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## IX. The hereditary blood factors of the Yemenite and Kurdish Jews

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Blood specimens were collected in the Negev, Israel, from 75 Yemenite Jews and 121 Kurdish Jews, who were also subjected to a variety of measurements and physiological tests. The specimens were tested for the antigens of 10 blood group systems, for the genetic variants of four systems of plasma proteins and of eight systems of red-cell enzymes. The gene frequencies calculated from the results show that these two Jewish populations differ widely from one another genetically. The Yemenite Jews show a rather close resemblance to the Yemenite Arabs. The Kurdish Jews show a fair resemblance to the indigenous Kurds of Iran, but differ markedly in having a very much higher incidence of glucose 6-phosphate dehydrogenase deficiency. As there are reasons for thinking that, in general, a high incidence of this deficiency results from natural selection in response to endemic malarial infection, the marked difference between Kurdish Jews and the indigenous Kurds who were for many generations their close neighbours is difficult to explain.

## INTRODUCTION

A large amount of previous work by numerous authors, summarized by Mourant (1959), shows that there is a close resemblance between the blood group frequencies of most of the Jewish communities of Europe and North Africa, whereas those of the communities of Asia differ widely from one another and from those of the European and North African communities. Previous work on the Yemenite Jews, however, shows a close resemblance to the Yemenite Arabs. The frequencies in the Jewish communities of Iraq, Iran, Kurdistan and Turkestan not only were found to differ considerably from those of the western communities and from one another, but also appeared to differ considerably from those of the indigenous communities of the regions concerned; information on the indigenous peoples was, however, inadequate. The Jews of Iraq have been found to possess very high frequencies of glucose 6-phosphate dehydrogenase deficiency, and the highest known frequencies of this condition have proved to occur in the Jews from Kurdistan (northern Iraq and northwestern Iran) (Cohen *et al.* 1963).

The opportunity to study in greater detail the hereditary blood factors of the Yemenite and Kurdish Jews as part of a multidisciplinary Israeli–British project thus promised to yield information of considerable anthropological and genetic interest. Owing to an interruption of the investigations, due to causes beyond the control of the participants, the numbers tested are fewer than would have been desirable, but have nevertheless yielded results of considerable value, especially when considered in comparison with extensive new data on the indigenous populations of Arabia and Kurdistan respectively.

Specimens of about 10 ml of whole blood were taken in vacutainers containing potassium EDTA solution and sent by air freight to London where they were tested at the Serological

Population Genetics Laboratory by standard methods for the blood group antigens: A, A<sub>1</sub>, B, H, M, N, S, He, P<sub>1</sub>, C, C<sup>w</sup>, c, D, D<sup>u</sup>, E, e, V, Lu<sup>a</sup>, K, Js<sup>a</sup>, Fy<sup>a</sup>, Fy<sup>b</sup>, Jk<sup>a</sup>, Di<sup>a</sup>, Wr<sup>a</sup>. Tests were carried out for the serum immunoglobulin groups Gm and Inv, haptoglobins, and transferrins, and for variants of the red-cell enzymes glucose 6-phosphate dehydrogenase, acid phosphatase, adenylate kinase, 6-phosphogluconate dehydrogenase, phosphoglucomutase, lactate dehydrogenase, malate dehydrogenase and phosphohexose isomerase.

Tests for haemoglobin variants were carried out by Dr G. H. Beaven at the National Institute for Medical Research (Hampstead Laboratories).

No Di<sup>a</sup>-positives or Wr<sup>a</sup>-positives were found, and among the Kurdish Jews no He-positives, V-positives or Js<sup>a</sup>-positives. No variant types were found for lactate dehydrogenase, malate dehydrogenase or phosphohexose isomerase. All transferrins were of type B, except for one BC in a Kurdish Jew.

The results of the remaining tests are set out in tables 1 to 26 together with the results of gene-frequency calculations.

TABLE 1. THE A<sub>1</sub>A<sub>2</sub>BO GROUPS OF THE YEMENITE JEWS

group	no. observed	frequency observed	frequency expected	no. expected
O	47	0.6267	0.6277	47.08
A <sub>1</sub>	12	0.1600	0.1523	11.42
A <sub>2</sub>	9	0.1200	0.1264	9.48
B	6	0.0800	0.0782	5.87
A <sub>1</sub> B	0	0.0000	0.0080	0.60
A <sub>2</sub> B	1	0.0133	0.0073	0.55
total	75	1.0000	0.9999	75.00

gene frequencies

$p_1$	0.0837
$p_2$	0.0761
$q$	0.0479
$r$	0.7923
total	1.0000

TABLE 2. THE MNS GROUPS OF THE YEMENITE JEWS

phenotype†	no. observed	frequency observed	frequency expected	no. expected
MMS	50	0.6667	0.6273	47.05
MsMs	9	0.1200	0.1353	10.15
MNS	7	0.0933	0.1647	12.35
MsNs	6	0.0800	0.0567	4.25
NNS	3	0.0400	0.0101	0.76
NsNs	0	0.0000	0.0059	0.44
total	75	1.0000	1.0000	75.00

frequencies of gene complexes

$MS$	0.5055
$Ms$	0.3678
$NS$	0.0496
$Ns$	0.0771
total	1.0000

† Seven He-positives were present, all MMS.

TABLE 3. THE Rh GROUPS OF THE YEMENITE JEWS

phenotype†	no. observed	frequency observed	frequency expected	no. expected
CCDee	17	0.2267	0.1936	14.52
CcDEe	10	0.1333	0.1584	11.88
CcDeeV	2	0.0267	0.0884	6.63
CcDee	20	0.2667	0.2460	18.45
ccDEE	2	0.0267	0.0324	2.43
ccDEeV	5	0.0667	0.0361	2.71
ccDEe	8	0.1067	0.1007	7.55
ccDeeV	7	0.0933	0.0662	4.97
ccDee	1	0.0133	0.0195	1.46
ccddee	3	0.0400	0.0587	4.40
total	75	1.0001	1.0000	75.00

frequencies of gene complexes

<i>CDe</i>	$R_1$	0.4400
<i>cDe</i>	$R_2$	0.1800
<i>cDeV</i>	$R_0^V$	0.1004
<i>cDe</i>	$R_0$	0.0374
<i>cde</i>	$r$	0.2422
total		1.0000

† All  $C^w$  negative

TABLE 4. THE DUFFY GROUPS OF THE YEMENITE JEWS

phenotype	no. observed	frequency observed	frequency expected	no. expected
Fy(a-b-)	46	0.6133	0.5866	44.00
Fy(a+b-)	19	0.2533	0.2813	21.10
Fy(a-b+)	6	0.0800	0.1094	8.20
Fy(a+b+)	4	0.0533	0.0227	1.70
total	75	0.9999	1.0000	75.00

gene frequencies

$Fy^a$	0.1657
$Fy^b$	0.0684
$Fy$	0.7659
total	1.0000

TABLE 5. SUNDRY BLOOD GROUPS OF THE YEMENITE JEWS

system	phenotype	no. observed	frequency observed	gene	gene frequency
P	$P_1+$	50	0.6667	$P_1$	0.4227
	$P_1-$	25	0.3333	$P_2(+p)$	0.5773
Lutheran	Lu(a+)	2	0.0267	$Lu^a$	0.0134
	Lu(a-)	73	0.9733	$Lu^b$	0.9866
Kell-Sutter	K-Js(a+)	1	0.0133	$kJ_s^a$	0.0067
	K-Js(a-)	74	0.9867	$kJ_s^b$	0.9933
Kidd	Jk(a+)	67	0.8933	$kJ^a$	0.6734
	Jk(a-)	8	0.3266	$kJ^b$	0.3266

TABLE 6. THE Gm GROUPS OF THE YEMENITE JEWS

phenotype Gm						no. observed	frequency observed	frequency expected	no. expected
1	2	4	5	10	11				
+	-	-	-	-	-	7	0.0933	0.0637	4.78
+	-	+	+	+	+	28	0.3733	0.4391	32.93
+	-	-	+	+	+	3	0.0400	0.0475	3.56
+	-	-	-	+	+	1	0.0133	0.0090	0.68
+	+	+	+	+	+	1	0.0133	0.0253	1.90
+	+	-	+	+	+	2	0.0267	0.0031	0.23
-	+	+	+	+	+	33	0.4400	0.4011	30.08
+	+	-	+	+	+	0	0.0000	0.0105	0.79
+	+	-	-	-	-	0	0.0000	0.0007	0.05
total						75	0.9999	1.0000	75.00

  

allele frequencies	
$Gm^1$	0.2524
$Gm^{4, 5, 10, 11}$	0.6333
$Gm^{1, 5, 10, 11}$	0.0770
$Gm^{1, 10, 11}$	0.0173
$Gm^{1, 2}$	0.0200
total	1.0000

TABLE 7. THE Inv GROUPS OF THE YEMENITE JEWS

phenotype Inv		no. observed	frequency observed	allele	allele frequency
1	2				
+	+	14	0.1867	$Inv^{1, 2}$	0.0982
-	-	61	0.8133	$Inv^3$	0.9018

TABLE 8. THE HAPTOGLOBINS OF THE YEMENITE JEWS

phenotype Hp	no. observed	frequency observed	frequency expected	no. expected
1	3	0.0395	0.1039	7.90
2-1	43	0.5658	0.4369	33.21
2	30	0.3947	0.4591	34.89
total	76	1.0000	0.9999	76.00

  

gene frequencies	
$Hp^1$	0.3224
$Hp^2$	0.6776
total	1.0000

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TABLE 9. THE ADENYLATE KINASE VARIANTS OF THE YEMENITE JEWS

phenotype AK	no. observed	frequency observed	frequency expected	no. expected
1	68	0.8947	0.8976	68.22
2-1	8	0.1053	0.0996	7.57
2	0	0.0000	0.0028	0.21
total	76	1.0000	1.0000	76.00
gene frequencies				
		<i>AK</i> <sup>1</sup>	0.9474	
		<i>AK</i> <sup>2</sup>	0.0526	
		total	1.0000	

TABLE 10. THE ACID PHOSPHATASE VARIANTS OF THE YEMENITE JEWS

phenotype AP	no. observed	frequency observed	frequency expected	no. expected
A	0	0.0000	0.0209	1.59
BA	17	0.2237	0.2152	16.36
B	42	0.5526	0.5528	42.01
CA	5	0.0658	0.0324	2.46
CB	12	0.1579	0.1662	12.63
C	0	0.0000	0.0125	0.95
total	76	1.0000	1.0000	76.00
gene frequencies				
		<i>P</i> <sup>a</sup>	0.1447	
		<i>P</i> <sup>b</sup>	0.7435	
		<i>P</i> <sup>c</sup>	0.1118	
		total	1.0000	

TABLE 11. THE PHOSPHOGLUCOMUTASE (*PGM*<sub>1</sub>) VARIANTS OF THE YEMENITE JEWS

phenotype <i>PGM</i> <sub>1</sub>	no. observed	frequency observed	frequency expected	no. expected
1	42	0.5526	0.5825	44.27
2-1	32	0.4211	0.3614	27.47
2	2	0.0263	0.0561	4.26
total	76	1.0000	1.0000	76.00
gene frequencies				
		<i>PGM</i> <sub>1</sub> <sup>1</sup>	0.7632	
		<i>PGM</i> <sub>1</sub> <sup>2</sup>	0.2368	
		total	1.0000	

TABLE 12. THE 6-PHOSPHOGLUCONATE DEHYDROGENASE VARIANTS OF THE YEMENITE JEWS

phenotype 6-PGD	no. observed	frequency observed	frequency expected	no. expected
A	62	0.8158	0.8243	62.65
CA	14	0.1842	0.1672	12.71
C	0	0.0000	0.0085	0.64
total	76	1.0000	1.0000	76.00
gene frequencies				
		<i>PGD</i> <sup>A</sup>	0.9079	
		<i>PGD</i> <sup>C</sup>	0.0921	
		total	1.0000	

TABLE 13. THE GLUCOSE 6-PHOSPHATE DEHYDROGENASE VARIANTS OF YEMENITE JEWISH MALES

phenotype†	no. observed	frequency observed
B+	34	0.9189
negative	3	0.0811
total	37	1.0000

† As these are X-linked characters the gene frequencies are identical with the phenotype frequencies in males. The gene present in negative males is presumed to be  $Gd^{B-}$ .

TABLE 14. THE  $A_1A_2BO$  BLOOD GROUPS OF THE KURDISH JEWS

group	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
O	24	0.2553	0.2532	23.80	8	0.2963	0.3301	8.91
$A_1$	27	0.2872	0.3046	28.63	10	0.3704	0.3009	8.13
$A_2$	7	0.0745	0.0602	5.66	0	0.0000	0.0293	0.79
B	24	0.2553	0.2610	24.53	8	0.2963	0.2504	6.76
$A_1B$	11	0.1170	0.0968	9.10	0	0.0000	0.0799	2.16
$A_2B$	1	0.0106	0.0242	2.28	1	0.0370	0.0094	0.25
total	94	0.9999	1.0000	94.00	27	1.0000	1.0000	27.00

## gene frequencies

	Iran	Iraq
$p_1$	0.2263	0.2131
$p_2$	0.0566	0.0250
$q$	0.2139	0.1874
$r$	0.5032	0.5745
total	1.0000	1.0000

TABLE 15. THE MNS BLOOD GROUPS OF THE KURDISH JEWS

phenotype	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
MMS	12	0.1276	0.1497	14.07	2	0.0741	0.0385	1.04
MsMs	15	0.1596	0.1741	16.37	2	0.0741	0.0853	2.30
MNS	30	0.3192	0.2674	25.14	3	0.1111	0.2237	6.04
MsNs	23	0.2447	0.2231	20.97	8	0.2963	0.2325	6.28
NNS	8	0.0851	0.1142	10.73	9	0.3333	0.2617	7.07
NsNs	6	0.0638	0.0715	6.72	3	0.1111	0.1583	4.27
total	94	1.0000	1.0000	94.00	27	1.0000	1.0000	27.00

## frequencies of gene complexes

	Iran	Iraq
$MS$	0.1518	0.0598
$M_s$	0.4173	0.2921
$NS$	0.1636	0.2502
$N_s$	0.2673	0.3979
total	1.0000	1.0000

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TABLE 16. THE Rh BLOOD GROUPS OF THE KURDISH JEWS

phenotype	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
CCDEE	0	0.0000	0.0001	0.01	—	—	—	—
CCDEe	2	0.0213	0.0123	1.16	—	—	—	—
CCDee	32	0.3404	0.3341	31.40	7	0.2593	0.2318	6.26
CCddee	0	0.0000	0.0021	0.20	—	—	—	—
CcDEE	0	0.0000	0.0051	0.48	—	—	—	—
CDE/cde†	0	0.0000	0.0036	0.34	—	—	—	—
Ce/cDE†	23	0.2447	0.2776	26.09	3	0.1111	0.1605	4.33
CcDee	18	0.1915	0.1918	17.10	9	0.3333	0.3388	9.15
Ccddee	2	0.0213	0.0155	1.46	—	—	—	—
ccDEE	8	0.0851	0.0573	5.38	1	0.0370	0.0278	0.75
ccDEe	6	0.0638	0.0815	7.66	4	0.1481	0.1173	3.17
ccddee	3	0.0319	0.0290	2.73	3	0.1111	0.1238	3.34
total	94	1.0000	1.0001	94.01	27	0.9999	1.0000	27.00

† Within the type CcDEe, an anti-CE reagent has been used to distinguish genotypes *CE/ce* and *Ce/cE*.

## frequencies of gene complexes

	Iran	Iraq
<i>CDE R<sub>z</sub></i>	0.0106	—
<i>CDe R<sub>1</sub></i>	0.5343	0.4815
<i>Cde r'</i>	0.0455	—
<i>cDE R<sub>2</sub></i>	0.2394	0.1667
<i>cde r</i>	0.1702	0.3518
total	1.0000	1.0000

TABLE 17. THE DUFFY GROUPS OF THE KURDISH JEWS

phenotype	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
Fy(a-b-)	4	0.0425	0.0231	2.17	2	0.0741	0.0489	1.32
Fy(a+b-)	34	0.3617	0.4041	37.98	8	0.2963	0.3424	9.24
Fy(a-b+)	17	0.1809	0.2253	21.18	7	0.2592	0.3058	8.26
Fy(a+b+)	39	0.4149	0.3475	32.67	10	0.3704	0.3029	8.18
total	94	1.0000	1.0000	94.00	27	1.0000	1.0000	27.00

## gene frequencies

	Iran	Iraq
<i>Fy<sup>a</sup></i>	0.5016	0.4044
<i>Fy<sup>b</sup></i>	0.3464	0.3745
<i>Fy</i>	0.1520	0.2211
total	1.0000	1.0000



TABLE 18. SUNDRY BLOOD GROUPS OF THE KURDISH JEWS

phenotype	no. observed		frequency observed		gene	gene frequency	
	Iran	Iraq	Iran	Iraq		Iran	Iraq
$P_1+$	65	15	0.6915	0.5556	$P_1$	0.4446	0.3334
$P_1-$	29	12	0.3085	0.4444	$P_2(+p)$	0.5554	0.6666
$Lu(a+)$	0	1	0.0000	0.0370	$Lu^a$	0.0000	0.0187
$Lu(a-)$	94	26	1.0000	0.9630	$Lu^b$	1.0000	0.9813
$K+\dagger$	3	0	0.0319	0.0000	$K$	0.0160	0.0000
$K-$	91	27	0.9681	1.0000	$k$	0.9840	1.0000
$Jk(a+)$	64	15	0.6809	0.5556	$Jk^a$	0.4351	0.3334
$Jk(a-)$	30	12	0.3191	0.4444	$Jk^b$	0.5649	0.6666

† No  $J_s(a+)$  specimens were found.

TABLE 19. THE  $G_m$  GROUPS OF THE KURDISH JEWS

phenotype† Gm	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
1 4 5 10 11								
+ - - - -	1	0.0114	0.0138	1.21	—	—	—	—
+ + + + +	33	0.3750	0.3541	31.16	8	0.3077	0.3106	8.08
+ - + + +	1	0.0114	0.0154	1.36	1	0.0385	0.0370	0.96
+ + - - -	1	0.0114	0.0154	1.36	—	—	—	—
- + + + +	52	0.5909	0.6012	52.91	17	0.6538	0.6524	16.96
total	88	1.0001	0.9999	88.00	26	1.0000	1.0000	26.00

† All specimens  $G_m^{2-}$  negative.

## allele frequencies

allele	Iran	Iraq
$G_m^1$	0.1176	—
$G_m^{4,5,10,11}$	0.7754	0.8077
$G_m^{1,5,10,11}$	0.0535	0.1923
$G_m^{1,4}$	0.0535	—
total	1.0000	1.0000

TABLE 20. THE  $Inv$  GROUPS OF THE KURDISH JEWS

phenotype Inv	no. observed		frequency observed		allele	allele frequency	
	Iran	Iraq	Iran	Iraq		Iran	Iraq
1 2							
+ +	1	0	0.0114	0.0000	$Inv^{1,2}$	0.0057	0.0000
- -	87	26	0.9886	1.0000	$Inv^3$	0.9943	1.0000

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TABLE 21. THE HAPTOGLOBINS OF THE KURDISH JEWS

phenotype	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
1	7	0.0753	0.0486	4.52	3	0.1111	0.1111	3.0
2-1	27	0.2903	0.3436	31.95	12	0.4444	0.4444	12.0
2	59	0.6344	0.6078	56.53	12	0.4444	0.4444	12.0
0	1	—†	—	—	—	—	—	—
total	94	1.0000	1.0000	93.00	27	0.9999	0.9999	27.0

† Hp0 is omitted from computations.

## gene frequencies

	Iran	Iraq
$Hp^1$	0.2204	0.3333
$Hp^2$	0.7796	0.6667
total	1.0000	1.0000

TABLE 22. THE ADENYLATE KINASE VARIANTS OF THE KURDISH JEWS

phenotype AK	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
1	82	0.8723	0.8765	82.39	26	0.9630	0.9633	26.01
2-1	12	0.1277	0.1194	11.22	1	0.0370	0.0363	0.98
2	0	0.0000	0.0041	0.39	0	0.0000	0.0003	0.01
total	94	1.0000	1.0000	94.00	27	1.0000	0.9999	27.00

## gene frequencies

	Iran	Iraq
$AK^1$	0.9362	0.9815
$AK^2$	0.0638	0.0185
total	1.0000	1.0000

TABLE 23. THE ACID PHOSPHATASE VARIANTS OF THE KURDISH JEWS

phenotype AP	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
A	11	0.1170	0.1195	11.23	3	0.1111	0.1238	3.34
BA	41	0.4362	0.4229	39.75	13	0.4815	0.4039	10.90
B	34	0.3617	0.3742	35.17	7	0.2593	0.3296	8.90
CA	2	0.0213	0.0295	2.77	0	0.0000	0.0521	1.41
CB	6	0.0638	0.0521	4.90	4	0.1481	0.0851	2.30
C	0	0.0000	0.0018	0.17	0	0.0000	0.0055	0.15
total	94	1.0000	1.0000	93.99	27	1.0000	1.0000	27.00

## gene frequencies

	Iran	Iraq
$P^a$	0.3457	0.3518
$P^b$	0.6117	0.5741
$P^c$	0.0426	0.0741
total	1.0000	1.0000

TABLE 24. THE PHOSPHOGLUCOMUTASE ( $PGM_1$ ) VARIANTS OF THE KURDISH JEWS

phenotype $PGM_1$	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
1	55	0.5851	0.5705	53.63	17	0.6296	0.6639	17.93
2-1	32	0.3404	0.3696	34.74	10	0.3704	0.3018	8.15
2	7	0.0745	0.0599	5.63	0	0.0000	0.0343	0.93
total	94	1.0000	1.0000	94.00	27	1.0000	1.0000	27.01

## gene frequencies

	Iran	Iraq
$PGM_1^1$	0.7553	0.8148
$PGM_1^2$	0.2447	0.1852
total	1.0000	1.0000

TABLE 25. THE 6-PHOSPHOGLUCONATE DEHYDROGENASE VARIANTS OF THE KURDISH JEWS

phenotype 6-PGD	Iran				Iraq			
	no. observed	frequency observed	frequency expected	no. expected	no. observed	frequency observed	frequency expected	no. expected
A	85	0.9043	0.9065	85.21	26	0.9630	0.9633	26.01
CA	8	0.0851	0.0811	7.62	1	0.0370	0.0363	0.98
C	0	0.0000	0.0018	0.17	0	0.0000	0.0003	0.01
HA	1	0.0106	0.0101	0.95	—	—	—	—
HC	0	0.0000	0.0005	0.05	—	—	—	—
H	0	0.0000	0.0000	0.00	—	—	—	—
total	94	1.0000	1.0000	94.00	27	1.0000	0.9999	27.00

## gene frequencies

	Iran	Iraq
$PGD$	0.9521	0.9815
$PGD^c$	0.0426	0.0185
$PGD^H$	0.0053	0.0000
total	1.0000	1.0000

TABLE 26. THE GLUCOSE 6-PHOSPHATE DEHYDROGENASE VARIANTS OF MALE KURDISH JEWS

phenotype† G6PD	Iran		Iraq		total	
	no. observed	frequency observed	no. observed	frequency observed	no. observed	frequency observed
B+	35	0.6140	6	0.4286	41	0.5775
negative	22	0.3860	8	0.5714	30	0.4225
total	57	1.0000	14	1.0000	71	1.0000

† As these characters are X-linked, the gene frequencies are identical with the phenotype frequencies in males. The negative type is presumed to carry the gene  $Gd^{B-}$ .

Some near relatives are included in all the series tested, but as the numbers are already small it has been thought best to retain all results rather than cut down these numbers still further. The Yemenite Jews came from a fairly compact single area and so have all been combined for the purpose of gene-frequency calculations. There were, however, some differences, probably to be attributed to founder effects, between those from the village of Bitha and those from Pedwin. The Kurdish Jews were at first classified by their places of residence in Israel and in their case only very slight differences were found between the present local communities. When, however, they were divided according to whether they came from the portion of Kurdistan falling in Iraq or that in Iran, marked differences were seen, and this subdivision has therefore been adopted in the tables, although only 27 from Iraq were tested.

#### THE YEMENITE JEWS

The Jews from the Yemen show very high O and low B frequencies which agree well with those found in the non-Jewish inhabitants of most parts of Arabia, but almost nowhere else in Asia (Maranjian, Ikin, Mourant & Lehmann 1966).

For the MNSs system, again, the very high total *M* and especially the high *MS* are characteristic features of the peoples of Arabia generally. The relatively high frequency of the African Henshaw gene in the Jews is noteworthy.

In the Rh system, frequencies agree well with the rest of Arabia. This applies not only to the frequencies of the typically caucasoid gene complexes *CDe* and *cde*, but also to the negroid *cDe* and *V*. Rather surprisingly, no K-positives were found, for Arabians generally have some of the highest frequencies known of the *K* gene, around 10%. One example of the negroid *J<sub>s</sub><sup>a</sup>* antigen of the Kell system was found. The typically negroid amorph *Fy* gene of the Duffy system has an unexpectedly high frequency. The *Jk<sup>a</sup>* gene of the Kidd system, which has a very high frequency in Africans, is found at well above the normal European level. In the immunoglobulin systems, the negroid allele *Gm<sup>1,5,10,11</sup>* is present and the *Inu<sup>1,2</sup>* allele is above the European level. Most of the typically negroid genes were found at a higher frequency in Bitha than in Pedwin.

With one exception the gene frequencies of the plasma protein and red-cell enzyme systems so far examined fit well into the regional pattern. The exception is the acid phosphatase system in which the *P<sup>o</sup>* gene has rarely been found outside Europe. It is almost completely absent from the Arab population of Sinai (Bonné *et al.* 1971) and appears to be completely so from that of southern Arabia, which suggests that it is also absent from the nuclear population of central Arabia. Yet it is present in extremely high frequency in the Yemenite Jews and relatively high in the Habbanites.

Its presence at a high level in this isolate, though absent from the surrounding Arab tribes, suggests that it has been maintained among the former since their ancestors left Palestine more than 1500 years ago. The maintenance of a relatively rare gene in a small isolate suggests some active balanced polymorphism, perhaps related to their special mode of life, or the operation of genetic drift. Alternatively we may suppose that it has been kept up by occasional intermarriage with members of other Jewish communities, for even the extremely isolated and almost forgotten Habbanites were found to possess medieval Jewish writings, including the Talmud, the writings of Maimonides and Spanish poetry. However, any entry of genes from outside must have been sufficiently small to avoid disturbing the genetic similarity with the southern Arabs, which holds for most genetic systems other than acid phosphatase.

Some of the authors have examined specimens from a number of South Arabian tribes, collected by Dr A. Marengo-Rowe. The unpublished results show that there is on the whole little difference between the frequencies of typical negroid blood group genes in these tribes and in the Yemenite Jews, but where there is a difference the Jews tend to have more negroid genes. A small isolate of Jews formerly living in Habban, a town and region between the Yemen and the Hadhramaut, quite isolated from the main Yemenite Jews, has been studied in collaboration with Dr B. Bonn . In spite of their high degree of isolation and inbreeding, this population has a distinctly higher frequency of negroid genes than either the southern Arabs or the Yemenite Jews (Bonn  *et al.* 1970).

#### THE KURDISH JEWS

In view of the unusual genetic traits of the Kurdish Jews tested in Israel, it appeared to be desirable to compare them on the one hand with other Oriental Jewish communities and on the other with the Kurds and other neighbouring peoples of western Iran, northern Iraq and south-eastern Turkey. Both for this reason and because of their intrinsic interest, a detailed study has recently been made of the Kurds of Iran, among whom most of the Kurdish Jews examined in the present investigation, or their parents, had lived (Lehmann *et al.* 1973, this volume). Our knowledge of the blood factors of other surrounding indigenous non-Jewish populations must be considered very incomplete and patchy in relation to the ethnic diversity of the region, and our information on these factors in other Jewish communities from east of the River Euphrates is mainly confined to their ABO, Rh and MN groups, and the frequency of glucose 6-phosphate dehydrogenase deficiency.

A large proportion of the Kurdish Jews who migrated to Israel came from northern Iraq, and previous work refers largely to these, whereas the present investigation concerns communities who came mainly from western Iran. The Kurdish Jews, like the Kurds themselves and like most other peoples of the region, have rather high frequencies of both the *A* and the *B* genes. However, Gurevitch, Hermoni & Margolis (1953-4) and Gurevitch & Margolis (1955) report *A* gene frequencies of 36 and 32 % respectively in Kurdish Jews from Iraq, apparently two separate samples. Both these figures are higher than our observation of 28 % in Kurdish Jews from Iran and much higher than the 24 % in our very small sample of those from Iraq. It is thus almost certain that the frequency of the *A* gene is higher in the Jews from Iraq than in those from Iran, and it may be related to the high *A* frequencies found in the neighbouring Turkish and Armenian populations.

There is also a considerable difference between the MN frequencies found in Kurdish Jews from Iraq and from Iran, 57 and 35 % respectively of *M* genes. The second figure is almost certainly due to the smallness of the sample, and the *M* gene frequency of 53 % found by Gurevitch & Margolis (1955) for Jews from Iraqi Kurdistan is probably much nearer the truth. From the combined data there is almost certainly a genuine increase in *M* frequencies from west to east among these Jews.

In none of these populations, however, are *M* frequencies anywhere near as high as in the Yemenite Jews and other Arabian populations. The low frequency of *MS*, and high *NS*, in the Kurdish Jews, also contrast strongly with findings in the Yemenite Jews.

The frequencies of the Rh gene complexes in the Kurdish Jews, with relatively high *CDe* and *cDE* and low *cde*, fit well into the regional picture. A somewhat surprising finding is the absence of the negroid *cDe* and *V*. The former was, however, found by Gurevitch *et al.* (1953)



and by Gurevitch & Margolis (1954). Differences in the Rh system between the Iranian Kurdish Jews and the Iranian Kurds are very small.

The relatively low  $P_1$ ,  $Lu^a$  and  $K$  frequencies in both these populations are about what would be expected in this region. The frequencies of around 15% of the amorph  $Fy$  gene in both Kurds and Jews, suggest that this gene, usually regarded as of African origin, extends as an indigenous feature into southwest Asia. The frequency of the  $Jk^a$  gene is below European levels and far below those for African populations.

Examination of the haptoglobins shows an  $Hb^1$  gene frequency in Iranian Kurdish Jews of 22%. This is somewhat below the figures for Iranian Kurds, for other Iranians, and for south-east Europeans, and within the range of those for India and southeastern Asia.

Nearly all populations tested for adenylate kinase variants show an  $AK^1$  gene frequency above 90% (Rapley, Robson, Harris & Maynard Smith 1967, Tills, Van den Branden, Clements & Mourant 1970a, 1971a); it is therefore convenient to consider variations in the frequency of the next most common gene,  $AK^2$ . European frequencies of the latter are around 4%; the gene is extremely rare in Africa. The frequencies in Iranian Kurds are over 7%, in other Iranians 5%, and in the peoples of the Indian region (including Cochin Jews) 10 to 15%. Iraqi Jews have 4.7% of this gene. Thus the Iranian Kurdish Jews with 6.4% agree well with the local distribution.

In the phosphoglucosylase system, variants of interest in population studies are mainly those at the  $PGM_1$  locus;  $PGM_2$  variants are very rare and almost entirely confined to Africa (Giblett 1969). The  $PGM_1^1$  gene is the commonest, followed by  $PGM_1^2$  with a frequency of 24% in the Iranian Kurdish Jews. This is indeed the approximate level found in most peoples of the Old World, with certain notable exceptions. In particular, while the Yemenite Jews have 24% of this gene, the Habbanite Jews have the extremely high frequency of 58%, presumably as a result of genetic drift. The Jews of Cochin, India, probably for the same reason, have 59%.

In the 6-phosphogluconate dehydrogenase system the Iranian Kurdish Jews have 4.3% of  $PGD^c$ , the second most common gene. Frequencies of  $PGD^c$  seldom differ greatly from this level in Europe, western Asia and Africa, but in the western group of Iranian Kurds the level exceeds 7%, and in Bhutan it was found to be 25% (Mourant *et al.* 1968; Tills, Van den Branden, Clements & Mourant 1970b, 1971b). As we have seen, it is 9% in the Yemenite Jews, and frequencies between 10 and 20% are common in southwest Asia. One example of a heterozygote for the very rare  $PGD^H$  gene was also found among the Iranian Kurdish Jews.

The frequencies of the variants of glucose 6-phosphate dehydrogenase are of particular interest, and it was indeed these which first drew special attention to the population genetics of the Kurdish Jews (Cohen *et al.* 1963). Tests on the latter in Israel have consistently shown very high frequencies for the deficient type of gene. In many series the frequency exceeds 50%, and the only other known population with about equally high frequencies consists of the Shi'a Moslems of the oases of eastern Saudi Arabia (Gelpi 1965).

It has long been known that in the Mediterranean area there is a tendency for high frequencies of the deficiency to be present in populations in which malaria was formerly endemic, and it has been concluded that the deficient gene has been favoured by natural selection in a malarious environment. In Africa several workers (Allison & Clyde 1961; Gilles *et al.* 1967; Bienzle, Ayeni, Lucas & Luzzato 1972) have shown that the deficiency has a direct protective effect against *falciparum* malaria; there is, however, an electrophoretically detectable difference between the Mediterranean and African types of enzyme with deficient activity.

Haemoglobin S also is known to protect against *falciparum* malaria, and Lehmann *et al.* (1963) have shown that in Saudi Arabia high frequencies of the Hb S gene are confined to the inhabitants of the eastern oases, but the frequencies of the abnormal gene are much higher in the Shi'a Moslems who are the indigenous inhabitants of the oases than in the Sunni who are recent immigrants from the rest of Arabia. The Sunni also appear to have much lower frequencies of G6PD deficiency than the Shi'a, but the evidence is not quite conclusive. Though the religious dichotomy goes back to the very early days of the Moslem religion, Maranjian *et al.* (1966) have demonstrated that there is very little difference between the blood group frequencies of the members of the two sects who have at the same time been shown to share a number of marked genetic characteristics with the rest of the inhabitants of the Arabian peninsula, including the Yemenite Jews, characteristics which distinguish the peoples of the peninsula from all surrounding populations (who are nearly all Moslems).

While many parts of Iran were formerly malarious, and moderate frequencies of G6PD deficiency are indeed widespread, it seemed unlikely that there had been a high incidence of the disease in mountainous areas such as Kurdistan. Nevertheless, the present group of Iranian Kurdish Jews showed a frequency of the deficient gene of 39% (and the small sample from Iraq, 42%).

The two main groups of indigenous Kurds tested (Lehmann *et al.* 1973, this volume) showed, on the other hand, frequencies of only 6.5 and 2.8% respectively. We must therefore look for a cause for the high frequency in the Jews (and perhaps also in the Shi'a Moslems) in some circumstances peculiar to their own ancestry or mode of life. The gene itself may have accompanied the Jewish community, which presumably migrated a very long time ago from the region of Baghdad, or perhaps even directly from northern Israel at the time of the Assyrian deportations. It is unlikely, however, that it is solely through inbreeding that the high frequency found in this community has persisted and even increased in the long subsequent period, in the absence of some special environmental influence favouring this normally slightly deleterious gene. It seems unlikely that there has been any substantial amount of immigration in recent centuries from Baghdad, or from more malarious parts of Kurdistan, for our relatively slight historic information suggests the existence of communities that, by comparison with other Jews, were singularly immobile. One is led to look for some cultural peculiarity resembling in a general way found by Undevia (1969) in the Parsis of Bombay. These have a significantly higher incidence of G6PD deficiency than their Hindu neighbours, and he shows that this was associated with a former much higher incidence of malaria in the Parsi community. Public health investigations had shown that this was due to the open bathing tanks attached to Parsi dwellings which, though favouring hygiene in other respects, also enabled malarial mosquitos to breed.

Though the parallel is by no means complete, it may be remarked that the Shi'a Moslems of eastern Arabia are likewise the descendants of a very ancient inbred religious group originating (and in this case living until very recently) in a malarious environment. There is undoubtedly a close connexion between the distributions of the genes for G6PD deficiency, haemoglobin S, thalassaemia (which we have not considered here) and exposure to *plasmodium falciparum*. The problems presented are complex and we can claim only to have applied a few additional pieces of the jig-saw puzzle, and perhaps shown how some of them may fit together.

There is, as pointed out by Lightman, Carr-Locke & Pickles (1970) another genetic system where a rather great difference exists between Iranian Kurds and Kurdish Jews (Ashkenazi *et al.*

1963). This is the phenylthiocarbamide taster system: the Kurds include 27% of non-tasters, whereas there are only 14% among Kurdish Jews. The difference is statistically highly significant.

One may sum up by saying that the Kurdish Jews show a broad but by no means close agreement, in the frequencies of their genetic factors, with the other populations of the region in which they lived, with the notable exception of their very high frequency of glucose 6-phosphate dehydrogenase deficiency. This is in contrast to the case of the Yemenite Jews, who show quite a close agreement in most respects, including low frequency of this deficiency, with the local Arab pattern. This would suggest a much higher degree of exclusive endogamy in the Kurdish than in the Yemenite Jewish community.

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