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The Hereditary Blood Factors of the Kurds of Iran

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XI. The hereditary blood factors of the Kurds of Iran

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[Plate 26]

Blood specimens were collected from 184 Kurds living in those parts of northwest Iran from which many of the Kurdish Jews, tested in Israel, or their parents, came. Tests were done for the antigens of 10 blood group systems, for the genetic variants of six systems of plasma proteins, and of nine systems of red cell enzymes, and for abnormal haemoglobins. The gene frequencies calculated from the results do not differ greatly from those found in neighbouring populations. They also show a general resemblance to those of the Kurdish Jews, except that the latter have a very much higher incidence of glucose 6-phosphate dehydrogenase deficiency. The possible reasons for this marked difference affecting one genetic system only, are discussed.

Introduction

The Kurds are an ethnic group inhabiting adjacent mountainous regions in western Iran, northern Iraq and southeastern Turkey. There are also small numbers of Kurds in Syria, Jordan, the Soviet Union, Afghanistan and Pakistan. There are today about 9000000 Kurds of whom over 3000000 are in Iran. The Kurdish language belongs to the Indo-European family, being most closely related to, though quite distinct from, Iranian.

It is only about the beginning of the Christian era that the Kurds become recognizable in history as a distinct people. There is considerable disagreement as to their origin and previous history. Nikitine (1943) has summarized the views of numerous authorities and supports the theory of Minorsky (1945) that the Kurds originated as an amalgam of two related tribes, the Mardoi and the Kyrtioi, speaking related Median dialects. If we accept these views then the Kardoukhoi whom Xenophon encountered in the country which is now Kurdistan were not the main or sole ancestors of the Kurds (but migrated to become the Kartvelians of the Caucasus).

We may, however, regard the Kurds as descended at least in part from the Medes whose king Cyrus had his capital at Sanandaj, now the chief town of Iranian Kurdistan.

No adequate anthropometric survey of the Kurds exists, and we are dependent almost entirely on verbal descriptions from which it is clear that there are considerable variations of physical type from tribe to tribe. It is at least certain that some of the Kurds are blonde and blue-eyed while others are much darker in both hair and eyes. A comprehensive anthropometric survey is much to be desired of the Kurds, as well as of the many other ethnic groups who are their neighbours.

In view of the growing relative importance of the blood groups and other inherited blood factors in characterizing populations and defining their relationships to other populations, it had for many years been realized that such a study of the Kurds would be of great importance.

There was, however, a further reason for wishing to obtain such data. A numerous and important group of Jews, the Kurdish Jews, formerly lived in Kurdistan. Some still remain but many have migrated to Israel where they have been found to possess the highest known frequency of glucose 6-phosphate dehydrogenase (G6PD) deficiency (Cohen *et al.* 1959). The initial discovery has been followed by numerous further studies of the incidence of this and other hereditary blood factors.

Subsequent work in Iran by Beaconsfield *et al.* (1966), Beaconsfield, Mahboubi & Rainsbury (1967) and Hedeyat, Amirshany & Khademy (1969) showed a high incidence of G6PD deficiency in various populations both of Kurds and of Kurdish Jews. High frequencies of this deficiency have a special interest because of the finding (Bernini *et al.* 1960; Siniscalco, Bernini, Latte & Motulsky 1961; Gilles *et al.* 1967) of a probable relationship between the deficiency and resistance to malarial infection. Thus communities exposed to such infection and having acquired the deficiency gene might be expected to develop a raised frequency of the deficiency by means of natural selection. A similar process is almost certainly the cause of a raised frequency of haemoglobin S in populations, especially in Africa, which are or have been exposed to endemic *falciparum* malaria. The main homeland of the Kurds, and the region from which most of the Kurdish Jews migrated is, however, mountainous and might therefore not be expected to be favourable to malaria.

These considerations led to the organization of a joint expedition of Iranian and British workers to areas in Iran where Kurds live who are believed to be almost completely unmixed genetically with other populations. The area visited is shown in the map (figure 1). The investigators made their headquarters in Sanandaj and from there visited the districts of Baneh, Marivan, Sanandaj and Bijar. Though the Kurdish populations of all these districts are regarded as unmixed, there is a greater chance of slight Turkish admixture in Sanandaj and Bijar than in the other two districts. Sagres and Gorveh, where the populations were not sampled, have a somewhat greater likelihood still of Turkish admixture. In the Sanandaj district itself the southern half is regarded as more Kurdish than the northern half.

Within each village extensive intermarriage takes place, and cousin marriage is the rule rather than the exception. It was therefore decided to examine no more than one member of any one family, and not more than two or three persons from any one village. The main object of the investigation was to ascertain the incidence of glucose 6-phosphate dehydrogenase deficiency; this is an X-linked condition and the genotype of females cannot be diagnosed with certainty. The sampling was therefore limited to males, whose type can always be diagnosed.

Visits were made to the villages themselves but as it was the harvest season a large proportion

FIGURE 1. Sketch map of Iranian Kurdistan (Kordestan). (Scale about 1 in 5 500 000.)

of the men were in the fields and much of the collecting was done there. This had the advantage that men from different villages were often found working together. The name and age of each man was recorded as well as his family and village. The latter information helped to ensure that the sample was a random one. The object was not to take as many samples as possible, but to obtain samples from as many villages as possible.

As well as taking blood samples, the medical members of the team gave advice on minor medical complaints and handed out medicines.

Blood was taken from the antecubital vein into vacutainers containing sequestrene (EDTA). Two vacutainers were filled from each person, one for retention in Tehran and one for sending to London and Cambridge. Thirty-eight pairs of samples were obtained in Marivan, 39 in Baneh, 36 in Sanandaj and 71 in Bijar, 184 in all.

The samples were taken in refrigerated containers to Tehran and one set was sent on from there by air freight to London, where tests were carried out at the Medical Research Council Serological Population Genetics laboratory for the antigens of the blood group systems: ABO, MN, P, Rh, Lutheran, Kell, Duffy, Diego, Radin and Wright, and for the serum groups: Gm, Gc, Ag, haptoglobins and transferrins, also for the red cell isoenzymes: glucose 6-phosphate dehydrogenase (by electrophoresis), acid phosphatase, 6-phosphogluconate dehydrogenase, phosphoglucomutase, adenylate kinase, lactate dehydrogenase, adenosine deaminase, and phosphohexose isomerase. At Cambridge specimens were tested for abnormal haemoglobins, for glucose 6-phosphate dehydrogenase deficiency and for variants of serum pseudocholinesterase.

RESULTS

All transferrins were of type TfC apart from one TfC/TfD heterozygote among the specimens from Sanandaj. All specimens tested for lactate dehydrogenase were normal, except for one heterozygous slow variant from Sanandaj. The tests for atypical pseudocholinesterase were carried out by the method of Morrow & Motulsky (1968). No atypical variant was found in any of the specimens. Whenever there was a doubtful result the dibucaine number and the fluoride number were ascertained by the usual methods and it was confirmed that the specimens were normal. The screening method used detects the homozygote and heterozygotes of the variant gene E_1^a as well as the homozygotes of the genes E_1^s and E_1^f. It does not, however,

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detect the heterozygotes of E_1^s and of E_1^f with the normal gene E_1^u, and any such heterozygotes present would have remained undetected. The results of all the remaining tests are set out in tables 1 to 15.

TABLE 1. THE ABO GROUPS

phenotype	nos. observed: Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected Sanandaj and Bija
0	15	18	33	32.57	13	25	38	36.58
A_1	11	8	19	19.65	9	14	23	23.94
A_2†	4	2	6	5.83	4	6	10	10.71
B	6	8	14	14.53	6	19	25	26.66
A_1B	1	3	4	3.29	1	6	7	5.95
A_2B†	1	0	1	1.13	3	1	4	3.16
total	38	39	77	77.00	36	71	107	107.00

	gene frequencies: Marivan and Baneh	Sanandaj and Bija
p_1	0.1621	0.1511
p_2†	0.0558	0.0801
q	0.1317	0.1841
r	0.6504	0.5847
total	1.0000	1.0000

† The individual from Marivan listed as A_2B was A_3B: in computations A_2, A_2B and p_2 have been treated as including A_3, A_3B and p_3 respectively.

TABLE 2. THE MNSs GROUPS

phenotype†	nos. observed: Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected Sanandaj and Bija
MMSS	4	7	11	7.78	3	8	11	8.42
MMSs	7	6	13	15.59	9	13	22	19.63
MMss	3	6	9	7.81	3	8	11	11.44
MNSS	6	1	7	5.41	1	3	4	6.16
MNSs	5	5	10	17.81	7	10	17	24.59
MNss	7	8	15	12.41	6	15	21	20.28
NNSS	1	1	2	0.94	1	2	3	1.12
NNSs	3	0	3	4.31	1	6	7	6.37
NNss	2	5	7	4.93	5	6	11	8.99
total	38	39	77	76.99	36	71	107	107.00

	frequencies of gene complexes: Marivan and Baneh	Sanandaj and Bija
MS	0.3179	0.2806
Ms	0.3185	0.3269
NS	0.1106	0.1026
Ns	0.2530	0.2899
total	1.0000	1.0000

† All individuals were Henshaw-negative.

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TABLE 3. THE Rh GROUPS

phenotype	nos. observed: Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected Sanandaj and Bija
CCDEE	—	—	—	—	0	0	0	0.01
CCDEe	—	—	—	—	0	0	0	1.09
C^{w}CDEe	—	—	—	—	0	0	0	0.02
CCDee	10	11	21	21.84	14	24	38	32.26
C^{w}CDee	—	—	—	—	0	1	1	1.09
C^{w}C^{w}Dee	—	—	—	—	0	0	0	0.01
CCddee	—	—	—	—	0	0	0	0.27
CcDEE	—	—	—	—	0	0	0	0.28
CcDEe	11	14	25	19.71	4	8	12	16.54
CDE/ce†	—	—	—	—	1	1	2	0.58
C^{w}cDEe	—	—	—	—	0	1	1	0.28
CcDee	8	7	15	18.63	8	18	26	31.57
C^{w}cDee	—	—	—	—	0	0	0	0.58
CcD^{u}ee	—	—	—	—	2	0	2	0.30
Ccddee	—	—	—	—	0	1	1	2.33
ccDEE	2	0	2	4.39	2	1	3	2.11
ccDEe	5	2	7	7.51	3	8	11	8.69
ccDee	—	—	—	—	1	2	3	2.55
ccD^{u}Ee‡	0	1	1	0.07	—	—	—	—
ccD^{u}ee	0	0	0	0.58	0	0	0	1.37
ccddEE	0	0	0	0.05	—	—	—	—
ccddEe	0	0	0	0.84	—	—	—	—
ccddee	2	4	6	3.39	1	6	7	5.07
total	38	39	77	77.01	36	71	107	107.00

	frequencies of gene complexes: Marivan and Baneh	Sanandaj and Bija
CDE (*Rz*)	—	0.0093
CDe (R_1)	0.5325	0.5014
Cde (*r′*)	—	0.0500
C^{w}De (R_1^{w})	—	0.0093
cDE (R_2)	0.2144	0.1402
cDE (R_0)	—	0.0445
cD^{u}e (R_0^{u})	0.0171	0.0275
cDe (*r″*)	0.0259	—
cde (*r*)	0.2101	0.2178
total	1.0000	1.0000

† The use of anti-Ce serum enables genotypes of the type *CE/ce* to be distinguished from *Ce/cE* which constitute most of the phenotype CcDEe.

‡ Assumed to be of genotype *cD^{u}e/cdE*, not *cD^{u}E/cde*.

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Table 4. The Kell groups†

genotype	nos. observed: Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected Sanandaj and Bija
KK	0	0	0	0.00	0	0	0	0.02
Kk	1	0	1	1.00	0	3	3	2.95
kk	37	39	76	76.00	36	68	104	104.02
total	38	39	77	77.00	36	71	107	106.99

	gene frequencies: Marivan and Baneh	Sanandaj and Bija
K	0.0065	0.0140
k	0.9935	0.9860
total	1.0000	1.0000

† All individuals were Js (a−).

Table 5. The Duffy groups

phenotype	nos. observed: Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected Sanandaj and Bija
Fy (a−b−)	0	4	4	2.16	1	3	4	1.94
Fy (a+b−)	10	20	30	33.70	9	19	28	33.01
Fy (a−b+)	7	5	12	15.95	12	15	27	32.00
Fy (a+b+)	21	10	31	25.18	14	34	48	40.05
total	38	39	77	76.99	36	71	107	107.00

	gene frequencies: Marivan and Baneh	Sanandaj and Bija
Fy^a	0.5150	0.4368
Fy^b	0.3175	0.4285
Fy	0.1675	0.1347
total	1.0000	1.0000

Table 6. Sundry blood groups†

phenotype	nos. observed: Marivan	Baneh	Sanandaj	Bija	Marivan and Baneh	Sanandaj and Bija	gene	gene frequency: Marivan and Baneh	Sanandaj and Bija
P_1	29	30	27	51	59	78	P_1	0.5165	0.4860
P_2	9	9	9	19	18	28	$P_2(+p)$	0.4835	0.5140
L^u (a+)	1	0	2	3	1	5	Lu^a	0.0065	0.0236
L^u (a−)	37	39	34	68	76	102	Lu^b	0.9935	0.9764
Di (a+)	1	0	0	0	1	0	Di^a	0.0065	0.0000
Di (a−)	37	39	36	71	76	107	Di^b	0.9935	1.0000
Wr (a+)	0	0	1	1	0	2	Wr^a	0.0000	0.0094
Wr (a−)	38	39	35	70	77	105	*Wr*	1.0000	0.9906

† All individuals were Rd (−).

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TABLE 7. THE HAPTOGLOBINS

phenotype Hp	nos. observed: Marivan	Baneh	total	no. expected† Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected† Sanandaj and Bija
1	3	5	8	7.67	2	5	7	7.46
2–1	16	15	31	31.66	16	26	42	41.08
2	17	16	33	32.67	16	40	56	56.46
negative	2	3	5		2	0	2	
total	38	39	77	72.00	36	71	107	105.00

	gene frequencies†: Marivan and Baneh	Sanandaj and Bija
Hp^1	0.3264	0.2667
Hp^2	0.6736	0.7333
total	1.0000	1.0000

† Gene frequencies and expected numbers have been calculated omitting the Hp-negatives.

TABLE 8. THE Gc GROUPS

phenotype Gc	nos. observed: Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected Sanandaj and Bija
1	16	20	36	36.48	20	45	65	62.85
2–1	19	15	34	33.04	11	23	34	38.31
2	3	4	7	7.48	5	3	8	5.84
total	38	39	77	77.00	36	71	107	107.00

	gene frequencies: Marivan and Baneh	Sanandaj and Bija
Gc^1	0.6883	0.7664
Gc^2	0.3117	0.2336
total	1.0000	1.0000

TABLE 9. THE Ag GROUPS

phenotype Ag	nos. observed: Marivan	Baneh	Sanandaj	Bija	Marivan and Baneh	Sanandaj and Bija	gene	gene frequency: Marivan and Baneh	Sanandaj and Bija
x+	17	17	16	34	34	50	Ag^x	0.2527	0.2701
x−	21	22	20	37	43	57	Ag^y	0.7473	0.7299
total	38	39	36	71	77	107		1.0000	1.0000

TABLE 10. THE ACID PHOSPHATASE VARIANTS

phenotype Ac Ph	nos. observed: Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected Sanandaj and Bija
A	2	6	8	9.12	5	8	13	10.69
BA	18	17	35	34.06	14	27	41	44.66
B	16	16	32	31.82	13	35	48	46.67
CA	2	0	2	0.69	0	0	0	0.95
CB	0	0	0	1.29	2	1	3	2.01
C	0	0	0	0.02	0	0	0	0.02
total	38	39	77	77.00	34	71	105	105.00

	gene frequencies: Marivan and Baneh	Sanandaj and Bija
P^a	0.3441	0.3190
P^b	0.6429	0.6667
P^c	0.0130	0.0143
total	1.0000	1.0000

TABLE 11. THE ADENYLATE KINASE VARIANTS

phenotype AK	nos. observed: Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected Sanandaj and Bija
1	35	34	69	68.27	30	60	90	89.61
2–1	3	4	7	8.47	3	11	14	14.78
2	0	1	1	0.26	1	0	1	0.61
total	38	39	77	77.00	34	71	105	105.00

	gene frequencies: Marivan and Baneh	Sanandaj and Bija
AK^1	0.9416	0.9238
AK^2	0.0584	0.0762
total	1.0000	1.0000

TABLE 12. THE PHOSPHOGLUCOMUTASE (PGM_1) VARIANTS

phenotype PGM_1	nos. observed: Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed: Sanandaj	Bija	total	no. expected Sanandaj and Bija
1	20	25	45	46.75	16	32	48	49.37
2–1	17	13	30	26.50	17	31	48	45.26
2	1	1	2	3.75	1	8	9	10.37
total	38	39	77	77.00	34	71	105	105.00

	gene frequencies: Marivan and Baneh	Sanandaj and Bija
PGM_1^1	0.7792	0.6857
PGM_1^2	0.2208	0.3143
total	1.0000	1.0000

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TABLE 13. THE 6-PHOSPHOGLUCONATE DEHYDROGENASE VARIANTS

phenotype PGD	nos. observed Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed Sanandaj	Bija	total	no. expected Sanandaj and Bija
A	36	30	66	66.40	33	66	99	99.10
CA	2	9	11	10.21	1	5	6	5.82
C	0	0	0	0.39	0	0	0	0.08
total	38	39	77	77.00	34	71	105	105.00

	gene frequencies Marivan and Baneh	Sanandaj and Bija
PGD^A	0.9286	0.9715
PGD^C	0.0714	0.0285
total	1.0000	1.0000

TABLE 14. THE GLUCOSE 6-PHOSPHATE DEHYDROGENASE VARIANTS

phenotype	nos. observed Marivan	Baneh	total	frequency observed Marivan and Baneh	nos. observed Sanandaj	Bija	total	frequency observed Sanandaj and Bija
B+	36	36	72	0.9351	33	69	102	0.9714
B−	2	3	5	0.0649	1	2	3	0.0286
total	38	39	77	1.0000	34	71	105	1.0000

The deficient types, in the absence of evidence to the contrary, are assumed to be all of type B−. As the subjects are all males, gene frequencies are identical with phenotype frequencies.

TABLE 15. THE ADENOSINE DEAMINASE VARIANTS

phenotype ADA	nos. observed Marivan	Baneh	total	no. expected Marivan and Baneh	nos. observed Sanandaj	Bija	total	no. expected Sanandaj and Bija
1	31	35	66	66.40	21	52	73	72.77
2–1	7	4	11	10.21	11	17	28	28.44
2	0	0	0	0.39	1	2	3	2.78
total	38	39	77	77.00	33	71	104	103.99

	gene frequencies Marivan and Baneh	Sanandaj and Bija
ADA^1	0.9286	0.8365
ADA^2	0.0714	0.1635
total	1.0000	1.0000

Haemoglobin tests

The examination for abnormal haemoglobins followed the techniques described by Lehmann & Huntsman (1966). Three heterozygotes were found for the genes for haemoglobins A and D. The D variant concerned was shown by 'finger-printing' (figure 3, plate 26), to be haemoglobin

D Punjab. Four persons, one from Baneh, three from Sanandaj, showed a raised haemoglobin A_2 level (6.0–6.5 %) without any significant elevation of haemoglobin F; they may be regarded as heterozygotes for the high A_2 variety of β-thalassaemia. Two persons, one from Baneh, one from Marivan, showed a high level of haemoglobin F, 16 and 7 % respectively, with a normal one of haemoglobin A_2 and are considered to be heterozygotes for $\delta\beta$-thalassaemia. One of these (from Baneh) has a particularly high level (16 %) of foetal haemoglobin. Smears from this one were stained for intracellular foetal haemoglobin by the method of Kleihauer, Braun & Betke (1957) (see figure 2, plate 26). The distribution of haemoglobin between the red cells was found to be uneven, some appearing devoid of the foetal type. This confirmed the diagnosis of high F β-thalassaemia (in contradistinction to persistence of high foetal haemoglobin, PHDH, into adult life, a condition characterized by an even distribution of haemoglobin F). The second specimen (from Marivan) was too small to allow this confirmatory test to be done.

The tables

Owing to the large amount of data needing to be presented in the tables, and to the need to keep them to a reasonable size, it has been decided, in tables 1 to 15, to give for each main population only observed and expected numbers for each phenotype, and gene frequencies, omitting the expected and observed proportional phenotype frequencies usually given in presenting such data. Anyone requiring to use these frequencies can readily calculate them from the figures given in the tables.

Discussion

The two geographical subdivisions of the Kurds here adopted differ significantly only with respect to one genetic system, but the other systems nevertheless show small differences which tend to support the initial supposition that the Kurds of Marivan and Baneh might be less mixed than those of Sanandaj and Bijar. The one exception is the adenosine deaminase system, where the inhabitants of Sanandaj and Bijar have a significantly higher frequency of the ADA^2 allele. When using over twenty systems it might be expected that one system would, by the chances of sampling alone, show an apparently significant difference, at the conventional probability level of one in twenty, but the difference actually found for this system gives a χ^2 value for the comparison of the two samples of 6.7, showing a probability of less than 0.01 that they are drawn from a single homogenous population. Moreover, ADA^2 is a gene which was already known to increase markedly in frequency from west to east in Europe and Asia.

Vergnes & Gherardi (1971) have reported the results of an isoenzyme survey of about 160 Kurds living in Syria and Jordan (the numbers tested are slightly different for different tests). Frequencies of the variants of acid phosphatase, adenylate kinase and 6-phosphogluconate dehydrogenase are very close to those found in the present series. The frequency of the phosphoglucomutase gene PGM_1^2 is 33 %, appreciably higher than the overall frequency of 27.5 % found in the present survey. The difference is not quite significant at the 5 % level of probability. The Kurds of Jordan and Syria were not tested for variants of adenosine deaminase. There is no significant difference between the two communities in the incidence of glucose 6-phosphate dehydrogenase deficiency.

The study of the genetics of Kurdish Jews in Israel (Godber, Kopeć, Mourant, Tills &

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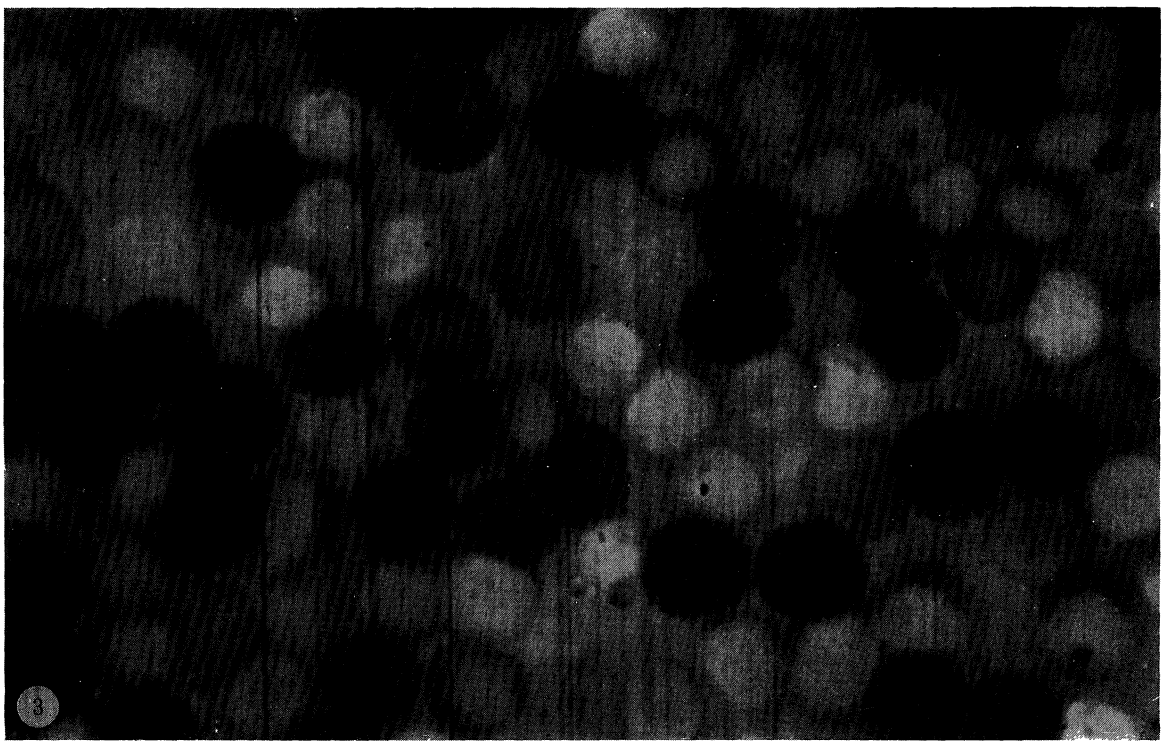

FIGURE 2. 'Fingerprint' of haemoglobin D (Punjab) from Kurdistan. The tryptic peptides and amino acid residues are indicated by roman and arabic numerals respectively.

FIGURE 3. Film of blood from individual from Baneh heterozygous for $\delta\beta$-thalassaemia, stained by Kleihauer method.

(*Facing p.* 204)

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Lehmann 1973, this volume), shows that the genetic differences between them and the Iranian Kurds as a whole, though considerably greater than between the two main subdivisions of the present series of Kurds, are not outstandingly great except with regard to the incidence of glucose 6-phosphate dehydrogenase deficiency. If the distribution of this deficiency is an evolutionary response to malaria, there is no reason to think that the frequencies found among the Iranian Kurds are far from the equilibrium frequencies corresponding to the incidence of *falciparum* malaria in recent times. It is the frequencies among the Kurdish Jews which require a special explanation, and possible explanations are discussed by Godber *et al.* (1973).

The results of a survey of the incidence of tasters of phenylthiocarbamide, and of males with defective colour vision among the Kurds was carried out immediately following the present investigation, and the results have been published by Lightman, Carr-Locke & Pickles (1971).

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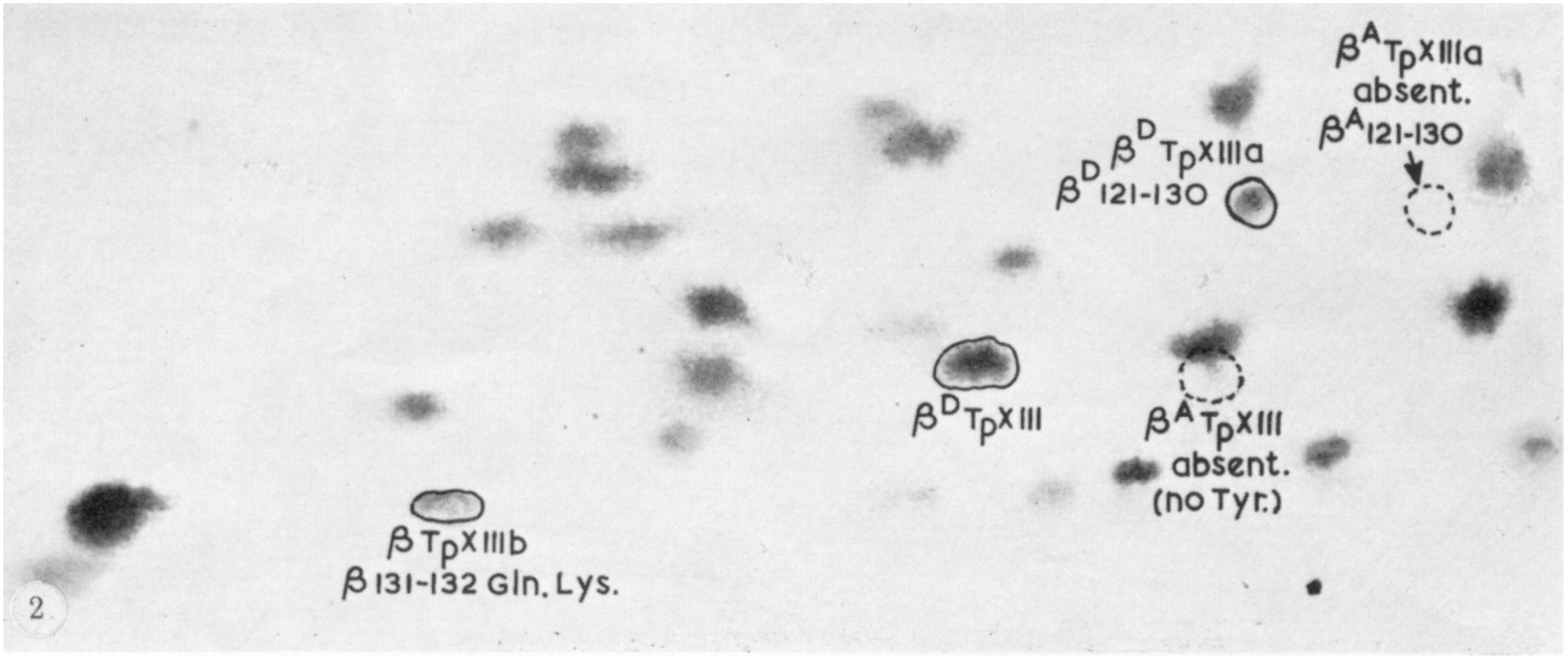

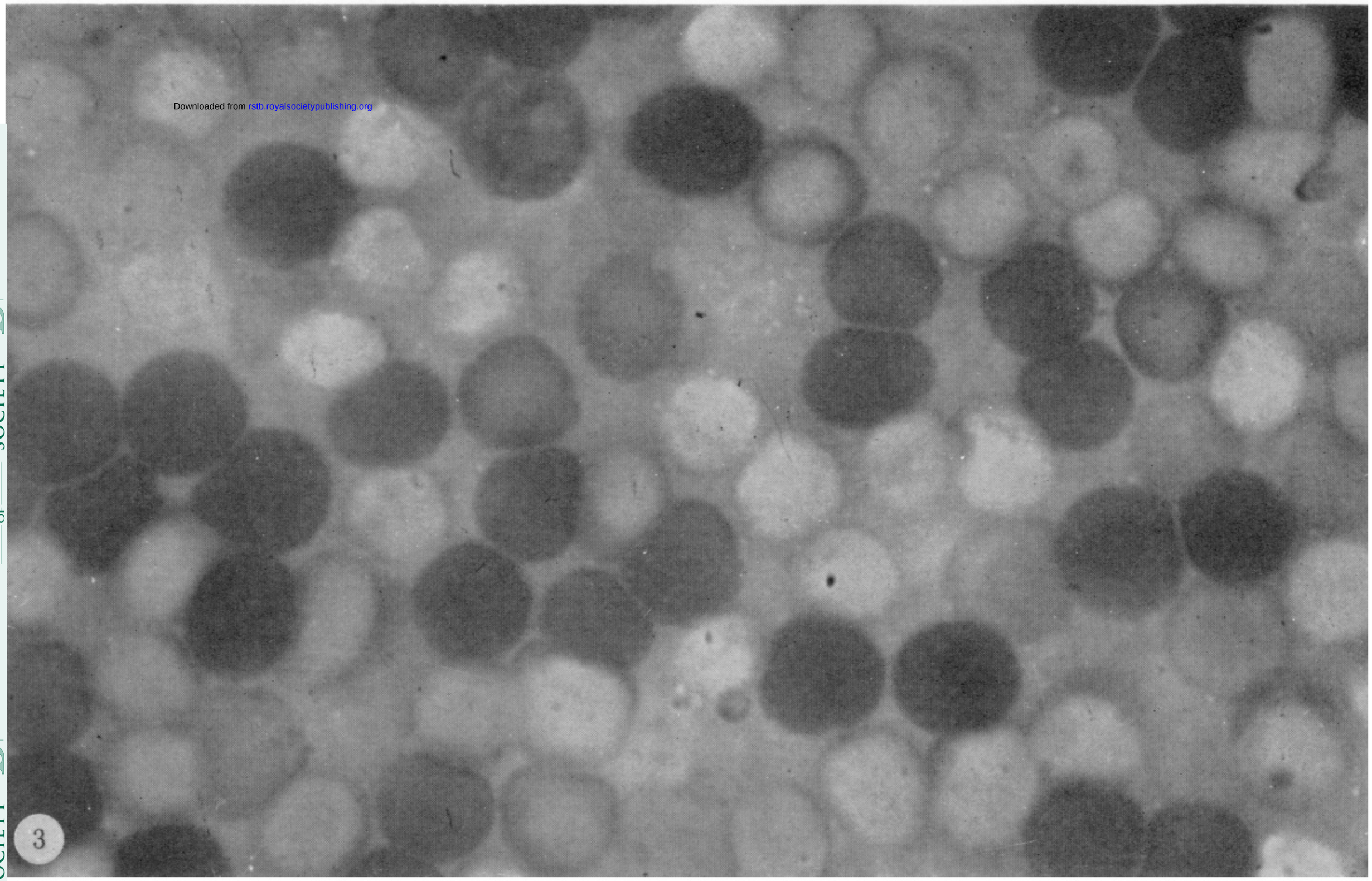

FIGURE 2. 'Fingerprint' of haemoglobin D (Punjab) from Kurdistan. The tryptic peptides and amino acid residues are indicated by roman and arabic numerals respectively.

FIGURE 3. Film of blood from individual from Baneh heterozygous for $\delta\beta$-thalassaemia, stained by Kleihauer method.