Original Paper

European Journal of Human Genetics

Eur J Hum Genet 1997;5:390–396

Received: February 24, 1997
Revision received: July 18, 1997
Accepted: August 8, 1997

Haemoglobin D-Ouled Rabah among the Mozabites: A Relevant Variant to Trace the Origin of Berber-Speaking Populations

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Key Words
Haemoglobin D-Ouled Rabah
Blood groups
Berber
Tuareg
Mzab

Abstract

We have studied haemoglobin (Hb) variants and blood groups (ABO, RH, and Kell) in 598 children from the Berber population of the Mzab. Hb D-Ouled Rabah, considered as a private marker of the Kel Kummer Tuaregs, and Hb C were found at the same gene frequency (0.015). Haplotype analysis suggests a single origin to the Hb D mutation. Genetic distances calculated from the blood group data cluster Mozabites and Tuaregs with the other Berber-speaking groups, Arabic-speaking populations being more distant. But, we found no specific relationship between Mozabites and Kel Kummers. Tuaregs in general exhibit features that tend to differentiate them from other Berber-speaking groups. Hb D-Ouled Rabah may be specific of Berber-speaking populations.

Introduction

The origin of the Berber people is not clearly established. North Africa was peopled around the 16th millennium B.C. by a late Palaeolithic culture (Iberomarusian) [1] and then by a more advanced Mesolithic culture (Capsian). Transition to agriculture (Neolithic) occurred around 9,500–7,000 B.C., spreading from the Near East to Egypt. Berbers may be the descendants of the Capsian and the later Neolithic peoples [2]. Berber kingdoms declined under the impact of Greek invasions (457–404 B.C.), Roman Punic wars (264-6 B.C.), and Roman settlements in the area [3–5]. The Arab invasion (7/8th centuries) brought islamisation and dispersal of the Berber culture, even seeing their leader Tariq invade Spain in 710 A.D. and reaching as far as Poitiers in France.

Present-day populations of North Africa are mostly Arabic-speaking, whatever their remote origin. Berbers, however, with their languages and customs, still live in small niches of Northern Morocco and Algeria, and in some Northern Oases of the Sahara, including those of the Mzab (Algeria). The Tuaregs also speak Berber languages. They inhabit the South of the Sahara and have been involved for centuries in trans-Saharan trade. Tuaregs have their own culture that probably diverged from the Berber world through isolation.

We studied haemoglobin (Hb) variants and blood groups in the population of the Mzab. Due to social, religious, and geographic isolation, it has remained unmixed for centuries and may be one of the most representative of Berber identity. The finding of Hb D-Ouled Rabah, considered as a 'private marker' of the Kel Kummer Tuaregs, suggested a common origin.

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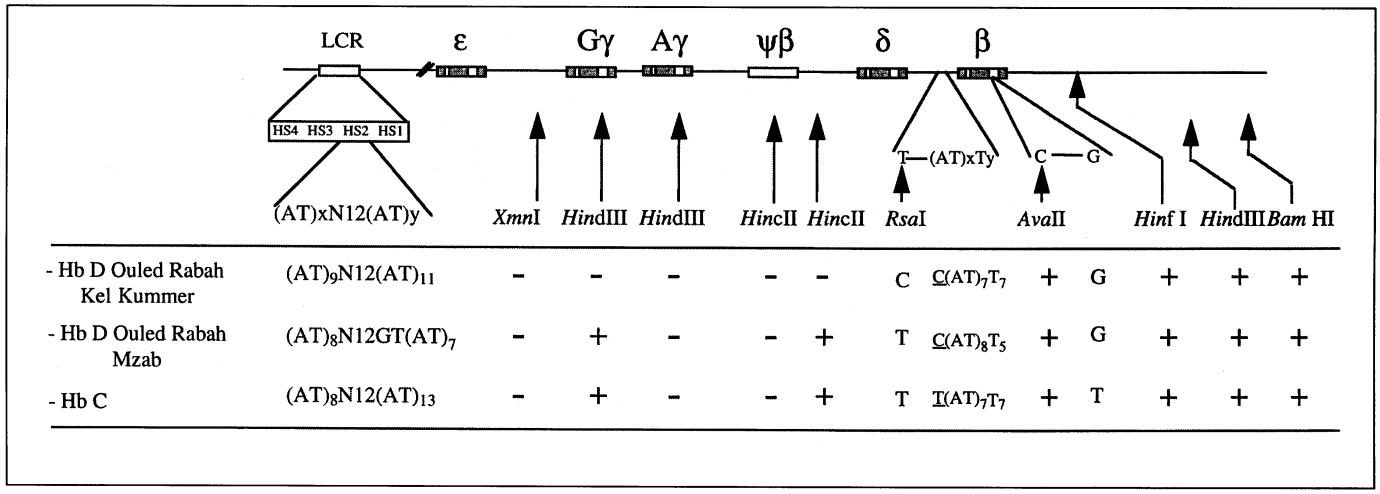


- Hb D Ouled Rabah Kel Kummer	$(AT)_9N12(AT)_{11}$	-	-	-	-	-	C	$\underline{C}(AT)_7T_7$	+	G	+	+	+
- Hb D Ouled Rabah Mzab	$(AT)_8N12GT(AT)_7$	-	+	-	-	+	T	$\underline{C}(AT)_8T_5$	+	G	+	+	+
- Hb C	$(AT)_8N12(AT)_{13}$	-	+	-	-	+	T	$\underline{T}(AT)_7T_7$	+	T	+	+	+

Fig. 1. β-Globin gene haplotypes. Locations of the polymorphic sites that make up the haplotype, together with the restriction enzymes with which they are detected, are shown below the map; + and – indicate the presence or absence of the polymorphic restriction sites, respectively. Sequences of the polymorphic AT repeats in both the (AT)xN12 (AT)y region of the LCR-HS2 and the (AT)xTy region in the β-silencer are also indicated. The β-gene frameworks are indicated by their corresponding sequence and restriction site polymorphisms.

Material and Methods

We studied 598 children (359 males, 239 females; aged 5–19 years) attending Koranic schools of the Ibadite rite in three oases of the Mzab and in Ghardaia, the main city, where pupils come from all the oases. Sampling was at random and informed parental consent was obtained.

Hb screening was performed by alkaline electrophoresis, eventually completed by isoelectric focusing (IEF) [6]. The Hb D mutation was identified by direct sequencing of PCR-amplified DNA from a homozygote subject. It abolishes a *Mae*II site and this was used for confirmation in all the Hb D carriers. β-Globin gene haplotypes were based upon the markers shown in figure 1. PCR-RFLPs were determined as described [7–10], repeats by DNA sequencing [9, 10], and the β-gene framework by denaturing gradient gel electrophoresis [11].

Blood group typing used classical methods. Maximum likelihood allele and haplotype frequencies were estimated using GENEF2, a Lalouel's adaptation of Yasuda's ALL-TYPE program [12]. Genetic distances between any pair of populations were obtained both as co-ancestry coefficient or linearised Fst values [13], and a

$$D_{ij} = \frac{1}{2} \sum_{i=1}^{k} |x_i - y_i|,$$

where x_i and y_i are the frequencies of allele or haplotype i in populations x and y, respectively, and k the size of the frequency vector [14]. The two genetic distance matrices were compared by a Mantel [15] test, using the NTSYS package [16]. D_{ij} distances were used for principal co-ordinate analysis [17]. The significance of the genetic distances between populations was tested using a resampling algorithm [18]. The genetic structure of three population groups (Arabic-speaking, Berbers and Tuaregs) was estimated by analysis of molecular variance (AMOVA) [18] with the ARLEQUIN package [Schneider, Kuffner, Roesseli, Excoffier, unpubl. data].

Results

Haemoglobin Analysis

Two Hb variants (Hb C and D) were observed in the Mozabites with a similar frequency (table 1). Carriers belonged to different families and one D/D homozygote was observed. The D variant was identified as Hb D-Ouled Rabah (codon 19 C → A, β^{19} Asn → Lys) [6, 19], up to now considered a private marker of the Kel Kummer Tuaregs [20]. The genetic background of the mutation was established on DNA from the D/D individual from the Mzab and from an EBV-immortalised cell line from a D/D Kel Kummer individual. In both cases, the mutation was the same. Studied markers included RFLPs, repeats in the LCR HS2 and 5′ to the β-gene, and point mutations in β-IVS2 defining the β-gene frameworks (fig. 1). Only markers 3′ from the mutation are identical in the two populations. In the 5′ region, 5 out of 8 markers are different, including the repeat 639 nt 5′ from the mutation.

Table 1. Red blood cell phenotypes and gene frequencies in the Mozabites

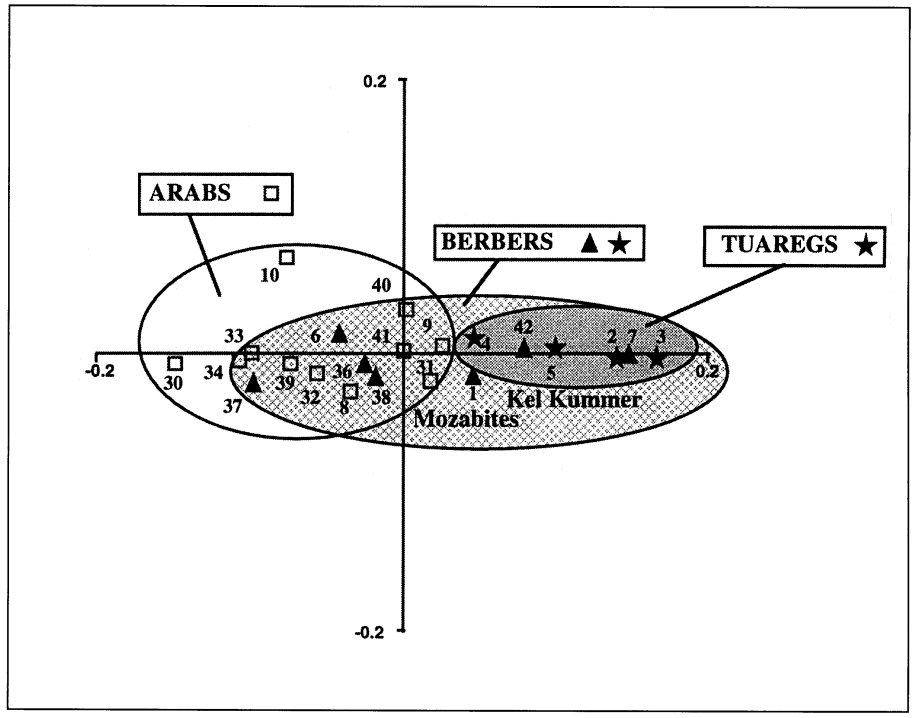
Phenotype	Observed	Allele/haplotype	Frequency[1]	Kel Kummer [34]
Haemoglobins				
AA	564	β^A	0.97	0.86
AD	16	β^D	0.015	0.13
DD	1			
AC	17	β^C	0.014	0.00
Total	598			382
Blood group antigens (ABO, RH and Kell)				
A	155	A	0.164	0.116
B	39	B	0.042	0.041
AB	5	O	0.793	0.843
O	332			
Total	531			286
K	22	K	0.021	0.000
k	503	k	0.979	1.000
Total	525			286
DCE	3	R1 (DCe)	0.286	0.570
DCEe	4	R2 (DcE)	0.072	0.049
DCe	54	R0 (Dce)	0.329	0.211
DCcE	5	Rz (DCE)	0.025	0.000
DCcEe	34	r (cde)	0.253	0.170
DCce	168	r′ (dCe)	0.004	0.000
DcE	9	r″ (dcE)	0.032	0.000
DcEe	53			
Dce	150			
Cce	1			
cE	2			
cEe	6			
ce	36			
Total	535			286

[1] Hardy-Weinberg equilibrium: Hb: χ^2 [3] = 1.058 (n.s.), ABO: χ^2 [1] = 0.97 (n.s.), Rh: χ^2 [8] = 40.16 ($p < 0.01$), but due to one rare phenotype (DCE).

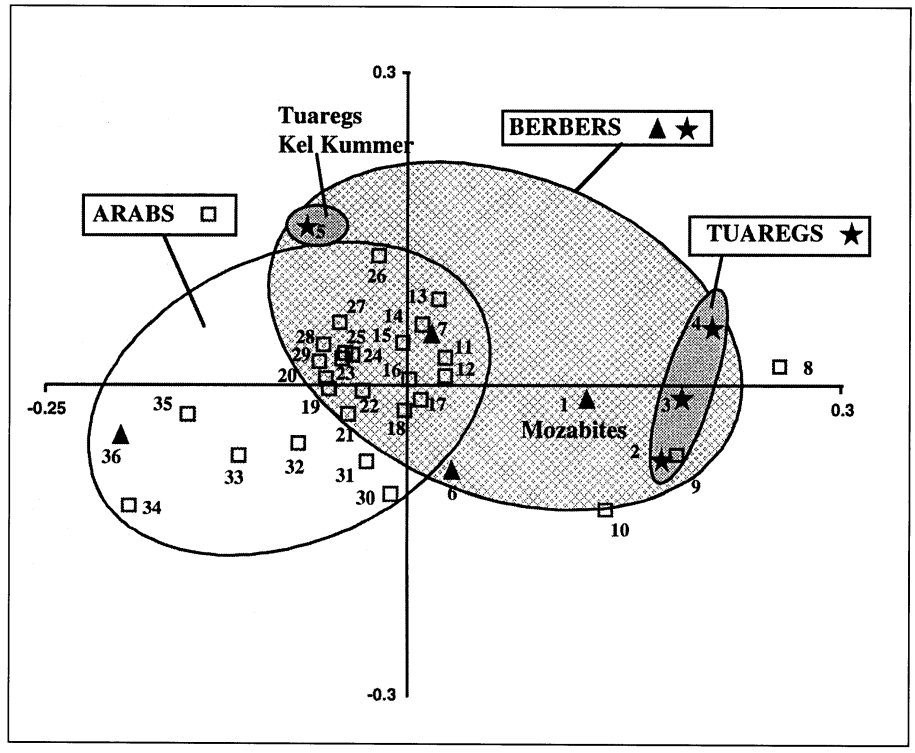
Blood Group Analysis

Results obtained for the Mozabite population are given in table 1 in comparison with those published for the Kel Kummers. Mozabites exhibit a high frequency of allele O (79.3%) and 2.1% of allele K. Among seven RH haplotypes, R0 is the most frequent (32.0%), followed by R1 (28.6%) and r (25.3%). These data were compared to those published for geographically related populations. Genetic distances independently obtained from the D_{ij}'s and co-ancestry coefficients are significantly correlated (ABO: $r = 0.95$, $p < 0.001$; RH: $r = 0.79$, $p < 0.001$).

According to the ABO system, the Mozabites are closely related to other Berber-speaking groups among whom they are genetically intermediate (fig. 2A). For this system, they are not significantly different from the Kel Kummers, but the same result is obtained between Mozabites and other Berbers (No. 2 and 36 in fig. 2A) and non-Berbers (No. 8, 9, 31, 40, and 41). Most Berber groups exhibit high O frequencies, in contrast to the Arabic groups. Among the Berbers, the Tuaregs are genetically the most distant from the latter. Overall, Berber groups are more differentiated from each other ($Fst = 0.023$, $p < 0.001$) than the Arabic-speaking groups ($Fst = 0.006$, $p < 0.001$), while the Tuaregs are homogeneous ($Fst = 0.009$, $0.03 < p < 0.04$). The genetic differentiation between Arabic- and Berber-speaking groups (including Tuaregs)



A



B

Fig. 2. Principal co-ordinate analysis for geographically related populations tested for ABO and RH systems. **A** 22 populations tested for the ABO system. First axis (horizontal): 88% of total variance; second axis (vertical): 5% of total variance. **B** 36 populations tested for the RH system. First axis (horizontal): 49% of total variance; second axis (vertical): 17% of total variance. Among Berber-speaking populations (▲, ★) Tuaregs are individualised (★); Arabic-speaking populations (□). *Mozabites* (the figures in parentheses indicate the size of the studied population for both blood group systems, or for ABO then for RH when it is different for each system; the references are indicated in square brackets): 1 = Ghardaia, Algeria (531; [present study]); *Berbers*: 6 = Zaian, Oran, Algeria (985–630; [35]); 7 = Ait Haddidou, Central Atlas, Morocco (256; [51]); 37 = Kossovitch, M'sirda-Fouaga, Algeria (503; [35]); 38 = Gaud, M'sirda-Fouaga, Algeria (191; [35]); 42 = Messerlin, M'sirda-Fouaga (850; [35]); 36 = Arabs M'Sirdas Fouaga, Oran, Algeria (245; [35]); *Tuaregs*: 2 = Isseq-qamaren, Ahaggar, Algeria (160; [36]); 3 = Isseqqamaren, Tassili N'Ajjer, Algeria (129; [36]); 4 = Air, Niger (164–93; [37]); 5 = Kel Kummer, Adras des Iforas, Mali (286; [38]); *Arabic-speaking populations:* 8 = Algerians, Tidikelt, Algeria (268; [39]); 9 = Algerians, Hoggar, Algeria (132; [40]); 10 = Moroccans, Ksar Glagla (149; [41]); 11 = Mostaganem (127); 12 = Chlef (199); 13 = Blida (172); 14 = Guelma (262); 15 = Jijel (168); 16 = Tiaret (114); 17 = Sidi bel Abbes (112); 18 = Medea (104); 19 = Tizi Ouzou (455); 20 = Constantine (220); 21 = Bouira (186); 22 = Tlemcen (137); 23 = Algiers (315); 24 = Tebessa (125); 25 = Annaba (135); 26 = Batna (155); 27 = Setif (333); 28 = Skikda (148); 29 = Bejaia (164) (11–29 = Algerian samples from Aireche et al. [42]); 30 = Egyptians, Mansurah (250; [43]); 31 = Arabs Chaamba, Metlili, Algeria (232; [35]); 32 = Tunisians (1986-474; [44, 45]); 33 = Libyans, Benghazi, Tripoli (168; [46]); 34 = Libyans, Benghazi (6,000–2,071; [47]); 35 = Moroccan Jews, Tafilalet (146; [48]); 39 = Reguibat, M'Sirda-Fouaga, Algeria (401; [35]); 40 = Algerians, Saoura (293; [49]); 41 = Towara Bedouins, Sinai (202; [50]).

is low but significant (Fct = 0.013, $p < 0.01$). However, it loses significance (Fct = 0.009, $0.04 < p < 0.05$) when the Tuaregs are removed from the comparison. The Arabic groups vs. the Tuaregs taken alone are highly significantly differentiated (Fct = 0.054, $p < 0.001$).

Overall, the analysis on the RH system (fig. 2B) reveals a similar pattern. The Mozabites are genetically close to the other Berbers. But in this analysis, the Kel Kummers occupy a peculiar position due to a high R1 and a low r frequencies. Accordingly, unlike for ABO, the genetic distance between Mozabites and Kel Kummers is highly significant ($p < 0.001$). Other differences are observed compared to ABO. Berber and Arabic groups show comparable levels of intra-group diversity (Arabs: Fst = 0.033, $p < 0.001$; Berbers: Fst = 0.031, $p < 0.001$). Indeed, some Arabic-speaking populations in Southern Algeria and the Moroccan Atlas (No. 8, 9, 10; fig. 2) are very distant from the others and close to the Berbers. Thus, Arabs and Berbers are not significantly differentiated (Fct = 0.012, p = 0.18). This also stands when removing the Tuaregs (Fct = 0.008, p = 0.14), and even when comparing Arabic groups to the Tuaregs alone (Fct = 0.021, p = 0.19), apparently because of the peculiar Kel Kummer RH distribution.

Discussion

Next to blood groups, Hb variants have been studied more extensively and in more human populations than any other genetic marker. Common variants (Hbs S, C, E, A'_2, D-Punjab ...) are found in polymorphic frequencies among populations geographically widely distributed [21]. Some rare variants are frequent in specific populations or isolates because of genetic drift and/or founder effect. Distribution of Hb variants in Algeria reflects successive waves of migration, endogamy, and selective pressure by malaria [22]. Up to now, little was known from the Southern desert part of the country. Our study in the Mzab reveals only two variants, Hb C and D, with a similar frequency. The largest epidemiological study in Algeria included 69 subjects of Mozabite origin in whom only Hb C was observed (gene frequency 0.051 vs. 0.014 in our series) [23]. As in our study, Hb S was not observed in the Mozabites.

Hb C presence in North Africa probably relates to war expeditions and slave trade from its epicentre on the Voltaic plateau during the 15th century [21, 24–26]. Its high frequency among Mozabites may be the result of genetic drift or it may indicate that their isolation was not as tight as believed. Alternatively, as for Hb S in Sicily [27], it may have been introduced at a low frequency and may have later expanded by selective pressure.

Finding Hb D-Ouled Rabah in the Mzab is surprising as it was considered a 'private marker' of the Kel Kummer Tuaregs [28]. The β-globin haplotype surrounding the mutation is different in the two ethnic groups, but all the differences are 5′ from the mutation. A sporadic case of Hb D-Ouled Rabah was reported in China [29], but the hypothesis of independent mutations in two linguistically and geographically close populations (Mozabites and Kel Kummer) is improbable. A hot spot of recombination spans over 9.1 kb from 5′ to the δ-gene to 5′ of the β-globin gene [30]. Thus, a common origin to the mutation is likely, and recombination must have occurred 3′ of, or within, the β-silencer AT repeat. Its origin should be rather ancient to explain the high gene frequency of a rare variant in two groups considered as ethnic isolates.

As this observation suggests a link between the settled Mozabites and the nomadic Tuaregs, we determined blood group frequencies in the Mozabites, and compared our results to those published for the Tuaregs. This comparison was also extended to all the data reported to date concerning Berber- and Arabic-speaking groups in North Africa. Independent analyses of the ABO and RH data converge to show that the Mozabites are close to other Berber-speaking populations. However, they do not confirm a particularly close relationship between Mozabites and Kel Kummers. Actually, most Tuareg communities, although speaking Berber languages, exhibit common genetic features which tend to differentiate them from other Berbers and relate them to sub-Saharan Africans. Cavalli-Sforza et al. [2] propose a common origin between the Tuaregs and an Afro-Asiatic population, the Beja from Sudan. The Tuaregs may have differentiated from the Beja 5,000 years ago and migrated westwards where they were exposed to a Berber influence. Previous results on RH polymorphism indicate that the Beja are genetically closer to North Africans than all other tested Afro-Asiatic populations, except the Tutsi [31, 32]. Another explanation would be a common origin with the Berber people, and a differentiation of the Tuaregs due to their nomadic way of life and higher isolation in the Sahara desert.

Among the Tuaregs, the Kel Kummers distinguish themselves by one of the highest O and R1 frequency ever observed in North Africa, and by the apparent lack of K antigen. A founder effect has been invoked to explain the high frequency of Hb D-Ouled Rabah in the Kel Kummers, and the analysis of blood groups and other classical polymorphisms (for example HLA) reinforces this hypothesis [33, 34].

In conclusion, the finding of Hb D-Ouled Rabah in the Mozabites contradicts the idea that it is a Kel Kummer private marker. However, these two populations are not particularly close genetically. Hb D-Ouled Rabah may be sporadic in North Africa [19], and eventually specific to Berber-speaking populations.

Acknowledgements

This work was supported in part through the INSERM/DRS Algerian-French co-operative aggreement and by grant 3100-039847.93 of the FNRS (Switzerland). We thank S. Zergoune, C. Klouche, Y. Yahia, and S. Merghoub for assistance in collecting blood samples, G. Lenoir for providing the EBV-immortalised cell line, and R. Nagel for his helpful comments.

References

1 Murdock GP: Africa, Its People and Their Culture History. New York, McGraw-Hill Book Company, 1959.

2 Cavalli-Sforza LL, Menozzi P, Piazza A: The History and Geography of Human Genes. Princeton, Princeton University Press, 1994.

3 Camps G: Les Berbères: Mémoire et Identité. Paris, Errance, 1987.

4 Julien C-A: Histoire de l'Afrique du Nord: Tunisie, Algérie, Maroc. Paris, Payot Press, 1978.

5 Mc Eveddy C: Penguin Atlas of Ancient History. Harmondsworth, Penguin, 1967.

6 Basset P, Beuzard Y, Garel MC, Rosa J: Isoelectric focusing of human hemoglobin: Its application to screening, to the characterization of 70 variants, and to the study of modified fractions of normal hemoglobins. Blood 1978; 51:971–982.

7 Semenza GL, Delgrosso K, Poncz M, Malladi P, Schwartz E, Surrey S: The silent carrier allele: β-Thalassemia without a mutation in the β-globin gene or its immediate flanking regions. Cell 1984;39:123–128.

8 Sutton M, Bouhassira EE, Nagel RL: Polymerase chain reaction amplification applied to the determination of β-like globin gene cluster haplotypes. Am J Hematol 1989;33:66–69.

9 Elion J, Berg PE, Lapouméroulie C, Trabuchet G, Mittelman M, Krishnamoorthy R, Schechter A, Labie D: DNA sequence variation in a negative control region 5′ to the β-globin gene correlates with the phenotypic expression of the βs mutation. Blood 1992;79:787–792.

10 Périchon B, Ragusa A, Lapouméroulie C, Romand A, Moi P, Ikuta T, Labie D, Elion J, Krishnamoorthy R: Inter-ethnic polymorphism of the β-globin gene locus control region (LCR) in sickle-cell anemia patients. Hum Genet 1993;91:464–468.

11 Ghanem N, Girodon M, Vidaud M, Martin J, Fanen P, Plassa F, Goosens M: A comprehensive scanning method for rapid detection of β-globin gene mutations and polymorphisms. Hum Mutat 1992;1:229–239.

12 Yasuda N: Estimation of the inbreeding coefficient from phenotype frequencies by a method of maximum likelihood scoring. Biometrics 1968;24:915–935.

13 Reynolds J, Weir BS, Cockerham CC: Estimation of the coancestry coefficient: Basis for a short-term genetic distance. Genetics 1983; 105:767–779.

14 Powell JR, Levene H, Dobzanski T: Chromosomal polymorphisms in *Drosophila pseudoobscura* used for diagnosis of geographic origin. Evolution 1972;26:553–559.

15 Mantel NA: The detection of disease clustering and a generalized regression approach. Cancer Res 1967;27:209–220.

16 Rohlf FJ: NTSYS-pc, Version 1.80 Setauket: Applied Biostatistics, Inc. 1993.

17 Gower JC: Some distance properties of latent root and vector methods used in multivariate analysis. Biometrika 1966;53:325–338.

18 Excoffier L, Smouse P, Quattro JM: Analysis of molecular variants inferred from metric distances among DNA haplotypes: Applications to human mitochondrial DNA restriction data. Genetics 1992;131:479–491.

19 Elion J, Belkhodja O, Wajcman H, Labie D: Hemoglobin D in the Algerian population: Hemoglobin D-Ouled Rabah β19 (B1) Asn → Lys and hemoglobin D-Iran β22 (B4) Glu → Gln. Biochim Biophys Acta 1973;310:360–364.

20 Mauran-Sendrail A, Lefévre-Witier P, Lehmann H, Casey R: Haemoglobin D Ouled Rabah (β19 [B1] Asn → Lys) in a Tuareg tribe of the southern Sahara. J Med Genet 1977;14: 245–249.

21 Lehmann H, Huntsman RG: Man's Haemoglobins, ed 2. Amsterdam, North-Holland Publishing Co, 1974.

22 Labie D, Benabadji M, Elion J: Genetic disorders in North African populations of the Maghreb: Morocco, Algeria, Tunisia; in Teebi A, Farag TI (eds): Genetic Disorders among Arab Populations. Oxford, Oxford University Press, 1996, pp 290–321.

23 Cabanne R: Etude des hémoglobines rencontrées dans la population de la partie occidentale du continent Africain; thèse, Toulouse, 1962.

24 Labie D, Richin C, Pagnier J, Gentilini M, Nagel RL: Hemoglobin S and C in Upper Volta. Hum Genet 1984;65:300–302.

25 Linvingstone FB: Frequencies of Haemoglobin Variants. Oxford, Oxford University Press, 1985.

26 Trabuchet G, Elion J, Baudot G, Pagnier J, Bouhass R, Nigon VM, Labie D, Krishnamoorthy R: Origin and spread of β-globin gene mutations in India, Africa, and Mediterranean: Analysis of the 5′ flanking and intragenic sequences of βs and βc genes. Hum Biol 1991;63: 241–252.

27 Schiliro G: Sicily: The world reservoir for thalassaemia and haemoglobinopathies. Nature 1978;276:761.

28 Junien C, Chaventré A, Fofana Y, Lapouméroulie C, Floury B, Duflo B, Labie D, Kaplan JC: Glucose-6-phosphate dehydrogenase and hemoglobin variants in Kel Kummer Tuareg and related groups. Hum Hered 1982;32:318–328.

29 Ren Y, Chen SS, LIang CC, Zhang MJ, Huang MX, Zhang GL, Zen XS: Hb D-Ouled Rabah [β 19 (B1) Asn → Lys] a rare β chain variant found in a Chinese family. Hemoglobin 1988; 12:77–79.

30 Chakravarti A, Bueto KH, Antonarakis SE, Waber PG, Boehm CD, Kazazian HH: Non-uniform recombination within the human β-globin gene cluster. Am J Hum Genet 1984;36: 1239–1258.

31 Excoffier L, Pellegrini B, Sanchez-Mazas A, Simon C, Langaney L: Genetics and history of sub-Saharan Africa. Yearb Phys Anthropol 1987;30:151–194.

32 Sanchez-Mazas A: Polymorphisme des systèmes immunologiques rhésus, Gm et HLA et histoire du peuplement humain; thèse, Genève, 1990.

33 Chaventré A: Un isolat du Sud Sahara: Les Kel Kummer. Population 1972;4–5:699–803.

34 Chaventré A: Evolution anthropo-biologique d'une population touarègue. Les Kel Kummer et leurs apparentés. Institut National d'Etudes Démographiques, Travaux et Documents, Cahier No 103. Paris, Presses Universitaires de France, 1983.

35 Ruffié J, Cabannes R, Larrouy G: Etude hémotypologique des populations berbères de M'Sirda-Fouaga (Nord-Ouest Oranais). Bull Mém Soc Antrophol Paris 1962;11(suppl 8):294–314.

36 Lefèvre-Wittier Ph: Ecology and biological structures of pastoral Isseqqamaren Tuareg; in Crawford MH, Mielke J (eds): Current Developments in Anthropoligical Genetics 2. New York, Plenum Press, 1982, p 93.

37 Barnicot NA, Ikin EW, Mourant AE: Les groupes sanguins ABO, MNS et Rh des Touareg de l'Aïr. Anthropologie (Paris) 1954;58: 231–240.

38 Langaney A, Chaventré A, Lefèvre-Wittier P, Jacquard A: Un isolat du Sud -Sahara: Les Kel Kummer. VI. Structure génétique des systèmes sanguins érythrocytaires et sériques. VII. Conclusions provisoires. Population 1973;6:1109–1124.
39 Ruffié J, Ducos J, Vergnes H: Etude hémotypologique des populations du Tidikelt (Sahara central). Bull Soc Anthropol Paris 1963;11/4: 531–544.
40 Benabadji M, Ruffié J, Larrouy G, Ducos J, Vergnes H: Etude hémotypologique des populations du massif du Hoggar et du plateau de l'Aïr. 1. Les groupes érythrocytaires. Bull Mém Soc Anthropol Paris 1965;7/XI:171–180.
41 Méchali D, Lévèque J, Faure P: Les groupes sanguins ABO et Rh des Haratins du Maroc. Bull Soc Anthropol Paris 1957;10/8:196–204.
42 Aireche H, Benabadji M: Rh and Duffy gene frequencies in Algeria. Gene Geogr 1988;2:1–8.
43 Mahmoud LA-N, Ibrahim AA-K, Ghonem HR: Human blood groups in Dakahlya, Egypt. Ann Hum Biol 1987;14:487–493.
44 Gherib B, Nicoli RM, Ranque J, Battaglini PF: Recherches sur les populations tunisiennes. Etudes séro-anthropologiques. 12. Bull Soc Anthropol Paris 1965;7(suppl 11):165–179.
45 Moullec J, Abdelmoula H: Quelques données sur les groupes sanguins des Tunisiens. Sem Hôp Paris 1954;30:3061–3062.
46 Walter H, Arndt-Hanser A, Raffa MA, Gumbel A: On the distribution of some genetic markers in Libya. Hum Genet 1975;27:129–139.
47 Woodfield G: 1970 cited as a personal communication; in Mourant AE, Kopec AC, Domaniewska-Sobczak K: The Distribution of the Human Blood Groups and Other Polymorphisms. Oxford, Oxford University Press, 1976.
48 Ikin EW, Mourant AE, Kopec AC, Lehmann H, Scott RAP, Horsfall J: The blood groups and haemoglobin of the Jews of the Tafilalet oases of Morocco. Man 1972;7:595–600.
49 Ruffié J, Benabadji M, Larrouy G: Etude hémotypologique des populations sédentaires de la Saoura, Sahara occidental. I. Les groupes érythrocytaires. Bull Soc Anthropol Paris 1966; 9(suppl 11):45–53
50 Bonné B, Godber M, Ashbel S, Mourant AE, Tills D: South-Sinai Beduin. A preliminary report on their inherited blood factors. Am J Phys Anthropol 1971;34:397–408.
51 Johnson RH, Ikin EW, Mourant AE: Blood groups of the Ait Haddidu Berbers of Morocco. Hum Biol 1963;35:514–523.