Genetic polymorphisms in selected populations in south west and south Asia

Sawhney, Kanwarjit

GENETIC POLYMORPHISMS IN SELECTED POPULATIONS

IN SOUTH WEST AND SOUTH ASIA

By

KANWARJIT SINGH SAWHNEY

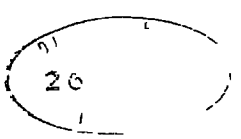
A THESIS SUBMITTED FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

DEPARTMENT OF ANTHROPOLOGY

UNIVERSITY OF DURHAM

1975



ABSTRACT

This thesis describes the genetic polymorphisms in selected populations in South West and South Asia. Samples from Kuwait and Iran represented the Middle East, while the Panjabis of northern India and the Nepalese illustrated the South of Asia. The methods used to detect these polymorphisms were serological and electrophoretic techniques. A total of 162 Kuwaitis were screened for 5 blood group , 2 serum protein and 6 enzyme systems. No variants were detected in the transferrin, lactate dehydrogenase and malate dehydrogenase systems. The Kuwaitis were found to fit reasonably well into the known Arabian gene frequency distributions with regard to many genetic parameters. Two groups from Iran - the Tehran (196) and Isfahan Iranians (112) - were similarly tested for 7 blood group , 2 serum protein and 7 enzyme systems, as well as for abnormal haemoglobin variants. The groups were found to be genetically homogeneous. Of especial interest was the finding of an appreciable frequency of the Lutheran gene in the Isfahanis and a high frequency of the ABO gene q in the Tehranis.

In the Indian Sub-continent, a series of 360 Panjabis tested for six blood groups were compared with selected populations of northern India. Phenotype and gene frequencies were found to be comparable with the northern values. Of the 313 specimens, typed for 2 serum proteins, a single individual exhibited a transferrin CB variant. The results of six enzyme systems did not reveal notable frequency differences between the Panjabis and the state populations of India. The Nepalese (212) examined for the forementioned systems were compared with selected populations from India and South East Asia. Their results differed from the Indians but showed resemblances with the Mongoloids. Finally, a general comparison was made and some of the characteristic features were discussed in the light of factors that influence them.

CONTENTS

LIST OF FIGURES

INTRODUCTION

Anthropology defined as the study of the human species is primarily concerned with ethnology, phylogeny and taxonomy. Ethnology includes the archaeological study of the prehistoric fossil man and his evolutionary progenitors. Hence the study of primates closely related to man is vital in the determination of man's place in the animal kingdom and his origin. Phylogeny on the other hand looks into the origin and inter-relationship of the different human races. Numerous questions regarding racial affinity and differences, whether or not these relationships are the result of human adaptibility in response to environmental and other selective forces have been raised. To facilitate the comparative study of races, a taxonomy of man is therefore essential. However, race taxonomy has been a controversial topic in the study of Anthropology. Race taxonomies in the past depended almost exclusively on the descriptive physical characters of man. But these taxonomic units are prone to environmental influences and subjective judgements which are clearly recognised by anthropologists. Equally important is the realization that racial differences transcend differences in size and appearance and extend to the metabolism, biochemical functioning and other immunochemical properties. Here then lies the important link between biological anthropology and biochemical genetics.

Since the 1900s, a new class of physical characters, the blood groups, has entered into the realm of modern Anthropology. These characters are susceptible to accurate statistical analysis. Their mode of inheritance is simple, straightforward and abide by Mendelian laws. The gene frequencies of the populations tested can be calculated from the observable phenotypic frequencies. The differences of gene frequencies observed in the populations lead to the study of natural

selection, migration, racial admixture, mutation, disease resistance and environmental influences like season, climate etc. The fact that these biochemical traits once genetically determined at conception will remain fixed for life (Mourant 1954) makes them very reliable taxonomic tools. Hence a classification based on these biochemical traits will be more scientific and fundamental in character than that based on external morphology.

Although the ABO blood group by itself is of limited use in the comparative study of human populations, when combined with the other blood group systems like MNSs, P, Rhesus, Lutheran, Kell, Lewis, Duffy, Kidd, Diego, Yt, Dombrock and Auberger, it is very useful in anthropological research. Such a serological taxonomy helps to solve many puzzles in Anthropology. The world distributions of blood groups have been extensively reviewed by a number of workers (Boyd 1939, Race and Sanger 1950, Mourant 1954 and Mourant et al 1958). Suffice it to say that the blood group systems are invaluable in the study of races, and the development of palaeoserology - a technique of blood typing material from the dead - makes it possible to investigate gene frequencies of not only the contemporary populations but also of their ancestors and other extinct populations.

Apart from the blood group systems discussed earlier, the new biochemical markers such as serum proteins and red cell isoenzymes are of immense importance in anthropological research. Such markers of human blood exhibit hereditary variations in different populations. These variant forms occur so frequently that they cannot be considered as recurrent mutations but could be considered to be polymorphic in man according to Ford's definition (Ford 1940). These biochemical markers are being extensively used in modern population surveys. It is the purpose of this thesis to study in detail these polymorphic systems in selected populations of South West and South Asia.

The organization of this thesis is as follows :

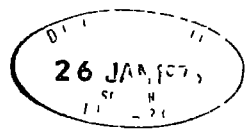
Chapter one gives an account of the polymorphic systems employed in this investigation. Chapter two summarizes the collection of samples, different serological techniques, various electrophoretic methods and statistical techniques used to interpret the data. The analysis of the data collected in Kuwait is given in Chapter three The data on the two Iranian samples - the Tehran Iranians and the Isfahan Iranians - is included in Chapter four. Chapter five deals with the Panjabis results and their comparison with other Indian populations. In Chapter six, the Nepalese material is given and compared with selected populations of India and South East Asia. Finally, in Chapter seven, a general comparison is made and some of these characteristics are discussed in the light of evolutionary mechanisms.

CHAPTER 1

AN ACCOUNT OF POLYMORPHIC SYSTEMS EMPLOYED IN THE PRESENT INVESTIGATION

1.1 Introduction

During the last fifty years, and especially over the last twenty years, the discovery of new techniques has led to the detection of many genetically determined polymorphisms in man. Genetic traits now commonly investigated in population surveys include the blood group antigens, the serum proteins and red cell isoenzymes. The most important technique developed in recent years is the starch-gel electrophoresis (Smithies 1955[a]). Following its application, workers like Smithies and Walker made a major contribution to the establishment of genetic individuality in man by demonstrating the genetic control of the phenotypic expression of the serum proteins, the haptoglobins. In the early 60s Harris and his co-workers extended this work to study the extent of such individuality in the red cell isoenzymes. It is with these genetic polymorphic systems that the present study is concerned. All these systems introduced below have been reviewed separately, touching on their discovery and genetics.



1.2 Blood Group Antigens

1.2.1 ABO blood group system

The earliest observations on differences between the blood of normal human individuals were made by Landsteiner (1900). He tested red cells and sera and found that in some cases agglutination occurred, whereas in others there was no reaction. On the basis of these agglutination reactions, Landsteiner (1901) was able to divide human beings into three distinct groups and, with the discovery of Von Decastello and Sturli (1902) of a fourth group, the system now known as ABO was completed.

The classification of four groups - A, B, AB and O - is based on the presence of blood group substances situated on the surface of the red cells. The red cells of an individual possess either one or both, or neither of the antigens, A and B; and his serum possesses either one or other, neither, or both of the antibodies, anti-A and anti-B. Red cells containing antigen A are agglutinated by anti-A, cells containing antigen B by anti-B. Both anti-A and anti-B agglutinate AB cells, while neither of them react with O cells. The relationships are shown in Table 1.1.

Table 1.1 The ABO blood group antigens and antibodies

Blood Groups	Antigens on red cells	Reactions with anti-A	Reactions with anti-B	Antibodies present in serum
A	A	+	-	anti-B
B	B	-	+	anti-A
AB	A and B	+	+	None
O	None	-	-	anti-A anti-B

The inheritance of blood group characters, based on Mendelian principles, was reported by Dungern and Hirszfeld (1911). Bernstein (1924) showed that the four groups were inherited by means of three allelic genes A, B and O (sometimes also called p, q and r). The A gene determines the presence of A antigen on the red cells and is present on one or both the chromosomes. The B gene similarly determines the presence of B antigen. The O gene does not determine the presence of either of these antigens, but neither does it suppress them if the other allelic gene present is the antigen producing A or B. Thus, the four blood groups represent six genotypes as shown below in Table 1.2.

Table 1.2 The ABO groups and their corresponding genotypes

Groups	Genotypes
A	AA) AO)
B	BB) BO)
O	OO
AB	AB

In 1930, Thomsen, Friendenreich and Warsaae discovered that antigen A could be further divided into two groups, A_1 and A_2, with a corresponding subdivision of groups A and AB into A_1, A_2 and A_1B and A_2B respectively. Anti-A reacts with both antigen A_1 and antigen A_2, but anti-A_1 reacts only with antigen A_1. Red cells classified as A_1 are agglutinated by anti-A and anti-A_1, whereas cells classified as A_2 are agglutinated by anti-A and do not show any reaction

with anti-A_1. Both antigens, A_1 and A_2, are produced by corresponding allelic genes. A_1 is dominant to A_2 and O, A_2 is dominant to O, neither A_1 nor A_2 is dominant to B. Thus, the recognition of four alleles - A_1, A_2, B and O - leads to the expectation of ten different genotypes and six phenotypes as shown in Table 1.3.

Table 1.3 The A_1A_2BO genotypes and groups

Genotypes	Groups or Phenotypes
OO	O
A_2O, A_2A_2	A_2
A_1O, A_1A_2, A_1A_1	A_1
BB, BO	B
A_1B	A_1B
A_2B	A_2B

1.2.2 MNSs blood group system

Landsteiner and Levine (1927a and b) were the first to describe the existence of two human antigens, which they called M and N. Antibodies against M and N are not usually found in humans, but are prepared by injecting human blood into rabbits. The serum of the rabbit injected with M blood agglutinates both M and MN erythrocytes, and the serum treated with N blood agglutinates N and MN erythrocytes.

The inheritance of these antigens is based on the two allele theory advanced by Landsteiner and Levine in 1928. According to this theory there are two alleles, M and N, either of which determines the presence of corresponding antigens on the red cells. Thus there are three genotypes MM, MN and NN and three corresponding phenotypes M, MN and N.

In 1947, Walsh and Montgomery reported the existence of another antigen in an Australian blood sample. The new antigen called S was shown to be serologically different from M and N (Sanger and Race 1947). Family studies suggested that persons who possess S are homozygous or heterozygous for one allele and persons who do not have S are homozygous for another allele. In 1951, Levine et al discovered the expected antithetical antibody anti-s which agglutinated the red cells of homozygotes as well as heterozygotes, thus indicating that there are two antigens, S and s, and three blood types, S, Ss and s. Table 1.4 demonstrates the genetical interpretation of serological reactions of the complete MNSs system.

Table 1.4 Genetical interpretation of the reactions of anti-M, anti-N, anti-S and anti-s sera

Anti M	N	S	s	All 4 Sera : genotype	First 3 Sera : genotype or phenotype
+	-	+	-	MS/MS)	MM.S
+	-	+	+	MS/Ms)	
+	-	-	+	Ms/Ms	Ms/Ms
+	+	+	-	MS/NS)	MN.S
+	+	+	+	(M^S/N_s) (Ms/NS)	
+	+	-	+	Ms/Ns	Ms/Ns
-	+	+	-	NS/NS)	NN.S
-	+	+	+	NS/Ns)	
-	+	-	+	Ns/Ns	Ns/Ns

(After Race and Sanger 1950)

The relationship of the M and N gene locus to that for S and s is considered to be very close, and it becomes difficult to postulate whether these are two gene loci close together on the same chromosome or whether complex alleles at a single gene locus are concerned in producing both MN and Ss substances. Race and Sanger (1970) suggested that the linkage between the loci is very close since crossing over occurs very occasionally.

1.2.3 Rhesus blood group system

One of the most important works in the field of blood groups was the discovery of the Rh blood group system by Landsteiner and Wiener in 1940. They showed that the antibodies, produced by immunizing rabbits and guinea pigs with the red cells of the monkey *Macacus rhesus*, not only agglutinated the red cells of the monkey but also about 85% of the people of European descent. Red cells agglutinated by anti-rhesus serum were classified as Rh-positive and those showing no agglutination were classified as Rh-negative. The antibody was shown to be indistinguishable from that reported in Levine and Stetson's case of 1939. Finally, in 1941 Levine and his colleagues showed that the destruction of the red cells in the new born was due to sensitization of a Rh-negative mother by a Rh-positive child.

Further investigations showed that the antigens of the Rhesus system and the genes responsible for them were complicated. The nomenclature used to communicate the various findings of Rh system was based on two classic hypotheses concerning the genetics of the system, one of which was suggested by Fisher and Race (cf Race and Sanger 1962), the other by Wiener (cf Wiener and Wexler 1963). In the present investigation, the nomenclature of Fisher and Race was used.

According to Fisher's hypothesis there are three closely linked genes, arranged in a linear sequence D, C and E. At each gene locus there are two main alternative genes named C and c, E and e, D and d. The occurrence of the d antigen was presumed, but it has never been demonstrated. As only one of each pair can be carried on each chromosome, there are eight alternative Rhesus gene combinations. and these are shown in Figure 1.5. Considering the fact that these Rh gene complexes occur in three orders of frequency in the English population (Frequent DCe, dce, DcE; Infrequent : Dce, dce, dCe, DCE; Very infrequent . dCE), Fisher-Race suggested that the very infrequent combinations may be maintained by crossing over from the three frequent complexes. The emergence of DCE was shown due to a cross over with one of the infrequent complexes Dce, dce, dCe and DCE.

E.....CDE

D

e.....CDe

C

E.....CdE

d

e.....Cde

E.....cDE

D

e.....cDe

c

E.....cdE

d

e.....cde

Figure 1.5 Alternative Rhesus Chromosomes

The Wiener hypothesis suggests that the Rh antigens are produced by a series of multiple alleles of one gene. He postulates that a gene gives rise to an agglutinogen and this in turn possesses a number of blood factors, whereas the Fisher-Race hypothesis makes no such distinction between genes and antigens. The relationship between the Wiener and Fisher-Race notation is set out in Table 1.6.

Table 1.6 Comparison of the Fisher-Race linked gene theory and the Wiener multiple allele theory

CDE SYSTEM			Rh-Hr SYSTEM		
Gene Complex	Symbol	Antigens	Gene	Agglutinogen	Blood Factors
CDE	R_z	C,D,E	R^z	Rh_z	rh', Rh_o, rh"
CDe	R_1	C,D,e	R^1	Rh_1	rh', Rh_o, hr"
cDE	R_2	c,D,E	R^2	Rh_2	hr', Rh_o, rh"
ede	r	c,D,e	r	rh	hr', hr", hr
cDe	R_o	c,D,e	R^o	Rh_o	hr', Rhohr", hr
cdE	R"	c,d,E	r"	rh"	hr', hr"
Cde	R'	C,d,e	r'	rh'	rh', hr"
CdE	R_y	C,d,E	r_y	rh^y	rh', rh"

A number of additional alleles have been reported by various investigators to occur at the Rh loci. They are labelled D^u, C^w, C^u, C^x, E^w, E^u, e^s and e^1. However, with the exception of D^u and C^w, all these alleles are unusual or rare in Caucasian populations. Specific antibodies demonstrating the antigens D^u, C^u, E^u and e^1 have not been found.

Although the Rh system is one of the most complex genetic systems investigated in man, it has been of great value in describing the gene pools of various human populations. Detailed information regarding racial differences in the frequency of gene complexes has been reported by Mourant (1954).

1.2.4 Lutheran blood group system

In 1945 the antibody which defines the Lutheran blood group system was found in the serum of a patient suffering from *lupus erythematosus diffusus* (Callender, Race and Paykoc 1945). The antibody was shown to be immune in nature. Family studies indicated that the antigen Lu^a was inherited as a Mendelian character, and the blood group system is controlled by two genes. The notation suggested for the system was as follows :

genes	:	Lu^a, Lu^b
phenotypes	:	Lu (a+), Lu (-)
antibodies	:	anti-Lu^a, anti-Lu^b (to be discovered)

Ten years later, in 1956, the expected antibody anti-Lu^b was described by Cutbush and Chanarin (1956). The finding of anti-Lu^b established the existence of the antigen and gene Lu^b. In 1961, Crawford et al showed the system to be more complicated with the detection of a new phenotype that reacted like Lu(a-b-) to the known antibodies. Finally, an antibody Lu^aLu^b was discovered which reacted with all cells except those of phenotype Lu(a-b-) (Darnborough et al 1963). The following Table 1.7 indicates the correspondence of genotypes and phenotypes of the Lutheran system.

Table 1.7 Phenotypes and genotypes of the Lutheran system

Reactions with :			
anti-Lu^a	anti-Lu^b	Phenotypes	Genotypes
+	-	Lu(a+b-)	Lu^aLu^a
+	+	Lu(a+b+)	Lu^aLu^b
-	+	Lu(a-b+)	Lu^bLu^b

1.2.5 Kell blood group system

The antibody which recognised the Kell antigen was described by Coombs, Mourant and Race in 1946. The antigen called K was found to occur in 10 percent of the British population and appeared to be inherited as a dominant Mendelian character. The discovery of the expected antithetical antibody anti-k by Levine et al (1949) made it clear that the system was governed by a pair of allelomorphic genes, K and k, which control the production of the corresponding antigens K and k. The groups of the system as defined by anti-K and anti-k are shown in Table 1.8.

Table 1.8 Phenotypes and genotypes of the Kell system

Red cells React with :	Phenotypes	Genotypes
anti-K	K	KK
anti-K and anti-k	Kk	Kk
anti-k	k	kk

A new antigen called Kp^a was reported by Allen and Lewis (1957). Family studies showed that it was associated with the Kell system. Later, the same workers described an anti-Kp^b antibody. It was suggested that these antigens were produced by a pair of allelomorphic genes within the Kell system.

1.2.6 Duffy blood group system

In 1950, Cutbush, Mollison and Parkin reported the discovery of the Duffy system, unrelated to the ABO, MNS, Rhesus and Kell blood types. The antibody anti-Fy^a was discovered in the serum of an individual suffering from haemophilia and who had received a number of blood transfusions over a period of twenty years. Family studies indicated that the antigen was inherited by means of a gene expressing itself in a single and double dose. The discovery of an antithetical antibody, antiFy^b, by Ikin et al (1951), suggested that the system was governed by two allelic genes, Fy^a and Fy^b.

A third allele in the Duffy system was postulated when Sanger et al (1955) discovered that the red cells of many American Negroes did not react with either antiserum and the phenotype was like Fy(a-b-). This allele, now known as Fy, is exceedingly rare in Whites but is quite common in Negroes. The existence of the Fy allele in Whites was first reported by Chown et al (1965). Table 1.9 demonstrates the genetical interpretation of the Duffy system.

Table 1.9 Phenotypes and genotypes of the Duffy system

Reactions with :		Interpretation	Phenotypes
anti-Fy^a	anti-Fy^b		
+	-	Fy^aFy^a) Fy^aFy)	Fy(a+b-)
+	+	Fy^aFy^b	Fy(a+b+)
-	+	Fy^bFy^b) Fy^bFy)	Fy(a-b+)
-	-	FyFy	Fy(a-b-)

1.2.7 Kidd blood group system

The Kidd blood group system was discovered by Allen, Diamond and Niedziela in 1951. The finding of this 'new' antibody was the result of a haemolytic disease in a newborn infant. The antigen,called JK^a, was shown to be independent of the other blood group systems. Family studies carried out by Race et al (1951) showed that the antigen was inherited by means of a gene capable of expressing itself in single or double dose. The phenotypes and genotypes of the system as defined by anti-JK^a were as follows :

Phenotypes	Genotypes
JK(a+)	JK^aJK^a) JK^aJK^b)
JK(a-)	JK^bJK^b

The existence of the expected antibody, anti-JK^b, was described by Plaut et al (1953). At present, tests have been made with both antisera and it has been suggested that the genes are inherited as non-dominant autosomal alleles. A new phenotype like JK(a-b-) was reported by Pinkerton et al (1959) in Filipinos with some Spanish and Chinese ancestry Such individuals were presumed to be homozygous for a third allele JK. The genetic background of the JK(a-b-) phenotype is not yet clear.

1.2.8 Diego system

The Diego blood group system was first reported by Layrisse, Arends and Dominguez in 1955. The antibody, anti-Di^a, was the result of haemolytic disease of the new born. In 1967, Thompson et al identified the expected antithetical antibody anti-Di^b. It has been suggested that the system is controlled by two allelic genes, Di^a and Di^b. No phenotype like Di(a-b-) has yet been reported.

Information about the distribution of the Diego system has been summarised by Layrisse and Wilbert (1960). According to them the Diego antigen appears to be confined to ethnic groups of Mongoloid origin. Geographically, it is distributed widely, having been found in South American Indians, Mexican Indians, Japanese, Chinese and in mixtures of Mongoloids with other ethnic groups. It is excremely rare in Caucasians, North American and African Negroes.

1.3 Serum Proteins

1.3.1 Haptoglobin

The first report of the existence of the plasma protein now known as 'haptoglobin' was made by Polonovski and Jayle (1938). Smithies (1955b) demonstrated that when human serum is subjected to electrophoresis in starch gel as a supporting medium, haptoglobins differentiate into one of three patterns : I, IIA and IIB. The groups were later termed as Hp1-1, Hp2-1 and Hp2-2.

Family study carried out by Smithies and Walker (1955-1956) suggested that the three groups are controlled by two autosomal genes, Hp^1 and Hp^2, without dominance. Homozygous Hp^1 individuals are type 1-1, homozygous Hp^2 are type 2-2 and heterozygotes are type 2-1. Figure 1.10 illustrates the electrophoretic patterns of common haptoglobin types. Type 1-1 has a single, intensely staining band somewhat slower than free haemoglobin. Hp2-2 has no band in the position of the Hp1-1 component, but slower bands of diminishing intensity towards the origin. These bands vary in width and the extent to which they stain. Hp2-1 has one band with the same mobility as that of Hp1-1, but it stains less strongly and contains a series of bands that move more slowly than the Hp1-1 component.

Smithies et al (1962) and Connell et al (1962) reported that the haptoglobin molecules can be broken by reducing their disulphide groups and carrying out electrophoresis in an acid gel urea. This revealed that haptoglobin consists of two kinds of polypeptide chains, designated as α- and β-chains. The β-chains are found to be the same in all the three Hp types, whereas structural variations in the α-chains show different electrophoretic patterns of the intact molecules. Thus, a series of phenotypes in the

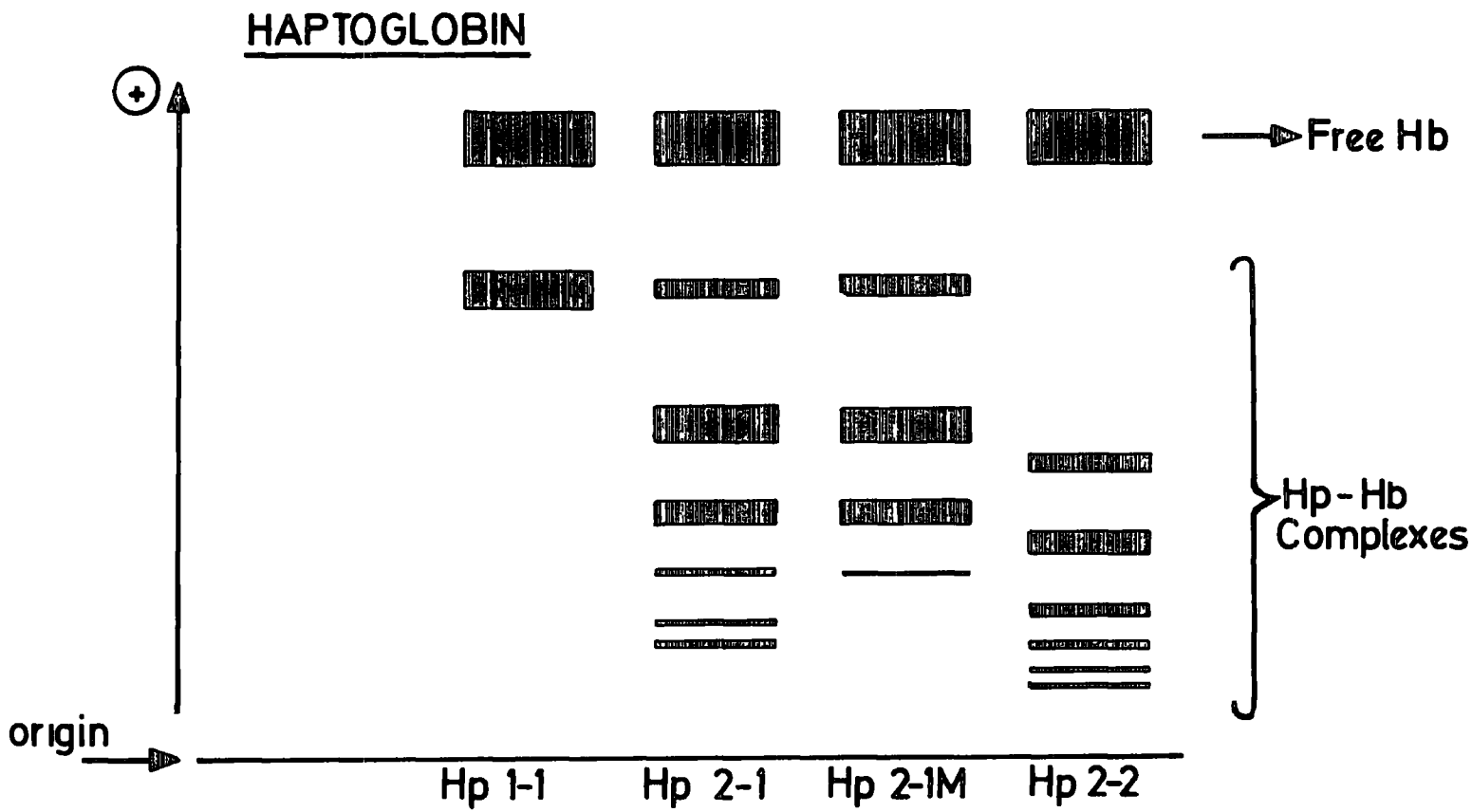


Fig 1 10 Diagram showing the common Haptoglobin Phenotypes.

haptoglobin system can now be recognised. Individuals with type Hp1-1 can have a fast migrating α-polypeptide (Hp1F), a slow migrating α-polypeptide (Hp1S) and a mixture of both Hp1F and Hp1S. Type 2-2 persons exhibit only one band which is considerably slower than Hp1F or Hp1S. Type 2-1 persons have Hp-2 band combined either with Hp1F or Hp1S. These subtypes are controlled by two alleles, Hp^{1F} and Hp^{1S}.

A considerable number of rare phenotypes found in the haptoglobin system are classified as quantitative and qualitative variants. The first report of a quantitative variant called 2-1M or 2-1 (modified) was made by Connell and Smithies (1959). Allison et al (1958) described another interesting missing haptoglobin in Nigerians. This type, designated as Hp0-0 or anhaptoglobinaemia, is also found in other populations. There is, as yet, no genetic explanation for this type. Other rare types of this category are Hp2-1 'Carlberg', Hp2-1 (Haw) and Hp2-1 (Trans.). The qualitative variants are very rare and the best known are Hp-1J, Hp-2J and Hp-Mb.

Factors affecting the maintenance of the Hp^{2} gene have been attributed to selective advantage conferred by environment such as malaria (cited in Walter and Steegmüller 1969). It is also well established that the capacity of haptoglobin to bind haemoglobin varies considerably among Hp types. The Hp1-1 type has a greater capacity than others. It is possible that the relatively high incidence of haemolytic conditions, especially anaemias, known to occur among tropical populations, is related to a possible advantage in haemolytic binding capacity, which one haptoglobin type may have over others. From this it has been inferred that Hp2-2 is selected against in areas with prevalent haemolytic diseases (Baxi and Camoens 1969a).

1.3.2 Transferrin

Transferrin or siderophilin is the iron-binding protein component of the serum. Smithies (1957) described a slowly migrating β- globulin component in human serum termed β- globulin D, which occurred together with the normal β- globulin C. A faster migrating β- globulin B was observed in White Canadians by Smithies (1958). Family studies by Horsfall and Smithies (1958) suggested that the genes responsible for the variation were genetically determined. Smithies and Miller (1959) reported that the formation of β- globulins B, C, D is determined by three co-dominant autosomal allelic genes, Tf^B, Tf^C and Tf^D, the C gene being much more common than the B or D genes. At present, the polymorphism of transferrin is attributed to 19 allelic genes, and about 28 phenotypes have been reported (Giblett 1969). Figure 1.11 illustrates the twenty eight phenotypes, of which twenty one are heterozygous and seven are homozygous (TfC, D_1, D_{chi}, B_2, B_1, B_{Lae} and Bo-1).

Walter and Bajatzadeh (1971) suggested that the frequencies of the Tf alleles are not distributed equally within the human species and show marked racial differences with respect to alleles, Tf^C and Tf^D. Races living in tropical biotopes show high Tf^D frequencies and the populations in non-tropical biotopes have low frequencies. They assumed that such geographical distribution is a result of selective adaptations to particular environmental conditions. Ashton (1965) reported a positive association between the slow variant, Tf^E, and a tolerance to hotter climates in cattle. The assumption that Tf is associated with resistance to infectious diseases such as malaria appears to be unfounded (Curtain et al 1965). Selective mechanisms which might be responsible for maintaining the four common "aberrant" phenotypes in certain populations are still unknown.

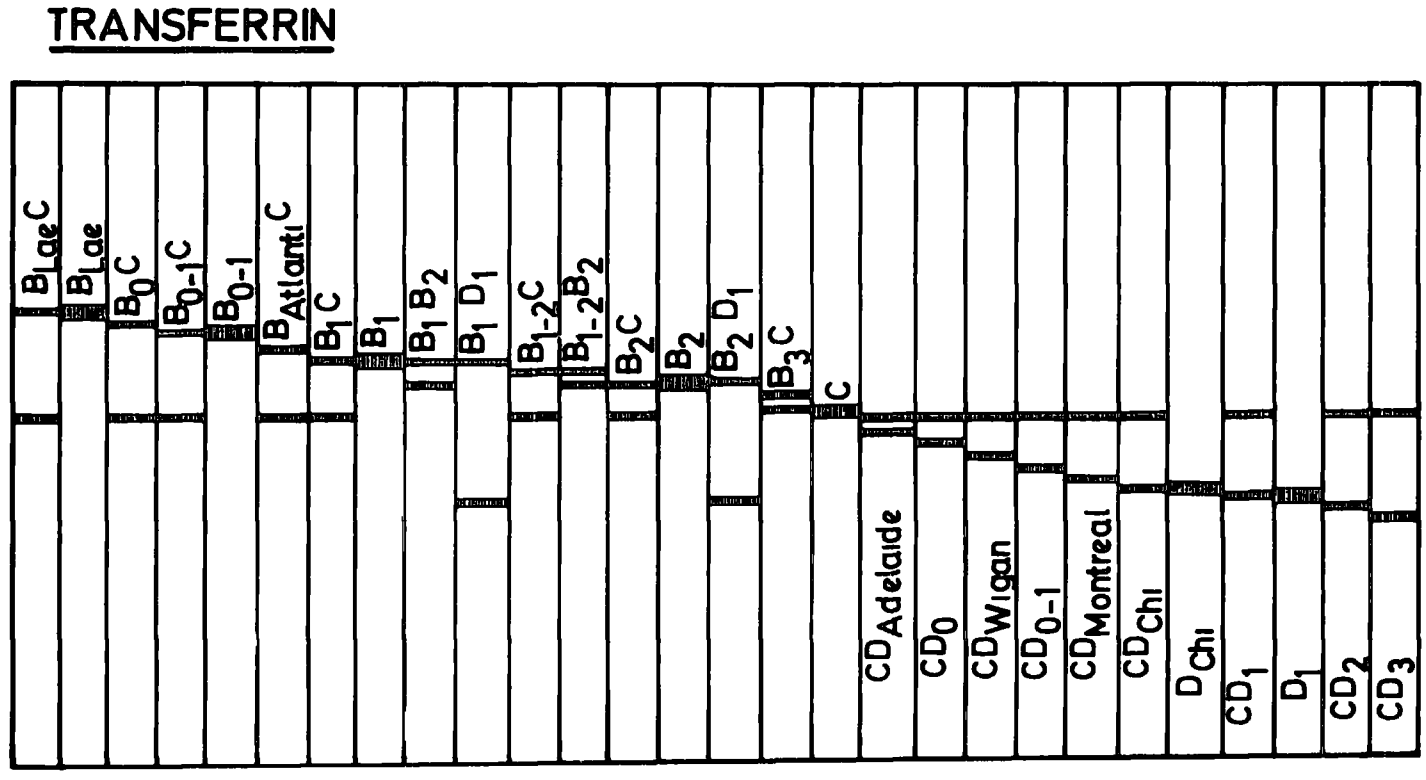


Fig.1-11. Diagram showing electrophoretic variants of Transferrin (After Giblett 1969).

1.4 Red Cell Isoenzymes

1.4.1 Acid phosphatase

Hopkinson et al (1963) found that the enzyme acid phosphatase which catalyses the reaction involving phosphorus transfer exists in several heritable forms. They demonstrated five distinct electrophoretic patterns, designated A, BA, B, CA and CB (Figure 1.12) The sixth pattern called homozygous C was detected in Brazilian families by Lai et al (1964). Figure 1.12 shows that each type consists of two or more isozymes. The isozyme of type A exhibits two bands of equal intensity, and migrates faster than the other homozygous types. Type B has two bands but the faster band is more active than the slow band. The mobility of the B band is slightly faster than the A band. The isozyme of type C has the same electrophoretic mobility, but the slow band is much more active than the fast band. The pattern in types BA, CA and CB appears to be the mixture of the isozymes of their corresponding homozygous types. Family studies indicate that the different types are controlled by three co-dominant alleles, P^a, P^b and P^c, at an autosomal locus. Phenotypes A, B and C represent homozygous genotypes P^aP^a, P^bP^b, P^cP^c respectively, and phenotypes BA, CA and CB represent heterozygous genotypes P^aP^b, P^aP^c and P^bP^c.

In addition to the common types, two rare phenotypes, RA and RB, were reported by Giblett and Scott (1965) and Giblett (1967). The isozyme of type RA is characterised by a pair of fast moving components with either A or B or C zones. Type RB consists of two bands which migrate faster towards the anode. P^r is therefore suggested as a fourth allele and was confirmed by Karp and Sutton (1967). It is found to determine approximately the same amount

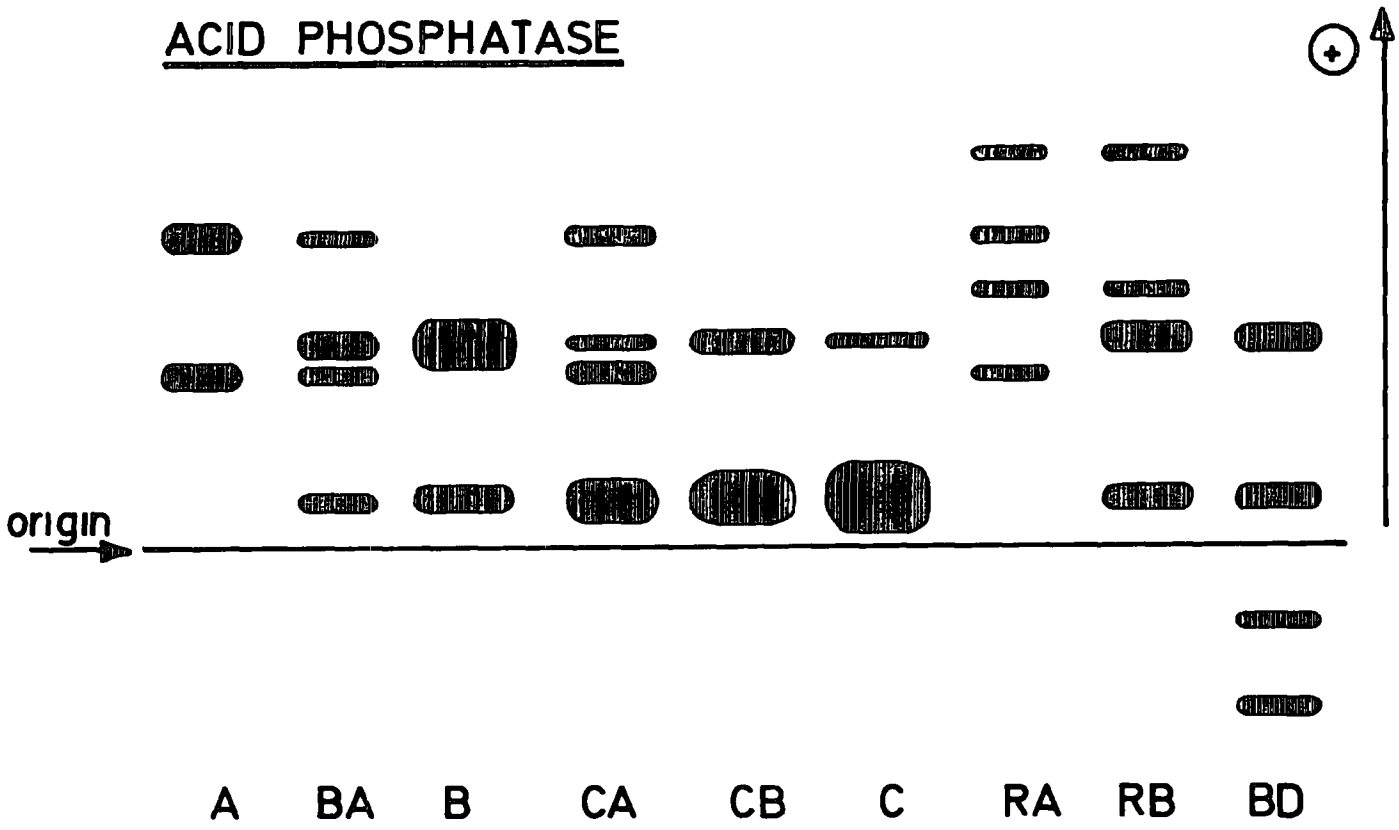


Fig 1.12. Diagram showing electrophoretic variants of Acid Phosphatase.
(After Hopkinson 1969)

of AP as does the P^a allele, and is allelic to P^a and P^b (Jenkins and Corfield 1972). The detection of another rare phenotype BD pointed to the existence of a fifth allele, P^d. Type BD has the two most cathodal bands which are not observed in other types. Lamm (1970) described DA and DB variants in a Danish family and found the P^d allele segregating at the AP locus.

Walter and Bajatzadeh (1968) attributed the high incidence of P^b to selective factors specific to tropical living conditions. Ananthakrishnan and Walter (1972) supported this suggestion by finding a possible correlation between decreasing P^a frequency and increasing mean annual temperature. Jenkins and Corfield (1972) speculated that selection could be responsible for the current low P^c frequencies. They further suggested that the P^c allele could be the product of a recent mutation which is advantageous.

1.4.2 Phosphoglucomutase

Phosphoglucomutase is a phosphotransferase which catalyses the transfer of a phosphate group between the 1- and 6-positions of glucose. Spencer et al (1964) demonstrated that when red cell lysates are subjected to starch gel electrophoresis, seven different zones of PGM can be observed (a - g, Figure 1.13). Three common phenotypes are referred to as PGM1-1, PGM2-1 and PGM2-2. The a and c isozymes are present in type 1 and type 2-1 but not in type 2. Isozymes b and d are absent on type 1 but occur in type 2-1 and type 2. Isozymes e, f and g are present in all the three types. Phenotype 2-1 includes the components present in types 1 and 2.

Family studies show that the three types are determined by two autosomal allelic genes, PGM^1 and PGM^2. Phenotypes PGM1-1 and

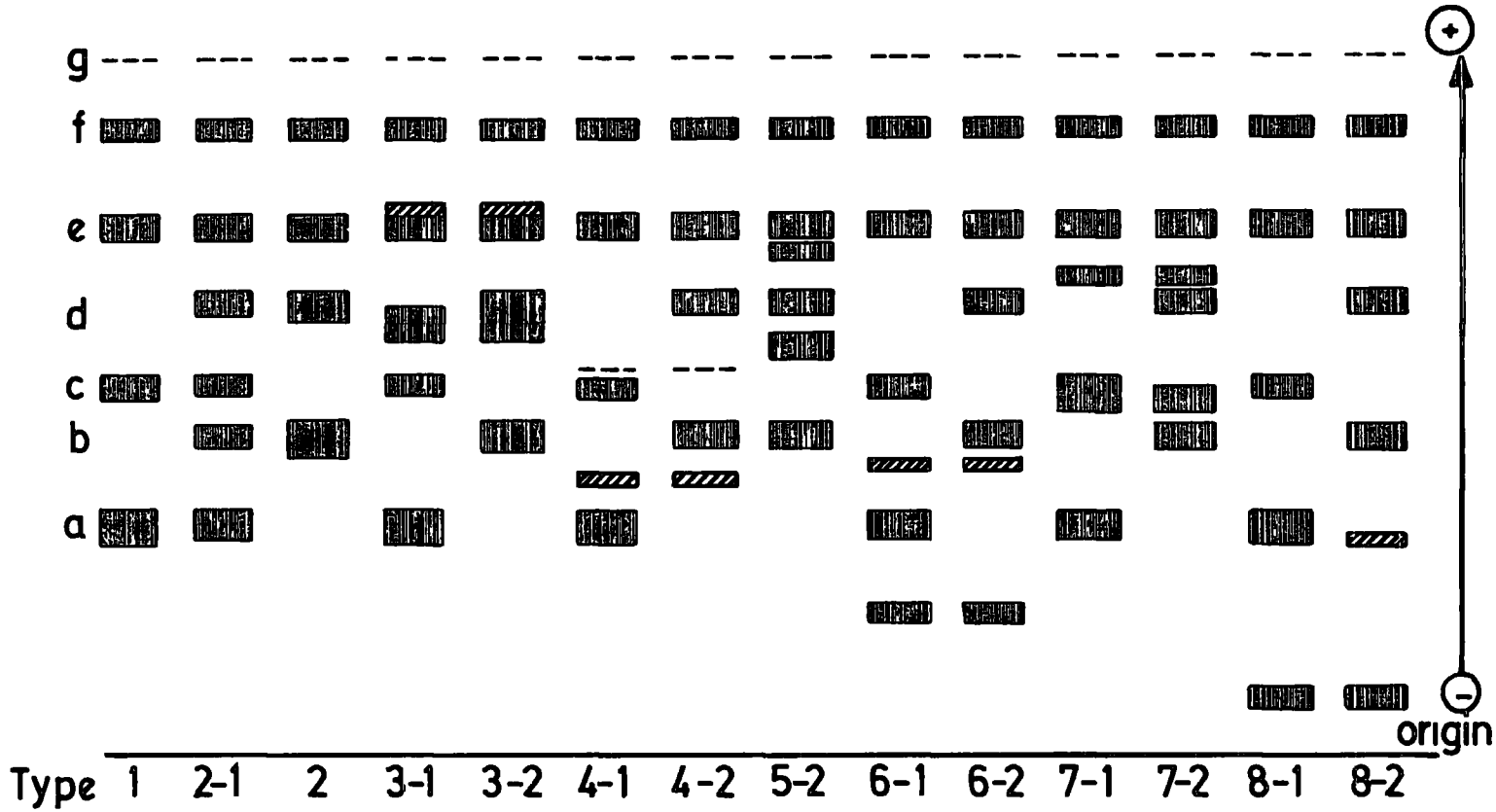


Fig 1.13. Diagram showing electrophoretic variants of Phosphoglucomutase.
(After Hopkinson and Harris 1966).

PGM2-2 correspond to the homozygotes PGM^1PGM^1 and PGM^2PGM^2 respectively and phenotype PGM2-1 represents the heterozygote PGM^1PGM^2. The isozymes a and c are controlled by the PGM^1 gene while b and d isozymes are products of the allelic gene PGM^2. A number of rare alleles have been shown to occur at the PGM_1 locus (Hopkinson and Harris 1966). Figure 1.13 illustrates these rare phenotypes observed in human red cells. They are determined by six alleles designated as PGM_1^3, PGM_1^4, PGM_1^5, PGM_1^6, PGM_1^7 and PGM_1^8.

At present nothing is known of the factors that maintain the genetic polymorphism of the PGM_1 locus.

1.4.3 Adenylate kinase

The enzyme adenylate kinase catalyses the reversible reaction, 2-adenosine diphosphate $\rightleftharpoons$ adenosine triphosphate + adenosine monophosphate within the red cells and other tissues. The polymorphism of the human red cell adenylate kinase was first described by Fildes and Harris (1966). Three different isozyme patterns referred to as AK1-1, AK2-1 and AK2-2 are shown in Figure 1.14. AK1-1 shows two active zones, of which the most intense is found near the margin and the less active zone towards the anode. The third zone is fast (very anodal), weak and generally diffused. AK2-1 consists of the same three zones but in addition a fourth zone which moves towards the cathode is present. The pattern seen in AK2-1 represents a mixture of isozymes present in AK1-1 and AK2-2. Population and family studies indicate that the variants of AK are genetically determined by two co-dominant autosomal alleles, AK^1 and AK^2, such that the genotypes AK^1AK^1, AK^2AK^2 and AK^2AK^1 determine the phenotypes AK1-1, AK2-1 and AK2-2. This hypothesis was confirmed by the studies of Bowman et al (1967), Rapley et al (1967) and Berg (1969).

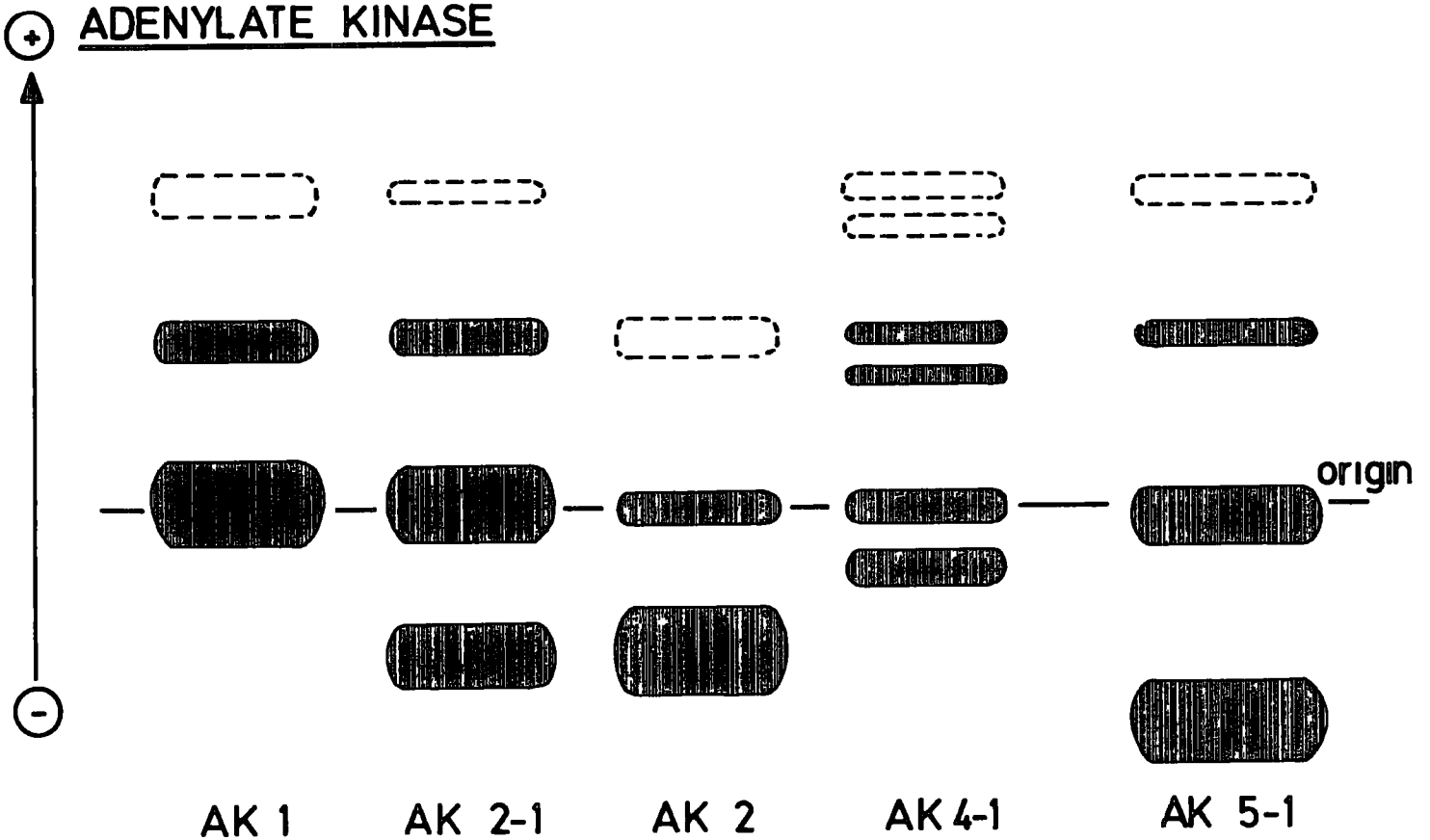


Fig. 1 14 Diagram showing electrophoretic variants of Adenylate Kinase.

In addition to the common types, three variants referred to as AK3-1, AK4-1 and AK5-1 have been described (Bowman et al 1967, Rapley et al 1967, Benercetti et al 1972). Figure 1.14 shows that type 4-1 exhibits a series of bands which migrate more slowly than the usual isozyme of AK1-1. The fastest moving band is anodal and very faint. Isozyme 3-1 shows a series of components which migrate faster than AK1-1. Type 5-1 consists of two major bands . One corresponds to the gene product of the common AK^1 allele and the other occupies a position more cathodic than that of the AK2-2 main band. Individuals with such aberrant phenotypes are heterozygotes for the usual allele AK^1 and the new allele AK^3, AK^4 and AK^5.

1.4.4 6-Phosphogluconate dehydrogenase

The enzyme 6-phosphogluconate dehydrogenase catalyses the conversion of 6-phosphogluconate to ribulose-5-phosphate and involves an important step in the hexose monophosphate shunt leading to the conversion of hexoses to pentoses for the biosynthesis of nucleic acids. Genetically determined variation of 6-PGD was first demonstrated by starch gel electrophoresis of human haemolysates (Fildes and Parr 1963). The distinct electrophoretic patterns are shown in Figure 1.15. Isozyme patterns of the usual type (AA) consist of a single anodal bond a. The 'common variant' (CA) exhibits an anodal band (a) and a cathodal band (b). Bands a and b have equal intensity. A third variant, designated the 'canning variant' (CC) consists of bands a, b and c, with the anodal band as a minor component. Family studies show that the inheritance of these variants is controlled by two alleles, PGD^A and PGD^C. It is assumed that the enzyme 6-PGD is a dimer consisting of two subunits, S^A and S^C. The S^A unit dimerizes to form S^CS^C. The heterovariant is thought to be a mixed dimer, S^AS^C (Parr and Fitch 1967).

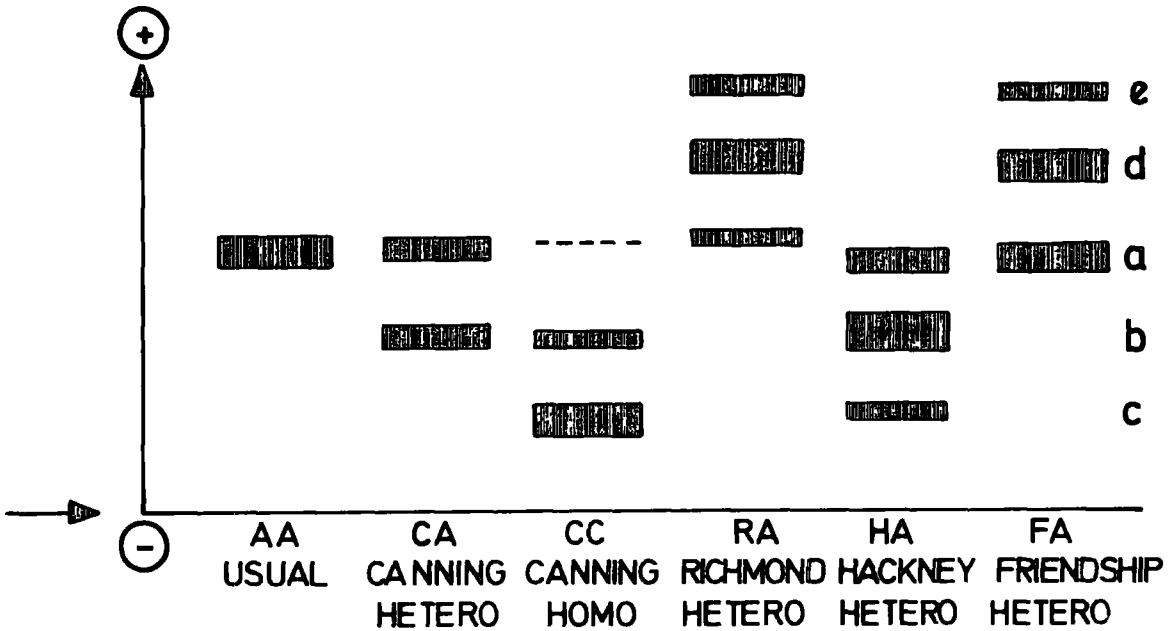


Fig. 1.15 Diagram showing electrophoretic variants of red cell 6 Phosphogluconate Dehydrogenase

Apart from common variants, a rare phenotype called the 'Richmond variant' was detected in an American family by Davidson (1967). Figure 1.15 illustrates the phenotypic pattern of this variant. It consists of three bands, a, d and e. The most cathodal band corresponds to the usual a band, while the intermediate band is intensively stained and predominates. Another rare variant called the 'Hackney variant' was reported by Parr (1966). It differs from others in showing slow mobility of all the bands. The 'Friendship variant' described by Parr (1966) exhibits bands a, d and e (Figure 1.15). The most anodal band, e, is weakly stained, and the other two cathodal bands are of equal intensity. All these variants are controlled by a group of alleles at a single autosomal locus and are found normally in the heterozygous state.

1.4.5 Glucose-6-phosphate dehydrogenase

Glucose-6-phosphate dehydrogenase is an essential enzyme in the human system. Most of the information relating to the biochemical, genetic and anthropological aspects of the enzyme have emerged from the study of red cells in which G-6PD forms part of the hexose shunt, converting G-6-phosphate to 6-phosphogluconate. It is inherited as a sex linked gene. The deficiency is expressed fully in homozygous males, but its expression varies in heterozygous females. Some heterozygous females are entirely normal, some have intermediate enzyme activity, and some are enzyme deficient.

Investigations of the enzyme in different human populations have shown that a wide variety of genetic variants exists. Two molecular forms, the B and A types, recognised by their electrophoretic mobility on starch gel, are usual (Figure 1.16). Caucasians, black Africans and Asians

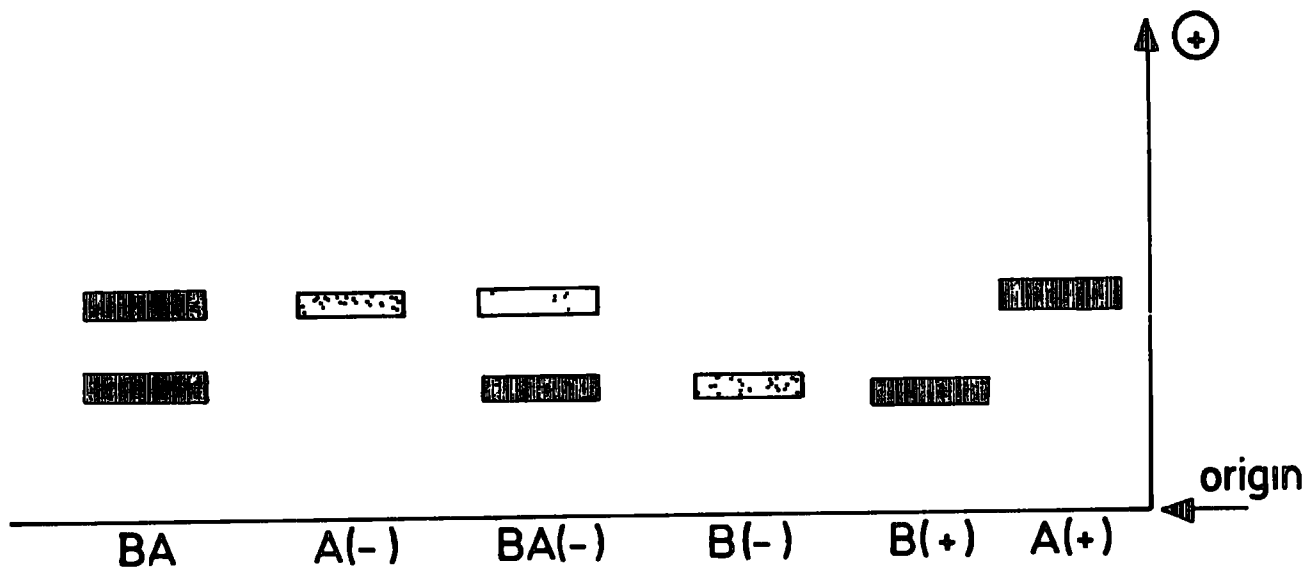


Fig 1 16 Diagram showing electrophoretic variants of Glucose-6-Phosphate Dehydrogenase

usually possess the B type, designated as B(+) (WHO 1966). Enzyme deficient persons in Caucasian populations also have the B type with very low enzyme activity designated as B(-). The enzyme present in Africans shows an increased electrophoretic mobility and is referred to as A(+). American Negroes,who are not enzyme deficient, possess an enzyme of high electrophoretic mobility, designated as A(-), or simply A. It is assumed that variants of G-6PD represent single amino-acid substitutions caused by specific base pair mutations within the structural gene for G-6PD. Motulsky and Yoshida (1969) demonstrated the exact nature of amino-acid substitution and showed that the mutation for the A-type is not associated with the enzyme deficiency observed in Negroes.

1.4.6 Lactate dehydrogenase

Lactate dehydrogenase catalyses the interconversion of lactate and pyruvate with the oxidation and reduction of coenzyme NAD. The normal LDH consists of five isozymes. Each isozyme is a tetramer composed of electrophoretically distinct polypeptide subunits. The subunits are referred to as A and B. They combine randomly to give isozymes with four peptides. The structure of each isozyme is as follows :

LDH-1 = BBBB

LDH-2 = BBBA

LDH-3 = BBAA

LDH-4 = BAAA

LDH-5 = AAAA

LDH-1 with four B subunits is the fastest moving isozyme, whereas LDH-5 with four A subunits is the slowest one. LDH from erythrocytes shows three distinct electrophoretic patterns (Figure 1.17). Enumerating,

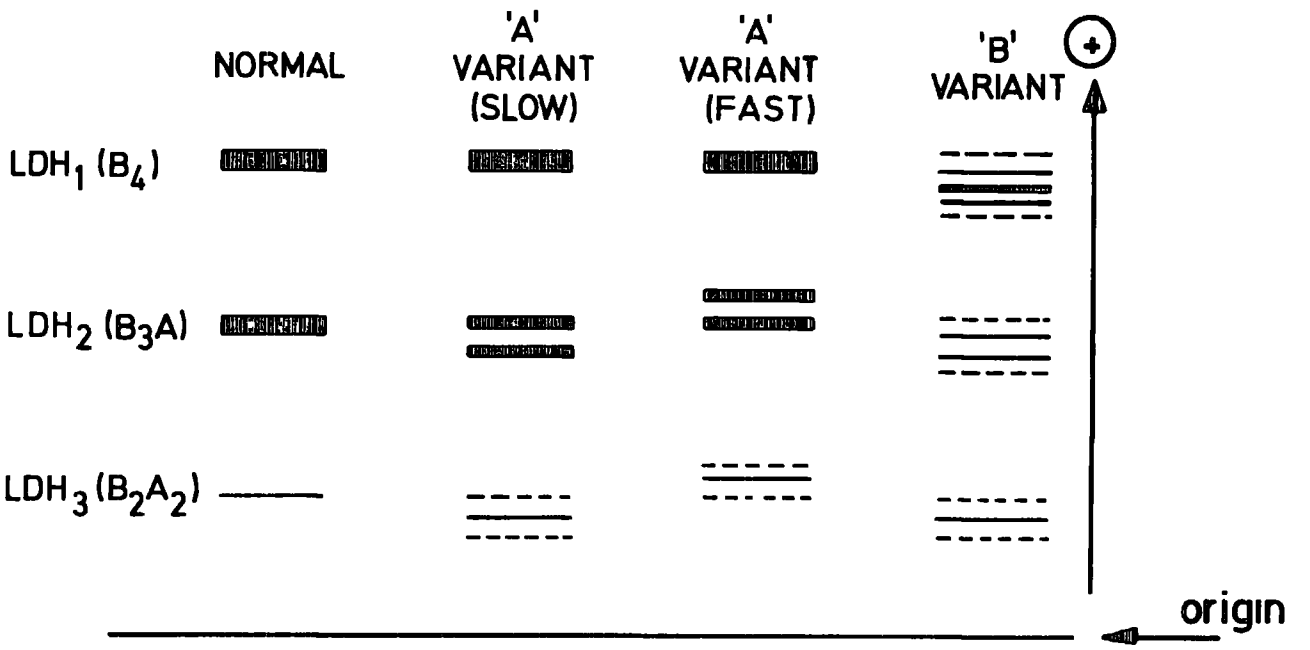


Fig 1.17 Diagram showing electrophoretic patterns of Lactate Dehydrogenase (Drawn from Harris 1969).

from the anodal end. they correspond to the isozymes, LDH-1 (BBBB), LDH-2 (BBBA) and LDH-3 (BBAA).

A B-subunit variant of LDH was first demonstrated by Boyer et al (1963) in the erythrocytes of a Nigerian male. The variant exhibited five components in the position of the LDH-1 zone, four in the LDH-2, three in the LDH-3. and two in the LDH-4. LDH-5 was not demonstrable in the haemolysate. The data indicated autosomal inheritance. Another LDH variant of A subunit was described by Nance et al (1963) in a Brazilian family. The isozyme pattern of the variant showed no alteration in the LDH-1 zone. LDH-2 had two components of which the slower one corresponded with the normal LDH-2. The LDH-3 zone exhibited only two components. Changes in electrophoretic patterns were thought to be due to mutation at the A- and B- loci.

Recently, four genetically deteimined variants of LDH were reported in Indian populations (Das et al 1970, Ananthakrishnan et al 1970, Das et al 1972). They are referred to as 'LDH Cal-1', 'LDH Mad-1', 'LDH Cal-2 and 'LDH Del-1' respectively. LDH Cal-1 is a Faster-A subunit variant and its zymogram exhibited a single LDH-1 band, a double LDH-2b and a triple LDH-3 band. LDH Cal-2 is faster and had distinct 'A'-subunit variation. LDH Mad-1 and LDH Del-1 are classified as slower 'B'-subunit variants.

1.4.7 Malate dehydrogenase

Malate dehydrogenase catalyses the reversible conversion of malate to oxaloacetate. Two forms of malate dehydrogenase are found in man, one in cytoplasm and the other in mitochondria. The forms have different chemical and physical properties. The genetically controlled polymorphism of cytoplasmic MDH was first described by Davidson and

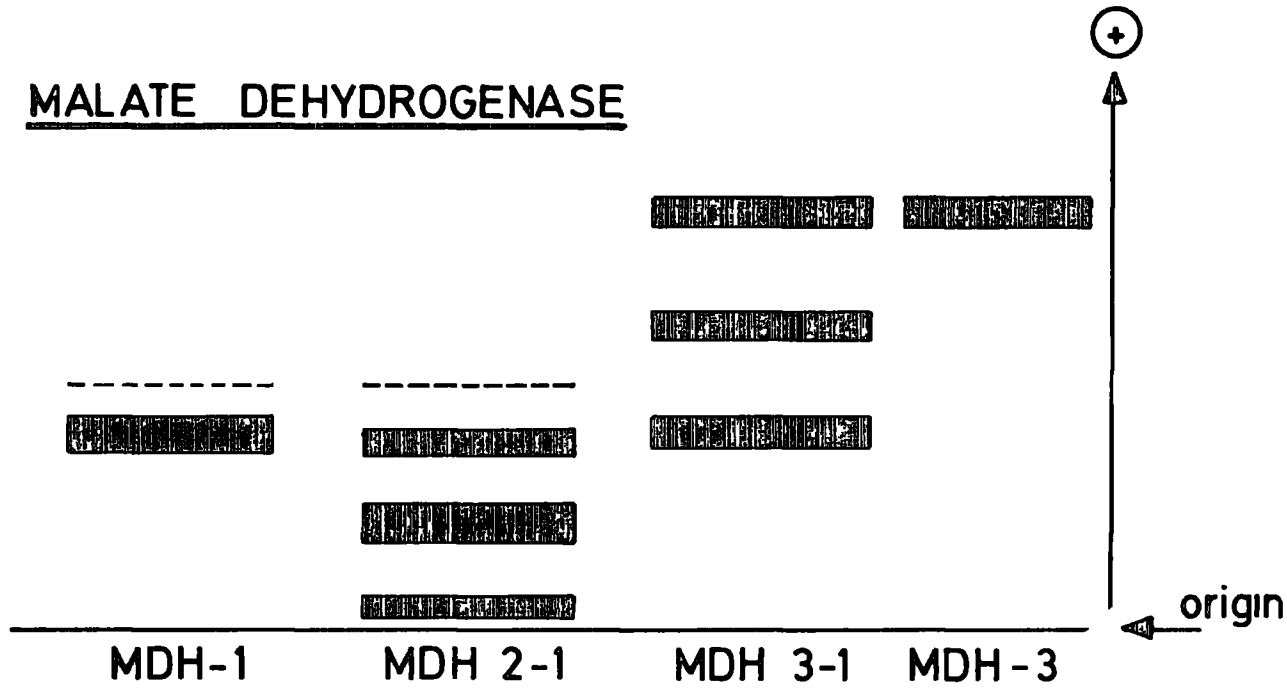


Fig.1-18 Diagram showing electrophoretic variants of Malate Dehydrogenase (Drawn from the photographs of Leakey et al 1972)

Cortner (1967). Figure 1.18 illustrates the pattern of the usual and MDH2-1 types detected by them in a survey of 1,440 White and 1,470 Negro subjects. The usual MDH exhibit one major anodal band. The variant pattern (MDH2-1) consists of three major bands, one corresponding to that of the common pattern and the other two with slower mobility. The minor bands have the same mobility as in the usual type. Family studies indicate that the usual and variant types are controlled by a pair of co-dominant alleles at an autosomal locus.

Blake et al (1970b)demonstrated another fast variant called MDH3-1 in a number of persons from New Guinea. Its zymogram exhibits three bands, of which the slowest one corresponds with the major anodal band of the usual type (Figure 1.18). Leakey et al (1972) reported one MDH2-1 among the Amhara of Ethiopia and one hundred and twenty MDH3-1 and three homozygous MDH-3 among people of New Guinea. These variants are shown in Figure 1.18. The homozygous MDH-3 has one band which corresponds with the most anodal band of MDH3-1 type.

1.4.8 Phosphohexose isomerase

Phosphohexose isomerase catalyses the conversion of glucose-6-phosphate to fructose-6-phosphate. Genetically, variation of PHI was first demonstrated by Detter et al (1968). In addition to the usual type, PHI 1, eight variant phenotypes called PHI2-1, 3-1, 4-1, 5-1, 6-1, 7-1, 8-1 and 9-1, were reported. Family studies suggest that the variant phenotypes are found in individuals who are heterozygous for a common allele PHI^1 at an autosomal locus. Nothing is known about the inheritance of phenotypes 2-1, 4-1, 7-1 and 8-1.

PHOSPHOHEXOSE ISOMERASE

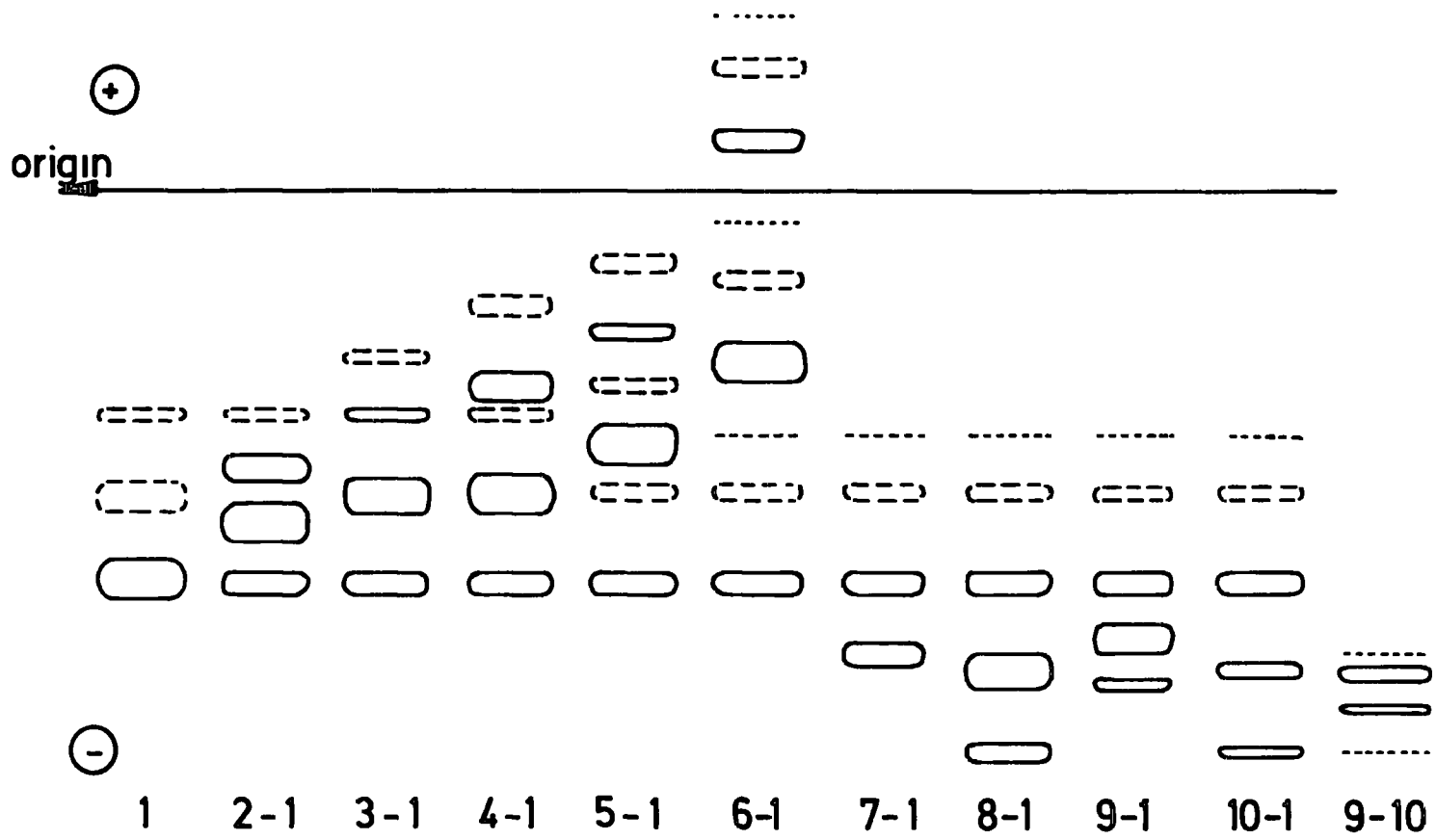


Fig 1.19 Diagram showing electrophoretic patterns of Phosphohexose Isomerase.
(After Detter et al 1968).

Isozyme patterns of the different phenotypes are shown in Figure 1.19. The usual type (PHI 1) exhibits three bands, one major and two minor. The major band is cathodal. All the variant phenotypes, except PHI9-10, consist of three major bands, one of which corresponds to the major cathodal band of the usual type. The triplet pattern of the variant phenotypes suggests a dimeric structure. It is assumed that the heterozygosity at the PHI locus produces two subunits, one usual and one variant. Phenotypes such as 2-1, 3-1, 4-1, 5-1 and 6-1 have a negatively charged variant subunit, whereas in types like 8-1, 9-1 and 7-1 the variant subunit is positively charged. Phenotype PHI7-1 represents a hybrid dimer.

PHI variants are very rare in human populations, except for Asiatic Indians, where the frequency of PHI3-1 seems to be considerably higher.

1.5 Human Haemoglobins

Human haemoglobins, the oxygen carrying pigment of erythrocytes, are complex protein molecules. The globin molecule in adults consists of four polypeptide chains each composed of amino acids linked by peptide bands and arranged in a definite sequence. These different polypeptides are called the α- and β-chains, and the formula for adult haemoglobin is written as $\alpha_2\beta_2$. The α-chain contains 141 amino acid residues and the β-chain contains 146 amino acid residues. Each of the polypeptide chains is coiled and folded in such a way that the whole molecule shows a three dimensional arrangement. There is one haem group located on the surface of the molecule. This haem is attached to the polypeptide by a linkage between the iron atom and a histidine residue in the chain.

The existence of three normal haemoglobins, adult (Hb-A), Fetal (Hb-F) and Hb-A_2 is well established (Huehns and Shooter 1965). Hb-A comprises about 98 percent of the total haemoglobin and Hb-A_2 the remaining 2 percent. In the newborn, Hb-F contains about 50-70 percent of the total haemoglobin. The polypeptide chain composition of these normal haemoglobins is as follows :

$$\text{Hb-A} = \alpha_2\beta_2$$

$$\text{Hb-}A_2 = \alpha_2\delta_2$$

$$\text{Hb-F} = \alpha_2\gamma_2$$

Hb-A_2 and Hb-F, like Hb-A, both contain two distinct polypeptides, each of which is represented twice in the molecule. One of these is identical with the α-chain in Hb-A. The difference lies in the other pair of the chain, which in Hb-A_2 is called the δ-chain and in Hb-F the γ-chain.

All the four chains are determined at separate loci on the chromosome. Thus there are four loci concerned with determining the structure of the three normal haemoglobins (Figure 1.20).

Locus	1	2	3	4
Genotype	α/α	β/β	δ/δ	γ/γ
Polypeptide chains formed	α	β	δ	γ
Haemoglobins formed	$\alpha_2\beta_2$ Hb-A	$\alpha_2\delta_2$ Hb-A_2		$\alpha_2\gamma_2$ Hb-F

Figure 1.20 Formation of normal haemoglobins

A mutant allele at the locus determining the α-chain produces variant forms of all three haemoglobins A, A_2 and F. A mutant allele at other loci determining the β- δ- or-γ chains only results in a variant form of the haemoglobin which contains the corresponding chain. Such abnormalities in polypeptide chains initiated a search for other variants and at present about a hundred different genetically determined haemoglobin types have been identified (Lehman and Carell 1969). The majority of these are rare and have only been seen in the heterozygous state, occurring together with the normal haemoglobin. However, there are some aberrant types like Hb-S, Hb-C, Hb-D and Hb-E which are quite common in certain parts of the world.

The maintenance of the high incidence of the sickle cell trait (which represents AS heterozygotes) in Africa and other parts of the world is attributed to malaria. It is believed that the heterozygotes (Hb_β^A

Hb_{β}^{S}) are more resistant to malaria produced by a specific parasite called *Plasmodium falciparum*. Allison (1954) put forward three types of evidence to support the hypothesis that heterozygotes are more resistant to malaria than are homozygotes, $Hb_{\beta}^{S}{}_{\beta}Hb^{S}$ or $Hb_{\beta}^{A}\ Hb_{\beta}^{A}$. First, high frequencies of Hb_{β}^{S} were correlated with the high rates of infection with *falciparum* malaria. Second, the number of parasites was shown to be smaller in sicklers than in non sicklers. Third, sicklers were less susceptible to experimentally induced malaria than were non-sicklers. This evidence could not be confirmed but Allison's conclusions were fully vindicated by Raper (1955). He found that though there was no difference in the parasite rate between sicklers and non-sicklers, sicklers once infected showed a significantly lower density of *P. falciparum*. As the degree of parasitaemia determines the malarial death rate in an endemic area, it therefore supports the evidence that balanced polymorphism exists in man. Another supporting finding is that no sickle cell trait carrier has been discovered with cerebral malaria. It is also well established that malignant malaria normally kills before immunity is acquired, roughly between the ages of two and five years. This is the period when the frequency of heterozygotes rises in the population, suggesting that the children with normal haemoglobin are being selectively eliminated.

At present the theory of malaria as a factor in the explanation of the high frequencies of the Hb-S gene is accepted by most authors. Recent investigations have shown this to be correct for Hb-C also (Thomson 1962). Again it is believed that malaria may be a selective factor in *thalassemia* (Haldane 1949) but the explanation of the high incidence of the Hb-E gene in East Asian ethnic groups is still to be provided. Although this abnormality in itself is "harmless", the combination of the Hb-E gene with *thalassemia* causes anaemia, which leads to death in childhood. It is therefore postulated that the carriers of this haemoglobin have advantages over normal individuals.

CHAPTER II

MATERIALS AND METHODS

2.1 Blood Collection

Blood samples for the present investigation were obtained from India, Iran, Kuwait and Nepal. Most of the samples from India and Iran were collected by the author during field work conducted from July to October 1973. The following sub-sections give an account of the collection of samples from various areas.

2.1.1 India

The blood samples from India were obtained from four different cities of the Panjab in northern India. Dr J G Jolly of the Blood Bank, Post Graduate Institute of Medical Sciences, Chandigarh, kindly allowed the collection of blood taken in donor sessions held in Ludhiana, Patiala, Jullundhur and Chandigarh. Altogether some 350 samples were collected during this period and sent by air at wet ice temperature to London and subsequently to the Department of Anthropology, Durham. Each batch was received within 72 hours of its collection. Personal information, regarding name and sex of the donors was obtained from the blood bank. A hundred blood samples sent by Mr Kulwant Singh Sawhney were badly haemolysed due to the breakage of the thermos flask and delay in transportation from London. These samples were typed only for red cell isoenzymes. All the samples were taken in 5ml sequestrene tubes.

2.1.2 Iran

The specimens from Iran were collected from Tehran and Isfahan. Blood samples from Tehran, mainly obtained from blood donors, were provided by Professor A Adjir and Dr J Arbabzadeh of the Red Lion and Sun Society. Samples from Isfahan were personally collected by Dr M Suzangar of the Clinical Biochemistry Department, Isfahan University, Isfahan. The samples, well-packed in thermocool packages, were sent by air to Durham. No information apart from name and sex was obtained.

2.1.3 Nepal

Nepalese blood samples were collected from the Gurkha Regiment stationed at Aldershot, Hampshire. Captain I Bethell of the Army Blood Supply Depot, Aldershot, was kind enough to send 10 to 20 samples at weekly intervals. Blood typing, except for one of the Indirect Coombs test, was done in Aldershot. Analysis of plasma proteins and red cell isoenzymes was carried out in the Anthropology Laboratory, Durham. A few specimens were stored in liquid nitrogen by the sucrose method described for us by Mr Davison of the General Hospital N.B.T.S. Unit, Newcastle upon Tyne. The procedure involved placing equal volumes of washed red cells and cryogenic agent (prepared by dissolving 45 grams of sucrose in 100ml of distilled water) in the coded polyproplene tubes and dropping them immediately into liquid nitrogen containers. Personal details regarding name, sex and caste were obtained from the blood depot.

2.1.4 Kuwait

The Kuwaiti samples, collected by Dr Eid and Mr A A Bashir of the Central Blood Bank, were sent in two batches. The first collection was brought by Professor E Sunderland and the second was despatched by Miss Jasmiya-al-Marzook. All the samples contained in 5ml sequestrene tubes were kept at wet ice temperature during the journey. No information apart from that they are Kuwaiti Arabs was obtained.

2.2 Blood Group Serology

Blood received in the laboratory was either grouped immediately or stored in liquid nitrogen. The two different methods employed to prepare blood for grouping were as follows :

2.2.1 Blood in anti-coagulant E.D.T.A.

The blood received in sequestrene tubes was centrifuged for 10 minutes at 1000 R.P.M. to separate the serum from the red cells. The serum was pipetted off and stored at -20°C until required for use. The red cells were washed thrice in normal saline (0.9% NaCl) to remove anti-coagulant E.D.T.A. A saline suspension of red cells was prepared by adding 5 drops of whole blood to 5ml of saline. The rest of the red cells were stored at -20°C for preparing haemolysates.

2.2.2 Blood stored in liquid nitrogen

A glass beaker of physiological saline was placed in a water bath and warmed to 40°C. The tubes were quickly removed in batches of six from the liquid nitrogen containers and dropped into the warm saline. The frozen contents were poured into a cooled centrifuge tube. It was spun at low speed for 10 minutes and the supernatant removed. The red cells were washed thrice in normal saline. The saline suspension was prepared as described in sub-section 2.2.1 and grouping was carried out as rapidly as possible.

2.2.3 Blood grouping techniques

Three different method used for blood grouping are given below .

2.2.4 Tile technique

This test was carried out by adding a drop of blood cell suspension (in physiological saline) to a drop of antiserum. The red cells/serum mixture was left for a specific period at a certain temperature. The tile was then moved gently back and forth and inspected for agglutination. The following antisera required a tile technique; anti-A, anti-B, anti-A+B, anti-A_1, anti-A hel, anti-M and anti-N.

2.2.5 Tube technique

The tube technique was performed by adding one volume of 5% saline suspension to one volume of antiserum in a precipitin tube. The serum/cell mixture was incubated for specific periods of time at a certain temperature. After incubation, bovine albumin was carefully run down the side of the tube to cover the red cells. The cells were re-incubated for 30 minutes and the results were read microscopically. The following antisera required this method; anti-C, anti-c, anti-D, anti-E, anti-S and anti-C^W. Only Lu^a was tested without the addition of bovine albumin.

2.2.6 Indirect Coombs technique

This technique involved placing equal volumes of red cells with antiserum in a precipitin tube. The serum/cell mixture was incubated for a specific length of time at certain temperature. After incubation, the cells were washed four times with physiological saline. The red cells were shaken well at the end of each washing. The tile was washed properly to ensure that there was no contamination to inhibit the coming test. A drop of shaken red cells was mixed well with a drop of anti-human globulin. The tile was then rocked gently for 5-10 minutes and agglutination was observed over a strong light. The following antisera required use of the anti-globulin technique; anti-Fy^a, anti-Fy^b, anti-JK^a, anti-JK^b, anti-K, anti-Cellano, anti-Kp^b, anti-S and anti-s.

All the controls were set up at the same time, under the same conditions and were read immediately before the tests.

2.2.7 Antisera

The antisera obtained from various sources, along with their methods and temperature conditions, are tabulated below :

Antiserum	Source	Method
anti-A	Blood Group.Ref.Lab.	Tile, Room Temp. for 10mins
anti-B	Blood Group.Ref.Lab.	Tile, Room Temp. for 10mins
anti-A+B	Blood Group.Ref.Lab.	Tile, Room Temp. for 10mins
anti-A_1	Blood Group.Ref.Lab.	Tile, Room Temp. for 10mins
anti-A_1	Newcastle B.T.S.	Tile, Room Temp. for 10mins
anti-A hel	Biotest Diagnostics	Tile, Room Temp. for 30secs
anti-M	Newcastle B.T.S.	Tile, Room Temp. for 10mins

Antiserum	Source	Method
anti-N	Blood Group.Ref.Lab.	Tile, Room Temp. for 10mins
anti-N	Ortho Diagnostics	Tile, Room Temp. for 1min
anti-S	Newcastle B.T.S.	IDC, 37°C for 1hr
anti-s	Ortho Diagnostics	IDC, 37°C for 1hr
anti-C	Newcastle B.T.S.	Tube, albumin addition 37°C for 2hrs
anti-C	Hyland Laboratories	Tile, 37°C for 2mins
anti-c	Newcastle B.T.S.	Tube,albumin addition 37°C for 2hrs
anti-D	Newcastle B.T.S.	Tube,albumin addition 37°C for 2hrs
anti-e	Newcastle B.T.S.	Papanised cell technique 37°C for 50mins
anti-E	Newcastle B.T.S.	Tube,albumin addition 37°C for 2hrs
anti-C^w	Blood Group.Ref.Lab.	Tube,albumin addition 37°C for 2hrs
anti-Fy^a	Newcastle B.T.S.	IDC, 37°C for 1hr
anti-Fy^b	Biotest Diagnostics	IDC, 37°C for 1hr
anti-K	Newcastle B.T.S.	IDC, 37°C for 1hr
anti-K	Ortho Diagnostics	Tile, Room Temp. for 2mins
anti-Cellano	Hyland Laboratories	IDC, 37°C for 1hr
anti-Cellano	Ortho Diagnostics	IDC, 37°C for 1hr
anti-Lu^a	Blood Group.Ref.Lab.	IDC, 37°C for 1hr
anti-JK^a	Biotest Diagnostics	IDC, 37°C for 1hr
anti-JK^b	Biotest Diagnostics	IDC, 37°C for 1hr
anti-Kp^b	Biotest Diagnostics	IDC, 37°C for 1hr
anti-Kp^a	Biotest Diagnostics	IDC, 37C for 1hr

2.3 Haemolysate Preparation

Haemolysates were prepared by the carbon tetrachloride method of Ager and Lehman (1961); briefly described below :

An equal volume of water was added to the washed red cells and stored at -20°C. The red cells were then thawed before adding a volume of carbon tetrachloride at least equal to twice the volume of cells and all the contents thoroughly mixed. The tubes containing the mixture were centrifuged at 3000 R.P.M. for 40-50 minutes. The supernatant was placed in tubes and stored at -20°C until required for subsequent analysis.

2.4 Starch-Gel Electrophoresis

Starch gel as a supporting medium was introduced for zone electrophoresis by Smithies (1955a). The resolving power of this medium has a greater ability to separate complex serum proteins, hormones, tissue extracts, etc. The mechanism for high resolving power is unknown but it is believed that molecular sieving plays an important role. Another advantage of the method lies in the relative ease of manipulation and the sharpness of the zones obtained. The following sub-sections describe the necessary equipment, sample insertion, gel slicing and details of electrophoretic methods.

2.4.1 Apparatus used

Electrophoresis on starch gel was carried out in the horizontal position and the equipment consisted of (i) a power pack capable of providing a constant current up to 50mA and a constant voltage up to 500 volts, (ii) plastic trays with internal dimensions of 20 x 15cm x 7mm and plastic lids, (iii) a plastic tank for bridge buffer solution; (iv) platinum electrode; (v) a pH meter, (vi) dental forceps for inserting the specimens; (vii) plastic boxes for staining the gel; and (viii) a slicer board fitted with a horizontal wire for cutting the gel.

2.4.2 Preparation of gel

For the preparation of the gel, starch obtained from Connaught Medical Research Laboratories, Toronto, was used. A suspension of starch in buffer solution was heated over a gas flame in a 50ml pyrex round-

bottomed flask. The contents were agitated by an electrical stirrer. Heating was continued until the solution became opaque and semi-solid. Care was taken to prevent charring. A vacuum was applied to the flask for a few seconds to expel the air bubbles. After disconnecting the vacuum, the viscous translucent solution was poured into the plastic trays. The trays were lightly smeared with liquid paraffin before pouring the gel. The gel was left to set from 2-3 hours at room temperature.

2.4.3 Sample insertion

After the gel had set, a cut was made with a surgical blade across the gel about two inches from the cathode end. The gel was pushed back gently to allow space for the insertion of samples. Care was taken to avoid breakage or distortion of the gel. A piece of filter paper (Whatman No.3 or 17) about 10 x 15mm was held by forceps and immersed in the serum or haemolysate sample. The moist filter was inserted into the cut so that it adhered to the surface of the gel. The slot was closed by applying slight pressure on the gel so that it came back to its original position. A space of 2-3mm was left between the adjacent inserts.

2.4.4 Slicing the gel

After electrophoresis the inserted sample strips of paper were removed with forceps and the gel was laid flat on the slicer board. The required height of the horizontal wire was fixed with the height adjuster. The plastic tray with the gel on it was moved gently towards the wire to cut it in half. The sliced gel was then transferred to a plastic box containing the stain solution.

2.4.5 Electrophoretic methods

The protein and enzyme systems all require fairly strict control of electrophoretic method, pH, temperature, strength of buffer solution and purity of ingredients used in the buffer and incubation mixtures. Electrophoretic conditions should be designed to give optimum separation of isoenzymes without any loss of activity. The plasma proteins and red cell isoenzymes employed in this survey were analysed in the Physical Anthropology Laboratory at Durham. Phosphohexose isomerase typing was performed in the Laboratory of Human Genetics, Newcastle upon Tyne. All electrophoretic runs were read under the supervision of Dr R Cartwright. The ambiguous results were re-run using a thicker insert. The experimental conditions and procedure of different methods are described in the following sub-sections.

2.4.6 Haptoglobin and transferrin

Resolution of haptoglobin and transferrin was carried out using Smithies (1955a) horizontal electrophoresis and Poulik's (1957) discontinuous buffer system. One drop of a 4 per cent suspension of fresh haemoglobin was added to 3 drops of plasma, and the sample so treated was inserted into the gel using a Whatman No. 3 filter paper insert. The electrophoresis was carried out at 30 mA, 500 V for 3 hours at $+4^{o}C$.

Tank Buffer

0.3 M	Boric Acid	46.3g
0.05M	Sodium Hydroxide	6.0g
	Distilled Water	1.0L
	pH 8.5	

Diluted for use 1/5

Gel Buffer

0.076M	Tris	23.0g
0.005M	Citric Acid	2.62g
	Distilled Water	1.0L
	pH 8.7	

Dilute 100ml with 150ml distilled water for each gel.

The gels were cut horizontally in half. The upper half was stained with the solution given below, which detects the presence of Hb/Hp complex. The benzidine stain used by Smithies (1959) contained 100ml distilled water, 0.5ml glacial acetic acid, 0.2g benzidine and 0.2ml 30% hydrogen peroxide. The lower half was stained with 1% amido black in 50 : 50 : 10 methanol : water and acetic acid solution for 3-4 minutes. Then the gels were washed to remove the excess of stain and decolourised in the above solution until the gels were cleared for all protein zones.

2.4.7 Acid phosphatase

The method used is that described by Hopkinson et al (1963). The electrophoretic conditions and procedure are given below :

Tank Buffer

0.41M	Citric Acid	86.1615g
	NaOH	45.0g
	Distilled Water	1.0L

adjust to pH 6.0 with 4 N NaOH

Gel Buffer

0.0025M	Succinic Acid	0.2952g
0.0046M	Trisma Base	0.5552g
	Distilled Water	1.0L

The gels were prepared with 0.0931g of EDTA and before degassing the starch 1ml of 2-mercapto-ethanol was mixed. The haemolysate sample was applied on Whatman No. 17 filter paper inserts and electrophoresis carried out at 6 volts/cm for 17 hours at +4^{o}C. The gels were sliced and covered with Whatman No. 17 filter paper to which was applied the incubation buffer containing 0.005M phenolphthalein diphosphate pentasodium, 0.2944g.

Incubation Buffer

Citric Acid	1.05g
Distilled Water	100ml

adjust to pH 6.0

After the incubation was complete, the filter papers were removed and concentrated ammonia solution 1ml/gel was spread over the gel.

2.4.8 Phosphoglucomutase

The method used is that described by Spencer et al (1964).

Bridge Buffer

0.1M	Tris	12.11g
0.1M	Maleic Acid	11.608g
0.01M	EDTA	3.7225g
0.01M	$MgCl_2$	2.0333g
	NaOH	6.5g
	Distilled Water	1.0L

adjust to pH 7.4 with 4N NaOH

Gel Buffer

Bridge buffer diluted 1 : 10 with distilled water.

The electrophoresis was carried out at 5.5 volts/cm for 17 hours at $+4^{o}C$. The sliced gel was incubated for 3 hours at $+37^{o}C$ in the incubation buffer which contained the following ingredients :

Incubation Buffer

$4.6 \times 10^{-3}M$	Glucose-1-phosphate	0.1713g
$5.0 \times 10^{-5}M$	Glucose-1-6-diphosphate	
	(this is present as an impurity of G-1-P)	
$1.2 \times 10^{-4}M$	NADP	0.0100g
$10^{-2}M$	$MgCl_2$	0.2033g
0.04 units/ml	G-6-PD	4.0 units
0.1mg/ml	PMS	0.0100g
0.1mg/ml	MTT	0.0100g

All these ingredients were dissolved in 100ml of 0.03M Tris Buffer, pH 8.0.

2.4.9 Adenylate kinase

The method used is that described by Fildes and Harris (1966). Horizontal electrophoresis was carried out at 10 volts/cm for 4 hours at +4°C. The haemolysate samples were placed on Whatman No. 3 filter paper inserts in the centre of the gel.

Tank Buffer

0.41M	Citric Acid	86.1615g
	NaOH	45.0g
	Distilled Water	1.0L

adjust to pH 7.0 with 4N NaOH

Gel Buffer

0.005M	Histidine	1.0487g
	Distilled Water	1.0L

adjust to pH 7.0 with 4N NaOH

Incubation Mixture

To 100ml of 0.1M Trisma base, pH 8.0, is added

10.0M	Glucose	0.1802g
1.0mM	ADP	0.0439g
0.4mM	NADP	0.0255g
0.012%	MTT	0.0120g
0.012%	PMS	0.0120g
20.0mM	$MgCl_2$	0.4067g
0.04 units/ml	G-6-PD	4.0 units
0.08 units/ml	Hexokinase	8.0 units
	Agar	0.75g

0.75g of Agar was added to 50ml of incubation buffer. It was heated to 95°C and then cooled in a water bath to 45°C. The agar was then poured into the rest of the 50ml of buffer and mixed thoroughly by continual stirring. The mixture was spread over the cut gel and incubated for 2 hours at +37°C.

2.4.10 Dehydrogenase enzymes

Tests for 6-phosphogluconate dehydrogenase, glucose-6-phosphate dehydrogenase, lactate dehydrogenase and malate dehydrogenase were performed on the same gel, since the conditions required for electrophoresis were identical. The gel was cut in half. The top half was stained for 6-PGD and G-6PD and the bottom half for LDH and MDH enzymes. The details of the method used are described below :

Tank Buffer

A	0.2M	Mono potassium phosphate (KH_2PO_4)	27.22g
B	0.2M	Disodium hydrogen phosphate ($Na_2PO_4 2H_2O$)	28.44g
These were mixed in proportions			508A 492B

Gel Buffer

The Tank Buffer was diluted 1 : 20 with distilled water. Horizontal electrophoresis was carried out at 12 volts/cm for 3 hours at +4°C.

After electrophoresis the gel was sliced and one half was stained with the undermentioned buffers :

a) 6-PGD

Incubation Buffer

To 10ml of 0.1M Tris, pH 8.0, was added :

NADP	0.002g
Sodium-6-phosphogluconate	0.01g
PMS	0.0004g
MTT	0.002g

and incubated on the gel for approximately 15 mins at +37°C.

b) G-6-PD

Incubation Buffer

To 10ml of 0.1M Tris, pH 8.0, is added :

NADP	0.002g
G-6-P	0.01g
PMS	0.0004g

The gel was incubated for 30 minutes at +37°C.

The other half was stained with the following incubation buffer for the determination of LDH and MDH phenotypes.

c) LDH

Incubation Buffer

10% Lactic Acid	0.25ml
NAD (DPN)	0.005g
PMS	0.005g
MTT	0.005g

These were dissolved in 0.1M Tris, pH 8.0, placed on the gel and then incubated for 1 hour at +37°C.

d) MDH

Incubation Buffer

50ml of 0.1M Tris, pH 8.0, contained :

L-Malic Acid	0.1g
NAD(DPN)	0.01g
PMS	0.01g
MTT	0.01g

After recording the LDH results the gel was washed with distilled water to remove stains. The gel was restained for MDH and incubated for one hour at +37°C.

2.4.11 Phospho-hexose isomerase

The method used for the PHI analysis was as follows :

Buffers

0.21M	Tris	25.4g
0.15M	Borate	9.3g
0.006M	EDTA	1.75g
	Distilled Water	1.0L

Gel Buffer

Used above buffer diluted 1 : 10, pH 8.6

Bridge Buffer

Used above buffer undiluted, pH 8.0.

The samples were applied on Whatman No. 3 filter paper inserts and horizontal electrophoresis was carried out at 12V/cm for 20 hours at +4°C. After electrophoresis the gel was sliced and the lower half was stained for 10 mins at room temperature, with the following stains :

Stain

0.00032M	F-6-P	0.0110g
0.005M	$MgCl_2$	0.1g
0.00013M	NADP	0.01g
0.00024M	MTT	0.01g
0.00013M	PMS	0.004g
	G-6-PD	0.05mg

The stains were dissolved in 100ml 0.05M Tris, pH 8.0.

2.5 Cellulose Acetate Electrophoresis

Cellulose acetate was introduced and developed as a medium for electrophoresis by Kohn (1957). Adsorption of proteins on this medium is minimal so that the tailing of zones is largely eliminated. This results in sharper bands and also renders more visible the minor components. Further advantages of cellulose acetate are the speed of separation and the small quantities of material which can be handled. Haemoglobin screening was carried out by this method.

2.5.1 Apparatus used

Electrophoresis was carried out in a horizontal tank (designed by Shandon Scientific Co.) which had four compartments, two central electrodes and two buffer components on the outside. The middle partition separated the anode from the cathode compartment. A lid made of Perspex was fitted well in the groove of the wall to ensure adequate vapour saturation, thus avoiding drying out of the strips during a run. A power pack capable of providing 400 volts was used.

2.5.2 Buffers used

The composition of buffer solutions was as follows :

a) At the anode :

Tris	25.2g
EDTA	2.5g
Boric acid	1.9g
Distilled water	1.0 L

b) At the cathode :

Sodium diethylbarbiturate	5.15g
Diethylbarbituric acid	0.92g
Distilled water	1.0 L

The solutions a and b were mixed in equal volumes for the impregnation of strips.

2.5.3 Procedure

The cellulose acetate strip was floated on the electrophoresis buffer. After soaking, it was blotted slightly between sheets of filter paper so that no excess buffer was left. A sharp pencil was used to draw a straight line in the middle of the strip. Blood samples were applied by moving a micro-pipette along the edge of the ruler. A 5mm margin was left on either side of the strip. After applying samples, the strip was immediately transferred to the tank and the strip holders were placed on it to hold it taut.

2.5.4 Staining

After electrophoresis, the strip was floated on to a solution of Ponceau S (0.20% W/V) in 3% W/V trichloro-acetic acid. It was kept in Ponceau solution for 3-4 minutes and then washed in 5% W/V acetic acid until the background became white.

2.6 Zip Zone : Titan IV Citrate Electrophoresis

Abnormal haemoglobins were confirmed in the Metropolitan Forensic Laboratory, London, using Titan IV citrate technique.* The method is briefly described below.

Electrophoresis was carried out in horizontal positions. A 100ml of citrate buffer, pH 8.6 (diluted with distilled water) was poured into each of two compartments of the electrophoresis chamber fitted with a zip zone sponge. The haemoglobin specimens were prepared in micro-preparation dish by adding one part packed red cells to 20 parts of haemolysate reagent. Application of samples was made by pressing the tip of the applicator on to the gel surface. A Hemo AFSC control was run by placing it in the middle of the gel. After electrophoresis the gel was stained in a benzidine solution with the following composition :

(i) 20ml of 5% Acetic acid

(ii) 10ml of 2% Benzidine solution

(iii) 2ml of Hydrogen peroxide

(iv) 2ml of Sodium nitro ferricyanide

*The method used for identifying the abnormal haemoglobins has been taken from the paper "Titan IV Citrate Hemoglobin Procedure" produced by Helena Laboratories, Beaumont, Texas.

2.7 Statistical Techniques

This section deals with the statistical techniques and formulae used to analyse and interpret the data. The derivation of the formulae is beyond the scope of this thesis.

2.7.1 Gene frequencies

Since genotype could be deduced directly from the results of tests in the case of some blood groups, the serum proteins and the red cell enzyme systems, gene frequencies were determined by direct gene counting. In the case of ABO, MNSs and Rh blood group systems, the gene frequency calculations were carried out by using the formulae given in Mourant (1954). In the Duffy system which involved phenotype Fy(a-b-) the frequencies were calculated by the method of Race and Sanger (1958). Some systems which involved the use of only one antiserum e.g. Lutheran system, the gene frequencies were calculated in the following manner. The frequency of Lu^b was obtained by square rooting the observed frequency of Lu(a-) in the sample. The frequency of Lu^a is therefore found to be 1- frequency of Lu^b. In the Iranian sample this works as follows :

$$Lu^b = \sqrt{0.8687} = 0.9319$$

$$Lu^a = 1-0.9319 = 0.0681$$

2.7.2 Chi squared test

To test the Hardy-Weinberg equilibrium and internal goodness of fit for phenotype frequencies, the chi-squared values were calculated using the following well known equation :

$$X^2 = \frac{(\text{Observed} - \text{Expected})^2}{\text{Expected}}$$

In order to make the results statistically plausible, in certain cases where an incidence was found to be less than 5, it was added to that of the next lowest phenotype to evaluate the X^2, e.g. in the acid phosphatase the CC phenotype was added to the CB phenotype.

Only X^2 values exhibiting a probability of 0.05 were considered to be statistically significant and the actual values of X^2 were calculated and set out in the text. However, the systems showing the level of probability greater than 0.05 are reported as non-significant throughout this work.

2.7.3 Contingency tables

In testing populations for possible relationships in two gene marker systems, the contingency table used is shown below. In certain protein and enzyme systems each phenotype included one genotype, and comparisons could be made in terms of the number of genes as well as phenotypes.

2 x 2 Contingency Table

Gene	Population I	Population II	Total
P	a	b	a+b
Q	c	d	c+d
Total	a+c	b+d	a+b+c+d

The standard formula for this is :

$$X^2 = \frac{[(axd) - (cxb)]^2 \times N}{(a+c) \times (b+d) \times (c+d) \times (a+b)}$$

Systems which offer three alternative gene forms such as acid phosphatase, the relationship of two population groups can be obtained using a 2x3 contingency table by the method shown below.

2 x 3 Contingency Table e.g. AP system

Gene	Population I	Population II	Total
P^a	A^1	A^2	$A^1 + A^2$
P^b	B^1	B^2	$B^1 + B^2$
P^c	C^1	C^2	$C^1 + C^2$
Total	$A^1+B^1+C^1$	$A^2+B^2+C^2$	$A^1+A^2+B^1+B^2+C^1+C^2 = N$

A^1, A^2, B^1, B^2, C^1 and C^2 are the number of genes of each type in the population and N is the total number of the genes.

Expected values are then obtained for each of the genes as follows :

$$\text{Exp}A^1 = \frac{(A^1+B^1+C^1) \times (A^1+A^2)}{N}$$

The X^2 is then obtained for each cell as follows :

$$X^2 = \frac{(\text{Obs } A^1 - \text{Exp } A^1)^2}{\text{Exp } A^1}$$

The values of the X^2 given by the six cells are then summed to obtain the total X^2. The number of degrees of freedom is obtained by the following formula :

$$(1-r)\ (1-c)$$

Where r is the number of rows and c is the number of columns. Hence, in the above system the number of degrees of freedom is two.

In other cases, for example in 6-PGD and PGM systems, a rare allele can be added to the next nearest allele with confidence, owing to the fact that they have not been found in any population in high frequency, and then a 2 x 2 contingency table is adequate.

In the case of blood groups e.g. the ABO system, genotypes were not obtainable from the phenotypes and statistical comparison had to be done in terms of phenotypic numbers only. Any expected value found to be less than 5 was added to the next lowest number e.g. in the ABO system A and AB were added together, in the acid phosphatase system the CC phenotype was added to the CB phenotype. X^2 values of the blood group systems exhibiting a probability of 0.05 were classified as statistically significant and are given in the text.

CHAPTER III

GENETIC POLYMORPHISMS IN THE KUWAITI ARABS

3.1 Introduction

3.1.1 Geographical Setting

Kuwait is an independent Arabian state and lies between 28° and 30° north and between 46° and 48° east. Its territory occupies about 15,900 square kilometres of desert and low offshore islands and is a continuation of the great desert that covers large areas of the neighbouring Arabian countries. Located in the northeastern corner of the Arabian Peninsula, Kuwait is bordered on the north and west by Iraq and on the southwest by Saudi Arabia (Figures 3.1, 3.2). To the southeast lies the neutral Zone which has an area of 4,200 square kilometres and is jointly administrated by Kuwait and Saudi Arabia. On the western side of the country is the valley of Batin. Mishrif and Sirra are isolated hills in the south. Of the offshore islands the largest are the uninhabited islands of Bubiyan and Warba. The island of Failaka, situated near the entrance of the Kuwait Bay, has been populated since prehistoric times.

Among the principal cities, the city of Kuwait is the largest and is located in the Bay of Kuwait. The population of the country is largely concentrated in this city. The other large communities are Ahmadi, the centre of the oil industry, Al-Jahra, an oasis on the main road to Basra, and the settlements of Failaka Island. The climate of Kuwait is characterized by extremely hot, dry summers with an average maximum daily temperature of 45°C and mild to cool winters in which temperatures of -1°C may occur. The relative humidity is highest in December and January, with average maxima of 85 per cent. Summer humidity is generally less than 45 per cent.

Figure 3 1 KUWAIT – GEOGRAPHICAL SETTING.

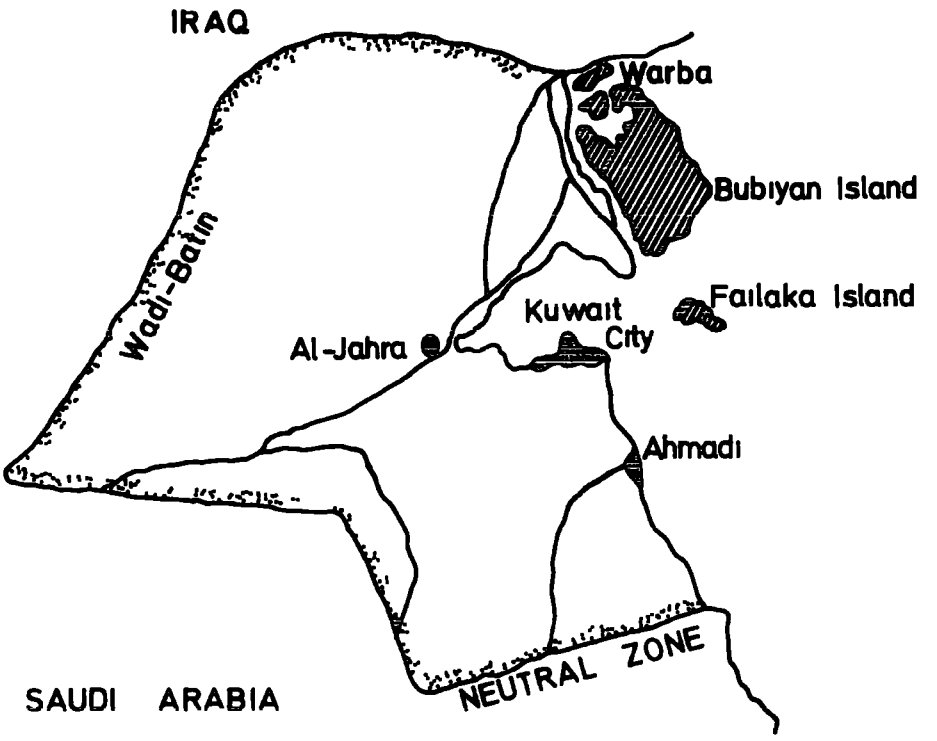
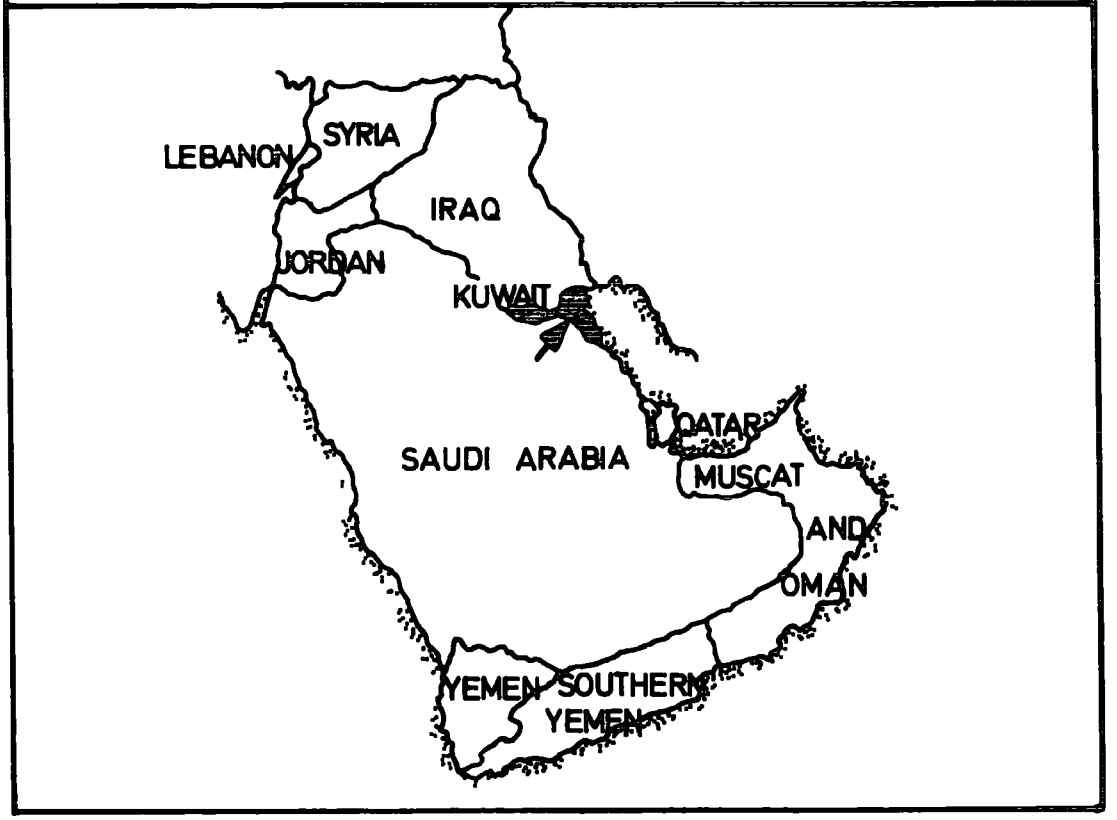


Figure 3 2 ARABIAN PENINSULA SHOWING THE POSITION OF KUWAIT AND NEIGHBOURING ARAB STATES



3.1.2 Historical review

The early history of Kuwait is rather enigmatic. The scanty knowledge available is exclusively based on the excavations on Failaka Island. The Bronze-age dwellings found at Failaka suggest the existence of a civilization some three thousand years ago (Ffrench and Hill 1971). The Sumerians were a non-Semitic people who originally occupied South Babylonia but gradually spread down to the Arabian Gulf to form a strong link with the Indus valley. The evidence for this comes from the excavations at Ur just over Kuwait's northern border and at Mohenjo-Daro in Pakistan. The burial mounds on Bahrain Island are also believed to have belonged to these early mariners whose artefacts can still be seen today stretching over 2,000 miles from Iraq to India. In the 4th century B.C. the Greeks came to Failaka Island. They colonised the Island as a defence against the Arabs which failed to pay Alexander the respect he considered due. The Portugese inhabited the Island in the 16th and 17 centuries. They established two defensive positions, one on the Island of Quam in Kuwait Bay and the other at the eastern tip of Failaka Island.

The founding of the original settlement of Kuwait is believed to have taken place about 1710. In the early years of the 18th century a group of bedu from central Arabia were driven by drought to leave their own lands and move in search of water and pasture. After much wandering, this group, who were of the Dahamshah section of the Amarat, a subtribe of the Aniza confederation, arrived on the southern shore of Kuwait Bay. Among them were the ancestors of the Al-Sabah, the ruling family of Kuwait, and also the Al-Khalifah who are today the ruling family of Bahrain. At the time of the settlement of these groups, the present day Kuwaiti formed part of the territory of the Bani-Khalid, the powerful Hasa tribe which

dominated north-east Arabia. Abu-Hakima (1965) thinks that at the time of the arrival of the Aniza colonists there were other isolated groups of fishermen called Bani-Utub. These people once inhabited Najd and the northern part of Arabia. It is also believed that some of these early settlers came from Iraq and belonged to the Aniza and Shammar tribes.

In 1937, with the establishment of the Kuwait Oil Company, the large real inflow of immigrant populations from south-west Asia, south Asia and north Africa started.

3.2 Blood Group Antigens

The results of the Kuwaitis tested for different blood group antigens are given below :

(i) Table 3.1 shows the ABO blood group distributions in a sample of 162 Kuwaiti Arabs, together with the respective gene frequencies. There was good agreement between the observed and expected phenotype values, thus confirming the assumption of Hardy-Weinberg equilibrium and that it is a randomly mating population. The gene frequencies are as follows : $p_1 = 0.1518$, $p_2 = 0.0213$, $q = 0.1268$ and $r = 0.7001$.

(ii) The results for 159 Kuwaiti Arabs, tested with anti-M, anti-N and anti-S, are set out in Table 3.2. Good agreement was found between the observed and expected phenotype values confirming the Hardy-Weinberg equilibrium. The calculated gene frequencies are : MS = 0.2224, Ms = 0.3814, NS = 0.0509 and Ns = 0.3453.

(iii) Table 3.3 presents the results for 110 Kuwaitis, tested with anti-D, -C, -c, -E and -e. Again good agreement was found between the observed and expected phenotypic values confirming the assumption of Hardy Weinberg equilibrium. The gene complex frequencies are as follows : $R_z = 0.0217$, $R_1 = 0.4920$, $R_2 = 0.0504$, $r'' = 0.0234$, $R_o = 0.0880$ and $r = 0.3246$.

(iv) Table 3.4 reveals the results for the Kell and Duffy blood groups, tested respectively with anti-K and anti-Fy^a serum. The Kell gene frequencies are as follows : K = 0.0187 and k = 0.9813 and the Duffy frequencies were found to be; $Fy^a = 0.2879$ and $Fy^b = 0.7121$.

3.3 Discussion

3.3.1 ABO blood group system Table 3.5

The ABO gene frequencies show wide variations throughout the world and in the Arabian Peninsula and neighbouring Arab states, the general distribution of the ABO genes, with the exception of a few tribal isolates, show frequency ranges of 15-30 per cent for p_A, 10-20 per cent for q_B and 60-75 percent for r_O. The gene frequencies of the present sample are within this range and close to the values reported for the Kuwaitis by Onsi and El-Alfi (1968).

The available ABO gene frequencies for the Arab groups from which the indigenous Kuwaitis are known to be historically derived, viz · the 'Aniza of Najd and the Bedouin of South-Iraq', show some similarities with the gene frequencies of the Kuwaiti Arabs. The Arabs of Najd studied by Moshkovski et al (1931) have p_A, q_B and r_O of 0.1420, 0.1782 and 0.6798 respectively. A report by Kayassi et al (1938) on the Bedouin of Iraq showed the three frequencies to be 0.1843, 0.1789 and 0.6368 respectively. The present sample shows a higher r_O and slightly lower p_A and q_B than these groups of figures which probably represent the main ancestral populations, and one is tempted to assume that a proportion of the present day Kuwaitis was derived from a population with higher r_O and lower p_A and q_B. Three Arab tribes living in Syria - the Rwala, the Maulay and the Akeydat - qualify for these gene frequencies (see Table 3.5). It is interesting to note that the Rwala are a branch of the Aniza tribe which migrated from Najd to Syria in the first half of the 18th century as a part of the 'great migration of the Aniza' (Musil 1928). It is probable that the Rwalas had either joined the 'Bani Utub' on their way to Kuwait at that early date or arrived at some later date. The second

tribe - the Maulay - had dominated the area of south-east Iraq before migrating to Syria and may have contributed a proportion to the Kuwaiti population. Although the third tribe - Akeydat - may have contributed to the present day population, there is nothing in the history of Kuwait that substantiates this assumption.

Using a Chi-squared test, it is seen that the present day Kuwaitis exhibit ABO phenotypes consistent with those found in the Arabs of Najd, Yemen and Zabid, but they differ significantly from the Arabs of South Western Arabia, $X_2^2 = 14.829$ $P<0.001$ (Marengo-Rowe et al 1974). The Kuwaitis also differ from the Arabs of Iraq, Syria and Lebanon (Kennedy and Macfarlane 1936, Altounyan 1928 and Parr 1931).

Kuwaiti Arabs v Iraqi Arabs	$X_2^2 = 12.721$	$0.01<P>0.001$
Kuwaiti Arabs v Syrian Arabs	$X_2^2 = 12.109$	$0.01<P>0.001$
Kuwaiti Arabs v Lebanon Arabs	$X_2^2 = 14.469$	$P<0.001$

It is the higher frequency of O and lower of B in the Kuwaitis that contributes to the large Chi-squared value.

3.3.2 MNS blood group system Table 3.6

To the author's knowledge there has been no investigation of the MNS blood group distributions in the neighbouring Arab states, with the exception of Iraq and Syria which have been tested only for the MN blood groups.

After statistical analysis, the Kuwaiti Arabs are found to exhibit MNS phenotype frequencies consistent with those found in the Zabidis (Ikin 1963) and the Shiah of eastern Saudi Arabia (Maranjian et al 1966), whereas they differ from the Yemenis, Bedouin and Sunni samples of Saudi Arabia and the Arabs of Southern Arabia (Marengo-Rowe et al 1974).

Kuwaiti Arabs v Yemenis	$X^2_4 = 18.059$	$0.01<P>0.001$
Kuwaiti Arabs v Bedouin	$X^2_4 = 38.696$	$P<0.001$
Kuwaiti Arabs v Sunni (Najd)	$X^2_4 = 27.463$	$P<0.001$
Kuwaiti Arabs v Sunni (E.Arabia)	$X^2_4 = 10.963$	$0.05<P>0.02$
Kuwaiti Arabs v Sunni (W.Arabia)	$X^2_4 = 26.009$	$P<0.001$
Kuwaiti Arabs v S. Arabia	$X^2_4 = 22.473$	$P<0.001$

It is the lower incidence of MMS and the higher frequency of MNs in the Kuwaiti Arabs compared with these samples that contribute largely to the difference noted between them.

The differences noted in the phenotype distribution between the Kuwaiti Arabs and some of the populations of the Arabian Peninsula are reflected in the frequencies of the gene complexes. The total M frequency of 60 per cent is comparable with the Beduoin of Baghdad (Kayassi et al 1938) but is considerably lower than the values reported for the populations of Arabia. The Kuwaitis exhibit a lower frequency of the gene complex MS, whereas the other Arab populations included in Table 3.6 are characterized by a higher frequency of MS. They have a similar frequency of Ms as found in the Shiah, Sunni of Saudi Arabia and the Arabs of Southern Arabia, but is lower than the Yemenis, Zabidis and the Bedouin. The gene complex NS has a similar incidence and does not show much difference. Except for Zabidis, the Kuwaiti Arabs are found to exhibit a high frequency of the gene complex Ns.

3.3.3 Rh blood group system Table 3.7

As regards the Rh blood group distributions, the statistical analysis was performed by using 6 x 2 contingency table. The different Rh type groups employed were as follows :

R_1R_1 ;

R_1R_2 ;

R_1r ;

R_or :

rr ;

R_1R_z, R_2R_z, r'r, r"r, R_2R_2 and R_2r

It is found that the Kuwaitis are similar to the Sunni and Bedouin of Saudi Arabia (Maranjian et al 1966) and the Arabs living in the south west of Southern Arabia (Marengo-Rowe et al 1974). However, they exhibit a significant variation from the Zabidis, $X_5^2 = 22.317$ $P<0.001$ and the Shiah, $X_5^2 = 19.278$ $0.01<P>0.001$. It is the lower incidence of R_or and the higher frequency of R_1R_1 and rr in the Kuwaitis that accounts for the large Chi-squared value. The Kuwaitis also exhibit just a significant difference from the Hadhramaut Arabs, $X_5^2 = 12.855$ $0.05<P>0.02$. It is the excess of type R_1R_1 in the Kuwaitis that produce the difference noted between the two populations.

The frequency distribution of the Rh gene complexes also reflect some variation in the Arabian Peninsula. The gene complex R_1 is found to rise in incidence from 0.3833 in the Sunni to 0.4564 in the Shiah of Saudi Arabia. In Southern Arabia, the Arab samples show frequencies lying between 0.3286 and 0.5400. The frequency of 0.4920 exhibited by the Kuwaiti Arabs is typical Mediterranean and resembles some of the Arab values. The other notable feature of the Arab populations is the abundance of the gene comples R_o. Its frequency lies between the limits of 0.1099 and 0.3356. However, the present figure of 0.0880 is lower but it shows the presence of this Negroid component. Except for the Yemenite Arabs, the gene complex r exhibits its highest incidence (0.3231) in the Hadhramaut Arabs, while the other Arab samples show frequencies lying between 0.1899 and 0.2989. Again.

the frequency of 0.3246 reported here is comparable with some Arab samples from Saudi Arabia and Southern Arabia.

3.3.4 Other blood group systems Tables 3.8 - 3.9

As shown in Table 3.8, the frequency of the gene K varies between 3.17 and 10.56 per cent in Saudi Arabia and is found to be 4.73 per cent in the inhabitants of Southern Arabia. In the present sample, however, the figure is only 1.87 per cent which is lower than the Arabian values but falls within the wide range reported for the Indian region. Regarding the Kell groups, the Kuwaiti Arabs are found to be consistent with those found in the Shiah of eastern Saudi Arabia, whereas they differ significantly from the Bedouin and Sunni samples of Saudi Arabia and the Arabs of Southern Arabia (Maranjian et al 1966 and Marengro-Rowe et al 1974).

Kuwaiti Arabs v Bedouin	$X_1^2 = 20.909$	$P<0.001$
Kuwaiti Arabs v Sunni (E.Arabia)	$X_1^2 = 7.808$	$0.01<P>0.001$
Kuwaiti Arabs v Sunni (Najd)	$X_1^2 = 20.019$	$P<0.001$
Kuwaiti Arabs v Sunni (W.Arabia)	$X_1^2 = 12.302$	$P<0.001$
Kuwaiti Arabs v Southern Arabia	$X_1^2 = 4.852$	$0.05<P>0.02$

The frequency of the Fy^a gene is found to be very low in Arabia. Maranjian et al (1966) recorded considerable variation in the populations of Arabia, with values from 4.51 per cent for the Shiah to the high figure of 32.51 in a sample of Sunni from western Saudi Arabia. The present figure of 28.32 per cent is in line with the Bedouin (26.32) and Sunni of Najd (32.51). The Kuwaiti Arabs also display a similar distribution of Duffy phenotypes to that found in the Bedouin and Sunni of Najd, whereas they differ from the inhabitants of Southern Arabia, $X_1^2 = 36.926$ $P<0.001$ and Sunni of eastern Saudi Arabia, $X_1^2 = 20.350$ $P<0.001$.

3.4 Serum Proteins

The results of two serum protein systems are as follows :

(i) Table 3.10 shows the distribution of haptoglobin groups and respective gene frequencies in the Kuwaiti Arabs. There was close agreement between the observed and expected phenotypic values, thus confirming the assumption of Hardy-Weinberg equilibrium and that it is a random mating population. Three subjects were found to have the missing phenotype Hp 0-0. The gene frequencies were calculated by excluding the phenotype Hp 0-0. The gene frequencies are : $Hp^1 = 0.3449$ and $Hp^2 = 0.6551$.

(ii) All 161 samples for the transferrin variants were found to have the common type CC.

3.5 Discussion

3.5.1 Haptoglobin Table 3.11

Table 3.11 summarizes the distribution of serum haptoglobin groups and respective allele frequencies in the Arab populations. Unfortunately, no tests have been carried out on the populations of Saudi Arabia, Iraq and Syria so the comparison was made with the Arabs of Southern Arabia, Jordan and the Palestinian Arabs living in Israel.

After statistical analysis, it is seen that the Kuwaiti sample exhibits Hp phenotype and gene distributions consistent with those found in the Jordanian and the Palestinian Arabs (Tills 1969), whereas they are significantly different from the Arabs of Southern Arabia (Tills 1969, Marengo-Rowe et al 1974).

Kuwaiti Arabs v S.Arabian Arabs	$X_2^2 = 8.820$	$0.02<P>0.01$
,, ,, v ,, ,,	$X_1^2 = 7.969$	$0.01<P>0.001$
Kuwaiti Arabs v Southern Arabia	$X_2^2 = 8.030$	$0.02<P>0.01$
,, ,, v ,, ,,	$X_1^2 = 7.952$	$0.01<P>0.001$

Regarding the Hp allele frequencies, it appears that the Arabs are characterized by higher frequencies of the Hp^1 allele. Kirk (1968) states that the Hp^1 frequencies in Europe range from 0.31 to 0.45, with the majority of values from 0.36 to 0.43. He further mentions that the Hp^1 values drop as one passes from Europe into the Middle East and this decline is continued into India. In the present sample, the value of 0.3449 confirms this tendency and fits well into the European range. However, the Hp^1 values found in the Kuwaitis and other Arabs are considerably higher than in India.

3.6 Red Cell Isoenzymes

The results of various enzyme systems are given below :

(i) Table 3.12 reveals the distribution of AP groups and respective allele frequencies in 155 Kuwaiti Arabs. A Chi-squared test showed close agreement between the observed and expected phenotype values confirming the assumption of Hardy-Weinberg equilibrium and that it is a random mating population. The gene frequencies are : $P^a = 0.2032$, $P^b = 0.7774$ and $P^c = 0.0194$.

(ii) The results of the PGM Locus 1 system are set out in Table 3.13. Like the AP system, good agreement was found between the observed and expected phenotype values. The gene frequencies are : $PGM_1^1 = 0.7041$ and $PGM_1^2 = 0.2959$.

(iii) Table 3.14 shows the distribution of AK groups and respective gene frequencies in a sample of 159 Kuwaiti Arabs. Again, close agreement was found between the observed and expected phenotype values confirming the Hardy-Weinberg equilibrium. The calculated gene frequencies are : $AK^1 = 0.9717$ and $AK^2 = 0.0283$.

(iv) Table 3.15 shows the results of the 6-PGD system. The observed and expected values were found to be very close. The gene frequencies are : $PGD^A = 0.9667$ and $PGD^C = 0.0333$.

(v) 150 samples tested for the LDH and MDH variants were found to have the normal type (Table 3.16).

3.7 Discussion

3.7.1 Acid phosphatase Table 3.17

In the AP polymorphism, one of the earliest red cell enzyme systems established in man (Hopkinson et al 1963), genes P^a and P^b are the most common and are found in all populations, but the characteristic distribution of the P^c gene is interesting. The P^c gene is highest in eastern and southern European areas, in Poland approximately 10 per cent (Wysolochowa 1970) and in Italy 8-10 per cent (Modiano et al 1967), but the European frequencies on average range from 5-7 per cent. The P^c gene frequency falls in the Middle East to 0-3 per cent, and still lower values are found in Negro populations, 0-1 per cent (Bhasin and Fuhrmann 1972). For the Arab populations, the highest frequency of the P^c gene is given by Tills (1969) for the Palestinian Arabs living in Israel, 7 per cent. In other samples the P^c gene frequency is 2 per cent. It is absent in the Arabs of Southern Arabia (Tills 1969, Marengo-Rowe et al 1974). In the present sample, the figure of 2 per cent is within the Middle Eastern range. A Chi-squared test of AP phenotypes shows that the Kuwaiti Arabs have the same distribution as found in the Arabs of Jordan and Southern Arabia, whereas they differ from the Palestinian Arabs, $X^2_3 = 8.176$ $0.05<P>0.02$. With respect to the AP alleles, the Kuwaitis are similar to the Jordanians but differ from the Arabs of Southern Arabia, $X^2_2 = 12.981$ $0.01<P>0.001$ and the Palestinian Arabs, $X^2_2 = 7.711$ $0.05<P>0.02$.

3.7.2 Phosphoglucomutase locus 1 Table 3.18

Although the PGM polymorphism was described recently, many populations of the world have been tested for this trait. The investigations show that the distribution of PGM Locus 1 genes is not uniform among the European populations. A higher frequency is found among the Mediterranean peoples (Greece, Italy, Turkey) than among those from England, Germany or the Scandinavian countries (Hopkinson and Harris 1966, Hummel et al 1970 and Modiano et al 1970). The comparative data summarized by Bhasin and Führmann (1972) demonstrate that the Kuwaiti Arabs share the Mediterranean populations trait of high frequency of the PGM_1^2 gene. On the other hand, some of the Arab samples included in Table 3.18 show a low frequency of the PGM_1^2 and are similar to some non-Mediterranean populations. Since the data are missing from the Arab communities in Iraq, Syria and Saudi Arabia, it is difficult to say whether the Arabs of Kuwait exhibit gene frequencies different from these populations or not; but a comparison with the available data show that the present sample exhibits PGM allele and phenotype distributions consistent with those found in the Arabs of Southern Arabia (Marengo-Rowe et al 1974), Jordanian Arabs (Tills 1969) and the Arabs living in Israel (Szeinberg and Tomashevsky 1971)

3.7.3 Adenylate kinase Table 3.19

Data on the distribution of AK groups and their respective allele frequencies are set out in Table 3.19. The AK^2 gene has a frequency of 0.05 in European populations but is rarer in black Africans (Hopkinson 1968) and absent in New Guinea (Kirk et al 1969). In the Middle East, the AK^2 gene frequency is found to lie between the limits

of 0.0252 in a sample of South Arabian Arabs and 0.0687 in the Kurds (Tills et al 1970a). The frequency of AK^2 found in the Kuwaiti Arabs, 0.0283, lie within the Middle Eastern and European range. The Kuwaiti Arabs also exhibit phenotype and allele distributions consistent with those found in all the samples included in Table 3.19.

3.7.4 6-Phosphogluconate dehydrogenase Table 3.20

Among the wide fluctuations in the frequency of the Canning gene (PGD^C) in World populations, one of the extremely high values of PGD^C (0.2305) was found in the population of Bhutan (Mourant et al 1968). Among the Arab populations, the inhabitants of Southern Arabia gave the highest frequency of 0.1149 (Marengo-Rowe et al 1974), but otherwise most PGD^C gene frequencies range from 0.0437 to 0.0747. Compared with the European figures, the Arab populations are found to have a higher frequency of PGD^C, whereas the Kuwaiti Arabs have a frequency of 0.0333 which shows little difference from the European figures and is considerably lower than most of the Arab values.

Using a Chi-squared test, it is noted that the Kuwaiti Arabs differ from the Arabs of Southern Arabia (Marengo-Rowe et al 1974) and the Palestinian Arabs (Tills 1970b), with respect to phenotypes and genes. These differences are accounted for by the high frequency of PGD^A and phenotype AA and the lower incidence of PGD^C and phenotype CC.

Kuwaiti Arabs v Southern Arabia	$X_1^2 = 16.065$	$P<0.001$
,, ,, v ,, ,,	$X_1^2 = 16.291$	$P<0.001$
Kuwaiti Arabs v Palestinian Arabs	$X_1^2 = 4.312$	$0.05<P>0.02$
,, ,, v ,, ,,	$X_1^2 = 4.088$	$0.05<P>0.02$

However, the Kuwaitis are similar to the Jordanian Arabs.

3.8 Conclusion

The serological findings of the Kuwaitis which have been presented for the first time show some resemblances with the Arabs of the Arabian Peninsula and neighbouring Arab States. In the case of the ABO blood group system, the Kuwaitis fit well into the framework of the Arabs of the Arabian Peninsula. However, they differ from the Arabs of Iraq, Syria, Jordan and Lebanon in having a rather higher frequency of O and a lower one of B. On the whole, the MNS frequencies of the Kuwaiti Arabs are typical of the "Mediterranean" region. The main difference from the populations of the Arabian Peninsula is the low frequency of the gene complex MS. Considering only the single MN groups. it appears that the total M frequency is comparable with the Arabs and Bedouin of Iraq but is lower than the Arabian average. Also the Rh frequencies fit reasonably well into the Arabian picture and agree with those found in the Arabs living in the south west of Southern Arabia The Kell gene is of particular interest. Its frequency, shown by the Kuwaiti Arabs, is considerably lower than the Arabian average and falls in the European and Indian range. The Duffy gene, lower than the European range, is close to the Bedouin and Sunni of Saudi Arabia.

Of the two serum protein systems, the transferrin is found to be invariant. The Hp^1 frequency is within the normal range found in Europe and the Middle East. Among the six red cell enzyme systems investigated, lactate dehydrogenase and malate dehydrogenase are found to be invariant. The frequencies of the three systems - AP, PGM and AK - are within the Middle Eastern range. The low frequency of the Canning gene, PGD^C, in the Kuwaitis, is much below the Arabian values and is close to the European figures. However, much more research is needed to be done on the populations of the Arabian Peninsula and other Arab countries in order to know the exact affinities of the Kuwaiti Arabs.

CHAPTER IV

GENETIC POLYMORPHISMS IN IRAN

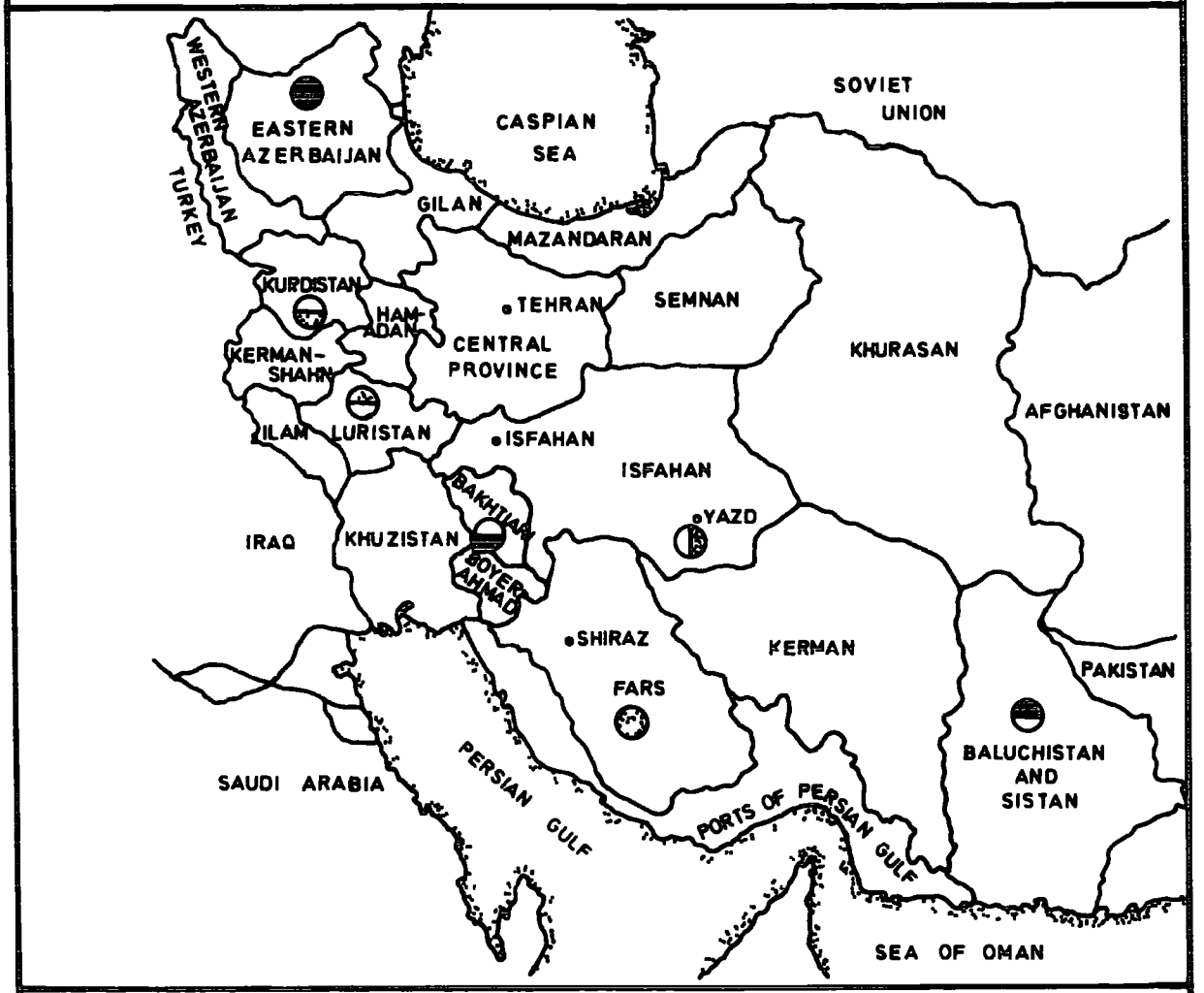
4.1 Introduction

4.1.1 Geographical setting

"Iran" means a land of Aryans, what the Greeks and then the West called Persia. It is situated between 44° and 63° longitude and 25° and 40° latitude. It has an area of 628,000 square miles. The Iranian plateau is a triangle set between two depressions - the Caspian sea to the north and the Persian Gulf to the south. Further, between central and western Asia, it forms a link between the steppes of inner Asia and the plateau of Asia Minor and beyond Europe. Geography can thus account for the historic past which the plateau was called on to play in the course of thousands of years of history.

The country is bounded on the north by the Soviet Union and the Caspian Sea, on the east by Afghanistan and Pakistan, on the south by the Persian Gulf and sea of Oman, and on the west by Turkey and Iraq. High mountains cover half the total area and form two famous ranges, one in the north (Alborz) and the other stretching from northwest to southeast (Zagros). Its centre is dominated by two deserts, the great Kavir (Dash-i-Kavir) and Southern Lut (Dash-i-Lut). The heart of the country is the Tehran-Isfahan area. To the south is the important province of Fars, with its principal city of Shiraz. Further to the east is the province of Kirman and Bam. This area is isolated from the rest of Iran by deserts to the west between Kirman and Yazd and to the east between Bam and Seistan. In the northeast is the province of

Figure 4 1 IRAN - GEOGRAPHICAL SETTING AND MAJOR ETHNIC GROUPS



Khurusan with its largest city, Meshed. Further to the south and east is the barren land of Baluchistan (Figure 4.1).

4.1.2 Historical review

Although Iran has more than her share of prehistoric mounds and sites, far too little archaeological work has been done to permit a reconstruction of her history before the advent of the Achaemenids. Excavations in one cave at Behistun and in the caves called Belt and Hotu near Behshahr on the Caspian coast represent a splendid beginning. The finds indicate an active flint industry in the Middle Palaeolithic period as well as skeletons and skulls of the Mesolithic period. This material has been dated to about 10,000 B.C. by the carbon-14 method. About 6,000 B.C. some of the inhabitants of Iran discovered agriculture, practised animal husbandry, and made painted pottery and polished stone implements. The physical characteristics of these people are not known. However, the finds at Susa and Khuzistan showed the Indo-European names in the cuneiform tablets of the Mittani period in Mesopotamia and indicate the presence of these people in Iran.

About 2,000 B.C., the Aryans came to Iran from the plains of the southern U.S.S.R. One stream of this great migration moved into present day India and the other stream came into Iran. Gradually these people took the place of the original inhabitants. In the 19th century B.C., nomadic tribes like the Medes, Persians (Parsa) and Parthians entered Iran from the northern side of the Caspian Sea and by way of the Caucasus. They mixed with the former inhabitants of the country. The Medes settled in what is today the north-west of Iran; the Persians lived further south, roughly in the modern provinces of Fars and Khuzistan. The inflow of small communities of Greek origin after 331 B.C. also changed the ethnic

map of Iran. Following the Greek period, the Sassanian dynasty (226 - 641 A.D.) was established.

The next major movements which resulted in the present diversity was the inflow of Arabs, Turks and Mongols. Arabs came to Iran in the 7th century A.D. The transfer of tribes and the incursion of wandering Arab groups from Iraq had an effect on the racial mixing within the country. The Turkish invasion (1050 A.D.) changed the ethnic face of Iran. All of Anatolia, Azerbaijan and other areas were Turkified. The Turkish speaking tribes scattered throughout the country. People of the north-east and north-west showed strong intrusion of Turkish and Tartar blood. The Mongols (1200 A.D.) ruled Iran for a century and strongly influenced the people of the country. Eventually the Mongols were converted to Islam and this facilitated the mixing with Persians already living in Iran.

4.1.3 Ethnic groups in Iran

The ethnic groups inhabiting Iran today are in many cases the descendants of the invaders who came to conquer but who remained and became assimilated, in part or in whole. Least assimilated have been those groups who remained nomadic and retained their own languages and customs. To the author's knowledge, very little is known about the physical anthropology of these groups. A classification based on language, tribal affiliations and small size of certain groups is as follows :

(i) Linguistic groups - Persian, Turkish and Arabic speaking peoples

(ii) Tribal groups - Kurds, Lurs, Ghash Quays etc

(iii) Minority groups - Armenians, Arabs, Jews etc.

Of the two major language groups, Persian and Turkish, Persian predominates. Those who speak Persian or a related dialect constitute three-fourths of the population. They form the bulk of the urban population and also inhabit the villages on the Iranian plateau. Most of them belong to the Shia'a sect, but a few continue to practise Zorastrianism and a smaller number belong to the Ba'hai sect. The Turkish-speaking people live in the northern province of Azerbaijan. These people live in villages and occupy themselves with farming. The Arabic-speaking group is the least prevalent of the three groups.

Among the tribal groups, the most important are Kurds, Bakhtiaris, Lurs, Ghash Qays and Baluchis. All these tribes, except Ghash Qays speak dialects related to Persian. The Kurds constitute the largest tribal group in Iran (about two million), in addition to those who live in Iraq, Turkey and the Soviet Union. They belong to the Sunni sect of Islam and live in the highlands of the Zagros in western Iran. South of the Kurds are the Bakhtiaris and Lurs, who together may number half a million. The Bakhtiaris are a nomadic people, not intermixing until very recently with other Iranians. The Luis live in Luristan and are related to the Kurds. The origin of the Lurs is still unknown. Henry Field (1939) thinks that they are a part of the original Iranian stock that migrated from the regions to the east of the Caspian sea during the first half of the first millenium B.C. They have, however, a strong admixture of Arabic blood.

The Ghash Qays occupy a region south of Shiraz. They are a group of Turkish origin with a language of their own. Their Mongoloid features are stronger than, for instance, those of Ottoman Turks in the N.W. of Iran, who are considered to be a product of intermixing with the original ancient population of Asia Minor. In general, the Ghash Qays resemble the Lur nomads (Henry Field 1939). In appearance

they compare favourably with their Lur compatriots. They seem on the whole to be taller and fairer than the Bakhtiaris, and certainly more than the Lurs. The Baluchis live in the barren province of Baluchistan.

Of the minority groups, the well known are the Armenians, Zoroastrians, Assyrians and Arabs. The Armenians account for a substantial number of peasants in Azerbaijan. They are non-Moslem. The difference in religion is the reason why no intermixing with other populations takes place. Zoroastrians are found as an isolated group in Yazd. They rarely inter-marry with the Shi'a Moslems. They are also the ancestors of the modern Parsis of Bombay. The Assyrians were originally an Arab-Semitic agricultural people. It is believed that in 650 A.D. when their country was conquered by Moslems, they fled to the mountains where they maintained themselves as a separate group among the Zoroastrians. Up to now this group has maintained its own language and script. The Arabic groups in Iran have not mixed with other population elements.

4.1.4 Genetic studies in Iran

Knowledge on the distribution of blood groups, serum proteins and red cell isoenzymes in Iran has been rather limited. Extensive data are only available regarding the ABO polymorphism (Boué and Boué 1955-56). Very little is known about the other blood group polymorphisms - MNSs, Rh, Kell, Duffy etc. Nijenhuis (1964), Sunderland and Smith (1966), Bajatzadeh and Walter (1969) published some frequency data in detail but this information is not sufficient to understand the distribution pattern of genes for these polymorphisms in Iran as a whole. Data regarding the serum protein (Hp) were first reported by Walter and Djahanschahi (1963). Similar information about the Iranians living in different parts of Iran was reported by Bajatzadeh and Walter.

Few published observations are available on the red cell enzymes of the inhabitants of Iran. Bowman and Ronaghy (1967) first studied the distribution of some isoenzymes in the Moslems of Iran. Subsequently others looked at some other populations in Iran (Farhud et al 1973, Lehman et al 1973).

The aim of this chapter is to enlarge our knowledge of the distribution of blood, serum and enzyme groups in Iran, and to contribute to the better understanding of genetic polymorphisms in South West Asia.

4.2 Blood Group Antigens

The results of the Iranians tested with different antigens are as follows :

(i) Table 4.1 presents the distribution of the ABO blood groups and respective gene frequencies, after testing with and without anti-A_1 serum, in the Tehran and Isfahan Iranians. There was good agreement between the observed and expected phenotypic values in both samples, thus confirming the Hardy-Weinberg equilibrium. Using the Chi-squared test, both samples were found to exhibit homogeneity with respect to the common ABO phenotypes. The two samples, being similar, were pooled to give a total sample of 205.

The results of the A_1A_2BO system show that the allele p_2 exhibits very little variation, having a frequency of 0.0325 in the total sample. Similarly, the gene p_1 shows little variation between the two samples. The gene q shows a range of 4%, from 0.1199 in the Tehran Iranians, to 0.1595 in the Isfahan Iranians. The frequency of the gene r is slightly higher in the Tehran Iranians (0.7034) whereas it is 0.6582 in Isfahan and 0.6798 in the total sample. The gene frequencies of the 99 Tehran Iranians, tested without anti-A_1 serum, are :
$p = 0.1565$, $q = 0.1748$ and $r = 0.6687$.

(ii) Table 4.2 exhibits the frequency of MN blood groups and respective gene frequencies in the Tehran and Isfahan Iranian samples, expressed in terms of 3 phenotypes, after testing with 2 antisera. Close agreement was found between the observed and expected phenotype values, thus confirming the assumption of Hardy-Weinberg equilibrium. Statistical analysis demonstrated no difference between the two samples so they were combined into one relatively large representative Iranian sample.

Table 4.3 shows the results for 137 Tehran Iranians tested with 4 antisera. Again, good agreement was found between the observed and expected phenotypic values.

The gene frequencies in the samples included in Table 4.2 show some variability. The gene M rises from 0.6293 in the Tehran Iranians to 0.6847 in the Isfahan Iranians, with an incidence of 0.6657 in the total sample. Regarding the N gene, the frequency is found to be lower in the Isfahan Iranians and the variability is about 6%. The frequency of the gene complexes, tested with four antisera, are : MS = 0.3187, Ms = 0.3455, NS = 0.0938 and Ns = 0.2420.

(iii) Table 4.4 illustrates the distribution of Rh types and gene complex frequencies in two Iranian samples. The specimens were tested with the following antisera; anti-D, -C, -E, -c, -e and $-C^{W}$. No statistically significant heterogeneity could be demonstrated between the Tehran and Isfahan Iranians. Also, the Rh gene complexes do not exhibit much variability. The gene complex frequencies are : R_z = 0.0151, R_1 = 0.4871, R_1^{W} = 0.0147, r' = 0.0175, R_2 = 0.1229, r'' = 0.0090, R_o = 0.0141 and r = 0.3196.

(iv) Table 4.5 shows the observed numbers, their frequency and calculated gene frequencies for the Kell system. The two Iranian samples, being similar, were pooled to produce a relatively large series having a frequency of K(+) of 4%. Table 4.6 demonstrates the results for 49 Tehran Iranians tested with anti-JK^a serum. The frequencies for the Kidd system are : JK^a = 0.4849 and JK^b = 0.5151. The frequencies exhibited by the Isfahan Iranians, tested with anti-Lu^a serum, are : Lu^a = 0.0681 and Lu^b = 0.9319.

4.3 Discussion

4.3.1 ABO blood group system Table 4.7a

As can be seen from the table, all population groups, except the Yazdi Iranians and Turkomans, show a higher frequency of gene A than of gene B. In this investigation, the 99 Tehran Iranians tested without anti-A_1 serum showed a higher frequency of gene B than that found in the series from Tehran tested by Motamed (1949), Azhir (1951) and Boué and Boué (1955). Using a Chi-squared test, it is seen that the Tehran Iranians exhibit overall similarity in ABO group distribution to the Iranians of Tehran, Azerbaijan, Kurdistan and Yazd (Boué and Boué 1955, Sunderland and Smith 1966). However, the Tehran Iranians differ significantly from the Turkomans, $X^2_3 = 14.620$ $0.01<P>0.001$.

Table 4.7b presents comparative data on the Iranian populations tested with anti-A_1 serum. The A_2 gene is present in all the populations and its frequency ranges from 0.013 to 0.080. The value of 0.0325 obtained in this study is within the range and compares favourably with previously obtained values of 0.029 for the Iranians (Nijenhuis 1964), 0.0404 for Tehran Iranians (Boué and Boué 1956) and 0.048 for Iranians (Bajatzadeh and Walter 1969). The frequency of the gene B varies between 10% and 25% and that of O between 50% and 70%. The frequencies of genes B and O exhibited by the Iranians are again within the range and closer to the figures on Iranians given by Nijenhuis.

After statistical analysis, the present Iranians are found to be similar to the Iranian series of Bajatzadeh and Walter, to the samples of Tehran Iranians of Boué and Boué and to the Kurdish series of Lehman et al (1973). However, they are significantly different from the Armenian sample of Nijenhuis, $X^2_4 = 12.545$ $0.02<P>0.01$, who exhibit a

very high frequency of A_1 and a lower incidence of O. The Iranians also differ from the Yazdi Shia sample of Sunderland and Smith (1966), $X^2_4 = 10.510$ $0.05<P>0.02$. This difference is largely due to the higher frequency of gene ^{B} exhibited by the Yazdi Shia.

4.3.2 MNSs blood group system Table 4.8a - 4.8b

Mourant (1963) suggests that the frequency of the M gene is well above 60 per cent in nearly every Indian population tested, whereas it is below 60 percent and mostly below 55 percent in the Mediterranean area. In this study the M frequency of 0.6657 is much higher than in Western Europe, however, and is characteristic of the belt 60-65 percent which sweeps across Finland to south-east Asia, including the Caspian Sea area and much of the Arabian peninsula. In the Iranian populations, the frequency of the gene M varies between 56 and 67 percent and the present value of 67 percent is within the range. After statistical analysis, it is seen that the present sample of Iranians is similar to the Iranian samples of Nijenhuis (1964) and Bajatzadeh and Walter (1969), with respect to phenotypes and genes. Compared with the ethnic samples of Nijenhuis, it is found that the Iranians differ only from the Armenians with regard to the genes, as $X^2_1 = 4.743$ $0.05<P> .02$.

The Tehran Iranians, tested with four antisera, exhibit MNSs phenotypes consistent with those found in the Kurdish series of Lehman et al (1973) and the Yazdi Shia sample of Sunderland and Smith (1966). The gene complexes do not show much variability. The frequency of the gene complex MS is comparable with the Kurds of Baneh, but is slightly higher than the Yazdi Shia. The frequency of Ms is close to the Kurds and Shia. The gene complex NS is the same and Ns has a lower frequency than that found in the above-mentioned samples.

4.3.3 Rh blood group system Table 4.9

As shown in the table, the Iranian populations appear to exhibit a higher frequency of the gene complex r which ranges between 0.205 and 0.393. The frequency of 0.3196 obtained for the present Iranians is within the range. The gene complex R_1 (0.4871) is comparable with the figures of 0.492 for the Iranians and 0.501 for the Bachtiari (Nijenhuis 1964). It is, however, mugh higher than the Yazdi Shia. The incidence of R_2 is again in line with most of the Iranian groups. Furthermore, the frequencies of the rare gene complexes do not show much variation, only with the exception of Arabs who possess a higher frequency of R_o. Overall, it appears that the Iranians exhibit Rh gene complexes as found in the Mediterranean.

For statistical purposes, the Rh type groupings employed were as follows : R_1R_1, $R_1^WR_1$; R_1R_2, $R_1^WR_2$; R_1r, R_1^Wr; R_2r; rr; R_1R_z, r'r', R_2R_2, R_or. Using the 6x2 contingency table, it is found that the present Iranians are similar to the Iranian samples of Nijenhuis. They differ from the Yazdi Shia samples of Sunderland and Smith (1966) who exhibit a lower incidence of type R_1R_2 and a higher frequency of type R_2r. Also the differences exist between the Iranians and the ethnic samples of Nijenhuis.

Iranians v Bachtiari	$X^2_5 = 25.230$	P<0.001
Iranians v Kurds	$X^2_5 = 13.972$	0.02<P>0.01
Iranians v Arabs	$X^2_5 = 14.228$	0.02<P>0.01

These differences are generally accounted for by the types R_1r and R_2r compared with the Iranians.

4.3.4 Other blood group systems Table 4.10

In European populations, the frequency of the gene Lu^a ranges from 2 to 4 percent. It is absent in the Asians and Australian aborigines (Mourant 1954). The present value of 7 percent found among the Isfahan Iranians is higher than the European values and shows the presence of gene Lu^a at an appreciable level in the Middle East.

Regarding the Kell system, the frequency of the Kell gene (K) is relatively uniform throughout Europe, ranging between 1.54 and 5.82 percent (Mourant 1954). Data summarized in Table 4.10 show that the frequency of the gene K in the Iranian populations varies between 2 and 6 percent. In this survey, however, the figure is only 2 percent but is within the range. Again the variation in Kell groups between the present sample and the two Iranian series of Nijenhuis (1964) and Bajatzadeh and Walter (1969) is statistically not significant. The Iranians are also found to be similar to most of the ethnic samples of Nijenhuis. They are statistically different from the Yazdi Shia, $X_1^2 = 4.315$ $0.05<P>0.02$ and Arabs, $X_1^2 = 7.099$ $0.01<P>0.001$, with respect to the Kell groups.

The frequencies of the Kidd system exhibited by the Tehran Iranians are comparable with the English series of Race et al (1951).

4.4 Serum Proteins

The results of two serum protein systems are given below :

(i) The distribution of Hp groups and respective gene frequencies in the Tehran and Isfahan Iranian samples is shown in Table 4.11. The gene frequencies were calculated excluding the phenotype Hp 0-0. Both the samples showed close agreement between the observed and expected phenotypic values, thus confirming the assumption of Hardy-Weinberg equilibrium. No Hp 2-1 (modified) or other rarer phenotype was detected. Two subjects with phenotype Hp 0-0 were detected in the Isfahan sample. No such individual was found in the Tehran sample. Statistical analysis demonstrated that there is no significant heterogeneity among the samples, with respect to either phenotypes or genes, so they were combined into one sample. The gene frequencies in the combined sample of Iranians are : $Hp^1 = 0.2964$ and $Hp^2 = 0.7036$.

(ii) The distribution of Tf groups and respective gene frequencies in Isfahan Iranians are set out in Table 4.12. All the Iranians from Tehran exhibited the type CC. A single individual from Isfahan was typed as CD. The sub-typing of the D variant was not performed, but when run with a CD control on the starch-gel the present CD variant showed similar mobility.

4.5 Discussion

4.5.1 Haptoglobin Table 4.13

Data on the distribution of the Hp^1 gene frequencies in the various populations of Iran are set out in Table 4.13. With the exception of the Iranian Zoroastrians (0.2157) tested in Bombay, the Hp^1 gene frequencies in the Iranian populations are relatively higher. It is seen from the table that some differences in the distribution of the Hp gene frequencies exist in Iran. The Hp^1 values are found to be lower in the North-West (0.272), Centre (0.286) and East (0.291) of Iran (Bajatzadeh and Walter 1968). Similar low values were reported by Bajatzadeh and Walter (1969) in the North-Western (0.276), Central (0.263) and Eastern (0.249) parts of Iran. By contrast, slightly higher frequencies are found in Tehran (0.324), the North (0.310) and among the Kurds (0.3264). After statistical analysis, it is found that these differences are not statistically significant. Regarding the Hp^2 gene frequencies it is however higher in the eastern part of Iran. Bajatzadeh and Walter (1969) give Hp^1 value of 0.288 for the Iranian Moslems, and the present value of 0.2964 is not statistically different from Bajatzadeh's value.

4.5.2 Transferrin Table 4.14

The frequency of Tf variants, other than type Tf CC, found in any Middle East population, is low, usually 1%, and no work has been carried out to study the distribution of Tf variants in Iran. For comparative purposes the distribution of Tf variants in Middle East populations is summarized in Table 4.14. Except for the Yemenite and

(Tills 1969 and Berne et al 1970)

Habbanite Jews, the variant Tf CB is absent in the populations of the Middle East. Transferrin CD appears to be more common and has been reported among Palestinian Arabs (Tills 1969) and the Arabs of Southern Arabia (Marengo-Rowe et al 1974). The frequency of Tf CD found among the Isfahan Iranians is comparable with the above mentioned samples from the Middle East.

4.6 Red Cell Isoenzymes

The results of the red cell isoenzymes are as follows :

(i) Table 4.15 presents the observed number and frequency of common AP phenotypes together with the respective gene frequencies found in Tehran and Isfahan Iranians. Both the samples showed good agreement between the observed and expected phenotypic values, thus confirming the assumption of Hardy-Weinberg equilibrium and that they are random mating populations. No rarer AP phenotype was detected. Both the samples showed an absence of phenotype CC. Statistical analysis demonstrated that there is no significant heterogeneity among the samples, with respect to either phenotypes or genes and therefore the Iranians can be regarded as a homogenous population.

The frequency of the P^a allele exhibits a variability of 7% within the two samples. The P^b allele shows a range of 7% rising from 0.6367 in Tehran Iranians to 0.7143 in Isfahan Iranians. The rarest allele P^c does not exhibit much variability. The gene frequencies exhibited by the combined sample are : $P^a = 0.3193$, $P^b = 0.6618$ and $P^c = 0.0189$.

(ii) The frequencies of the PGM Locus 1 phenotypes and genes are given in Table 4.16. Close agreement, calculated on the basis of Hardy-Weinberg equilibrium, was found between the observed and expected phenotype values. No heterogeneity was found between the two Iranian samples. A rarer phenotype PGM 6-1 was detected in an Isfahan subject. The allele PGM_1^2 do not show much variability within the two samples. The frequencies shown by the Iranians are : $PGM_1^1 = 0.7590$, $PGM_1^2 = 0.2390$ and $PGM_1^6 = 0.0020$.

(iii) Table 4.17 shows the distribution of AK phenotypes and respective allele frequencies in samples from Tehran and Isfahan. Close agreement was found between the observed and expected phenotype values which confirms the assumption of Hardy-Weinberg equilibrium. No significant heterogeneity was demonstrated among the samples, so they were pooled together into one. No rare AK phenotypes were discovered in any of the samples examined.

The variation in the frequency of the AK^2 allele is 1% rising from 0.0422 in the Isfahan Iranians to 0.0536 in the Tehran Iranians, the incidence being 0.0498 in the combined sample.

(iv) The observed number and frequency of 6-PGD phenotypes together with the respective gene frequencies found in two samples of the Iranians are set out in Table 4.18. Like the AK system, good agreement was found between the observed and expected phenotypic values. No significant heterogeneity was found among the samples, with respect to either phenotypes or genes. No variants other than the 'common' CA type was found. The PGD^C allele rises from 0.0242 in Tehran Iranians to 0.0281 in Isfahan Iranians. The frequencies exhibited by the combined sample are : $PGD^A = 0.9744$ and $PGD^C = 0.0256$.

(v) All the specimens from Tehran and Isfahan showed the normal LDH phenotype (Table 4.19).

(vi) Table 4.20 shows the distribution of PHI phenotypes and genes in the Tehran Iranians. All the 88 Isfahan Iranians showed the normal type PHI 1-1. The gene frequencies exhibited by the Tehran Iranians are : $PHI_1^1 = 0.9939$ and $PHI_1^3 = 0.0061$.

(vii) Of the 254 specimens tested for soluble MDH phenotypes, all were found to be type 1 (Table 4.19).

4.7 Discussion

4.7.1 Acid phosphatase Table 4.21

Table 4.21 shows that P^b is the most common of the three major alleles in each Iranian sample investigated. Its frequency ranges from 0.6429 in the Kurds of Marivan and Baneh (Lehman et al 1973) to 0.7500 in the Zoroastrian Iranis of Bombay (Undevia et al 1972). A lower range of values (0.2292 to 0.4043) characterizes the P^a allele. The present values of P^a (0.3193) and P^b (0.6618) are within the range of variation. The P^c gene is present in all the Iranian populations tested so far, the highest frequency being 0.030 in the Iranians coming from different parts of the country. In the present sample, the allele P^c is 0.0189 and is comparable with the other Iranian samples. The frequencies of the three alleles in the Iranian sample of Walter and Bajatzadeh (1968) are consistent with those found in the present sample but it is different in the proportion of common phenotypes, $X_3^2 = 8.687$ $0.05<P>0.02$ (phenotype C, CA and CB were amalgamated for statistical purposes). The difference between the two samples was found in the case of phenotype BA which exhibited variability of 10%. The Iranians are similar to the Kurdish sample of Lehman et al and the Zoroastrian Iranian sample of Undevia et al. However, they are found to be statistically different from the Parsis sample of Undevia et al, with respect to the phenotypes, $X_2^2 = 8.722$ $0.02<P>0.01$ and genes $X_1^2 = 9.358$ $0.01<P>0.001$. Compared with the Caucasoid populations, it appears that the Iranians are characterized by a higher frequency of P^b and the P^c frequency does not differ much.

4.7.2 Phosphoglucomutase locus 1 Table 4.22

In general, the PGM_1 frequencies of the Iranians show marked similarities to Middle East and European values. The frequencies of PGM_1^2 in the Iranian populations lie between the limits of 0.2208 and 0.3150. The value of 0.2390 observed in this investigation is within the range. The frequencies of PGM_1 alleles and phenotypes in the Iranians are consistent with those found in the Zoroastrian Iranis and Parsis sample of Undevia et al (1972). However, the present Iranians show slightly significant difference from the Iranian sample of Farhud et al (1973), with respect to genes only, as $X_1^2 = 4.455$ $0.05<P>0.02$. They also differ from the Kurdish sample of Lehman et al (1973), with regard to phenotypes, $X_2^2 = 7.249$ $0.05<P>0.02$ and genes, $X_1^2 = 3.869$ $0.05<P>0.02$. This difference is mainly due to the higher incidence of PGM_1^2 and type 2-2.

Besides the common three phosphoglucomutase phenotypes, some variant type, PGM 6-1, was present in the Isfahan Iranians. No rare variants of the PGM Locus 1 have been reported from Iran so far. Hopkinson and Harris (1966) demonstrated nine rare phenotypes determined by the rare alleles PGM_1^3 to PGM_1^7. Out of all these phenotypes, type PGM 6-1 was observed in a Turkish Cypriot and two Nigerian Negroes. Among the Asiatic populations, it occurs frequently and the present investigation confirms.

4.7.3 Adenylate kinase Table 4.23

As can be seen from the table, very few observations for the AK system on Iranians have been reported so far. Bowman and Ronaghy (1967) found a frequency of 0.0497 for the AK^2 allele in 322 Iranian

Moslems and Undevia et al (1972) give a value of 0.0670 among Parsis living in Bombay. These values are similar to those found in the present investigation for the Iranians (0.0498). The Iranians are also similar in AK phenotypes and genes to the Kurdish series of Lehman et al (1973), even though the frequency of 0.0762 in Kurds is one of the highest reported so far in an Iranian population. The AK^2 allele is low in Iranian Zoroastrians (0.0208) but not statistically different from the earlier samples.

In European populations, the frequencies of the AK^2 gene lie between the limits of 0.015 and 0.056 (Bhasin and Fuhrman 1972). Reported values in the Middle East range from 0.0252 to 0.0687 (Tills et al 1970a). The present value of 0.0498 falls within this range and demonstrates a similarity between different populations of the Middle East and Europe.

4.7.4 6-Phosphogluconate dehydrogenase Table 4.24

As for the AK system, observations for 6-PGD in Iranians are very limited. Farhud et al (1973) gave a value of 0.0153 for the PGD^C allele in Iranians and Bowman and Ronagny (1967) found a value of 0.0280 in Iranian Moslems. Similar low values were reported in the Parsis (0.0263) of Bombay (Undevia et al 1972). In this survey, the PGD^C frequency of 0.0256 for the combined sample of Iranians is comparable with the earlier samples. Further comparison with the Kurdish series of Lehman et al (1973) shows that the Kurds of Marivan and Baneh have the highest frequency of PGD^C (0.0714) in Iran but are statistically insignificantly different from the Iranians with respect to phenotypes and genes.

The PGD^C frequencies in European populations range from 0.0086 to 0.0392 (Tills et al 1970b). In the Middle East the PGD^C allele varies

from 0.0279 in the Moslems of Iran to 0.1098 for South Arabians (Tills et al). The value of PGD^C for the Iranians reported here lies within the range for Europeans. However, it is lower than for some other Middle Eastern populations.

4.7.5 Phosphohexose isomerase

Variants of PHI have been reported to be absent in Iranians (Farhud et al 1973). In this survey, however, two Iranians from Tehran exhibited the 3-1 phenotype. Detter et al (1968) found that the Asiatic populations exhibit an appreciable frequency of the allele PHI_1^3 and the present study confirms this tendency. Also, the frequency of 0.0061 here is comparable with the values given for the North Indians by Blake et al (1971).

4.8 Discussion

4.8.1 Haemoglobin Table 4.25

Table 4.25 shows that a single individual from Tehran exhibited a heterozygous Hb-AD. All the subjects from Isfahan showed the normal type Hb-AA. Knowing the fact that the electrophoretic properties of haemoglobins S and D are indistinguishable on the cellulose acetate paper, the confirmation was established by the technique of Titan IV citrate electrophoresis.

The present estimated allele frequency in the Tehran Iranians (0.0030) is lower than the value given for the Iranian Moslems (0.006) by Bowman and Ronaghy (1967). Overall, it confirms the reported existence of Hb-AD in Iran.

4.9 Conclusion

The serological results do not reveal significant differences between the Tehran and Isfahan Iranians. Of all the blood group antigens studied here, the ABO and Lutheran systems are of great interest. Concerning the ABO system, it is seen that all the previous tests carried out on Iranians from Tehran showed a higher frequency of gene A. Similarly, the present sample of Iranians, tested with anti-A_1 serum confirmed this tendency. But a small sample of the Tehran Iranians(79), tested without anti-A_1 serum, exhibited a higher frequency of gene B. This is an unexpected finding and requires further investigation. On the other hand, the distributions of MN, Rh and Kell groups are consistent with those found in the Iranians and certain other groups tested. Frequency distributions of allelic genes controlling the Lu^a and JK^a antigens have been presented for the first time. The frequency of the Lu^a gene found in the Isfahan Iranians is higher than in Europeans and clearly shows the presence of the Lu^a gene in Iran. However, it must be substantiated by further research whether this high frequency is due to small sample size or is simply a characteristic of the Iranians.

Analysis of the serum protein polymorphism Hp reveals that the Iranians exhibit frequencies of the alleles consistent with those found in European populations. Statistical analysis did not demonstrate any difference between the Tehran and Isfahan Iranians with respect to the Hp phenotypes and genes. Results for transferrins have been reported for the first time and could be valuable in differentiating various population groups in Iran. However, much more research needs to be done to understand the exact distribution of transferrin variants in the country.

Of the seven red cell enzymes investigated, five were found to be polymorphic. These are AP, PGM, AK, 6-PGD and PHI. No major

difference was found between the two Iranian samples. In general, the frequencies of the first three systems show marked similarities with other Middle Eastern and European values. However, the value of PGD^C shown by the present sample of Iranians lies within the range for Europeans and is lower than some of the Middle Eastern populations. Again the phenotype and allele frequencies of the Iranians are similar to those found in the earlier Iranian samples. Regarding the acid phosphatase system, the Iranians differ from the Parsis of Bombay. Such a difference was reported by Undevia et al (1972) between the Iranian Zoroastrians and the Parsis. However, Undevia et al suggest that the differences observed among the Parsis are the result of selection, genetic drift and hybridization. The MDH system has not been studied previously in Iran. Both LDH and MDH systems have been found to be invariant. The results of LDH are in agreement with the previous studies in Iran. The PHI system is of potential interest because the PHI_1^3 allele frequency is found to be higher in the Asiatic populations. Like the Asiatics, the Iranians also show an appreciable frequency of the PHI_1^3 allele, and this finding suggests the importance of conducting further studies in Iran. The presence of Hb-AD is of great anthropological interest, but still the information regarding the distribution of abnormal haemoglobins in Iran is scanty.

Finally, the information supplied by the present study leads to the conclusion that the Iranians from Tehran and Isfahan comprise a genetically homogenous group.

CHAPTER V

GENETIC POLYMORPHISMS IN PANJABIS OF NORTHERN INDIA

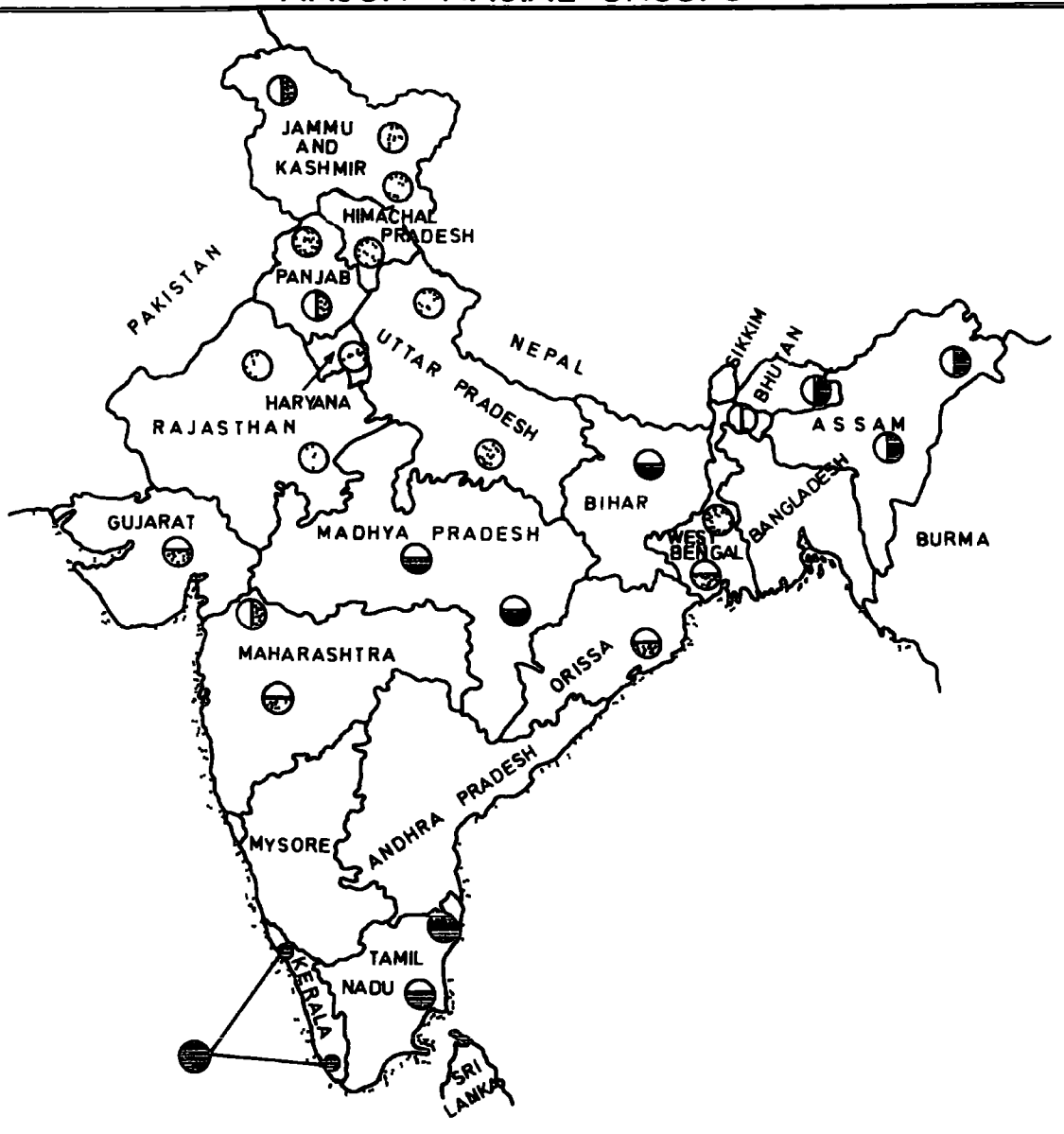
5.1 Introduction

5.1.1 Historical review

Traces of man in his primitive stage are as yet few and confused in India. Archaeologists surmise that the traces of human presence on the Indian sub-continent stem from the time of the second interglacial period and immediately after, perhaps as long ago as 400,000 B.C. The evidence for this period consists of the stone tools which show striking uniformity with those found in some other parts of the world, especially of the west of India, and of the same age. No skeletal remains of Palaeolithic man have been discovered in India so far. The excavations at Mohenjo-daro and Harrapa prove the existence of a great civilization in the west of India, which is believed to have reached its height between about 3250 and 2750 B.C.

The racial history of India opens with the settlement of Aryans in the North-West. They were nomadic tribes who injected new life into the aboriginal cultures. These people were not homogeneous but were composed of various communities or tribes with independent traditions, the only bond between them being a common language a form of Sanskrit. At the time of the Indo-Aryan settlement, the greater part of the country was occupied by Dravidians, also known as Dasas or Dasus. As the Aryan tribes spread eastward through and beyond the "land of five rivers", they came in contact with aboriginal races. This imperfect fusion resulted in the formation of the caste and religious systems of India. In Peninsular India to the south of the Vindhya mountains, the spread of Aryan civilization was

Figure 5 1 INDIA - GEOGRAPHICAL SETTING AND MAJOR RACIAL GROUPS



NEGRITOS PROTO-AUSTRALOIDS MONGOLOIDS
MEDITERRANEANS WESTERN BRACHYCEPHALS
NORDICS

slow. The races there, which are known as Dravidians, were powerful and civilized communities. Some Aryan states were founded, but even today the mass of people in southern India show little trace of Aryan blood and speak non-Aryan languages.

Further diversity in the Indian races was due to the massive inflow of foreign populations. On Alexander's invasion in 326 B.C., small communities of Greek or semi-Greek origin settled in the Panjab and in the North-West frontier province. The next extensive immigration was that of the Sakas, which began in the second century. Sakas included squat, narrow eyed Mongols, and also races like the Turks, who resemble the Aryans in physique. In the first century after Christ, another nomadic tribe from Central Asia, called the Yuch-Chi, descended upon the plains of northern India. They were akin to the Iranian Aryans and introduced a large element of foreign blood into the Indian population.

The next movement which introduced a large new class of recruits to the Indian population was that of Muslims, beginning with the inroads of the Arabs at the start of the eighth century and ending with the establishment of the Mogul dynasty in the sixteenth century. The Muslim invaders and settlers belonged to various Asiatic races, including a certain number of Mongols. But the majority were collected from nations or tribes of different appearance. They comprised Persians akin to the Indo-Aryans, Turks, Afghans of many varieties and sundry people of mixed descent. The admixture of Mongol blood having been overborne by other elements has left little trace in the features of the modern Indian Muslims.

In the seventeenth century, with the establishment of the East India Company, the massive European inflow started. Since then a considerable population of mixed Indo-Europeans originating from the union of Portuguese, English and other Europeans with Indian women has grown

up and forms an important element in the population of great cities, the Bombay konkon, and the settlements on the lower Himalayan ranges. Apart from the above described invasion and races, the Jews, Parsis, Zoroastrians, Armenians and certain other small communities maintain their isolation so strictly that they hardly affect the racial character of the general population.

5.1.2 Racial types

Racial types that occur in the present day populations of India show many extremely primitive strains and represent certain elements from the four major stocks of mankind : Negroid, Australoid, Mongoloid and Caucasoid. According to the morphological classification of Guha (1931-46), the different racial groups in India come from six main races and are as follows :

1. Negritos
2. The Proto-Australoid
3. (i) Palaeo-Mongoloids of (a) long headed and
   (b) broad headed types
   (ii) Tibeto-Mongoloids
4. The Mediterranean comprising :
   (i) Palaeo-Mediterranean
   (ii) Mediterranean
   (iii) Oriental type
5. The Western Brachycephals, consisting of .
   (i) Alpinoid
   (ii) Dinaric
   (iii) Armenoid
6. The Nordic

Of all these races, the Negritos are considered to be the first to come to India. In the mainland, the Negrito element is found among Kadars and Pulayans in the hills of Cochin and Travancore. It also exists among the Irulas and primitive tribes of Wynaad. Besides the Negritos, the aboriginal population of peninsular India contains another primitive element called Proto-Australoid. In stature, shape of head, broad flat nose, fleshy everted lips, the difference between the two is slight. The only characteristic that distinguishes Proto-Australoid from the Negrito type is the complete absence of frizzy or woolly hair. This element is found to be dominant in southern and central India (e.g. Munda, Santal, Bhil, Kurumbas, Gonds etc.). The other main physical substructure of the Indian races today is the Mongoloid type. This group contains three types of which the Palaeo-Mongoloids are of a primitive nature. Its first type characterized by scanty growth of hair and moderately developed epicanthic fold is found in the sub-Himalayan region and in the tribes of Assam. The second type is dominant in the hill tribes of Chittagong. The Tibeto-Mongoloid type occurs in Darjeeling and the areas around Sikkim and Bhutan.

The first three racial types, namely the Negritos, Proto-Australoid and the Mongoloid, with their sub-types, constitute the tribal populations living in India today.

In addition to these, the main constituent of India is that of the Aryan-speaking peoples or, more properly, the Caucasian or Mediterranean physical type of tall, relatively fair, long headed and aquiline featured people. It is the mixture of these types in varied proportions and in different localities which has produced the various physical strands in India. One of the most ancient elements of this race is the "Palaeo-Mediterranean type". The main "Mediterranean group" is dominant in northern India and in the upper section of the people of the

rest of the country. One of the diagnostic features of this group is the presence of large eyes which is not found to the same extent in other races. The "Oriental type" occurs mainly in the Panjab and Rajasthan. Morphologically it is close to the Mediterranean race except in the form of nose which is large and complex. The sub-types of the Western Brachycephals are found in Gujarat, Maharashtra, Bengal and Orissa.

Finally, there is mention of the Nordic race which in the Panjab, Rajputana and some upper castes of India is marked by increasing admixture with the older long headed races of the Mediterranean stock. It is mainly found in Western India and East Bengal.

5.1.3 Development of genetic studies in India

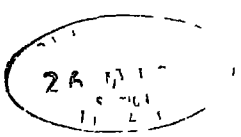
Investigations concerning the morphological measurements of the people of India were begun during the latter half of the last century. Comprehensive studies on the subject were undertaken by Risley (1915) and Guha (1931), both of whom studied a large number of individuals comprising several different groups. However, investigations concerning genetical traits have been few and far between in India as compared with some other countries of the world.

Preliminary studies carried out on Indians to study the genetic polymorphic system started in the early decades of the century with the ABO blood groups (Hirszfeld and Hirszfeld 1919). At present most of the data on the blood group systems are restricted to the ABO, Rhesus and MN systems. Knowledge about the other systems like Duffy, Kell, Lutheran etc. is still lacking. Several workers like Sen (1960), Vyas et al (1962), Parikh et al (1969) and Roberts et al (1974) have published reports about the frequency distribution of other systems, but such information is too

scanty to understand the genetic diversity exhibited by several endogamous groups of the Indian population. Later researches carried out by Sanghvi and his co-workers at Tata Cancer Research Institute, Bombay, revealed striking genetical differences between different endogamous groups and suggested that it may be of great interest to examine other such isolated groups who could contribute to the better understanding of polymorphism in the Indian sub-continent. The distribution of haemoglobin type frequency distribution is rather better documented (Chatterjea 1966). Kirk and Lai (1961) were the first to investigate the other polymorphic systems (e.g. Haptoglobin, Transferrin) in south Indian subjects. Subsequently, other inquiries regarding these polymorphic systems were looked into for other populations of the Indian sub-continent (Baxi and Hakim 1966, Baxi and Camoens 1969a, Chopra 1970, Blake et al 1971, Sunderland et al 1975).

The next step in solving some of the anthropological problems presented by the heterogeneous population was the use of red cell enzymes discovered in the last decade. The first enquiry into the isoenzyme systems of two endogamous groups of Madras was initiated by Ananthakrishnan and Kirk (1969). In subsequent years, information regarding the other enzyme systems was published about the populations of the states of Bengal, Maharashtra and Gujrat, Panjab, Madhya Pradesh, Assam (Das and Mukherjee 1970, Blake et al 1970a, Blake et al 1971, Undevia et al 1972, Goedde et al 1972, Singh et al 1974b and Roberts et al 1974). Similar researches were carried out among the neighbouring populations of Bhutan, Nepal and Ceylon (Mourant et al 1968, Tills et al 1970 and Roberts et al 1972). However, the studies conducted on the various populations of India are limited and information about the frequency distribution of various genetic parameters is scanty, not only among the tribal populations, but also within the urban communities.

Keeping in view the studies conducted on the populations of Northern India, plans were made to study the Panjabis in order to collect detailed information about the frequency distribution of polymorphic systems in the hope of adding to the knowledge of population genetics in India.



5.2 Analysis of Data

This section deals with the gene and phenotype frequencies of different genetical systems investigated in this survey. The results for the Panjabis are discussed in four different sections. They are : blood group antigens, serum proteins, red cell isoenzymes and haemoglobin. In the case of the blood group antigens, the results were discussed by comparing them with other state populations of Northern India, and where the data was limited (e.g. in the case of Duffy, Kell, Kidd etc), it was thought advisable to compare them with the rest of the populations of India. Studies conducted on the serum proteins and isoenzymes of Indian populations are limited, so the present results were compared with the available data of neighbouring as well as indigenous populations of different Indian states.

5.3 Blood Group Antigens

The results of different blood group antigens are given below :

(i) Table 5.1 shows the distribution of the ABO blood groups in the Panjabis of Northern India. Two levels of discrimination are shown, depending upon whether $-A_1$ serum was employed in testing the samples. Good agreement was found between the observed and expected phenotype frequencies, thus confirming the Hardy-Weinberg equilibrium.

(ii) The results for the MNSs phenotypes and gene frequencies are set out in Table 5.2. Two levels of discrimination are shown, depending upon whether -s serum was employed or not in testing the specimens. Good agreement was found between the observed and expected phenotypic values, confirming the assumption of Hardy-Weinberg equilibrium.

(iii) Table 5.3 presents the distribution of Rh types and gene complex frequencies in the Panjabi sample. All the specimens were tested with the following antisera : anti-D, -C, -E, -c, -e and-C^W. The statistical analysis demonstrated a significant difference between the observed and expected phenotype values, $X^2_6 = 12.688$ $0.05<P>0.02$.

(iv) Table 5.4a presents the observed number and frequency of K(+) individuals together with the calculated gene frequencies. The results of 86 subjects tested with anti-Kp^b serum are set out in Table 5.4b.

(v) Data on the distribution of the Duffy blood groups are shown in two parts as a result of some specimens being tested with anti-Fy^a and anti-Fy^b sera, whereas others were tested with anti-Fy^a serum only. The results set out in Table 5.5a show good agreement between the observed and expected phenotype values, thus confirming the assumption of Hardy-Weinberg law. Table 5.5b presents the observed number, their frequency and calculated gene frequencies.

(vi) The results of 163 Panjabi samples tested with anti-JK^a serum for the Kidd blood group system are given in Table 5.6. The table also reveals the Kidd groups, JK(a+) and JK(a-), along with their observed frequencies.

5.4 Discussion

5.4.1 ABO blood group system Table 5.7

Table 5.7 presents data of the ABO blood groups and respective gene frequencies in the state populations of Kashmir, Panjab, Uttar Pradesh (U.P.) and the cis-Himalayan region of Himachel Pradesh (H.P) and Uttar Pradesh. It is seen from the table that all the populations tested in the Panjab, such as Jat, Sikh, Muslim or Hindu, irrespective of the fact that they have been categorized as a religious or a linguistic group, exhibit, without any exception, a higher frequency of gene B than that of gene A. Such is also the case with the populations of Rajasthan, Kashmir and Uttar Pradesh. In the higher caste groups of Uttar Pradesh, there is a tendency for A to approach more closely the frequency of B, while in low caste groups like the Chamar, Bhoksa etc. (Majumdar and Kishen 1947) the differences are more prominent. The distribution of gene frequencies in the cis-Himalayan populations present an interesting picture. The populations bordering Himachal Pradesh, particularly in the regions of Chamba and Rampur Bushar, exhibit a higher incidence of gene B than gene A. A similar case is seen in certain populations bordering the Uttar Pradesh plains. In remote areas, however, there are isolated groups, such as the Kinner Kanets of the Chinni valley (Bhalla 1961) and the upper caste of Jaunsar Bawar (Banerjee and Kumar 1953) which reveal a higher frequency of the gene A (see Table 5.7).

The gene frequency distribution in the North Indian populations reveals a preponderance of gene r with a frequency of 40 to 60 percent. The frequency of q varies generally between 20 and 30 percent and that of p between 15 and 20 percent. The frequencies exhibited by the present sample of Panjabis fit into this range. The Panjabis are similar to

Panjabis soldiers sample of Hirszfeld and Hirszfeld (1919). Compared with the North Indian sample of Allen and Scott (1947), the Panjabi sample of Bhalla (1963) and Anand (1957), and the Sikh sample of Bird et al (1956), the Panjabis are found to exhibit a higher frequency of gene B (35%) and a slightly lower frequency of gene O (47%). It appears that the high frequency of gene B is a characteristic feature of the North Indian populations, but the difference could be due to small sample size. Further, no difference is seen when the Panjabis, tested with anti-A_1 serum, are compared with the Panjabi sample of Papiha et al (1972) and Seth (1968), and the Sikh sample of Bird et al (1956).

Studies conducted with anti-A_1 serum are limited to Northern India. However, the A_2 gene is low among the Panjabis (0.0062), but is comparable to the populations of North India tested so far (Papiha et al 1972, Bhattacharjee 1966 and Seth 1968).

5.4.2 MNSs blood group system Table 5.8

In the Indian sub-continent the incidence of a higher frequency of the M* gene and a relatively low frequency of the N gene "fits well into the Mediterranean and European picture" (Mourant 1954). The highest frequency of gene M (89.00%) was reported among the Riangs of Tripura (Kumar and Sastry 1961), and the lowest (38.36%) among Mela-pantarums of Travancore, South India (Buchi 1955). In Northern India, Bhalla (1963) examined three upper castes of Panjabi Hindus and reported

* For the sake of comparison and convenience the comparable results of the MNS and MNSs systems obtained by using anti-M, -N, -S and -s were converted into terms of M and N gene by adding the values of MS and Ms to get M and NS and Ns to get N.

M gene frequencies varying from 53.58% in the Arora to 57.37% in the Brahmin. In Kashmir the Pandits and Muslims studied by Bhattacharjee (1966) showed the frequency of the M gene to be 57%. The Sikhs tested by Bird et al (1956) showed M frequency of 64% and similar figures were given by Papiha et al (1972) in his Panjabi sample.

In the present survey the Panjabis show a higher frequency of the M gene (65% and 67%). Mourant (1954) described a lower value of 58% in the province of Panjab, West Pakistan. Similar low values have been reported in Panjabi Hindus (55%) by Bhalla (1963). A plausible explanation for this difference could be that Mourant's sample consisted of Panjabi Muslims and Bhalla's only the Panjabi Hindus, whereas the present sample consists of predominantly Panjabi Sikhs and a few Hindus. However, it appears that the M gene in the north averages around 62% and the present value for the Panjabis is close to those reported for other North Indian populations.

From the comparative data summarized in Table 5.8b, it appears that, like Europe, the gene S tends to accompany gene M rather than gene N. The Panjabis show these tendencies and confirm the general Indian pattern.

5.4.3 Rh blood group system Table 5.9

In general, the findings for the Rh blood groups reported here show resemblances with the populations of Northern India. The frequency of Rh negative type, rr, for the present sample is 7.02%. Similarly, the Rh negative incidence has been reported as 6.66% in Panjabis (Talwar 1962), 6.12% in Panjabi Hindus (Bhalla 1963) and 6.67% in the U.P. Brahmins (Shivaraman et al 1971). However, the present value is higher than the figure of 3.8% for the Sikh (Bird et al 1956)

and 4.4% for the Panjabis (Papiha et al 1972), but is within the range of 6 to 10% reported for the North Indian populations. The Panjabis also show the presence of rare phenotypes R_2R_z, r'r and R_or. Papiha et al (1972) has, however, reported the absence of type R_2R_z and r'r in the Panjabis.

The gene frequencies set out in Table 5 9 suggest that the gene r has frequencies in the range between 0.2377 and 0.2833, except in the Sikh, where the value is low (0.176). The present value of 0.2457 is within this range. The gene R_1 varies from 0.5279 in Thakkur to 0.639 in Sikh and the frequency of 0.5985 here is close to the value of 0.610 for the Panjabis. The frequency of gene R_2, 0.1095, is again in line with the values of 0.118 and 0.097 reported for the Sikh and the Panjabis. Also, the frequencies of the rare genes are comparable with the populations of Northern India.

5.4.4 Kell blood group system Table 5.10

Table 5.10 shows the distribution of the Kell blood group system in selected Indian populations. As can be seen from the table, the frequency of the gene K is lower in India than in Europe. Again not many state populations are available for comparison, but it is clear from the available data that the Kell positive phenotype frequency approximates 1% in the Indian populations. An exceptionally high frequency of the Kell positive phenotype (about 12%) was reported in an upper caste of Bengal by Sen (1960). This aberrant case could be attributed either to sampling or technique. The low frequency of the gene K found in this survey (0.0140) is similar to that from other samples from the Panjab (Papiha et al 1972) and Madhya Pradesh, Central India (Roberts et al 1974).

No tests appear ever to have been carried out on specimens from Indians with either of the Penny antisera, Kp^a and Kp^b. The Panjabis tested with anti-Kp^b serum only gave the following frequencies · kp^a = 0.3235 and Kp^b = 0.6765. These values seem to be much higher than in European populations, and this may be due to a small sample size

5.4.5 Duffy blood group system Table 5.11

Generally speaking, the Asiatic populations are characterized by high Fy^a gene frequencies as compared with European populations (Mourant 1954). Cutbush and Mollison (1950) tested 55 Asiatic Indians and found a Fy^a phenotype frequency of 93%, a figure which may be inflated by small sample size. But there is no doubt that a high frequency of the Fy^a gene is present in certain Indian populations. Lehman and Cutbush (1952) reported the highest frequency of the Fy^a gene in the Irulas (74%) of South India. Similar high values were found in the Todas (71%) of the Nilgiri hills. The lowest frequency reported is 32% found in a Jalaiguri caste of West Bengal (Das et al 1967). As shown in Table 5.11a, the Western and Central populations of India show the Fy^a gene frequency varying between 35% and 53% (Vyas et al 1958, Parikh et al 1969, Roberts et al 1974). Thus it appears that the incidence of the Fy^a antigen varies in different parts of India and the present figure of 57% for the Panjabis is well within the range of variation of the earlier samples.

Table 5.11b shows the distribution of the Duffy blood groups tested with anti-Fy^a and anti-Fy^b serum. To the author's knowledge no other population from North India, apart from the present Panjabi sample, has been tested for the complete Duffy system. In the present survey, a single subject showed the presence of the phenotype Fy(a-b-).

A similar finding was reported in the Muslims of Madhya Pradesh (Roberts et al 1974). It seems that the presence of the Fy gene is common in these areas but it is as yet premature to indicate the general picture for the Fy gene in India. Furthermore, the Panjabis exhibit Duffy gene frequencies consistent with those found in the . Hindus and Muslims of Madhya Pradesh, Central India (Roberts et al 1974).

5.4.6 Kidd blood group system

Studies conducted with either of the Kidd anti-sera, anti-JK^a or JK^b, are few in India. Among the state populations available for comparison are the Hindus and Muslims of Madhya Pradesh (Roberts et al 1974) and the Muslims of Bombay (Hakim et al 1973). The Panjabis appear to have similar frequencies to those found in the Muslim sample of Madhya Pradesh and Bombay. The Panjabis, as well as the other Indian populations, are found to be similar to European populations.

5.5 Serum Proteins

The results of the two serum systems employed in this study are as follows .

(i) Table 5.12 shows the distribution of haptoglobin groups and respective gene frequencies in the Punjabis of Northern India. No significant difference between the observed distribution of phenotypes and the expectation determined according to the Hardy-Weinberg law was found. No Hp 2-1 (modified) or other rarer phenotypes were detected. Seven subjects with phenotype Hp 0-0 or anhaptoglobinaemia were detected. As the phenotype Hp 0-0 is not considered to be inherited (Barnicot et al 1960), it was excluded from the gene frequency calculations. The allele frequencies exhibited by the Punjabis are as follows :
$Hp^1 = 0.2204$, $Hp^2 = 0.7796$.

(ii) The distribution of the Tf groups and their respective gene frequencies are shown in Table 5.13. A single subject showed a CB phenotype; all others were CC, no D variant being found. It was not possible to sub-type the B variant, but when run with a CB control on the starch-gel, the present CB variant exhibited similar mobility.

5.6 Discussion

5.6.1 Haptoglobin Table 5.14

Previous results of haptoglobin typing in the Indian sub-continent are summarized in Table 5.14. World values for the Hp^1 gene range from 0.07 to 0.89 (Kirk 1968). The Indian sub-continent is an area of low Hp^1 values. In the South, the Nadar (0.037), the Irulas (0.070) and the Tamils (0.090) show the lowest frequencies in the world, whereas the Todas are exceptional in having the highest known frequency of 0.37 (Kirk and Lai 1961). The Central and Eastern Indian populations show an increased Hp^1 frequency (see Table 5.14). Baxi and Camoens (1969a) value of Hp^1 0.1165 for the Marathi is low and to the North West there is a slight increase in the Hp^1 frequency, the value rising to 0.271 among the Jats (Sunderland et al 1975). Comparison with neighbouring areas shows a similar increase in the value, rising to 0.24 among the Pathans (Kirk and Lai 1961) and 0.276 among the Peshawarans of West Pakistan (Walter et al 1966). The frequencies of Hp^1 plotted against latitude confirm the suggestion of a south to north gradient of increasing frequency.

In the present survey the Hp^1 frequency of 0.2204 for the Panjabis is comparable with values of 0.2094 and 0.237 for the Panjabis obtained by Tiwari (1961) and Papiha (1973). This value is also in agreement with the range of variation (0.037 - 0.37) reported for other Indian populations hitherto investigated (Kirk and Lai 1961, Blake et al 1971, Seth et al 1971 and Singh et al 1974a). Apart from the usual haptoglobin patterns, the presence of phenotype Hp 0-0 in the Panjabis is consistent with the report of Tiwari (1961). Blake et al (1971) also reported the Hp 0-0 type in the scheduled caste individual from

North India, but Sunderland et al (1975) did not find it in any of the four castes of the Panjabis. The present appreciable frequency of Hp 0-0 in the Panjabis could be attributed to parasitaemia and other diseases in which haemolytic episodes occur.

5.6.2 Transferrin Table 5.15

Table 5.15 shows the distribution of transferrin variants in various populations of the Indian sub-continent. Variants of transferrin in some parts of India are rare. The existence of transferrin D has been reported in Madras (Ananthakrishnan and Kirk 1969), in Assam (Goedde et al 1972), in West Bengal (Walter et al 1972, Mukherjee et al 1974, and Das et al 1974) in a miscellaneous sample of Indians from different states (Baxi and Camoens 1969b). In addition, variants occur at high frequency in the tribal population of the Oraons and in the Veddas of Ceylon (Kirk and Lai 1961). Thus these results indicate that the distribution of transferrin D is confined to the South and East of India which separates the population of Dravidian and Austro-Asiatic linguistic affinity from the more northerly Aryan speakers in which the B variant is found to be more common (see Table 5.15).

The detection of a CB variant among the Panjabis in this survey has increased the number of subjects known with B variants. Goedde et al (1972) first reported a transferrin B variant in the Khasis of Assam. A similar variant was found in Delhi Muslims (Papiha and Wastell 1974), in the Jats (Singh et al 1974a), in Khatri (Sunderland et al 1975) and Pathans (Kirk and Lai 1961). This illustrates the fact that the CB variant is relatively frequent in the Northern, North-Western and North-Eastern populations of India compared with the populations of the South. However, much research is called for in order to assess the exact distribution of different transferrin variants in the Indian sub-continent.

5.7 Red Cell Isoenzymes

The results of different isoenzyme systems are given below .

(i) Table 5.16 reveals the distribution of acid phosphatase groups and respective allele frequencies in the Panjabi sample. Close agreement was found between the observed and expected phenotype values confirming the assumption of Hardy-Weinberg equilibrium, and it is a random mating population. The allele frequencies exhibited by the Panjabis are $P^a = 0.3217$, $P^b = 0.6687$ and $P^c = 0.0096$.

(ii) The distribution of PGM_1 groups and calculated gene frequencies are shown in Table 5.17. No significant difference between the observed distribution of phenotypes and the expectation according to the Hardy-Weinberg law was found. No rarer phenotype was found. The frequency of PGM_1^1 observed is 0.7044 and that of PGM_1^2 is 0.2956.

(iii) Table 5.18 shows the distribution of the adenylate kinase groups and respective gene frequencies in the Panjabis. There was close agreement between the observed and expected phenotypic values. No rarer phenotype was detected. The observed frequencies of gene AK^1 and AK^2 for the Panjabis are as follows : $AK^1 = 0.9177$ and $AK^2 = 0.0823$.

(iv) Table 5.19 gives the distribution of 6-PGD phenotypes and respective gene frequencies. No difference between the observed and expected phenotype values was found, thus confirming the Hardy-Weinberg equilibrium. The gene frequencies of 6-PGD of the Panjabis are : $PGD^A = 0.9772$ and $PGD^C = 0.0228$.

(v) The percentage deficiency of G-6-PD detected in the Panjabi males is set out in Table 5.20. No other variant, apart from noting whether the subject is deficient or not, was possible as the specimens were old.

(vi) All the 415 specimens analysed for lactate dehydrogenase (LDH) exhibited the normal phenotype and are shown in Table 5.21.

(vi) Also the 415 specimens tested for malate dehydrogenase (MDH) were found to have the type MDH-1 (Table 5.21).

5.8 Discussion

5.8.1 Acid phosphatase Table 5.22

Three alleles exist in many populations to control the phenotypic expression of red cell acid phosphatase, and two of these, P^a and P^b, are universally distributed. The third, P^c, appears to be absent or of very low frequency in black Africans and New Guineans (Hopkinson 1968) and Japanese (Shinoda 1969). Such polymorphic variation in India is controlled by three alleles, P^a, P^b and P^c, the last being very low as compared with the commonly occurring P^a and P^b genes. The highest P^c gene for an Indian sample is 0.047 for the Khasis of Assam (Goedde et al 1972). Hopkinson (1968) quotes unpublished results of 222 Indians sampled in England giving a P^c frequency of 0.02. In indigenous samples, most of the states show frequencies of less than 1%, and the P^c gene is absent in the Brahmins and Naickers of Madras (Ananthakrishnan and Kirk 1969), the Marathi of Bombay (Blake et al 1970a) and the Parsis of Bombay (Undevia et al 1972). The Bhutanese sample of Mourant et al (1968) also showed an absence of the P^c allele. In the present sample, the P^c allele occurred in eight heterozygotes, seven CB and one CA. This low frequency of the P^c gene (0.0096) is clearly in agreement with other Indian populations tested. However, the Indian populations also vary appreciably in the frequencies of the P^a and P^b genes. P^a ranges from 0.328 in a Muslim sample of Roberts et al (1974) and the North Indian sample of Blake et al (1971), down to 0.206 in Naickers of Madras and 0.171 in Bhutan (Mourant et al 1968). The present frequency of 0.3217 for the Panjabis is within this range and identical with the value of 0.318 for the Panjabis reported by Papiha et al (1972).

The highest P^b frequencies of 0.829 and 0.794 occur in Bhutan and among the Naickers of Madras ranging down to 0.5775 amongst the Arora of North India, and the present sample at 0.6687. Overall, highly significant heterogeneity appears amongst the samples in acid phosphatase phenotype frequency.

5.8.2 Phosphoglucomutase locus 1 Table 5.23

Data on the distribution of phosphoglucomutase locus 1 in the various populations of the Indian sub-continent are summarised in Table 5.23 . PGM_1^1 and PGM_1^2 have a universal distribution though their frequency varies from one population to another (Hopkinson 1968). In South India, Anathakrishnan (1972) noted PGM_1^1 values ranging from 0.721 to 0.796, in Marathi and Gujarati the corresponding values found by Blake et al (1970) were 0.6491 and 0.6909 and for the Bengalis, Das and Mukherjee (1970) found a PGM_1^1 frequency of 0.6989. In the North Indian sample of Blake et al (1971), PGM_1^1 ranged from 0.6341 among the Rajput to 0.7937 among the Vaish. Thus the frequency of the PGM_1^1 gene among the Panjabis (0.7044) fits into the range of 0.634 to 0.804 (see Table 5.23) reported for the Indian populations tested to date. Again the frequency of the PGM_1^1 allele is consistent with those found by Singh et al (1974b) in a combined sample of Panjabis and in Das et al's sample of Bengalis. A statistical comparison with neighbouring countries like Ceylon and Bhutan shows that the PGM_1 frequencies are somewhat similar. As shown in Table 5.23, the PGM_1^2 ranges from 0.196 to 0.3537, and the present value of 0.2956 for the Panjabis is well within the range. Overall, the PGM_1 frequencies appear to be relatively uniform over the sub-continent, no markedclines being apparent.

5.8.3 Adenylate kinase Table 5.24

Table 5.24 presents data on the distribution of adenylate kinase and respective gene frequencies in various populations of the Indian sub-continent.

Among Europeans, the AK^2 frequency approximates 0.05, but lower frequencies are found among U.S. Negroes and black Africans (Rapley et al 1967). The AK^2 gene is shown to be absent in New Guinea and Australia (Sinnett et al 1970, Kirk et al 1971) and has zero or very low frequencies in Mongoloids (Shih and Hsia 1969). By contrast, the frequencies in Indian populations are high. The highest frequencies of AK^2 reported so far are amongst Indians (0.0985) and Pakistanis (0.1296) sampled in England (Rapley et al (1967). Similar frequencies in the Brahmin (0.081) and Naicker (0.099) of South India were reported by Ananthakrishnan and Kirk (1969). The frequency of AK^2 in Bengal is slightly lower at 0.0867 (Das and Mukherjee 1970) and the Marathi and Gujarati have frequencies of 0.0909 and 0.1115, respectively (Blake et al 1970a). Among the North Indian upper caste groups, the range is from 0.0563 for the Arora and Brahmin to 0.1270 for the Vaish and 0.1406 for a small series of miscellaneous Hindu (Blake et al 1971). Exceptionally low values of AK^2 have been found to occur in the Reddiar (0.034) of South India (Ananthakrishnan 1972). The Panjabis show a striking similarity in the North Indian sample of Camoens (1971) and the Panjabi samples of Papiha et al (1972) and of Singh et al (1974b).

5.8.4 6-Phosphogluconate dehydrogenase Table 5.25

In the 6-PGD system, two alleles, PGD^A and PGD^C, have been shown to occur in all populations (Carter et al 1968). In Europeans

the PGD^C allele frequency approximates 0.02 and similar values have been found in the Brahmin and Naicker of South India (Ananthakrishnan and Kirk 1969), in North Indians (Blake et al 1971), among the Gujarati and Marathi of Bombay (Blake et al 1970a) and in Bengalis (Das and Mukherjee 1970). The highest PGD^C frequency reported so far in India is 0.048 among the Nadar of Madras (Ananthakrishnan 1972) and 0.047 among the Khasi of Assam (Goedde et al (1972). Overall, the frequency of the PGD^C gene in the Indian populations ranges from 0.006 to 0.048. The 0.0228 value for the Panjabis fits into this range and is comparable with European figures. Using the Chi-squared test, the Panjabis are found to exhibit 6-PGD phenotypes and genes consistent with the proportions found in Panjabis by Papiha et al (1972) and in Marathi and Gujarati of Bombay by Blake et al (1970a).

5.8.5 Glucose-6-phosphate dehydrogenase Table 5.26

Table 5.26 shows the incidence of G-6-PD deficiency in selected populations of the Indian sub-continent. The association of G-6-PD with malaria and its distribution in the Indian sub-continent has already been reviewed by Chatterjea (1966). It is seen from the table that the percentage of G-6-PD deficiency in Indian populations varies from one region to another. A high percentage is found among the tribal population (8.5%) of Andhra Pradesh (Meera Khan 1964) and among the Parsis (19%) of Bombay (Baxi et al 1963). G-6-PD is known to be more frequent among the North Indians (0.4 - 13.5%) than among South Indians of non-Tribal origin (0 - 3.3%).

In the present survey the incidence of G-6-PD deficiency is found to be 4.09% among the Panjabis*. This figure is higher than that reported for the North Indians (2.79%) by Saha and Banerjee (1971).

*A high frequency of G-6-PD (4.09%) could be attributed to the age of the specimens.

It is seen to be lower compared with a 2000 sample of Panjabis (6.9%) but is close to the Panjabi sample (3.42%) of Singh et al (1974b). Such difference among the above mentioned samples could be due to different techniques, different ethnicity, the effect of environment and nutritional factors.

5.8.6 Lactate dehydrogenase Table 5.27

The distribution of LDH variants detected in various Indian populations is shown in Table 5.27. The Eastern and Southern populations have a relatively high frequency of a genetic variant designated as LDH Cal-1, ranging in frequency from 1 to 4% (Das and Mukherjee 1970, Anantha Krishnan et al 1970). Among the North Indian sample, the frequency is shown to be 1% (Blake et al 1971). In the present investigation no LDH variant was found among the Panjabis and this result is in agreement with that of the Panjabis reported by Papiha et al (1972).

5.8.7 Malate dehydrogenase

MDH variants are rare in India. The absence of MDH variants in the Panjabis is consistent with the results of previous studies on the Indian populations (Blake et al 1970a, Blake et al 1971 and Undevia et al 1972).

5.9 Discussion

5.9.1 Haemoglobin Table 5.28

The establishment of the fact that all the abnormal haemoglobins were of type Hb-D has been discussed earlier in Chapter IV.

Haemoglobin-D has been shown to occur very frequently among the Sikh and Gujarati in India (Bird and Lehmann 1956, Ghai et al 1961). In the present sample, 9 subjects exhibited the heterozygous form Hb-AD and the frequency of 0.0132 here is in agreement with the value of 0.0108 given for the Sikhs by Bird et al (1956).

5.10 Conclusion

Serologically, it has been observed that the Panjabis show the greatest resemblance to the state populations of Kashmir, Rajasthan and Uttar Pradesh in Northern India. Regarding the ABO gene frequencies, the Panjabis exhibit a high B frequency which seems to be a characteristic feature of North Indian populations. The frequency of the A_2 gene is low. A higher frequency of the gene M and lower frequency of the gene N fit well within the Mediterranean and European picture. The Panjabis, like other Indian and European populations, confirm the tendencies of showing that the gene S accompanies gene M rather than gene N. The general pattern of the Rh frequency distributions also shows close resemblance with the northern populations. Among other blood group antigens, the data on the frequency distribution of the Penny, Duffy and Kidd systems has been presented for the first time. The Panjabis appear to have a high frequency of the Kp^a gene. Further study of this may prove to be of some importance in differentiating populations in India. Of great interest is the finding of phenotype Fy(a-b-). Compared with the only other population tested, in Madhya-Pradesh, it seems that the Fy gene is present in northern and central populations of India. Since not many populations from India have been tested for the complete Duffy system, it would be premature to attempt to predict the general picture of the Fy gene in India. Concerning the Kell and Kidd systems, the frequency distributions in the Panjabis are found to be the same as those observed in other Indian populations.

Of the two serum protein systems investigated, both were found to be polymorphic. No major difference was revealed by the present study between the Panjabi population and those in other parts of India for the haptoglobin system where comparable data are available. The frequency

of Hp^2 is high for the Panjabis, but similar to that of North Indians, Bengalis, Marathis and Gujaratis. The presence of the transferrin B variant is common in Northern India and the present study is in agreement with the previous findings. The occurrence of a single individual with a CB variant is of interest, therefore, and although the frequency of this variant is low, it could prove to be significant in distinguishing populations in different places of a vast country like India. Further research is urgently needed to know the exact distribution of transferrin B variants in India.

Among the seven enzyme systems studied, five were found to be polymorphic in the Panjabis. They were : acid phosphatase, phosphoglucomutase, adenylate kinase, 6-phosphogluconate dehydrogenase and glucose-6-phosphate dehydrogenase. In the Indian sub-continent most of the populations have been known to exhibit low frequencies of the gene P^c. The present low value of the P^c allele in the Panjabis has been confirmed by comparing with the earlier studies. P^b is the commonest allele, but again there is no significant difference between North and South. The same is true for Locus 1 in the PGM system, no clinal trends being apparent. Similarly, the PGD^C allele in the 6-phosphogluconate dehydrogenase system showed no obvious difference between populations in various parts of the country. The adenylate kinase system shows that Indian populations have very high values for the AK^2 gene. The Panjabis also display AK allele and phenotype frequencies similar to those reported in Bengalis, Marathis, Gujaratis and some South Indians.

Two enzyme systems, malate dehydrogenase and lactate dehydrogenase, were found to be invariant in the present survey. Malate dehydrogenase was studied for the first time in the Panjabis.

The invariant results of this system are consistent with the previous studies in India. Further study of the populations of Northern India may provide useful information as such variants distinguish certain populations from other peoples of the world where the system is found to occur as a polymorphism. Indian populations appear to have the highest frequency of LDH variants (1 - 4%) in the world. Similarly, an appreciable frequency has been reported in North Indian populations. The absence of LDH variants in the present study requires more detailed study to be conducted in other state populations of Northern India.

Finally, it appears that the frequency distributions of enzyme variants in Panjabis are similar to those in other states in peninsular India. The samples drawn from the Aryan speaking peoples of the North do not differ from those of the Dravidian speaking groups of Central and Southern India. Thus the ethnic distinction does not coincide with the isoenzyme genetic differences. For example, the Panjabis of the North and the South Indians share high frequencies of the AK^2 gene.

CHAPTER VI

GENETIC POLYMORPHISMS IN THE NEPALESE

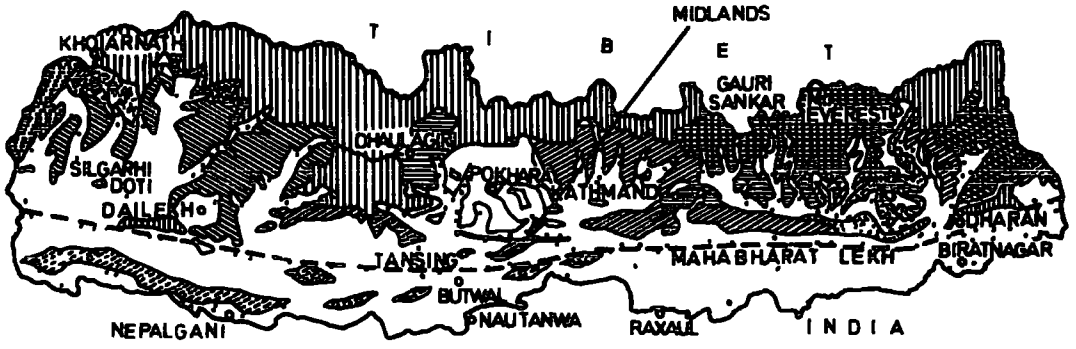
6.1 Introduction

6.1.1 Geography - history and people

Nepal, roughly rectangular in shape, comprises an area of about 55,000 square miles and is situated between longitude 80 and 88E and latitude 27 and 30N. It is sandwiched between the great Gange plains of India to the south and the Tibetan plateau to the north. The country shows different topographical units which are clearly distinguishable from each other. The heart of the country called 'the Midlands' is protected in the south by the Mahabarat Lekh and in the north by the mountains of the High Himalayas (Figure 6.1).

According to Regmi (1965), the valley of Nepal had received all kinds of settlers by the advent of 14th century A.D. The majority of these people came from South and South-east Asia. Lack of archaeological and historical evidence makes it difficult to determine who were the early settlers and what time they came to Nepal. What is certain is that the ancient Nepalese tribes settled in the Midlands and constituted the original Nepalese population. In the 12th century, the Khasa tribes migrated to the south and west and around 1500 B.C. the Aryan invasion introduced a new stock into the Nepal Midlands. In the early middle ages, two Tibetan tribes - the Sherpas and the Thakals - migrated to Nepal and this complex situation gave rise to a composite population. No one of these groups could preserve its ethnic isolation and idiosyncracies in toto.

Figure 6 1 NEPAL - GEOGRAPHICAL SETTING AND ETHNIC GROUPS



Tibeto-Nepalese races

Ancient Nepalese groups

- Newars
- Thamangs
- Gurungs
- Mangars
- Sunwars
- Rais
- Limbus
- Buras, Rukhas
- Tharus

Tibetan groups

- Bhotiyas
- Sherpas
- Thakals

Indo-Nepalese races

Nepalese groups

- Brahmans, Kshatriyas
- Thakurs

Today, as a result of such complex situations, the ethnic map of the country shows three major races with many ethnic groups (Hagen 1961). These races are as follows :

(i) Ancient-Nepalese races

(ii) Indo-Nepalese races

(iii) Tibeto-Nepalese races

Of all these races, the ancient Nepalese group consists of different ethnic groups like Newars, Thamangs, Gurungs, Rais, Limbus, Tharus, Sunwars, Rukhas and Buras. The Newars are small and graceful in build, their features well cut, the complexion slightly yellowish or sallow, and the nose small. Many of the Indo-Nepalese elements have been absorbed by the Newars. The Thamangs are found in the east, while the Gurungs, who have pronounced Mongol features, are concentrated in the west of Nepal. They are short, with slightly yellowish complexions. In the east, the other two important groups are the Rais and the Limbus. The Rais have the root of the nose especially low and the Limbus are characterized by their broad cheekbones. It is believed that they were the first people to settle in the valley of Nepal. The Sunwars, Rukhas and Buras form minority groups and are scattered throughout the country.

Among the Indo-Nepalese races the important groups are the Brahmins, Khastriyas, Khas and Thakurs. The first two groups, belonging to the sacredotal and warrior classes, have maintained their social status and introduced the Indian caste system into Nepal. At present they form a considerable part of the Midland population. The Khas group includes the people who are the result of intermarriage between the Brahmins or Khastriyas with the members of the Newar, Thamang, Gurung, Rai and Limbu tribes. The Thakurs, who live in central Nepal, resemble the Khastriyas with regard to the physical characters.

Sherpa, Thakals and Bhotiyas represent the Tibetan ethnic groups in Nepal. The Sherpas inhabit different regions in the high mountains of east Nepal. The Thakals resemble the Sherpas with regard to physical features. The Bhotiyas, who are considered to be pure Tibetans, migrated to Nepal very recently.

Finally, it is worth mentioning that the aforementioned groups offer a great opportunity for the geneticists and anthropologists to study the genetic diversity in the country. With the exception of two groups - the Gurkha and the Newar - no other tribe has been screened for different genetic systems. Nijenhuis and Runia's (1963) work on the Nepalese is limited to the blood groups only. The present investigation aims at presenting a detailed picture of the distribution of blood groups, serum proteins and red cell enzymes in the Nepalese.

6.2 Blood Group Antigens

The results of the Nepalese tested for different blood group antigens are as follows :

(i) Table 6.1 shows the results for the ABO system, tested with anti-A, anti-B, anti-A+B and anti-A_1. Good agreement was found between the observed and expected phenotype numbers calculated on the basis of Hardy-Weinberg equilibrium. A_2 and A_2B phenotypes were found to be absent. The gene frequencies exhibited by the sample are : $p_1 = 0.3429$, $q = 0.1371$ and $r = 0.5200$.

(ii) The results of the MNSs system are set out in Table 6.2. No significant deviation from the expected numbers calculated on the basis of Hardy-Weinberg equilibrium was observed. The gene complex frequencies are : MS = 0.1347, Ms = 0.6058, NS = 0.0280 and Ns = 0.2315.

(iii) Table 6.3 presents the Rh results of 211 Nepalese, tested with anti-D, -C, -c, -E and -e. Again, the observed numbers were found to be very close to the expected ones, showing that the population is in genetic equilibrium. The gene complex frequencies are : $R_z = 0.0186$, $R_1 = 0.7492$, $R_2 = 0.1346$, $R_2^U = 0.0016$, $r'' = 0.0016$, $R_o = 0.0399$ and $r = 0.0545$.

(iv) Table 6.4 reveals the results from the Kell system, tested with anti-K and anti-k sera. The gene frequencies are K = 0.0189 and k = 0.9811. A sample of 44 Nepalese, tested with anti-Kp^a and anti-Kp^b sera, exhibited the following frequencies : $Kp^a = 0.0227$ and $Kp^b = 0.9773$. Good agreement was found between the observed and expected phenotypic values in both systems.

(v) Table 6.5 shows the Nepalese samples tested with anti-Di^a serum only. The calculated gene frequencies are : Di^a = 0.0217 and Di^b = 0.9783.

6.3 Discussion

6.3.1 ABO blood group system Table 6.6

As illustrated in Table 6.6, the Nepalese show gene A_1 to be more frequent than gene B. Similar high frequencies of gene A_1 have been found in the Gurkha, the Nepalese and the Newar (Bird et al 1957, Nijenhuis and Runia 1963 and Bhasin 1970). On the other hand, the frequency of gene B in the Nepalese is somewhat lower than in most Indian peoples. It is also interesting to observe that the frequency of gene B is higher in the Tibetans, Burmese and some Mongoloids of India, whereas it is lower in the Bhutanese and the Baltis (see Table 6.6). However, the frequency of gene A_1, 0.3429, reported here, is higher than the Nepalese samples and the Mongoloids of South and South-East Asia.

The A_2 gene has been reported among the Gurkha and the Newar of Nepal In this study, however, the gene A_2 was not found. Again, the lack of A_2 is not surprising as this gene is either rare or absent in the Asiatic Mongoloids.

After statistical analysis, the Nepalese are found to be similar to the Newar and Baltis (Clegg et al 1961), with regard to phenotypes. They differ from the Gurkha sample of Bird et al, as $X^2_3 = 12.318$ $0.01<P>0.001$. The Nepalese also differ from the Tibetans, Bhutanese, Burmese, Chinese and some Mongoloids of India.

Nepalese v Tibetans	(Bhattacharjee 1968a)	$X^2_3 = 56.110$	$P<0.001$
Nepalese v Bhutanese	(Mourant et al 1968)	$X^2_3 = 8.524$	$0.05<P>0.02$
Nepalese v Burmese	(Ikin 1958)	$X^2_3 = 18.173$	$P<0.001$
Nepalese v Ladakhi	(Bhattacharjee 1968b)	$X^2_3 = 19.671$	$P<0.001$
Nepalese v Chinese	(Layrisse et al 1956)	$X^2_3 = 14.477$	$P<0.001$

These differences are accounted for by the generally higher incidence of A and the lower frequency of phenotype B, compared with the aforementioned populations.

6.3.2 MNSs blood group system Table 6.7

As shown in Table 6.7, the Nepalese have a higher frequency of gene M as found in India, Bhutan, Tibet, Baltistan and Burma. On the contrary, they are dissimilar from the Chinese who possess a low value of M. With regard to the MNSs system as a whole, the incidence of the gene complex MS, 0.1347, is less frequent than most of the Indian samples but is closer to the Himalayan Mongoloid peoples - the Gurkha (0.1578) (Bird et al 1957), the Bhutanese (0.1406) (Mourant et al 1968) and the Ladakhis (0.1355) (Bhattacharjee 1968a). The incidence of MS is also similar to the Burmese (0.1163) but is very low in the Tibetans (0.0888). The Ms frequency, 0.6058, is decidedly higher than in the Caucasoid peoples in India where MS is preponderant, and similar to that observed in the Gurkha (0.5447), the Bhutanese (0.5449), the Tibetans (0.5813), the Burmese (0.6367) and the Lepechas (0.6324). Overall, the high level of gene s, 0.8373, exhibited by the Nepalese, may be due to the contribution of neighbouring peoples of Mongoloid stock.

As all the samples included in the table were tested with anti-M, anti-N and anti-S, statistical comparison could not be performed.

6.3.3 Rh blood group system Table 6.8

With respect to the Rh frequencies, the Nepalese appear to have R_1 as the most common gene complex followed by R_2, r, R_o, R_z and r". The frequency of R_1, 0.7492, which is considerably higher than in the Newar (0.590); Bhutanese of Luana (0.5493); Tibetans (0.505); is near the

frequency in the Gurkha (0.7394), Chinese (0.760), Lepcha (0.7022) and Burmese (0.6666). Similarly, the frequency of the gene complex R_2, 0.1346, is lower than in Bhutanese (0.2890), but is comparable with the figures of 0.1794 for the Gurkha, 0.1606 for the Burmese and 0.195 given for the Chinese by Simmons et al (1950). The incidence of r is reported to be either rare or absent in most of the Asian Mongoloids. However, the value of 0.0545 here is lower than in the Nepalese samples and is in agreement with other Mongoloid populations (see Table 6.8). Furthermore, like the Bhutanese of Thimbu and the Tibetans, the frequency of Ro is lower in the Nepalese.

A direct comparison shows that the Nepalese are similar to the Gurkha sample of Bird et al (1957), whereas they differ from the Newar sample of Bhasin (1970), as $X^2_4 = 14.498$ $0.01<P>0.001$. The Nepalese also display significant difference from the Bhutanese, $X^2_3 = 21.474$ $P<0.001$ (Mourant et al 1968), the Tibetans, $X^2_3 = 46.195$ $P<0.001$ (Nijenhuis and Runia 1963) and the Chinese, $X^2_3 = 12.445$ $0.01<P>0.001$ (Simmons et al 1950). These differences are due to a higher frequency of R_1R_o/R_1r, but in certain cases it is the lower incidence of R_2R_2 and the higher frequency of R_1R_1 that contributes to the large Chi-squared value. The present sample also differs from the Burmese who exhibit a higher frequency of type rr, $X^2_3 = 6.591$ $0.01<P>0.05$ (Ikin et al 1969).

6.3.4 Other blood group systems Table 6.9 - 6.10

Among the Asiatic Mongoloids, the K gene is found to be either rare or absent. It appears to be fairly well represented in Nepal, Bhutan and Burma (see Table 6.9). However, the present frequency of 0.0189 is lower than in the Newar and other Mongoloid samples, but is comparable with the figure of 0.0222 given for the Tibetans by Nijenhuis

and Runia (1963). The frequency of Kp^a, 0.0227, exhibited by the Nepalese, is also similar to the English (0.0109) series tested by Cleghorn (1961).

The Diego gene, a characteristic of the Mongoloids, is present in the populations of Bhutan, Tibet, Burma and Baltistan (see Table 6.10). The incidence of 0.0217 shown by the Nepalese is in agreement with the Bhutanese, Tibetans and the Chinese (Layrisse and Arends 1956).

6.4 Serum Proteins

The results for two serum protein systems are as follows :

(i) Table 6.11 shows the distribution of the haptoglobin groups and respective gene frequencies in the Nepalese. Good agreement was found between the observed and expected phenotypic values, thus confirming the assumption of Hardy-Weinberg equilibrium. All the subjects showed an absence of phenotype Hp 0-0. No Hp 2-1M (modified) or other rarer phenotype was found. The gene frequencies are : $Hp^1 = 0.2571$ and $Hp^2 = 0.7429$.

(ii) All 212 samples tested for the transferrin variants showed the common type CC.

6.5 Discussion

6.5.1 Haptoglobin Table 6.12

The Nepalese have here been tested for the first time for the serum proteins and most of the red cell enzymes, and comparisons were made with the selected populations of South and South-east Asia.

In the case of the serum haptoglobin system, it has been reported by several investigators that the Indian populations are characterized by low values of the Hp^1 gene. In contrast, the Mongoloid populations of South-east Asia show a slightly higher frequency, ranging between 0.23 and 0.28 (Kirk and Lai 1961). The Hp^1 gene frequency of 0.2571 reported here is within this range and similar to the values of 0.28 and 0.285 for the Chinese and 0.24 for the Thais (Kirk and Lai 1961, Blackwell et al 1962). The statistical analysis also shows that the Nepalese exhibit Hp phenotype and allele frequencies consistent with the Bhutanese, Chinese, Thais and the Malayans.

6.6 Red Cell Isoenzymes

The results of the Nepalese tested for six enzyme systems are given below .

(i) Table 6.13 shows the distribution of AP phenotypes and the respective gene frequencies. No significant deviation from the expected numbers calculated on the basis of Hardy-Weinberg equilibrium was observed. The gene frequencies exhibited by the sample are : $P^a = 0.1529$, $P^b = 0.8398$ and $P^c = 0.0073$.

(ii) The distribution of PGM Locus 1 phenotypes and the respective gene frequencies are set out in Table 6.14. A significant deviation from the expected numbers calculated on the basis of Hardy-Weinberg* equilibrium was observed, $X_2^2 = 11.130$ $0.01<P>0.001$. The gene frequencies are : $PGM_1^1 = 0.7919$ and $PGM_1^2 = 0.2081$.

(iii) Table 6.15 presents the distribution of AK phenotypes and respective gene frequencies in the Nepalese. Good agreement was found between the observed and expected phenotypic values, thus showing that the population is in genetic equilibrium. The gene frequencies are : $AK^1 = 0.9953$ and $AK^2 = 0.0047$.

(iv) Table 6.16 gives the distribution of 6-PGD groups and respective gene frequencies. No significant difference was found between the observed and expected phenotypic values. The calculated gene frequencies are : $PGD^A = 0.9135$ and $PGD^C = 0.0865$.

(v) All the 207 samples tested for the LDH variants showed the normal type and are shown in Table 6.17.

(vi) No variant was found in the MDH system (Table 6.17).

*Although for this system there is a departure from the condition of Hardy-Weinberg equilibrium, it seems none the less appropriate to present the data as found.

6.7 Discussion

6.7.1 Acid phosphatase Table 6.18

From the comparative data set out in Table 6.18 it appears that the gene P^b is the most common of all the three major alleles of the AP system. Its frequency lies between 0.578 and 0.794 in the Indians (see Table 5.22). By contrast, the Mongoloids of South-east Asia show frequencies ranging between 0.654 and 0.778. The frequency exhibited by the Nepalese, 0.8398, is considerably higher and is similar to the 0.8289 given for the Bhutanese by Mourant et al (1968). Similarly, the gene P^a is lower than the Indian and Mongoloid values and is close to the value of the Bhutanese. The gene P^c is found to be absent in the Bhutanese and the Chinese living in Singapore. However, the Nepalese exhibit a lower frequency which is in agreement with the Indian range (0.003 - 0.047, Table 5.22).

A Chi-square test shows that the Nepalese are similar to the Bhutanese with regard to the distribution of AP phenotypes, whereas they differ from the Indians, $X_1^2 = 8.062$ $0.01<P>0.001$, the Chinese, $X_1^2 = 5.945$ $0.05<P>0.02$ and the Malayans, $X_1^2 = 32.197$ $P<0.001$. Concerning the AP alleles, the Nepalese are found to be similar to the Bhutanese, but again they differ from the Indians, $X_1^2 = 7.701$ $0.01<P>0.001$, the Chinese, $X_1^2 = 6.903$ $0.01<P>0.001$ and the Malayans, $X_1^2 = 44.747$ $P<0.001$. It is the lower incidence of P^a and phenotype BA and the higher frequency of P^b and phenotype B in the Nepalese that accounts for these differences.

6.7.2 Phosphoglucomutase locus 1 Table 6.19

Generally speaking, the Indians are characterized by a higher frequency of PGM_1^2 and most of the values are between 30 and 40 percent.

In comparison, the Mongoloids tend to exhibit a slightly lower frequency which is comparable with that found in the North and middle of Europe. The Nepalese seem to exhibit a lower frequency of PGM_1^2, 0.2081, which is below the Indian values and is similar to the figures of 0.2240 given for the Bhutanese and 0.2159 and 0.243 reported for the Chinese tested in Indonesia and San Francisco (Mourant et al 1968, Lie-Injo et al 1968 and Lie-Injo and Poey 1970). Furthermore, the statistical analysis also displays similarities between the Nepalese, the Bhutanese and the Chinese samples. However, the Nepalese differ from the Thais with regard to phenotypes, X_2^2 = 12.854 $0.01<P>0.001$ and genes, X_1^2 = 6.068 $0.02<P>0.01$.

6.7.3 Adenylate kinase Table 6.20

As discussed earlier in Chapter V, the AK^2 gene in India ranges between 0.046 and 0.099. It is either absent or has a very low frequency among the Asiatic Mongoloids. The frequency of 0.0047 here is considerably lower than in India and also differs from the value of 0.0379 given for the Nepalese by Tills et al (1970a). However, the present value is slightly higher but comparable with the figures of 0.0024 for the Thais (Giblett and Scott, unpublished data) and 0.002 for the Chinese tested in Taiwan (Shih et al 1968). Statistical analysis shows that the present Nepalese differ from the Nepalese sample of Tills et al, with respect to genes, X_1^2 = 10.461 $0.01<P>0.001$ and phenotypes X_1^2 = 9.074 $0.01<P>0.001$. The Nepalese exhibit phenotype and gene frequencies similar to those of the Chinese and the Thais, but they show significant variation from the Indians and the Malayans (Chan 1971). It is the higher frequency of phenotype 1-1 and low incidence of 2-1 in the Nepalese that contributes to the observed Chi-squared value.

Nepalese v Indians	$X_1^2 = 53.719$	P<0.001	Phenotypes
Nepalese v Indians	$X_1^2 = 43.100$	P<0.001	Genes
Nepalese v Malayans	$X_1^2 = 4.043$	0.05<P>0.02	Phenotypes
Nepalese v Malayans	$X_1^2 = 3.926$	0.05<P>0.02	Genes

6.7.4 6-Phosphogluconate dehydrogenase Table 6.21

In the Indian sub-continent, the highest frequency of the PGD^C gene (0.2305) is found among the Bhutanese (Mourant et al 1968). Similarly, a high frequency has been observed in Nepal (0.0956) by Tills et al (1970b). The present sample exhibits a frequency of 0.0865 which is greater than the Indian samples included in the table and is similar to the values of 0.0658 for the Chinese and 0.0703 for the Thais (Shih et al 1968 and Giblett and Scott, unpublished data). Statistically, the Nepalese also exhibit gene and phenotype frequencies consistent with those found in the Chinese and the Thais, whereas they differ from the Bhutanese who show a very low incidence of PGD^A and phenotype AA and a higher frequency of PGD^C and phenotype CA. The Nepalese also differ from the Indians (Goedde et al 1972) and the Malayans (Gordon et al 1966), with regard to phenotypes and genes.

Nepalese v Bhutanese	$X_1^2 = 27.534$	P<0.001	Phenotypes
Nepalese v Bhutanese	$X_1^2 = 29.127$	P<0.001	Genes
Nepalese v Assamese (India)	$X_1^2 = 12.749$	P<0.001	Phenotypes
Nepalese v Assamese (India)	$X_1^2 = 11.754$	P<0.001	Genes
Nepalese v Malayans	$X_1^2 = 8.862$	0.01<P>0.001	Phenotypes
Nepalese v Malayans	$X_1^2 = 8.230$	0.01<P>0.001	Genes

6.8 Conclusion

The data from the present study provide a basis for the establishment of a possible relationship between the Nepalese and other populations of South and South-east Asia.

Most of the Nepalese blood group systems studied here show striking differences from those of the Indian peoples, but they resemble the Mongoloids. Considering the A_1A_2BO blood groups only, it is observed that the high frequency of A_1 and the lower incidence of B possessed by the Nepalese is similar to that found in the Bhutanese and the Chinese. However, the Nepalese differ from the Tibetans, the Burmese and some of the Mongoloids of India. With regard to the MNSs system, the high level of gene s indicates close affinity with the Mongoloids of Bhutan, Tibet, China and Burma. Similarly, the general pattern of the Rh system suggests a great connection with the Mongoloids. The low frequency of the Kell gene again is similar to that in the Tibetans and the Bhutanese. Furthermore, the presence of the Diego gene makes it easier to establish a Mongoloid connection.

Of the two serum proteins, the transferrin is found to be invariant and the Hp^1 gene frequency appears to be intermediate between the Indians and the South-eastern peoples.

Among the six enzymes investigated, lactate dehydrogenase and malate dehydrogenase are invariant. Of particular interest is the AP system. The characteristic frequency of P^c is considerably lower than in the Chinese tested in Taiwan and resembles Indian values. On the other hand, the frequency of P^a and P^b is similar to the Bhutanese, but differs from the Indians. The frequency of PGM_1^2 deviates from the Indian range and resembles the Bhutanese and the Chinese, as is the case with the AK^2 gene. The gene PGD^C also shows close relationship with the Mongoloids rather than the Indians.

Finally, the information supplied by this investigation strengthens the view that the Nepalese appear to be closest to the Mongoloids of South and South-east Asia.

CHAPTER VII

GENERAL COMPARISON AND PROPOSALS FOR FURTHER RESEARCH

The aim of the present study was to obtain data on the frequencies of various allelic genes at some of the blood group, serum protein and red cell isoenzyme loci known to exhibit genetic polymorphism, in the Kuwaiti Arabs, the Iranians, the Panjabis and the Nepalese. Geographically, all four populations are widely separated and represent two major populations, the Asiatic Caucasoids and the Mongoloids. The analysis of data shows that the Kuwaiti Arabs, the Iranians and the Panjabis of Northern India illustrate the Caucasoid groups, whereas the Nepalese exhibit genetic characteristics quite different from the forementioned groups and resemble more the Mongoloids of South and South East Asia. Based on observational inspection of the data, an attempt is made here to summarise the features of various genetic markers that distinguish them from each other. Furthermore, some of these characteristics will be discussed in the light of factors that help in viewing the population differences.

Analysis of different blood group systems, as set out in Table 7.1, show that each of the Caucasoid groups possess certain distinguished characteristics. Similarly, the Mongoloid group shows marked differences from the Caucasoids with regard to almost every system. The ABO gene frequencies show a good deal of variation. The Nepalese appear to have a very high frequency of A_1, whereas it is considerably lower in the other populations. The gene A_2, considered as the European gene, is present in all the Caucasoid groups but is absent in the Nepalese. Except for the Panjabis, the incidence of B is relatively low in the other groups. The frequency of the gene O is higher in the Kuwaitis and the

Iranians, whereas it is comparatively low in the Panjabis and the Nepalese. These observable variations show that the frequency of the gene A_1 increases and A_2 disappears as one moves from the Middle East and the Indian region into the Himalayas. With regard to the MNSs system, the Caucasoid groups show the total M frequency varying between 60 and 65 per cent which is higher than the Mediterranean values. By contrast, the Nepalese have a high frequency of the gene M (74%) and gene s (84%). Further comparison shows that the gene S suffers an abrupt fall as one moves into the land of varied peoples broadly described as Mongoloids. The frequency distribution of the Rh gene complexes presents another interesting picture. As in the Mediterranean, the gene complex R_1 is predominant in all the populations. The Kuwaitis and the Iranians do not exhibit much variation, but the Panjabis appear to have a high frequency of R_1. In contrast, the Nepalese again differ from the other groups in possessing a very high frequency of the gene complex R_1 (75%). Thus it appears that the high value of R_1 is another marked feature of the Mongoloids and its frequency increases as one moves to the South East. Except for the Kuwaitis, the incidence of R_2 does not vary much. The gene complex R_o, known as the Negroid component, has a considerably higher frequency in the Kuwaitis. Whereas the value found in the Iranians, Panjabis and Nepalese is like that in Europeans. Again the gene complex r does not exhibit much variation between the Kuwaitis and the Iranians. Its frequency, however, is lower in the Panjabis. By contrast, the Nepalese differ very much in having a moderate frequency of r. The Kell genes do not show much variation. Among the two serum protein systems, it is observed that the Hp^1 frequency is higher in the Kuwaiti Arabs and it starts declining as one moves to Iran and then to Panjab. The Hp^1 frequency exhibited by the Nepalese is also comparatively low. Regarding the transferrin variants, the Kuwaiti Arabs and the Nepalese show the common type CC, whereas the Iranians and the Panjabis are differentiated from others by exhibiting fast and slow variants like Tf CB and Tf CD.

TABLE 7.1 General comparison : frequency distribution of blood group, serum protein and enzyme systems in the Kuwaitis, Iranians, Panjabis and Nepalese

System	Kuwaitis	Iranians	Panjabis	Nepalese
ABO				
A_1	.1518	.1470	.1568	.3429
A_2	.0213	.0325	.0062	-
B	.1268	.1407	.2604	.1371
O	.7001	.6798	.5766	.5200
MNSs				
MS	.2224	.3187	.1665	.1347
Ms	.3814	.3455	.4957	.6058
NS	.0509	.0938	.0537	.0280
Ns	.3453	.2420	.2841	.2315
Rh				
R_z	.0217	.0151	.0042	.0186
R_1	.4920	.4871	.5985	.7492
R_1^W	-	.0147	.0116	-
r'	-	.0175	.0206	-
R_2	.0504	.1229	.1095	.1362
r''	.0234	.0141	-	.0016
R_o	.0880	.0141	.0098	.0399
r	.3246	.3196	.2457	.0545
Kell				
K	.0187	.0214	.0140	.0189
k	.9813	.9786	.9860	.9811

TABLE 7.1 (Contd.)

System	Kuwaitis	Iranians	Panjabis	Nepalese
Hp				
Hp^1	.3449	.2964	.2204	.2571
Hp^2	.6551	.7036	.7796	.7429
Tf				
Tf^C	1.0000	.9944	.9984	1.0000
Tf^B	-	-	.0016	-
Tf^D	-	.0056	-	-
Ap				
P^a	.2032	.3193	.3217	.1529
P^b	.7774	.6618	.6687	.8398
P^c	.0194	.0189	.0096	.0073
PGM_1^1	.7041	.7590	.7044	.7919
PGM_1^2	.2959	.2410	.2956	.2081
AK				
AK^1	.9717	.9502	.9177	.9953
AK^2	.0283	.0498	.0823	.0047
6-PGD				
PGD^A	.9667	.9744	.9772	.9135
PGD^C	.0333	.0256	.0228	.0865
LDH	N	N	N	N
MDH	N	N	N	N

Of all the red cell enzyme systems studied, the lactate dehydrogenase and the malate dehydrogenase systems are found to be invariant in all the populations. In the case of acid phosphatase, no difference exists between the Iranians and the Panjabis with regard to the frequency distribution of the three major alleles P^a, P^b and P^c. The Kuwaitis appear to have a lower incidence of P^a and a higher frequency of P^b. Again the Nepalese also show the presence of P^c. With regard to the phosphoglucomutase locus 1 system, no major difference is observable. But the adenylate kinase and 6-phosphogluconate dehydrogenase systems present an interesting picture. With the exception of the Panjabis, the AK^2 frequency is low in all the groups. The gene PGD^C does not show variation between the Caucasoids, but its incidence is found to be higher in the Nepalese. Furthermore, it seems that the frequency of PGD^C increases as one moves towards the Himalayas.

Now the question arises as to what accounts for such geographical variations. Are these variations the result of evolutionary mechanisms that operate on these populations? Since the biological explanation of most of the polymorphisms is not yet clear, certain characteristics have been selected for discussion in the light of selective or other factors. These characteristics are as follows :

a. Wide variation shown by the ABO gene frequencies
b. High frequency of the gene M in all four populations
c. An appreciable high frequency of the gene complex R_o in the Kuwaiti Arabs
d. Presence of low frequency of r in the Nepalese
e. Considerably high frequency of AK^2 in the Panjabis
f. Comparatively high incidence of PGD^C shown by the Nepalese.

The wide variation shown by the ABO genes appears to be due to a complex interplay of the genetic composition of these populations and the environmental circumstances in which they live. Thus it has been suggested that

certain genotypes are differentially susceptible to diseases such as plague and smallpox. Since the blood types OO, A_2O and A_2A_2 are susceptible to plague, so the populations long subjected to plague could show high frequencies of A_1, B and A_1B (cited in Sunderland 1973). Vogel and Chakravartti (1966) have hypothesised that smallpox shows a higher mortality in subjects of blood groups A and AB as compared to groups B and O. So it is probable that the high incidence of B could be the result of such a factor. Further, the haemolytic disease due to ABO incompatability may be considered responsible for lowering the frequencies of A and B and raising that of O. But again one has to take into account that most of these studies are rather superficial and these considerations do not imply that population differences in the ABO blood group frequencies are the result of these diseases only. For the MNSs system, little is known about the operative selective factors. It appears that the high frequency of the gene M in the Middle East and the Indian sub-continent may be adaptive.

To explain the appreciable frequency of the gene complex R_o in the Kuwaiti Arabs, one has to speculate first whether this is the result of chance, adaptive selection or gene flow. Considering the fact that the incidence of R_o is very high in Africa, roughly 60%, and in the Arabian peninsula, around 20%, it is very likely that this is due to gene flow from Africa. Also the moderate frequency of r shown by the Nepalese could be attributed to the Caucasoid components. Another approach, which may help to give a better explanation, is the study of demographic structure of these widely separated populations. The factors like mortality and morbidity at different ages and fertility may have been selective agents in the past and may have shaped many of the differences that we observe today.

With regard to the red cell enzymes, some suggestions have been put forward to explain the distributional patterns. Of great interest is the presence of a high frequency of AK^2 in the Panjabis. Tills et al (1970a) speculate that this is due either to limited migration from Europe to the Indian sub-continent or progressive diffusion from India to Europe. As the AK^2 frequencies found in the Middle East, the Nepalese and other South Eastern populations is similar to Europeans, so it is probable that the high incidence shown by the Panjabis and other Indian populations is the result of natural selection. The high frequency of PGD^C shown by the Nepalese could be attributed to the long outstanding racial differences between the Mongoloids and non-Mongoloids, or possibly the climate. To interpret such findings, one has to keep in mind that the data are scarce and above all the functional aspects of most of the red cell enzymes in relation to environmental variables are not clearly understood. But again it is worth mentioning here that the similarities in the occurrence of certain polymorphic genes in these populations do not necessarily indicate a common ancestry or other form of correlation as they could very well be the result of similar selective forces.

Finally, from what has been discussed above and in the earlier chapters of this thesis, it can clearly be seen that in the Middle East and the Indian Sub-continent very limited studies have been done on the lines that the International Biological Programme planned. Our knowledge about the genetic structure of different populations living in this region is very meagre. The majority of these populations have been examined only for their ABO groups and the picture which they present generally remains a confused one. Some points which suggest the investigation of untouched populations are as follows :

a. For the better understanding of genetic polymorphism in the South-west of Asia it is important to screen the many tribal groups inhabiting Saudi Arabia, Iraq, Jordan and Syria.

b. Iran, with its varied ethnic groups, some certainly of different origin, presents an almost completely untouched field for serum protein and red cell enzymes investigation.

c. To fill the gap, the populations of Afghanistan and Pakistan await investigation.

d. In India, the ABO studies of the state populations have been numerous but patchy - much remains to be done on the other systems, and despite several recent studies, a great many aboriginal tribes await investigation.

e. In the Himalayas, several population groups living at different altitudes have to be screened for different genetic systems.

ACKNOWLEDGEMENTS

The author's gratitude starts far back - to his late father who encouraged and taught him how to read; to his mother and brothers who saw that he received an education; and to everyone responsible for educating him.

He wishes to thank his supervisor, Professor E Sunderland, for his ever-willing guidance, invaluable assistance, stimulating suggestions and careful reading of the manuscript. He is also grateful to Dr R Cartwright for his encouragement, interest and help at the various stages of the work.

On the other side of the world, he would like to thank Dr J G Jolly, Dr V Bhalla, Mr B S Grewal, Mr K K Bagai, Mr Kulwant Singh Sawhney from the Panjab, India; Dr A Azhir, Dr J Arbabzadeh and Dr M Suzangar from Iran; Dr I Eid, Mr A A Bashir and Miss Jasmiya-Al-Marzook from Kuwait, who were kind enough to provide and send the blood samples even at their personal inconvenience.

Here in England, acknowledgement is gratefully made to : Captain I Bethell for making available the Nepalese specimens for examination; Mr A Berrisford for transporting the blood from London; Dr D Tills for the supply of antisera and kind help in doing the Rh calculations; Dr H Lehman for sending the haemoglobin controls; Dr S S Papiha and Mr D Teasdale for analysing specimens for the Phosphohexose isomerase and haemoglobin systems.

His thanks are also due to the departmental colleagues, and in particular to Mr M Carr for his help during the course of the experimental work. He would like to thank Mrs D Robinson and Mrs B Elvin foi their help and patience in typing all the correspondence regarding the collection of the blood samples. He is also thankful to Mrs R L Reed for her careful typing of this thesis.

He is also thankful to The British Council for the grant of an award for the last two years of this study.

Last but not least he would like to thank the members of his family, in particular his brothers who supported him financially with so much beneficence that his own labours were hardly disturbed.

REFERENCES

Abu-Hakima, A.

(1965) History of Eastern Arabia - The rise and development of Bahrain and Kuwait. 1st edn. Khayats - Beirut, Lebanon.

Ager, J.A.M. and Lehman, H

(1961) Laboratory detection of abnormal haemoglobins. Ass. Clin. Path., broadsheet No. 33.

Allen, G.V. and Scott MacGregor, R.G.

(1947) The emergency blood transfusion in Singapore. Med. J. Malaya., 2, 136.

Allen, F.H. and Diamond, L.K. and Niedziela, B.

(1951) A new blood group antigen. Nature, Lond., 167, 482.

Allen, F.H. and Lewis, S.J.

(1957) Kp^a (Penney), a new antigen in the Kell blood group system. Vox Sang., 2, 81.

Allison, A.C.

(1954) Protection afforded by sickle-cell trait against subtertain malarial infection. Brit. med. J., 1, 290.

Allison, A.C., Blumberg, B.S. and AP Rees, W.

(1958) Haptoglobin types in British, Spanish, Basque and Nigerian African populations. Nature, Lond., 181, 824.

Altounyan, E.H.R.

(1928) Blood group percentages for Arabs, Armenians and Jews. Brit. med. J., 1, 546.

Anand, S.

(1957) A genetic survey of ABO blood group among the Punjabis. Anthropologist, Delhi., 4, 28.

Ananthakrishnan, R.

(1972) Further studies on the distribution of some serum protein and enzyme groups in South India. Humangenetik, 15, 172.

Ananthakrishnan, R. and Kirk, R.L.

(1969) The distribution of some serum protein and enzyme group systems in two endogamous groups in South India. Ind. J. med. Res., 57, 1011.

Ananthakrishnan, R., Blake, N.M., Kirk, R.L. and Baxi, A.J.

(1970) Further studies on the distribution of genetic variants of lactate dehydrogenase (LDH) in India. Med. J. Aust., 2, 787.

Ananthakrishnan, R. and Walter, H.

(1972) Some notes on the geographical distribution of the human red cell acid phosphatase phenotypes. Humangenetik, 15, 177.

Ashton, G.C.

(1965) Cattle serum transferrin : a balanced polymorphism. Genetics, 52, 983.

Azhir, A.

(1951) Personal Communication. Cited by Mourant (1954).

Bajatzadeh, M. and Walter, H.

(1968) Serum protein polymorphisms in Iran. Humangenetik, 6, 40.

Bajatzadeh, M. and Walter, H.

(1969) Investigations on the distribution of blood and serum groups in Iran. Hum. Biol., 41, 401.

Banerjee, S. and Kumar, N.

(1953) Blood group distributions of the people of Jaunsar - Bawar. Bull. Dept. Anthrop. India., 2, 55.

Barnicot, N.N., Garlick, J.P. and Roberts, D.F.

(1960) Haptoglobin and transferrin inheritance in northern Nigerians. Ann. hum. Genet., 24, 171.

Baxı, A.J., Balakrıshnan, V. and Sanghvı, L.D.

(1961) Defıcıency of glucose-6-phosphatase dehydrogenase observatıons on a sample from Bombay. Curr. Scı., 30, 16.

Baxı, A.J., Balakrıshnan, V., Undevıa, J.V. and Sanghvı, L.D.

(1963) Glucose-6-phosphate dehydrogenase defıciency ın thc Parsee Communıty, Bombay. Ind. J. med. Scı., 17, 493.

Bayı, A.J. and Hakım, M.A. Shırın.

(1966) Haptoglobın types ın Marathas of Bombay. Ind. J. med. Res., 54, 1150.

Baxı, A.J. and Parıkh, N.P.

(1967) Dıstrıbutıon of haptoglobıns, haemoglobıns and glucose-6-phosphate dehydrogenase (G-6-PD) defıcıency ın three Gujaratı speakıng Communıtıes from Western Indıa. 4th Congr. Asıan Pacıfıc Soc. of Haemat., New Delhı.

Baxı, A.J. and Camoens, H.

(1969a) Studies on haptoglobın types in varıous Indıan populations. Hum. Hered., 19, 65.

Baxı, A.J. and Camoens, H.

(1969b) Transferrın polymorphısm ın Indıans. Proc. Indıan Soc. Haemat. and Blood Transfusıon. Hyderabad, p. 41.

Beckett, P.H.

(1950) Unpublished ıeport of Oxford Unıversıty expedıtion to Persıa : Cıted ın Mourant (1954).

Benercettı, A. Silvano, S., Cattanei, A. and Khan, P.M.

(1972) A new variant allele Ak^5 of the red cell adenylate kınase polymorphısm ın a non-trıbal Indıan populatıon. Hum. Hered., 22, 171.

Berg, K.

(1969) Genetıc studıes of the adenylate kınase (AK) polymorphısm. Hum. Hered., 19, 239.

Bernstein, F.

(1924) Ergebnisse einer biostatischen zus ammenfassenden Betrachtung uber die erblichen Blutstrukturen des.Menschen. Klin. Wschr., 3, 1495, cited in Race, R.R. and Sanger, R. (1970).

Bhalla, V.

(1961) A study of ABO blood groups in Kinner-Kanets of Chinni Valley (H.P.). Ind. Sci. Proc., part iii, p. 442.

Bhalla, V.

(1963) The genetic analysis of ABO, MNSs, Rh-Hr and Duffy blood group systems in three endogamous groups of Panjabi Hindus and an association of blood groups with tuberculosis. Ph.D. Thesis. University of Delhi.

Bhasin, M.K.

(1970) The blood groups of the Newars of Nepal. Hum. Biol., 42, 369.

Bhasin, B.K. and Fuhrmann, W.

(1972) Geographic and ethnic distribution of some red cell enzymes. Humangenetik, 14, 204.

Bhattacharjee, P.N.

(1966) Distribution of the blood groups (A_1 A_2BO, MNSs, Rh), and the secretor factor among the Muslims and the Pandits of Kashmir. Z. Morph. Anthrop., 58, 86.

Bhattacharjee, P.N.

(1968a) The blood groups (A_1 A_2BO, MNS and Rh) of the Ladakhis. Acta genet., Basel, 18, 78.

Bhattacharjee, P.N.

(1968b) Sero-genetical variations in the Lepchas of Darjeeling district with special reference to the Indian Mongoloids. Bull. Anthrop. Surv. India.,17, 382.

Bhattacharjee, P.N.

(1968c) Further study of the Tibetan blood groups. J. Indian anthrop. Soc., 3, 57.

Bird, G.W.G. and Lehman, H.

(1956) Haemoglobin-D in India. Brit. med. J., i, 514.

Bird, G.W.G., Ikin, E.W., Lehman, H. and Mourant, A.E.

(1956) The blood groups and haemoglobins of the Sikhs. Heredity, 10, 425.

Bird, G.W.G., Jayaram, T.K., Ikin, E.W., Mourant, A.E. and Lehman, H.

(1957) The blood groups and haemoglobins of the Gorkhas of Nepal. Amer. J. phys. Anthrop., 15, 163.

Blackwell, R.Q., Lin, Tsau-Yen and Shiao, D.D.F.

(1962) Distribution of haptoglobin among Chinese in Taiwan. Nature, Lond., 193, 284.

Blackwell, R.Q. and Thephusdin, C.

(1963) Distribution of haptoglobin among Thais. Nature, Lond., 197, 503.

Blake, N.M., Kirk, R.L. and Baxi, A.J.

(1970a) The distribution of some enzyme group systems among Marathis and Gujaratis in Bombay. Hum. Hered., 20, 409.

Blake, N.M., Kirk, R.L., Simons, M.J. and Alpers, M.P.

(1970b) Genetic variants of soluble malate dehydrogenase in New Guinea populations. Humangenetik, 11, 72.

Blake, N.M., Kirk, R.L., McDermid, E.M., Omoto, K. and Ahuja, Y.R.

(1971) The distribution of serum protein and enzyme group systems among north Indians. Hum. Hered., 21, 440.

Bonne, B.

(1966) Genes and phenotypes in the Samaritan Isolate. Amer. J. phys. Anthrop., 24, 1.

Bonne, B., Ashbel, S., Modai, M., Godber, M.J., Mourant, A.E., Tills, D. and Woodhead, B.G.

(1970) The Habbanite Isolate 1. Genetic markers in the blood. Hum. Hered., 20, 609.

Boue, A. and Boue, J.

(1955) Etude sur la repartition des groupes sanguins en Iran. Le Sang.,26, 705.

Boue, A. and Boue, J.

(1956) Etude sur la repartition des groupes sanguins en Iran. II. Ann. Inst. Pasteur.,91, 898.

Bowman, J.E. and Ronaghy, H.

(1967) Haemoglobin, glucose-6-phosphate dehydrogenase, and adenylate kinase polymorphism in Moslems in Iran. Amer. J. phys. Anthrop., 27, 119.

Bowman, J.E., Frischer, H., Ajmar, F., Carson, P.E. and Gower, M.K.

(1967) Population, family and biochemical investigation of human adenylate kinase polymorphism. Nature, Lond., 214, 1156.

Boyd, W.C.

(1939) Blood groups. Tabul. biol. Hague, 17, 113.

Boyd, W.C. and Boyd, L.G.

(1938) Blood groups of the Rwala Bedouin. J. Immunol., 34, 441.

Boyd, W.C. and Boyd, L.G.

(1941) Blood groups and types in Baghdad and vicinity. Hum. Biol., 13, 398.

Boyer, S.H., Fainer, D.C. and Watson-Williams, E.J.

(1963) Lactate dehydrogenase variant from human blood : evidence for molecular subunits. Science, 141, 642.

Buchi, E.C.

(1955) A genetic survey among the Malapantarum. A hill tribe of Travancore. Anthropologist, Delhi.,2. 11.

Callender, S., Race, R.R. and Paykoc, Z.V.

(1945) Hypersensitivity to transfused blood. Brit. med. J., ii, 83.

Camoens, H.

(1971) Adenylate kinase (AK) phenotypes in an Indian population sample. Hum. Hered., 21, 297.

Carter, N.D., Fildes, R.A., Fitch, L.I. and Parr, C.W.

(1968) Genetically determined electrophoretic variations of human phosphogluconate dehydrogenase. Acta genet., Basel, 18, 109.

Chan, K.L.

(1971) Human red cell adenylate kinase polymorphism in West Malaysian population. Hum. Hered., 21, 173.

Chatterjea, J.B.

(1966) Haemoglobinopathies : glucose-6-phosphate dehydrogenase deficiency and allied problems in the Indian sub-continent. Bull. Wld. Hlth. Org., 35, 837.

Chopra, V.P.

(1970) Studies on the serum groups in the Kumaon region, India. Humangenetik, 10, 35.

Chown, B., Lewis, M. and Kaita, H.

(1965) The Duffy blood system in Caucasians : evidence for a new allele. Amer. J. hum. Genet., 17, 384.

Clegg, E.J., Ikin, E.W. and Mourant, A.E.

(1961) The blood groups of the Baltis. Vox Sang., 6, 604.

Cleghorn, T.E.

(1961) Unpublished observations in Race, R.R. and Sanger, R. (1970)

Connell, G.E. and Smithies, O.

(1959) Human Haptoglobins : estimation and purification. Biochem. J., 72, 115.

Connell, G.E., Dixon, G.H. and Smithies, O.

(1962) Subdivision of three common Haptoglobin types based on 'hidden' differences. Nature, Lond., 193, 505.

Coombs, R.R.A., Mourant, A.E. and Race, R.R.

(1946) In-vivo isosensitization of red cells in babies with haemolytic disease. Lancet, i, 264.

Crawford, M.N., Greenwalt, T.J., Saski, T., Tippett, P., Sanger, R. and Race, R.R.

(1961) The phenotype Lu (a-b-) together with unconventional Kidd groups in one family. Transfusion, Philad., I, 228.

Curtain, C.C., Gajdusek, D.C., Kidson, C., Gorman, J.G., Champness, L. and Rodrigue, R.

(1965) Haptoglobin and transferrin in Melanesia : Relation to haemoglobin, serum haptoglobin and serum iron levels in population groups in Papua, New Guinea. Amer. J. phys. Anthrop., 23, 363.

Cutbush, M. and Mollison, P.L.

(1950) The 'Duffy' blood group system. Heredity, 4, 383.

Cutbush, M., Mollison, P.L. and Parkin, D.M.

(1950) A new human blood group. Nature, Lond., 165, 188.

Cutbush, M. and Chanarin, I.

(1956) The expected blood-group antibody, anti-Lu^b. Nature, Lond., 178, 855.

Da Costa, H., Pattani, J., Mehandle, K., Desai, M. and Merchant, S.M.

(1967) Glucose-6-phosphate dehydrogenase defect in Indian children. A preliminary report. Indian Pediat., 4, 163.

Darnborough, J., Firth, R., Giles, C.M., Goldsmith, K.L.G. and Crawford, M.N.

(1963) A 'new' antibody anti-Lu^a Lu^b and two further examples of the genotype Lu(a-b-). Nature, Lond., 198, 796.

Das, S.R., Mukherjee, D.P. and Bhattacharjee, P.N.

(1967) Survey of the blood groups and PTC taste among the Rajbanshi caste of West Bengal. Acta genet., Basel, 17, 433.

Das, S.R. and Mukherjee, B.N.

(1970) The distribution of some enzyme group systems among Bengalis. Ind. J med. Res., 58, 866.

Das, S.R., Mukherjee, B.N., Das, S.K., Ananthakrishnan, R., Blake, N.M. and Kirk, R.L.

(1970) LDH variants in India. Humangenetik, 9, 107.

Das, S.R., Mukherjee, B.N. and Das, S.K.

(1972) Caste and age variations of the incidence of LDH variants in the Bengali Hindus. Humangenetik, 14, 151.

Das, S.R., Mukherjee, S.K.D., Monami, R. and Chhatui, S.S.

(1974) Blood groups, serum proteins, haemoglobin and some serum and red cell enzymes among the Kaoras of 24-Paragans in West Bengal (India). Hum. Hered., 24, 24.

Davidson, R.G.

(1967) Electrophoretic variants of human 6-phosphogluconate dehydrogenase : population and family studies and description of a new variant. Ann. hum. Genet., 30, 355.

Davidson, R.G. and Cortner, J.A.

(1967) Genetic variant of human erythrocyte malate dehydrogenase. Nature, Lond., 215, 761.

Davison, G.

(1974) Personal Communication.

Decastello, A.V. and Sturli, A.

(1902) Ueber die Isoagglutinine in serum gesunder und kranker Menschen. Munch. med. Wschr., 1090, in Race, R.R. and Sanger, R. (1970).

Delhi University

(1957) Brahmins, Kanets and scheduled castes of Simla hills. (Unpubl. dissertation work).

Delhi University

(1958) Brahmins and Rajputs of Kulu and Katrain. (Unpubl. dissertation work).

Delhi University

(1959) Brahmins, Mahajans and Aryas of Chamba. (Unpubl. dissertation work).

Detter, J.C., Ways, P.O., Giblett, E.R., Baughan, M.A., Hopkinson, D.A., Poey, S. and Harris, H.

(1968) Inherited variations in human phosphohexose isomerase. Ann. hum. Genet., 31, 329.

Dimson, S.B. and McMartin, R.B.

(1946) Pamaquine hemoglobinuria. Quart. J. med., 15, 25.

Dungern, E.V. and Hirszfeld, L.

(1911) Ueber Verebung gruppenspezifischer strukturen des Blutes. Z. Immun. Forsch., 6, 284, cited in Race, R.R. and Sanger, R. (1970).

Furhud, D.D., Ananthakrishnan, R., Walter, H. and Laser, J.

(1973) Electrophoretic investigations of some red cell enzymes in Iran. Hum. Hered., 23, 263.

Ffrench, G.E. and Hill, A.G.

(1971) Kuwait. Springer-Verlag Berlin - Heidelberg - New York.

Field, H.

(1939) Contributions to the Anthropology of Iran. Anthrop. Series, Field Museum of Natural History, New York, Kraus.

Field, H.

(1957) Personal communication. Cited by Mourant et al (1958).

Fildes, R.A. and Parr, C.W.

(1963) Human red cell phosphogluconate dehydrogenase. Nature, Lond., 200, 890.

Fildes, R.A. and Harris, H.

(1966) Genetically determined variation of adenylate kinase in man. Nature, Lond., 209, 261.

Ford, E.B.

(1940) The New Systematics, ed. Julian Huxley. Clarendon Press, Oxford.

Ghai, O.P., Varma, K.P.S. and Taneja, P.N.

(1961) Haemoglobinopathies in North India. Haemoglobin 'D' trait and haemoglobin 'D' Thalassaemia. Indian J. Child Health.,10, 334.

Giblett, E.R.

(1967) Variant phenotypes : haptoglobin, transferrin and red cell enzymes, in Greenwalt, T.J. (ed.). Advances in Immunogenetics, p. 99, J.B. Lippincott, Philadelphia.

Giblett, E.R.

(1969) Genetic markers in human blood. Blackwell Scientific Public., Oxford.

Giblett, E.R. and Scott, N.M.

(1965) Red cell acid phosphatase : racial distribution and report of a new phenotype. Amer. J. hum. Genet., 17, 425.

Giblett, E.R. and Scott, N.M.

Unpublished data : cited in Giblett, E.R. (1969).

Glasgow, B.G., Goodwin, M.J., Jackson, F., Kopec, A.C., Lehman, H., Mourant, A.E., Tills, D., Turner, R.W.D. and Ward, M.P.

(1968) The blood groups, serum groups and haemoglobins of the inhabitants of Lunana and Thimbu, Bhutan. Vox Sang., 14, 31.

Goedde, H.W., Benkmann, H.G., Singh, S., Das, B.M., Chakravarti, M.R., Delbruck, H. and Flatz, G.

(1972) Genetic Survey in the population of Assam 2 : Serum protein and erythrocyte enzyme polymorphism. Hum. Hered., 22, 331.

Gordon, H., Vooijis, M. and Kerran, M.M.

(1966) Genetical variation in some human red cell enzymes : an interracial Study. S. Afr. med. J., 40, 1031.

Goyal, R.K. and Anand, B.R.

(1953) Distribution of blood groups (A, B, O and AB) in Rajasthan. J. Indian med. Ass., 22, 233.

Guha, B.S.

(1931) Racial affinities of the people of India. Census of India, 1, part III. Delhi : Manager of Publications.

Guha, B.S.

(1946) Racial elements in the population. Oxford pamphlets on Indian affairs No. 22. Oxford University Press.

Gupta, S., Ghai, O.P. and Chandra, R.K.

(1970) Glucose-6-phosphate dehydrogenase deficiency in the new born and its relation to serum bilirubin. Ind. J. Pediat., 37, 169.

Hagen, T.

(1961) Nepal. Geographical Publications, Bonn.

Hakim, S.M.A., Baxi, A.J., Balakrishnan, V., Kulkarni, K.V., Rao, S.S. and Jhala, H.I.

(1973) Blood groups, secretor status and ability to taste phenylthiocarbamide in some Muslim groups. Hum. Hered., 23, 72.

Haldane, J.B.S.

(1949) Disease and evolution. La Ricerca Scientif. (suppl.), 19, 68.

Harris, H.

(1970) The principles of human biochemical genetics. North Holland Public., Amsterdam - London.

Hirszfeld, L. and Hirszfeld, H.

(1919) Serological differences between the blood of different races. Lancet, ii. 675.

Hopkinson, D.A.

(1968) Genetically determined polymorphisms of erythrocyte enzymes in man. Adv. Clin. Chem., 11, 21.

Hopkinson, D.A., Spencer, N. and Harris, H.

(1963) Red cell acid phosphatase variants : a new human polymorphism. Nature, Lond., 199, 969.

Hopkinson, D.A. and Harris, H.

(1966) Rare phosphoglucomutase phenotypes. Ann. hum. Genet., 30, 167.

Horsfall, W.R. and Smithies, O.

(1958) Genetic control of some human serum β-Globulins. Science, 128, 35.

House, R.J. and Mahalanobis, P.C.

(1939-45) Personal communication. Cited by Mourant, A.E. (1954).

Huehns, E.R. and Shooter, E.M.

(1965) Human Haemoglobins. J. med. Genet., 2, 48.

Hummel, K., Pulverer, G., Schaal, K.P. and Weidtman, V.

(1970) Haufigkeit der Sichttypen in den Erbsystemen Haptoglobin Gc, saure erythrozytenphosphatase, Phosphoglucomutase und Adenylate kinase sowie den Erbeigenschaften Gm (1), GM (2) und Inv (1) bei Deutschen (aus dem Raum Freiberg i. Br und koln) und bei Turken. Humangenetik, 8, 330.

Hurkat, P.C., Mittal, S.R. amd Sharda, S.

(1971) Distribution of ABO blood groups in Rajasthan (Ajmer). Curr. med. Pract., 15, 628.

Ikin, E.W.

(1958) Personal Communication. Cited by Mourant et al (1958).

Ikin, E.W.

(1963) The incidence of the blood group antigens in different populations. Ph.D. Thesis. University of London.

Ikin, E.W., Mourant, A.E., Pettenkofer, H.J. and Blumenthal, G.

(1951) Discovery of the expected haemagglutin, anti-Fy^b. Nature, Lond., 168, 1077.

Ikin, E.W., Lehman, H., Mourant, A.E. and Thein, H.

(1969) The blood groups and haemoglobins of the Burmese. Man, 4, 118.

Jenkins, T. and Corfield, V.

(1972) The red cell phosphatase polymorphism in South Africa. Population data and studies on the R, RA and RB phenotypes. Ann. hum. Genet., 35, 379.

Jolly, J.G., Sarup, B.M., Bhatnagar, D.P. and Maini, S.C.

(1972) Glucose-6-phosphate deficiency in India. Indian J. med. Asso., 58, 196.

Karp, G.W. and Sutton, H.E.

(1967) Some new phenotypes of human red cell acid phosphatase. Amer. J. hum. Genet., 19, 54.

Kayassi, A.I., Boyd, W.C. and Boyd, L.G.

(1938) Blood groups of the bedouin near Baghdad. Amer. J. phys. Anthrop., 23, 295.

Kennedy, W.P. and Macfarlane, J.

(1936) Blood groups in Iraq. Amer. J. phys. Anthrop., 21, 87.

Khan, A.M.

(1952) Blood group frequencies in the Punjab. Medicus, Karachi., 5, 29.

Khanduja, P.C., Agarwal, K.N., Julka, S., Bhargava, S.K. and Taneja, P.N.

(1966) Incidence of glucose-6-phosphate dehydrogenase deficiency and some observations on patients with haemoglobinuria. Ind. J. Pediat., 33, 341.

Kirk, R.L.

(1968) Monographs in Human Genetics - The haptoglobin groups in man. Karger, Basel. Switzerland.

Kirk, R.L. and Lai, L.Y.C.

(1961) The distribution of haptoglobins and transferrins in South and South East Asia. Acta genet; Basel, 11, 97.

Kirk, R.L., Lai, L.Y.C., Vos, G.H., Wickremasinghe, R.L. and Perera, D.J.B.

(1962) The blood and serum groups of selected populations in South India and Ceylon. Amer. J. phys. Anthrop., 20, 485.

Krik, R.L , Blake, N.M., Lai, L.Y.C., and Cooke, D.R.

(1969) The distribution of some serum protein and enzyme groups among the Malag of Elcho Island. Archaeol. phys. Anthrop. Oceania., 4, 238.

Kirk, R.L., Blake, N.M., Moodie, P.M. and Tibbs, G.J.

(1971) The distribution of some protein and enzyme groups among populations at various localities in the Northern Territory of Australia. Hum. Biol. Oceania., 1, 54.

Kohn, J.

(1957) A new supporting material for zone electrophoresis. Biochem. J., 65, 9.

Kumar, N. and Sastry, D.E.

(1961) A genetic Survey among the Riang : A Mongoloid tribe of Tripura. Z. Morp. Anthrop., 51, 346.

Lai, L., Nevo, S. and Steinberg, A.G.

(1964) Acid phosphatase of human red cells : predicted phenotype conforms to a genetic hypothesis. Science, 145, 1187.

Lai, L.Y.C. and Kwa, S.B.

(1968) Red cell acid phosphatase types in some populations of South-East Asia. Acta gent. Basel., 18, 45.

Lamm, L.U.

(1970) Family studies of red cell acid phosphatase types. Report of a family with the D variant. Hum. Hered., 20, 329.

Landsteiner, K.

(1900) Zur kenntnis der antifermentativen Iytischen und agglutinierenden Wirkungen des Blutserums und der lympha. Zbl. Bakt., 27, 357. Cited in Race, R.R. and Sanger, R. (1970).

Landsteiner, K.

(1901) Uber agglutination ser scheinungen normalen menschlichen Blutes. Wien klin. Wschr., 14, 1132. Cited in Race, R.R. and Sanger, R. (1970).

Landsteiner, K. and Levine, P.

(1927a) A new agglutinable factor differentiating individual human bloods. Proc. Soc. exp. Biol. N.Y., 24, 600.

Landsteiner, K. and Levine, P.

(1927b) Further observations on individual differences of human blood. Proc. Soc. exp. Biol. N.Y., 24, 941.

Landsteiner, K. and Levine, P.

(1928) On the inheritance of agglutinogens of human blood demonstrable by immune agglutinins. J. exp. Med., 48, 731.

Landsteiner, K. and Wiener, A.S.

(1940) An agglutinable factor in human blood recognised by immune sera for Rhesus blood. Proc. Soc. Exper. Biol. and Med., 43, 223.

Layrisse, M., Arends, T. and Dominguez Sisco, R.

(1955) Nuevo grupo sanguineo encontrado en descendientes de Indios. Acta Medica Venzolana., 3, 132. Cited in Race, R.R. and Sanger, R. (1970).

Layrisse, M. and Arends, T.

(1956) The Diego blood factor in Chinese and Japanese. Nature, Lond., 177, 1083.

Layrisse, M. and Wilbert, J.

(1960) El Antigen del Sistema Sanguineo Diego, pp. 160, La Fundacion Creole y la Fundacion Eugenio Mendoza, Caracas. Cited in Race, R.R. and Sanger, R. (1970).

Leakey, T.E.B., Coward, A.R., Warlow, A. and Mourant, A.E.

(1972) The distribution in human populations of electrophoretic variants of cytoplasmic malate dehydrogenase. Hum. Hered., 22, 542.

Lehman, H. and Cutbush, M.

(1952) Sub-division of some Southern Indian communities according to the incidence of sickle-cell trait and blood groups. Trans. R. Soc. trop. Med., 46, 380.

Lehman, E. and Ikin, E.W.

(1953) Personal communication. Cited by Mourant (1954).

Lehman, H. and Carrell, R.W.

(1969) Variations in the Structure of human haemoglobin. Brit. Med. Bull., 25, 14.

Lehman, H., Ala, F., Hedeyat, S., Montazemi, K., Nejd, H.K., Lightman, S., Kpoec, A.C., Mourant, A.E., Teesdale, P. and Tills, D.

(1973) The hereditary blood factors of the Kurds of Iran. Phil. Trans. R. Soc. Lond., 266, 195.

Levine, P. and Stetson, R.E.

(1939) An unusual case of intragroup agglutination. J. Amer. med. Ass., 113, 126.

Levine, P., Burnham, L., Katzin, E.M. and Vogel, P.

(1941) The role of isoimmunization in the pathogenesis of erythroblastosis fetalis. Amer. J. Obst. Gynec., 42, 925.

Levine, P., Backey, M., Wigod, M. and Ponder, R.

(1949) A new human hereditary blood property (Cellano) present in 99.8% of all bloods. Science, 109, 464.

Levine, P., Kuhmichel, A.B., Wigod, M. and Koch, E.

(1951) A new blood factor, s_1, allelic to S. Proc. Soc. exp. Biol. N.Y., 78, 218.

Lie-Injo, L.E. and Ti, T.S.

(1964) Glucose-6-phosphate dehydrogenase deficiency in Malayans. Trans. roy. Soc. trop. Med. Hyg., 58, 500.

Lie-Injo, L.E., Lopez, C.G. and Poey-Oey-Hoey, G.

(1968) Erythrocyte and leucocyte phosphoglucomuta in Chinese. Amer. J. hum. Genet., 20, 101.

Lie-Injo, L.E. and Poey-Oey-Hoey, G.

(1970) Phosphoglucomutase, carbonic anhydrase and catalase in Indonesians. Hum. Hered., 20, 215.

Majumdar, D.N. and Kishen, K.

(1947) Report on the serological survey of the United Provinces. Eastern Anthropologist, 1, 8.

Malone, R.H. and Lahiri, M.N.

(1928-29) The distribution of the blood groups on certain races and castes of India. Ind. J. med. Res., 16, 963.

Maranjian, G., Ikin, E.W., Mourant, A.E. and Lehman, H.

(1966) The blood groups and haemoglobins of the Saudi Arabians. Hum. Biol., 38, 394.

Marengo-Rowe, A.G., Godber, M.G., Kopec, A.C., Mourant, A.E., Tills, D. and Woodhead, B.G.

(1974) The inherited blood factors of the inhabitants of Southern Arabia. Ann. hum. Biol., 1, 311.

Meera Khan, P.

(1964) Glucose-6-phosphate dehydrogenase deficiency in an Indian rural area. J. Genet., 59, 14.

Miller, E.B., Rosenfield, E.R. and Vogel, P.

(1951) On the incidence of some of the new agglutinogens in Chinese and Negores. Amer. J. phys. Anthrop., 9, 115.

Modiano, G., Filippi, G., Brunelli, F., Frattaroli, W. and Siniscalco, M.

(1967) Studies on red cell acid phosphatase in Sardinia and Rome. Absence of correlation with past malarial morbidity in Sardinia. Acta genet., Basel, 17, 17.

Modiano, G., Scozzari, R., Gigliani, F., Santolamazza, C., Afeltra, P. and Frattaroli, W.

(1970) Red cell Phosphoglucomutase polymorphism. I. Enzyme activity of different red cell PGM phenotypes. Hum. Hered., 20, 86.

Moshkovski, S., Said, S. and Rihab, M.

(1931) Quoted from Steffan, P. and Wellisch, S. (1933). Die geographische verteilung de Blutgruppen. Z. Rassenphysiol., 6, 28.

Motamed, M.

(1949) Personal Communication. Cited by Mourant, A.E. (1954).

Motulsky, A.G. and Yoshida, A.

(1969) Methods for the study of red cell Glucose-6-phosphate dehydrogenase. Biochemical methods in red cell genetics. ed. by Yunis, J.J. p. 52. Academic Press, New York.

Mourant, A.E.

(1954) The distribution of the human blood groups. Blackwell Scientific Public., Oxford.

Mourant, A.E.

(1963) Blood groups in South-west Asia. in E. Goldschmidt (ed.). The genetics of migrant and isolate populations. Williams and Wilkins.

Mourant, A.E., Kopec, A.C. and Domaniewski-Sobczak, K.

(1958) The ABO blood groups. Comprehensive tables and maps of world distribution. Blackwell Scientific Public., Oxford.

Mourant, A.E., Godber, E.M., Kopec, A.C., Lehman, H., Steele, P.R. and Tills, D.

(1968) The hereditary blood factors of some populations of Bhutan. Anthropologist, Delhi.,29.

Mukherjee, B.N. and Das, S.K.

(1970) The haptoglobin and transferrin types in West Bengal and a case of Haptoglobin 'Johnson'. Hum. Hered., 20, 209.

Mukherjee, B.N., Das, S.K. and Kellermann, G.

(1974) Study of some serum group systems in the Mahishyas and the Muslims in 24- Paraganas district, West Bengal. Humangenetik, 21, 27.

Musil, A.

(1928) The manners and customs of the Rwala Bedouins. Amer. Geo. Soc., Oriental Explorations and Studies, No. 6.

Nagaratnam, N., Leelawaithie, P.K. and Weerasinghe, W.M.T.

(1969) Enzyme glucose-6-phosphate dehydrogenase (G-6-PD) deficiency amongst Sinhalese in Ceylon as revealed by methaemoglobin reduction test. Ind. J. med. Res., 57, 569.

Nance, W.E., Claflin, A. and Smithies, O.

(1963) Lactic dehydrogenase : genetic control in man. Science, 142. 1075.

Nijenhuis, L.E.

(1964) Blood group frequencies in Iran. Vox Sang., 9, 723.

Nijenhuis, L.E. and Runia, J.

(1963) Blood group frequencies and haemoglobin types in Tibetans and Nepalese. Vox Sang., 8, 622.

Onsi, A.A. and El-Alfi, O.S.

(1968) The ABO blood groups in Kuwait. J. Kwt. med. Assoc., 2, 3.

Orjasaeter, H., Kornstad, L. and Larsen Heier, A.M.

(1966) A contribution to the blood group frequencies in Tibetans. Vox Sang., 11, 726.

Panjab University.

(1962) Brahmins and Rajputs of Rampur Bushehar. (unpubl. dissertation work).

Papiha, S.S.

(1973) Haptoglobin and abnormal haemoglobin types in Sinhalese and Panjabis. Hum. Hered., 23, 147.

Papiha, S.S., Roberts, D.F., Wig, N.N. and Singh, S.

(1972) Red cell enzyme polymorphisms in Punjabis in North India. Amer. J. phys. Anthrop., 37, 293.

Papiha, S.S. and Wastell, H.J.

(1974) Transferrin variants in the Indian Sub-Continent. Humangenetik, 21, 69.

Parikh, N.P., Baxi, A.J. and Jhala, H.I.

(1969) Blood groups, abnormal haemoglobins and other genetical characters in three Gujarati speaking groups. Hum. Hered., 19, 486.

Parr, C.W.

(1931) Blood studies on peoples of Western Asia and North Africa. Amer. J. phys. Anthrop., 16, 15.

Parr, C.W.

(1966) Erythrocyte phosphogluconate dehydrogenase polymorphism. Nature, Lond., 210, 487.

Parr, C.W. and Fitch, L.I.

(1967) Inherited quantitative variations of human phosphogluconate dehydrogenase. Ann. hum. Genet., 30, 339.

Pinkerton, F.J., Mermod, L.E., Liles, Bobbie Ann, Jack, J.A. and Noades, Jean.

(1959) The phenotype JK (a-b-) in the Kidd blood group system. Vox Sang., 4, 155.

Plato, C.C., Rucknagel, D.L. and Gershowitz, H.

(1964) Studies on the distribution of glucose-6-phosphate dehydrogenase deficiency. Thalassemia and other genetic traits in the coastal and mountain villages of Cyprus. Amer. J. hum. Genet., 16, 267.

Plaut, G., Ikin, E.W., Mourant, A.E., Sanger, R. and Race, R.R.

(1953) A new blood-group antibody, anti-JK^b. Nature, Lond., 171, 431.

Polonovski, M. and Jayle, M.F.

(1938) Existence dans le plasma Sanguin d'une substance activant l'action peroxydasique de l'hemoglobine. C.r. Soc. Biol., 129, 457.

Poulik, M.D.

(1957) Starch-gel electrophoresis in a discontinuous system of buffers. Nature, Lond., 180, 1477.

Race, R.R. and Sanger, R.

(1950) Blood groups in Man. 1st edn. Blackwell Scientific Public., Oxford.

Race, R.R., Sanger, R., Allen, R.H., Diamond, L.K. and Niedziela, B.

(1951) Inheritance of the human blood group antigen JK^a. Nature, Lond., 168, 207.

Race, R.R. and Sanger, R.

(1958) Blood groups in Man. 3rd edn. Blackwell Scientific Public., Oxford.

Race, R.R. and Sanger, R.

(1962) Blood groups in Man. 4th edn. Blackwell Scientific Public., Oxford.

Race, R.R. and Sanger, R.

(1970) Blood groups in Man. 5th edn. Blackwell Scientific Public., Oxford.

Raper, A.B.

(1955) Malaria and the sickling trait. Brit. med. J., 1, 1186.

Rapley, S., Robson, E.B., Harris, H. and Smith, M.

(1967) Data on the incidence segregation and linkage relations of the adenylate kinase (AK) polymorphism. Ann. hum. Genet., 31,237.

Regmi, D.R.

(1965) Mediaeval Nepal. Vols. 1-3. Calcutta.

Risley, H.

(1915) People of India. Calcutta. Thaker Spink and Co.

Roberts, D.F., Papiha, S.S. and Abeyaratne, K.P.

(1972) Red cells enzyme polymorphisms in Ceylon Sinhalese. Amer. J. hum. Genet., 24, 181.

Roberts, D.F., Papiha, S.S., Green, C.K., Chhaparwal, B.C. and Mehta, S.

(1974) Red cell enzyme and other polymorphic systems in Madhya Pradesh, Central India. Ann. hum. Biol., 1, 159.

Saha, N.

(1969) Incidence of glucose-6-phosphate dehydrogenase deficiency in patients of three different ethnic origins suffering from pulmonary tuberculosis. J. med. Genet., 6, 292.

Saha, N. and Banerjee, B.

(1971) Incidence of erythrocyte glucose-6-phosphate dehydrogenase deficiency among different ethnic groups of India. Hum. Hered., 21, 78.

Sanger, R. and Race, R.R.

(1947) Sub-divisions of MN blood groups in man Nature, Lond., 160, 505.

Sanger, R., Race, R.R. and Jack, J.A.

(1955) Unpublished data : cited in Race, R.R. and Sanger, R. (1970).

Sen, D.K.

(1960) Blood group and haemoglobin variants in some upper castes of Bengal. J. R. anthrop. Inst., 90, 161.

Seth, S.

(1968) The study of the A_1A_2BO blood group system and ABO (H) secretion in 6 endogamous groups of Punjabis. Amer. J. phys. Anthrop., 29, 387.

Seth, P.K., Seth, S., Rao, M.B. and Mani, S.B.

(1969) Genetical study of the Gujars : A_1A_2BO blood groups, P.T.C.; somatometry, mid-phalangeal hair, ear lobes, hand clasping. arm folding and leg folding. Hum. Hered., 19, 190.

Seth, S., Bansal, I.J.S. and Seth, P.K.

(1971) Studies on haptoglobin and transferrin types in Khatris and Aroras of Punjab, India. Humangenetik , 14, 44.

Shanklin, W.M.

(1935) Blood grouping of Rwala Bedouin. J. Immunol., 29, 427.

Shanklin, W.M.

(1936) Blood Grouping of Maulay and Akeydat Bedouin. Amer. J. phys. Anthrop., 21, 39.

Shih, L.Y., Hsia, D.Y.Y., Bowman, J.E., Shih, S.C. and Shih, P.L.

(1968) The electrophoretic phenotypes of red cell 6-phosphogluconate dehydrogenase and adenylate kinase in Chinese populations. Amer. J. hum. Genet., 20, 474.

Shih, L.Y. and Hsia, D.Y.Y.

(1969) The distribution of genetic polymorphism among Chinese and Taiwan. Hum. Hered., 19, 227.

Shinoda, T.

(1969) A note on the frequency of the red cell acid phosphatase types in Japan. Jap. J. hum. Genet., 13, 249.

Shivaraman, E.K., Saran, R.K. and Bhatia, H.M.

(1971) The distribution of ABO and Rh Blood groups in North Indian populations. Hum. Hered., 21, 326.

Simmons, R.T., Graydon, J.J., Semple, N.M. and Green, R.

(1950) The A_1A_2BO, MN and Rh blood groups in Southern Chinese : Hak-Kas, Cantonese and Hokkas. Med. J. Aust., ii, 917.

Singh, S., Sareen, K.N. and Goedde, H.W.

(1974a) Investigation of some biochemical genetic markers in four endogamous groups from Panjab (N.W. India). I. Protein and enzyme polymorphisms in serum. Humangenetik, 21, 341.

Singh, S., Sareen, K.N. and Goedde, H.W.

(1974b) Investigations of some biochemical genetic markers in four endogamous groups from Panjab (N.W. India). II. Red cell enzyme polymorphisms. Humangenetik, 22, 133.

Sinnett, P., Blake, N.M., Kirk, R.L., Walsh, R.J. and Lai, L.Y.C.

(1970) Blood serum protein and enzyme groups among the Enga speaking people of the Western Highlands, New Guinea, with an estimate of genetic distance between clans. Arch. phys. Anthrop. Oceania., 5, 236.

Smithies, O.

(1955a) Zone Electrophoresis in starch gel : group variations in the serum proteins of normal human adults. Biochem. J., 61, 629.

Smithies, O.

(1955b) Grouped variation in the occurience of new protein component in human serum. Nature, Lond., 175. 307.

Smithies, O.

(1957) Variations in human serum β-globulins. Nature, Lond., 180, 1482.

Smithies, O.

(1958) Third allele at the serum β-globulin locus in humans. Nature, Lond., 181, 1203.

Smithies, O.

(1959) Zone electrophoresis in starch gels and its application to studies of serum proteins, in Recent Advances in Protein Chemistry, Anfinsen, C.B., Anson, M.L., Bailey, K. and Edsall, J.T. (eds.), 14, 65, Academic Press, N.Y.

Smithies, O. and Walker, N.F.

(1955) Genetic control of some serum protein groups and the gene controlling their inheritance. Nature, Lond., 178, 694.

Smithies, O. and Walker, N.F.

(1956) Notation for serum protein groups and genes controlling their inheritance. Nature, Lond., 178, 694.

Smithies, O. and Hiller, O.

(1959) The genetic control of transferrin in humans. Biochem. J., 72, 121.

Smithies, O., Connell, G.E. and Dixon, G.H.

(1962) Inheritance of haptoglobin subtypes. Amer. J. hum. Genet., 14, 14.

Spencer, N., Hopkinson, D.A. and Harris, H.

(1964) Phosphoglucomutase polymorphism in man. Nature, Lond., 204, 742.

Srivastva, R.V.

(1965) Blood groups in the Tharus of Uttar Pradesh and their bearing on ethnic genetic relationships. Hum. Biol., 37, 1.

Sunderland, E.

(1973) Elements of human and social geography. Pergamon Press, Oxford.

Sunderland, E. and Smith, H.M.

(1966) The blood groups of the Shia in Yazd, Central Iran. Hum. Biol., 38, 50.

Sunderland, E., Sawhney, K.S., Cartwright, R. and Jolly, J.G.

(1975) Studies of haptoglobin and transferrin types in four castes of the Panjab, Northern India. Hum. Hered., (in press).

Szeinberg, A. and Tomashevsky-Tamir, S.

(1971) Red cell adenylate kinase and phosphoglucomutase polymorphisms in several population groups in Israel. Hum. Hered., 21, 289.

Talwar, C.L.

(1962) The Rh subgroups in Panjab (India). Indian J. med. Sci., 16, 717.

Thomsen, O., Friendenreich, V. and Worsaae, E.

(1930) Uber die moglichkeit der existenz zweier neuer Blutgruppen; auch eim Beitrag zur Belenchtung sogennanter untergruppen. Acta path. microbiol. scand., 7, 157. Cited in Race, R.R. and Sanger, R. (1970).

Thomson, G.R.

(1962) Significance of haemoglobins S and C in Ghana. Brit. med. J., 1, 682.

Thompson, P.R., Childers, D.M. and Hatcher, D.E.

(1967) Anti-Di^b : first and second examples. Vox Sang, 13, 314.

Tills, D.

(1969) Protein and enzyme polymorphisms in human populations with special reference to Middle East populations including blood groups. M. Phil. Thesis (C.N.A.A.).

Tills, D., Vanden Branden, J.L., Clements, V.R. and Mourant, A.E.

(1970a) The world distribution of electrophoretic variants of red cell adenylate kinase. Hum. Hered., 21, 517 and (1971) Hum. Hered., 21, 302.

Tills, D., Vanden Branden, J.L., Clements, V.R. and Mourant, A.E.

(1970b) The distribution in man of genetic variants of 6-phosphogluconate dehydrogenase. Hum. Hered., 20, 523 and (1971) Hum. Hered., 21, 305.

Tiwari, S.C.

(1952) The distribution of blood groups among the Bhotias of the Almora district Uttar Pradesh. Man in India.,32, 148.

Tiwari, S.C.

(1954) The blood groups of Kumaonis. Anthropologist, Delhi.,1, 50.

Tiwari, S.C.

(1960) Haptoglobins and transferrin variants in some upper castes of Bengal. Anthropologist, Delhi.,7, 17.

Tiwari, S.C.

(1961) The frequencies of some serum haptoglobin and transferrin types in a Panjabi population. Anthropologist, Delhi.,8, 23.

Tyagi, S.P.

(1968) Distribution of ABO blood groups in Uttar Pradesh. Ind. J. med. Ass., 51, 57.

Undevia, J.V., Blake, N.M., Kirk, R.L. and Medermid, E.M.

(1972) The distribution of some enzyme group systems among Parsis and Iranis in Bombay. Hum. Hered., 22, 274.

Undevia, J.V., Kirk, R.L. and McDermid, E.M.

(1973) Serum protein systems among Parsis and Iranis in Bombay. Hum. Hered., 23.

Vogel, F. and Chakravatti, M.R.

(1966) ABO blood groups and smallpox in a rural population of West Bengal and Bihar (India). Humangenetik, 3. 166.

Vyas, G.N., Bhatia, H.M., Banker, D.D. and Purandare, N.M.

(1958) Study of blood groups and other genetical characters in six Gujarati endogamous groups in Western India. Ann. hum. Genet., 22, 185.

Vyas, G.N., Bhatia, H.M., Sukumaran, P.K., Balakrishnan, V. and Sangvi, L.D.

(1962) Study of blood groups, abnormal haemoglobins and other genetical characters in some tribes of Gujrat. Amer. J. phys. Anthrop., 20, 255.

Walsh, R.J. and Montgomery, C.

(1947) A new human isogglutin sub-dividing the MN blood groups. Nature, Lond., 160, 504.

Walter, H. and Djahanschahi, I.

(1963) Zur Haufugkeit der Serum - gruppen in Persien. Homo., 14, 70.

Walter, H., Bernhard, W., Tahir Hassan, S. and Bajatzadeh, M.

(1966) Untersuchungen uber die Verteilung der Hp-, Gc- und Gm-Gruppen in Pakistan. Humangenetik, 2, 262.

Walter, H. and Bajatzadeh, M.

(1968) Studies on the distribution of the human red cell acid phosphatase in Iranians and other populations. Acta. genet., Basel, 18, 421.

Walter, H. and Steegmuller, H.

(1969) Studies on the geographical and racial distribution of haptoglobin and Gc polymorphisms. Hum. Hered., 19, 209.

Walter, H. and Bajatzadeh, M.

(1971) Investigations on the geographical variability of the human transferrins. Humangenetik, 12, 267.

Walter, H., Kellermann, G., Bajatzadeh, M., Kruger, J. and Chakravartti, M.R.

(1972) Hp. Gc. Cp. Tf. Bg. and Pi phenotypes in leprosy patients and healthy controls from West Bengal (India). Humangenetik, 14, 314.

Weiner, A.S. and Wexler, I.B.

(1963) An Rh-Hr syllabus. Grunne and Stratton, New York.

World Health Organization (WHO)

(1966) Haemoglobinopathies and allied disorders. Wld. Hlth. Org. techn. Rep. Ser., No. 338, 5.

Wyslouchowa, B.

(1970) Red cell acid phosphatase types in Poland : Population and genetic studies. Hum. Hered., 20, 199.

Younovitch, Rina.

(1933) Etude, Serologique des Juifs Samaritans. C.R. Soc. Biol., Paris., 112,970.

TABLE 3.1 DISTRIBUTION OF ABO BLOOD GROUPS - KUWAITI ARABS

Tested with anti-A, anti-B, anti-A+B and anti-A_1 sera

Phenotype	Obs. No.	Freq. obs.	Exp. No.	Freq. Exp.
A_1	40	.2469	39.22	.2421
A_2	5	.0309	4.91	.0303
B	32	.1975	31.36	.1936
O	81	.5000	79.40	.4901
A_1B	2	.0123	6.24	.0385
A_2B	2	.0123	.87	.0054
Total	162	.9999	162.00	1.0000

Gene Frequencies

P_1	.1518
P_2	.0213
q	.1268
r	.7001

TABLE 3.2 DISTRIBUTION OF MNSs BLOOD GROUPS - KUWAITI ARABS

Tested with 3 antisera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
MMS	29	.1824	34.84	2191
MMss	23	.1447	23.13	.1455
MNS	43	.2704	34.19	.2150
MNss	45	.2830	41.88	.2634
NNS	3	.0189	6.01	.0378
NNss	16	.1006	18.95	.1192
Total	159	1.0000	159.00	1.0000

Gene Frequencies

MS	.222[illegible]
Ms	.3814
NS	.0509
Ns	.3453

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
MM	52	.3270	57.97	.3646
MN	88	.5535	76.07	.4784
NN	19	.1195	24.96	.1570
Total	159	1.0000	159.00	1.0000

Gene Frequencies

M	.6038
N	.3962

TABLE 3.3 DISTRIBUTION OF Rh BLOOD GROUPS - KUWAITI ARABS

Tested with anti-D, -C, -c, -E and -e sera

Rh Type	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
R_zR_z	-	-	.06	.0005
R_1R_z	1	.0091	2.34	.0213
R_1R_1	27	.2455	26.62	.2420
R_2R_z	1	.0091	.35	.0032
R_1R_2	14	.1273	9.96	.0905
R_1r	42	.3818	44.65	.4059
r"r	1	.0091	1.67	.0152
R_2R_2	-	-	.54	.0049
R_2r	3	.0273	5.03	.0457
r"r"	-	-	.06	.0005
R_or	8	.0727	7.13	.0648
rr	13	.1182	11.59	.1054
Total	110	1.0000	110.00	1.0000

Gene Complex	Frequencies
R_z	.0217
R_1	.4920
R_2	.0504
r"	.0234
R_o	.0880
r	.3246

TABLE 3.4 DISTRIBUTION OF KELL AND DUFFY BLOOD GROUPS - KUWAITI ARABS

Tested with anti-K serum only

Phenotype	Obs. No.	Freq. Obs.
K(+)	6	.0370
K(-)	156	.9630
Total	162	1.0000

Gene Frequencies

K	.0187
k	.9813

Tested with anti-Fy^a serum only

Phenotype	Obs. No.	Freq. Obs.
Fy(a+)	69	.4929
Fy(a-)	71	.5071
Total	140	1.0000

Gene Frequencies

Fy^a	.2879
$Fy^b + Fy$	.7121

TABLE 3.5 DISTRIBUTION OF ABO BLOOD GROUPS IN SELECTED POPULATIONS OF THE ARABIAN PENINSULA AND NEIGHBOURING ARAB STATES

(a) Tested with anti-A, anti-B and anti-AB Sera

Sample	Number Tested	Phenotypes								Gene Frequencies			Author(s)
		O		A		B		AB					
ARABIA		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	O	A	B	
Kuwaiti Arabs	162	81	.5000	45	.2778	32	.1975	4	.0247	.7001	.1731	.1268	Present Study
Najd Arabs	65	29	.4461	15	.2308	19	.2923	2	.0308	.6798	.1420	.1782	Moshkovski et al (1931)
Yemenite Arabs	158	88	.5570	51	.3228	17	.1076	2	.0126	.7524	.1854	.0622	,, ,, ,,
Kuwaiti Arabs	2632	1244	.4726	638	.2424	635	.2413	115	.0437	.6906	.1552	.1542	Onsi and El-Alfi (1968)
Palestinians (Kuwait)	4163	1383	.3322	1641	.3942	802	.1926	337	.0810	.5766	.2753	.1481	,, ,, ,,
South Western Arabia	2203	1264	.5738	678	.3078	220	.0999	41	.0186	.7594	.1794	.0612	Marengo-Rowe et al (1974)
Zabidi Arabs	113	61	.5398	37	.3274	13	.1150	2	.0177	.7524	.1854	.0622	,, ,, ,, ,,
IRAQ													
Arabs	493	174	.3529	150	.3043	133	.2698	36	.0730	.5987	.2115	.1898	Kennedy and Macfarlane (1936)
Kurds	1500	531	.3540	449	.2993	293	.1953	227	.1513	.5598	.2529	.1873	,, ,, ,, ,,
Bedouin (Baghdad	338	138	.4083	90	.2663	87	.2574	23	.0680	.6368	.1843	.1789	Kayassi et al (1938)
Moslems	386	130	.3368	121	.3135	109	.2824	26	.0673	.5906	.2145	.1949	Boyd and Boyd (1941)
Arabs	123	48	.3902	51	.4146	18	.1463	6	.0488	.6288	.2681	.1031	Field (1957)
Iraqis	313	127	.4058	89	.2843	81	.2588	16	.0511	.6444	.1858	.1698	Onsi and El-Alfi (1968)

TABLE 3.5 (Contd.)

Sample	Number Tested	O		A		B		AB					Author(s)
JORDAN		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	O	A	B	
Arabs and Syrians (Nablus)	347	112	.3228	138	.3977	71	.2046	26	.0749	.5736	.2747	.1517	Younovitch (1933)
SYRIA													
Arabs	933	333	.3569	345	.3698	194	.2079	61	.0654	.6027	.2493	.1480	Altounyan (1928)
Rwala Bedouin	77	67	.8701	7	.0909	2	.0260	1	.0130				Shanklin (1935)
Akeydat Bedouin	159	119	.7484	27	.1698	9	.0566	4	.0252	.8561	.1023	.0416	Shanklin (1936)
Maulay Bedouin	213	190	.8920	16	.0751	7	.0329	-	-	.9451	.0383	.0166	,, ,,
Bedouin	304	121	.3980	71	.2336	92	.3026	20	.0658	.6315	.1631	.2054	Boyd and Boyd (1938)
Syrians	505	168	.3328	199	.3941	98	.1940	40	.0792	.5742	.2768	.1490	Onsi and El-Alfi (1968)
LEBANON													
Moslems	1777	622	.3500	650	.3658	340	.1913	165	.0929	.5839	.2630	.1531	Parr (1931)
Lebanese	496	169	.3407	196	.3952	86	.1734	45	.0907	.5734	.2841	.1425	Onsi and El-Alfi (1968)

TABLE 3.5 (b) Tested with anti-A, anti-B, anti-A+B and anti-A_1 Sera

Sample	Number Tested	Phenotypes												Gene Frequencies				Author(s)
		A_1		A_2		B		O		A_1B		A_2B		A_1	A_2	B	O	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.					
Kuwaiti Arabs	162	40	.2469	5	.0309	32	.1975	81	.5000	2	.0123	2	.0123	.1518	.0213	.1268	.7001	Present study
Arabs (Aden)	111	19	.1712	10	.0901	12	.1081	66	.5946	2	.0180	2	.0180	.0995	.0619	.0746	.7641	Lehman & Ikin (1953)
Yemenis	110	19	.1727	10	.0909	12	.1091	65	.5909	2	.0182	2	.0182	.1001	.0626	.0753	.7620	Ikin (1963)
Zabidis	114	17	.1491	12	.1053	19	.1667	65	.5702	1	.0088	-	-	.0828	.0596	.0925	.7651	,, ,,
Arabia, Socotra	99	15	.1515	10	.1010	6	.0606	66	.6667	1	.0101	1	.0101	.0842	.0627	.0412	.8120	,, ,,
Saudi Arabians	1384	203	.1467	100	.0723	277	.2001	760	.5491	23	.0166	21	.0152	.0853	.0491	.1207	.7419	Maranjian et al (1966)
Southern Arabia	261	43	.1647	38	.1456	28	.1073	148	.5070	4	.0153	-	-	.0902	.0890	.0636	.7572	Marengo-Rowe et a (1974)

TABLE 3.6 DISTRIBUTION OF MNS BLOOD GROUPS IN SELECTED POPULATIONS OF THE ARABIAN PENINSULA

Tested with anti-M, anti-N and anti-S Sera

Sample	Number Tested	Phenotypes										Gene Frequencies				Author(s)
		MSS		Mss		MNSS		MNss		NSS		Nss		MS	Ms	NS	Ns	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.					
Kuwaiti Arabs	159	29	.1824	23	.1447	43	.2704	45	.2830	3	.0189	16	.1006	.2224	.3814	.0509	.3453	Present study
Yemenis	110	40	.3636	23	.2091	23	.2091	15	.1364	3	.0273	6	.0545	.3127	.4328	.0549	.1997	Ikin (1963)
Zabidis	114	23	.2018	15	.2193	17	.1491	29	.2544	3	.0263	17	.1491	.1779	.4449	.0329	.3442	,, ,,
Shiah (E. Saudi Arabia)	463	123	.2657	65	.1404	109	.2354	99	.2138	27	.0583	40	.0864	.2556	.3751	.0807	.2887	Maranjian et al (1966)
Sunni ,,	323	92	.2848	59	.1827	75	.2322	59	.1827	8	.0248	30	.0929	.2826	.3923	.0405	.2846	,, ,, ,,
Sunni (Najd)	180	74	.4111	30	.1667	39	.2167	23	.1278	3	.0167	11	.0611	.3653	.3847	.0384	.2116	,, ,, ,,
Sunni (W. Saudi Arabia)	176	69	.3920	31	.1761	40	.2273	21	.1193	6	.0341	9	.0511	.3493	.3921	.0619	.1966	,, ,, ,,
Bedouin	178	73	.4101	36	.2022	44	.2472	15	.0843	5	.0281	5	.0281	.3670	.4111	.0721	.1498	,, ,, ,,
Saudi Arabians	1382	450	.3256	232	.1679	319	.2308	226	.1635	54	.0391	101	.0731	.2980	.3926	.0660	.2434	,, ,, ,,
Southern Arabia	26	108	.4138	37	.1418	62	.2375	30	.1149	13	.0498	11	.0421	.3658	.3660	.0876	.1806	Marengo-Rowe et al (1974)

TABLE 3.7 DISTRIBUTION OF Rh BLOOD GROUPS IN SELECTED POPULATIONS OF THE ARABIAN PENINSULA

Tested with anti-D, -C, -c. -E and -e Sera

	Present study		Yemenite Zabidis Lehman and Ikin (1953)				Shiah		Sunni (E.Arabia) Maranjian et al (1966)		Sunni (Najd)		Sunni (W.Arabia)		Bedouin		Hadhramaut Arabs		S.E.Arabia Marengo-Rowe et al (1974)		S.W.Arabia	
Rh Type	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
R_1R_z	1	.0091	-	-	-	-	-	-	-	-	-	-	2	.0114	-	-	1	.0097	-	-	-	-
R_1R_1	27	.2455	32	.2883	13	.1140	99	.2129	58	.1796	28	.1556	37	.2102	32	.1798	10	.0971	20	.2667	55	.2511
R_2R_z	1	.0091	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
R_1R_2	14	.1273	11	.0991	13	.1140	47	.1011	30	.0929	22	.1222	17	.0966	30	.1685	20	.1942	3	.0400	28	.1279
R_1r	42	.3881	43	.3874	37	.3246	188	.4043	110	.3406	66	.3667	60	.3409	54	.3033	40	.3883	38	.5067	73	.3333
r'r	-	-	-	-	-	-	2	.0043	3	.0093	3	.0167	1	.0057	-	-	-	-	-	-	3	.0137
r"r	1	.0091	-	-	-	-	-	-	-	-	-	-	1	.0057	-	-	-	-	-	-	-	-
R_2R_2	-	-	1	.0090	2	.0175	10	.0215	15	.0464	1	.0056	5	.0284	5	.0281	4	.0388	-	-	3	.0137
R_2r	3	.0273	7	.0631	13	.1140	43	.0925	48	.1486	21	.1167	16	.0909	19	.1067	9	.0874	8	.1067	14	.0639
R_o ı	8	.0727	15	.1351	31	.2719	60	.1291	39	.1208	23	.1278	21	.1193	21	.1180	10	.0970	4	.0533	21	.0959
rr	13	.1182	2	.0180	5	.0439	16	.0344	20	.0619	16	.0889	16	.0909	17	.0955	9	.0874	2	.0267	22	.1005
Total	110	1.0000	111	1.0000	114	.9999	465	1.0001	323	1.0001	180	1.0002	176	1.0000	178	.9999	103	.9999	75	1.0001	219	1.0000

Table 3.7 (cont).

Gene Complex Frequencies	Present Study	Yemenite Arabs	Zabidis Arabs	Shiah	Sunni (E.Arabia)	Sunni (Najd)	Sunni (W.Arabia)	Bedouin	Hadhramaut Arabs	S.E.Arabia	S.W.Arabi
R_z	.0217	-	-	-	-	-	.0117	-	.0154	-	-
R_1	.4920	.5315	.3333	.4564	.3833	.3805	4227	.4157	.3286	.5400	.4665
r'	-	-	-	.0113	.0176	.0279	.0089	-	-	-	.0220
R_2	.0504	.0902	.1316	.1183	.1672	.1250	.1124	.1657	.1690	.0733	.1096
r''	.0234	-	-	-	-	-	.0066	-	-	-	-
R_o	.0880	.2486	.3356	.224C	.1805	.1678	.1499	.1386	.1099	.1633	.1140
r	.3246	.1298	.1994	.1899	.2515	.2989	.2879	.2799	.3231	.2234	.2879

TABLE 3.8 DISTRIBUTION OF KELL BLOOD GROUPS IN THE ARABIAN PENINSULA

Tested with anti-K Serum

Sample	Number Tested	Phenotypes				Gene Frequencies		Author(s)
		K(+)		K(-)		K	k	
		No.	Freq.	No.	Freq.			
Kuwaiti Arabs	162	6	.0370	156	.9630	.0187	.9813	Present study
Shi ah (S. Arabia)	465	29	.0624	436	.9376	.0317	.9683	Maranjian et al (1966)
Sunni (E. Saudi Arabia)	318	36	.1132	282	.8868	.0583	.9417	,, ,, ,,
Sunni (Najd)	180	35	.1944	145	.8056	.1025	.8975	,, ,, ,,
Sunni (W. Saudi Arabia)	174	26	.1494	148	.8506	.0777	.9223	,, ,, ,,
Bedouin (Saudi Arabia)	175	35	.2000	140	.8000	.1056	.8944	,, ,, ,,
Saudi Arabians	1374	170	.1237	1204	.8763	.0639	.9361	,, ,, ,,
Southern Arabia	243	23	.0947	220	.9053	.0473	.9527	Marengo-Rowe et al (1974)

TABLE 3.9 DISTRIBUTION OF DUFFY BLOOD GROUPS IN THE ARABIAN PENINSULA

Tested with anti-Fy^a Serum

Sample	Number Tested	Phenotypes				Gene Frequencies		Author(s)
		Fy(a+)		Fy(a-)		Fy^a	Fy^b+Fy	
		No.	Freq.	No.	Freq.			
Kuwaiti Arabs	140	69	.4929	71	.5071	.2879	.7121	Present study
Shi ah (S. Arabia)	465	41	.0882	424	.9118	.0451	.9549	Maranjian et al (1966)
Sunni (E.Saudi Arabia)	319	88	.2759	231	.7241	.1491	.8509	,, ,, ,,
Sunni (Najd)	180	98	.5444	82	.4556	.3251	.6749	,, ,, ,,
Sunni (W.Saudi Arabia)	173	49	.2832	124	.7168	.1534	.8466	,, ,, ,,
Bedouin (S. Arabia)	175	80	.4571	95	.5429	.2632	.7368	,, ,, ,,
Saudi Arabians	1368	372	.2719	996	.7281	.1467	.8533	,, ,, ,,
Southern Arabia	243	48	.1975	195	.8025	.1038	.8962	Marengo-Rowe et al (1974)

TABLE 3.10 DISTRIBUTION OF SERUM HAPTOGLOBIN GROUPS - KUWAITI ARABS

Phenotype Hp	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
1-1	24	.1519	18.80	.1190
2-1	61	.3861	71.40	.4519
2-2	73	.4620	67.80	.4291
0-0	3	-	-	-
Total	158	1.0000	158.00	1.0000

Gene Frequencies

Hp^1 .3449

Hp^2 .6551

TABLE 3.11 DISTRIBUTION OF SERUM HAPTOGLOBIN GROUPS IN POPULATIONS OF THE ARABIAN PENINSULA AND NEIGHBOURING ARAB STATES

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		1-1		2-1		2-2		Hp^1	Hp^2	
		No.	Freq.	No.	Freq.	No.	Freq.			
Kuwaiti Arabs	158	24	.1519	61	.3861	73	.4620	.3449	.6551	Present study
Jordanian Arabs	30	2	.0667	13	.4333	15	.5000	.2833	.7167	Tills (1969)
Palestinian Arabs	81	12	.1482	33	.4074	36	.4444	.3519	.6481	,, ,,
South Arabian Arabs	231	50	.2165	108	.4675	73	.3160	.4502	.5498	,, ,,
Southern Arabia	238	51	.2143	110	.4622	77	.3235	.4454	.5546	Marengo-Rowe et al (1974)

TABLE 3.12 DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS - KUWAITI ARABS

Phenotype AP	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A	8	.0516	6.40	.0413
BA	46	.2968	48.96	.3159
B	95	.6129	93.67	.6043
CB	5	.0323	4.68	.0302
CA	1	.0064	1.22	.0079
C	-	-	.06	.0004
Total	155	1.0000	154.99	1.0000

Gene Frequencies

P^a	.2032
P^b	.7774
P^c	.0194

TABLE 3.13 DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 - KUWAITI ARABS

Phenotype PGM_1	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
1-1	77	.5238	72.88	.4958
2-1	53	.3605	61.25	.4167
2-2	17	.1156	12.86	.0875
Total	147	.9999	146.99	1.0000

Gene Frequencies

PGM_1^1 .7041

PGM_1^2 .2959

TABLE 3.14 DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS - KUWAITI ARABS

Phenotype AK	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
1-1	150	.9434	150.13	.9442
2-1	9	.0566	8.74	.0550
2-2	-	-	.13	.0008
Total	159	1.0000	159.00	1.0000

Gene Frequencies

AK^1	.9717
AK^2	.0283

TABLE 3.15 DISTRIBUTION OF RED CELL 6-PHOSPHOGLUCONATE DEHYDROGENASE GROUPS - KUWAITI ARABS

Phenotype 6-PGD	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A	140	.9333	140.18	.9345
CA	10	.0667	9.66	.0644
C	-	-	.16	.0011
Total	150	1.0000	150.00	1.0000

Gene Frequencies

PGD^A .9667

PGD^C .0333

TABLE 3.16 LACTATE DEHYDROGENASE TYPES - KUWAITI ARABS

Sample	Number Tested	LDH Normal	LDH Variant	% Variant
Kuwaiti Arabs	150	150	-	-

TABLE 3.16 MALATE DEHYDROGENASE TYPES - KUWAITI ARABS

Sample	Number Tested	MDH Normal	MDH Variant	% Variant
Kuwaiti Arabs	150	150	-	-

TABLE 3.17 DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS IN POPULATIONS OF THE ARABIAN PENINSULA AND NEIGHBOURING ARAB STATES

Sample	Number Tested	Phenotypes												Gene Frequencies			Author(s)
		A		BA		B		CA		CB		C		P^a	P^b	P^c	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Kuwaiti Arabs	155	8	.0516	46	.2968	95	.6129	1	.0064	5	.0323	-	-	.2032	.7774	.0194	Present study
Jordanian Arabs	31	1	.0323	7	.2258	22	.7097	-	-	1	.0322	-	-	.1452	.8387	.0161	Tills (1969)
Palestinian Arabs	87	5	.0575	24	.2759	46	.5287	1	.0115	11	.1264	-	-	.2011	.7299	.0690	,, ,,
South Arabian Arabs	255	7	.0275	67	.2627	181	.7098	-	-	-	-	-	-	.1588	.8412	-	,, ,,
Southern Arabia	261	7	.0268	69	.2644	185	.7088	-	-	-	-	-	-	.1590	.8410	-	Marengo-Rowe et al (1974)

TABLE 3.18 DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 IN POPULATIONS OF THE ARABIAN PENINSULA AND NEIGHBOURING ARAB STATES

Sample	Number Tested	Phenotypes									Gene Frequencies			Author(s)
		1-1		2-1		2-2		7-1			PGM_1^1	PGM_1^2	PGM_1^7	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.					
Kuwaiti Arabs	147	77	.5238	53	.3605	17	.1156	-	-		.7041	.2959	-	Present study
Jordanian Arabs	31	17	.5288	11	.3547	3	.0968	-	-		.7258	.2742	-	Tills (1969)
Palestinian Arabs	87	49	.5632	33	.3793	5	.0575	-	-		.7529	.2471	-	,, ,,
South Arabian Arabs	255	157	.6157	85	.3333	12	.0471	1	.0039		.7843	.2137	.0020	,, ,,
Arabs (Israel)	203	103	.5074	78	.3842	22	.1084	-	-		.6995	.3005	-	Szeinberg and Tomashevsky (1971)
Southern Arabia	261	160	.6130	86	.3295	14	.0536	1	.0038		.7797	.2184	.0019	Marengo-Rowe et al (1974)

TABLE 3.19 DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS IN POPULATIONS OF THE ARABIAN PENINSULA AND NEIGHBOURING ARAB STATES

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		1-1		2-1		2-2		AK^1	AK^2	
		No.	Freq.	No.	Freq.	No.	Freq.			
Kuwaiti Arabs	159	150	.9434	9	.0566	-	-	.9717	.0283	Present study
Jordanian Arabs	31	29	.9355	2	.0645	-	-	.9677	.0323	Tills (1970a)
Palestinian Arabs	86	80	.9302	6	.0698	-	-	.9651	.0349	,, ,,
South Arabian Arabs	258	245	.9496	13	.0504	-	-	.9748	.0252	,, ,,
Arabs (Israel)	262	192	.9505	10	.0495	-	-	.9753	.0247	Szeinberg and Tomashevsky (1971)
Southern Arabia	261	249	.9540	12	.0460	-	-	.9770	.0230	Marengo-Rowe et al (1974)

TABLE 3.20 DISTRIBUTION OF RED CELL 6-PHOSPHOGLUCONATE DEHYDROGENASE GROUPS IN POPULATIONS OF THE ARABIAN PENINSULA AND NEIGHBOURING ARAB STATES

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		AA		CA		CC		PGD^A	PGD^C	
		No.	Freq.	No.	Freq.	No.	Freq.			
Kuwaiti Arabs	150	140	.9333	10	.0667	-	-	.9667	.0333	Present study
Jordanian Arabs	31	28	.9032	2	.0645	1	.0323	.9355	.0645	Tills (1970b)
Palestinian Arabs	87	74	.8506	13	.1494	-	-	.9253	.0747	,, ,,
South Arabians	255	201	.7882	52	.2039	2	.0078	.8902	.1098	,, ,,
Southern Arabia	261	204	.7816	54	.2069	3	.0115	.8851	.1149	Marengo-Rowe et al (1974)

TABLE 4.1 DISTRIBUTION OF ABO BLOOD GROUPS - IRAN

(a) Tested with anti-A, anti-B, anti-A+B and anti-A_1 Sera

	TEHRAN IRANIANS				ISFAHAN IRANIANS				TEHRAN AND ISFAHAN IRANIANS			
Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A_1	21	.2234	21.23	.2259	25	.2252	26.14	.2355	46	.2244	47.36	.2310
A_2	5	.0532	5.07	.0539	4	.0360	4.18	.0377	9	.0439	9.27	.0452
B	17	.1808	17.21	.1831	25	.2252	26.13	.2354	42	.2049	43.28	.2111
O	46	.4894	46.51	.4948	46	.4144	48.09	.4332	92	.4488	94.73	.4621
A_1B	5	.0532	3.14	.0334	8	.0721	5.46	.0492	13	.0634	8.49	.0414
A_2B	-	-	.84	.0089	3	.0271	.99	.0089	3	.0146	1.87	.0091
Total	94	1.0000	94.00	1.0000	111	1.0000	110.99	.9999	205	1.0000	205.00	.9999

Gene Frequencies

p_1	.1394	.1542	.1470
p_2	.0373	.0281	.0325
q	.1199	.1595	.1407
r	.7034	.6582	.6798

(b) Tested with anti-A, anti-B and anti-AB Sera

TEHRAN IRANIANS

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A	23	.2323	23.15	.2338
B	26	.2626	26.17	.2643
O	44	.4444	44.27	.4472
AB	6	.0606	5.41	.0547
Total	99	.9999	99.00	1.0000

Gene Frequencies

p	.1565
q	.1748
r	.6687

TABLE 4.2 DISTRIBUTION OF MN BLOOD GROUPS - IRAN

Tested with anti-M and anti-N Sera

	TEHRAN IRANIANS				ISFAHAN IRANIANS				TEHRAN AND ISFAHAN IRANIANS			
Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
MM	24	.4138	22.97	.3960	56	.5045	52.04	.4688	80	.4734	74.88	.4431
MN	25	.4210	27.06	.4666	40	.3604	47.93	.4318	65	.3846	75.22	.4451
NN	9	.1552	7.97	.1374	15	.1351	11.03	.0994	24	.1420	18.89	.1118
Total	58	1.0000	58.00	1.0000	111	1.0000	111.00	1.0000	169	1.0000	168.99	1.0000

Gene Frequencies

M	.6293	.6847	.6657
N	.3707	.3153	.3343

TABLE 4.3 DISTRIBUTION OF MNSs BLOOD GROUPS - TEHRAN IRANIANS

Tested with anti-M, anti-N, anti-S and anti-s Sera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
MMSS	18	.1314	13.91	.1016
MMSs	19	.1387	30.17	.2202
MMss	23	.1679	16.36	.1194
MNSS	9	.0657	8.19	.0598
MNSs	32	.2336	30.02	.2191
MNss	21	.1533	22.91	.1672
NNSS	2	.0146	1.20	.0088
NNSs	4	.0292	6.22	.0454
NNss	9	.0657	8.02	.0586
Total	137	1.0001	137.00	1.0000

Gene Frequencies

MS	.3187
Ms	.3455
NS	.0938
Ns	.2420

TABLE 4.4

DISTRIBUTION OF Rh BLOOD GROUPS - IRAN

Tested with anti-D, -C, -c, -E, -e and C^W Sera

	TEHRAN IRANIANS				ISFAHAN IRANIANS				TEHRAN AND ISFAHAN IRANIANS			
Rh Type	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.
R_zR_z	-	-	.02	.0001	-	-	.08	.0007	-	-	.06	.0002
R_1R_z	-	-	1.80	.0092	1	.0091	2.93	.0266	1	.0033	4.65	.0152
$R_1^WR_z$	-	-	.08	.0004	-	-	.02	.0002	-	-	.12	.0004
R_1R_1	42	.2143	48.16	.2457	26	.2364	29.66	.2696	68	.2222	77.82	.2543
$R_1^WR_1$	3	.0153	3.96	.0202	1	.0091	.52	.0047	4	.0131	4.53	.0148
$R_1^WR_1^W$	-	-	.08	.0004	-	-	-	-	-	-	.06	.0002
r'r'	-	-	.04	.0002	-	-	.07	.0006	-	-	.09	.0003
R_2R_z	2	.0102	.47	.0024	2	.0182	.75	.0068	4	.0131	1.22	.0040
R_1R_2	35	.1786	26.71	.1363	25	.2273	16.92	.1538	60	.1961	43.73	.1429
$R_1^WR_2$	2	.0102	1.04	.0053	-	-	.13	.0012	2	.0065	1.19	.0039
R_1r	72	.3672	65.01	.3317	36	.3273	34.57	.3143	108	.3529	99.63	.3256
R_1^Wr	3	.0153	2.74	.0140	-	-	.32	.0029	3	.0098	3.00	.0098
r'r	2	.0102	1.74	.0089	2	.0182	1.63	.0148	4	.0131	3.43	.0112

TABLE 4.4 (Contd.)

	TEHRAN IRANIANS				ISFAHAN IRANIANS				TEHRAN AND ISFAHAN IRANIANS			
Rh Type	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.
r"r	-	-	-	-	1	.0091	1.57	.0143	1	.0033	1.77	.0058
r"r'	-	-	-	-	-	-	.12	.0011	-	-	.09	.0003
R_2R_2	3	.0153	3.37	.0172	-	-	1.90	.0173	3	.0098	5.29	.0173
R_2r	8	.0408	17.64	.0900	4	.0364	7.72	.0702	12	.0392	25.18	.0823
r"r"	-	-	-	-	-	-	.07	.0006	-	-	.03	.0001
R_or	2	.0102	1.92	.0098	1	.0091	.90	.0082	3	.0098	2.82	.0092
rr	22	.1122	21.19	.1081	11	.1000	10.12	.0920	33	.1078	31.24	.1021
Total	196	.9999	195.98	.9999	110	1.0000	110.00	.9999	306	1.0000	305.95	.9999

Gene Complex	Frequencies		
R_z	.0093	.0256	.0151
R_1	.4823	.4954	.4871
R_1^W	.0204	.0045	.0147
r'	.0136	.0244	.0175
R_2	.1310	.1099	.1229
r"	-	.0235	.0090
R_o	.0146	.0132	.0141
r	.3289	.3033	.3196

TABLE 4.5 DISTRIBUTION OF KELL BLOOD GROUPS - IRAN

Tested with anti-K Serum only

	TEHRAN IRANIANS		ISFAHAN IRANIANS		TEHRAN AND ISFAHAN IRANIANS	
Phenotype	Obs. No.	Freq. Obs.	Obs. No.	Freq. Obs.	Obs. No.	Freq. Obs.
K(+)	6	.0308	7	.0625	13	.0423
K(-)	189	.9692	105	.9375	294	.9577
Total	195	1.0000	112	1.0000	307	1.0000

Gene Frequencies

K	.0155	.0318	.0214
k	.9845	.9682	.9786

TABLE 4.6 DISTRIBUTION OF KIDD AND LUTHERAN BLOOD GROUPS - IRAN

(a) Tested with anti-JK^a Serum only

Phenotype	Obs. No.	Freq. Obs.	Gene Frequencies
JK(a+)	36	.7347	JK^a = .4849
JK(a-)	13	.2653	JK^b = .5151
Total	49	1.0000	

(b) Tested with anti-Lu^a Serum only

Phenotype	Obs. No.	Freq. Obs.	Gene Frequencies
Lu(a+)	10	.1316	Lu^a = .0681
Lu(a-)	66	.8684	Lu^b = .9319
Total	76	1.0000	

TABLE 4.7a

DISTRIBUTION OF ABO BLOOD GROUPS IN IRAN

(a) Tested with anti-A, anti-B and anti-AB Sera

Sample	Number Tested	Phenotypes								Gene Frequencies			Author(s)
		O		A		B		AB		O	A	B	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Tehran Iranians	99	44	.4444	23	.2323	26	.2626	6	.0606	.6687	.1565	.1748	Present study
Persians (Tehran)	565	230	.4071	173	.3062	133	.2354	29	.0513	.6446	.1993	.1561	Motamed (1949)
Persians (Tehran)	40	23	.5750	7	.1750	8	.2000	2	.0500	.7675	.1090	.1235	Beckett (1950)
Persian Moslems (Kirman)	10000	3789	.3789	3327	.3327	2224	.2224	660	.0660	.6183	.2250	.1567	Azhir (1951)
Azerbaijan	157	54	.3439	61	.3885	31	.1975	11	.0701	.5905	.2649	.1446	Boue and Boue (1955)
Kurdistan	148	61	.4122	48	.3243	30	.2027	9	.0608	.6424	.2158	.1418	,, ,,
Tehran	3049	1063	.3486	1036	.3398	721	.2365	229	.0751	.5937	.2356	.1707	Boue and Boue (1956)
Gurgan Town	537	200	.3724	169	.3147	120	.2235	48	.0894	.6027	.2270	.1703	,, ,,
Gurgan Province	134	45	.3358	46	.3433	29	.2164	14	.1045	.5699	.2553	.1748	,, ,,
Turkish, Shasavan, Azerbaijan	247	68	.2753	89	.3603	62	.2510	28	.1134	.5232	.2742	.2026	,, ,,
Turkomans	374	95	.2540	101	.2701	134	.3583	44	.1176	.5058	.2178	.2764	,, ,,
Gilan Province	225	70	.3111	77	.3422	54	.2400	24	.1067	.5523	.2567	.1910	,, ,,
Hamadan Province	115	34	.2957	38	.3304	31	.2696	12	.1043	.5434	.2480	.2086	,, ,,
Yazdi Iranians	307	96	.3127	81	.2638	100	.3257	30	.0977	.5585	.2009	.2406	Sunderland and Smith (1966)

TABLE 4.7b (Contd.) (a) Tested with anti-A, anti-B, anti-A+B and anti-A_1 Sera

Sample	Number Tested	Phenotypes									
		A_1		A_2		B		O		A_1B		A_2B	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Tehran Iranians	94	21	.2234	5	.0532	17	.1808	46	.4894	5	.0532	-	-
Isfahan Iranians	111	25	.2252	4	.0360	25	.2252	46	.4144	8	.0721	3	.0271
Pooled Data	205	46	.2244	9	.0439	42	.2049	92	.4488	13	.0634	3	.0146
Tehran Iranians	1239	351	.2833	61	.0492	301	.2429	427	.3446	83	.0670	16	.0130
Yazdı Musılman	258	45	.1745	12	.0465	90	.3488	82	.3178	19	.0736	10	.0387
Irarıans	348	78	.224	13	.037	86	.247	148	.425	19	.055	4	.011
Armenıans	78	33	.423	5	.064	12	.154	24	.308	3	.038	1	.013
Assyrıans	32	12	.375	2	.063	2	.063	12	.375	3	.094	1	.031
Bachtıiarı	138	25	.181	3	.022	30	.217	79	.572	1	.007	-	-
Kurds	127	29	.228	12	.094	28	.220	47	.370	7	.055	4	.031
Arabs	158	24	.152	9	.057	51	.323	59	.373	11	.070	4	.025
Ghash Qays	66	19	.288	4	.061	14	.212	25	.379	4	.061	-	-
Yazdı Shıa's	151	28	.1882	12	.0767	48	.3179	49	.3245	10	.0642	4	.0285
Iranıans	565	168	.297	35	.062	114	.202	225	.398	17	.030	6	.011
Kurds													
(ı) Marıvan and Baneh	77	19	.2467	6	.0779	14	.1818	33	.4286	4	.0519	1	.0130
(ıı) Sanandaj and Bıja	107	23	.2149	10	.0935	25	.2336	38	.3551	7	.0654	4	.0374

TABLE 4.7b (Contd.) (a) Tested with anti-A, anti-B, anti-A+B and anti-A_1 Sera

Sample	Gene Frequencies				Author(s)
	A_1	A_2	B	O	
Tehran Iranians	.1394	.0373	.1199	.7034	Present Study
Isfahan Iranians	.1542	.0281	.1595	.6582	,, ,,
Pooled Data	.1470	.0325	.1407	.6798	,, ,,
Tehran Iranians	.1951	.0404	.1792	.5853	Boue and Boue (1956)
Yazdi Musilman	.1313	.0401	.2544	.5741	,, ,,
Iranians	.151	.029	.171	.650	Nijenhuis (1964)
Armenians	.269	.056	.110	.566	,, ,,
Assyrians	.270	.060	.100	.580	,, ,,
Bachtiiari	.100	.013	.120	.766	,, ,,
Kurds	.154	.077	.168	.601	,, ,,
Arabs	.117	.047	.235	.601	,, ,,
Ghash Qays	.192	.041	.148	.619	,, ,,
Yazdi Shia's	.1354	.0631	.2322	.5693	Sunderland and Smith (1966)
Iranians	.190	.048	.144	.618	Bajatzadeh and Walter (1969)
Kurds					
(i) Marivan and Baneh	.1621	.0558	.1317	.6504	Lehman et al (1973)
(ii) Sanandaj and Bija	.1511	.0801	.1841	.5847	,, ,,

TABLE 4.8a DISTRIBUTION OF MNSs BLOOD GROUPS IN IRAN

(a) Tested with anti-M and anti-N Sera

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		MM		MN		NN		M	N	
		No.	Freq.	No.	Freq.	No.	Freq.			
Tehran Iranians	58	24	.4138	25	.4310	9	.1552	.6293	.3707	Present study
Isfahan Iranians	111	56	.5045	40	.3604	15	.1351	.6847	.3153	,, ,,
Pooled Data	169	80	.4734	65	.3846	24	.1420	.6657	.3343	,, ,,
Iranians	348	128	.368	181	.520	39	.112	.628	.372	Nijenhuis (1964)
Armenians	78	27	.346	34	.436	17	.218	.564	.436	,, ,,
Assyrians	32	12	.375	16	.500	4	.125	.625	.375	,, ,,
Bachtiari	138	62	.449	60	.435	16	.116	.666	.334	,, ,,
Kurds	127	47	.370	58	.457	22	.173	.599	.401	,, ,,
Arabs	158	62	.392	77	.487	19	.120	.636	.364	,, ,,
Gnash Qays	66	27	.409	33	.500	6	.091	.659	.341	,, ,,
Iranians *	56	19	.3393	28	.5000	9	.1607	.5893	.4107	,, ,,
Iranians *	520	222	.4269	213	.4096	85	.1635	.6317	.3683	Bajatzadeh and Walter (1969)

* For comparative purposes the six phenotypes of these two samples were combined to get M, MN and N. Similarly, the gene complexes were combined to get M and N.

TABLE 4.8b (Contd.) Tested with anti-M, anti-N, anti-S and anti-s

Sample	Number Tested	Phenotypes								
		MSS	MSs	Mss	MNSS	MNSs	MNss	NSS	NSs	Nss
		No. Freq.	No. Freq.	No. Freq.	No.Freq.	No. Freq.	No. Freq.	No. Freq.	No. Freq.	No. Freq.
Tehran Iranians	137	18 .1314	19 .1387	23 .1679	9 .0657	32 .2336	21 .1533	2 .0146	4 .0292	9 .0657
Yazdi Shia	151	10 .0682	25 .1636	22 .1457	10 .0662	30 .1987	35 .2318	3 .0199	4 .0265	12 .0795
Kurds										
(i) Marivan and Baneh	77	11 .1428	13 .1688	9 .1169	7 .0909	10 .1299	15 .1948	2 .0260	3 .0390	7 .0909
(ii) Sanandaj and Bija	107	11 .1028	22 .2056	11 .1028	4 .0374	17 .1589	21 .1963	3 .0280	7 .0654	11 .1028

	Gene Frequencies				Author(s)
	MS	Ms	NS	Ns	
Tehran Iranians	.3187	.3455	.0938	.2420	Present study
Yazdi Shia	.2497	.3761	.0990	.2752	Sunderland and Smith (1966)
Kurds					
(i) Marivan and Baneh	.3179	.3185	.1106	.2530	Lehman et al (1973)
(ii) Sanandaj and Bija	.2806	.3269	.1026	.2899	,, ,, ,,

TABLE 4.9 DISTRIBUTION OF Rh BLOOD GROUPS IN SELECTED POPULATIONS OF IRAN

Tested with anti-D, -C, -c, -E, -e and with or without anti-C^W

	Iranians		Iranians		Bachtiiari		Kurds		Arabs		Ghash Qays		Yazdi Shia	
	Present study				Nijenhuis (1964)								Sunderland and Smith (1966)	
Rh Type	Obs. No.	Freq. Obs.	Obs. No.	Freq. Obs.	Obs. No.	Freq. Obs.	Obs. No.	Freq. Obs.	Obs. No.	Freq. Obs.	Obs. No.	Freq. Obs.	Obs. No.	Freq. Obs.
R_1R_z	1	.0033	2	.0057	3	.0217	-	-	-	-	1	.0152	-	-
R_1R_1	68	.2222	87	.2500	40	.2898	27	.2126	44	.2785	20	.3030	31	.2077
$R_1^WR_1$	4	.0131	2	.0057	1	.0072	1	.0079	-	-	-	-	-	-
r'r'	-	-	-	-	1	.0072	-	-	-	-	-	-	-	-
R_2R_z	4	.0131	-	-	1	.0072	-	-	-	-	-	-	1	.0070
R_1R_2	60	.1961	59	.1695	30	.2174	17	.1339	21	.1329	11	.1667	18	.1188
$R_1^WR_2$	2	.0065	-	-	-	-	-	-	-	-	-	-	-	-
R_1r	108	.3529	114	.3276	31	.2247	46	.3622	59	.3734	16	.2424	54	.3552
R_1^Wr	3	.0098	-	-	-	-	-	-	-	-	-	-	-	-
r'r	4	.0131	3	.0086	-	-	-	-	-	-	1	.0151	1	.0066
r"r	1	.0033	1	.0029	1	.0072	-	-	2	.0126	-	-	-	-
R_2R_2	3	.0098	7	.0201	2	.0145	1	.0079	6	.0380	3	.0455	2	.0132
R_2r	12	.0392	30	.0862	18	.1304	16	.1260	13	.0823	8	.1212	18	.1192
R_or	3	.0098	14	.0402	4	.0290	13	.0236	6	.0380	-	-	5	.0331
rr	33	.1078	29	.0833	6	.0435	16	.1260	7	.0443	6	.0909	21	.1391
	306	1.0000	348	.9998	138	1.0000	127	1.0000	158	.9999	66	1.0000	151	.9999

TABLE 4.9 (Contd.)

Gene Complex Frequencies							
R_z	.0151	.005	.021	-	-	.010	.028
R_{l_W}	.4871	.492	.501	.464	.531	.493	.415
R_l	.0147	.003	.004	.004	-	-	-
r'	.0175	.014	.028	-	-	.027	.008
R_2	.1229	.141	.167	.138	.125	.187	.111
r''	.0090	.006	.019	-	.030	-	-
R_o	.0141	.061	.055	.033	.085	-	.044
r	.3196	.278	.205	.361	.229	.283	.393

TABLE 4.10 DISTRIBUTION OF KELL BLOOD GROUPS IN IRAN

Tested with anti-K Serum

Sample	Number Tested	Phenotypes				Gene Frequencies		Author(s)
		K(+)		K(-)		K	k	
		No.	Freq.	No.	Freq.			
Tehran Iranians	195	6	.0308	189	.9692	.0155	.9845	Present study
Isfahan Iranians	112	7	.0625	105	.9375	.0318	.9682	,, ,,
Pooled Data	307	13	.0423	294	.9577	.0214	.9786	,, ,,
Iranians	302	19	.063	283	.937	.032	.968	Nijenhuis (1964)
Armenians	67	2	.030	65	.970	.016	.984	,, ,,
Bachtiari	138	6	.043	132	.957	.023	.977	,, ,,
Kurds	127	14	.110	113	.890	.057	.943	,, ,,
Arabs	153	11	.072	142	.928	.037	.963	,, ,,
Ghash Qays	66	4	.061	62	.939	.032	.968	,, ,,
Yazdi Shi'a	142	13	.0915	129	.9085	.0469	.9531	Sunderland and Smith (1966)
Iranians	507	33	.065	474	.935	.033	.967	Bajatzadeh and Walter (1969)

TABLE 4.11 DISTRIBUTION OF SERUM HAPTOGLOBIN GROUPS - IRAN

	TEHRAN IRANIANS				ISFAHAN IRANIANS				TEHRAN AND ISFAHAN IRANIANS			
Phenotype Hp	Obs. No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.
1-1	19	.1022	15.97	.0858	6	.0674	8.20	.0921	25	.0909	24.15	.0878
2-1	71	.3817	77.06	.4143	42	.4719	37.62	.4227	113	.4109	114.70	.4171
2-2	96	.5161	92.96	.4998	41	.4607	43.18	.4852	137	.4982	136.15	.4951
0-0	-	-	-	-	2	-	-	-	2	-	-	-
Total	186	1.0000	185.99	.9999	89	1.0000	89.00	1.0000	275	1.0000	275.00	1.0000

Gene Frequencies

Hp^1	.2930	.3034	.2964
Hp^2	.7070	.6966	.7036

TABLE 4.12 DISTRIBUTION OF SERUM TRANSFERRIN GROUPS - IRAN

(a) ISFAHAN IRANIANS

Phenotype Tf	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
CC	88	.9888	88.00	.9888
CD	1	.0112	.99	.0111
DD	—	—	—	—
Total	89	1.0000	88.99	.9999

Gene Frequencies

Tf^C .9944

Tf^D .0056

(b) All the 186 Tehran Iranians showed the common transferrin type CC

TABLE 4.13 Hp^1 GENE FREQUENCIES IN IRAN

Sample	Total Number	Hp^1 Frequency	Author(s)
Tehran Iranians	186	.2930	Present study
Isfahan Iranians	89	.3034	,, ,,
Pooled Data	275	.2964	,, ,,
Shiras	97	.356	Walter and Djahanschahi (1963)
Iranians			
Tehran	305	.319	Bajatzadeh and Walter (1968)
North Iran	95	.358	,, ,, ,,
North West Iran	169	.272	,, ,, ,,
West Iran	210	.305	,, ,, ,,
Central and South Iran	138	.286	,, ,, ,,
East Iran	103	.291	,, ,, ,,
Iranians			
Tehran	400	.324	Bajatzadeh and Walter (1969)
North	179	.310	,, ,, ,,
North West	250	.276	,, ,, ,,
West	313	.282	,, ,, ,,
Central and South	245	.263	,, ,, ,,
East	179	.249	,, ,, ,,
Total Iranians	1566	.288	,, ,, ,,
Iranis (Bombay)	113	.2157	Undevia et al (1973)
Kurds (Baneh)	72	.3267	Lehman et al (1973)
Kurds (Bija)	105	.2667	,, ,,

TABLE 4.14 DISTRIBUTION OF TRANSFERRIN VARIANTS IN SELECTED POPULATIONS OF THE MIDDLE EAST

Sample	Phenotype Variants			Author(s)
Iranians	CC	CB	CD	
Iranians	88	-	1	Present study
Cypriots	197	-	-	Plato et al (1964)
Samaritan Isolate	125	-	-	Bonne (1966)
South Arabian Arabs	252	-	2	Tills (1969)
Jordanian Arabs	31	-	-	,, ,,
Palestinian Arabs	88	-	-	,, ,,
Sinai Bedouin	164	-	-	,, ,,
Jebeliya	74	-	-	,, ,,
Yemenite Jews	103	1	-	,, ,,
Samaritan Nablus	39	-	-	,, ,,
Habbanite Jews	595	3	-	Bonne et al (1970)
Southern Arabia	260	-	1	Marengo-Rowe et al (1974)

TABLE 4.15 DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS - IRAN

	TEHRAN IRANIANS				ISFAHAN IRANIANS				TEHRAN AND ISFAHAN IRANIANS			
Phenotype AP	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.
A	21	.1304	18.79	.1167	6	.0779	5.73	.0744	27	.1134	24.28	.1020
BA	68	.4224	70.04	.4350	30	.3896	30.00	.3896	98	.4118	100.58	.4226
B	65	.4037	65.27	.4054	39	.5065	39.28	.5102	104	.4370	104.24	.4380
CB	7	.0435	4.44	.0276	2	.0260	1.43	.0186	9	.0378	5.95	.0250
CA	-	-	2.38	.0148	-	-	.55	.0071	-	-	2.88	.0121
C	-	-	.08	.0005	-	-	.02	.0002	-	-	.07	.0003
Total	161	1.0000	161.00	1.0000	77	1.0000	77.01	1.0001	238	1.0000	238.00	1.0000

Gene Frequencies

P^a	.3416	.2727	.3193
P^b	.6367	.7143	.6618
P^c	.0217	.0130	.0189

TABLE 4.16 DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 GROUPS - IRAN

	TEHRAN IRANIANS				ISFAHAN IRANIANS				TEHRAN AND ISFAHAN IRANIANS			
Phenotype PGM	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.
1-1	101	.6121	96.21	.5831	50	.5814	48.38	.5625	151	.6016	144.60	.5761
2-1	50	.3030	59.57	.3610	28	.3256	31.50	.3663	78	.3107	91.06	.3628
2-2	14	.0848	9.22	.0559	7	.0814	5.13	.0596	21	.0837	14.33	.0571
6-1	-	-	-	-	1	.0116	.75	.0087	1	.0040	.75	.0030
6-2	-	-	-	-	-	-	.24	.0028	-	-	.25	.0010
6-6	-	-	-	-	-	-	-	-	-	-	-	-
Total	165	.9999	165.00	1.0000	86	1.0000	86.00	.9999	251	1.0000	250.99	1.0000

Gene Frequencies

PGM_1^1	.7636	.7500	.7590
PGM_1^2	.2364	.2442	.2390
PGM_1^6	-	.0058	.0020

TABLE 4.17 DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS - IRAN

	TEHRAN IRANIANS				ISFAHAN IRANIANS				TEHRAN AND ISFAHAN IRANIANS			
Phenotype AK	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.
1-1	150	.8929	150.48	.8957	76	.9157	76.14	.9174	226	.9004	226.63	.9029
2-1	18	.1071	17.03	.1014	7	.0843	6.71	.0808	25	.0996	23.74	.0946
2-2	-	-	.49	.0029	-	-	.15	.0018	-	-	.63	.0025
Total	168	1.0000	168.00	1.0000	83	1.0000	83.00	1.0000	251	1.0000	251.00	1.0000

Gene Frequencies

AK^1	.9464	.9578	.9502
AK^2	.0536	.0422	.0498

TABLE 4.18 DISTRIBUTION OF RED CELL 6-PHOSPHOGLUCONATE DEHYDROGENASE GROUPS - IRAN

	TEHRAN IRANIANS				ISFAHAN IRANIANS				TEHRAN AND ISFAHAN IRANIANS			
Phenotype-6PGD	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.	Obs.No.	Freq.Obs.	Exp.No.	Freq.Exp.
A	157	.9515	157.11	.9522	84	.9438	84.07	.9446	241	.9488	241.17	.9495
CA	8	.0485	7.79	.0472	5	.0562	4.86	.0546	13	.0512	12.67	.0499
C	-	-	.10	.0006	-	-	.07	.0008	-	-	.15	.0006
Total	165	1.0000	165.00	1.0000	89	1.0000	89.00	1.0000	254	1.0000	253.99	1.0000

Gene Frequencies

PGD^A	.9758	.9719	.9744
PGD^C	.0242	.0281	.0256

TABLE 4.19 LACTATE DEHYDROGENASE TYPES IN IRAN

Population	Numbers Tested	Phenotype		% Variant
		LDH Normal	LDH Variant	
Tehran Iranians	165	165	-	-
Isfahan Iranians	89	89	-	-

TABLE 4.19 MALATE DEHYDROGENASE TYPES IN IRAN

Population	Numbers Tested	Phenotype		% Variant
		MDH Normal	MDH Variant	
Tehran Iranians	165	165	-	-
Isfahan Iranians	89	89	-	-

TABLE 4.20 (a) DISTRIBUTION OF RED CELL PHOSPHOHEXOSE ISOMERASE - TEHRAN IRANIANS

Phenotype PHI	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
1-1	163	.9879	162.99	.9878
3-1	2	.0121	2.00	.0121
3-3	—	—	—	—
Total	165	1.0000	164.99	.9999

Gene Frequencies

PHI^1	.9939
PHI^3	.0061

(b) All the 88 Isfahan Iranians showed the normal type PHI 1-1

TABLE 4.21 DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS IN IRAN

Sample	Number Tested	Phenotypes												Gene Frequencies			Author(s)
		A		BA		B		CA		CB		C		P^a	P^b	P^c	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Tehran Iranians	161	21	.1304	68	.4224	65	.4037	-	-	7	.0435	-	-	.3416	.6367	.0217	Present study
Isfahan Iranians	77	6	.0779	30	.3896	39	.5065	-	-	2	.0260	-	-	.2727	.7143	.0130	,, ,,
Pooled Data	238	27	.1134	98	.4118	104	.4370	-	-	9	.0378	-	-	.3193	.6618	.0189	,, ,,
Iranians	449	61	.136	137	.305	223	.497	13	.029	15	.033	-	-	.304	.666	.030	Walter and Bajatzadeh (1968)
Iranians (Bombay)	48	2	.0417	18	.3750	26	.5416	-	-	2	.0417	-	-	.2292	.7500	.0208	Undevia et al (1972)
Parsis (Bombay)	418	76	.1818	186	.4450	156	.3732	-	-	-	-	-	-	.4043	.5957	-	,, ,, ,,
Kurds																	
(i) Marivan and Baneh	77	8	.1039	35	.4545	32	.4156	2	.0260	-	-	-	-	.3441	.6429	.0130	Lehman et al (1973)
(ii) Sanandaj and Bija	105	13	.1238	41	.3905	48	.4571	-	-	3	.0286	-	-	.3190	.6667	.0143	,, ,, ,,

TABLE 4.22 DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 GROUPS IN IRAN

Sample	Number Tested	Phenotypes								Gene Frequencies			Author(s)
		1-1		2-1		2-2		6-1		PGM_1^1	PGM_1^2	PGM_1^6	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Tehran Iranians	165	101	.6121	50	.3030	14	.0848	-	-	.7636	.2364	-	Present study
Isfahan Iranians	86	50	.5814	28	.3256	7	.0814	1	.0116	.7500	.2442	.0058	,, ,,
Pooled Data	251	151	.6016	78	.3107	21	.0837	1	.0040	.7590	.2390	.0020	,, ,,
Iranians (Bombay)	46	26	.5652	13	.2826	7	.1522	-	-	.7065	.2935	-	Undevia et al (1972)
Parsis ,,	401	219	.5461	153	.3815	29	.0722	-	-	.7369	.2631	-	,, ,, ,,
Iranians	127	61	.4803	52	.4094	14	.1102	-	-	.6850	.3150	-	Farhud et al (1973)
Kurds													
(i) Marivan and Baneh	77	45	.5844	30	.3896	2	.0260	-	-	.7792	.2208	-	Lehman et al (1973)
(ii) Sanandaj and Bija	105	48	.4571	48	.4571	9	.0857	-	-	.6857	.3143	-	,, ,, ,,

TABLE 4.23 DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS IN IRAN

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		1-1		2-1		2-2		AK^1	AK^2	
		No.	Freq.	No.	Freq.	No.	Freq.			
Tehran Iranians	168	150	.8929	18	.1071	-	-	.9464	.0536	Present study
Isfahan Iranians	83	76	.9157	7	.0843	-	-	.9578	.0422	,, ,,
Pooled Data	251	226	.9004	25	.0996	-	-	.9502	.0498	,, ,,
Moslems	322	290	.9006	32	.0994	-	-	.9503	.0497	Bowman and Ronaghy (1967)
Iranians (Bombay)	48	46	.9583	2	.0417	-	-	.9792	.0208	Undevia et al (1972)
Parsis ,,	418	363	.9684	54	.1292	1	.0024	.9330	.0670	,, ,, ,,
Kurds										
(i) Marivan and Baneh	77	69	.8961	7	.0909	1	.0130	.9416	.0584	Lehman et al (1973)
(ii) Sanandaj and Bija	105	90	.8571	14	.1333	1	.0095	.9238	.0762	,, ,, ,,

TABLE 4.24 DISTRIBUTION OF RED CELL 6-PHOSPHOGLUCONATE DEHYDROGENASE IN IRAN

Sample	Number Tested	Phenotypes								Gene Frequencies			Author(s)
		AA		CA		CC		HH		PGD^A	PGD^C	PGD^H	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Tehran Iranians	165	157	.9515	8	.0485	-	-	-	-	.9758	.0242	-	Present study
Isfahan Iranians	89	84	.9438	5	.0562	-	-	-	-	.9719	.0281	-	,, ,,
Pooled Data	254	241	.9488	13	.0512	-	-	-	-	.9744	.0256	-	,, ,,
Moslems	322	304	.9441	18	.0559	-	-	-	-	.9720	.0280	-	Bowman and Ronaghy (1967)
Iranians (Bombay)	48	48	1.0000	-	-	-	-	-	-	1.0000	-	-	Undevia et al (1972)
Parsis ,,	418	395	.9450	22	.0526	-	-	1	.0024	.9725	.0263	.0012	,, ,, ,,
Iranians	132	128	.9696	4	.0303	-	-	-	-	.9847	.0153	-	Farhud et al (1973)
Kurds													
(i) Marivan and Baneh	77	66	.8571	11	.1429	-	-	-	-	.9286	.0714	-	Lehman et al (1973)
(ii) Sanandaj and Bija	105	99	.9429	6	.0571	-	-	-	-	.9715	.0285	-	,, ,, ,,

TABLE 4.25 (a) DISTRIBUTION OF ABNORMAL HAEMOGLOBINS - TEHRAN IRANIANS

Haemoglobin type	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
AA	164	.9939	164.01	.9940
AD	1	.0061	.99	.0060
DD	-	-	-	-
Total	165	1.0000	165.00	1.0000

Gene Frequencies

Hb^A	.9970
Hb^D	.0030

(b) All the 89 Isfahan Iranians showed the normal haemoglobin type AA

TABLE 5.1 DISTRIBUTION OF ABO BLOOD GROUPS - PANJABIS (N. INDIA)

(a) Tested with anti-A, anti-B, anti-A+B and anti-A_1 Sera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A_1	58	.2042	58.87	.2073
A_2	2	.0070	2.04	.0072
B	103	.3627	104.54	.3681
O	93	.3275	94.43	.3325
A_1B	21	.0739	23.20	.0817
A_2B	7	.0246	.91	.0032
Total	284	.9999	283.99	1.0000

Gene Frequencies

p_1	.1568
p_2	.0062
q	.2604
r	.5766

(b) Tested with anti-A, anti-B and anti A+B Sera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A	16	.2162	15.18	.2051
B	35	.4730	33.20	.4486
O	17	.2297	16.12	.2179
AB	6	.0811	9.50	.1284
Total	74	1.0000	74.00	1.0000

Gene Frequencies

p	.1336
q	.3496
r	.4668

TABLE 5.2 DISTRIBUTION OF MNSs BLOOD GROUPS - PANJABIS (N. INDIA)

Tested with 3 antisera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
MMS	13	.1757	14.27	.1928
MMss	19	.2567	18.18	.2457
MNS	14	.1892	12.26	.1657
MNss	20	.2703	20.85	.2817
NNS	2	.0270	2.46	0333
NNss	6	.0811	5.97	.0807
Total	74	1.0000	73.99	.9999

Gene Frequencies

MS	.1665
Ms	.4957
NS	.0537
Ns	.2841

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
MM	32	.4324	32.45	.4385
MN	34	.4595	33.11	.4474
NN	8	.1081	8.44	.1141
Total	74	1.0000	74.00	1.0000

Gene Frequencies

M	.6622
N	.3378

TABLE 5.2 (Contd.) DISTRIBUTION OF MNSs BLOOD GROUPS - PANJABIS (N. INDIA)

Tested with 4 antisera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
MMSS	21	.0755	15.65	.0563
MMSs	56	.2014	59.32	.2134
MMss	53	.1906	56.27	.2024
MNSS	11	.0396	13.07	.0470
MNSs	49	.1762	52.99	.1906
MNss	62	.2230	53.49	.1924
NNSS	3	.0108	2.72	.0098
NNSs	12	.0432	11.79	.0424
NNss	11	.0396	12.70	.0457
Total	278	.9999	278.00	1.0000

Gene Frequencies

MS	.2372
Ms	.4499
NS	.0991
Ns	.2138

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
M	130	.4676	131.24	.4721
MN	122	.4388	119.54	.4300
N	26	.0935	27.22	.0979
Total	278	.9999	278.00	1.0000

Gene Frequencies

M	.6871
N	.3129

TABLE 5.3 DISTRIBUTION OF Rh BLOOD GROUPS - PANJABIS (N. INDIA)

Tested with anti-D, -C, -c, -E, -e and C^W Sera

Rh Type	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
R_1R_Z	-	-	1.85	.0052
$R_1^WR_Z$	-	-	.93	.0001
R_1R_1	126	.3539	134.03	.3765
$R_1^WR_1$	4	.0112	5.09	.0143
$R_1^WR_1^W$	-	-	.11	.0003
r'r'	-	-	.07	.0002
r'^Wr'	-	-	.07	.0002
R_2R_z	2	.0056	.32	.0009
R_1R_2	64	.1798	48.63	.1366
$R_1^WR_2$	2	.0056	1.32	.0037
R_1r	112	.3146	109.01	.3062
R_1^Wr	6	.0169	4.38	.0123
r'r	3	.0084	2.70	.0076
r'^Wr	-	-	.93	.0026
R_2R_2	1	.0028	4.27	.0120
R_2r	9	.0253	19.54	.0560

TABLE 5.3 (Contd.)

Rh Type	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
$R_o r$	2	.0056	1.74	.0049
rr	25	.0702	21.50	.0604
Total	356	1.0000	356.00	1.0000

TABLE 5.3 (Contd.) DISTRIBUTION OF Rh BLOOD GROUPS - PANJABIS (N. INDIA)

Tested with anti-D, -C, -c, -E, -e and C^W Sera

Gene Complex	Frequencies
R_z	.0042
R_1	.5985
R_1^W	.0116
r'	.0154
r'^W	.0052
R_2	.1095
R_o	.0098
r	.2457

TABLE 5.4 DISTRIBUTION OF KELL BLOOD GROUPS - PANJABIS (N. INDIA)

(a) Tested with anti-K serum

Phenotype	Obs. No.	Freq. Obs.	Gene Frequencies	
K(+)	10	.0278	K	.0140
K(-)	350	.9722	k	.9860
Total	360	1.0000		

(b) Tested with anti-Kp^b Serum

Phenotype	Obs. No.	Freq. Obs.	Gene Frequencies	
Kp(b+)	77	.8953	Kp^a	.3235
Kp(b-)	9	.1047	Kp^b	.6765
Total	86	1.0000		

TABLE 5.5 DISTRIBUTION OF DUFFY BLOOD GROUPS - PANJABIS (N. INDIA)

(a) Tested with anti-Fy^a and anti-Fy^b Sera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.	Gene Frequencies
Fy(a+b-)	57	.3931	57.62	.3974	Fy .0831
Fy(a+b+)	59	.4069	58.38	.4026	Fy^b .3642
Fy(a-b+)	28	.1931	28.00	.1931	Fy^a .5527
Fy(a-b-)	1	.0069	1.00	.0069	
Total	145	1.0000	145.00	1.0000	

(b) Tested with anti-Fy^a Serum

Phenotype	Obs. No.	Freq. Obs.	Gene Frequencies
Fy(a+)	156	.8125	Fy^a .5670
Fy(a-)	36	.1875	Fy^b .4330
Total	192	1.0000	

TABLE 5.6 DISTRIBUTION OF KIDD BLOOD GROUPS - PANJABIS (N. INDIA)

Tested with anti-JK^a serum only

Phenotype	Obs. No.	Freq. Obs.
JK a(+)	122	.7485
JK a(-)	41	.2515
Total	163	1.0000

Gene Frequencies

JK^a	.4985
JK^b	.5015

TABLE 5.7 DISTRIBUTION OF ABO BLOOD GROUPS IN SELECTED POPULATIONS OF NORTHERN INDIA

(a) Tested with anti-A, anti-B and anti-AB Sera

Sample	Number Tested	Phenotypes								Gene Frequencies			Author(s)
		O		A		B		AB		A	B	O	
PANJAB		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Panjabis	74	17	.2297	16	.2162	35	.4730	6	.0811	.1836	.3496	.4668	Present study
Panjab soldiers	262	63	.2405	41	.1565	121	.4618	37	.1412	.1602	.3658	.4740	Hirszfeld and Hirszfeld (1919)
Jats	277	92	.3322	68	.2455	98	.3538	19	.0685	.1728	.2414	.5858	Malone and Lahiri (1928-29)
Hindu soldiers	615	199	.3236	137	.2228	213	.3463	66	.1073	.1805	.2593	.5602	House and Mahalanobis (1939-45)
Sikhs	2278	805	.3534	575	.2524	697	.3060	201	.0882	.1876	.2212	.5912	,, ,, ,,
N. Indians	1478	507	.3430	369	.2497	482	.3261	120	.0812	.1822	.2304	.5874	Allen and Scott (1947)
Panjabis	10000	3064	.3064	2448	.2448	3478	.3478	1010	.1010	.1910	.2573	.5517	Khan (1952)
Sikhs	600	205	.3417	152	.2533	199	.3317	44	.0733	.1801	.2295	.5904	Bird et al (1956)
Khatris	1708	513	.3004	365	.2137	694	.4063	136	.0796	.1687	.2912	.5411	Anand (1957)
Aroras	1598	462	.2891	343	.2146	659	.4124	134	.0839	.1708	.2990	.5302	,, ,,
Comb.Panjabis	3306	975	.2949	708	.2142	1353	.4092	270	.0817	.1687	.2952	.5361	,, ,,
Panjab Hindus	1284	Figures unavailable								.1741	.2863	.5396	Bhalla (1963)
Aroras	422	125	.2962	86	.2038	172	.4075	39	.0924	.1697	.2911	.5392	Shivaraman et al (1971)
Brahmin	360	102	.2833	78	.2167	144	.4000	36	.1000	.1747	.2941	.5312	,, ,, ,,
Sikh	460	125	.2717	99	.2152	188	.4087	48	.1044	.1762	.3032	.5206	,, ,, ,,
Khatri	475	140	.2947	102	.2147	192	.4042	41	.0863	.1697	.2911	.5392	,, ,, ,,
Kholi	845	245	.2899	180	.2130	340	.4024	80	.0947	.1898	.2116	.5986	,, ,, ,,
Seighal	395	104	.2633	82	.2076	171	.4329	38	.0962	.1718	.3189	.5093	,, ,, ,,
Seth	210	59	.2809	47	.2238	83	.3952	21	.1000	.1799	.2915	.5286	,, ,, ,,
Kapoor	345	102	.2956	71	.2058	139	.4029	33	.0956	.1644	.2920	.5436	,, ,, ,,
RAJASTHAN													
Hindus	111	37	.3333	24	.2162	39	.3514	11	.0991	.1718	.2576	.5706	House and Mahalanobis (1939-45)
Hindus and Muslims	600	195	.3250	147	.2450	215	.3583	43	.0717	.1744	.2464	.5792	Goyal and Anand (1953)
Rajasthanis	14286	3230	.3301	5415	.2261	4714	.3791	927	.0647	.1580	.2545	.5746	Hurkat et al (1971)

TABLE 5.7 (Contd.)

Sample	Number Tested	Phenotypes								Gene Frequencies			Author(s)
		O		A		B		AB		A	B	O	
UTTAR PRADESH		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Chamars	150	55	.3667	28	.1867	59	.3933	8	.0533	.1288	.2574	.6138	Majumdar and Kishen (1947)
Bhoksa	144	44	.3056	28	.1944	52	.3611	20	.1389	.1808	.2886	.5306	,, ,, ,,
Kurmis	107	37	.3458	21	.1963	37	.3458	12	.1121	.1700	.2600	.5700	,, ,, ,,
Kayasthas	111	40	.3607	22	.1982	36	.3243	13	.1171	.1703	.2494	.5803	,, ,, ,,
Kshtriyas	415	128	.3084	111	.2675	136	.3277	40	.0964	.2025	.2412	.5563	,, ,, ,,
Khasa	246	75	.3049	74	.3008	69	.2805	28	.1138	.2339	.2207	.5454	,, ,, ,,
Uttar Pradesh	13105	4103	.3131	3072	.2344	4876	.3721	1054	.0804	.1729	.2611	.5660	Tyagi (1968)
Bania Group													
Aggarwal	244	81	.3320	54	.2213	91	.3729	18	.0738	.1665	.2615	.5720	Shivaraman et al (1971)
Gupta	310	102	.3290	68	.2194	115	.3710	25	.0806	.1663	.2621	.5716	,, ,, ,,
Kumaoni Group													
Rajput	360	101	.2806	83	.2305	144	.4000	32	.0889	.1834	.2923	.5243	,, ,, ,,
Thakur	285	80	.2807	66	.2316	110	.3860	29	.1017	.1855	.2860	.5285	,, ,, ,,
Other Groups													
Jain	165	54	.3273	39	.2364	57	.3454	15	.0909	.1738	.2485	.5727	,, ,, ,,
Jaswal	430	130	.3023	88	.2047	166	.3861	46	.1069	.1635	.2821	.5544	,, ,, ,,
Gujjar	300	92	.3067	63	.2100	110	.3667	35	.1166	.1674	.2707	.5619	,, ,, ,,
Jat	322	96	.2981	83	.2578	115	.3571	28	.0870	.1978	.2611	.5411	,, ,, ,,
Anir	270	77	.2852	65	.2407	99	.3667	29	.1074	.1915	.2737	.5348	,, ,, ,,
Brahmin	140	49	.3500	29	.2072	45	.3214	17	.1214	.1589	.2338	.6073	,, ,, ,,
CIS-HIMALAYAN REGION OF PANJAB AND H.P.													
Kanets (Simla)	196	Figures unavailable								.273	.291	.457	Delhi University (1957)
Brahmins (Kulu)	110	,,		,,						.2929	.2739	.4156	,, ,, (1958)
Brahmins (Chamba)	147	,,		,,						.229	.260	.511	,, ,, (1959)

TABLE 5.7 (Contd.)

Sample	Number Tested	Phenotypes							Gene Frequencies			Author(s)
		O		A		B		AB				
		No.	Freq.	No.	Freq.	No.	Freq.	No. Freq.				
Mahajans(Chamba)	112	Figures unavailable							.140	.234	.625	Delhi University (1959)
Kinner Kanets (Chinni)	310	,,				,,			.258	.162	.579	Bhalla (1961)
Rajputs (Rampur)	126	,,				,,			.113	.275	.541	Panjab University (1962)
CIS-HIMALAYAN REGION OF U.P.												
Upper Castes (Jaunsar-Bawar)	148	38	.2567	53	.3581	38	.2567	19 .1284	.2826	.2153	.5021	Banerjee and Kumar (1953)
Rajputs (Kumaon)	124	36	.2903	30	.2419	42	.3387	16 .1290	.2052	.2682	.5266	Tiwari (1954)
Bhotias(Almora)	144	26	.1805	22	.1528	73	.5069	23 .1597	.1692	.4187	.4121	,, ,,
Tharus	412	74	.1796	93	.2257	194	.4709	51 .1238	.1953	.3669	.4376	Srivastva (1965)

TABLE 5.7 (Contd.) (b) Tested with anti-A, anti-B, anti-A+B and anti-A_1 Sera

Sample	Number Tested	Phenotypes														Author(s)
		A_1		A_2		B		O		A_1B		A_2B		Gene Frequencies				
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	A_1	A_2	B	O	
Panjabis	284	58	.1737	2	.0070	103	.3627	93	.3275	21	.0739	7	.0246	.1568	.0062	.2604	.5766	Present study
Sikhs	213	37	.1533	11	.0516	79	.3650	69	.3239	12	.0563	5	.0234	.1229	.0442	.2596	.5734	Bird et al (1956)
Pandits and Moslems (Kashmir)	614	110	.1792	9	.0146	239	.3893	210	.3420	43	.0700	3	.0049	.1345	.0123	.2697	.5835	Bhattacharjee (1966)
Panjabis	5225	969	.1855	252	.0482	1947	.3726	1593	.3049	375	.0718	89	.0170	.1394	.0415	.2697	.5494	Seth (1968)
Gujars	68	17	.2500	4	.0588	21	.3088	23	.3382	1	.0147	2	.0294	.1454	.0540	.1994	.6012	Seth et al (1969)
Panjabis	137	21	.153	1	.007	50	.365	48	.350	12	.087	5	.037	.121	.006	.262	.611	Papiha et al (1972)

TABLE 5.8 DISTRIBUTION OF MNSs BLOOD GROUPS IN SELECTED POPULATIONS OF NORTHERN INDIA

(a) Tested with anti-M, anti-N and anti-S Sera

Sample	Number Tested	Phenotypes						Gene Frequencies				Author(s)
		MMSS	MMss	MNSS	MNss	NNSS	NNss	MS	Ms	NS	Ns	
		No. Freq.	No. Freq.	No. Freq.	No. Freq.	No. Freq.	No. Freq.					
Panjabis	74	13 .1757	19 .2567	14 .1892	20 .2703	2 .0270	6 .0811	.1665	.4957	.0537	.2841	Present study
Sikhs	213	50 .235	39 .183	51 .239	24 .207	8 .038	21 .099	.242	.399	.060	.299	Bird et al (1956)
Pandits and Muslims (Kashmir)	390	79 .2026	51 .1308	113 .2897	70 .1795	33 .0846	44 .1128	.2335	.3391	.1160	.3114	Bhattacharjee (1966

TABLE 5.8 (Contd.) (b) Tested with anti-M, anti-N, anti-S and anti-s Sera

Sample	Number Tested	MMSS		MMSs		MMss		MNSS		MNSs		MNss		NNSS		NNSs		NNss	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Panjabis	278	21	.0755	56	.2014	53	.1906	11	.0396	49	.1762	62	.2230	3	.0108	12	.0432	11	.0396
Panjabi Hindus	287	10	.0348	38	.1324	41	.1429	18	.0627	50	.1742	71	.2473	7	.0244	28	.0976	24	.0836
Panjabis	137	20	.146	22	.168	18	.131	14	.102	23	.168	19	.139	5	.036	6	.044	9	.066

Gene Frequencies				(Author(s)
MS	Ms	NS	Ns	
.2372	.4499	.0991	.2138	Present study
.1712	.3808	.1527	.2953	Bhalla (1963)
.335	.315	.140	.211	Papiha et al (1972)

TABLE 5.9 DISTRIBUTION OF Rh BLOOD GROUPS IN SELECTED POPULATIONS OF NORTHERN INDIA

Tested with anti-D. -C, -c, -E, -e and C^W Sera

	Present study		Sikh Bird et al (1956)		Hindus and Sikhs Talwar (1962)		P. Hindus Bhalla (1963)		U.P.Brahmins Shivaraman et al (1971)		Rajput		Thakkur		Panjabis Papiha et al (1972)	
Rh Type	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
R_zR_z	-	-	-	-	1	.0033	-	-	-	-	-	-	-	-	-	-
R_1R_z	-	-	-	-	4	.0133	-	-	-	-	3	.0323	2	.0182	-	-
R_1R_1	126	.3539	93	.435	105	.3500	403	.4112	22	.3667	29	.3118	32	2902	48	.350
$R_1^WR_1$	4	.0112	4	.020	-	-	-	-	-	-	-	-	-	-	-	-
R_2R_z	2	.0056	-	-	7	.0233	-	-	-	-	2	.0215	2	0182	-	-
R_1R_2	64	.1798	30	.141	39	.1300	116	.1184	8	.1333	18	.1935	17	.1545	14	.102
$R_1^WR_2$	2	.0056	-	-	-	-	-	-	-	-	-	-	-	-	-	-
R_1r	112	.3146	57	.268	97	.3233	312	.3184	20	.3333	25	.2683	35	.3182	57	.416
R_1^Wr	6	.0169	2	.009	-	-	-	-	-	-	-	-	-	-	-	-
r'r	3	.0084	1	.005	1	.0033	9	.0092	2	.0333	-	-	1	.0091	-	-
R_2R_2	1	.0028	5	.023	10	.0333	10	.0102	-	-	2	.0215	4	.0364	3	.022
R_2r	9	.0253	9	.042	10	.0333	51	.0520	4	.0667	4	.0430	6	.0545	6	.044
R_or	2	.0056	4	.019	6	.0200	19	.0194	-	-	2	.0215	1	.0091	3	.022
rr	25	.0702	8	.038	20	.0666	60	.0612	4	.0667	8	.0860	9	.0909	6	.044
Total	356	1.0000	213	.1000	300	.9997	980	1.0000	60	1.0000	93	1.0000	110	1.0000	137	1.000

Table 5.9 (cont).

Gene Complex Frequencies	Present Study	Sikh	Hindus and Sikhs	P. Hindus	U. P. Brahmins	Rajput	Thakkur	Panjabis
R_z	.0042	-	.011	.0156	-	.0291	.0163	-
R_1	.5985	.639	.573	.5994	.5514	.5569	.5279	.610
R_1^w	.0116	.015	-	- -	-	-	-	-
r'	.0154	.013	.00(	.0187	.0653	-	.0148	-
r'^w	.0052	-	-	-	-	-	-	-
R_2	.1095	.118	.097	.0898	.1000	.1484	.1519	.095
R_o	.0098	.040	.036	.0359	-	.0279	.0135	.054
r	.2457	.176	.258	.2406	.2833	.2377	.2756	.241

TABLE 5.10 DISTRIBUTION OF KELL BLOOD GROUPS IN SELECTED POPULATIONS OF INDIA

Tested with anti-K Serum only

Sample	Number Tested	Phenotypes				Gene Frequencies		Author(s)
		K(+)		K(-)		K	k	
		No.	Freq.	No.	Freq.			
Panjabis	360	10	.0278	350	.9722	.0140	.9860	Present study
Brahmin (E. India)	64	8	.1250	56	.8750	.0646	.9354	Sen (1960)
Kayastha ,,	95	7	.0737	88	.9263	.0375	.9625	,, ,,
Vaidya ,,	44	1	.0227	43	.9773	.0114	.9886	,, ,,
Panjabis (N. India)	137	1	.007	136	.993	.004	.996	Papiha et al (1972)
Hindus and Muslims (M.P.)	333	4	.012	329	.988	.006	.994	Roberts et al (1974)

TABLE 5.11a DISTRIBUTION OF DUFFY BLOOD GROUPS IN SELECTED POPULATIONS OF INDIA

(a) Tested with anti-Fy^a Serum

Sample	Number Tested	Phenotypes				Gene Frequencies		Author(s)
		Fy a(+)		Fy a(-)		Fy^a	Fy^b	
		No.	Freq.	No.	Freq.			
Panjabis	192	156	.8125	36	.1875	.5670	.4330	Present study
Asiatic Indians	55	51	.9273	4	.0727	.7304	.2696	Cutbush and Mollison (1950)
Irulas (S. India)	100	93	.9300	7	.0700	.7354	.2646	Lehman and Cutbush (1952)
Todas ,,	60	55	.9167	5	.0833	.7114	.2886	,, ,, ,,
Bhangi (W. India)	192	125	.651	67	.349	.4093	.5907	Vyas et al (1958)
Leva Patidars (W. India)	50	34	.680	16	.320	.4344	.5656	,, ,,
Kapul Vania ,,	173	119	.6679	54	.3321	.4409	.5591	,, ,,
Cutchi Lohana ,,	188	148	.7872	40	.2128	.5297	.4703	,, ,,
A. Brahmin	196	.38	.7041	58	.2959	.4560	.5440	,, ,,
Talavia Dubla ,,	198	130	.6566	58	.3434	.4140	.5860	,, ,,
Rajbanshi (E. India)	252	155	.615	97	.385	.380	.620	Das et al (1967)
Midnapur ,,	102	60	.588	42	.412	.358	.642	,, ,, ,,
Jalaiguri ,,	75	40	.533	35	.467	.317	.683	,, ,, ,,
Cooch Behar ,,	75	55	.733	20	.267	.484	.516	,, ,, ,,
Audich Brahmin (W. India)	100	64	.640	36	.360	.400	.600	Parikh et al (1969)
Lad Vania ,,	100	58	.580	42	.420	.368	.632	,, ,,
Visa Oswal Jain ,,	127	74	.583	53	.417	.354	.646	,, ,,
Panjabis (N. India)	137	98	.715	39	.285	.466	.534	Papiha et al (1972)
Mixed Muslims (W. India)	150	Figures unavailable				.434	.566	Hakim et al (1973)

TABLE 5.11b (Contd.) (b) Tested with anti-Fy^a and anti-Fy^b Sera

Sample	Number Tested	Phenotypes								Gene Frequencies			Author(s)
		Fy(a-b-)		Fy(a+b-)		Fy(a-b+)		Fy(a+b+)		Fy^a	Fy^b	Fy	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Panjabis	145	1	.0069	57	.3931	28	.1931	59	.4069	.5527	.3642	.0831	Present study
Hindus and Muslims (M.P.)	277	1	.004	104	.375	61	.220	111	.401	.536	.392	.073	Roberts et al (1974)

TABLE 5.12 DISTRIBUTION OF SERUM HAPTOGLOBIN GROUPS - PANJABIS (N. INDIA)

Phenotype Hp	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
1-1	17	.0543	15.21	.0486
2-1	104	.3323	107.55	.3436
2-2	192	.6134	190.24	.6078
0-0	7	-	-	-
Total	313	1.0000	313.00	1.0000

Gene Frequencies

Hp^1 .2204

Hp^2 .7796

TABLE 5.13 DISTRIBUTION OF SERUM TRANSFERRIN GROUPS - PANJABIS (N. INDIA)

Phenotype Tf	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
CC	312	.9968	312.00	.9968
CB	1	.0032	1.00	.0032
BB	-	-	-	-
Total	313	1.0000	313.00	1.0000

Gene Frequencies

Tf^C	.9984
Tf^B	.0016

TABLE 5.14 HP^1 GENE FREQUENCIES IN THE INDIAN SUB-CONTINENT

Sample	Total No.	HP^1 Frequency	(Author(s)
Nadar (S. India)	-	.037	Ananthakrishnan (1972)
Naickers (S. India)	135	.050	,, ,,
Irulas (S. India)	74	.070	Kirk and Lai (1961)
Tamils (S. India)	133	.090	,, ,,
Muslims (E. India)	121	.0990	Mukherjee et al (1974)
Thevar (S. India)	-	.100	Ananthakrishnan (1972)
Parsis (W. India)	30	.1034	Baxi and Camoens (1969a)
Marathi (W. India)	497	.1165	,, ,,
Sch. Castes (N. India)	85	.1190	Blake et al (1971)
Doms (N. India)	81	.130	Chopra (1970)
Konkon Christians (W. India)	44	.1363	Baxi and Camoens (1969a)
Parsis (W. India)	611	.137	Undevia et al (1973)
Panjabi Khatris (N.India)	100	.1377	Seth et al (1971)
Marathas (W. India)	145	.140	Baxi and Hakim (1966)
Panjabi Aroras (N.India)	100	.1443	Seth et al (1971)
Rajputs (N. India)	130	.150	Chopra (1970)
Oraons (E. India)	125	.150	Kirk and Lai (1961)
Bengali Hindus (E.India)	310	.1543	Mukherjee and Das (1970)
Kaoras (E. India)	202	.1700	Das et al (1974)
Brahmins (N. India)	109	.174	Chopra (1970)
Mahishya (E. India)	100	.1750	Mukherjee et al (1974)
Gujerati Hindus (W.India)	140	.1798	Baxi and Camoens (1969a)
Audich Brahmins (W.India)	187	.181	Baxi and Parikh (1967)
Reddiar (S. India)	-	.182	Ananthakrishnan (1972)
Muslims (E. India)	54	.1827	Mukherjee and Das (1970)
Lad Vania (W. India)	200	.185	Baxi and Parikh (1967)
Khatri (N. India)	73	.185	Sunderland et al (1975)
Brahmin (N. India)	61	.1885	Blake et al (1971)
Kurumbas (S. India)	49	.190	Kirk and Lai (1961)
Bengalees (E. India)	176	.1960	Tiwari (1960)
Khatris (N. India)	76	.197	Blake et al (1971)
Jat (N.W. India)	159	.1975	Singh et al (1974a)
Konkon Saraswats (W.India)	69	.2014	Baxi and Camoens (1969a)
Vaish (N. India)	62	.2016	Blake et al (1971)
Rajput (N. India)	37	.2027	Blake et al (1971)
Aroras (N. India)	71	.2042	,, ,,
Panjabis (N. India)	161	.2094	Tiwari (1961)
Khasi (E. India)		.213	Goedde et al (1972)
Iranis (W. India)	113	.215	Undevia et al (1973)
Khatri (N.W. India)	132	.2197	Singh et al (1974a)
Panjabis (N.W. India)	313	.2204	Present study
Muslims (C. India)	163	.222	Roberts et al (1974)
Tharus (N. India)	152	.223	Chopra (1970)
Aroras (N.W. India)	102	.2254	Singh et al (1974a)
Brahmin (N. India)	37	.229	Sunderland et al (1975)
Hindus (C. India)	143	.231	Roberts et al (1974)
Panjabis (N. India)	114	.237	Papiha (1973)
Brahmin (N.W. India)	106	.2429	Singh et al (1974a)
Assamese (E. India)	75	.243	Goedde et al (1972)

TABLE 5.14 (Contd.)

Sample	Total No.	HP[1] Frequency	Author(s)
Bania (N. India)	39	.269	Sunderland et al (1975)
Jat (N. India)	48	.271	,, ,,
Oswal Visa Jains (W. India)	203	.276	Baxi and Parikh (1967)
Todas (S. India)	89	.37	Kirk and Lai (1961)
PAKISTAN			
Panjabis	207	.20	,, ,,
Pathans	185	.24	,, ,,
Peshawar	135	.276	Walter et al (1966)
BHUTAN			
Bhutanese	152	.200	Mourant et al (1968)
Thimbu	31	.209	Glasgow et al (1968)
Lunana	21		,, ,,
CEYLON			
Sinhalese	87	.16	Kirk and Lai (1961)
Tamils	46	.14	,, ,,
Veddahs	64	.19	,, ,,
Sinhalese	151	.180	Papiha (1973)

TABLE 5.15 THE DISTRIBUTION OF TRANSFERRIN VARIANTS IN THE INDIAN SUB-CONTINENT

Sample	Phenotype Variants			Author(s)
INDIA	CC	CB	CD	
1. NORTH INDIA				
Panjabis	313	1		Present study
Punjab	102			Papiha and Wastell (1974)
Haryana	74			,, ,,
Panjabis	161			Tiwari (1961)
PANJAB				
(a) Khatri	100			Seth et al (1971)
(b) Arora	100			,, ,,
N. Indians	485			Blake et al (1971)
Delhi Muslims	120	1		Papiha and Wastell (1974)
PANJAB				
(a) Brahmin	106			Singh et al (1974a)
(b) Arora	102			,, ,,
(c) Khatri	132			,, ,,
(d) Jat	158	1		,, ,,
N. INDIA				
(a) Brahmin	109			Chopra (1970)
(b) Rajput	130			,, ,,
(c) Doms	81			,, ,,
(d) Tharus	152			,, ,,
PANJAB				
(a) Khatri	72	1		Sunderland et al (1975)
(b) Brahmin	37			,, ,, ,,
(c) Bania	39			,, ,, ,,
(d) Jat	48			,, ,, ,,
2. E. INDIA				
Bengal Muslim	48			Mukherjee and Das (1970)
Bengal Hindu	288			,, ,, ,,
W. Bengal	384		3	Walter et al (1972)
Mahishya	98		3	Mukherjee et al (1974)
Muslim (W. Bengal)	115		4	,, ,,
ASSAM				
(a) Assamese	74		1	Goedde et al (1972)
(b) Khasi	77	2		,, ,,
Bengalees	176			Tiwari (1960)
Oraons	117		8	Kirk and Lai (1961)
Kaoras	202		5	Das et al (1974)

TABLE 5.15 (Contd.)

Sample	Phenotype Variants			Author(s)
	CC	CB	CD	
3. W. INDIA				
Visa Oswal Jain	203			Bakshi and Parikh (1967)
Lada Vania	192			,, ,,
Audich Brahmin	160			,, ,,
Parsis	609	3		Undevia et al (1973)
Iranis	109			,, ,,
4. C. INDIA				
MADHYA PRADESH				
Hindus	143			Roberts et al (1974)
Muslims	161		2	,, ,,
5. S. INDIA				
Todas	89			Kirk and Lal (1961)
Irulas	74			,, ,,
Kurumbas	49			,, ,,
Tamils	133			,, ,,
Kerala	66		1	Papiha and Wastell (1974)
S. INDIA				
(a) Brahmin	136		1	Ananthakrishnan and Kirk (1969
(b) Naickers	135			,, ,, ,,
6. Indians	1067		1[a]	Bakshi and Camoens (1969b)
7. PAKISTAN				
Panjabis (W.Pakistan)	207			Kirk and Lal (1961)
Pathans (W. Pakistan)	183	2		,, ,,
8. BHUTAN				
Bhutan	149		3	Mourant et al (1968)
Thimbu and Lunana	31		1	Glasgow et al (1968)
9. CEYLON				
Sinhalese	220			Papiha and Wastell (1974)
Tamils	140			Kirk et al (1962)
Veddahs	57		6[a]	,, ,,
Sinhalese	157	1	1	,, ,,
10. BALGLADESH				
Bengali Hindus	16	1		Papiha and Wastell (1974)
Bengali Muslims	196	1	1	,, ,,

[a] There was one homozygote D observed

TABLE 5.16 DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS - PANJABIS (N. INDIA)

Phenotype AP	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A	44	.1060	42.95	.1035
AB	178	.4289	178.53	.4302
B	185	.4458	185.59	.4472
CB	7	.0169	5.31	.0128
CA	1	.0024	2.57	.0062
C	-	-	.04	.0001
Total	415	1.0000	414.99	1.0000

Gene Frequencies

P^a	.3217
P^b	.6687
P^c	.0096

TABLE 5.17 DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 GROUPS- PANJABIS (N. INDIA)

Phenotype PGM	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
1-1	208	.5061	203.94	.4962
2-1	163	.3966	171.14	.4164
2-2	40	.0973	35.92	.0874
Total	411	1.0000	411.00	1.0000

Gene Frequencies

PGM_1^1 .7044

PGM_1^2 .2956

TABLE 5.18 DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS - PANJABIS (N. INDIA)

Phenotype AK	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
1-1	352	.8401	352.88	.8422
2-1	65	.1551	63.27	.1510
2-2	2	.0048	2.85	.0068
Total	419	1.0000	419.00	1.0000

Gene Frequencies

AK^1	.9177
AK^2	.0823

TABLE 5.19 DISTRIBUTION OF RED CELL 6-PHOSPHOGLUCONATE DEHYDROGENASE GROUPS - PANJABIS (N. INDIA)

Phenotype 6-PGD	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A	397	.9543	397.24	.9549
CA	19	.0457	18.55	.0446
C	-	-	.21	.0005
Total	416	1.0000	416.00	1.0000

Gene Frequencies

PGD^A	.9772
PGD^C	.0228

TABLE 5.20 THE INCIDENCE OF GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENCY IN PANJABIS - (N. INDIA)

Population	Number Tested	Phenotype		G6PD Deficiency (%)
		Normal	Deficient	
Panjabis	415	398	17	4.09

TABLE 5.21 LACTATE DEHYDROGENASE TYPES - PANJABIS (N. INDIA)

Population	Number Tested	LDH Normal	LDH Variant	% Variant
Panjabis	415	415	-	-

TABLE 5.21 MALATE DEHYGROGENASE TYPES - PANJABIS (N. INDIA)

Population	Number Tested	MDH Normal	MDH Variant	% Variant
Panjabis	415	415	-	-

TABLE 5.22 DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS IN THE INDIAN SUB-CONTINENT

Sample	Number Tested	Phenotypes									
INDIA		A		BA		B		CA		CB		C	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Panjabis (N.India)	415	44	.1060	178	.4289	185	.4458	1	.0024	7	.0169	-	-
Indians (Singapore)	116	6	.052	46	.396	64	.552	-	-	-	-	-	-
Brahmins (S.India)	139	10	.072	61	.439	68	.489	-	-	-	-	-	-
Naickers (S.India)	129	6	.046	41	.318	82	.636	-	-	-	-	-	-
Marathis (W.India)	399	28	.0702	164	.4110	207	.5188	-	-	-	-	-	-
Gujaratis (W.India)	442	39	.0882	174	.3937	225	.5090	-	-	4	.0090	-	-
Bengalis (E.India)	257	24	.0934	89	.3463	142	.5525	2	.0078	-	-	-	-
Arora (N.India)	71	9	.1268	40	.5634	21	.2958	1	.0141	-	-	-	-
Brahmin ,,	61	7	.1148	24	.3934	30	.4918	-	-	-	-	-	-
Khatri ,,	78	13	.1667	36	.4615	28	.3590	1	.0128	-	-	-	-
Rajput ,,	41	6	.1463	15	.365?	9	4634	1	.0244	-	-	-	-
Vaish ,,	63	7	.1111	24	.3810	32	.5079	-	-	-	-	-	-
Sch.Castes ,,	85	6	.0706	32	.3765	45	.5294	1	.0118	1	.0118	-	-
Misc.Hindu ,,	32	1	.0313	9	.2812	22	.6875	-	-	-	-	-	-
,,non-Hindu (N.India)	62	8	.1290	21	.3387	33	.5323	-	-	-	-	-	-
All Groups Combined (N.India)	493	57	.1156	201	.4077	230	.4665	4	.0081	1	.0020	-	-
Panjabis (N.India)	140	17	.121	54	.386	67	.479	1	.007	1	.007	-	-
Parsis (W.India)	418	76	.1818	186	.4450	156	.3732	-	-	-	-	-	-
Iranis ,,	48	2	.0417	18	.3750	26	.5417	-	-	2	.0417	-	-
Assamese	136	8	.059	56	.412	68	.500	2	.015	2	.015	-	-
Khasi (Assam)	43	6	.139	14	.326	19	.442	-	-	4	.093	-	-
Brahmin (N.W.India)	106	8	.075	44	.415	54	.509	-	-	-	-	-	-
Khattri ,,	130	7	.054	52	.400	71	.546	-	-	-	-	-	-
Arora ,,	103	8	.078	42	.408	52	.505	1	.010	-	-	-	-
Jat ,,	158	10	.063	59	.373	86	.544	-	-	3	.019	-	-
Total Panjabi,,	497	33	.066	197	.396	263	.529	1	.002	3	.006	-	-
Hindu (C.India)	174	21	.121	65	.373	87	.500	1	.006	-	-	-	-

TABLE 5.22 (Contd.)

Sample	Gene Frequencies			Author(s)
	p^a	p^b	p^c	
Panjabis (N.India)	.3217	.6687	.0096	Present Study
Indians (Singapore)	.250	.750	-	Lai and Kwa (1968)
Brahmins (S.India)	.292	.708	-	Ananthakrishnan and Kirk (1969)
Naickers (S.India)	.206	.794	-	,, ,, ,,
Marathis (W.India)	.2757	.7243	-	Blake et al (1970[a])
Gujaratis (W.India)	.2851	.7104	.0045	,, ,,
Bengalis (E.India)	.2704	.7257	.0039	Das and Mukherjee (1970)
Arora (N.India)	.4155	.5775	.0070	Blake et al (1971)
Brahmin ,,	.3115	.6885	-	,, ,,
Khatri ,,	.4038	.5897	.0064	,, ,,
Rajput ,,	.3415	.6463	.0122	,, ,,
Vaish ,,	.3016	.6984	-	,, ..
Sch. Castes (N.India)	.2647	.7235	.0118	,, ,,
Misc.Hindus ,,	.1719	.8281	-	,, ,,
Misc.non-Hindu ,,	.2984	.7016	-	,, ,,
All Groups Combined (N.India)	.3235	.6714	.0051	,, ,,
Panjabis (N.India)	.318	.675	.007	Papiha et al (1972)
Parsis (W.India)	.4043	.5957	-	Undevia et al (1972)
Iranis ,,	.2292	.7500	.0208	,, ,,
Assamese	.272	.714	.014	Goedde et al (1972)
Khasi (Assam)	.302	.651	.047	,, ,,
Brahmin (N.W.India)	.283	.717	-	Singh et al (1974b)
Khattri ,,	.254	.746	-	,, ,,
Arora ,,	.286	.709	.005	,, ,,
Jat ,,	.250	.741	.009	,, ,,
Total Panjabi (N.W.India)	.266	.730	.004	,, ,,
Hindu (C.India)	.310	.687	.003	Roberts et al (1974)

TABLE 5.22 (Contd.)

Sample	Number Tested	Phenotypes											
		A		BA		B		CA		CB		C	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
INDIA													
Muslim (C. India)	163	13	.080	80	.491	69	.423	1	.006	-	-	-	-
Kaoras (E.India)	87	11	.1264	29	.3333	46	.5287	1	.0115	-	-	-	-
CEYLON													
Sinhalese	156	13	.083	53	.340	86	.551	-	-	4	.026	-	-
BHUTAN													
Bhutanese	152	5	.033	42	.276	105	.691	-	-	-	-	-	-

Sample	Gene Frequencies			Author(s)
	p^a	p^b	p^c	
INDIA				
Muslim (C. India)	.328	.669	.003	Roberts et al (1974)
Kaoras (E. India)	.2989	.6954	.0057	Das et al (1974)
CEYLON				
Sinhalese	.253	.734	.013	Roberts et al (1972)
BHUTAN				
Bhutanese	.171	.829	-	Mourant et al (1968)

TABLE 5.23 DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 GROUPS IN THE INDIAN SUB-CONTINENT

Sample	Number Tested	Phenotypes					Gene Frequencies		Author(s)
		1-1		2-1		2-2	PGM_1^1	PGM_1^2	
INDIA		No.	Freq.	No.	Freq.	No. Freq.			
Panjabis (N.India)	411	208	.5061	163	.3966	40 .0973	.7044	.2956	Present study
Marathis (W.India)	352	150	.4261	156	.4432	46 .1306	.6491	.3508	Blake et al (1970[a])
Gujaratis (W.India)	296	146	.4932	116	.3919	34 .1149	.6909	.3091	,, ,,
Bengalis (E.India)	269	140	.5204	95	.3532	34 .1264	.6989	.3012	Das and Mukherjee (1970)
Arora (N.India)	71	35	.4929	26	.3662	10 .1409	.6901	.3099	Blake et al (1971)
Brahmin ,,	61	37	.6065	18	.2951	6 .0984	.7623	.2377	,, ,,
Khatri ,,	78	38	.4872	27	.3462	13 .1656	.6731	.3269	,, ,,
Rajput ,,	41	18	.4390	15	.3659	8 .1951	.6341	.3659	,, ,,
Vaish ,,	63	41	.6508	18	.2857	4 .0635	.7937	.2063	,, ,,
Sch. Castes ,,	85	42	.4941	31	.3647	12 .1412	.6824	.3177	,, ,,
Misc. Hindu ,,	32	17	.5313	11	.3437	4 .1250	.7031	.2969	,, ,,
Misc. non-Hindu(N.India)	62	33	.5323	24	.3871	5 .0806	.7258	.2742	,, ,,
Combined Groups ,,	493	261	.5294	170	.3448	62 .1258	.7089	.2911	,, ,,
Panjabis (N.India)	140	63	.450	60	.429	17 .121	.668	.332	Papiha et al (1972)
Parsis (W.India)	401	219	.5461	153	.3815	29 .0723	.7369	.2631	Undevia et al (1972)
Iranis ,,	46	26	.5652	13	.2826	7 .1522	.7065	.2935	,, ,,
Nadar (S.India)				Figures unavailable			.721	.279	Ananthakrishnan (1972)
Thevar ,,				,,		,,	.796	.204	,, ,,
Reddiar ,,				,,		,,	.804	.196	,, ,,
Assamese (E.India)	136	71	.522	55	.404	10 .073	728	.272	Goedde et al (1972)
Khasi ,,	43	18	.419	23	.535	2 .046	.687	.313	,, ,,
Hindu (C.India)	174	96	.552	59	.339	19 .109	.724	.276	Roberts et al (1974)

TABLE 5.23 (Contd.)

Sample	Number Tested	Phenotypes 1-1		2-1		2-2		Gene Frequencies PGM_1^1	Gene Frequencies PGM_1^2	Author(s)
		No.	Freq.	No.	Freq.	No.	Freq.			
Muslim (C.India)	164	90	.549	58	.354	16	.097	.726	.274	Roberts et al (1974)
Brahmin (N.W.India)	106	57	.5377	40	.3774	9	.0849	.7264	.2736	Singh et al (1974b)
Khatri ,,	130	68	.5231	52	.4000	10	.0769	.7231	.2769	,, ,,
Arora ,,	103	52	.5049	41	.3980	10	.0971	.7039	.2961	,, ,,
Jat ,,	158	88	.5570	60	.3797	10	.0633	.7500	.2400	,, ,,
Total Panjabi,,	497	265	.5332	193	.3883	39	.0785	.7284	.2716	,, ,,
Kaoras (E.India)	134	64	.4776	54	.4030	16	.1194	.6791	.3209	Das et al (1974)
CEYLON										
Sinhalese	155	90	.581	49	.316	16	.103	.739	.261	Roberts et al (1972)
BHUTAN										
Bhutanese	154	93	.6039	49	.3182	12	.0779	.7695	.2305	Mourant et al (1968)

TABLE 5.24 DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS IN THE INDIAN SUB-CONTINENT

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		1-1		2-1		2-2		AK^1	AK^2	
INDIA		No.	Freq.	No.	Freq.	No.	Freq.			
Panjabis (N.India)	419	352	.8401	65	.1551	2	.0048	.9177	.0823	Present study
Indians (England)	132	107	.811	24	.182	1	.008	.902	.098	Rapley et al (1967)
Brahmins (S.India)	124	106	.855	16	.129	2	.016	.919	.081	Ananthakrishnan and Kirk (1969)
Naickers (S.India)	122	101	.8281	18	.147	3	.025	.901	.099	,, ,, ,,
Marathis (W.India)	352	292	.8295	56	.1591	4	.0114	.9091	.0909	Blake et al (1970a)
Gujaratis (W.India)	296	233	.7872	60	.2027	3	.0101	.8885	.1115	,, ,,
Bengalis (E.India)	271	226	.8339	43	.1587	2	.0074	.9133	.0867	Das and Mukherjee (1970)
Arora (N.India)	71	64	.9014	6	.0845	1	.0141	.9437	.0563	Blake et al (1971)
Brahmin (N.India)	61	53	.8689	8	.1311	-	-	.9344	.0656	,, ,,
Khatri (N.India)	78	65	.8333	12	.1538	1	.0128	.9103	.0897	,, ,,
Rajput (N.India)	41	32	.7805	9	.2195	-	-	.8902	.1098	,, ,,
Vaish (N.India)	63	47	.7460	16	.2540	-	-	.8730	.1270	,, ,,
Sch. Castes (N.India)	85	72	.8471	13	.1529	-	-	.9235	.0765	,, ,,
Misc. Hindu (N.India)	32	24	.7500	7	.2188	1	.0312	.8594	.1406	,, ,,
Misc. non-Hindu ,,	62	52	.8387	9	.1452	1	.0161	.9113	.0887	,, ,,
Combined Groups	493	409	.8296	80	.1623	4	.0081	.9108	.0892	,, ,,
N. Indians	46	41	.89	5	.11	-	-	.95	.05	Camoens (1971)
S. Indians	153	119	.778	34	.222	-	-	.889	.111	,, ,,
Khasi (E.India)	43	39	.906	4	.094	-	-	.954	.046	Goedde et al (1972)
Indians	132	107	.811	24	.182	1	.007	.985	.015	Tills et al (1970a)
Panjabis (N.India)	140	112	.800	28	.200	-	-	.900	.100	Papiha et al (1972)
Parsis (W.India)	418	363	.8684	54	.1292	1	.0024	.9330	.0670	Undevia et al (1972)
Iranis (W.India)	48	46	.9583	2	.0417	-	-	.9792	.0208	,, ,,
Nadar (S.India)	figures unavailable							.949	.051	Ananthakrishnan (1972)

TABLE 5.24 (Contd.)

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		1-1		2-1		2-2		AK^1	AK^2	
INDIA		No.	Freq.	No.	Freq.	No.	Freq.			
Reddiar (S.India)		figures unavailable						.949	.051	Ananthakrishnan (1972)
Thevar (S.India)		,,		,,				.966	.034	,, ,,
Brahmin (N.W.India)	106	88	.8302	18	.1698	-	-	.9150	.0850	Singh et al (1974b)
Khatri (N.W.India)	131	108	.8244	23	.1756	-	-	.9122	.0878	,, ,, ,,
Arora (N.W.India)	103	83	.8058	20	.1942	-	-	.9030	.0970	,, ,, ,,
Jat (N.W.India)	149	117	.7852	32	.2148	-	-	.8926	.1074	,, ,, ,,
Total Panjabis (N.W.India)	489	396	.8098	93	.1902	-	-	.9049	.0951	,, ,, ,,
CLYLON										
Sinhalese	156	131	.840	24	.154	1	.006	.917	.083	Roberts et al (1972)
BHUTAN										
Bhutanese	154	154	1.0000	0	-	0	-	1.0000	.000	Mourant et al (1968)
PAKISTAN										
Pakistanis (England)	54	40	.7407	14	.2593	0	-	.8704	.1296	Rapley et al (1967)
NEPAL										
Nepalese	132	123	.9318	8	.0606	1	.0076	.9621	.0379	Tills et al (1970a)

TABLE 5.25 DISTRIBUTION OF RED CELL 6-PHOSPHOGLUCONATE DEHYDROGENASE IN THE INDIAN SUB-CONTINENT

Sample	Number Tested	Phenotypes									Gene Frequencies			Author(s)
		AA		CA		CC		RA			PGD^A	PGD^C	PGD^R	
INDIA		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.					
Panjabis (N. India)	416	397	.9543	19	.0457	-	-	-	-		.9772	.0228	-	Present study
Brahmins (S. India)	137	135	.985	2	.015	-	-	-	-		.993	.007	-	Ananthakrishnan and Kirk (1969)
Naickers (S. India)	130	121	.931	9	.069	-	-	-	-		.965	.035	-	,, ,, ,,
Marathis (W. India)	504	477	.9464	27	.0536	-	-	-	-		.9732	.0268	-	Blake et al (1970a)
Gujaratis (W. India)	498	482	.9679	15	.0301	-	-	1	.0020		.9839	.0151	.0010	,, ,, ,,
Bengalis (E. India)	271	260	.9594	11	.0406	-	-	-	-		.9797	.0203	-	Das and Mukherjee (1970)
Arora (N. India)	71	67	.9437	4	.0563	-	-	-	-		.9718	.0282	-	Blake et al (1971)
Brahmin (N. India)	61	60	.9836	1	.0164	-	-	-	-		.9918	.0082	-	,, ,,
Khatri (N. India)	78	77	.9872	1	.0128	-	-	-	-		.9936	.0064	-	,, ,,
Rajput (N. India)	41	40	.9756	1	.0244	-	-	-	-		.9878	.0122	-	,, ,,
Vaish (N. India)	63	59	.9365	4	.0635	-	-	-	-		.9683	.0317	-	,, ,,
Sch. Castes (N. India)	85	81	.9529	4	.0471	-	-	-	-		.9765	.0235	-	,, ,,
Misc. Hindu (N. India)	32	29	.9063	3	.0937	-	-	-	-		.9531	.0469	-	,, ,,
Misc. non-Hindu (N. India)	62	59	.9516	3	.0484	-	-	-	-		.9758	.0242	-	,, ,,
All Groups Combined	493	472	.9574	21	.0426	-	-	-	-		.9787	.0213	-	,, ,,
Panjabis (N. India)	140	134	.957	6	.043	-	-	-	-		.979	.021	-	Papiha et al (1972)
Parsis (W. India)	418	395	.9450	22	.0526	-	-	1	.0024		.9725	.0263	.0012	Undevia et al (1972)
Iranis (W. India)	48	48	1.0000	-	-	-	-	-	-		1.0000	-	-	,, ,,
Nadar (S. India)	figures not available										.952	.048	-	Ananthakrishnan (1972)
Reddiar (S. India)	,,			,,		,,					.993	.007	-	,, ,, ,,
Therar (S. India)	,,			,,		,,					.994	.006	-	,, ,, ,,
Assamese (E India)	136	130	.956	6	.044	-	-	-	-		.986	.014	-	Goedde et al (1972)
Khasi (E. India)	43	39	.907	4	.093	-	-	-	-		.953	.047	-	,, ,,

TABLE 5.25 (Contd.)

Sample	Number Tested	Phenotypes						Gene Frequencies			Author(s)
		AA		CA		CC		RA		PGD^A	PGD^C	PGD^R	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
CELYON													
Sinhalese	156	151	.968	5	.032	-	-	-	-	.984	.016	-	Roberts et al (1972)
BHUTAN													
Bhutanese	154	89	.5779	59	.3831	6	.0390	-	-	.7695	.2305	-	Mourant et al (1968)
NEPAL													
Nepalese	136	111	.8162	24	.1765	1	.0073	-	-	.045	.955	-	Tills et al (1970b)

TABLE 5.26 INCIDENCE OF G-6PD DEFICIENCY AMONG INDIAN MALES

Sample	Number Tested	Deficient %	Method	Author(s)
Panjabis	415	4.09	E*	Present study
Delhi	363	2.8	S	Khanduja et al (1966)
Delhi	238	2.1	S	Gupta et al (1970)
Army (Panjabi)	200	13.5	D	Dimson and Mcmartan (1946)
Punjab	215	2.8	S	Saha and Banerjee (1971)
Chandigarh	2000	6.9	MR	Jolly et al (1972)
Punjab (Sikhs)	94	2.1	S	Saha and Banerjee (1971)
Bombay	81	7.4	S	Baxi et al (1961)
Bombay (Parsees)	100	19.0	S	Baxi et al (1963)
Bombay	233	0.4	S	Da Costa et al (1967)
Panjabis	322	3.42	E	Singh et al (1974b)
Andhra Pradesh (non-tribal)	241	1.2	S	Meera Khan (1964)
,, ,, (tribal)	224	8.5	S	Meera Khan (1964)
Malaya	204	1.0	S	Lie-Injo and Ti (1964)
Singapore	425	1.9	S	Saha (1969)
Kerala (Dravidians)	1372	1.3	S	Saha and Banerjee (1971)
Sinhalese	320	1.2	MR	Nagaratnam et al (1969)

* The explanation of symbols used is as follows :

S = Screening; D = Drug sensitivity; E = Electrophoresis; MR = methaemoglobin reduction

TABLE 5.27 DISTRIBUTION OF RED CELL LACTATE DEHYDROGENASE IN THE INDIAN SUB-CONTINENT

Sample	Number Tested	LDH Variants			Author(s)
		Normal	LDH Cal-1	LDH Cal-2	LDH Mad-1	
INDIA						
Panjabis	415	415	-	-	-	Present study
Marathis (W. India)	504	495	9	-	-	Blake et al (1970[a])
Gujaratis ,,	501	496	5	-	-	,, ,,
Bengalis (E. India)	614	604	10	-	-	Das and Mukherjee (1970)
Tamilnadu (S. India)	717	710	5	-	2	,, ,,
Tamilnadu ,,	1171	1144	25	-	2	Ananthakrishnan et al (1970)
Arora (N. India)	71	71	-	-	-	Blake et al (1971)
Brahmin ,,	61	60	1	-	-	,, ,,
Khatri ,,	78	77	1	-	-	,, ,,
Rajput ,,	41	40	1	-	-	,, ,,
Vaish ,,	63	62	1	-	-	,, ,,
Sch. Castes (N. India)	85	83	2	-	-	,, ,,
Misc. Hindu ,,	32	32	-	-	-	,, ,,
Misc. non-Hindu ,,	62	61	1	-	-	,, ,,
Total N. Indians	493	486	7	-	-	,, ,,
Parsis	418	403	15	-	-	Undevia et al (1972)
Iranis	48	48	-	-	-	,, ,,
CEYLON						
Sinhalese	156	156	-	-	-	Roberts et al (1972)

TABLE 5.28 DISTRIBUTION OF HAEMOGLOBIN TYPES - PANJABIS (N. INDIA)

Haemoglobin type	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
AA	331	.9735	331.09	.9738
AD	9	.0265	8.84	.0260
DD	-	-	.07	.0002
Total	340	1.0000	340.00	1.0000

Gene Frequencies

Hb^A	.9868
Hb^D	.0132

TABLE 6.1 DISTRIBUTION OF ABO BLOOD GROUPS - NEPALESE

Tested with anti-A, anti-B, anti-A+B and anti-A_1 Sera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A_1	100	.4717	100.53	.4742
A_2	-	-	-	-
B	34	.1604	34.22	.1614
O	57	.2689	57.32	.2704
A_1B	21	.0990	19.93	.0940
A_2B	-	-	-	-
Total	212	1.0000	212.00	1.0000

Gene Frequencies

p_1	.3429
p_2	-
q	.1371
r	.5200

TABLE 6.2 DISTRIBUTION OF MNSs BLOOD GROUPS - NEPALESE

Tested with anti-M, anti-N, anti-S and anti-s Sera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
MMSS	2	.0094	3.84	.0181
MMSs	37	.1745	34.60	.1632
MMss	83	.3915	77.80	.3670
MNSS	3	.0142	1.59	.0075
MNSs	18	.0849	20.41	.0963
MNss	49	.2311	59.47	.2805
NNSS	1	.0047	.17	.0008
NNSs	2	.0094	2.76	.0130
NNss	17	.0802	11.36	.0536
Total	212	.9999	212.00	1.0000

Gene Frequencies

MS	.1347
Ms	.6058
NS	.0280
Ns	.2315

TABLE 6.3 DISTRIBUTION OF Rh BLOOD GROUPS - NEPALESE

Rh Type	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
R_zR_z	-	-	.06	.0003
R_1R_z	6	.0284	5.87	.0278
R_1R_1	114	.5403	118.43	.5613
R_2R_z	1	.0047	1.08	.0051
R_1R_2	50	.2370	44.31	.2100
R_1R_o	33	.1564	29.84	.1414
r"r	-	-	.04	.0002
R_2R_2	4	.0190	4.01	.0190
R_2R_o	-	-	5.42	.0257
R_or	2	.0095	1.24	.0059
$R_o^Ur"$	-	-	.04	.0002
rr	1	.0047	.63	.0030
Total	211	1.0000	210.97	.9999

Gene Complex	Frequencies
R_z	.0186
R_1	.7492
R_2	.1346
R_2^U	.0016
r"	.0016
R_o	.0399
r	.0545

TABLE 6.4 DISTRIBUTION OF KELL BLOOD GROUPS - NEPALESE

(a) Tested with anti-K and anti-k Sera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
KK	1	.0047	.08	.0004
kk	205	.9670	204.05	.9625
Kk	6	.0283	7.87	.0371
Total	212	1.0000	212.00	1.0000

Gene Frequencies

K	.0189
k	.9811

(b) Tested with anti-Kp^a and anti-Kp^b Sera

Phenotype	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
Kp^akp^a	-	-	.02	.0005
Kp^aKp^b	2	.0455	1.95	.0444
Kp^bKp^b	42	.9545	42.03	.9551
Total	44	1.0000	44.00	1.0000

Gene Frequencies

Kp^a	.0227
Kp^b	.9773

TABLE 6.5 DISTRIBUTION OF DIEGO BLOOD GROUPS - NEPALESE

Tested with anti-Di^a Serum

Phenotype	Obs. No.	Freq. Obs.
Di(a+)	6	.0429
Di(a-)	134	.9571
Total	140	1.0000

Gene Frequencies

Di^a	.0217
Di^b	.9783

TABLE 6.6 DISTRIBUTION OF ABO BLOOD GROUPS IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

Tested with anti-A, anti-B, anti-A+B, anti-A_1 Sera

Sample	Number Tested	Phenotypes										Gene Frequencies				Author(s)
		A_1		A_2		B		O		A_1B		A_2B		A_1	A_2	B	O	
NEPAL		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.					
Nepalese	212	100	.4717	-	-	34	.1604	57	.2689	21	.0990	-	-	.3429	-	.1371	.5200	Present study
Gorkhas	200	66	.3300	5	.0250	59	.2950	55	.2750	15	.0750	-	-	.2306	.0168	.2088	.5438	Bird et al (1957)
Nepalese	19	7	.36	-	-	6	.31	4	.21	2	.11	-	-	.28	-	.24	.48	Nijenhuis and Runia (1963)
Newars	260	84	.323	15	.058	57	.219	70	.269	26	.100	8	.031	.24	.05	.18	.53	Bhasin (1970)
TIBET																		
Tibetans	42	10	.2381	1	.0238	16	.3809	13	.3095	2	.0476	-	-	Figures unavailable				Orjasaeter et al (1966)
,,	233	40	.1717	-	-	89	.3820	87	.3734	17	.0730	-	-	.1277	-	.2590	.6133	Bhattacharjee (1968c)
BHUTAN																		
Bhutanese	154	52	.3377	-	-	39	.2532	49	.3182	14	.0909	-	-	.2443	-	.1903	.5654	Mourant et al (1968)
BALTISTAN																		
Baltis	80	25	.3125	2	.0250	21	.2625	20	.2500	9	.1125	3	.0375	.2396	.0420	.2315	.4869	Clegg et al (1961)
MONGOLOIDS OF NORTH AND N.E. INDIA																		
Riang	206	40	.1942	1	.0048	89	.4320	45	.2184	30	.1456	1	.0049	.1861	.0060	.3513	.4566	Kumar and Sastry (1961)
Ladakhis	141	38	.2695	1	.0071	45	.3191	47	.3333	10	.0709	-	-	.1953	.0060	.2278	.5709	Bhattacharjee (1968a)
Lepchas	250	94	.3760	-	-	41	.1640	99	.3960	16	.0640	-	-	.2499	-	.1193	.6308	,, ,,
CHINESE (Venezuela)	100	28	.280	4	.040	25	.250	41	.410	2	.020	-	-	.1650	.0249	.1475	.6626	Layrisse and Arends (1956)
BURMA																		
Burmese	115	35	.3043	1	.0087	40	.3478	33	.2869	5	.0435	1	.0087	.1961	.0113	.2294	.5632	Ikin (1958)
,,	83	20	.2410	1	.0120	32	.3855	24	.2892	5	.0602	1	.0120	.1667	.0134	.2663	.5536	Ikin et al (1969)

TABLE 6.7 FREQUENCY DISTRIBUTION OF MNSs SYSTEM IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

Sample	Number	Gene Complexes				Author(s)
		MS	Ms	NS	Ns	
NEPAL						
Nepalese	212	.1347	.6058	.0280	.2315	Present study
Gorkhas	2..	.1578	.5447	.0422	.2553	Bird et al (1957)
Nepalese	93	.20	.36	-	.45	Nijenhuis and Runia (1963)
Newars	15	.21	.48	.12	.18	Bhasin (1970)
BHUTAN						
Bhutanese	49	.1406	.5449	.0342	.2803	Mourant et al (1968)
Thimbu	21	.1655	.5925	.0725	.1694	Glasgow et al (1968)
TIBET						
Tibetans	18	.08	.54	.04	.34	Nijenhuis and Runia (1963)
,,	152	.0888	.5813	.0548	.2751	Bhattacharjee (1968c)
BALTISTAN						
Baltis	80	.2429	4329	.1346	.1904	Clegg et al (1961)
MONGOLOIDS OF NORTH AND N.E. INDIA						
Ladakhis	128	.1355	.5207	.0839	.2599	Bhattacharjee (1968a)
Lepchas	20	.0835	.6324	-	.2841	,, ,,
Chinese (New York)	154	.0405	.5663	.0144	.3788	Miller et al (1951)
BURMA						
Burmese	83	.1163	.6367	.0263	.2207	Ikin et al (1969)

TABLE 6.8 DISTRIBUTION OF Rh BLOOD GROUPS IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

Tested with anti-D, -C, -c, -E and -e Sera

	Present study		Gorkhas Bird et al (1957)		S. Newars Bhasin (1971)		Bhutanese Mourant et al (1968)		Tibetans Nijenhuis and Runia (1963)		S. Chinese Simmons et al (1950)		Burmese Ikin et al (1969)		Baltis Clegg et al (1961)	
Rh Type	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
R_1R_z	6	.0284	3	.015	12	.0656	1	.0065	-	-	2	.0080	2	.0241	-	-
R_1R_1	114	.5403	107	.535	74	.4044	74	.4805	25	.2451	145	.5800	39	.4699	25	.3125
r'r'	-	-	-	-	2	.0109	-	-	-	-	-	-	-	-	-	-
R_2R_z	1	.0047	-	-	-	-	2	.0065	-	-	-	-	-	-	-	-
R_1R_2	50	.2370	53	.265	40	.2186	50	.3247	43	.4216	74	.2960	16	.1928	22	.2750
R_1R_o/R_1r	33	.1564	26	.130	36	.1967	6	.0390	7	.0686	14	.0560	15	.1807	16	.2000
r"r	-	-	-	-	2	.0109	-	-	-	-	-	-	-	-	-	-
R_2R_2	4	.0190	8	.040	1	.0054	17	.1104	17	.1667	11	.0440	3	.0361	2	.0278
R_2R_o	-	-	-	-	9	.0492	-	-	-	-	-	-	-	-	-	-
R_2r	-	-	3	.015	-	-	4	.0260	8	.0784	2	.0080	5	.0602	8	.0972
R_or	2	.0095	-	-	5	.0273	-	-	1	.0098	2	.0080	-	-	1	.0125
rr	1	.0047	-	-	2	.0109	1	.0065	1	.0098	-	-	3	.0361	6	.0750
Total	211	1.0000	200	1.000	183	.9999	154	1.0001	102	1.0000	250	1.0000	83	.9999	80	1.0000

TABLE 6.8 FREQUENCY DISTRIBUTION OF Rh GENE COMPLEXES IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

Sample	Gene Complexes								Author(s)
NEPAL	R_z	R_1	-'	R_2	R^U_2	r''	R_o	r	
Nepalese	.0186	.7492	-	.1346	.0016	.0016	.0399	.0545	Present study
Gurkhas	.0081	.7394	-	.1794	-	-	.0731	-	Bird et al (1957)
Nepalese	-	.75	-	.14	-	-	-	.11	Nijenhuis and Runia (1963)
S. Newar	.052	.590	.053	.120	-	-	.086	.099	Bhasin (1970)
BHUTAN									
Ihimbu	-	.5806	-	.2903	-	-	.0378	.0912	Glasgow et al (1968)
Luana	.0142	.5493	-	.3963	-	-	-	.0402	,, ,,
Bhutanese	.0064	.6656	-	.2890	-	-	-	.0390	Mourant et al (1968)
TIBET									
Tibetans	-	.505	-	.397	-	-	.028	.070	Nijenhuis and Runia (1963)
,,	.0133	.5536	-	.2658	-	-	.1673	-	Bhattacharjee (1968c)
BALTISTAN									
Baltis	-	.5500	-	.2139	-	-	.0175	.2186	Clegg et al (1961)
SELECTED INDIAN MONGOLOIDS									
Ladakhis	.0067	.5045	-	.1653	-	.0896	.1293	.1046	Bhattacharjee (1968a)
Lepcha	.0103	.7022	-	.2515	-	-	.0300	-	,, ,,
BURMA									
Burmese	.0141	.6666	-	.1606	-	-	-	.1587	Ikin et al (1969)
S. Chinese	.005	.760	-	.195	-	-	-	.040	Simmons et al (1950)

TABLE 6.9 DISTRIBUTION OF KELL BLOOD GROUPS IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

(a) Tested with anti-K and anti-k Sera

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		KK		kk		Kk		K	k	
NEPAL		No.	Freq.	No.	Freq.	No.	Freq.			
Nepalese	212	1	.0047	205	.9670	6	.0283	.0189	.9811	Present study
Newars	284	1	.003	30	.106	253	.891	.06	.94	Bhasin (1970)

(b) Tested with anti-K Serum

NEPAL		k(+)		K(-)		K	k	
Gorkhas	200	16	.0800	184	.9200	.0408	.9592	Bird et al (1957)
BHUTAN								
Thimbu	30	2	.0667	28	.9333	.0333	.9667	Glasgow et al (1968)
TIBET								
Tibetans	91	4	.0440	87	.9560	.0222	.9778	Nijenhuis and Runia (1963)
,,	42	-	-	42	1.0000	-	1.0000	Orjaseter et al (1966)
Chinese (New York)	103	-	-	103	1.0000	-	1.0000	Miller et al (1951)
BURMA								
Burmese	82	7	.0854	75	.9146	.0437	.9563	Ikin et al (1969)

TABLE 6.10 DISTRIBUTION OF DIEGO BLOOD GROUPS IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

Tested with anti-Di^a Serum

Sample	Number Tested	Phenotypes				Gene Frequencies		Author(s)
		Di(a+)		Di(a-)		Di^a	Di^b	
NEPAL		No.	Freq.	No.	Freq.			
Nepalese	140	6	.0429	134	.9571	.0217	.9783	Present study
BHUTAN								
Bhutanese	128	6	.0469	122	.9531	.0237	.9763	Mourant et al (1968)
Thimbu	28	5	.1786	23	.8214	.0937	.9063	Glasgow et al (1968)
TIBET								
Tibetans	84	2	.1428	82	.9762	.0120	.9880	Nijenhuis and Runia (1963)
,,	42	1	.0238	41	.9762	.0120	.9880	Orjasaeter et al (1966)
BALTISTAN								
Baltis	79	1	.0127	78	.9873	.0063	.9937	Clegg et al (1961)
Chinese (Canton)	100	5	.0500	95	.9500	.0253	.9747	Layrisse and Arends (1956)
BURMA								
Burmese	6	1	.1667	5	.8333	.0871	.9129	Ikin et al (1969)

TABLE 6.11 DISTRIBUTION OF SERUM HAPTOGLOBIN GROUPS - NEPALESE

Phenotype Hp	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
1-1	12	.0566	14.01	.0661
2-1	85	.4009	80.98	.3820
2-2	115	.5425	117.00	.5519
Total	212	1.0000	211.99	1.0000

Gene Frequencies

Hp^1	.2571
Hp^2	.7429

TABLE 6.12 DISTRIBUTION OF SERUM HAPTOGLOBIN GROUPS IN SELECTED POPULATIONS OF SOUTH AND SOUTH-EAST ASIA

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		1-1		2-1		2-2		Hp^1	Hp^2	
NEPAL		No.	Freq.	No.	Freq.	No.	Freq.			
Nepalese	212	12	.0566	85	.4009	115	.5425	.2571	.7429	Present Study
BHUTAN										
Thimbu	31	1	.0323	11	.3548	19	.6129	.2097	.7903	Glasgow et al (1968)
INDIA										
Nadar (S. India)		Figures unavailable						.037	.963	Ananthakrishnan (1972)
Marathi (W. India)	485	8	.0165	97	.2000	380	.7835	.1165	.8835	Baxi and Camoens (1969b)
Gujarati Hindus (W.India)	136	2	.0147	45	.3309	89	.6544	.1798	.8202	,, ,, ,,
North Indians	484	9	.0186	171	.3533	304	.6281	.1952	.8048	Blake et al (1971)
Panjabis (N. India)	197	11	.056	69	.350	117	.594	.230	.770	Sunderland et al (1975)
Bengalis (E. India)	173	8	.046	52	.301	113	.653	.196	.804	Tiwari (1960)
Assamese (E. India)	75	7	.0933	22	.2933	46	.6133	.240	.760	Goedde et al (1972)
Khasi (E. India)	79	4	.0506	25	.3165	50	.6329	.209	.791	,, ,, ,,
PAKISTAN										
Pathans	180	10	.056	67	.372	103	.572	.24	.76	Kirk and Lai (1961)
TAIWAN										
Chinese	172	14	.081	70	.407	88	.512	.285	.715	Blackwell et al (1962)
THAILAND										
Thais	408	20	.049	155	.380	233	.571	.24	.76	Kirk and Lai (1961)
Thais	666	38	.057	243	.365	385	.578	.24	.76	Blackwell and Thephusdin (1963)
MALAYSIA										
Malays	234	12	.051	85	.363	137	.585	.23	.77	Kirk and Lai (1961)
Chinese	165	18	.109	57	.345	90	.545	.28	.72	,, ,, ,,

TABLE 6.13 DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS - NEPALESE

Phenotype AP	Obs. No.	Freq. Obs.	Exp. No.	Exp. Freq.
A	3	.0146	4.82	.0234
BA	57	.2767	52.90	.2568
B	143	.6942	145.29	.7053
CB	3	.0146	2.53	.0123
CA	-	-	.45	.0022
C	-	-	.02	.0001
Total	206	1.0001	206.01	1.0001

Gene Frequencies

p^a	.1529
p^b	.8398
p^c	.0073

TABLE 6.14 DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 GROUPS - NEPALESE

Phenotype PGM	Obs. No.	Freq. obs.	Exp. No.	Freq. Exp.
1-1	139	.6651	131.06	.6271
2-1	53	.2536	68.89	.3296
2-2	17	.0813	9.05	.0433
Total	209	1.0000	209.00	1.0000

Gene Frequencies

PGM_1^1 .7919

PGM_1^2 .2081

TABLE 6.15 DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS - NEPALESE

Phenotype AK	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
1-1	210	.9906	210.01	.9906
2-1	2	.0094	1.99	.0094
2-2	-	-	-	-
Total	212	1.0000	212.00	1.0000

Gene Frequencies

AK^1	.9953
AK^2	.0047

TABLE 6.16 DISTRIBUTION OF RED CELL 6-PHOSPHOGLUCONATE DEHYDROGENASE GROUPS - NEPALESE

Phenotype 6-PGD	Obs. No.	Freq. Obs.	Exp. No.	Freq. Exp.
A	172	.8269	173.58	.8345
CA	36	.1731	32.86	.1580
C	-	-	1.56	.0075
Total	208	1.0000	208.00	1.0000

Gene Frequencies

PGD^A .9135

PGD^C .0865

TABLE 6.17 LACTATE DEHYDROGENASE TYPES IN NEPALESE

Population	Number Tested	LDH Normal	LDH Variant	% Variant
Nepalese	207	207	-	-

TABLE 6.17 MALATE DEHYDROGENASE TYPES IN NEPALESE

Population	Number Tested	MDH Normal	MDH Variant	% Variant
Nepalese	207	207	-	-

TABLE 6.18 DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

	Number Tested	Phenotypes												Gene Frequencies			Author(s)
		A		BA		B		CA		CB		C		P^a	P^b	P^c	
NEPAL		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Nepalese	206	3	.0146	57	.2767	143	.6942	-	-	3	.0145	-	-	.1529	.8398	.0073	Present study
BHUTAN																	
Bhutanese	152	5	.0329	42	.2763	105	.6908	-	-	-	-	-	-	.1711	.8289	-	Mourant et al (1968)
INDIA																	
Indians (Singapore)	116	6	.052	46	.396	64	.552	-	-	-	-	-	-	.250	.750	-	Lai and Kwa (1968)
Brahmins (S.India)	139	10	.072	61	.439	68	.489	-	-	-	-	-	-	.292	.708	-	Ananthakrishnan and Kirk (1969)
Marathis (W.India)	399	28	.0702	164	.4110	207	.5188	-	-	-	-	-	-	.2757	.7243	-	Blake et al (1970)
Panjabis (N.India)	497	33	.066	197	.396	263	.529	1	.002	3	.006	-	-	.266	.730	.004	Singh et al (1974b)
Assamese (E.India)	136	8	.059	56	.412	68	.500	2	.015	2	.015	-	-	.272	.714	.014	Goedde et al (1972)
Kaoras (E. India)	87	11	.1264	29	.3333	46	.5287	1	.0115	-	-	-	-	.2989	.6954	.0057	Das et al (1974)
Bengalis (E.India)	257	24	.0934	89	.3463	142	.5225	2	.0078	-	-	-	-	.2704	.7257	.0039	Das and Mukherjee (1970)
SINGAPORE																	
Chinese	620	36	.058	203	.327	381	.615	-	-	-	-	-	-	.222	.778	-	Lai and Kwa (1968)
Malayans	260	35	.135	108	.415	116	.446	1	.004	-	-	-	-	.344	.654	.002	,, ,,
TAIWAN																	
Chinese	100	7	.070	21	.210	60	.600	3	.030	7	.070	2	.020	.190	.740	.070	Shih and Hsia (1969)

TABLE 6.19 DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

Sample	Number Tested	Phenotypes										Gene Frequencies				Author(s)
		1-1		2-1		2-2		6-1		7-1		PGM_1^1	PGM_1^2	PGM_1^6	PGM_1^7	
NEPAL		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.					
Nepalese	209	139	.6651	53	.2536	17	.0813	-	-	-	-	.7919	.2081	-	-	Present study
BHUTAN																
Bhutanese	154	93	.6039	49	.3182	10	.0649	2	.0130	-	-	.7695	.2240	.0065	-	Mourant et al (1968)
INDIA																
Marathis (W.India)	352	150	.4261	156	.4432	45	.1278	1	.0028	-	-	.6491	.3494	.0014	-	Blake et al (1970)
Bengalis (E.India)	269	140	.5204	95	.3532	33	.1227	1	.0037	-	-	.6989	.2993	.0019	-	Das and Mukherjee (1970)
N. Indians	493	261	.5294	170	.3448	62	.1258	-	-	-	-	.7089	.2911	-	-	Blake et al (1971)
Assamese (E.India)	136	71	.522	55	.404	10	.073	-	-	-	-	.728	.272	-	-	Goedde et al (1972)
TAIWAN																
Chinese	100	53	.530	41	.410	6	.060	-	-	-	-	.735	.265	-	-	Shih and Hsia (1969)
THAILAND																
Thais	503	268	.532	198	.393	37	.074	-	-	-	-	.730	.270	-	-	Giblett and Scott (unpubl.)
INDONESIA																
Chinese	88	51	.5795	28	.3182	5	.0568	2	.0227	2	.0227	.7614	.2159	.0114	.0114	Lie-Injo and Poey (1970)
Chinese (from San Francisco, Kuala Lumpur, Djarta)	427	242	.567	150	.351	35	.082	-	-	-	-	.757	.243	-	-	Lie-Injo et al (1968)

TABLE 6.20 DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

Sample	Number Tested	Phenotypes						Gene Frequencies		Author(s)
		1-1		2-1		2-2		AK^1	AK^2	
NEPAL		No.	Freq.	No.	Freq.	No.	Freq.			
Nepalese	212	210	.9906	2	.0094	-	-	.9953	.0047	Present study
Nepal	132	123	.9318	8	.0606	1	.0076	.9621	.0379	Tills et al (1970a)
BHUTAN										
Bhutanese	154	154	1.0000	-	-	-	-	1.0000	-	Mourant et al (1968)
INDIA										
Indians (Malaysia)	227	180	.7929	44	.1938	3	.0132	.8899	.1101	Chan (1971)
Indians (England)	132	107	.8106	24	.1818	1	.0076	.9015	.0985	Rapley et al (1967)
Brahmins (S.India)	124	106	.855	16	.129	2	.016	.919	.081	Ananthakrishnan and Kirk (1969)
Bengalis (E.India)	271	226	.834	43	.159	2	.007	.913	.087	Das and Mukherjee (1970)
TAIWAN										
Chinese	100	100	1.0000	-	-	-	-	1.0000	-	Shih and Hsia (1969)
Chinese	227	226	.995	1	.004	-	-	.998	.002	Shih et al (1968)
MALAYSIA										
Malays	400	385	.9625	15	.0375			.9813	.0187	Chan (1971)
Malays (S.Africa)	100	93	.9300	7	.0700			.9650	.0350	Gordon et al (1966)
Chinese	318	318	1.0000	-	-	-	-	1.0000	-	Chan (1971)
THAILAND										
Thais	201	192	.9552	9	.0448	-	-	.9976	.0024	Giblett and Scott (unpubl.)

TABLE 6.21 DISTRIBUTION OF RED CELL 6-PHOSPHOGLUCONATE DEHYDROGENASE GROUPS IN SELECTED POPULATIONS OF SOUTH AND SOUTH EAST ASIA

Sample	Number Tested	Phenotypes					Author(s)
		AA	CA	CC	PGD^A	PGD^C	
NEPAL		No. Freq.	No. Freq.	No. Freq.			
Nepalese	208	172 .8345	36 .1580	- -	.9135	.0865	
Nepal	136	111 .8162	24 .1765	1 .0073	.9044	.0956	Tills et al (1970b)
BHUTAN							
Bhutanese	154	89 .5779	59 .3831	6 .0390	.7695	.2305	Mourant et al (1968)
INDIA							
Naickers (S.India)	130	121 .931	9 .069	- -	.965	.035	Ananthakrishnan et al (1970)
Assamese (E. India)	136	130 .956	6 .044	- -	.986	.014	Goedde et al (1972)
Bengalis ,,	271	260 .9594	11 .0406	- -	.9797	.0203	Das and Mukherjee (1970)
Panjabis (N.India)	140	134 .957	6 .043	- -	.979	.021	Papiha et al (1972)
N. Indians	493	472 .9574	21 .0426	- -	.9787	.0213	Blake et al (1971)
TAIWAN							
Chinese	228	199 .8728	28 .1228	1 .0044	.9342	.0658	Shih et al (1968)
THAILAND							
Thais	441	379 .8594	62 .1406	- -	.9297	.0703	Giblett and Scott (unpubl.)
MALAYSIA							
Malays (S.Africa)	100	95 .9500	5 .0500	- -	.9750	.0250	Gordon et al (1966)