras semi-isoladas sul-americanas. M.Sc. thesis. Porto Alegre: Federal University of Rio Grande do Sul.

Bortolini MC, Weimer TA, Franco MHLP, Salzano FM, Layrisse Z, Schneider H, Schneider MPC, Harada ML. 1992. Genetic structure in three South American Black populations. Gene Geogr 6:1–16.

Bortolini MC, Weimer TA, Salzano FM, Callegari-Jacques SM, Schneider H, Layrisse Z, Bonatto SL. 1995a. Evolutionary relationships between Black South American and African populations. Hum Biol 67:547–559.

Bortolini MC, Castro de Guerra D, Salzano FM, Weimer TA. 1995b. Inter- and intrapopulational genetic diversity in Afro-Venezuelan and African populations. Interciencia 20:90–93.

Bortolini MC, Weimer TA, Salzano FM, Moura LB, Silva MCBO. 1997a. Genetic structure of two urban Afro-Brazilian populations. Int J Anthropol 12:5–16.

Bortolini MC, Zago MA, Salzano FM, Silva-Junior WA, Bonatto SL, Silva MCBO, Weimer TA. 1997b. Evolutionary and anthropological implications of mitochondrial DNA variation in African Brazilian populations. Hum Biol 69:141–159.

Bortolini MC, Silva-Junior WA, Weimer TA, Zago MA, Castro de Guerra D, Schneider MPC, Layrisse Z, Mendez Castellano H, Salzano FM. 1998. Protein and hypervariable tandem repeat diversity in eight African-derived South American populations: Inferred relationships do not coincide. Hum Biol 70:443–461.

Bravi C, Sans M, Bailliet G, Martinez-Marignac VL, Portas M, Barreto I, Bonilla C, Bianchi NO. 1997. Characterization of mitochondrial and Y-chromosome haplotypes in a Uruguayan population of African ancestry. Hum Biol 69:641–652.

Calafell F, Underhill P, Tolun A, Angelicheva D, Kalaydjieva L. 1996. From Asia to Europe: Mitochondrial DNA sequence variability in Bulgarians and Turks. Ann Hum Genet 60:35–49.

Castro de Guerra D, Pinto-Cisternas J, Rodriguez-Larralde A. 1990. Inbreeding as measured by isonymy in two Venezuelan populations and its relationship to other variables. Hum Biol 62:269–278.

Castro de Guerra D, Arvelo H, Pinto-Cisternas JP. 1993. Estructura de población y factores influyentes en dos pueblos negros venezolanos. Am Negra 5:37–47.

Castro de Guerra D, Arvelo H, Rodriguez Larralde A, Salzano FM. 1996. Genetic study in Panaquire, a Venezuelan population. Hum Hered 46:323–328.

Castro de Guerra D, Hutz MH, Bortolini MC, Salzano FM. 1997. Beta globin gene cluster haplotypes in an admixed Venezuelan population. Am J Hum Biol 9: 323–327.

Chakraborty R. 1985. Gene identity in racial hybrids and estimation of admixture rates. In: Ahuja YR, Neel JV, editors. Genetic differentiation in human and other animal populations. Delhi: Indian Anthropological Association, p, 171–180.

Chakraborty R. 1986. Gene admixture in human populations: Models and predictions. Yrbk Phys Anthropol 29:1–43.

Chakraborty R, Kamboh MI, Ferrel RE. 1991. "Unique" alleles in admixed populations: A strategy for determining hereditary population differences of disease frequencies. Ethn Dis 1:245–256.

Chen Y-S, Torroni A, Excoffier L, Santachiara-Benerecetti AS, Wallace DC. 1995. Analysis of mtDNA variation in African populations reveals the most ancient of all human continent-specific haplogroups. Am J Hum Genet 57:133–149.

Cumming AM, Armstrong JG, Pendry K, Burn AM, Wensley RT. 1991. Polymerase chain reaction amplification of two polymorphic sample repeat sequences within the von Willebrand factor gene: Application to family studies in von Willebrand disease. Hum Genet 89:194–198.

Deka R, Chakraborty R, De Croo S, Rothhammer F, Barton AS, Ferrel RE. 1992. Characteristics of polymorphism at a VNTR Locus 3′ to the apolipoprotein B gene in five human populations. Am J Hum Genet 51:1325–1333.

Deka R, De Croo S, McGarvey ST, Chakraborty R, Jin L, Rothhammer F, Ferrel RE. 1994. Population genetic characteristics of the D1S80 locus in seven human populations. Hum Genet 94:252–258.

Destro-Bisol G, Presciuttini S, d'Apoja E, Dobosz M, Spedini G, Pascali VL. 1994. Genetic variation at the Apo B 3′ HVR, D2S44, and D7S21 loci in the Ewondo ethnic group of Cameroon. Am J Hum Genet 55:168–174.

Falcone E, Spadafora P, De Luca M, Ruffolo R, Brancati C, De Benedictis G. 1995. DYS19, D12S67, and D1S80 polymorphisms in population samples from southern Italy and Greece. Hum Biol 67:689–701.

Fernandes F. 1965. A Integração do Negro na Sociedade de Classes. São Paulo: Editora Dominus.

Forster P, Harding R, Torroni A, Bandelt H-J. 1996. Origin and evolution of Native American mtDNA variation: A reappraisal. Am J Hum Genet 59:935–945.

Franco MHLP, Weimer TA, Salzano FM. 1982. Blood polymorphisms and racial admixture in two Brazilian populations. Am J Phys Anthropol 58:127–132.

Gené M, Huguet E, Sánchez-Garcia C, Moreno P, Corbella J, Mezquita J. 1995. Study of the 3′ Apo B minisatellite performed by PCR in the population of Catalonia (Northeast Spain). Hum Hered 45:70–74.

Graven L, Passarino G, Semino O, Boursot P, Santachiara-Benerecetti S, Langaney A, Excoffier L. 1995. Evolutionary correlation between control region sequence and restriction polymorphisms in the mitochondrial genome of a large Senegalese Mandenka sample. Mol Biol Evol 12:334–345.

Hammer MF, Spurdle AB, Karafet T, Bonner MR, Wood ET, Novelletto A, Malaspina P, Mitchell RJ, Horai S, Jenkins T, Zegura SL. 1997. The geographic distribution of human Y chromosome variation. Genetics 145: 787–805.

Heidrich EM, Hutz MH, Salzano FM, Coimbra CEA Jr, Santos RV. 1995. D1S80 locus variability in three Brazilian ethnic groups. Hum Biol 67:311–319.

Horn GT, McClatchey AI, Richards B, MacDonald ME, Gusella JF. 1991. Deletion by PCR of a VNTR polymorphism at D4S43. Nucleic Acids Res 19:4772.

Hsieh CL, Sutton HE. 1992. Mitochondrial and nuclear variants in a US Black population: Origin of a hybrid population. Ann Hum Genet 56:105–112.

Kayser M, Cagliá A, Corah D, Fretwell N, Gehring C, Graziosi G, Heidorn F, Herrmann S, Herzog B, Hidding M, Honda K, Jobling M, Krawezak M, Leim K, Meuser S, Meyer E, Oesterreich W, Pandya A, Parson W, Penacino G, Perez-Lezaun A, Piccinini A, Prinz M, Schmitt C, Schneider PM, Szibor R, Teifel-Greding J, Weichhold G, Knijff P, Roewer L. 1997. Evaluation of Y-chromosomal STRs: A multicenter study. Int J Legal Med 110:125–133.

Knijff P de, Kayser M, Cagliá A, Corach D, Fretwell N, Gehring C, Graziosi G, Heidorn F, Herrmann S, Herzog B, Hidding M, Honda K, Jobling M, Krawezak M, Leim K, Meuser S, Meyer E, Oesterreich W, Pandya A, Parson W, Penacino G, Perez-Lezaun A, Piccinini A, Prinz M, Schmitt C, Schneider PM, Szibor R, Teifel-Greding J, Weichhold G, Roewer L. 1997. Chro-

mosome Y microsatellite: Population genetic and evolutionary aspects. Int J Legal Med 110:134–140.
Long JC, Smouse PE. 1983. Intertribal gene flow between the Ye'cuana and Yanomama: Genetic analysis of an admixed village. Am J Phys Anthropol 64:411–422.
Long JC. 1991a. The genetic structure of admixed populations. Genetics 127:417–428.
Long JC. 1991b. Genetic variations in Arizona Mexican Americans: Estimation and interpretation of admixture proportions. Am J Phys Anthropol 84:141–157.
Lorente M, Lorente JA, Wilson MR, Budowle B, Villanueva E. 1997. Spanish population data on seven loci: D1S80, D17S5, HUMTH01, HUMVWA, ACTBP2, D21S11, and HLA-DQA1. Forensic Sci Int 86: 163–171.
Ludwig EH, Friedel W, McCarthy BJ. 1989. High-resolution analysis of a hipervariable region in the human apolipoprotein B gene. Am J Hum Genet 45: 458–464.
Mattoso K. 1982. Ser Escravo no Brasil. São Paulo: Editora Brasiliense.
Merriwether DA, Huston S, Iyengar S, Hamman R, Norris JM, Shetterly SM, Kamboh MI, Ferrel RE. 1997. Mitochondrial vs nuclear admixture estimates demonstrate a past history of directional mating. Am J Phys Anthropol 102:153–159.
Nei M, Tajima F. 1981. DNA polymorphisms detectable by restriction endonucleases. Genetics 97:145–163.
Peréz-Lezaun A, Calafell F, Mateu E, Comas D, Bosch E, Bertranpetit J. 1997. Allele frequencies for 20 microsatellites in a worldwide population survey. Hum Genet 47:189–196.
Pinheiro MF, Pontes ML, Huguet E, Gené M, Pinto da Costa J, Moreno P. 1996. Study of three AMPFLPs (D1S80, 3′ Apo B and YNZ22) in the population of the North of Portugal. Forensic Sci Int 79:23–29.
Pinto F, González AM, Hernández M, Larruga JM, Cabrera VM. 1996. Genetic relationship between the Canary Islanders and their African and Spanish ancestors inferred from mitochondrial sequences. Ann Hum Genet 60:321–330.
Renges HH, Peackoc R, Dunning A, Talmud P, Humphries SE. 1992. Genetic relationship between the 3′-VNTR and diallelic apolipoprotein B gene polymorphisms: Haplotype analysis in individuals of European and South Asian origin. Ann Hum Genet 56: 11–33.
Ribeiro-dos-Santos AK, Santos SEB, Machado L, Guapindaia V, Zago MA. 1996. Heterogeneity of mitochondrial DNA haplotypes in pre-Columbian natives of the Amazon river. Am J Phys Anthropol 101:29–37.
Rose G, De Luca M, Falcone E, Spadafora P, Garrieri G, De Benedictis G. 1996. Allele frequency distributions at seven DNA hypervariable loci in a population sample from Calabria (Southern Italy). Gene Geogr 10:135–145.
Richards M, Côrte-Real H, Forster P, Macaulay V, Wilkinson-Herbots H, Demaine A, Papiha S, Hedges R, Bandelt H, Sykes B. 1996. Paleolithic and neolithic lineages in the European mitochondrial gene pool. Am J Hum Genet 59:185–203.
Ruiz-Linares A, Nayar K, Goldstein DB, Hebert JM, Seielstad MT, Underhill PA, Lin AA, Feldman MW, Cavalli-Sforza LL. 1996. Geographic clustering of human Y-chromosome haplotypes. Ann Hum Genet 60: 401–408.
Sagnes MA. 1995. Matrimonios de Esclavos. Caracas: Suma Universitaria.
Sajantila A, Budowle B, Ström M, Johnsson V, Lukka M, Peltonen L, Etnholm C. 1992. PCR amplification of alleles at the D1S80 locus: Comparison of a Finnish and a North American Caucasian population sample, and forensic casework evaluation. Am J Hum Genet 50:816–825.
Santos SEB, Guerreiro JF. 1995. The indigenous contribution to the formation of the population of the Brazilian Amazon region. Rev Bras Genet 18:311–315.
Santos SEB, Ribeiro-dos-Santos AK, Meyer D, Zago MA. 1996. Multiple founder haplotypes of mitochondrial DNA in Amerindians revealed by RFLP and sequencing. Ann Hum Genet 60:305–319.
Schnee-Greise J, Bläß G, Herrmann S, Schneider HR, Förster R, Bäßler G, Pflug W. 1993. Frequency distribution of D1S80 alleles in the German population. Forensic Sci Int 59:131–136.
Schnee-Greise J, Teifel-Greding J. 1991. DNA length polymorphism of the Apo B 3′ region: Frequency distribution of the alleles in the German population. Forensic Sci Int 51:173–178.
Schneider H, Guerreiro JF, Santos SEB, Weimer TA, Schneider PC, Salzano FM. 1987. Isolate breakdown in Amazonia. The Blacks of the Trombetas river. Rev Bras Genet 10:565–574.
Shriver MD, Smith MW, Jin L, Marcini A, Akey JM, Deka R, Ferrel RE. 1997. Ethnic-affiliation by use of population-specific DNA markers. Am J Hum Genet 60:957–964.
Soodyall H, Vigilant L, Hill AV, Stoneking M, Jenkins T. 1996. MtDNA control-region sequence variation suggested multiple independent origins of an "Asian-specific" 9-bp deletion in sub-Saharan Africans. Am J Hum Genet 58:595–608.
Torroni A, Schurr TG, Yang CC, Szathmary EJE, Williams RC, Schanfield MS, Troup GA, Knowler WC, Lawrence DN, Weiss KM, Wallace DC. 1992. Native American mitochondrial DNA analysis indicates that the Amerind and Nadene populations were founded by two independent migrations. Genetics 130:153–162.
Torroni A, Schurr TG, Cabell MF, Brown MD, Neel JV, Larsen M, Smith DG, Vullo CM, Wallace DC. 1993. Asian affinities and continental radiation of the four founding native American mitochondrial DNAs. Am J Hum Genet 53:563–590.
Torroni A, Lott MT, Cabell MF, Chen Y-S, Lavergne L, Wallace DC. 1994. Mitochondrial DNA and the origin of Caucasians. Identification of ancient Caucasian-specific haplogroups, one of which is prone to a recurrent somatic duplication in the D-loop region. Am J Hum Genet 55:760–776.
Torroni A, Petrozzi M, Santomazza P, Sellito D, Cruciani F, Scozzari R. 1995. About the "Asian-specific" 9-bp deletion of mtDNA. Am J Hum Genet 57:507–508.
Valle-Silva N. 1991. Estabilidade temporal e differenças regionais no casamento inter-racial. Estudos Afro-Asiáticos 21:49–60.
Wallace DC. 1995. Mitochondrial DNA variation in human evolution, degenerative disease, and aging. Am J Hum Genet 57:201–223.
Ward RH, Salzano FM, Bonatto SL, Hutz MH, Coimbra CEA Jr, Santos RV. 1996. Mitochondrial DNA polymorphism in three Brazilian Indian tribes. Am J Hum Biol 8:317–323.
Watson E, Forster P, Richards M, Bandelt H. 1997. Mitochondrial footprints of human expansion in Africa. Am J Hum Genet 61:691–704.
Zago MA, Silva-Junior WA, Tavella MH, Santos SEB, Guerreiro JF, Figueiredo MS. 1996. Interpopulational and intrapopulational genetic diversity of Amerindians as revealed by six variable number of tandem repeats. Hum Hered 46:274–289.

APPENDIX

TABLE 1. Weighted average allele frequencies for the hypervariable loci used in the interethnic admixture estimates[1]

		Parental populations		
Loci	Alleles	African	European	Amerindian
F13A1*	1	0.033		0.188
	2	0.170		0.394
	3	0.434	0.058	0.229
	4		0.038	
	5	0.073	0.178	0.078
	6	0.105	0.320	0.111
	7		0.378	
	8	0.105		
	9		0.003	
	12	0.008	0.001	
	13	0.024	0.001	
	14		0.004	
	15	0.048	0.014	
	16		0.005	
D1S80*	14			0.002
	16	0.009	0.002	
	17	0.033	0.005	0.002
	18	0.025	0.237	0.382
	19		0.005	
	20	0.029	0.021	
	21	0.203	0.027	0.002
	22	0.112	0.037	
	23	0.031	0.010	0.020
	24	0.161	0.359	0.089
	25	0.055	0.042	0.094
	26	0.017	0.021	0.020
	27	0.033	0.010	
	28	0.090	0.062	0.036
	29	0.009	0.051	0.010
	30		0.015	0.301
	31	0.009	0.056	0.035
	32		0.007	
	33		0.003	0.002
	34	0.177	0.005	
	35	0.007	0.001	
	36		0.010	
	37		0.007	
	38		0.006	
	40		0.001	
	46			0.004
D4S43*	1	0.584	0.250	0.563
	2	0.249		
	3	0.014		0.004
	6	0.110	0.070	
	7	0.007	0.300	0.154
	8	0.007	0.020	
	9		0.040	
	10		0.070	0.017
	11	0.022	0.160	0.187
	12	0.007	0.040	
	13		0.030	
	14		0.010	0.008
	16			0.059
	17			0.008
	22		0.010	
APOB*	22	0.006		
	23	0.006		
	25	0.016		
	26	0.003	0.002	
	27	0.006		
	29	0.031	0.001	
	30	0.012		
	31	0.071	0.085	0.028
	33	0.062	0.055	
	34	0.003		
	35	0.133	0.213	0.062
	36		0.005	
	37	0.209	0.382	0.470
	38	0.003		
	39	0.099	0.058	0.012
	40	0.006		
	41	0.074	0.014	
	43	0.093	0.002	
	45	0.078	0.012	0.049
	47	0.050	0.073	0.370
	48	0.003		
	49	0.015	0.081	0.008
	51	0.012	0.015	
	53	0.003	0.002	
	57	0.006		
VWF-1*	5	0.213	0.170	0.032
	6	0.347	0.440	0.483
	7	0.139	0.090	
	8	0.015	0.030	0.008
	9	0.118		0.081
	10	0.088	0.090	0.227
	11	0.080	0.180	0.108
	12			0.061
DYS19*	178			0.006
	182			0.019
	186	0.043	0.084	0.692
	190	0.135	0.493	0.205
	194	0.455	0.247	0.072
	198	0.257	0.132	0.006
	202	0.110	0.044	

[1]Sources: (1) Africans: Destro-Bisol et al. (1994); Hammer et al. (1997); Silva-Junior et al. (unpublished); (2) Europeans: Boerwinkle et al. (1989); Ludwig et al. (1989); Horn et al. (1991); Schnee-Greise and Teifel-Greding (1991); Cumming et al. (1991); Deka et al. (1992, 1994); Renges et al. (1992); Sajantila et al. (1992); Schnee-Greise et al. (1993); Falcone et al. (1995); Gené et al. (1995); Pinheiro et al. (1996); Rose et al. (1996); Hammer et al. (1997); Kayser et al. (1997); Knijff et al. (1997); Lorente et al. (1997); Pérez-Lezaun et al. (1997); (3) Amerindians: Heidrich et al. (1995); Ruiz-Linares et al. (1996); Zago et al. (1996); Hammer et al. (1997); Silva-Junior et al. (unpublished).

The number of populations and of individuals studied for the different systems varied. For Africans they are as follows: F13A1: 2, 62; D1S80: 2, 68; D4S43: 2, 68; APOB: 3, 161; VWF-1: 2, 68; DYS19: 18, 236. Europeans: F13A1: 4, 212; D1S80: 8, 1683; D4S43: 1, 81; APOB: 7, 1441; VWF-1: 1, 39; DYS19: 34, 2601. Amerindians: F13A1: 5, 122; D1S80: 10, 255; D4S43: 5, 123; APOB: 5, 123; VWF-1: 5, 123; DYS19: 10, 155.