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A SEROLOGICAL - GENETIC STUDY OF IRANIAN AND NEIGHBOURING POPULATIONS

By

PARIVASH. AMIRSHAHI, Pharm.D., M.P.H.

A Thesis Submitted for the Degree of

Doctor of Philosophy

Department of Anthropology

University of Durham. U.K.

1983

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ABSTRACT

A total of 1709 blood specimens collected from ten population groups of Iran have been studied for genetic variations of some blood group, serum protein and red cell enzyme systems. The results were combined with those of the previous studies on other Iranian groups and compared with reported frequencies for neighbouring populations.

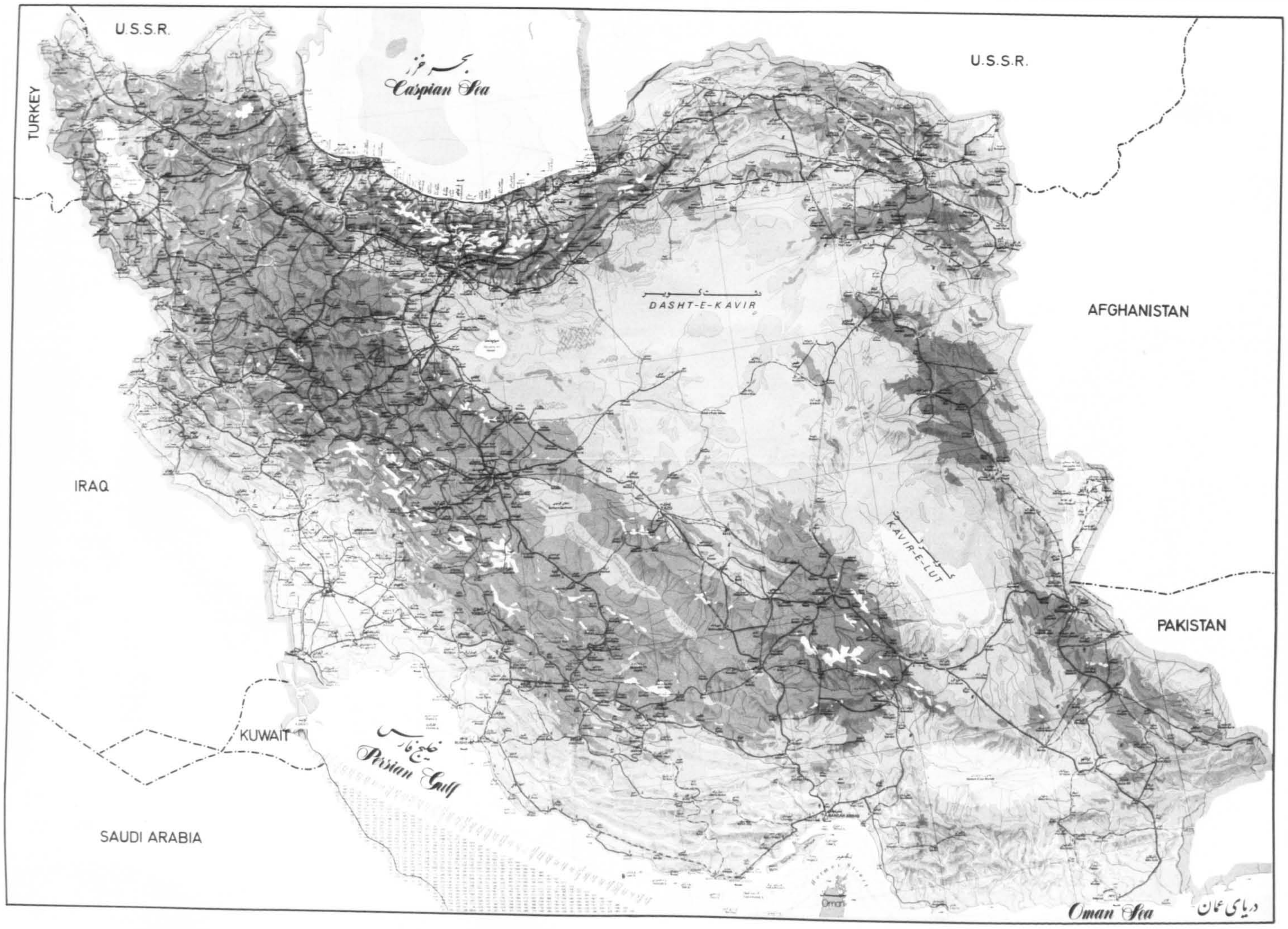
The average gene frequencies for Iran, in general, show a departure from the values found in the countries to the West and an approach to those of the Indian region. By contrast, the generalized feature of gene frequencies for both the Caucasus and Turkey show connections rather with the West than with the East.

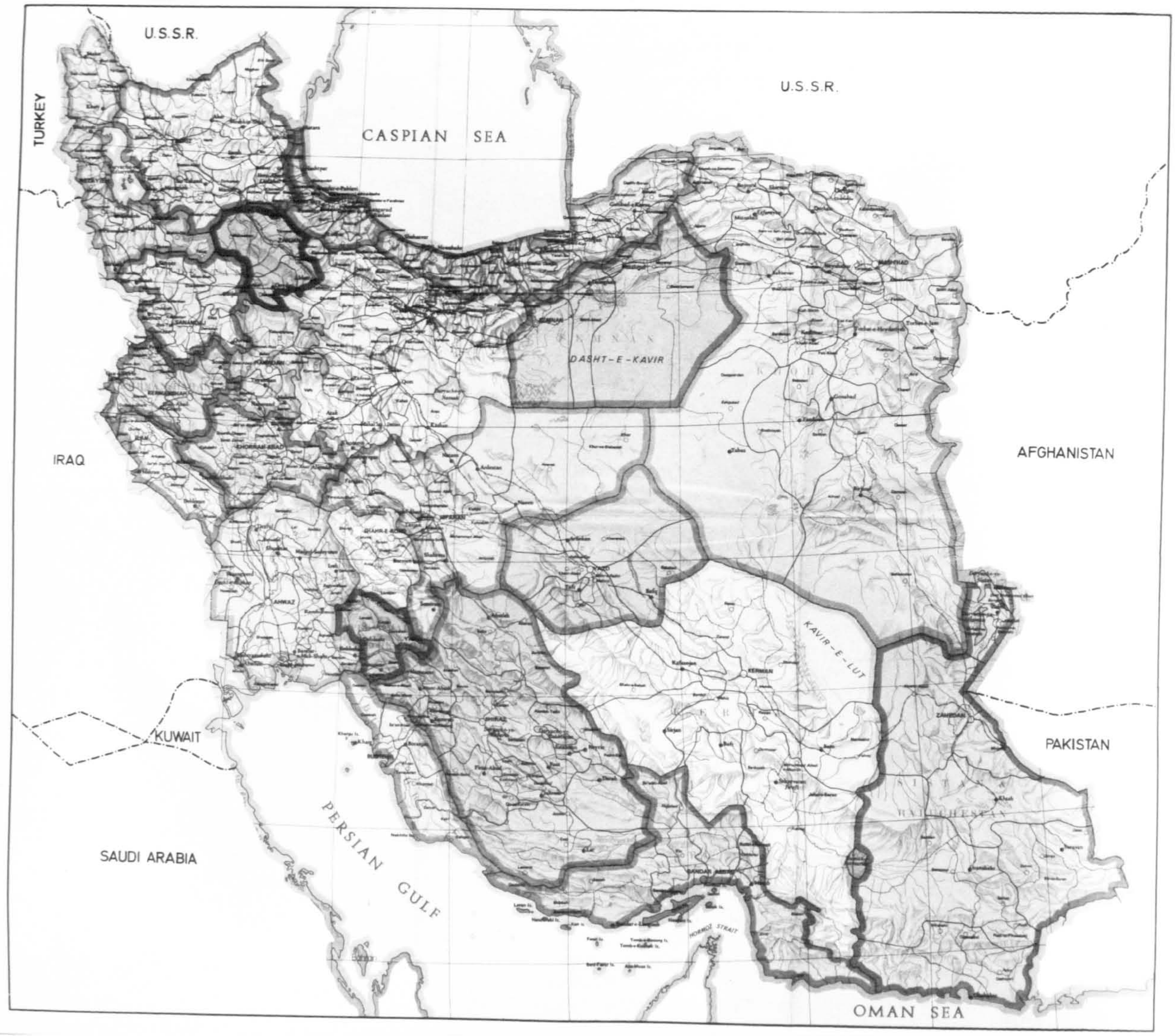
Taken as a whole the gene frequencies of the population of Iraq suggest Eastern rather than Western connections. The Arabs of Kuwait, Saudi Arabia and the United Arab Emirates, having high frequencies of African marker genes, show a considerable degree of admixture from Africans. It is notable that the Arabic speakers of South Western Iran show little resemblance to the Arabs of Arabia. On thewhole, the serological evidence suggests that over a long period of time there has been little migration into Iran across the Persian Gulf. The generalized feature of gene frequencies for Pakistan show relationships rather with India than with the neighbouring countries to the West. The average gene frequencies for the Afghans suggest both Caucasoid and Mongoloid characteristics.

To :

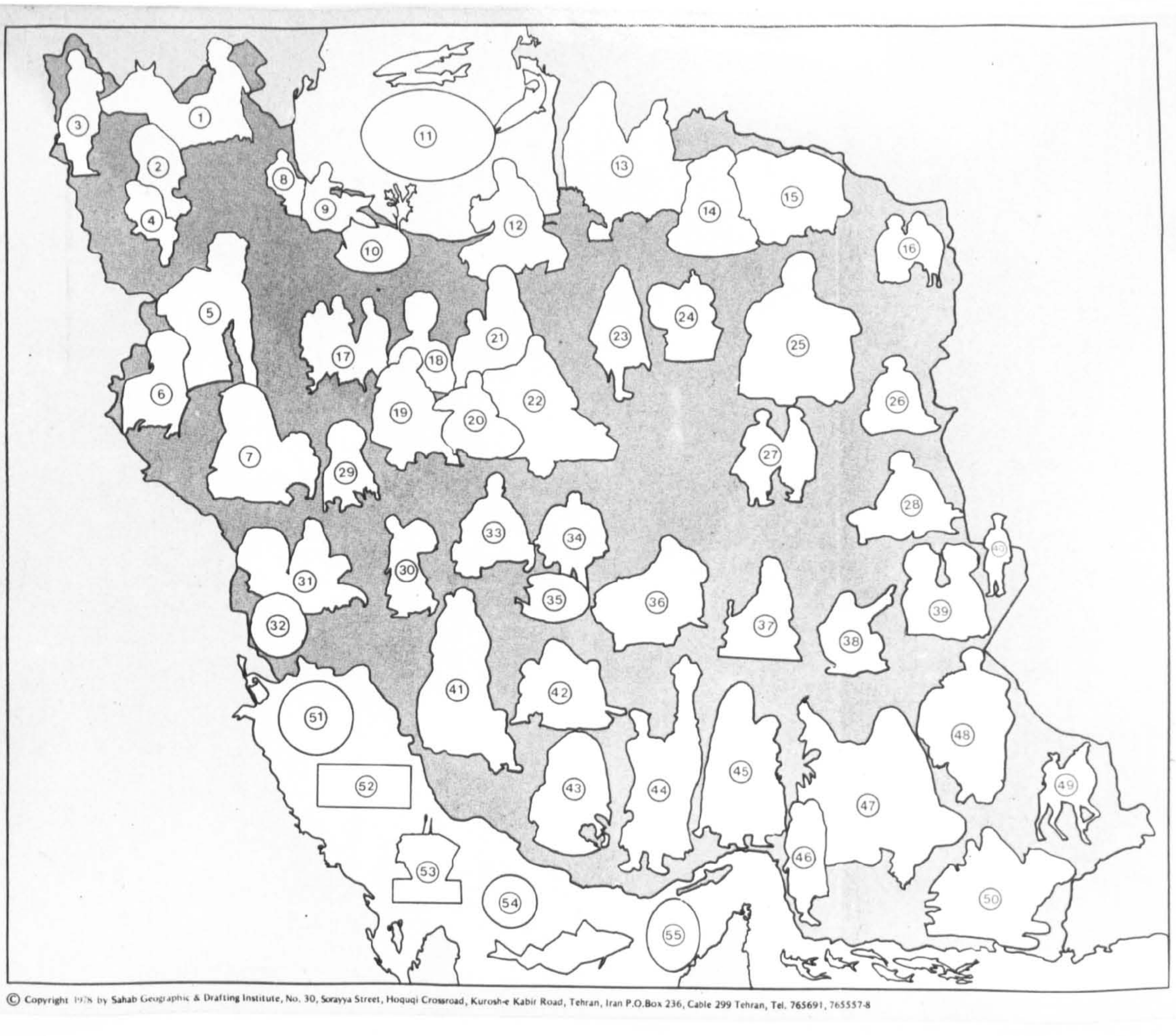
My Country




U.S.S.R.
U.S.S.R.
TURKEY
بحر خزر
Caspian Sea
دشت کویر
DASHT-E-KAVIR
AFGHANISTAN
IRAQ
KAVIR-E-LUT
PAKISTAN
KUWAIT
خلیج فارس
Persian Gulf
SAUDI ARABIA
Oman
Oman Sea
دریای عمان


U.S.S.R.
U.S.S.R.
TURKEY
CASPIAN SEA
DASHT-E-KAVIR
IRAQ
AFGHANISTAN
KAVIR-E-LUT
KUWAIT
PAKISTAN
SAUDI ARABIA
PERSIAN GULF
OMAN SEA





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Ethno Composition

LOCAL COSTUMES – HANDICRAFTS – CERTAIN AGRICULTURAL PRODUCTS

1. Shahsavan horse-rider
2. Rural girl from Eastern Azarbaijan
3. Rural woman from Western Azarbaijan
4. Old man from Oshnoviyeh strings tobacco for drying
5. Kurdish woman from the area around Sanandaj
6. Kurdish man from Javanrood area
7. Kurdish woman from the environs of Kermanshah
8. Kaviar-production, Bandar Pahlavi
9. Girl from Lahijan picking tea leaves.
10. Riceplanting by Gilani women
11. Fishing in the Caspian Sea
12. Beautiful local costume of the Northern Provinces
13. Carpet-weaving Turkomans (Yamut tribe)
14. Turkoman girl, sorting wool
15. Turkoman Yurt
16. Shepherd
17. Children of the Central Province
18. A city-girl (Tehran)
19. Qalamkar-maker (Isfahan)
20. Engraving a tray (Isfahan)
21. Girl and boy from rural area of the Central Province
22. Roses gathered for Golab-distillation (Rose-water) in Qamsar near Kashan
23. Woman from Sanguesar-e Shahmirzad
24. Traditional transportation of straw
25. Man in local costume of northern Khorasan
26. Costume of the Barbar and Teymouri tribes of eastern Khorasan
27. Carpet pedlar in eastern Khorasan
28. Shoemaker from southern Khorasan
29. Woman of the Lor tribe from the environs of Khoramabad
30. Young Bakhtiary mother carries her baby
31. Bread-baking in the environs of Shushtar
32. Lovely girl from southern Khuzestan
33. Rural man from Isfahan area, spinning wool
34. Transportation of dyed wool for carpet-weaving, near Isfahan
35. Carpet-weaving girl, Isfahan
36. Woman from Yazd area spinning with an old spinning wheel
37. Woman from a village near the desert
38. Baluchi woman spinning wool
39. Two men from Sistan
40. Man from Zabol
41. Qashqa'i woman spinning wool in the old traditional way
42. Rural woman from the environs of Shiraz with her children
43. Woman from southern Fars Province
44. Qashqa'i camel driver
45. Woman and children from Bandar Abbas
46. Young girl from around Minab
47. Baluchi-woman weaving a net for the transportation of straw
48. Pedlar from Zahedan
49. Dromedary-rider
50. Two girls riding on a camel
51. Diving in the Persian Gulf
52. Trade-Vessel
53. Motor-Launch on the Persian Gulf
54. Sunset on the Persian Gulf
55. Pearl-diver

For my husband and my two children
Amir Ali and Nazli without whose
moral support, patience, love and
understanding this research could
never have been under taken.

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NOTE ON THE TABLES

1- All the tables are arranged by order of gene frequencies.

2- All the tables of each serological system for nine countries in chapter 5 will have the same number and each country will take a code as follows:

I	Iran
II	Caucasus
III	Turkey
IV	Iraq
V	Kuwait
VI	Saudi Arabia
VII	The United Arab Emirates
VIII	Pakistan
IX	Afghanistan

3- The population numbers in the tables correspond to those on the maps.

INTRODUCTION

There are obvious phenotypic differences between human populations throughout the world. Physical characteristics, such as skin, hair and eye colour, stature, and the shape of the cranium have long been used for purposes of classification of humanity. The differences between populations with regard to such characters are obvious to the layman, but the anthropologist has reduced the results of examining them to mathematical form. It is, however, well known that, with one or two possible exceptions, such characters are not inherited according to any simple rule, and geneticists have shown that, in the case of certain of these characters, the quantitative expression of any one of them is controlled by a number of independent genes. Furthermore, most of the measurable features of human beings bear the marks not only of heredity but in a varying degree of environment, and especially of the state of nutrition during early life. Though the relationship is not a simple one, it is generally true that characters whose individual expression tends to be modified by the environment are also particularly subject to natural selection by the environment in the population as a whole. For instance,it is probable that the individual who is genetically small-boned will be at an advantage in a lime-poor environment, and so will tend to have more surviving offspring than a large-boned person.

In spite of these combinations and the apparently continuous grading of the characters concerned, morphological anthropologists have come to realize that the underlying factor in their classifications of human beings consists in the dis-

crete genes which are reproduced and so transmitted from generation to generation in a given population, and which, taken together, determine its innate physical composition.

Although obvious phenotypic features distinguish one population from another, it is also recognized that genotypic factors, such as blood group gene frequencies, differ from population to population. In the case of the blood factors we are dealing with unitary and obviously discontinously characters which are determined at conception and remain fixed for life that is to say,with the separate effects of individual genes which are sharply differentiated from one anther and which are therefore less subject to personal errors of observation than are many of the characters hitherto used.It appears likely that natural selection due to the external environment is slight for most of the blood factors(though certain combinations of blood groups are known to be intrinsically unstable).Moreover,the observed characters are a much more direct consequence of the genetic constitution than are the directly observable characters of the body.

It is evident that regional differences have biological significance and the functioning of the physical organism towards its environment is determined by the genes responses. Physical anthropologists are devoted to the understanding of these differences and their biological significance.

The study of population structure can show the interrelationship of biological and cultural variation with reference to a variety of models. Since the genetic structure of a population refers to the distribution and relationship of gene and genotype frequencies, it is ultimately a reflection of the population's mating structure(Yasuda and Morton, 1967).

In studies of population structure, the effect of barriers to gene flow,which create a subdivided population, are of major importance. These barriers may be geographic and/or cultural in nature. Man has complex rules about marriage and mating which can affect the amount of inbreeding in any population. In some small groups, the prohibition of mating with a close relative may force a man to seek a bride from another community, thus expanding the gene pool. Many thousands of years ago the movement of both large and small groups of people and their consequent admixture have made it difficult for any clear classification of human types to be made. In fact, much of today's research into human population variation seems to emphasise the need to examine the various factors contributing to human diversity.

The ABO blood groups were discovered at the beginning of the century(Landsteiner, 1900), their Mendelian inheritance indicated in 1911 by Von Dungern and Hirszfeld. Hirszfeld and Hirszfeld(1919),introduced the distribution of human blood groups as a new basis of classification. An enormous mass of information has accumulated since then on the distribution of ABO blood groups, the results of which were collected by Mourant et al(1954 and 1976) whose investigations suggested their variation in frequency differed from one population to another(Sunderland, 1973; Papiha,1979). Sunderland(1973), considers that,"...from an anthropological view point the most interesting matter is that the distribution of the A,B and O genes is extremely variable among the World's People." Distribution of other genetical characters such as MNS, Rh and taster reactions to phenylthiocarbamide and the later discovered blood groups are still being studied. These charact-

ers are susceptible to accurate statistical analysis. Their mode of inheritance is by Mendelian laws.

Apart from blood group systems, the new biochemical markers such as serum proteins, red cell isoenzymes and haemoglobins are of great importance in anthropological research. Their frequencies change slowly and the mode of heritance is relatively simple. Surveys from various parts of the world have shown that most of these markers are polymorphic,that is, most of them occur in two or more forms,maintaining a frequency of greater than one percent and less than ninety nine percent. Not all characters determined by single genes can be used for anthropological classification. To be of value, the gene frequency must vary significantly from one population to another, and the frequency of the second most common gene in a given system must be sufficiently high in some populations to enable a useful estimate of its value to be made by exami ning or testing only a few hundred perons. This eliminates nearly all congenital pathological conditions as being too rare, but a few inherited haematological abnormalities, such as thalassaemia, the sicklecell trait and glucose-6-phosphate dehydrogenase deficiency, are sufficiently common in some populations to render them taxonomically useful.

At the present time the study of genetic markers,such as the blood group systems can provide very useful information on the differentiation of human populations.

The aim of the present study is to add to the genetic data already existing, to attempt to appreciate the genetic structure of Iranian population as a whole and to compare it with those of the neighbouring groups.

CHAPETER 1. INTRODUCTION TO IRAN

1.1. Geographical setting.

1.1.1. Physical features.

Iran means " Land of the 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 (1,648,000 sq.Km) and is the fourth largest country in Asia.

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.

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 the sea of Oman, and on the west by Turkey and Iraq. 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.

Iran is a land of high mountains, deep valleys, great flat - lands and vast arid deserts. The average altitude is 3,300 feet, while many peaks rise as high as 10,000 feet. Mount Damavand, the highest point in the Alborz range, rises to nearly 19,000 feet.

Two major ranges divide Iran in an east-west direction: the Alborz, beginning in the Caucasus, passes through northern Iran and continues into Afghanistan. The second range, the Zagros begins in Anatolia and continues right down to Baluchistan. Iran has two big deserts: the salt Desert (Kavir-e-Namak) south east of Tehran and the Lut Desert (Kavir-e-Lut)

further to the south-east. There are several major lakes in Iran, apart from the great in land sea, the Caspian, which lies to the north. The largest is Lake Rezaiyeh in west Azarbaijan, followed the Hamun in Sistan and the Bakhtegan in Fars. The heart of the country is the Tehran-Esfahan area.To the south is the important province of Fars, with its principal city of Shiraz. Further to the south-east is the province of Kerman. In the north-east is the province of Khorasan with its largest city,Mashhad. Further to the south and east is the barren land of Baluchistan.

1.1.2 Climate

Almost all of Iran lies in the north temperate Zone,but the plateau as a whole offers a variety of climates which,in turn, yield a great variety of agricultural products. Iran has great extremes of climate, the Caspian littoral is humid and sub-tropical while the central deserts are almost completely dry. Annual rainfall varies from an average of 40 inches in Gilan and Mazandaran to an average of less than two inches in the desert regions. Temperatures also vary greatly; in parts of the deserts and the areas of the Persian Gulf temperatures are rise to 130 degrees Fahrenheit,while in certain parts of the north-west temperatures of less than 18^{o} F. are not unusual in winter. Rain generally falls between October and May, the heaviest being recorded in December and March. The heihgt of the plateau and the presence of high mountains contribute to cold winters; frost occurs all over the country with the exception of southern Khuzistan and the coastal areas of the Persian Gulf and the sea of Oman. In central and north-eastern as well as in north-western regions, he-

avy snows fall from November to early March.

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 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 replaced the original inhabitants. In the 19th Century B.C., nomadic tribes like the Medes, Persians (Parsa) and Parthians entered Iran from the north of the Caspian sea and by way of the Caucasus and mixed with the former inhabitants. The Medes settled in what is today north-west 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 dyna-

stry (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, Azarbaijan and other areas were Turkified, the Turkish speaking tribes being scattered throughout the area. The north-east and north-west saw strong intrusions of Turkish and Tartar elements. The Mongols (1200 A.D.) ruled Iran for a century and strongly influenced the population. Eventually the Mongols were converted to Islam and this facilitated the mixing with earlier established Persians.

Persian history falls naturally into two distinct periods: the pre-Islamic and the Islamic, the dividing point being the overthrow of the Sassanians by the Arabs.in the third decade of the Seventh century A.D.

The Persians themselves always speak of their country as Iran, an ethnic name implying the habitat of the Iranians - a branch of the Indo-European family. The term Persia is derived from the name of the south-westerly province of Pars (Modern Fars), where the first kings of united Iran, the Achaemenids, had their Capitals Pasargadae and Persepolis(Parsa).

As far as we know, the earliest Iranians to settle in what we now call Persia were the Medes who, migrating from the region of Transoxania or Turkistan,established themselves in the country lying between Rhages and Ekbatana (now Hamadan) in the north-west, probably in the eighth century B.C. Of the early Medes we know very little beyond what may be learnt from the

Greek historians and occasional allusions in Assyrian inscriptions for the Medes had frequent intercourse with their neighbours the Assyrians, from whom they acquired the art of writing, and much else besides.

Three general factors characterize Persian history: general continuity, periodic cataclysms, and the extraordinary influence of physical environment. For 2,500 years Persia had possessed an educated ruling class and a tradition of civilization which had survived the most barbarous invasions and the most alien conquests. From about 600 B.C. to the Moslem conquest there existed a native Persian civilization which nearly reached its perfection, modified by Hellenic influence after the conquest of Alexander and later, Islam. This civilization blossomed again in the Abbasid period, and was modified a third time by Chinese influences after the Turkish and Mongolian conquests. Persian civilization produced the safavid culture in the sixteenth and seventeenth centuries, the artistic productions of which are well known in Europe. In the twentieth century Persian life has again become subject to violent external influence with the introduction of European thought and mechanical techniques, and at present the old way of life and the new are still imperfectly blended. During her long history, Persia has always preserved her frontiers more or less unchanged, and she even emerged from the great war without loss of territory or status.

During certain periods, notably after Alexander the Great's conquest, and for more than two hundred years after the invasion of the country by the arms of Islam, Persia lost her position as an independent empire, and during the remaining periods of her history, though she has often formed merely one

of several states in the dominions of some world-conqueror, she has always retained her national individuality.

1.3. Ethnic groups

The Iranian population is among the most heterogeneous in the world. The region, now called Iran, has been invaded by people of many different races, and each has left its contribution to the gene pool of the local population. Kirk et al (1977) have reviewed the pertinent literature and indicate that the invaders include Assyrians, Greeks, Arabs, Turks, and Mongols. They point out also that many trade routes, including one from China, have crossed Iran, and that Africans were brought as slaves during the Moslem conquest. The invasions and the trade routes have provided ample opportunity for admixture.

Around the rim of Persia are group after group of people who are organized into pastoral tribes. Many of them live near settled folk who speak their language and often have a tradition of having formerly been tribesmen themselves. Such an ehtnic group is likely to be dominated by the tribesmen rather than the settled folk even through the latter are more numerous. The tribesmen are better organized for war and hence have more political influence.It is not surprising therefore that geneticists have made many studies of the people of Iran.Field (1939 and 1959) has reviewed extensively the studies in prehistory and physical anthropology. More recently there has been an increasing number of investigations into the distribution of blood genetic markers in Iranians. These offer a means of dissecting the varied genetic structure of the modern populations and relating these populations to others out-

side the country.

The inhabitants of Persia have neither racial nor linguistic unity. The principal elements are commonly called Persian but there are also Kurds, Lurs, Turks, Baluchis,Arabs, Georgians, Dravidian Brahuis, and Afghan Hazaras of Mongolian origin, whilst religious minorities include Armenians, Jews, Nestorian Christians or " Assyrians" and Zoroastrians.

Exact measurements have been made of only a few small groups of the population, but it seems that the bulk of the Persian population is derived from the Indo-European Aryan-speaking Iranian invaders of the second millennium B.C., and from their intermarriage with the previous inhabitants. The latter belonged to the 'Caucasian' branch which originally colonized the territories from the Caucasus to the Arabian Peninsula. There are several widely different constituents within the 'Persian' block. In the most recent anthropological work on Persia the term " Iranian" is reserved for a widespread physical type, on the border of the medium- and long -headed classifications, that occurs Persistently in Persian-speaking areas. But within the Persian block there are some large groups which may prove to be racially distinct, and possibly purely Caucasian, such as the Bakhtiari tribes of the Central Zagros, who are an extremely short-headed people..The Kurds, too, tend to be short-headed, and the exact affinities of the Persian-speaking stock of Khorasan, commonly called Tajiks, and of the Baluchis, are not yet determined. In general, racial mixture is greatest in those part of Persia which have formed the historical Zones of passage for invading armies and wandering hordes, namely Azarbaijan,

Khorasan, and the intermediate provinces, and also in the Zones of Sistan and Baluchistan which are open to the east. The traditional Persian policy of transferring troublesome tribal groups from one part of the country to another, and the persistent wandering of Arab tribes, have also complicated the racial map.

Thus Arab, Turkish, and Kurdish tribes appear in relatively homogeneous areas such as Fars and Mazandaran and add to the confusion of Khorasan and east Persian Baluchistan With these notable exceptions, there is a block of Iranian peoples speaking Persian dialects or allied languages throughout the Zagros regions and the populated basin west of the central deserts, and in the Alborz and Caspian provinces, while a similar element is considerable in the cultivated areas of Khorasan and continues through the Birjand highlands to Sistan; of specific elements Kurds live in the northern Zagros and north Khorasan, Lurs in the Central and Southern Zagros. The principal area of the Turkish element, descendants of the Seljuk and later invaders, is from Azerbaijan eastwards to Tehran and Saveh districts; there is also a large Turkish group in eastern Fars and northern Khorasan. Arabs are found in the plains of Khuzistan where they form the eastern fringe of the principal body of Arab peoples, in southern Khorasan, in south - eastern Fars, and along the southern Coasts, where they arrived by sea continuously in ancient and recent times. Armenians are most numerous as peasants in Azerbaijan, in the Burujerd and Faridan districts of the north-eastern Zagros, and as townsmen in many cities of north-western and northern Persia, particulary in the Julfa suburb of Esfahan. Jews are widely scattered in towns throughout Persia.

Georgians exist in much the same areas as the Armenians, but have generally been assimilated to Persians.

1.3.1.The Turks

The Turks of Persia have few racial affinities with the Ottoman Turks of Anatolia, and are generally grouped with the Uzbek and other Turkemans of the Adjoining Russian Republics as ' Iranian Turks '. They derive from central Asiatic Stocks, but probably contain by intermarriage a considerable Iranian element, especially in Azarbaijan.

1.3.2. Th Kurds

The Kurds are an *ethnic* group inhabiting adjacent mountainous regions in western Iran, northern Iraq and southeastern Turkey. There are also small numbers of Kurds in Syria, Jordan, the Soviet Union, Afghanistan, and Pakistan. There are today about 9,000,000 Kurds of whom over 3,000,000 are in Iran.

It is only about the beginning of the Christian era that the Kurds become recognizable as a distinct people. There is considerable disagreement as to their origin and privious history. Nikitin (1943) has summarized the views of numerous authorities and supports the theory of Minorsky (1945) that the Kurds originated as an amalgam of two related tribes, the Mardoi and the Kyrtioi, speaking related Median dialects. If we accept these views then the Kardoukhoi whom xenophon encountered in the country which is now Kurdistan were not the main or sole ancestors of the Kurds (but migrated to become the Kartvelians of the Caucasus). We may, however, regard the Kurds as descended at least in part from the Medes whose king Cyrus had his capital at Sanandaj, now the chief town of Iranian

Kurdistan. No adequate anthropometric survey of the Kurds exists, and we are dependent almost entirely on verbal descriptions from which it is clear that there are considerable variations of physical type from tribe to tribe. It is certain that at least some of the Kurds are blond and blue-eyed while others are much darker. In view of the growing relative importance of the blood groups and other inherited blood factors in characterizing populations and defining their inter relationships, it had for many years been realized that such a study of the Kurds would be of great importance. There was, however, a further reason for wishing to obtain such data. A numerous and important group of Jews, the Kurdish Jews, formerly lived in Kurdistan. Some still remain but many have migrated to Israel.

The Kurds follow the sunnite branch of Islam, an important feature which sets them apart from most of the remaining population of Iran. On the whole, however, linguistic affinity may have been a greater force than religious differences in the relation between the two people. The Iranians seem to have had less trouble with the Kurds than have their sunnite neighbours, the Turks and Iraqis. However, the Kurds have been a difficult element in Iranian history. In the seventeenth century Shah Abbas found it necessary to move a part of the tribe to Khorasan, partly to defend the area against Turkman raiders, but partly also to break up a dangerously concentrated force in the west. The Kurdish communities of today vary from highly nomadic tribal groups to settled town dwellers.

1.3.3. The Lurs

South of the Kurds in the Zagros chain, the Lurs still pursue a largely tribal existence. Together with the closely

associated Bakhtiari, they are said to number somewhat less than half a million. They live in Luristan. Their language is a special dialect of Persian and they are shiites. 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.

1.3.4. The Arabs.

Several Arab tribes are found in the Zagros and many settled Arab villages and towns are found along the coast of Persian Gulf. The Arabs scattered through Persia are, like the Turks, no longer a racially pure element. In Khuzistan, where they are most homogenous, their affinities are with the Arabs of southern Iraq, themselves a mixed people with a preponderance of Caucasian elements. The Arabs of Khuzistan region are most probably the original population of the region. They are not mixed with other local population groups. However, in the course of history they must have become intermixed to a certain degree with negro elements, as can still be observed from the form of hair, nose, and skin colour.
The existance of a negrito type in southern Iran, said to be of 'Sumerian' or Dravidian origin and distinct from imported negro stocks, is still a matter of dispute. There are about two hundred thousand Arabs in Iran. The Iranian Arabs are Sunnite by religion.

1.3.5. The Bakhtiaris

All along the Zagros mountain-range from Azarbaijan to the Persian Gulf, pastoral tribes move with their herds back and forth every year over the spine of the mountains between summer ranges in the high valleys of the east and winter ran-

ges in the low foothills of the west. A segment of the Zagros about 150 miles long and centered due west of Esfahan is the home land of the Bakhtiari tribe. Some Bakhtiari tribesmen speak a dialect containing many words of Arab origin not found in other persian dialects; others speak a Turkish dialect differing little if any from that of the neighbouring Ghashghai tribesmen.

The Bakhtiari were until 1942 an autonomous political unit: they still have a common tradition of unity; and they reveal their feeling of belonging together in legends of a common origin. The Bakhtiaris are nominally shi'ah Moslems.

1.3.6.The Ghashghais

The Ghashghais occupy a region south of Shiraz. They are a group of Turkish origin with a language of their own, and a Mongol-like (very often a Bakhtiari-like) habitus. Their Mongoloid features are stronger than for instance those of the Ottoman Turks in the N.W. of Iran, who are perhaps more a product of intermixing with the original ancient population of Asia Minor. According to the tribal leaders, the Ghashghais were brought to the province of Fars by Shah Abass in the 17th century from what is now Russian Azarbaijan. Some Iranians state that they are of Seljuk Turkish origin; however, the Khans emphasize that although their language is a Turkish dialect, it was probably aquired in the not too distant past,and that their music and legendary tales are non-Turkish. Other Iranians believe that the Ghashghais originated from one of the Mongol tribes of Turkemanistan in Russia.

The Ghashghais are primarily a Turkish-speaking group,though a few Arab and perhaps even some Persian-speaking bands have allied themselves to the group. They are reported to have about

thirty-three thousand families in the area of Fars, though their numbers may have been much reduced lately by defection to other groups. Their annual migration from winter to summer pasturage is the longest among Iranian nomads, extending almost from the area south of Esfahan to the Gulf. They follow the shi'a branch of Islam.

1.3.7. The Mamassanis

The Mamassanis are a branch of the Lur tribe in southern Iran. According to the Khan, this tribe migrated from the Kurdistan region more than 800 years ago.

1.3.8. The Basseris

The Basseris are a tribe in the province of Fars near Shiraz. Their history is sparse with the exception of the belief that they have always occupied their present lands. They are variously described as Arab and Persian.

1.3.9. The Baluchis

The Baluchis of Iran are part of the larger group which forms the major part of the population of Baluchistan in west Pakistan. They speak an Aryan language akin to Persian and are quite conscious of bonds with Iran. However, they are sunnites by religion (though with several shiite modifications.) They may number as many as a million in Iran, in an area streching from near the Gulf of Oman to the province Sistan on the Afghan border.

1.3.10. The Brahuis

The Brahuis in Iran represent a very small fragment, under one hundred thousand, of a much larger group in Pakistan and Afghanistan. They live in the neighbourhood of Sistan and speak a Dravidian language, unrelated to any other language or dialect in Iran.

1.3.11. The Turkmans

Historically, Iran has been much troubled by the nomadic, raiding Turkmans, who have crossed the Russian border for many centuries, wreaking havoc on the Persian inhabitants of the area. With rather broad heads, Mongoloid eye folds, and straight hair, not always dark, they present a markedly different appearance from other Iranians. No accurate estimates of their numbers are availabe; their distribution is throughout most of northern Khorasan and down into the eastern part of Mazandaran.

1.3.12. The Assyrians

The Assyrians were originally an Arab-semitic agricultural people, who in ancient times occupied (several thousand years B.C.) the area between the Euphrates and Tigris rivers. They conquered extensive areas of the Middle East and became the elite class in this region, who did not intermix with the conquered peoples. In 175 A.D. approximately they were converted to Christianity. In 650 approximately their country was conquered by the Moslems, and the Assyrians fled, partly to the mountains where they became perhaps the ancetors of some of the Kurdish tribes, and partly to Persia where they maintained themselves as a separate group. Upto now this group has maintained its own language and script, quite different from Iranian and related to Arabic. The Assyrians are probably not a pure "race" as Christian Iranians are called " Assyrians" and taken up in the Assyrian Community. The christian Assyrians, live chiefly in the neighbourhood of Lake Urumieh, numbering about twenty thousand. Their language, of the semitic group, is distantly related to Arabic; their religion, of the

Nestorian branch of Christianity, provides a great Cohesive force.

1.3.13. The Armenians

Some Armenians, as mentioned, live in north western Iran, near the borders of their traditional native land. Others were settled in Julfa by Shah Abbas in 1604 across the river from Esfahan where they engaged their skills in the production of brocades and rugs in the royal factories. Still others live in Tehran and other major cities. Their total number is probably about sixty thousand. Their language is of the Indo-European group, but not at all closely related to Persian. They were not encouraged to intermarry with the Moslem group. Even today, those Armenians who have intermarried live outside the area of Julfa.

1.3.14. The Jews

Very little is known about the history of the Jews in Persia and about the social and cultural conditions of their life. It is definite, however, that a Jewish community was established there during the Babylonian exile(Levy, 1948 , Fishel, 1948). This community appears to have existed continuously since then. Its members sought to guard their Cultural individuality, carefully guarded themselves against all foreign influences and observed all of their religious laws, despite many vicissitudes during which many of them were killed or converted to Islam. Nevertheless, it is difficult to assume that the Persian Jewish community isolated itself so successfully that it can be considered anthropologically pure. The mother tongue of the Jews today is Persian but most of them jealously preseve their ethnic group integrity (including religion) through inbreeding and segregation.Jews in

Iran number perhaps seventy-five thousand with the greatest concentration in Tehran, Hamadan, Esfahan, and Tabriz.

1.3.15. The Zoroastrians

The Zoroastrians are the closest to the original Persians now in existence and today number only 15 to 20,000. Most Zoroastrians were born in Yazd and now live in Yazd, Kerman and Tehran. The Parsis of India, of similar origin, are descendants of Zoroastrians who fled to India at the time of the Islamic conquest. They are probably different ethnically from the Zoroastrians of Iran in that, according to local belief, those who left Iran were mainly soldiers.

Zoroastrians have a strict unwritten code:

They do not proselytize, outsiders are not accepted into the religion, and if a Zoroastrian marries into another group,neither he nor his children are considered Zoroastrian.

Zoroastrians believe that they were untouched by the Mongol invasions of the 13th-14th centuries. They most certainly were not the defending forces. If this is true, it suggests that the Zoroastrians, with their extensive Empire, were also reservoirs of the blood group, B gene. The importance of this group for future studies is incalculable. Unfortunately, if the present rate of loss of its members continues, they will probably be extinct within the next 50 years. The followers of the Zoroastrian religion in India form two distinctive groups, the Parsis and Iranis, numbering respectively just over 100,000 and approximately 6,000. The Parsis migrated to Gujarat at the end of the 7th century AD after the fall of the Sassanian Empire in Persia under the impact of the Moslem invasion. Since that time they have maintained a high degree of sociocultural isolation from other Indian communities, but

they have spread from Gujarat to other areas, chiefly in the north-west. Today approximately 70% of the Parsis in India live in Bombay having migrated there from the end of the 17th century. By contrast the Iranis are derived from the remnants of the Zoroastrian. Communities in the Yazd region of Iran who have migrated to India in the last 150 years and now live predominantly in Bombay, Poona and Dahanu regions.

1.3.16. Other ethnic groups.

The north-west corner of Iran is a potential trouble spot because of its proximity to Turkey and Russia. This largely mountainous area, Azarbaijan, before the nineteenth century included what is now the Soviet Republic of Azarbaijan in the USSR. The dominant people are called Azari or Azarbaijani. A small part of the area, near the Turkish border, is inhabited by Assyrians.

The Azari are a Turkish-speaking group, numbering about two million, indentical in language and certainly origin with the inhabitants of the Soviet Republic of Azarbaijan, immediately across the Russian border.

1.4.Demography and population density.

Iran is located in a region of complex population movement throughout the whole historical and protohistorical period. Some of this movement is documented, other parts we can only infer: the result has been to leave the population of Iran divided into many religious and ethnic groups of great interest not only to the Iranian people themselves but also to the researchers of human population structure and evolution.

We know little of the earliest inhabitants of what is now called Iran though there is evidence of human occupation from

palaeolithic times onwards, and increasingly abundant information is being discovered about occupation during the post-pleistocence or proto-neolithic era (Field,H.,1939,1959;Braidwood et al., 1961; sunderland,E., 1968; wilber, 1973).At some time during the second millennium B.C., Aryan invaders from the north or north-east entered the area and left their mark in myth and language and in the physical appearance of the peoples. Olmstead (1948) interprets the archaeological and traditional history of the Iranians as indicating that Aryans settled the southern shores of the Caspian sea in what was called Hyrcania (later Mazandaran) the capital of which was not far from Asterabad Bay at the south east corner of the Caspian. From the coastal area the Aryans crossed the barrier of the Alborz Mountains and spread out over the Iranian Plateau.

The Aryan invaders were succeeded by many others including Assyrians, Greeks, Arabs, Turks and Mongols. The present day populations are descended from the remnants of these successive waves of peoples who became assimilated with the original inhabitants. This process of assimilation seems to have progressed further in the more settled groups: it has had the least effect on nomadic groups (Arasteh, 1964). In addition to many invasions, northern Iran, particularly, has been on major trade routes and its people have been influenced, at least in part, by the repeated passage of caravans. With their assortment of exotic wares and of people bringing new ideas, and from time to time, new genes. of these trade routes, by far the most important was that running east to west passing close to modern Tehran and forming the end of the old "silk" road which connected China with Europe.

The total population of Iran is about 36 million.since Iran covers a vast territory, this means there is a fairly low numerical density. Movements of population in and out of the country (i.e. external migration)are not of great importance.But, on the other hand, the rural exodus, the drift from the country to the towns, presents a highly intractable problem; and the seasonal migrations of nomads are also considerable. It is the disposition of relief which could be held mainly to determine the geographical distribution of the population.

The centre of the country is largely empty and life has been driven either towards the exterior or towards the interior of the mountains, to the points where there is an adequate water-supply; people live among the foothills of all three mountain ranges, and each group has settled half in the plain and half on the mountain side. There are extraordinary variations in density. The most heavily populated cities are:Tehran, the shores of the Caspian sea, Tabriz,Shiraz, Esfahan, Mashhad, etc.

By way of simplification, the most heavily populated areas may be classified as follows:

the shores of the Caspian sea, the watered mountain regions, the naturally watered or irrigated oases of the plateau.

The elements which determine this distribution are the same that influence every dispersal of men throughout the world: natural factors and human factors(sociological, economic, and historical). The most important natural factors affecting the population distribution throughout Iran,as in a number of other developing countries, are water and temperature.

1.5. Consanguinity

Consanguinity and endogamy are among the characteristic features of marriage in Iran. Apart from considerations of convention such as the very frequent marriages between relatives,geographical, ethnic, religious, cultural, social and economic factors exert considerable influence.

There exists different rate of consanguineous matings in Iran as the results of variations in the above mentioned factors. The types of consanguineous marriages also differ among Iranians. First cousin matings occur more than the second cousin marriages among most population groups. Uncle-niece matings happen only among the Jews. This type of mating is forbidden in Islamic and Christian rules.

The incidence of consanguineous marriages ranges from 24.46 (Esfahanis) to 31.59 (Tehranis) percent in Urban; 30.59 (Azaris) to 65 (Bandaris) percent in rural; 18 (Turkmans) to 73.46 (Ghashghais) percent in tribal; and 2.81(Christians) to 25.39(Jews) percent in religious groups of Iran (Farhud et al, in preparation).

On the whole the observed frequencies of the consanguineous matings in Iran appear to be higher than that of 30 percent in the Middle Eastern populations (Frazer Roberts and Pembrey, 1978).

1.6. Religion

In religion, however, Persia has played an important part, a part not wholly laid aside, and an outline of her religious history is here appropriate. Zoroastrianism was the ancient faith of Iran and is important because of its similarity to Judaism and Christianity. A phase of this religion known as Mithraism penetrated into the Roman world during the early Christian ages and spread so rapidly in many parts of Europe that altars were set up and cave-temples built to celebrate the mysteries of the Persian divinity Mithra and to glorify this personification of light, the sun, and truth. Furthermore, the system of Manichaeism, which sprang up on Persian soil, was powerful enough to compete for a time with Neo-platonism and Christianity for the religious and intellectual supremacy of the Roman Empire. Islam is the religion of Persia to day, but Persia belongs to the Shiite sect of the faith and acknowledges Ali, Mohammed's first cousin and son-in-law, as the prophet's successor in opposition to the Sunnite branch of Islam. Iran is in fact the chief representative of Shiism and has been largely instrumental in the growth of this factional movement which divides the Islam world.

Except for one or two hundred thousand Christians, Jews, and Zoroastrians, all the inhabitants of Persia are Moslems, and Shia Islam of the" Jafari"form is the official religion of the state.

Persia is peculiar in that the dominant form of Islam is

not the Sunnism which is supposed to be "orthodox" in other Moslem countries. Most of the Kurdish, Turkomans, Baluchis and the Arabs people inhabitants in the Kurdistan, Gonbad, Baluchistan and Khuzistan, are Moslem Sunnis, More than two million of the 36 million Persians being Sunni Moslems.

Since the Islamic conquest in the seventh century A.D., vast areas of Iran have been overrun by Arabs, Turks, Mongols and Afghans. These peoples arrived as Moslems or were afterwards converted to Islam. Large numbers of the invaders remained and their descendants today form the tribes in Iran ; however, many of their progeny are now mixed with the Moslem population.

1.7. Language

Persian or Farsi is the official national and literary language of Iran, spoken by three quarters of the population. Arabic, a number of Turkish dialects, and a few other languages are spoken in Iran, in addition to Persian, which is ,of course, the most important language. It is also, as the official language of the country, known one way or another by most of the other inhabitants. Persian is a member of the Indo-European family of languages and belongs to the Indo-Aryan(Indo Iranian) branch of this family. It is spoken in various dialect forms by a majority of the population. It is an aryan language, derived from Middle and old Persian, but has been modified by Arabic in general vocabulary and in the constructions of written prose. The present tendency is towards the expulsion of Arabic words and forms in favour of Persian terms. In sound, Persian is sonorous and has been called the most musical of all languages. It is written in Arabic characters, but in an elementary form it is capable of Romanization.

Inhabitants of various cities can be identified by minor variations in pronunciation and choice of words. Most Persians believe that the "purest" Persian is spoken in Shiraz. These regional differences do not seriously affect mutual understanding.

Several provincial dialects are spoken,especailly in Gilan and Mazandaran, and there are several small communities speaking patois of their own. The speech of Gilani(Gilaki) and Mazandarani is far enough from standard Persian as to be almost incomprehensible to a Persian.

Two other languages, that of the Kurds on the west and the Baluchis on the east, are related languages of the Indo-European group, Kurdish is distinct from Persian, being directly connected neither to old nor to middle Persian. Of the three principal forms of the Kurdish language two are spoken in Persia, Kurdi and Gorani, but these two have numerous dialects.

Baluchi is another aryan language akin to old Persian and often modified by modern Persian. Luri, the language spoken by most Faili Lurs of Luristan proper, by the Bakhtiari,Mamassani tribes is closer to old Persian than is Kurdish. Brahuis of the eastern border speak a Dravidian language,unrelated to any other language of Iran.

The Arabic dialects spoken by the Arab tribes of Khuzistan, along the coast of the Gulf and of Mokran, and,with varying mixture of Persian or Turki elements, by Arab groups of the interior such as the Khamseh tribes of Fars, are modern variants of the same older Arabic that formed the base of the classic literary language and all the dialects from Morocco to India. Arabic is a semitic language,related to Hebrew,

Syriac, and Ethiopic. There is thus no clear relationship between Arabic and Persian, but conquest and the legacy of the Islamic religion and culture have resulted in an enormous infiltration of Arabic words into Persian. Arabic words actually incorporated into Persian have been modified to fit the Persian sound pattern.

The Turkish languages, unrelated to Arabic or Persian , belong to the Uralic-Altaic family, which includes languages spoken as far west as the European shores of the Bosphorus and east to Mongolia. Turkish dialects are spoken in Iran: Azari, the Turkish language spoken by the Turks of Azarbaijan, north west of Iran, is grammatically close to the Ottoman Turkish of Anatolia. Various tribes, including the Ghashghai and Khamseh, in the south, speak Turkish dialects. The nomadic Turkmans of Khorasan in the north east, also speak a form of Turki, which is much closer to east Turki.

These languages are not written to any extent in Iran . In Azarbaijan, however, the language is written as well as spoken, and though the educated classes understand Persian, they prefer to be addressed in their own language. Many Turkish words have been incorporated into Persian, and vice versa.

Of minor nationalities, the Armenians retain their native language. Armenian is an Indo-European language, but is entirely incomprehensible to a Persian. Almost all Armenians in Iran speak Persian in addition to their native tongue.
The Assyrians around Lake Urumieh speak Aramaic dealects related to the older East Syriac, an ancient language of the Semitic group, intermingled with Persian, Turki and Arabic words. With their religion, this is a great binding force, which

unites them with the Assyrians of Iraq, and makes them a foreign minority in both countries.

Jewsspeak Persian and also a Persianized Hebrew.

CHAPTER 2.

AN ACCOUNT OF POLYMORPHIC SYSTEMS EMPLOYED IN THE PRESENT INVESTIGATION

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, and the frequency of the genes and phenotypes can be used in classifying and comparing populations. Genetic traits now commonly investigated in population surveys include the major blood group antigens and the serum protein and red cell enzyme polymorphisms. Search for genetic variation in human populations is important because genetic marker systems constitute a prerequiste for several kinds of genetic studies, particularly for the attempts to map the human genome.If genetic variants governed by one locus shall be efficient as a maker system, more than one of the alleles at the locus in question must be relatively frequent in the population under study. Recent research has demonstrated that approximately 10 percent of randomly selected gene loci controlling biochemical traits in man show polymorphic variation and that nearly all gene loci reveal the presence of rare mutant forms with frequencies of 1 per 1000 or less (Harris, 1970; Harris and Hopkinson, 1972). The ability to detect such genetic variants provides a powerful tool for tracing population affinities.

The study of serum protein and red cell enzyme polymorphisms by electrophoretic techniques has become a valuable tool for the modern physical anthropologists. It contributes to the description of genetic stocks, helps to trace migrations and exchanges, and may confirms the genetic isolation of some

groups. As more refined techniques have developed, a large number of new alleles have been demostrated in many serum protein and red cell enzyme systems. This permits a more precise definition of a population's genetic composition. When many populations have been studied, maps can be drawn showing the distribution of the various alleles. If these maps show that clines of gene frequencies cross linguistic, cultural, and racial barriers but are correlated with geographical factors, one can suppose an environmental (selective) effect which may be as important as migration and genetic exchanges(Bodmer and Cavalli-Sforza, 1976).

Little or nothing is known regarding the adaptive value of erythrocyte enzyme polymorphisms, except for the recognized role of G-6-PD. But if the allelic frequencies of most of these systems are represented geographically, it is observed that their distribution within mankind cannot be considered merely as random phenomenon. Furthermore, it is known that, in general terms, for each polymorphic enzyme of differing phenotype there exists a different enzymatic activity, as a result of which it is reasonable to suppose that, in the presence of certain biotic and/or abiotic factors, an adaptive role attaches to these alleles. Since the first step in ascertaining whether this is true is obviously to gather the largest possible volume of data regarding genetically and/or geographically heterogeneous populations; a number of surveys are now being conducted to that end, especially of populations still heavily exposed to the natural environment.

2.1. Blood group antigens.

Since Landsteiner's work (1900-1901) on the ABO groups, other separate blood groups systems have been discovered, in

all of which inheritance has been shown to conform to Mendelian principles. The ABO blood groups of man have been intensively studied and the distributions well-mapped because of the dramatic ill-effects arising from incompatibel transfusion. Remarkably, the incidence of haemolytic disease of the newborn arising from ABO incompatibility is low, but some specialists are doubtful of the sensivity of the standard techniques used for its detection. The Rhesus group in all its complexity has received much attention in connection with haemolytic disease of the newborn, whilst other systems such as Duffy, Kell and Kidd are known to incite haemolytic antibody formations but the specific conditions for this to occur are unknown. All these serological systems and others besides, are variable in human populations.

2.1.1. The ABO blood group system

Three of the ABO blood groups were discovered in 1900 by Landsteiner. He described the agglutination which occurred when red cells of one individual were exposed to the action of serum from another (Landsteiner, 1900, 1901). He found that in some cases agglutination occurred, whereas in others there was no reaction.On the basis of these agglutination reactions Landsteiner was able to divide human beings into three distinct groups.Indication of a fourth group (AB) were found in 1902 by Decastello and Sturil but they tested only the serum which they described as ' without group '. We have been unable to ascertain who first described the reactions of AB red cells (Mourant, et al. 1976), but the situation which emerged within a few years was that human

beings are divisible into four classes according to the reactions which their red cells show with normal human sera. The classification of four groups- A, B, AB and O, is based on the presence upon the surface of the red cells, of either, both, or neither of two substances known as A and B substances, the active parts of their molecules being of a polysaccharide nature. In serological terms the A and B substances are described as antigens or agglutinogens.

The red cells of an individual possess either one or both, or neither of the antigens A and B. A cells carry only A substances, B cells only B; AB cells carry both and O cells neither. All or very nearly all the red cells of an individual are qualitatively indentical in blood group constitution and reactions..

Human sera may contain either,both, or neither of the corresponding antibodies or haemagglutinins, 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 anit-A in a serum containing it can attach itself to the A substance on A or AB cells, and it then alters their properties in such a way that they stick to each other, or agglutinate. Anti-B behaves similarly with B or AB cells.

No blood contains one of these antibodies which will cause its own red cells to agglutinate, but subject to this restriction all possible antibodies are present in all bloods after the first few months of extra-uterine life. Thus all normal human sera contain those antibodies which do not react with the individual's own red cells, The serum of an A person contains anti B, that of a B person, anti-A. The serum of an O

person contains both antibodies, and that of an AB person, niether of them. The relations between genotypes,phenotypes, and antibodies are summarized in Table 2.1.1.a., which forms the basis of AB testing, known sera containing anti-A and anti-B, and know A and B cells, being used as reagents to test unknown bloods.

TABLE 2.1.1.a.

THE ABO BLOOD GROUPS ANTIGENS AND ANTIBODIES

Blood groups (Phenotype)	Blood groups substances on red cells	Antibodies present in plasma(or serum).	Genotype
O	none	anti-A,anti-B	OO
A	A	anti-B	AO or AA
B	B	anti-A	BO or BB
AB	A and B	none	AB

The inheritance of blood group characters, based on Mendelian principles, was reported by Dungern and Hirszfeld(1911). But the genetics of the ABO system were first correctly worked out by Bernstein(1924) who showed that the four groups were inherited by means of a set of three allelomorphic genes A,B and O(for the frequencies of which the symbols p, q and r are commonly used). Of these, A and B each gives rise to a characteristic antigenic (and ultimately biochemical) structure, the A or the B antigen, while the O gene behaves as an amorph, not giving rise to any antigen peculiar to itself. These antigens are usually

sought on the surface of the red cells, but are in fact found widely distributed elsewhere in the body.

Thus, the A gene, when present at all, on one or both of the chromosomes concerned, determines the presence of the A substance on the red cells. The B gene similarly determined the presence of B antigen. The 0 gene give rise to neither antigen, and in the heterozygote with an A or B gene it has little influence on the expression of that gene in producing the A or B antigen, but homozygous 00 bloods carry neither antigen. As the 0 gene is really an amorph, therefore group A and B behave as dominant traits. Thus group A blood may be of genotype AA or AO, B blood BB or BO, but group 0 blood can only be of genotype 00 and AB blood of genotype AB. Thus, the four blood groups represent six genotypes as shown in Table. 2.1.1.a. Because of the amorph status of the 0 gene, the genotypes AA and AO are indistinguishable by any routine tests on the cells of the individual, as are the genotypes BB and BO.

The anti-A and anti-B antibodies in human serum are still often described as 'naturally occuring', but it is now generally accepted that they are the result of immunization in infancy by substances closely related to the A and B antigens, and derived from the environment through the respiratory or alimentary tracts. It may appear at first sight surprising that these antibodies, the formation of which is stimulated by a variety of environmental antigens, should apparently be so precisely tailored to the blood group antigens of other human beings. It must, however, be remembered that the normal individual is tolerant of, (or does not make antibodies to), antigens forming part of his own body,(or

at any rate parts which are accessible to plasma antibodies). Thus, for example, any antibody produced in response to an A-like antigen derived from the environment will be so constituted as to react as well as possible with A-like antigens but subject to the restriction of not reacting with the individual's own antigens, (which may include substances which are more or less A-like, present in all individuals, whether or not they possess the gene-determined and biochemically sharply defined blood group A antigen). Such an antibody may also react with non-human A-like substances, such as red cell antigens of other mammals, but this will not show in tests restricted to human red cells and human plasma or serum. Similar considerations apply to B and anti-B.

The sub-groups of A and B

Considerable numbers of variants of the A antigen are known, most of which are rare; the B antigen is less variable but several rare variants have been described.

At the variant level, the most important distinction is between A_1, the commonest antigen, and A_2 which has a frequency of several percent in most European, African, and West Asiatic populations. Though the distinction has been known for about 50 years, its basic nature is still not completely understood, but most of the facts are covered by the following conventional explanation. Thomsen et al(1930) observed that there were two varieties of the A antigen, A_1 and A_2, allowing the blood groups A and AB to be classified respectively as A_1 and A_2, and as A_1B and A_2B. Both types of antigen react with the ordinary antibody anti-A, but only A_1 reacts with anti-A_1, while A_2 fails to do so. Thus, 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. Reaction of antigens is shown,as usual, by agglutination. Anti-A_1 is present in the serum of most B persons together with ordinary anti-A.

The A_1 and A_2 antigens are produced by corresponding allelomorphic genes, so that what we have called the A gene is really of two possible kinds, A_1 and A_2. A_1 is dominant to A_2 and 0; A_2 is dominant to 0; neither A_1 nor A_2 is dominant to B. In the genotype A_1A_2, the A_1 gene causes the production of A_1 antigen, and thus the genotypes A_1A_2, A_10 and A_1A_1 are indistinguishable, by methods at present available, since all react both with anti-A and anti-A_1.

Thus, the recognition of four alleles -A_1, A_2, B and 0 - leads to the expectation of ten possible genotypes and six distinguishable phenotypes.

The complete genotypic system of the A_1A_2B0 groups is shown in Table 2.1.1.b.

TABLE 2.1.1.b.

THE A_1A_2BO GENOTYPES AND GROUPS

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

A considerable number of other variants of blood group A are known; they are mostly very rare but at least two of them are not uncommon in Africans, and possibly in other non-European populations.

A type intermediate between groups A_1 and A_2 ($A_{1,2}$ or A_{int}), reacting positively but weakly with anti-A_1, was described by Landsteiner and Levine(1929) and first shown by Wiener et al

(1945) to found especially in Negroes. In addition, many sub-groups of A have been described which are negative with anti-A_1 and weaker than A_2 in their reactions with anti-A. There has been some confusion as to their identity and nomenclature. One such group has been shown to be present in South African Negroes (Bantu). Its distinctiveness has now been established by Brain (1966) who has called it A_{bantu}. Though he is able to define four fairly distinct types, A_1, A_{int}, A_2, and A_{bantu}, among the Bantu, the sharp distinction between the A subgroups, found among Europeans, certainly breaks down in the Bantu; a similar finding has been made by Vos and Comley(1967) in West Australian aborigines. As group A_2 is intermediate in several of its properties between A_1 and O, it would be of great interest to know whether this applies to its associations with disease, but unfortunately very few investigations of associations have determined the sub-groups of A in their patients.

2.1.2. The Rhesus blood group system

The discovery of the Rhesus or Rh blood group system by Landsteiner and Wiener in 1940 was the most important discovery in the blood group field since the ABO groups. They showed that the antibodies, produced by injecting red cells of the monkey Macacus rhesus into rabbits and guinea-pigs(produced by immunizing them) not only agglutinated the red cells of the monkey but also about 85 percent of the people of European descent. They classified these 85 percent whose red cell were agglutinated by rabbit anti-rhesus serum as Rh-positive and those showing no agglutination as Rh-negative. The antibody was shown to be indistinguishable from that reported in Levine and Stetson's case of 1939.Finally, it was shown by Levine et al in 1941 that the destruction of the red cells

in the new-born was the result of Rh blood group incompatibility between a Rh-negative mother and a Rh-positive child.

Intensive work on this system led to the view that the Rh groups were not as simple as they seemed at first. Soon after the discovery of the original anti-Rh (the anti-rhesus monkey-guinea pig serum actually defines a different antigen to D as found in man) other reactions were noted and antibodies clearly connected with the Rh system, but having different specificities from the original one, were discovered, and it became necessary to recognize subtypes of the Rh groups.

The nomenclature used to communicate the various findings of the 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).

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 2.1.2. a .

TABLE 2.1.2.a

COMPARISON OF THE FISHER-RACE LINKED GENE THEORY AND THE WIENER MULTIPLE ALLELE THEORY

Gene Complex	Symbol	Anti-gens	Gene	Agglu-tinogen	Blood Factors
CDE	R_z	C,D,E	R^z	Rh_z	rh',Rh_o,rh"
CDe	R_1	C,D,e	R'	Rh_1	rh',Rh_o,hr"
cDE	R_2	c,D,E	R^2	Rh_2	hr',Rh_o,rh"
cde	r	c,d,e	r	rh	hr',hr",hr
cDe	R_o	c,D,e	R^o	Rh_o	hr',Rh_ohr",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"

The system of antigenic and genetic notation most commonly used is the CDE system based on the work of Fisher and Race (1944); but Wiener and others describe it in terms of Rh and R with indices and suffices. As Race and Sanger (1968) point out, disagreement is almost entirely on interpretation and not on the observed facts. They also say, with reference to the CDE notation, ' we think that this is at present the only notation in which a detailed account can be communicated'. The Rh groups are thus regarded as determined by a set of alleles at three very closely adjacent loci on each of a pair of chromosomes, which may be called the C, D, and E loci.

The principal antigen of the system is know as D, determined by a gene D, the allele d of which behaves as an amorph. Very closely linked to the Dd locus are two other loci each

characterized by a pair of major alleles, Cc and Ee respectively. Each of these four genes gives rise to a correspondingly named antigen.

As only one of each pair can be carried on each chromosome, there are thus eight possible chromosomic combinations of genes, all of which are known to exist, but of which CDe, cDE, cDe and cde are the commonest. The eight alternative Rhesus gene combinations are shown in Table 2.1.2.b.

TABLE 2.1.2.b.

ALTERNATIVE RHESUS CHROMOSOMES

	D	E	CDE
		e	CDe
C		E	CdE
	d	e	Cde
	D	E	cDE
		e	cDe
c		E	cdE
	d	e	cde

When these letters were assigned by Fisher there was no evidence as to the serial order of the loci, but there is now strong evidence that it is DCE.

At each locus two common alleles may be distinguished as well as a considerable number of rarer ones. In general, each allelic gene gives rise to a distinct antigen, corres-

ponding to a distinct antibody which causes agglutination of the red cells containing that antigen. Some of the rarer alleles, however, differ only quantitatively in their effects from one or other of the common alleles.

The D locus can be occupied by either of two common allelic genes D and d. Of these, D gives rise to the D antigen which is the strongest antigen of the system, both in its immunizing potential and also usually in agglutination for which it is responsible. The d gene, on the other hand is, like O in the ABO system, an amorph, giving rise to no specific antigen. There are, in addition, a number of other genes which are known collectively as D^u(Stratton, 1946). The D^u antigen produced by the genotypes $D^u d$ and $D^u D^u$, gives rise to agglutination by some but not all anti-D sera, and those agglutinations which do occur tend to be weak. Wiener and his colleagues (1959, 1962) have, however, described a series of specific weak variants of the D antigen which they call Rh^A, Rh^B, Rh^C and Rh^D, of which the first three at least may be regarded as forming part of the D^u series, but precisely definable by reactions with appropriate antisera. They give rise to the possibility of immunization of persons carrying one component of the D cc complex, by another component of the same complex, a phenomenon which has been reviewed by Tippett and Sanger (1962).

The Gonzales (Go^a) antigen of Alter et al (1962) is present in about 2 percent of American Negroes. It is recognized by a positive agglutination test with a specific human anti-Go^a serum. The antigen was originally thought to belong to a completely new blood group system, but Lewis et al (1967) have shown that it is part of the Rh system, and is apparently produced by a variant of the D gene in the cDe complex. All Go^a-

positives are strongly positive with most anti-D sera, but a number of Go^a-positives have produced an antibody which agglutinates all D-positives except those whose only D gene is of the Go^a-variety. Tippett has shown that the antigen fits into the class of D variants described above and, according to Race and Sanger(1968), 'The antigen maybe pictured as replacing part of the usual D structure previously thought merely to be missing'.

At the C locus there may exist either of two common alleles, C and c, each giving rise to a specific antigen. Rarer specific alleles are C^w and C^x and one quantitative one like D^u, known as C^w. A further variant, more nearly resembling c, has been described by Huestis et al (1964).

At the E locus the common alleles are E and e. The rare antigen E^w (Race and Sanger, 1968) is recongnized by a specific anti-E^w antibody, and depends on a gene E^w.

However, with the exception of D^u and c^w, all additional alleles are unusual or rare in Caucasian populations.

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 the various human populations. There are wide racial differences in the frequency of Rh gene complexes. Detailed information regarding racial differences has been reported by Mourant et al (1954, 1976).

From a clinical point of view the Rhesus or Rh system is by far the most important of the blood group systems other than ABO. Because of its greater clinical importance it has been the subject of much more extensive and detailed research, and also of intense controversy, mainly centring upon its genetics.

Rh incompatibility is the main cuase of haemolytic disease of the newborn, and a major cause of transfusion reactions. Incompatibility with respect to the D antigen is the main cuase of haemolytic disease of the newborn, but Cc and E**e** incompatibilities are rare causes, as they are also of transfusion reactions. Unlike the antibodies of the ABO system, each of which is universally present in persons lacking the corresponding antigen, antibodies of the Rh antigens are virtually never found except as a result of immunization by pregnancy or transfusion, and reactions appear only at a second exposure to the antigen. This applies also to all the other blood group systems except the ABO. Very few searches have been made for any other kinds of diseases associations of the Rh groups, and nearly all of these are confined to a comparison of the D-positive and D-negative types.

The Rh genetical complexes

Fisher's theory of the genetics of the Rh blood groups postulates the existence of closely linked sets of three genes, one from each of the sets of alleles mentioned above.The number of such complexes described in the literature is very large.

While the CDE formulae are essential for understanding both the genetics and the antigens, it is convenient to have a 'shorthand' notation for at least the commoner complexes, and for this purpose symbols based on those of Wiener are most convenient. In the Table 2.1.2.c the CDE genetical formulae of these selected complexes are set out, together with the 'shorthand' notation which is based on, but not indentical with, that of Wiener.

TABLE 2.1.2.c.

Rh GENE COMPLEXES FOUND IN POPULATION STUDIES, SHOWING THE CDE (FISHER) NOTATION AND THE USUAL ABBREVIATED NOTATION, BASED ON THAT OF WIENER.

Fisher's notation	Abbreviated notation
CDE	RZ
CDe	R_1
CdE	r^y
Cde	r'
cDE	R_2
cDe	R_o
cdE	r"
cde	r
$C^w DE$	R_Z^w
$C^w De$	R^w_1
$C^w de$	r'^w
$CD^u e$	R_1^u
$cD^u E$	R_2^u
$cD^u e$	R_o^u

Most of the antigenic manifestations of the Rh genes can be expressed in terms of separate antigens determined by allelic genes at three distinct though very closely linked loci. There are however, a number of antigens which cannot be attributed to any one of these three hypothetical loci. Nearly ev-

ery such antigen can, however, be explained as a joint product of genes at two adjacent loci on one chromosome.

2.1.3. The MNSs blood group system

There was a long interval between the discovery of the ABO blood groups and that of the next system, the MN, which was first described by Landsteiner and Levine(1927).This system depends upon a pair of allelomorphic genes which behave in nearly all respects as though they determined the antigens M and N respectively.

Antibodies against M and N are not usually found in humans, but are prepared by the injecting of 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. Thus, the two antigens,M and N were detectable on human red cells by means of immune rabbit sera, one or both of which were present on all cells.

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 antigenes on red cells. Thus there are three genotypes MM, MN, and NN and three corresponding phenotypes M, MN and N.

It has, however, recently been shown that the N antigen is a precursor substance, and that the so-called N gene is an amorph which leaves the N antigen unchanged, while the M gene in the heterozygote converts part of the N antigen into M, and in the homozygote converts nearly but not quite the whole of it.

In 1947 Walsh and Montgomery discovered the existence of a new antigen, S, which Race and Sanger found to be related to the MN system (Race and Sanger, 1949).

The S antigen was shown to be serologically different from M and N (Race and Sanger, 1947). Antigen S was shown not to be an allele of M and N but that it was related to MN as the alleles of Rh system are related. Using the analogy of Fisher's theory of the genetics of the Rh system, they showed that the S antigen was the product of one of a pair of allelic genes S and s, very closely linked to M and N.

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 the discovery of the expected antithetical antibody anti-s which agglutinated the red cells of homozygotes as well as heterozygotes was reported by Levine et al (1951), indicating that there are two antigens, S and s and three blood types, S, Ss and s. These findings confirmed the hypothesis that S and s form another pair of genes closely linked with M and N. 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. Table 2.1.3. demonstrates the genetical interpretation of serological reactions of the complete MNSs system.

TABLE 2.1.3.

GENETICAL INTERPRETATION OF THE REACTIONS OF ANTI-M, ANTI-N, ANTI-S AND ANTI-s SERA

(After Race and Sanger, 1950).

Anti M N S s	All 4 sera: genotype	First 3 sera: genotype or phenotype
+ - + -	MS/MS	MMS
+ - + +	MS/Ms	
+ - - +	Ms/Ms	Ms/Ms
+ + + -	MS/NS	
+ + + +	MS/Ns	MN.S
	Ms/NS	
+ + - +	Ms/Ns	Ms/Ns
- + + -	NS/NS	
		NN.S
- + + +	NS/Ns	
- + - +	Ns/Ns	Ns/Ns

Race and Sanger (1970) suggested that the linkage between the loci must be very close since crossing over has been shown to occur only very occasionally.

On a given chromosome any one of the four combinations MS, Ms, NS, and Ns could exist, but S was more usually linked with M than with N. To explain this uneven association it was necessary to postulate very close linkage.

In addition to the common genes M and N, a number of rare alleles are known at the same locus. The only one which is of anthropological interest is M^g, discovered by Allen et al

(1958). The antigen M^g reacts very weakly with anti-N and not at all with anti-M, but a specific anti-M^g is present in one percent or more of normal human sera.

M^c is an antigen intermediate in properties between M and N, reacting with some anti-M and some anti-N sera.The gene responsible is an allele of M and N(Dunsford et al, 1953).

The M^k gene appears to be an allele at the combined MNSs locus, and gives rise to none of the antigens M,N,S and s.It does, however, gives rise to a specific antigen to which a very weakly reacting antibody can be prepared in rabbits(Metaxas and Metaxas, 1964).

Two kinds of antiserum are known which define subtypes of M.Anti-M_1, corresponding to anti-A_1 of the ABO system, was found by Jack et al(1960), to be present in some human anti-M sera, but not in rabbit anti-M. The M_1 antigen is commoner in Negroes than in Europeans. If the antisera were more readily available, the subtyping of M would probably be of considerable anthropological interest. The M^v antigen is defined by a single human serum which reacts with all N and MN bloods and with about 1 in 400 M samples from Europeans. The latter are regarded as containing an M Variant, mostly of course in the heterozygous form. (Gershowitz and Fired, 1966).

A third allele, which determines the absence of both S and s, and is know as S^u, is not uncommon in Negroids.

The system is one of considerable complexity, involving numerous variants of M and N, and a variety of antigens determined by other closely linked genes. While the system is thus of considerable genetical and anthropological interest, it seldom gives rise to haemolytic disease of the newborn, or of transfusion reactions, and shows few disease associations.

Antigens due to genes linked with MNSs

A considerable number of antigens are known, each individually very rare, which in family studies have proved to be due to genes closely linked to the loci for MN and Ss. Because of their individual rarity they seldom occur together, and hence little is known as to possible allelomorphism among the genes concerned.

The Hunter (H^u) antigen was found as early as 1934 by Landsteiner, Strutton and Chase in 7 percent of American Negroes but only 0.5 percent of whites, all of whom were N or MN. Chalmers et al (1953) obtained evidence that the antigen was the product of a gene closely linked to Ns. It is possible that the anti-Hunter serum defines a sub-group of N rather than distinct antigen.

The Henshaw (He) antigen of Ikin and Mourant (1951) is found mainly in Africans. The He gene is almost invariably closely linked with NS in West Africans but with MS in South Africans. It is also sometimes linked with Ns or with Ms. The antigen does not appear to have been found, in tests controlled by comparison with known He-positives, in any populations not known on other grounds to be of African ancestry (Shapiro, 1956).

The Miltenberger complex of antigens (Mi^a,VW, Mur) is certainly due to a number of genes closely linked with MNSs, and may plausibly though not certainly be regarded as due to a set of alleles at a single locus closely adjacent to those for MN and Ss (Levine et al, 1951).

Of the other antigens known to have close linkage relations with MNSs, several are actually or potentially of anthropological interest. They are as follows: the st^a(stones) anti-

gen, the m^a(Martin) antigen, the Vr (Verdegaal) and N^a (Nyberg) antigens, the Tm antigen, the sj antigen, the Ri^a (Ridley) antigen.

2.1.4. The p blood group system

The p blood groups were discovered by Landsteiner and Levine (1927) in the course of the same investigations that led to the discovery of the MN groups. It was at first thought that only one antigen, p, was involved, determined by a gene p, the allele p being an amorph. Thus, bloods were classified as p-positive and p-negative, the positives possessing p gene. Thus, p-positives were either pp or Pp, and p- negatives pp. The antibody agglutinated approximately 75% of the population and these were termed.p+. Owing to the existence of bloods which reacted weakly to the early anti-p sera, the frequency of the two groups could not be established with certainty. Racial differences however, were recognized (Landsteiner and Levine, 1927, 1929) and the p antigen was shown to be inherited as a mendelian dominant character (Landsteiner and Levine, 1931).

Since then the Tj^a antigen has been discovered by Levine et al (1951), coupled with the recognition by Sanger (1955) that the antigen is part of the p system. It became clear that p-persons shared a powerful antigen (Tj^a) with p+ people, and a third and extremely rare group is defined in which this antigen is lacking. This discovery led to a modification of the original notation of the p system. Race and Sanger (1954)gave a table of results of testing with anti-p_1 on the blood of 'Caucasians'. The frequency of negatives, now p_2, varied from 18-30 percent. Such differences Race and Sanger (1970) stated reflect serological and not anthropological differences.

Further investigation has disclosed a system of considerable complexity and has shown that p as originally defined is not in fact an amorph but a weak variant of the original p. By analogy with the ABO system the two genes are now called p_1 and p_2, and a third extremely rare amorph gene, the new p,has been recognized, the homozygous form of which fails to react with a very rare but extremely potent antibody, anti-p,formerly known as anti-Tj^a(anti-Tay) and then thought to belong to an independent system.

The antigen Tj^a found by Levine et al (1951) was at first regarded as the product of a gene present in nearly all human beings, the extremely rare allele being an amorph, with the homozygotes usually having a strong anti-Tj^a antibody. Sanger (1955) then showed that Tj^a was part of the p system, with three alleles, p_1 (formerly p), P_2(formerly p), and the new p (formerly regarded as the amorph allele of Tj^a). These relationships are similar to those existing between A_1, A_2 and 0 of the ABO system. P_2 bloods sometimes show anti-p_1 in the plasma, usually with a very low titre, but the rare bloods always have a high titre of anti-p+ anti-p_1. The p system has now taken the form shown in Table 2.1.4.

TABLE 2.1.4.

THE P BLOOD GROUP SYSTEM

Phenotype under old system	modern phenotype	Genotype	Reaction with anti-sera	
			anti-P+ anti-P_1 (formerly anti-Tj^a)	anti-P_1 (formerly anti-P)
P+	P_1	P_1P_1	+	+
		P_1P_2		
		P_1P		
P-	P_2	P_2P_2	+	-
		P_2P		
	P	PP	-	-

The gene P, and hence all the genotypes containing it, are exceedingly rare.

The only recognizable genotype, PP, has never been found in any random population survey, and Race and Sanger(1968) estimate its frequency at about one per million. Population data are thus concerned only with the genes P_1 and P_2. The strength of the antigen P_1 in P_1- positives varies quite considerably. Fisher(1953)has shown that much of the variation can be explained as due to the difference between P_1P_1 and P_1P_2.By no means the whole of the observed variation can be explained in this way,but it is still uncertain whether,and if so how far, the remaining variation is due to a series of quantitative variants of the P_1 gene. Many papers by Japanese workers describe the incidence and other properties of blood group antigen Q. This has been shown by Henningsen (1954, 1955) to

be indentical with that here called P_1.

Two very rare antigens, p^k and Luke, are related to the p system; as Race and Sanger (1968) show, p^k and probably also Luke, behave as though controlled by a series of allelic genes separate from those of the P_1P_2P series, but interacting with them in a similar manner to the interaction of the Hh genes with those of the ABO series. p^k individuals (perhaps analogous in the above sense with those of Bombay type) seem to be almost confined to the Finnish people, but even among them have never been found in a random series of tests. The Luke system or subsystem remains rather obscure; the Luke antiserum fails to react with p(and also with p^k) people,but is negative also with about 2 percent of $p_1$and p_2 people.

The p_1 antigen is present in hydatidcyst fluid (Cameron and Staveley, 1957) and in a considerable variety of worms, both parasitic and free living. It is likely that the anti-P_1, not infrequently found in the plasma of p_2 individuals and in that of several species of mammals, is a response to worm infestation, but nowork appears to have been done on possible associations between the p groups and such infestation.

Women of genotype pp, who always have anti-p+ anti-P_1 in their plasma, are particularly subject to abortion, apparently resulting from the action of the antibody upon the almost invariably p-positive foetus.

Paroxymal cold haemoglobinuria is due to the presence in the patient's plasma of a cold-reacting auto-antibody. It was shown by Levine et al (1963) that this usually has anti-p specificity.

2.1.5 The Kell blood group system

The kell blood group system which initially, like all other systems, appeared simple, has gradually been shown to have a comparable complexity, and a similar organisation to the MNSs and Rh systems. The Kell antigen was first described by Coombs, Mourant, and Race in 1946. It was soon shown that it was the product of a gene K. The antigen K was found in only 9 percent of an English population sample, who were termed Kell-positive. The discovery of the expected antithetical antibody anti-K (Cellano) by Levine et al (1949) made it clear that the system was governed by a pair of allelomorphic genes, K and k and the inheritance of the Kell groups by means of two allelic genes without dominance was proved. The two genes K and k 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 2.1.5.a.

TABLE 2.1.5.a.

PHENOTYPES AND GENOTYPES OF THE KELL SYSTEM

Reaction with antisera anti-K	anti-k	Phenotype	Genotype
+	-	K	KK
+	+	Kk	Kk
-	+	k	kk

This simple view of the Kell system lasted until 1957 when Allen and Lewis (1957, 1958) described two further antigens Kp^a and Kp^b, associated with the Kell system and shown to be the products of a pair of allelic genes.

The sutter system of Giblett (1958) at first appeared to be an independent one. Again two antithetical antigens Js^a (found by Giblett, 1958) and Js^b (found by Walker et al,1963) are known, the products of a pair of allelic genes, first shown by Stroup et al 1965 to belong to the Kell system.

Finally, the Karhula or Ul^a antigen of Furuhjelm et al (1968, 1969) has been shown to have a similar association.

By analogy with the MNSs and Rh systems, the Kell system may be regarded as determined by three or possibly four closely linked loci, occupied by the respective allelic genes K,k; Kp^a, Kp^b; and Js^a, Js^b. The details of the relationship with Ul^a are not yet published. If we disregard Ul^a, about which little is yet known, only four of the eight theoretically expected gene complexes have been found, as shown in Table 2.1. 5. b.

TABLE 2.1.5.b.

GENE COMPLEXES OF THE KELL SYSTEM

$KK^b\ Js^b$
kKp^aJs^b
kKp^bJs^a
kKp^bJs^b

Some very rare individuals are known whose red cells are negative with all six diagnostic antisera. They may be homozygous for a rare amorph gene complex, but it is possible that some of them are, as suggested by Race and Sanger (1968) , homozygous for a rare allele of a common gene which controls the synthesis of a basic substance from which the observed

antigens are elaborated.

The great majority of human beings in all parts of the world are homozygous kKp^b Js^b/kKp^b Js^b, the genes K, Kp^a, and Js^a being everywhere relatively rare and none of them having yet been found occurring together in one gene complex. Thus most of the facts about the distribution of the complexes can be covered by separate consideration of the distributions of the genes K, Kp^a and Js^a:

2.1.6. The Duffy blood group system

The discovery of the Duffy blood group system was first briefly reported by Cutbush, Mollison and Parkin (1950) and more fully reported by Cutbush and Mollison (1950). 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, and developed an immune antibody in response to transfusion. It soon became clear that the antigen was the product of a gene,giving rise to the recognizable antigen, called Fy^a which must possess an allele Fy^b though no product of the latter gene could at the time be detected. The first example of the antithetical antibody, anti-Fy^b, which did detect this product, was reported in 1951 by Ikin et al. They also found that the antigens were inherited by two allelic genes, Fy^a and Fy^b, without dominance.

The allelic genes Fy^a and Fy^b account for nearly all the phenotypes found in European populations, but a third allele in the Duffy system was postulated when Sanger et al (1955) discovered that the red cells of most American Negroes did not react with either antiserum and that they belonged to a new phenotype which was like Fy(a-b-). They thought that they

were homozygous for a third gene, Fy. This allele is exceedingly rare in whites but Race and Sanger, from family data, consider that Fy may have a frequency as high as 3 percent among Europeans. Chown et al (1962, 1965) have reached a similar conclusion; they however, find that the third gene among Europeans is not a complete amorph but gives rise to weak Fy^b reactions.

Table 2.1.6. demonstrates the genetical interpretation of the Duffy system.

TABLE 2.1.6.

PHENOTYPES AND GENOTYPES OF THE DUFFY SYSTEM

Reaction with: anti-Fy^a	anti-Fy^b	Genotype	Phenotype
+	-	Fy^aFy^a	Fy(a+b-)
+	+	Fy^aFy^b	Fy(a+b+)
-	+	Fy^bFy^b Fy^bFy	Fy(a-b+)
-	-	Fy Fy	Fy(a-b-)

An inspection of the latest tables of Mourant et al (1976) will show that, except in the South African Republic, over 95 percent of African Negroes are of this type, that is homozygotes of a third allelic gene, at first regarded as an amorph and so named Fy. It is now known that two distinct genes have in the past been confused under this term. One, Fy^x, with a frequency of approximately 1.6 percent in Euro-

peans, gives a product which is negative with anti-Fy^a but which reacts feebly with anti-Fy^b(Chown et al, 1972). The other, almost universal in Africans, gives a product which completely fails to react with either of these antibodies, but is not a true amorph, for Behzad et al (1973) have shown its product to react specially with an antibody which they called anti-Fy^4; the gene should presumably be called Fy^4. Miller et al (1975) have shown that the homozygote of this type is probably specifically resistant to Vivax malaria, to which Africans have long been known to be resistant. It is known that Africans are mostly of the Duffy blood type Fy(a-b-), the homozygote of which was formerly regarded as the amorph gene Fy but is now recognized positively as Fy^4. They are also mostly highly resistant to the malarial parasite plasmodium vivax (the formerly common European type.). Miller et al have now shown that the point of attachment and entry into the human red cell is the antigen Fy^a or Fy^b, but that they will not enter cells of type Fy(a-b-). One may speculate that Africans developed resistance to p. vivax by natural selection of the 'amorph' Fy gene, biologically an efficient and 'cheap' process. When, however, p. vivax had thus almost disappeared from tropical Africa, the much more virulent p. falciparum was evolved, to which at first there was no inborn resistance. Thus when the haemoglobin S gene appeared, possibly introduced from outside Africa, or evolving in some limited focus, the fact that heterozygotes were resistance to p. falciparum led to the spread of the new haemoglobin by natural selection. This, however, was a much more 'costly' form of resistance, since it led to the early deaths of nearly all homozygotes.

2.1.7. The Kidd blood group system

The Kidd blood group system was discovered by Allen et al in 1951. The finding of this 'new' antibody, anti-JK^a, was the result of a haemolytic disease in a newborn infant. The antigen JK^a, was shown by these authors,in collaboration with Race and Sanger (1951) to be the product of a gene JK^a. The existence of the expected antibody, anti-JK^b, reacting with the product of its allele, JK^b, was described by Plaut et al in 1953. It has been suggested that the two genes, JK^a and JK^b, are inherited as non-dominant autosomal alleles.At present, tests have been made with both antisera. 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.

The phenotypes and genotypes of the system as defined by using anti-JK^a antisera is shown in Table 2.1.7.a

TABLE 2.1.7.a.

PHENOTYPES AND GENOTYPES OF THE KIDD SYSTEM

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

The phenotypes and genotypes of the system as defined by both anti-JK^a and anti-JK^b antisera is presented in Table 2.1.7.b.

TABLE 2.1.7.b

PHENOTYPES AND GENOTYPES OF THE KIDD SYSTEM.

Reactions with:		Phenotypes	Genotypes
Anti-JK^a	Anti-JK^b		
+	-	JK(a+b-)	JK^a JK^a
+	+	JK(a+b+)	JK^a JK^b
-	+	JK(a-b+)	JK^b JK^b

Race and Sanger(1968), however, think that many sera used as anti-JK^a reagents contain an additional unrecognized antibody, probably belonging to the JK system, since tests carried out in England have rather consistently shown an excess of apparent heterozygotes, above the frequency required by genetic equilibrium. Studies on Canadians by Chown et al (1965), however, show internal consistency, suggesting that a pure anti-JK^a serum was used. The question of the purity of anti-JK^a sera is at the moment mainly of theoretical interest in population studies, since such sera, pure or otherwise,are in extremely short supply. It could, however, have considerable practical importance if adequate supplies of anti-JK^b as well as of anti-JK^a should become available and show the JK (a-b-) phenotype, mentioned below, to be at all widespread. Even the antiserum anti-JK^a however is rather rare, so that its use has to be somewhat restricted. It is moreover a difficult serum to use requiring either an enzyme technique or a complement-fixing anti-human-globulin test.The knowledge of the distribution of the genes of the JK system is based mainly

on tests with anti-JK^a alone.

2.1.8 The Lutheran blood group system

The Lutheran blood group system was discovered in 1945 by Callender and Race who found the antibody now known as anti-Lu^a in the serum of a patient suffering from Lupus erythematosus diffusus. The antibody was shown to be immune in nature. The system was more completely reported by Callender and Race (1946). Family studies indicated that the antigen Lu^a was inherited as a Mendelian dominant character and the blood group system depends upon a pair of allelic genes, Lu^a and Lu^b. The gene Lu^b was only recognized as the absence of Lu^a until 1956. The notation suggested for the system was as follows:

genes : Lu^a, Lu^b

phenotypes : Lu(a+), LU(a-)

antibodies : anti-Lu^a, anti-Lu^b (to be discovered).

Positive evidence of the existence of an Lu^bgene was first found by Cutbush and Chanarin (1956) when they indentified an expected antibody anti-Lu^b which reacts with the product of this gene. The finding of anti-Lu^b established the existence of the antigen and gene Lu^b. With the use of the two antisera three phenotypes can be identified each corresponding to one of the genotypes Lu^aLu^a, Lu^aLu^b and Lu^bLu^b.

Table 2.1.8 indicates the correspondence of genotypes and phenotypes of the Lutheran system.

TABLE 2.1.8.

PHENOTYPES AND GENOTYPES OF THE LUTHERAN SYSTEM

Reactions with: anti-Lu^a	anti-Lu^b	Phenotype	Genotype
+	-	Lu(a+b-)	Lu^a Lu^a
+	+	Lu(a+b+)	Lu^a Lu^b
-	+	Lu(a-b+)	Lu^b 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. This fourth phenotype fails to react with either of the anti-sera.

Most examples of this phenotype show dominant inheritance possibly due to a gene at a separate locus, but no such example has ever been found in a random population survey. Others show recessive inheritance and may be due to an amorph allele at the Lutheran locus; only three examples have been found in population surveys one by Darnborough et al (1963) in testing 18069 British blood donors, and two by Candanayingyong et al (1967) among 455 Thai donors.

The Lutheran system is notable genetically as having provided the first known example of ordinary autosomal linkage in man, between the loci for Lutheran blood group and for salivary secretion of the antigens of the ABO system (Mohr,1951). As, however, there is a recombination frequency of about 15 percent the linkage does not affect the independence of the two systems in population studies.

Both the diagnostic sera required for this system are in short supply, anti-Lu^b extremely so. Thus only a very few population samples have been tested with both antisera, and only moderate numbers with anti-Lu^a alone.

It has been shown that the Sw^a (Swann antigen) gene is part of, or closely linked to, the Lutheran system. (Metaxas -Buhler et al, 1972).

2.1.9. The Diego blood group system

The Diego blood group system was first reported by Layrisse et al in 1955. The antibody which reacted with the antigen had been produced by a Venezuelan woman, Mrs. Diego, through pregnancy immunization, and had caused haemolytic disease of the newborn. The authors recognized that the family involved was probably partly of American Indian descent, and they showed the antigen to be present in various Indian and mixed populations, the phenotype frequency rising as high as 35 percent in Carib Indians. The antigen is the product of a gene Di^a with dominant (or co-dominant) expression.
The expected antithetical antibody anti-Di^b, detecting the product of the allelic gene Di^b was found by Thompson et al (1967). It has been suggested that the system is controlled by two allelic genes, Di^a and Di^b. No example has yet been found of a phenotype Di(a-b-) nor is there any other genetical evidence of the existence of a third allele.

Information about the distribution of the Di^a gene has been studied extensively by numerous investigators, but especially by Layrisse and his colleagues (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 extremely rare in Caucasians,North American and African Negroes. Thus, the gene is essentially characteristic of Mongoloids and has never been found in any survey of unselected persons of presumed unmixed European descent.

Of the Diego blood groups more than of those of any other system it can be said that their main scientific interest lies in the field of anthropology.

2.1.10 The ABH secretion system

The antigens A,B and H of the ABO blood group system, as well as occurring in the erythrocytes, are also found in water soluble form in body fluids, including saliva (Yamakami, 1926; Lehrs, 1930, Putkonen, 1930). However, some individuals do and others do not secrete into their saliva antigens corresponding to their ABO blood group. Schiff and Sasaki (1932) found that the ability to secrete the ABH group-specific substances is inherited as an autosomal dominant trait. Groups A, B and AB persons who are secretors secrete the antigens corresponding to their blood groups. Group O persons who are secretors secrete the H substance, as do all other secretors to a somewhat less extent. The non-secretor individuals do not secrete their ABH substances, no antigenic substances being found in their body fluids. Secretion is controlled by a pair of allelic genes, Se and se, giving three possible genotypes SeSe, Sese and sese, the first two constituting the secretors, the third category the non-secretors.

2.2. Serum proteins

All immediate gene products are now known to be proteins, but the direct detection and examination of the products of

specific genes is a very recent development, beginning with the discovery by Pauling et al in 1949 of a physio-chemical difference between two genetically distinct haemoglobins.

From the standpoint of the methods used in identifying them,as well as from that of their innate functions the proteins of the blood are divided into two main classes by the surface membrane of the red cell. Outside are the plasma proteins. Inside are the red-cell proteins, of which by far the most abundant is haemoglobin. Of the others, present in smaller amounts, the enzymes are of the greatest interest to the geneticist, because they can be visualized by highly specific staining reactions, and because many of them show two or more readily distinguishable gene-controlled variants. Numerous enzymes, many of them genetically polymorphic are,however, also present in the plasma.

The object of a series of population surveys is not,except in rare instances, to determine in any absolute sense the properties and constitutions of the products of a series of allelic genes. This has in general already been done, or is being done, by workers in other disciplines. The object is rather to distinguish clearly in an empirical manner between known gene products, while always keeping an eye open for an unknown one. The geneticist must not,however, lose sight of the functions of the proteins with which he is dealing, from the standpoint of the body's total metabolism. In many cases the genetical variants of a single protein can be shown to differ quantitatively in their functional chemical activity, and such differences must be taken into account in studying interactions between genetical constitution and the environment.

However, the blood plasma contains in solution a great

variety of proteins, many of which show genetical polymorphism. Unlike the blood groups, these proteins mostly have known functions, for instance as enzymes, or as carriers of simple substances like metals and vitamins. As mentioned above the genetical variants of a particular protein often, and perhaps nearly always, differ quantitatively in their functional activity, thus allowing the possibility of natural selection, which may be the cause of the observed variability of gene frequencies between populations. Only in a few cases have the frequencies of the variants of a particular protein been ascertained in patients suffering from specified diseases (Mourant et al, 1976, 1979).

2.2.1. The haptoglobin (Hp) system

Haptoglobin has long been known (Polonovski and Jale, 1940) as a glycoprotein (a protein with attached carbohydrate) present in the plasma, and having the well defined function of combining with any dissolved haemoglobin entering the plasma as a result of the lysis of red cells. This prevents the haemoglobin from being excreted by the kidney, but the share of the haptoglobins in conserving the body's supply of iron is not fully understood. The literature of the biochemistry and genetics of haptoglobin is extensive and complicated. A full and excellent summary is given by Giblett (1969).

The first report of the existence of the plasma protein now known as 'haptoglobin' was made by polonovski and Jale (1938). Smithies (1955) and Smithies and Walker (1956) demonstrated that genetical variation occurred in the α_2-globulin, haptoglobin, when sera were subjected to starch-gel

electrophoresis. The complex electrophoretic patterns could be attributed to three common phenotypes, known as Hp1-1, Hp2-1, and Hp2-2, which behaved genetically as though the proteins concerned were the products of two allelomorphic genes, Hp^1 and Hp^2, at an autosomal locus (Smithies et al 1962).Phenotypes 1-1 and 2-2 are the expression of the homozygous forms and phenotype 2-1 of the heterozygote of the Hp^1 and Hp^2genes. Extensive family studies (Smithies and Walker, 1955;Gala - tius-Jensen, 1958; Harris et al, 1959; Smithies et al, 1962) confirmed this hypothesis.

The Hp^1gene has a single product which yields a fast moving band, intensely stained and somewhat slower than free haemoglobin. The Hp^2gene gives rise to a series of polymers with different mobilities. It has no band in the position of the Hp^1 component, but slower bands of diminishing intensity towards the origin. These bands vary in width and extent to which they stain. The heterozygote of these two genes, HP2-1, shows a band in the position of the Hp^1band, but it stains less trongly, and also has multiple bands in the Hp^2 position which have a faster mobility than Hp^2 bands, due to the fact that they are polymers of the products of both the Hp^1 and Hp^2 genes (Figure 2.2.1.a and b).

The haptoglobin molecule, like those of so many other proteins, consists of two types of polypeptide chains, α and β ,presumably the products of two distinct sets of allelomorphic genes. Black and Dixon(1968) who have studied the structure in great detail point out resemblances with the immunoglobulins and suggest that the two types of protein have evolved from a common origin.

Variants of the β chains are exceedingly rare; almost all

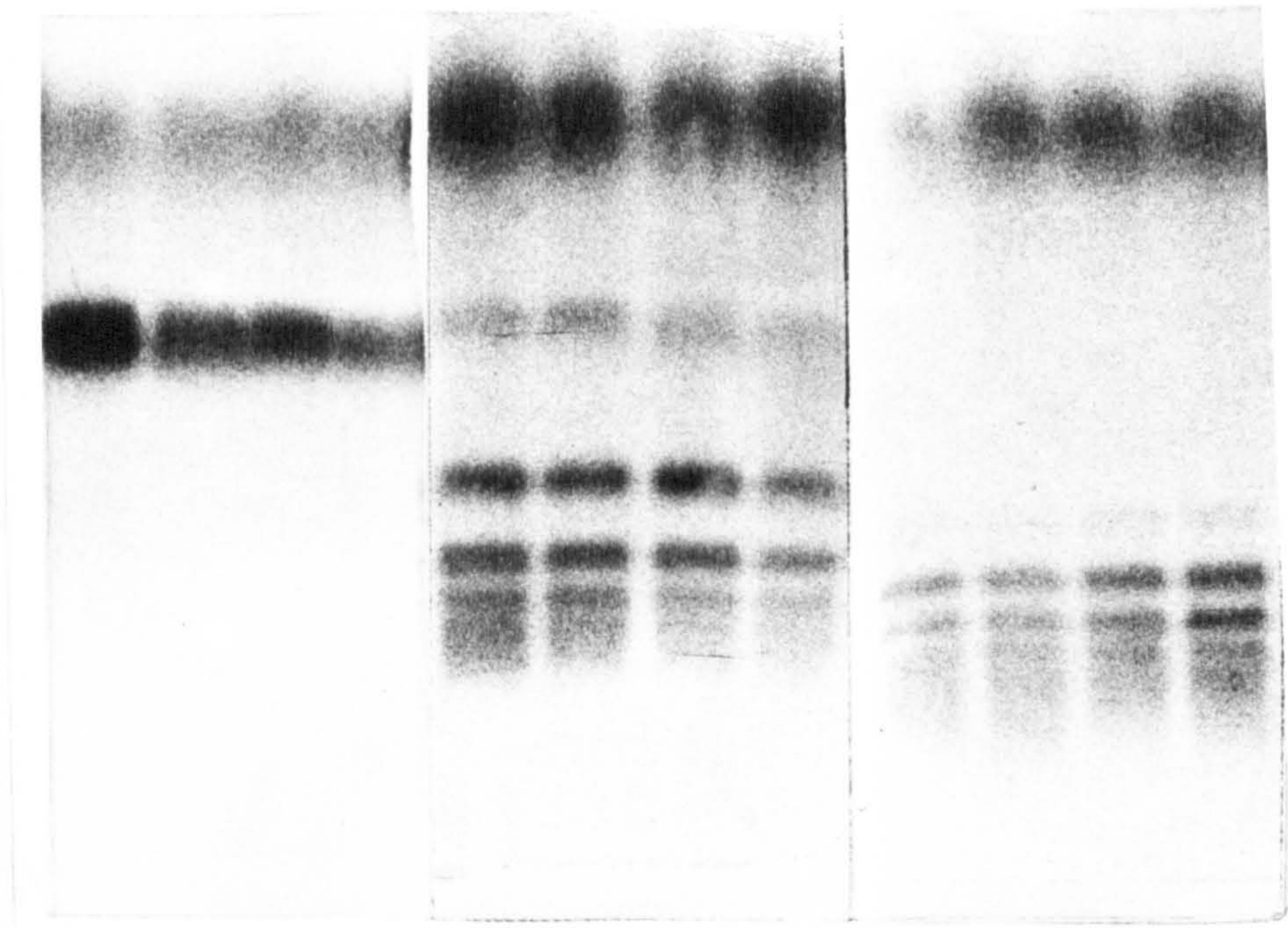


Fig.2.2.1.a. Starch gel electrophoretic patterns of the three common haptoglobin phenotypes (From left to right)HP 1, 2-1 and 2.

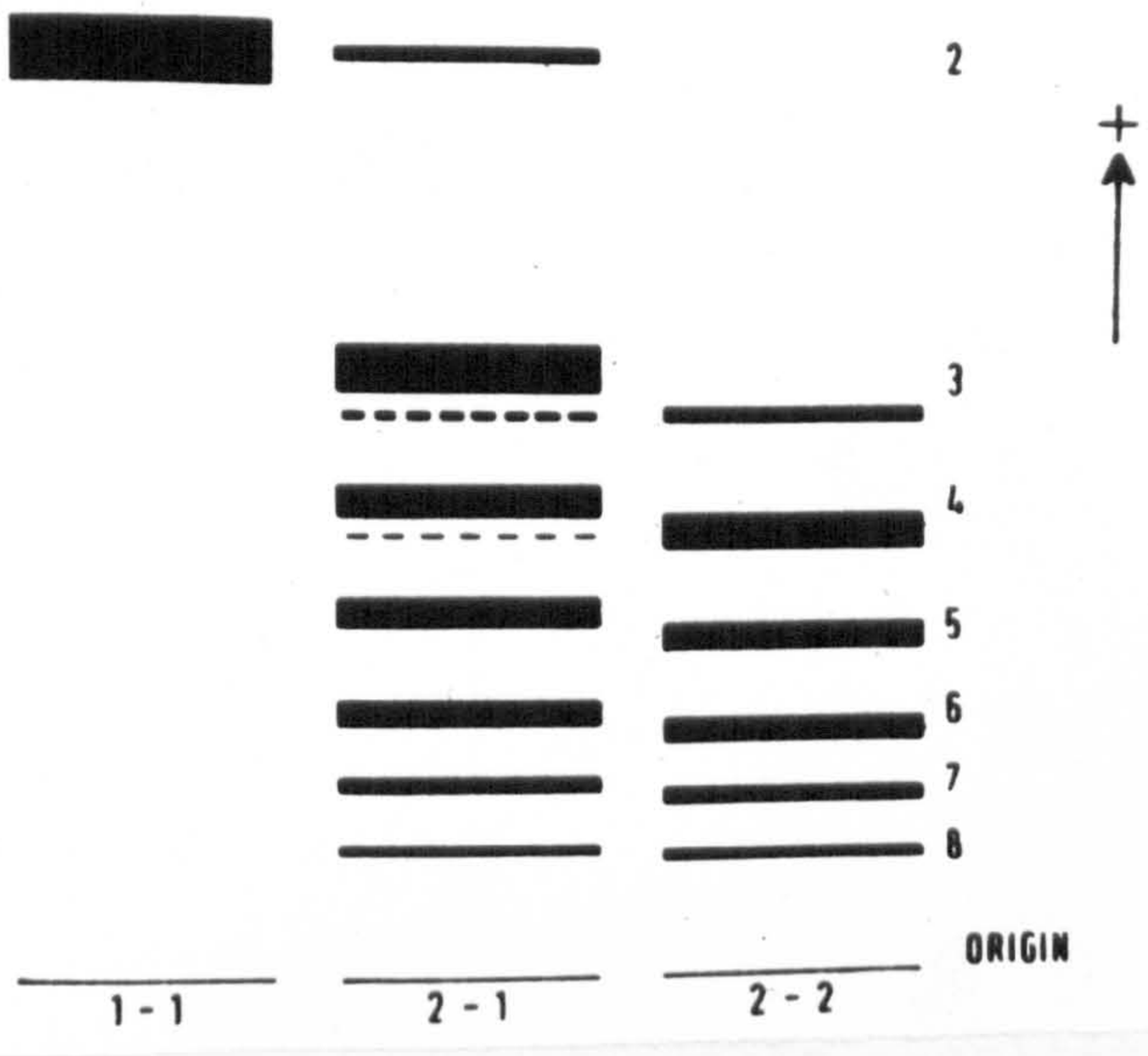


Fig.2.2.1. b. Diagram of the three common haptoglobin phenotypes, HP 1, 2-1 and 2.

the known variation is in the ∝ chains, the main polymorphism being determined by genes at the ∝ locus.

The β -chains are found to be the same in all the three haptoglobin types, whereas structural variations in the ∝ -chains show different electrophoretic patterns of the intact molecules. Thus, a series of phenotypes in the haptoglobin system can now be recognized.

When the haptoglobin molecule is cleaved into its constituent chains by means of 8 M urea and mercaptoethanol, the chains determining the apparently single Hp1 phenotype are shown to be of two alternative types, differing slightly in their speed of migration during electrophoresis, and by a single amino-acid substitution in their polypeptide chains, corresponding to alleles Hp^{1F} and Hp^{1S}.Thus, individuals with Hp1-1 phenotype can have a fast migrating ∝ -polypeptide (Hp_{1F-1F}), a slow migrating ∝ -polypeptide(Hp_{1S-1S}) or a mixture of both fast and slow migrating ∝ -polypeptide (Hp_{1F-1S}). Phenotypes Hp2-2 persons exhibit only one band which is considerably slower than Hp1F or Hp1S. Individuals with Hp2-1 phenotype have Hp2 band combined either with Hp1F or Hp1S. These subtypes are controlled by two alleles, Hp^{1F} and Hp^{1S}. The single type of ∝ chain derived from the Hp^{2} molecule is of nearly twice the molecular weight of that of Hp1 of either kind; it is in fact a combination of the greater part of the ∝ chains of the two types of Hp1, and appears to arise from a partial gene duplication resulting from unequal crossing over between Hp^{1F} and Hp^{1S}.The retention of the original amino-acid sequences suggests that this crossing over was a comparatively recent event in the history of mankind, while the wide distribution of the Hp^{2}gene suggest that it

must in some circumstances have had a considerable selective advantage over both the Hp^1 genes.The single band of Hpl and the fastest band of Hp2 each correspond to molecules containing two α and two β chains.

The molecular weights of all Hp2 molecules present in the plasma are thus greater than any of those of Hpl. Hp2-2 presumably for this reason,tends to bind less haemoglobin,weight for weight, than Hpl-1,but the former,because of the larger molecular size of the complexes,is more fully resistant to excretion by the Kidney: this is presumably the basis of the success of the mutation.Some more nearly direct evidence of the presumably balanced advantage enjoyed by the Hp^1 molecule has recently come to light,since Ritter and Hinkelmann(1966)and subsequently Kirk and his colleagues(Kirk et al,1970;Kirk,1971)and Ananthakrishnan et al (1973)have shown,on a wide variety of populations,that in matings where the father is incompatible with the mother with respect to the ABO groups, the children show a higher frequency of the Hp^1 gene than do those of families, drawn from the same population,inwhich the father is ABO compatible.This was explained as due to deaths from haemolytic disease of the newborn resulting from ABO incompatibility(though mostly unrecognized as such),and related to the fact that the product of the Hp^1gene is more efficient than that of Hp^2in removing dissolved haemoglobin from the plasma conserving the contained iron. Thus in cases where foetuses,or newborn are affected by haemolytic disease due to foetal A or B inherited from the father, a child who is of type Hp1-1 should have the best, and one of type 2-2 the worst chance of survival. Family studies by Vana and Stienberg(1975) confirmed the tendency towards raised Hp^1frequencies among the offspring of ABO incompatible matings, but fur-

ther analysis of the data has shown that the Hp^1 frequencies are intrinsic to the blood groups themselves,irrespective of the type of mating from which they come. They conclude that there is a tendency,which they cannot at present explain,for HP^1frequencies to increase with blood group in the order:O,A, B,AB and indeed in the order of genotypes:OO,AO,BO,AB,AA,BB. This explanation does not, of course, exclude some form of underlying natural selection either of gametes or zygotes.

In most populations only the three phenotypes 1-1,2-1, and 2-2 are commonly distinguished, a number which is increased to six if tests for the variants of Hp^1 are performed. Beside these, however, a few variant phenotypes are sufficiently common to be found in some random population surveys. One of these is known as HP2-1M.,the M standing for 'modified'. This phenotype shows the bands of Hp2-1, but with the slower bands (higher polymer) increasingly faint. This type, found mostly in Negroes, is probably due to variant of the Hp2 gene which, however, gives rise to a homozygote indistinguishable from normal Hp^2/Hp^2, or sometimes to Hpo.Another type,even commoner,and also found mostly in Africans,is that usually known as Hpo,and characterized by an apparent absence of haptoglobin; it was originally regarded as the product of an amorph allele. Though a very few cases may be due to such an allele, the majority are not directly due to genes at the Hp loci.There are in fact a series of grades of deficiency of haptoglobin, and by physicochemical methods of concentration it can often be shown to be present,and its type can be determined,when initial tests appeared to show complete absence.Haptoglobin is shown to be depleted or absent in cases of haemolytic anaemia.However,the type is found with undue frequency in children both of whose parents

are Hp2-1M. It is likely that Hp0 or ahaptoglobinaemia is the result of a combination of environmental and genetic factors, not necessarily all at the Hp locus.

Though the quantitative variant phenotype Hp0, may in most cases be of environmental origin,e.g. haemolysis, there seems no doubt that there also occur individuals with no detectable haptoglobin,or with only very minute amounts,in the absence of any haemolytic process (Harris,1961).

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 Steegmuller,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 other: Hp1-1 > Hp2-1 > Hp2-2. 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 haemoglobin 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,1969).Weitkamp et al(1972)also found a positive correlation between ahaptoglobinaemia and malaria infection.

A considerable number of rare phenotypes found in the haptoglobin system are classified as quantitative and qualitative variants. In addition to the Hp2-1M and Hp0 phenotypes, other rare phenotypes of quantitative category are Hp2-1'Carlber', 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.

Despite much investigation the relation between genetics

and physiology in this system is by no means fully understood. The Hp and ABO genes appear to interact ante-natally through some form of selection, to affect the frequencies of various combinations of ABO and Hp phenotypes, as shown by numerous family studies.

2.2.2. The transferrin (Tf) system.

Transferrin or siderophilin is the iron-binding protein component of the plasma. It is a β -globulin, containing 5.5 percent of carbohydrate, which combines with inorganic iron present in the plasma and transfers it to the receptor cells of the bone marrow and other storage depots. Like haptoglobin, trnasferrin has long been known as a well defined plasma protein when Smithies (1957) first demonstrated by means of Starch-gel electrophoresis, the existence of inherited variation in its molecular structure. It exists as a number of genetically determined variants, migrating at different speeds. The protein part of the molecule appears to be one long polypeptide chain coded by a single gene.

By far the most common type in all populations is known as C. The other variants have been labelled with regard to their electrophoretic mobility in relation to this type. Types which migrate faster than TfC are known as TfB with an appropriate suffix and those which migrate more slowly are similarly classed as TfD. Suffixes are sometimes geographical, or ethnographic, as in D_{chi} and $D_{montreal}$. In other cases the relative speed of migration is indicated as in B_{1-2}, intermediate between B_1 and B_2. Gene notation is typographically awkward, involving suffixes to indices as $Tf^{D}chi$. Variants other than C are relatively uncommon, and have generally been found in combination with C, the usual variant being present in amounts

about equal to that of C.

Family studies by Horsfall and Smithies (1958) suggested that there exists a series of allelic genes, each of which determines the formation of a particular Tf type. Individuals with two of the above Tf genes appear to be heterozygotes, individuals with one, homozygotes. Smithies and Hiller (1959) reported that the formation of β-globulins B, C, and 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 B or D genes. The band or bands of transferrin can as a rule be recognized by inspection of a general-protein-stained electrophoretogram, lying between haptoglobin and albumin (Figure 2.2.2.a).

For definite identification, however, one of a number of specific tests should be used. As a rule only a single band is seen, corresponding to the common TfC phenotype, representing the homozygote of the most common gene. A great many genetically determined variants of transferrin are now known, but all are rare, and most are very rare, so that they are found almost exclusively as heterozygotes in combination with the common type. Variant homozygotes, and heterozygotes of two variants combined, are very rare indeed. At present, the polymorphism of transferrin is attributed to 22 allelic genes. (Smithies, 1957; Giblett, 1969; Walter, 1975). (Figure 2.2. 2.b).

But only four variant phenotypes are known to have frequencies above one percent in any population, these are the heterozygotes of Tf^D1, Tf^Dchi, Tf^B2, and $Tf^B0\text{-}1$.

The application of isoelectric focusing to the analysis of transferrin has revealed considerably more genetic hetero-

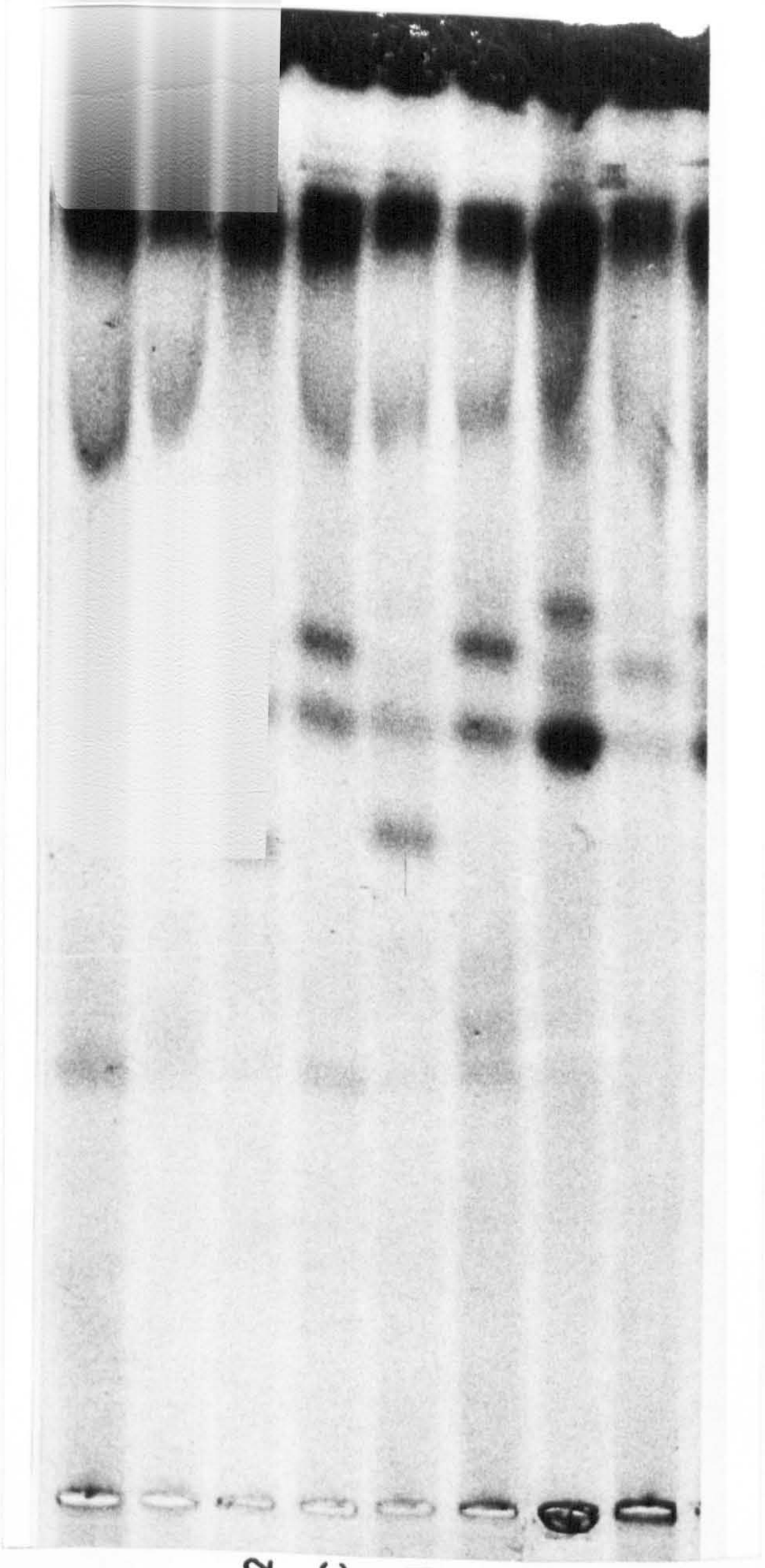


Fig. 2.2.2. a. Agarose gel electrophoretic patterns of the common transferrin C and some rare CB and CD phenotypes (Farhud, D.D. 1972).

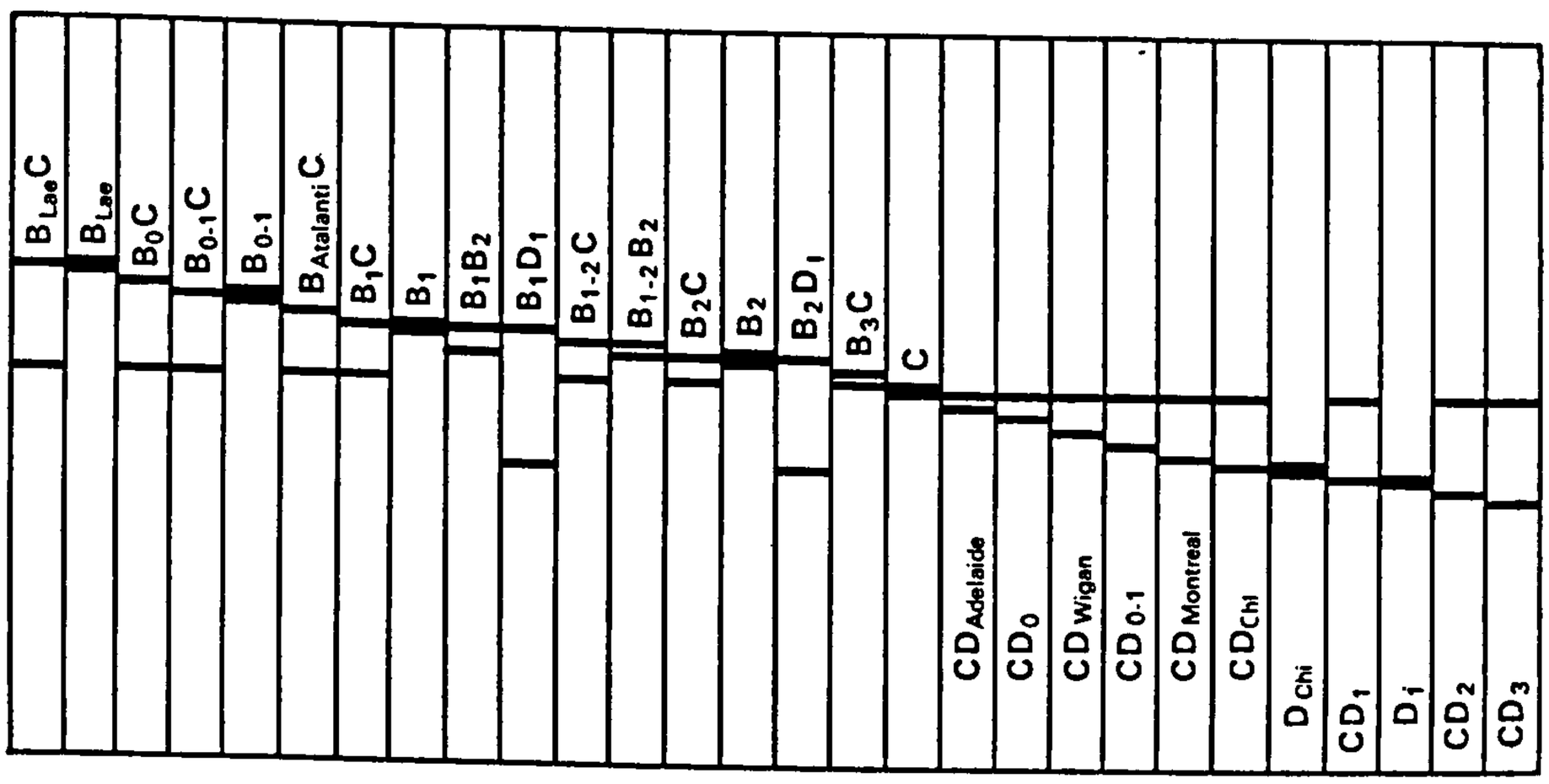


Fig.2.2.2.b. Diagram of the 28 reported transferrin phenotypes in human serum, representing TfC and 17 variants with slower or faster mobility (Giblett, E.R. 1969).

geneity than was apparent by electrophoretic techniques. Kuhnl and Spielmann (1978), Thymann (1978), Kuhnl and Spielmann (1979), Kuhnl et al (1979), Altland et al (1980) and Constans et al (1981) have recently described two common subtypes of the C allele, C_1 and C_2, and three rare ones C_3,C_4, C_5 in some European populations, which revealed a clear distribution heterogeneity of this polymorphism within Europe. As far as we know, the only transferrin subtyping performed on population samples from other parts of the world is that of Walter et al (1981) on six Indian population samples.

No differences have been detected in iron-binding capacity of the common type and the different variants of the transferrin molecule. Whatever functional differences may exist, the rarity of the variants suggests that the common type has under most circumstances a selective advantage over all the others. This contrasts with the situation in cattle where several alleles are of comparable frequency and a complex system of selective fertility exists (Ashton, 1965).
Because of the rarity of variants, and the even greater rarity of families including members with two different ones, there has been no comprehensive proof of the hypothesis that all variants are the result of mutations at a single locus. However, as pointed out by Giblett, this hypothesis is supported by the simple Mendelian behaviour of the individual variants, the absence of any record of individuals with three kinds of transferrin, the fact that the molecules appear to differ only in electrical charge and not appreciably in molecular weight, and in a few cases by the demonstration that the variants differ from the common type only in the substitution of a single amino-acid for another.

So far no specific association between an unusual Tf variant and any particular clinical disorder has been found. All transferrin variants, other than TfC, are rare, so that even if they showed disease associations or other selective effects, this would be difficult to prove. However, because of the occurrence in cattle of a well-defined system of balanced polymorphism of the transferrins (Ashton, 1955), it is possible that such a situation could emerge in a human population having a high frequency of one of the variants. Walter and Bajatzadeh (1971) have suggested that the frequencies of the Tf alleles are not distributed equally within the human species and show marked racial differences with respect to the 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, so that the relatively high Tf^D gene frequencies in tropical biotopes could indicate a better physiological functionning of these variants. Ashton (1965) reported a positive association between another slow variant, Tf^E, and tolerance to hotter climate 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.

2.2.3. The third component of human complement (C3) system

Complement is a complex mixture of blood proteins which are activated sequentially, many by conversion of a proteolytic zymogen into an active proteinase, and which play an

important role in the host defence against infection. They were recognized at the end of the last century by their ability to cause the lysis of bacterial cells that had been agglutinated by specific antibodies. Antibody-coated red blood cells soon replaced the bacterial cells as the test system in the assay of complement, as measurement of the haemoglobin released offered an easy method of estimating lysis. Complement is an important part of an animal's defence mechanism against pathogenic microorganisms, but subsequent work has suggested that some of the proteins in the system may have a direct role in the immune response itself by facilitating intercellular interactions. Most recently, four of the complement components have been found to be coded by structural genes located in the major histocompatibility complex. This again has suggested, by the analogy with other products of this genetic complex, that complement proteins may have a function in the cell surface in addition to their activity as an effector mechanism in the destruction and elimination of foreign antigenic material.

As many as 20 proteins in the blood may be part of the complement system, which has two pathways of activation as well as a variety of control mechanisms which include inhibitors and inactivators specific for different steps or components. Most of these proteins have now been isolated and have been characterized at least in part (Gotz and Muller-Eberhard, 1976; Lachman, 1973; Muller-Eberhard, 1975).

The major component of human complement (C_3 or β_{1c} - globulin) was first isolated by Muller-Eberhard (1960). He

isolated a beta globulin from normal serum and showed that it was one of the proteins comprising the classic third component of complement. With the characterization of the remaining five proteins of this group, the term C_3 or third component has been reserved for the first protein of the group to be purified and characterized and the first of this group to be activated during complement fixation, formerly called $beta_{1C}$-globulin. The remaining proteins of the classic third component of complement have been numbered C_5 to C_9, in order of their participation in full complement activation (Muller-Eberhard et al, 1966; Bulletin of World Health Organization, 1968). The numbering of the first three complement components in the activation sequence, C_1, C_4 and C_2, persists with all the illogical force of conservative tradition.

In addition to being part of the classic third component of complement, β_{1C}- globulin (Muller-Eberhard et al, 1960) has been shown to play a key role in the mechanism of immune adherence (Nishioka and Linscott, 1963), conglutination, immune conglutination (Lachmann and Coombs, 1965), and erythrophagocytosis in vitro (Nelson, 1962) and in vivo (Mollison, 1965). Thus, C_3 has many important functions in the immune mechanisms (Muller-Eberhard et al, 1966).

C_3(β_{1C})is labile on storage in serum, and the main conversion product is a β-globulin called β_{1A} globulin. The conversion of β_{1C} to β_{1A} globulin, whatever its underlying mechanism, is of immunological importance because it leads to complete loss of the serological activity associated with the intact β_{1C}- globulin (Muller-Eberhard and Nilsson, 1960). Thus, C_3 converts during electrophoresis or when aged into β_{1A}- globulin (West et al, 1965; Laurell and Laurell,

1967) which has a faster electrophoretic mobility than β_{1C}. After electrophoresis the phenotypes are characterized by proteins appearing into regions. The slower group (β_{1C})migrates behind α2-macroglobulin, the faster one(β_{1A})behind transferrin. When sera were stored for 24 hrs at room-temperature or at -20° C for more than 2 months, proteins in the region behind α2-macroglobulin were found to disappear. The pattern of proteins in the region behind transferrin remains stable. It is advisable to use fresh serum samples. The interpretation of the bands in the faster regions is uncertain, because the protein bands of one type may overlap the bands of another type(Goedde et al, 1970).

Study of the genetically controlled molecular variation in human C_3 has been greatly facilitated by the circumstance that this protein occurs in normal serum in concentrations allowing it to be visualized easily in almost any zone electrophoresis of whole serum. The visualization is further improved if calcium is added to the electrophoresis buffers so that C_3 is slowed relative to transferrin and beta lipoprotein, an observation first made by Laurell, Laurell and Skoog (1956).

A polymorphism of the third component of complement(C_3) in serum was described by Ropartz et al in 1965,using methods of hemagglutination inhibition of C_3-coated red cells.No family studies were reported.With the usual methods of agar or starch-gel electrophoresis C_3(β_{1C}-globulin)is seen as a single band in the slow β -globulin region.Variants of this protein were suspected when Wieme(1965)noted that a few rare sera had an extra band in the C_3region of either slower or faster electrophoretic mobility than the usual C_3on agar electrophoresis

and that the extra bands disappeared with storage of the sera. By the use of high voltage electrophoresis on agar Wieme and Demeulenaere and Wieme and Seyers in 1967 and 1968,respectively, demonstrated duplication of C_3 bands in certain families from a Flemish and a Bantu population. Family studies indicated simple autosomal codominant inheritance of variant allele.

Genetically determined polymorphism of the third component of human complement (C_3) was demonstrated simultaneously by two research groups. Alper and Propp (1968) used a prolonged high voltage agarose electrophoresis and Azen and Smithies (1968), high voltage starch-gel electrophoresis. Both groups made observations consistent with the theory that the phenotype of C_3 is governed by autosomal co-dominant inheritance and presented evidence for the existence of two relatively frequently occurring genes as well as of rarer genes controlling this polymorphism. The discoverers of the C_3 polymorphism use different nomenclatures. Alper and Propp propose the symbols F and S for the common allele products, where F is the fastest (anodal) and S the slowest (cathodal) migrating band on electrophoresis. The F and S bands correspond to the bands 1 and 2 in the nomenclature of Azen and Smithies. The two groups exchanged sera with different C_3 phenotypes and found this correspondence of C_3 components: 1=F, 2=S. The most frequenty occurring phenotypes thus are SS(or 2-2), FS (or 2-1), and FF (or 1-1).

As mentioned above, C_3 proteins can be seen as distinct bands in the slow β -globulin region (β_{1C}- globulin). It has been postulated that each allele in the system controls a pair of protein bands, a heavy-staining band and, somewhat anodal to this, a lighter-staining band. C_3 types always re-

fer to the pattern of the cathodal heavy-staining bands.Normally, the C_3 fast band stains slightly more intensely than C_3slow bands in C_3 heterozygous individuals(i.e.C3FS)(McLean and Hoefnagel, 1980).

At the moment, our present assemblage consists of more than 20 structural electrophoretically distinct variants of C_3 in human sera. Two genes C_3^S and C_3^F and three phenotypes C_3SS, C_3FS and C_3FF are found in more than 99% of the populations. (Figure 2.2.3.a). All the other variants due to mutated alleles at the C_3 locus are very rare. The rare variants of C_3 have been described by different authors (Rittner and Rittner, 1974; Alper and Rosen, 1976). Figure 2.2.3.b. shows the twenty structural electrophoretically distinct forms of C_3 in human sera.

If the original fast variant is called C_3F_1 and the original slow variant is S_1, then any new variant may be assigned as subscript which expresses its migration relative to C_3S and either one of these variants. Thus, a variant migrating 60% of the distance between C_3S and C_3S_1 is called $S_{0.6}$. Another variant migrating a head of F_1 is $C_3F_{1.1}$ and so on.

The variant protein bands reported so far have been named $F_{1.2}$' $F_{1.1}$' F_1' $F_{0.85}$' $F_{0.8}$' $F_{0.5}$' $S_{0.4}$' $S_{0.6(or\ 3)}$'$S_{0.8}$'$S_{0.9}$ and $S_{1(or\ 4)}$in order of descreasing electrophoretic mobility (Alper, 1969; Azen and Smithies, 1968; Teisberg, 1971; Farhud, 1972).

In addition, the existence of a 'silent' or non-expressed allele of the system has been suggested.

Genetically controlled deficiencies of almost all complement components in humans have been described (Donaldson and Evans,1963; Klemperer et al, 1966;Abramson et al,1971; Leddy

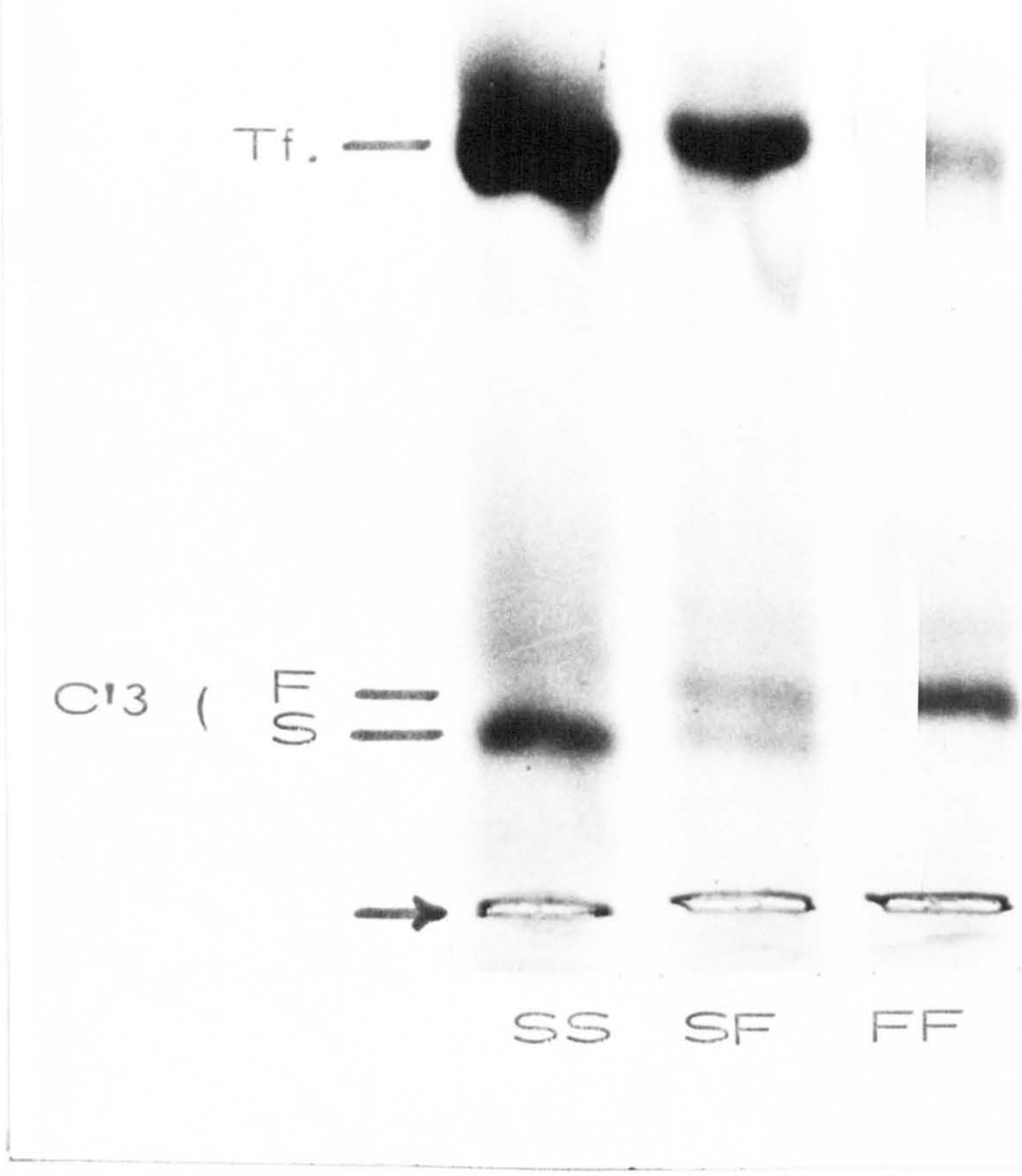


Fig.2.2.3. a. Agarose gel electrophoretic patterns of the three common C3 phenotypes, C3 SS, SF and FF (Farhud,D.D. 1972).

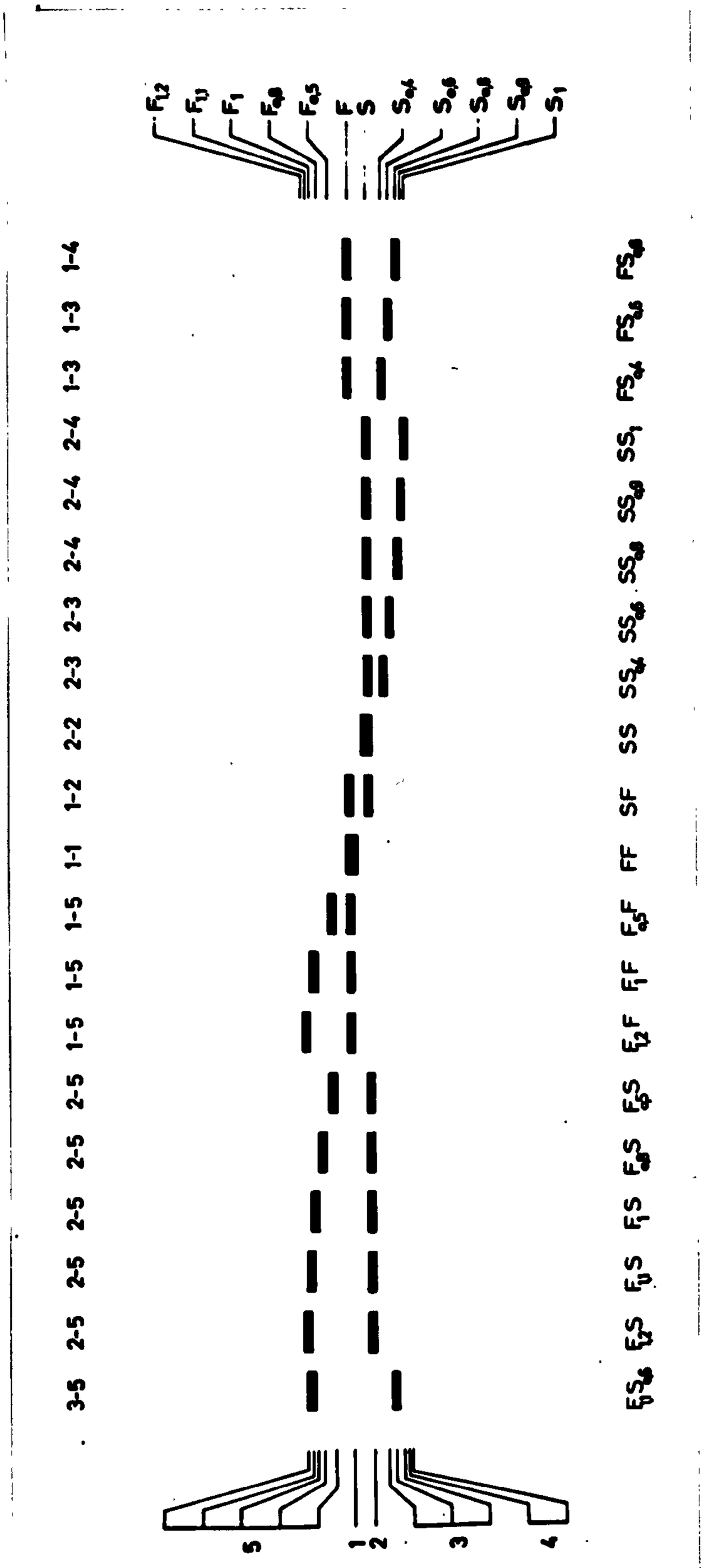


Fig.2.2.3.b. Diagram showing the twenty structural electrophoretically distinct variants of C3 in human sera. The nomenclature of Azen and Smithies (1968) is shown on the left and that of Alper and Propp (1968) on the right (Farhud, D.D. 1972).

et al, 1974; Boyer et al, 1975; Peterson et al, 1975; Rosenfeld et al, 1976). Most of these cases were discovered more or less incidentally due to their increased susceptibility to infections or during other clinical investigations.A case of inherited partial deficiency of $C_3$was first discovered by Alper et al in 1969.The family survey revealed a C_3^- gene in 7 family members.Other cases of inherited partial deficiency of C_3 were reported by Alper et al(1972),Alper and Rosen(1976). C_3deficiency regarded as a rare homozygote has been reported by Alper et al (1976)and by Papiha et al (1979). The association of C_3 phenotypes and various diseases were first reported by Farland and Raeder(1972).The C_3S phenotype has been found to be associated with higher titre of immune antibodies than the C_3FS and C_3F phenotypes in normal persons(Bronnestam and Cedergren,1973).The observation that there is a significantly lower C_3^Fgene frequency in pyelonephritis could support the assumption that the C_3S phenotype is in some way less efficient than C_3F phenotype in combating infections(Stoffersen and Jorgensen, 1980).

All C_3 phenotypes have the same haemolytic activity (Alper and Propp,1968)and are found in the same quantity in human blood (Agarwal et al, 1972).Arvilommi(1973)has shown that there is a difference between the common phenotypes (C_3F, C_3FS, C_3S) in their ability to bind mononuclear cells. The C_3F has a greater binding capacity than the C_3S type. The different phenotypes of the C_3polymorphism seem to have different biological properties, as shown by their association to various diseases.

A high incidence of C_3F phenotype has been found in patients with Rheumatiod arthritis (Farhud and Walter, 1972; Bronnestam, 1973), atherosclerosis (Disssing et al, 1972),hepatitis

(Farhud et al, 1972), renal diseases (Stoffersen and J rgensen, 1980), cystic fibrosis (Schi tz et al, 1978).

Family studies have so far supported the theory of autosomal codominant inheritance of C_3^{F1}, C_3^{F}, C_3^{S}, $C_3^{S}0.6$, and C_3^{S1}.

It was of interest to determine whether the C_3 variants all functioned normally as the third component of complement. Total haemolytic complement was normal in all variant-containing sera. This, of course, is a rather gross measure and even if a variant were totaly inactive, haemolytic complement might be normal or only slightly reduced since, except for C_3F and C_3S all the variants have been found only in heterozygotes and C_3 concentration must be less than 50% of normal to effect a lowering of the CH_{50} (or total haemolytic capacity).C_3functional activity paralleled immunochemically determined concentration in C_3 heterozygous sera containing each of the variants (Alper, 1970).

There seem to be racial differences in C_3 allele frequencies which may become valuable in anthropological studies (Teisberg,1971). It is clear that the gene frequencies differ strikingly among the races of man. The allele frequencies of human C_3 polymorphism found in some population groups indicate that the system may become a valuable tool in practical work in the field of human genetics.

The S gene is common in the three major races of man. The F gene is relatively common in Caucasians,less common in American Negroes,and relatively uncommon in Orientals.The F_1,$S_1$and $F_{0.8}$ genes are probably rare in all populations. In most Caucasians, the C_3S allele has an approximate gene frequency of 0.80, while the F gene has a frequency of about 0.20 (Seth

and Seth, 1976). The other allotypes, F_1, $F_{0.8}$, $F_{0.5}$, $S_{0.6}$ and S_1, are relatively rare. However, the SS type is the common in all populations studied thus far (Alper and Propp, 1968; Azen and Smithies, 1968; Berg et al, 1970; Bronnestman, 1971). As mentioned above, the F gene appears to be rare in Negroes since it may be that the low incidence of C_3^F in American Negroes represents a mixture of Caucasian genes (Alper, 1970). At present, nothing can be said about the possible selective effects of climate and living conditions on the allele frequencies. The allele frequencies of C_3 in Caucasian population groups (Alper and Propp, 1968; Azen et al, 1969; Teisberg, 1970; Farhud and Walter, 1973) indicate that the system may become useful in cases of disputed paternity. Much reference material is a prerequisite to the use of this polymorphic system in forensic serology. It appears that the rare C_3 proteins described up to now can be used as genetic markers in forensic serology. It was feared, however, that the lability of C_3would prove to be a major obstacle to its forensic use as these blood samples may be exposed to unfavourable conditions for several days during shipment.

Since C_3 is present in serum in a concentration higher than that of the other known complement components and is vitally important in immune mechanism (Muller-Eberhard, 1968), investigations of the C_3 polymorphism is of interest for both geneticists and immunologists.

The results obtained so far on C_3 polymorphism suggest that the system should be a valuable marker in population studies. The instability of the complement component C_3 may, however, cause some practical problems in the field of population genetics, since a certain fraction of serum samples may be diffi-

cult to type with certainty, due to storage alteration.

2.2.4. The group specific component (Gc) system

The Gc group of the plasma is the expression of genetically controlled variants of one of the ∝2-globulins. It was not recognized as a distinct protein until 1959 when, Hirschfeld, by means of immunoelectrophoresis of human serum, showed the precipitation pattern of this ∝2-globulin besides haptoglobin as showing systematic variation in appearance and migration rate. The three phenotypic patterns consist of a fast, a slow, and an intermediate (bimodal) arc. Mixture of sera containing the slow and fast types resulted in a pattern indistinguishable from the intermediate type. This variable globulin was named "Group specific component" or Gc. Family studies by Hirschfeld et al (1960) showed that the two main Gc proteins are the product of a pair of autosomal codominant alleles, called Gc^1 and Gc^2, giving rise to the expected three phenotypes, Gc1-1, Gc2-1, and Gc2-2, both genes being expressed in the heterozygote to give a mixture of the two proteins. Thus, individuals homozygous for Gc^1 have the fast moving component; those homozygous for Gc^2 have the slow-moving component; and heterozygotes have the intermediate arc containing both components. By means of immunoelectrophoresis each homozygous serum appears to contain a single Gc protein, and the heterozygote a mixture of the two. Starch-gel electrophoresis shows two bands for each homozygote, and three for the heterozygote, but the two bands for a given gene product may represent a single substance with different electrical charges. On starch-gel electrophoresis this protein migrates between transferrin and albumin (Figure 2.2.4.a).Recently, a new technique of Gc typing based on isoelectric focusing, polyacrylamide gel

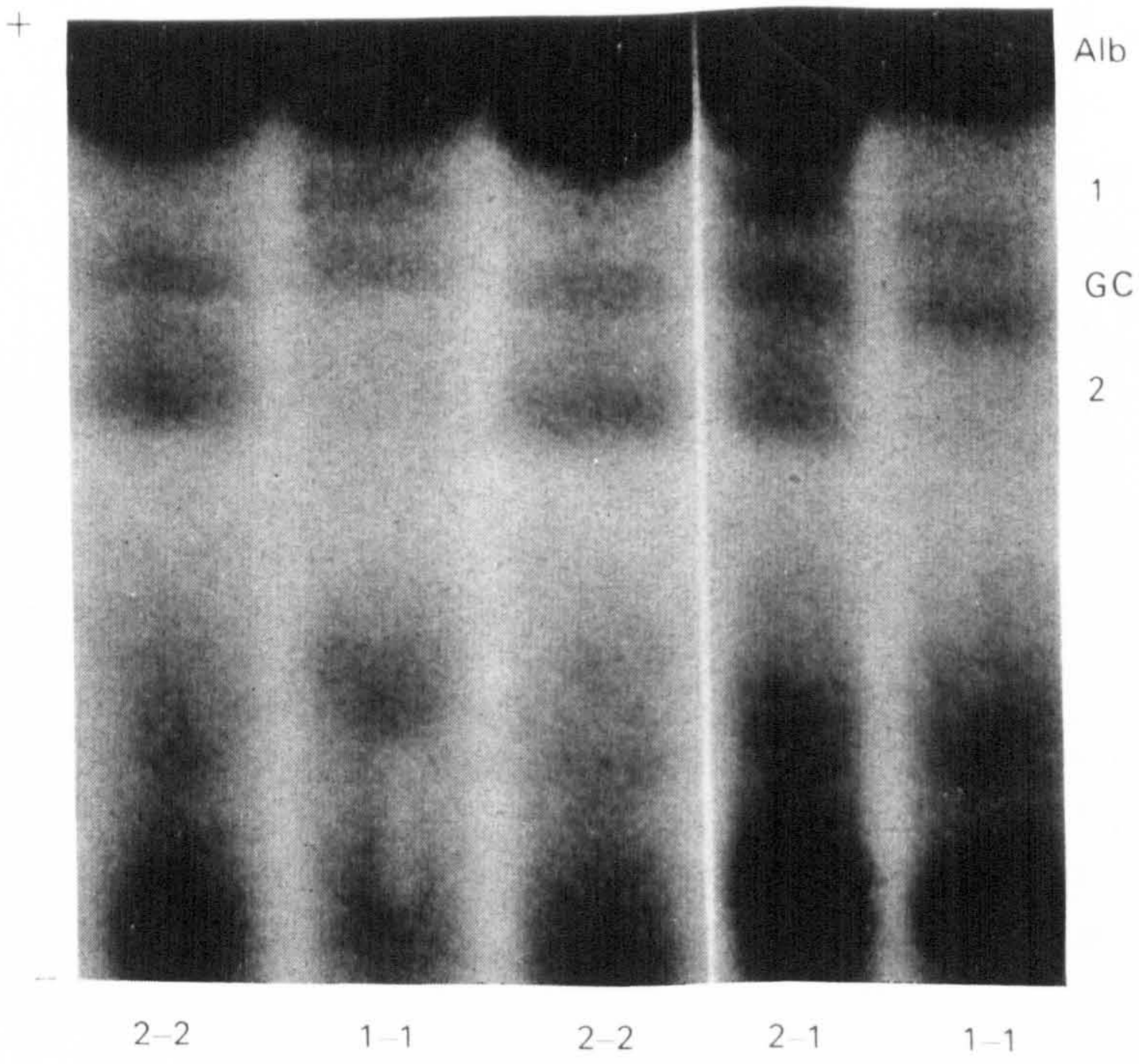


Fig. 2.2.4.a. Starch gel electrophoretic patterns of the three common Gc phenotypes Gc 1-1, 2-1 and 2-2(Bearn,A.G.,et al.1964).

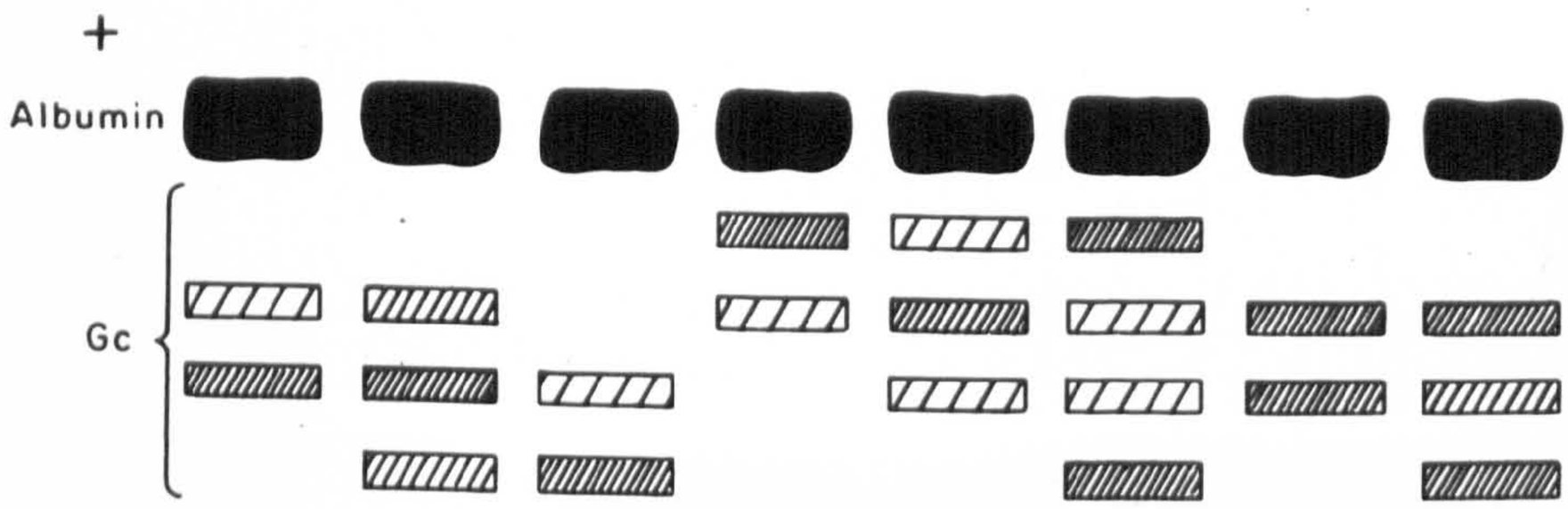


Fig.2.2.4.b. Diagram of the three common Gc 1-1, 2-1 and 2-2 and some rare Gc phenotypes (Kitchin, F.D., and Bearn,A.G. 1966).

isoelectrofocusing (PAGIF) with immunofixation was elaborated by Constans and Viau (1977), Constans et al (1978). Thereby, it was possible to demonstrate subtypes of the Gc^1 genes and several additional rare Gc variants. This method revealed heterogeneity in the Gc^1 band zone, which is explained by the existence of two Gc^1 suballeles, Gc^{1F} and Gc^{1S}; the resultant phenotypes are Gc1F-1F, Gc1F-1S, Gc1S-1S,Gc2-1F, Gc2-1S and Gc2-2. This six Gc phenotypes can be explained by the three common alleles, Gc^{1F}, Gc^{1S} and Gc^2. In addition to the common alleles, a considerable number of alleles are known, all of which are everywhere rare. In fact, of the aberrant Gc genes, only Gc^{chip} and Gc^{Ab} occur with a frequency of one percent or more in any known population.(Figure 2.2. 4.b). At an international workshop on the Gc system held in July 1978 (Constans et al, 1979) the Gc phenotypes determined by a total of 30 different alleles were demonstrated. Of these Gc variants, 21 are presumably mutants of the Gc^1 allele, and 9 are presumably derived from the allele Gc^2. The existence of a rare Gc^O ' silent' allele was suggested by Henningsen (1966) from studies of a family with anomalous inheritance. In nearly all previous population studies, the Gc^2gene has a lower frequency than Gc^1:

The importance of the Gc groups, other than as genetical markers, has recently been realized as a result of the discovery by Schanfield et al (1975) that the Gc proteins are the vitamin D carriers of the plasma. Subsequently Daiger et al (1975; 1977) showed that this protein is capable of binding vitamin D, a feature confirmed by many other experiments (Van Baelen et al, 1978; Haddad and Walgate, 1976).

The metabolic role of the protein and its polymorphism contribute to its importance in clinical and biochemical research. In addition, the fact that it is observed at different frequencies of the Gc genes in different population studies, makes a knowledge of the genetic composition of a population imperative in studies of many disease states. Wendt et al (1968) and Cleve (1973) have summarized work on associations of diseases with Gc types. There are indications that Gc1-1 may be associated with psoriasis (Jorgensen and Hopfer,1967) and less probably with carcinoma of the uterus, neurodermatitis, diabetes mellitus, and rheumatoid arthritis. Kitchin et al (1972) suggest an association of the Gc^{Ab} variant with Kuru in New Guinea. As shown by Mourant et al (1976) Gc has in general, though with several exceptions, a high frequency in sunny climates and Gc^{2} a relatively high one in dull ones, suggesting that the Gc types are affected by natural selection related to the availability of vitamin D. It is therefore desirable that Gc distribution should be investigated in patients with rickets, which results from vitamin D deficiency, as well as if possible, in the very rare condition of hyper-vitaminosis-D.

Kirk et al (1963), Walter and Steegmuller (1969), Mourant et al (1976), and later Daiger (1979) have demonstrated that the frequency of the gene Gc^{2} follows a cline, diminishing as the mean intensity of solar radiation increases. The data recovered in the investigations show the presence of a gradient in the frequency of the gene Gc^{1F} which increases from Europe to equatorial Africa. This gradient may be superposed on that of skin pigmentation (Loomis, 1967) which has always been considered to be linked to genetic and adaptative

conditions (Hiernaux, 1977). The lowest frequencies of the gene Gc^2 correspond to regions where sunlight is stronger. There is also a north-south gradient in the Gc^{1F} gene frequency. This seems to parallel the gradient seen in skin pigmentation. Group specific component polymorphism, as studied by modern electrophoretic techniques, represents an extremely instructive marker system for anthropological research. The frequencies of the Gc alleles and their presence or absence in the populations studied help us to evaluate the population dynamics. Every human population studied to date is polymorphic for the Gc protein. Even in a population completely lacking the Gc^2 allele, a polymorphism is present, because the Gc system is multiallelic. The distribution of the Gc alleles seems to correlate with geographical clines, notably the incidence of solar radiation as might be expected for the protein which transports vitamin D.

2.2.5. The ceruloplasmin (Cp) system

Ceruloplasmin is an α 2-globulin, which contains more than 90 percent of the plasma copper. It is a glycoprotein with a 7 percent carbohydrate content. Each molecule contains eight copper atoms (Holmberg and Laurell, 1951). It was discovered in 1948 by Laurell and Holmberg. The protein is known to have oxidase activity in vitro (Holmberg and Laurell, 1951; Walaas et al, 1967).

Though its biological function is not yet quite clear, it seems to play a role in copper metabolism, such as being involved in the enzymatic transfer of copper to copper containing enzymes like cytochromoxidase (Bearn, 1966; Schultz and Heremans, 1966; Schreffler et al, 1967, 1968, Shokeir and Schreffler 1969; Marceau et al, 1970).

Genetically determined structural variants of ceruloplasmin was detected by McAlister et al (1961); and Martin et al (1961). They described a fast moving electrophoretic variant of this protein, which was found to be controlled genetically. However, no further evidence of inherited variants of this protein was obtained until the studies of Schreffler et al (1967, 1968), demonstrated a genetically controlled polymorphism of the ceruloplasmin . It has long been known that different plasmas tend to show different band patterns of ceruloplasmin on electrophoresis, but as the molecule is somewhat unstable it proved difficult to distinguish between gene controlled variants and the effect of molecular dissociation. As Schreffler et al (1967) showed, however, under suitable conditions clean band patterns can be obtained, each of five alleles giving rise to a characteristic band, and each heterzygote showing the bands of the two constituent genes. On the basis of family studies Schreffler et al assumed the existance of three allelic genes: Cp^{A}, Cp^{B} and Cp^{C} at the autosomal Cp-locus, determining six phenotypes: CpA, CpB, CpC, CpAB, CpAC and CpBC. Two further phenotypes: CpB-NH and CpA-NH produced by an additional allele Cp^{NH} (NH. Stands for new haven) have been demonstrated in 1967 by Shokeir et al (Figures 2.2.5.a, b and c). The electrophoretic migration rates of the product of the alleles in the starch gel decrease in the order: Cp^{A}, Cp^{B}, Cp^{NH}, Cp^{C}. Finally, Shokeir et al (1968) found a further ceruloplasmin variant in Tailand, controlled by an allele Cp^{Th}, the product of which migrates with a mobility similar but not identical to that of Cp^{A}.

The common phenotype, CpB, represent homozygosity for the gene, Cp^{B}, which has a frequency of about 94 percent in Negroes and



Fig.2.2.5.a. Ceruloplasmin patterns of (from left to right) CPB, CPAB, CPB, CPAB, CPAC, and CPBC sera after electrophoresis on starch gel and staining with O-dianizidine. The anode is at the top(Shokeir, M.H.K., and Shreffler,D.C. 1973).

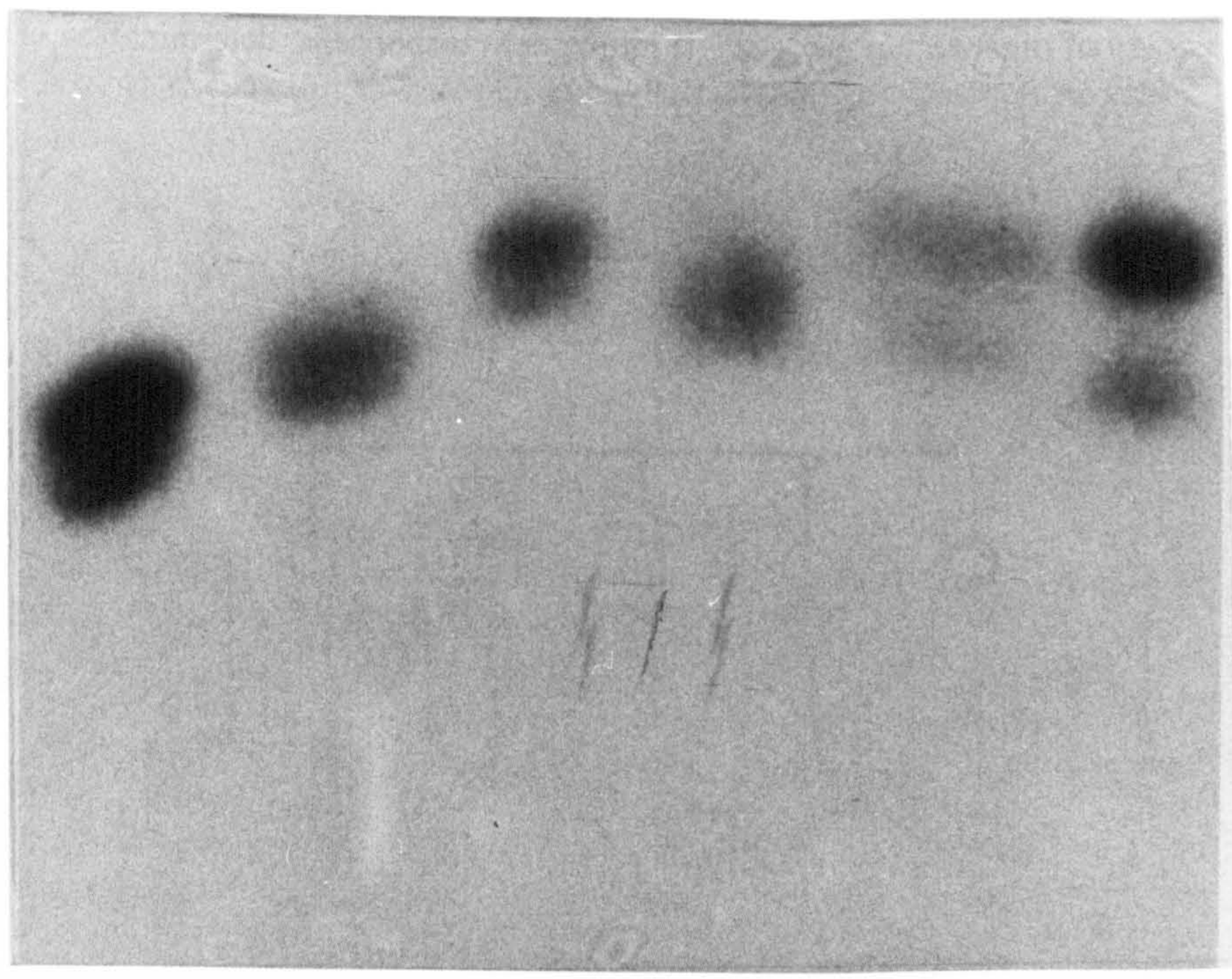


Fig.2.2.5.b. Ceruloplasmin patterns of (from left to right) CPBC, CPBNH, CPAB, CPBNH, CPANH, and CPAC sera after electrophoresis on starch gel and staining with O-dianizidine.The anode is at the top (Shokeir,M.H.K., and Shreffler, D.C. 1973).

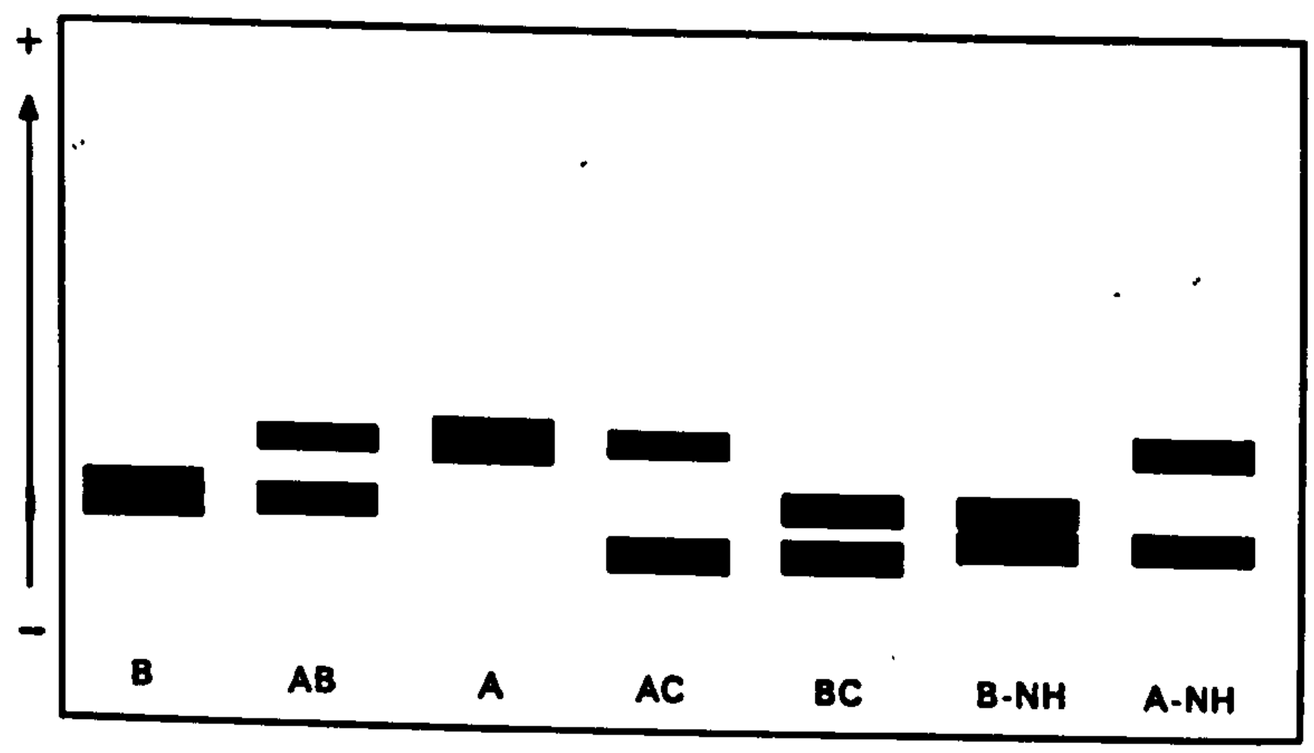


Fig.2.2.5.C. Diagram of the starch gel electrophoretic patterns of the seven ceruloplasmin phenotypes described by Shreffler et al. (1967) and Shokeir et al.(1967).The rare CP phenotype B-NH is very difficult to differentiate from the common CPB phenotype(Giblett, E.R.1969).

over 99 percent in non-Negroes tested. The Cp^{A} gene, with a frequency of about 5 percent in Negroes, was also found in the homozygous state, as designated by the phenotype CpA.The genes Cp^{C} and Cp^{NH} have very low frequencies, estimated as 0.4 percent and their gene products have been seen only in combination with Cp^{B} and Cp^{A}.

In all populations hitherto tested Cp^{B} is of universal distribution and very high frequencies, whereas the alleles Cp^{A}, Cp^{C}, Cp^{NH} and Cp^{Th} show much lower frequencies. An exception seems to be Cp^{A}, because its frequency ranges between 0.04 and 0.15 in Negroes, it was therefore suggested that it can be used as a genetic marker indicating Negroid influence(Giblett, 1969). However, some remarkable racial differences seems to exist in this system. Though the ceruloplasmin polymorphism has been well known for several years, there are, in contrast to other recently detected polymorphism, surprisingly few publications on its allele distribution in different ethnic groups. The frequency of variants in all hitherto tested populations except the Negroes is very low.

The ceruloplasmins are probably of considerable importance in mineral metabolism, and it may be that certain alleles are selectively favoured in conditions of copper deficiency in the environment. However, it seems that it is too early to make general statements about its importance or non-importance for population genetical studies.

2.2.6 The α_1-antitrypsin (α_1AT=pi) system

The term 'protease inhibitor' is applied to a group of serum proteins which have the property of inhibiting the activity of proteolytic enzymes such as trypsin, plasmin and thrombin. The protease inhibitors which have been most intensively

investigated (Ganrot, 1967) are in the α_1 and α_2 fractions. Human plasma contains several protease inhibitors both in the α_1- globulin and the α_2-globulin fractions, such as α_1AT, α_2 macroglobulin, α -anti-chymotrypsin and inter- α -tripsin inhibitor.

Only the α_1 variety has been found to exhibit electrophoretic polymorphism, and it is the main trypsin inhibitor present in plasma and serum (Schultze et al, 1962). The protease inhibitor effect of α_1-antitrypsin is due to the formation of an inactive complex with the proteolytic enzyme (Rimon et al, 1966.).

The history of investigations into its genetics has been summarized by Giblett (1969). Among the protease inhibitors present in human serum, α_1AT is of special interest as it exists in high concentration, has approximately 90 percent of the serum's trypsin inhibitory capacity, and is active against other proteolytic enzymes, including chymotrypsin, elastase, collagenase, plasmin, thrombin and protease from human leukocytes. This protein shows a high degree of genetically determined heterogeneity; at least 25 alleles already have been identified. Nearly one half of the individuals in European and United States population samples are heterozygous for this system (Kueppers and Christopherson, 1978). There are, however, substantial differences in gene frequencies among human populations. When only the types detected by starch gel electrophoresis were considered, Asian populations were apparently more homozygous or almost monomorphic in the pi system(Ashley et al, 1980). With the availability of 'subtyping' by isoelectric focusing it is of interest to examine these groups for heterogeneity.

Genetic variants with severe deficiency can predispose to disease, as to pulmonary emphysema (Laurell and Eriksson, 1963), or be associated with Cirrhosis (Sharp, 1970). This deficiency was found to be hereditary and to form only part of a complex polymorphism at the chromosome locus which has been called pi. Famuly studies showed that the deficient state represents homozygosity for a mutant gene, and that heterozygotes have about half normal activity (Eriksson,1964, 1965; Kueppers et al, 1964; Lopez et al, 1964; Talamo et al, 1966; Ganrot et al, 1967).

The existence of other α_1-antitrypsin variants in serum was first reported by Eriksson and Laurrell (1963). They described a slow moving variant subsequently shown to be inherited as an autosomal codominant, and its gene was found to be allelic to the gene determining the deficient variant (Axelsson and Laurell, 1965). Fagerhol and Braend (1965) reported that when human serum is subjected to starch gel electrophoresis, several bands with variable patterns in the prealbumin region, appear. These prealbumin zones were recognized α_1-antitrypsin by Kueppears and Bearn (1966),Fagerhol and Braend (1966) and Fagerhol and Laurell (1967). The first three phenotypes described are the common type, called MM, a rare type, FF, and the ZZ type, which designates α_1-antitrypsin deficiency. These three types represent homozygosity for the common gene, Pi^M , and the rare genes Pi^F and Pi^Z, respectively. The product of Pi^F gene has faster mobility and that of Pi^Z gene slower mobility than that of the common Pi^M gene. (Figure 2.2.6.). Among twenty five alleles now known, all of them but one Pi^M, are rare in nearly all populations. The commonest allele, Pi^M has a frequency of at

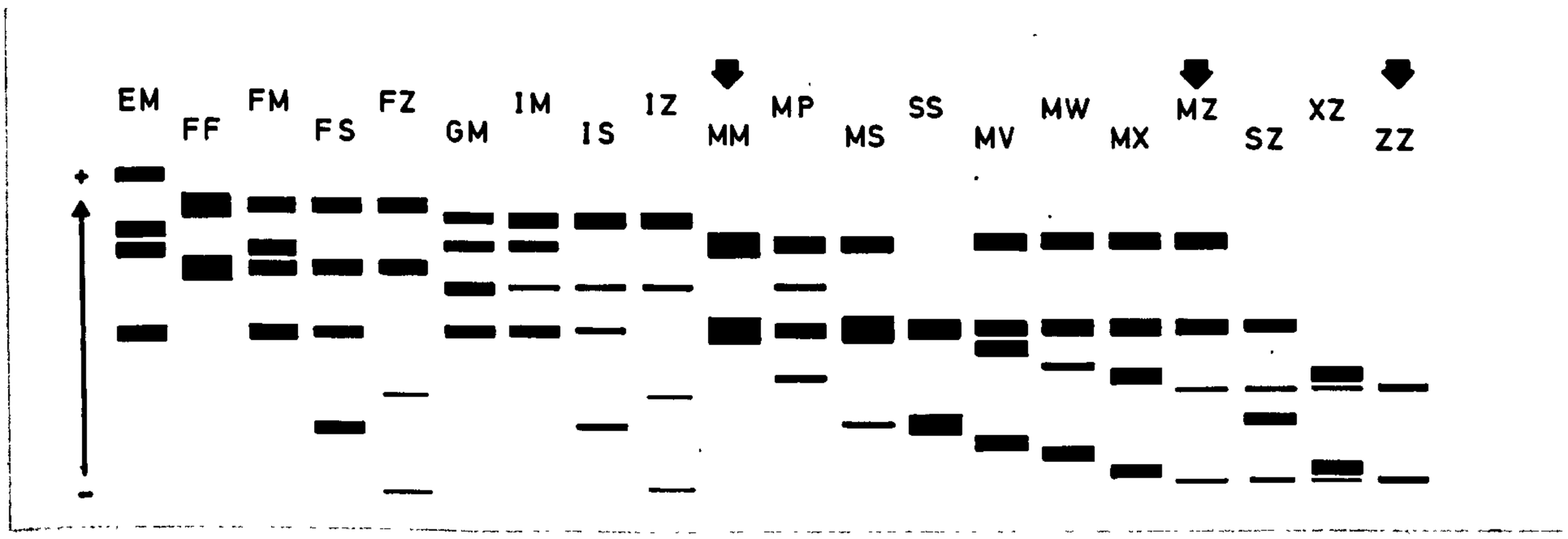


Fig. 2.2.6. Diagram of twenty Pi phenotypes, migrating ahead of albumin on starch gel electrophoresis at acid PH.(Fagerhol,M.K.1972).

least 85 percent in all populations tested.

The product of the gene Pi^S has somewhat reduced activity, but two alleles, Pi^Z and the very rare Pi^- (or Pi^0) are responsible, in the homozygous state, for most of the severe deficiencies causing pulmonary disease. The Pi^Z gene product has a small but measurable protease inhibitor activity, about 16 percent of that of Pi^M (Cook, 1975), and the protein itself gives the normal precipitin reaction of the α_1 protease inhibitors. The plasma of Pi^- homozygotes gives no detectable protease inhibitor activity and no precipitin reaction, so that it may represent a gene deletion. Most of the electrophoretically abnormal variants are present in normal concentration in serum and show unimpaired inhibitory capacity. α_1AT levels can be depressed, however, in case of the Pi^Z, Pi^S, Pi^{WI}, Pi^P, and Pi^{null} alleles(Fagerhol and Tenfjord, 1968; Martin et al, 1975). Recently Fineman et al (1976) found a significantly increased frequency of Pi heterozygotes in Down syndrome patients whose mothers were more than 35 years old at the time of the patient's birth; whereas the frequency in patients whose mothers were less than 35 years old was not significantly different from that in the general population. These data suggest that Pi^- phenotypes which comport a decreased α_1AT activity may in some way predispose to chromosomal errors.

In view of the known pathological association of one of the alleles and the marked variations in frequency of the electrophoretically detectable alleles, this system merits further application both anthropologically and in disease surveys.

2.2.7. The Pseudocholinesterase system

Red cells contain a cholinesterase (in official notation, acetylcholine acetylhydrolase, EC 3.1.1.7) which has the essential function of breaking down the acetylcholine produced at nerve endings of various kinds, especially those controlling voluntary muscles. It thus prevents the muscles from going into permanent spasm. Another cholinesterase, now commonly called pseudocholinesterase, and officially as acylcholine acylhydrolase, EC 3.1.1.8, is present in the plasma; its normal function is uncertain, but the existence of genetically determined variations in its activity came to light as a result of using another acylated choline compound, succinylcholine, as a muscle relaxant in surgical anaesthesia. This substance blocks the action of acetylcholine on voluntary muscles and so produces relaxation. Its effect is usually moderated by, and after some minutes most of it is broken down by,the action of pseudocholinesterase,so that the tone of muscles,including those of respiration, returns to normal. From time to time, however, individuals have been found who remain relaxed and without spontaneous respiration for prolonged periods.This has been found to be due to a deficiency of plasmatic pseudocholinesterase activity.Sometimes it could be explained as a secondary effect of the disease from which the patient was suffering,but it was liable also to occur in persons whose general health was excellent.In 1953,Forbat et al found one patient with a deficiency of the enzyme who had a healthy brother

with a similar deficiency.This group of workers then, and several others at about the same time (Harris, H., and Whittaker, M. 1962 and Kalow, W. 1959) began to look for further examples of inherited variants of the enzyme, and several came to light differentiated by their normal activity and its percentage inhibition in vitro under specified conditions by fluoride ions and by dibucaine. As a result of quantitative tests for enzyme activity with and without these inhibitors, and a further inhibitor which is a neostigmine analogue with the code name Ro2-0863, together with starch-gel electrophoresis and staining for enzyme activity, it has been possible to account for the various phenotypes in terms of the products of four allelic genes, the normal E_1^u, E_1^a which gives prdoducts with a reduced activity but relatively resistant to dibucaine inhibition, E_1^f whose products are relatively resistant to inhibition by fluoride, and E_1^s, the homozygote of which is almost totally inactive. E_1^s is, however, very much rarer than E_1^a, and it is the E_1^a homozygote which is the usual cause of trouble following relaxation anaesthesia.

Despite the rarity of variants, their clinical importance and biochemical interest have led to the performance of numerous population surveys. Some of these, depending upon inhibition with dibucaine, have distinguished only the genotypes containing E_1^a, and the very rare homozygote E_1^s E_1^s if present. Others, using also inhibition with fluoride have distinguished types containing E_1^f as well. The genetics and serology have been fully reviewed by Giblett (1969).

Besides the immediate products of the genes at the E_1 locus, some sera contain another similar esterase which is partly independent genetically, known as C5. This has been

described in a series of papers by Harris et al (1963). It is most easily demonstrated by electrophoresis in a rather acid gel, when all the bands directly due to E_1 alleles come together, and that due to C5 moves slightly more slowly towards the positive pole.

To a first approximation this enzyme behaves as the product of a gene at a locus independent of E_1, and known as E_2, with dominant expression. However, it appears not always to be expressed in the heterozygote, and the expression is also affected by the E_1 genotype. This is, therefore, not a very useful genetic marker.

2.3. Red cell enzymes

Enzymes have long been known as proteins which catalyse biochemical reactions, the name 'enzyme' having been proposed by Kuhne in 1878.

The concept of specific enzymes as products of single genes is inherent in Garrod's paper in 1902 on alkaptonuria as well as in his later classical work, Inborn Errors of Metabolism(1909). The concept was, however, given much more concrete expression by Beadle and Tatum (1941) in their 'One gene, one enzyme ' hypothesis of 1941.

Human genetics, however, had until recently concerned itself mainly with hereditary diseases and abnormalities, and the work of Krebs and others on the enzymatic control of normal metabolism was not at first seen as having any important relation to genetics.

Interest in variants of the normal proteins of the body was greately stimulated by Pauling's discovery in 1949 of sickle-cell haemoglobin, Smithies's discovery of genetic variants of haptoglobin (1962), and Lehmann and Ryan's demonstration of

genetic variants of pseudocholinesterase (1959); but the genetic study of variants of normal enzymes owes most to the work of Professor Harry Harris and his colleagues. Some enzymes showing polymorphism are present in the plasma, but a much greater variety of them is to be found inside the red cell.

Many enzymes of which genetic variants (isozymes) have been studied are involved in various cycles of normal glucose metabolism. In order to understand the methods used in testing for the enzyme variants it is necessary to have some understanding of these normal cycles and of the function of the enzymes in them. The isozymes have hitherto been treated mainly as formal genetic markers in family studies involving such phenomena as linkage, in which their precise functions are of secondary importance.

However, the wide differences in frequency of certain isozymes in different populations is almost certainly due largely to natural selection.

A knowledge of quantitative and qualitative functional differences between the genetic variants of a given enzyme is thus an essential part of the study, as yet scarcely begun, of the evolutionary mechanisms involved in the interaction between human populations and their physiological and microbiologicalenvironment.

Inside the boundary membrane of the red cell is a solution, possibly with an elaborate microscopic structure, containing haemoglobin and a variety of other substances, especially enzymes. Haemoglobin and a great many of the enzymes show genetical polymorphism. The enzymes have, of course, precisely known functions and in some cases the products of al-

lelic genes differ quantitatively in functional activity. For each offa considerable number of the enzymes there is at least one allele conferring low or absent activity, and homozygotes for such alleles may suffer from distinct congenital diseases.

The physical anthropologist is not directly interested in the quantitative substrate activity of particular enzymes, but only with their multiple molecular forms. These multiple molecular enzymes were termed isoenzymes by Markert and Moller (1959). The application of standard histochemical staining techniques to starch gels has enabled a large number of enzymes to be visualised, the resulting pattern being known as a zymogram.

2.3.1. The acid phosphatase (AcP) system

Human erythrocyte acid phosphatase (orthophosphate monoester phosphohydrolase, EC 2.1.3.2) was first shown to be genetically polymorphic by Hopkinson et al (1963), by means of starch-gel electrophoresis and appropriate staining. It catalyses the reaction involving the transfer of the phosphate ion from its ester to the inorganic state or to suitable acceptor substances. The precise function of this enzyme in the red cell is unknown. Five distinct electrophoretic patterns, designated A, BA, B, CA and CB, were described by Hopkinson, Spencer and Harris (1963 and 1964). 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 $p^a p^a$, $p^b p^b$, $p^c p^c$ respectively, and phenotypes BA, CA and CB represent heterozygous genotypes $p^a p^b$, $p^a p^c$ and $p^b p^c$. They have shown that the five different types were controlled by

three autosomal allelic genes p^a, p^b, and p^c. According to the three allele hypothesis, the BA, CA and CB types represent the heterozygous combinations, and A and B are homozygotes. This hypothesis predicted the occurrence of a sixth phenotype C, which should be relatively rare. Lai et al (1964) presented confirmatory family data and the finding of the C phenotype. At about the same time Harris and his colleagues also found the C type.

Two alleles, p^a and p^b, have been found to be polymorphic in all populations studied so far. The third allele, p^c, is polymorphic in some populations while totally absent in others. The relatively high frequency of p^c in European populations led to it being regarded as a 'Caucasian' gene by Scott et al (1966) and its occurrence in other populations is thought to be due to Caucasoid admixture (Tashian et al, 1967).

Each homozygous phenotype consists of two electrophoretically distinct isozymes. The AcP_1A phenotype has unique electrophoretic mobility, while AcP_1 types B and C are electromorphs, distinguished by differences in the relative intensity of the two isozymes. The erythrocyte enzyme is genetically and functionally distinct from the lysosomal AcP_1s of other tissues, although these tissues also contain the erythrocyte enzyme.

Figure 2.3.1.a shows that each type consists of two isozymes. The isozyme of type A exhibits two bands with about equal intensity and migrates faster than the other homozygous types. Type B has also two bands, but the faster band is more intense than the slow band. The mobility of the B band is slightly faster than the A band. The C type has two bands corresponding to the ones found in the B and CB types but the fast band is

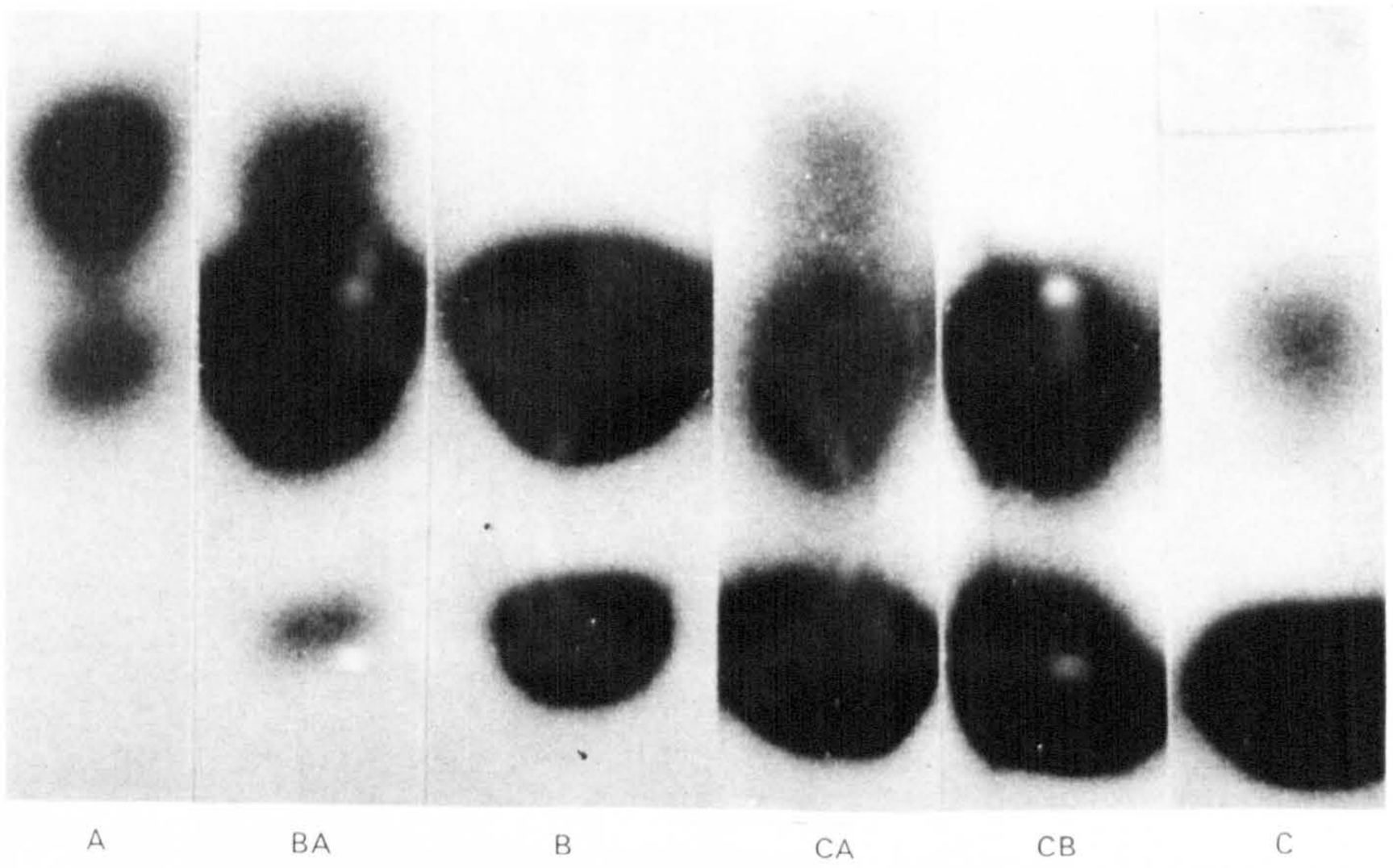


Fig.2.3.1.a. Electrophoretic patterns of red cell haemolysates with the six phenotypes representing homozygosity and heterozygosity for the three genes at the acid phosphatase locus(Giblett,E. R. 1969).

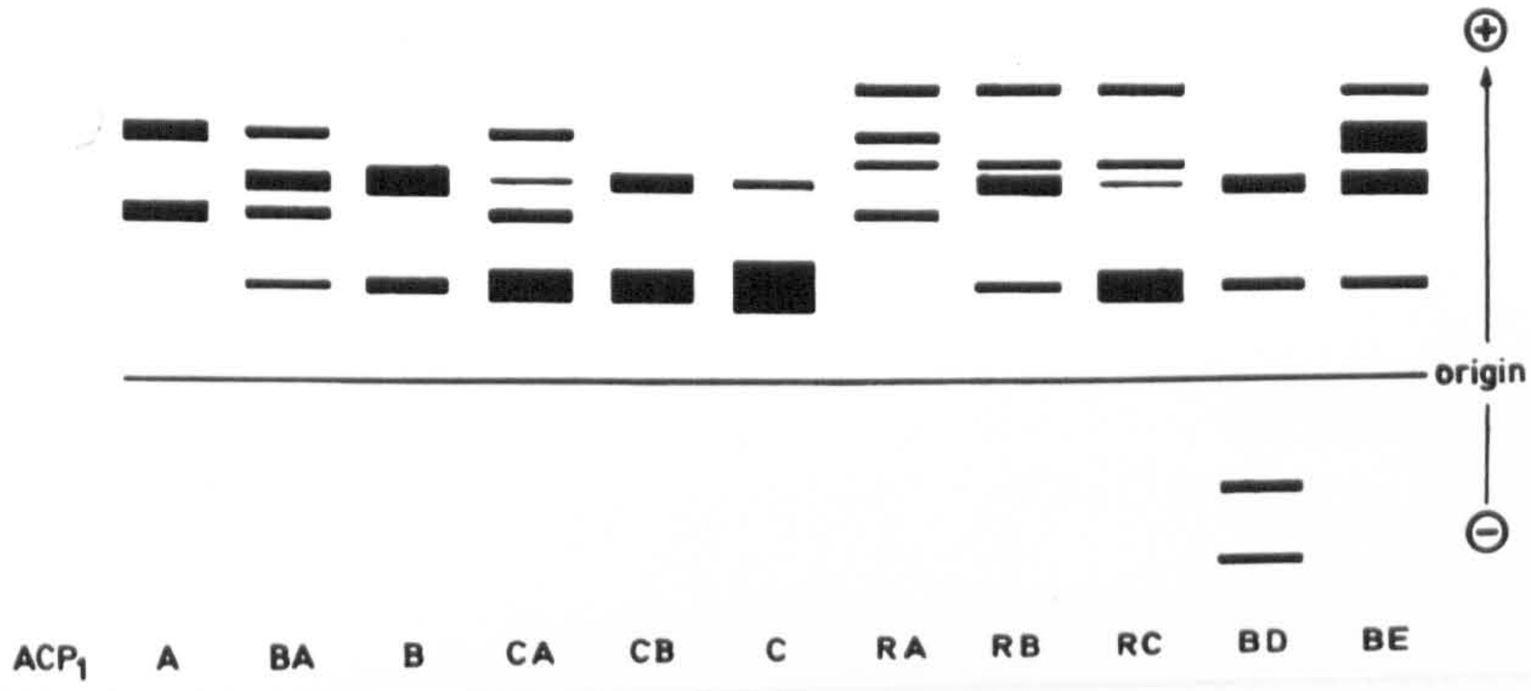


Fig.2.3.1.b. Diagram showing AcP isozymes in red cells from different phenotypes.Note that each of the alleles appears to determine two isozymes (Hopkinson,D.A., and Harris,H. 1969).

very faint and the slow band has a very high activity. The pattern in type BA, CA and CB, appears to be a mixture of the isozymes of their corresponding homozygous types. Type BA shows a complex pattern that is a combination of the components seen in the A and B types. The BA type can be reproduced by the electrophoresis of a mixture of haemolysates of type A and B in equal amounts. The part of the BA pattern that corresponds to the fast A components does not separate clearly, however, but appears as a smear. Both type CA and CB show a very intense slow band. Type CA has, in addition, two fast bands similar to the ones seen in type A. In type CB the intense slow band is associated with a faster band similar to the main band of the B type. 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 characterized 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 determines approximately the same amount of AcP as does the P^aallele,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 AcP locus (Figure 2.3.1.b). The occurrence of a rare AcP_1^0 allele has been described in two Australian (Herbich et al, 1970; Herbich and Meinhart, 1972) and several German families (Brinkmann, 1974), and in one Danish family (Dissing and Svensmark, 1976) and one fa-

tribution of AcP alleles indicated that some environmentally induced selective pressures are at work.The earliest observations on the possible adaptive value of the AcP polymorphism are attributed to Ananthakrishnan and Walter(1972),who discovered that the P^aallele frequencies are negatively and significantly correlated with temperature;they thus reached the conclusion that the P^ballele would be advantageous for populations living in hot climates.They found a marked gradient in the world distribution of the AcP alleles.The frequency of the P^ballele rises with the increase in mean annual temperature of the various biotopes whereas P^a shows a decrease.Even in a relatively small area,West Germany,they found a significant negative correlation between mean annual temperature and the frequency of the P^a allele.

Jenkins and Corfield (1972) speculated that selective forces could be acting against the P^c allele which carried the highest activity thus accounting for the current low P^c frequencies or, alternatively, that the P^c allele could be a relatively recent mutation which is advantageous and so is gradually increasing in frequency.

As regards the P^c allele distribution, Bottini et al (1972)and Palmarino et al (1975) hypothesised that this allele has some adaptive relevance concerning malaria, since its frequency is correlated positively with altitude and negatively with malaria morbidity in Sardinia in the past. There is some evidence from New Guinea of a negative association between the AB phenotype and the presence of malarial parasites (Harrison et al, 1976). The hypothesis formulated by Jenkins and Corfield(1972) postulated that in sub-Saharan Africa there could be a connection between the distribution of the P^rallele and distribution of various cultures,since this allele is more frequent

among the herder and hunter-gatherer peoples than among settled farming populations.

Observing that this latter distribution is closely associated with the geographical environment, Spedini et al (1978) surmised that the p^r allele could be negatively correlated with malaria, as appears to be the case with the p^c allele in the Mediterranean peoples subject to malaria. They have therefore attempted to ascertain whether the p^r allele, known as "Negro allele" is correlated with certain climatic factors more directly responsible for the incidence of malarial morbidity. The wide range of gene frequencies, and the quantitatively different activities of the gene products, make this system a most promissing one for studies of ecology and possible natural selection.

2.3.2. The adenylate kinase (AK) system

The enzyme adenylate kinase (AK) or ATp: AMP phospho-transferase (EC 2.7.4.3.) reversibly catalyses the reaction: Adenosine diphosphate $\rightleftharpoons$ Adenosine triphosphate + Adenosine monophosphate ($2ADP \rightleftharpoons ATP + AMP$)
within the red cell and in muscle and other tissues. Its molecule is extremely stable, which renders it highly suitable for investigation in far-travelled blood specimens.

The polymorphism of the human red cell adenylate kinase was first described by Filds and Harris (1966), by means of starch-gel electrophoresis. Population and family studies indicate that the variants o AK are genetically determined by two co-dominant autosomal alleles, called AK^1 and AK^2 . Individuals homozygous for the alleles are phenotypes AK1-1 and AK2-2 respectively, and heterozygous persons possessing bothe AK^1 and AK^2 genes have the AK2-1 phenotype. Three dif-

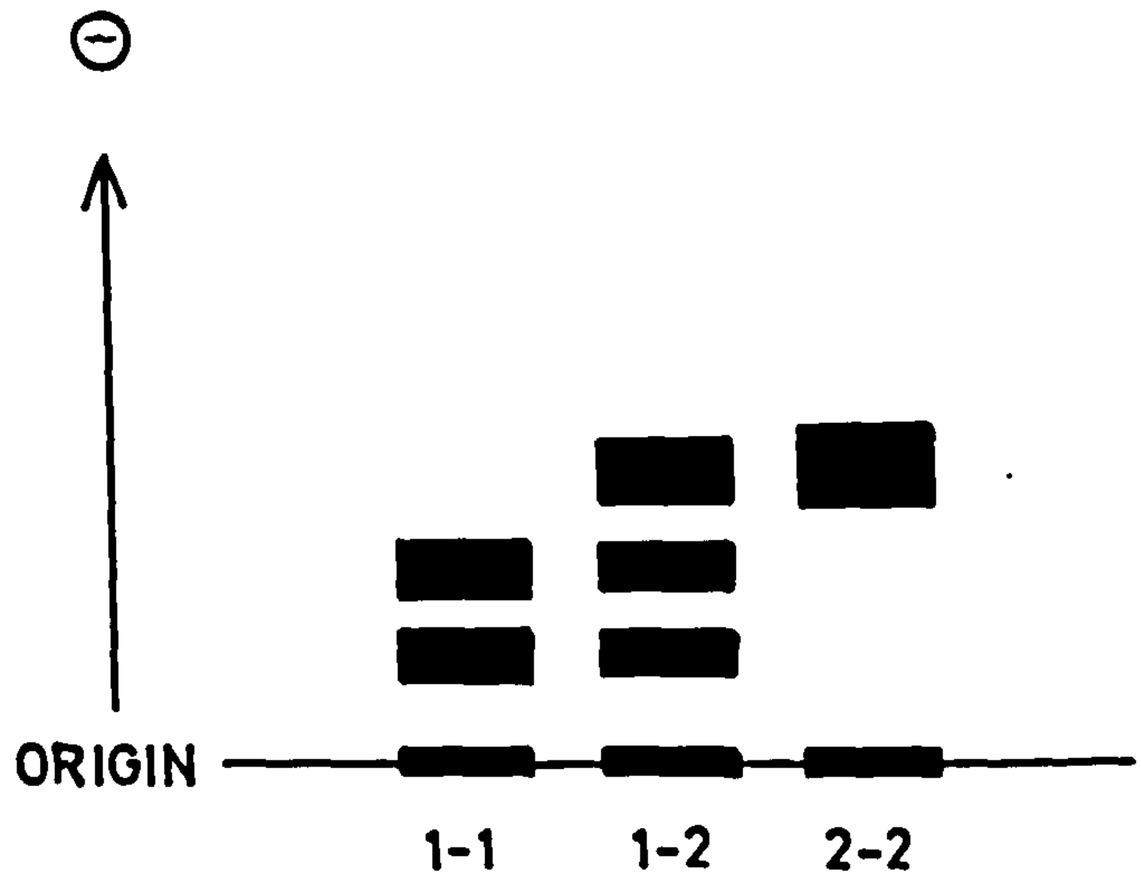


Fig. 2.3.2.a. Diagram of the three common AK phenotypes AK1-1, AK2-1 and AK2-2. Electrophoresis at PH 5.0.

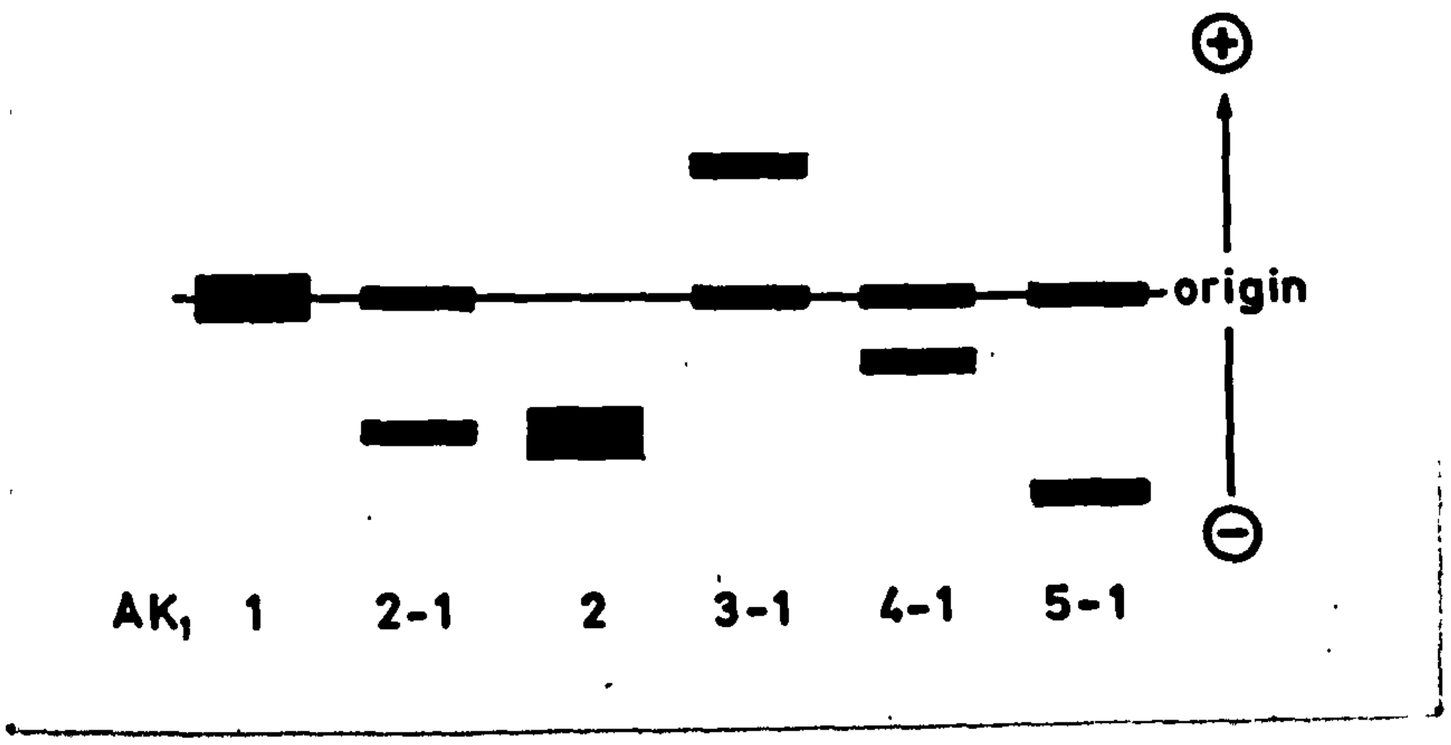


Fig. 2.3.2.b. Diagram of various AK phenotypes. Only the main isozymes are shown. Electrophoresis at PH 7.0(Harris,H., and Hopkinson,D.A. 1976).

ferent isozyme patterns referred to as AK1-1, AK2-1 and AK2-2 are shown in Figure 2.3.2. a. The most common phenotype, AK1-1 consists of two major components, of which the most intense remains to the fastest, and the less active zone migrates slower, a third slower-moving component stains poorly and tends to be diffuse. The AK2-1 pattern consists of the same three components,but with relatively less activity, and in addition a fourth zone which moves faster. The pattern seen in AK2-1 represents a mixture of isozymes present in AK1-1 and AK2-2. The AK2-2 pattern consists of one fast component which stains strongly. In addition to the common types, further variants due to the very rare genes, AK^3, AK^4 and AK^5 have been described (Bowman et al 1967, Rapley et al 1967, Benercetti et al 1972). Individuals with such aberrant phenotypes are heterozygotes for the usual allele AK^1 and the rare allele AK^3, AK^4 and AK^5 (Figure 2.3.2.b.).

The only alleles of any substantial importance in population studies are AK^1 and AK^2, the AK^2 gene being by far the rarer of the two with a frequency of only about 5 percent. With the large differences in adenylate kinase frequencies found in different parts of Asia, the system may prove of considerable value in tracing population relationship in this continent.

2.3.3. The phosphoglucomutase (PGM) system

The enzyme phosphoglucomutase PGM_1 (α-D-glucose 1.6. diphosphate: α-D-glucose-1-phosphate phosphotransferase, EC 2.7.5.1) reversibly catalyses the transfer of a phosphate group between the 1 and 6 positions of glucose. It occurs in the red cells and several other human tissues (Hopkinson & Harris, 1965, 1966; Renninger, 1969; McAlpine et al, 1970) .

The existence of multiple isozymes of human PGM detectable by starch-gel electrophoersis, and inherited variation in this enzyme were demonstrated by Spencer et al (1964). Since then, three independent loci, PGM_1, PGM_2 and PGM_3 have been indentified (Spencer et al, 1964; Hopkinson and Harris, 1965, 1966, 1968), and assigned to chromosomes 1, 4 and 6 respectively (Nguyen et al, 1971; Douglas et al, 1973; McAlpine et al, 1975; Jongsma et al, 1973). In most tissues the isozymes of PGM_1 predominate, exhibiting about 90 percent of the total PGM activity; the residual activity is yielded mainly by PGM_2 and to a slight extent by PGM_3 (Harris and Hopkinson, 1976). In the red cells, however, only PGM_1 and PGM_2 isozymes are expressed, and they display nearly equal activities. This means that erythrocytes have a smaller amount of PGM_1, but a correspondingly higher content of PGM_2, approximately 50 percent (McAlpine et al, 1970). The PGM_3 contribution is relatively small in all tissues and apparently not detectable in erythrocytes at all (Harris and Hopkinson, 1976; Quick et al,1974). Bissbort et al (1975), however, were able to demonstrate the polymorphism of PGM_3 in erythrocyte haemolysates by means of horizontal starch-gel electrophoresis. The three common phenotypes in whites, detectable on PGM-stained starch gels are expressions of two common alleles at PGM_1 (PGM_1^1 and PGM_1^2) and analogously at PGM_3 (PGM_3^1 and PGM_3^2).

The PGM_2 isozymes can be characterized by a remarkable phosphoribomutase (PRM) activity (Quick et al, 1972), whereas PGM_1 and especially PGM_3 are not efficient in this reaction(Quick et al,1974). Due to low PGM_3 activities in all tissues, Harris(1975) suspected that its isozymes have another main catalytic activity, which is so far unrecognized.

The different loci of phosphoglucomutase probably arose from gene duplications during early vertebrate evolution (Engle and Schmidtke, 1975). Spencer et al (1964) demonstrated that when red cell haemolysates are subjected to starch -gel electrophoresis, seven different zones of PGM can be observed,labelled a, b, c, d, e, f and g from the cathodal end,which appear to be due to alleles at two distinct and not closely linked autosomal loci, PGM_1 and PGM_2. The PGM_1 locus determines the a, b, c and d bands while the PGM_2 locus determines the e, f and g bands. There are two common alleles at the PGM_1 locus, PGM^1 and PGM^2. Three common phenotypes are referred to as PGM1-1 PGM2-1 and PGM2-2. PGM_1^1 produces the a and c bands and its homozygote is called PGM1-1, while PGM_1^2 , gives rise to the bands b and d and its homozygote is type PGM2-2. The heterozygote PGM2-1 has all four bands; a, b, c and d which has the appearence of a mixture of PGM1 and PGM2. The three fast moving bands; e, f and g are found in all three phenotypes although the g band is usually weak or absent (Figure 2.3.3.a.) Family studies showed that the results could be explained by the segregation of two autosomal allelic genes, PGM_1^1 and PGM_1^2, at the PGM_1 locus. Thus, the phenotypes PGM1-1 and PGM 2-2 correspond to the homozygotes PGM^1 PGM^1 and PGM^2 PGM^2 respectively and phenotype PGM2-1 represents the heterozygote PGM^1 PGM^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. Studies by Hopkinson and Harris (1965, 1966) indicate that in addition to the common alleles, PGM_1^1 and PGM_1^2 , there are at least six rare alleles at this locus. (Figures 2.3.3. b & c.). 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 (Harris et al,1973; Fiedler

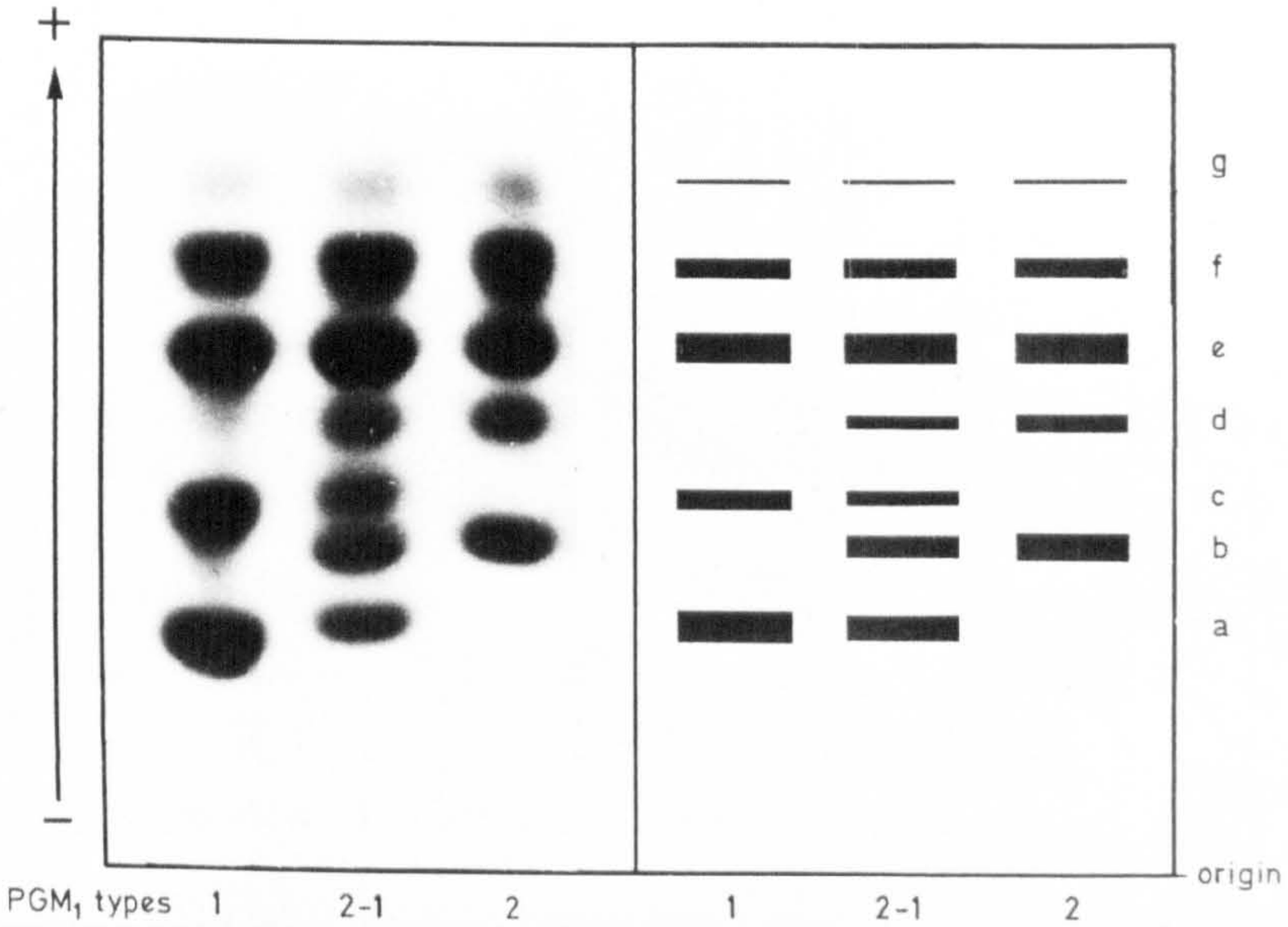


Fig.2.3.3.a. Red cell phosphoglucomutase isozymes in the three common phenotypes, PGM_1 1, 2-1 and 2. Isozymes c and d are thought to be secondary isozymes derived from a and b respectively. Isozymes f and g are thought to be secondary isozymes derived from e (Harris, H., and Hopkinson,D.A. 1976).

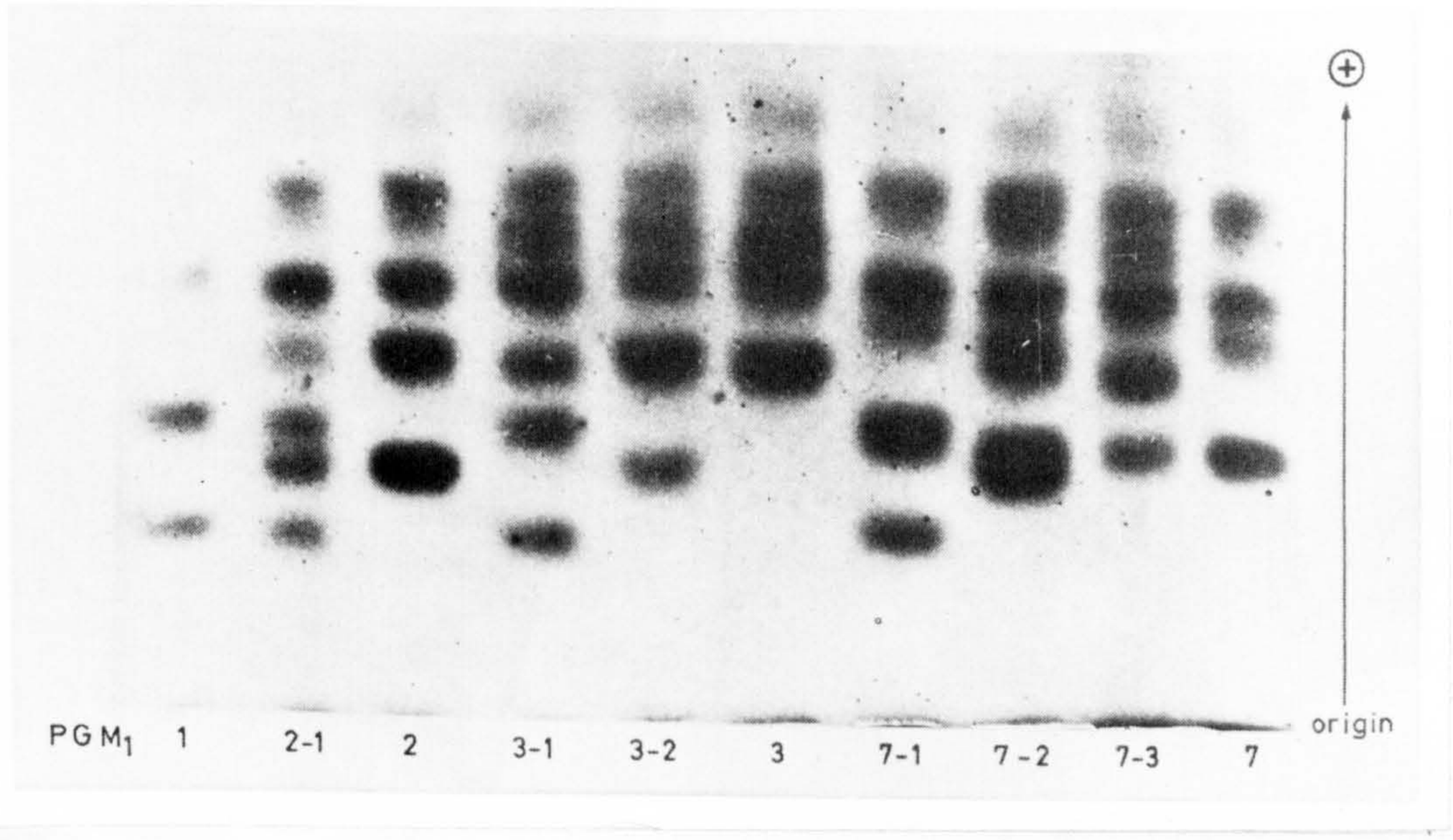
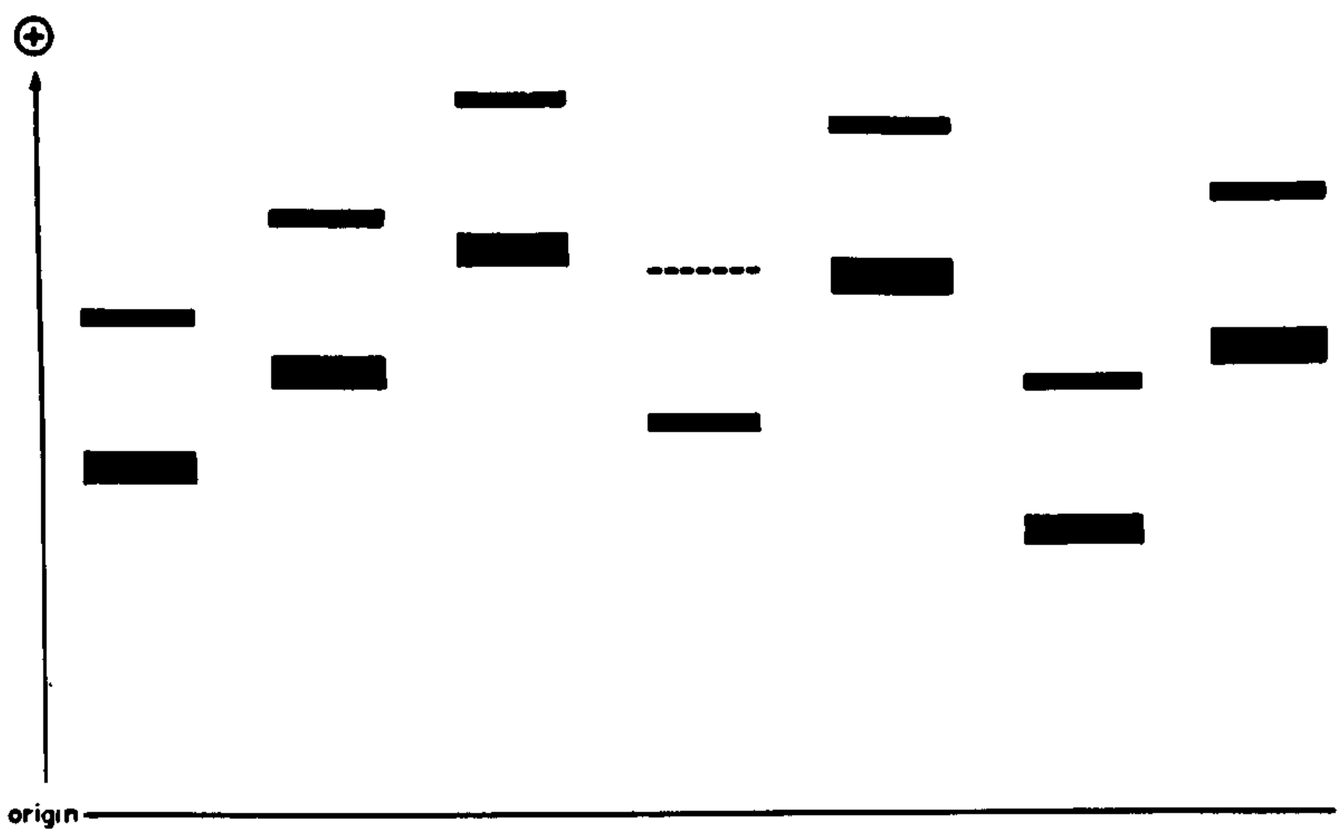


Fig.2.3.3.b. Red cell phosphoglucomutase isozymes in three common phenotypes PGM_1 1, 2-1 and 2 and several rare PGM_1 phenotypes (Blake, N.M., and Omoto, K. 1975).



Allele	PGM_1^1	PGM_1^2	PGM_1^3	PGM_1^4	PGM_1^5	PGM_1^6	PGM_1^7
Number detected	15,649	5,005	3	1	1	3	4
Frequency	0·757	0·242	0·00014	0·00005	0·00005	0·00014	0·00019

Fig.2.3.3.C. Diagram of the isozymes determined by seven different alleles at the PGM_1 locus. The isozyme patterns seen in heterozygotes appear to be simple mixtures of the products of the two alleles. The incidence of the different alleles found in a survey of 10333 unrelated individuals(20666 alleles)is given (Harris,H., and Hopkinson,D.A. 1976).

and Pettenkofer, 1969).

Most individuals are homozygous for the commonly occuring allele at the PGM_2 locus, PGM_2^1, which determines a set of bands, e, f and g, well separated from PGM_1 components. Variation in the PGM_2 locus have been found only in Negroes. So far nothing is known of the factors which maintain the genetic polymorphism at the PGM_1 locus. There does not seem to be any apparent differential superiority of one phenotype over the other with regard to enzyme activity.

In population studies the most useful system is PGM_1 with two common genes PGM_1^1 and PGM_1^2 varying widely in frequency in different populations, and at least six rare genes.
With a very few known exceptions, the frequency of PGM_1^1 always exceeds 50 percent. It is therefore convenient to describe populations in terms of the frequencies of the genes PGM_1^2, and of the rarer genes where they occur.

2.3.4. The adenosine deaminase (ADA) system

The enzyme adenosine deaminase (ADA; adenosine aminohydrolase E.C. 3.5.4.4.) acts as an aminohydrolase catalysing the deamination of adenosine into inosine. It was found in different tissues and in red blood cells. The enzyme is know to exist in multiple molecular forms.

Genetically determined variants of this enzyme were first demonstrated by means of starch-gel electrophoresis of red-cell haemolysates, by Spencer et al (1968). These were shown to be the products of a pair of allelic genes ADA^1 and ADA^2 at an outosomal locus.
They observed three different patterns. (Figure 2.3.4 a), ADA 1-1, ADA2-1 and ADA2-2, and family studies indicated the patterns to be genetically controlled by two co-dominant alleles at an autosomal locus, termed ADA1 and ADA^2. The phenotypes

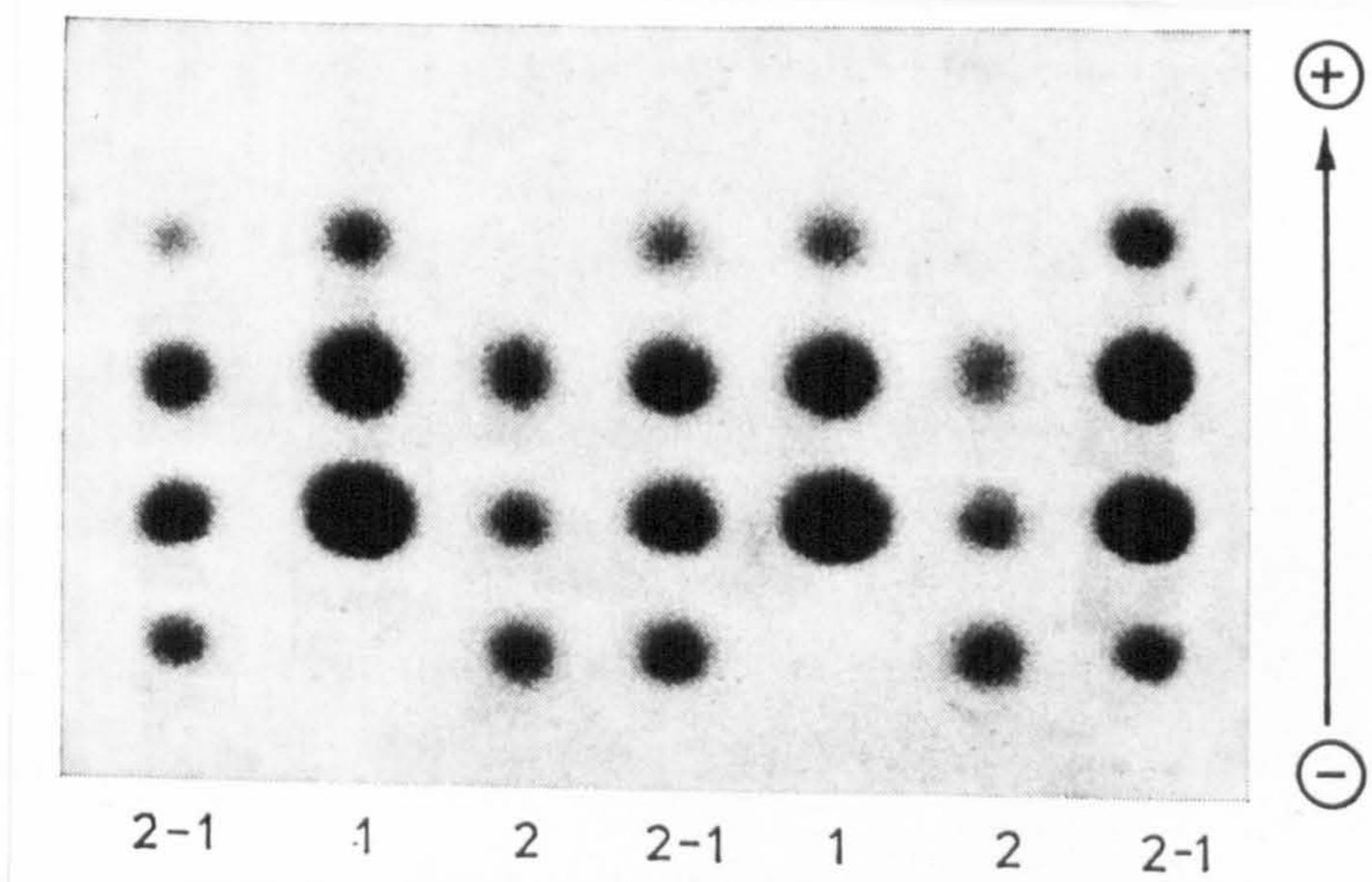


Fig. 2.3.4.a. ADA isozymes in red cells of the three common phenotypes ADA 1, 2-1 and 2.(Walter, H., and Ananthakrishnan,R.1975).

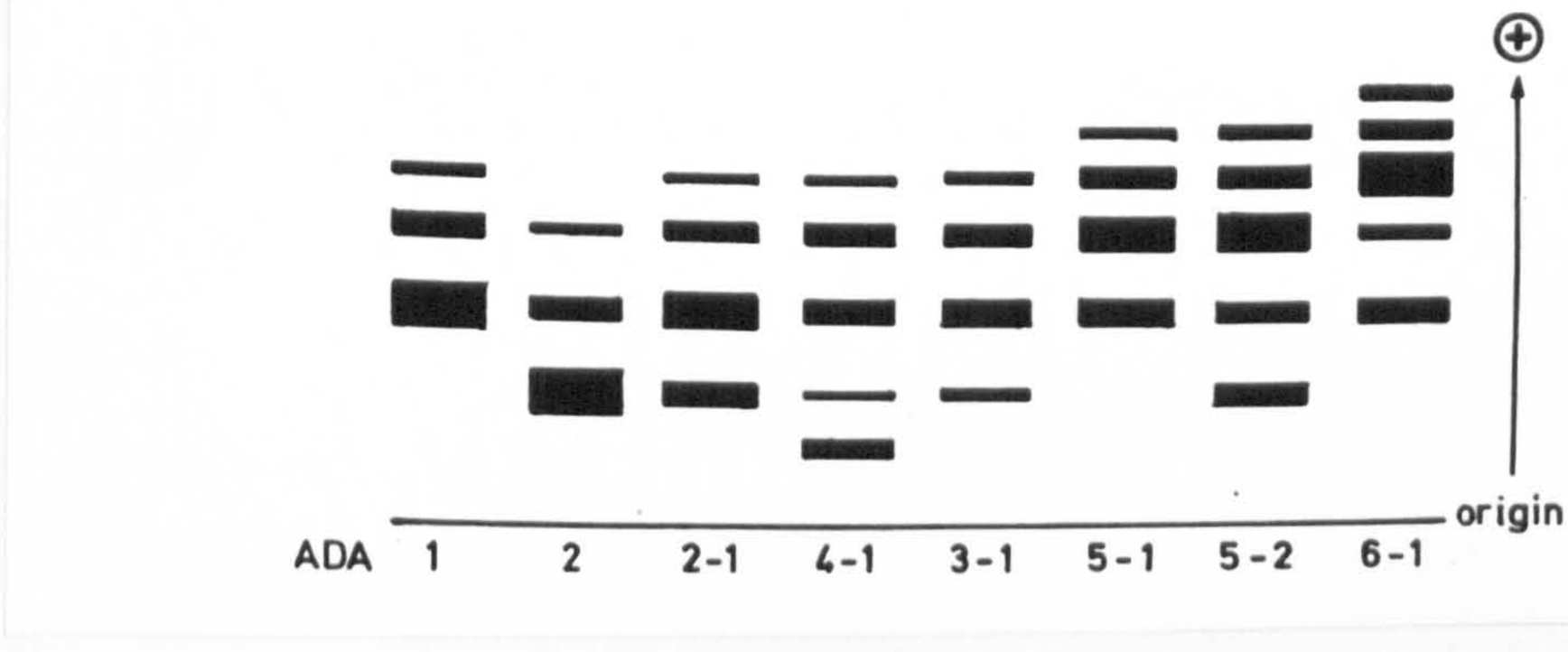


Fig.2.3.4.b. Diagram of the red cell ADA isozymes in various phenotypes (Radam, R., et al. 1974).

ADA1-1, ADA2-1 and ADA2-2 according to this hypothesis represent the genotypes ADA^1/ADA^1, ADA^1/ADA^2 and ADA^2/ADA^2 respectively. In a further paper Hopkinson, Cook and Harris (1969) described another allele ADA^3 at the same locus which, however, appears to be very rare, if present at all, in all populations. Since then several additional rare phenotypes have been observed (Figure 2.3.4.b.), which were attributed to the alleles ADA^3, ADA^4, ADA^5, ADA^6, and ADA^8(Dissing and Knudsen, 1970, and Detter et al, 1969). In addition, a 'silent' allele ADA^0 has been described (Chen et al, 1974; Giblett et al,1972) which appears to be associated with combined immunodeficiency. Adenosine deaminase is an importan enzyme in purine metabolism as evidenced by the occurrence of severe combined immunodeficiency disease in invidviduals deficient in leucocyte ADA activity(Giblett et al, 1972; Meuwissen, Pollara and Pickering,1975) and in one case elevated red cell ADA activity appears to be a cause of haemolytic anaemia (Valentine et al,1977).Since thediscovery ofgeneticallydétermined variants of red cell adenosine deaminase,it has been screened in numerous populations. The gene frequencies of the common alleles ADA^1 and ADA^2 have been established in many areas and several rare variant phenotypes, attributable to uncommon alleles at the ADA locus have been identified (Harris and Hopkinson, 1976).

The wide range of frequencies and the well-marked geographical differentiation suggest that his system will prove of considerable value in population studies. In Asiatic populations and to a less extent in Europeans, the frequency of the rarer of the two common genes is sufficient to make the system of value in family investigations of such features as linkage. Other ADA genes ADA^3, ADA^4, ADA^5, ADA^6 and ADA^8, are so rare as to be of very little interest in populations so far studied,

and even ADA^2 is not known to exceed 20 percent in frequency in any population.

2.3.5. The esterase D(EsD) system

The enzyme esterase D(EC 3.1.1.1.) is a ubiquitous tissue esterase of unknown biological function. This enzyme,which is present in red cells and most other tissues, was shown to be biochemically and genetically distinct from the previously recognized red cell esterases A, B and C(Tashian,1961,1969). So it represents one of the four ester isozymes described in human erythrocytes. It is distinguished from other tissue esterases by its electrophoretic mobility and by its narrow specificity for methylumbelliferyl esters as substrates.

Genetically determined variants of human red cell EsD was first described by Hopkinson et al(1973),by means of starch-gel electrophoresis,using an ester of fluorescent compounds (4-methylumbelliferyl acetate) as substrate. The appearance of fluorescent bands is due to the release of umbelliferon at the site of enzyme activity. Three distinct common phenotypes can be distinguished designated according to their electrophoretic mobilities ESD1-1, EsD2-1 and EsD2-2.(Figures 2.3.5.a and b). Family studies confirmed that these three phenotypes are determined by the co-dominant expression of two alleles,EsD^1 and EsD^2, segregating at an autosomal locus.Formal genetic researches have concluded to assignment of EsD locus on chromosome 13. The homozygous phenotype EsD 1-1 and EsD2-2 patterns each consist of three isozymes and EsD2-1 heterozygous phenotype, of five isozymes. However, the fastest moving band of the EsD2-2 and EsD2-1 cannot be clearly seen as they superimpose the slowest of the Esterase A_2 bands. The EsD1-1 and EsD 2-2 bands show decreasing anodal intensities with the slowest band of EsD2-2 having the same mobility as that of the fastest

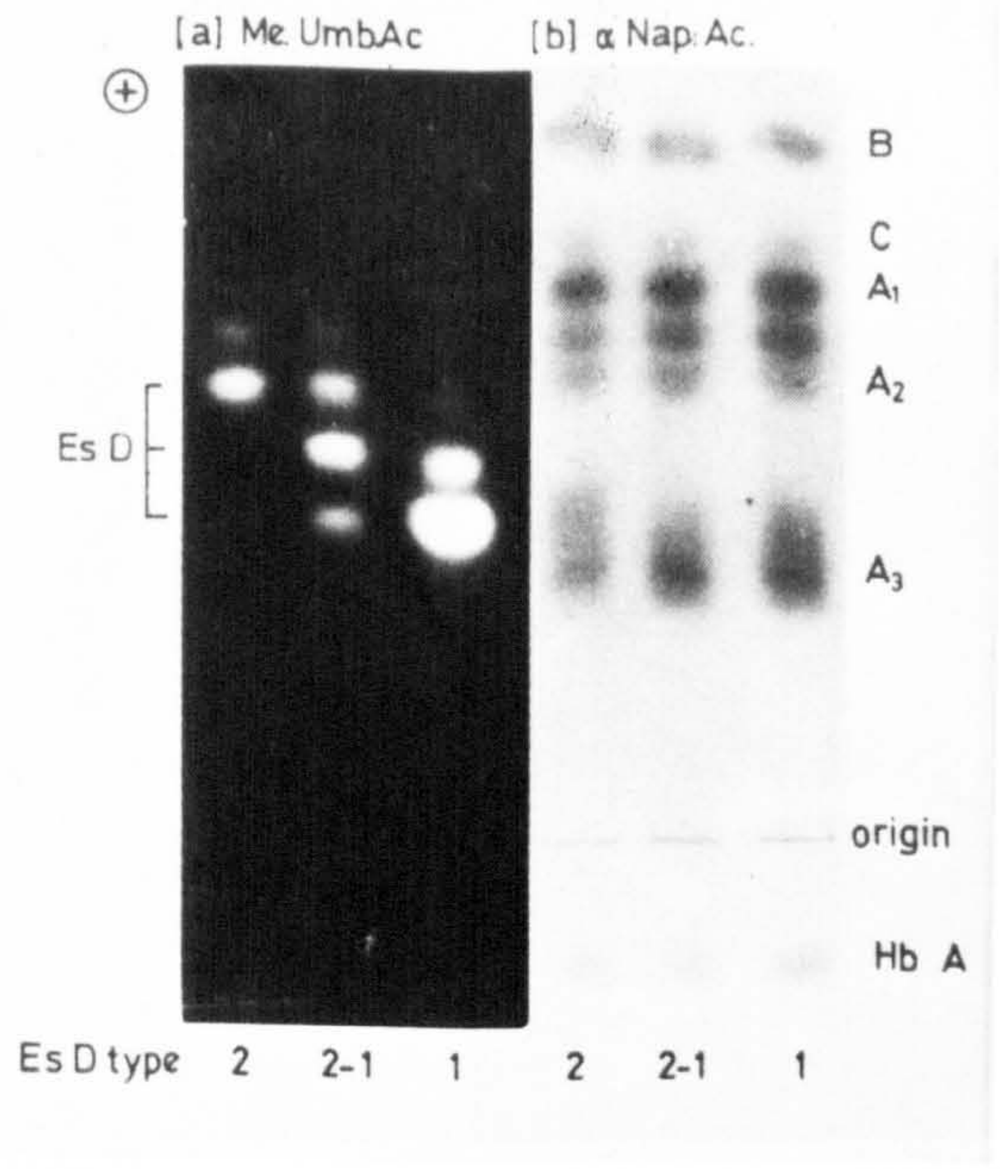


Fig.2.3.5.a. Red cell esterase isozymes stained with 4-methylumbelliferyl and ∝ -naphtyl acetate (Harris, H., and Hopkinson,D.A.1976).

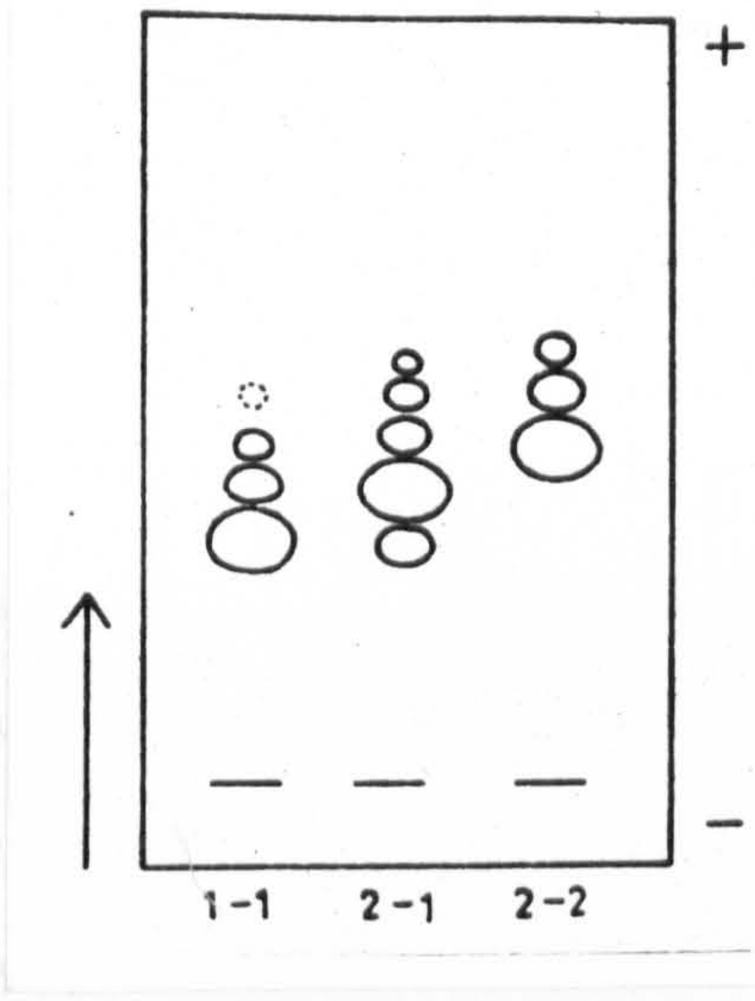


Fig.2.3.5.b. Starch gel electrophoretic patterns of the three common EsD phenotypes (Benkmann,H.G., and Goedde, H.W. 1974).

band of the EsD1-1 pattern. EsD2-2 can be easily distinguished due to its relatively greater mobility. Care must be taken in distinguishing EsD1-1 and EsD2-1. In the electrophoretic pattern of EsD1-1 phenotype, band 1 gradually loses its intensity until bands 1 and 2 are about the same intensity, with band 3 much weaker. In EsD2-1, the strongest band, band 2, loses intensity until bands 1, 2 and 3 are of the same intensity. Thus, when interpreting the EsD types,it has been found that the comparison of bands 1 and 3 in each type gives the most consistent interpretation. In EsD1-1,band 1 is stronger than band 3 and in EsD2-1,bands 1 and 3 are approximately of the same intensity.

Since the discovery of EsD polymorphism by Hopkinson et al (1973), several reports have been published of its distribution in European and Asian samples(Welch and Lee,1974; Koster et al, 1975; Cartwright et al 1976; Olaisen et al,1976). Of the two alleles,the $Es D^{1}$ gene is the more frequent in all populations so far studied, but the $Es D^{2}$ gene is commoner in Asia than in most European populations(Hopkinson et al, 1973; Benkmann and Goedde, 1974).

To date, two rare alleles for EsD has been described. Bender and Frank(1974) have reported the slow electrophoretic variant EsD3-1 in an individual from South West Germany, and Rittner and Muller(1975)have reported an example of EsD3-1 inna blood donor from Bonn and Bargagna et al(1975)have found the variant in Tuscany, Italy. This very rare phenotype indicates the existence of a third allele $Es D^{3}$; at the EsD locus. More recently, Berg et al(1976) have described even slower-electrophoretic variant EsD4-1, attributed to a fourth allele $Es D^{4}$. One instance of an individual apparently homozygous for a null allele, has also been reported. This was detected in an elec-

trophoretic study and there was no further family analysis, nor was there any quantitative characterization of esterase D activity (marks et al, 1977).

This system has already proved to be of considerable interest for genetic delineation of human populations.

2.3.6 The 6-phosphogluconate dehydrogenase (6PGD) system

The enzyme 6-phosphogluconate dehydrogenase (D-glucose-1-phosphate phosphotransferase, EC 1.1.1.4.1.)takes part in the hexose monophosphate shunt, that is to say, a relatively minor series of reactions branching off from the main anaerobic (Embden-Meyerhof)glucose metabolic cycle, and converting hexoses into pentoses needed for making nucleic acids. It catalyses the next step in the chain of reactions after that catalysed by glucose-6-phosphate dehydrogenase, causing the conversion of 6-phosphogluconate to ribulose-5-phosphate, the co-enzyme, nicotinamide adenine dinucleotide phosphate (NADP) being simultaneously reduced to NADPH.

Though the enzyme occurs in many tissues, studies have been performed mainly on red cells in the form of haemolysates.

The existence of genetically determined variation of this enzyme in man was first demonstrated by Fildes and Parr(1963) by means of starch-gel electrophoresis of human red cell haemolysates. They found three electrophoretic patterns and showed that these isozymes represented the two homozygotes and the heterozygote of a pair of allelic genes, PGD^A and PGD^C at an autosomal locus. The commonest phenotype, AA, consists of a single anodal band in the 'a' position due to the homozygous presence of the $6PGD^A$ allele. The second most frequent.phenotype is the 'common' variant, CA,which is heterozygous for the PGD^A& PGD^C genes.It exhibits an anodal band(a) and a cathodal band(b).Bands a

and b have equal intensity. Individuals homozygous for the PGD^C gene exhibit the third variant designated the 'canning variant' CC. It consists of bands a, b and c, with the anodal band as a minor component.(Figure 2.3, 6.a.), Family studies showed that the inheritance of these variants is controlled by two alleles, PGD^A and PGD^C at an autosomal locus.

It is assumed that the enzyme 6PGD exists as a dimer, so that in the two homozygous types, the gene product is a subunit, (i.e. S^A or S^C), which dimerizes to form either the a band (S^AS^A) or the c ban S^CS^C). In the heterozygote, both bands a and c are present, as well as the b band, which is thought to be a mixed dimer, S^AS^C(Parr, 1966; Parr and Fitch, 1967).

Apart from common variants, futhter variants due to alleles at the same locus have been found and examined by Fildes and Parr (1964), Parr and Fitch (1964), Parr and Parr (1965), Parr (1966), and Parr and Fitch (1967). These variants include two, recognizable electrophoretically, due to the alleles PGD^H and PGD^R, as well as alleles producing unstable enzymes, and one which, in the homozygous condition, gives no detectable enzyme activity. The rare phenotype, due to the allele PGD^R, called the 'Richmond variant' was detected by Parr (1966) and in an American family by Davidson (1967). The corresponding phenottype appears to be PGD^A PGD^R. The phenotypic pattern 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 due to the allele PGD^H, called the 'Hackney variant' was reported by Parr (1966). This phenotype apparently represents heterozygosity for PGD^A and PGD^H. It differs from others in showing slow mobility of all the bands. Davidson (1967) has described

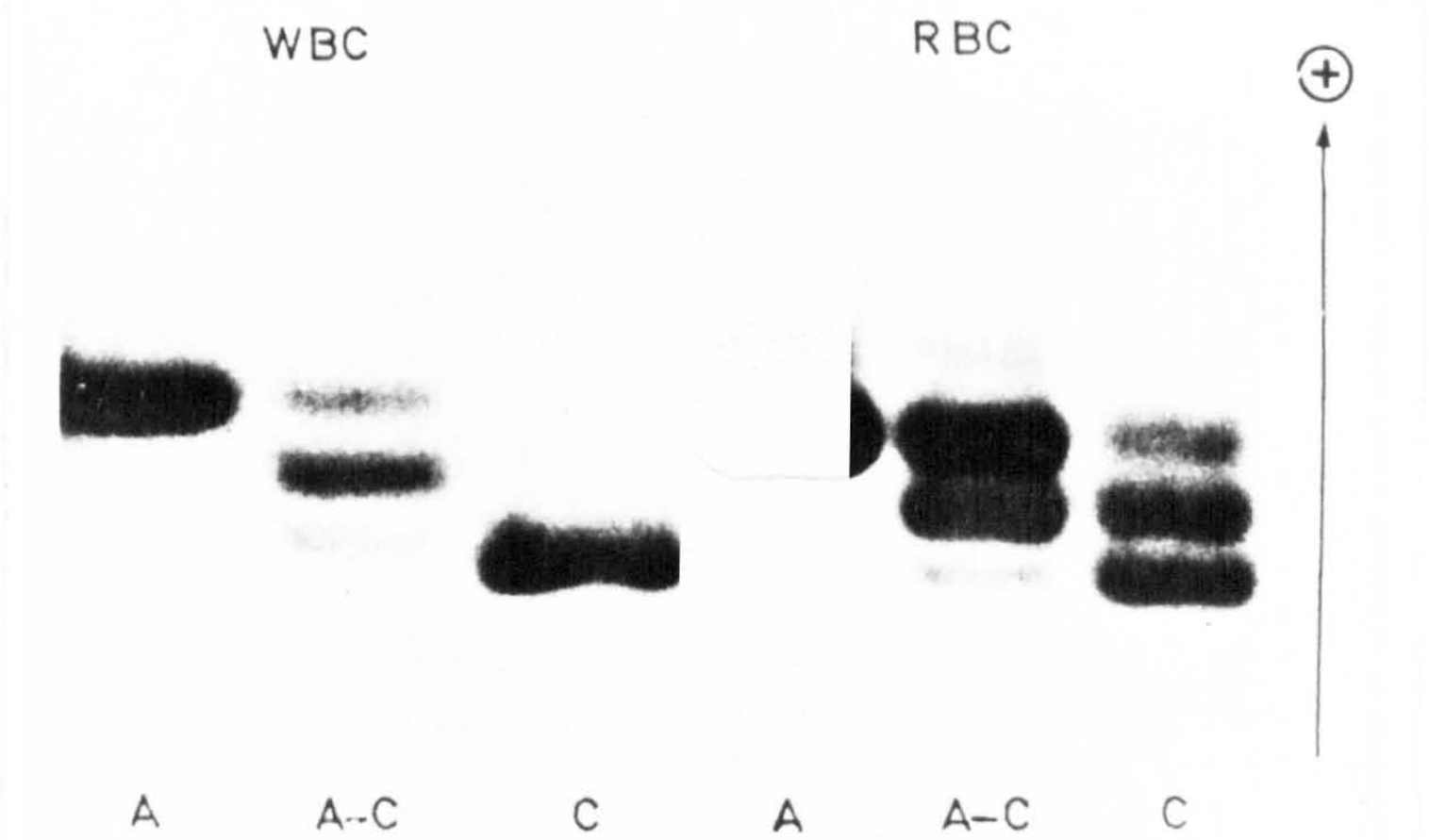


Fig. 2.3.6.a. Isozyme patterns in the three phenotypes PGDA, PGDA-C,PGDC in red cells and white cells. Note the more anodal secondary isozymes seen in the red cell samples(Giblett,E.R. 1969).

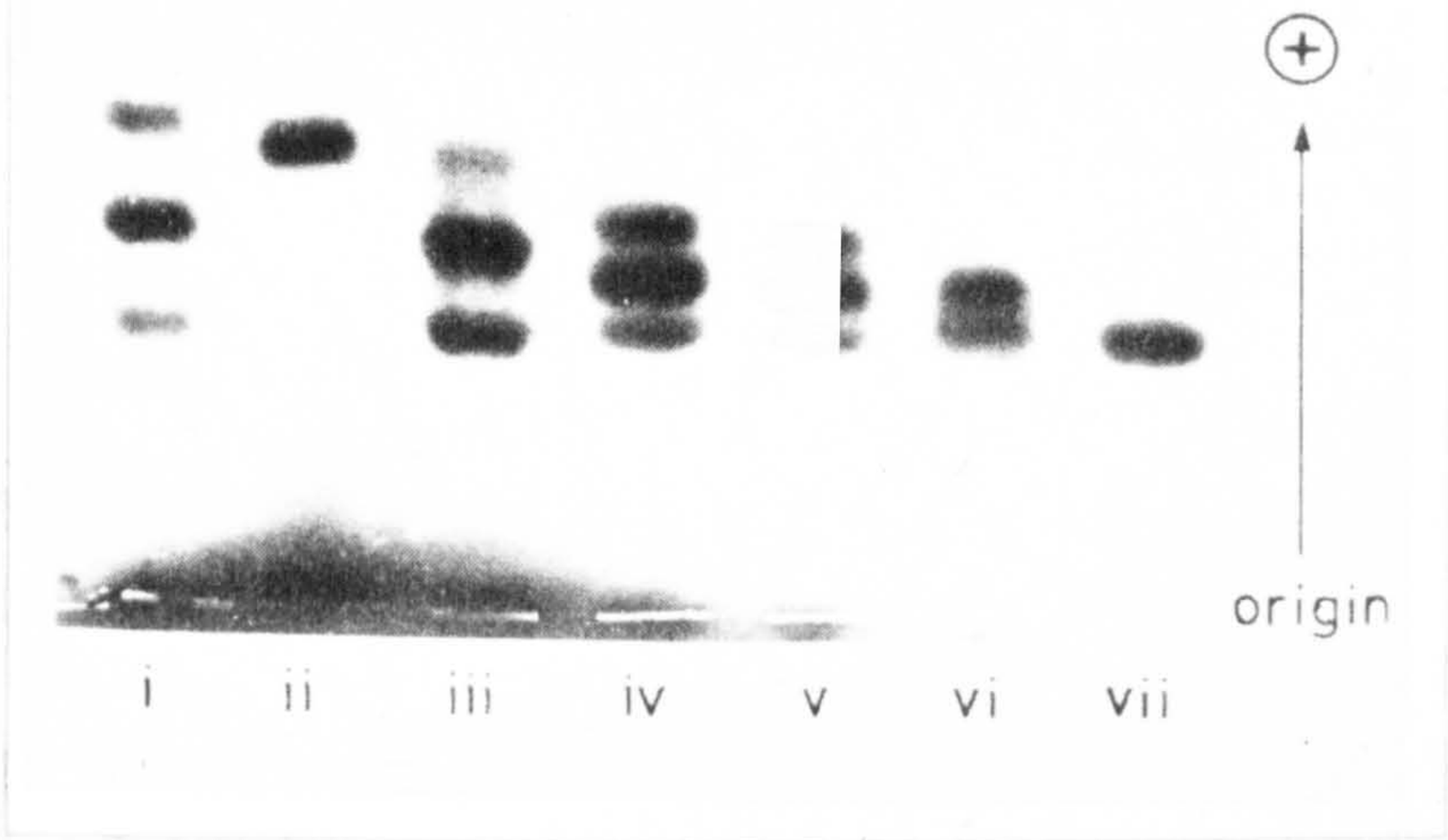


Fig.2.3.6.b. Some rare PGD phenotypes:(i)PGDA-Elcho;(ii)PGD Kadar (Presumed homozygote);(iii)PGDA-Kadar;(iv)PGDA-Caspian;(v)PGDA-Richard;(vi)PGDA-Bombay;(vii)PGDA(Blake,N.M.,et al. 1974).

a rare variant due to an allele PGD^F, called the 'Friendship variant'. Its electrophoretic pattern exhibits bands a, d and e. 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. The whole subject of the variants and their relationships has been reviewed by Carter et al (1968) and by Giblett (1969), but Blake and Kirk (1969) have since described a variant of considerable anthropological interest. This is the 'Elcho' variant, so far found only in Australian aborigines(Figure 2.3. 6.b.).

In nearly all populations PGD^A has a frequency exceeding 90 percent and PGD^C comes next in frequency. All other variants are rare and sporadic.

Parr (1966) has shown that both the heterozygote PGD^A/ PGD^C and the homozoygote PGD^C/PGD^C have slightly less enzyme activity than the common PGD^A/PGD^A homozygote. The heterozygotes of PGD^A with PGD^R and PGD^H which have been found in some random population surveys, as well as that with PGD^F, appear to have normal activity but some of the very rare variants give rise to considerably reduced enzyme activity. It would not be surprising to find that such variations were associated with selective advantages or disadvantages.

The common genetic phenotypes consituting this genetic polymorphism, i.e., AA, AC and CC, as determined by the codominant alleles PGD^A and PGD^C, are also characterized by variations in specific enzyme activity (Parr, 1966; Gordon et al, 1969; cf. Giblett, 1969). However, unlike G-6-PD, gross enzyme deficiency appears not to be of significance with regard to the mani-

festation and geographic distribution of this genetic polymorphism (Parr, 1966; Parr and Fitch, 1967; cf. Giblett, 1969) . Thus, while contributing to the intraerythrocyte level of NADPH, like G-6-PD, the genetic variation underlying the 6-PGD polymorphism has not been implicated in the occurrence of endemic falciparum malaria. In fact, the significance of the anthropogenetic variation of this polymorphism in terms of adaptation, anthropogeography, etc, is still poorly understood (Tills et al, 1970, 1971; cf. Giblett, 1969; Ananthakrishnan, 1975).

2.3.7 The glucose-6-phosphate dehydrogenase (G-6-PD) system

The enzyme Glucose-6-phosphate dehydrogenase (G6PD: D-glucose-6-phosphate : $NADP^+$1-oxido-rductase, EC 1.1.1.49) is an essential catalyst in one of the body's method of oxidizing glucose, known as the pentose phosphate pathway, since it involves a breakdown from the 6-carbon molecule of glucose to a 5-carbon chain (G-6-phosphate to 6-phosphogluconate). Through the action of the enzyme the hydrogen so lost is transferred to nicotinamide adenine dinucleotide phosphate (NADP) to give rise to its reduced form NADPH. The latter is responsible through the action of the enzyme glutathione reductase, for maintaining an adequate level of reduced glutathion (a tripeptide with an SH group) necessary, in a manner not fully understood for the continuing integrity of the red cells. Thus in the absence of adequate G6PD activity red cells tend to haemolyse. The enzyme is present in red cells and also in other tissues.

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, recognized by their electrophoretic mobility on starch gel, ar usual (Figure 2.3.7.a).

A very large number of variants of G6PD are now known (150

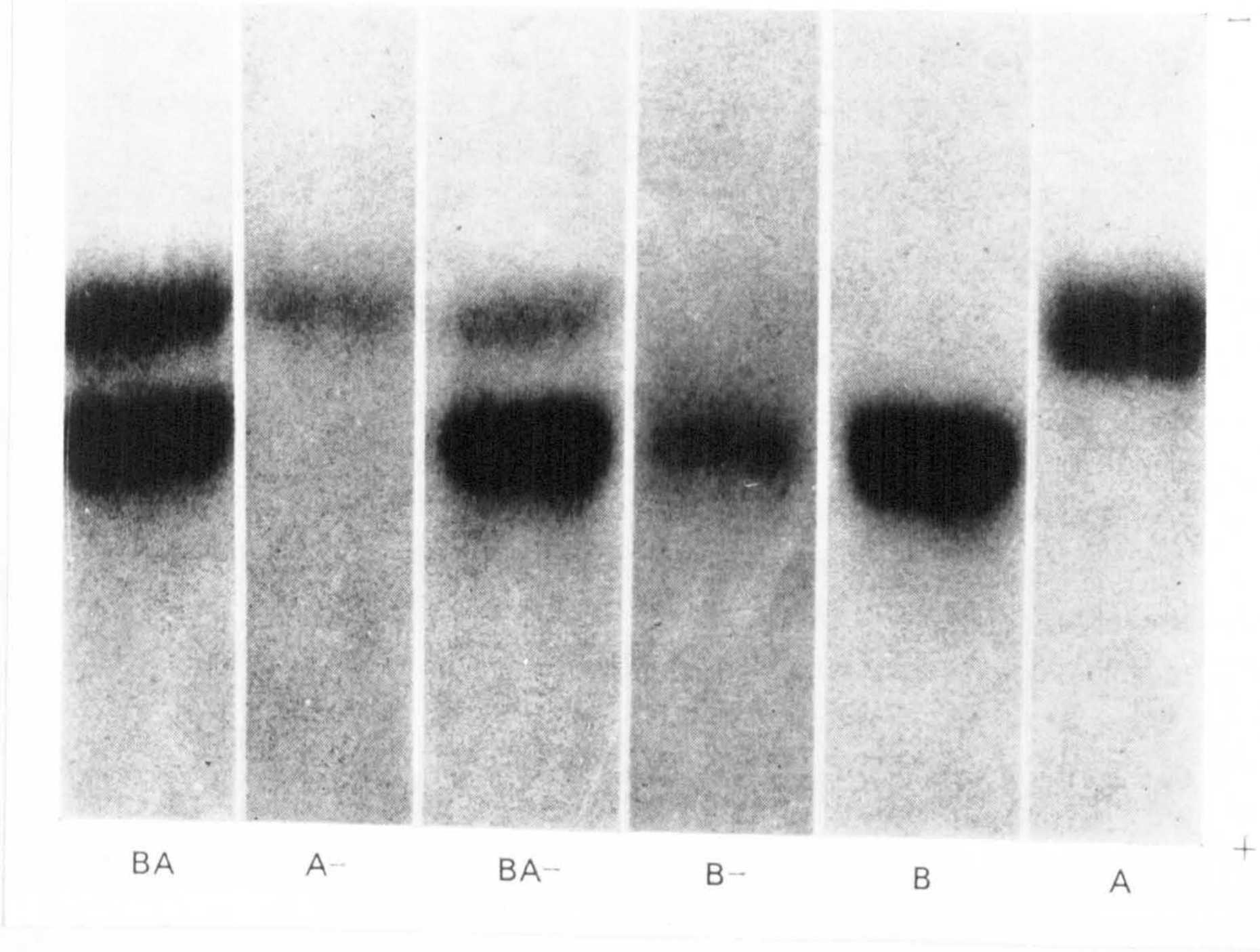


Fig.2.3.7. a. Starch gel electrophoretic patterns of six different G6PD phenotypes in red cell haemolysates (Giblett,E.R. 1969).

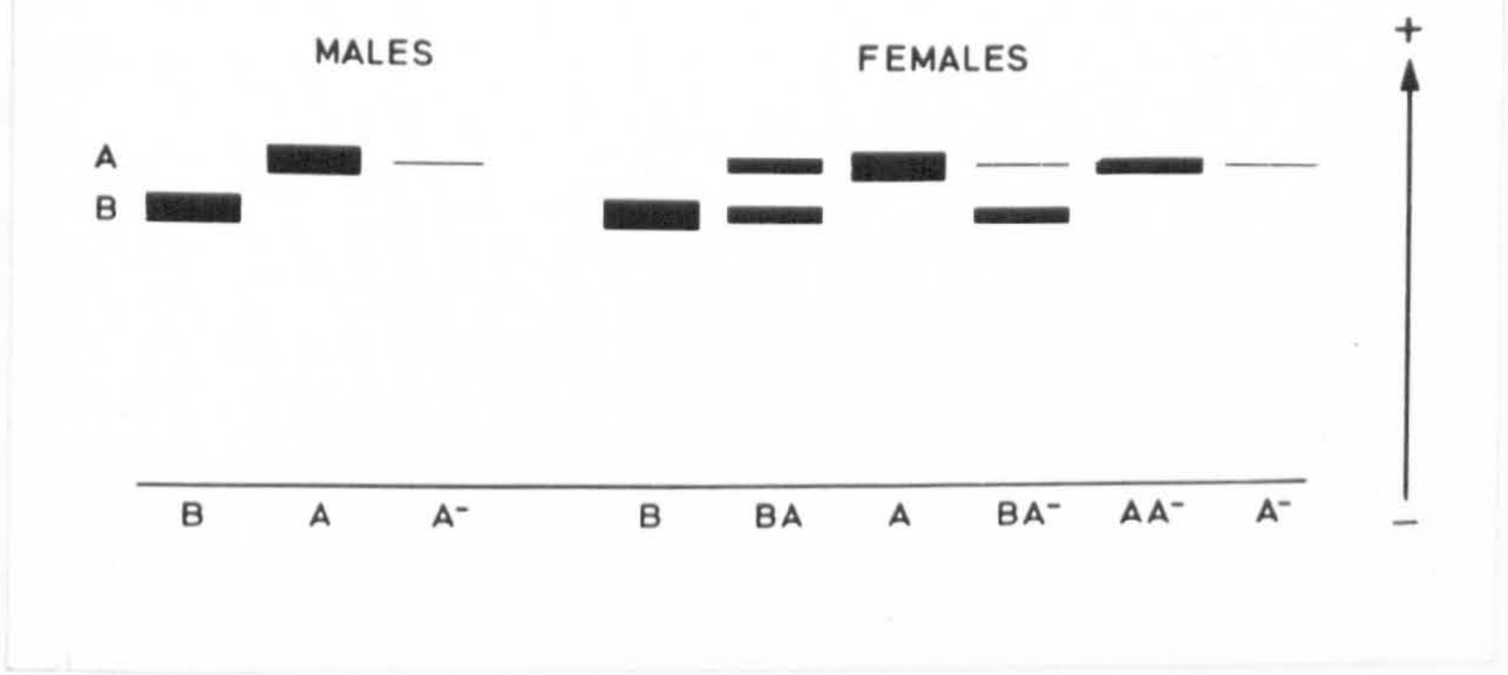


Fig.2.3.7.b. Diagram of red cell glucose-6-phosphate dehydrogenase isozymes in Blacks of different phenotypes (Harris,H., and Hopkinson, D.A. 1976).

G6PD variants tabulated by Yoshida and Beutler, 1971, 1978 ; and Beutler and Yoshida, 1973) but, in addition to the normal type, only three variants are commonly recognized as being of any importance in population studies. The common type in all populations is known as B^+, the B referring to its rate of migration on electrophoresis and the + to its normal enzymatic activity. The common abnormal type in the Mediterranean area is B-, migrating at the same speed as the normal but with an activity between 0 and 7 percent of normal. It is, of course, possible to determine the speed of migration by the standard electrophoretic method only if at least a trace of enzyme activity is present.

In African populations two abnormal types are known, A+ which has normal activity but which migrates faster than the normal, and A- which migrates at the same speed as A+ but has only 8 to 20 percent of normal activity.

The variants of G6PD are determined by a series of allelomorphic genes on the X chromosome. Thus a male with only one X chromosome can have only one type of enzyme, but a female, having a pair of X chromosomes, can be heterozygous and have genes for two different alleles (Figure 2.3.7.b.).

Tests are of three main kinds, screening tests for the presence of enzyme deficiency, precise quantitative tests to measure the degree of deficiency, mainly of use in identifying rare variants, and tests by starch gel electrophoresis, which, provided that the blood specimens are fresh so that all the variants retain some enzyme activity, will in males,and to a large extent also in females, show both the relative electric charge and the activity of any variants present.

Deficiency of erythrocyte glucose-6-phosphate dehydroge-

nase is the commonest genetic enzymatic abnormality in human beings, probably affecting more than a hundred and fifty million males. It has long been known that some persons in many Mediterranean countries suffer from favism, a haemolytic anaemia wiht an appreciable mortality, precipitated by consumption of the common horse bean or broad bean, vicia fava. During World War II it was found that some American Negro soldiers suffered from haemolysis when treated with certain antimalarial drugs derived from quinoleine, such as primaquine. Investigations by numerous workers finally traced both these conditions, and a number of others such as jaundice among newborn Chinese infants in Singapore, to a deficiency in the red cells, of the enzyme, glucose-6-phosphate dehydrogenase, the complicated history of research on the genetics of this enzyme has been reviewed by many authors including Giblett (1969)who gives full references to previous works.

Over the past decade the genetic variability in man associated with the enzyme G-6-PD has rapidly developed into a subject of major concern in medical, genetic (Pharmacogenetic), and anthropobiological research.

The central theme underlying this interest concerns the occurrence of two major types of genetically determined enzyme deficiencies, i.e., the Negro-type (10%-15% of normal enzyme activity in red cells) and the Mediterranean type (8% of normal enzyme activity in red cells), both affecting millions of people, with all the clinical, public health and anthropogeographical implications associated with it. Genetically, the two types of enzyme deficiency are attributable to the manifestation of two alleles, Gd^{A-} and Gd^{B-}, respectively, at the sex-lniked Gd locus. The alleles Gd^{A} and Gd^{B}, on the other

hand, are characterized by the synthesis of enzyme molecules with 'normal' enzyme activity and electrophoretic mobilities indistinguishable from those of their defective counterpart molecules. Biochemically, the dimeric enzyme G-6-PD is responsible for the first and rate-controlling reaction in the hexose monophosphate shunt pathway in red cells. With this reaction NADPH is generated, an ample supply of which is essential for the stability and longevity of red cells.However, under certain dietary conditions, after treatment with a variety of specific drugs, or after infections by certain bacteria, the genetically determined enzyme defficiencies impair the normal metabolic function of the red cells, cause their premature destruction, and thus enhance the possibilities of overt manifestation of severe haemalytic anaemia, especially in children (allison, 1960, 1963; Motulsky, 1960, 1961, 1964, 1965; Motulsky and Campbell-Kraut, 1961; Motulsky et al, 1966; cf.Giblett, 1969; Livingstone, 1971; Allison, 1975; Flatz and Xirotiris, 1975). The postulated association between the worldwide distribution of these enzyme-deficient genetic markers and the endemicity of falciparum malaria represents a significant step in the understanding of the selective mechanism possibly under - lying the maintenance of this polymorphic genetic system and provides a basis for specifically directed population qenetic and anthropogeographical research. The malaria hypothesis is based mainly on the striking correlation between the geographic distribution of endemic falciparum malaria and that of the enzyme-deficient alleles Gd^{A-} and Gd^{B-}, and also on supporting comparative parasitological studies. More specifically, it is postulated that whereas hemizygous males and homozygous females for normal enzyme activity are highly susceptible to mala-

ria infections, they are in no undue danger when exposed to certain dietary or pharmacological haematolytic agents. Conversely, whereas enzyme-deficient hemizygous males and homozygous females are likely to develop severe haemolysis upon exposure to the conditions and agents mentioned(leading to elevated mortality, especially during childhood), they are at a relative selective advantage in malarious environments, in that they enjoy a measure of natural resistance against malaria parasites.Follwoing this reasoning, heterozygous females are regarded as optimally equipped genetically against malaria infections, without undue risk of suffering severe bouts of haemolytic anaemia when exposed to especific drugs, diets, or certain bacterial infections. It follows that this genetic polymorphism appears to be maintained in the population by a balance of selective forces in heterozygotes analogous to the sicklecell polymorphism. A significant difference, however, is the fact that whereas the latter is autosome-linked, the Gd locus is sex-linked, entailing its subjection to lyonization and a more complex selective meachanism and approach to genetic equilibrium. The crucial factor in this type of balanced polymorphism obviously is the selective advantage and greater genetic fitness enjoyed by the heterozygous female (Allision, 1960, 1963; Motulsky, 1960, 1961, 1964, 1965; Motulsky and Campbell-Kraut, 1961; Motulsky et al, 1966; Luzatto et al, 1969; Bienzle et al, 1972; cf. Giblett, 1969; Livingstone, 1971; Allison, 1975; Flatz and Xirotiris, 1975).

2.3.8. The lactate dehydrogenase (LDH) system

Lactate dehydrogenase (L-lactate: NAD oxidoreductase, EC 1.1.1.27) catalyses the reversible reduction of pyruvate to lactate. This enzyme is present in many tissues and has been

the subject of much investigation. Work done on it up to 1965 has been very fully reviewed by Vessel(1965).

The molecules of this enzyme are tetramers of two types of polypeptide chain, A and B. Each tetramer molecule can contain any number from 0-4 of any type of chain. Thus, in the normal case, where only the single common homozygote of each chain type is present, five molecular types are present, A4, A3B, A2B2, AB3, and B4, giving rise to five electrophoretically distinct bands. (Figure 2.3.8). The two chain types are the products of two sets of alleles at different autosomal loci which are not closely linked. The relative strength of the various electrophoretic bands differ according to the tissue or red cell type from which the enzyme is derived. The isozymes are numbered from LDH1 (the fastest moving towards the positive pole) for B4 to LDH5 (which migrates slowly towards the negative pole) for A4. The structure of each isozyme is as follows:

LDH-1 = BBBB (B4)

LDH-2 = BBBA (B3A)

LDH-3 = BBAA (B2A2)

LDH-4 = BAAA (BA3)

LDH-5 = AAAA (A4)

Only bands 1, 2 and 3 are well developed on electrophoretic patterns of red cell haemolysates, variants at either locus are all of very low frequency but a considerable number of rare variants only in the form of heterozygotes with the normal allele, are known (Vesell, 1965), some apparently affecting the A chain and some the B chain. Because of the lack of constraint as to the manner in which tetramers can be built up, a heterozygote at one locus should be able to give rise to fifteen bands, and a bouble heterozygote (a type not yet identified) to thirty five.

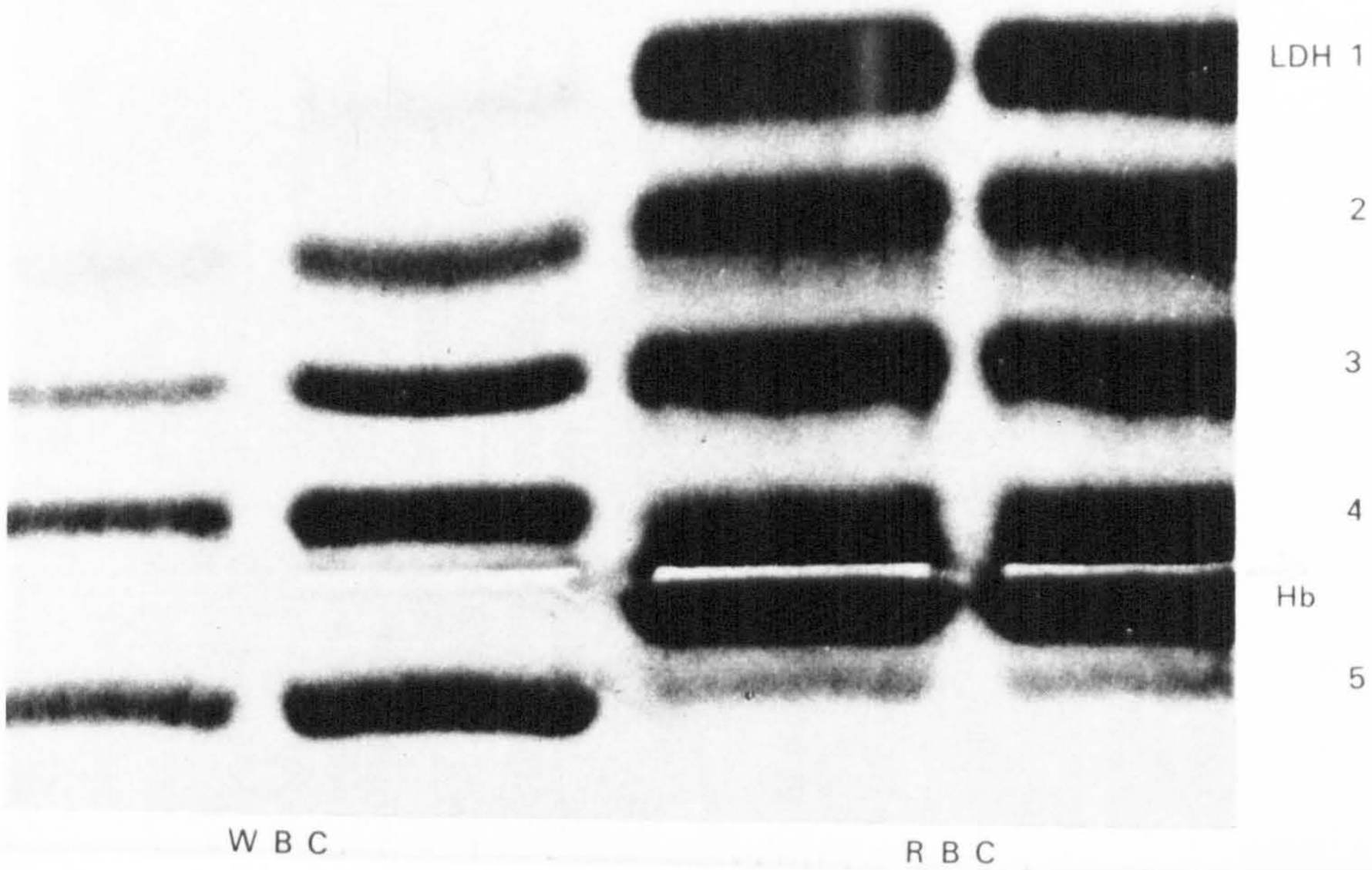


Fig.2.3.8. Electrophoretic patterns of LDH isozymes in lysates of white cells (left) and red cells (right). From Giblett,E.R.1969.

The patterns actually observed involve a splitting of the main bands LDH 1-5 into spectra of fine lines. The complete analysis of a given variant so as to interpret it in terms of an allele at the A or B locus is a very complicated process. Thereafter, newly observed variants can with more or less confidence be identified by running them alongside a known variant on the same gel.

Variants are in general very rare, occurring with a frequency of the order of 1 in 1000 in most populations.

The most widespread variant appears to be memphis -4 of Kraus and Neely (1964) which is a slow A variant. This (or a type or types indistinquishable from it) is the main variant in European populations. Type Calcutta 1, heterozygous for a fast A variant, has a frequency of somewhat over one percent in various Indian populations. A number of other variants are known, all with very low frequencies indeed.

2.3.9 The malate dehydrogenase (MDH) system

The enzyme malate dehydrogenase (l-malate: NAD reductase, EC 1.1.1.37) catalyses the reversible oxidation of malate to oxaloacetate. Two electrophoretically distinct forms of the enzyme are foundrin man, one present in solution in cytoplasma, including red cells and one localised within the mitochondria (Christie and Judah, 1953; Shrago, 1965). These are the products of genes at separate autosomal loci, not closely linked. Nearly all populations are uniformly homozygous for one common gene, but variants are known in a few populations.

Genetically controlled polymorphism of the cytoplasmic enzyme was first described by Davidson and Cortner (1967) in a survey of 1,440 North American whites and 1,470 North American Negroes. The common MDH phenotype, MDH1-1, which is homozygote

for the usual allele MDH^1, exhibits on electrophoresis, when fresh, one strong band migrating towards the anode, with some faint and variable faster bands which disappear in older specimens. All the whites and all but one of the Negroes showed the common phenotype, but one Negro exhibited a variant pattern, MDH2-1 heterozygote phenotype, migrating more slowly than the normal, and consisting of three bands, the fastest of which had the same mobility as the single major band of the common pattern and the other two with slower mobility. From a family sutdy of the propositus, they were able to show that the variant and the usual types behave as if controlled by a pair of co-dominant alleles at an autosomal locus.

Blake et al (1970) described a fast variant, termed MDH3-1 in a number of persons from New Guinea. This heterozygote phenotype exhibits two moderately strong bands, together with a stronger intermediate band, and a fainter and more transient band in front of each.

Leakey et al (1972) reported the homozygote phenotype MDH3-3 among some people of New Guinea. It exhibits one strong band, faster than that for the common type, together with one fainter faster band which is absent in older specimens(Figures 2.3.9. a and b.).

The heterozygote patterns are presumably due to each molecule being a dimer, with the heterozygote giving two pure dimers and one mixed one.

The slow variant has been found only in the heterozygous 2-1 form. The fast variant, mostly as the 3-1 heterozygote, but including a few 3-3 homozygotes, has up to now been found only in Papua, New Guinea.

Many thousands of persons from different parts of the world

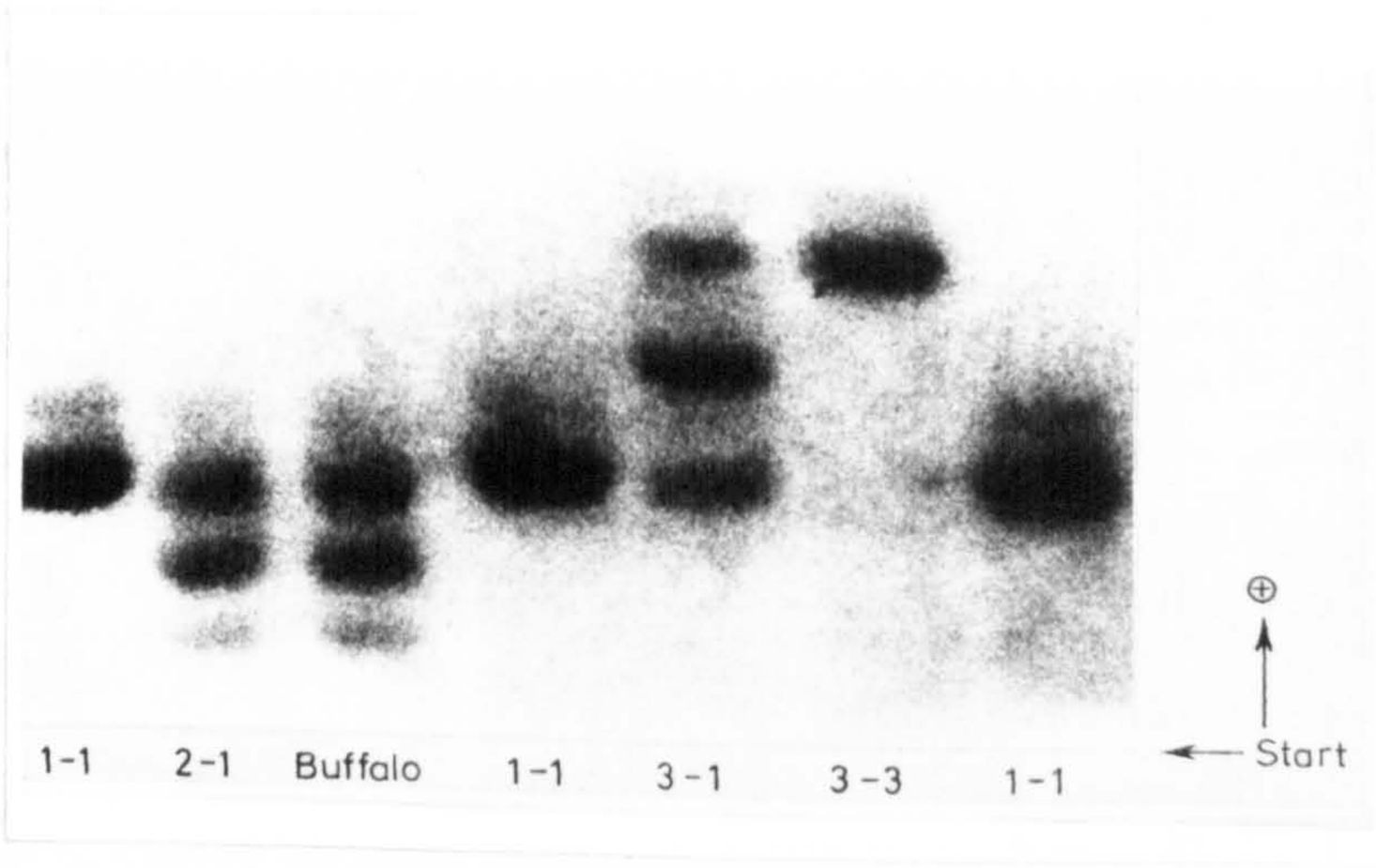


Fig.2.3.9.a. Electrophoretic patterns of the usual (MDH 1-1) and some variant phenotypes of soluble malate dehydrogenase in red cells (Leakey, T.E.B., et al. 1972).

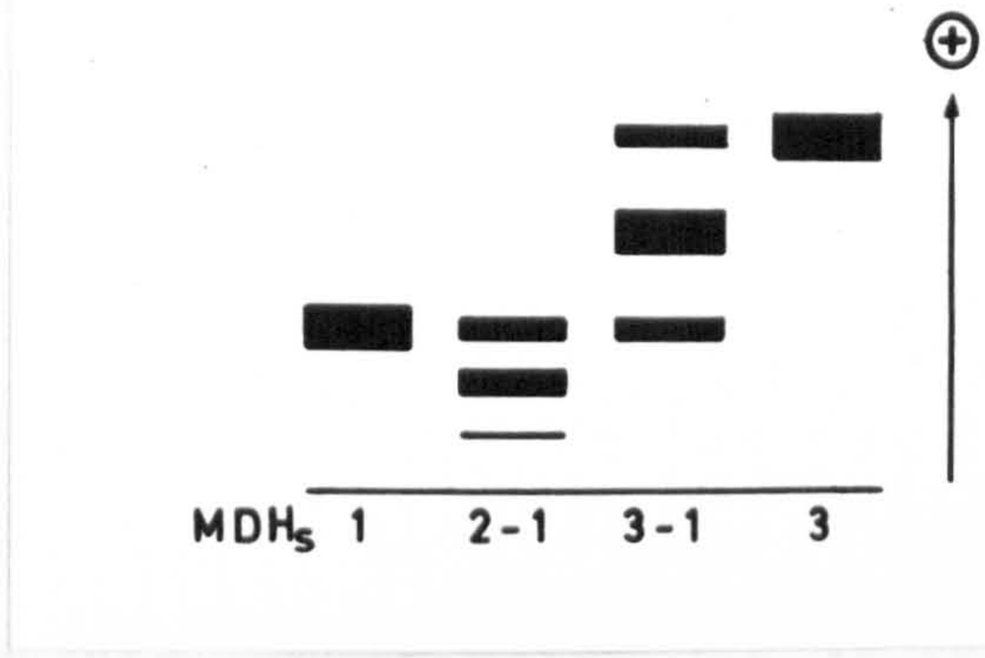


Fig. 2.3.9.b. Diagram showing the usual (MDH$_S$ 1) and some variant phenotypes of soluble malate dehydrogenase in red cells. Only the primary isozymes are shown (Leakey,T.E.B., et al. 1972).

tested for MDH system have shown only the common 1-1 homozygote type.

2.3.10 The phospho hexose isomerase (PHI) system

The enzyme phosphohexose isomerase, also known as phosphoglucose isomerase, glucose phosphate isomerase, or D-glucose-6-phosphate Ketol isomerase (EC 5.3.1.9) catalyses the reversible conversion of glucose-6-phosphate to fructose-6-phosphate.

Genetically determined variants of this enzyme were first demonstrated by Detter et al (1968). They found, in addition to the usual phenotype, PHI1-1, ten distinct variant phenotypes called PHI2-1, 3-1, 4-1, 5-1, 6-1, 7-1, 8-1, 9-1, and 10-1. Family studies showed an autosomal codominant mode of inheritance for those variants that have been subjected to it, and PHI1-1 individuals are homozygotes for the common allele at the PHI locus. The variant phenotypes are found in individuals who are heterozygous for the common allele PHI^1 and one of the rare alleles at this locus. Nothing is known about the inheritance of phenotypes 2-1, 4-1, 7-1, and 8-1.

The usual type PHI1-1 exhibits three bands, one major and two minor. The major band is cathodal. All the vairant phenotypes, except PHI9-10, consist of three major bands, one of which corresponds to the major cathodal band of the usual type. (Figure 2.3.10). The triplet pattern of the variant phenotypes suggests a dimeric structure. It is assumed that the heterozigosity at the PHI locus produces two subunits, one usual and one variant. Phenotypes such as 2-1, 3-1, 4-1 and 6-1 have a negatively charged variant subunit, whereas in types like 7-1, 8-1, and 9-1 the variant subunit is positively charged. Phenotype PHI7-1 represents a hybrid dimer. All the variant isozyme patterns are rare in the populations reported upon to date.

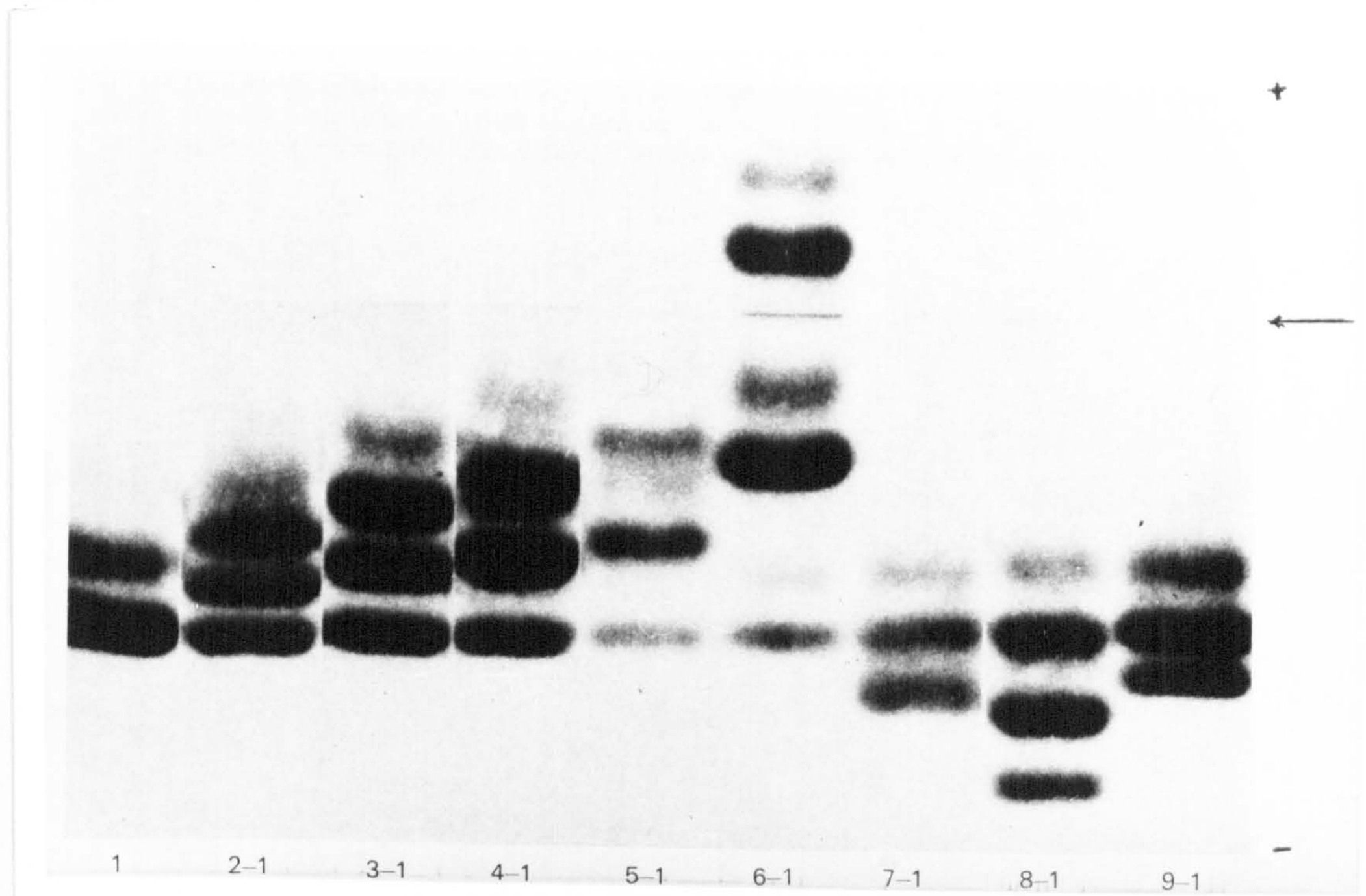


Fig.2.3.10. Electrophoretic patterns of nine phosphohexose isomerase(PHI) phenotypes. The usual phenotype, PHI 1, is on the left. The arrow points the origin (Giblett, E.R. 1969).

2.3.11 The glutamic-pyruvic trasaminase (GpT) system

The enzyme glutamic-pyruvic trasaminase, synonym alanine aminotransferase (GpT; EC 2.6.1.2), catalyses the reversible interconversion of L-alanine and α-ketoglutarate to L-glutamate and pyruvate. In rat liver, the enzyme occurs in two distinct molecular forms: a cytoplasmic or soluble form and a mitochondiral or granular form (Swick et al, 1965). It is probable that a similar state prevails in human liver and other human tissues. In human red blood cells, a soluble form occurs which has been shown, by means of starch-gel electrophoresis, to exhibit genetic polymorphism. The polymorphism is determined by two alleles GpT^1 and GpT^2. An autosomal codominant mode of inheritance is known for this enzyme system. Three common phenotypes, (Figure 2.3.11.a), called GpT1-1, GpT2-1, and GpT2-2 are distinguishable (Chen and Giblett, 1971). Apart from their different electrophoretic patterns, each of the phenotypes has a different mean level of enzyme activity. GpT1-1 individuals have, on the average, 2-3 times the red cell GpT activity of GpT2-2 individuals, while the GpT2-1 phenotype shows intermediate levels (Welch, 1972; Chen et al,1972; Olaisen, 1973; Kompf and Bissbort, 1974). Unfortunately, measurements of enzyme activity alone do not adequately distinguish members of the different phenotypes, as considerable overlaps in activity occur (Welch, 1972; Chen et al, 1972). In addition to the common alleles GpT^1 and GpT^2, population studies have led to the discovery of several rare phenotypes (Figure 2.3.11 b), and it has been postulated that six other alleles, GpT^3, GpT^4, GpT^5, GpT^6, GpT^7, and GpT^8, exist (Chen et al, 1972;Olaisen, 1973; Santachiara Benerecetti et al, 1975; Gussmann and Schwarzfischer, 1972; Spielmann et al, 1973; McAlpine et al,

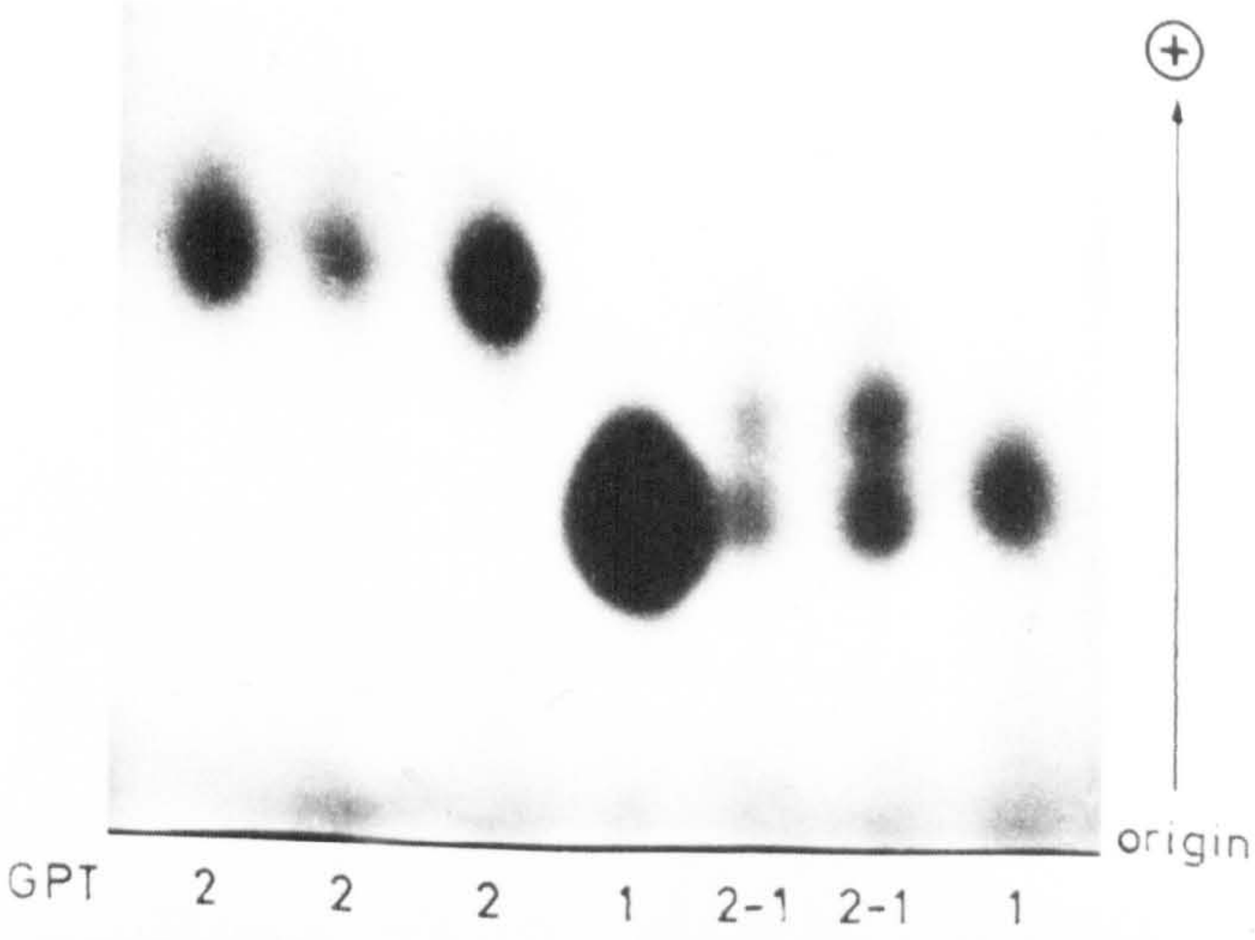


Fig.2.3.11.a. GPT in haemolysates from individuals of the three common phenotypes GPT 1, 2-1 and 2 (Harris,H., and Hopkinson,D.A. 1976).

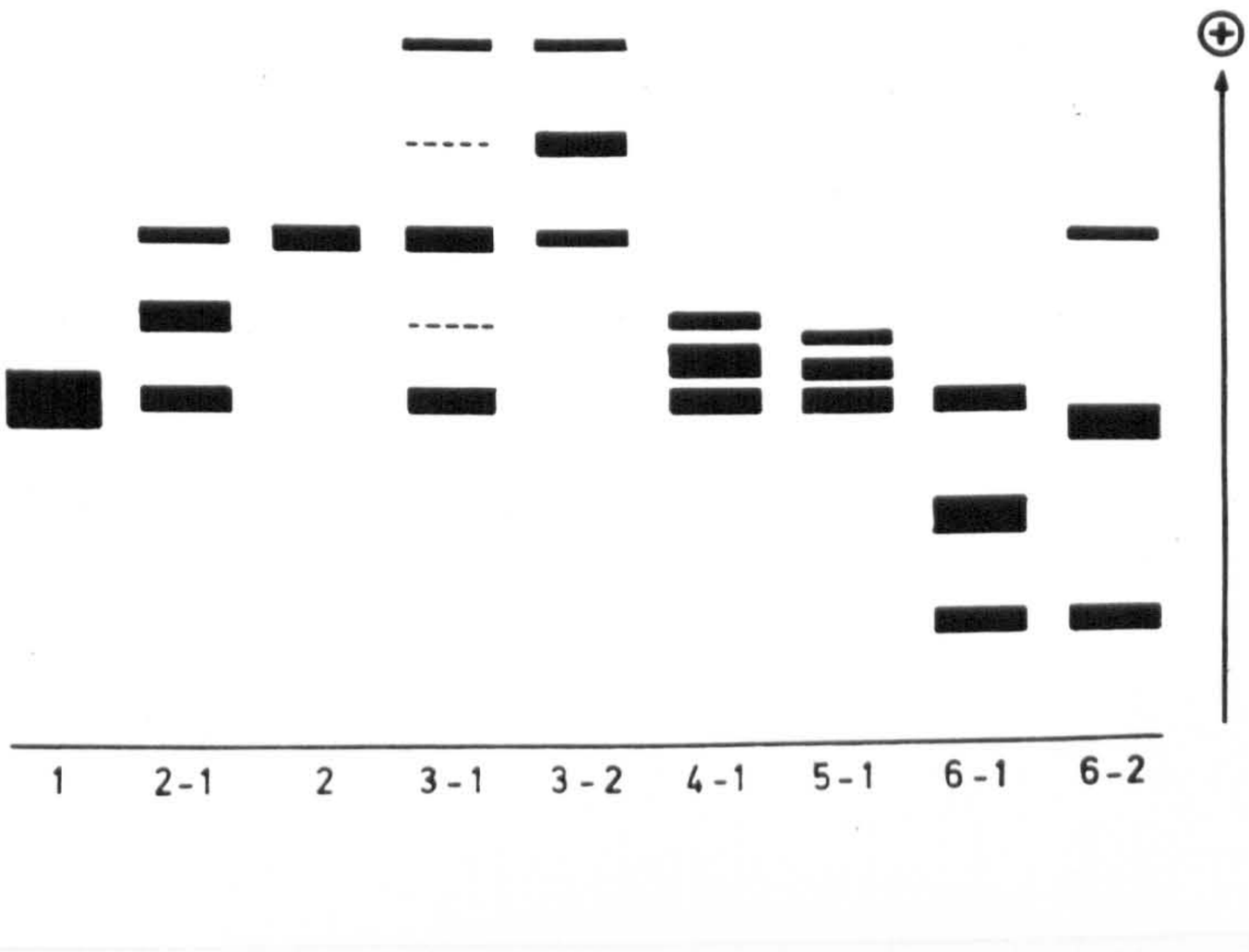


Fig. 2.3.11.b. Diagram of electrophoretic patterns of nine different GPT phenotypes(Chen, S.H., et al. 1972).

1974; Ishimoto and Kuwata, 1974; Olaisen, 1973; Brinkmann et al, 1972; Martin and Niebuhr, 1973; Wiebecke and Brackebush, 1974). Evidence for the existence of a GpT "silent" allele (GpT^0) has been reported by Olaisen (1973) in a Norwegian family, by Spielmann et al (1973) and very recently by Mithal et al (1980).

CHAPTER 3. MATERIALS AND METHODS

3.1. Blood collection

A total of 1709 blood samples were collected from ten different population groups of Iran for the purpose of the present study.

The areas and the population groups belonging to them were as follows:

Areas	Population groups
Tehran	The Moslems
	The Zoroastrians
Rezaieh	The Turks
	The Kurds
Luristan	The Lurs
Kerman	The Kermanis
Sistan and Baluchistan	The Zabolis
	The baluchis
Shirvan,Khorasan	The Turks
	The Kurds

All the blood donors were healthy and unrelated. These samples were taken in several occasions during 1979, 1980, 1981 and 1982.

In the present investigation blood grouping was carried out for the following blood group systems:

ABO, MNSs, Rh, P, Kell, Duffy and Kidd, and electrophoresis was performed for the following serum protein and red cell enzyme systems:

Haptoglobin(HP), Transferrin (Tf), the third component of human complement (C3), Acid phosphatase (AcP), Adenylate Kinase(AK), Phosphoglucomutase Locus 1 (PGM_1), Adenosine deaminase (ADA),

Esterase D (EsD) and 6-phosphogluconate dehydrogenase(6-PGD). Two out of the seven blood group (ABO and Rh), all the three serum protein and four out of the six red cell enzyme systems (AcP, AK, PGM_1, and EsD) were done for all the above mentioned samples but the remaining blood group and red cell enzyme systems were done if not for all the samples but for most of them.

A total of 645 blood samples of Tehran and Kerman were tested for blood group and serum protein systems in Tehran(Unit of human genetics and anthropology, school of public health, University of Tehran) and the lysates were brought to Durham by air and at dried iCe temperature (-78^{o}C) for the red cell enzyme systems analysis.

Another total of 670 blood specimens of Rezaieh, the Zoroastrians and Shirvan were analysed for all the blood group, serum protein and red cell enzyme systems in Tehran.

The final collection of blood samples of Luristan and Sistan, Baluchistan(total = 394) were tested only for serum proteins in Tehran and the fresh red cell suspensions and the lysates were taken to Durham within 72 hours of their collection and were tested for blood groups and for the red cell enzyme systems.

3.2.Laboratory procedures

When the blood samples were received at the laboratory:

1)The plasma was separated from the red cells by centrifugation into tubes and stored at -20^{o}C until required for serum proteins analysis. A few drops of plasma were placed in another tube for ABO grouping procedures.

2)For blood grouping procedures a few drops of red cells (0.5-1.0 ml) were separated into tubes and washed three times in normal Saline (0.85 percent) and then diluted to a 4 per-

cent suspension.

3)Haemolysates were prepared by the Carbon tetrachloride method of Ager and Lehmann (1961); briefly described below: The remaining red cells were washed three times in normal Saline. An equal volume of distilled water was added to the washed red cells, a volume of carbon tetrachloride,at least equal to twice the volume of cells plus distilled water was added and all the contents throughly mixed The tubes containing the mixture were centrifuged at 3000 R.P.M. for 20 minutes. The supernatant was placed in tubes and stored at -20^{o}C untill required for red cell enzymes analysis.

3.3.Blood grouping techniques

Three main blood grouping techniques were used and these will be described briefly.

3.3.1.Tile technique

The tile technique involves the use of an equal volume (one drop) of antiserum and 4 percent saline suspension of red cells.The cells were mixed with the particular antiserum on a clean white tile and left for a fixed period of time at a certain temperature(these two points being dependent on the antiserum being used).The tile is then rocked gently and inspected for agglutination over a strong light.

The following antisera required this technique; anti-A, anti-B, anti-AB, anti-A1, anti-A2, and anti-P1.

3.3.2.Tube technique

The tube technique involves placing an equal volumes of 4 percent red cell suspension and the appropriate anti-serum into precipitin tube and leaving at a prescribed temperature for a specific period of time. After this time the mixture is examined for agglutination microscopically.

The following antisera required this method; anti-M, anti-N,

anti-D, anti-C, anti-c, anti-E, anti-e and anti-Lu^a.
In the case of incomplete antibodies, a layer of 30 percent bovine serum albumin was added as an overlay for the reason described above after one and one and a half hours, and left for a further thirty minutes. Only anti-C^w serum required this latter technique. The presence of agglutination is investigated microscopically.

3.3.3. Indirect coombs technique

The remainder of the blood grouping required the Indirect coombs test. In this test one volume of the antiserum is incubated with one volume of 4 percent red cell suspension at 37°C in a precipitin tube for a specific period of time.
After-wards the cells, removed of antisera by washing four times in large volumes of saline, are placed on a clean tile with one drop of anti-human globulin reagent. The tile is then rocked for 5-10 minutes and the mixture is inspected for agglutination over a strong light.
This method was used with the following antisera; anti-S, anti-s, anti-Fy^a, anti-Fy^b, anti-Jk^a, anti-Jk^b, anti-K, anti-k (anti cellano), anti-KP^a, and anti-KP^b. Also all Rh(D) negative cells were tested by this method for the presence of the D^u antigen.

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

3.4.Thick layer starch gel electrophoresis

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, and the frequency of the genes and phenotypes can be used in classifying and comparing populations. Genetic traits now com-

monly investigated in population surveys include the major blood group antigens and the serum protein and red blood cell isoenzyme polymorphisms. The single most important technique developed in recent years is starch-gel electrophoresis (Smithies 1955).

Demonstration of serum and red cell enzyme phenotypes by horizontal starch-gel electrophoresis depends upon separating the individual components on the basis of their molecular size as well as their electrical charge. The resolving power of this medium has a greater ability to separate complex serum proteins, enzymes, 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.

In the present investigation this method was only used for typing of the haptoglobin groups.

3.4.1.Apparatus used:

Electrophorsis on starch gel was carried out in the horizontal position and the equipment consisted of:

1)A power pack capable of providing a constant current up to 50 mA and a constant voltage up to 500 volts.

2) Plastic tanks for tank buffer solution.

3)Platinum electrodes.

4)PH meter.

5)Gel plates: plastic plates were used with formers. The formers were placed over the plates and held secure with elastic bands around each end.The internal dimensions of the plates were 20 cmx 15 cm x 7mm.The plates were greased with liquid paraffin, before pouring the gel, to stop the gel sticking.

6) A electrical stirrer.

7)A vaccum pump.

8)Dental forceps for inserting the specimens on the gels.

9)Shandon comb for making slits in the gels.

10) A slicer board fitted with a horizontal wire for cutting the gels.

11) Plastic boxes for staining the gels.

3.4.2. Preparation of gel

For the preparation of the gel, starch obtained from Sigma or merck company was used. For each gel, a suspension of 25 gms of starch in 250 mls of gel buffer was heated over a gas flame in a one litre pyrex flask. The contents were agitated by an electrical stirrer. Heating was continued until boilling. A vaccum was applied to the flask for a few seconds for expel the air bubbles. After disconnecting the vaccum, the starch solution was poured slowly into the plastic plates until they were full. The gels were left to set for one hour at room temperature.

3.4.3. Sample insertion

After the gels had set, a cut was made with a surgical blade across each gel about two inches from the cathodal 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 gels. A piece of whatman No.3.filter paper was held by forceps and immersed in the serum sample. The moist filter paper 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 1-2 mm was left between the adjacent inserts.

3.4.4. Slicing the gel

After electrophoresis the formers were removed and the

inserted sample strips of paper were removed too, with forceps and then the gels were cut just above the insert line and 3-4 inches above the insert line. This was discarded leaving the middle protein. The gels were sliced horizontally by drawing them slowly through the wire of slicer. The required height of the horizontal wire was fixed with the height adjuster. The plastic plates with the gel on them were removed gently towards the wire to cut them in half. The sliced gels were then transfered to plastic boxes for staining.

3.5.Thin layer starch gel electrophoresis

This method was used in the present investiation for typing of all the red cell enzyme systems.

3.5.1. Apparatus used:

Electrophorsis on starch gel was carried out in the horizontal position and the equipment consisted of:

1) A power pack capable of providing a constant current up to 50 m A and a constant voltage up to 500 volts.

2) Plastic tanks for tank buffer solution.

3) Platinum electrodes.

4) PH meter.

5) Gel plates: Glass plates were used with 1 mm high glass side pieces set at the edge with araldite to provide a former for the gel. The internal dimensions of the plates were 20 cm x 15 cm x 1 mm.

6) A electrical stirrer

7) A vaccum pump

8) Dental forceps for inserting the specimens on the gles.

9)Shandon comb for making slits in the gels.

10) Plastic staining formers: These are rectangles of plastic which hold the agar overlay in place. Two size were required

for the different electrophoretic runs, their external dimensions were 13½ cm x 8 cm and 13½ cm x 11½ cm; they were 3-4 mm deep.

3.5.2. Gel making

For the preparation of the gel, starch obtained from sima or Merck company was used. For each gel, a suspension of 3.5 gms of starch in 40 mls of gel buffer was heated over a gas flame in a 250ml pyrex flask. The contents were agitated by an electrical stirrer. Heating was continued until boilling. A vaccum was applied to the flask for a few seconds for expel the air bubbles. After disconnecting the vaccum, the starch solution was poured slowly into one end of the plates and immediately spread down the plates using a bevelled edged scraper which is as wide or wider than the gel plates. The excess starch solution was scraped away from the gels and the gels were allowed to set for half an hour in the cold room.

3.5.3. Insertion of the specimens

After the gels had set, slits were made with a Shandon comb across each gel about two inches from the cathodal end. The inserts were threads drawn from a well-washed sheet of cotton. The threads were held by forceps and soaked in the lysate samples. The moist threads were inserted into the slits.

3.5.4. Clelland's reagent

This reagent is required for old specimens only and is used to eliminate any storage effect so to produce clear electrophoretic patterns.

The clelland's reagent can be used to improve 6-PGD, ADA, ESD and AcP. This reagent should not be used for PGM and AK.

Preparation of the clelland's reagent:

75 mgs of Dithiothreitol was added to 10 mls of the app-

ropriate gel buffer,then was mixed and stored at 4°C. One drop of this solution was added to one drop of lysate in a disposable 10mmx64mm tube.This mixture was applied to the gel in the usual way.

3.6.Thin layer agarose gel electrophoresis

In the present investigation this method was used for typing of trnaferrin and the fhird component of human complement.

3.6.1. Apparatus used.

Electrophoresis on agarose gel was carried out in the horizontal position and the equipment consisted of:

1) A power pack capable of providing a constant current up to 50 mA and a constant voltage upto 500 volts.

2) A apparatus designed for thin layer electrophoresis(Behringwerke AG. Marburg.Lahn).This apparatus was chosen because of its effective cooling system consisting of a centrally placed,metal cooling block which is perfused with tapwater.The glass plates rest directly on this block.On each side of the cooling block are two buffer troughs.The lid of the apparatus has a circuit breaker which interrupts the current if the lid is opened during electrophoresis.

3)Platinum electrodes.

4)PH meter.

5)Gel plates: Glass plates with dimensions of 20 cmx20cmx2mm were used.

6)Plastic boxes for staining the gels.

3.6.2.Preparation of gel

For the preparation of the gel, agarose obtained from Behringwerke AG, Marburg Lahn,Germany(OP.No.1285)was used.For each gel,a suspension of 0.4 gm of agarose in 40 mls of gel buffer was heated over a boilling water bath in a 250 ml pyrex flask.Heating was continued until boiling.

The plates were covered with a 1.5 mm thick layer of the above mentioned 1% agarose gel. The gels were allowed to set for one hour in room temperature and then were stored in a airlight moist chamber in the cold room for 1-3 days before use.

3.6.3. Sample insertion

10-15 slots for application of serum were made by gently pressing the end of a 0.9 cm broad piece of whatman filter paper No.3 into the gels about 2 cm from the cathodal end of the plates. In each slot 5 microlitres of serum was applied.

3.7.Electrophoretic methods

The serum protein and red cell enzyme systems all require fairly strict control of electrophoretic method,PH,temperature, strength of buffer solution and purity of the ingredients used in the buffer and incubation mixtures.

Electrophoretic conditions should be designed to give optimum separation of isozymes without any loss of activity.

It is often necessary to put a sheet of glass over the gels to hold the wicks in place.It is adivisable also to lay a sheet of polythen over the gels for overnight runs to help prevent evaporation.

In the present investigation all electrophoretic runs were read by two persons.All discrepancies were re-run as a double check and any weakly reacting samples were also re-run using a thicker insert and if unreliable results were still obtained the sample was not included in the overall results.

3.7.1. Haptoglobin (HP)

The method used was that of smithies(1955),but using the modification of a discontinuous system of buffers as described by Poulik(1957).One drop of a 4 percent suspension of fresh haemoglobin is added to three drops of plasma and the resulting mixture so treated in inserted into the gel using a whatman No.3

filter paper insert. Horizontal thick layer starch gel elecro-phoresis was carried out at constant voltage(100 volts) and 20 mA for 16-17 hours(over night)at 4°c., or until the borate line has moved about 3 inches from the origin.The gels were run cathode to anode(-ve to +ve).

After electrophoresis the gels were sliced horizontally in half. Stain was applied(see details bellow)and the bands appeared within 2 to 3 minutes.

The buffer system was discontinuous and was made up as follow:

Gel buffer:

Tris	9.196	gms
Citric acid	1.05	gms
Distilled water	1	litre

Adjusted to PH 8.65

Tank buffer:

NaOH	8	gms
Boric acid	74.172	gms
Distilled water	4	litres

Adjusted to PH 8.5.

Staining:

Leuco-malachite green method:

Glacial acetic acid	100	mls
Distilled water	150	mls
Leuco-malachite green	1	gm
Zinc dust powdered	1	hand full

The above ingredients were boiled in a one litre beaker until the green colour of the Leuco-malachite green disappeared. The mixture was then filtered to remove the solids and stored at 4°c. until required.The above mixture was poured over the sliced gels and left for 10 minutes; the excess stain was then poured off and 10 mls of 1/10 20 vols H202 was

poured on the gels. The bands then appeared after a few minutes.

3.7.2. Transferrin (Tf) and the third component of human complement (C3).

Tf and C3 typing was performed on the same gel because the electrophoretic conditons required are identical.

The method used was high voltage thin layer agarose gel electrophoresis described by Teisberg (1970).

5 microlitres of serum samples were applied in 10-15 slots on the gel plates.

Horizontal electrophoresis was carried out at constant voltage (20 v/cm) for 3-4 hours at room temperature. The voltage was measured by inserting two platinum wires connected to a voltmeter into the gel 15 cm appart.

A double layer of whatman filter paper No. 3 acted as connection between the buffer compartments on each side and betwe-en the two central buffer compartments on the gel.

The gels were run cathode to anode (-ve to +ve).

The buffers used were as follows:

Gel buffer: barbital/calcium lactate buffer

Natrium barbital	11.855 gms
Barbital acid	1.704 gms
Calcium lactate	0.693 gm
Distilled water	2500 mls

Adjusted to PH 8.6.

Tank buffer: barbital/calcium lactate buffer

Natrium barbital	31.443 gms
Barbital acid	4.881 gms
Calcium lactate	1.837 gms
Distilled water	2500 mls

Adjusted to PH 8.6

After electrophoresis, the gels were fixed in a 4%(v/v) solution of acetic acid, washed in distilled water and dried under filter paper at room temperature for 12-18 hours.

Staining:

Staining was performed with Amidoshwarz 10B obtained from E. Merck AG, Darmstadt, Germany.

A 1-2% solution of amidoshwarz was made up in methanol, acetic acid and water mixture. This was poured on the gels and left for 10 secs. Then rinsed off with tapwater and placed in methanol, acetic acid, water mixture.

This mixture was made up in the following ratios:

Methanol	6 parts
Acetic acid	1 part
Water	4; parts

The gels were allowed to stand in this mixture until amidoshwarz was cleared and the transferrin and C3 bands were visible. C3 bands could be seen about 2-3 cm and those of Tf about8-10 cm anodal from the application slots.

3.7.3. Acid phosphatase (AcP)

The method used was that outlined by Hopkinson et al (1963) with slight modification.

All the lysates had clleland's reagent mixed with them, volume for volume, and the resulting mixture was soaked onto threads of cotton which acted as the inserts. Horizontal thin layer starch gel electrophoresis was performed at constant voltage (105 Volts) and low amperage (5mA) for 16-17 hours at 4^{o}c. The gels were run cathode to anode(-ve to +ve). The acid phosphatase bands were then demonstrated by staining with a reac-

tion mixture containing 4- methylumbelliferyl phosphate soacted into No. 3 whatman filter paper which was placed on the gel surface for 15 minutes at 37°c. and the resulting bands could then be read under u.v. light.

The buffers used were as follows:

Tank buffer:

Natrium dehydrogen phosphate (Na H2 Po4, 2 H20)	152.8	gms
Tri-Natrium citrate (Na 3 C6H507, 2H20)	128.4	gms
Distilled water	4	litres

Adjusted to PH 6.3 with NaoH.

Gel buffer:

The tank buffer was diluted 1 in 40 and adjusted to PH 6.3 with 0.2 M citric acid.

Incubation buffer:

Citric acid	105.074	gms
Distilled water	1	litre

Adjusted to PH 6.0 with 0.05 M NaOH.

Staining (per gel):

4-methylumbelliferyl phosphate	8	mgs
Incubation buffer	10	mls

The stain was applied on the gels using whatman filter paper overlay. The gels were then incubated at 37°C. for approximately 15 minutes and then read under U.V light.

3.7.4. Adenylate Kinase(AK)

The method used was that described by Fildes and Harris (1966) with slight modification.

Horizontal thin layer starch gel electrophoresis was carried out at constant voltage (100 volts) and low amperage (5 mA)

for 16-17 hours (over night).

The gels were run anode to cathode(+ve to -ve). Clelland's reagent should not be added to the specimens for AK's.

Lysates were soaked onto threads of cotton which acted as the inserts.

The gels were stained using an agar overlay, and when the agar has set the gels were placed in an 37°c oven, electrophoretic bands appearing after about 30 minutes.

The buffers used were as follows:

Gel buffer:

Succinic acid	1.89	gms
Tris	2.226	gms
Distilled water	1	litre
Adjusted to PH 5.0		

Tank buffer:

Citric acid	114.88	gms
NaoH	42.7464	gms
Distilled water	4	litres
Adjusted to PH 4.9		

Incubation buffer:

Tris	12.12	gms
Distilled water	1	litre
Adjusted PH to 8.0 using concentrated Hcl.		

Staining (Mixture per gel):

2% Agar solution (in H20)	10	mls
Incubation buffer	10	mls
Glucose	18	mgs
Mgcl2	40	mgs
A.D.P.	4.9	mgs
N.A.D.P.	3.1	mgs

M.T.T.	2.5.	mgs
P.M.S.	2.5	mgs
G6PDH	20	microlitres
Hexokinase	20	microlitres

The ingredients were mixed in the incubation buffer and placed in the 37^{o} oven. The agar was boiled, and then cooled. When the agar solution was hand hot (approximately 55-60^{o}c) the incubation buffer was mixed in and this was poured in the previously placed former. When the agar was set, the gels were placed in the 37^{o} c oven. Bands appeared after 30 minutes.

3.7.5. Phosphoglucomutase (PGM_1).

The method used was that described by Spencer et al (1964), with slight modification.

Horizontal thin layer starch-gel electrophoresis was carried out at constant voltage (110.V) and 5-8 mA for 16-17 hours at 4^{o}c.

The gels were run cathode to anode(-ve to +ve). Clleland's reagent should not be added to the specimens for PGM's.

The haemolysates were soaked onto threads of cotton which acted as the inserts.

The buffers used were as follows:

Tank buffer:

Tris	48.44	gms
Maleic acid	46.48	gms
EDTA(acid)	11.68	gms
Mgcl2	8.12	gms
Distilled water	4	litres

Adjusted to PH 7.4 with 40% NaoH.

Gel buffer:

The tank buffer was diluted 1 in 15 with distilled water.

Incubation buffer

Tris	7.28	gms
Distilled water	1	litre

Adjusted to PH 8.0 with concentrated Hcl.

Staining (Mixture per gel):

2% Agar solution (in H20)	10	mls
Incubation buffer	10	mls
Mgcl2	20	mgs
G-1-phosphate (containing G-1:6 diphosphate)	30	mgs
N.A.D.P.	1.5	mgs
M.T.T.	2.5	mgs
P.M.S.	2.5	mgs
G-6PDH	20	microlitres

The ingredients were mixed in the incubation buffer and placed in the 37°c oven. The agar was boiled, and then cooled. When the agar solution was hand hot (55-60°c) the incubation buffer was mixed in and this was poured in the previously placed former. When the agar was set the gels were placed in the 37°c oven.

PGM, electrophoretic bands appeared after 30-60 minutes.

3.7.6. Adenosine deaminase (ADA)

The method used was that outlined by Spencer et al (1968) with slight modification.

All the lysates had cleland's reagent mixed with them, volume for volume, and the resulting mixture was soaked onto threads of cotton which acted as the inserts. Horizontal thin layer starch gel electrophoresis was performed at constant voltage (60volts) and low amperage (5mA) for 17 hours at 4°c.

The gels were run cathode to anode (-ve to +ve).

The buffers used were as follows:

Tank buffer:

NaH2P04 46.802 gms

Solution A:

Distilled water 3. litres

Na2HP04 19.874 gms

Solution B:

Distilled water 1.4 litres.

Solution A was added to solution B in order to be adjusted to PH 6.5.

Gel buffer:

The tank buffer was diluted 1 in 10 with distilled water.

Incubation buffer:

NaH2P04 0.195 gm

Solution A:

Distilled water 50 mls

Na2HP04 0.7098 gm

Solution B:

Distilled water 200 mls

Solution A was added to solution B in order to be adjusted to PH 7.5. This solution was then diluted 1 in 4 for use.

Staining (Mixture per gel):

2% Agar solution (in H20)	10	mls
Incubation buffer	10	mls
Adenosine	15	mgs
M.T.T.	2	mgs
P.M.S.	2	mgs
Xanthine oxidase	10	microlitres
Nucleoside phosphorylase	10	microlitres

The ingredients were mixed in the incubation buffer and placed in the 37^{o}c oven. The agar was boiled and then cooled.

When the agar solution was hand hot (55-60°c) the incubation buffer was mixed in and this was poured in the previously placed former. When the agar was set the gels were placed in the 37°c oven.

ADA electrophoretic bands appeared after 30 minutes.

3.7.7. Esterase D(EsD)

The method used was that outlined by Koster et al (1975), with slight modification.

All the lysates had clelland's reagent mixed with them, volume for volume, and the resulting mixture was soaked onto threads of cotton which acted as the inserts. Horizontal thin layer starch gel electrophoresis was performed at constant voltage (300 volts) and low amperage (5 mA), for 2 hours at 4°C. The gels were run negative to positive (-ve to +ve). Th esterase D bands were then demonstrated by staining with a reaction mixture containing 4 - methylumbelliferyl acetate(as described by Hopkinson et al, 1973) soacted into No.3 whatman filter paper which was placed on the gel surface for 5 minutes at 37°c. and the resulting bands could then be read under u.v. light.

The buffers used were as follows:

Gel buffer:

Tris	1.636	gms
Citric acid	0.756	gm
Boric acid	0.272	gm
Lithium hydroxide	0.016	gm
Distilled water	1	litre

Adjusted to PH 7.4.

The gel buffer was diluted volume for volume with distilled water for use.

Tank buffer:

Boric acid	108.84	gms
Lithium hydroxide	6.72	gms
Distilled water	4	litres

Adjusted to PH 7.2

Staining (per gel):

1- 41 mgs anhydrous sodium acetate was added to 10 mls distilled water and adjusted to PH 6.9.

2- 4 mgs 4-methylumbelliferyl acetate was mixed with a few drops of aceton.

3- Solutions 1 and 2 were mixed for incubation buffer and placed on 3 mm strips on each gel. The gels were incubated at 37°c. for 5 minutes and then were read under U.V. light.

3.7.8. 6- Phosphogluconate dehydrogenase (6-PGD)

The method used was that described by Hopkinson et al (1976), with slight modification.

All the lysates had clleland's reagent mixed with them volume for volume, and the resulting mixture was soaked onto threads of cotton which acted as the inserts. Horizontal thin layer starch gel electrophoresis was performed at constant voltage (100 volts) and low amperage (5 mA) for 17 hours at 4°c. The gels were run cathode to anode (-ve to +ve).

The buffers used were as follows:

Tank buffer:

NaH2PO4	46.802	gms

Solution A:

Distilled water	3	litres
Na2HPO4	19.874	gms

Solution B:

Distilled water	1.4	litres

Solution A was added to solution B in order to be adjusted to PH 7.0 and then 10 mgs NADP was added to each 500 mls of tank buffer.

Gel buffer:

The tank buffer was diluted 1 in 10 and then 4 mgs NADP was added to each 200 mls of gel buffer.

Incubation buffer:

Tris	15.14	gms
Distilled water	250	mls

Adjusted to PH 8.0 with concentrated Hcl.

Staining (Mixture per gel):

2% Agar solution (in H20)	10	mls
Incubation buffer	10	mls
6-Phosphogluconate	10	mgs
Mgcl2	0.2	gm
NADP	5	mgs
M.T.T.	5	mgs
P.M.S.	5	mgs

The ingredients were mixed in the incubation buffer and placed in the 37^{o}c oven. The agar was boiled and then cooled. When the agar solution was hand hot (55-60^{o}c) the incubation buffer was mixed in and this was poured into the previously placed formers. 6PGD, electrophoretic bands appeared after 30 minutes.

CHAPTER 4. RESULTS

4.1. Blood group antigens

4.1.1. The ABO blood group system

The distribution of the ABO blood groups and respective gene frequencies in the ten Iranian samples, expressed in term of four phenotypes after testing with two antisera, is demonstrated in Table 4.1.1.a. There was good agreement between the observed and expected phenotypic values in all samples, thus confirming the assumption of Hardy-weinberg equilibrium and that they are random mating populations.

The frequency of the A gene ranges from 19.00 percent in the Baluchis of Sistan and Baluchistan to 27.46 percent in the Turks of Rezaieh; that of the B gene from 11.97 percent in the Kurds of Rezaieh to 22.99 percent in the Kermani series. The 0 gene frequency varies between 56.13 percent in the Turks of Shirvan, Khorasan and 65.33 percent in the Lurs of Luristan.

Table 4.1.1.b shows the distribution of the A_1 A_2 BO system in six out of the ten Iranian samples, expressed in term of six phenotypes after testing with three antisera.

Close agreement was found between the observed and expected phenotypic values which confirms the assumption of Hardy-weinberg equilibrium.

The frequency of the allele A_2 ranges from 0.56 percent in the Baluchis of Sistan and Baluchistan to 7.28 percent in the Kurds of Rezaieh and that of the A_1 gene from 15.25 percent in the Zoroastrians to 24.38 percent in the Turks of Rezaieh.

4.1.2. The Rhesus blood group system

Table 4.1.2.a illustrates the distribution of Rh types and respective gene frequencies in the ten Iranian samples, expressed in term of two phenotypes after testing with one

antiserum.

The frequency of the gene d ranges from 22.36 percent in the Turks of Shirvan, Khorasan to 39.92 percent in the Kurds of Rezaieh.

The distribution of Rh types and respective gene complex frequencies in three out of the ten Iranian samples is shown in Table 4.1.2. b. The specimens were tested with the following antisera: anti-C, anti-D, anti-E, anti-c, and anti-e. There was good agreement between the observed and expected phenotypic values in all samples, thus confirming the assumption of Hardy-weinberg equilibrium. The frequencies of the principal Rh complexes are as follows:

CDe(R_1) ranges from 47.95 percent in the Lurs of Luristan to 59.63 percent in the Baluchis of Sistan and Baluchistan.

cde(r) varies between 21.81 percent in the Baluchis of Sistan and Baluchistan and 25.80 percent in the Zabolis of the same area.

cDE(R_2) ranges from 14.29 percent in the Baluchis of Sistan and Baluchistan to 22.65 percent in the Lurs of Luristan.

cDe(Ro) varies between Zero in the Zabolis of Sistan and Baluchistan and 3.52 percent in the Lurs of Luristan.

4.1.3. The MNSs blood group system

The distribution of the MNSs blood groups and respective gene combination frequencies, expressed in term of nine phenotypes after testing with four antisera, in six out of the ten Iranian samples is presented in Table 4.1.3. close agreement was found between the observed and expected phenotypic values in all samples which confirms the assumption of Hardy-weinberg equilibrium and that they are random mating populations.

The frequencies of the MNSs gene complexes are as follows:

MS ranges from 11.99 percent in the Kurds of Rezaieh to 48.95 percent in the Baluchis of Sistan and Baluchistan.

Ms varies between 26.28 percent in the Baluchis of Sistan and Baluchistan and 42.80 percent in the Zoroastrians.

NS ranges from 7.02 percent in the Lurs of Luristan to 15.73 percent in the Turks of Rezaieh.

Ns varies between 17.69 percent in the Baluchis of Sistan and Baluchistan and 40.18 percent in the Kurds of Rezaieh.

4.1.4.The P blood group system

Table 4.1.4. illustrates the distribution of the P blood groups and respective gene frequencies in three out of the ten Iranian samples.

The frequency of the P_1 gene ranges from 23.27 percent in the Turks of Rezaieh to 31.90 percent in the Kurds of the same region.

4.1.5. The Kell blood group system

Table 4.1.5.shows the distribution of the Kell blood groups and respective gene frequencies in six out of the ten Iranian samples.

The frequency of the K gene ranges from 0.46 percent in the Baluchis of Sistan and Baluchistan to 3.41 percent in the Lurs of Luristan.

4.1.6.The Duffy blood group system

The distribution of the Duffy blood groups and respective gene frequencies in six out of the ten Iranian samples is presented in Table 4.1.6.

The frequency of the Fy^a gene varies between 33.10 percent in Lurs of Luristan and 45.23 percent in the Turks of Rezaieh.

4.1.7.The Kidd blood group system

Table 4.1.7. exhibits the distribution of the Kidd blood

groups and respective gene frequencies in six out of the ten Iranian samples.

The frequency of the JK^a gene ranges from 25.52 percent in the Lurs of Luristan to 45.02 percent in the Zoroastrians.

4.2. Serum proteins

4.2.1. The haptoglobin (HP) system

The distribution of haptoglobin types and respective gene frequencies in the ten Iranian samples is shown in Table 4.2.1. The gene frequencies were calculated excluding the phenotype HP0-0. All the samples showed close agreement between the observed and expected phenotypic values, thus confirming the assumption of Hardy-weinberg equilibrium. NO HP2-1 (modified) or other rarer phenotype was detected.

The frequency of the HP^1 gene varies between 21.79 percent in the Zabolis of Sistan and Baluchistan and 30.71 percent in the Kurds of Rezaieh.

4.2.2. The transferrin (Tf) system

Table 4.2.2. presents the distribution of transferrin types and respective gene frequencies in the ten Iranian samples. Seven samples, out of ten, exhibited only the type CC. A single Zoroastrian and a single individual from Kerman were typed as CD.

In the Kurdish sample from Shirvan, Khorasan one CD and one CB were typed.

The subtyping of the variants was not performed.

4.2.3. The third component of human complement (C3) system

The distribution of C3 types and respective gene frequencies in the ten Iranian samples is shown in Table 4.2.3. The observed numbers were in close agreement with those expected, assuming Hardy-weinberg equilibrium. No rare variant was detected in the present investigation.

The frequency of the C^F3 gene ranges from 5.78 percent in the Kermani series to 22.43 percent in the Baluchis of Sistan and Baluchistan.

4.3. Red cell enzymes

4.3.1. The acid phosphatase (AcP) system

The distribution of acid phosphatase types and respective gene frequencies in the ten Iranian samples is given in Table 4.3.1. Close agreement, calculated on the basis of Hardy-weinberg equilibrium, was found between the observed and expected phenotypic values in all the samples.

No rarer AcP phenotype was detected in the present investigation. All the samples showed an absence of the CC phenotype. The frequency of the gene AcP^A ranges from 21.01 percent in the Zoroastrians to 36.99 percent in the Kurds of Rezaieh, that of the AcP^B allele varies between 63.01 percent in the Kurds of Rezaieh and 77.73 percent in the Zoroastrians. The frequency of the rarest allele AcP^C ranges from Zero both in the Turks and the Kurds of Rezaieh to 1.96 percent in the Lurs of Luristan.

4.3.2. The adenylate Kinase (AK) system

Table 4.3.2. shows the distribution of adenylate Kinase types and respective gene frequencies in the ten Iranian samples. All the samples showed close agreement between the observed and expected phenotypic values which confirms the assumption of Hardy-weinberg equilibrium and that they are random mating populations. No rare AK phenotypes were discovered in any of the samples examined.

The frequency of the AK^2 gene ranges from 1.38 percent in the Kurds of Rezaieh to 10.81 percent in the Baluchis of Sistan and Baluchistan.

4.3.3. The Phosphoglucomutase Locus 1(PGM_1) system

The frequencies of PGM_1 types and respective genes in the ten Iranian samples are given in Table 4.3.3.
Close agreement, calculated on the basis of Hardy-weinberg equilibrium, was found between the observed and expected phenotypic values.
No rare variant was detected in the present investigation.
The frequency of the PGM_1^2 gene ranges from 23.08 percent in the Zoroastrians to 43.56 percent in the Turks of Rezaieh.

4.3.4. The adenosine deaminase (ADA) system

Table 4.3.4 exhibits the frequencies of ADA types and respective genes in five out of the ten Iranian samples.
Good agreement was found between the observed and expected phenotypic values which confirms the assumption of Hardy-weinberg equilibrium.
The rarer ADA^4 and ADA^6 alleles were found only in the Turkish series of Rezaieh.
The frequency of the ADA^2 gene varies between 13.48 percent in the Turks of Rezaieh and 19.56 percent in the Zoroastrians.

4.3.5. The esterase D (EsD) system

The distribution of EsD types and respective gene frequencies in the ten Iranian samples is presented in Table 4.3.5.
There was good agreement between the observed and expected phenotypic values in all the samples, thus confirming the assumption of Hardy-weinberg equilibrium and that they are random mating populations.
No phenotypes other than common 1-1, 2-1 and 2-2 types were found in the present investigation.
The frequency of the EsD^2 gene ranges from 12.61 percent in the Turks of Shirvan, Khorasan to 29.55 percent in the Baluchis

of Sistan and Baluchistan.

4.3.6. The 6-phosphogluconate dehydrogenase (6-PGD) system

The frequncies of 6-PGD types and respective genes in five out of the ten Iranian samples are set out in Table 4.3.6. Good agreement was found between the observed and expected phenotypic values confirming the assumption of Hardy-weinberg equilibrium and that they are random mating populations. With the exception of the only CC phenotype which was found in the 301 blood specimens from Kerman, no variant other than the 'common' CA type was found in the present investigation. The frequency of the PGD^{C} allele ranges from zero in the Zoroastrians to 6.43 percent in the Kurds of Shirvan, Khorasan.

Table 4.1.1.a ABO blood groups distribution in Iran tested with anti-A, and Anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			
			A	B	AB	O	A	B	O	X^2
1-Kurds.Rezaieh	138	obs.	50	24	7	57	23.43	11.97	64.60	0.11
		Exp.	49.36	23.32	7.74	57.58				
2-Lurs.Luristan	167	obs.	56	31	9	71	21.87	12.80	65.33	0.02
		Exp.	55.69	30.68	9.35	71.28				
3-Turks.Rezaieh	142	obs.	58	27	9	48	27.46	13.66	58.88	0.41
		Exp.	56.61	25.50	10.65	49.23				
4-Tehran	328	obs.	119	71	28	110	25.63	16.44	57.93	0.01
		Exp.	118.93	71.33	27.63	110.10				
5-Baluchis.Sistan & Baluchistan	111	obs.	32	30	6	43	19.00	17.90	63.10	0.48
		Exp.	30.63	28.62	7.55	44.20				
6-Kurds.Shirvan Khorasan	142	obs.	52	38	9	43	24.75	18.42	56.83	1.96
		Exp.	48.65	34.54	12.95	45.86				

Table 4.1.1.a (Cont) ABO Blood groups distribution in Iran tested with anti- A, and Anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			
			A	B	AB	O	A	B	O	X^2
7-Zabolish.Sistan & Baluchistan	116	obs.	29	26	14	47	20.32	18.73	60.95	4.67
		Exp.	33.52	30.56	8.83	43.09				
8-Turks.Shirvan Khorasan	160	obs.	53	40	16	51	24.53	19.34	56.13	0.07
		Exp.	53.70	40.71	15.18	50.41				
9-Zoroastrians	88	obs.	20	25	10	33	18.59	22.12	59.29	1.67
		Exp.	22.43	27.39	7.24	30.94				
10-Kerman	317	obs.	82	101	28	106	19.19	22.99	57.82	0.00
		Exp.	82.03	101.03	27.97	105.98				

Table 4.1.1.b ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-A_1 sera

Population	Number Tested		Phenotypes						Gene frequencies				
			A_1	A_2	B	A_1B	A_2B	0	A_1	A_2	B	0	X^2
1-Baluchis.Sistan & Baluchistan	111	obs	32	0	30	5	1	43	18.50	0.56	17.89	63.05	4.49
		Exp.	29.92	0.79	28.60	7.35	0.22	44.12					
2-Zabolis.Sistan & Baluchistan	116	obs.	28	1	26	13	1	47	19.30	1.04	18.73	60.93	4.91
		Exp.	32.06	1.48	30.55	8.39	0.45	43.06					
3-Zoroastrians	88	obs.	17	3	25	8	2	33	15.25	3.35	22.12	59.28	1.72
		Exp.	18.86	3.59	27.38	5.94	1.30	30.92					
4- Turks.Rezaieh	92	obs.	36	5	18	3	1	29	24.38	4.55	12.94	58.13	2.12
		Exp.	33.59	5.06	15.38	5.80	1.08	31.09					
5- Lurs.Luristan	167	obs.	47	9	31	5	4	71	17.10	4.86	12.80	65.24	2.97
		Exp.	44.91	10.98	30.64	7.31	2.08	71.07					
6- Kurds.Rezaieh	138	obs.	38	12	24	3	4	57	16.26	7.28	11.97	64.49	2.47
		Exp.	35.85	13.70	23.28	5.37	2.41	57.39					

Table 4.1.2.a Rh blood groups distribution in Iran tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies	
		D+	D-	D	d
1-Turks.Shirvan Khorasan	160	152	8	77.64	22.36
2-Kerman	317	301	16	77.53	22.47
3-Lurs.Luristan	162	152	10	75.16	24.84
4-Baluchis.Sistan & Baluchistan	111	104	7	74.89	25.11
5-Kurds.Shirvan Khorasan	142	132	10	73.47	26.53
6-Zabolis.Sistan & Baluchistan	115	104	11	69.07	30.93
7-Tehran	302	268	34	66.40	33.60
8-Turks.Rezaieh	141	124	17	65.27	34.73
9-Zoroastrians	88	77	11	64.64	35.36
10-Kurds.Rezaieh	138	116	22	60.08	39.92

Table 4.1.2.b. Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Phenotypes																		
			CCDEE	CCDEe	CCDee	CCdEE	CCdEe	CCdee	CcDEE	CcDEe	CcDee	CcdEE	CcdEe	Ccdee	ccDEE	ccDEe	ccDee	ccdEE	ccdEe	ccdee
1-Baluchis.Sistan & Baluchistan	111	obs.	0	0	44	0	0	0	1	18	30	0	0	1	3	7	1	0	0	0
		Exp.	0	0.78	42.06	0	0	0.04	0.18	19.84	31.24	0	0	0.95	2.27	7.47	0.87	0	0	5.28
2-Lurs.Luristan	162	obs.	0	0	40	0	0	0	2	30	46	0	0	0	11	20	3	0	0	10
		Exp.	0.01	1.26	37.25	0	0	0	0.59	35.94	44.42	0	0	0	8.31	20.98	3.06	0	0	10.18
3-Zabolis.Sistan & Baluchistan	115	obs.	0	0	40	0	0	1	2	13	32	0	0	2	8	9	0	0	0	8
		Exp.	0.01	1.31	35.67	0	0	0.31	0.40	22.79	30.10	0	0	3.10	3.42	10.23	0	0	0	7.66

Population	Number Tested	Gene complex frequencies								
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	X^2
1-Baluchis.Sistan & Baluchistan	111	0.57	59.63	0.00	1.96	14.29	1.74	0.00	21.81	5.21
2-Lurs.Luristan	162	0.81	47.95	0.00	0.00	22.65	3.52	0.00	25.07	6.75
3-Zabolis.Sistan & Baluchistan	115	1.02	50.71	0.00	5.22	17.24	0.00	0.00	25.80	20.67

Table 4.1.3 MNSs blood groups distribution in Iran tested with anti-M, anti-N, anti-S, and anti-s sera

Pupolation	Number Tested	Phenotypes								
		MMSS	MMSs	MMss	MNSS	MNSs	MNss	NNSS	NNSs	NNss
1-Kurds.Rezaieh	124	obs. 2	11	18	2	19	40	2	9	21
		Exp. 1.29	9.97	19.25	2.62	20.25	39.12	1.33	10.29	19.88
2-Turks.Rezaieh	139	obs. 4	19	26	7	28	30	4	11	10
		Exp. 4.79	20.68	22.32	6.76	29.18	31.49	2.38	10.29	11.11
3-Zoroastrians	74	obs. 2	12	13	4	16	16	2	4	5
		Exp. 3.00	12.48	13.00	3.86	16.09	16.75	1.24	5.18	5.40
4-Zabolis.Sistan & Baluchistan	114	obs. 6	28	18	4	18	23	2	4	11
		Exp. 5.13	21.34	22.21	5.44	22.63	23.56	1.44	6.00	6.25
5-Lurs.Luristan	153	obs. 22	41	20	9	30	15	1	6	9
		Exp. 16.80	39.31	22.99	13.13	30.72	17.97	2.57	6.00	3.51
6-Baluchis.Sistan & Baluchistan	111	obs. 27	28	8	10	22	9	1	2	4
		Exp. 20.89	30.67	11.26	13.75	20.20	7.42	2.26	3.33	1.22

Table 4.1.3 (Cont) MNSs blood groups distribution in Iran tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Gene complex frequencies				
		MS	Ms	NS	Ns	X^2
1-Kurds.Rezaieh	124	11.99	37.60	10.23	40.18	1.39
2-Turks.Rezaieh	139	16.15	42.48	15.73	25.64	2.27
3-Zoroastrians	74	18.01	42.80	14.24	24.95	1.33
4-Zabolis-Sistan & Baluchistan	114	24.62	40.73	7.84	26.81	8.87
5-Lurs.Luristan	153	36.81	35.08	7.02	21.09	13.43
6-Baluchis.Sistan & Baluchistan	111	48.95	26.28	7.08	17.69	12.04

Table 4.1.4. P blood groups distribution in Iran tested with anti-P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies	
		P_1	P_2	P_1	P_2+P
1-Turks.Rezaieh	141	58	83	23.27	76.73
2-Zoroastrians	86	41	45	27.66	72.34
3-Kurds.Rezaieh	138	74	64	31.90	68.10

Table 4.1.5. Kell blood groups distribution in Iran tested with anti- K serum only

Population	Number Tested	Phenotypes		Gene frequencies	
		K(+)	K(-)	K	k
1-Baluchis.Sistan & Baluchistan	110	1	109	0.46	99.54
2-Zoroastrians	86	1	85	0.58	99.42
3-Kurds.Rezaieh	138	3	135	1.09	98.91
4-Zabolis.Sistan & Baluchistan	115	3	112	1.31	98.69
5-Turks.Rezaieh	141	7	134	2.51	97.49
6-Lurs.Luristan	149	10	139	3.41	96.59

Table 4.1.6 Duffy blood groups distribution in Iran tested with anti-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies	
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+ Fy
1-Lurs.Luristan	143	79	64	33.10	66.90
2-Baluchis.Sistan & Baluchistan	47	28	19	36.42	63.58
3-Zoroastrians	86	52	34	37.12	62.88
4-Zabolis.Sistan & Baluchistan	115	70	45	37.45	62.55
5-Kurds.Rezaieh	138	89	49	40.41	59.59
6-Turks.Rezaieh	140	98	42	45.23	54.77

Table 4.1.7 Kidd blood groups distribution in Iran tested with anti-JK^a serum only

Population	Number Tested	Phenotypes		Gene frequencies	
		JK(a+)	JK(a-)	JK^a	Jk^b+JK
1-Lurs.Luristan	146	65	81	25.52	74.48
2-Kurds.Rezaieh	136	69	67	29.81	70.19
3-Baluchis.Sistan & Baluchistan	111	58	53	30.90	69.10
4-Turks.Rezaieh	141	74	67	31.07	68.93
5-Zabolis .Sistan & Baluchistan	115	70	45	37.45	62.55
6-Zoroastrians	86	60	26	45.02	54.98

Table 4.2.1. Haptoglobin (HP) Phenotypes and gene frequencies distribution in Iran

Population	Number Tested		Phenotypes HP 1-1	HP 2-1	HP 2-2	HP 0	Gene frequencies HP^1	HP^2	X^2
1-Zabolis.Sistan & Baluchistan	118	obs.	3	45	69	1	21.79	78.21	1.92
		Exp.	5.56	39.88	71.56				
2-Lurs.Luristan	178	obs.	10	61	106	1	22.88	77.12	0.10
		Exp.	9.27	62.47	105.27				
3-Kurds.Shirvan Khorasan	102	obs.	4	39	58	1	23.27	76.73	0.67
		Exp.	5.47	36.06	59.47				
4-Turks.Shirvan, Khorasan	106	obs.	3	43	57	3	23.79	76.21	2.36
		Exp.	5.83	37.34	59.83				
5-Baluchis.Sistan & Baluchistan	111	obs.	5	43	59	4	24.77	75.23	0.66
		Exp.	6.56	39.87	60.56				
6-Turks.Rezaieh	145	obs.	11	51	79	4	25.89	74.11	0.46
		Exp.	9.45	54.10	77.45				
7-Zoroastrians	171	obs.	15	56	95	5	25.90	74.10	2.44
		Exp.	11.14	63.72	91.14				

Table 4.2.1 (Cont) Haptoglobin (HP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested		Phentoypes HP 1-1	HP 2-1	HP 2-2	HP 0	Gene frequencies HP^1	HP^2	X^2
8-Tehran	136	obs.	9	51.	71	5	26.34	73.66	0.00
		Exp.	'9.09	50.83	71.08				
9-Kerman	294	obs.	31	108	141	14	30.36	69.64	2.16
		Exp.	25.80	118.39	135.80				
10-Kurds.Rezaieh	145	obs.	18	50	72	5	30.71	69.29	3.62
		Exp.	13.21	59.59	67.21				

Table 4.2.2. Transferrin(Tf) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies		
		TfCC	TfCB	TfCD	Tf^C	Tf^B	Tf^D
1-Turks.Shirvan, Khorasan	122	120	1	1	99.18	0.41	0.41
2-Zoroastrians	175	174	-	1	99.71	0.00	0.29
3-Kerman	313	312	-	1	99.84	0.00	0.16
4-Tehran	186	186	-	-	100.00	0.00	0.00
5-Zabolis .Sistan & Baluchistan	118	118	-	-	100.00	0.00	0.00
6-Baluchis.Sistan & Baluchistan	111	111	-	-	100.00	0.00	0.00
7-Kurds.Shirvan, Khorasan	112	112	-	-	100.00	0.00	0.00
8-Kurds.Rezaieh	147	147	-	-	100.00	0.00	0.00
9-Turks.Rezaieh	149	149	-	-	100.00	0.00	0.00
10-Lurs.Luristan	178	178	-	-	100.00	0.00	0.00

Table 4.2.3. The third component of human complement(C3) phenotypes and gene frequencies distribution in Iran

Population	Number Tested		Phenotypes C3 SS	C3 SF	C3 FF	Gene frequencies F C3	S C3	2 X
1-Kerman	199	obs.	177	21	1	5.78	94.22	0.18
		Exp.	176.66	21.67	0.67			
2-Kurds.Shirvan, Khorasan	60	obs.	53	6	1	6.67	93.33	2.32
		Exp.	52.27	7.47	0.27			
3-Teheran	51	obs.	44	7	0	6.87	93.13	0.27
		Exp.	44.23	6.53	0.24			
4-Turks.Rezaieh	90	obs.	77	12	1	7.78	92.22	0.43
		Exp.	76.54	12.91	0.55			
5-Kurds.Rezaieh	89	obs.	76	12	1	7.87	92.13	0.43
		Exp.	75.55	12.90	0.55			
6-Turks.Shirvan, Khorasan	82	obs.	70	11	1	7.92	92.08	0.55
		Exp.	69.53	11.96	0.51			

Table 4.2.3. (Cont.) The third component of human complement (C3) phenotypes and gene frequencies distribution in Iran

Population	Number Tested		Phenotypes C3 SS	C3 SF	C3 FF	Gene frequencies $C3^F$	$C3^S$	X^2
7-Zabolis.Sistan & Baluchistan	109	obs.	80	27	2	14.22	85.78	0.02
		Exp.	80.21	26.59	2.20			
8-Zoroastrians	131	obs.	93	34	4	16.03	83.97	0.17
		Exp.	92.37	35.26	3.37			
9-Lurs.Luristan	143	Obs.	101	37	5	16.43	83.57	0.60
		Exp.	99.87	39.27	3.86			
10-Baluchis.Sistan & Baluchistan	107	Obs.	67	32	8	22.43	77.57	2.11
		Exp.	64.38	37.23	5.38			

Table 4.3.1 Red cell Acid phosphatase(AcP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested		Phenotypes						Gene frequencies			
			AcP A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	A AcP	B AcP	C AcP	X^2
1-Zoroastrians	119	obs.	4	42	70	-	3	-	21.01	77.73	1.26	1.44
		Exp.	5.25	38.87	71.90	0.63	2.33	0.02				
2-Kurds.Shirvan Khorasan	101	obs.	8	28	63	-	2	-	21.78	77.23	0.99	3.91
		Exp.	4.79	33.98	60.24	0.44	1.54	0.01				
3-Lurs.Luristan	178	obs.	16	61	94	2	5	-	26.69	71.35	1.96	1.76
		Exp.	12.68	67.79	90.62	1.86	4.98	0.06				
4-Turks.Shirvan Khorasan	115	obs.	11	40	62	-	2	-	26.96	72.17	0.87	2.18
		Exp.	8.36	44.75	59.90	0.54	1.44	0.01				
5-Turks.Rezaieh	125	obs.	12	48	65	-	-	-	28.80	71.20	0.00	0.51
		Exp.	10.37	51.26	63.37	0.00	0.00	0.00				
6-Kerman	307	obs.	31	133	134	4	5	-	32.41	66.12	1.47	0.68
		Exp.	32.25	131.59	134.23	2.92	5.95	0.07				

Table 4.3.1. (cont) Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested		Phenotypes						Gene frequencies			x^2
			AcP A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	A AcP	B AcP	C AcP	
7-Teheran	352	obs.	49	145	146	1	11	-	34.66	63.64	1.70	5.81
		Exp.	42.28	155.27	142.55	4.16	7.64	0.10				
8-Zabolis.Sistan & Baluchistan.	117	obs.	19	44	51	1	2	-	35.47	63.25	1.28	3.00
		Exp.	14.72	52.50	46.81	1.06	1.89	0.02				
9-Baluchis.Sistan &Baluchistan.	111	obs.	17	47	46	-	1	-	36.49	63.06	0.45	1.32
		Exp.	14.78	51.08	44.14	0.37	0.63	0.00				
10-Kurds.Rezaieh	146	obs.	17	74	55	-	-	-	36.99	63.01	0.00	1.11
		Exp.	19.97	68.05	57.97	0.00	0.00	0.00				

Table 4.3.2. Red cell Adenylate Kinase (AK) Phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies		
			AK 1-1	AK 2-1	AK 2-2	AK^1	AK^2	X^2
1-Baluchis.Sistan & Baluchistan	111	obs.	88	22	1	89.19	10.81	0.09
		Exp.	88.30	21.40	1.30			
2-Zabolis.Sistan & Baluchistan	118	obs.	94	23	1	89.40	10.60	0.10
		Exp.	94.31	22.36	1.33			
3-Lurs.Luristan	178	obs.	154	22	2	92.70	7.30	1.35
		Exp.	152.96	24.09	0.95			
4-Tehran	357	obs.	306	51	-	92.86	7.14	2.11
		Exp.	307.82	47.36	1.82			
5-Kerman	309	obs.	268	38	3	92.88	7.12	1.52
		Exp.	266.57	40.87	1.57			
6-Zoroastrians	120	obs.	105	15	-	93.75	6.25	0.53
		Exp.	105.47	14.06	0.47			
7-Kurds.Shirvan Khorasan	103	obs.	91	12	-	94.17	5.83	0.39
		Exp.	91.35	11.30	0.35			

Table 4.3.2. (cont) Red cell Adneylate Kinase (AK) Phenotypes and gene frequencies distribution in Iran

Population	Number Tested		Phenotypes AK 1-1	AK 2-1	AK 2-2	Gene frequencies AK^1	AK^2	X^2
8-Turks.Shirvan, Khorasan	116	obs.	103	13	-	94.40	5.60	0.41
		Exp.	103.36	12.27	0.36			
9.Turks.Rezaieh	127	obs.	120	7	-	97.24	2.76	0.10
		Exp.	120.10	6.81	0.10			
10-Kurds.Rezaieh	145	obs.	141	4	-	98.62	1.38	0.03
		Exp.	141.03	3.94	0.03			

Table 4.3.3 Red cell phosphoglucomutase Locus1(PGM_1) phenotypes and gene frequencies distribution in Iran

Pupolation	Number Tested		Phenotypes PGM 1-1	PGM 2-1	PGM 2-2	Gene frequencies PGM_1^1	PGM_1^2	X^2
1-Turks.Rezaieh	101	obs.	36	42	23	56.44	43.56	2.40
		Exp.	32.17	49.66	19.17			
2-Kurds.Rezaieh	138	obs.	47	68	23	58.70	41.30	0.04
		Exp.	47.55	66.91	23.54			
3-Kerman	310	obs.	123	144	43	62.91	37.09	0.01
		Exp.	122.69	144.67	42.64			
4-Kurds.Shirvan, Khorasan	92	obs.	40	40	12	65.22	34.78	0.16
		Exp.	39.13	41.78	11.13			
5-Baluchis.Sistan & Baluchistan	110	obs.	45	54	11	65.45	34.55	0.80
		Exp.	47.12	49.75	13.13			
6-Tehran	346	obs.	152	153	41	66.04	33.96	0.07
		Exp.	150.90	155.20	39.90			

Table 4.3.3. (Cont) Red cell Phosphoglucomutase Locus 1(PGM_1) phenotypes and gene frequencies distribution in Iran

Population	Number Tested		Phenotypes PGM 1-1	PGM 2-1	PGM 2-2	Gene frequencies PGM_1^1	PGM_1^2	X^2
7-Zabolis.Sistan & Baluchistan	117	obs.	53	51	13	67.09	32.91	0.02
		Exp.	52.66	51.67	12.67			
8-Turks.Shirvan, Khorasan	110	obs.	50	50	10	68.18	31.82	0.25
		Exp.	51.13	47.73	11.14			
9-Lurs.Luristan	175	obs.	90	68	17	70.86	29.14	0.61
		Exp.	87.87	72.27	14.86			
10-Zoroastrians	104	Obs.	60	40	4	76.92	23.08	0.72
		Exp.	61.53	36.93	5.54			

Table 4.3.4. Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Iran.

Population	Number Tested		Phenotypes ADA 1-1	ADA 2-1	ADA 2-2	ADA 4-1	ADA 6-1	Gene frequencies ADA^1	ADA^2	ADA^4	ADA^6	X^2
1-Zoroastrians	46	obs.	31	12	3	-	-	80.44	19.56	0.00	0.00	1.35
		Exp.	29.76	14.48	1.76	0.00	0.00					
2-Turks.Shirvan, Khorasan	118	obs.	83	33	2	-	-	84.33	15.67	0.00	0.00	0.39
		Exp.	83.92	31.18	2.90	0.00	0.00					
3-Kurds.Shirvan, Khorasan	101	obs.	74	23	4	-	-	84.66	15.34	0.00	0.00	1.54
		Exp.	72.39	26.23	2.38	0.00	0.00					
4-Kurds.Rezaieh	147	obs.	106	38	3	-	-	85.04	14.96	0.00	0.00	0.04
		Exp.	106.31	37.40	3.29	0.00	0.00					
5-Turks.Rezaieh	141	obs.	104	30	4	1	2	85.46	13.48	0.35	0.71	1.09
		Exp.	102.98	32.49	2.56	0.84	1.71					

Table 4.3.5. Red cell Esterase D (EsD) Phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies		
			EsD 1-1	EsD 2-1	EsD 2-2	EsD^1	EsD^2	X^2
1-Baluchis.Sistan & Baluchistan	110	obs.	57	41	12	70.45	29.55	1.21
		Exp.	54.59	45.80	9.60			
2-Turks.Rezaieh	129	obs.	66	54	9	72.09	27.91	0.21
		Exp.	67.05	51.91	10.05			
3-Kurds.Rezaieh	143	obs.	81	57	5	76.57	23.43	1.76
		Exp.	83.85	51.30	7.85			
4-Kerman	306	obs.	204	91	11	81.54	18.46	0.05
		Exp.	203.43	92.14	10.43			
5-Tehran	345	obs.	229	105	11	81.60	18.40	0.06
		Exp.	229.72	103.60	11.68			
6-Zoroastrians	111	obs.	74	35	2	82.43	17.57	0.87
		Exp.	75.43	32.15	3.43			
7-Kurds.Shirvan, Khorasan	101	obs.	71	27	3	83.66	16.34	0.05
		Exp.	70.70	27.61	2.70			

Table 4.3.5. (Cont.) Red cell Esterase D(EsD)phenotypes and gene frequencies distribution in Iran

Population	Number Tested		Phenotypes EsD 1-1	EsD 2-1	EsD 2-2	Gene frequencies EsD^1	EsD^2	X^2
8-Zabolis.Sistan & Baluchistan	117	obs.	86	28	3	85.47	14.53	0.16
		Exp.	85.47	29.06	2.47			
9-Lurs.Luristan	178	obs.	136	36	6	86.52	13.48	3.15
		Exp.	133.25	41.52	3.23			
10-Turks.Shirvan, Khorasan	115	obs.	88	25	2	87.39	12.61	0.02
		Exp.	87.83	25.34	1.83			

Table 4. 3. 6. Red cell 6-Phosphogluconate dehydrogenase (6-PGD)phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Pehnotypes				Gene frequencies		
			PGD AA	PGD AC	PGD CC	PGD^A	PGD^C	X^2
1-Kurds.Shirvan, Khorasan	70	obs.	61	9	-	93.57	6.43	0.33
		Exp.	61.29	8.42	0.29			
2-Turks.Shirvan, Khorasan	90	obs.	80	10	-	94.44	5.56	0.31
		Exp.	80.28	9.44	0.28			
3-Kerman	301	obs.	278	22	1	96.01	3.99	0.62
		Exp.	277.48	23.04	0.48			
4-Tehran	82	obs.	79	3	-	98.17	1.83	0.03
		Exp.	79.03	2.95	0.03			
5-Zoroastrians	66	obs.	66	-	-	100.00	0.00	0.00
		Exp.	66.00	0.00	0.00			

CHAPTER 5. DISCUSSION AND CONCLUSION

5.1. Blood group antigens

5.1.1. The ABO blood group system

The distribution of the ABO blood groups and respective gene frequencies in Iranian and neighbouring populations, expressed in terms of four and six phenotypes after testing with two and three antisera, respectively, is set out in Tables 5.1.1.a and b.

The frequency of the A gene ranges from 11.19 to 38.17 percent in Iranians, being lowest in the Bakhtiaris (NiJenhuis, 1964) and highest in the Armenians of Esfahan (Bowman and Walker,1961); that of the gene B varies between 9.59 percent in the Assyrians of Abadan (Nijenhuis, 1964) and 34.54 percent in the Zoroastrians (Bowman and Walker, 1961). The frequency of the 0 gene in Iran ranges from 50.32 percent in the Kurdish Jews (Godber et al, 1973) to 76.80 percent in the Bakhtiaris(Nijenhuis, 1964). Values obtained in the present investigation are within this range of variation.
On the whole, with an average A gene frequency of 22.24 percent, B gene frequency of 16.98 percent and 0 gene frequency of 60.78 percent, the Iranian population appears to exhibit higher B and lower A gene frequencies than those found in Europeans (Mourant et al, 1976).

Some differences in the distribution of the ABO gene frequencies seem to exist in Iran, as the B values increase from west to east and those of the A gene decrease. In the present investigation also two groups of Kurds and Turks of Rezaieh (north west) exhibit lower B gene frequencies of 11.97 and 13.66 percent compared with those of 18.42 and 19.34 percent in the same two population groups of Shirvan, Khorasan (north east).

Some other remarkable things must be noted:

It appears from the table that the Kurds of western Iran,like most other populations of the region,exhibit low B gene frequencies but the Kurdish Jews have rather high frequencies of both the A and the B genes. The frequencies of the gene A in the non-Kurdish Jews of Iran as in those of Iraq are also high. It is thus almost certain that the Jewish populations on the whole exhibit high frequencies of the A gene both in Iran and in Iraq.

The Arab population of Khuzistan shows ABO frequencies that are outside the range of the Arabian frequencies found by several investigators in various studies. However, in the course of history they must have become intermixed to a certain degree with negro elements, as can still be observed from the forms of hair and nose.

The Zoroastrians are separable from the Moslems by ABO blood groups frequencies as they appear to exhibit higher B and lower O gene frequencies compared with those found in the Moslems. Whatever the Zoroastrians were before the Islamic era, there is no doubt that they are a separate and genetically different breeding group from that of the present Moslem majority.

The Armenian and the Assyrian populations of Iran both seem to be characterized by high frequencies of the A and low frequencies of the B genes.

The high B and low O gene frequencies in the Turkmans of north eastern Iran suggest Mongoloid ancestry.

In neighbouring areas, the frequency of the A gene in the populations of the Caucasus ranges from 13.76 percent in the Adighes of north western Caucasus to 36.64 percent in the Khevsurs of highlands of Georgia (Bunak, 1960); that of the gene B

from 3.67 percent in the Megrelians, Abashi(Voronov, 1973) to 18.98 percent in the Lakts of Dagestan (Gadzhiev, 1964). The 0 gene frequency in the Caucasus varies between 52.54 percent in the Khevsurs of highlands of Georgia.(Bunak, 1960) and 78.14 percent in the Mingrelians of Georgia, Abkhazskaya (Aseyeva, 1936). With an average A gene frequency of 23.68 percent, B gene frequency of 10.73 percent and 0 gene frequency of 65.59 percent, the population of the Caucasus appears to exhibit a much lower B and a slightly higher A gene frequency than those found in Iranians. In general, ABO system frequencies show that the populations living in the Caucasus are characterized by low frequencies of the gene B and considerable presence of the 0 gene.

The frequency of the A gene in the populations of Turkey ranges from 22.94 percent in south east Anatolia to 32.97 percent in west Black sea (Saatcioglu , 1979); that of the gene B from 8.89 percent in the Eti-Turks(Aksoy et al, 1958) to 17.00 percent in the Kurds (Richard, 1976). The 0 gene frequency in Turkey varies between 53.86 percent in Aegean and 64.64 percent in East Black Sea (Saatcioglu, 1979). With an average A gene frequency of 28.97 percent, B gene frequency of 12.79 percent and 0 gene frequency of 58.24 percent, the population of Turkey seems to show a much lower B and a higher A gene frequency than those found in Iranians. The frequency of the 0 gene in Turkish population is only slightly lower than in Iranians.

The frequency of the A gene in the populations of Iraq ranges from 11.73 percent in the Kurdish Jews of the south east (Tills et al, 1977) to 36.00 percent in the Kurdistani Jews (Gurevitch et al, 1955); that of the B gene from 10.31

percent in the Arabs(Field, 1957) to 34.58 percent in the Karaite Jews (Goldschmidt et al, 1976). The 0 gene frequency in Iraq varies between 42.89 percent in the Karaite Jews (Goldschmidt et al, 1976) and 64.44 percent in the Iraqis abroad(Onsi and El-Alfi, 1968). With an average A gene frequency of 24.22 percent, B gene frequency of 18.73 percent and 0 gene frequency of 57.05 percent, the population of Iraq appears to exhibit slightly higher B and A but lower 0 gene frequencies than those found in Iranians.

The extremely high B gene frequency of 34.58 percent and low 0 gene frequency of 42.89 percent in the Karaite Jews of Iraq (Goldschmidt et al, 1976) clearly indicate the strong influence of isolation and genetic drif operating in this community.

The frequency of the A gene in the Kuwaiti populations ranges from 14.00 percent in the Suluba tribe to 18.00 percent in the Ajman tribe; that of the B gene from 4.00 percent in the Ajman tribe to 17.00 percent in the Suluba tribe. The 0 gene frequency in Kuwait varies between 69.00 percent in the Suluba tribe and 78.00 percent in the Ajman tribe (Khaled et al, 1981). With an average A gene frequency of 15.97 percent, B gene frequency of 12.63 percent and 0 gene frequency of 71.40 percent, the population of Kuwait seems to show much lower A and B but higher 0 gene frequencies than those found in Iranians.

The frequency of the gene A in Saudi Arabians ranges from 11.42 percent in the Sunnis of Asir and Najran (Maranjian et al, 1966) to 21.90 percent in the Saudi Arabian sample of Goedde et al (1979); that of the B gene from 4.12 percent in the Sunni sample of Asir, Najran (Maranjian et al, 1966) to 28.90 percent in the Saudi Arabian sample of Goedde et al

(1979). The O gene frequency in Saudi Arabia varies between 49.20 percent in the Saudi Arabian sample(Goedde et al,1979) and 84.46 percent in the Sunni sample of Asir, Najran (Maranjian et al, 1966). With an average A gene frequency of 15.38 percent, B gene frequency of 13.21 percent and O gene frequency of 71.41 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit much lower A and B but higher O gene frequencies than those found in Iranians.

Similar low A and B gene frequencies of respectively 19.60 and 8.20 percent and a high O gene frequency of 72.20 percent are reported by Kamel et al (1980) for the Abu-Dhabians of the United Arab Emirates.

In general, the Arab populations of Kuwait, Saudi Arabia and the United Arab Emirates are characterized by the high Overall frequency of O and the frequencies of the A and the B genes are correspondingly low. These findings agree with what is at present known about the frequencies of ABO in the indigenous desert populations (Mourant et al, 1976).

The frequency of the A gene in the populations of Pakistan ranges from 15.42 percent in the Multans (Lodhi,1960) to 28.37 percent in the Baltis of Baltistan (Clegg et al, 1961); that of the gene B from 22.99 percent in Dacca (Attabudin, 1954) to 29.23 percent in the Pathan, Dari, Timurgara sample of Bernhard (1967). The O gene frequency in Pakistan varies between 48.53 percent in the Baltis of Baltistan (Clegg et al, 1961) and 58.79 percent in the Multans (Lodhi,1960). With an average A gene frequency of 19.50 percent, B gene frequency of 25.24 percent and O gene frequency of 55.26 percent, the Pakistani population appears to exhibit lower A, much higher B and lower O gene frequencies than those found in Iranians.

The frequency of the gene A in the populations of Afghanistan ranges from 13.91 percent in the Uzbeks (Samin,1965) to 28.60 percent in the Pushtus (Papiha et al, 1977); that of the B gene from 17.80 percent in the Pushtus (Papiha et al, 1977) to 36.21 percent in the Hazaras (Samin, 1965). The 0 gene frequency in Afghanistan varies between 45.04 percent in the Hazaras (Samin, 1965) and 60.80 percent in the Daris(Papiha et al, 1977). With an average A gene frequency of 20.42 percent, B gene frequency of 25.25 percent and 0 gene frequency of 54.33 percent, the population of Afghanistan, like that of Pakistan, seems to show lower A, much higher B and lower 0 gene frequencies than those found in Iranians.

The generalized features of blood groups frequencies in Afghanistan suggest Caucasoid ancestry on the one hand and Mongoloid on the other. The frequency of B in Afghanistan is higher than has been found in any Iranian population, except for those on the northern frontier with Soviet Turkmenistan. Further east, in northern Pakistan and India, and in the neighbouring areas north of the main mountain barrier, similar high frequencies of B are found in both Caucasoids and Mongoloids. In summary the Afghan populations appear to be intermediate in allele frequencies between Caucasoid and Mongoloid populations, with unmistakable evidence of both in their ancestry.

Regarding the A sub-alleles of the ABO system (Tables 5.1.1.b), the A_2 allele is present in all populations tested and its frequency ranges from 0.56 to 8.01 percent in Iranians' the lowest being in the Baluchis of Sistan and Baluchistan(present investigation) and the highest in the Kurds of Sanandaj (Lehmann et al, 1973). With an average A_2 allele frequency of 4.20 percent, the Iranian population on the whole appears to

exhibit a lower A2 allele frequency than that of about 10 percent found in Europeans (Mourant et al,1976). In Iranians about 19 percent of the total A genes are A2.

From the table it seems that some differences in the distribution of the A2 allele exist in Iran as the Kurds and the Kurdish Jews of western Iran appear to exhibit higher A2 allele frequencies than those found in other parts of the country.

The Arab population of Khuzistan (south west) also seems to be characterized by high frequencies of the A2 allele. By contrast the Baluchis and the Zabolis of Sistan and Baluchistan (south east) show the lowest frequencies of this allele in Iran. There are very few reports on the distribution of the A2 allele frequencies in the populations of eastern Iran, so it is difficult to see any west-east decrease of the A2 allele frequencies.

In neighbouring populations, the A2 allele frequency of 2.66 percent in the Svanis of Georgia, Abkhazskaya of the Caucasus (Verbitsky et al, 1971) is lower than that found in Iranians and lower than the frequency in Europeans. In the population of the Caucasus about 12 percent of the total A genes are A2.

The A2 allele frequencies of 6.77 and 8.10 percent in the Turks and the Eti-Turks of Turkey (Aksoy et al, 1958) are higher than the average for Iran but still lower than the European frequencies. In the Turkish populations about 25 percent of the total A genes are A2.

The frequency of the A2 allele in the Iraqi populations varies between 1.12 percent in the Kurdish Jews of the south east (Tills et al, 1977) and 18.27 percent in the Karaite Jews

(Goldschmidt et al, 1976). With the exception of the extremely high A2 allele frequency of 18.27 percent in the Karaite Jews, the population of Iraq with an average A2 frequency of 3.41 percent seems to exhibit lower A2 values than those found in Iranians and lower than the European frequencies. In Iraq about 16 percent of the total A genes are A2.

The very high A2 frequency of 18.27 percent in the Karaite Jews of Iraq clearly indicates the strong influence of isolation and genetic drift operating in this community.

The A2 allele frequency in the Kuwaiti populations ranges from 2.13 percent in the Kuwaiti Arabs (Sawhney, 1975) to 11.00 percent in the Ajman tribe (Khaled et al,1981). With an average A2 allele frequency of 7.03 percent, the population of Kuwait appears to show higher A2 values than those found in Iranians but lower than the frequencies in Europeans. In the Kuwaiti population about 43 percent of the total A genes are A2.

The frequency of the A2 allele in Saudi Arabians varies between 3.60 percent in the Shia sample of Qatif and Hasa Oases and 8.34 percent in the Sunni sample of Hejaz(Maranjian et al, 1966). With an average A2 allele frequency of 6.02 percent, the Arab population of Saudi Arabia, like that of Kuwait, seems to exhibit higher A2 values than those found in Iranians but lower than the European frequencies. In Saudi Arabians about 42 percent of the total A genes are A2.

The A2 allele frequency in the populations of Pakistan ranges from 2.59 percent in the Hunza, Gilgit (Ikin et al, 1959)to 4.19 percent in the Baltis of Baltistan(Clegg et al, 1961). With an average A2 allele frequency of 3.46 percent, the Pakistani population appears to show lower A2 allele fre-

quencies than those found in Iranians and lower than the frequencies in Europeans.In the population of Pakistan about 16 percent of the total A genes are A2.

The frequency of the A2 allele in the populations of Afghanistan varies between 3.62 percent in the Timuri and related tribes(Woodd-Walker et al,1967)and 8.00 percent in the Pushtus(Papiha et al,1977).With an average A2 allele frequency of 5.21 percent,the Afghan population seems to exhibit slightly higher A2 frequencies than those found in Iranians but still lower than the European frequencies. In Afghanistan about 22 percent of the total A genes are A2.

Conclusion

Frequencies of the gene A are high in Europe, especially in Scandinavia, in parts of central Europe, and in an area extending from Bulgaria into Asia Minor and Turkistan. The frequency of A2 appears to vary somewhat irregularly, but it .nowhere greatly exceeds 10 percent.

The distribution of B is more definite and less Patchy than that of A. There is a well marked maximum frequency of B in central Asia and northern India. In Europe the frequency of the B gene diminishes steadily from the borders of Asia to a minimum somewhere below 5 percent in parts of the Netherlands, France, Spain and Portugal. There is a slight but significant rise to over 7 percent further in the celtic countries and in Iceland, with higher local maxima near 10 percent in parts of wales, Scotland and Ireland (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab populations of Kuwait, Saudi Arabia and the United Arab Emirates with their low frequencies of both the A and the B genes which seem to be characteristics of these population groups, the frequency of the A gene is lower and that

of the B gene much higher than those found in Europeans. On the whole, in the area under discussion, frequencies of the total A genes and the A2 allele decrease and those of the B gene increases from the west (Turkey) to the east (Pakistan).

Table 5.1.1.a I ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

Population	Number tested	Phenotypes				Gene grequencies			Authors	
		A	B	AB	0	A	B	0		
1-Assyrians.Abadan	32	14	2	4	12	33.10	9.59	57.31	Nijenhuis, L.E.	1964
2-Southern Gorgan,Behshahr,Sari	53	18	7	3	25	22.30	9.90	67.80	Kirk, R.L., et al.	1977
3-Armenians	78	38	12	4	24	32.38	10.92	56.70	Nijenhuis, L.E.	1964
4-Armenians	145	71	22	10	42	35.20	11.00	53.80	Tabatabai, H.	1977
5-West	110	38	19	4	49	21.50	11.10	67.40	Bajatzadeh,M.,& Walter,H.	1969
6-Armenians.Julfa	145	79	21	10	35	38.17	11.43	50.40	Bowman,J.E.,& Walker,D.G.	1961
7-North	73	25	13	3	32	21.61	11.66	66.73	Bajatzadeh,M.,& Walter,H.	1969
8-Teheran	115	46	22	3	44	24.52	11.66	63.82	Bajatzadeh,M.,& Walter,H.	1969
9-East	78	38	13	4	23	32.42	11.66	55.92	Bajatzadeh,M.,& Walter,H.	1969
10-Mamassanis.Fars	113	29	21	4	59	15.87	11.76	72.37	Bowman,J.E., & Walker, D.,G.	1961
11-Kurds.Rezaieh	138	50	24	7	57	23.43	11.97	64.60	Present study.	
12-Bakhtiaris	138	28	30	1	79	11.19	12.01	76.80	Nijenhuis, L.E.	1964
13-Tavalesh.Astara	49	18	10	1	20	22.10	12.10	65.70	Kirk, R.L., et al.	1977
14-Shahsavar,Rudsar,Rudbar,Rasht Langarud,Lahijan,Bandar-Pahlavi	87	18	15	5	49	14.10	12.20	73.70	Kirk, R.L., et al.	1977

Table 5.1.1.a.I (Cont.) ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Genefrequencies			Authors	
		A	B	AB	O	A	B	O		
15-Tehran	140	17	8	2	23	10.90	12.35	76.75	Beckett, P.H.	1950
16-Lurs. Luristan	167	56	31	9	71	21.87	12.80	65.33	Present study.	
17-Kurds.Baneh, Marivan	77	25	14	5	33	21.79	13.17	65.04	Lehmann,H., et al.	1973
18-Turks.Rezaieh	142	58	27	9	48	27.46	13.66	58.88	Present study.	
19-Kurdistan	148	48	30	9	61	21.58	14.18	64.24	Boue' & Boue'.	1955
20-Central and South	113	40	24	6	43	23.19	14.34	62.47	Bajatzadeh,M.,& Walter,H.	1969
21-Azarbaijan	157	61	31	11	54	26.49	14.46	59.05	Boue' and Boue'.	1955
22-Ghashghais.Abadeh	1518	476	321	72	469	20.57	14.47	64.96	Montazami,K.	1978
23-Ghashghais	66	23	14	4	25	23.13	14.75	62.12	Nijenhuis,L.E.	1964
24-Azarbaijan	1467	547	296	108	516	25.63	14.90	59.47	Montazami, K.	1978
25-Basseris.Fars	101	30	24	40	43	18.67	15.08	66.25	Bowman,J.E,& Walker,D.G.	1961
26-Tehran	20000	7000	4600	1000	7400	22.71	15.26	62.03	Vazin, H.	1969
27-Tehran	565	173	133	29	230	19.93	15.61	64.46	Motamed, M.	1949
28-Kerman	10000	3327	2224	660	3789	22.50	15.67	61.83	Azhir, A.	1951
29-Esfahan	111	29	25	11	46	18.23	15.95	65.82	Sawhney, K.S.	1975
30-Jews	225	83	45	22	75	26.85	16.13	57.02	Silberstein,W., & Goldstein,N.	1958

Table 5.1.1.a.I (Cont)

ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	O	A	B	O		
31-Khuzestan	2043	525	503	113	902	17.08	16.42	66.50	Montazami, K.	1978
32-Tehran	328	111	63	37	117	25.63	16.44	57.93	Present study.	
33-Mashhad	17277	5259	4535	641	6842	19.03	16.47	64.50	Afkari, A.H.	1967
34-Fars.Recipients	16368	4652	3872	1094	6750	19.42	16.52	64.06	Mohallatee, E.A.,& Haghshenas, M .	1969
35-Tehran	150000	48060	34815	10995	56130	22.14	16.66	61.20	Tehranchian..	1963
36-Kurds.Kermanshah	127	41	28	11	47	23.12	16.66	60.22	Nijenhuis,L.E.	1964
37-Jews	108	43	29	6	30	30.60	16.70	52.70	Tabatabai, H.	1977
38-Tehran	992	337	224	80	351	23.86	16.71	59.43	Roshan,G., et al.	1969
39-Tehran	42329	13037	7894	5337	16061	24.35	16.80	58.85	Berelian-Jahanshahi,F.	1973
40-Mobarakeh.Esfahan	1211	357	284	93	477	20.68	16.97	62.35	Montazami, K.	1978
41-Esfahan	9753	2992	2326	709	3726	21.22	17.01	61.77	Mirdamadi, M.,et al.	1978
42-Gorgan twon	537	169	120	48	200	22.70	17.03	60.27	Boue' and Boue'.	1956
43-Shiraz	5407	1633	1333	353	2088	20.50	17.07	62.43	Tahmasbian A.	1972
44-Tehran	3049	1036	721	229	1063	23.56	17.07	59.37	Boue' and Boue'.	1956
45-South east	348	91	86	23	148	17.99	17.08	64.93	Nijenhuis, L.E.	1964

Table 5.1.1.a.I (Cont)

ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
46-Esfahan	4385	1426	1093	274	1592	21.86	17.12	61.02	Montazami, K.	1978
47-Khuzestani. Non Arab	2555					19.40	17.30	63.30	Fakhrai, H., et al.	1978
48-Gonbad	156	63	37	12	44	28.30	17.30	54.40	Kirk, R.L., et al.	1977
49-Tehran	99	23	26	6	44	15.65	17.48	66.87	Sawhney, K.S.	1975
50-Gorgan Province	134	46	29	14	45	25.53	17.48	56.99	Boue' and Boue'.	1956
51-Baluchis.Sistan & Baluchistan	111	32	30	6	43	19.00	17.90	63.10	Present study.	
52-Tehran	1196	374	300	90	432	21.79	17.93	60.28	Defai, H.	1971
53-Babol,Shahi,Amol	70	21	19	4	26	20.00	18.10	61.90	Kirk, R.L., et al.	1977
54-Persian Gulf area	4574	1084	1277	233	1980	15.65	18.19	66.16	Montazami, K.	1978
55-Tehran	2334	748	579	196	811	22.82	18.27	58.91	Sadatzadeh,H., & Hamidi, A.H.	1972
56-Kurds.Sanandaj	107	33	25	11	38	23.12	18.41	58.47	Lehmann, H.,et al.	1973
57-Kurds.Shirvan, Khorasan	142	52	38	9	43	24.75	18.42	56.83	Present study.	
58-Jews	200	75	51	16	58	26.37	18.59	55.04	Gurevitch,J.,et al.	1956

Table 5.1.1.a.I (Cont)

ABO blood groups distribution in Iran tested with anti-A, and Anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
59-Arabs.Khuzestan	97					21.20	18.60	60.20	Marzban, M.	1978
60-Zabolis.Sistan & Baluchistan	116	29	26	14	47	20.32	18.73	60.95	Present study.	
61-North west	76	16	23	3	34	13.51	18.95	67.54	Bajatzadeh, M., & Walter, H.	1969
62-Moslems.Fars	768	211	205	60	292	19.53	19.05	61.42	Bowman,J.E., & Walker, D.G.	1961
63-Ghashgais	103	22	25	11	45	17.30	19.06	63.64	Bowman,J.E., & Walker, D.G.	1961
64-Gilan.Province	225	77	54	24	70	25.67	19.10	55.23	Boue' and Boue'.	1956
65-Turks.Shirvan,Khorasan	160	53	40	16	51	24.53	19.34	56.13	Present study .	
66-Northern Gorgan	43	19	13	2	9	29.30	19.90	50.80	Kirk, R.L.,et al.	1977
67-Turkish.Shahsavan Azerbaijan	247	89	62	28	68	27.42	20.26	52.32	Boue' and Boue'.	1956
68-Hamadan.Province	115	38	31	12	34	24.80	20.86	54.34	Boue' and Boue'.	1956
69-Kurdish Jews	94	34	24	12	24	28.29	21.39	50.32	Godber, Marilyn J.,et al .	1973

Table 5.1.1.a.I(Cont) ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
70-Kurdish Jews	106	35	29	12	30	25.31	21.66	53.03	Tills, D., et al.	1977
71-Zoroastrians	88	20	25	10	33	18.59	22.12	59.29	Present study.	
72-Arabs.Khuzestan	364					16.50	22.90	60.60	Fakhrai, H.,et al.	1978
73- Kerman	317	82	101	28	106	19.19	22.99	57.82	Present study.	
74-Arabs.Abadan	158	33	51	15	59	16.46	23.50	60.04	Nijenhuis, L.E.	1964
75-Shi'a . Yazd	307	81	100	30	96	20.09	24.06	55.85	Sunderland,E., & Smiht, H.M.	1966
76-Moslems. Yazd	258	57	90	29	82	17.14	25.44	57.41	Boue' and Boue'.	1956
77-Turkmans	1163	296	401	116	350	19.64	25.48	54.88	Montazami, K.	1978
78-Turkmans	374	101	134	44	95	21.78	27.64	50.58	Boue' and Boue'.	1956
79-Zoroastrians. Tehran and Yazd	150	25	69	17	39	15.09	34.54	50.37	Bowman, J.E., & Walker D.G.	1961

Table 5.1.1.a. II

ABO blood groups distribution in the Caucasus tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
1-Megrelians.Abashi	195	66	11	3	115	19.62	3.67	76.71	Voronov, A.A.	1973
2-Abkhazians Ochamchiri	91					18.29	4.27	77.44	Voronov, A.A.	1973
3-Zugdidi.Mingrelians.Georgia	168	58	15	3	92	20.22	5.52	74.26	Aseyeva, S.M.	1936
4-Georgia.Except Gudauta	397	121	40	9	227	18.00	6.37	75.63	Aseyeva, S.M.	1936
5-Mingrelians .Georgia,Abkhazeskaya	229	58	24	6	141	15.09	6.77	78.14	Aseyeva, S.M.	1936
6-Svanis.Georgia,Abkhazeskaya	659	234	62	26	337	22.12	6.89	70.99	Verbitsky,M.Sh.,et al.	1971
7-Imeretians.Trans.Caucasia	287	85	32	9	161	17.97	7.40	74.63	Bunak, V.V.	1960
8-Imeretians.Southern Caucasus	80	28	9	3	40	21.71	7.80	70.49	Aseyeva, S.M.	1936
9-Gudauta	191	57	25	4	105	17.54	7.93	74.53	Aseyeva, S.M.	1936
10-Gurians.Southern Caucasus	32	16	4	1	11	31.81	8.22	59.97	Aseyeva, S.M.	1936
11-Gurians.Lunchkhuti	98					14.32	9.68	75.99	Voronov, A.A.	1973
12-Azerbaijanians.Barda	147	76	17	11	43	36.07	10.02	53.93	Voronov, A.A.	1973
13-Armenians	317	170	43	17	87	36.27	10.05	53.68	Aseyeva, S.M.	1936
14-Goris	428	180	61	24	163	27.68	10.49	61.83	Solovyeva, T.G.	1967

Table 5.1.1.a. II(Cont) ABO blood groups distribution in the Caucasus tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	O	A	B	O		
15-Osetes.Northern Caucasus	220	82	33	11	94	24.03	10.56	65.41	Bunak, V.V.	1960
16-Khevsurs.High lands of Georgia	69	37	10	4	18	36.64	10.82	52.54	Bunak, V.V.	1960
17-Azerbaijanians.Nukha	287	110	48	14	115	24.71	11.53	63.75	Voronov, A.A.	1973
18-Adighes.North-Western Caucasus	486	115	98	9	264	13.76	11.75	74.49	Bunak, V.V.	1960
19-Karachay-Cherkesskaya oblast	811	282	145	35	349	22.03	11.83	66.14	Grikurov, V.S.	1963
20-Ingushes.North-Eastern Caucasus	407	165	76	17	149	25.87	12.27	61.86	Bunak, V.V.	1960
21-Khumarin,Uchkulan	269	100	49	13	107	23.94	12.33	63.73	Lauer, V.V.	1929
22-Kakhetians.Gurdjaani	196	86	27	19	64	32.45	12.36	55.18	Voronov, A.A.	1973
23-Lezgians.Dagestan	208	72	38	13	85	23.08	13.11	63.81	Gadzhiev,A.G.	1964
24-Darghians.Dagestan	189	66	35	12	76	23.35	13.32	63.33	Gadzhiev, A.G.	1964
25-Chechens.North-Eastern Caucasus	756	299	159	38	260	25.78	14.14	60.08	Bunak, V.V.	1960
26- Azerbaijanians.Shemakha	180	72	35	14	59	27.80	14.70	57.49	Voronov, A.A.	1973

Table 5.1.1.a.II (Cont) ABO blood groups distribution in the Caucasus tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
27-Kabardins.Northern Caucasus	106	29	23	6	48	18.14	14.75	67.11	Bunak, V.V.	1960
28-Kumyks.Dagestan	187	61	38	18	70	23.83	16.18	59.99	Gadzhiev, A.G.	1964
29-Avars.Dagestan	210	68	45	25	72	25.07	18.14	56.79	Gadzhiev, A.G.	1964
30-Lakts.Dagestan	168	52	41	17	58	23.12	18.98	57.90	Gadzhiev, A.G.	1964

Table 5.1.1.a.III ABO blood groups distribution in Turkey tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
1-Eti-Turks.Asia Minor, near Mersin	118	55	14	6	43	30.59	8.89	60.55	Aksoy,M.,et al.	1958
2-Black sea region·Eastern	5295	2284	652	259	2100	27.93	9.01	63.06	Mizan, N., et al.	196?
3-East Black sea	195	78	29	8	80	25.33	10.03	64.64	Saatci oglu, Armagan.	1979
4-Marmara	649	292	93	42	222	30.00	11.00	59.00	Saatci oglu, Armagan.	1979
5-Turks.Istanbul	8674	3833	1245	592	3004	30.00	11.22	58.78	Benbanaste, M.	1963
6-Black sea region.Western	3612	1581	550	253	1228	29.84	11.82	58.34	Mizan, N.,et al.	196?
7-Anatolia central.Eastern part	7023	3126	1091	511	2295	30.59	12.15	57.26	Mizan, N., et al.	196?
8-Anatolia.Eastern	7598	3474	1148	593	2383	31.83	12.20	55.97	Mizan, N., et al.	196?
9-Anatolia central.Middle part	5453	2501	818	444	1690	32.15	12.32	55.53	Mizan, N., et al.	196?
10-Middle Black sea	360	157	54	30	119	30.59	12.40	57.01	Saatci oglu,Armagan.	1979
11-West Black sea	173	82	28	13	50	32.97	12.70	54.33	Saatci oglu,Armagan.	1979
12-Marmar and Thrace	8430	3681	1390	626	2733	30.10	12.79	57.11	Mizan, N., et al..	196?
13-Mediterranean region	3122	1251	548	211	1112	27.11	13.02	59.87	Mizan, N., et al.	196?

Table 5.1.1.a.III (Cont) ABO blood groups distribution in Turkey tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
14.Turks.Abroad.Recruits	364	146	64	25	129	27.21	13.09	59.70	Paidoussis, M.	1967
15-East Anatolia	399	158	72	26	143	26.64	13.17	60.19	Saatci oglu, Armagan.	1979
16-Central Anatolia	1141	486	193	91	371	29.68	13.33	56.99	Saatci oglu, Armagan.	1979
17-Mediterranean region	321	122	51	30	118	27.20	13.40	59.40	Saatci oglu, Armagan.	1979
18-Asia Minor Aegean region	3166	1291	546	249	1080	28.31	13.45	58.24	Mizan, N., et al.	196?
19-Anatolia Central.Western part	4096	1819	700	331	1246	31.12	13.51	55.37	Mizan, N., et al.	196?
20-Anatolia.South-Eastern	5200	2020	1016	390	1774	26.91	14.59	58.50	Mizan, N., et al.	196?
21-Turks.Asia Minor,Mersin	108	43	20	10	35	28.54	14.94	56.52	Aksoy,M.,et al.	1958
22-Aegean	423	178	79	42	124	30.67	15.47	53.86	Saatci oglu, Armagan.	1979
23-South east Anatolia	220	76	50	13	81	22.94	15.60	61.46	Saatci oglu, Armagan.	1979
24-Kurds						27.00	17.00	56.00	Richard, P.	1976

Table 5.1.1.a IV ABO blood groups distribution in Iraq tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
1-Arab	123	51	18	6	48	26.81	10.31	62.88	Field, H.	1957
2-Kurdistani Jews	250	117	34	32	67	36.00	14.04	49.96	Gurevitch, J.,et al.	1955
3-Kurds						23.00	16.00	61.00	Al-Khafaji, S.D.,et al.	1976
4-Kurdish Jews.North West	61	27	12	6	16	32.42	16.08	51.50	Tills, D., et al.	1977
5-Kurdistani Jews	129	53	23	17	36	32.02	16.76	51.22	Gurevitch, J.,et al.	1956
6-Iraqi abroad	313	89	81	16	127	18.56	17.00	64.44	Onsi, A.A., & El-Alfi,	1968
7-Jews	308	128	61	38	81	31.94	17.53	50.53	Silberstein,W., & Goldstein, N.	1958
8-Civilian donors	1185	406	286	98	395	24.23	17.81	57.96	Fakhrie, S.D.	1966
9-Bedouin.Baghdad	338	90	87	23	138	18.43	17.89	63.68	Kayassi,A.I.,et al.	1938
10-Assyrians	99	27	25	8	39	19.53	18.26	62.21	Ikin,Elizabeth W.,et al.	1965
11-Paid donors	4056	1209	1037	320	1490	21.06	18.42	60.52	Fakhrie, S.D.	1966
12-Kurds	1500	449	293	227	531	25.29	18.73	55.98	Kennedy, W.P., and MaC-farlane, J.	1936
13-Kurdish Jews	27	10	8	1	8	23.81	18.74	57.45	Godber,Marilyn J.,et al.	1973

Table 5.1.1.a. IV (Cont) ABO blood groups distribution in Iraq tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
14-Arabs	493	150	133	36	174	21.15	18.98	59.87	Kennedy,W.P., and MaCfarlane, J.	1936
15-Moslems	386	121	109	26	130	21.54	19.49	59.06	Boyd,W.C., and Boyd,L.G.	1941
16-Military donors	1067	306	300	77	384	19.98	19.63	60.39	Fakhrie, S.D.	1966
17-Baghdadi Jews	162	62	39	21	40	30.09	20.60	49.31	Gurevitch,J.,et al.	1956
18-Kurdish Jews.South East	50	8	19	3	20	11.73	25.17	63.10	Tills, D., et al.	1977
19-Karaite Jews	72	22	34	6	10	22.53	34.58	42.89	Goldschmidt,E.,et al.	1976

Table 5.1.1.a. V ABO blood groups distribution in Kuwait tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	O	A	B	O		
1-Ajman tribe	52	17	3	1	31	18.00	4.00	78.00	Khaled, E., et al.	1981
2-Kuwaiti Arabs	162	45	32	4	81	17.31	12.68	70.01	Sawhney, K.S.	1975
3-General population	74	19	17	2	36	15.00	14.00	71.00	Khaled,E., et al.	1981
4-Kuwaitis	2632	638	635	115	1244	15.53	15.47	69.00	Onsi, A. A.,et al.	1968
5-Suluba tribe	52	12	14	2	24	14.00	17.00	69.00	Khaled, E., et al.	1981

Table 5.1.1.a. VI ABO blood groups distribution in Saudi Arabia tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	O	A	B	O		
1-Sunnis.Asir,Najran	74	14	4	2	54	11.42	4.12	84.46	Maranjian,G.,et al.	1966
2-Sunnis. Hejaz	102	27	14	4	57	16.50	9.22	74.28	Maranjian,G.,et al.	1966
3-Bedouins	178	49	28	7	94	17.25	10.35	72.40	Maranjian, G.,et al.	1966
4-Sunnis. Najd	180	42	35	6	97	14.44	12.12	73.44	Maranjian, G.,et al.	1966
5-Western Saudi Arabia	210	54	41	8	107	16.18	12.57	71.38	Saha, N.,et al.	1980
6-Sunnis.Qatif & Hasa Oases	323	73	74	7	169	13.36	13.49	73.15	Maranjian, G.,et al.	1966
7-Shias.Qatif & Hasa Oases	465	88	112	17	248	12.05	14.98	72.97	Maranjian, G.,et al.	1966
8-Saudi Arabians	311	63	85	10	153	21.90	28.90	49.20	Goedde, H.W., et al.	1979

Table 5.1.1.a. VII

ABO blood groups distribution in the United Arab Emirates

tested with

anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		A	B	AB	0	A	B	0	
1- Abu-Dhabians	624	208	86	12	318	19.60	8.20	72.20	Kamel,K., et al. 1980

Table 5.1.1.a. VIII ABO blood groups distribution in Pakistan tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		A	B	AB	0	A	B	0	
1-Dacca	1000	254	311	97	338	19.38	22.99	57.63	Attabudin.ed.by Boyde, 1954 W.C. & Boyde,Lyle G.
2-Baltis.Baltistan	80	23	9	3	25	28.37	23.10	48.53	Clegg, E.J., et al. 1961
3-Hunza.Gilgit	23	5	8	2	8	16.77	24.78	58.45	Ikin,Elizabeth W.,et al.1959
4-Pathans.Swat,Saidu Sharif	133	31	42	18	42	20.32	25.54	54.14	Alciati, G. 1968
5-Multans	201	43	76	14	68	15.42	25.79	58.79	Lodhi, M.A.K. 1960
6-Pathan.Dari,Timurgara	78	16	31	8	23	16.76	29.23	54.01	Bernhard, W. 1967

Table 5.1.1.a. IX ABO blood groups distribution in Afghanistan tested with anti-A, and anti-B sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		A	B	AB	0	A	B	0		
1-Pushtus	104	42	24	7	31	28.60	17.80	53.60	Papiha,S.S.,et al.	1977
2-Daris	179	51	48	14	66	20.10	19.10	60.80	Papiha,S.S.,et al.	1977
3-Timuri & related tribes	118	30	38	14	36	20.73	25.02	54.25	Woodd-Walker,R.B.,et al.	1967
4-Uzbeks	369	90	171	3	105	13.91	28.10	57.99	Samin, P.	1965
5-Hazaras	1052	263	533	77	179	18.75	36.21	45.04	Samin, P.	1965

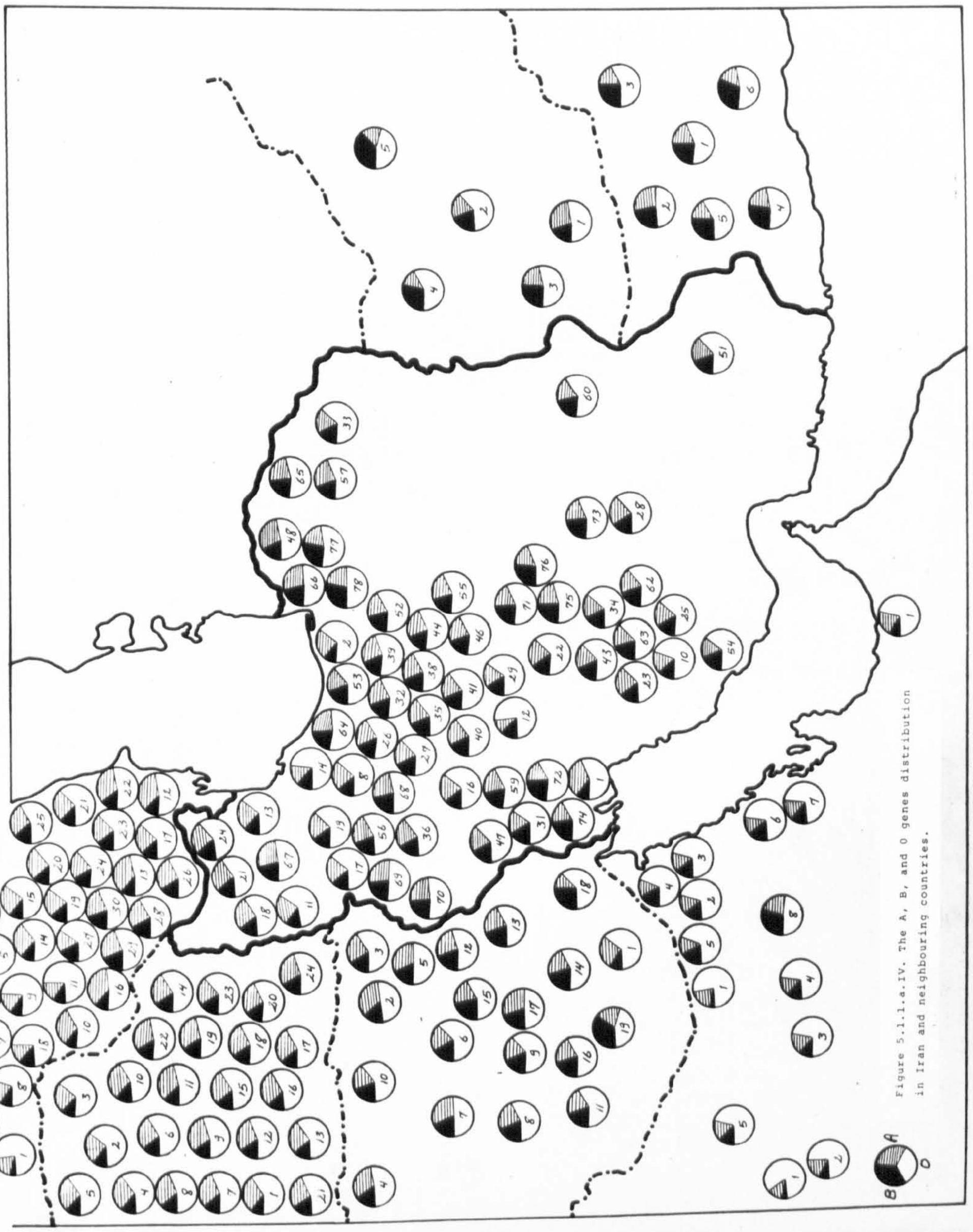


Figure 5.1.1.a.IV. The A, B, and O genes distribution in Iran and neighbouring countries.

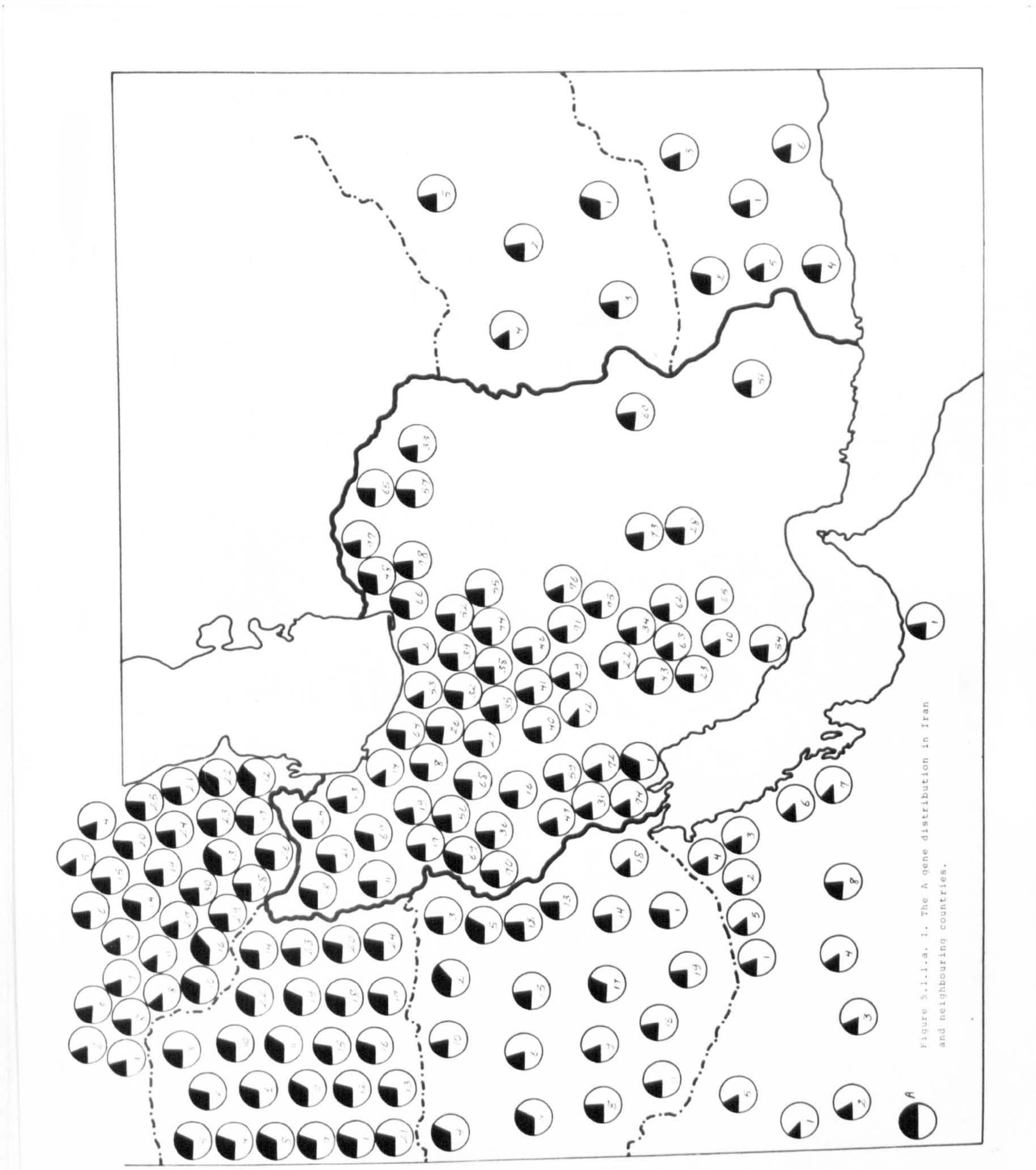


Figure 5.1.1.a. 1. The A gene distribution in Iran and neighbouring countries.

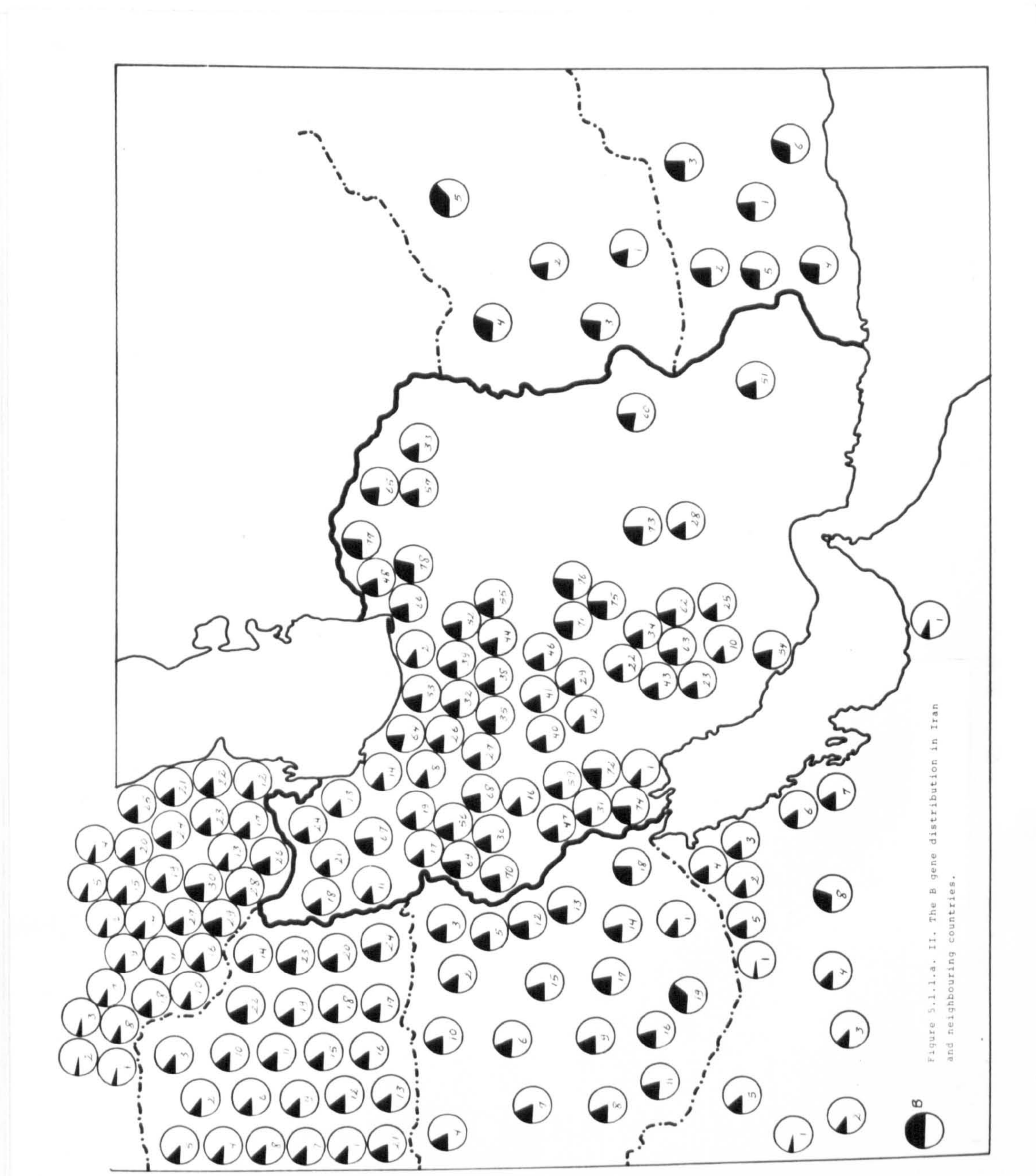


Figure 5.1.1.a. II. The B gene distribution in Iran and neighbouring countries.

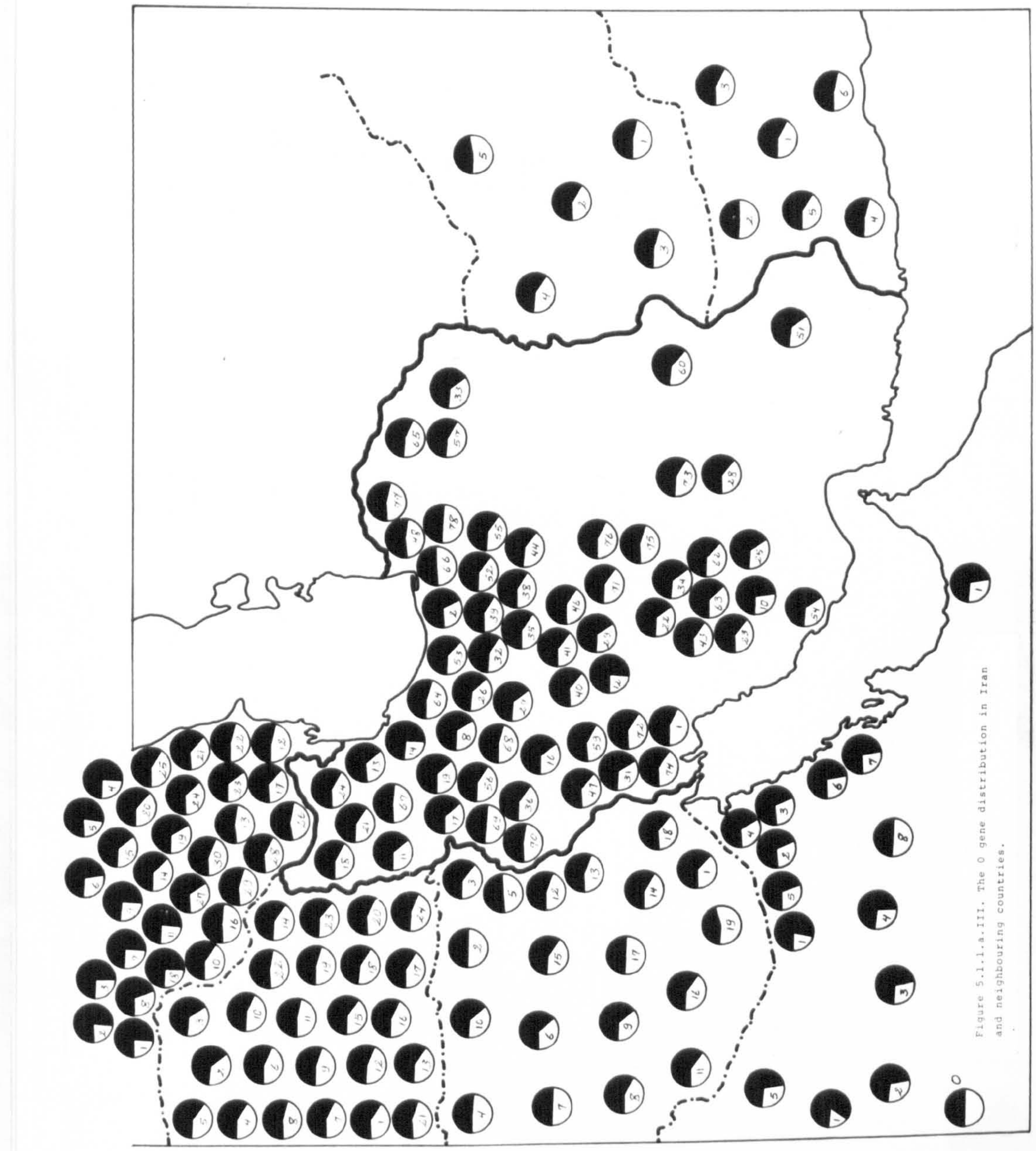


Figure 5.1.1.a.III. The O gene distribution in Iran and neighbouring countries.

Table 5.1.1.b.I ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-A1 sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		A1	A2	B	A1B	A2B	0	A1	A2	B	0	
1-Baluchis.Sistan & Baluchistan	111	32	-	30	5	1	43	18.50	0.56	17.89	63.05	Present study.
2-Zabolis. Sistan & Baluchistan	116	28	1	26	13	1	47	19.30	1.04	18.73	60.93	Present study..
3-Southern Goran,Sari, Behshahr	53	18	-	7	2	1	25	21.10	1.20	9.90	67.80	Kirk,R.L.,et al. 1977
4-Tavalesh,Astara	49	18	-	10	-	1	20	20.80	1.30	12.10	65.70	Kirk,R.L.,et al. 1977
5-Bakhtiaris	138	25	3	30	1	-	79	9.99	1.32	12.01	76.77	Nijenhuis,L.E. 1964
6-Shahsavar,Rudsar,Langa-rud,Lahijan,Bandar-Pah-Pahlvi,Rudbar,Rasht	87	16	2	15	4	1	49	12.20	1.90	12.20	73.70	Kirk,R.L.,et al. 1977
7-Armenians	145	68	3	22	8	2	42	33.30	1.90	11.00	53.80	Tabatabai,H. 1977
8-North West	76	14	2	23	2	1	34	11.23	2.28	18.95	67.54	Bajatzadeh,M.,& Walter, H. 1969
9-Babol, Shahi,Amol	70	18	3	19	4	-	26	17.20	2.70	18.20	61.90	Kirk,R.L.,et al. 1977
10-Esfahan	111	25	4	25	8	3	46	15.42	2.81	15.95	65.82	Sawhney,K.S. 1975
11-Tehran	115	41	5	22	3	-	44	21.63	2.89	11.66	63.82	Bajatzadeh,M.,& Walter, H. 1969

Table 5.1.1.b.I(Cont)

ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-A1 sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors	
		A1	A2	B	A1B	A2B	0	A1	A2	B	0		
12-South-east	348	78	13	86	19	4	148	15.07	2.92	17.08	64.93	Nijenhuis,L.E.	1964
13-Zoroastrians	88	17	3	25	8	2	33	15.25	3.35	22.12	59.28	Present study.	
14-Jews	108	39	4	29	4	2	30	27.20	3.40	16.70	52.70	Tabatabai,H.	1977
15-Gonbad	156	57	6	37	10	2	44	24.70	3.60	17.30	54.40	Kirk,R.L.,et al.	1977
16-Tehran	94	21	5	17	5	-	46	13.94	3.73	11.99	70.34	Sawhney,K.S.	1975
17-Ghashghais	66	19	4	14	4	-	25	19.27	3.86	14.75	62.12	Nijenhuis,L.E.	1964
18-Moslems.Yazd	258	45	12	90	19	10	82	13.13	4.01	25.44	57.41	Boue' and Boue'.	1956
19-Tehran	1239	351	61	301	83	16	427	19.51	4.04	17.92	58.53	Boue' and Boue'.	1956
20-West	110	31	7	19	3	1	49	16.96	4.54	11.10	64.70	Bajatzadeh,M., & Walter, H.	1969
21-Turks.Rezaieh	92	36	5	18	3	1	29	24.38	4.55	12.94	58.13	Present study.	
22-Arabs.Abadan	158	24	9	51	11	4	59	11.72	4.74	23.50	60.04	Nijenhuis,L.E.	1964
23-Lurs.Luristan	167	47	9	31	5	4	71	17.10	4.86	12.80	65.24	Present study.	
24-Kurdish Jews	106	29	6	29	10	2	30	20.42	4.89	21.66	53.03	Tills,D.,et al.	1977
25-Northern Gorgan	43	16	3	13	2	-	9	24.30	5.00	19.90	50.80	Kirk,R.L.,et al.	1977

Table 5.1.1.b.I(Cont.) ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-A1 sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors	
		A1	A2	B	A1B	A2B	0	A1	A2	B	0		
26-Armenians	78	33	5	12	3	1	24	26.83	5.55	10.92	56.70	Nijenhuis,L.E.	1964
27-Kurds.Baneh,Marivan	77	19	6	14	4	1	33	16.21	5.58	13.17	65.04	Lehmann,H.,et al.	1973
28-Central & South	113	32	8	24	4	2	43	17.57	5.62	14.34	62.47	Bajatzadeh,M., & Walter, H.	1969
29-Kurdish Jews	94	27	7	24	11	1	24	22.63	5.66	21.39	50.32	Godber,Marilyn J., et al.	1973
30-North	73	19	6	13	2	1	32	15.68	5.93	11.66	66.73	Bajatzadeh,M., & Walter, H.	1969
31-Shi'a.Yazd	151	28.42	11.58	48	9.69	4.31	49	13.55	6.32	23.17	56.96	Sunderland,E.,& Smith,H.M.	1966
32-Assyrians.Abadan	32	12	2	2	3	1	12	26.69	6.41	9.59	57.31	Nijenhuis,L.E.	1964
33-Arabs.Khuzestan	97							14.70	6.50	18.60	60.20	Marzban,M.	1978
34-Kurds.Rezaieh	138	38	12	24	3	4	57	16.26	7.28	11.97	64.49	Present study.	
35-East	78	31	7	13	3	1	23	25.09	7.33	11.66	55.92	Bajatzadeh,M., & Walter, H.	1969

Table 5.1.1.b.I (Cont.) ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-A1 sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		A1	A2	B	A1B	A2B	O	A1	A2	B	O	
36-Kurds.Kermanshah	127	29	12	28	7	4	47	15.36	7.76	16.66	60.22	Nijenhuis,L.E. 1964
37-Kurds.Sanandaj	107	23	10	25	7	4	38	15.11	8.01	18.41	58.47	Lehmann,H.,et al. 1973

Table 5.1.1.b.II ABO blood groups distribution in the Caucasus tested with anti-A, anti-B, and anti-Al sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		A1	A2	B	A1B	A2B	0	A1	A2	B	0	
1-Svanis . Georgia.Abkhazskaya	358	119	12	36	11	3	177	20.18	2.66	7.23	69.93	Verbitsky,M.Sh., 1971 et al.

Table 5.1.1.b.III ABO blood groups distribution in Turkey tested with anti-A, anti-B, and anti-Al sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		A1	A2	B	A1B	A2B	0	A1	A2	B	0	
1-Turks.Asia Minor,Mersin	108	34	9	20	8	2	35	21.77	6.77	14.94	56.52	Aksoy,M.,et al. 1958
2-Eti-Turks.Asia Minor,near Mersin	118	43	12	14	4	2	43	22.49	8.10	8.89	60.55	Aksoy,M.,et al. 1958

Table 5.1.1.b.IV ABO blood groups distribution in Iraq tested with anti-A, anti-B, and anti-A1 sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		A1	A2	B	A1B	A2B	0	A1	A2	B	0	
1-Kurdish Jews.South east	50	8	-	19	2	1	20	10.61	1.12	25.17	63.10	Tills,D.,et al. 1977
2-Kurdish Jews	27	10	-	8	-	1	8	21.31	2.50	18.74	57.45	Godber,Marilyn J., et al. 1973
3-Assyrians	99	23	4	25	7	1	39	16.47	3.06	18.26	62.21	Ikin,Elizabeth W., et al. 1965
4- Kurdis Jews.North west	61	23	4	12	4	2	16	25.45	6.97	16.08	51.50	Tills ,D.,et al. 1977
5-Karaite Jews	72	4	18	34	2	4	10	4.26	18.27	34.58	42.89	Goldschmidt,E., et al. 1976

Table 5.1.1.b V ABO blood groups distribution in Kuwait tested with anti-A, anti-B, and anti-Al sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		Al	A2	B	A1B	A2B	O	Al	A2	B	O	
1-Kuwaiti Arabs	162	40	5	32	2	2	81	15.18	2.13	12.68	70.01	Sawhney, K.S. 1975
2-General population	74	13	6	17	-	2	36	9.00	6.00	14.00	71.00	Khaled,E.,et al. 1981
3-Suluba tribe	52	5	7	14	-	2	24	5.00	9.00	17.00	69.00	Khaled,E.,et al. 1981
4-Ajman tribe	52	7	10	3	-	1	31	7.00	11.00	4.00	78.00	Khaled,E.,et al. 1981

Table 5.1.1.b. VI

ABO blood groups distribution in Saudi Arabia tested with anti-A, anti-B, and anti-A1 sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors	
		A1	A2	B	A1B	A2B	0	A1	A2	B	0		
1-Shias.Qatif & Hasa Oases	465	66	22	112	9	8	248	8.45	3.60	14.98	72.97	Maranjian,G., et al.	1966
2-Sunnis.Qatif & Hasa Oases	323	54	19	74	5	2	169	9.65	3.71	13.49	73.15	Maranjian,G., et al.	1966
3- Western Saudi Arabia	210	37	17	41	7	1	107	10.72	5.46	12.57	71.38	Saha,N.,et al.	1980
4-Sunnis.Najd	180	26	16	35	2	4	97	8.15	6.29	12.12	73.44	Maranjian,G., et al.	1966
5-Sunnis.Asir,Najran	74	5	9	4	1	1	54	4.14	7.28	4.12	84.46	Maranjian,G., et al.	1966
6-Bedouins	178	30	19	28	3	4	94	9.78	7.47	10.35	72.40	Maranjian,G., et al.	1966
7-Sunnis. Hejaz	102	13	14	14	3	1	57	8.16	8.34	9.22	74.28	Maranjian,G., et al.	1966

Table 5.1.1.b. VIII ABO blood groups distribution in Pakistan tested with anti-A, anti-B, and anti-A1 sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		A1	A2	B	A1B	A2B	O	A1	A2	B	O	
1-Hunza.Gilgit	23	5	-	8	1	1	8	14.18	2.59	24.77	58.44	Ikin,Elizabeth W., et al. 1959
2-Pathans.Swat, Saidu Sharif	133	27	4	42	14	4	42	16.71	3.61	25.54	54.14	Alciati, G. 1968
3-Baltis.Baltistan	60	2	21	9	3	-	25	24.18	4.19	23.10	48.53	Clegg,E.J., et al. 1961

Table 5.1.1.b. IX

ABO blood groups distribtution in Afghanistan tested with anti-A, anti-B, and anti-Al sera

Population	Number Tested	Phenotypes						Gene frequencies				Authors	
		A1	A2	B	A1B	A2B	O	A1	A2	B	O		
1-Timuri & related tribes	118	26	4	38	11	3	36	17.11	3.62	25.02	54.25	Woodd-Walker, R. B., et al.	1967
2- Daris	179	42	9	48	12	2	66	16.10	4.00	19.10	60.80	Papiha, S.S., et al.	1977
3- Pushtus	104	32	10	24	6	1	31	20.60	8.00	17.80	53.60	Papiha, S.S., et al.	1977

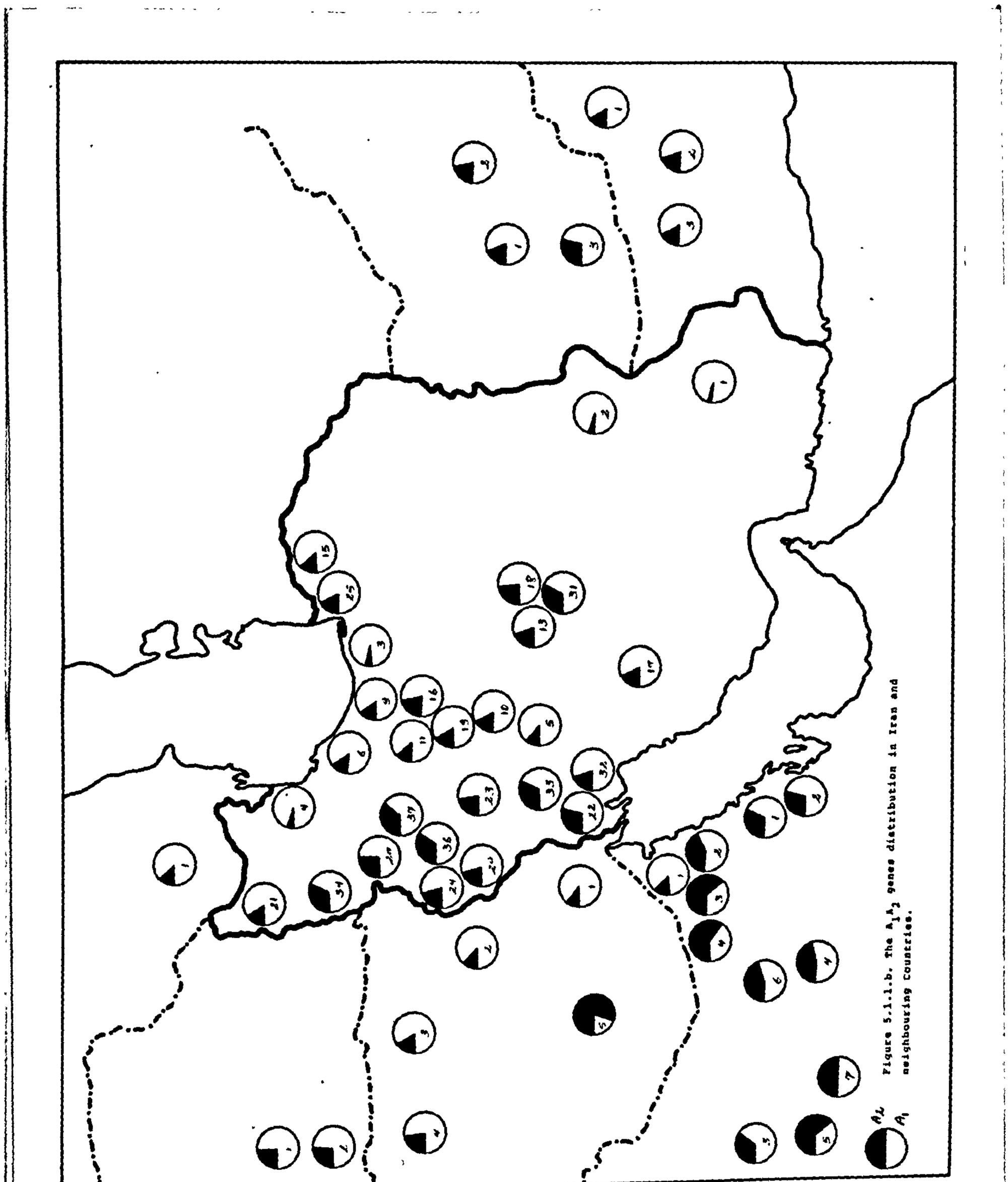


Figure 5.1.1.b. The A_1A_2 genes distribution in Iran and neighbouring Countries.

5.1.2. The Rhesus blood group system

The distribution of the Rhesus blood groups and respective gene frequencies in Iranian and neighbouring populations, expressed in term of two phenotypes after testing with one antiserum, is shown in Tables 5.1.2.a.

The frequency of the d gene ranges from 12.17 to 48.20 percent in Iranians, being lowest in the Kurdish Jews of western Iran (Gurevitch et al, 1947) and highest in the Gilani series (Shahsavar, Rudsar, Rasht, Langarud, Lahijan, Rudbar, Bandar-Pahlavi) of Kirk et al (1977). Values obtained in the present investigation are within this range of variation.
On the whole, with an average d gene frequency of 30.08 percent, the Iranian population appears to exhibit a much lower d gene frequency than that of around 40 percent found in Europeans (Mourant et al, 1976).
It seems that some differences in the distribution of the d gene frequencies exist in Iran as with the exception of the Kurdish Jews and the Arabs of Khuzistan, the populations of western Iran appear to exhibit higher d gene frequencies than those found in the populations of eastern Iran. In the present investigation also two population groups, the Kurds and the Turks, of Rezaieh(north west) show higher d gene frequencies of 39.92 and 34.73 percent compared with those of 26.53 and 22.36 percent in the population groups of Shirvan, Khorasan (north east). The d gene frequency of 26.53 percent obtained in the present investigation in the Kurds of Shirvan is also lower than all d gene frequencies reported in various studies in the Kurds of western Iran.

The d gene frequency seems also to decrease from north to south in Iran, in addition to its decreasing frequency from west to east.

The Kurds of western Iran like most other populations of the region exhibit high frequencies of the gene d but the Kurdish Jews like the Jews themselves have low frequencies of this gene.

The Arabs of south western Iran, unlike the other population groups in western Iran, also seem to exhibit low frequencies of the gene d.

The Turkmans of north eastern Iran appear to be characterized by low frequencies of the gene d, suggesting Mongoloid features.

High frequencies of the gene d seem to be characteristics of the Armenians and the Assyrians of Iran.

Regarding neighbouring populations, the frequency of the gene d in the populations of the Caucasus ranges from 21.49 percent in the Azerbaijanians of Barda (Voronov, 1973) to 41.50 percent in the Goris of Georgia(Solovyeva, 1967). With an average d gene frequency of 31.81 percent, the population of the Caucasus appears to exhibit a higher d gene frequency than that found in Iranians but lower than the European frequency.

The d gene frequency in Turkish populations varies between 29.68 percent in the Mediterranean region(Mizan et al , 1963) and 35.55 percent in the Turkish sample of Paidoussis (1967). With an average d gene frequency of 32.61 percent, the population of Turkey, like that of the Caucasus, seems to show a higher d gene frequency than that found in Iranians but still lower than the European frequency.

The frequency of the gene d in the Iraqi populations

ranges from 14.14 percent in the Kurdish Jews of the south east (Tills et al, 1977) to 52.70 percent in the Karaite Jews (Goldschmidt et al, 1976). With an average d gene frequency of 30.13 percent, the population of Iraq appears to exhibit nearly similar d gene frequencies to those found in Iranians and lower than the European frequencies.

The extremely high d gene frequency of 52.70 percent reported by Goldschmidt et al (1976) in the Karaite Jews of Iraq, clearly indicates the strong influence of isolation and genetic drift operating in this community.

On the whole the Kurdish Jews and the Jewish populations of Iraq seem to show higher d gene frequencies compared with those found in the same two population groups of Iran.

The d gene frequency in the populations of Kuwait varies between 19.61 percent in the Suluba tribe and 39.22 percent in the Ajman tribe (Khaled et al, 1981). With an average d gene frequency of 31.34 percent, the Kuwaiti population appears to exhibit a higher d gene frequency than that found in Iranians though lower than the European frequency.

The frequency of the d gene in Saudi Arabians ranges from 26.68 percent in the Sunni sample of Qatif and Hasa Oases to 31.98 percent in the Sunni sample of Asir, Hejaz, Najran(Maranjian et al, 1966). With an average d gene frequency of 29.28 percent, the Arab population of Saudi Arabia seems to show a slightly lower d gene frequency than that found in Iranians and lower than the European frequency.

The d gene frequency of 27.44 percent reported by Kamel et al (1980) in the Abu-Dhabians of the United Arab Emirates is also lower than that found in Iranians and lower than the European frequency.

The frequency of the d gene in the populations of Pakistan ranges from 20.85 percent in the Hunza, Gilgit (Ikin et al, 1959) to 33.12 percent in Peshawar (Boyd and Boyd, 1954).With an average d gene frequency of 27.82 percent, the population of Pakistan appears to exhibit a lower d gene frequency than Iranians and lower than the European frequency.

The d gene frequency in the Afghan populations varies between 18.41 percent in the Timuri and related tribes (Woodd-Walker et al, 1967) and 35.06 percent in the Daris (Papiha et al,1977). With an average d gene frequency of 26.91 percent, the population of Afghanistan, like that of Pakistan, seems to show a lower d gene frequency than that found in Iranians and lower than the European frequency.

The distribution of Rh types and respective gene complex frequencies in Iranian and neighbouring populations, expressed in terms of 8, 12, 18, ... phenotypes after testing with 3,4,5,.....antisera respectively, is presented in Tables 5.1.2. b, c, d, ...

The gene complex frequencies were combined in Tables 5.1.2.* for comparative purposes.

The frequencies of the principal Rh complexes in the Iranian populations are as follows:

CDe(R_1) ranges from 36.50 percent in a sample from north of Iran (Bajatzadeh and Walter, 1969) to 60.50 percent in the Jews (Gurevitch et al, 1956).

cde (r) varies between 17.02 percent in the Kurdish Jews (Godber et al, 1973) and 38.90 percent in the Armenians (Nijenhuis, 1964).

cDE(R_2) ranges from 4.00 percent in a sample from east of Iran (Bajatzadeh and Walter, 1969) to 25.56 percent in the Ma-

massani tribe of Fars (Bowman, 1969).

cDe(Ro) varies between zero in the Kurdish Jews (Godber et al, 1973, and Tills et al, 1977); in the Zabolis of Sistan and Baluchistan (present study); and in the Ghashghais (Nijenhuis, 1964), and 20.58 percent in the Arabs of Khuzistan (Marzban, 1978).

Furthermore, the frequencies of the rare gene complexes do not show much variation. Values obtained in the present investigation are within this range of variation.

The approximate frequencies of Rh gene complexes in the English population are as follows:

Gene complex	% frequency
CDe(R_1)	41
cde(r)	39
cDE(R_2)	14
cDe(Ro)	3
$C^wDe(R_1^w)$	1
Cde(r')	1
cdE(r")	1
CDE(R_Z)	0.2

Most northern and central European populations differ only slightly (Mourant et al, 1976).

On the whole, with average Rh gene complex frequencies of:

Gene complex	% frequencies
CDe(R_1)	48
cde(r)	28
cDE(R_2)	14
cDe(Ro)	6
Cde(r')	3
CDE(R_z)	1
cdE(r")	0.57

the Iranian population appears to exhibit much lower cde (r) but higher CDe(R_1) and cDe(Ro) gene complex frequencies than those found in northern European populations. The Rh gene complex frequencies in Iranians seem to be similar to those found in southern Europe and the Mediterranean region.

The Kurdish Jews of western Iran are characterized by the relatively low frequency of the gene complex cde(r), but high frequencies of both the CDe(R_1) and cDE(R_2) gene complexes.

The Iranian Jews appear to exhibit low frequencies of both the gene complex cde(r) and cDE(R_2), but very high CDe(R_1) frequency.

The Arabs of south western Iran seem to show low frequencies of both the gene complexes cde(r) and cDE(R_2), but high frequencies of CDe(R_1) and African cDe(Ro).

The Turkmans of north eastern Iran appear to be characterized by low frequency of the gene complex cde(r), but a high CDe(R_1) frequency, suggesting Mongoloid features.

High frequencies of both the gene complexes cde(r) and cDE (R_2), but a low frequency of CDe(R_1) seem to be characteristics of the Armenians and the Assyrians of Iran.

Regarding neighbouring populations, the frequencies of the principal Rh complexes in the populations of the Caucasus are as follows:

CDe(R_1) ranges from 34.76 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al,1972) to 46.30 percent in the

Georgians (Kherumian et al, 1954).

cde (r) varies between 34.09 percent in the Georgians (Kherumian et al, 1954) and 35.61 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1972).

cDE(R_2)ranges from 15.61 percent in the Armenians (Kherumian et al, 1954) to 17.73 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1972).

cDe(Ro) varies between 3.25 percent in the Georgians and 4.82 percent in the Armenians(Kherumian et al, 1954). With average Rh gene complex frequencies of :

Gene complex	% Frequency
CDe(R_1)	41
cde(r)	35
cDE(R_2)	17
cDe(Ro)	4
Cde(r')	2
CDE(R_Z)	1
cdE(r")	0.26

the population of the Caucasus seems to exhibit higher cde(r) but lower CDe(R_1) and cDe(Ro) gene complex frequencies than those found in Iranians and more similar to the European frequencies.

The frequencies of the principal Rh complexes in the populations of Turkey(Aksoy et al, 1958) are as follows:

CDe(R_1) ranges from 48.18 percent in the Turks to 54.66 percent in the Eti-Turks.

cde(r) varies between 30.85 percent in the Eti-Turks and 32.00 percent in the Turks.

cDE(R_2) ranges from 13.14 percent in the Eti-Turks to 17.14 percent in the Turks.

cDe(Ro) varies between 1.30 percent in the Turks and 1.35 percent in the Eti-Turks.

With average Rh gene complex frequencies of:

Gene complex	%Frequency
CDe(R_1)	51
cde(r)	32
cDE(R_2)	15
cDe(Ro)	1
Cde(r')	1
CDE(R_Z)	0
cdE(r")	0

the Turkish population appears to exhibit higher frequencies of both cde(r) and CDe(R_1) gene complexes compared with those found in Iranians, but surprisingly much lower African cDe(Ro) values which are relatively common in Iranian and in nearly every other Mediterranean populations.

The frequencies of the principal Rh complexes in the populations of Iraq are as follows:

CDe (R_1) ranges from 29.93 percent in the Karaite Jews (Goldschmidt et al, 1976) to 64.51 percent in the Kurdish Jews of the south east (Tills et al, 1977).

cde(r) varies between 7.49 percent in the Kurdish Jews of the south east (Tills et al, 1977) and 52.70 percent in the Karaite Jews (Goldschmidt et al, 1976).

cDE(R_2) ranges from 3.47 percent in the Karaite Jews (Goldschmidt et al, 1976) to 20.51 percent in the Kurdish Jews of the south east (Tills et al, 1977).

cDe(Ro) varies between zero in the Kurdish Jews (Godber et al, 1973; and Tills et al, 1977) and 20.90 percent in the Karaite Jews (Goldschmidt et al, 1976). With average Rh gene complex frequencies of :

Gene complex	%Frequency
CDe(R_1)	48
cde(r)	27
cDE(R_2)	15
cDe(Ro)	6
Cde(r')	1
CDE(R_Z)	3
cdE(r")	0.30

the population of Iraq seems to exhibit Rh gene complex frequencies similar to those found in Iranians.

The very high frequency of the so called "African gene",cDe (20.90 percent) in the Karaite Jews of Iraq is notable, as is the frequency of cde (52.70 percent). This results clearly indicate the strong influence of isolation and genetic drift operating in this community.

The frequencies of the principal Rh complexes in the populations of Kuwait are as follows:

CDe(R_1) ranges from 30.00 percent in the Ajman tribe (Khaled et al, 1981) to 49.20 percent in the Kuwaiti Arabs (Sawhney, 1975).

cde(r) varies between 15.00 percent in the Suluba tribe and 37.00 percent in the Ajman tribe (Khaled et al, 1981).

cDE (R_2) ranges from 5.04 percent in the Kuwaiti Arabs(Sawhney, 1975) to 33.00 percent in the Suluba tribe (Khaled et al, 1981).

cDe(Ro) varies between 3.00 percent in the Suluba tribe(Khaled et al, 1981) and 14.42 percent in the Kuwaiti sample of Onsi et al (1969). With average Rh gene complex frequencies of:

Gene complex	% Frequency
CDe(R_1)	41
cde (r)	28
cDE (R_2)	18
cDe (Ro)	9
Cde (r')	2
CDE (R_z)	1
cdE (r")	1

the population of Kuwait appears to exhibit lower CDe(R_1) but higher African cDe(Ro) frequencies than those found in Iranians.

The frequencies of the principal Rh complexes in the po-

pulations of Saudi Arabia are as follows:

CDe(R_1) ranges from 38.46 percent in the Sunni sample of Qatif and Hasa Oases to 42.46 percent in the Sunni sample of Asir, Hejaz, Najran (Maranjian et al, 1966).

cde (r) varies between 25.02 percent in Saudi Arabians and 28.74 percent in the Sunni sample of Asir, Hejaz, Najran (Maranjian et al, 1966).

cDE(R_2) ranges from 9.96 percent in western Saudi Arabia(Saha et al, 1980) to 16.72 percent in the Sunni sample of Qatif and Hasa Oases (Maranjian et al, 1966).

cDe(Ro) varies between 14.77 percent in the Sunni sample of Asir, Hejaz, Najran (Maranjian et al, 1966) and 22.61 percent in western Saudi Arabia (Saha et al, 1980). With average Rh gene complex frequencies of :

Gene complex	% Frequency
CDe(R_1)	14
cde (r)	26
cDE (R_2)	13
cDe (Ro)	18
Cde (r')	1
CDE (R_Z)	0.25
cdE (r")	1

the Arab population of Saudi Arabia, like that of Kuwait,seems to exhibit lower frequencies of both CDe(R_1) and cde (r) , but much higher African cDe(Ro) frequency than those found in Iranians.

Generally, if seems that the frequency of the typical negro gene cDe(Ro) in the Arab populations is higher than in Europeans, but lower than in Africans. Rh is only one of the several systems suggesting the flow of African genes in the Arabs

of Peninsula (Mourant et al, 1976). On the whole, it appears that the gene frequency distribution of the Rh system in Arab populations is intermediate between Caucasoids and Negroids.

The frequencies of the Rh principal complexes in the populations of Pakistan are as follows:

CDe (R_1) ranges from 55.02 percent in the Baltis of west Pakistan (Clegg et al, 1961) to 66.69 percent in the Moslems of Punjab and west Pakistan (Chaudhri et al, 1952.).

cde (r) varies between 17.08 percent in Dacca, Bengal (Boyd et al,1954) and 28.26 percent in the Hunza , Gilgit(Ikin et al, 1959).

cDE (R_2) ranges from 7.71 percent in Dacca, Bengal (Boyd et al, 1954) to 21.39 percent in the Baltis of west Pakistan(Clegg et al, 1961).

cDe(Ro) varies between zero in the Hunza, Gilgit (Ikin et al, 1959) and 7.21 percent in the Punjabis of Lahore (Boyd et al, 1954). With average Rh gene complex frequencies of:

Gene complex	% Frequency
CDe (R_1)	60
cde (r)	24
cDE(R_2)	11
cDe(Ro)	3
Cde(r')	1.50
CDE(R_Z)	0.76
cdE(r'')	0.10

the population of Pakistan appears to exhibit lower frequencies of both cde (r) and cDe(Ro), but much higher CDe(R_1)frequency than those found in Iranians.

The frequencies of the Rh principal complexes in the populations of Afghanistan are as follows:

CDe(R_1) ranges from 46.10 percent in the Pushtus (Papiha et al, 1977) to 60.61 percent in the Timuri and related tribes (woodd-Walker et al, 1967).

cde (r) varies between 13.63 percent in the Timuri and related tribes (Woodd-Walker et al, 1967) and 28.60 percent in the Daris (Papiha et al, 1977).

cDE(R_2) ranges from 13.80 percent in the Daris (Papiha et al, 1977) to 24.15 percent in the Timuri and related tribes (Woodd-Walker et al, 1967).

cDe (Ro) varies between 1.61 percent in the Timuri and related tribes (Woodd-Walker et al, 1967) and 6.80 percent in the Pushtus (Papiha et al, 1977).

With average Rh gene complex frequencies of:

Gene complex	% Frequency
CDe(R_1^1)	53
cde(r)	23
cDE(R_2)	19
cDe(Ro)	4
Cde(r')	0.26
CDE(R_Z)	0.13
cdE(r")	0

the population of Afghanistan, like that of Pakistan, seems to exhibit lower frequencies of both cde(r) and cDe(Ro), but much higher CDe(R_1) frequency than those found in Iranians. The low frequency of cde(r) seems to be suggestive of some Mongoloid features in the Afghan population.

Conclusion

In northern Europe the frequency of the d gene is about 40 percent and the frequencies of the principal Rh complexes are approximately:

CDe(R_1) 40 percent; cDE(R_2) 15 percent; cde(r) 40 percent; cDe (Ro) 3 percent.

In southern Europe and the Mediterranean area generally, the frequency of cde(r) is usually much lower and that of CDe (R_1) higher than further north; and the variations between populations are greater.

South-western Asia, including most of the Indian region, shows a similar Rh distribution to the Mediterranean region but with a Marked admixture of African genes in the Arab countries.

Africa, south of Sahara Desert, shows a uniform absolute preponderance of cDe(Ro) which is unique. Here the cde(r)complex has a frequency of about 20 percent and there are lower frequencies of CDe(R_1) and cDE(R_2) (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab populations of Kuwait and Saudi Arabia with their lower CDe(R_1) and much higher African cDe(Ro), the frequencies of the total d gene and the cde(r)complex appear to be much lower but that of the CDe(R_1) complex higher than those found in Europeans. Therefore with slightly lower cde (r) and higher CDe(R_1), the frequencies of the principal Rh complexes in Iranian and neighbouring populations seem to be more similar to those in southern European and Mediterranean populations.

In general, regarding the whole area under discussion,the frequencies of the total d gene and the cde(r) complex seem to decrease from the west(Turkey) to the east (Pakistan) and that of CDe(R_1) increases.

Table 5.1.2. a I

Rh blood groups distribution in Iran tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
1-Kurdish Jews	203	200	3	87.83	12.17	Gurevitch,J.,et al.	1947
2-Turkmans	1263	1244	19	87.74	12.26	Montazami, K.	1978
3-Arabs.Khuzestan	97	94	3	82.41	17.59	Marzban, M.	1978
4-Southern Gorgan, Behshahr,Sari	53	51	2	80.60	19.40	Kirk,R.L.,et al.	1977
5-Mashhad	11240	10767	473	79.49	20.51	Afkari,A.H.	1967
6-Jews	225	214	11	77.89	22.11	Silberstein W., & Goldstein.	1958
7-Jews	200	190	10	77.64	22.36	Gurevitch,J.,et al.	1956
8-Turks.Shirvan,Khorasan	160	152	8	77.64	22.36	Present study.	
9-Kerman	317	301	16	77.53	22.47	Present study.	
10-Kurdish Jews	94	89	5	76.94	23.06	Godber, Marilyn.J., et al.	1973
11-Turkmans	292	276	16	76.59	23.41	Boue' and Boue'.	1956
12-Arabs.Abadan	158	149	9	76.13	23.87	Nijenhuis, L.E.	1964

Table 5.1.2.a. I (Cont.) ^{Rh} blood groups distribution in Iran tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		D+	D-	D	d	
13-Zoroastrians. Yazd	182	171	11	75.42	24.58	Boue' and Boue'. 1956
14-Lurs.Luristan	162	152	10	75.16	24.84	Present study.
15-Gonbad	143	134	9	74.90	25.10	Kirk,R.L., et al. 1977
16-Baluchis.Sistan & Baluchistan	111	104	7	74.89	25.11	Present study.
17-Bakhtiaris	138	129	9	74.46	25.54	Nijenhuis, L.E. 1964
18-Shahsavan.Azari Dashte Moghan	137	128	9	74.37	25.63	Boue' and Boue'. 1956
19-Kurdish Jews	106	99	7	74.30	25.70	Tills, D., et al. 1977
20-Tehran	992	925	67	74.01	25.99	Roshan, G.,et al. 1969
21-Gorgan	133	124	9	73.99	26.01	Boue' and Boue'. 1956
22-Ghashghais.Abadeh	1516	1412	104	73.81	26.19	Montazami,K. 1978
23-Khuzestan	2043	1902	141	73.73	26.27	Montazami, K. 1978
24-Moslems. Yazd	145	135	10	73.73	26.27	Boue' and Boue'. 1956
25-Bassaris.Fars	101	94	7	73.67	26.33	Bowman, J.E. 1959

Table 5.1.2.a.I (Cont.)

Rh blood groups distribution in Iran tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
26-Kurds.Shirvan,Khorasan	142	132	10	73.47	26.53	Present study.	
27-Mamassanis.Fars	113	105	8	73.39	26.61	Bowman, J.E.	1959
28-Moslems.Fars	768	712	56	73.00	27.00	Bowman,J.E., & Ronaghy, H.	1967
29-Mobarakeh.Esfahan	1211	1121	90	72.74	27.26	Montazami, K.	1978
30-Kurds.Sanandaj	107	99	8	72.66	27.34	Lehmann, H.,et al.	1973
31-Shiraz	1171	1083	88	72.59	27.41	Tahmasbian,A.	1972
32-Gilan.Turkish speakers	198	183	15	72.48	27.52	Boue' and Boue'.	1956
33-Tehran	1196	1103	93	72.12	27.88	Defai, H.	1971
34-Kurds.Baneh,Marivan	77	71	6	72.09	27.91	Lehmann,H.,et al.	1973
35-Esfahan	4385	4031	354	71.59	28.41	Montazami, K.	1978
36-Tehran	500	457	43	70.67	29.33	Boue' and Boue'.	1956
37-Shiraz	16368	14942	1426	70.49	29.51	Mohallatee,E.A., & Haghshenas.	1969

Table 5.1.2.a.I(Cont.) Rh blood groups distribution in Iran tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
38-North west	68	62	6	70.30	29.70	Bajatzadeh,M., & Walter, H.	1969
39-Azarbaijan	1467	1326	131	70.12	29.88	Montazami.K.	1978
40-Jews	108	98	10	69.57	30.43	Tabatabai, H.	1977
41-Moslems.Tehran	203	184	19	69.41	30.59	Boue' and Boue'.	1956
42-South east	348	315	33	69.21	30.79	Njenhuis, L.E.	1964
43-Zabolis.Sistan & Baluchistan	115	104	11	69.07	30.93	Present study.	
44-Babol,Shahi,Amol	70	63	7	68.40	31.60	Kirk,R.L.,et al.	1977
45-Esfahan	9753	8779	974	68.40	31.60	Mirdamadi,M.,et al.	1978
46-Tehran	20000	18000	2000	68.38	31.62	Vazin, H.	1969
47-Kurds.Sanandaj	129	116	13	68.26	31.74	Boue' and Boue'.	1955
48-Tavalesh.Astara	49	44	5	68.10	31.90	Kirk, R.L.,et al.	1977
49-Tehran	150000	134640	15360	68.00	32.00	Tehranchian ..	1963

Table 5.1.2.a.I (Cont.) Rh blood groups distribution in Iran tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
50-Assyrians.Abadan	32	29	3	67.84	32.16	Nijenhuis,L.E.	1964
51-Hamadan,Akinlou Turkish Speakers	115	103	12	67.70	32.30	Boue' and Boue'.	1956
52-Ghashghais	66	59	7	67.43	32.57	Nijenhuis,L.E.	1964
53-Tehran	42330	37631	4699	66.68	33.32	Berelian-Jahanshahi,F.	1973
54-Tehran	302	268	34	66.40	33.60	Present study.	
55-Turks.Rezaieh	141	124	17	65.27	34.73	Present study.	
56-Shi'a Yazd	270	237	33	65.04	34.96	Sunderland,E., & Smith,H.M.	1966
57-Tehran	196	172	24	65.01	34.99	Sawhney, K.S.	1975
58-Zoroastrians	88	77	11	64.64	35.36	Present study.	
59-Kurds.Kermanshah	127	111	16	64.51	35.49	Nijenhuis, L.E.	1964
60-Esfahan	110	96	14	64.33	35.67	Sawhney,K.S.	1975
61-Kurds.Mahabad	125	108	17	63.12	36.88	Boue' and Boue'.	1955

Table 5.1.2.a I.(Cont.) Rh blood groups distribution in Iran tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
62-Northern Gorgan	43	37	6	62.60	37.40	Kirk, R.L.,et al.	1977
63-East	70	60	10	62.20	37.80	Bajatzadeh,M., & Walter, H.	1969
64-Central & South	109	93	16	61.69	38.31	Bajatzadeh,M., & Walter, H.	1969
65-Armenians	144	122	22	60.91	39.09	Tabatabai, H.	1977
66-Kurds.Rezaieh	138	116	22	60.08	39.92	Present study.	
67-Armenians.Esfahan	144	119	25	58.33	41.67	Bowman, J.E.	1959
68-Tehran	113	93	20	57.93	42.07	Bajatzadeh,M., & Walter, H.	1969
69-Armenians	78	64	14	57.63	42.37	Nijenhuis, L.E.	1964
70-West	107	86	21	55.70	44.30	Bajatzadeh,M., & Walter H.	1969
71-Tehran	1432	1145	287	55.23	44.77	Sadatzadeh,H., Hamidi, A.H.	1972

Table 5.1.2.a.I (Cont.) Rh blood groups distribution in Iran tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
72-North	63	49	14	52.86	47.14	Bajatzadeh,M., & Walter, H.	1969
73- Shahsavar, Rudsar, Rudbar, Rasht, Langarud, Lahijan,Bandar Pahlavi	86	66	20	51.80	48.20	Kirk,R.L., et al.	1977

Table 5.1.2.a. II Rh blood groups distribution in the Caucasus tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
1-Azerbaijanians.Barda	136	130	6	78.51	21.49	Voronov, A.A.	1973
2-Kakhetians.Gurgaani	195	184	11	76.25	23.75	Voronov, A.A.	1973
3-Georgia, Gurdzhaani. Autochthonous	699	653	46	74.35	25.65	Voronov, A.A.,& Potapov, M.I..	1967
4-Azerbaijanians Shemakha	176	162	14	71.79	28.21	Voronov, A.A.	1973
5-Azerbaijanians.Nukha	191	175	16	71.06	28.94	Voronov, A.A.	1973
6-Megrelians.Mikha-Tsakai	121	108	13	67.23	32.77	Voronov, A.A.	1973
7-Georgian abroad.Autochthonous	40	35	5	64.65	35.35	Kherumian,R.,et al.	1954
8-Georgians.Different region	802	695	107	63.48	36.52	Vaisblit,R.I.,et al.	1965
9-Armenians.Autochthonous	199	171	28	62.49	37.51	Kherumian,R.,et al.	1954

Table 5.1.2.a. II (Cont.) Rh blood groups distribution in the Caucasus tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		D+	D-	D	d	
10-Svanis. Georgia.Abkhazskaya	643	549	94	61.77	38.23	Verbitsky,M.Sh., et al. 1972
11-Goris.Georgia	331	274	57	58.50	41.50	Solovyeva,T.G. 1967

Table 5.1.2.a.III

Rh blood groups distribution in Turkey tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
1-Mediterranean region	3122	2847	275	70.32	29.68	Mizan,N., et al.	1963
2-Asia Minor.Aegean region	3166	2875	291	69.68	30.32	Mizan,N., et al.	1963
3-Eti-Turks.Asia Minor.near Mersin	118	107	11	69.47	30.53	Aksoy,M., et al.	1958
4-Western part	4096	3711	385	69.34	30.66	Mizan,N., et al.	1963
5-Anatolia,South-Eastern	5200	4695	505	68.84	31.16	Mizan,N., et al.	1963
6-Middle part	5453	4890	563	67.88	32.12	Mizan,N., et al.	1963
7-Marmar and Thrace	8430	7517	913	67.09	32.91	Mizan,N., et al.	1963
8-Anatolia Central	7023	6255	768	66.92	33.08	Mizan,N., et al.	1963
9-Black sea region. western	3612	3202	410	66.31	33.69	Mizan,N., et al.	1963
10-Anatolia,Eastern	7598	6684	914	65.32	34.68	Mizan,N., et al.	1963
11-Turks.Asia Minor.Mersin	108	95	13	65.31	34.69	Aksoy,M., et al.	1958

Table 5.1.2.a. III (Cont.) Rh blood groups distribution in Turkey tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		D+	D-	D	d	
12-Black sea region. Eastern	5295	4650	645	65.10	34.90	Mizan, N., et al. 1963
13-Turks abroad.Recruits	364	318	46	64.45	35.55	Paidoussis, M. 1967

Table 5.1.2.a:IV Rh blood groups distribution in Iraq tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
1-Kurdish Jews. south east	50	49	1	85.86	14.14	Tills,D., et al.	1977
2-Kurdish Jews	129	124	5	80.31	19.69	Gurevitch,J., et al.	1956
3-Jews	1146	1063	83	73.09	26.91	Levence, C.	1968
4-Kurdish Jews	250	231	19	72.44	27.56	Gurevitch,J., et al.	1953
5-Jews.Baghdad	162	149	13	71.67	28.33	Gurevitch,J., et al.	1956
6-Kurdish Jews.North West	61	56	5	71.37	28.63	Tills,D., et al.	1977
7-Military donors	1067	977	90	70.97	29.03	Fakhrie, S.D., et al.	1966
8-Paid donors	4056	3697	359	70.25	29.75	Fakhrie, S.D., et al.	1966
9-Baghdadi Jews	200	181	19	69.18	30.82	Sirat,Satyavati M.	1956
10-Assyrians	99	89	10	68.22	31.78	Ikin,Elizabeth W.,et al.	1965
11-Jews	308	276	32	67.77	32.23	Silberstein,W., & Goldstein.	1958
12-Civilian donors	1185	1060	125	67.52	32.48	Fakhrie,S.D., et al.	1966

Table 5.1.2.a. IV (Cont.) Rh blood groups distribution in Iraq tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		D+	D-	D	d	
13-Kurdish Jews	27	24	3	66.67	33.33	Godber,Marilyn J., et al. 1973
14-Arabs.Baghdad	300	264	36	65.36	34.64	Kayssi, A.I. 1940
15-Karaite Jews	72	52	20	47.30	52.70	Goldschmidt,E.,et al. 1976

Table 5.1.2. a V

Rh blood groups distribution in Kuwait tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d		
1-Suluba tribe	52	50	2	80.39	19.61	Khaled, E., et al.	1981
2-Kuwaitis	2328	2154	174	72.67	27.33	Onsi,A., et al.	1969
3-General population	74	65	9	65.13	34.87	Khaled,E.,et al.	1981
4-Kuwaiti Arabs	110	96	14	64.33	35.67	Sawhney, K.S.	1975
5-Ajman tribe	52	44	8	60.78	39.22	Khaled,E., et al.	1981

Table 5.1.2.a. VI Rh blood groups distribution in Saudi Arabia tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		D+	D-	D	d	
1-Sunnis. Qatif and Hasa Oases	323	300	23	73.32	26.68	Maranjian,G.,et al. 1966
2-Saudi Arabians	355	328	27	72.42	27.58	Goedde,H.W., et al. 1979
3-Western Saudi Arabia	178	161	17	69.10	30.90	Saha,N., et al. 1981
4-Sunnis. Asir,Hejaz,Najran	176	158	18	69.02	31.98	Maranjian,G.,et al. 1966

Table 5.1.2. a VII

Rh blood groups distribution in the United Arab Emirates tested with anti -D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		D+	D-	D	d	
1-Abu-Dhabians	624	577	47	72.56	27.44	Kamel,K.,et al. 1980

Table 5.1.2. a. VIII Rh blood groups distribution in Pakistan tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		D+	D-	D	d´		
1-Hunza.Gilgit	23	22	1	79.15	20.85	Ikin,Elizabeth W., et al.	1959
2-Bengalis.Dacca	236	222	14	75.64	24.36	Boyd,W.C., & Boyd, lyle.G.	1954
3-Moslems.Borne in Punjab and West Pakistan	101	95	6	75.63	24.37	Chaudhri,I.M., et al.	1952
4- Punjabis.Lahore	203	189	14	73.74	26.26	Boyd,W.C., & Boyd, lyle.G.	1954
5-Moslems.Karachi	150	139	11	72.92	27.08	Moten,A.N., et al.	1956
6-Baltis.Baltistan	80	74	6	72.61	27.39	Clegg,E.J., et al.	1961
7-Pathans.Swat. Saidu Sharif	133	121	12	69.96	30.04	Alciati, G.	1968
8-Pathan. Dari,Timurgara	78	70	8	67.97	32.02	Bernhard, W.	1967
9-Parsis.Karachi	103	92	11	67.32	32.68	Moten,A.N.,et al.	1956
10-Peshawar	155	138	17	66.88	33.12	Boyd,W.C.,& Boyd Lyle.G.	1954

Table 5.1.2.a. IX Rh blood groups distribution in Afghanistan tested with anti-D serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		D+	D-	D	d	
1-Timuri and related tribes	118	114	4	81.59	18.41	Woodd-Walker,R.B., et al. 1967
2-Fara,Girishk	98	92	6	75.26	24.74	Boue',A., et al. 1956
3-Pushtus	104	95	9	70.58	29.42	Papiha,S.S., et al. 1977
4-Daris	179	157	22	64.94	35.06	Papiha,S.S., et al. 1977

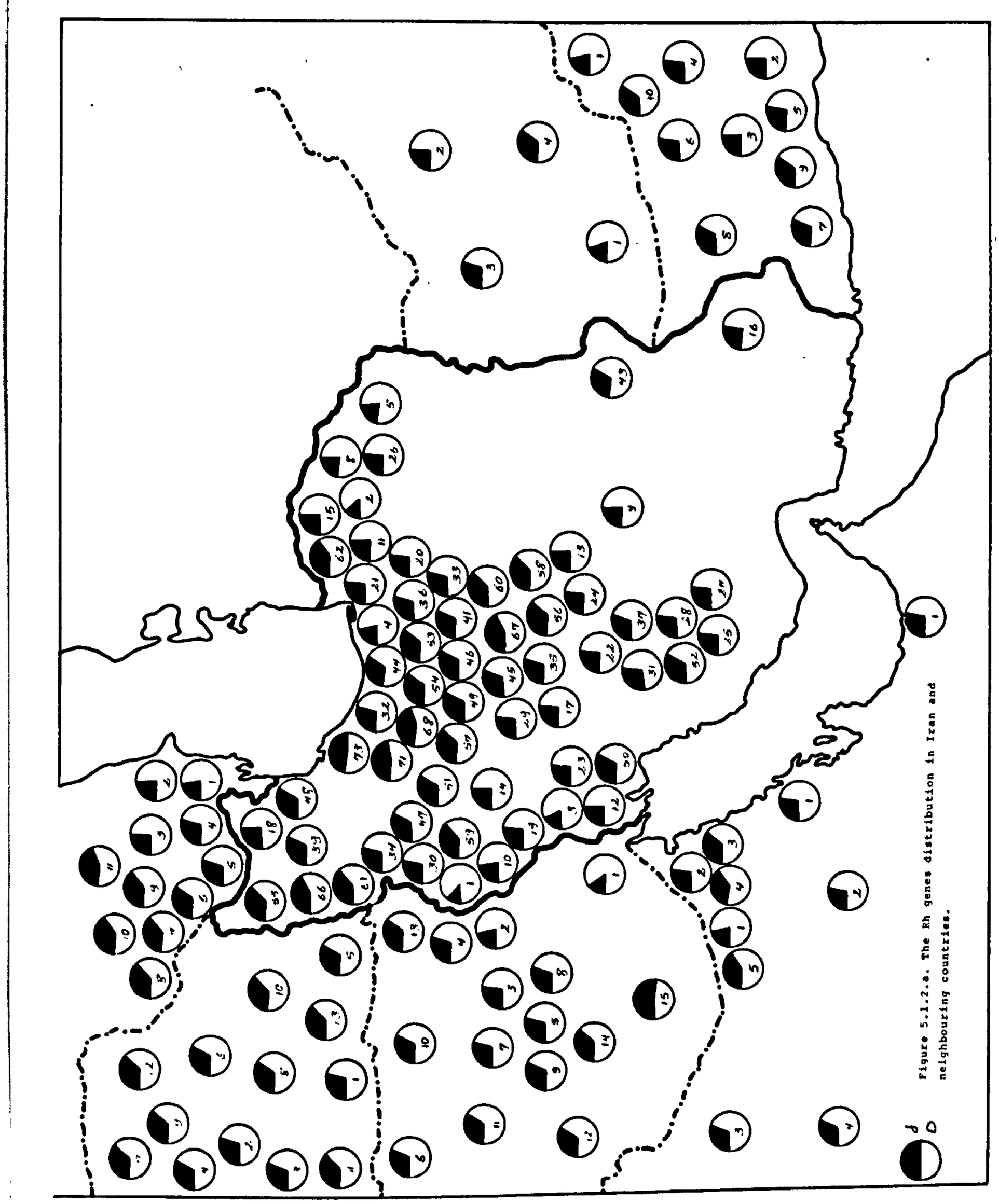


Figure 5.1.2.a. The Rh genes distribution in Iran and neighbouring countries.

Table 5.1121b.I

Rh blood groups distribution in Iran tested with anti-C, anti-D, and anti-E sera

Population	Number Tested	Phenotypes								Gene complex frequencies						Authors
		CCDEE	CCDee	CCddEE	CCddee	ccDEE	ccDee	ccddEE	ccddee	CDe	Cde	cDE	cDe	cdE	cde	
1-Zoroastrians . Yazd	182	42	111	-	2	16	2	-	9	57.79	2.61	17.15	2.14	0.00	20.31	Boue' & Boue'. 1956

Table 5.1.2.c.I

Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, and anti-c sera

Population	Number Tested	Phenotypes											
		CCDEE	CCDee	CCddEE	CCddee	CcDEE	CcDee	CcddEE	Ccddee	ccDEE	ccDee	ccddEE	ccddee
1-Turkmans. Gonbad	292	3	104	-	-	34	82	-	3	44	9	-	13
2-Gorgan	133	-	46	-	-	19	45	-	1	7	7	-	8
3-Jews	200	-	73	-	-	26	70	-	-	15	6	-	10
4-Gilan.Turkish Speakers	198	4	49	-	1	43	56	-	1	27	4	-	13
5-North West	68	1	17	-	-	10	23	-	2	7	4	-	4
6-Kurds.Sanandaj	129	3	21	-	1	26	49	-	2	9	8	-	10
7-Kurds.Mahabad	125	3	34	-	-	18	35	-	2	6	12	-	15
8-Moslems.Tehran	203	-	38	-	-	42	82	-	2	18	4	-	17
9-Tehran	113	3	24	-	-	11	36	-	6	10	9	2	12
10-North	63	1	16	-	-	1	19	-	4	6	6	3	7
11-Central and South	109	4	28	-	-	5	41	-	2	7	8	-	14
12-East	70	2	17	-	-	5	29	-	1	3	4	-	9
13-West	107	1	24	-	-	9	37	-	3	8	7	2	16

Table5.1.2.c.I.(Cont) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, and anti-c sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Turkmans. Gonbad	292	0.73	54.01	0.00	2.30	14.78	6.45	0.00	21.73	Boue' and Boue'. 1956
2-Gorgan	133	0.00	57.40	0.00	1.64	10.15	8.27	0.00	22.54	Boue' and Boue'. 1956
3-Jews	200	0.00	60.50	0.00	0.00	10.85	6.00	0.00	22.65	Gurevitch,J.,et al. 1956
4-Gilan.Turkish Speakers	198	2.04	47.81	0.00	2.70	18.75	3.80	0.00	24.90	Boue' and Boue'. 1956
5-North West	68	1.50	44.90	0.00	5.80	12.70	9.80	0.00	25.30	Bajatzadeh,M.,& Walter, H. 1969
6-Kurds.Sanandaj	129	3.92	41.07	0.00	4.27	11.97	10.42	0.00	28.35	Boue' and Boue'. 1956
7-Kurds.Mahabad	125	3.39	45.20	0.00	3.06	8.04	10.21	0.00	30.10	Boue' and Boue'. 1956
8-Moslems. Tehran	203	0.00	48.49	0.00	1.29	15.60	3.46	0.00	31.16	Boue' and Boue'. 1956
9-Tehran	113	2.70	36.90	0.00	7.70	7.20	10.50	2.20	32.70	Bajatzadeh, M., & Walter, H. 1969
10-North	63	1.30	36.50	0.00	8.20	4.20	11.50	3.70	34.60	Bajatzadeh, M., & Walter, H. 1969
11-Central and South	109	3.30	45.70	0.00	2.40	4.30	8.90	0.00	35.40	Bajatzadeh, M., & Walter, H. 1969

Table 5.1.2.c.I.(cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, and anti-c sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
12- East	70	2.90	47.30	0.00	1.90	4.00	7.30	0.00	36.50	Bajatzadeh, M.,& Walter, H. 1969
13-West	107	1.00	41.80	0.00	3.40	6.50	7.50	2.30	37.40	Bajatzadeh, M., & Walter, H. 1969

Table 5.1.2.d.I

Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Phenotypes																		
		CCDEE	CCDEe	CCDee	CCddEE	CCddEe	CCddee	CcDEE	CcDEe	CcDee	CcddEE	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Kurdish Jews	94	-	2	32	-	-	-	-	23	18	-	-	2	8	6	-	-	-	3
2-Arabs.Khuzestan	97	-	1	24	-	-	-	3	12	33	-	-	1	1	13	7	-	-	2
3-Bakhtiaris	138	-	4	40	-	-	1	1	30	31	-	1	-	1	18	4	-	1	6
4-Kurdish Jews	106	-	5	31	-	-	-	3	28	21	-	-	1	6	5	-	-	-	6
5-Kurds.Baneh,Marivan	77	-	-	21	-	-	-	-	25	15	-	-	-	2	8	-	-	-	6
6-Kurds.Sanandaj	107	-	-	39	-	-	-	-	15	28	-	-	1	3	11	3	-	-	7
7-Baluchis.Sistan & Baluchistan	111	-	-	44	-	-	-	1	18	30	-	-	1	3	7	1	-	-	6
8-Arabs.Abadan	158	-	-	44	-	-	-	-	21	59	-	-	-	6	13	6	1	1	7
9-Lurs.Luristan	162	-	-	40	-	-	-	2	30	46	-	-	-	11	20	3	-	-	10
10-Zabolis.Sistan & Baluchistan	115	-	-	40	-	-	1	2	13	32	-	-	2	8	9	-	-	-	8
11-Moslems.Fars	768	-	1	196	-	-	-	1	134	262	-	-	3	21	73	24	-	1	52
12-South east	348	-	4	87	-	-	-	-	59	114	-	-	3	7	30	14	-	1	29
13-Ghashghais	66	-	1	20	-	-	-	-	11	16	-	-	1	3	8	-	-	-	6

Table 5.1.2. d. I (Cont) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Phenotypes																		
		CCDEE	CCDEe	CCDee	CCddEE	CCddEe	CCddee	CcDEE	CcDEe	CcDee	CcddEE	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
14-Bassaris. Fars	101	-	-	17	-	-	-	-	15	46	-	-	-	4	6	6	-	-	7
15-Mamassanis. Fars	113	-	3	20	-	-	-	-	25	29	-	-	-	7	20	1	-	-	8
16-Esfahan	110	-	1	27	-	-	-	2	25	36	-	-	2	-	4	1	-	1	11
17-Assyrians. Abadan	32	-	-	7	-	-	-	-	5	9	-	-	-	-	7	1	-	-	3
18-Tehran	196	-	-	45	-	-	-	2	37	75	-	-	2	3	8	2	-	-	22
19-Kurds.Kermanshah	127	-	-	28	-	-	-	-	17	46	-	-	-	1	16	3	-	-	16
20-Shi'a.Yazd	151	-	-	31	-	-	-	1	18	54	-	-	1	2	18	5	-	-	21
21-Armenians.Esfahan	144	-	1	23	-	-	-	1	23	45	-	-	1	2	16	8	-	1	23
22-Armenians	78	-	-	13	-	-	-	-	12	24	-	-	-	3	9	3	-	-	14

Table 5.1.2.d I (Cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Gene complex frequencies								Authors	
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde		
1-Kurdish Jews	94	1.06	53.43	0.00	4.55	23.94	0.00	0.00	17.02	Godber,Marilyn. J., et al..	1973
2-Arabs.Khuzestan	97	1.04	46.28	0.00	3.59	10.15	20.58	0.00	18.36	Marzban, M.	1978
3-Bakhtiaris	138	2.00	48.73	0.00	4.62	16.48	6.97	2.17	18.93	Nijenhuis,L.E.	1964
4-Kurdish Jews	106	4.73	51.86	0.00	2.37	21.69	0.00	0.00	19.35	Tills,D., et al.	1977
5-Kurds.Baneh,Marivan	77	0.00	53.25	0.00	0.00	21.44	1.71	2.59	21.01	Lehmann,H., et al.	1973
6-Kurds.Sanandaj	107	0.93	51.07	0.00	5.00	14.02	7.20	0.00	21.78	Lehmann,H., et al.	1973
7-Baluchis.Sistan & Baluchistan	111	0.57	59.63	0.00	1.96	14.29	1.74	0.00	21.81	Present study.	
8-Arabs.Abadan	158	0.00	53.18	0.00	0.00	12.13	8.91	3.37	22.41	Nijenhuis,L.E.	1964
9-Lurs.Luristan	162	0.81	47.95	0.00	0.00	22.65	3.52	0.00	25.07	Present study.	
10-Zabolis.Sistan & Baluchistan	115	1.02	50.71	0.00	5.22	17.24	0.00	0.00	25.80	Present study.	
11-Moslems. Fars	768	0.19	50.83	0.00	0.68	16.03	5.52	0.26	26.49	Bowman,J.E.,& Ronaghy, H.	1967

Table 5.1.2.d I (Cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Gene complex frequencies								Authors	
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde		
12-South east	348	0.46	49.52	0.00	1.48	14.24	6.01	0.53	27.76	Nijenhuis,L.E.	1964
13-Ghashgais.Fars	66	0.99	49.30	0.00	2.47	18.71	0.00	0.00	28.26	Nijenhuis,L.E.	1964
14-Bassaris.Fars	101	0.00	47.10	0.00	0.00	14.39	10.24	0.00	28.27	Bowman,J.E.	1959
15-Mamassanis. Fars	113	1.88	42.39	0.00	0.00	25.56	1.72	0.00	28.54	Bowman,J.E.	1959
16-Esfahan	110	2.56	49.99	0.00	2.44	10.99	1.32	2,35	30.33	Sawhney, K.S.	1975
17-Assyrians. Abadan	32	0.00	43.80	0.00	0.00	18.76	5.01	0.00	32.43	Nijenhuis,L.E.	1964
18-Tehran	196	0.93	50.27	0.00	1.36	13.10	1.46	0.00	32.89	Sawhney, K.S.	1975
19-Kurds.Kermanshah	127	0.00	46.88	0.00	0.00	13.79	3.24	0.00	36.09	Nijenhuis,L.E.	1964
20-Shi'a.Yazd	151	0.58	43.72	0.00	0.89	13.35	4.20	0.00	37.26	Sunderland,E.,& Smith, H.M/	1966
21-Armenians.Esfahan	144	1.52	38.55	0.00	0.94	13.84	6.12	0.97	38.06	Bowman,J.E.	1959
22-Armenians	78	0.00	39.81	0.00	0.00	17.32	3.97	0.00	38.90	Nijenhuis, L.E.	1964

Table 5.1.2.e.I Rh blood groups distribtuion in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-C^W sera

Population	Number Tested	Phenotypes																								
		CCDEE	CCDEe	C^WCDEe	CCDee	C^WCDee	C^WC^WDee	CCddee	C^WCddee	C^WC^Wddee	CcDEE	CcDEe	C^WcDEe	CcDee	C^WcDee	CcddEe	C^WcddEe	Ccddee	$C^Wccddee$	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Bakhtiaris	138	-	3	1	40	-	-	1	-	-	1	30	-	31	-	1	-	-	-	1	18	4	-	1	6
2-South east	348	-	2	2	87	-	-	-	-	-	-	59	-	114	-	-	-	3	-	7	30	14	-	1	29
3-Esfahan	110	-	1	-	26	1	-	-	-	-	2	25	-	36	-	-	-	2	-	-	4	1	-	1	11
4-Tehran	196	-	-	-	42	3	-	-	-	-	2	35	2	72	3	-	-	2	-	3	8	2	-	-	22
5-Kurds.Kermanshah	127	-	-	-	27	1	-	-	-	-	-	17	-	46	-	-	-	-	-	1	16	3	-	-	16

Table 5.1.2.e I (Cont)

Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-C^W sera

Population	Number Tested	Gene complex frequencies									Authors	
		CDE	CDe	C^WDe	Cde	C^Wde	cDE	cDe	cdE	cde		
1-Bakhtiaris	138	2.00	48.47	0.36	4.62	0.00	16.48	6.97	2.17	18.93	Nijenhuis,L.E.	1964
2-South east	348	0.46	49.23	0.29	1.48	0.00	14.24	6.01	0.53	27.76	Nijenhuis,L.E.	1964
3-Esfahan	110	2.56	49.54	0.45	2.44	0.00	10.99	1.32	2.35	30.33	Sawhney,K.S.	1975
4-Tehran	196	0.93	48.23	2.04	1.36	0.00	13.10	1.46	0.00	32.89	Sawhney,K.S.	1975
5-Kurds.Kermanshah	127	0.00	46.49	0.39	0.00	0.00	13.79	3.24	0.00	36.09	Nijenhuis,L.E.	1964

able 5.1.2.f I

Rh blood groups distribution in Iran tested with

anti-C, anti-D, anti-E, anti-c, anti-e, and anti-D^u sera

Population	Number Tested	Phenotypes																				
		CCDEE	CCDEe	CCDee	CCD^{u}ee	CCddee	CcDEE	CcDEe	CcD^{u}Ee	CcDee	CcD^{u}ee	CcddEe	Ccddee	ccDEE	ccD^{u}EE	ccDEe	ccD^{u}Ee	ccDee	ccD^{u}ee	ccddEE	ccddEe	ccddee
1-Kurds.Baneh, Marivan	77	-	-	21	-	-	-	25	-	15	-	-	-	2	-	7	1	-	-	-	-	6

Table 5.1.2. f. I (cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-D^u sera

Population	Number Tested	Gene complex frequencies										Authors
		CDE	CDe	CD^ue	Cde	cDE	cD^uE	cDe	cD^uc	cdE	cde	
1-Kurds.Baneh,Marivan	77	0.00	53.25	0.00	0.00	21.44	0.00	0.00	1.71	2.59	21.01	Lehmann,H., 1973 et al.

Table 5.1.2.h.I Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-Ce(f) sera

Population	Number Tested	Phenotypes															
		CCDEE	CCDEe	CCDee	CCdee	CcDEE	CE/D/Ce	Ce/D/cE	CcDee	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccDee
1-Kurdish Jews	94	-	2	32	-	-	-	23	18	-	2	8	6	-	-	-	3

Table 5.1.2.h.I (cont.)

Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-Ce(f) sera

Population	Number Tested	Gene complex frequencies							Authors
		CDE	CDe	Cde	cDE	cDe	cdE	cde	
1-Kurdish Jews	94	1.06	53.43	4.55	23.94	0.00	0.00	17.02	Godber,Marilyn.J., et al. 1973

Table 5.1.2.i.I

Rh blood groups distribution in Iran tested with

anti-C, anti-D, anti-E, anti-^{c}, anti-^{e}, anti-CE, anti-C^{w}, and anti-D^{u} sera

Population	Number Tested	Phenotypes																			
		CCDEE	CCDEe	C^{w}CDEe	CCDee	c^{w}CDee	$C^{w}C^{w}$Dee	CCddee	CcDEE	CE/D/Ce	Ce/D/cE	C^{w}cDEe	CcDee	C^{w}cDee	CcD^{u}ee	Ccddee	ccDEE	ccDEe	ccDee	ccD^{u}ee	ccddee
1-Kurds. Sanandaj	107	-	-	-	38	1	-	-	-	2	12	1	26	-	2	1	3	11	3	-	7

Table 5.1.2. i.I (Cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, anti-CE, anti-C^w, and anti-D^u, sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	$C^w De$	Cde	cDE	cDe	$cD^u e$	cde	
1-Kurds.Sanandanj	107	0.93	50.14	0.93	5.00	14.02	4.45	2.75	21.78	Lehmann, H., et al. 1973

Table 5.1.2. k I. Rh blood groups distribution in Iran tested with anti-C^w serum

Population	Number Tested	C^w+	C^w-	C^w+%	Authors	
1-Arabs	158	158		CDEce	Nijenhuis,L.E.	1964
2-Armenians	78	78		CDEce	Nijenhuis,L.E.	1964
3-Assyrians	32	32		CDEce	Nijenhuis,L.E.	1964
4-Ghashgais	66	66		CDEce	Nijenhuis,L.E.	1964
5-Kurds.Baneh,Marivan	77	77		CDEce	Lehmann,H.,et al.	1973
6-Kurdish Jews	121	121		CDEce	Godber,Marylin J.,et al.	1973

Table 5.1.2. 1 I. Rh blood groups distribution in Iran tested with anti-V serum

Population	Number Tested	V+	V-	V+%	Authors
Kurdish Jews	94	94	CDEcef		Godber,Maryline,J.,et al. 1973

Table 5.1.2. * I. Rh gene complex frequencies distribution in Iran

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Kurdish Jews	94	1.06	53.43	0.00	4.55	23.94	0.00	0.00	17.02	Godber,Marilyn J., et al. 1973
2-Arabs.Khuzestan	97	1.04	46.28	0.00	3.59	10.15	20.58	0.00	18.36	Marzban, M. 1978
3-Bakhtiaris	138	2.00	48.73	0.00	4.62	16.48	6.97	2.17	18.93	Nijenhuis,L.E. 1964
4-Kurdish Jews	106	4.73	51.86	0.00	2.37	21.69	0.00	0.00	19.35	Tills,D., et al. 1977
5-Kurds.Baneh,Marivan	77	0.00	53.25	0.00	0.00	21.44	1.71	2.59	21.01	Lehmann,H., et al. 1973
6-Turkmans.Gobad	292	0.73	54.01	0.00	2.30	14.78	6.45	0.00	21.73	Boue' and Boue'. 1956
7-Kurds.Sanandaj	107	0.93	51.07	0.00	5.00	14.02	7.20	0.00	12.78	Lehmann,H.,et al. 1973
8-Baluchis.Sistan & Baluchistan	111	0.57	59.63	0.00	1.96	14.29	1.74	0.00	21.81	Present study.
9-Arabs.Abadan	158	0.00	53.18	0.00	0.00	12.13	8.91	3.37	22.41	Nijenhuis,L.E. 1964
10-Gorgan	133	0.00	57.40	0.00	1.64	10.15	8.27	0.00	22.54	Boue' and Boue'. 1956
11-Jews	200	0.00	60.50	0.00	0.00	10.85	6.00	0.00	22.65	Gurevitch,J.,et al.1956
12-Gilan.Turkish Speakers	198	2.04	47.81	0.00	2.70	18.75	3.80	0.00	24.90	Boue' and Boue'. 1956
13-Lurs.Luristan	162	0.81	47.95	0.00	0.00	22.65	3.52	0.00	25.07	Present study.

Table 5.1.2.*I(Cont.) Rh gene complex frequencies distribution in Iran

Population	Number Tested	Gene complex frequencies								Authors	
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde		
14-North west	68	1.50	44.90	0.00	5.80	12.70	9.80	0.00	25.30	Bajatzadeh,M.,& Walter,H.	1969
15-Zabolis.Sistan & Baluchistan	115	1.02	50.71	0.00	5.22	17.24	0.00	0.00	25.80	Present study.	
16-Moslems.Fars	768	0.19	50.83	0.00	0.68	16.03	5.52	0.26	26.49	Bowman,J.E.,et al.	1967
17-South east	348	0.46	49.52	0.00	1.48	14.24	6.01	0.53	27.76	Nijenhuis,L.E.	1964
18-Ghashghais	66	0.99	49.30	0.00	2.47	18.71	0.00	0.00	28.26	Nijenhuis,L.E.	1964
19-Bassaris.Fars	101	0.00	47.10	0.00	0.00	14.39	10.24	0.00	28.27	Bowman,J.E.	1959
20-Kurds.Sanandaj	129	3.92	41.07	0.00	4.27	11.97	10.42	0.00	28.35	Boue' and Boue'.	1956
21-Mamassanis.Fars	113	1.88	42.39	0.00	0.00	25.56	1.72	0.00	28.54	Bowman,J.E.	1959
22-Kurds.Mahabad	125	3.39	45.20	0.00	3.06	8.04	10.21	0.00	30.10	Boue' and Boue'.	1956
23-Esfahan	110	2.56	49.99	0.00	2.44	10.99	1.32	2.35	30.33	Sawhney,K.S.	1975
24-Moslems. Tehran	203	0.00	48.49	0.00	1.29	15.60	3.46	0.00	31.16	Boue' and Boue'.	1956
25-Assyrians.Abadan	32	0.00	43.80	0.00	0.00	18.76	5.01	0.00	32.43	Nijenhuis,L.E.	1964
26-Tehran	113	2.70	36.90	0.00	7.70	7.20	10.50	2.20	32.70	Bajatzadeh,M.,& Walter,H.	1969

Table 5.1.2.* I(Cont) Rh gene complex frequencies distribution in Iran

Population	Number Tested	Gene complex frequencies								Authors	
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde		
27-Tehran	196	0.93	50.27	0.00	1.36	13.10	1.46	0.00	32.89	Sawhney, K.S.	1975
28-North	63	1.30	36.50	0.00	8.20	4.20	11.50	3.70	34.60	Bajatzadeh,M., & Walter, H.	1969
29-Central and South	109	3.30	45.70	0.00	2.40	4.30	8.90	0.00	35.40	Bajatzadeh,M., & Walter, H.	1969
30-Kurds.Kermanshah	127	0.00	46.88	0.00	0.00	13.79	3.24	0.00	36.09	Nijenhuis, L.E.	1964
31-East	70	2.90	47.30	0.00	1.90	4.00	7.30	0.00	36.50	Bajatzadeh,M., & Walter, H.	1969
32-Shi'a . Yazd	151	0.58	43.72	0.00	0.89	13.35	4.20	0.00	37.26	Sunderland,E., and Smith, H. M.	1966
33-West	107	1.00	41.80	0.00	3.40	6.50	7.50	2.30	37.40	Bajatzadeh,M., & Walter,H.	1969
34-Armenians.Esfahan	144	1.52	38.55	0.00	0.94	13.84	6.12	0.97	38.06	Bowman, J.E.	1959
35-Armenians	78	0.00	39.81	0.00	0.00	17.32	3.97	0.00	38.90	Nijenhuis,L.E.	1964

Table 5.1.2.C. 11 Rh blood groups distribution in the Caucasus tested with anti-C, anti-D, anti-E, and anti-c sera

Population	Number Tested	Phenotypes											
		CCDEE	CCDee	CCddEE	CCddee	CcDEE	CcDee	CcddEE	Ccddee	ccDEE	ccDee	ccddEE	ccddee
1-Georgians abroad. Autocthonous	40	-	9	-	-	6	13	-	-	6	1	-	5
2-Armenians autochthonous	199	2	41	-	-	28	62	-	1	30	8	-	27

Table 5.1.2.C II (Cont)

Rh blood groups distribution in the Caucasus tested with anti-C, anti-D, anti-E, and anti-c sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Georgians abroad. Autocthonous	40	0.00	46.30	0.00	0.00	16.36	3.25	0.00	34.09	Kherumian,R., et al. 1954
2-Armenians autochthonous	199	0.88	42.78	0.00	0.77	15.61	4.82	0.00	35.14	Kherumian,R., et al. 1954

Table 5.1.2.d. II

Rh blood groups distribution in the Caucasus tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Phenotypes																		
		CCDEE	CCDEe	CCDee	CCddEE	CCddEe	CCddee	CcDEE	CcDEe	CcDee	CcddEE	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Svanis.Georgia, Abkhazskaya	643	-	10	75	-	-	-	6	113	217	-	-	24	18	92	18	-	4	66

Table 5.1.2.d II (Cont.)

Rh blood groups distribution in the Caucasus tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Svanis. Georgia, Abkhazskaya.	643	2.24	34.76	0.00	4.21	17.73	4.65	0.80	35.61	Verbitsky,M. Sh., et al. 1972

Table 5.1.2. K II Rh blood groups distribution in the Caucasus tested with anti-C^w serum

Population	Number Tested	C^w+	C^w-	C^w+%		Authors	
Svanis.Georgia. Abkhazskaya	643	6	637	0.93	CDEce	Verbitsky,M.Sh.,et al.	1972

Table 5.1.2. * II Rh gene complex frequencies distribution in the Caucasus

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Georgians abroad., autochthonous	40	0.00	46.30	0.00	0.00	16.36	3.25	0.00	34.09	Kherumian,R., et al. 1954
2-Armenians.auto-chthonous	199	0.88	42.78	0.00	0.77	15.61	4.82	0.00	35.14	Kherumian,R., et al. 1954
3-Svanis.Georgia, Abkhazskaya	643	2.24	34.76	0.00	4.21	17.73	4.65	0.80	35.61	Verbitsky,M. Sh., et al. 1972

Table 5.1.2. d * III Rh blood groups distribution in Turkey tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Phenotypes																	
		CCDEE	CCDEe	CCDee	CCddEE	CCddEe	CCddee	CcDEE	CcDEe	CcDee	CcddEE	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Eti-Turks.Asia Minor, near Mersin	118	-	-	33	-	-	-	-	17	46	-	-	-	4	6	1	-	-	11
2-Turks.Asia Minor, Mersin	108	-	-	25	-	-	-	-	21	35	-	-	1	3	10	1	-	-	12

Table 5.1.2. d.* III (Cont.) Rh blood groups distribution in Turkey tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Gene complex frequencies								Authors	
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde		
1-Eti-Turks.Asia Minor, near Mersin	118	0.00	54.66	0.00	0.00	13.14	1.35	0.00	30.85	Aksoy,M., et al.	1958
2-Turks.Asia Minor, Mersin	108	0.00	48.18	0.00	1.38	17.14	1.30	0.00	32.00	Aksoy,M., et al.	1958

Table 5.1.2. f. III

Rh blood groups distribution in Turkey tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-D^u sera

Population	Number Tested	Phenotypes																				
		CCDEE	CCDEe	CCDee	$CCD^{u}ee$	CCddee	CcDEE	CcDEe	$CcD^{u}Ee$	CcDee	$CcD^{u}ee$	CcddEe	Ccddee	ccDEE	$ccD^{u}EE$	ccDEe	$ccD^{u}Ee$	ccDee	$ccD^{u}ee$	ccddEE	ccddEe	ccddee
1-Eti-Turks.Asia Minor , near Mersin	118	-	-	33	-	-	-	17	-	42	4	-	-	4	-	6	-	1	-	-	-	11

Table 5.1.2. f III (Cont.)

Rh blood groups distribution in Turkey tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-D^u sera

Population	Number Tested	Gene complex frequencies										Authors
		CDE	CDe	C^uDe	Cde	cDE	c^uDE	cDe	c^uDe	cdE	cde	
1-Eti-Turks.Asia Minor, near Mersin	118	0.00	50.20	4.46	0.00	13.14	0.00	1.35	0.00	0.00	30.85	Aksoy,M.,et al. 1958

Table 5.1.2. K III Rh blood groups distribution in Turkey tested with anti-C^W serum

Population	Number Tested	C^W+	C^W-	C^W+%	Authors
Eti-Turks.Asia Minor,near Mersin	118	118	CDEceD^u		Aksoy,M., et al. 1968

Table 5.1.2.b. IV

Rh blood groups distribution in Iraq tested with anti-C, anti-D, and anti-E sera

Population	Number Tested	Phenotypes								Gene complex frequencies						Authors	
		CCDEE	CCDee	CCdEE	CCddee	ccDEE	ccDee	ccddEE	ccddee	CDe	Cde	cDE	cDe	cdE	cde		
1-Kurdish Jews	250	54	103	-	2	48	26	4	13	37.42	1.59	20.15	15.61	3.55	21.68	Gurevitch,J., et al.	1955

Table 5.1.2. c IV

Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, and anti-c sera

Population	Number Tested	Phenotypes											
		CCDEE	CCDee	CCddEE	CCddee	CcDEE	CcDee	CcddEE	Ccddee	ccDEE	ccDee	ccddEE	ccddee
1-Kurdish Jews	129	14	38	-	-	34	22	-	-	12	4	-	5
2-Baghdad.Jews	162	9	55	-	-	34	28	-	1	18	5	1	11
3-Baghdad.Arabs	300	-	72	-	-	47	79	-	3	41	25	2	31

Table 5.1.2.c IV (Cont.) Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, and anti-c sera

Population	Number Tested	Gene complex frequencies								Authors	
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde		
1-Kurdish Jews	129	9.82	52.24	0.00	0.00	17.01	5.32	0.00	15.61	Gurevitch,J., et al.	1956
2-Baghdad.Jews	162	4.77	52.37	0.00	1.83	15.46	4.11	1.29	20.17	Gurevitch,J., et al.	1956
3-Baghdad.Arabs	300	0.00	43.56	0.00	1.95	15.13	9.73	1.13	28.50	Kayssi,A.I.	1940

Table 5.1.2.d. IV Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Phenotypes																	
		CCDEE	CCDEe	CCDee	CCddEE	CCddEe	CCddee	CcDEE	CcDEe	CcDee	CcddEE	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Kurdish Jews. South east	50	-	-	24	-	-	-	7	12	5	-	-	-	1	-	-	-	-	1
2-Jews	1146	1	4	338	-	-	1	5	208	340	-	-	8	29	115	23	-	2	72
3-Kurdish Jews. North west	61	-	-	13	-	-	-	4	15	19	-	-	1	1	4	-	-	-	4
4-Assyrians	99	-	-	18	-	-	-	-	12	39	-	-	-	-	11	9	-	-	10
5-Kurdish Jews	27	-	-	7	-	-	-	-	3	9	-	-	-	1	4	-	-	-	3
6-Karaite Jews	72	-	-	4	-	-	-	-	1	24	-	-	-	-	4	19	-	-	20

Table 5.1.2.d. IV (Cont.) Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Kurdish Jews.South east	50	7.49	64.51	0.00	0.00	20.51	0.00	0.00	7.49	Tills,D.,et al, 1977
2-Jews	1146	0.67	52.20	0.00	1.63	16.43	3.74	0.31	25.02	Levence,C. 1968
3-Kurdish Jews.North west	61	4.86	46.17	0.00	2.24	18.91	0.00	0.00	27.82	Tills,D.,et al. 1977
4-Assyrians	99	0.00	43.99	0.00	0.00	11.63	12.92	0.00	31.46	Ikin,Elizabeth W., et al. 1965
5-Kurdish Jews	27	0.00	48.15	0.00	0.00	16.67	0.00	0.00	35.18	Godber,Marilyn. J., et al.. 1973
6-Karaite Jews	72	0.00	22.93	0.00	0.00	3.47	20.90	0.00	52.70	Goldschmidt,E., et al. 1976

Table 5.1.2.f. IV

Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-D^u sera

Population	Number Tested	Phenotypes																				
		CCDEE	CCDEe	CCDee	CCD^ue	CCddee	CcDEE	CcDEe	CcD^uEe	CcDee	CcD^uee	CcddEe	Ccddee	ccDEE	ccD^uEE	ccDEe	ccD^uEe	ccDee	ccD^uee	ccddEE	ccddEe	ccddee
1-Jews	1146	1	4	338	-	1	5	208	-	339	1	-	8	29	-	115	-	23	-	-	2	72

Table 5.1.2.f IV (Cont.) Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-D^u sera

Population	Number Tested	Gene complex frequencies										Authors
		CDE	CDe	CD^ue	Cde	cDE	cD^uE	cDe	cD^ue	cdE	cde	
1-Jews	1146	0.67	52.04	0.16	1.63	16.43	0.00	3.74	0.00	0.31	25.02	Levence,C. 1968

Table 5.1.2.g. IV

Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-V sera

Population	Number Tested	Phenotypes																	
		CCDEE	CCDEe	CCDee	CCddee	CcDEE	CcDEeV	CcDEe	CcDeeV	CcDee	CcddeeV	Ccddee	ccDEE	ccDEeV	ccDEe	ccDeeV	ccDee	ccddeeV	ccddee
1- Assyrians	99	-	-	18	-	-	-	12	2	37	-	-	-	-	11	-	9	-	10

Table 5.1.2.g. IV (Cont.)

Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-V sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	Cde	cDE	cDeV	cDe	cdeV	cde	
1-Assyrians	99	0.00	43.99	0.00	11.63	1.01	11.91	0.00	31.46	Ikin,Elizabeth W., et al. 1965

Table 5.1.2. h. IV

Rh blood groups distribution in Iraq tested with

anti-C, anti-D, anti-E, anti-c, anti-e, and anti-Ce(f)sera

Population	Number Tested	Phenotypes																
		CCDEE	CCDEe	CCDee	CCddee	CcDEE	CE/D/Ce	Ce/D/cE	CcDee	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1- Kurdish Jews	27	-	-	7	-	-	-	3	9	-	-	1	4	-	-	-	3

Table 5.1.2.h. IV (Cont.) Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-Ce (f) sera

Population	Number Tested	Gene complex frequencies							Authors
		CDE	CDe	Cde	cDE	cDe	cdE	cde	
1-Kurdish Jews	27	0.00	48.15	0.00	16.67	0.00	0.00	35.18	Godber,Marilyn.J., 1973 et al.

Table 5.1.2. K IV. Rh blood groups distribution in Iraq tested with anti-C^W serum

Population	Number Tested	C^W+	C^W-	C^W+%	Authors
Assyrians	99	99		CDEceV	Ikin,Elizabeth W.,et al. 1965

Table 5.1.2. 1 IV Rh blood groups distribution in Iraq tested with anti-V serum

Population	Number Tested	V+	V-	V+%	Authors
Kurdish Jews	27	27	CDEcef		Godber,Marylin,J.,et al. 1973

Table 5.1.2. * IV Rh gene complex frequencies distribution in Iraq

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Kurdish Jews, South east	50	7.49	64.51	0.00	0.00	20.51	0.00	0.00	7.49	Tills,D., et al. 1977
2-Kurdish Jews	129	9.82	52.24	0.00	0.00	17.01	5.32	0.00	15.61	Gurevitch,J., et al. 1956
3-Baghdad.Jews	162	4.77	52.37	0.00	1.83	15.46	4.11	1.29	20.17	Gurevitch,J., et al. 1956
4-Jews	1146	0.67	52.20	0.00	1.63	16.43	3.74	0.31	25.02	Levence, C. 1968
5-Kurdish Jews.North west	61	4.86	46.17	0.00	2.24	18.91	0.00	0.00	27.82	Tills,D., et al. 1977
6-Baghdad.Arabs	300	0.00	43.56	0.00	1.95	15.13	9.73	1.13	28.50	Kayssi,A.I. 1940
7-Assyrians	99	0.00	43.99	0.00	0.00	11.63	12.92	0.00	31.46	Ikin,Elizabeth W.,1965 et al.
8-Kurdish Jews	27	0.00	48.15	0.00	0.00	16.67	0.00	0.00	35.18	Godber,Marilyn.J.,1973 et al.
9-Karaite Jews	72	0.00	22.93	0.00	0.00	3.47	20.90	0.00	52.70	Goldschmidt,E., 1976 et al.

Table 5.1.2.d. * V

Rh blood groups distribution in Kuwait tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Phenotypes																	
		CCDEE	CCDEe	CCDee	CCddEE	CCddEe	CCddee	CcDEE	CcDEe	CcDee	CcddEE	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Suluba tribe	52	-	-	13	-	-	-	-	16	9	-	-	-	7	4	1	-	-	2
2-Kuwaitis	200	1	4	44	-	-	1	5	24	55	-	1	3	6	30	15	1	-	10
3-General population	74	-	-	16	-	-	-	-	8	24	-	-	2	1	11	5	-	-	7
4-Kuwaiti Arabs	110	-	1	27	-	-	-	1	14	42	-	-	-	-	3	8	-	1	13
5-Ajman tribe	52	-	-	4	-	-	-	-	9	14	-	-	-	4	9	4	-	-	8

Table 5.1.2.d.*V(Cont.)

Rh blood groups distribution in Kuwait tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Suluba tribe	52	0.00	49.00	0.00	0.00	33.00	3.00	0.00	15.00	Khaled,E., et al. 1981
2-Kuwaitis	200	3.53	38.12	0.00	5.37	15.39	14.42	2.25	20.92	Onsi,A., et al,. 1969
3-General population	74	0.00	41.00	0.00	4.00	14.00	10.00	0.00	32.00	Khaled,E.,et al. 1981
4-Kuwaiti Arabs	110	2.17	49.20	0.00	0.00	5.04	8.80	2.34	32.46	Sawhney,K.S. 1975
5-Ajman	52	0.00	30.00	0.00	0.00	25.00	8.00	0.00	37.00	Khaled,E., et al.. 1981

Table 5.1.2.d. VI

Rh blood groups distribution in Saudi Arabia tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Phenotypes																	
		CCDEE	CCDEe	CCDee	CCddEE	CCddEe	CCddee	CcDEE	CcDEe	CcDee	CcddEE	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Saudi Arabians	1167	-	2	225	-	-	-	-	137	413	-	-	7	32	129	148	-	-	74
2-Sunnis.Qatif and Hasa Oases	323	-	-	58	-	-	-	-	30	110	-	-	3	15	48	39	-	-	20
3-Western Saudi Arabia	178	-	-	39	-	-	-	1	8	53	-	-	-	14	6	40	-	2	15
4-Sunnis.Asir,Hejaz, Najran	176	-	2	37	-	-	-	-	17	60	-	-	1	5	16	21	-	1	16

Table 5.1.2.d. VI(Cont.) Rh blood groups distribution in Saudi Arabia tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Gene complex frequencies								Authors	
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde		
1-Saudi Arabians	1167	0.13	42.06	0.00	1.13	14.09	17.57	0.00	25.02	Maranjian,G., et al.	1966
2-Sunnis.Qatif and Hasa Oases	323	0.00	38.46	0.00	1.63	16.72	17.93	0.00	25.26	Maranjian,G., et al.	1966
3-Western Saudi Arabia	178	0.00	38.97	0.36	0.00	9.96	22.61	2.61	25.50	Saha,N., et al.	1980
4-Sunnis.Asir,Hejaz,Najran	176	0.90	42.46	0.00	0.96	11.23	14.77	0.94	28.74	Maranjian,G., et al.	1966

Table 5.1.2.f VI

Rh blood groups distribution in Saudi Arabia tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-D^u sera

Population	Number Tested	Phenotypes																				
		CCDEE	CCDEe	CCDee	CCD^ue	CCddee	CcDEE	CcDEe	CcD^uEe	CcDee	CcD^uee	CcddEe	Ccddee	ccDEE	ccD^uEE	ccDEe	ccD^uEe	ccDee	ccD^uee	ccddEE	ccddEe	ccddee
1-Sunnis.Qatif and Hasa Oases	323	-	-	58	-	-	-	30	-	110	-	-	3	15	-	48	-	37	2	-	-	20
2-Sunnis.Asir,Hejaz , Najran	176	-	2	37	-	-	-	17	-	60	-	-	1	5	-	16	-	19	2	-	1	16

Table 5.1.2. f VI (Cont.) Rh blood groups distribution in Saudi Arabia tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-D^u sera

Population	Number Tested	Gene complex frequencies										Authors
		CDE	CDe	CD^ue	Cde	cDE	cD^uE	cDe	cD^ue	cdE	cde	
1-Sunnis.Qatif and Hasa Oases	323	0.00	38.46	0.00	1.63	16.72	0.00	16.78	1.15	0.00	25.62	Maranjian, G., et al. 1966
2-Sunnis.Asir,Hejaz, Najran	176	0.90	42.46	0.00	0.96	11.23	0.00	13.13	1.64	0.94	28.74	Maranjian, G., et al. 1966

Table 5.1.2.j. VI

Rh blood groups distribution in Saudi Arabia tested with anti-C+C^w, anti-D,anti-E,anti-c,anti-e,anti-C^w,anti-V,and anti-D^u sera

Population	Number Tested	Phenotypes																										
		CCDEE	CCDEe	CC^wDEe	CCDee	CC^wDee	CCddee	CcDEE	CcDEev	CcDEe	C^wcDEe	CcDeev	CcDee	C^wcDeev	C^wcDee	CcD^ueev	CcD^uee	CcddeeV	Ccddee	ccDEE	ccDEev	ccDEe	ccDeev	ccDee	ccD^ueev	ccD^uee	ccddeev	ccddee
1-Saudi Arabians	1167	-	2	-	224	1	-	-	-	136	1	41	368	1	3	-	-	-	7	32	17	112	56	85	2	5	1	73

Table 5.1.2.j. VI (cont.) Rh blood groups distribution in Saudi Arabia tested with anti-C+C^w, anti-D, anti-E, antic, anti-e, anti-c^w, anti-V, and anti-D^u sera

Population	Number Tested	Gene complex frequencies											Authors
		CDE	CDe	C^wDe	Cde	cDE	cDeV	cDe	cD^uev	cD^ue	cdev	cde	
1-Saudi Arabians	1167	0.13	41.80	0.26	1.13	14.09	4.81	11.67	0.27	0.82	0.13	24.89	Maranjian, 1966 G., et al.

Table 5.1.2. K VI Rh blood groups distribution in Saudi Arabia tested with anti-C^w serum

Population	Number Tested	C^w+	C^w-	C^w+ %		Authors	
Sunnis.Asir,Hejaz, Najran	176	176			CDEceD^u	Maranjian,G.,et al.	1966
Sunnis,Qatif,Hasa Oases	323	323			CDEceD^u	Maranjian,G.,et al.	1966

Table 5.1.2.* VI Rh gene complex frequencies distribution in Saudi Arabia

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Saudi Arabians	1167	0.13	42.06	0.00	1.13	14.09	17.57	0.00	25.02	Maranjian,G., et al. 1966
2-Sunnis.Qatif & Hasa Oases	323	0.00	38.46	0.00	1.63	16.72	17.93	0.00	25.26	Maranjian,G., et al. 1966
3-Western Saudi Arabia	178	0.00	38.97	0.36	0.00	9.96	22.61	2.61	25.50	Saha,N., et al. 1980
4-Sunnis.Asir,Hejaz, Najran	176	0.90	42.46	0.00	0.96	11.23	14.77	0.94	28.74	Maranjian,G., et al. 1966

Table 5.1.2.c. VIII

Rh blood groups dsitribution in Pakistan tested with anti-C, anti-D, anti-E, and anti-c sera

Population	Number Tested	Phenotypes											
		CCDEE	CCDee	CCddEE	CCddee	CcDEE	CcDee	CcddEE	Ccddee	ccDEE	ccDee	ccddEE	ccddee
1-Bengalis.Dacca	236	6	118	-	2	24	58	-	3	12	4	-	9
2-Punjabis.Lahore	203	5	75	-	-	24	64	-	2	12	9	-	12
3-Moslems.Karachi	150	-	62	-	-	20	48	-	1	8	1	-	10
4-Peshawar	155	4	62	-	-	16	38	-	2	12	6	1	14
5-Parsis.Karachi	103	-	44	-	-	12	31	-	-	4	1	-	11

Table 5.1.2.c. VIII (Cont.) Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, and anti-c sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Bengalis.Dacca	236	1.71	63.08	0.00	6.62	7.71	3.80	0.00	17.08	Boyd,W.C., et al. 1954
2-Punjabis.Lahore	203	2.04	57.39	0.00	2.17	8.64	7.21	0.00	22.55	Boyd,W.C., et al. 1954
3-Moslems.Karachi	150	0.00	63.03	0.00	1.31	9.76	1.20	0.00	24.70	Moten,A.N., et al. 1956
4-Peshawar	155	1.94	55.82	0.00	2.91	8.53	4.82	0.92	25.06	Boyd,W.C., et al. 1954
5-Parsis.Karachi	103	0.00	63.61	0.00	0.00	8.02	1.20	0.00	27.17	Moten,A.N., et al. 1956

Table 5.1.2. d VIII Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Phenotypes																	
		CCDEE	CCDEe	CCDee	CCddEE	CCddEe	CCddee	CcDEE	CcDEe	CcDee	CcddEE	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Baltis.Baltistan	80	-	-	25	-	-	-	-	22	16	-	-	-	2	8	1	-	-	6
2-Moslems.Punjab & West	101	-	-	46	-	-	-	1	10	33	-	-	1	-	5	-	-	-	5
3-Pathans.Swat.Saidu Sharif	133	-	1	42	-	-	-	-	18	44	-	-	-	2	8	6	-	-	12
4-Hunza.Gilgit	23	-	-	7	-	-	-	-	3	9	-	-	-	1	2	-	-	-	1

Table 5.1.2.d. VIII (Cont.) Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Baltis.Baltistan	80	0.00	55.02	0.00	0.00	21.39	1.74	0.00	21.85	Clegg,E.J., 1961 et al.
2-Moslems.Punjab & West	101	0.63	66.69	0.00	0.49	7.78	0.05	0.00	24.36	Chaudhri,I. 1952 M.,et al.
3-Pathans.Swat.Saidu Sharif	133	0.59	55.08	0.00	0.00	11.08	6.09	0.00	27.16	Alciati,G. 1968
4-Hunza.Gilgit	23	0.00	56.52	0.00	0.00	15.22	0.00	0.00	28.26	Ikin,Eliza- 1959 beth W.,et al.

Table 5.1.2. e. VIII

Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-C^w sera

Population	Number Tested	Phenotypes																						
		CCDEE	CCDEe	C^wCDEe	CCDee	C^wCDee	C^wC^wDee	CCddee	C^wCddee	C^wC^wddee	CcDEE	CcDEe	C^wcDEe	CcDee	C^wcDee	CcddEe	C^wcddEe	Ccddee	C^wcddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Moslems.Punjab & West	101	-	-	-	46	-	-	-	-	-	1	10	-	33	-	-	-	-	1	-	5	-	-	-	5

Table 5.1.2.e. VIII (Cont.)

Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-C^w sera

Population	Number Tested	Gene complex frequencies									Authors
		CDE	CDe	C^wDe	Cde	C^wde	CDE	cDe	cdE	cde	
1-Moslems.Punjab & West	101	0.63	66.69	0.00	0.00	0.49	7.78	0.05	0.00	24.36	Chaudhri,I.M., 1952 et al.

Table 5.1.2. 1 VIII Rh blood groups distribution in Pakistan tested with anti-V serum

Population	Number Tested	V+	V-	V+%	Authors	
1-Pathans	139	139			Vos,G.H., and Kirk,R.L.	1961
2-Punjabis	168	168			Vos,G.H., and Kirk,R.L.	1961

Table 5.1.2. * VIII Rh gene complex frequencies distribution in Pakistan

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Bengalis.Dacca	236	1.71	63.08	0.00	6.62	7.71	3.80	0.00	17.08	Boyd,W.C., et al. 1954
2-Baltis.Baltistan	80	0.00	55.02	0.00	0.00	21.39	1.74	0.00	21.85	Clegg,E., et al. 1961
3-Punjabis.Lahore	203	2.04	57.39	0.00	2.17	8.64	7.21	0.00	22.55	Boyd,W.C.,et al. 1954
4-Moslems.Punjab & West	101	0.63	66.69	0.00	0.49	7.78	0.05	0.00	24.36	Chaudhri,I.M., et al. 1952
5-Moslems.Karachi	150	0.00	63.03	0.00	1.31	9.76	1.20	0.00	24.70	Moten,A.N.,et al.1956
6-Peshawar	155	1.94	55.82	0.00	2.91	8.53	4.82	0.92	25.06	Boyd,W.C.,et al. 1954
7-Pathans.Swat. Saidu Sharif	133	0.59	55.08	0.00	0.00	11.08	6.09	0.00	27.16	Alciati,G. 1968
8-Parsis.Karachi	103	0.00	63.61	0.00	0.00	8.02	1.20	0.00	27.17	Moten,A.N.,et al.1956
9-Hunza.Gilgit	23	0.00	56.52	0.00	0.00	15.22	0.00	0.00	28.26	Ikin,Elizabeth W.,et al. 1959

Table 5.1.2.d*IX Rh blood groups distribution in Afghanistan tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Populations	Number tested	Phenotypes																		
		CCDEE	CCDEe	CCDee	CCddEE	CCddEe	CCddee	CcDEE	CcDEe	CcDee	CcddEE	CcddEe	Ccddee	ccDEE	ccDEe	ccDee	ccddEE	ccddEe	ccddee
1-Timuri and related tribes	118	-	-	38	-	-	-	-	43	24	-	-	-	6	2	1	-	-	4
2- Pushtus	104	-	-	27	-	-	-	-	16	26	-	-	-	4	17	5	-	-	9
3- Daris	179	-	1	60	-	-	1	-	24	43	-	-	1	3	20	6	-	-	20

Table 5.1.2.d*IX (Cont). Rh blood groups distribution in Afghanistan tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

Population	Number Tested	Gene complex frequencies								Authors
		CDE	CDe	CdE	Cde	cDE	cDe	cdE	cde	
1-Timuri and related tribes	118	0.00	60.61	0.00	0.00	24.15	1.61	0.00	13.63	Woodd-Walker, 1967 R.B., et al.
2-Pushtus	104	0.00	46.10	0.00	0.00	19.70	6.80	0.00	27.40	Papiha,S.S., 1977 et al.
3-Daris	179	0.40	52.40	0.00	0.80	13.80	4.00	0.00	28.60	Papiha,S.S., 1977 et al.

Table 5.1.2. K IX Rh blood groups distribution in Afghanistan tested with anti-C^W serum

Population	Number Tested	C^W+	C^W-	C^W+%		Authors
Timuri and related tribes	118		118		CDEce	Woodd-Walker,R.B.,et al. 1967

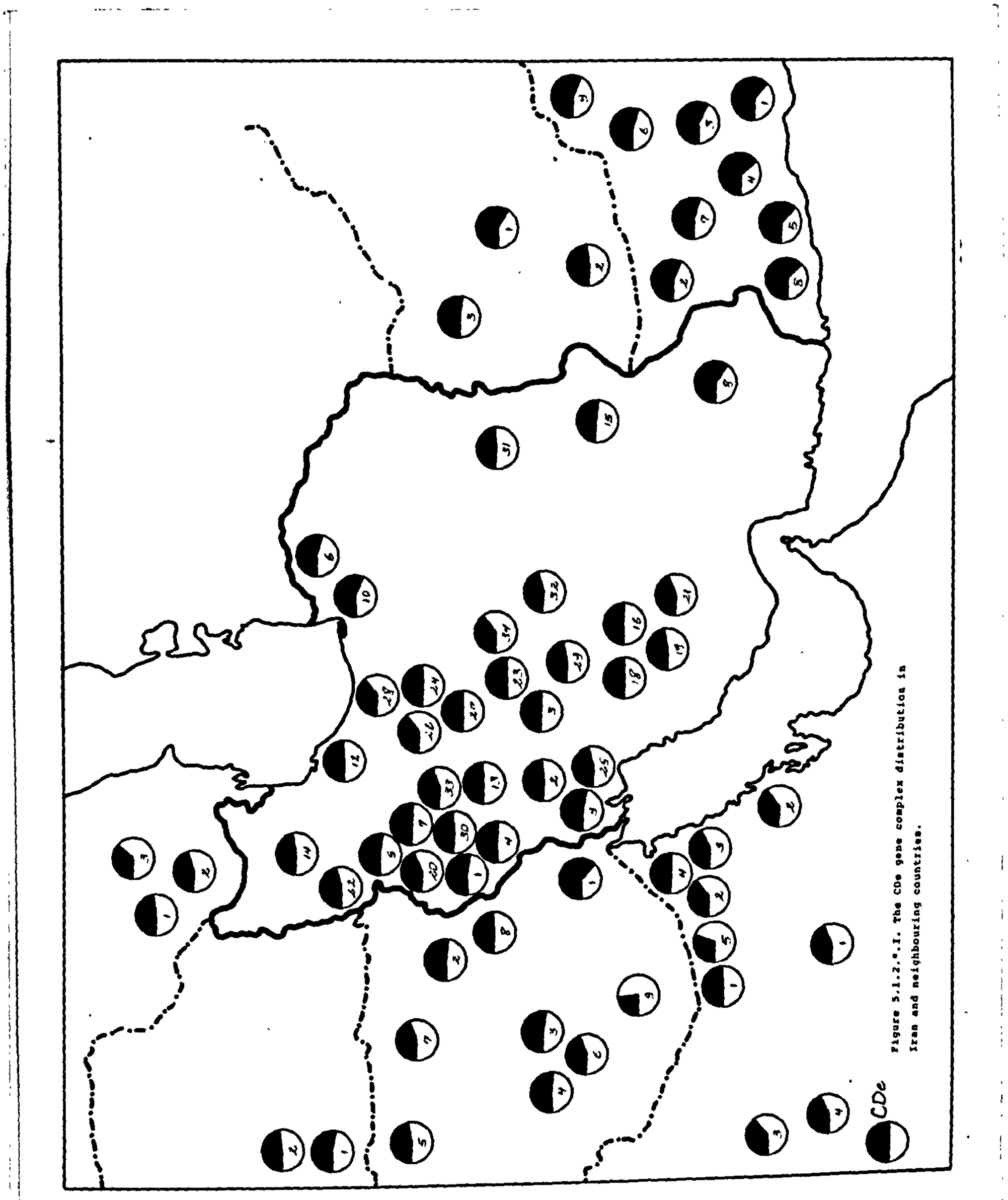


Figure 5.1.2.e.1. The CDe gene complex distribution in Iran and neighbouring countries.

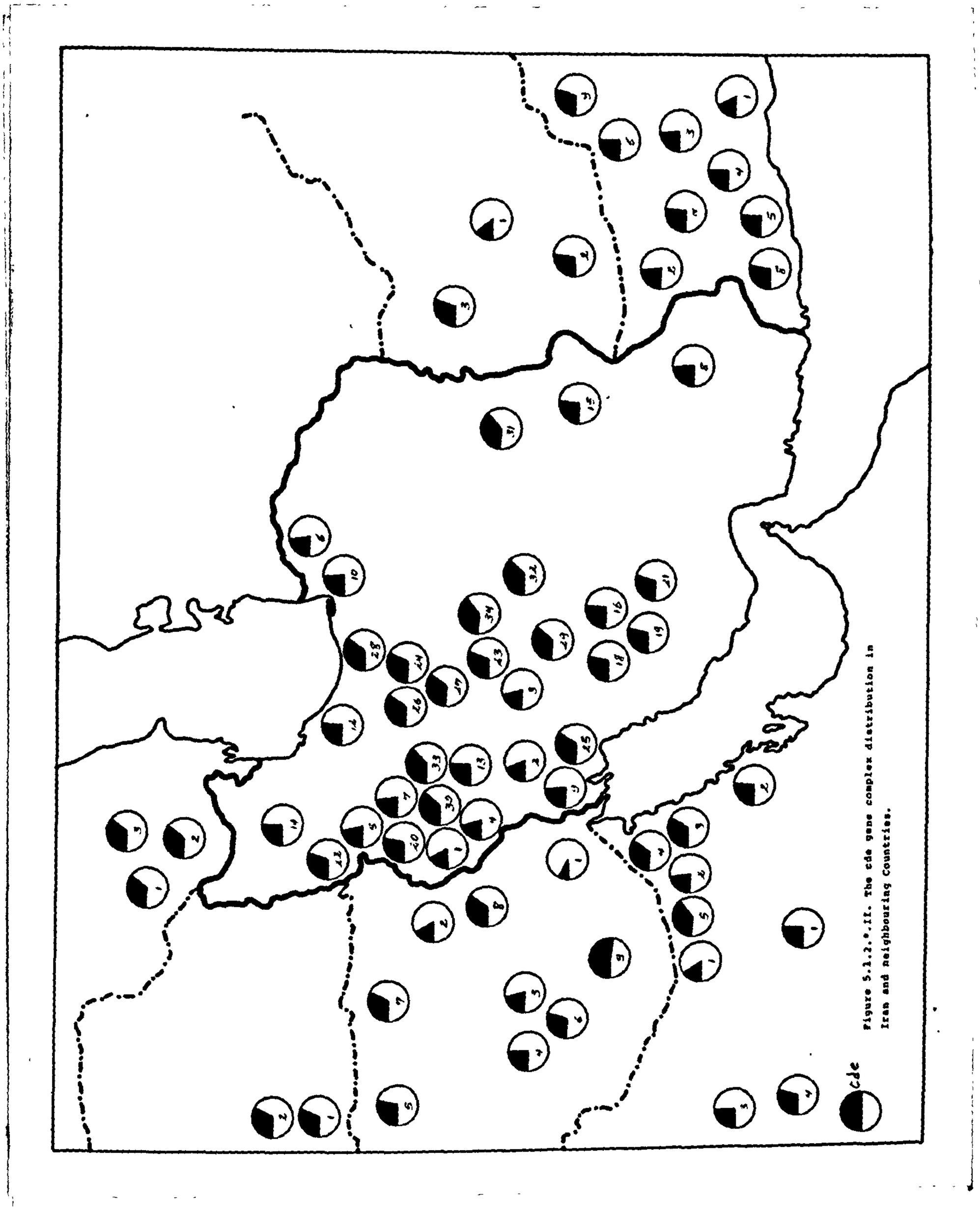


Figure 5.1.2.*.II. The cde gene complex distribution in Iran and neighbouring Countries.

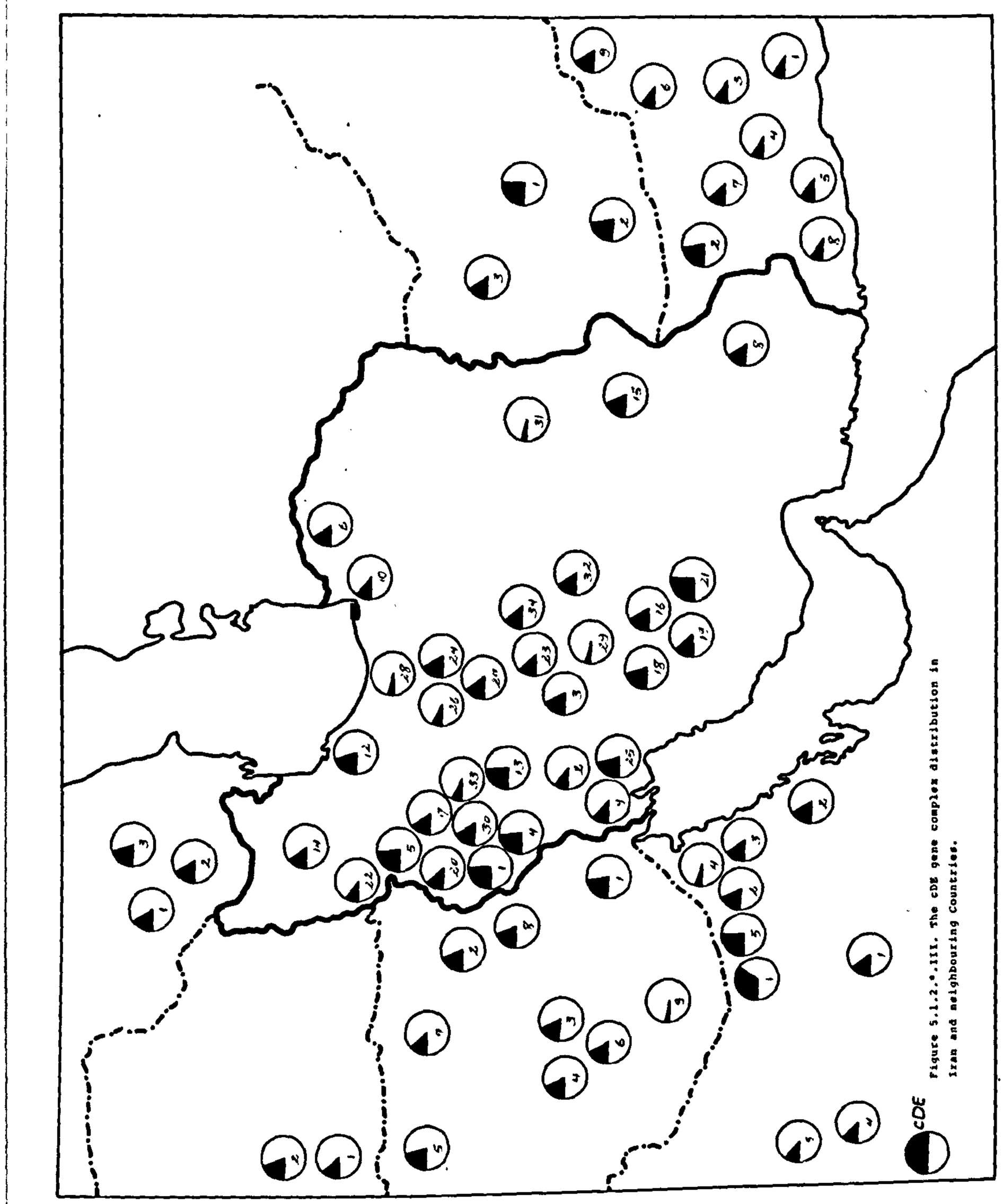


Figure 5.1.2.*.III. The cDE gene complex distribution in Iran and neighbouring Countries.

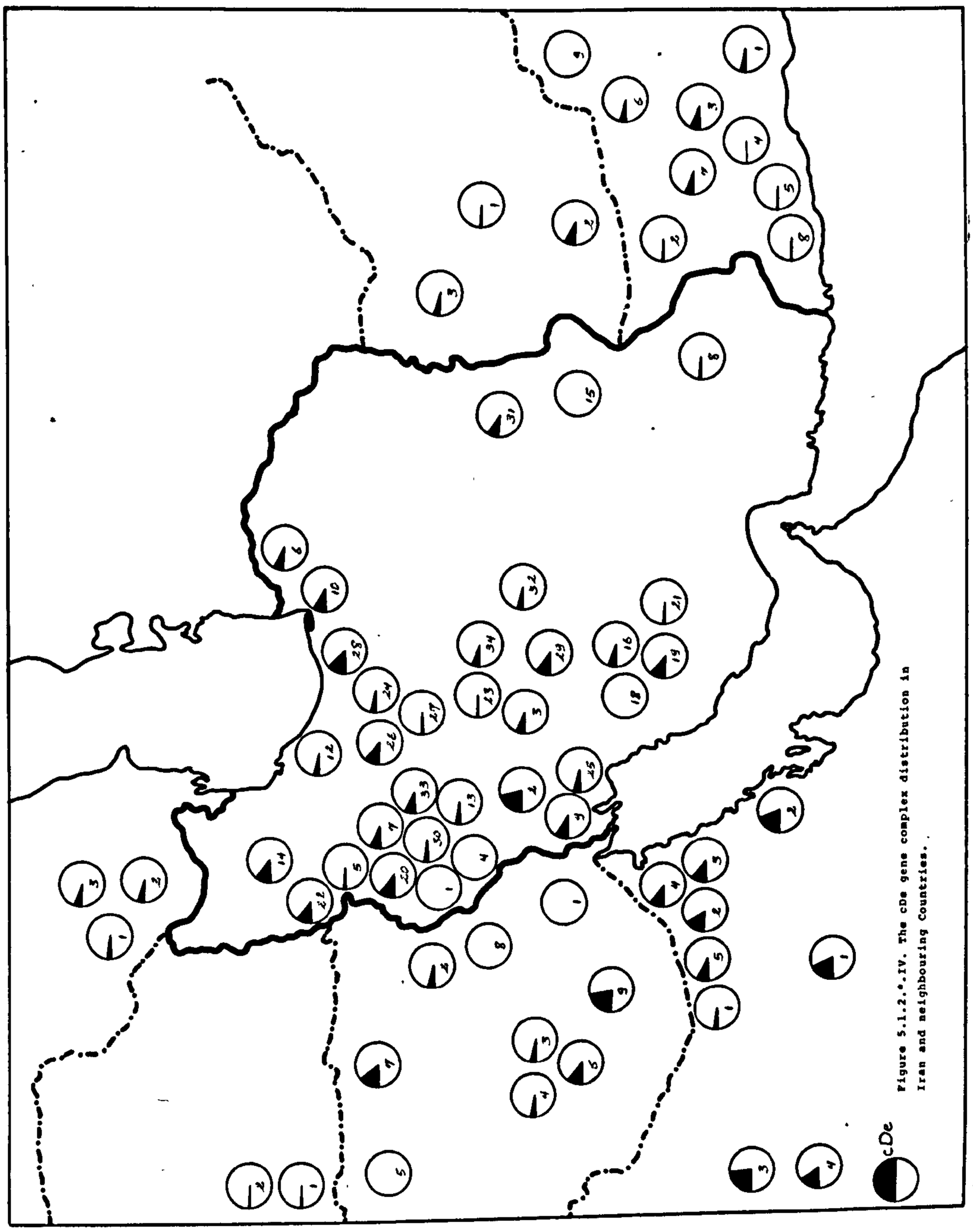


Figure 5.1.2.4.IV. The cDe gene complex distribution in Iran and neighbouring Countries.

5.1.3. The MNSs blood group system

The distribution of MN blood groups and respective gene frequencies in Iranian and neighbouring populations, expressed in term of three phenotypes after testing with two anti-sera, is shown in Tables 5.1.3.a.

The frequency of the M gene ranges from 49.59 to 75.23 percent in Iranians, being lowest in the Kurds of Rezaieh and highest in the Baluchis of Sistan and Baluchistan both obtained in the present investigation.

On the whole, with an average M gene frequency of 62.54 percent, the Iranian population appears to exhibit a much higher M frequency than that of around 55 percent found in most European populations(Mourant et al, 1976).

From the table, some differences in the distribution of the M gene frequencies seem to exist in Iran, as the M gene with an average frequency of 59.33 percent in western Iran appears to increases eastwards with an average frequency of 66.80 percent in eastern Iran.

The Kurds both in western Iran and in Iraq show an M gene frequency averaging 61.41 and 64.20 percent respectively which are near the average for Iran and for Iraq. It is, therefore,interesting that the Kurdish Jews both in Iran and in Iraq exhibit much lower M gene frequencies, averaging only 56.75 and 48.76 percent respectively,figures near those for Europeans. The non-Kurdis Jews of Iran and Iraq have an average M gene frequency of 62.27 and 55.24 percent respectively which are, on the whole,also rather low in relation to the regional level.

The Arabs of south western Iran, with an M gene frequency averaging 60.67 percent which is near the average for Iran, show no resemblance to the Arabs of Arabia with the extremely high M gene frequency averaging 74.17 percent.

Regarding neighbouring groups, the frequency of the M gene in the populations of the Caucasus ranges from 55.43 percent in the Azerbaijanians of Barda (Voronov,1973) to 71.00 percent in the Avars of Dagestan (Gadzhiev,1964), with an average M gene frequency of 63.80 percent, the population of the Caucasus appears to exhibit slightly higher M frequencies than those found in Iranians and higher than the European frequencies.

The M gene frequency in the Turkish populations varies between 49.07 percent in the Turks and 57.33 percent in the Eti-Turks (Aksoy et al, 1958). With an average M gene frequency of 55.49 percent, the population of Turkey shows more similarity to the populations of other east Mediterranean countries than to Iranians and other Asiatic populations generally, with their much higher M frequencies.

The frequency of the gene M in the populations of Iraq ranges from 35.19 percent in the Kurdish Jews(Godber et al, 1973) to 98.61 percent in the Karaite Jews (Goldschmidt et al,1976). With an average M gene frequency of 60.00 percent, the Iraqi population appears to exhibit a lower M frequency than that found in Iranians but higher than the European frequency.

The extremely high M gene frequency of 98.61 percent in the Karaite Jews clearly indicates the strong influence of isolation and genetic drift operating in this community.

The M gene frequency in the populations of Kuwait varies between 51.96 percent in the Suluba tribe and 70.19 percent in the Ajman tribe (Khaled et al, 1981). With an average M gene frequency of 59.16 percent, the Kuwaiti population seems to show a lower M gene frequency than that found in

Iranians but higher than that found in Europeans.

The frequency of the gene M in the populations of Saudi Arabia ranges from 63.07 percent in the Shia sample of Qatif and Hasa Oases (Maranjian et al, 1966) to 87.50 percent in western Saudi Arabians (Saha et al, 1980).With an average M gene frequency of 74.17 percent, the Arab population of Saudi Arabia appears to exhibit a much higher frequency of the gene M than that found in Iranians and ofcourse much higher than the European frequency.

High frequencies of the M gene characterize many of the indigenous populations of southern Asia, and from a peak in Arabia, the M gene frequency declines eastwards before rising again in the Indian subcontinent (Mourant et al, 1976).

The Abu-Dhabians of the United Arab Emirates with an M gene frequency of 61.00 percent (Kamel et al, 1980) seem to exhibit a much lower M frequency compared with that found in Saudi Arabians and even lower than the Iranian frequency but still higher than the frequency in Europeans.

The frequency of the M gene in the populations of Pakistan varies between 58.04 percent in the Bengalis (Boyd and Boyd, 1954) and 67.80 percent in the Pathans (Alciati, 1968). With an average M gene frequency of 63.01 percent, the Pakistani population appears to show a slightly higher M gene frequency than that found in Iranians and higher than the European frequency. The M gene frequency tends to be higher than this in most of the Indian region, varying between 65 and 70 percent (Mourant et al, 1976).

The M gene frequency in the Afghan populations ranges from 62.02 percent in the Pushtus (Papiha et al, 1977) to 66.-10 percent in the Timuri and related tribes (Woodd-Walker et

al, 1967). With an average M gene frequency of 64.22 percent, the population of Afghanistan seems to exhibit a higher M gene frequency than that found in Iranians and higher than the European frequency.

The distribution of the MNSs blood groups and respective gene combination frequencies in Iranian and neighbouring populations, expressed in terms of six and nine phenotypes after testing with three and four antisera, respectively, is set out in Tables 5.1.3. b and c.

The gene complex frequencies were combined in Tables 5.1.3* for comparative purposes.

The frequencies of the MNSs complexes in Iranian populations are as follows:

MS ranges from 10.24 percent in a sample from the north of Iran (Bajatzadeh and Walter, 1969) to 48.95 percent in the Baluchis of Sistan and Baluchistan (Present investigation).

Ms varies between 26.28 percent in the Baluchis of Sistan and Baluchistan (Present investigation) and 51.92 percent in the northern sample of Bajatzadeh and Walter (1969).

NS ranges from 3.43 percent in the Ghashghai tribe (Nijenhuis, 1964) to 16.36 percent in the Kurdish Jews (Godber et al, 1973).

Ns varies between 17.69 percent in the Baluchis of Sistan and Baluchistan and 40.18 percent in the Kurds of Rezaieh (Present study).

The approximate MNSs-complex frequencies in European populations, calculated by the author on the basis of the data available in Mourant et al (1976), are as follows:

Gene complex	% Frequency
MS	25.33
Ms	32.55
NS	7.87
Ns	34.66

On the whole,with average MNSs-complex frequencies of:

Gene complex	% Frequency
MS	22.92
Ms	39.43
NS	9.60
Ns	28.05

the Iranian population appears to exhibit lower MS and Ns but higher Ms and NS-complex frequencies than those found in Europeans.

In Iranians about 37 percent of the M chromosomes carry S while the proportion of NS chromosomes is 25 percent,somewhat more than 1/6 given by Mourant et al (1976) a characteristic of Europeans.

The Kurdish Jews both in Iran and in Iraq appear to be characterized by low MS(average 15.44 and 17.13 percent respectively) and high NS(average 15.98 and 17.62 percent respectively)frequencies.

The Arabs of south western Iran with MNSs complex frequencies of MS 21.21 percent;Ms 36.52 percent; NS 7.14 percent;and Ns 35.13 percent,show no resemblance to the Arabs of Arabia with their extremely high MS(31.40 percent)and low Ns(19.66 percent) frequencies.

Regarding neighbouring populations,the frequencies of the MNSs complexes in the populations of the Caucasus are as follows: MS ranges from 31.02 percent in the Svanis of Georgia, Abkha-

zskaya (Verbitsky et al, 1971) to 38.85 percent in the Georgians (Kherumian et al, 1954).

Ms varies between 27.08 percent in the Georgians (Kherumian et al, 1954) and 34.48 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1971).

NS ranges from 11.67 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1971) to 14.50 percent in the Georgians (Kherumian et al, 1954).

Ns varies between 19.57 percent in the Georgians (Kherumian et al, 1954) and 22.83 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1971). With average MNSs-complex frequencies of:

Gene complex	% Frequency
MS	34.94
Ms	30.78
NS	13.08
Ns	21.20

the population of the Caucasus appears to exhibit much higher MS and NS but lower Ms and Ns frequencies than those found in Iranians.

In the population of the Caucasus about half the M genes carry S and half s, while of the N genes about 38 percent carry S which is much higher than that in Iranians.

The frequencies of the MNSs complexes in the populations of Turkey (Aksoy et al, 1958) are as follows:

MS ranges from 19.04 percent in the Turks to 31.96 percent in the Eti-Turks.

Ms varies between 25.43 percent in the Eti-Turks and 30.11 percent in the Turks.

NS ranges from 8.52 percent in the Eti-Turks to 9.07 percent

in the Turks.

Ns varies between 34.09 percent in the Eti-Turks and 41.78 percent in the Turks. With average MNSs-complex frequencies of:

Gene complex	% Frequency
MS	25.50
Ms	27.77
NS	8.79
Ns	37.94

the population of Turkey seems to show higher MS and Ns but lower Ms and NS frequencies than those found in Iranians. In the population of Turkey, as in Europeans, about half the M genes carry S and half s, while of the N genes about one-sixth carry S and the rest s.

The frequencies of the MNSs complexes in the populations of Iraq are as follows:

MS ranges from 5.98 percent in the Kurdish Jews (Godber et al, 1973) to 36.10 percent in the Assyrians (Ikin et al, 1965).

Ms varies between 16.59 percent in the Kurdish Jews of the south east (Tills et al, 1977) and 80.60 percent in the Karaite Jews (Goldschmidt et al, 1976).

NS ranges from zero in the Karaite Jews (Goldschmidt et al, 1976) to 27.00 percent in the Kurdish series of Al- Khafaji et al (1976).

Ns varies between 1.40 percent in the Karaite Jews (Goldschmidt et al, 1976) and 41.24 percent in the Kurdish Jews of the south east (Tills et al, 1977).

With average MNSs-complex frequencies of :

Gene complex	% Frequency
MS	22.08
Ms	37.49
NS	15.60
Ns	24.83

the population of Iraq appears to exhibit higher NS but lower MS, Ms and Ns frequencies than those found in Iranians. In the Iraqi population about 37 percent of the M genes carry S, while of the N genes about 38 percent carry S which is more than ¼ a characteristic of Iranians.

The frequencies of the MNSs complexes in the populations of Kuwait are as follow:

MS ranges from 14.00 percent in the Suluba tribe to 38.00 percent in the Ajiman tribe (Khaled et al, 1981).

Ms varies between 30.00 percent in general population of Kuwait (Khaled et al, 1981) and 38.14 percent in the Kuwaiti Arabs (Sawhney, 1975).

NS ranges from 5.09 percent in the Kuwaiti Arabs (Sawhney, 1975) to 25.00 percent in the Suluba tribe (Khaled et al,1981).

Ns varies between 8.00 percent in the Ajiman tribe (Khaled et al, 1981) and 34.53 percent in the Kuwaiti Arabs (Sawhney, 1975).

With average MNSs-complex frequencies of:

Gene complex	% Frequency
MS	24.56
Ms	34.54
NS	18.02
Ns	22.88

the Population of Kuwait seems to show higher MS and NS but lower Ms and Ns frequencies than those found in Iranians.

In the Kuwaiti population about 41 percent of the M genes carry S, while the proportion of NS chromosomes is .44 percent, higher than ¼ in Iranians and much higher than 1/6 in Europeans.

The frequencies of the MNSs complexes in the populations of Saudi Arabia are as follows:

MS ranges from 25.61 percent in the Shia sample of Qatif and Hasa Oases to 36.50 percent in the Sunni sample of Najd(Maranjian et al, 1966).

Ms varies between 37.49 percent in the Shia sample of Qatif and Hasa Oases (Maranjian et al, 1966) and 57.06 percent in western Saudi Arabia (Saha et al, 1980).

NS ranges from 3.37 percent in western Saudi Arabia (Saha et al, 1980) to 9.63 percent in the Bedouins (Maranjian et al, 1966).

Ns varies between 9.13 percent in western Saudi Arabia (Saha et al, 1980) and 28.83 percent in the Shia of Qatif and Hasa Oases (Maranjian et al, 1966).

With average MNSs-complex frequencies of:

Gene complex	% Frequency
MS	31.40
Ms	42.80
NS	6.14
Ns	19.66

the population of Saudi Arabia appears to exhibit higher MS and Ms but lower NS and Ns frequencies than those found in Iranians.

In the Saudi Arabian population about 42 percent of the M genes carry S which is higher than that in Iranians, while of the N genes about 24 percent carry S, a figure similar to

that in Iranians.

The frequencies of the MNSs complexes in the Abu-Dhabians of the United Arab Emirates are as follows:

Gene complex	% Frequency
MS	24.70
Ms	36.30
NS	10.50
Ns	28.50

With the above mentioned MNSs - complex frequencies, the Abu-Dhabians seem to show higher MS, NS and Ns but lower Ms frequencies than those found in Iranians.

In the Abu-Dhabians of the United Arab Emirates about 40 percent of the M chromosomes carry S while the proportion of NS chromosomes is 27 percent, figures nearly similar to those is Saudi Arabians.

The frequencies of the MNSs complexes in the populations of Pakistan are as follows:

MS ranges from 17.71 percent in the Punjabis of Lahore to 32.16 percent in Peshawar (Body and Boyd, 1954).

Ms varies between 32.47 percent in the Gilgit, Hunza (Ikin et al, 1959) and 43.49 percent in the Pathans (Alciati, 1968).

NS ranges from 6.65 percent in Peshawar to 13.02 percent in the Bengali series of Dacca (Boyd and Boyd, 1954).

Ns varies between 19.79 percent in the Pathans (Alciati,1968) and 29.84 percent in the Gilgit, Hunza (Ikin et al, 1959).

With average MNSs-complex frequencies of:

Gene complex	% Frequency
MS	24.70
Ms	38.36
NS	11.05
Ns	25.89

the Pakistani population appears to exhibit slightly higher MS and NS but lower Ms and Ns frequencies than those found in Iranians.

In the population of Pakistan about 39 percent of the M genes carry S, while the proportion of NS chromosomes is about 30 percent, somewhat more than that in Iranians.

The MNSs-complex frequencies in the populations of Afghanistan are as follows:

MS ranges from 17.26 percent in the Timuri and related tribes (Woodd-Walker et al,1967)to 29.00 percent in the Pushtus (Papiha et al, 1977).

Ms varies between 33.00 percent in the Pushtus (Papiha et al 1977) and 48.84 percent in the Timuri and related tribes (Woodd-Walker et al, 1967).

NS ranges from 7.50 percent in the Pushtus (Papiha et al,1977) to 9.67 percent in the Timuri and related tribes(Woodd-Walker et al, 1967).

Ns varies between 24.23 percent in the Timuri and related tribes (Woodd-Walker et al,1967)and 30.50 percent in the Pushtus (Papiha et al,1977).

With average MNSs-complex frequencies of:

Gene complex	% Frequency
MS	23.99
Ms	40.21
NS	8.79
Ns	27.01

the Afghan population seems to show slightly higher MS and Ms but lower NS and Ns frequencies than those found in Iranians.

In the population of Afghanistan about 37 percent of the M gene carry S While of the N genes about 24 percent carry S, figures similar to that found in Iranians.

Conclusion

MN frequencies vary less, on the whole, than those of the ABO and Rh groups. Most of the populations tested show M gene frequencies between 50 and 60 percent. Such frequencies are found throughout most of Europe and Africa, and in eastern Asia.

Frequencies of M around the Mediterranean sea, being below 60 percent and mostly below 55 percent, are similar to those found in north western Europe, but eastwards the frequency of M rises steadily to values well above 60 percent in India (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the population of Turkey which shows more similarity to the European frequency, the frequency of the M gene, being 60 percent and over, is much higher than that in Europeans, and is characteristic of the 60-65 percent belt which sweeps across Finland to south east Asia, including the Caspian sea area and much of the Arabian Peninsula.

Saudi Arabia shows a very high local maximum with M frequencies above 70 percent.

Regarding the MNSs complexes, In the European populations about half the M genes carry S and halfss, while of theN genes about one-sixth carry S and the rest s (Mourant et al, 1976).

In Iranian and neighbouring populations, again with the

exception of the population of Turkey which shows more similarity to the Europeans, the proportion of MS chromosomes is less while that of NS chromosomes is more than ½ and 1/6 respectively given by Mourant et al (1976) as characteristics of the *European populations*.

The high frequency of 34.94 percent of the MS complex in the population of the Caucasus is noteworthy.

The Arabs of Saudi Arabia, as mentioned above, have an exceptionally high frequency of M.

Frequencies of M are high nearly everywhere in southern Asia, but Arabia represents a high Peak, separated by lower frequencies from the high ones of India and south east Asia. Moreover, M in Arabia is accompanied by exceptionally high frequencies of S, and it is here that the MS combination reaches its highest known frequency other than in American Indians.

Table 5.1.3.a. I MN blood groups distribution in Iran tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
1-Kurds Rezaieh	124	31	61	32	49.59	50.41	Present study.	
2-Turks.Azerbaijan	1043	312	489	242	53.36	46.64	Bidar,A.	1956
3-Armenians	78	27	34	17	56.41	43.59	Nijenhuis, L.E.	1964
4-Kurdish Jews	106	30	60	16	56.60	43.40	Tills, D., et al.	1977
5-Kurdish Jews	94	27	53	14	56.91	43.09	Godber,Marilyn J., et al.	1973
6-Arabs.Khuzistan	97				57.73	42.27	Marzban, M.	1978
7-Turks.Rezaieh	139	49	65	25	58.63	41.37	Present study.	
8-Iranians	56	19	28	9	58.93	41.07	Nijenhuis, L.E.	1964
9-Jews	200	63	111	26	59.25	40.75	Gurevitch, J.,et al.	1956
10-Tehran	113	43	49	21	59.73	40.27	Bajatzadeh,M., & Walter, H.	1969
11-KUrds.Kermanshah	127	47	58	22	59.84	40.16	Nijenhuis, L.E.	1964
12-Kurds.Sanandaj	107	44	42	21	60.75	39.25	Lehmann,H.,et al.	1973

Table 5.1.3.a.I(Cont.) MN blood groups distribution in Iran tested with anti-M and anti- N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
13-Zoroastrians	74	27	36	11	60.81	39.19	Present study.	
14-North	58	24	24	10	62.07	37.93	Bajatzadeh,M., & Walter, H.	1969
15-Assyrians.Abadan	32	12	16	4	62.50	37.50	Nijenhuis, L.E.	1964
16-Shi'a Yazd	151	57	75	19	62.58	37.42	Sunderland,E., & Smith, H.M.	1966
17-Turks.Mainly south east	348	128	181	39	62.79	37.21	Nijenhuis,L.E.	1964
18-Central and South	109	48	41	20	62.85	37.15	Bajatzadeh,M., & Walter, H.	1969
19-Moslems.Tehran	256	104	117	35	63.48	36.52	Boue' and Boue'.	1956
20-Arabs. Abadan	158	62	77	19	63.61	36.39	Nijenhuis,L.E.	1964
21-Kurds.Baneh , Marivan	77	33	32	12	63.64	36.36	Lehmann,H., et al.	1973
22-East	67	28	30	9	64.18	35.82	Bajatzadeh,M., & Walter, H.	1969
23-Jews	108	54	33	21	65.30	34.70	Tabatabai, H.	1977

Table 5.1.3.a. I (Cont) MN blood groups distribution in Iran tested with anti-M and Anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
24-Zabolis.Sistan & Baluchistan	114	52	45	17	65.35	34.65	Present study.	
25-West	107	48	44	15	65.42	34.58	Bajatzadeh,M., & Walter, H.	1969
26-North west	66	31	25	10	65.91	34.09	Bajatzadeh,M., & Walter, H.	1969
27-Ghashghais	66	27	33	6	65.91	34.09	Nijenhuis,L.E.	1964
28-Tehran	137	60	62	15	66.42	33.58	Sawhney, K.S.	1975
29-Bakhtiaris	138	62	60	16	66.67	33.33	Nijenhuis, L.E.	1964
30-Esfahan	111	56	40	15	68.47	31.53	Sawhney, K.S.	1975
31-Armenians	145	80	46	19	71.10	28.90	Tabatabai, H.	1977
32-Lurs.Luristan	153	83	54	16	71.90	28.10	Present study.	
33-Baluchis.Sistan & Baluchistan	111	63	41	7	75.23	24.77	Present study.	

Table 5.1.3.a. II MN blood groups distribution in the Caucasus tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
1-Azerbaijanians.Barda	147	38	87	22	55.43	44.57	Voronov, A.A.	1973
2-Dagestan.Various tribes	161	75	39	47	58.70	41.30	Kherumian, R.	1948
3-Caucasus(different regions)	641	218	321	102	59.05	40.95	Voronov, A.A.,et al.	1967
4-Azerbaijanians. Shemakha	180	70	74	36	59.44	40.56	Voronov, A.A.	1973
5-Gurians.Lanchkhut	98				61.23	38.77	Voronov ,A.A.	1973
6-Georgia,Gori	428	164	204	60	62.15	37.85	Solovyeva, T.G.	1967
7- Kakhetians ,Gurdjaani	196	73	99	24	62.50	37.50	Voronov, A.A.	1973
8-Georgia,Tbilisi	500	198	230	72	62.60	37.40	Boyd,W.C.,et al.	1937
9-Dagestan.Kumky	178	79	66	33	62.92	37.08	Gadzhiev, A.G.	1964
10-Azerbaijanians. Nukha	248	104	105	39	63.15	36.85	Voronov, A.A.	1973
11-Megrelians.Abashi	192	85	75	32	63.85	36.15	Voronov, A.A.	1973
12-Georgia,Abkhazskaya. Svanis	655	275	308	72	65.50	34.50	Verbitsky,M.Sh., et al.	1971

Table 5.1.3.a. II (Cont.) MN blood groups distribution in the Caucasus tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
13-Georgians abroad . Autochthonous	41	20	14	7	65.85	34.15	Kherumian,R., et al.	1954
14-Dagestan.Darghian	179	82	77	20	67.32	32.68	Gadzhiev, A.G.	1964
15-Dagestan. Lezgian	196	98	72	26	68.37	31.63	Gadzhiev, A.G.	1964
16-Dagestan.Lakts	151	76	55	20	68.54	31.46	Gadzhiev, A.G.	1964
17-Abkhazians.Ochamchiri	91				70.88	29.12	Voronov, A.A.	1973
18-Dagestan.Avar	200	100	84	16	71.00	29.00	Gadzhiev, A.G.	1964

Table 5.1.3.a. III MN blood groups distribution in Turkey tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
1-Turks.Asia Minor, Mersin	108	27	52	29	49.07	50.93	Aksoy,M., et al..	1958
2-Turkish	1266	411	619	236	56.91	43.09	Baecher.	1935
3-Kurds					57.00	43.00	Richard, P.	1976
4-Turkish Donors	2361	763	1172	426	57.14	42.86	Mizan, N., et al.	1965
5-Eti- Turks. Asia Minor near Mersin	116	43	47	26	57.33	42.67	Aksoy,M., et al.	1958

Table 5.1.3.a. IV MN blood groups distribution in Iraq tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
1-Kurdish Jews	27	4	11	12	35.19	64.81	Godber,Marilyn J., et al.	1973
2-Kurdish Jews.South east	50	11	21	18	43.00	57.00	Tills, D., et al.	1977
3-Jews.Baghdad	215	57	101	57	50.00	50.00	Boyd,W.C., & Boyd, Lyle G.	1941
4-Jews. Kurdistan	120	37	53	30	52.92	47.08	Gurevitch,J., & Margolis, E.	1955
5-Kurds					56.00	44.00	Al-Khafagi,S.D., et al.	1976
6-Bedouins	139	47	64	28	56.83	43.17	Kayssi,A.I.,et al.	1938
7-Moslems.Baghdad	387	143	182	62	60.47	39.53	Boyd,W.C., & Boyd,Lyle G.	1941
8-Jews. Baghdad	162	66	64	32	60.49	39.51	Gurevitch,J., & Margolis, E.	1955
9-Assyrians	99	37	46	16	60.61	39.39	Ikin,Elizabeth W., et al.	1956

Table 5.1.3.a. IV (Cont.) MN blood groups distribution in Iraq tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
10-Bedouins.Mosul	206	76	102	28	61.65	38.35	Boyd, W.C.	1939
11-Christians.Baghdad	63	24	30	9	61.90	38.10	Boyd,W.C., and Boyd, Lyle.G.	1941
12-Kurdish Jews.North west	61	29	20	12	63.93	36.07	Tills.D., et al.	1977
13-Bedouins	199	82	99	18	66.08	33.92	Kayssi,A.I.,et al.	1938
14-Kurds.Baghdad	29	17	8	4	72.41	27.59	Boyd,W.C., and Boyd, Lyle,G.	1941
15-Karaite Jews	72	70	2	-	98.61	1.39	Goldschmidt,E., et al.	1976

Table 5.1.3.a. V MN blood groups distribution in Kuwait tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
1-Suluba tribe	51	8	37	6	51.96	48.04	Khaled, E., et al.	1981
2-General population	73	16	47	10	54.11	45.89	Khaled, E., et al.	1981
3-Kuwaiti Arabs	159	52	88	19	60.38	39.62	Sawhney,K.S.	1975
4-Ajman tribe	52	22	29	1	70.19	29.81	Khaled,E., et al.	1981

Table 5.1.3.a. VI MN blood groups distribution in Saudi Arabia tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		MM	MN	NN	M	N	
1-Shias.Qatif and Hasa Oases	463	188	208	67	63.07	36.93	Maranjian,G.,et al. 1966
2-Sunnis.Qatif and Hasa Oases	323	151	134	38	67.49	32.51	Maranjian,G.,et al. 1966
3-Sunnis.Hejaz,Asir,Najran	176	100	61	15	74.15	25.85	Maranjian,G.,et al. 1966
4-Sunnis. Najd	180	104	62	14	75.00	25.00	Maranjian,G.,et al. 1966
5-Bedouins	178	109	59	10	77.81	22.19	Maranjian,G.,et al. 1966
6-Western Saudi Arabia	176	145	18	13	87.50	12.50	Saha, N.,et al. 1980

Table 5.1.3.a. VII MN blood groups distribution in the United Arab Emirates tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		MM	MN	NN	M	N	
1-Abu-Dhabians	100	39	44	17	61.00	39.00	Kamel,K.,et al. 1980

Table 5.1.3.a VIII MN blood groups distribution in Pakistan tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		MM	MN	NN	M	N		
1-Bengalis. Dacca	230	79	109	42	58.04	41.96	Boyd,W.C., & Boyd,L.G.	1954
2-Punjabis.Lahore	202	69	97	36	58.17	41.83	Boyd,W.C., & Boyd,L.G.	1954
3-Gilgit.Hunza	23	10	8	5	60.87	39.13	Ikin,Elizabeth W., et al.	1959
4-Peshawar	153	64	73	16	65.69	34.31	Boyd,W.C., & Boyd,L.G.	1954
5-Baltis.Baltistan	80	38	32	10	67.50	32.50	Clegg,E.J.,et al.	1961
6-Pathans.Swat.Saidu Sharif	132	65	49	18	67.80	32.20	Alciati, G.	1968

Table 5.1.3.a IX MN blood groups distribution in Afghanistan tested with anti-M and anti-N sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		MM	MN	NN	M	N	
1-Pushtus	104	39	51	14	62.02	37.98	Papiha, S.S.,et al. 1977
2-Daris	179	82	67	30	64.53	35.47	Papiha, S.S.,et al. 1977
3-Timuri and related tribes	118	52	52	14	66.10	33.90	Woodd-Walker,R.B., 1967 et al.

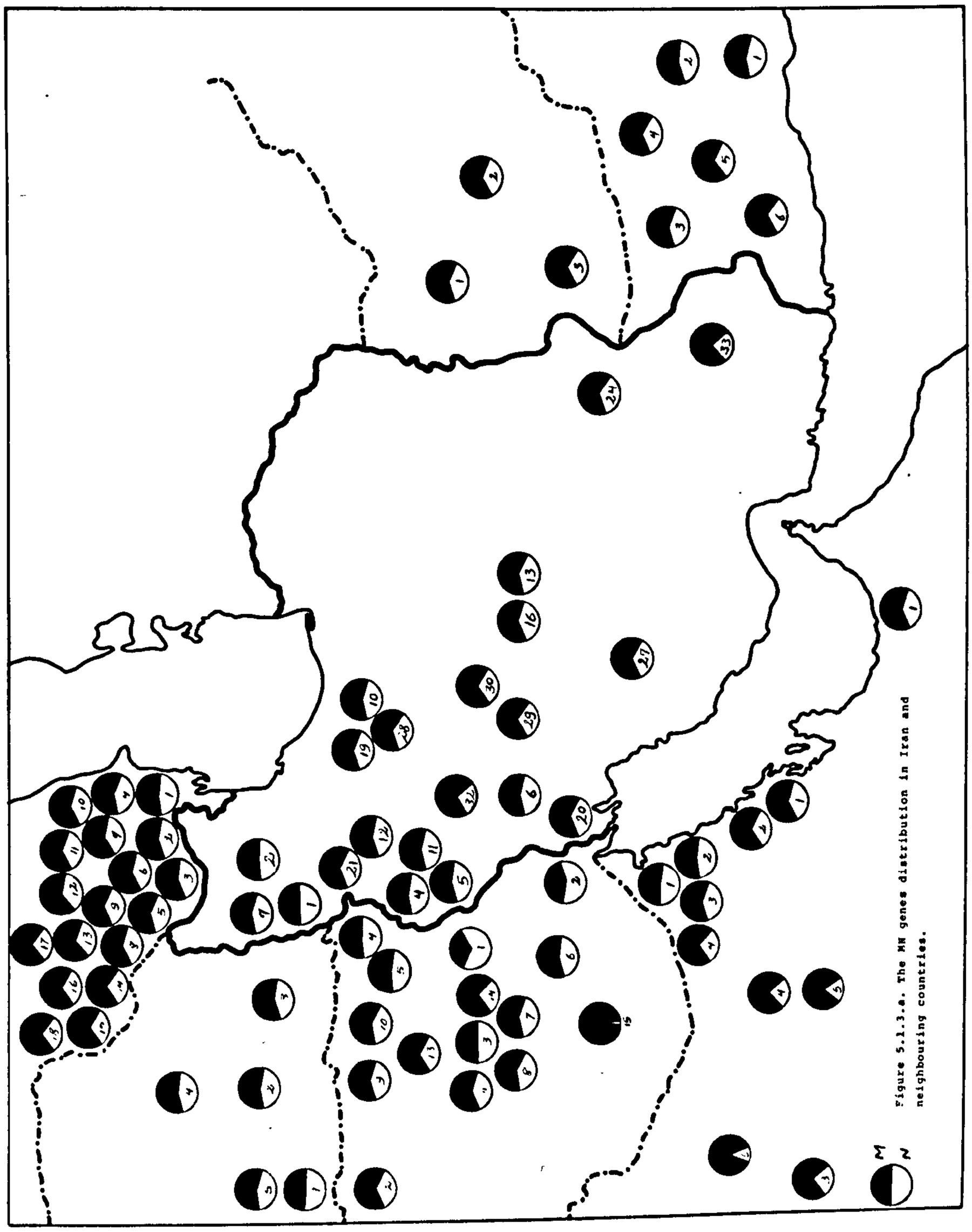


Figure 5.1.3.4. The MM genes distribution in Iran and neighbouring countries.

Table 5.1.3.b. I MNSs blood groups distribution in Iran tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Phenotypes					
		MMSS	MMss	MNSS	MNss	NNSS	NNss
1-North	58	5	19	13	11	1	9
2-Kurdish Jews	94	12	15	30	23	8	6
3-Tehran	113	20	23	20	29	10	11
4-Kurdish Jews	106	13	17	35	25	8	8
5-East	67	13	15	10	20	3	6
6-Iranians	56	13	6	10	18	6	3
7-Central and South	109	25	23	21	20	8	12
8-North West	66	15	16	12	13	2	8
9-West	107	24	24	24	20	5	10
10-Ghashghais	37	12	2	9	10	1	3

Table 5.1.3.b.I.(Cont). MNSs blood groups distribution in Iran tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Gene complex frequencies				Authors	
		MS	Ms	NS	Ns		
1-North	58	10.24	51.92	7.80	30.04	Bajatzadeh,M., & Walter, H.	1969
2-Kurdish Jews	94	15.18	41.73	16.36	26.73	Godber,Marilyn.J., et al.	1973
3-Tehran	113	15.32	44.47	10.05	30.16	Bajatzadeh,M., & Walter, H.	1969
4-Kurdish Jews	106	15.70	40.90	15.60	27.80	Tills,D., et al.	1977
5-East	67	16.34	47.93	5.55	30.18	Bajatzadeh,M., & Walter, H.	1969
6-Iranians	56	19.90	39.08	10.73	30.29	Nijenhuis, L.E.	1964
7-Central and South	109	20.03	42.87	8.99	28.11	Bajatzadeh,M., & Walter, H.	1969
8-North West	66	20.44	45.52	4.74	29.30	Bajatzadeh,M.,& Walter, H.	1969
9-West	107	20.89	44.61	8.15	26.35	Bajatzadeh,M., & Walter, H.	1969
10-Ghashghais	37	33.18	30.40	3.43	32.99	Nijenhuis,L.E.	1964

Table 5.1.3. C.I MNSs blood groups distribution in Iran tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Phenotypes								
		MMSS	MMSs	MMss	MNSS	MNSs	MNss	NNSS	NNSs	NNss
1-Kurds.Rezaieh	124	2	11	18	2	19	40	2	9	21
2-Turks.Rezaieh	139	4	19	26	7	28	30	4	11	10
3-Zoroastrians	74	2	12	13	4	16	16	2	4	5
4-Arabs.Khuzistan	97									
5-Shi'a.Yazd	151	10	25	22	10	30	35	3	4	12
6-Zabolis.Sistan & Baluchistan	114	6	28	18	4	18	23	2	4	11
7-Kurds.Sanandaj	107	11	22	11	4	17	21	3	7	11
8-Kurds.Baneh,Marivan	77	11	13	9	7	10	15	2	3	7
9-Tehran	137	18	19	23	9	32	21	2	4	9
10-Lurs.Luristan	153	22	41	20	9	30	15	1	6	9
11-Baluchis.Sistan & Baluchistan	111	27	28	8	10	22	9	1	2	4

Table 5.1.3.C.I.(Cont.) MNSs blood groups distribution in Iran tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Gene complex frequencies				Authors	
		MS	Ms	NS	Ns		
1-Kurds.Rezaieh	124	11.99	37.60	10.23	40.18	Present study.	
2-Turks.Rezaieh	139	16.15	42.48	15.73	25.64	Present study.	
3-Zoroastrians	74	18.01	42.80	14.24	24.95	Present study.	
4-Arabs.Khuzistan	97	21.21	36.52	7.14	35.13	Marzban,M.	1978
5-Shi'a,Yazd	151	24.62	38.04	10.29	27.05	Sunderland,E.,& Smith, H.M.	1966
6-Zabolis.Sistan & Baluchistan	114	24.62	40.73	7.84	26.81	Present study .	
7-Kurds.Sanandaj	107	28.06	32.69	10.26	28.99	Lehmann, H., et al.	1973
8-Kurds.Baneh,Marivan	77	31.79	31.85	11.06	25.30	Lehmann, H., et al.	1973
9-Tehran	137	31.87	34.55	9.38	24.20	Sawhney, K.S.	1975
10-Lurs.Luristan	153	36.81	35.08	7.02	21.09	Present study.	
11-Baluchis.Sistan & Baluchistan	111	48.95	26.28	7.08	17.69	Present study.	

Table 5.1.3.d.I. MN blood groups distribution in Iran tested with anti-He (Henshaw) serum

Population	Number Tested	He+	He-	He+%	Authors	
1-Kurds.Kermanshah	122	-	122		Nijenhuis,L.E.	1964
2-Ghashghai.Fars.South of Shiraz	8	-	8		Nijenhuis,L.E.	1964
3-Bakhtiari.Zagros Mountain	131	-	131		Nijenhuis,L.E.	1964
4-Iranians	76	-	76		Nijenhuis,L.E.	1964
5-Assyrians	14	-	14		Nijenhuis,L.E.	1964
6-Armenians	9	-	9		Nijenhuis,L.E.	1964
7-Kurdish Jews	121	-	121		Godber,Marilyn,J., et al.	1973
8-Kurds.Sanandaj	107	-	107		Lehmann,H.,et al.	1973
9-Kurds.Baneh,Marivan	77	-	77		Lehmann,H.,et al.	1973
10-Kurdish Jews	106	-	106		Tills,D., et al.	1977

Table 5.1.3. * I MNSs-complex frequencies distribution in Iran

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-North	58	10.24	51.92	7.80	30.04	Bajatzadeh,M.,& Walter, H. 1969
2-Kurds.Rezaieh	124	11.99	37.60	10.23	40.18	Present study.
3-Kurdish Jews	94	15.18	41.73	16.36	26.73	Godber,Marilyn J., et al. 1973
4-Tehran	113	15.32	44.47	10.05	30.16	Bajatzadeh,M., & Walter, H. 1969
5-Kurdish Jews	106	15.70	40.90	15.60	27.80	Tills, D., et al. 1977
6-Turks.Rezaieh	139	16.15	42.48	15.73	25.64	Present study.
7-East	67	16.34	47.93	5.55	30.18	Bajatzadeh,M., & Walter, H. 1969
8-Zoroastrians	74	18.01	42.80	14.24	24.95	Present study.
9-Iranians	56	19.90	39.08	10.73	30.29	Nijenhuis,L.E. 1964
10-Central & South	109	20.03	42.87	8.99	28.11	Bajatzadeh,M.,& Walter, H. 1969
11-North west	66	20.44	45.52	4.74	29.30	Bajatzadeh,M., & Walter,H. 1969

Table 5.1.3. * I (Cont.) MNSs-complex frequencies distribution in Iran

Population	Number Tested	Gene complex frequencies				Authors	
		MS	Ms	NS	Ns		
12-West	107	20.89	44.61	8.15	26.35	Bajatzadeh,M., & Walter, H.	1969
13-Arabs.Khuzistan	97	21.21	36.52	7.14	35.13	Marzban, M.	1978
14-Shi'a . Yazd	151	24.62	38.04	10.29	27.05	Sunderland,E.,& Smith, H.M.	1966
15-Zabolis. Sistan & Baluchistan	114	24.62	40.73	7.84	26.81	Present study.	
16-Kurds. Sanandaj	107	28.06	32.69	10.26	28.99	Lehmann, H.,et al.	1973
17-Kurds.Baneh, Marivan	77	31.79	31.85	11.06	25.30	Lehmann, H.,et al.	1973
18-Tehran	137	31.87	34.55	9.38	24.20	Sawhney, K.S.	1975
19-Ghashghais	37	33.18	30.40	3.43	32.99	Nijenhuis, L.E.	1964
20-Lurs.Luristan	153	36.81	35.08	7.02	21.09	Present study.	
21-Baluchis.Sistan & Baluchistan	111	48.95	26.28	7.08	17.69	Present study.	

Table 5.1.3.b.* II MNSs blood groups distribution in the Caucasus tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Phenotypes					
		MMSS	MMss	MNSS	MNss	NNSS	NNss
1-Svanis.. Georgia.Abkhazskaya	655	196	79	206	102	38	34
2-Georgians	41	17	3	10	4	5	2

Table 5.1.3.b.*II (Cont.). MNSs blood groups distribution in the Caucasus tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Svanis . Georgia.Abkhazskaya	655	31.02	34.48	11.67	22.83	Verbitsky,M.Sh.,et al.1971
2-Georgians	41	38.85	27.08	14.50	19.57	Kherumian,R.,et al. 1954

Table 5.1.3.b.* III MNSs blood groups distribution in Turkey tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Phenotypes					
		MMSS	MMss	MNSS	MNss	NNSS	NNss
1-Turks.Asia Minor, Mersin	108	18	9	24	28	10	19
2-Eti-Turks. Asia Minor, near Mersin	116	36	7	28	19	10	16

Table 5.1.3.b.*III (Cont.) MNSs blood groups distribution in Turkey tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Gene complex frequencies				Authors	
		MS	Ms	NS	Ns		
1-Turks. Asia Minor, Mersin	108	19.04	30.11	9.07	41.78	Aksoy,M.,et al.	1958
2-Eti-Turks.Asia Minor, near Mersin	116	31.96	25.43	8.52	34.09	Aksoy,M.,et al.	1958

Table 5.1.3.b. IV MNSs blood groups distribution in Iraq tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Phenotypes					
		MMSS	MMss	MNSS	MNss	NNSS	NNss
1-Kurdish Jews	27	2	2	3	8	9	3
2-Kurdish Jews. North west	61	15	14	10	10	7	5
3-Kurdish Jews; South east	50	10	1	14	7	9	9
4-Kurds							
5-Assyrians	99	32	5	32	14	10	6

Table 5.1.3.b. IV (Cont.) MNSs blood groups distribution in Iraq tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Kurdish Jews	27	5.98	29.21	25.02	39.79	Godber,Marilyn.J., et al. 1973
2-Kurdish Jews.North west	61	18.99	44.94	12.07	24.00	Tills,D., et al. 1977
3-Kurdish Jews. South east	50	26.41	16.59	15.76	41.24	Tills,D., et al. 1977
4-Kurds		27.00	29.00	27.00	17.00	Al-Khafaji,S.D.,et al.1976
5-Assyrians	99	36.10	24.59	13.78	25.53	Ikin,Elizabeth W., et al. 1965

Table 5.1.3.C. IV MNSs blood groups distribution in Iraq tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Phenotypes								
		MMSS	MMSs	MMss	MNSS	MNSs	MNss	NNSS	NNSs	NNss
1-Karaite Jews	72	2	22	46	-	-	2	-	-	-

Table 5.1.3.C.IV (Cont.) MNSs blood groups distribution in Iraq tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Karaite Jews	72	18.00	80.60	0.00	1.40	Goldschmidt,Elizabeth., 1976 et al.

Table 5.1.3.d. IV MN blood groups distribution in Iraq tested with anti-He (Henshaw) serum

Population	Number Tested	He+	He-	He+%	Authors
1-Kurdish Jews..South east	50	-	50		Tills,D., et al. 1977
2-Kurdish Jews..North west	61	-	61		Tills,D., et al. 1977

Table 5.1.3. * IV. MNSs - complex frequencies distribution in Iraq

Population	Number Tested	Gene complex frequencies				Authors	
		MS	Ms	NS	Ns		
1-Kurdish Jews	27	5.98	29.21	25.02	39.79	Godber,Marilyn J.,et al.	1973
2-Karaite Jews	72	18.00	80.60	0.00	1.40	Goldschmidt,Elizabeth., et al.	1976
3-Kurdish Jews. North west	61	18.99	44.94	12.07	24.00	Tills,D., et al.	1977
4-Kurdish Jews. South east	50	26.41	16.59	15.76	41.24	Tills,D., et al.	1977
5-Kurds		27.00	29.00	27.00	17.00	Al-Khafaji,S.D.,et al.	1976
6-Assyrians	99	36.10	24.59	13.78	25.53	Ikin,Elizabeth W.,et al.	1965

Table 5.1.3.b. V MNSs blood groups distribution in Kuwait tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Phenotypes					
		MMSS	MMss	MNSS	MNss	NNSS	NNss
1- Kuwaiti Arabs	159	29	23	43	45	3	16

Table 5.1.3.b. V (Cont.). MNSs blood groups distribution in Kuwait tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Kuwaiti Arabs	159	22.24	38.14	5.09	34.53	Sawhney, K.S. 1975

Table 5.1.3.C.V MNSs blood groups distribution in Kuwait tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Phenotypes								
		MMSS	MMSs	MMss	MNSS	MNSs	MNss	NNSS	NNSs	NNss
1-Suluba tribe	51	1	3	4	4	21	12	2	2	2
2-General population	73	5	3	8	13	18	16	1	5	4
3-Ajman tribe	52	7	8	7	14	11	4	-	1	-

Table 5.1.3.C.V (Cont.) MNSs blood groups distribution in Kuwait tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Gene complex frequencies				Authors	
		MS	Ms	NS	Ns		
1-Suluba tribe	51	14.00	38.00	25.00	23.00	Khaled,E., et al.	1981
2-General population	73	24.00	30.00	20.00	26.00	Khaled,E., et al.	1981
3-Ajman tribe	52	38.00	32.00	22.00	8.00	Khaled,E., et al.	1981

Table 5.1.3. * V MNSs- complex frequencies distribution in Kuwait

Population	Number Tested	Gene complex frequencies				Authors	
		MS	Ms	NS	Ns		
1-Suluba tribe	51	14.00	38.00	25.00	23.00	Khaled,E., et al.	1981
2-Kuwaiti Arabs	159	22.24	38.14	5.09	34.53	Sawhney, K.S.	1975
3-General population	73	24.00	30.00	20.00	26.00	Khaled, E., et al.	1981
4-Ajman tribe	52	38.00	32.00	22.00	8.00	Khaled, E., et al.	1981

Table 5.1.3.b. VI MNSs blood groups distribution in Saudi Arabia tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Phenotypes					
		MMSS	MMss	MNSS	MNss	NNSS	NNss
1-Shias.Qatif & Haza Oases	463	123	65	109	99	27	40
2-Sunnis.Qatif & Haza Oases	323	92	59	75	59	8	30
3-Sunnis.Hejaz,Asir, Najran	176	69	31	40	21	6	9
4-Bedouins	178	73	36	44	15	5	5
5-Sunnis.Najd	180	74	30	39	23	3	11

Table 5.1.3.b. VI (Cont.) MNSs blood groups distribution in Saudi Arabia tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Shias.Qatif & Haza Oases	463	25.61	37.49	8.07	28.83	Maranjian,G.,et al. 1966
2-Sunnis. Qatif & Haza Oases	323	27.45	40.09	4.81	27.65	Maranjian,G.,et al. 1966
3-Sunnis.Hejaz,Asir,Najran	176	34.07	40.111	7.11	18.71	Maranjian,G.,et al. 1966
4-Bedouins	178	34.31	43.53	9.63	12.53	Maranjian,G.,et al. 1966
5-Sunnis.Najd	180	36.50	38.52	3.85	21.13	Maranjian,G.,et al. 1966

Table 5.1.3.C. VI MNSs blood groups distribution in Saudi Arabia tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Phenotypes								
		MMSS	MMSs	MMss	MNSS	MNSs	MNss	NNSS	NNSs	NNss
1-Western Saudi Arabia	176	38	26	81	1	7	10	3	2	8

Table 5.1.3.C.VI (Cont.) MNSs blood groups distribution in Saudi Arabia tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Western Saudi Arabia	176	30.44	57.06	3.37	9.13	Saha, N., et al. 1980

Table 5.1.3.d. VI MN blood groups distribution in Saudi Arabia tested with anti-He (Henshaw) serum

Population	Number Tested	He+	He-	He+%	Authors	
1-Bedouins	156	-	156		Maranjian,G.,et al.	1966
2-Sunnis.Najd	180	-	180		Maranjian,G.,et al.	1966
3-Sunnis.Asir,Hejaz, Najran	168	-	168		Maranjian,G.,et al.	1966
4-Shias.Qatif & Hasa Oases	412	4	408	0.97	Maranjian,G.,et al.	1966
5-Sunnis. Qatif & Hasa Oases	287	4	283	1.39	Maranjian,G.,et al.	1966

Table 5.1.3. *. VI MNSs - complex frequencies distribution in Saudi Arabia

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Shias.Qatif & Hasa Oases	463	25.61	37.49	8.07	28.83	Maranjian,G.,et al. 1966
2-Sunnis.Qatif & Hasa Oases	323	27.45	40.09	4.81	27.65	Maranjian,G.,et al. 1966
3-Western Saudi Arabia	176	30.44	57.06	3.37	9.13	Saha,N.,et al. 1980
4-Sunnis,Hejaz,Asir,Najran	176	34.07	40.11	7.11	18.71	Maranjian,G.,et al. 1966
5-Bedouins	178	34.31	43.53	9.63	12.53	Maranjian,G.,et al. 1966
6-Sunnis.Najd	180	36.50	38.52	3.85	21.13	Maranjian,G.,et al. 1966

Table 5.1.3.b.*VII MNSs blood groups distribution in the United Arab Emirates tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Phenotypes					
		MMSS	MMss	MNSS	MNss	NNSS	NNss
1-Abu-Dhabians	100	25	14	25	19	8	9

Table 5.1.3.b.*VII (Cont.) MNSs blood groups distribution in the United Arab Emirates tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Abu-Dhabians	100	24.70	36.30	10.50	28.50	Kamel,K.,et al. 1980

Table 5.1.3.b.*VIII MNSs blood groups distribution in Pakistan tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Phenotypes					
		MMSS	MMss	MNSS	MNss	NNSS	NNss
1-Punjabis.Lahore	202	34	35	53	44	17	19
2-Bengalis.Dacca	230	44	35	62	47	21	21
3-Pathans.Swat,Saidu Sharif	132	38	27	30	19	11	7
4-Baltis.Baltistan	80	24	14	18	14	7	3
5-Gilgit.Hunza	23	7	3	5	3	2	3
6-Peshawar	153	46	18	45	28	5	11

Table 5.1.3.b*.VIII (Cont.) MNSs blood groups distribution in Pakistan tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Gene complex frequencies				Authors	
		MS	Ms	NS	Ns		
1-Punjabis.Lahore	202	17.71	40.51	12.67	29.11	Boyd,W.C., & Boyd, Lyle.G.	1954
2-Bengalis.Dacca	230	20.09	38.00	13.02	28.89	Boyd,W.C., & Boyd, Lyle.G.	1954
3-Pathans.Swat,Saidu Sharif	132	24.38	43.49	12.34	19.79	Alciati,G.	1968
4-Baltis.Baltistan	80	25.48	42.09	12.35	20.08	Clegg,E.J.,et al.	1961
5-Gilgit.Hunza	23	28.41	32.47	9.28	29.84	Ikin,Elizabeth W., et al.	1959
6-Peshawar	153	32.16	33.58	6.65	27.61	Boyd,W.C., & Boyd, Lyle.G.	1954

Table 5.1.3.b. IX MNSs blood groups distribution in Afghanistan tested with anti-M, anti-N, and anti-S sera

Population	Number Tested	Phenotypes					
		MMSS	MMss	MNSS	MNss	NNSS	NNss
1-Timuri & related tribes	118	27	25	20	32	8	6

Table 5.1.3.b. IX (Cont). MNSs blood groups distribution in Afghanistan tested with anti-M , anti-N, and anti-S sera

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Timuri & related tribes	118	17.26	48.84	9.67	24.23	Woodd-Walker,R.B., 1967 et al.

Table 5.1.3.C. IX MNSs blood groups distribution in Afghanistan tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Phenotypes								
		MMSS	MMSs	MMss	MNSS	MNSs	MNss	NNSS	NNSs	NNss
1-Daris	179	17	37	28	24	29	34	2	13	15
2-Pushtus	104	6	21	12	5	28	18	1	3	10

Table 5.1.3.C. IX (Cont.) MNSs blood groups distribution in Afghanistan tested with anti-M, anti-N, anti-S, and anti-s sera

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Daris	179	25.70	38.80	9.20	26.30	Papiha,S.S.,et al. 1977
2-Pushtus	104	29.00	33.00	7.50	30.50	Papiha,S.S.,et al. 1977

Table 5.1.3. * IX MNSs - complex frequencies distribution in Afghanistan

Population	Number Tested	Gene complex frequencies				Authors
		MS	Ms	NS	Ns	
1-Timuri & related tribes	118	17.26	48.84	9.67	24.23	Woodd-Walker,R.B.,et al.1967
2-Daris	179	25.70	38.80	9.20	26.30	Papiha,S.S.,et al. 1977
3-Pushtus	104	29.00	33.00	7.50	30.50	Papiha,S.S.,et al. 1977

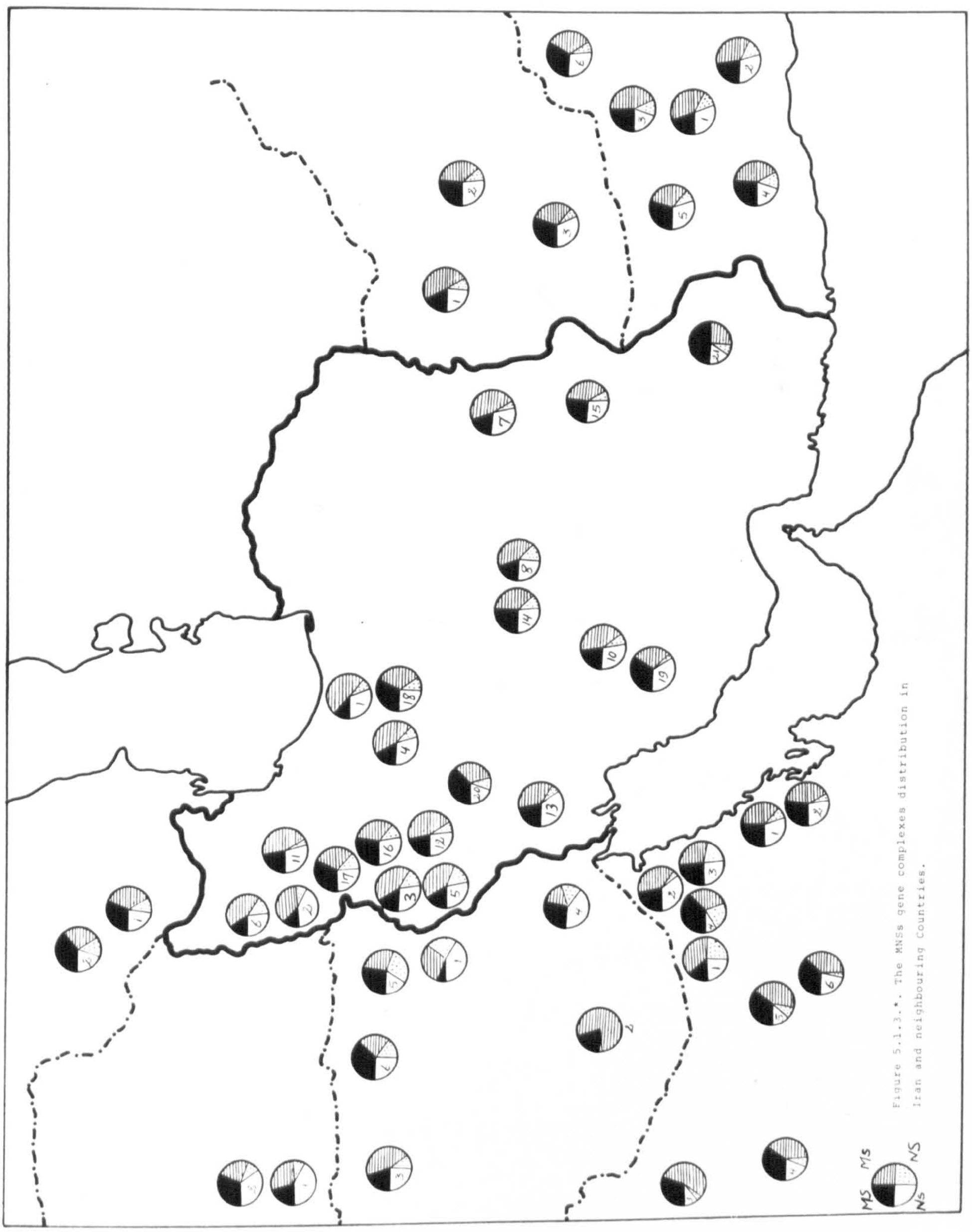


Figure 5.1.3.*. The MNSs gene complexes distribution in Iran and neighbouring Countries.

5.1.4. The P blood group system

The distribution of the P blood groups and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.1.4.

The frequency of the P_1 gene ranges from 23.27 to 53.22 percent in Iranians, being lowest in the Turks of Rezaieh (present investigation) and highest in the Assyrians(Nijenhuis, 1964). With the exception of the Turks of Rezaieh, values obtained in the present investigation are within the range of variation.

On the whole, with an average P_1 frequency of 42.62 percent, the Iranian population appears to exhibit a lower P_1 gene frequency than that of over 50 percent found in Europeans (Mourant et al, 1976).

In general, the allele P_1 does not show much variability within Iran.

Regarding neighbouring areas, the frequency of the gene P_1 in the populations of the Caucasus varies between 46.79 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1971) and 53.15 percent in the Georgians (Kherumian et al, 1954). With an average P_1 frequency of 49.97 percent, the population of the Caucasus seems to show a higher P_1 frequency than that found in Iranians. The P_1 frequency in the Caucasus is similar to that in Europe.

The P_1 gene frequency of 35.00 percent in the Kurds of Turkey (Richard, 1976) is much lower than the values ranging from 47.50 to 51.65 percent in the Kurds of western Iran (Nijenhuis, 1964; and Lehmann et al, 1973).

The frequency of the P_1 gene in the populations of Iraq

ranges from 21.26 percent in the Kurdish Jews of the south east(Tills et al, 1977) to 41.40 percent in the Assyrians (Ikin et al, 1965).

With an average P_1 frequency of 30.34 percent, the Iraqi population appears to exhibit a much lower frequency of the P_1 gene than that found in Iranians. The Iraqi Kurds and Kurdish Jews seem to show much lower P_1 frequencies than those found in the same two population groups in Iran.

The P_1 gene frequency in the Kuwaiti populations varies between 45.00 percent in the Ajman tribe and 61.00 percent in the Suluba tribe (Khaled et al, 1981). With an average P_1 frequency of 52.67 percent, the population of Kuwait has a higher P_1 frequency than that found in Iranians and is more similar to the European frequency.

The frequency of the P_1 gene in the populations of Saudi Arabia ranges from 42.42 percent in the Bedouins to 57.18 percent in the Sunni sample of Najd(Maranjian et al, 1966). With an average P_1 frequency of 50.78 percent, the population of Saudi Arabia, like that of Kuwait, appears to exhibit a higher P_1 frequency than that found in Iranians and more similar to the European frequency.

The P_1 frequency of 46.10 percent in the Abu-Dhabians of the United Arab Emirates (Kamel et al, 1980) is also higher than in Iran but lower than the European frequency.

The frequency of the P_1 gene in the populations of Pakistan varies between 35.77 percent in the Baltis(Clegg et al, 1961) and 48.92 percent in the Gilgit, Hunza (Ikin et al, 1959). With an average P_1 frequency of 40.52 percent, the Pakistani population seems to show a slightly

lower P_1 frequency than that found in Iranians and lower than the European frequency.

The P_1 gene frequency in the populations of Afghanistan ranges from 38.93 percent in the Timuri and related tribes (Woodd-Walker et al, 1967) to 50.40 percent in the Daris (Papiha et al, 1977). With an average P_1 frequency of 42.98 percent, the Afghan population appears to exhibit a similar P_1 frequency to that found in Iranians and lower than the frequency in Europeans.

Conclusion

In the European populations the gene P_1 has a frequency of slightly over 50 percent. In Negroes the frequency is much higher, around 80 percent. In India and south east Asia frequencies are mostly similar to those found in Europe but in eastern Asia is considerably lower (Mourant et al,1976).

In the Iranian and neighbouring populations, with the exception of the Arab populations of Kuwait and Saudi Arabia with relatively higher values, the frequency of the P_1 gene is below the typical European level, though tending rather towards the low values found in Asia than to the high ones in Africa. The low frequency of P_1 is consistent with the suggestion of Mongoloid contribution to the gene pool.

Table 5.1.4. I P blood groups distribution in Iran tested with anti-P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		P_1	P_2	P_1	P_2+ P	
1-Turks.Rezaieh	141	58	83	23.27	76.73	Present study.
2-Jews	108	51	57	27.35	72.65	Tabatabai,H. 1977
3-Zoroastrians	86	41	45	27.66	72.34	Present study.
4-Kurds.Rezaieh	138	74	64	31.90	68.10	Present study.
5-North	58	35	23	37.02	62.98	Bajatzadeh,M.,& Walter, H. 1969
6-Tehran	112	71	41	39.49	60.51	Bajatzadeh,M.,& Walter, H. 1969
7-Kurdish Jews	106	71	35	42.54	57.46	Tills,D.,et al. 1977
8-Ghashghais.Fars	66	45	21	43.59	56.41	Nijenhuis,L.E. 1964
9-Armenians	143	98	45	43.90	56.10	Tabatabai,H. 1977
10-Bakhtiaris	137	94	43	43.97	56.03	Nijenhuis,L.E. 1964
11-Central and South	106	73	33	44.21	55.79	Bajatzadeh,M.,& Walter, H. 1969
12-West	106	73	33	44.21	55.79	Bajatzadeh,M.,& Walter, H. 1969

Table 5.1.4.I (Cont.) P blood groups distribution in Iran tested with anti-P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		P_1	P_2	P_1	P_2+P		
13-Kurdish Jews	94	65	29	44.46	55.54	Godber,Marilyn. J., et al.	1973
14-East	66	46	20	44.95	55.05	Bajatzadeh,M.,& Walter, H.	1969
15-North west	65	46	19	45.94	54.06	Bajatzadeh,M.,& Walter,H.	1969
16-Kurds.Kermanshah	127	92	35	47.50	52.50	Nijenhuis,L.E.	1964
17-Turks.Mainly south East	348	256	92	48.58	51.42	Nijenhuis,L.E.	1964
18-Kurds.Sanandaj	106	78	28	48.60	51.40	Lehmann,H.,et al.	1973
19-Armenians	78	59	19	50.64	49.36	Nijenhuis,L.E.	1964
20-Kurds.Baneh,Marivan	77	59	18	51.65	48.35	Lehmann,H.,et al.	1973
21-Arabs.Abadan	158	123	35	52.94	47.06	Nijenhuis,L.E.	1964
22-Assyrians	32	25	7	53.22	46.78	Nijenhuis,L.E.	1964

Table 5.1.4.II P blood groups distribution in the Caucasus tested with anti-P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		P_1	P_2	P_1	P_2+P		
1-Svanis.Georgia, Abkhazskaya	650	466	184	46.79	53.21	Verbitsky,M.Sh., et al.	1971
2-Georgians	41	32	9	53.15	46.85	Kherumian,R.,et al.	1954

Table 5.1.4. III P blood groups distribution in Turkey tested with anti-P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		P_1	P_2	P_1	P_2+P	
1-Kurds				35.00	65.00	Richard, P. 1976

Table 5.1.4. IV P blood groups distribution in Iraq tested with anti-P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		P_1	P_2	P_1	P_2+P	
1-Kurdish Jews.south east	50	19	31	21.26	78.74	Tills,D.,et al. 1977
2-Kurds				27.00	73.00	Al-Khafaji,S.D.,et al. 1976
3-Kurdish Jews. North west	61	30	31	28.71	71.29	Tills,D.,et al. 1977
4-Kurdish Jews	27	15	12	33.34	66.66	Godber,Marilyn J.,et al.1973
5-Assyrians	99	65	34	41.40	58.60	Ikin,Elizabeth W.,et al.1965

Table 5.1.4.V P blood groups distribution in Kuwait tested with anti-P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		P_1	P_2	P_1	P_2+P	
1-Ajman tribe	52	36	16	45.00	55.00	Khaled,E.,et al.1981
2-General population	74	57	17	52.00	48.00	Khaled,E.,et al.1981
3-Suluba tribe	52	44	8	61.00	39.00	Khaled,E.,et al.1981

Table 5.1.4. VI P blood groups distribution in Saudi Arabia tested with anti-P_1 serum

Population	Number Tested	Phenotypes		Gene fruequencies		Authors
		P_1	P_2	P_1	P_2+P	
1-Bedouins	178	119	59	42.42	57.58	Maranjian,G.,et al. 1966
2-Shias.Qatif & Hasa Oases	465	320	145	44.16	55.84	Maranjian,G.,et al. 1966
3-Sunnis.Qatif & Hasa Oases	323	253	70	53.45	46.55	Maranjian,G.,et al. 1966
4-Sunnis.Asir,Hejaz, Najran	176	143	33	56.70	43.30	Maranjian,G.,et al. 1966
5-Sunnis. Najd	180	147	33	57.18	42.82	Maranjian,G.,et al. 1966

Table 5.1.4. VII P blood groups distribution in the United Arab Emirates tested with anti- P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		P_1	P_2	P_1	P_2+P	
1-Abu-Dhabians	100	71	29	46.10	53.90	Kamel,K.,et al. 1980

Table 5.1.4. VIII

P blood groups distribution in Pakistan tested with anti-P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		P_1	P_2	P_1	P_2+P	
1- Baltis.Baltistan	80	47	33	35.77	64.23	Clegg,E.J.,et al. 1961
2-Pathans.Swat,Saidu Sharif	133	80	53	36.87	63.13	Alciati,G. 1968
3-Gilgit,Hunza	23	17	6	48.92	51.08	Ikin,Elizabeth W., et al. 1959

Table 5.1.4. IX P blood groups distribution in Afghanistan tested with anti- P_1 serum

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		P_1	P_2	P_1	P_2+P		
1-Timuri and related tribes	118	74	44	38.93	61.07	Woodd-Walker,R.B.,et al.	1967
2-Pushtus	104	66	38	39.60	60.40	Papiha,S.S.,et al.	1977
3-Daris	179	135	44	50.40	49.60	Papiha,S.S.,et al.	1977

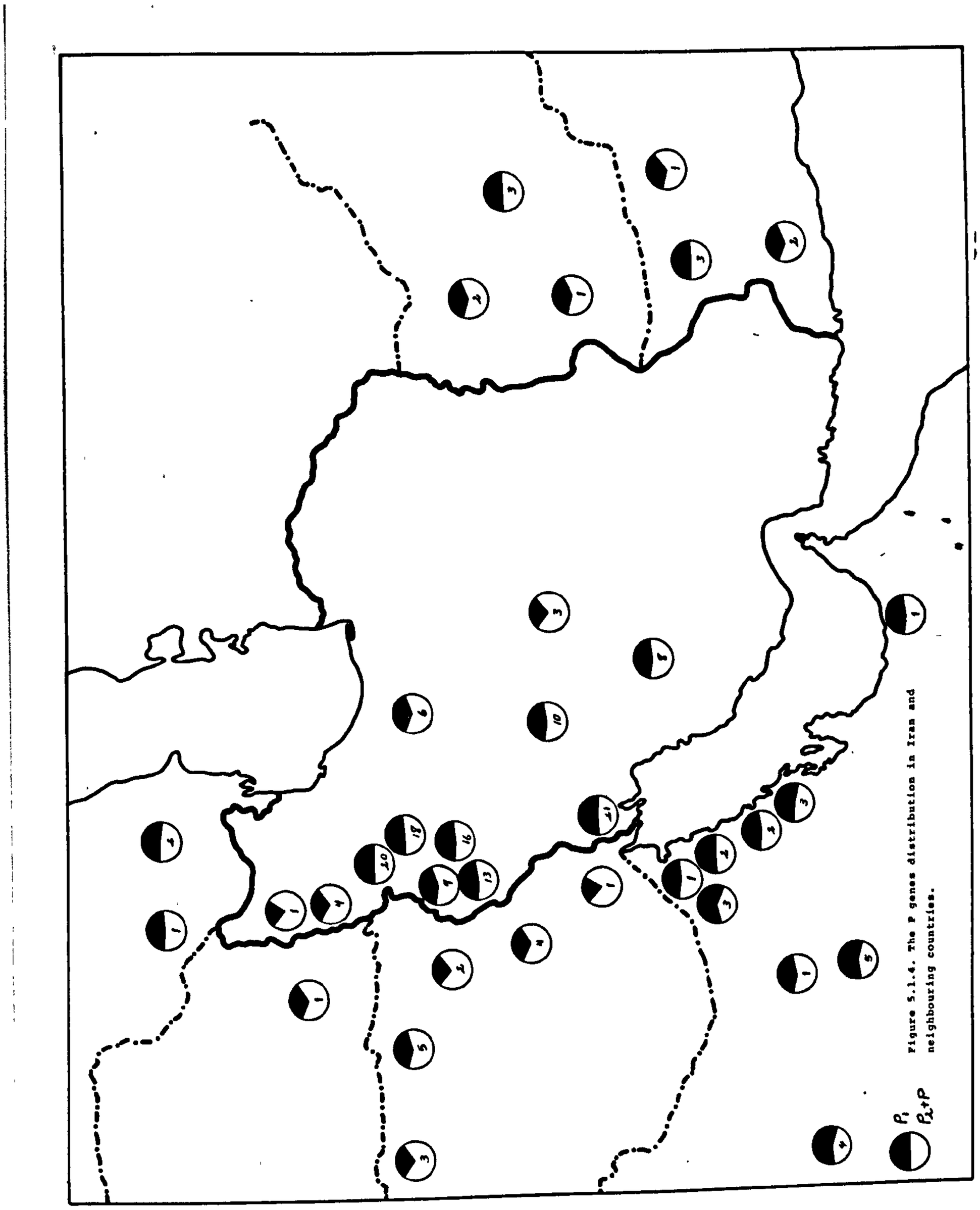


Figure 5.1.4. The P genes distribution in Iran and neighbouring countries.

5.1.5. The Kell blood group system

The distribution of the Kell blood groups and respective gene frequencies in Iranian and neighbouring populations, expresssed in terms of 2, 3 and 6 phenotypes after testing with 1, 2 and 3 antisera respectively, is set out in Tables 5.1.5. a, b and c.

Data summarized in the tables (5.1.5.a) show that the frequency of the gene K varies between zero and 5.67 percent in Iranians, being lowest in the Assyrians and highest in the Kurds of Kermanshah (Nijenhnis, 1964). Values obtained in the present investigation are within this range of variation.

On the whole, with an average K gene frequency of 2.46 percent, the Iranian population appears to exhibit a K frequency similar to that varying between 1.54 and 5.82 percent in the European populations(Mourant et al, 1976).

From the table, some differences in the distribution of the K gene frequencies seem to exist in Iran, as the K values ranging from 0.46 to 3.20 percent in eastern Iran (average 1.43 percent) appear to be lower than those varying between 0.56 and 5.67 percent (average 2.55 percent) in western Iran.

The Arabs of south western Iran, with a K gene frequency of 3.66 percent which is near the average for Iran, show no resemblance to the Arabs of Arabia with their much higher K frequency of above 6 percent.

The Zoroastrians with a K gene frequency of 0.58 percent, which is one of the lowest frequencies within Iran, differ strongly from the Shia from Yazd in the same region, with a much higher K gene frequency of 4.69 percent.

Regarding neighbouring groups , the frequency of the K gene in the populations of the Caucasus ranges from 2.53 percent in the Georgians(Kherumian et al, 1954) to 2.88 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al 1972). With an average K frequency of 2.70 percent, the population of the Caucasus appears to exhibit a K gene grequency similar to that found in Iranians and comparable with the European frequency.

The frequency of the K gene in the Turkish populations varies between 1.20 percent in the Kurds (Richard, 1976) and 5.31 percent in the Eti-Turks (Aksoy et al, 1958). With an average K gene frequency of 2.80 percent, the population of Turkey also seems to show a K gene frequency comparable with that found in Iranians and similar to the European frequency.

The frequency of the gene K in the populations of Iraq ranges from zero in the Kurdish Jews (Godber et al, 1973) to 9.48 percent in the Karaite Jews (Goldschmidt et al, 1976). With an average K gene frequency of 3.1 percent, the Iraqi population appears to exhibit a K gene frequency similar to that found in Iranians and comparable with the frequency in Europe.The very high K gene frequency of 9.48 percent in the Karaite Jews is remarkable and clearly indicates the strong influence of isolation and genetic drift operating in this community.

The freqeuncy of the K gene in the Kuwaiti populations varies between 1.87 percent in the Kuwaiti Arabs(Sawhney, 1975) and 12.00 percent in the Ajman tribe (Khaled et al, 1981). With an average K gene frequency of 6.47 percent, the population of Kuwait seems to show a much higher K frequency than that found in Iranians and in Europeans.

The K gene frequency in the Saudi Arabian populations ranges from 3.17 percent in the Shia sample of Qatif and Hasa Oases to 10.56 percent in the Bedouins (Maranjian et al, 1966), With an average K gene frequency of 6.38 percent, the population of Saudi Arabia, like that of Kuwait, appears to exhibit a much higher K frequency than that found in Iranians and in Europeans.

The K gene frequency of 5.00 percent in the Abu-Dhabians of the United Arab Emirates (Kamel et al, 1980), though lower than the frequencies in the Arab populations of Kuwait and Saudi Arabia, is still higher than the Iranian and the European frequencies.

The few studies on the inhabitants of the Arabian Peninsula have already shown that the K gene of the Kell system reaches its highest frequency there. The average K frequency of around 6 percent in the Arabs of Kuwait and Saudi Arabia seems to be suggestive of African admixture.

The frequency of the K gene in the populations of Pakistan varies between zero in the Baltis (Clegg et al, 1961) and 8.40 percent in the Punjabis (Boyd and Boyd, 1954). With an average K gene frequency of 4.15 percent, the Pakistani population seems to show a higher K frequency than that found in Iranians and higher than the European frequency.

The K gene frequency in the Afghan populations ranges from 1.40 percent in the Pushtus to 2.00 percent in the Daris(Papiha et al, 1977). With an average K gene frequency of 1.70 percent, the population of Afghanistan appears to exhibit a lower K frequency than that in Iranians and other neighbouring populations and in almost all Europeans.

In north west India, and in some Himalayan populations there

appears to be a zone of low K frequency, into which the Afghan frequency appears to fit.

Conclusion

The frequency of the K gene is relatively uniform throughout Europe, ranging usually between 1.54 and 5.82 percent (Mourant et al, 1976).

In general, the Kell blood groups do not vary much on a geographical basis. The K gene frequencies in Iranians are in line with studies in neighbouring populations, showing K gene frequencies similar to those found in Europeans. The only exception is among the Arabs of Arabia in whom the highest frequencies of the K gene, exceeding 12 percent, are found. The very high frequencies of the K gene in the Arab populations appear to be suggestive of African admixture.

Table 5.1.5.a. I Kell blood groups distribution in Iran tested with anti-K serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		K(+)	K(-)	K	k		
1-Assyrians	27	-	27	0.00	100.00	Nijenhuis,L.E.	1964
2-Baluchis.Sistan & Baluchistan	110	1	109	0.46	99.54	Present study.	
3-Zoroastrians	86	1	85	0.58	99.42	Present study.	
4-Kurds.Baneh,Marivan	77	1	76	0.65	99.35	Lehmann,H.,et al.	1973
5-East	66	1	65	0.76	99.24	Bajatzadeh,M., & Walter,H.	1969
6-West	103	2	101	0.97	99.03	Bajatzadeh,M., & Walter, H.	1969
7-Kurds.Rezaieh	138	3	135	1.09	98.91	Present study.	
8-Zabolis.Sistan & Baluchistan	115	3	112	1.31	98.69	Present study.	
9-Kurds.Sanandaj	107	3	104	1.40	98.60	Lehmann,H.,et al.	1973
10-Armenians	67	2	65	1.51	98.49	Nijenhuis,L.E.	1964
11-Tehran	195	6	189	1.55	98.45	Sawhney, K.S.	1975
12-Kurdish Jews	94	3	91	1.60	98.40	Godber,Marilyn J.,et al.	1973

Table 5.1.5.a. I(Cont.) Kell blood groups distribution in Iran tested with anti-K serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		K(+)	K(-)	K	k		
13- Bakhtiaris	138	6	132	2.20	97.80	Nijenhuis,L.E.	1964
14-Kurdish Jews	106	5	101	2.36	97.64	Tills,D., et al.	1977
15-Turks.Rezaieh	141	7	134	2.51	97.49	Present study.	
16-Ghashghais	66	4	62	3.08	96.92	Nijenhuis,L.E.	1964
17-Esfahan	112	7	105	3.18	96.82	Sawhney,K.S.	1975
18-South-East	302	19	283	3.20	96.80	Nijenhuis,L.E.	1964
19-Lurs.Luristan	149	10	139	3.41	96.59	Present study.	
20-North	58	4	54	3.51	96.49	Bajatzadeh,M., & Walter, H.	1969
21-Arabs.Abadan	153	11	142	3.66	96.34	Nijenhuis,L.E.	1964
22-Central and South	106	9	97	4.34	95.66	Bajatzadeh,M., & Walter, H.	1969
23-Tehran	110	10	100	4.65	95.35	Bajatzadeh,M.,& Walter, H.	1969
24-Shi'a.Yazd	142	13	129	4.69	95.31	Sunderland,E., & Smith,H.M.	1966

Table 5.1.5.a.I (Cont.) Kell blood groups distribution in Iran tested with anti-K serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		K(+)	K(-)	K	k		
25-North west	64	7	57	5.63	94.37	Bajatzadeh,M.,& Walter, H.	1969
26-Kurds.Kermanshah	127	14	113	5.67	94.33	Nijenhuis, L.E.	1964

Table . 5.1.5.C. I

Kell blood groups distribution in Iran tested with anti-K, anti-k, and anti-Js^a sera

Population	Number Tested	Phenotypes					
		KKJs(a+)	KKJs(a-)	KkJs(a+)	KkJs(a-)	kkJs(a+)	kkJs(a-)
1-Kurds.Baneh, Marivan	77	-	-	-	1	-	76
2-Kurds.Sanandaj	107	-	-	-	3	-	104
3-Kurdish Jews	94	-	-	-	3	-	91

Table 5.1.5.C.I (Cont.) Kell blood groups distribution in Iran tested with anti-K, anti-k, and anti-Js^{a} sera

Population	Number Tested	Gene frequencies			Authors
		KJs^{b}	kJs^{a}	kJs^{b}	
1-Kurds.Baneh, Marivan	77	0.65	0.00	99.35	Lehmann,H.,et al. 1973
2-Kurds.Sanandaj	107	1.40	0.00	98.60	Lehmann,H.,et al. 1973
3-Kurdish Jews	94	1.60	0.00	98.40	Godber,Marilyn J., et al. 1973

Table 5.1.5. d I. Kell blood groups distribution in Iran tested with anti-Js^a(Sutter) serum

Population	Number Tested	Js(a+)	Js(a-)	Authors	
Kurdish Jews	87	-	87	Tills,D.,et al.	1977

Table 5.1.5.a.II Kell blood groups distribution in the Caucasus tested with anti-K serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		K(+)	K(-)	K	k	
1-Georgians.Autochthonous	40	2	38	2.53	97.47	Kherumian,R.,et al. 1954
2-Svanis.Georgia,Abkhazskaya	659	38	621	2.88	97.12	Verbitsky,M.Sh.,et al. 1972

Table 5.1.5.b. II Kell blood groups distribution in the Caucasus tested with anti-K and anti-k sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		KK	Kk	kk	K	k	
1-Svanis.. Georgia,Abkhazskaya	659	-	38	621	2.88	97.12	Verbitsky,M.Sh., et al. 1972

Table 5.1.5.a. III Kell blood groups distribution in Turkey tested with anti-K serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		K(+)	K(-)	K	k		
I- Kurds				1.20	98.80	Richard, P.	1976
2-Turks.Asia Minor,Mersin	107	4	103	1.89	98.11	Aksoy,M., et al.	1958
3-Eti-Turks. Asia Minor. ,near Mersin	116	12	104	5.31	94.69	Aksoy,M., et al.	1958

Table 5.1.5. a. IV Kell blood groups distribution in Iraq tested with anti- K serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		K(+)	K(-)	K	k	
1- Kurdish Jews	27	-	27	0.00	100.00	Godber,Marilyn.J., et al. 1973
2-Kurdish Jews.South east	50	1	49	1.00	99.00	Tills,D., et al. 1977
3-Assyrians	99	3	96	1.53	98.47	Ikin,Elizabeth W., et al. 1965
4-Kurdish Jews.North west	61	4	57	3.28	96.72	Tills,D., et al. 1977
5-Kurds				3.30	96.70	Al-Khafaji,S.D., et al. 1976
6-Karaite Jews	72	13	59	9.48	90.52	Goldschmidt,E., et al. 1976

Table 5.1.5.b. IV Kell blood groups distribution in Iraq tested with anti-K and anti-k sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		KK	Kk	kk	K	k	
1-Kurdish Jews	27	-	-	27	0.00	100.00	Godber,Marilyn.J., et al. 1973
2-Kurdish Jews.South east	50	-	1	49	1.00	99.00	Tills,D., et al. 1977
3-Kurdish Jews.North west	61	-	4	57	3.28	96.72	Tills,D., et al. 1977
4-Kurds					3.30	96.70	Al-Khafaji,S.D.,et al. 1976

Table 5.1.5.C. IV Kell blood groups distribution in Iraq tested with anti-K, anti-k, and anti-Js^{a} sera

Population	Number Tested	Phenotypes					
		KKJs(a+)	KKJs(a-)	KkJs(a+)	KkJs(a-)	kkJs(a+)	kkJs(a-)
1- Kurdish Jews	27	-	-	-	-	-	27

Table 5.1.5.C. IV (Cont.) Kell blood groups distribution in Iraq tested with anti-K, anti-k, and anti-Js^{a} sera

Population	Number Tested	Gene frequencies			Authors
		KJs^{b}	KJs^{a}	kJs^{b}	
1-Kurdish Jews	27	0.00	0.00	100.00	Godber,Marilyn J., 1973 et al.

Table 5.1.5. d IV Kell blood groups distribution in Iraq tested with anti-Js^{a}(Sutter) serum

Population	Number Tested	Js(a+)	Js(a-)	Authors
Kurdish Jews. North west	20	-	20	Tills, D., et al. 1977
Kurdish Jews. South East	9	-	9	Tills, D., et al. 1977

Table 5.1.5.a. V Kell blood groups distribution in Kuwait tested with anti-K serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		K(+)	k(-)	K	k		
1-Kuwaiti Arabs	162	6	156	1.87	98.13	Sawhney, K.S.	1975
2-General population	74	8	66	5.00	95.00	Khaled,E.,et al.	1981
3-Suluba tribe	51	6	45	7.00	93.00	Khaled,E.,et al.	1981
4-Ajman tribe	52	12	40	12.00	88.00	Khaled,E.,et al.	1981

Table 5.1.5.b. V Kell blood groups distribution in Kuwait tested with anti-K and anti-k sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		KK	Kk	kk	K	k		
1-General population	74	-	8	66	5.00	.95.00	Khaled,E.,et al.	1981
2-Suluba tribe	51	1	5	45	7.00	93.00	Khaled,E.,et al.	1981
3-Ajman tribe	52	-	12	40	12.00	88.00	Khaled,E.,et al.	1981

Table 5.1.5.a. VI Kell blood groups distribution in Saudi Arabia tested with anti-K serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		K(+)	K(-)	K	k		
1-Shias.Qatif & Hasa Oases	465	29	436	3.17	96.83	Maranjian,G.,et al.	1966
2-Sunnis.Najd	180	35	145	4.84	95.16	Maranjian,G.,et al.	1966
3-Sunnis.Qatif &Hasa Oases	318	36	282	5.83	94.17	Maranjian, G.,et al.	1966
4-Western Saudi Arabia	205	23	182	6.10	93.90	Saha, N., et al.	1980
5-Sunnis.Asir,Hejaz,Najran	174	26	148	7.77	92.23	Maranjian,G.,et al.	1966
6-Bedouins	175	35	140	10.56	89.44	Maranjian,G.,et al.	1966

Table 5.1.5.b. VI Kell blood groups distribution in Saudi Arabia tested with anti-K and anti-k sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		KK	Kk	kk	K	k	
1- Western Saudi Arabia	205	2	21	182	6.10	93.90	Saha,N., et al. 1980

Table 5.1.5.b. VII Kell blood groups distribution in the United Arab Emirates tested with anti-K and anti-k sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		KK	Kk	kk	K	k	
1-Abu-Dhabians	100	2	6	92	5.00	95.00	Kamel,K.,et al. 1980

Table 5.1.5.C. VII Kell blood groups distribution in the United Arab Emirates tested with anti-K, anti-k, and anti-Js^{a} sera

Population	Number Tested	Phenotypes					
		KKJs(a+)	KKJs(a-)	KkJs(a+)	KkJs(a-)	kkJs(a+)	kkJs(a-)
1-Abu-Dhabians	100	-	2	1	5	2	90

Table 5.1.5.C.VII (Cont.) Kell blood groups distribution in the United Arab Emirates tested with anti-K, anti-k, and anti-Js^{a} sera

Population	Number Tested	Gene frequencies			Authors
		KJs^{b}	kJs^{a}	kJs^{b}	
1-Abu-Dhabians	100	5.00	1.50	93.50	Kamel,K.,et al. 1980

Table 5.1.5.a. VIII Kell blood groups distribution in Pakistan tested with anti-K serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		K(+)	K(-)	K	k		
1-Baltis.Baltistan	80	-	80	0.00	100.00	Clegg,E.J.,et al.	1961
2-Pathans.Swat,Saidu Sharif	133	10	123	3.76	96.24	Alciati, G.	1968
3-Gilgit,Hunza	23	2	21	4.45	95.55	Ikin,Elizabeth W.,et al.	1959
4-Punjabis.Lahore	87	14	73	8.40	91.60	Boyd,W.C., & Boyd,L.G.	1954

Table 5.1.5.b. VIII Kell blood groups distribution in Pakistan tested with anti-K and anti-k sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		KK	Kk	kk	K	k		
1-Pathans.Swat,Saidu Sharif	133	-	10	123	3.76	96.24	Alciati, G.	1968

Table 5.1.5. d VIII. Kell blood groups distribution in Pakistan tested with anti-Js^{a}(Sutter)serum

Population	Number Tested	Js(a+)	Js(a-)	Authors
Pathans	139	-	139	Vos,G.H.,and Kirk,R.L. 1961
Punjabis	168	-	168	Vos,G.H.,and Kirk,R.L. 1961

Table 5.1.5.b. IX Kell blood groups distribution in Afghanistan tested with anti-K and anti-k sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		KK	Kk	kk	K	k	
1-Pushtus	104	-	3	101	1.40	98.60	Papiha,S.S.,et al. 1977
2-Timuri and related tribes	118	1	2	115	1.69	98.31	Woodd-Walker,R.B.,et al.1967
3-Daris	179	1	5	173	2.00	98.00	Papiha,S.S., et al. 1977

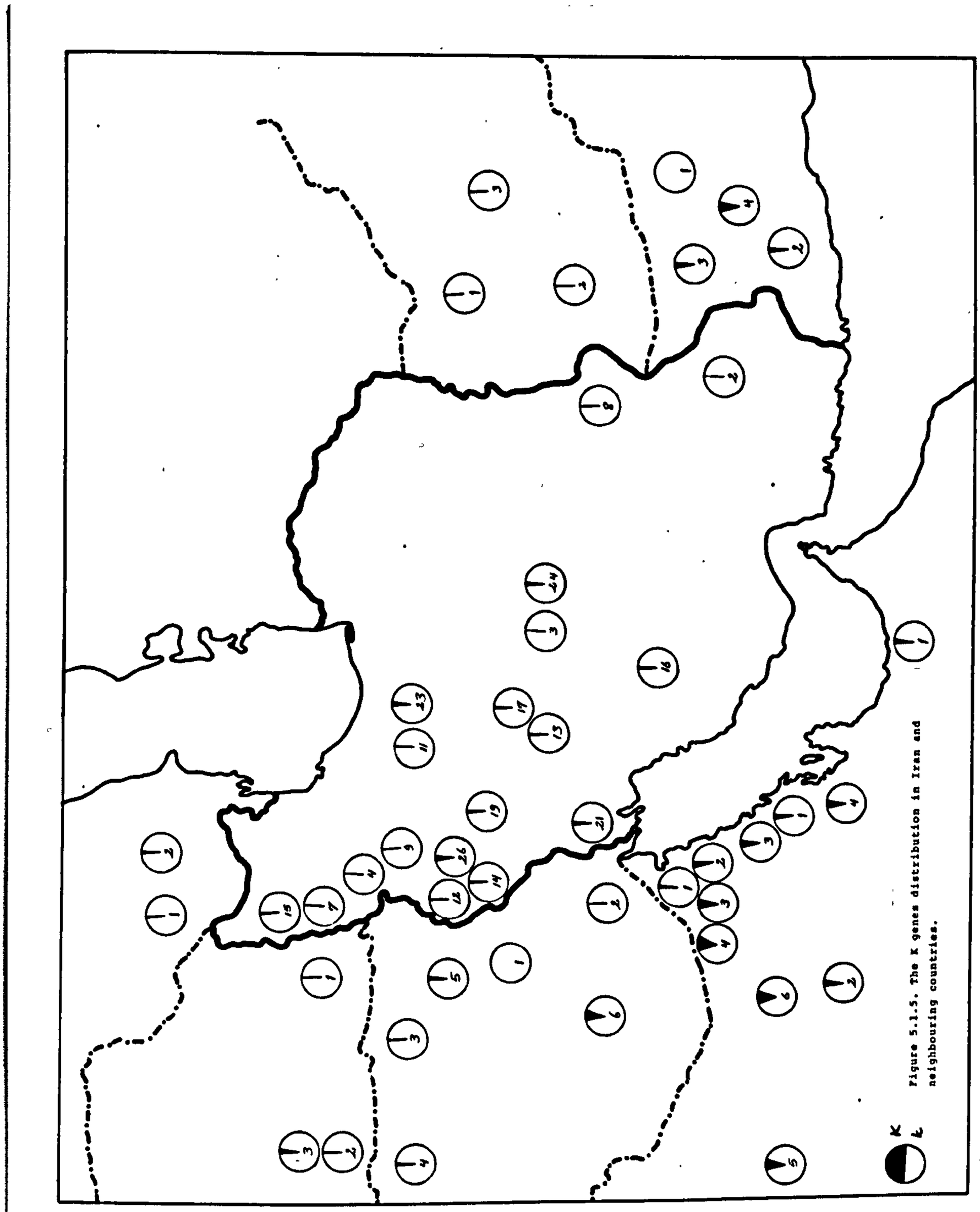


Figure 5.1.5. The K genes distribution in Iran and neighbouring countries.

5.1.6.The Duffy blood group system

The distribution of the Duffy blood groups and respective gene frequencies in Iranian and neighbouring populations, expressed in terms of 2 and 4 phenotypes after testing with 1 and 2 antisera, resepctively, is shown in Tables 5.1.6 a and b.

The frequency of the Fy^a gene ranges from 24.97 to 57.83 percent in Iranians, being lowest in the Shi'a Moslems of Yazd (Sunderland and Smith, 1966) and highest in the Tehrani series of Boue and Boue (1956). Values obtained in the present investigation are within this range of variation.

On the whole with an average Fy^a frequency of 43.58 percent, the Iranian population appears to exhibit a higher Fy^a frequency than that of around 40 percent found in Europeans(Mourant et al, 1976). Some differences in the distribution of the Fy^a gene frequencies seem to exist in Iran, as the Fy^a values ranging from 35.24 to 53.42 percent (average 46.47 percent) in western Iran appear to be higher than those varying between 36.42 and 43.02 percent (average 39.67 percent) in eastern Iran.

The Kurdish Jews both in Iran and in Iraq appear to exhibit higher Fy^afrequencies, averaging 51.79 and 42.04 percent respectively, compared with those averaging 45.22 and 20 percent respectively found in the Kurds themselves.

The Arabs of south western Iran though seem to show one of the lowest Fy^a frequencies within Iran but the frequency is still much higher than the very low ones found in the Arab populations of Arabia.

The Fy^a frequency of 37.12 percent in the Zoroastrians (present investigation) is also one of the lowest frequencies

within Iran and differs strongly from that of 24.97 percent in the Shi'a Moslems of Yazd (Sunderland and Smith, 1966), the region that the Zoroastrians come from. It should be pointed out here that the extremely low Fy^a frequency of 24.97 percent in the Yazdi Shi'a series of Sunderland and Smith (1966) may possibly be due to technical difficulties as was mentioned by the authors themselves.

Regarding neighbouring populations, with an Fy^a frequency of 48.76 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1972), the population of the Caucasus appears to exhibit a higher Fy^a frequency than that found in Iranians and higher than the European frequency.

The frequency of the Fy^a gene in the populations of Turkey ranges from 42.76 percent in the Eti-Turks to 48.84 percent in the Turks (Aksoy et al, 1958). With an average Fy^a frequency of 45.80 percent, the Turkish population seems to show a somewhat higher Fy^a frequency than that found in Iranians and higher than the frequency in Europeans.

The Fy^a gene frequency in the populations of Iraq varies between the limits of 20.00 percent in the Kurds (Al-Khafaji et al, 1976) and 75.38 percent in the Assyrians (Ikin et al, 1965).

With an average Fy^a frequency of 45.48 percent, the Iraqi population appears to exhibit a slightly higher Fy^a frequency than that found in Iranians and higher than the European frequency.

The very high Fy^a gene frequency of 75.38 percent in the Assyrians of Iraq is more like that found in India and further east than that usual in the Near East.

The frequency of the Fy^a gene in the Arab populations of Kuwait ranges from 23.00 percent in the Ajman tribe to 34.00 percent in the Suluba tribe (Khaled et al, 1981). With an average Fy^a frequency of 28.45 percent, the Kuwaiti population seems to show a much lower Fy^a frequency than that found in Iranians and much lower than the European frequency.

The Fy^a gene frequency in the Arab populations of Saudi Arabia varies between 4.51 percent in the Shia sample of Qatif and Hasa Oases and 32.50 percent in the Sunni series of Najd (Maranjian et al, 1966). With an average Fy^a frequency of 17.88 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit a much lower Fy^a frequency than that found in Iranians and much lower than the frequency in Europeans.

A similarly low Fy^a frequency of 18.70 percent is reported by Kamel et al (1980) in the Abu-Dhabians of the United Arab Emirates.

Low Fy^a gene frequencies seems to be characteristic of Arab populations and is suggestive of African admixture.

The frequency of the Fy^a gene in the populations of Pakistan ranges from 44.84 percent in the Hunza, Gilgit(Ikin et al, 1959) to 73.04 percent in the Pakistani series of Cutbush et al (1950). With an average Fy^a frequency of 57.68 percent, the Pakistani population seems to show a much higher Fy^a frequency than that found in Iranians and much higher than the European frequency. The frequency of the Fy^a gene in the population of Pakistan appears to be more like that found in Indians.

The Fy^a gene frequency in the populations of Afghanistan varies between the limits of 53.90 percent in the Pushtus and

59.80 percent in the Daris (Papiha et al, 1977). With an average Fy^a frequency of 57.17 percent, the Afghan population,like the population of Pakistan,appears to exhibit a much higher Fy^a frequency than that found in Iranians and much higher than the frequency in Europeans.

With regard to the null allele (Fy^-) of the Duffy system (Tables 5.1.6.b), since the majority of the samples were tested with anti-Fy^aserum only, it was impossible to detect the presence of the silent Fy gene.

The frequency of the Fy null allele in the Kurdish populations of Iran, both Jewish and non-Jewish, ranges from 13.47 percent in the Kurds of Sanandaj to 16.75 percent in the Kurds of Baneh, Marivan (Lehmann et al, 1973). With an average Fy null frequency of 15.01 percent, the Kurdish population of Iran appears to exhibit a much higher Fy null frequency than that of below 3 percent found in Europeans (Mourant et al, 1976).

The Fy null allele frequency in the Iraqi Kurdish Jews varies between 21.36 percent (Tills et al, 1977) and 22.11 percent (Godber et al, 1973). With an average Fy null frequency of 21.73 percent, the Iraqi Kurdish Jews seem to exhibit even a higher Fy null frequency than that found in Iranians. These findings suggest that the Fy null allele, usually regarded as of African origin, extends as an indigenous feature into south west Asia.

Although in the Kurdish Jews the African marker genes are mostly absent or present in only a low frequency, the Fy null allele persists at higher levels than the other markers.

Evidence for the presence of African genes in the Arabian pool is suggested from the widespread distribution of the

Duffy null allele. Among the three Kuwaiti groups investigated by Khaled et al (1981), the Suluba sample has the lowest Fy null frequency (24.00 percent) and the Ajman, the highest (38.00 percent).

African admixture can also be expressed by the presence of the high Fy null frequency of 68.00 percent in the Abu-Dhabians of the United Arab Emirates (Kamel et al, 1980).

The Fy null allele was not present in the Pathans of west Pakistan investigated by Alciati (1968).

The frequency of the Fy null allele in the Afghan populations ranges from 2.00 percent in the Daris to 9.70 percent in the Pushtus (Papiha et al, 1977). With an average Fy null frequency of 5.85 percent the population of Afghanistan appears to exhibit a lower Fy null frequency than that found in Iranians but higher than the European frequency.

The presence of the Fy null allele in Kurdish Jews and Kurds has already been mentioned as an African marker.It is however present in many populations of the Near East at frequencies implying a higher proportion of African admixture than that indicated by other markers. This may in part be due to the fact that its detection depends upon negative results in tests with both anit-Fy^a and anti-Fy^b, both of which tend to be weak reagents which may sometimes give false negative results. Now that a positive reagent, anti-Fy^4, has been discovered, it is important that it should be used to establish the distribution of the gene in a positive manner. However, following the findings of Miller et al (1975) that homozygotes for this gene are probably protected against malaria due to Plasmodium Vivax, the distribution of the gene needs further study in an epidemiological context, and in

comparison with that of other genes having a protective effect against malaria.

Conclusion

The frequency of the Fy^a gene in most populations of European origin is around 40 percent. In India and eastern Asia it is considerably higher.

The frequency of Fy^a is only about 5 percent in American Negroes, and very low indeed in African Negroes. Fy null may have a frequency as high as 3 percent among Europeans. The few African populations which have been tested show very low frequencies of both Fy^a and Fy^b, with an Fy null frequency of around 90 percent (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arabs of Arabia with their much lower frequencies, the frequency of the Fy^a gene appears to be higher than that in Europeans but lower than that in Indians.

The low Fy^a gene frequencies together with the high values of Fy null seems to be characteristic of the Arab populations and suggestive of African admixture.

The frequency of the Fy^a gene in the populations of Pakistan and Afghanistan appears to be much higher than that in Iranians and more like that found in Indians.

Until recently, owing to the great rarity of the anti-Fy^b reagent, very few populations had been tested with both anti-Fy^a and anti-Fy^b sera. The number of the tests completed is still relatively small but the value of the system in tracing and measuring an African component in populations is becoming evident. This is particularly the case in the Near East where a series of isogenic lines for the amorph Fy gene can now be drawn.

Table 5.1.6.a. I Duffy blood groups distribution in Iran tested with anti-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		FY(a+)	Fy(a-)	Fy^a	Fy^b+Fy	
1-Shi'a. Yazd	151	66	85	24.97	75.03	Sunderland,E., & Smith,H.M. 1966
2-Lurs.Luristan	143	79	64	33.10	66.90	Present study.
3-Arabs.Abadan	155	90	65	35.24	64.76	Nijenhuis,L.E. 1964
4-Baluchis.Sistan & Baluchistan	47	28	19	36.42	63.58	Present study.
5-Zoroastrians	86	52	34	37.12	62.88	Present study.
6-Zabolis.Sistan & Baluchistan	115	70	45	37.45	62.55	Present study.
7-Armenians	78	48	30	37.98	62.02	Nijenhuis,L.E . 1964
8-Central and South	95	59	36	38.45	61.55	Bajatzadeh,M., & Walter, H. 1969
9-Kurds. Rezaieh	138	89	49	40.41	59.59	Present study.
10-Tehran	92	60	32	41.02	58.98	Bajatzadeh,M., & Walter,H. 1969
11-East	62	41	21	41.80	58.20	Bajatzadeh,M., & Walter, H. 1969

Table 5.1.6.a.I.(Cont.) Duffy blood groups distribution in Iran tested with anti-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy		
12-South east	348	235	113	43.02	56.98	Nijenhuis,L.E.	1964
13-Kurds.Sanandaj	107	76	31	43.68	56.32	Lehmann,H.,et al.	1973
14-Bakhtiaris	137	94	43	43.97	56.03	Nijenhuis,L.E.	1964
15-Assyrians	32	22	10	44.10	55.90	Nijenhuis,L.E.	1964
16-Turks.Rezaieh	140	98	42	45.23	54.77	Present study.	
17-Kurds.Kermanshah	127	89	38	45.30	54.70	Nijenhuis,L.E.	1964
18-West	95	68	27	46.69	53.31	Bajatzadeh,M., & Walter, H.	1969
19-Kurdish Jews	94	73	21	50.16	49.84	Godber,Marilyn.J.,et al.	1973
20-Kurds.Baneh,Marivan	77	61	16	51.50	48.50	Lehmann,H., et al.	1973
21-North west	59	46	13	53.06	46.94	Bajatzadeh,M., & Walter, H.	1969
22-Kurdish Jews	106	83	23	53.42	46.58	Tills , D., et al.	1977
23-North	56	44	12	53.71	46.29	Bajatzadeh,M., & Walter, H.	1966

Table 5.1.6.a. I (Cont.) Duffy blood groups distribution in Iran tested with anti-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy		
24-Ghashghais	66	52	14	53.95	46.05	Nijenhuis, L.E.	1964
25-Tehran	135	111	24	57.83	42.17	Boue' & Boue'..	1956

Table 5.1.6.a.II Duffy blood groups distribution in the Caucasus tested with anti-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy	
1-Svanis. Georgia, Abkhazeskaya	339	250	89	48.76	51.24	Verbitsky,M.Sh., 1972 et al.

Table 5.1.6.a. III Duffy blood groups distribution in Turkey tested with anti-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy	
1-Eti-Turks.Asia Minor, near Mersin.	116	78	38	42.76	57.24	Aksoy,M.,et al. 1958
2-Turks.Asia Minor,Mersin	107	79	28	48.84	51.16	Aksoy,M.,et al. 1958

Table 5.1.6.a. IV Duffy blood groups distribution in Iraq tested with anti-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy	
1-Kurds				20.00	80.00	Al-Khafaji,S.D., et al. 1976
2-Kurdish Jews.South east	50	32	18	40.00	60.00	Tills,D.,et al. 1977
3-Kurdish Jews	27	18	9	40.44	59.56	Godber,Marilyn.J., et al. 1973
4-Kurdish Jews.North West	61	43	18	45.68	54.32	Tills,D., et al. 1977
5-Karaite Jews	72	55	17	51.41	48.59	Goldschmidt,E.,et al. 1976
6-Assyrians	99	93	6	75.38	24.62	Ikin,Elizabeth W., et al. 1965

Table 5.1.6.a. V Duffy blood groups distribution in Kuwait tested with anti-Fy^a serum only

Population	Number Tested	Phenotype		Gene frequencies		Authors
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy	
1-Ajman tribe	52	21	31	23.00	77.00	Khaled,E.,et al. 1981
2-General population	74	36	38	28.00	72.00	Khaled,E.,et al. 1981
3-Kuwaiti Arabs	140	69	71	28.79	71.21	Sawhney, K.S. 1975
4-Suluba tribe	51	29	22	34.00	66.00	Khaled,E.,et al. 1981

Table 5.1.6.a. VI

Duffy blood groups dsitribution in Saudi Arabia tested with anti-Fy^a serum only.

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy	
1-Shias.Qatif & Hasa Oases	465	41	424	4.51	95.49	Maranjian,G.,et al. 1966
2-Western Saudi Arabia	208	53	155	13.68	86.32	Saha, N., et al. 1980
3-Sunnis.Qatif & Hasa Oases	319	88	231	14.91	85.09	Maranjian,G.,et al. 1966
4-Sunnis Asir,Hejaz,Najran	173	49	124	15.34	84.66	Maranjian,G.,et al. 1966
5-Bedouins	175	80	95	26.32	73.68	Maranjian,G.,et al. 1966
6-Sunnis. Najd	180	98	82	32.50	67.50	Maranjian,G.,et al. 1966

Table 5.1.6.a.VII Duffy blood groups distribution in the United Arab Emirates tested with anti-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy		
1-Abu-Dhabians	100	34	66	18.70	81.30	Kamel,K.,et al.	1980

Table 5.1.6.a. VIII Duffy blood groups distribution in Pakistan tested with anit-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy	
1-Hunza,Gilgit	23	16	7	44.84	55.16	Ikin.,Elizabeth W., et al. 1959
2-Punjabis.Lahore	68	52	16	51.49	48.51	Boyd,W.C., & Boyd,L.G. 1954
3-Pathans.Swat,Saidu Sharif	72	55	17	52.78	47.22	Alciati,G. 1968
4-Baltis.Baltistan	79	70	9	66.25	33.75	Clegg,E.J.,et al. 1961
5-Pakistanis	55	51	4	73.04	26.96	Cutbush,Marie.,et al.1950

Table 5.1.6.a IX Duffy blood groups distribution in Afghanistan tested with anti-Fy^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Fy(a+)	Fy(a-)	Fy^a	Fy^b+Fy	
1-Pushtus	104	82	22	53.90	46.10	Papiha,S.S.,et al. 1977
2-Timuri & related tribes	118	97	21	57.81	42.19	Woodd-Walker,R.B., et al. 1967
3-Daris	179	152	27	59.80	40.20	Papiha,S.S.,et al. 1977

Table 5.1.6.b.I Duffy blood groups distribution in Iran tested with anti-Fy^a and anti-Fy^b sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		Fy(a+b-)	Fy(a+b+)	Fy(a-b+)	Fy(a-b-)	Fy^a	Fy^b	Fy	
1-Kurds. Sanandaj	107	28	48	27	4	43.68	42.85	13.47	Lehmann,H.,et al. 1973
2-Kurdish Jews	87	31	38	14	4	51.10	34.28	14.62	Tills,D.,et al. 1977
3-Kurdish Jews	94	34	39	17	4	50.16	34.64	15.20	Godber,Marilyn J., 1973 et al.
4-Kurds.Baneh,Marivan	77	30	31	12	4	51.50	31.75	16.75	Lehmann,H.,et al. 1973

Table 5.1.6.b. IV Duffy blood groups distribution in Iraq tested with anti-Fy^a and anti-Fy^b sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		Fy(a+b-)	Fy(a+b+)	Fy(a-b+)	Fy(a-b-)	Fy^a	Fy^b	Fy	
1-Kurdish Jews. North west	20	9	6	4	1	49.59	29.05	21.36	Tills,D.,et al. 1977
2-Kurdish Jews	27	8	10	7	2	40.44	37.45	22.11	Godber,Marilyn. 1973 J., et al.

Table 5.1.6.b. V

Duffy blood groups distribution in Kuwait tested with anti-Fy^a and anti-Fy^b sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		Fy(a+b-)	Fy(a+b+)	Fy(a-b+)	Fy(a-b-)	Fy^a	Fy^b	Fy	
1-Suluba tribe	51	14	15	19	3	34.00	42.00	24.00	Khaled,E.,et al.1981
2-General population	74	21	15	27	11	28.00	34.00	38.00	Khaled,E.,et al.1981
3-Ajman tribe	52	11	10	23	8	23.00	39.00	38.00	Khaled,E.,et al.1981

Table 5.1.6.b. VII Duffy blood groups distribution in the United Arab Emirates tested with anti-Fy^a and anti-Fy^b sera

Popualtion	Number Tested	Phenotypes				Gene frequencies			Authors	
		Fy(a+b-)	Fy(a+b+)	Fy(a-b+)	Fy(a-b-)	Fy^a	Fy^b	Fy		
1-Abu-Dhabians	100	28	6	19	47	18.70	13.30	68.00	Kamel,K.,et al.	1980

Table 5.1.6.b. VIII Duffy blood groups distribution in Pakistan tested with anti-Fy^a and anti-Fy^b sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		Fy(a+b-)	Fy(a+b+)	Fy(a-b+)	Fy(a-b-)	Fy^a	Fy^b	Fy	
1-Pathans.Swat,Saidu Sharif	72	21	34	17	-	52.78	47.22	0.00	Alciati,G. 1968

Table 5.1.6.b. IX Duffy blood groups distribution in Afghanistan tested with anti-Fy^a and anti-Fy^b sera

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		Fy(a+b-)	Fy(a+b+)	Fy(a-b+)	Fy(a-b-)	Fy^a	Fy^b	Fy	
1-Daris	179	66	86	27	-	59.80	38.20	2.00	Papiha,S.S.,et al. 1977
2-Pushtus	104	41	41	21	1	53.90	36.40	9.70	Papiha,S.S.,et al. 1977

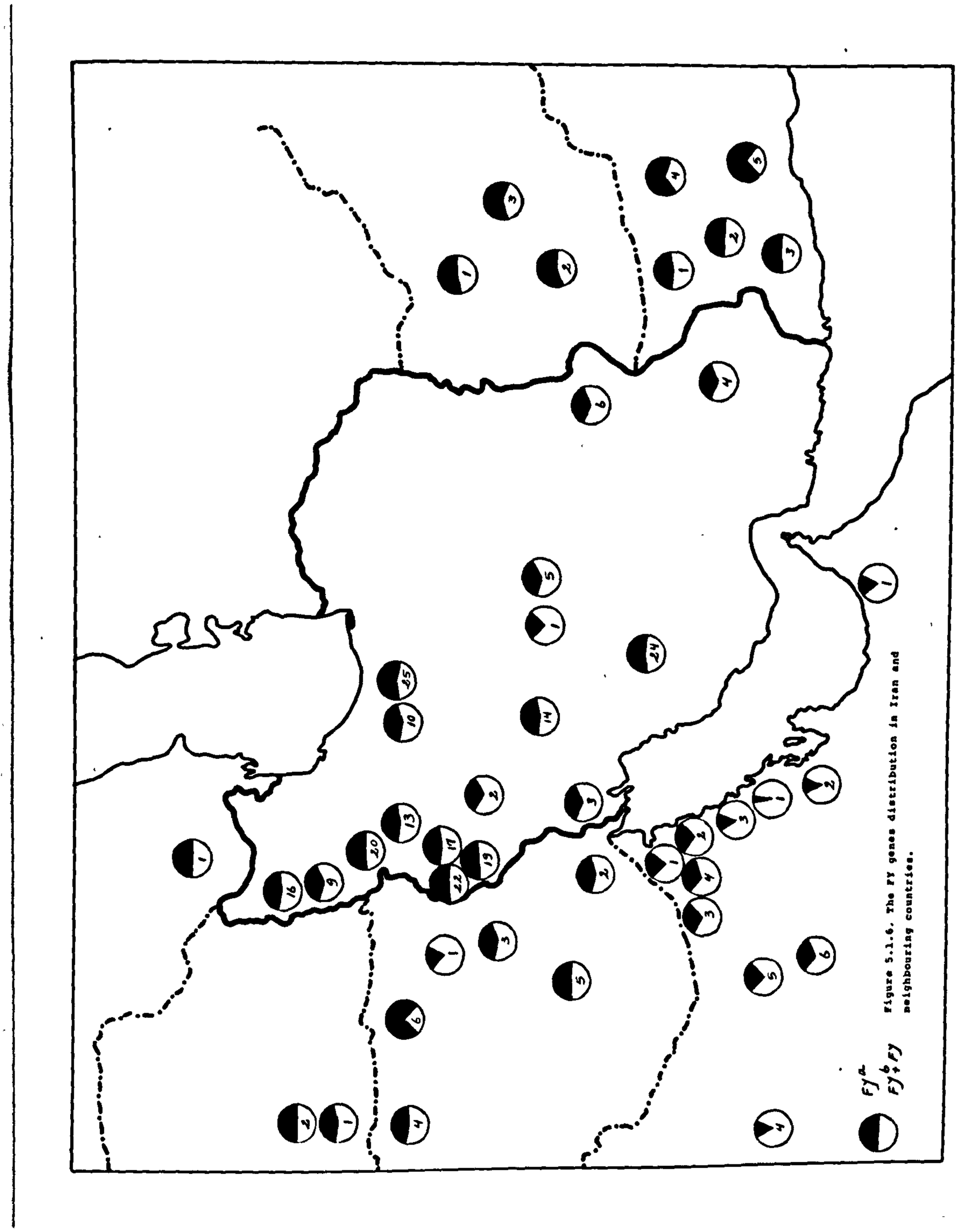


Figure 5.1.6. The FY genes distribution in Iran and neighbouring countries.

5.1.7 The Kidd blood group system

The distribution of the Kidd blood groups and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.1.7.

There are only a few reports of the Kidd blood groups in Iranian and neighbouring populations.

The frequency of the JK^a gene ranges from 25.52 to 48.49 percent in Iranians, being lowest in the Lurs of Luristan (present investigation) and highest in the Tehrani series of Sawhney (1975).

On the whole, with an average JK^a frequency of 37.34 percent, the Iranian population appears to exhibit a much lower JK^a frequency than that of around 50 percent found in Europeans (Mourant et al, 1976).

Since the data available on the distribution of the Kidd system in various populations of Iran are still not large enough, full discussion is not yet possible.

Regarding neighbouring groups, a JK^a frequency of 43.36 percent is reported by Verbitsky et al (1972) in the Svanis of Georgia, Abkhazskaya of the Caucasus, a figure higher than that found in Iranians but lower than the European frequency.

Information on the distribution of the Kidd genes in the inhabitants of Turkey is lacking.

The frequency of the JK^a gene in the Iraqi populations varies between 32.92 percent in the Kurdish Jews of the north west and 52.86 percent in the Kurdish Jews of the south east (Tills et al, 1977). With an average JK^a frequency of 41.03 percent, the Kurdish population of Iraq seems to show a higher JK^a frequency than that found in Iranians but lower than the frequency in Europeans. The Iraqi Kurds show, however, a high-

er JK^a frequency than the average for Iran but the frequency is lower than that of average 43.90 percent found in the Kurdish Jews of western Iran (Godber et al, 1973; and Tills et al, 1977).

The JK^a gene frequency in the populations of Kuwait ranges from 56.00 percent in the Suluba tribe to 76.00 percent in the Ajman tribe (Khaled et al, 1981). With an average JK^a frequency of 63.33 percent, the Kuwaiti population appears to exhibit a much higher JK^a frequency than that found in Iranians and even much higher than the frequency in Europeans. As with cDe(Ro) and Duffy null (Fy^-), the distribution of the JK^a gene reflects the effect of African gene flow into the Arabian pool.

Unfortunately, there are no data available on the distribution of the Kidd genes in the Arab populations of Saudi Arabia and the United Arab Emirates.

The frequency of the JK^a gene in the populations of Pakistan varies between 43.88 percent in the Pathans (Alciati, 1968) and 48.16 percent in the Punjabis (Boyd and Boyd, 1954). With an average JK^a frequency of 46.02 percent, the Pakistani population seems to show a higher JK^a frequency than that found in Iranians but still lower than the European fequency.

The JK^a frequency in the populations of Afghanistan ranges from 50.00 percent in the Daris to 65.90 percent in the Pushtus (Papiha et al, 1977). With an average JK^a frequency of 57.95 percent, the Afghan population appears to exhibit a much higher JK^a frequency than that found in Iranians and somewhat higher than the frequency in Europeans.

Conclusion

The frequency of the JK^a gene in all European populations tested is near 50 percent (average 51.40 percent). It is high, around 75 percent, in all African populations investigated (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab population of Kuwait with their very high JK^a frequency, the frequency of the JK^a gene is below the typical European level. The average frequency of the JK^a gene appears to be somewhat higher than the European level in the Afghan populations.

Again, as with cDe (Ro) and Duffy null (Fy^-), the high frequency of the JK^a gene in the Arab population of Kuwait reflects the effect of African gene flow into the Arabian pool.

Table 5.1.7. I Kidd blood groups distribution in Iran tested with anti-JK^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		JK(a+)	JK(a-)	JK^a	JK^b+JK	
1-Lurs.Luristan	146	65	81	25.52	74.48	Present study.
2-Kurds.Rezaieh	136	69	67	29.81	70.19	Present study.
3-Baluchis.Sistan & Baluchistan	111	58	53	30.90	69.10	Present study.
4-Turks.Rezaieh	141	74	67	31.07	68.93	Present study.
5-Zabolis.Sistan & Baluchistan	115	70	45	37.45	62.55	Present study.
6-Kurdish Jews	94	64	30	43.51	56.49	Godber,Marilyn J.,et al. 1973
7-Kurdish Jews	87	60	27	44.30	55.70	Tills,D.,et al. 1977
8-Zoroastrians	86	60	26	45.02	54.98	Present study.
9-Tehran	49	36	13	48.49	51.51	Sawhney,K.S. 1975

Table 5.1.7. II

Kidd blood groups distribution in the Caucasus tested with anti-JK^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		JK(a+)	JK(a-)	JK^a	JK^b+JK	
1-Svanis.Georgia, Abkhazskaya	159	108	51	43.36	56.64	Verbitsky,M.Sh.,et al. 1972

Table 5.1.7. IV Kidd blood groups distribution in Iraq tested with anti-JK^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		JK(a+)	JK(a-)	JK^a	JK^b+JK	
1-Kurdish Jews.North West	20	11	9	32.92	67.08	Tills,D.,et al. 1977
2-Kurdish Jews	27	15	12	33.34	66.66	Godber,Marilyn J., et al. 1973
3-Kurds				45.00	55.00	Al-Khafaji,S.D.,et al. 1976
4-Kurdish Jews.South east	9	7	2	52.86	47.14	Tills,D.,et al. 1977

Table 5.1.7. V Kidd blood groups distribution in Kuwait tested with anti-JK^a serum only

Population	Number tested	Phenotypes		Gene frequencies		Authors	
		JK(a+)	JK(a-)	JK^a	JK^b+JK		
1-Suluba tribe	51	41	10	56.00	44.00	Khaled,E.,et al.	1981
2-General population	74	61	13	58.00	42.00	Khaled,E.,et al.	1981
3-Ajman tribe	52	49	3	76.00	24.00	Khaled,E.,et al.	1981

Table 5.1.7. VIII

Kidd blood groups distribution in Pakistan tested with anti-JK^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		JK(a+)	JK(a-)	JK^a	JK^b+JK	
1-Pathans.Swat,Saidu Sharif	127	87	40	43.88	56.12	Alciati, G. 1968
2-Punjabis.Lahore	67	49	18	48.16	51.84	Boyd,W.C., & Boyd,L.G.1954

Table 5.1.7. IX Kidd blood groups distribution in Afghanistan tested with anti-JK^a and anti-JK^b sera

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		JK(a+b+)	JK(a+b-)	JK(a-b+)	JK^a	JK^b	
1-Daris	38	22	8	8	50.00	50.00	Papiha,S.S.,et al. 1977
2-Pushtus	22	9	10	3	65.90	34.10	Papiha,S.S.,et al. 1977

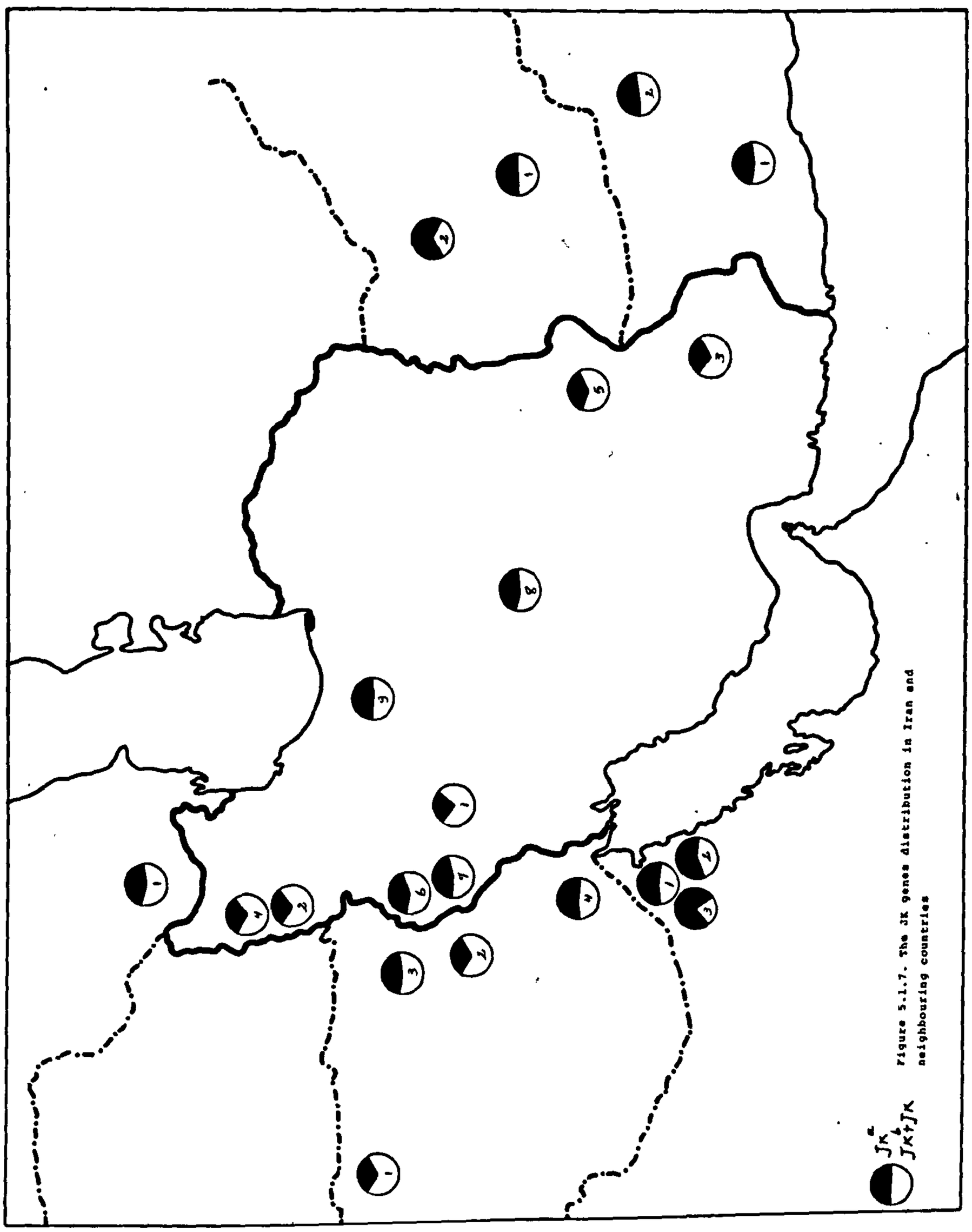


Figure 5.1.7. The JK genes distribution in Iran and neighbouring countries

5.1.8. The Lutheran blood group system

The distribution of the Lutheran blood groups and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.1.8.

The frequency of the Lu^a gene ranges from zero to 6.90 percent in Iranians, being lowest in the Kurdish Jews (Godber et al, 1973) and highest in the Tehrani series of Sawhney and Farhud (1979). There are only a few reports of Lutheran blood groups studies in Iranians. Godber et al (1973) and Tills et al (1977) reported Lu^a frequencies of zero and 0.47 percent in two groups of Kurdish Jews studied by them. Similar low Lu^a frequencies of 0.65 and 2.36 percent are given by Lehmann et al (1973) in two groups of Kurds of western Iran.

The relatively low Lu^a values in both the Kurds and the Kurdish Jews are about what would be expected in the region. More recently sawhney (1975) and Sawhney and Farhud (1979) reported high Lu^a frequencies of 6.81 and 6.90 percent in two Esfahani and Tehrani samples, respectively, which are much higher than Middle eastern and even higher than European values, showing the presence of the gene Lu^a at an appreciable level in Iran. However, as mentioned by the authors themselves, it must be substantiated by further research on ethnic, religious and geographically.isolated groups whether this frequency is a characteristic feature of Iranians or whether some other factors are involved.

On the whole, from the data available, the Iranian population with an average Lu^a frequency of 2.86 percent appears to exhibit a lower Lu^a frequency than that of about 4 percent in the populations of northern European origin. The average fre-

quency of the Lu^a gene in Iranians seems to be more similar to that of about 2 percent in the Mediterranean area (Mourant et al, 1976).

Regarding neighbouring populations, the frequency of the Lu^a gene in the populations of the Caucasus ranges from zero in the Georgians (Kherumian et al, 1954) to 8.44 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1971). Apart from the Lu^a frequency of zero which possibly is due to the very small sample size, the frequency of 8.44 percent is exceptionally high, even for Europe.

The relatively low Lu^a frequencies of 0.43 and 1.40 percent in the Eti-Turks and the Turks of Turkey (Aksoy et al, 1958) are about what would be expected for the Middle East.

Similar low Lu^a frequencies, varying between 0.82 and 1.87 percent are reported by Tills et al (1977) and Godber et al (1973) in Iraqi Kurdish Jews.

The frequency of the Lu^a gene in the populations of Saudi Arabia ranges from zero in the Sunni sample of Asir, Hejaz, Najran (Maranjian et al, 1966) to 12.21 percent in western Saudi Arabians (Saha et al, 1980). With the exception of the extremely high Lu^a value of 12.21 percent, which if true,possibly suggests African admixture, all other frequencies varying between zero and 1.09 percent are lower than the European frequency and about what would be expected for the region.

Clegg et al (1961) and Ikin and Warburton (1959) reported no Lu(a+) phenotype in the two Pakistani samples studied by them.

The Lu^a gene frequency in the populations of Afghanistan varies between zero in the Pushtus and 3.90 percent in the Daris (papiha et al, 1977), figures similar to those found

in Iranians and about what would be expected for the region.

Conclusion

It is suggested that the gene Lu^a has a frequency of about 4 percent in populations of northern European origin. In the Mediterranean area the frequency falls to about 2 percent, but in the Middle East the range lies between 1 and 2 percent. In Africa, though frequencies are variable, the average is nearly as high as in northern Europe. The gene is very rare or absent in all other indigenous populations examined (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Lu^a frequency of 8.44 percent in the Svanis of Georgia, Abkhazeskaya of the Caucasus (Verbitsky, et al,1971) which is exceptionally high, even higher than the European frequency, all other reported frequencies of the Lu^a gene (average between about 1 and 2 percent) are lower than the European frequency and about what would be expected for the region.

The extremely high Lu^a frequency of 12.21 percent in western Saudi Arabians (Saha et al, 1980) contrasts strongly with the low frequencies varying between zero and 1.09 percent in other Saudi Arabian samples (Maranjian et al, 1966) and, if true, seems to be suggestive of African admixture.

Table 5.1.8.I Lutheran blood groups distribution in Iran tested with anti-Lu^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		Lu(a+)	Lu(a-)	Lu^a	Lu^b		
1-Kurdish Jews	94	-	94	0.00	100.00	Godber,Marilyn J., et al.	1973
2-Kurdish Jews	106	1	105	0.47	99.53	Tills,D.,et al.	1977
3-Kurds.Baneh,Marivan	77	1	76	0.65	99.35	Lehmann,H.,et al.	1973
4-Kurds.Sanandaj	107	5	102	2.36	97.64	Lehmann,H.,et al.	1973
5-Esfahan	76	10	66	6.81	93.19	Sawhney,K.S.	1975
6-Moslems.Tehran	112	15	97	6.90	93.10	Sawhney,K.S.,& Farhud,D.D.	1979

Table 5.1.8. II Lutheran blood groups distribution in the Caucasus tested with anti-Lu^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Lu(a+)	Lu(a-)	Lu^a	Lu^b	
1-Georgians	19	-	19	0.00	100.00	Kherumian,R.,et al. 1954
2-Svanis.Georgia	77	10	67	8.44	91.56	Verbitsky,M.Sh., 1971 et al.
Abkhazskaya						

Table 5.1.8. III Lutheran blood groups distribution in Turkey tested with anti-Lu^a serum only.

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		Lu(a+)	Lu(a-)	Lu^a	Lu^b		
1-Eti-Turks.Asia Minor, near Mersin	116	1	115	0.43	99.57	Aksoy,M.,et al.	1958
2-Turks.Asia Minor, Mersin	108	3	105	1.40	98.60	Aksoy,M.,et al.	1958

Table 5.1.8.IV Lutheran blood groups distribution in Iraq tested with anti-Lu^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		Lu(a+)	Lu(a-)	Lu^a	Lu^b		
1-Kurdish Jews. North West	61	1	60	0.82	99.18	Tills,D., et al.	1977
2-Kurdish Jews. South east	50	1	49	1.01	98.99	Tills,D., et al.	1977
3-Kurdish Jews	27	1	26	1.87	98.13	Godber,Marilyn J., et al.	1973

Table 5.1.8.VI Lutheran blood groups distribution in Saudi Arabia tested with anti-Lu^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Lu(a+)	Lu(a-)	Lu^a	Lu^b	
1-Sunnis.Asir,Hejaz	176	-	176	0.00	100.00	Maranjian,G.,et al. 1966
2-Sunnis.Najd	180	3	177	0.84	99.16	Maranjian,G.,et al. 1966
3-Bedouins	178	3	175	0.85	99.15	Maranjian,G.,et al. 1966
4-Shias.Qatif & Hasa Oases	465	9	456	0.97	99.03	Maranjian,G.,et al. 1966
5-Sunnis,Qatif & Hasa Oases and east	323	7	316	1.09	98.91	Maranjian,G.,et al. 1966
6-Western Saudi Arabia	185	26	159	12.21	87.79	Saha, N., et al. 1980

Table 5.1.8. VIII Lutheran blood groups distribution in Pakistan tested with anti-Lu^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Lu(a+)	Lu(a-)	Lu^a	Lu^b	
1-Baltis.Baltistan	80	-	80	0.00	100.00	Clegg,E.J.,et al. 1961
2-Hunza,Gilgit	23	-	23	0.00	100.00	Ikin,Elizabeth W., & Warburton,K. 1959

Table 5.1.8. IX Lutheran blood groups distribution in Afghanistan tested with anti-Lu^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Lu(a+)	Lu(a-)	Lu^a	Lu^b	
1-Pushtus	22	-	22	0.00	100.00	Papiha,S.S.,et al. 1977
2-Timuri & related tribes	90	3	87	1.68	98.32	Woodd-Walker,R.B., et al. 1967
3-Daris	38	3	35	3.90	96.10	Papiha,S.S.,et al. 1977

5.1.9. The Diego blood group system

The distribution of the Diego blood groups and respective gene frequencies in Iranian and neighbouring populations is shown in Tables 5.1.9.

Only a single example of Di(a+) has been found in the Kurds of Iran (Lehmann et al, 1973), the Baltis of Pakistan (Clegg et al, 1961) and the Timuri and related tribes of Afghanistan (Woodd-Walker et al, 1967).

Conclusion

The Di^a gene is essentially characteristic of Mongoloids and has never been found in any survey of unselected persons of presumed unmixed European descent. It appears to be totally absent among Africans. The highest frequencies of the Di^a gene(up to 40 percent) are found among south and central American Indians.

In eastern Asia it is constantly present, though frequencies are not quite as high as in south and central America. The highest gene frequencies in Asia, around 5 percent, are found in Koreans and Tibetans. It is also present in Chinese, Japanese, the people of Thailand, northewest India and Nepal (Mourant et al, 1976).

The presence of even one Diego(Di^a) positive among the Iranian, the Pakistani, and the Afghan populations suggests a Mongoloid contribution to the gene pools.

It is most desirable that data should be obtained on its distribution in the Iranian Turkmans with Mongoloid origin and in the indigenous peoples of the Asiatic part of the Soviet Union.

Table 5.1.9.I Diego blood groups distribution in Iran tested with anti-Di^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Di(a+)	Di(a-)	Di^a	Di^b	
1-Arabs.Abadan	98	-	98	0.00	100.00	Nijenhuis,L.E. 1964
2-Iranians	16	-	16	0.00	100.00	Nijenhuis,L.E. 1964
3-Kurdish Jews	116	-	116	0.00	100.00	Gödber,Marilyn.J., et al. 1973
4-Arabs	100	-	100	0.00	100.00	Nijenhuis,L.E. 1958
5-Kurds.Sanandaj	107	-	107	0.00	100.00	Lehmann,H.,et al. 1973
6-Kurds.Baneh,Mar-vivan	77	1	76	0.65	99.35	Lehmann,H.,et al. 1973

Table 5.1.9. VIII Diego blood groups distribution in Pakistan tested with anti-Di^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Di(a+)	Di(a-)	Di^a	Di^b	
1-Hunza,Gilgit	23	-	23	0.00	100.00	Ikin,Elizabeth W.,1959 & Warburton,K.
2-Pathans	139	-	139	0.00	100.00	Vos,G.H.,& Kirk, R.L. 1961
3-Punjabis	168	-	168	0.00	100.00	Vos,G.H.,& Kirk, R.L. 1961
4-Baltis.Baltistan	79	1	78	0.64	99.36	Clegg,E.J.,et al. 1961

Table 5.1.9.IX Diego blood groups distribution in Afghanistan tested with anti-Di^a serum only

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Di(a+)	Di(a-)	Di^a	Di^b	
1-Timuri and related tribes	100	1	99	0.50	99.50	Woodd-Walker,R.B.,1967 et al.

5.1.10. The ABH secretion system

The distribution of secretor and non-secretor phenotypes and respective gene frequencies in Iranian and neighbouring populations is shown in Tables 5.1.10.
There are few reports on secretor status in Iranian and neighbouring populations.

The frequency of the Se allele ranges from 29.30 to 44.00 percent in Iranians, being lowest in southern Gorgan, Behshahr, Sari and highest in Tavalesh, Astara (Kirk et al, 1977). With an average Se frequency of 33.98 percent, the Iranian population appears to exhibit a lower Se frequency than that of around 50 percent in Europeans (Mourant et al, 1976).

Regarding neighbouring populations, the Se allele frequency of 49.29 percent in the Georgians of the Caucasus(Kherumian et al, 1954) is higher than the average for Iran and more similar to the European frequency.

Information on the distribution of secretor genes in other neighbouring populations is lacking.

Conclusion

The frequency of the Se gene in England is about 51 percent. Frequencies in continental Europe tend to be slightly higher than this, without any marked regional trend.
In the Indian region there is a wide Scatter of frequencies, on the whole high in the north and low in the south (Mourant et al, 1976).

In Iranians frequencies of the Se gene seem to be lower than those in Europeans.

Table 5.1.10.I ABH Secretor and Non-secretor phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		Sec	Non-Sec	Se	se		
1-Southern Gorgan, Behshahr,Sari	44	22	22	29.30	70.70	Kirk,R.L.,et al.	1977
2-Shahsavar,Rudsar, Rudbar,Rasht,Langa-rud,Lahijan,Bandar-Pahlavi	88	45	43	30.10	69.90	Kirk,R.L.,et al.	1977
3-Northern Gorgan	48	25	23	30.80	69.20	Kirk,R.L.,et al.	1977
4-Arabs.Khuzistan	91	48	43	31.20	68.80	Marzban,M.	1978
5-Gonbad	153	83	70	32.40	67.60	Kirk,R.L.,et al.	1977
6-Babol,Shahi,Amol	78	50	28	40.10	59.90	Kirk,R.L.,et al.	1977
7-Tavalesh,Astara	51	35	16	44.00	56.00	Kirk,R.L.,et al.	1977

Table 5.1.10. II ABH Secretor and Non-Secretor phenotypes and gene frequencies distribution in the Caucasus

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Sec	Non-Sec	Se	se	
1- Georgians	35	26	9	49.29	50.71	Kherumian,R., et al. 1954

5.1.11. Other blood group systems

In this part, data on some other blood group systems are presented. Since data available on these systems are not representative for the region, no discussion will be given.

Table 5.1.11. a. I. In a blood groups in Iran : tested with anti- In^a serum

Population	Number Tested	In(a+)	In(a+)%	Authors
Iranians	557	59	10.59	Badaker,S.S., et al. 1980

Table 5.1.11. b. I. Wright blood groups in Iran : tested with anit-Wr^a serum

Population	Number Tested	Wr(a+)	Wr(a+)%	Authors
1-Kurdish Jews	116	-	0.00	Godber,Marylin J., et al. 1973
2-Kurds.Baneh,Marivan	77	-	0.00	Lehmann,H.,et al. 1973
3-Kurds.Sanandaj	107	2	1.87	Lehmann,H.,et al. 1973

Table 5.1.11. C. I. Radin blood groups in Iran : tested with anti-Rd serum

Population	Number Tested	Rd+	Rd+%	Authors
1-Kurds.Baneh,Marivan	77	-	0.00	Lehmann,H., et al. 1973
2-Kurds. Sanandaj	107	-	0.00	Lehmann,H., et al. 1973

Table 5.1.11.d.VII. Vel blood groups in the United Arab Emirates: tested with anti-Ve^a serum

Population	Number Tested	Ve(a+)	Ve(a-)	Ve^a	Ve^b	Authors
Abu Dhabians	100	98	2	85.90	14.10	Kamel,K.,et al. 1980

5.2. SERUM PROTEINS

5.2.1. The haptoglobin(HP) system

The distribution of haptoglobin types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.2.1.

The frequency of the HP^1 gene ranges from 14.50 to 35.57 percent in Iranians, being lowest in northern Gorgan (Kirk et al, 1977) and highest in the Shirazi series of Walter and Djahanshahi (1963). Values obtained in the present investigation are within this range of variation.

On the whole, with an average HP^1 frequency of 26.85 percent, the Iranian population appears to exhibit a much lower HP^1 frequency than that of around 40 percent found in Europeans (Mourant et al, 1976).

It is difficult to see any definite regional trends but the HP^1 gene frequency tends to be lower in the east than the west.

The relatively lower HP^1 frequencies in the Caspian sea area of Iran (Kirk et al, 1977; Farhud et al, 1978) could be related to tropical disease syndromes, such as malaria, sickle cell disease, G6pD deficiency, etc, which are more common in this area.

Regarding neighbouring areas, the frequency of the HP^1 gene in the populations of the Caucasus ranges from 28.49 percent in the Azerbaijanians of Nukha to 35.04 percent in the Gurdjaanis of Georgia (Voronov, 1968). With an average HP^1 frequency of 31.76 percent, the population of the Caucasus appears to exhibit a higher HP^1 frequency than that found in Iranians but lower than the European frequency.

The frequency of the HP^1 gene in the populations of

Turkey varies between 25.09 percent (Hummel et al, 1970)and 31.72 percent (Erdem et al, 1966) both in the Turks of Turkey. with an average HP^1 frequency of 27.66 percent, the population of Turkey seems to show a slightly higher HP^1 frequency than that found in Iranians but lower than the frequency in Europeans.

The HP^1 gene frequency in the Jewish populations of Iraq ranges from 27.00 percent in the Kurdish Jews of the south east to 34.43 percent in the Kurdish Jews of the north west (Tills et al, 1977). With an average HP^1 frequency of 29.88 percent, the Jewish population of Iraq, like that of Iran, appears to exhibit a relatively higher HP^1 frequency than the average for the region.

The frequency of the HP^1 gene in the populations of Kuwait varies between 26.00 percent in the general population of Kuwait and 51.00 percent in the Ajman tribe (Khaled et al, 1981). With an average HP^1 frequency of 37.65 percent, the Arab population of Kuwait seems to show a much higher HP^1 frequency than that found in Iranians.

The HP^1 gene frequency in the populations of Saudi Arabia ranges from 36.64 percent in western Saudi Arabians (Saha et al, 1980) to 43.20 percent in the Saudi Arabian sample of Goedde et al (1979). With an average HP^1 frequency of 39.92 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit a much higher HP^1 frequency than that found in Iranians.

On the whole, the overall frequency of HP^1 in Arab populations seems to be higher than in other Asiatic populations but lower than an average of about 70 percent in Africans (Giblett, 1969; Mourant et al, 1976). Generally, haptoglobin

is a system indicating a substantial influx of African genes in the Arabian Peninsular populations.

The frequency of the HP^1 gene in the populations of Pakistan varies between 20.00 percent in the Punjabis (Kirk et al, 1961) and 30.49 percent in the Pathanis (Spedini,1969). With an average HP^1 frequency of 25.52 percent, the population of Pakistan seems to show a slightly lower HP^1 frequency than that found in Iranians and lower than the European frequency.

The HP^1 gene frequency in the populations of Afghanistan ranges from 23.70 percent in the Pushtus to 29.10 percent in the Daris (Papiha et al, 1977). With an average HP^1 frequency of 26.47 percent, the Afghan population appears to exhibit a HP^1 frequency similar to that found in Iranians and lower than the frequency in Europeans.

Conclusion

The frequency of the HP^1 gene in Europe varies around 40 percent; it is difficult to see any definite regional trends, but it tends to be lower in the south than the north. The HP^1 gene frequency is mostly low, around 15 percent, in the Indian region.

In Africa it is usually high, averaging about 70 percent(Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab populations of Kuwait and Saudi Arabia with their higher frequencies, the frequency of the HP^1 gene varying between 25 percent in the Pakistani population and 32 percent in the population of the Caucasus, seems to be lower than that found in Europeans but higher than that in Indians. The overall frequency of HP^1 in Arab populations seems to be

higher than in other Asiatic populations. Generally, haptoglobin is a system indicating a substantial influx of African genes in the Arabian Peninsular populations.

However, there appears to be a cline of decreasing HP^1 gene frequencies from Europe to India via the Middle East (Farhud, 1980). From the data reported in the present investigation on Iranian and neighbouring populations the same trend seems to exist, as the HP^1 frequency decreases obviously from the west towards the east, whilst the HP^2 frequency increases.

The precise significance of this cline in terms of environmental and genetic causation, can not at present be gauged but under the assumption that the HP frequencies obtained in the published samples from India are not due to chance, genetic drift or isolation effects, this observation might be of some anthropological value. In this sense one may consider the possibility of selective forces acting on the HP frequencies. Assuming that the high HP^2 frequencies in Iran, Pakistan and India may indicate some slective advantage of this allele, by which it might be able to extend considerably.. In this connection on may consider that the relatively large HP^2 moluecules can not easily pass the Kidney barrier, by which the organism will be protected from serious losses of Hb-connected Iron, which becomes disengaged with haemolytic ananemias, caused by G6pD deficiency, sickle cell disease, etc. Similar ideas have been expressed by Schultz and heremans (1966), who wrote: "since haptoglobin is in some way involved in the metabolism of haemoglobin being catabolized in excess during haemolytic episodes such as occur in malaria, it is not unthinkable that the latter disease may have

had a hand in the presently observed ascendancy of the HP^2 genes". Assuming these considerations the hypothesis may be formulated that the increase of HP^2 frequencies from the west towards the east is a result of genetic adaptation to specific environmental conditons, in which haemolytic anaemias such as occur in malaria seem to be of importance. Unfortunately we have no exact data concerning the frequency and distribution of haemolytic anaemias in south western and southern Asia, so that confidence in this hypothesis can not be checked. Nevertheless, attention should be paid to this problem. The solution will enable us to understand the geographical differences in the distribution of the HP alleles, and moreover it will improve our knowledge on the action of selective mechanisms in man.

Table 5.2.1.I Haptoglobin(HP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP 0	HP^1	HP^2		
1-Northern Gorgan	38	-	11	27	-	14.50	85.50	Kirk,R.L.,et al.	1977
2-Zoroastrians.Tehran & Yazd	145	6	43	96	-	18.97	81.03	Bowman,J.E.	1964
3-Southern Gorgan,Behshahr,Sari	52	2	17	33	-	20.20	79.80	Kirk,R.L.,et al.	1977
4-Babol,Shahi,Amol	61	1	24	36	-	21.30	78.70	Kirk,R.L.,et al.	1977
5-Gonbad	153	5	56	91	1	21.70	78.30	Kirk,R.L.,et al.	1977
6-Zabolis.Sistan & Baluchistan	118	3	45	69	1	21.79	78.21	Present study.	
7-Kurdish Jews	94	7	27	59	1	22.04	77.96	Godber,Marilyn.J.,et al.	1973
8-Shahsavar,Rudsar,Rudbar, Rasht,Langarud,Lahijan, Bandar-Pahlavi	84	3	30	48	3	22.20	77.80	Kirk,R.L.,et al.	1977
9-Lurs.Luristan	178	10	61	106	1	22.88	77.12	Present study.	
10-Kurds.Shirvan,Khorasan	102	4	39	58	1	23.27	76.73	Present study.	

Table 5.2.1.I (Cont.) Haptoglobin (HP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP 0	HP^1	HP^2		
11-Turks.Shirvan,Khorasan	106	3	43	57	3	23.79	76.21	Present study.	
12-Baluchis.Sistan & Baluchistan.	111	5	43	59	4	24.77	75.23	Present study.	
13-East	179	13	63	103	-	24.90	75.10	Bajatzadeh,M., & Walter,H.	1969
14-Caspian Sea area	240	11	96	130	3	24.90	75.10	Farhud,D.D.,et al.	1978
15-Iranians	34	2	13	19	-	25.00	75.00	Harris, H.	1959
16-Tavalesh.Astara	60	1	28	31	-	25.00	75.00	Kirk,R.L.,et al.	1977
17-Kurdish Jews	106	9	35	61	1	25.24	74.76	Tills,D.,et al.	1977
18-Turks.Rezaieh	145	11	51	79	4	25.89	74.11	Present study.	
19-Zoroastrians	171	15	56	95	5	25.90	74.10	Present sutdy.	
20-Central and South	245	20	89	136	-	26.30	73.70	Bajatzadeh,M., & Walter,H.	1969
21-Dezfool	545	33	217	288	7	26.30	73.70	Farhud,D.D., et al.	1977
22-Tehran	136	14	41	76	5	26.34	73.66	Present study.	

Table 5.2.1.I (Cont.) Haptoglobin (HP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP 0	HP^1	HP^2		
23-Bandar.Abass	1246	105	441	678	22	26.60	73.40	Farhud,D.D., et al.	1978
24-Kurds.Sanandaj	108	7	43	56	2	26.89	73.11	Lehmann,H.,et al.	1973
25-Tehran	366	28	145	193	-	27.00	73.00	Farhud,D.D., & Walter, H.	1972
26-Ghashghais	510	42	185	262	21	27.50	72.50	Farhud,D.D., & Amirshahi,P. un-published	
27-North West	250	29	80	141	-	27.60	72.40	Bajatzadeh,M., & Walter, H.	1969
28-Iranians	1061					28.00	72.00	Miyashita,T.K.,et al.	1975
29-Iranians	627	49	254	324	-	28.07	71.93	Farhud,D.D.	1980
30-West	313	33	111	169	-	28.20	71.80	Bajatzadeh,M., & Walter,H.	1969
31-Moslems.Shiraz	429	34	176	219	-	28.44	71.56	Bowman, J.E.	1964
32-Jews	101	8	43	50	-	29.21	70.79	Fried,K.,et al.	1963
33-Tehran	186	19	71	96	-	29.30	70.70	Sawhney, K.S.	1975
34-Jews	91	9	37	45	-	30.22	69.78	Ramot,Bracha.,et al.	1962

Table 5.2.1.I (Cont.) Haptoglobin (HP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP 0	HP^1	HP^2		
35-Esfahan	91	6	42	41	2	30.34	69.66	Sawhney,K.S.	1975
36-Kerman	294	31	108	141	14	30.36	69.64	Present study.	
37-Kurds.Rezaieh	145	18	50	72	5	30.71	69.29	Present study.	
38-North	179	27	57	95	-	31.00	69.00	Bajatzadeh,M., & Walter, H.	1969
39-Jews	158					31.00	69.00	Simhai,B.	1976
40-Shahsavar	168	16	75	77	-	31.80	68.20	Farhud,D.D.,et al.	1978
41-Jews	159	18	66	74	1	32.00	68.00	Tabatabai, H.	1977
42-Tehran	400	53	153	194	-	32.40	67.60	Bajatzadeh,M., & Walter, H.	1969
43-Ghashghais	117	15	46	56	-	32.48	67.52	Bowman,J.E.	1964
44-Kurds.Baneh,Marivan	77	8	31	33	5	32.64	67.36	Lehmann,H.,et al.	1973
45-Armenians	228	15	127	85	1	34.40	65.60	Tabatabai, H.	1977
46-Shiraz	97	12	45	40	-	35.57	64.43	Walter,H., & Djahanshahi, D.	1963

Table 5.2.1.b.I. Haptoglobin (HP) Subtypes in Iran

Population	Number Tested	Phenotypes						Gene frequencies			Authors
		HP 1s-1s	HP 1s-1F	HP 1F-1F	HP 2-1s	HP 2-1F	HP 2-2	1s HP	1F HP	Hp^2	
Tehran	366	8	13	7	88	57	193	16.20	10.80	73.00	Farhud,D.D., and Walter, H. 1972

Table 5.2.1. II Haptoglobin (HP) phenotypes and gene frequencies distribution in the Caucasus

Population	Number Tested	Phenotypes				Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP 0	HP^1	HP^2		
1- Azerbaijan .Nukha	87	6	37	43	1	28.49	71.51	Voronov, A.A.	1968
2-Gurdjaanis.Georgia	141	16	64	57	4	35.04	64.96	Voronov, A.A.	1968

Table 5.2.1.III Haptoglobin(HP) phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes					Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP 2-1M	HP 0	HP^1	HP^2		
1-Turks	274	12	113	148	-	1	25.09	74.91	Hummel,K.,et al.	1970
2-Turks	102	5	42	53	-	2	26.00	74.00	Aksoy,M.,et al.	1980
3-Turks	200	15	76	109	-	-	26.50	73.50	Erdem,S., & Aksoy,M.	1975
4-Kurds							29.00	71.00	Richard, P.	1976
5-Turks	299	24	138	136	1	-	31.27	68.73	Erdem,S., et al.	1966

Table 5.2.1. IV Haptoglobin (HP) phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes					Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP 2-1M	HP 0	HP^1	HP^2		
1-Kurdish Jews.South east	50	4	19	27	-	-	27.00	73.00	Tills,D.,et al.	1977
2-Jews.Baghdad	197	14	79	103	1	-	27.41	72.59	Fried,K.,et al.	1963
3-Karaite Jews	69	3	33	33	-	-	28.30	71.70	Goldschmidt,E.,et al.	1976
4-Jews	118	9	50	59	-	-	28.81	71.19	Ramot,Brancha.,et al.	1961
5-Kurdish Jews	27	3	12	12	-	-	33.33	66.67	Godber,Marilyn.J., et al.	1973
6-Kurdish Jews.North west	61	7	28	26	-	-	34.43	65.57	Tills,D.,et al.	1977

Table 5.2.1.V Haptoglobin (HP) phenotypes and gene frequencies distribution in Kuwait

Population	Number Tested	Phenotypes				Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP 0	HP^1	HP^2		
1-General population	88	6	33	49	-	26.00	74.00	Khaled,E.,et al.	1981
2-Kuwaiti Arabs	161	24	61	73	3	34.59	65.41	Sawhney,K.S.	1975
3-Suluba tribe	51	9	22	20	-	39.00	61.00	Khaled,E.,et al.	1981
4-Ajman tribe	52	13	27	12	-	51.00	49.00	Khaled,E.,et al.	1981

Table 5.2.1. VI Haptoglobin(HP) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes					Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP Calb.	HP 0	HP^1	HP^2		
1-Western Saudi Arabia	292	35	133	109	-	15	36.64	63.36	Saha,N., et al.	1980
2-Saudi Arabians	352	64	168	111	1	8	43.20	56.80	Goedde,H.W.,et al.	1979

Table 5.2.1. VIII Haptoglobin (HP) phenotypes and gene frequencies distribution in Pakistan

Population	Number Tested	Phenotypes				Gene frequencies		Authors	
		HP 1-1	HP 2-1	HP 2-2	HP 0	HP^1	HP^2		
1-Punjabis	201	12	52	130	7	20.00	80.00	Kirk,R.L.,et al.	1961
2-Pathans	185	10	67	103	5	24.00	76.00	Kirk,R.L., et al.	1961
3-Peshawar	135	12	50	72	1	27.61	72.39	Walter,H.,et al.	1966
4-Pathanis.Swat	131	11	53	59	8	30.49	69.51	Spedini,Gaberiella.	1969

Table 5.2.1.IX Haptoglobin (HP) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes				Gene frequencies		Authors
		HP 1-1	HP 2-1	HP 2-2	HP 0	HP^1	HP^2	
1-Pushtus	95	3	39	53	-	23.70	76.30	Papiha,S.S., et al. 1977
2-Uzbeks	124	6	49	69	-	24.59	75.41	Rahimi,A.G.,et al. 1977
3-Pushtus	210	13	83	114	-	25.95	74.05	Rahimi,A.G.,et al. 1977
4-Tajiks	310	23	118	169	-	26.45	73.55	Rahimi,A.G.,et al. 1977
5-Afghans	141	12	53	76	-	27.30	72.70	Shim,Bong-Sop.,et al.1964
6-Hazaras	172	12	73	87	-	28.19	71.81	Rahimi,A.G.,et al. 1977
7-Daris	158	13	66	79	-	29.10	70.90	Papiha,S.S., et al. 1977

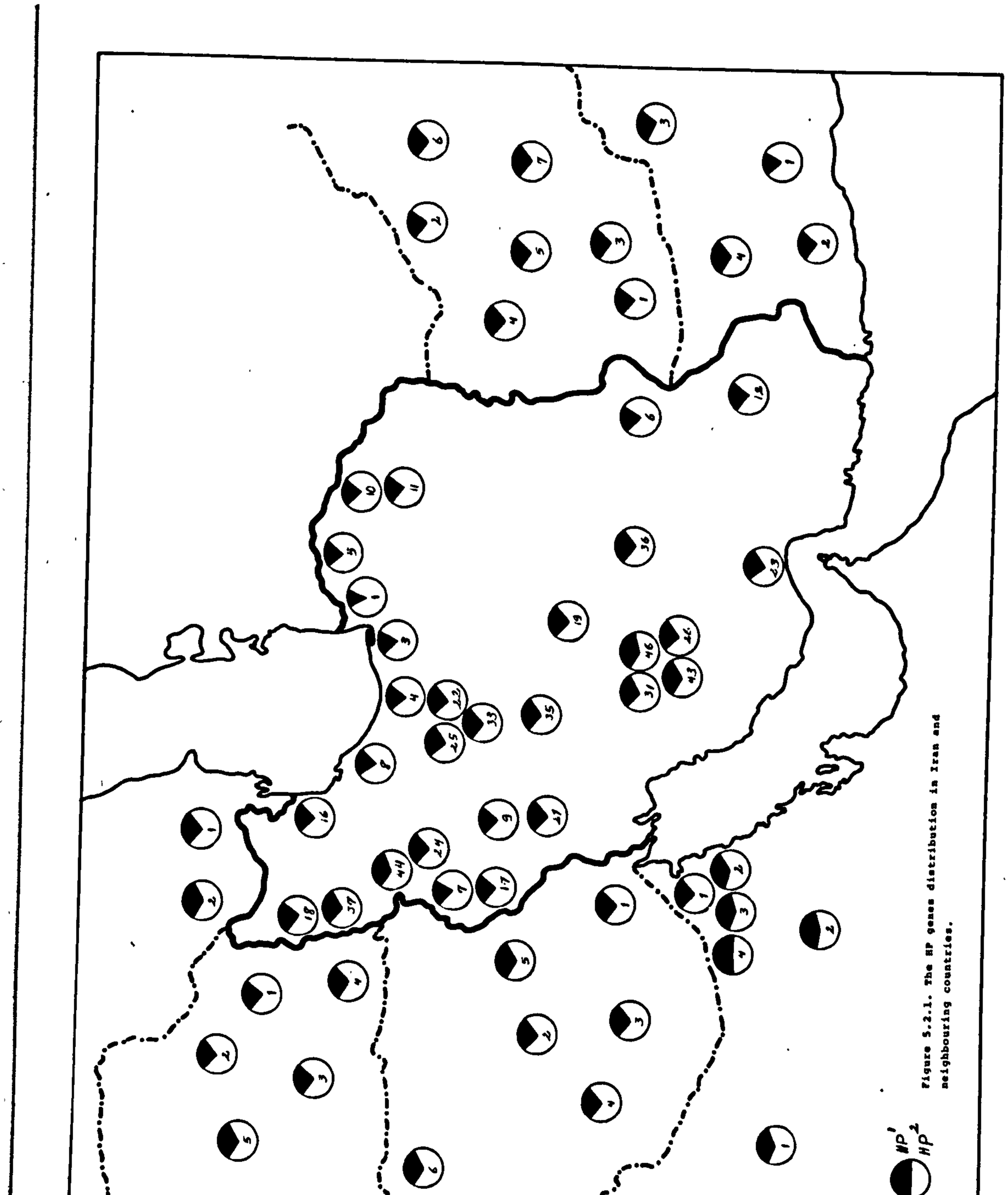


Figure 5.2.1. The HP genes distribution in Iran and neighbouring countries.

5.2.2. The transferrin (Tf) system

The distribution of transferrin groups and respective gene frequencies in Iranian and neighbouring populations is shown in Tables. 5.2.2.

Both TfB and TfD variants are found in Iranians but the TfD variant seems to be more frequent than TfB, the highest frequency of the Tf^D allele being 2.56 percent in the Ghashghai tribe (Bowman, 1964). It is difficult to see any definite regional trend of the distribution of the transferrin variants in Iran but it tends to be higher in the south than the north.

Regarding neighbouring groups, no transferrin variant other than type CC was observed in the Turks and the Kurds of Turkey (Dincol, Guncag et al, 1976; Richard, 1976).

Tills et al (1977) found no TfD variants in the two groups of Iraqi Kurdish Jews studied by them but they observed the relatively high Tf^B frequency of 1.64 percent in the Kurdish Jews of the north west and only TfCC type in the Kurdish Jews of the south east.

The only transferrin type observed in the three Kuwaiti samples investigated by Khaled et al (1981) was transferrin CC.

Saha et al (1980) and Goedde et al (1979) found no TfB but a high TfD variant frequency in the population of Saudi Arabia, the highest frequency of the Tf^D allele being 3.40 percent in western Saudi Arabians. A high frequency of TfD variant seems to be an indication of African admixture in the Arabs of Saudi Arabia.

Kirk et al (1961) observed no TfD variant in the Pathans

and the Punjabis of Pakistan but they found Tf^B variant with a low frequency of 0.50 percent in the Pathans.

Both TfD and TfB variants are found in the Afghan populations(Rahimi et al,1977;Papiha et al,1977).The high Tf^{Dchi} frequency of 1.73 percent in the Hazaras of Afghanistan(Rahimi et al,1977) can be explained as a Mongoloid marker(Kirk et al, 1968) because of its high frequency in Asiatic populations.

Conclusion

Frequencies of the transferrin variants other than Tf^C are very low in every population studied. The rarity of the variants suggests that the common TfC type has under most circumstances a selective advantage over all the others.
The transferrin B variants, though always rare, are the main ones found in European populations while the main distribution of the TfD variants are in the peoples of southern and south eastern Asia including India.
In Africa transferrin D variants are found to be more frequent(Mourant et al, 1976).

In Iranian and neighbouring populations,like other Asiatic populations,transferrin D variants seem to be more frequent than the B variants.

Generally, it is suggested that the transferrin D variants are much more frequent in tropical biotopes compared with non-tropical ones. Thus the finding of higher transferrin D variant frequencies than those of B variants in Iranian and neighbouring populations is in agreement with the hypothesis given by Walter and Bajatzadeh(1971) that: The nowadays recognizable geographical distribution of transferrin variants in man reflects the result of selective adaptations to particular environmental conditions . It is hypo-

thesized furthermore, that slowly moving transferrins in man are somehow associated with a better thermoregulation under the conditions of hot biotopes. On the other hand, faster moving transferrins might be of a greater adaptive value in non-tropical areas, indicating by higher Tf^B frequencies in human populations living in biotopes of moderate or arctic climate.

Table 5.2.2. I Transferrin (Tf) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies			Authors	
		TfCC	TfCB	TfCD	Tf^C	Tf^B	Tf^D		
1-Ghashghais	117	111	-	6	97.44	0.00	2.56	Bowman,J.E.	1964
2-Moslems.Shiraz	429	409	3	17	97.67	0.35	1.98	Bowman,J.E.	1964
3-Ghashghais	548	532	14	2	98.55	1.27	0.18	Farhud,D.D., & Daneshmand,P.	unpublished
4-Shahsavar	175	171	-	4	98.86	0.00	1.14	Farhud,D.D.,et al.	1978
5-Tehran	228	223	5	-	98.90	1.10	0.00	Farhud,D.D.	1979
6-Dezfool	587	576	5	6	99.07	0.42	0.51	Farhud,D.D.,& Daneshmand,P.	unpublished
7-Turks.Shirvan, Khorasan	122	120	1	1	99.18	0.41	0.41	Present study.	
8-Armenians	258	254	2	2	99.24	0.38	0.38	Farhud,D.D., & Daneshmand, P.	unpublished
9-Zoroastrians	145	143	-	2	99.31	0.00	0.69	Bowman,J.E.	1964
10-Jews	287	283	4	-	99.31	0.69	0.00	Farhud,D.D., & Daneshmand, P.	unpublished

Table 5.2.2. I(Cont.) Transferrin (Tf) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies			Authors	
		TfCC	TfCB	TfCD	Tf^C	Tf^B	Tf^D		
11-Caspian Sea area	344	340	1	3	99.42	0.15	0.43	Farhud,D.D.,et al.	1978
12-Esfahan	89	88	-	1	99.44	0.00	0.56	Sawhney,K.S.	1975
13-Bandar-Abass	901	893	3	5	99.56	0.16	0.28	Farhud,D.D., & Daneshmand, P.	unpublished
14-Zoroastrians	175	174	-	1	99.71	0.00	0.29	Present study.	
15-Kerman	313	312	-	1	99.84	0.00	0.16	Present study.	
16-Tehran	186	186	-	-	100.00	0.00	0.00	Sawhney,K.S.	1975
17-Tehran	136	136	-	-	100.00	0.00	0.00	Present study.	
18-Zabolis.Sistan & Baluchistan	118	118	-	-	100.00	0.00	0.00	Present study.	
19-Baluchis.Sistan & Baluchistan	111	111	-	-	100.00	0.00	0.00	Present study.	
20-Kurds.Shirvan, Khorasan	112	112	-	-	100.00	0.00	0.00	Present study.	
21-Kurds.Rezaieh	147	147	-	-	100.00	0.00	0.00	Present study.	
22-Kurds.Sanandaj	105	105	-	-	100.00	0.00	0.00	Lehmann,H.,et al.	1973

Table 5.2.2.I(Cont.) Transferrin(Tf) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies			Authors	
		TfCC	TfCB	TfCD	Tf^C	Tf^B	Tf^D		
23-Kurdish Jews	106	106	-	-	100.00	0.00	0.00	Tills,D.,et al.	1977
24-Turks.Rezaieh	149	149	-	-	100.00	0.00	0.00	Present study.	
25-Caspian Sea area	447	447	-	-	100.00	0.00	0.00	Kirk,R.L.,et al.	1977
26-Lurs.Luristan	178	178	-	-	100.00	0.00	0.00	Present study.	

Table 5.2.2. III Transferrin (Tf) phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes			Gene frequencies			Authors
		TfCC	TfCB	TfCD	Tf^C	Tf^B	Tf^D	
1-Turks	300	300	-	-	100.00	0.00	0.00	Dinçol, Gunçag., et al. 1976
2-Kurds					100.00	0.00	0.00	Richard,P. 1976

Table 5.2.2. IV Transferrin (Tf) phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes			Gene frequencies			Authors	
		TfCC	TfCB	TfCD	Tf^C	Tf^B	Tf^D		
1-Kurdish Jews. North west	61	59	2	-	98.36	1.64	0.00	Tills,D., et al.	1977
2-Kurdish Jews. South east	50	50	-	-	100.00	0.00	0.00	Tills,D., et al.	1977

Table 5.2.2.V Transferrin (Tf) phenotypes and gene frequencies distribution in Kuwait

Population	Number Tested	Phenotypes			Gene frequencies			Authors
		TfCC	TfCB	TfCD	Tf^C	Tf^B	Tf^D	
1-General population	89	89	-	-	100.00	0.00	0.00	Khaled,E., et al. 1981
2-Ajman tribe	52	52	-	-	100.00	0.00	0.00	Khaled,E., et al. 1981
3-Suluba tribe	52	52	-	-	100.00	0.00	0.00	Khaled,E., et al. 1981

Table 5.2.2. VI Transferrin (Tf) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes			Gene frequencies			Authors
		TfCC	TfCB	TfCD	Tf^C	Tf^B	Tf^D	
1-Western Saudi Arabia	191	178	-	13	96.60	0.00	3.40	Saha,N., et al. 1980
2-Saudi Arabians	359	355	-	4(chi)	99.40	0.00	0.60 Dchi	Goedde,H.W.,et al. 1979

Table 5.2.2. VIII Transferrin (Tf) phenotypes and gene frequencies distribution in Pakistan

Population	Number Tested	Phenotypes			Gene frequencies			Authors
		TfCC	TfCB	TfCD	Tf^C	Tf^B	Tf^D	
1- Pathans	185	183	2	-	99.50	0.50	0.00	Kirk,R.L.,et al. 1961
2-Punjabis	207	207	-	-	100.00	0.00	0.00	Kirk,R.L.,et al. 1961

Table 5.2.2. IX Transferrin (Tf) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		TfCC	Tf CB		TfCD				
			B_2C	B_1C		Tf^C	Tf^B	Tf^D chi	
1-Hazaras	172	164	1	1	6 chi	97.68	0.59	1.73	Rahimi,A.G.,et al. 1977
2-Pushtus	95	92	3		-	98.40	1.60	0.00	Papiha,S.S.,et al. 1977
3-Tajiks	310	305	3	1	1 chi	99.19	0.64	0.17	Rahimi,A.G.,et al. 1977
4-Pushtus	210	207	2	-	1 chi	99.28	0.48	0.24	Rahimi,A.G.,et al. 1977
5-Daris	160	160	-		-	100.00	0.00	0.00	Papiha,S.S.,et al. 1977
6-Uzbeks	124	124	-	-	-	100.00	0.00	0.00	Rahimi,A.G.,et al. 1977

5.2.3. The third component of human complement (C3)system

The distribution of C3 types and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.2.3.

There are only a few reports of C3 studies in Iranian and neighbouring populations.

The frequency of the $C3^F$ gene ranges from 5.78 to 23.30 percent in Iranians, being lowest in the Kermani series (present investigation) and highest in the Caspian sea area (Farhud et al, 1978).

On the whole, with an average $C3^F$ frequency of 13.00 percent, the Iranian population appears to exhibit a lower $C3^F$ frequency than that of 21 percent found in Europeans(Goedde et al, 1972).

Since the data available on the distribution of C3 types in various populations of Iran are still not large enough, full discussion is not yet possible.

Regarding neighbouring populations, the frequency of the $C3^F$ gene in the populations of Iraq varies between 19.00 percent in the Arabs of Baghdad and 27.20 percent in the Kurds of Mosul (Roberts and Al-Agidi, 1979). With an average $c3^F$ frequency of 23.30 percent, the population of Iraq seems to show a higher $C3^F$ frequency than that found in Iranians and more similar to the European frequency.

The $C3^F$ frequency of 9.20 percent in Saudi Arabians (Goedde et al, 1979) is lower than the average for Iran and much lower than the frequency in Europe.

The frequency of the $C3^F$ gene in the populations of Afghanistan ranges from 8.72 percent in the Hazaras to 14.03 percent in the Tajiks (Rahimi et al, 1977). With an average

$C3^F$ frequency of 12.11 percent, the Afghan population appears to exhibit a lower $C3^F$ frequency than that found in Iranians and lower than the frequency in Europeans.

Conclusion

The frequency of the $C3^F$ gene ranges from 19 to 23 percent (average 21 percent) in European populations(Goedde et al, 1972).

$C3^F$ frequency, ranging from 2.5 to 10 percent (average 6.4 percent), is consistently lower in the Indian region(Papiha et al, 1979).

Low frequencies of $C3^F$ have also been observed in Mongoloids and Negroids (Bender, 1975).

In Iranian and neighbouring populations, with the exception of the population of Iraq with their higher $C3^F$ values which are more similar to the European frequencies, the frequency of the $C3^F$ gene seems to be lower than that found in Europeans but higher than that in Indians.

Table 5.2.3.I The third component of human complement (C3) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		C3 SS	C3 SF	C3 FF	F C3	S C3	
1-Kerman	199	177	21	1	5.78	94.22	Present study.
2-Kurds.Shirvan, Khorasan	60	53	6	1	6.67	93.33	Present study.
3-Tehran	51	44	7	-	6.87	93.13	Present study.
4-Turks.Rezaieh	90	77	12	1	7.78	92.22	Present study.
5-Kurds.Rezaieh	89	76	12	1	7.87	92.13	Present study.
6-Turks.Shirvan , Khorasan	82	70	11	1	7.92	92.08	Present study.
7-Zabolis.Sistan & Baluchistan	109	80	27	2	14.22	85.78	Present study.
8-Zoroastrians	131	93	34	4	16.03	83.97	Present study.
9-Lurs.Luristan	143	101	37	5	16.43	83.57	Present study.
10-Iranians	101	66	28	7	20.79	79.21	Farhud,D.D., et al.1973
11-Baluchis.Sistan & Baluchistan	107	67	32	8	22.43	77.57	Present study.

Table 5.2.3.I (Cont.) The third component of human complement (C3) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		C3 SS	C3 SF	C3 FF	F C3	S C3	
12-Caspian Sea area	43	25	16	2	23.30	76.70	Farhud,D.D.,et al. 1978

Table 5.2.3. IV The third component of human complement (C3) phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes C3 SS	C3 SF	C3 FF	Gene frequencies F C3	S C3	Authors	
1-Arabs.Baghdad	174	119	44	11	19.00	81.00	Roberts,D.E., & Al-Agidi,S.K.	1979
2-Arabs.Mosul	112	70	35	7	21.90	78.10	Roberts,D.E., & Al-Agidi,S.K.	1979
3-Arabs.Ramadi	130	82	39	9	21.90	78.10	Roberts,D.E., & Al-Agidi,S.K.	1979
4-Arabs.Basrah	158	97	51	10	22.50	77.50	Roberts,D.E., & Al-Agidi,S.K.	1979
5-Turks.Kirkuk	120	74	36	10	23.30	76.70	Roberts,D.E., & Al-Agidi,S.K.	1979
6-Kurds.Baghdad	145	86	46	13	24.80	75.20	Roberts,D.E., & Al-Agidi,S.K.	1979
7-Turks.Baghdad	89	51	30	8	25.80	74.20	Roberts,D.E., & Al-Agidi,S.K.	1979
8-Kurds.Mosul	90	50	31	9	27.20	72.80	Roberts,D.E., & Al-Agidi,S.K.	1979

Table 5.2.3. VI The third component of human complement (C3) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes					Gene frequencies			Authors
		C3 SS	C3 SF	C3 FF	C3 SS0.4	C3 FS0.4	F C3	S C3	S0.4 C3	
1- Saudi Arabians	359	292	57	4	5	1	9.20	90.00	0.80	Goedde,H.W.,et al. 1979

Table 5.2.3. VIII The third component of human complement (C3) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		C3 SS	C3 SF	C3 FF	C3 SS0.4 SS0.25 FS0.25	$C3^F$	$C3^S$	rare variants C3	
1-Hazaras	172	142	26	2	2	8.72	9.69	0.59	Rahimi,A.G.,et al. 1977
2-Pushtus	210	164	42	4	-	11.91	88.09	0.00	Rahimi,A.G.,et al. 1977
3-Uzbeks	124	89	30	2	3	13.80	85.00	1.20	Rahimi,A.G.,et al. 1977
4-Tajiks	310	227	75	6	2	14.03	85.63	0.34	Rahimi,A.G.,et al. 1977

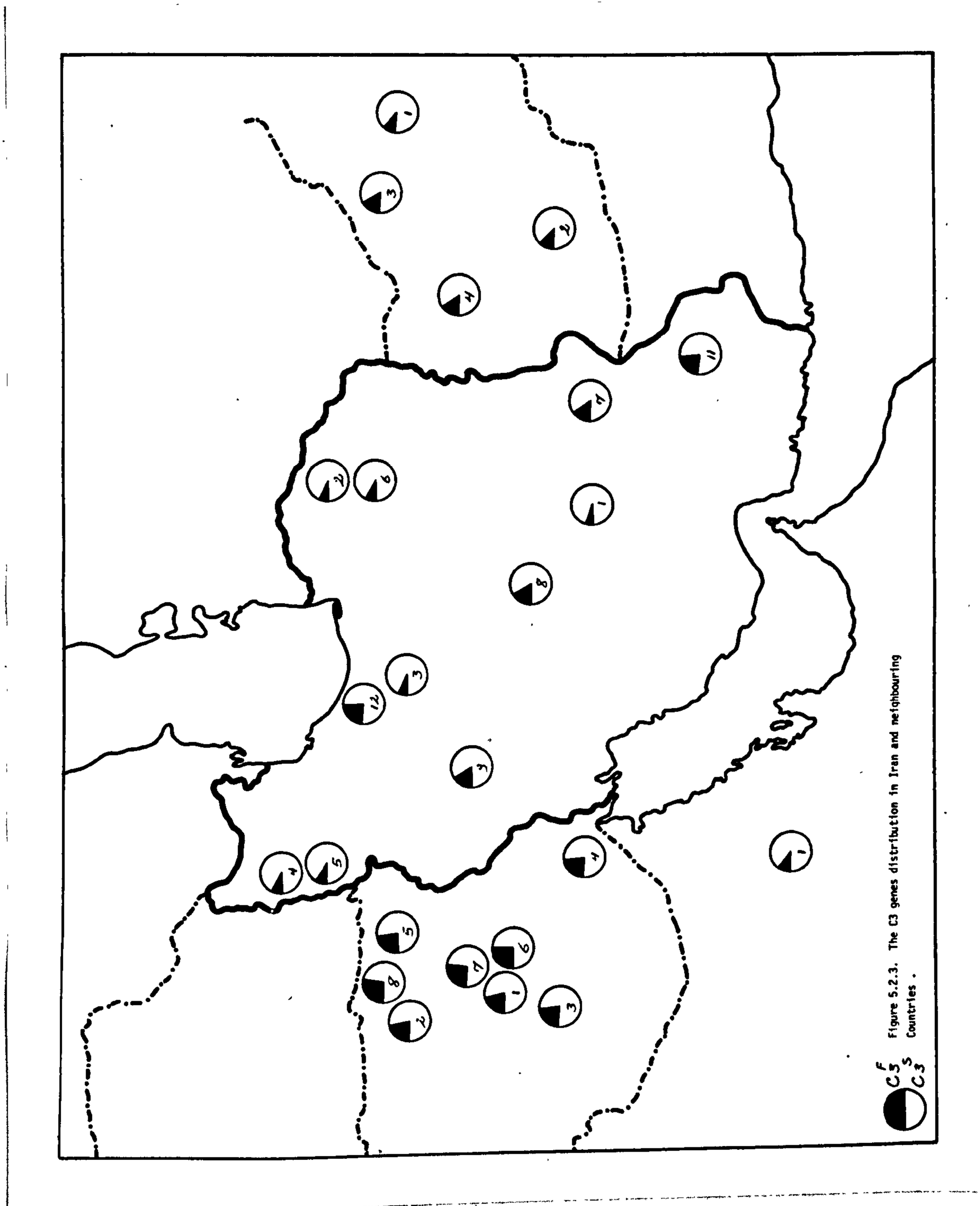


Figure 5.2.3. The C3 genes distribution in Iran and neighbouring Countries .

5.2.4. The group specific component(Gc) system

The distribution of Gc types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.2.4.

The frequency of the allele Gc^2 ranges from 19.05 to 37.90 percent in Iranians, being lowest in the Kurdish Jews(Cleve et al, 1962) and highest in the Tehrani series of Bajatzadeh and Walter(1969).

On the whole, with an average Gc^2 frequency of 30.40 percent, the Iranian population appears to exhibit a slightly higher Gc^2 frequency than that of around 27 percent found in Europeans (Mourant et al, 1976). It is difficult to see any definite regional trend of the distribution of Gc^2 in Iran but it tends to be lower in the south than the north.

Regarding neighbouring areas, the Gc^2 frequency of 25.73 percent in the Turks of Turkey (Hummel et al, 1970) seems to be lower than the average for Iran and also lower than the European frequency.

The frequency of the Gc^2 gene in the populations of Iraq varies between 17.20 percent in the Kurds (Constans et al, 1980) and 24.12 percent in the Jews (Cleve et al, 1962). With an average Gc^2 frequency of 20.66 percent, the Iraqi population appears to exhibit a lower Gc^2 frequency than that found in Iranians and also lower than the frequency in Europeans. The Gc^2 frequency of 24.12 percent in the Iraqi Jews, though lower than the average for Iran, is similar to the frequencies of 24.49 and 24.50 percent in the Iranian Jews (Kitchin and bearn,1964; Cleve et al, 1962). On the whole, it seems that Jewish populations both in Iran and Iraq resemble Europeans in Gc frequencies.

The Gc^2 frequency of 14.60 percent in Saudi Arabians(Goedde et al, 1979) appears to be much lower than the Iranian frequency and also much lower than the frequency in Europeans but more similar to the low frequencies in Africans (Mourant et al, 1976).

The frequency of the Gc^2 gene in the populations of Pakistan ranges from 19.63 percent in Peshawar (Walter, 1966) to 31.96 percent in the Pathanis (Spedini, 1969). With an average Gc^2 frequency of 26.09 percent, the Pakistani populations appears to exhibit a lower Gc^2 frequency than that found in Iranians and also lower than that in Europeans.

The Gc^2 gene frequency in the Afghan populations varies between 22.68 percent in the Hazaras (Rahimi et al, 1977) and 29.79 percent in the mixed Pakistani sample of Kitchin and Bearn (1964). With an average Gc^2 frequency of 24.70 percent, the population of Afghanistan, like that of Pakistan, seems to show a lower Gc^2 frequency than that found in Iranians and lower than the European frequency.

Conclusion

In Europe the frequency of the gene Gc^2 is nearly always between 20 and 30 percent.

In southern and south-western Asia frequencies are on the whole similar to those in Europe. There is considerable variation in the frequency of Gc^2 in India, varying between 10 and 37 percent. In south-east Asia Gc^2 frequencies are mostly a little over 20 percent.

The African populations differ considerably from those of Europe and Asia, mostly having less than 10 percent of Gc^2 (Mourant et al, 1976).

In Iranian and neighbouring populations, with the excep-

tion of Iranians with their slightly higher frequencies, the frequency of the Gc^2 allele appears to be lower than that found in Europeans but higher than that in Africans. The frequency of the gene Gc^2 in the Arab population of Saudi Arabia, being much lower than in the other population groups in the area, seems to be more similar to the African frequency.

It has recently been realized as a result of the discovery by Schanfield et al (1975) that the Gc proteins are the vitamin D carriers of the Plasma. Kirk et al (1963), Walter and Steegmuller (1969), Mourant et al (1976) and later Daiger (1979) and Constans(1980) have demonstrated that the frequency of the gene Gc^2 follows a cline, diminishing as the mean intensity of solar radiation increases. There is also a north-south gradient in the Gc gene frequencies. This seems to parallel the gradient seen in skin pigmentation. Thus Gc^1 has in general, though with several exceptions, a high frequency in sunny climates and Gc^2 a relatively high one in dull climates, suggesting that the Gc types are affected by natural selection related to the availability of vitamin D. However, the distribution of the Gc alleles seems to correlate with geographical clines, notably the incidence of solar radiation, as might be expected for the protein which transports vitamin D.

The very low Gc^2 frequency in the Arabs of Saudi Arabia, like that in African populations, can perhaps be best explained by the action of geographical factors in relation to the metabolic role of this protein in the organism (Daiger and Cavalli-Sforza, 1977; Constans et al, 1979). These findings confirm the assumption of the presence of a gradient in the frequency of the gene Gc^2 which decreases from Europe to equa-

torial Africa. This gradient may be superposed on that of skin pigmentation (Loomis, 1967) which has always been considered to be linked to genetic and adaptative conditions(Hiernaux, 1977).

Table 5.2.4. I Group specific component (Gc) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		Gc 1-1	Gc 2-1	Gc 2-2	Gc^1	Gc^2		
1-Kurdish Jews	42	27	14	1	80.95	19.05	Cleve,H.,et al.	1962
2- Shiraz	94	55	34	5	76.60	23.40	Walter,H.,& Djahan-shahi,D.	1963
3- Jews	49	30	14	5	75.51	24.49	Cleve,H.,et al.	1962
4- Jews	149	91	43	15	75.50	24.50	Kitchin,F.D., & Bearn,A.G.	1964
5-West	308	135	148	25	67.80	32.20	Bajatzadeh,M., & Walter, H.	1969
6-East	178	68	84	26	66.80	33.20	Bajatzadeh,M.,& Walter, H.	1969
7-Central and South	238	99	115	24	65.70	34.30	Bajatzadeh,M.,& Walter, H.	1969
8-North	177	68	95	14	65.20	34.80	Bajatzadeh,M., & Walter,H.	1969
9-Shahsavar	108	44	52	12	64.80	35.20	Farhud,D.D., et al.	1978

Table 5.2.4. I(Cont.) Group specific component (Gc) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		Gc 1-1	Gc 2-1	Gc 2-2	Gc^1	Gc^2		
10-North west	245	106	106	33	64.60	35.40	Bajatzadeh,M., & Walter, H.	1969
11-Tehran	385	144	190	51	62.10	37.90	Bajatzadeh,M., & Walter, H.	1969

Table 5.2.4. III Group specific component (Gc) phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		Gc 1-1	Gc 2-1	Gc 2-2	Gc^1	Gc^2		
1-Turks	274	148	111	15	74.27	25.73	Hummel,K.,et al.	1970

Table 5.2.4. IV Group specific component (Gc) phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		Gc 1-1	Gc 2-1	Gc 2-2	Gc^1	Gc^2	
1-Kurds	58	40	16	2	82.80	17.20	Constans,G.,et al. 1980
2-Jews	85	48	33	4	75.88	24.12	Cleve,H.,et al. 1962

Table 5.2.4.b. IV Group specific Component(Gc) subtypes in Iraq

Population	Number Tested	Phenotypes								
		Gc 1F-1F	Gc 1F-1s	Gc 1s-1s	Gc 1A1-1F	Gc 1A1-1s	Gc 2-1F	Gc 2-1s	Gc 1A1-2	Gc 2-2
Kurds	58	2	14	24	0	0	8	7	1	2

Table 5.2.4.b. IV (Cont.) Group specific component (Gc) subtypes in Iraq

Population	Number Tested	Gene frequencies				Authors
		Gc^{1F}	Gc^{1s}	Gc^{1A1}	Gc^{2}	
Kurds	58	22.40	59.50	0.80	17.20	Constans,G., et al. 1980

Table 5.2.4. VI Group specific component (Gc) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		Gc 1-1	Gc 2-1	Gc 2-2	Gc^1	Gc^2	
1-Saudi Arabians	352	256	89	7	85.40	14.60	Goedde,H.W.,et al. 1979

Table 5.2.4. VIII Group specific component (Gc) phenotypes and gene frequencies distribution in Pakistan

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		Gc 1-1	Gc 2-1	Gc 2-2	Gc^1	Gc^2		
1-Peshawar	135	86	45	4	80.37	19.63	Walter, H.	1966
2-Pathans.Peshawar	60	36	20	4	76.67	23.33	Kirk,R.L.,et al.	1963
3-Punjabis.Lahore	90	47	33	10	70.56	29.44	Kirk,R.L.,et al.	1963
4-Pathanis.Swat	61	32	19	10	68.04	31.96	Spedini,Gabriella.	1969

Table 5.2.4. IX Group specific component (Gc) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		Gc 1-1	Gc 2-1	Gc 2-2	Gc^1	Gc^2		
1-Hazaras	172	107	52	13	77.32	22.68	Rahimi,A.G.,et al.	1977
2-Pushtus	210	128	67	15	76.90	23.10	Rahimi,A.G.,et al.	1977
3-Uzbeks	124	76	38	10	76.61	23.39	Rahimi,A.G.,et al.	1977
4-Tajiks	310	183	102	25	75.48	24.52	Rahimi,A.G.,et al.	1977
5-Hazara,Tarjek,Timuri. Kabul	141	72	54	15	70.21	29.79	Kitchin,F.D., & Bearn,A.G.	1964

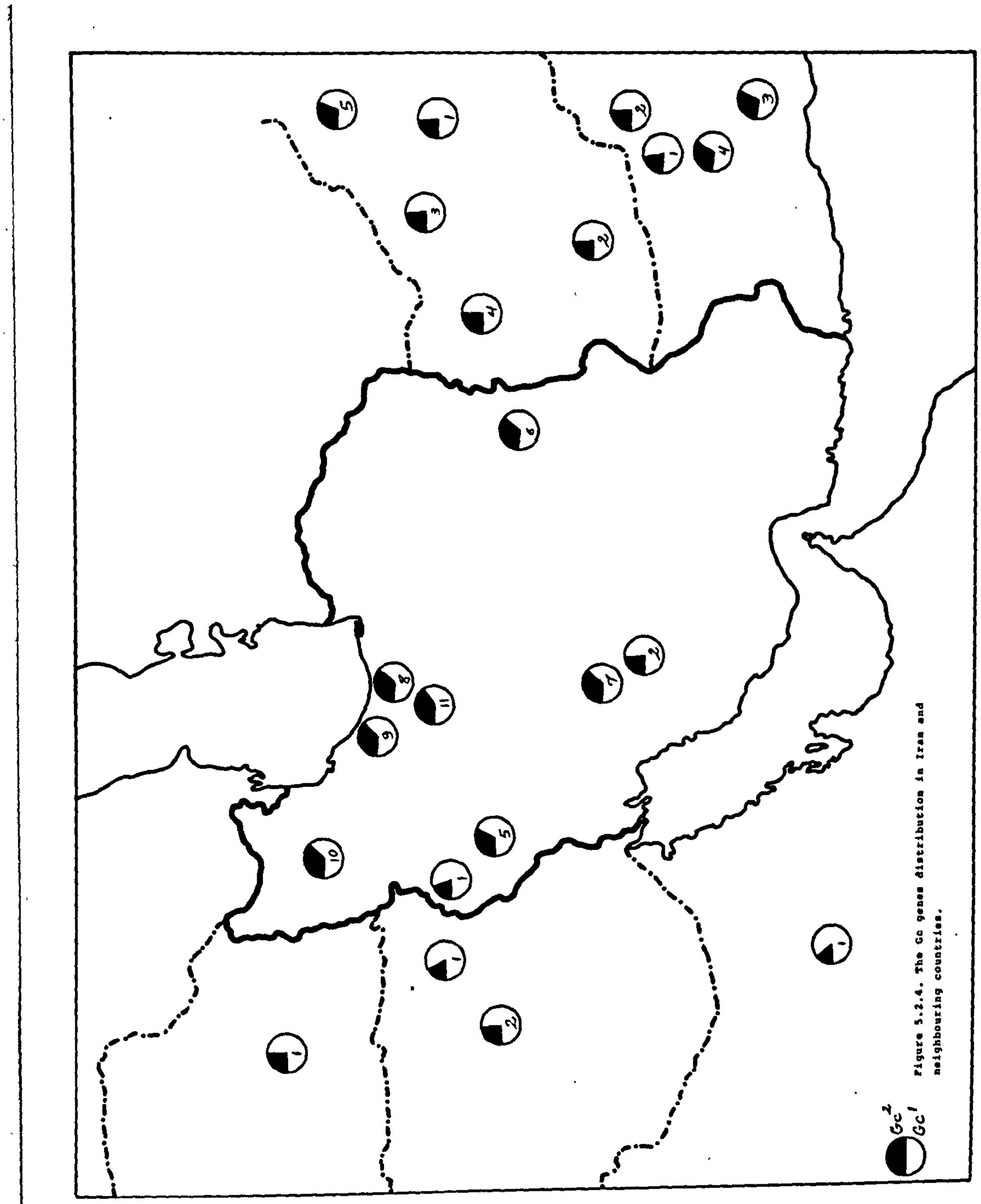


Figure 5.2.4. The Gc genes distribution in Iran and neighbouring countries.

5.2.5. The Caeruloplasmin (CP) system

The distribution of Caeruloplasmin groups and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.2.5.

There are few reports of Caeruloplasmin studies in Iranian and neighbouring populations but the results obtained confirm essentially the data given by Shreffler et al (1967/1968), who found very high frequencies of the common CP^B (98-99 percent) and very low frequencies of the CP^A and CP^C alleles in Caucasoids.

Conclusion

The allele CP^B accounts for about 99 percent of the Caeruloplasmin genes present in European populations and about 94 percent of those in American Negroes. The latter have 5 percent of the CP^A gene.

The other genes have very low frequencies in all the relatively few populations so far examined (Mourant et al,1976).

The Iranian and neighbouring populations, like other Caucasoids, seem also to be characterized by very high CP^B and low CP^A and CP^C frequencies.

Table 5.2.5. I Caeruloplasmin (CP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes						Gene frequencies			Authors	
		CP A	CP AB	CP B	CP AC	CP BC	CP C	CP^A	CP^B	CP^C		
1-Caspian Sea area	99	-	-	99	-	-	-	0.00	100.00	0.00	Farhud,D.D.,et al.	1978
2-Caspian Sea area	448	-	2	442	-	4	-	0.23	99.33	0.44	Kirk,R.L., et al.	1977
3-Iranians	198	-	1	195	-	1	1	0.25	98.99	0.76	Bajatzadeh,M.,& Walter, H.	1969
4-Shahsavar	134	-	5	127	-	2	-	1.80	97.40	0.80	Farhud,D.D.,et al.	1978

Table 5.2.5. VI Caeruloplasmin (CP) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes						Gene frequencies			Authors
		CP A	CP AB	CP B	CP AC	CP BC	CP C	CP^A	CP^B	CP^C	
I-Western Saudi Arabia	185	-	-	185	-	-	-	0.00	100.00	0.00	Saha,N.,et al. 1980

Table 5.2.5. VIII Caeruloplasmin(CP) phenotypes and gene frequencies distribution in Pakistan

Population	Number Tested	Phenotypes						Gene frequencies			Authors	
		CP A	CP AB	CP B	CP AC	CP BC	CP C	CP^A	CP^B	CP^C		
1-Pakistanis	96	-	1	95	-	-	-	0.52	99.48	0.00	Bajatzadeh,M.,& Walter, H.	1969

Table 5.2.5. IX Caeruloplasmin (CP) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes						Gene frequencies			Authors	
		CP A	CP AB	CP B	CP AC	CP BC	CP C	CP^A	CP^B	CP^C		
1-Tajiks	310	-	-	310	-	-	-	0.00	100.00	0.00	Rahimi,A.G.,et al.	1977
2-Uzbeks	124	-	-	124	-	-	-	0.00	100.00	0.00	Rahimi,A.G.,et al.	1977
3-Pushtus	210	-	-	210	-	-	-	0.00	100.00	0.00	Rahimi,A.G.,et al.	1977
4-Hazaras	172	-	-	172	-	-	-	0.00	100.00	0.00	Rahimi,A.G.,et al.	1977

5.2.6. The ∝ 1-antitrypsin (Pi) system

The distribution of ∝ 1-antitrypsin types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.2.6.

There are few reports of ∝ 1-antitrypsin studies in Iranian and neighbouring populations.

The Iranian sample consists of three population groups. Comparing the three groups, the lowest frequency of the commonest Pi^M gene is 88.38 percent in the Mixed Iranian sample of Kellermann and Walter(1970) and the highest, 99.64 percent in the Caspian sea area(Kirk et al, 1977).

The mixed Iranian sample of Kellermann and Walter(1970) and the Tehrani series of Daneshmand (1979)show the presence of the Pi^F and Pi^S genes with polymorphic frequencies but in the Caspian sea sample of Kirk et al (1977) the Pi^F and Pi^S genes show very low frequencies.

The Pi^Z gene is only present with a frequency of 2.21 percent in the mixed Iranian sample of Kellermann and Walter (1970)and totally absent in other Iranian samples.

On the whole, as in Europeans, the Pi^F gene seems to be more frequent than the Pi^S in Iranians.

Regarding neighbouring populations, the only report from Saudi Arabia is that of Goedde et al (1979). The most common Pi^M gene has a frequency of 98.30 percent in Saudi Arabians. Unlike that in Iranians, the Pi^S gene seems to be more frequent than the Pi^F in Saudi Arabians. The Pi^Z gene is present with a frequency of 0.70 percent which is much lower than that of 2.21 percent in the mixed Iranian sample of Kellermann and Walter (1970). On the whole the Pi variants other than Pi^M seem to be less frequent in Saudi Arabians than in Iranians.

The only report on the population of Pakistan(Kellermann and Walter, 1970) shows a Pi^M gene frequency of 95.28 percent. This is an intermediate frequency compared with those found in Iranians. Like that in Iranians, the Pi^F gene with a frequency of 3.78 percent appears to be the most common Pi variant other than Pi^M in the Pakistani population. The Pi^S is totally absent and the Pi^Z is present with a low frequency of 0.94 percent.

The afghan sample consists of four population groups(Rahimi et al, 1977). The frequency of the Pi^M gene ranges from 95.36 percent in the Hazaras to 99.59 percent in the Uzbeks. The Pi^S gene seems to be more frequent than the Pi^F gene in Afghanistan and appears to be the most common Pi variant other than Pi^M. The third common variant seems to be Pi^Z. This contrast with the findings in Iranian and other neighbouring populations.

Conclusion

The commonest Pi allele, Pi^M has a frequency of at least 85 percent in all populations tested, and over 97 percent in Negroes and Mongoloids examined, as well as in some Indian populations.

The commonest variant, Pi^F reaches a level of 11 percent in Ireland, only 1.3 percent in Norway, 7 percent in Germany and Hungry, but is rare or absent in most European populations other than those mentioned (Mourant et al, 1976).

In Iranian and neighbouring populations, the allele Pi^M has a frequency of at least 88.38 percent. The commonest variant, Pi^F reaches a level of 6.83 percent in Iranians and 3.78 percent in Pakistanis but is rare or absent in Afghan and Saudi Arabian populations as it is in most Europeans.

The other Pi variants are absent or very rare.

In view of the known pathological association of the deficient variant (Pi^Z) with pulmonary emphysema (Laurell and Eriksson, 1963) and Cirrhosis(Sharp, 1970), and the marked variations in frequency of the electrophoretically detectable alleles, this system merits further application both anthropologically and in disease surveys.

Table 5.2.6. I ∝1-antitrypsin(Pi) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes								
		Pi MM	Pi MS	Pi MF	Pi MZ	Pi MI	Pi SS	Pi SF	Pi FF	Pi ZZ
1-Iranians	271	218	7	28	6	2	1	3	3	3
2-Tehran	143	122	8	11	-	-	1	1	-	-
3-Caspian Sea area	412	409	2	1	-	-	-	-	-	-

Table 5.2.6. I (Cont.) ∝ 1-antitrypsin (Pi) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Gene frequencies					Authors	
		Pi^M	Pi^S	Pi^F	Pi^Z	Pi^i		
1-Iranians	271	88.38	2.21	6.83	2.21	0.37	Kellermann,G., & Walter, H.	1970
2-Tehran	143	91.95	3.85	4.20	0.00	0.00	Daneshmand,P.	1979
3-Caspian Sea area	412	99.64	0.24	0.12	0.00	0.00	Kirk,R.L.,et al.	1977

Table 5.2.6. VI ∝ 1- antitrypsin (Pi) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes								
		Pi MM	Pi MS	Pi MF	Pi MZ	Pi MI	Pi SS	Pi SF	Pi FF	Pi ZZ
1-Saudi Arabians	357	345	7	-	5	-	-	-	-	-

Table 5.2.6. VI (Cont.) ∝1- antitrypsin (Pi) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Gene frequencies					Authors
		Pi^M	Pi^S	Pi^F	Pi^Z	Pi^i	
1-Saudi Arabians	357	98.30	1.00	0.00	0.70	0.00	Goedde,H.W.,et al. 1979

Table 5.2.6.b. VI Alpha$_1$-antitrypsin(Pi) subtypes in Saudi Arabia

Population	Number Tested	Phenotypes								
		Pi M_1M_1	Pi M_1M_3	Pi M_3M_3	Pi M_1S	Pi M_3S	Pi SS	Pi M_1Z	Pi M_3Z	Pi ZZ
Saudi Arabians	178	112	52	10	1	1	-	1	1	-

Table 5.2.6.b. VI (Cont.) Alpha$_1$-antitrypsin (Pi) subtypes in Saudi Arabia

Population	Number Tested	Gene frequencies				Authors
		M_1	M_3	S	Z	
Saudi Arabians	178	78.00	21.00	0.50	0.50	Goedde,H.W.,et al. 1980

Table 5.2.6. VIII ∝ 1-antitrypsin(Pi) phenotypes and gene frequencies distribution in Pakistan

Population	Number Tested	Phenotypes								
		Pi MM	Pi MS	Pi MF	Pi MZ	Pi MI	Pi SS	Pi SF	Pi FF	Pi ZZ
1- Pakistanis	53	49	-	2	1	-	-	-	1	-

Table 5.2.6. VIII (Cont.) ∝1- antitrypsin (Pi) phenotypes and gene frequencies distribution in Pakistan

Population	Number Tested	Gene frequencies					Authors
		Pi^M	Pi^S	Pi^F	Pi^Z	Pi^i	
1-Pakistanis	53	95.28	0.00	3.78	0.94	0.00	Kellermann,G., & Walter, H. 1970

Table 5.2.6. IX α 1- antitrypsin(Pi) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes								
		Pi MM	Pi MS	Pi MF	Pi MZ	Pi MI	Pi SS	Pi SF	Pi FF	Pi ZZ
1-Hazaras	172	156	10	3	3	-	-	-	-	-
2-Pushtus	210	196	4	1	8	-	1	-	-	-
3-Tajiks	310	291	4	1	14	-	-	-	-	-
4-Uzbeks	124	123	1	-	-	-	-	-	-	-

Table 5.2.6. IX (Cont.) ∝1-antitrypsin (Pi) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Gene frequencies					Authors	
		Pi^M	Pi^S	Pi^F	Pi^Z	Pi^i		
1-Hazaras	172	95.36	2.90	0.87	0.87	0.00	Rahimi,A.G.,et al.	1977
2-Pushtus	210	96.44	1.42	0.23	1.91	0.00	Rahimi,A.G.,et al.	1977
3-Tajiks	310	96.94	0.64	0.16	2.26	0.00	Rahimi,A.G.,et al.	1977
4-Uzbeks	124	99.59	0.41	0.00	0.00	0.00	Rahimi,A.G.,et al.	1977

Table 5.2.6.b. IX Alpha$_1$-antitrypsin (Pi) subtypes in Afghanistan

Population	Number Tested	Phenotypes								
		Pi M_1M_1	Pi M_1M_3	Pi M_3M_3	Pi M_1S	Pi M_3S	Pi SS	Pi M_1Z	Pi M_3Z	Pi ZZ
1-Hazaras	135	90	40	5	-	-	-	-	-	-
2-Pushtus	167	118	39	8	1	-	-	1	-	-
3-Tajiks	232	171	50	9	1	-	-	1	-	-
4-Uzbeks	153	127	26	-	-	-	-	-	-	-

Table 5.2.6.b. IX (Cont.) Alpha$_1$-antitrypsin(Pi) subtypes in Afghanistan

Population	Number Tested	Gene frequencies				Authors	
		M_1	M_3	S	Z		
1-Hazaras	135	81.50	18.50	0.00	0.00	Goedde,H.W.,et al.	1980
2-Pushtus	167	82.90	16.50	0.30	0.30	Goedde,H.W.,et al.	1980
3-Tajiks	232	84.90	14.70	0.20	0.20	Goedde,H.W.,et al.	1980
4-Uzbeks	153	91.50	8.50	0.00	0.00	Goedde,H.W.,et al.	1980

5.2.7. The Pseudocholinesterase(E_1 locus) system

The distribution of Pseudocholinesterase types and respective gene frequencies in Iranian and neighbouring populations is shown in Tables 5.2.7.

The frequency of the E_1^a gene ranges from zero to 5.12 percent in Iranians, being lowest in the Kurds of Sanandaj (Lehmann et al, 1973) and highest in the Jews(Szeinberg et al, 1966). Apart from the Jews with their exceptionally high E_1^a frequency of 5.12 percent, the Iranian population appears to exhibit E_1^a frequencies similar to those varying between 1 and 2 percent in Europeans (Mourant et al, 1976).

Regarding neighbouring populations, the E_1^a gene frequencies in the populations of Turkey varying between 1.64 percent in the Jews (Szeinberg et al, 1966) and 3.31 percent in the Turks of Ankara (Sayek et al, 1967) seem to be slightly higher than those found in Europeans.

Frequencies of the E_1^a gene in the populations of Pakistan ranging from 1.24 percent in Peshawar (Neumann and Walter, 1968) to 3.20 percent in the Punjabis (Singh et al, 1971) appear also to be slightly higher than in Europeans.

The E_1^a gene frequencies in the populations of Afghanistan varying between zero in both the Uzbeks and Pushtus and 0.59 percent in the Hazaras (Rahimi et al, 1977) seem to be below the European level.

Conclusion

In nearly all European populations the frequency of the E_1^a gene lies between 1 and 2 percent, but somewhat higher frequencies around 3 percent are found in Czechoslovakia and Greece

Jews in general show similar frequencies to Europeans, but those of the Yemen have 3.6 percent.

In the Indian region the frequency of E_1^a is about 1.5 percent, but in Africa and the Far East is only a few per mille.

The frequency of E_1^f, where ascertained, is usually about 0.5 percent in Europe (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Irani Jews with their exceptionally high E_1^a frequency (5.12), frequencies of this gene seem to be similar to those in Europeans. The Turks of Turkey with somewhat higher E_1^a frequency (around 3 percent) appear to be more similar to the population of Greece.

Table 5.2.7.I. Pseudocholinesterase E_1 phenotypes and gene frequencies distribution in Iran

Populations	Number Tested	Phenotypes			Gene frequencies		Authors	
		U	I	A	E_1^u	E_1^a		
1-Kurds.Sanandaj	107	107	0	0	100.00	0.00	Lehmann,H., et al.	1973
2- Kurds.Baneh Marivan	77	77	0	0	100.00	0.00	Lehmann,H., et al.	1973
3- Arabs	36	35	1	0	98.61	1.39	Whittaker, M.	1968
4- Jews	381	343	37	1	94.88	5.12	Szeinberg,A.,et al.	1966

Table 5.2.7. III Pseudocholinesterase E_1 phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		U	I	A	UF	IF	S	E_1^u	E_1^a	E_1^f	F_1^s	
1- Jews	214	207	7	0	0	0	0	98.36	1.64	0.00	0.00	Szeinberg,A., et al. 1966
2- Turks.Ankara	725	675	43	1	3	3	0	96.28	3.31	0.41	0.00	Sayek,I., et al. 1967

Table 5.2.7. VIII Pseudocholinesterase E_1 phenotypes and gene frequencies distribution in Pakistan

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		U	I	A	E_1^u	E_1^a	
1-Peshawar	121	118	3	0	98.76	1.24	Neumann,S., and Walter,H. 1968
2-Punjabis	202				96.80	3.20	Singh,S.,et al. 1971

Table 5.2.7. IX Pseudocholinesterase E_1 phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		U	UA	A	E_1^u	E_1^a		
1-Pushtus	210	210	0	0	100.00	0.00	Rahimi,A.G., et al.	1977
2-Uzbeks	124	124	0	0	100.00	0.00	Rahimi,A.G., et al.	1977
3-Tajiks	310	308	2	0	99.67	0.33	Rahimi,A.G., et al.	1977
4-Hazaras	172	170	2	0	99.41	0.59	Rahimi,A.G., et al.	1977

5.2.8. Other serum protein systems

In this part, data on some other serum protein systems are presented. Since data available on these systems are not representative for the region, no discussion will be given.

Table 5.2.8. a.I. Albumin types in Iran

Population	Number Tested	Common type	Variant	Authors	
Caspian Sea area	448	448	-	Kirk,R.L., et al.	1977

Table 5.2.8.a. VI . Albumin types in Saudi Arabia

Population	Number Tested	Common type	Variant	Authors	
Western Saudi Arabia	191	191	-	Saha,N., et al.	1980

Table 5.2.8.b. VI Properdin factor B(Bf) types in Saudi Arabia

Population	Number Tested	Phenotypes							
		Bf SS	Bf FS	Bf FF	Bf SS_1	Bf FS_1	Bf F_1S	Bf FF_1	Bf F_1S_1
Saudi Arabians	246	68	78	62	13	15	6	3	1

Population	Number Tested	Gene frequencies				Authors
		Bf^S	Bf^F	Bf^F1	Bf^S1	
Saudi Arabians	246	47.40	44.70	2.00	5.90	Goedde, H.W., et al. 1979

Table 5.2.8. C.I. Betalipoprotein (Ag) types in Iran:tested with anti-Ag^X

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Ag(x+)	Ag(x-)	Ag^x	Ag^y	
Kurdish Jews	19	12	7			Tills,D.,et al. 1977

Table 5.2.8. C. IV Betalipoprotein (Ag) types in Iraq: tested with anti-Ag^X

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Ag(x+)	Ag(X-)	Ag^x	Ag^y	
Kurdish Jews North west	41	21	20	30.16	69.48	Tills, D., et al. 1977

Table 5.2.8.d.I. Betalipoprotein (Ag) types in Iran: tested with C. de B serum

Population	Number Tested	Ag(+)	Ag(-)	Ag(+)%	Authors	
Jews	37	26	11	70.27	Blumberg, B.S.	1963
Kurdish Jews	17	12	5	70.59	Blumberg, B.S.	1963

Table 5.2.8.e. I. Betalipoprotein (LP) types in Iran

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		LP(a+)	LP(a-)	LP^a	LP	
Kurdish Jews	19	12	7			Tills,D., et al. 1977

Table 5.2.8.e.IV Betalipoprotein (LP) types in Iraq

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		LP(a+)	LP(a-)	LP^a	LP		
1-Kurdish Jews. North west	41	14	27	18.85	81.15	Tills, D.,et al.	1977
2-Kurdish Jews. South west	41	20	21	28.43	71.57	Tills, D.,et al.	1977

Table 5.2.8.e. VIII. Betalipoprotein (LP) types in Pakistan

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		LP(a+)	LP(a-)	LP^a	LP		
Pakistanis	125	40	85	17.54	82.46	Wassenich,W.K., & Walter, H.	1968

Table 5.2.8.f.IX Beta$_2$ - Glycoprotein (Bg) types in Afghanistan

Population	Number Tested	Phenotypes			Gene frequencis		Authors	
		Bg NN	Bg DN	Bg DD	N Bg	D Bg		
1-Hazaras	172	112	60	-	82.55	17.45	Rahimi,A.G.,et al.	1977
2-Pushtus	210	160	48	2	87.61	12.39	Rahimi,A.G.,et al.	1977
3-Tajiks	310	242	62	6	88.06	11.94	Rahimi,A.G.,et al.	1977
4-Uzbeks	124	97	26	1	88.70	11.30	Rahimi,A.G.,et al.	1977

Table 5.2.8.g.IV. Pseudocholinesterase E_2 types in Iraq

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		C_5+	C_5-	E_2^+	E_2^-		
Jews	64	-	64	0.00	100.00	Robson,E.B.,& Harris,H.	1966

5.3. Red cell enzymes

5.3.1. The acid Phosphatase (AcP) system

The distribution of acid phosphatase types and respective allele frequencies in Iranian and neighbouring populations is shown in Tables 5.3.1.

The frequency of the AcP^A gene ranges from 21.01 to45.50 percent in Iranians, being lowest in the Zoroastrians (present study) and highest in northern Gorgan (Kirk et al, 1977), that of the AcP^B gene varies between 53.40 percent in northern Gorgan(Kirk et al, 1977) and 77.73 percent in the Zoroastrians (present investigation). The rarest AcP^C allele is present in most Iranian populations studied with frequencies ranging from 0.45 percent in the Baluchis of Sistan and Baluchistan (present study) to 4.26 percent in the Kurdish Jews (Godber et al, 1973).

Both the Turkish and the Kurdish series of Rezaieh(present investigation) and also two population groups from the Caspian sea area (Kirk et al, 1977) show an absence of this gene.With the exception of the Zoroastrians and the Kurds of Shirvan, Khorasan values obtained in the present investigation are within the range of variation.

On the whole, with an average AcP^A frequency of 31.97 percent, an AcP^B frequency of 66.18 percent and an AcP^Cfrequency of 1.85 percent, the Iranian population appears to exhibit a higher AcP^B but a lower AcP^C frequency than the average frequencies of about AcP^A 32, AcP^B 61 and AcP^C 6 percent found in Europeans (Mourant et al,1976). From the table it seems that the frequencies of the alleles $AcP^A$and AcP^C decrease but that of the AcP^B allele increases from west to east in Iran.

The Kurdish Jews, like the Jews themselves, both in Iran and

in Iraq appear to exhibit higher AcP^A and AcP^C but lower AcP^B frequencies than the regional averages.

The Iranian Zoroastrians seem to be characterized by lower AcP^A but higher AcP^B frequencies than the averages for the region.

Relatively higher AcP^A but lower AcP^B and AcP^C frequencies than the regional averages appear to be characteristic of the populations of the Caspian sea area of Iran.

Regarding neighbouring groups, the frequency of the AcP^A gene in the populations of Turkey ranges from 29.20 percent in the Turks (Hummel et al, 1970) to 35.00 percent in the Kurds (Richard, 1976), that of the AcP^B allele varies between 62.00 percent in the Kurdish sample of Richard (1976) and 68.06 percent in the Turkish series of Hummel et al (1970). The rarest AcP^C allele is present in both samples with frequencies ranging from 2.74 percent in the Turks to 3.00 percent in the Kurds. With an average AcP^A frequency of 32.10 percent, an AcP^B frequency of 65.03 percent and an AcP^C frequency of 2.87 percent, the population of Turkey, like that of Iran, seems to show a higher AcP^B but a lower AcP^C frequency than those found in Europeans.

The frequency of the AcP^A gene in the populations of Iraq ranges from zero in the Karaite Jews (Goldschmidt et al, 1976) to 40.98 percent in the Kurdish Jews of north western Iraq(Tills et al, 1977), that of the AcP^B allele varies between 54.92 percent in the north western Kurdish Jews series of Tills et al (1977) and 96.50 percent in the Karaite Jews sample of Goldschmidt et al (1976). The frequency of the rarest AcP^C gene ranges from zero in the Kurdish Jews of south eastern Iraq(Tills et al, 1977) to 7.41 percent in the Kurdish Jews series of

Godber et al (1973). With an average AcP^A frequency of 28.66 percent, an AcP^B frequency of 67.97 percent and an AcP^C frequency of 3.37 percent, the Jewish population of Iraq, though with slightly lower AcP^A but higher AcP^B and AcP^C frequencies than those found in Iranians, again appears to exhibit higher AcP^B but lower AcP^C frequencies than those found in Europeans. The complete absence of the allele AcP^A and a correspondingly high frequency of AcP^B (96.50 percent) in the Karaite Jews of Iraq clearly indicate the strong influence of isolation and genetic drift operating in this community.

The frequency of the AcP^A gene in the populations of Kuwait ranges from 16.00 percent in the Suluba tribe (Khaled et al, 1981) to 20.32 percent in the Kuwaiti Arabs (Sawhney,1975), that of the AcP^B allele varies between 77.74 percent in the Kuwaiti Arabs sample of Sawhney (1975) and 84.00 percent in the Suluba tribe series of Khaled et al (1981). The rarest AcP^C gene is present only in the Kuwaiti Arabs series of Sawhney (1975) with a frequency of 1.94 percent, the other Kuwaiti samples investigated showing complete absence of this gene. With an average AcP^A frequency of 18.58 percent, an AcP^B frequency of 80.94 percent and an AcP^C frequency of 0.48 percent, the population of Kuwait seems to show much lower AcP^A and AcP^C but higher AcP^B frequencies than those found in Iranians.

The frequency of the AcP^A gene in the populations of Saudi Arabia ranges from 9.79 percent in western Saudi Arabia(Saha et al, 1980) to 32.30 percent in Saudi Arabians (Goedde et al, 1979), that of the AcP^B allele varies between 66.70 percent in the Saudi Arabians sample of Goedde et al (1979) and 84.62 percent in western Saudi Arabian series of Saha et al (1980).The rarest AcP^C gene is present in both the

Saudi Arabian samples with frequencies varying between 1.00 percent in the Saudi Arabians sample of Goedde et al (1979) and 5.59 percent in the western Saudi Arabian series of Saha et al (1980). With an average AcP^A frequency of 21.05 percent, an AcP^B frequency of 75.66 percent and an AcP^C frequency of 3.29 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit much lower AcP^A but higher AcP^B frequencies than those found in Iranians. The frequency of the AcP^C allele seems to be higher in Saudi Arabians than in Kuwaiti Arabs and also in Iranians but, is still lower than the European frequency.

The frequency of the AcP^A gene in the populations of Afghanistan ranges from 26.82 percent in the Pushtus to 37.09 percent in the Uzbeks (Goedde et al, 1977), that of the AcP^B gene varies between 62.50 percent in the Uzbeks and 72.46 percent in the Pushtus (Goedde et al, 1977). The rarest AcP^C allele is present in all the Afghan samples with frequencies ranging from 0.28 percent in Hazaras (Goedde et al,1977)to 2.80 percent in the Daris (Papiha et al, 1977). With an average AcP^A frequency of 30.18 percent, an AcP^B frequency of 68.90 percent and an AcP^C frequency of 0.92 percent, the population of Afghanistan with higher AcP^B and lower AcP^C frequencies than in Iranians, also seems to show higher AcP^B but lower AcP^C values than those found in Europeans.

Conclusion

The average frequencies of the three AcP alleles in European populations are about 32, 61, and 6 percent respectively, with the frequency of AcP^A falling and that of AcP^B rising from north west to south east.

Negroes have about 20, 79 and 1 percent respectively of these

three alleles.

Asiatic populations vary considerably, they exhibit an AcP^A average of about 30 percent and an AcP^B of 70 percent,with very low frequencies of AcP^C(Mourant et al, 1976).

Iranian and neighbouring populations, like other Asiatic populations, seem to be characterized by lower AcP^A and AcP^C but higher AcP^B frequencies than those found in Europeans. The frequencies of the three AcP alleles in the Arab populations of Kuwait and Saudi Arabia, with much lower AcP^A but higher AcP^B frequencies, appear to be more like those found in Africans.

The high incidence of the allele AcP^B in Iranian and neighbouring populations agrees with the suggestion of a possible correlation of high AcP^B frequency and increasing mean annual temperature (Walter and Bajatzadeh, 1968; Wysloúchowa, 1970; Ananthakrishnan and Walter, 1972).

Table 5.3.1.I Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes						Gene frequencies			Authors	
		AcP A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	AcP^A	AcP^B	AcP^C		
1-Zoroastrians	119	4	42	70	-	3	-	21.01	77.73	1.26	Present study.	
2-Kurds.Shirvan,Khorasan	101	8	28	63	-	2	-	21.78	77.23	0.99	Present study.	
3-Armenians	180							24.70	72.10	3.20	Simhai,B.	1978
4-North west	59	5	18	32	2	2	-	25.50	71.10	3.40	Walter,H., & Bajatzadeh, M.	1968
5-Lurs.Luristan	178	16	61	94	2	5	-	26.69	71.35	1.96	Present study.	
6-Turks.Shirvan,Khorasan	115	11	40	62	-	2	-	26.96	72.17	0.87	Present study.	
7-Tavalesh,Astara	61	5	23	32	-	1	-	27.10	72.10	0.80	Kirk,R.L.,et al.	1977
8-Esfahan	77	6	30	39	-	2	-	27.27	71.43	1.30	Sawhney, K.S.	1975
9-North	54	6	17	27	2	2	-	28.80	67.40	3.80	Walter,H., & Bajatzadeh,M.	1968
10-Turks . Rezaieh	125	12	48	65	-	-	-	28.80	71.20	0.00	Present study.	
11-Babol,Shahi,Amol	64	3	31	30	-	-	-	28.90	71.10	0.00	Kirk,R.L.,et al.	1977
12-West	95	16	23	51	2	3	-	30.00	67.40	2.60	Walter,H.,& Bajatzadeh, M.	1968

Table 5.3.1.I (Cont) Red cell Acid phosphatase(AcP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes						Gene frequencies			Authors	
		Acp A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	AcP^A	AcP^B	AcP^C		
13-East	58	7	20	28	1	2	-	30.30	67.20	2.50	Walter,H., & Bajatzadeh, M.	1968
14-Central and South	92	11	32	43	3	3	-	30.90	65.70	3.40	Walter,H., & Bajatzadeh,M.	1968
15-Kurds.Sanandaj	105	13	41	48	-	3	-	31.90	66.67	1.43	Lehmann,H.,et al.	1973
16-Kerman	307	31	133	134	4	5	-	32.41	66.12	1.47	Present study.	
17-Tehran	91	16	27	42	3	3	-	34.10	62.70	3.20	Walter,H., & Bajatzadeh,M.	1968
18-Tehran	161	21	68	65	-	7	-	34.16	63.67	2.17	Sawhney,K.S.	1975
19-Jews	164							34.20	63.00	2.80	Simhai,B.	1974
20-Kurds.Baneh,Marivan	77	8	35	32	2	-	-	34.41	64.29	1.30	Lehmann,H.,et al.	1973
21-Kurdish Jews	94	11	41	34	2	6	-	34.57	61.17	4.26	Godber,Marilyn.,et al..	1973
22-Tehran	352	49	145	146	1	11	-	34.66	63.64	1.70	Present study.	
23-Kurdish Jews	131	19	53	55	1	3	-	35.11	63.36	1.53	Goldschmidt,Elizabeth.,et al.	1967
24-Zabolis.Sistan & Baluchistan	117	19	44	51	1	2	-	35.47	63.25	1.28	Present study.	

Table 5.3.1.I (Cont.) Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes						Gene frequencies			Authors
		AcP A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	AcP^A	AcP^B	AcP^C	
25-Shahsavar,Rudsar, Rudbar,Rasht,Langarud,Lahijan,Bandar-Pahlavi	86	8	44	30	1	3	-	35.50	62.20	2.30	Kirk,R.L.,et al. 1977
26-Baluchis.Sistan & Baluchistan	111	17	47	46	-	1	-	36.49	63.06	0.45	Present study.
27-Iranians	49	10	16	21	-	2	-	36.74	61.22	2.04	Goldschmidt,Elizabeth.,et al. 1967
28-Kurds.Rezaieh	146	17	74	55	-	-	-	36.99	63.01	0.00	Present study.
29-Gonbad	155	24	65	59	2	5	-	37.10	60.60	2.30	Kirk,R.L.,et al. 1977
30-Kurdish Jews	106	18	41	39	2	6	-	37.27	58.96	3.77	Tills,D.,et al. 1977
31-Southern Gorgan Behshahr, Sari	53	7	26	20	-	-	-	37.70	62.30	0.00	Kirk,R.L.,et al. 1977
32-Northern Gorgan	44	8	24	11	-	1	-	45.50	53.40	1.10	Kirk,R.L.,et al. 1977

Table 5.3.1. III Red cell Acid Phosphatase (AcP) phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes						Gene frequencies			Authors
		AcP A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	AcP^A	AcP^B	AcP^C	
1-Turks	274	23	109	127	5	10	-	29.20	68.06	.2.74	Hummel,K.,et al. 1970
2-Kurds								35.00	62.00	'3.00	Richard,P. 1976

Table 5.3.1. IV Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes						Gene frequencies			Authors
		AcP A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	AcP^A	AcP^B	AcP^C	
1-Karaite Jews	72	-	-	67	-	5	-	0.00	96.50	3.50	Goldschmidt, Elizabeth.,et al. 1976
2-Kurdish Jews.South east	50	6	21	23	-	-	-	33.00	67.00	0.00	Tills,D.,et al. 1977
3-Jews	82	11	33	35	1	2	-	34.15	64.02	1.83	Goldschmidt, Elizabeth.,et al. 1967
4-Kurdish Jews	27	3	13	7	-	4	-	35.18	57.41	7.41	Godber,Marilyn.J., et al. 1973
5-Kurdish Jews. North west	61	10	30	16	-	5	-	40.98	54.92	4.10	Tills,D.,et al. 1977

Table 5.3.1. V Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Kuwait

Population	Number Tested	Phenotypes						Gene frequencies			Authors
		AcP A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	AcP^A	AcP^B	AcP^C	
1-Suluba tribe	51	2	12	37	-	-	-	16.00	84.00	0.00	Khaled,E.,et al. 1981
2-Ajman tribe	50	5	9	36	-	-	-	19.00	81.00	0.00	Khaled,E.,et al. 1981
3-General population	86	3	26	57	-	-	-	19.00	81.00	0.00	Khaled,E.,et al. 1981
4-Kuwaiti Arabs	155	8	46	95	1	5	-	20.32	77.74	1.94	Sawhney,K.S. 1975

Table 5.3.1. VI Red cell Acid phosphatase(AcP) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes						Gene frequencies			Authors
		AcP A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	AcP^A	AcP^B	AcP^C	
1-Western Saudi Arabia	143	6	16	107	-	12	2	9.79	84.62	5.59	Saha,N.,et al. 1980
2-Saudi Arabians	350	36	153	154	1	6	-	32.30	66.70	1.00	Goedde,H.W., et al. 1979

Table 5.1.3.IX Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes						Gene frequencies			Authors
		AcP A	AcP BA	AcP B	AcP CA	AcP CB	AcP C	AcP^A	AcP^B	AcP^C	
1-Pushtus	210	17	77	113	1	2	-	26.82	72.46	0.72	Goedde,H.W.,et al. 1977
2-Pushtus	103	13	33	56	-	1	-	28.60	70.90	0.50	Papiha,S.S.,et al. 1977
3-Hazaras	174	11	78	84	-	1	-	28.74	70.98	0.28	Goedde,H.W.,et al. 1977
4-Tajiks	310	28	126	151	1	4	-	29.52	69.68	0.80	Goedde,H.W.,et al. 1977
5-Daris	178	20	66	82	2	8	-	30.30	66.90	2.80	Papiha,S.S.,et al. 1977
6-Uzbeks	124	17	58	48	-	1	-	37.09	62.50	0.41	Goedde,H.W.,et al. 1977

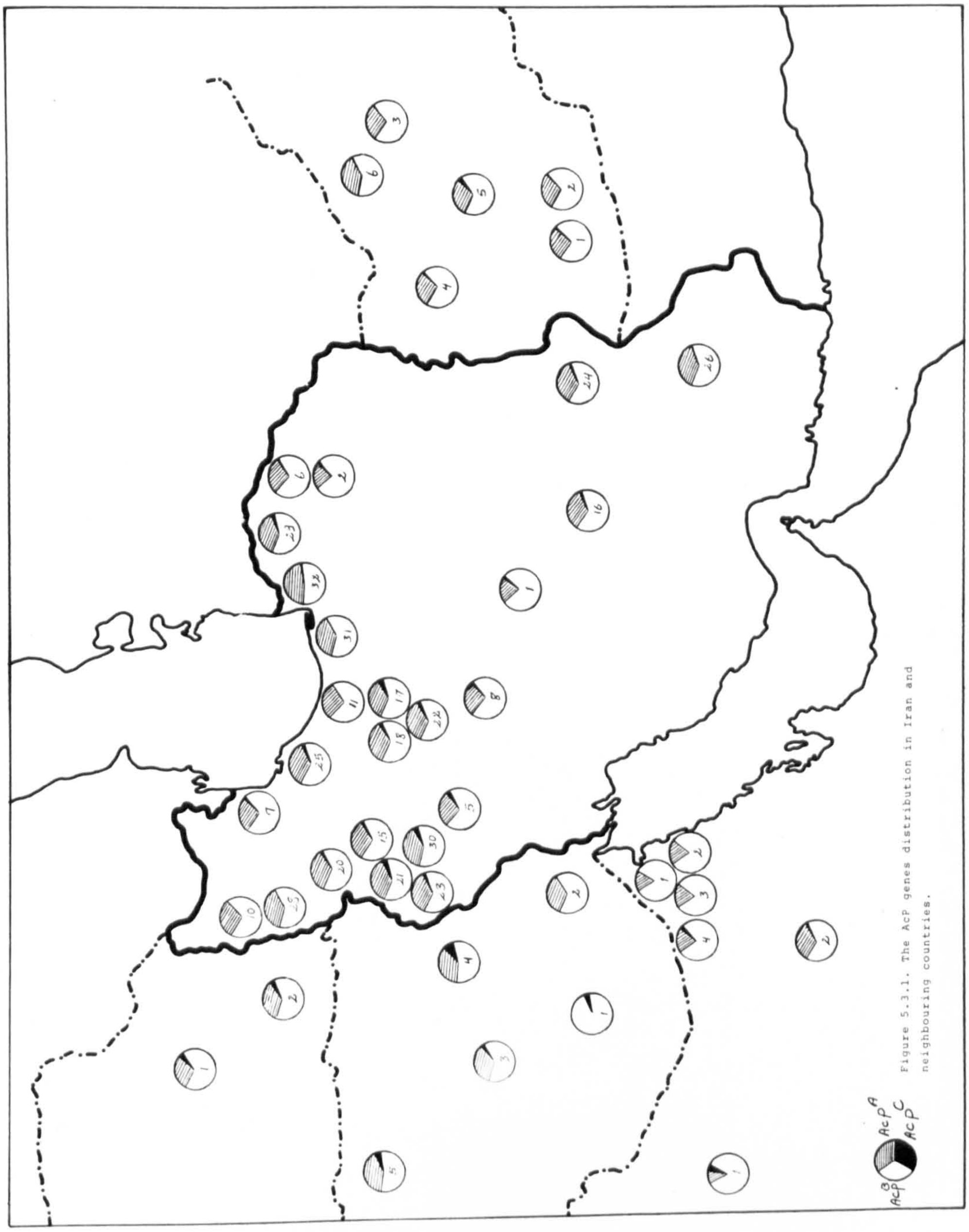


Figure 5.3.1. The AcP genes distribution in Iran and neighbouring countries.

5.3.2. The adenylate Kinase (AK) system

The distribution of adenylate kinase types and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.3.2.

The frequency of the AK^2 gene ranges from 1.38 to 10.81 percent in Iranians, being lowest in the Kurds of Rezaieh and highest in the Baluchis of Sistan and Baluchistan (Present investigation).

On the whole, with an average AK^2 frequency of 6.03 percent, the Iranian population appears to exhibit a higher AK^2 frequency than those varying between 2.5 and 6 percent in Europeans (Mourant et al, 1976).

It is difficult to see any definit regional trend of the distribution of the K^2 gene in Iran but it tends to be higher in the south east than the north west.

The extremely high AK^2 frequencies of 10.81 and 10.60 percent obtained in the present investigation in the Baluchis and the Zabolis of Sistan and Baluchistan (south eastern Iran) seem to be more similar to the high values found in Pakistan and India.

Regarding neighbouring areas, the frequency of the AK^2 gene in the populations of Turkey ranges from 4.00 percent in the Kurds (Richard, 1976) to 4.20 percent in the Turks (Hummel et al, 1970). With an average AK^2 frequency of 4.10 percent, the population of Turkey appears to exhibit a lower AK^2 frequency than that found in Iranians and more similar to the European frequency.

The AK^2 gene frequency in the populations of Iraq varies between zero in the Karaite Jews (Goldschmidt et al, 1976)

and 4.68 percent in the Jews (Adam, 1967). With an average AK^2 frequency of 2.04 percent, the Iraqi population seems to show a much lower AK^2 frequency than that found in Iranians and is even lower than the frequency in Europeans. The loss of the allele AK^2 in the Karaite Jews of Iraq clearly indicates the strong influence of isolation and genetic drift operating in this community.

The frequency of the AK^2 gene in the populations of Kuwait ranges from zero in the Ajman tribe to 5.00 percent in the general population of Kuwait (Khaled et al, 1981). With an average AK^2 frequency of 2.46 percent, the Kuwaiti population appears to exhibit a much lower AK^2 frequency than that found in Iranians and lower than the European frequency.

The AK^2 gene frequency of zero in the Ajman tribe of Kuwait may possibly be due to the relatively small sample size (N=50) and cannot be assumed as monomorphism of AK^1 though this situation occurs in several African populations (Tills et al, 1971).

The Ak^2 frequency of 3.50 percent in Saudi Arabians(Goedde et al, 1979) seems to be lower than the Iranian frequency and is more similar to that in Europeans.

The population of Pakistan with an AK^2 frequency of 12.96 percent (Cleghorn, 1967) appears to exhibit a much higher AK^2 frequency than that found in Iranians and of course much higher than the frequency in Europeans.

The frequency of the AK^2 gene in the populations of Afghanistan varies between 6.04 percent in the Hazaras and 10.72 percent in the Pushtus (Goedde et al, 1977). With an average AK^2 frequency of 8.53 percent, the Afghan population, like

that of Pakistan, shows a much higher AK^2 frequency than that found in Iranians and much higher than the European frequency. The frequency of the AK^2 gene is consistently higher in the Indian region than in Europe, and the AK^2 frequencies in Pakistan and Afghanistan are closer to the former.

Conclusion

In European populations the AK^2 gene nearly always has a frequency between 2.5 and 6 percent. In the populations of the Near East, including the Jews, frequencies are similar to those found in Europe.

In Africa AK^2 levels are about 1 percent and several Negroid populations tested have shown only the AK^1 allele.

The highest known frequencies of AK^2 are found in India(Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab populations of Iraq, Kuwait and Saudi Arabia with their relatively lower frequencies, the frequencies of the AK^2 gene appear to be higher than those in Europeans.

In Iranians though the AK^2 gene frequency seems to be higher than that in Europeans it is lower than in Indians but, the populations of Pakistan and Afghanistan with their much higher AK^2 frequencies appear to show more similarity with Indian populations.

On the whole, the frequency of the AK^2 gene seems to increases from Europe to India and the Iranian frequency is intermediate.

Table 5.3.2. I Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		AK 1-1	AK 2-1	AK 2-2	AK^1	AK^2		
1-Kurds.Rezaieh	145	141	4	-	98.62	1.38	Present study.	
2-Southern Gorgan, Behshahr,Sari	53	51	2	-	98.10	1.90	Kirk,R.L.,et al.	1977
3-Turks.Rezaieh	127	120	7	-	97.24	2.76	Present study.	
4-Jews	164				96.80	3.20	Simhai,B.	1974
5-Esfahan	83	76	7	-	95.78	4.22	Sawhney,K.S.	1975
6-Gonbad	155	140	15	-	95.20	4.80	Kirk,R.L.,et al.	1977
7-Tavalesh,Astara	61	55	6	-	95.10	4.90	Kirk,R.L.,et al.	1977
8-Moslems.Shiraz	322	290	32	-	95.03	4.97	Bowman,J.E., and Ronaghy,H.	1967
9-Tehran	168	150	18	-	94.64	5.36	Sawhney,K.S.	1975
10.Babol,Shahi,Amol	64	58	5	1	94.50	5.50	Kirk,R.L.,et al.	1977
11-Turks.Shirvan,Khorasan	116	103	13	-	94.40	5.60	Present study.	
12-Kurds.Shirvan,Khorasan	103	91	12	-	94.17	5.83	Present study.	

Table 5.3.2.I.(Cont.) Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		AK 1-1	AK 2-1	AK 2-2	AK^1	AK^2	
13-Zoroastrians	120	105	15	-	93.75	6.25	Present study.
14-Kurdish Jews	94	82	12	-	93.62	6.38	Godber,Marilyn.J.,et al. 1973
15-Northern Gorgan	44	38	6	-	93.20	6.80	Kirk,R.L.,et al. 1977
16-Kurds	182	159	21	2	93.13	6.87	Tills,D.,et al. 1971
17-Kurdish Jews	106	91	15	-	92.92	7.08	Tills,D.,et al. 1977
18-Kerman	309	268	38	3	92.88	7.12	Present study.
19-Tehran	357	306	51	-	92.86	7.14	Present study.
20-Kurds.Baneh,Marivan	77	68	7	2	92.86	7.14	Lehmann,H.,et al. 1973
21-Lurs.Luristan	178	154	22	2	92.70	7.30	Present study.
22-Kurds.Sanandaj	106	91	14	1	92.45	7.55	Lehmann,H.,et al. 1973
23-Shahsavar,Rudsar,Rudbar,Rasht,Langarud, Lahijan,Bandar Pahlavi	86	70	16	-	90.70	9.30	Kirk,R.L.,et al. 1977
24-Zabolis.Sistan & Baluchistan	118	94	23	1	89.40	10.60	Present study.
25-Baluchis.Sistan & Baluchistan	111	88	22	1	89.19	10.81	Present study.

Table 5.3.2. III Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		AK 1-1	AK 2-1	AK 2-2	AK^1	AK^2		
1- Kurds					96.00	4.00	Richard,P.	1976
2-Turks	274	251	23	-	95.80	4.20	Kummel,K.,et al.	1970

Table 5.3.2. IV Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Iraq.

Population	Number Tested	Phenotypes AK 1-1	AK 2-1	AK 2-2	Gene frequencies AK^1	AK^2	Authors
1-Karaite Jews	71	71	-	-	100.00	0.00	Goldschmidt,Elizabeth., et al. 1976
2-Kurdish Jews.North west	61	60	1	-	99.18	0.82	Tills,D.,et al. 1977
3-Kurdish Jews	27	26	1	-	98.15	1.85	Godber,Marilyn.J.,et al. 1973
4-Kurdish Jews.South east	50	48	2	-	98.00	2.00	Tills,D.,et al. 1977
5-Jews	190	-	-	-	97.10	2.90	Szeinberg,A.,et al. 1971
6-Jews	139	126	13	-	95.32	4.68	Adam.edited by : Rapley,S., et al. 1967

Table 5.3.2. V Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Kuwait

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		AK 1-1	AK 2-1	AK 2-2	AK^1	AK^2		
1-Ajman tribe	50	50	-	-	100.00	0.00	Khaled,E.,et al.	1981
2-Suluba tribe	51	49	2	-	98.00	2.00	Khaled,E.,et al.	1981
3-Kuwaiti Arabs	159	150	9	-	97.17	2.83	Sawhney,K.S.	1975
4-General population	86	77	9	-	95.00	5.00	Khaled,E.,et al.	1981

Table 5.3.2. VI Red cell Adenylate Kinase(AK) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		AK 1-1	AK 2-1	AK 2-2	AK^1	AK^2	
1-Saudi Arabians	359	335	23	1	96.50	3.50	Goedde,H.W.,et al. 1979

Table 5.3.2. VIII Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Pakistan

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		AK 1-1	AK 2-1	AK 2-2	AK^1	AK^2	
1-Pakistanis	54	40	14	-	87.04	12.96	Cleghorn et al.edited 1967 by: Rapley, S.,et al.

Table 5.3.2. IX Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		AK 1-1	AK 2-1	AK 2-2	AK^1	AK^2		
1-Hazaras	174	155	17	2	93.96	6.04	Goedde,H.W.,et al.	1977
2-Daris	178	152	24	2	92.10	7.90	Papiha,S.S.,et al.	1977
3-Pushtus	102	85	17	-	91.70	8.30	Papiha,S.S.,et al.	1977
4-Tajiks	310	259	49	2	91.45	8.55	Goedde,H.W.,et al.	1977
5-Uzbeks	124	103	18	3	90.32	9.68	Goedde,H.W.,et al.	1977
6-Pushtus	210	167	41	2	89.28	10.72	Goedde,H.W.,et al.	1977

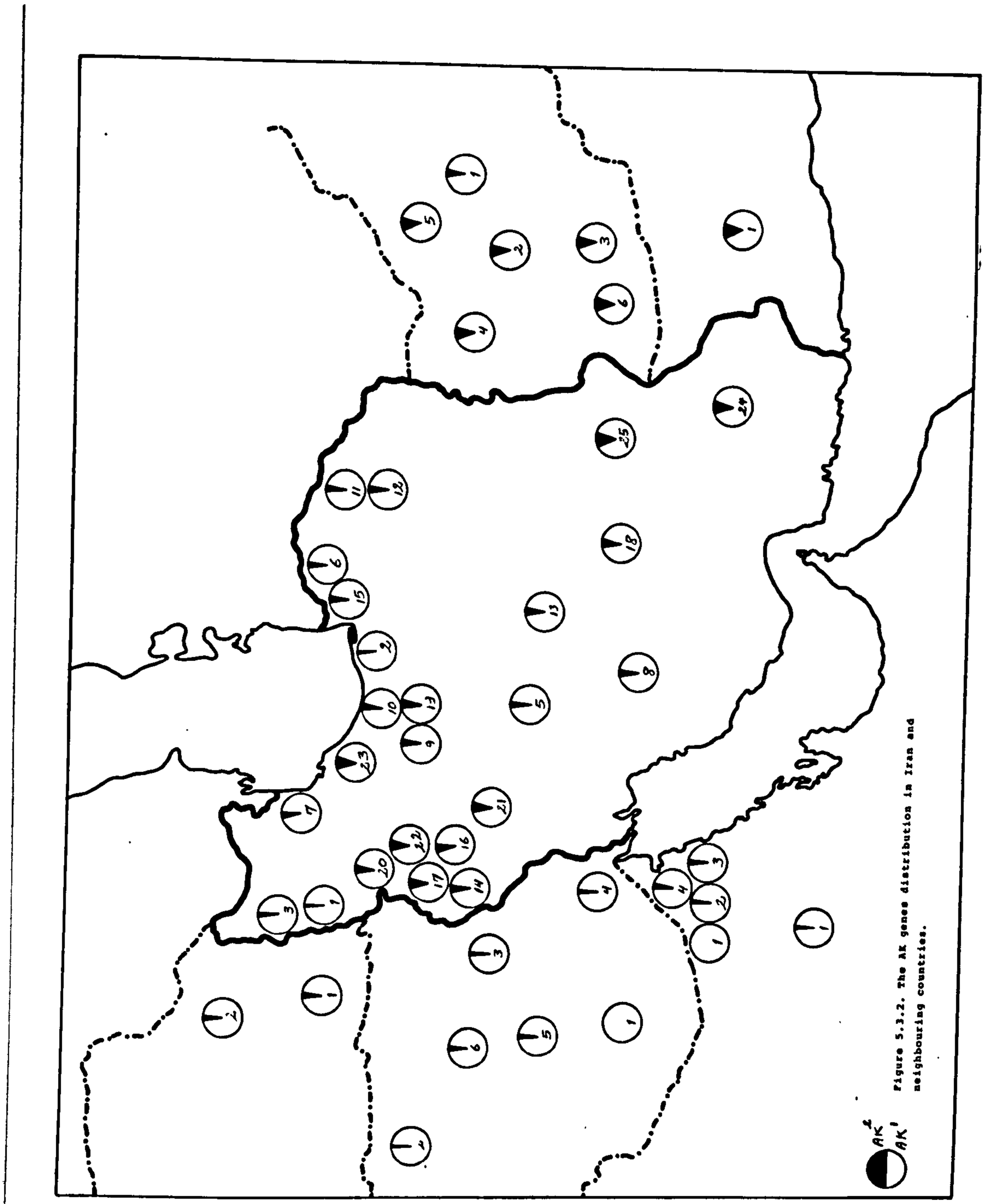


Figure 5.3.2. The AK genes distribution in Iran and neighbouring countries.

5.3.3. The Phosphoglucomutase Locus 1 (PGM_1) system

The distribution of PGM_1 types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.3.3.

The frequency of the PGM_1^2 gene varies between 22.08 and 34.56 percent in Iranians, being lowest in the Kurds of Baneh, Marivan (Lehmann et al, 1973) and highest in the Turks of Rezaieh (Present investigation). With the exception of the PGM_1^2 frequency of 43.56 percent in the Turks of Rezaieh, values obtained in the present study are within the range of variation. On the whole, with an average PGM_1^2 frequency of 31.43 percent, the Iranian population appears to exhibit a higher PGM_1^2 frequency than that of around 20 percent found in northern Europeans. The Iranian frequency seems to be more similar to that of around 30 percent in southern European and Mediterranean populations. Some differences in the distribution of the PGM_1^2 gene frequencies seem to exist in Iran as PGM_1^2 frequencies, averaging 30.82 percent, in the populations of western Iran are lower than those averaging 34.10 percent in the populations of eastern Iran.

The Kurdish Jews, both in Iran and in Iraq, seem to be characterized by lowerfrequencies of the PGM_1^2 gene but the Jews exhibit relatively higher values in both countries.

Regarding neighbouring populations, the frequency of the PGM_1^2 gene in the populations of Turkey ranges from 32.30 percent in the Turks (Hummel et al, 1970) to 34.00 percent in the Kurds (Richard, 1976). With an average PGM_1^2 frequency of 33.15 percent, the population of Turkey appears to exhibit a PGM_1^2 frequency similar to that found in Iranians and comparable with other southern European and Mediterranean populations.

The frequency of the PGM_1^2 gene in the populations of Iraq varies between 16.39 percent in the Kurdish Jews of the north west (Tills et al, 1977) and 32.60 percent in the Jews (Hopkinson et al, 1966). With an average PGM_1^2 frequency of 25.36 percent, the Jewish population of Iraq seems to show a lower PGM_1^2 frequency than the average for Iran, but similar to the relatively lower values found in Iranian Jews both Kurdish and non-Kurdish.

The PGM_1^2 gene frequency in the populations of Kuwait ranges from 25.00 percent in the Ajman tribe to 31.00 percent in the Suluba tribe(Khaled et al, 1981). With an average PGM_1^2 frequency of 29.15 percent, the Kuwaiti population appears to exhibit a PGM_1^2 frequency similar to that found in Iranians.

A similar PGM_1^2 frequency of 29.80 percent is reported by Goedde et al (1979) for Saudi Arabians.

The frequency of the PGM_1^2 gene in the populations of Afghanistan varies between 14.66 percent in the Hazaras and 27.42 percent in the Tajiks (Goedde et al,1977). With an average PGM_1^2 frequency of 23.65 percent, the Afghan population seems to show lower PGM_1^2 frequency than that found in Iranians.

Conclusion

European populations have frequencies of PGM_1^2 increasing from 14 percent in Ireland and 18 percent in Iceland,eastward and southward to 31 percent in Greece.

Frequencies of PGM_1^2 in India exceed 30 percent but the Chinese and Japanese have between 20 and 25 percent.

African Negroid populations vary between 15 and 24 percent (Mourant et al, 1976).

In Iranian and neighbouring populations,with the exception of the Afghans with their relatively lower frequency,the

frequency of PGM_1^2 appears to be higher than that found in northern Europeans and more similar to that in southern European and Mediterranean populations.

Table 5.3.3.I Red cell Phosphoglucomutase Locus 1 (PGM_1) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		PGM 1-1	PGM 2-1	PGM 2-2	PGM 6-1	PGM_1^1	PGM_1^2	PGM_1^6		
1-Kurds.Baneh,Marivan	77	45	30	2	-	77.92	22.08	0.00	Lehmann, H.,et al.	1973
2-Kurdish Jews	106	65	34	7	-	77.36	22.64	0.00	Tills,D., et al.	1977
3-Zoroastrians	104	60	40	4	-	76.92	23.08	0.00	Present study.	
4-Tehran	165	101	50	14	-	76.36	23.64	0.00	Sawhney,K.S.	1975
5-Esfahan	86	50	28	7	1	75.00	24.42	0.58	Sawhney,K.S.	1975
6-Kurdish Jews	94	55	32	7	-	75.53	24.47	0.00	Godber,Marilyn.J.,et al.	1973
7-Armenians	180	-	-	-	-	74.00	26.00	0.00	Simhai,B.	1978
8-Babol,Shahi,Amol	64	36	22	6	-	73.40	26.60	0.00	Kirk,R.L.,et al.	1977
9-Jews	164	-	-	-	-	71.70	28.30	0.00	Simhai,B.	1974
10-Lurs.Luristan	175	90	68	17	-	70.86	29.14	0.00	Present study.	
11-Northern Gorgan	44	20	22	2	-	70.50	29.50	0.00	Kirk,R.L.,et al.	1977
12-Tehran	127	61	52	14	-	68.50	31.50	0.00	Farhud,D.D.,et al.	1973
13-Kurds.Sanandaj	106	48	49	9	-	68.40	31.60	0.00	Lehmann,H.,et al.	1973

Table 5.3.3.I Red cell Phosphoglucomutase Locus 1 (PGM_1) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		PGM 1-1	PGM 2-1	PGM 2-2	PGM 6-1	PGM_1^1	PGM_1^2	PGM_1^6		
1-Kurds.Baneh,Marivan	77	45	30	2	-	77.92	22.08	0.00	Lehmann, H.,et al.	1973
2-Kurdish Jews	106	65	34	7	-	77.36	22.64	0.00	Tills,D., et al.	1977
3-Zoroastrians	104	60	40	4	-	76.92	23.08	0.00	Present study.	
4-Tehran	165	101	50	14	-	76.36	23.64	0.00	Sawhney,K.S.	1975
5-Esfahan	86	50	28	7	1	75.00	24.42	0.58	Sawhney,K.S.	1975
6-Kurdish Jews	94	55	32	7	-	75.53	24.47	0.00	Godber,Marilyn.J.,et al.	1973
7-Armenians	180	-	-	-	-	74.00	26.00	0.00	Simhai,B.	1978
8-Babol,Shahi,Amol	64	36	22	6	-	73.40	26.60	0.00	Kirk,R.L.,et al.	1977
9-Jews	164	-	-	-	-	71.70	28.30	0.00	Simhai,B.	1974
10-Lurs.Luristan	175	90	68	17	-	70.86	29.14	0.00	Present study.	
11-Northern Gorgan	44	20	22	2	-	70.50	29.50	0.00	Kirk,R.L.,et al.	1977
12-Tehran	127	61	52	14	-	68.50	31.50	0.00	Farhud,D.D.,et al.	1973
13-Kurds.Sanandaj	106	48	49	9	-	68.40	31.60	0.00	Lehmann,H.,et al.	1973

Table 5.3.3.I (Cont.) Red cell Phosphoglucomutase Locus 1 (PGM_1) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		PGM 1-1	PGM 2-1	PGM 2-2	PGM 6-1	PGM_1^1	PGM_1^2	PGM_1^6	
14-Turks.Shirvan,Khorasan	110	50	50	10	-	68.18	31.82	0.00	Present study.
15-Zabolis.Sistan & Baluchistan	117	53	51	13	-	67.09	32.91	0.00	Present study.
16-Tehran	346	152	153	41	-	66.04	33.96	0.00	Present study.
17-Baluchis.Sistan & Baluchistan	110	45	54	11	-	65.45	34.55	0.00	Present study.
18-Kurds.Shirvan,Khorasan	92	40	40	12	-	65.22	34.78	0.00	Present study.
19-Shahsavar,Rudsar, Rudbar,Rasht,Langarud,Lahijan,Bandar-Pahlavi	86	34	40	11	1	63.40	36.00	0.60	Kirk,R.L.,et al. 1977
20-Tavalesh,Astara	61	27	24	10	-	63.90	36.10	0.00	Kirk,R.L.,et al. 1977
21-Kerman	310	123	144	43	-	62.91	37.09	0.00	Present study.
22-Gonbad	155	49	88	18	-	60.00	40.00	0.00	Kirk,R.L.,et al. 1977

Table 5.3.3.I. (Cont). Red cell Phosphoglucomutase Locus 1(PGM_1) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		PGM 1-1	PGM 2-1	PGM 2-2	PGM 6-1	PGM_1^1	PGM_1^2	PGM_1^6		
23-Southern Gorgan Behshahr,Sari	53	18	25	9	1	58.50	40.60	0.90	Kirk,R.L.,et al.	1977
24-Kurds.Rezaieh	138	47	68	23	-	58.70	41.30	0.00	Present study.	
25-Turks.Rezaieh	101	36	42	23	-	56.44	43.56	0.00	Present study.	

Table 5.3.3. III Red cell Phosphoglucomutase Locus 1(PGM_1) phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		PGM 1-1	PGM 2-1	PGM 2-2	PGM_1^1	PGM_1^2		
1-Turks	274	119	133	22	67.70	32.30	Hummel,K.,et al.	1970
2-Kurds					66.00	34.00	Richard,P.	1976

Table 5.3.3. IV. Red cell Phosphoglucomutase Locus 1 (PGM_1) phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		PGM 1-1	PGM 2-1	PGM 2-2	PGM_1^1	PGM_1^2		
1-Kurdish Jews. North west	61	42	18	1	83.61	16.39	Tills,D.,et al.	1977
2-Kurdish Jews	27	17	10	-	81.48	18.52	Godber,Marilyn.J., et al.	1973
3-Karaite Jews	70	36	27	7	70.70	29.30	Goldschmidt,Elizabeth., et al.	1976
4-Kurdish Jews. South east	50	24	22	4	70.00	30.00	Tills,D.,et al.	1977
5-Jews	69				67.40	32.60	Hopkinson,D.A.,et al.	1966

Table 5.3.3. V Red cell Phosphoglucomutase Locus 1(PGM_1) phenotypes and gene frequencies distribution in Kuwait

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		PGM 1-1	PGM 2-1	PGM 2-2	PGM_1^1	PGM_1^2		
1-Ajman tribe	50	29	17	4	75.00	25.00	Khaled,E., et al.	1981
2-Kuwaiti Arabs	147	77	53	17	70.41	29.59	Sawhney,K.S.	1975
3-General population	88	45	32	11	69.00	31.00	Khaled,E., et al.	1981
4-Suluba tribe	51	22	26	3	69.00	31.00	Khaled,E., et al.	1981

Table 5.3.3. VI Red cell Phosphoglucomutase Locus 1 (PGM_1) phenotypes and gene frequencies distribution in Saudi Arabia.

Population	Number Tested	Phenotypes					Gene frequencies				Authors
		PGM 1-1	PGM 2-1	PGM 2-2	PGM 3-1	PGM 6-1	PGM_1^1	PGM_1^2	PGM_1^3	PGM_1^6	
1-Saudi Arabians	359	180	136	39	2	2	69.60	29.80	0.30	0.30	Goedde,H.W.,et al. 1979

Table 5.3.3. IX Red cell Phosphoglucomutase Locus 1(PGM_1) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		PGM 1-1	PGM 2-1	PGM 2-2	PGM 4-1	PGM 6-1	PGM 6-2	PGM_1^1	PGM_1^2	PGM_1^4	PGM_1^6	
1-Hazaras	174	124	46	2	1	-	1	84.78	14.66	0.28	0.28	Goedde,H.W.,et al. 1977
2-Uzbeks	124	80	36	8	-	-	-	79.03	20.97	0.00	0.00	Goedde,H.W.,et al. 1977
3-Pushtus	210	119	74	16	-	1	-	74.53	25.24	0.00	0.23	Goedde,H.W.,et al. 1977
4-Daris	173	100	54	19	-	-	-	73.40	26.60	0.00	0.00	Papiha,S.S., et al. 1977
5-Pushtus	102	60	29	13	-	-	-	73.00	27.00	0.00	0.00	Papiha,S.S., et al. 1977
6-Tajiks	310	163	124	23	-	-	-	72.58	27.42	0.00	0.00	Goedde,H.W.,et al. 1977

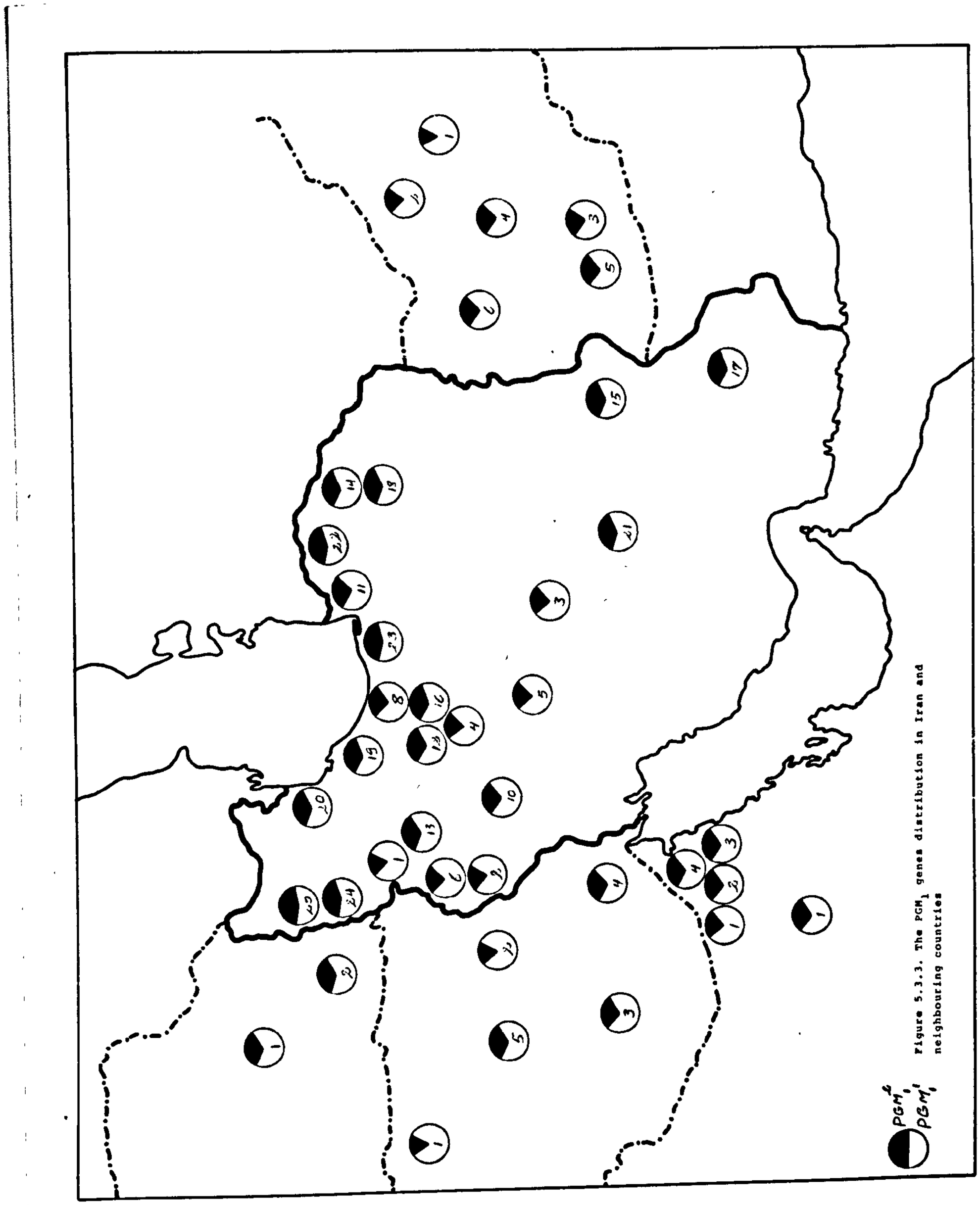


Figure 5.3.3. The PGM_1 genes distribution in Iran and neighbouring countries

5.3.4. The adenosine deaminase (ADA) system

The distribution of adenosine deaminase types and respective gene frequencies in Iranian and neighbouring populations is shown in Tables 5.3.4.

The frequency of the ADA^2 gene varies between 6.80 and 19.56 percent in Iranians, being lowest in northern Gorgan (Kirk et al, 1977) and highest in the Zoroastrians(Present investigation). With the exception of ADA^2 frequency of 19.56 percent in the Zoroastrians, values obtained in the present study are within the range of variation.

On the whole, with an average ADA^2 frequency of 13.29 percent, the Iranian population appears to exhibit a higher ADA^2 frequency than that of around 7 percent found in Europeans(Mourant et al, 1976).

Since the data available on ADA studies in Iran are still very few, complete discussion of the situation within the country is impossible.

Regarding neighbouring areas, the ADA^2 frequency of 9.60 percent in the Turks of Turkey (Altay et al, 1974) seems to be lower than that found in Iranians and quite close to those in Italians and Greeks.

The frequency of the ADA^2 gene in the populations of Iraq ranges from 9.76 percent in the Kurdish Jews of the south east to 24.39 percent in the Kurdish Jews of the north west(Tills et al, 1977). With an average ADA^2 frequency of 17.07 percent, the Iraqi Kurdish Jews seem to show a higher ADA^2 frequency than that found in Iranians and higher than the frequency in Europeans.

The ADA^2 gene frequency in the populations of Kuwait varies between 2.00 percent in the Suluba tribe and 16.00 per-

cent in the general population of Kuwait (Khaled et al,1981). With an average ADA^2 frequency of 8.00 percent, the Kuwaiti population appears to exhibit a lower ADA^2 frequency than that found in Iranians and also lower than the frequency in southern Europeans.

A similarly low ADA^2 frequency of 6.80 percent is reported by Goedde et al (1979) in Saudi Arabians.

The frequency of the ADA^2 gene in the Afghan populations ranges from 10.09 percent in the Uzbeks to 17.39 percent in the Pushtus (Goedde et al,1977). With an average ADA^2 frequency of 12.85 percent, the population of Afghanistan seems to show an ADA^2 frequency similar to that found in Iranians and higher than the European frequency.

Conclusion

The frequency of the ADA^2 gene ranges from 5 to 9 percent in European populations.Moreover,the European data seem to show an eastward increase in the frequency of ADA^2 from 5 percent in Ireland and 6 percent in England to 8 percent in Germany and 9 percent in Italy.

The frequency of the ADA^2 gene is consistently higher in the Indian region than in Europe.

In Africa the average frequency is under 1 percent(Mourant et al, 1976).

In Iranian and neighbouring populations,with the exception of the Arabs of Kuwait and Saudi Arabia with their relatively lower values, frequencies of the ADA^2 gene appear to be higher than those found in Europeans and more similar to the Indian frequencies. Thus as in Europe, the same trend seems to continue in Asia with around 9 percent in Turkey and 13 percent in Iran, Afghanistan and India.

Table 5.3.4.I Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes					Gene frequencies				Authors	
		ADA 1-1	ADA 2-1	ADA 2-2	ADA 4-1	ADA 6-1	ADA^1	ADA^2	ADA^4	ADA^6		
1-Northern Gorgan	44	38	6	-	-	-	93.20	6.80	0.00	0.00	Kirk,R.L.,et al.	1977
2-Kurds.Baneh,Marivan	77	66	11	-	-	-	92.86	7.14	0.00	0.00	Lehmann,H.,et al.	1973
3-Tavalesh,Astara	61	51	10	-	-	-	91.80	8.20	0.00	0.00	Kirk,R.L.,et al.	1977
4-Gonbad	155	125	27	3	-	-	89.40	10.60	0.00	0.00	Kirk,R.L.,et al.	1977
5-Kurds	182	142	37	3	-	-	88.20	11.80	0.00	0.00	Vanden Braden.,et al.	1971
6-Shahsavar,Rudsar, Rudbar,Rasht,Langa-rud,Lahijan,Band-ar Pahlavi	86	67	16	3	-	-	87.20	12.80	0.00	0.00	Kirk,R.L.,et al.	1977
7-Turks.Rezaieh	141	104	30	4	1	2	85.46	13.48	0.35	0.71	Present study.	
8-Jews	164	-	-	-	-	-	85.80	14.20	0.00	0.00	Simhai,B.	1974
9-Kurds.Rezaieh	147	106	38	3	-	-	85.04	14.96	0.00	0.00	Present study.	
10-Southern Gorgan, Behshahr,Sari	53	37	16	-	-	-	84.90	15.10	0.00	0.00	Kirk,R.L.,et al.	1977
11-Kurds.Shirvan, Khorasan	101	74	23	4	-	-	84.66	15.34	0.00	0.00	Present study.	

Table 5.3.4.I (Cont.) Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes					Gene frequencies				Authors	
		ADA 1-1	ADA 2-1	ADA 2-2	ADA 4-1	ADA 6-1	ADA^1	ADA^2	ADA^4	ADA^6		
12-Turks.Shirvan, Khorasan	118	83	33	2	-	-	84.33	15.67	0.00	0.00	Present study.	
13-Kurds. Sanandaj	105	75	27	3	-	-	84.29	15.71	0.00	0.00	Lehmann,H.,et al.	1973
14-Babol,Shahi,Amol	64	43	19	2	-	-	82.00	18.00	0.00	0.00	Kirk,R.L.,et al.	1977
15-Zoroastrians	46	31	12	3	-	-	80.44	19.56	0.00	0.00	Present study.	

Table 5.3.4. III Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		ADA 1-1	ADA 2-1	ADA 2-2	ADA^1	ADA^2	
1-Turks	566	462	99	5	90.40	9.60	Altay,C., et al. 1974

Table 5.3.4. IV Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		ADA 1-1	ADA 2-1	ADA 2-2	ADA^1	ADA^2		
1-Kurdish Jews. South east	41	33	8	-	90.24	9.76	Tills,D.,et al.	1977
2-Kurdish Jews. North west	41	22	18	1	75.61	24.39	Tills,D.,et al.	1977

Table 5.3.4. V

Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Kuwait

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		ADA 1-1	ADA 2-1	ADA 2-2	ADA^1	ADA^2		
1-Suluba tribe	52	50	2	-	98.00	2.00	Khaled,E.,et al.	1981
2-Ajman tribe	51	45	6	-	94.00	6.00	Khaled,E.,et al.	1981
3-General population	87	63	24	-	84.00	16.00	Khaled,E.,et al.	1981

Table 5.3.4. VI Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		ADA 1-1	ADA 2-1	ADA 2-2	ADA 4-1	ADA^1	ADA^2	ADA^4	
1- Saudi Arabians	359	318	31	9	1	93.00	6.80	0.20	Goedde,H.W.,et al. 1979

Table 5.3.4. IX Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes						Gene frequencies				Authors
		ADA 1-1	ADA 2-1	ADA 2-2	ADA 4-1	ADA 4-2	ADA 6-1	ADA^1	ADA^2	ADA^4	ADA^6	
1-Uzbeks	124	96	21	1	3	2	1	87.50	10.09	2.01	0.40	Goedde,H.W.,et al. 1977
2-Hazaras	174	144	25	5	-	-	-	89.88	10.12	0.00	0.00	Goedde,H.W.,et al. 1977
3-Tajiks	310	238	63	7	1	1	-	87.09	12.59	0.32	0.00	Goedde,H.W.,et al. 1977
4-Daris	177	131	45	1	-	-	-	86.70	13.30	0.00	0.00	Papiha,S.S.,et al. 1977
5-Pushtus	103	76	26	1	-	-	-	86.40	13.60	0.00	0.00	Papiha,S.S.,et al. 1977
6-Pushtus	210	143	59	7	1	-	-	82.38	17.39	0.23	0.00	Goedde,H.W.,et al. 1977

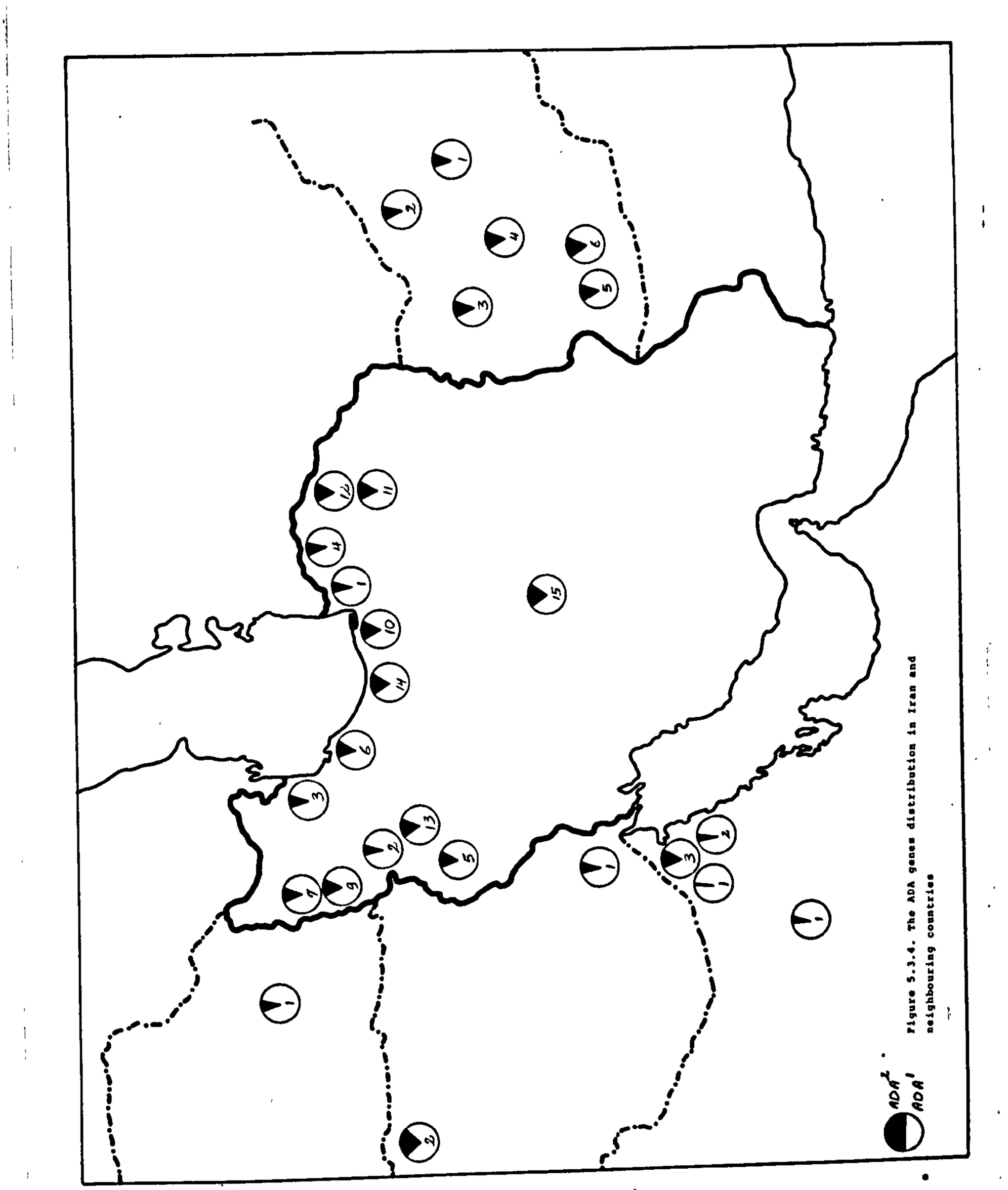


Figure 5.3.4. The ADA genes distribution in Iran and neighbouring countries

5.3.5. The esterase D (EsD) system

The distribution of the EsD types and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.3.5.

Esterase D is a relatively new polymorphic system, so that knowledge of the allele frequencies in different populations is, as yet, limited.

No observation of the EsD system among Iranians has hitherto been reported. The known heterogenity of the populations of Iran suggested that a searsh for variants there would be more rewarding than in more homogenous populations, so that the present study was under taken in samples from the ten different population groups in Iran.

The frequency of the EsD^2 gene varies between 12.61 and 29.55 percent in Iranians, being lowest in the Turks of Shirvan, Khorasan and highest in the Baluchis of Sistan and Baluchistan. With an average EsD^2 frequency of 19.23 percent, the Iranian population appears to exhibit a much higher EsD^2 frequency than that of about 11 percent in Europeans (Welch and Lee, 1974; Benkmann and Goedde, 1974; Koster et al, 1975).

In the ten Iranian samples, there does not appear to be any consistent pattern in the distribution of EsD genes.

The Turkish sample of Rezaieh in the north western region shows an EsD^2 frequency similar to that of the Baluchi sample of Sistan and Baluchistan in the south eastern region while the frequency in the Turks of Shirvan, Khorasan in the north east is similar to that in the Lurs of Luristan in the south western region.

Regarding neighbouring populations, the frequency of the EsD^2 gene in the populations of Iraq ranges from 18.90

percent in Baghdad to 24.20 percent in Ramadi(Papiha and Al-Agidi, 1976). With an average EsD^2 frequency of 21.62 percent, the population of Iraq, like that of Iran, seems to show a much higher EsD^2 frequency than that found in Europeans.

Similar high EsD^2 frequencies of 19.68 and 19.50 percent are reported by Cartwright et al(1976)and Goedde et al (1979)in the Kuwaitis and the Saudi Arabians,respectively.

The EsD^2 gene frequency in the populations of Afghanistan varies between 10.30 percent in the Afghan sample of papiha and Nahar(1977) and 20.96 percent in the Uzbeks(Goedde et al, 1977). With an average EsD^2 frequency of 16.50 percent, though the Afghan population appears to exhibit a lower EsD^2 frequency than that found in Iranians but the frequency is still higher than the European frequency.

Conclusion

Distinct geographical variation seems to exist in the frequency of the EsD^2 allele, particularly on the continental level.

The frequency of the EsD^2 gene in most populations of European origin is around 11 percent.

In eastern Asia it is considerably higher, around 34 percent.

India with an EsD^2 frequency of around 22 percent occupies an intermediate position between the European and east Asian levels.

The frequency of EsD^2 is only about 9 percent in Africans (Papiha and Nahar, 1977).

In Iranian and neighbouring populations, with the exception of the Afghans with their relatively lower frequency,the frequency of the EsD^2 gene appears to be much higher than that found in Europeans but slightly lower than that in Indians.

The position of Afghanistan, with a lower EsD^2 frequency than India, may indicate the historical association of the Afghans with the populations from the west.

Thus there is a clear increase in the frequency of the EsD^2 allele as one moves from Europe into Asia, and as one moves from peninsular India into the partially Mongoloid regions.

Already it seems clear that the major component of variation in the esterase D polymorphism is ethnic, strongly related to the continental groups to which the sampled populations belong, and this suggests that the frequencies owe little to ecological factors.

Table 5.3.5.I. Red cell Esterase D(EsD) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		EsD 1-1	EsD 2-1	EsD 2-2	EsD^1	EsD^2	
1-Turks.Shirvan,Khorasan	115	88	25	2	87.39	12.61	Present study.
2-Lurs.Luristan	178	136	36	6	86.52	13.48	Present study.
3-Zabolis.Sistan & Baluchistan	117	86	28	3	85.47	14.53	Present study.
4-Kurds.Shirvan,Khorasan	101	71	27	3	83.66	16.34	Present study.
5-Zoroastrians	111	74	35	2	82.43	17.57	Present study.
6-Tehran	345	229	105	11	81.60	18.40	Present study.
7-Kerman	306	204	91	11	81.54	18.46	Present study.
8-Kurds.Rezaieh	143	81	57	5	76.57	23.43	Present study.
9-Turks. Rezaieh	129	66	54	9	72.09	27.91	Present study.
10-Baluchis.Sistan & Baluchistan	110	57	41	12	70.45	29.55	Present study.

Table 5.3.5. IV Red cell Esterase D (EsD) phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		EsD 1-1	EsD 2-1	EsD 2-2	EsD^1	EsD^2	
1-Baghdad	122	81	36	5	81.10	18.90	Papiha,S.S., & Al-Agidi,S.K. 1976
2-Basra	92	59	30	3	80.40	19.60	Papiha,S.S., & Al-Agidi,S.K. 1976
3-Kirkuk	42	24	16	2	76.20	23.80	Papiha,S.S., & Al-Agidi,S.K. 1976
4-Ramadi	64	34	29	1	75.80	24.20	Papiha,S.S., & Al-Agidi,S.K. 1976

Table 5.3.5. V Red cell Esterase D(EsD) phenotypes and gene frequencies distribution in Kuwait

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		EsD 1-1	EsD 2-1	EsD 2-2	EsD^1	EsD^2		
1-Kuwaitis	160	101	55	4	80.32	19.68	Cartwright,R.A.,et al.	1976

Table 5.3.5. VI Red cell Esterase D (EsD) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		EsD 1-1	EsD 2-1	EsD 2-2	EsD^1	EsD^2	
1-Saudi Arabians	359	229	120	10	80.50	19.50	Goedde,H.W., et al. 1979

Table 5.3.5. IX

Red cell Esterase D (EsD) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		EsD 1-1	EsD 2-1	EsD 2-2	EsD 3-1	EsD^1	EsD^2	EsD^3		
1-Afghans	224	182	38	4	-	89.70	10.30	0.00	Papiha,S.S., & Nahar,A.	1977
2-Pushtus	210	153	51	5	1	85.24	14.53	0.23	Goedde,H.W.,et al.	1977
3-Tajiks	308	210	86	12	-	82.26	17.74	0.00	Goedde,H.W.,et al.	1977
4-Hazaras	174	117	48	9	-	81.04	18.96	0.00	Goedde,H.W.,et al.	1977
5-Uzbeks	124	74	40	6	4	77.41	20.96	1.63	Goedde,H.W.,et al.	1977

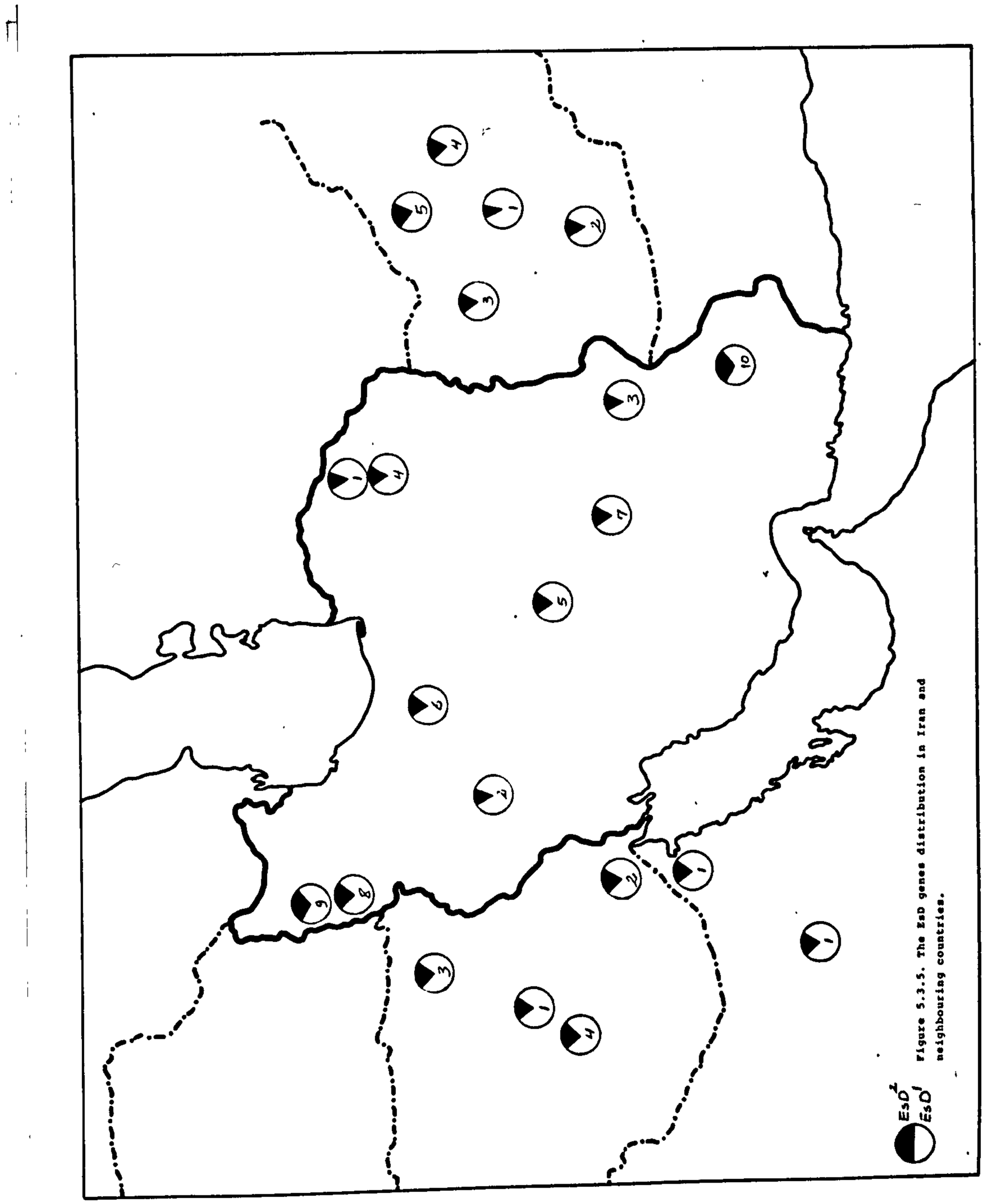


Figure 5.3.5. The EsD genes distribution in Iran and neighbouring countries.

5.3.6. The 6-phosphogluconate dehydrogenase(6PGD)system

The distribution of 6-phosphogluconate dehydrogenase types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.3.6.

The frequency of the PGD^C gene ranges from zero to 7.14 percent in Iranians, being lowest in Tavalesh, Astara (Kirk et al, 1977) and highest in the Kurds of Baneh, Marivan(Lehmann et al, 1973). Values obtained in the present investigation are within this range of variation.

On the whole, with an average PGD^C frequency of 3.19 percent, the Iranian population appears to exhibit a PGD^C frequency similar to that varying between 1 and 4 percent found in Europeans (Mourant et al, 1976). However, it seems to be lower than for some other Middle Eastern populations (Tills et al, 1971). It appears from the table that there is an apparent cline for the PGD^C allele in the Caspian sea area of Iran, declining from 5.20 percent in Gonbad (east Caspian) to zero in Tavalesh, Astara (west Caspian)

The absence of the 6PGD variants in the Iranian Zoroastrians could possibly be due to the small size of the series investigated.

The Kurdish populations of Iran both Jewish and non-Jewish seem to exhibit the highest PGD^C frequencies (average around 5 percent) within the country.

Regarding neighbouring groups, the frequency of the PGD^C allele in the populations of Turkey varies between 0.10 percent in the Turks(Altay et al, 1974) and 4.00 percent in the Kurds (Richard, 1976). With an average PGD^C frequency of 2.47 percent, the population of Turkey, like that of Iran, seems to show a PGD^C allele frequency similar to that found

in Europeans.

The PGD^C gene frequency in the populations of Iraq ranges from zero in the Karaite Jews (Goldschmidt et al, 1976) to 4.10 percent in the Kurdish Jews of the north west (Tills et al, 1977). With an average PGD^C frequency of 2.47 percent, the Jewish population of Iraq also appears to exhibit a PGD^C allele frequency similar to the European frequency. The loss of the allele PGD^C in the Karaite Jews of Iraq clearly indicates the strong influence of isolation and genetic drift operating in this community.

The frequency of the PGD^C gene in the populations of Kuwait varies between 3.33 percent in the Kuwaiti Arabs (Sawhney, 1975) and 9.00 percent in the Ajman tribe (Khaled et al, 1981). With an average PGD^C frequency of 6.58 percent, the Kuwaiti population seems to show a much higher PGD^C frequency than that found in Iranians and much higher than the frequency in Europeans.

The PGD^C gene frequency in the populations of Saudi Arabia ranges from 3.02 percent in western Saudi Arabia (Saha et al, 1980) to 11.20 percent in the Saudi Arabian sample of Goedde et al (1979). With an average PGD^C frequency of 7.11 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit a much higher PGD^C frequency than that found in Iranians and much higher than the European frequency. Similar high frequencies of the PGD^C allele were observed in other Arab populations (Tills et al, 1971). High PGD^C frequencies were also found in several Negro populations (Mourant et al, 1976).

The frequency of the PGD^C allele in the populations of Afghanistan varies between 2.02 percent in the Uzbeks and

10.63 percent in the Hazaras (Goedde et al,1977). Up to now the high frequency of 10.63 percent found in the Hazaras of Afghanistan has only been surpassed by the results of a survey in Bhutan (Fich and Parr, 1967), Chinese (Shih and Hsia, 1969), some African tribes (Mourant et al, 1976) and Saudi Arabians (Goedde et al, 1979). Thus, apart from the Hazaras, with an average PGD^C frequency of 3.17 percent, the Afghan population appears to exhibit PGD^C allele frequencies more like those in Iran, Turkey, Iraq and other populations to the west.

Conclusion

The frequency of the PGC^C gene varies between 1 and 4 percent in European populations, and tends to be higher in the south.

In Africa frequencies of this gene are mostly around 6 percent but reach about 15 percent in the Ethiopians, the Beja of the Sudan and the south African Bantu.

They are on the whole higher still in Asia, varying from 7 percent in much of the Far East to 10 percent in Nepal and 23 percent in Bhutan (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arabs of Kuwait and Saudi Arabia with their higher frequencies, the frequency of the PGD^C allele appears to be similar to that found in Europeans. However, it seems to be lower than for some other Middle Eastern populations (Tills et al, 1971). High frequencies of the PGD^C gene, similar to those found in the Arabs of Kuwait and Saudi Arabia, were also observed in other Arabs(Tills et al, 1971), and several Negro populations (Mourant et al, 1976).

Table 5.3.6.I Red cell 6-phosphogluconate dehydrogenase (6PGD) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		PGD AA	PGD AC	PGD CC	PGD Rare (HA)	PGD^A	PGD^C	PGD^H		
1-Tavalesh,Astara	61	61	-	-	-	100.00	0.00	0.00	Kirk,R.L.,et al.	1977
2-Zoroastrians	66	66	-	-	-	100.00	0.00	0.00	Present study.	
3-Shahsavar,Rudsar,Rudbar, Rasht,Langarud,Lahijan, Bandar-Pahlavi	86	84	2	-	-	98.80	1.20	0.00	Kirk,R.L.,et al.	1977
4-Tehran	132	128	4	-	-	98.47	1.53	0.00	Farhud,D.D.,et al.	1973
5-Tehran	82	79	3	-	-	98.17	1.83	0.00	Present study.	
6-Southern Gorgan,Behshahr,Sari	53	51	2	-	-	98.10	1.90	0.00	Kirk,R.L.,et al.	1977
7-Armenians	180					98.00	2.00	0.00	Simhai,B.	1978
8-Jews	164					97.70	2.30	0.00	Simhai, B.	1974
9-Tehran	165	157	8	-	-	97.58	2.42	0.00	Sawhney,K.S.	1975
10-Moslems.Shiraz	322	304	18	-	-	97.20	2.80	0.00	Bowman,J.E., & Ronaghy, H.	1967
11-Esfahan	89	84	5	-	-	97.19	2.81	0.00	Sawhney,K.S.	1975

Table 5.3.6.I(Cont.) Red cell 6-phosphogluconate dehydrogenase(6PGD)phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		PGD AA	PGD AC	PGD CC	PGD Rare(HA)	PGD^A	PGD^C	PGD^H		
12-Kurds.Sanandaj	106	100	6	-	-	97.17	2.83	0.00	Lehmann,H.,et al.	1973
13-Babol,Shahi,Amol	64	60	4	-	-	96.90	3.10	0.00	Kirk,R.L.,et al.	1977
14-Northern Gorgan	51	47	4	-	-	96.10	3.90	0.00	Kirk,R.L.,et al.	1977
15-Kerman	301	278	22	1	-	96.01	3.99	0.00	Present study.	
16-Kurdish Jews	106	96	9	-	1	95.28	4.25	0.47	Tills,D.,et al.	1977
17-Kurdish Jews	94	85	8	-	1	95.21	4.26	0.53	Godber,Marilyn.J., et al.	1973
18-Kurds	182	165	17	-	-	95.33	4.67	0.00	Tills,D.,et al.	1971
19-Gonbad	155	139	16	-	-	94.80	5.20	0.00	Kirk,R.L.,et al.	1977
20-Turks.Shirvan,Khorasan	90	80	10	-	-	94.44	5.56	0.00	Present study.	
21-Kurds.Shirvan,Khorasan	70	61	9	-	-	93.57	6.43	0.00	Present study.	
22-Kurds.Baneh,Marivan	77	66	11	-	-	92.86	7.14	0.00	Lehmann,H.,et al.	1973

Table 5.3.6. III Red cell 6-phosphogluconate dehydrogenase (6PGD) phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		PGD AA	PGD AC	PGD CC	PGD Rare (HA)	PGD^A	PGD^C	PGD^H	
1-Turks	500	499	1	-	-	99.90	0.10	0.00	Altay,C.,et al. 1974
2-Turks	198	185	13	-	-	96.70	3.30	0.00	Brinkmann,B.,et al.1973
3-Kurds.						96.00	4.00	0.00	Richard,P. 1976

Table 5.3.6. IV Red cell 6-phosphogluconate dehydrogenase(6PGD)phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		PGD AA	PGD AC	PGD CC	PGD Rare(HA)	PGD^A	PGD^C	PGD^H	
1-Karaite Jews	72	72	-	-	-	100.00	0.00	0.00	Goldschmidt, Elizabeth.,et al. 1976
2-Kurdish Jews	27	26	1	-	-	98.15	1.85	0.00	Godber,Marilyn.J.,et al. 1973
3-Kurdish Jews.South east	50	47	3	-	-	97.00	3.00	0.00	Tills,D.,et al. 1977
4-Kurdish Jews.North west	61	56	5	-	-	95.90	4.10	0.00	Tills,D.,et al. 1977

Table 5.3.6. V Red cell 6-phosphogluconate dehydrogenase(6PGD) phenotypes and gene frequencies distribution in Kuwait

Population	Number Tested	Phenotypes				Gene frequencies			Authors
		PGD AA	PGD AC	PGD CC	PGD Rare(HA)	PGD^A	PGD^C	PGD^H	
1-Kuwaiti Arabs	150	140	10	-	-	96.67	3.33	0.00	Sawhney,K.S. 1975
2-Suluba tribe	52	47	4	1	-	94.00	6.00	0.00	Khaled,E.,et al. 1981
3-General population	86	72	14	-	-	92.00	8.00	0.00	Khaled,E.,et al. 1981
4-Ajman tribe	51	43	7	1	-	91.00	9.00	0.00	Khaled,E.,et al. 1981

Table 5.3.6. VI Red cell 6-Phosphogluconate dehydrogenase (6PGD) phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		PGD AA	PGD AC	PGD CC	PGD rare(HA)	PGD^A	PGD^C	PGD^H		
1-Western Saudi Arabia	149	141	7	1	-	96.98	3.02	0.00	Saha,N.,et al.	1980
2-Saudi Arabians	354	280	69	5	-	88.80	11.20	0.00	Goedde,H.W.,et al.	1979

Table 5.3.6. IX Red cell 6-Phosphogluconate dehydrogenase(6PGD) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes				Gene frequencies			Authors	
		PGD AA	PGD AC	PGD CC	PGD rare(HA)	PGD^A	PGD^C	PGD^H		
1-Uzbeks	124	119	5	-	-	97.98	2.02	0.00	Goedde,H.W.,et al.	1977
2-Pushtus	210	200	10	-	-	97.62	2.38	0.00	Goedde,H.W.,et al.	1977
3-Tajiks	310	293	17	-	-	97.26	2.74	0.00	Goedde,H.W.,et al.	1977
4-Pushtus	103	95	8	-	-	96.10	3.90	0.00	Papiha,S.S.,et al.	1977
5-Daris	177	159	17	-	1	94.90	4.80	0.30	Papiha,S.S.,et al.	1977
6-Hazaras	174	138	35	1	-	89.37	10.63	0.00	Goedde,H.W.,et al.	1977

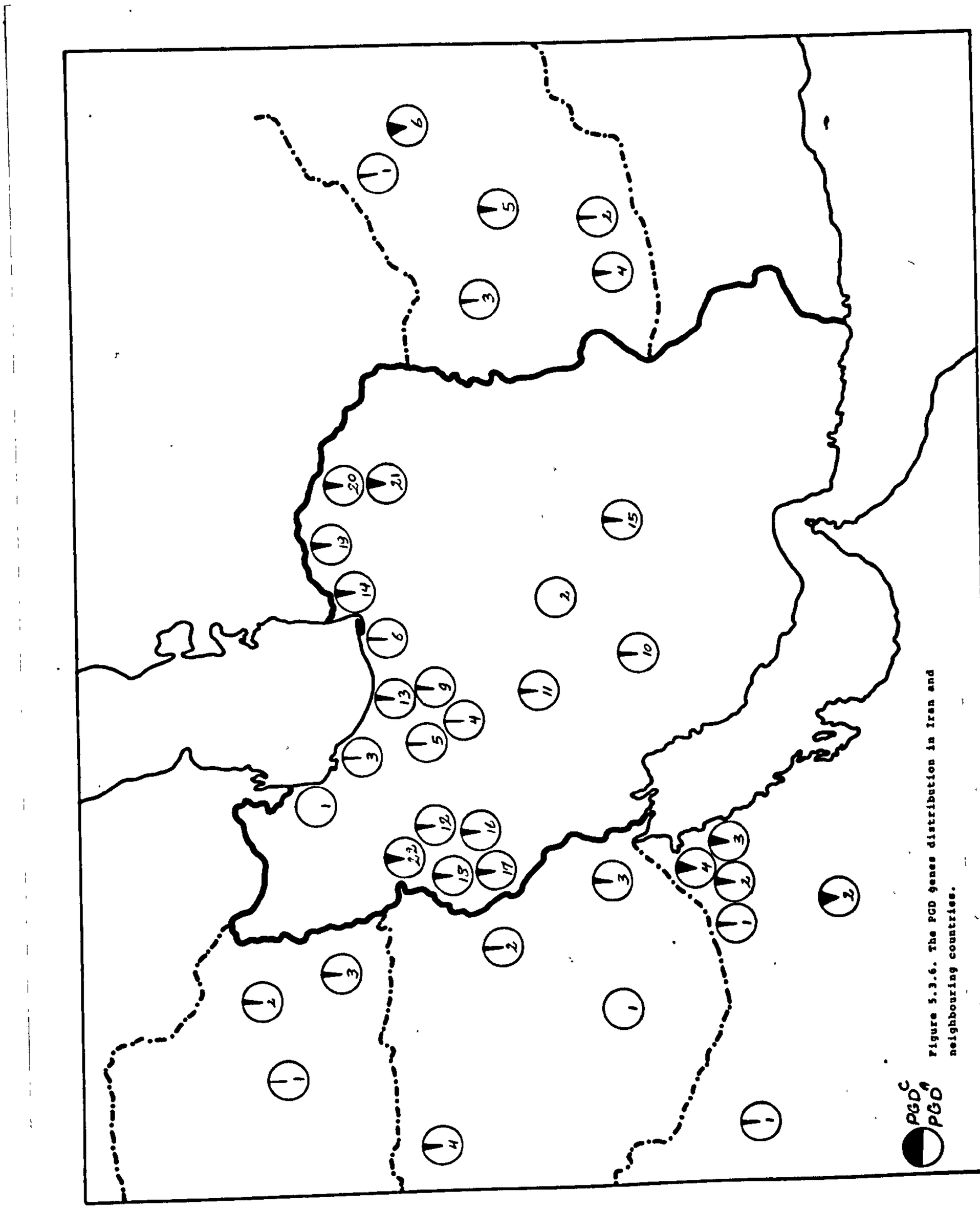


Figure 5.3.6. The PGD genes distribution in Iran and neighbouring countries.

5.3.7. The glucose-6-phosphate dehydrogenase(G6PD) system

Data on the distribution of glucose-6-phosphate dehydrogenase deficiency in Iranian and neighbouring populations are set out in Tables 5.3.7.

The frequency of the Gd^- gene ranges from zero to 58.16 percent in Iranians, being lowest in the Zoroastrians(Bowman and Walker, 1961) and highest in the Kurdish Jews of western Iran (Sheba et al, 1961).

A number of investigators have reported deficiency of G6PD in Iranians. Among them is a series of studies by Bowman and his collaborators of Moslems, Zoroastrians, Armenians and several tribal populations (summarized by Bowman and Ronaghy,1967). For male Moslems in Shiraz they found approximately 9 percent deficiency, a figure similar to that found for Moslems in other areas. Beaconsfield et al (1967) also surveyed the frequency of G6PD deficiency in various populations in Iran. In Yazd and environs, a desert plateau free from malaria, G6PD deficiency was reported to be 1 percent among Zoroastrians and 2.50 percent among Moslems, whereas among Moslems in the more malarial parts of the Caspian Littoral it was 25 percent. High values were also given for ethnic groups in and around the city of Kermanshah where malaria is common, while lower values (9 percent fro Moslems in Esfahan) were found where malaria has been intermittent. Kirk et al (1977) studied the frequency of G6PD deficiency for six areas in the Caspian Littoral. The male G6PD deficiency frequencies range from 1.20 to 17.90 percent, and there is a marked difference in frequency between the low values in Gonbad (1.20 percent) and Tavalesh, Astara (6.50 percent) and the other four areas from northern Gorgan to Bandar-Pahlavi where values are three to

four times higher. These latter areas comprise what used to be the highly malarial parts of the Caspian Littoral or include persons who have moved from such areas in the recent past, and corresponds with that part of the Caspian area included in Beaconsfield's (1967) study referred to above. The frequencies reported by Kirk et al (1977) therefore are in good agreement with those from previous work and add further support to the relationship between the frequency of G6PD deficiency and the level of malaria endemicity in the recent past. The most striking finding is the very high frequency of G6PD deficiency in the Kurdish Jews of western Iran on the one hand and the low frequency in the non-Jewish Kurds of the same region on the other. Thus, the Iranian Kurds carry the gene, but with a much lower frequency than the Kurdish Jews who live among them. It is difficult to account in terms of exposure to malaria for the marked difference between two populations living in such close proximity.

Though quite high frequencies are found in the indigenous populations in many parts of Iran, since one of the main determining conditions is probably a selective advantage of carriers of the gene, in the presence of endemic falciparum malaria, the very much higher frequency in Kurdish Jews than in Kurds poses a special problem.

It is suggested that the deficiency is almost certainly, like Haemoglobin S, protective against falciparum malaria. Malaria does not appear to have been endemic in the mountains of Kurdistan. However, the extreme contrast between the frequencies of the deficiency among the Kurdish Jews on the one hand and among the non-Jewish Kurds on the other makes it almost certain that the very high frequencies in the Kurdish Jews and

other oriental Jews is due mainly either to their ancestry or their way of life, rather than to any natural selection resulting from a common malarial environment.

We must therefore look for a cause for the high frequency in the Jews in some circumstances peculiar to their own ancestry or mode of life. The gene itself may have accompained the Jewish community, which presumably migrated a very long time ago from Israel at the time of Assyrian deportations. It is unlikely, however, that it is solely through inbreeding that the high frequency found in this community has persisted and even increased in the long subsequent period, in the absence of some special environmental influence favouring this normaly slightly deleterious gene. It seems unlikely also that there has been any substantial amount of immigration in recent centuries.

One is led to look for some cultural peculiarity resembling in a general way that found by Undevia (1969) in the parsis of Bombay. These have a significantly higher incidence of G6PD deficiency than their Hindu neighbours, and he shows that this was associated with a former much higher incidence of malaria in the Parsi community. Public health investigations had shown that this was due to the open bathing tanks attached to Parsi dwellings which, though favouring hygiene in other respcts, also enabled malarial mosquitos to breed.

There is, however, no new evidence as to why it has operated strongly on Jews but hardly at all on Kurds. The only suggestion is still that of Godber et al (1973): " some Jewish hygienic practices, as with the Parsis of Bombay, may in some way have promoted the breeding of malaria carrying mosquitoes near their dwellings."

Regarding neighbouring populations, the frequency of the Gd^- gene in the populations of the Caucasus ranges from zero in the Assyrians to 62.30 percent in the Jews (Voronov,1981). It can be seen from the table that in the Caucasus, as in Iran, the non-Jewish populations exhibit lower frequencies of the deficiency compared with the high ones in the Jews.

It should be pointed out that two neighbouring populations of different ethnic origin differ markedly in the frequency of abnormality. This observation refers to the mountain Jews of the Caucasus among whom the deficiency is frequent, and the Circassian Moslems living in the same region among whom it is absent.

If we accept the hypothesis of balanced polymorphism produced by the protective action of the enzyme deficiency against the lethal effect of falciparum malaria, then the findings of significantly different frequencies in neighbouring communities suggest a difference of their ethnic origin.

In a community where the abnormality is present, it could be concentrated by the protective action against a lethal environmental factor, whereas in a community originally free from the enzyme deficiency it do not arise throughout centuries of living in malarial areas.

Thus, these findings support the view, that G6PD deficiency may constitute a significant ethnological marker for the study of the origins of neighbouring communities.

The frequency of the Gd^- gene in the populations of Turkey varies between zero in the Armenians and 11.43 percent in the Eti-Turks (Say et al, 1965). On the whole, it seems that the overall incidence of G6PD deficiency in Turkish populations is low.

The frequency of the Gd^- gene in the populations of Iraq ranges from zero in the Karaite Jews (Goldschmidt et al,1976) to 7.63 percent in the Kurdish Jews (Szeinberg, 1963). Again, as in Iran, the Iraqi Kurdish Jews exhibit very high frequencies of the enzyme deficiency. The zero frequency of the enzyme deficiency in the Karaite Jews of Iraq is somewhat surprising since non-Karaite Iraqi Jews have a high prevalence of G6PD deficiency; the gene frequency is approximately 25 percent (Sheba et al, 1961; Szeinberg, 1963). This observation confirms the isolation of the Karaite from other Jews in Iraq.

A Gd^- gene frequency of 18.96 percent is reported by Shaker et al (1966) in the Kuwaiti population. This finding indicates that the gene for G6PD deficiency is quite prevalent in Kuwait.

The state of Kuwait, an arid land, is known to be free from malaria, although it is surrounded by several countries where malaria is highly endemic, especially Iraq and Saudi Arabia. This finding of a high frequency of G6PD deficiency would seem to contradict the malaria protection theory of Allison (1963), who in fact stated that the only convincing argument against his hypothesis would be " the finding of high frequencies of abnormal haemoglobin or G6PD deficiency genes among populations known to have lived for long periods in malaria-free environments", and that " no such populations have yet been found to exist". The discrepancy, however, could be explained by the intermingling of the racial groups in the Gulf area over the ages and the fact that the present day inhabitants of Kuwait originally migrated, early in the eighteenth century, from neighbouring Saudi Arabia, where malaria is

heavily endemic (Dickson, 1956), and later from other neighbouring countries, mainly Iraq and Iran, where malaria is also endemic.

The frequency of the Gd^- gene in the populations of Saudi Arabia ranges from 2.94 percent in the Sunnis to 65.38 percent in the Shias (Gelpi, 1965). These findings indicate that G6PD deficiency is significantly elevated in Saudi Arabians, as it is in other Arab populations, the national average being 31.67 percent.

A relatively lower frequency (10 percent) of the enzyme deficiency is reported by Kamel et al (1980) in the Abu-Dhabians of the United Arab Emirates.

The Gd^- gene frequency in the populations of Pakistan varies between 1.42 percent in the Bengalis (Ronald et al, 1968) and 7.89 percent in the Pathans (Stern et al, 1968). On the whole, with a national average of 3.30 percent, the overall incidence of the enzyme deficiency seems to be low in the Pakistani population.

Similar low frequencies ranging from 0.97 percent in the Pushtus (Papiha et al, 1977) to 10.34 percent in the Jews (Sheba et al, 1961) characterize the Afghan population, the national average being 4.9 percent.

Conclusion

Glucose-6-phosphate dehydrogenas deficiency, especially that of the B^- type can, particularly through favism, lead to serious illness with appreciable mortality. The genetical problem which it presents is thus similar to that of the abnormal haemoglobins and the thalassaemias. We need to explain how a gene with deleterious effects can nevertheless maintain very high frequencies in certain populations. In certain areas,

and especially in the Mediterranean region, the distribution of the gene shows a considerable degree of correspondence with that of present or recent malignant tertian malaria.There is also some direct evidence for protection of the individual against the effects of malarial infection(Allison and Clyde, 1961; Bienzle et al, 1972; Harris and Gilles, 1961). The deficiency is found in a belt extending from the Mediterranean area through south west Asia and India to south East Asia including Indonesia(Mourant et al, 1976).

Though quite high frequencies are found in the indigenous populations in many parts of Iran where malaria was formerly endemic, the highest frequencies of deficiency yet recorded are in the Kurdish Jews of Iran and Iraq. The Iranian Kurds carry the gene, but with a much lower frequency than the Kurdish Jews who live among them. It is difficult to account in terms of exposure to malaria for the marked difference between two populations living in such close proximity. Moreover, malaria does not appear to have been endemic in the mountainous area of Kurdistan.

However, the extreme contrast between the frequencies of the deficiency among the Kurdish Jews on the one hand and among the non-Jewish Kurds on the other makes it almost certain that the very high frequencies in the Kurdish Jews and other oriental Jews is due mainly either to their ancestry or their way of life, rather than to any natural selection resulting from a common malarial environment.There is, however, no new evidence as to why it has operated strongly on Jews but hardly at all on Kurds. The only suggestion is still that of Godber et al (1973):" some Jewish hygienic practices, as with the Parsis of Bombay(Undevia, 1969),may in some way have

promoted the breedings of malaria carrying mosquitoes near their dwellings."

The only other population known to have G6PD deficiency frequencies comparable with those of the Kurdish Jews consists of the indigenous Shia Moslems of the formerly malarial eastern Oases of Saudi Arabia. The more recent immigrants of the Sunni Moslem sect appear to have considerably lower frequencies of G6PD deficiency.

Finally, apart from the Kurdish Jews of western Iran and Iraq with their extremely high frequencies of G6PD deficiency and also the Shia Moslem Arabs of Saudi Arabia with high frequencies, comparable with those of the Kurdish Jews, there are substantial frequencies of this condition in most other populations in Iran and in neighbouring countries both in the indigenous populations and in Jews, but in general the condition is commoner in Jews.

Table 5.3.7. I Red cell Glucose-6-Phosphate dehydrogenase(G6PD) deficiency distribution in Iran (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors
1-Zoroastrians.Yazd	146	146	-	0.00	Bowman,J.E.,& Walker,D. 1961 G.
2-Armenians.Esfahan	158	157	1	0.63	Bowman,J.E.,& Walker,D. 1961 G.
3-Zoroastrians.Yazd				1.00	Beaconsfield,P.,et al. 1967
4-Gonbad	82	81	1	1.20	Kirk,R.L.,et al. 1977
5-Yazd				2.50	Beaconsfield,P.,et al. 1967
6-Kurds.Sanandaj	106	103	3	2.83	Lehmann,H., et al. 1973
7-Kurds.Baneh,Marivan	77	72	5	6.49	Lehmann,H., et al. 1973
8-Tavalesh,Astara	31	29	2	6.50	Kirk,R.L., et al. 1977
9-Chah Bahar,Jask	142	132	10	7.04	Hedayat,S.,et al. 1969
10-Mashhad				7.50	Beaconsfield,P.,et al. 1967
11-Kazeroun				7.50	Beaconsfield,P.,et al. 1967
12-Moslems.Fars	984	906	78	7.93	Bowman,J.E.,& Walker,D. 1961 G.
13-Esfahan				9.00	Beaconsfield,P.,et al. 1967
14-Shiraz				9.00	Beaconsfield,P.,et al. 1967

Table 5.3.7.I (Cont.) Red cell Glucose-6-Phosphate dehydrogenase (G6PD)deficiency distribution in Iran (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors	
15-Moslems.Shiraz	221	201	20	9.05	Bowman,J.E., & Ronaghy, H.	1967
16-Moslems.Tehran	557	502	55	9.87	Hedayat,S.,et al.	1969
17-Jews.Esfahan,Shiraz, Tehran	370	330	40	10.81	Szeinberg,A.	1963
18-Ghashghais	133	118	15	11.28	Bowman,J.E., & Walker, D.G.	1961
19-Jews	139			12.00	Simhai,B.	1974
20-Jews.Tehran	108	95	13	12.04	Hedayat,S.,et al.	1969
21-Babol,Shahi,Amol	32	28	4	12.50	Kirk,R.L.,et al.	1977
22-Basseris.Fars	83	72	11	13.25	Bowmann,J.E.,& Walker, D.G.	1961
23-Jews.Esfahan				14.00	Beaconsfield,P.,et al.	1967
24-Northern Gorgan	21	18	3	14.30	Kirk,R.L.,et al.	1977
25-Armenians.Tehran	102	87	15	14.71	Hedayat,S.,et al.	1969
26-Jews	557	473	84	15.08	Sheba,C.,et al.	1961

Table 5.3.7.I (Cont.) Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Iran (Males only).

Population	Number Tested	Normal	Deficient	Gd^-	Authors	
27-Shahsavar,Rudsar,Rud, bar,Rasht,Langarud, Lahijan,Bandar-Pahlavi	43	36	7	16.30	Kirk,R.L.,et al.	1977
28-Southern Gorgan, Behshahr,Sari	28	23	5	17.90	Kirk,R.L.,et al.	1977
29-Kermanshah				19.00	Beaconsfield,P.,et al.	1967
30-Mamassanis.Fars	91	73	18	19.78	Bowman,J.E., & Walker,G.	D.1961
31-Kurds.Kermanshah				25.00	Beaconsfield,P.,et al.	1967
32-Caspian Littoral area				25.00	Beaconsfield,P.,et al.	1967
33-Jews.Kermanshah				29.00	Beaconsfield,P.,et al.	1967
34-Kurdish Jews	63	40	23	36.51	Tills,D.,et al.	1977
35-Kurdish Jews	57	35	22	38.60	Godber,Marilyn.J., et al.	1973
36-Kurdish Jews,Kermanshah,Sanandaj	45	25	20	44.44	Szeinberg,A.	1963

Table 5.3.7.I (Cont.) Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Iran (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors	
37-Kurdish Jews	196	82	114	58.16	Sheba,C.,et al.	1961

Table 5.3.7.b. I Red cell Glucose-6-phosphate dehydrogenase(G6PD) types in Iran (Males onlY)

Population	Number Tested	Phenotypes				Gene frequencies				Authors	
		Normal		Deficient							
		A+	B+	A-	B-	Gd^{A+}	Gd^{B+}	Gd^{A-}	Gd^{B-}		
1-Jurds.Sanandaj	106	103		3		97.17		2.83		Lehmann,H.,et al.	1973
2-Kurds.Baneh, Marivan	77	72		5		93.51		6.49		Lehmann,H.,et al.	1973
3-Kurdish Jews	57	35		22		61.40		38.60		Godber,Marilyn J., et al.	1973

Table 5.3.7. II Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in the Caucasus (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors	
1-Assyrians	289			0.00	Voronov,A.A.	1981
2-Circassians	57			0.00	Sheba,C.,et al.	1961
3-Ingushes	210			0.00	Voronov, A.A.	1981
4-Imeritins.Georgian	421			0.20	Voronov. A.A.	1981
5-Megrels.Georgian	817			0.30	Voronov, A.A.	1981
6-Gurians.Georgian	625			0.30	Voronov, A.A.	1981
7-Cahetins.Georgian	212			0.50	Voronov, A.A.	1981
8-Armenians	5161			0.90	Voronov, A.A.	1981
9-Chechens	691			1.30	Voronov, A.A.	1981
10-Talyshes.Azerbaijan	237			8.00	Voronov, A.A.	1981
11-Azerbaijanians	18720			9.90	Voronov, A.A.	1981
12-Avars.Daghestan	100			19.00	Voronov, A.A.	1981
13-Udins.Azerbaijan	75			21.40	Voronov, A.A.	1981
14-Jews	25	18	7	28.00	Sheba,C.,et al.	1961
15-Mountain Jews	90			62.30	Voronov, A.A.	1981

Table 5.3.7.III Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Turkey (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors	
1-Armenians.Istanbul	44	44	-	0.00	Say,B.,et al.	1965
2-Jews.Istanbul	29	29	-	0.00	Say,B.,et al.	1965
3-Istanbul	37	37	-	0.00	Say,B.,et al.	1965
4-Asia Minor.Rize. Kurdish speakers	109	109	-	0.00	Say,B.,et al.	1965
5-Asia Minor,Ankara.Neonates	1000	995	5	0.50	Say,B.,et al.	1965
6-Asia Minor,Izmir	212	210	2	0.94	Say,B.,et al.	1965
7-Asia Minor,Diarbakir. Kurdish speakers	208	204	4	1.92	Say,B.,et al.	1965
8-Jews	256	251	5	1.95	Sheba,C.,et al.	1961
9-Jews	93	91	2	2.15	Sheba,C.,et al.	1961
10-Turks abroad	71	69	2	2.82	Kattamis,C.A.,et al.	1969
11-Kurds				4.00	Richard,P.	1976
12-Turks. Antalya	73			5.40	Aksoy,M.,et al.	1980
13-Eti-Turks	637	593	44	6.50	Altay,C.,et al.	1978

Table 5.3.7. III (Cont.) Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Turkey (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors	
14-Eti-Turks. Adana,Tarsus	105	93	12	11.43	Say,B.,et al.	1965

Table 5.3.7. IV Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Iraq (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors	
1-Karaite Jews	29	29	-	0.00	Goldschmidt,E.,et al.	1976
2-Moslems	305	267	38	12.40	Hamamy,H.A.& Saeed,T. Kh.	1981
3-Jews.Baghdad	286	216	70	24.48	Szeinberg, A.	1963
4-Jews	902	678	224	24.83	Sheba,C.,et al.	1961
5-Kurdish Jews	59	38	21	35.59	Szeinberg, A.	1963
6-Kurdish Jews	14	6	8	57.14	Godber,Marilyn.J., et al.	1973
7-Kurdish Jews.South east	19	8	11	57.89	Tills,D.,et al.	1977
8-Kurdish Jews.North west	36	11	25	69.44	Tills,D.,et al.	1977
9-Kurdish Jews	126	37	89	70.63	Szeinberg, A.	1963

Table 5.3.7. V Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Kuwait (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors
1-Kuwaitis	211	171	40	18.96	Shaker, Yehia.,et al. 1966

Table 5.3.7. VI Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Saudi Arabia (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors	
1-Sunnis .Eastern Provinces (except Al Qatif & Al Hasa)	34	33	1	.2.94	Gelpi,A.P.	1965
2-Western Provinces	97	93	4	4.12	Gelpi,A.P.	1965
3-Shias & Sunnis. Al .Hasa	64	61	3	4.69	Gelpi,A.P.	1965
4-Shias. Al Hasa,Qarah	104	79	25	24.04	Gelpi,A.P.	1965
5-Shias.Al Qatif	111	73	38	34.23	Gelpi,A.P.	1965
6-Shias.Al Hasa, Mansurah	92	43	49	53.26	Gelpi,A.P.	1965
7-Shias. Al Qatif,Safwah	51	18	33	64.71	Gelpi,A.P.	1965
8-Shias. Al Qatif, Al Ajman	104	36	68	65.38	Gelpi,A.P.	1965

Table 5.3.7. VII Red cell Glucose-6-Phosphate dehydrogenase(G6PD) deficiency distribution in the United Arab Emirates (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors
1-Abu-Dhabians	100	90	10	10.00	Kamel, K., et al. 1980

Table 5.3.7. VIII Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Pakistan (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors	
1-Bengalis.West	141	139	2	1.42	Ronald,A.R.,et al.	1968
2-Pathans.West	60	59	1	1.67	McCurdy,P.R., et al.	1970
3-Former residents of India	45	44	1	2.22	Ronald,A.R.,et al.	1968
4-Punjabis.West	133	130	3	2.25	McCurdy,P.R.,et al.	1970
5-Punjabis.West	185	180	5	2.70	Ronald,A.R.,et al.	1968
6-Other than Pathans and Punjabis.West	28	27	1	3.57	McCurdy,P.R.,et al.	1970
7-Pathans.West	85	81	4	4.71	Ronald,A.R.,et al.	1968
8-Pathans.West	114	105	9	7.89	Stern,M.A., et al.	1968

Table 5.3.7.IX Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Afghanistan (Males only)

Population	Number Tested	Normal	Deficient	Gd^-	Authors
1-Pushtus	103	102	1	0.97	Papiha,S.S.,et al. 1977
2-Daris	177	171	6	3.39	Papiha,S.S.,et al. 1977
3-Jews	29	26	3	10.34	Sheba,C.,et al. 1961

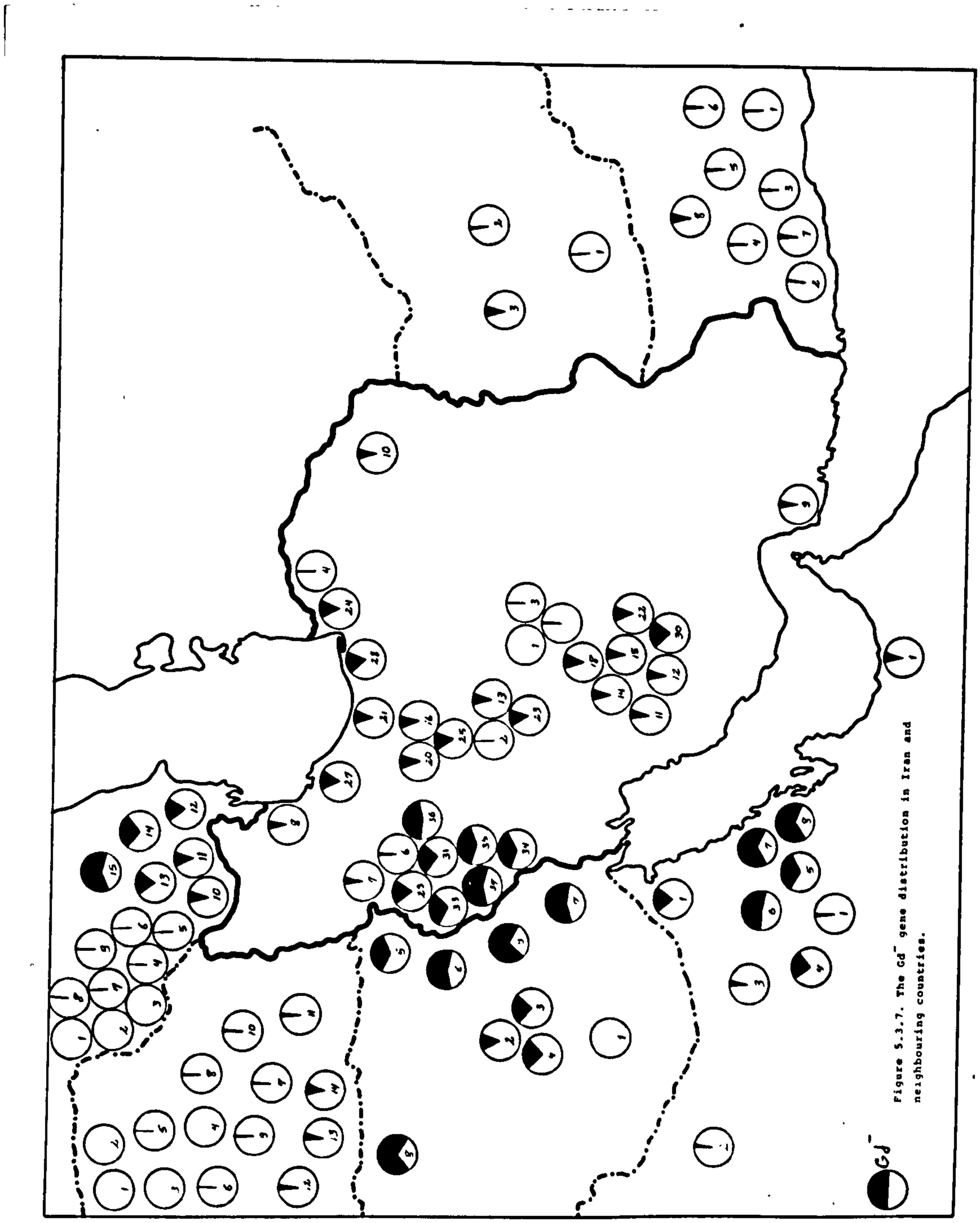


Figure 5.3.7. The Gd^- gene distribution in Iran and neighbouring countries.

5.3.8 The lactate dehydrogenase (LDH) system

The distribution of lactate dehydrogenase types in Iranian and neighbouring populations is presented in Tables 5.3.8.

With the exception of one Calcutta 1 type in the 463 specimens from the Caspian sea area of Iran (Kirk et al,1977), all the samples from Iran and neighbouring countries showed the common LDH type.

Conclusion

Variants of Lactate dehydrogenase are all very rare, occurring with a frequency of the order of 1 in 1000 in most populations. Type Calcutta 1 has a frequency somewhat over 1 percent in various Indian populations(Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of one Calcutta 1 type found in the Caspian sea area of Iran, the only type was found to be the Normal LDH type.

Table 5.3.8.I. Red cell Lactate dehydrogenase (LDH) types in Iran

Population	Number Tested	Phenotypes		%Variant	Authors
		LDH.N(Normal)	LDH Variant		
1-Kurds.Sanandaj	105	105	-	-	Lehmann,H.,et al. 1973
2-Tehran	132	132	-	-	Farhud,D.D.,& Walter, H.1973
3-Tehran	165	165	-	-	Sawhney,K.S. 1975
4-Esfahan	89	89	-	-	Sawhney,K.S. 1975
5-Caspian Sea area	463	462	1Calcutta 1	0.22	Kirk,R.L.,et al. 1977

Table 5.3.8. V. Red cell Lactate dehydrogenase (LDH) types in Kuwait

Population	Number Tested	Phenotypes		%Variant	Authors	
		LDH.N(Normal)	LDH Variant			
1-Kuwaiti Arabs	150	150	-	-	Sawhney,K.S.	1975
2-General population	89	89	-	-	Khaled,E.,et al.	1981
3-Suluba tribe	52	52	-	-	Khaled,E.,et al.	1981
4-Ajman tribe	52	52	-	-	Khaled,E.,et al.	1981

Table 5.3.8. VI Red cell Lactate dehydrogenase (LDH) types in Saudi Arabia

Population	Number Tested	Phenotypes		%Variant	Authors
		LDH.N(Normal)	LDH Variant		
1-Western Saudi Arabia	245	245	-	-	Saha,N.,et al. 1980

Table 5.3.8. IX Red cell Lactate dehydrogenase (LDH) types in Afghanistan

Population	Number Tested	Phenotypes		% Variant	Authors
		LDH.N (Normal)	LDH Variant		
1-Pushtus	103	103	-	-	Papiha,S.S.,et al. 1977
2-Daris	177	177	-	-	Papiha,S.S.,et al. 1977

5.3.9. The malate dehydrogenase (MDH) system

The distribution of malate dehydrogenase types in Iranian and neighbouring populations is shown in Tables 5.3.9.

With the exception of one MDH5-1 type in the 463 specimens from the Caspian sea area of Iran (Kirk et al, 1977),all the samples from Iran and neighbouring countries showed only the common MDH1-1 phenotype.

The MDH variant detected in a single individual in the Caspian sea sample was described by Blake (1977). It does not correspond with any previously reported MDH variant and the Caspian phenotype has been designated s-MDH5-1.

Conclusion

Many thousands of persons from Europe, the Near East and Asia have shown only the common MDH1-1 homozygous type (Mourant et al, 1976).

In Iranian and neighbouring populations, as in Europeans and in other Asiatic populations, the only type was found to be the normal MDH1-1 phenotype.

Table 5.3.9.I Red cell Malate dehydrogenase (MDH) types in Iran

Population	Number Tested	Phenotypes MDH.N(Normal) 1-1	Phenotypes MDH Variant	% Variant	Authors	
1-Kurds	182	182	-	-	Leakey,T.E.B.,et al.	1972
2-Tehran	165	165	-	-	Sawhney,K.S.	1975
3-Esfahan	89	89	-	-	Sawhney,K.S.	1975
4-Caspian Sea area	463	462	1s-MDH5-1	0.22	Kirk,R.L.,et al.	1977

Table 5.3.9. V. Red cell Malate dehydrogenase(MDH) types in Kuwait

Population	Number Tested	Phenotypes		%Variant	Authors
		MDH.N(Normal) 1-1	MDH Variant		
1-Kuwaiti Arabs	150	150	-	-	Sawhney,K.S. 1975

Table 5.3.9. VI Red cell Malate dehydrogenase (MDH) types in Saudi Arabia

Population	Number Tested	Phenotypes		%Variant	Authors
		MDH.N (Normal) 1-1	MDH Variant		
1- Western Saudi Arabia	93	93	-	-	Saha,N.,et al. 1980

Table 5.3.9. IX Red cell Malate dehydrogenase (MDH) types in Afghanistan

Population	Number Tested	Phenotypes		%Variant	Authors
		MDH.N(Normal) 1-1	MDH Variant		
1-Pushtus	103	103	-	-	Papiha,S.S.,et al. 1977
2-Daris	177	177	-	-	Papiha,S.S.,et al. 1977

5.3.10. The Phosphohexose isomerase (PHI) system

The distribution of phosphohexose isomerase types and respective gene frequencies in Iranian populations is presented in Table 5.3.10.

Information on the distribution of the PHI genes in neighbouring populations is lacking.

The Iranian sample consists of four population groups. Comparing the four groups, Farhud et al (1973) and Sawhney (1975) found no PHI3-1 phenotype in the 125 Tehrani and the 88 Esfahani Iranians studied by them, but Sawhney (1975) and Kirk et al (1977) found two PHI3-1 phenotypes in the 165 Tehrani sample and seven in the 463 sample from the Caspian sea area, respectively. Detter et al (1968) found that the Asiatic populations exhibit an appreciable frequency of the allele PHI^3.

PHI^3 frequencies of 0.61 and 0.76 percent in Iranians confirm this tendency. Also, these frequencies are comparable with the values given by Blake et al (1971) for the North Indians.

Conclusion

The PHI system is of potential interest because the PHI^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^3 allele. This finding suggests the importance of conducting further studies in Iranian and neighbouring populations.

Table 5.3.10. I Red cell Phosphohexose isomerase (PHI) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		PHI 1-1	PHI 3-1	PHI^1	PHI^3		
1-Tehran	125	125	-	100.00	0.00	Farhud,D.D.,et al.	1973
2-Esfahan	88	88	-	100.00	0.00	Sawhney,K.S.	1975
3-Tehran	165	163	2	99.39	0.61	Sawhney,K.S.	1975
4-Caspian Sea area	463	456	7	99.24	0.76	Kirk,R.L.,et al.	1977

5.3.11. The glutamic pyruvic transaminase (GPT) system

The distribution of GPT types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.3.11.

There are a few reports on GPT studies in Iranian and neighbouring populations.

The only report on Iranians is that of Kirk et al (1977) who studied six areas in the Caspian Littoral of Iran. The frequency of the GPT^2 allele ranges from 38.50 to 50.00 percent in northern Iran, being lowest in Tavalesh, Astara and highest in northern Gorgan. With an average GPT^2 frequency of 42.62 percent, the population of northern Iran appears to exhibit a lower GPT^2 frequency than that of 47.67 percent in Europeans (Kompf and Ritter, 1979).

Regarding neighbouring areas, the GPT^2 frequency of 45.60 percent in the Turks of Turkey (Brinkmann et al,1973) is higher than the average for northern Iran but lower than the European frequency.

The GPT^2 frequency of 41.10 percent in the Iraqi Jews is slightly lower than that in Iranians and lower than the frequency in Europeans.

The frequency of the GPT^2 gene being 38.20 percent in Saudi Arabians (Goedde et al, 1979) is also lower than that in Iranians and much lower than the European frequency.

The GPT^2 allele frequency in the populations of Afghanistan ranges from 50.41 percent in the Uzbeks to 55.81 percent in the Tajiks (Goedde et al, 1977). With an average GPT^2 frequency of 53.05 percent, the Afghan population appears to exhibit a much higher GPT^2 frequency than that found in Iranians and even higher than the frequency in Europeans.

Conclusion

The European populations frequencies of the GPT^2 gene lie between 46.10 and 49.66 percent (Kompf and Ritter,1979). Frequencies of the GPT^2 allele, ranging from 55.15 to 64.63 percent, are higher in the Indian region than in Europe (Blake, 1979).

In Chinese the GPT^2 gene frequencies are around 50 percent (Chen et al, 1973; Blake, 1976).

In south African Bantus it is only 12.71 percent(Blake,1976).

In Iranian and neighbouring populations, with the exception of the Afghans with their higher values, frequencies of the GPT^2 gene appear to be lower than those in Europeans.

Table 5.3.11. I. Red cell Glutamic Piruvic transaminase(GPT) phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		GPT 1-1	GPT 2-1	GPT 2-2	GPT^1	GPT^2		
1-Tavalesh,Astara	61	28	19	14	61.50	38.50	Kirk,R.L.,et al.	1977
2-Babol,Shahi,Amol	64	23	32	9	60.90	39.10	Kirk,R.L.,et al.	1977
3-Southern Gorgan Behshahr,Sari	53	19	26	8	60.40	39.60	Kirk,R.L.,et al.	1977
4-Shahsavar,Rudsar,Rudbar, Langarud,Lahijan,Bandar-Pahlavi	86	31	37	18	57.60	42.40	Kirk,R.L.,et al.	1977
5-Gonbad	155	47	73	35	53.90	46.10	Kirk,R.L.,et al.	1977
6-Northern Gorgan	44	9	26	9	50.00	50.00	Kirk,R.L.,et al.	1977

Table 5.3.11.III Red cell Glutamic Piruvic transaminase (GPT) phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		GPT 1-1	GPT 2-1	GPT 2-2	GPT^1	GPT^2	
1-Turks	213				54.40	45.60	Brinkmann,B.,et al. 1973

Table 5.3.11. IV Red cell Glutamic Piruvic transaminase (GPT) phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		GPT 1-1	GPT 2-1	GPT 2-2	GPT^1	GPT^2	
1- Jews	192				58.90	41.10	Lahav,M.,et al. 1972

Table 5.3.11. VI Red cell Glutamic Piruvic transaminase (GPT)phenotypes and gene frequencies distribution in Saudi Arabia

Population	Number Tested	Phenotypes			Gene frequencies		Authors
		GPT 1-1	GPT 2-1	GPT 2-2	GPT^1	GPT^2	
1-Saudi Arabians	359	123	198	38	61.80	38.20	Goedde,H.W.,et al. 1979

Table 5.3.11. IX Red cell Glutamic Piruvic transaminase(GPT) phenotypes and gene frequencies distribution in Afghanistan

Population	Number Tested	Phenotypes					Gene frequencies				Authors
		GPT 1-1	GPT 2-1	GPT 2-2	GPT 3-2	GPT 6-2	GPT^1	GPT^2	GPT^3	GPT^6	
1-Uzbeks	124	29	65	30	-	-	49.59	50.41	0.00	0.00	Goedde,H.W.,et al.1977
2-Hazaras	174	37	94	42	-	1	48.27	51.45	0.00	0.28	Goedde,H.W.,et al.1977
3-Pushtus	210	44	103	63	-	-	45.47	54.53	0.00	0.00	Goedde,H.W.,et al.1977
4-Tajiks	310	59	155	95	1	-	44.03	55.81	0.16	0.00	Goedde,H.W.,et al.1977

5.3.12. Other red cell enzyme systems

In this part, data on some other red cell enzyme systems are presented. Since data available on these systems are not representative for the region, no discussion will be given.

Table 5.3.12. a. I Red cell phosphoglucomutase Locus 2(PGM_2) in Iran

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		PGM_2 1-1	PGM_2 9-1	PGM_2^1	PGM_2^9	
Caspian Sea area	463	459	4	99.57	0.43	Kirk, R.L., et al. 1977

Table 5.3.12.a.IV. Red cell phosphoglucomutase Locus 2(PGM_2) in Iraq

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		PGM_2 1-1	PGM_2 rare	PGM_2^1	PGM_2^{rare}	
Jews	69	69	-	100.00	0.00	Hopkinson,D.A.,et al. 1966

Table 5.3.12.a. IX Red cell phosphoglucomutase Locus 2(PGM_2) in Afghanistan

Population	Number tested	Phenotypes		Gene frequencies		Authors	
		$PGM_{1\text{-}1}^{2}$	PGM_2^{rare}	PGM_2^1	PGM_2^{rare}		
1-Daris	178	178	-	100.00	0.00	Papiha,S.S.,et al.	1977
2-Pushtus	103	103	-	100.00	0.00	Papiha,S.S.,et al.	1977

Table 5.3.12.b.I. Red cell peptidase A (PeP A) in Iran

Population	Number Tested	Phenotypes			Gene frequencies		Authors	
		PePA 1-1	PePA 2-1	PePA 2-2	$PePA^1$	$PePA^2$		
Caspian Sea area	463	459	3	1	99.46	0.54	Kirk,R.L., et al.	1977

Table 5.3.12.C.I. Red cell peptidase B(PePB) in Iran

Population	Number Tested	Phenotypes			Gene frequencies			Authors	
		PePB 1-1	PePB 2-1	PePB 3-1	$PePB^1$	$PePB^2$	PeP^3		
Caspian Sea area	463	459	2	2	99.56	0.22	0.22	Kirk,R.L.,et al.	1977

Table 5.3.12.d.I. Red cell peptidase C (PePC) in Iran

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		PePC 1-1	PePC 3-1	$PePC^1$	$PePC^3$		
Caspian Sea area	463	462	1	99.89	0.11	Kirk,R.L.,et al.	1977

Table 5.3.12.e.I. Red cell peptidase D (PePD) in Iran

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		PePD 1-1	PePD 3-1	$PePD^1$	$PePD^3$		
Caspian Sea area	463	460	3	99.68	0.32	Kirk, R.L.,et al.	1977

Table 5.3.12.f.I. Red cell Isocitrate dehydrogenase (ICD) in Iran

Population	Number Tested	Phenotypes	Gene frequencies	Authors
		ICD 1-1	ICD^1	
1- Tehran	132	132	100.00	Farhud,D.D.,et al. 1973
2- Caspian Sea area	463	463	100.00	Kirk,R.L., et al. 1977

Table 5.3.12.g.I. Red cell phosphoglycerate Kinase(PGK) in Iran

Population	Number Tested	Phenotypes	Gene frequencies	Authors
		PGK 1-1	PGK^1	
Caspian Sea area	463	463	100.00	Kirk, R.L., et al. 1977

Table 5.3.12.h.I. Red cell Superoxide dismutase (SoD) in Iran

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		SoD 1-1	SoD 2-1	SoD^1	SoD^2	
Caspian Sea area	463	459	4	99.57	0.43	Kirk, R.L., et al. 1977

Table 5.3.12.h.VI. Red cell Superoxide dismutas (SoD) in Saudi Arabia

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		SoD 1-1	SoD 2-1	SoD^1	SoD^2	
Western Saudi Arabia	149	149	-	100.00	0.00	Saha,N., et al. 1980

Table 5.3.12.i.I. Red cell Nicotinamide Adenine Dinucleotide (NADH) Diaphorase in Iran

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Dia 1-1	Dia 2-1	Dia^1	Dia^2	
Caspian Sea area	463	461	2	99.78	0.22	Kirk,R.L.,et al. 1977

Table 5.3.12.j. I. Red cell glutamic oxaloacetic transaminase(GoT) in Iran

Population	Number Tested	Phenotypes	Gene frequencies	Authors
		GoT 1-1	GoT^1	
Tehran	134	134	100.00	Farhud,D.D.,et al. 1973

Table 5.3.12.k. IX Red cell phosphogluco-isomerase(PGI) in Afghanistan

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		PGI 1-1	PGI 1-3	PGI^1	PGI^3	
1-Daris	178	177	1	99.70	0.30	Papiha,S.S.,et al. 1977
2-Pushtus	102	102	-	100.00	0.00	Papiha,S.S.,et al. 1977

Table 5.3.12. 1. V. Red cell Catalase in Kuwait

Population	Number Tested	Normal type	variant	Authors
1- General population	89	89	-	Khaled,E.,et al. 1981
2-Ajman tribe	52	52	-	Khaled,E.,et al. 1981
3-Suluba tribe	52	52	-	Khaled,E.,et al. 1981

5.4. Genetic distance

5.4.1. Introduction

The study of genetic distance is now a major growth industry, and new measures of distance come off the assembly line almost as frequently as new car models (Kirk,1976).And like the automobile industry all distance measures have something in common: they manipulate variations in gene frequencies, package them in various styles and come up with a product that performs the same functions and appeals to the consumer on the basis of a variety of hard and soft 'sells'. What is of importance, however, is customer satisfaction, that is whether the product performs according to the demands made on it by the user.

Excellent reviews of the theory underlying genetic distance measurement have been provided by Balakrishnan et al (1975),Cavalli-Sforza(1973), Cannings and Cavallisforza(1974), Chakraborty et al (1976,1980,1982), Edwards and Cavalli-Sforza(1972), Goodman and Laseer (1974), Morton(1974) and Nei (1975,1978). Many of these authors have pointed out the close mathematical relationship between the various distance indices.

Several measures of genetic distance are now in common use. Those distance coefficients which make use of gene frequency results may appropriately be designated as ' genetic distances' (Constandse-westermann,1972). If two populations have precisely the same gene frequencies they will be considered to be at Zero' distance' apart. The greater the distance between the gene frequencies the greater the distance (C.A.B. Smith, 1977). Genetic distance is used for two reason: Firstly, to reduce a complicated mass of data to easily manageable and visuable form. The study of variation in gene fre-

quency at a single locus is comparatively easy. The difficulty of comprehending such variation at a large number of loci increases very rapidly, and it is necessary to have some composite measure to suggest relationships between population based on all the characteristics under consideration. This is the rational for having a measure of genetic distance(Sanghvi and Balakrishnan, 1972), and secondly to try to reconstruct something of the evolutionary history of the populations concerned (Smith, 1977). Most genetic distances care calculated from qualitative traits, which have been developed mostly over the last ten years. A massive amount of information about the different populations has been gathered and studied using other methods.

According to Constandse-westermann(1972) the coefficients which can be calculated from qualitative data (gene frequencies) may be divided into four categories.

The first category including those coefficients based on squared differences between percentages of frequency values,initially reported by Spuhler(1954).

The second category includes those distance coefficients based on the same principles as X^2, namely GS^2 considered by Edwards and Cavalli-Sforza(1972) and DK^2, which can be considered as a transition to a coefficient belonging to the third category.

The third category includes the coefficient by which the differences in frequency values between populations are expressed in terms of the elements of the pooled dispersion matrix of all investigated groups.

The fourth category contains those distance coefficients whose calculation is based on the angular transformation of the

original percentages of frequencies; this category was used in the present investigation for calculation of distance coefficient.Edwards and Cavalli-Sforza(1972) suggested that the angular transformation provides a measure of distance, which has some advantages over other measures, namely,that the angular transformation, being non-linear, standardises the variances and takes care of the correlations by reducing the number of dimensions and gives a better estimate of large distances. The genetic distance E is strictly comparable between different sets of data based on information from the same loci.

The study of genetic relationships between populations in Iran, either on the broad basis of comparisons across the sub-continent, or at the local level, is still in its infancy. For such an analysis, the first practical step is to collect data on gene frequencies of various polymorphic systems. This is as far as most of the work so far completed on the Iranian population goes, and indeed even at the present time no comprehensive and countrywide survey has yet been undertaken.

5.4.2. Materials and method

In the present investigation because of the large number of the population groups and the serological systems involved, finding of a considerable number of loci common to all the populations considered and calculating of a single genetic distance analysis, was impossible. Therefore, eleven groups of populations were selected on the basis of variability of gene frequency information on particular loci common to all of them. The resulting eleven sets of genetic distance

analysis together with the population groups and the loci considered in each set of analysis will be presented later.

The method employed in the present study for distance coefficient was Edwards and Cavalli-Sforza's ' new E^2'. The genetic distance statistic Edwards E^2 is a distance coefficient produced between groups for frequency data(Edwards and Cavalli-Sforza, 1972).

The Statistic E^2 is calculated from the following formula:

$$E^2 = 8\left(1 - \sum_{K=1}^{S_J+1} \sqrt{P_1 JK \cdot P_2 JK}\right) \Bigg/ \left(\left(1 + \sum_{K=1}^{S_J+1} \sqrt{\frac{P_1 JK}{S_J+1}}\right)\left(1 + \sum_{K=1}^{S_J+1} \sqrt{\frac{P_2 JK}{S_J+1}}\right)\right)$$

Where P_1 JK is the frequency of the K th class of the Jth character in the population.

SJ is the number of classes minus one.

The E^2 distance for the various population groups from Iran and neighbouring countries were examined on the basis of this formula, the gene frequencies data was analysed using the Fortran Program.

The output from the program consisted of:

1- A data input reprint.

2- Individual E^2 values for each locus separately.

3- The total E^2 values.

4- The standardised E^2 values.

Two types of analysis were used in the aid to interpretation:

1- Non-Metric Multidimensional Scaling the Program was called ' MINISSAN' from the ' MDS(X)' package.

2- Cluster Analysis-using maximum/complete link clustering.

5.4.3. Results

The results of the two types of the genetic distance analysis, based on Edwards E^2 standard genetic distance , for the eleven sets of data are presented in figures 5.4.3. 1-11.

Since in the eleven sets of analysis the population groups and the loci considered differ one from another, therefore, the Non-Metric two dimensional Scaling and the cluster analysis outputs (figures a and b respectively) together with the populations codesand the loci considered for each set of analysis are given separately below:

Set 1.

The 7 loci and the respective 17 alleles

Loci	Alleles
The ABO locus	A, B, O
The Rh locus	D, d
The HP locus	HP^1, HP^2
The Tf locus	Tf^C, Tf^B, Tf^D
The AcP locus	AcP^A, AcP^B, AcP^C
The AK locus	AK^1, AK^2
The PGM_1 locus	PGM_1^1, PGM_1^2

Set 2.

The 7 loci and the respective 18 alleles

Loci	Alleles
The A_1A_2BO locus	A_1, A_2, B, O
The Rh locus	D, d
The HP locus	HP^1, HP^2
The Tf locus	Tf^C, Tf^B, Tf^D
The AcP locus	AcP^A, AcP^B, AcP^C
The AK locus	AK^1, AK^2
The PGM_1 locus	PGM_1^1, PGM_1^2

Set 3.

The 8 loci and the respective 19 alleles

Loci	Alleles
The ABO locus	A, B, O
The Rh locus	D, d
The HP locus	HP^1, HP^2
The Tf locus	Tf^C, Tf^B, Tf^D
The AcP locus	AcP^A, AcP^B, AcP^C
The AK locus	AK^1, AK^2

Loci		Alleles
The PGM_1	locus	PGM_1^1, PGM_1^2
The PGD	locus	PGD^A, PGD^C

Set 4.

The 8 loci and the respective 19 alleles

Loci		Alleles
The ABO	locus	A, B, O
The Rh	locus	D, d
The HP	locus	HP^1, HP^2
The Tf	locus	Tf^C, Tf^B, Tf^D
The AcP	locus	AcP^A, AcP^B, AcP^C
The AK	locus	AK^1, AK^2
The PGM_1	locus	PGM_1^1, PGM_1^2
The ADA	locus	ADA^1, ADA^2

Set 5.

The 8 loci and the respective 19 alleles

Loci		Alleles
The ABO	locus	A, B, O
The Rh	locus	D, d
The HP	locus	HP^1, HP^2
The Tf	locus	Tf^C, Tf^B, Tf^D
The AcP	locus	AcP^A, AcP^B, AcP^C
The AK	locus	AK^1, AK^2
The PGM_1	locus	PGM_1^1 , PGM_1^2
The EsD	locus	EsD^1, EsD^2

Set 6.

The 9 loci and the respective 22 alleles

Loci	Alleles
The A_1A_2BO locus	A_1, A_2, B, O

Loci		Alleles
The Rh	locus	D, d
The MN	locus	M, N
The Kell	locus	K, k
The Hp	locus	HP^1 , HP^2
The Tf	locus	Tf^C, Tf^B, Tf^D
The AcP	locus	AcP^A, AcP^B, AcP^C
The AK	locus	AK^1 , AK^2
The PGM_1	locus	PGM_1^1 , PGM_1^2

Set 7.

The 9 loci and the respective 24 alleles

Loci		Alleles
The A_1A_2BO	locus	A_1, A_2, B, O
The Rh	locus	D, d
The MNSs	locus	M, N, S, s
The Kell	locus	K, k
The HP	locus	HP^1, HP^2
The Tf	locus	Tf^C, Tf^B, Tf^D
The AcP	locus	AcP^A, AcP^B, AcP^C
The AK	locus	AK^1, AK^2
The PGM_1	locus	PGM_1^1 , PGM_1^2

Set 8.

The 10 loci and the respective 26 alleles

Loci		Alleles
The A_1A_2 BO	locus	A_1, A_2, B, O
The Rh	locus	D, d
The MNSs	locus	M, N, S, s
The Kell	locus	K, k
The Duffy	locus	FY^a, Fy^b+ FY

Loci		Alleles
The HP	locus	HP^1, HP^2
The Tf	locus	Tf^C, Tf^B, Tf^D
The AcP	locus	AcP^A , AcP^B, AcP^C
The AK	locus	AK^1, AK^2
The PGM_1	locus	PGM_1^1 , PGM_1^2

Set 9.

The 10 loci and the respective 26 alleles

Loci		Alleles
The A_1A_2 BO	locus	A_1, A_2, B, O
The Rh	locus	D, d
The MNSs	locus	M, N, Ŝ, s
The Kell	locus	K, k
The Kidd	locus	JK^a, JK^b+Jk
The HP	locus	HP^1, HP^2
The Tf	locus	Tf^C, Tf^B, Tf^D
The AcP	locus	AcP^A , AcP^B, AcP^C
The AK	locus	AK^1, AK^2
The PGM_1	locus	PGM_1^1 , PGM_1^2

Set 10.

The 9 loci and the respective 26 alleles

Loci		Alleles
The A_1A_2 BO	locus	A_1, A_2, B, O
The Rh	locus	C, c, D, d, E, e
The MN	locus	M, N
The Kell	locus	K, k
The HP	locus	HP^1, HP^2
The Tf	locus	Tf^C, Tf^B, Tf^D
The AcP	locus	AcP^A, AcP^B, AcP^C

Loci	Alleles
The AK locus	AK^1, AK^2
The PGM_1 locus	PGM_1^1 , PGM_1^2

Set 11.

The 12 loci and the respective 30 alleles

Loci	Alleles
The A_1A_2BO locus	A_1, A_2, B, O
The Rh locus	D, d
The MNSs locus	M, N, S, s
The Kell locus	K, k
The Duffy locus	Fy^a , Fy^b + Fy
The Kidd locus	JK^a, JK^b + JK
The HP locus	HP^1, HP^2
The Tf locus	Tf^C, Tf^B, Tf^D
The AcP locus	AcP^A, AcP^B, AcP^C
The AK locus	AK^1, AK^2
The PGM_1 locus	PGM_1^1, PGM_1^2
The EsD locus	EsD^1, EsD^2

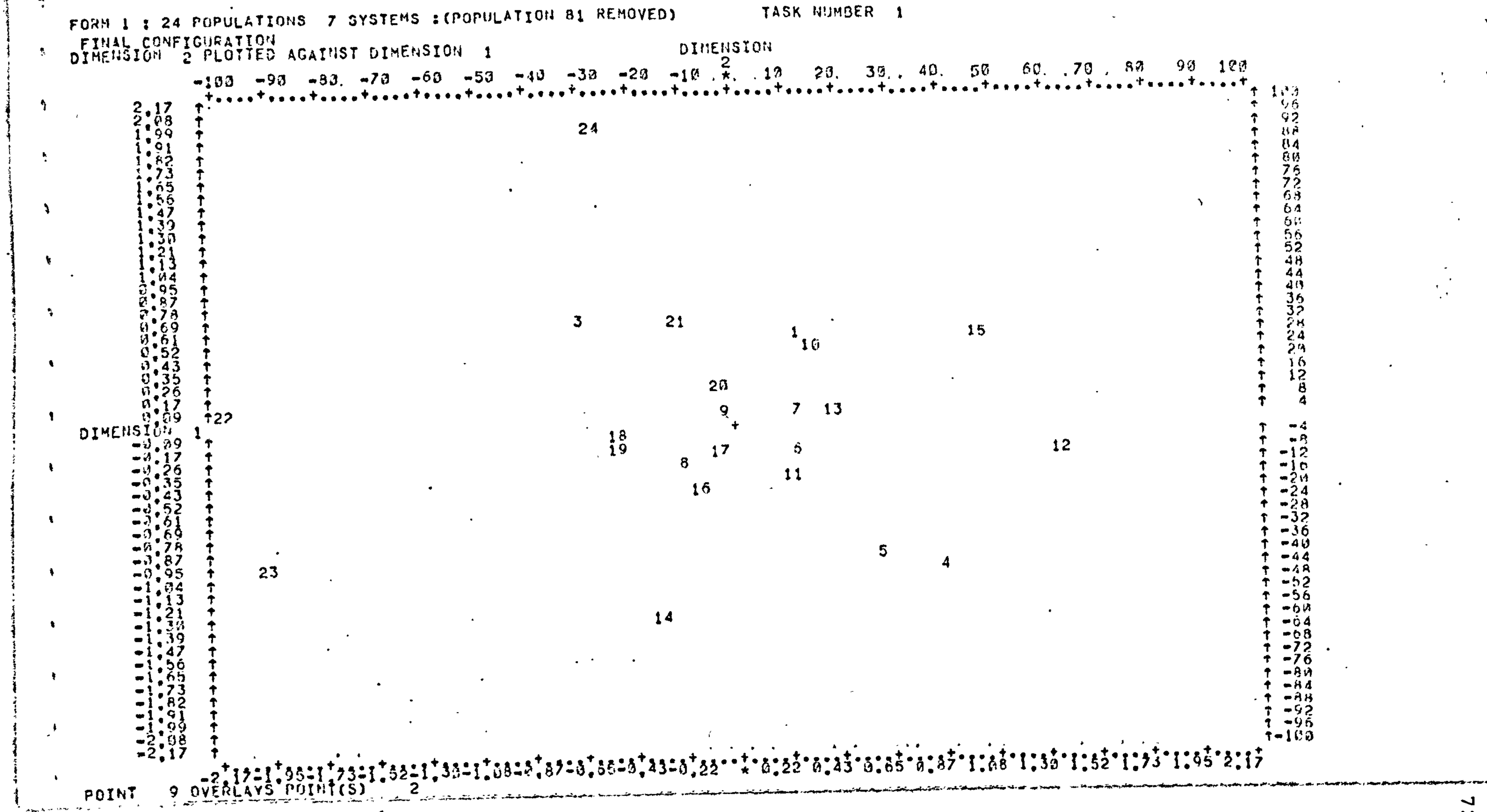


Fig. 5.4.3. 1a. Two- dimensional map of genetic distance for 24 Iranian and neighbouring populations.

Based on gene frequency data for 7 polymorphic loci.

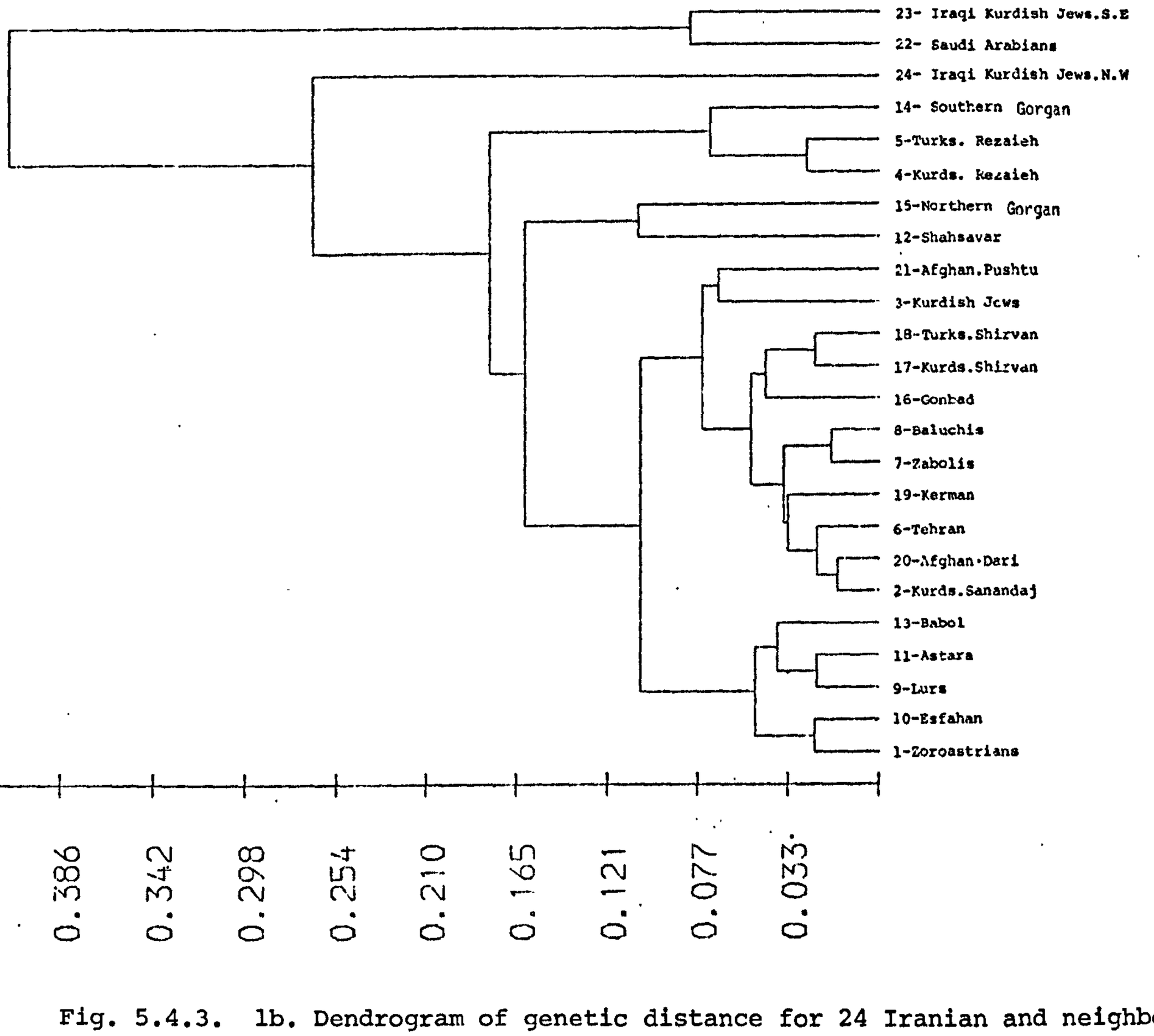


Fig. 5.4.3. 1b. Dendrogram of genetic distance for 24 Iranian and neighbouring populations. Based on gene frequency data for 7 polymorphic loci.

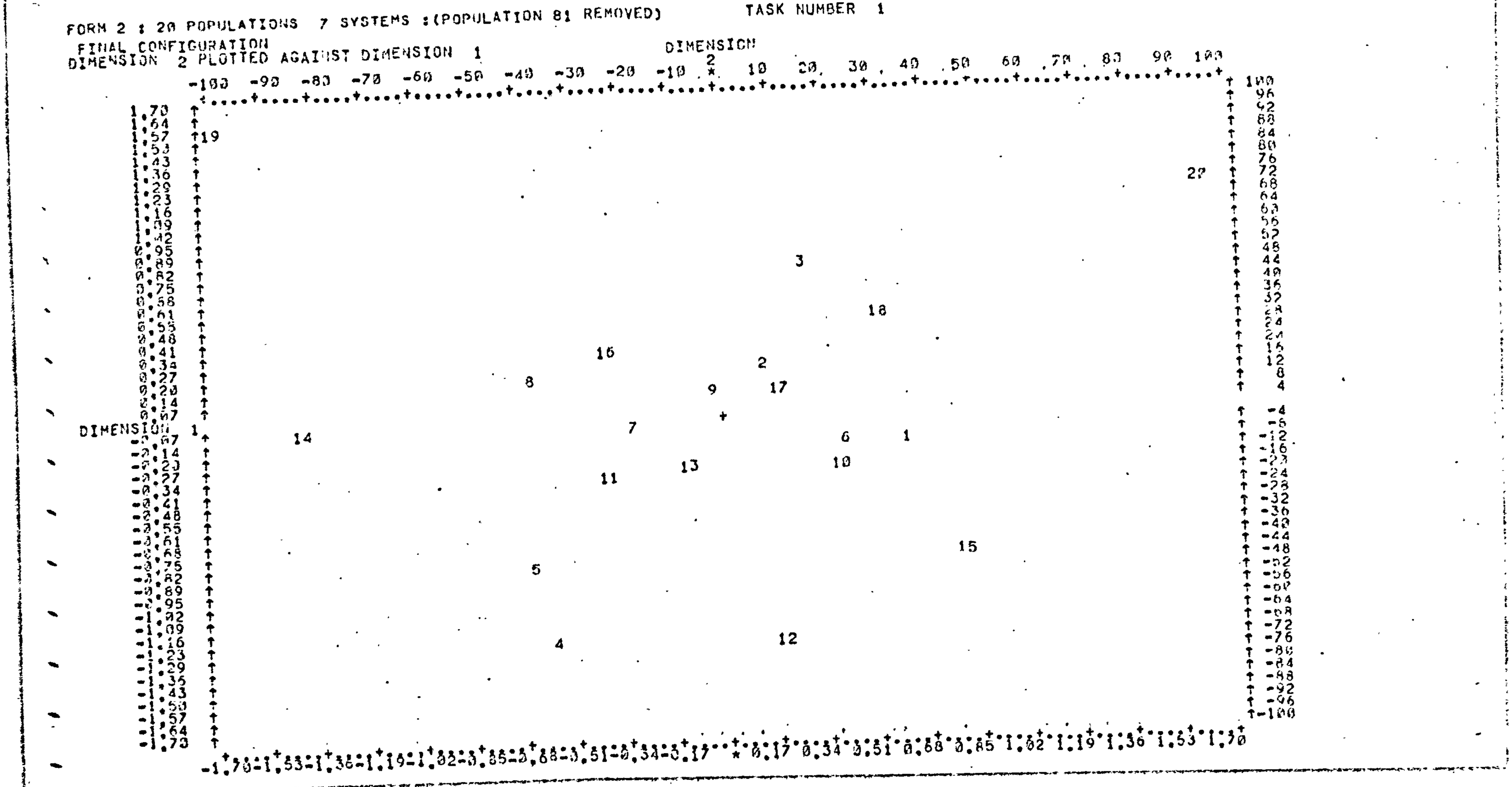


Fig. 5.4.3. 2a. Two- dimensional map of genetic distance for 20 Iranian and neighbouring populations.

Based on gene frequency data for 7 polymorphic loci.

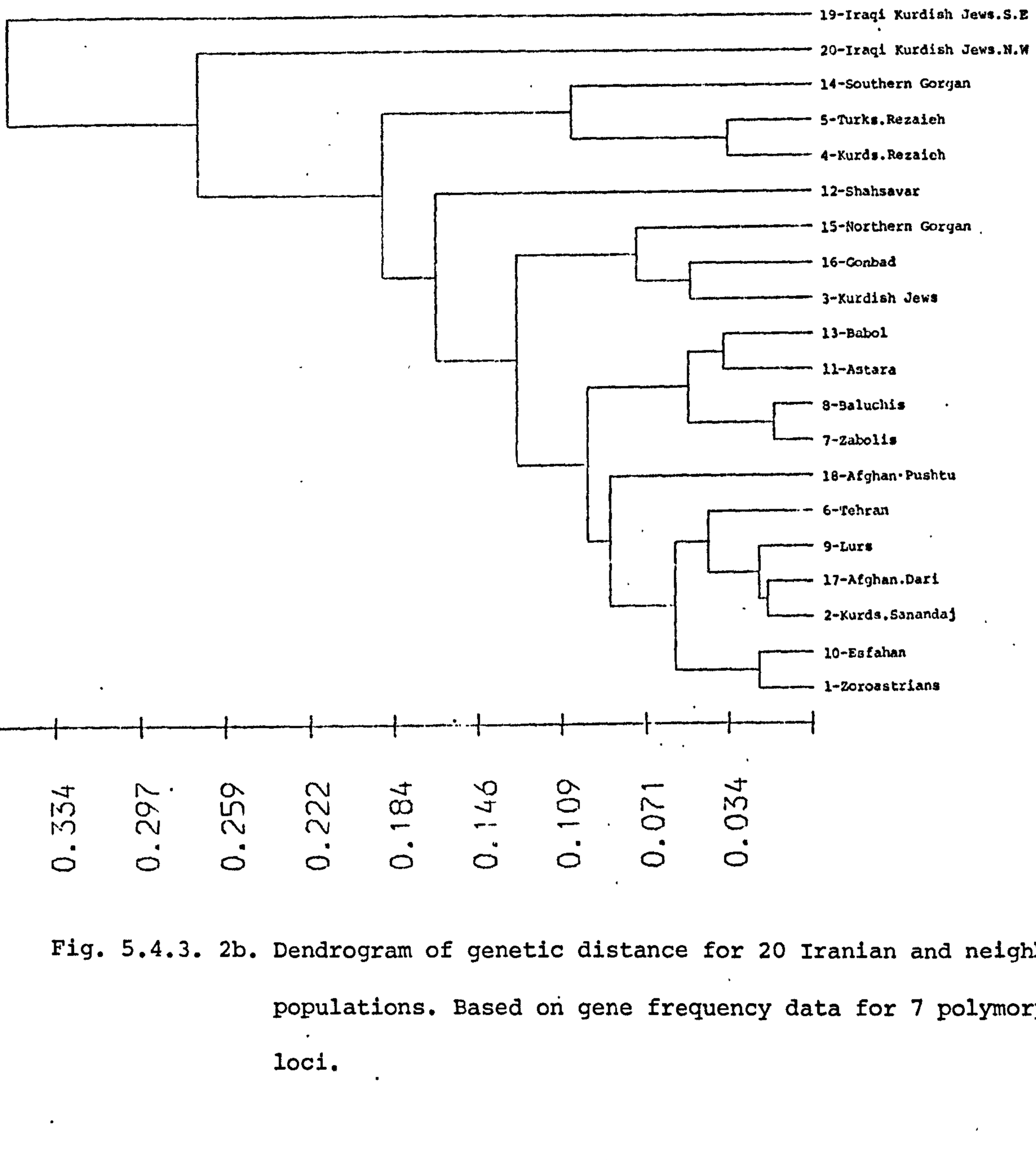


Fig. 5.4.3. 2b. Dendrogram of genetic distance for 20 Iranian and neighbouring populations. Based on gene frequency data for 7 polymorphic loci.

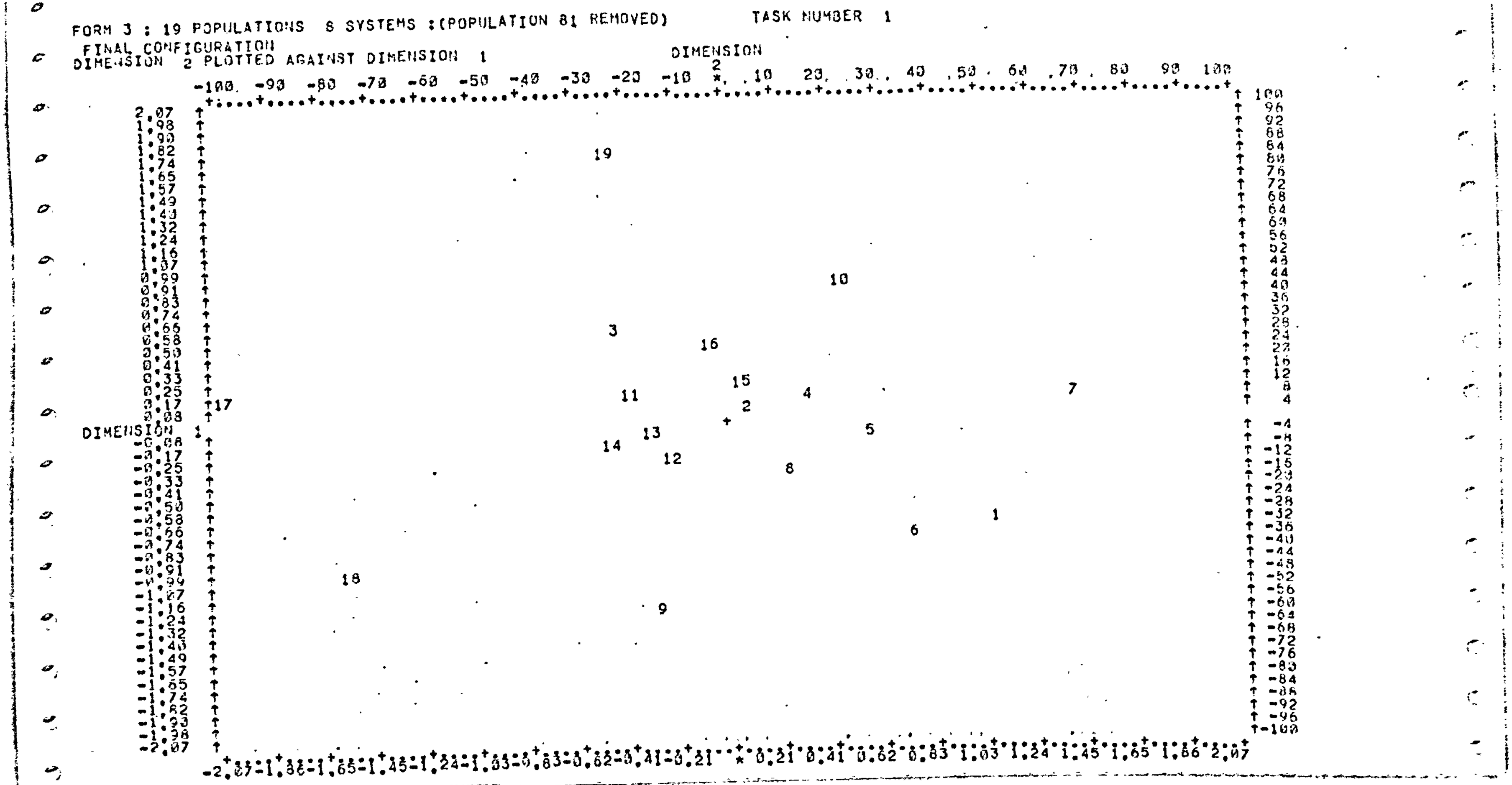


Fig. 5.4.3. 3a. Two- dimensional map of genetic distance for 19 Iranian and neighbouring populations.

Based on gene frequency data for 8 polymorphic loci.

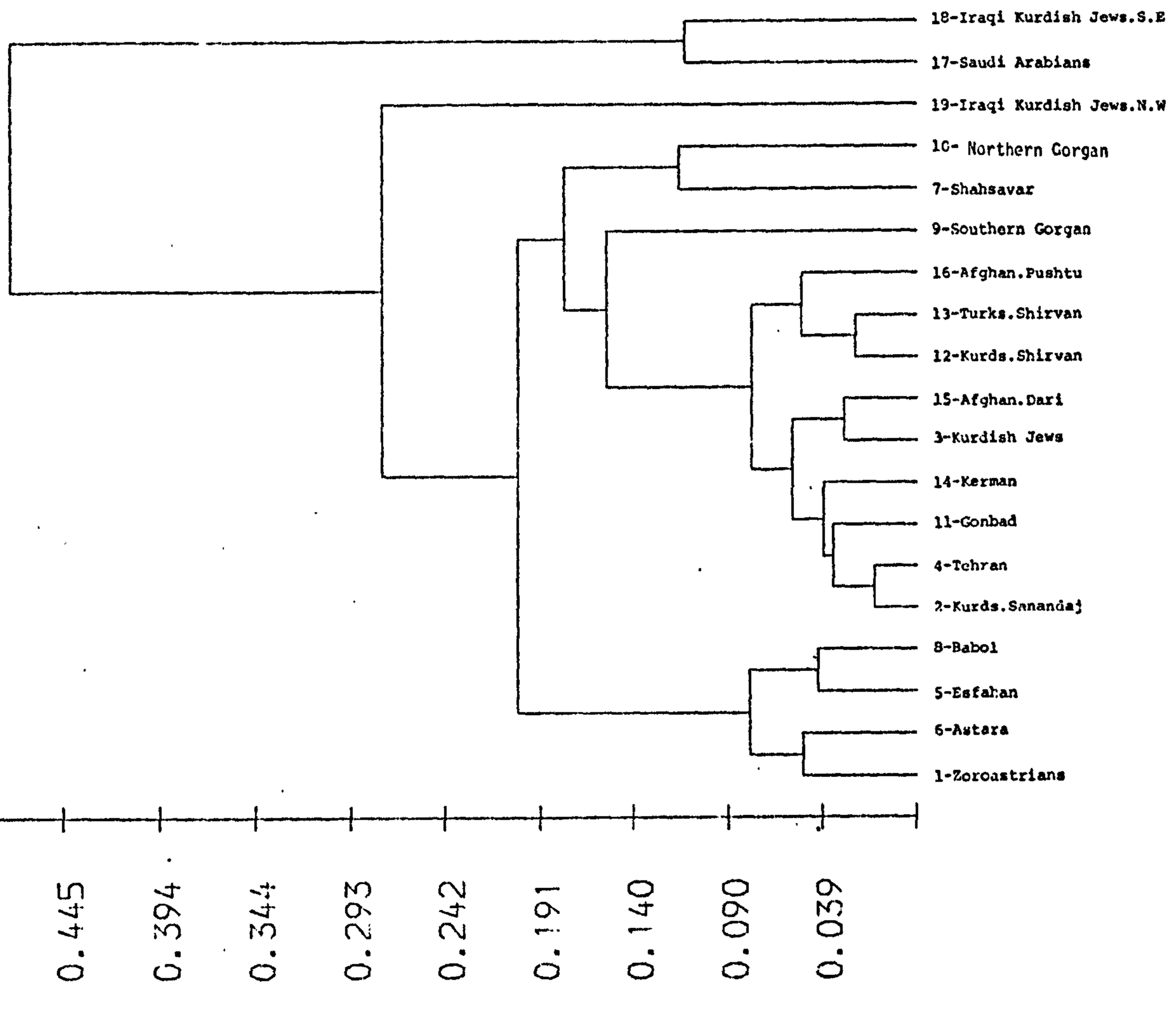


Fig. 5.4.3. 3b. Dendrogram of genetic distance for 19 Iranian and neighbouring populations. Based on gene frequency data for 8 polymorphic loci.

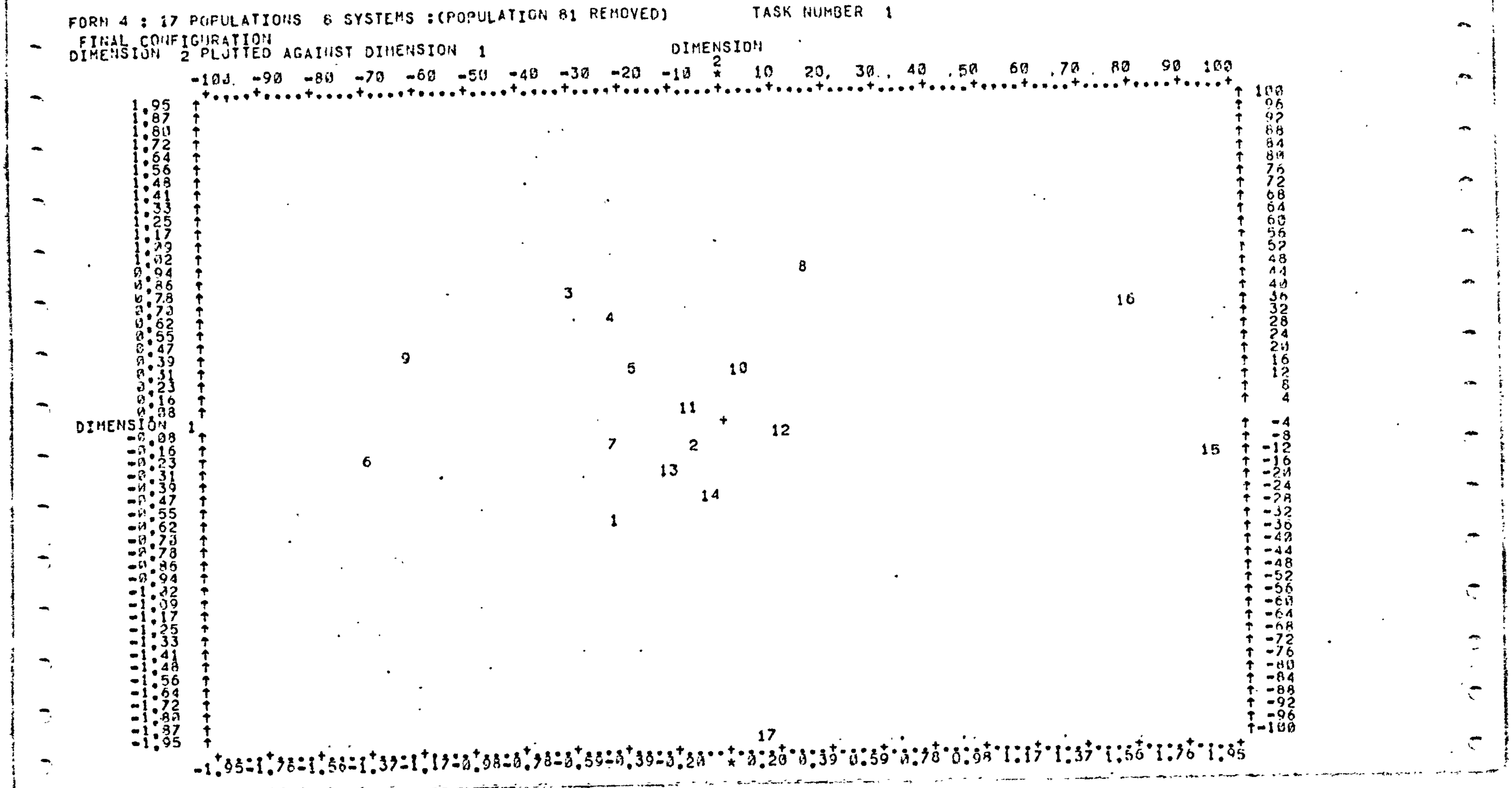


Fig. 5.4.3. 4a. Two-dimensional map of genetic distance for 17 Iranian and neighbouring populations.

Based on gene frequency data for 8 polymorphic loci.

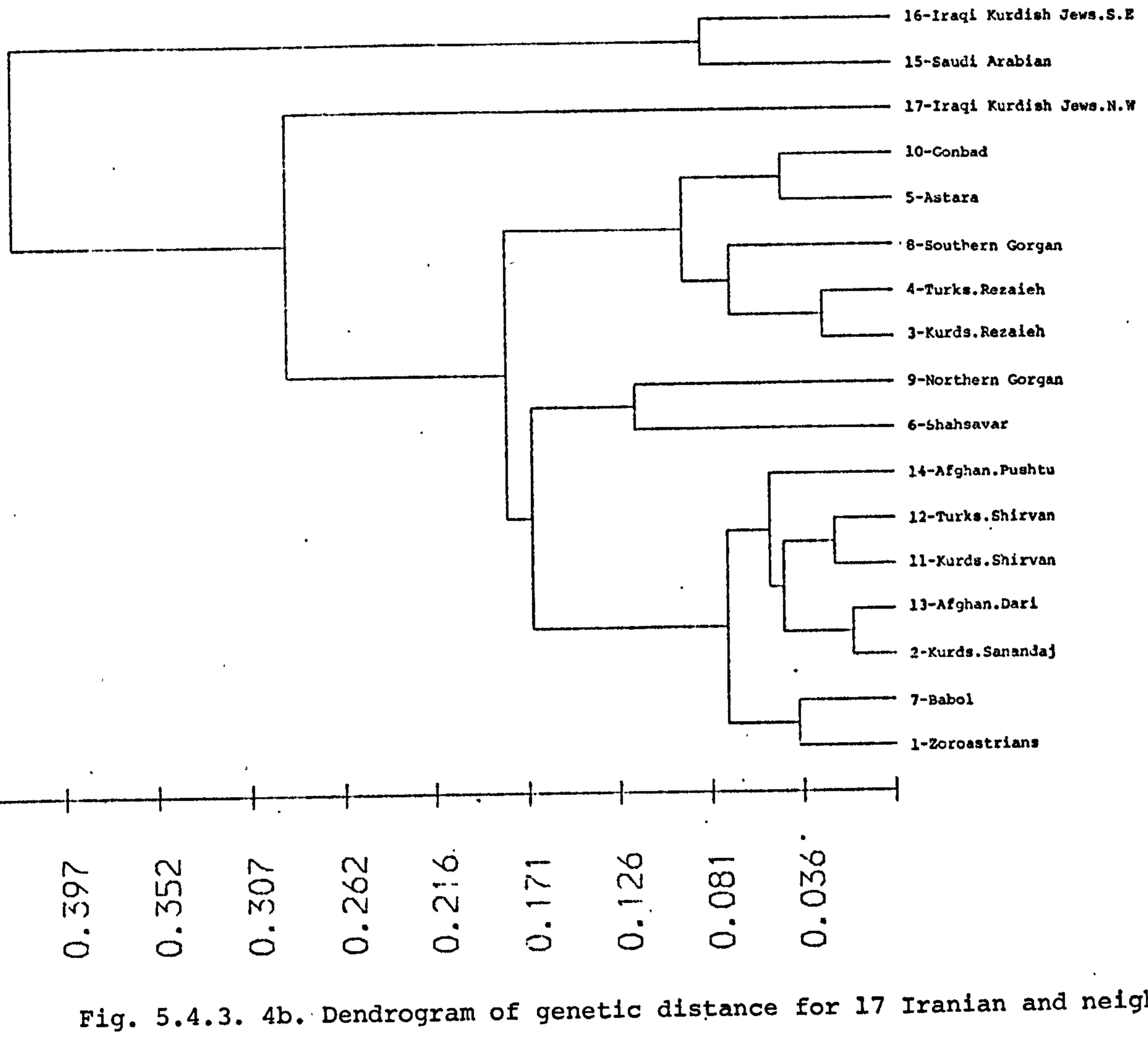


Fig. 5.4.3. 4b. Dendrogram of genetic distance for 17 Iranian and neighbouring populations. Based on gene frequency data for 8 polymorphic loci.

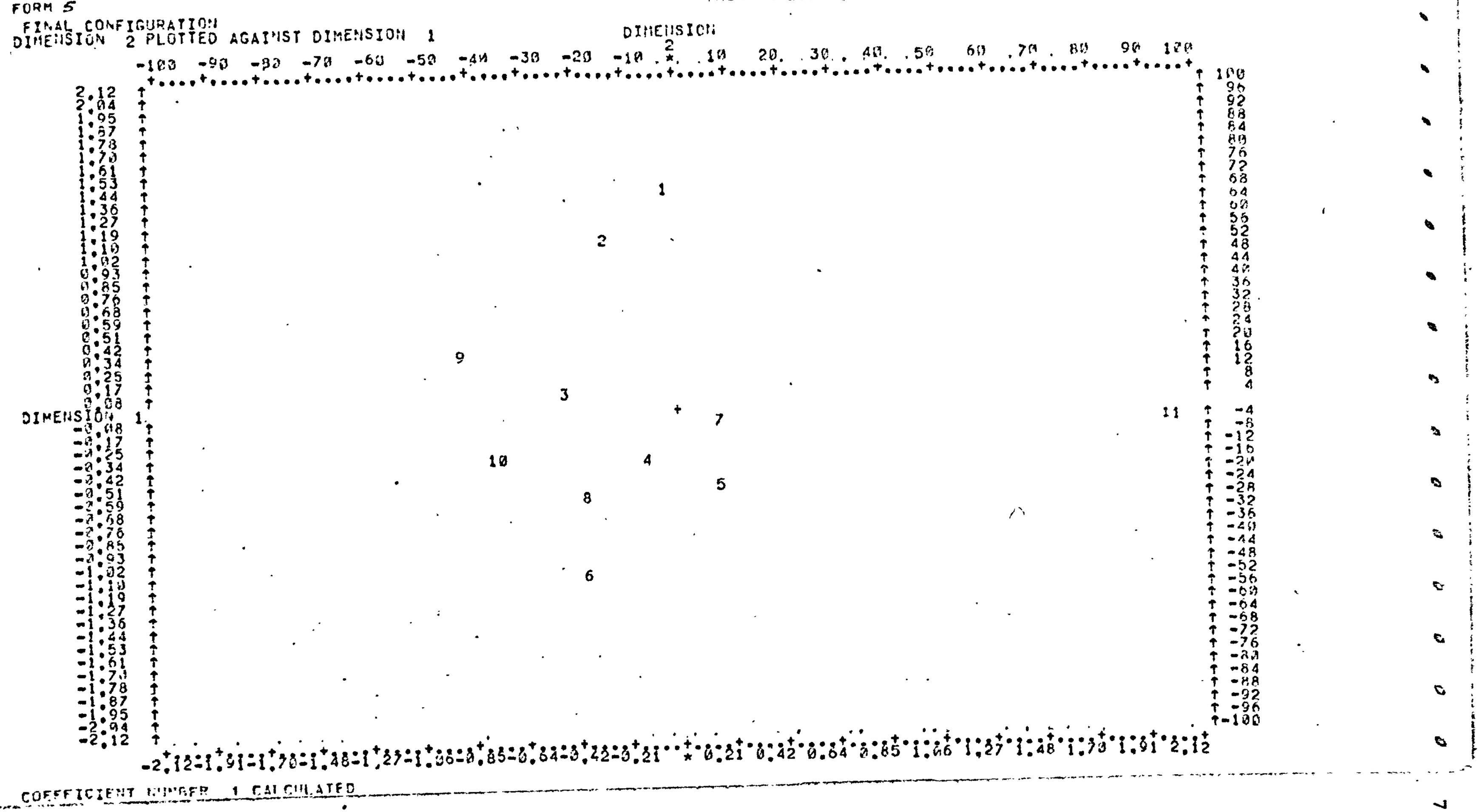


Fig. 5.4.3. 5a. Two-dimensional map of genetic distance for 11 Iranian and neighbouring populations.

Based on gene frequency data for 8 polymorphic loci.

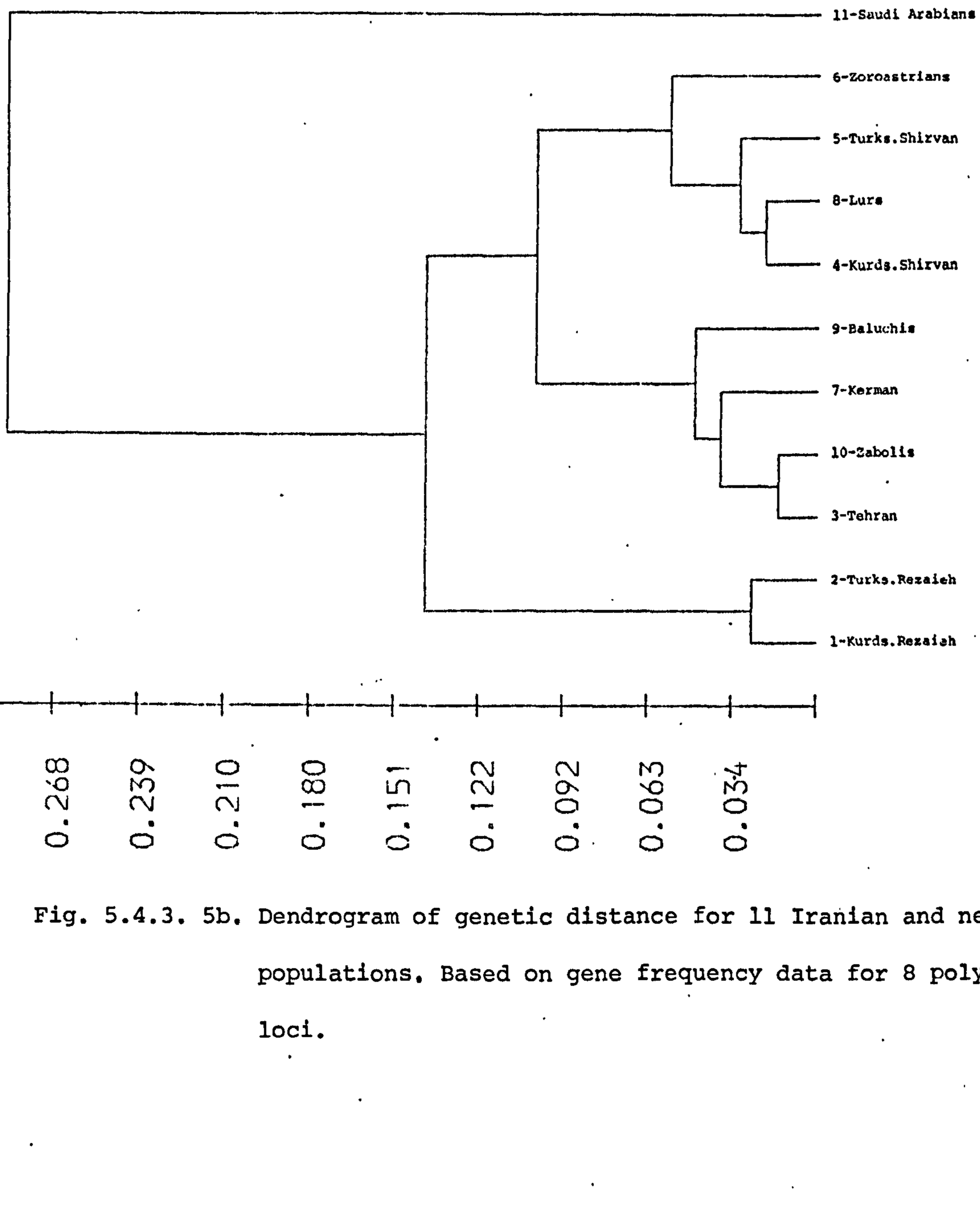


Fig. 5.4.3. 5b. Dendrogram of genetic distance for 11 Iranian and neighbouring populations. Based on gene frequency data for 8 polymorphic loci.

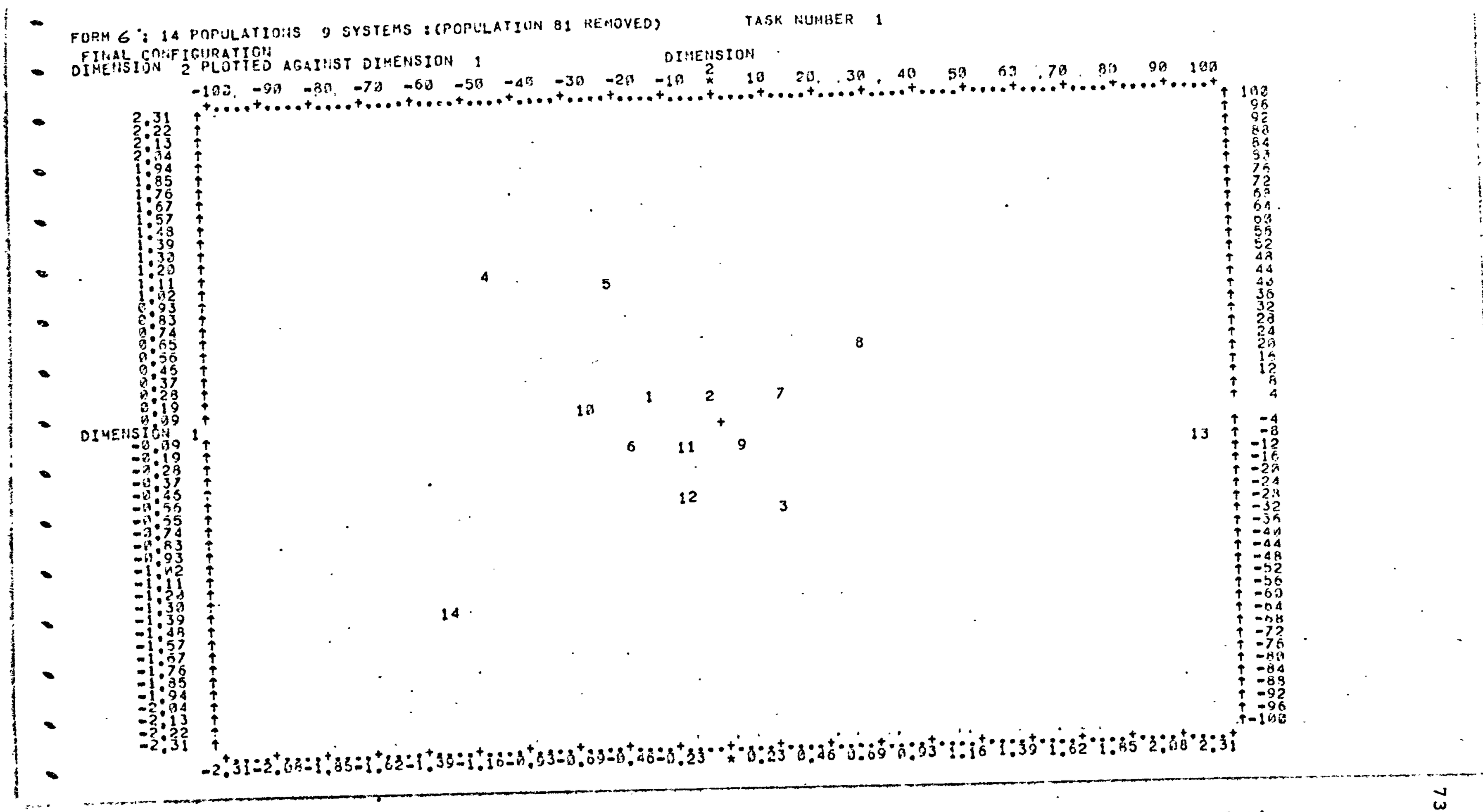


Fig. 5.4.3. 6a. Two- dimensional map of genetic distance for 14 Iranian and neighbouring populations.

Based on gene frequency data for 9 polymorphic loci.

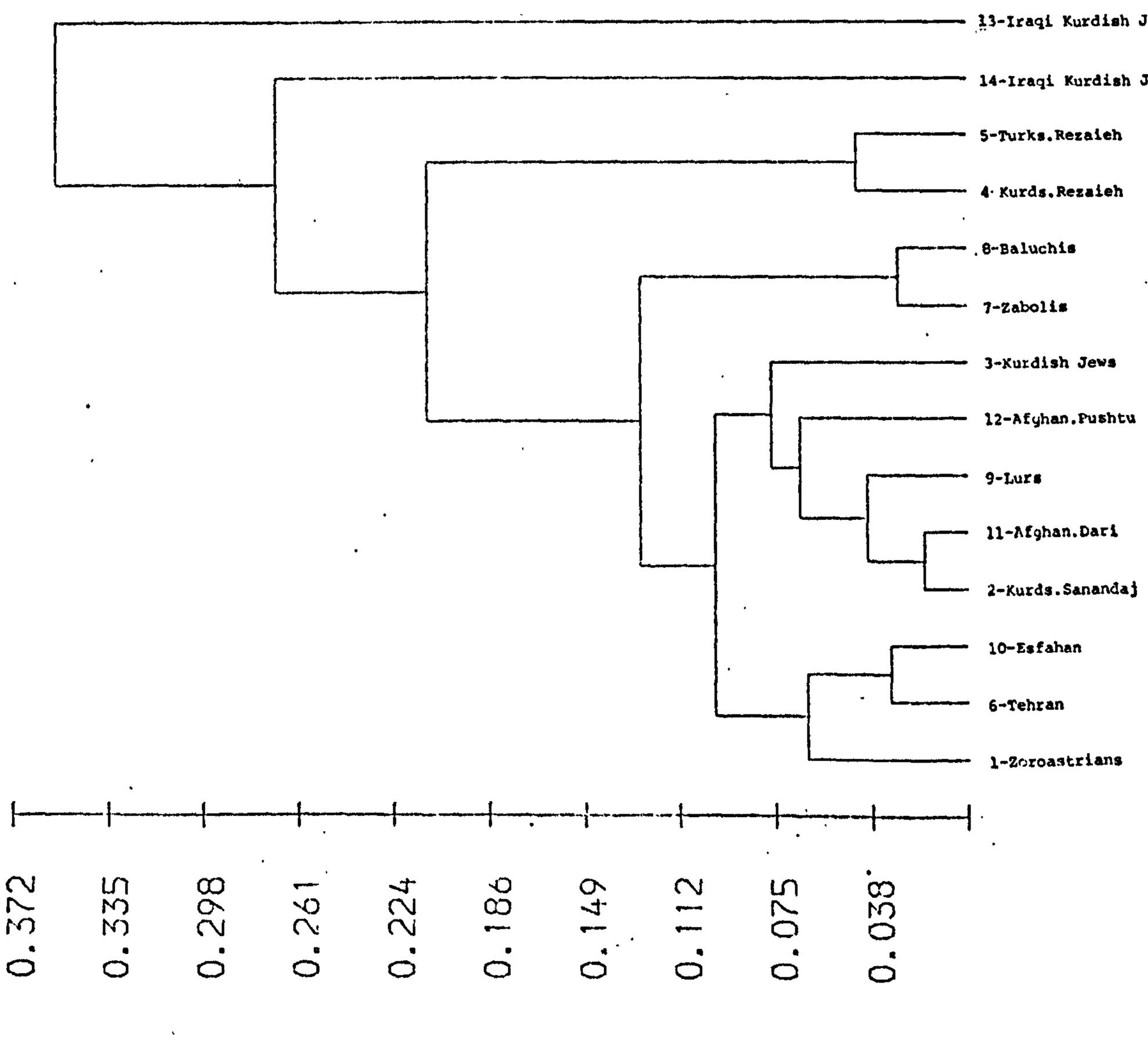


Fig. 5.4.3. 6b. Dendrogram of genetic distance for 14 Iranian and neighbouring populations. Based on gene frequency data for 9 polymorphic loci.

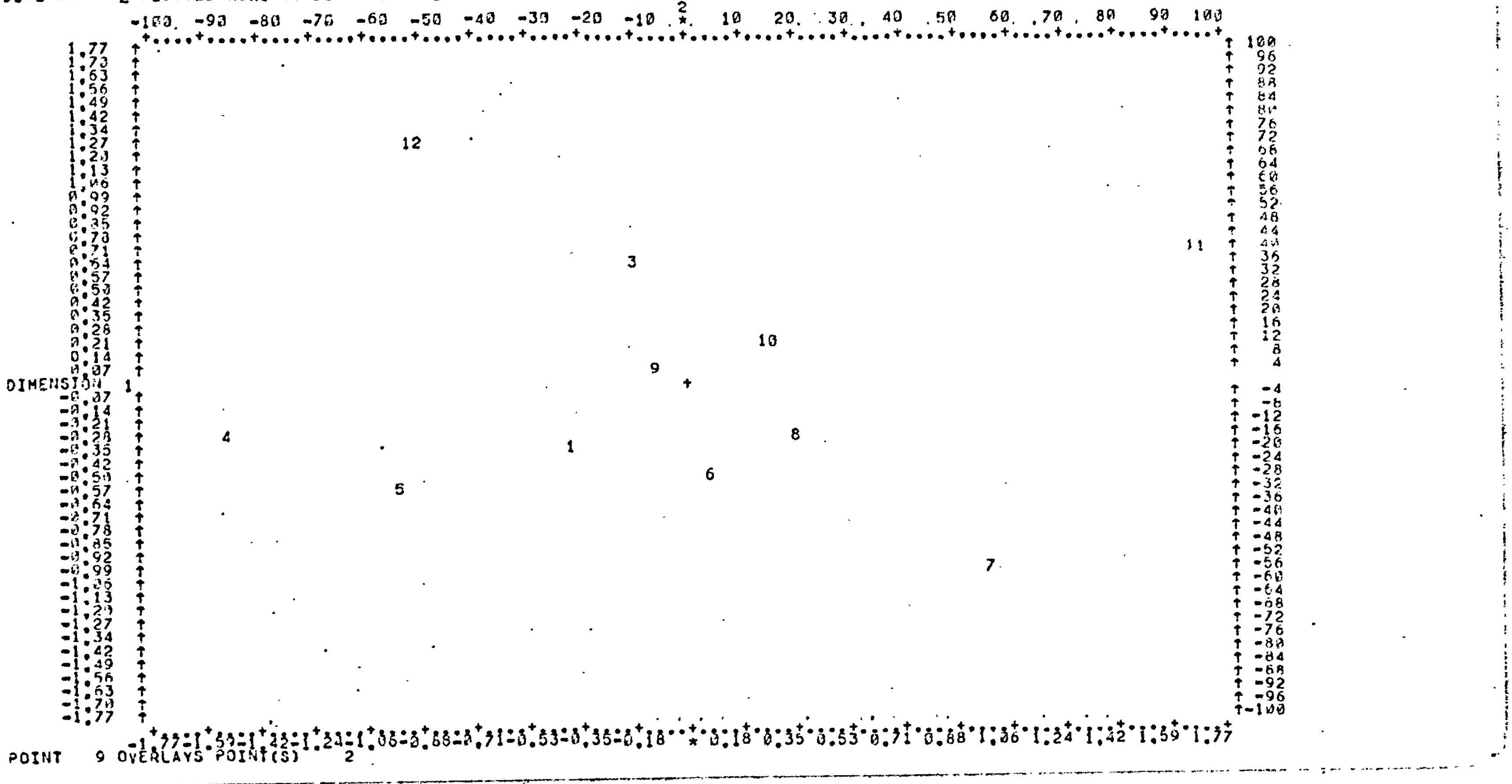


Fig. 5.4.3. 7a. Two- dimensional map of genetic distance for 12 Iranian and neighbouring populations.

Based on gene frequency data for 9 polymorphic loci.

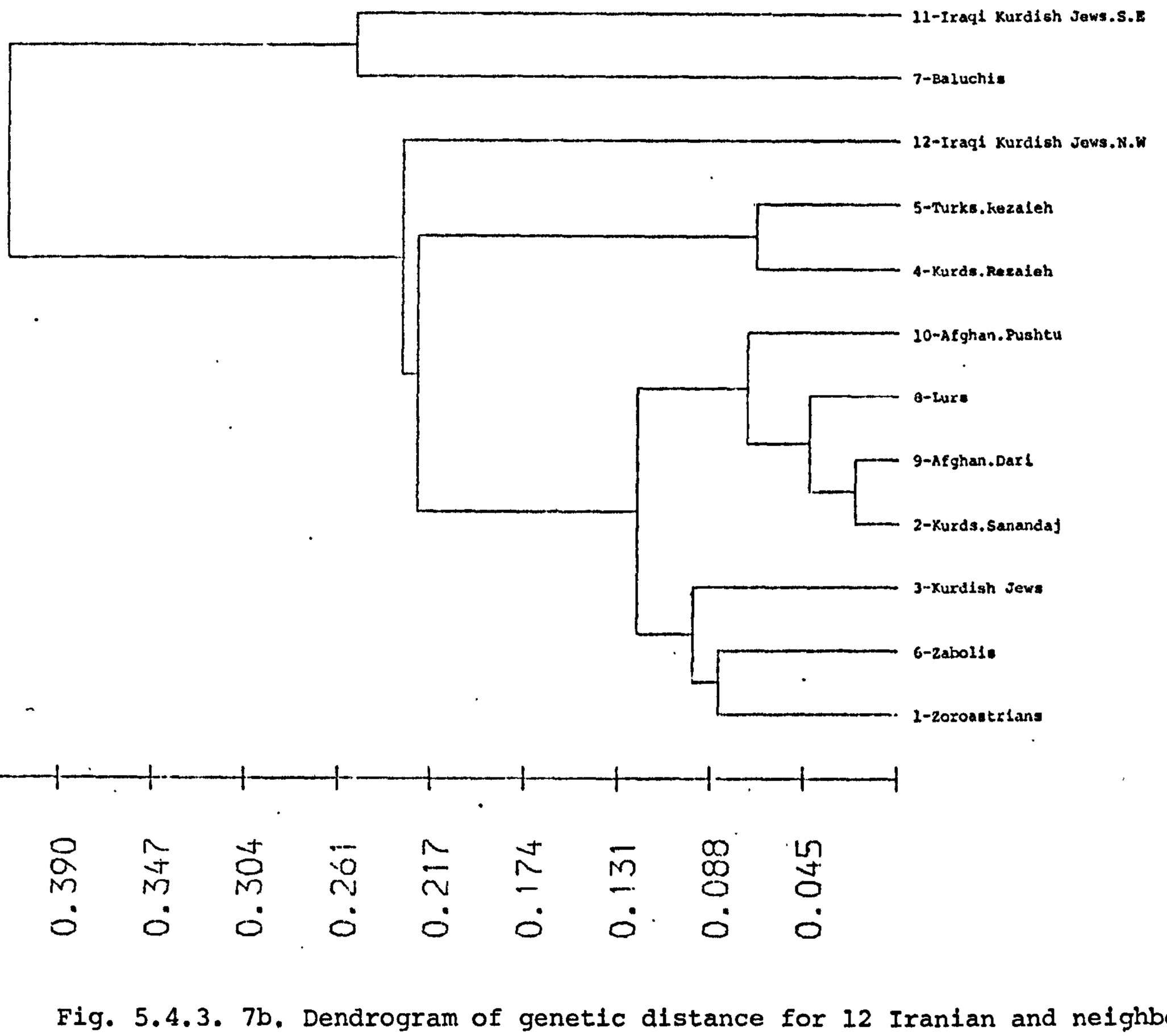


Fig. 5.4.3. 7b. Dendrogram of genetic distance for 12 Iranian and neighbouring populations. Based on gene frequency data for 9 polymorphic loci.

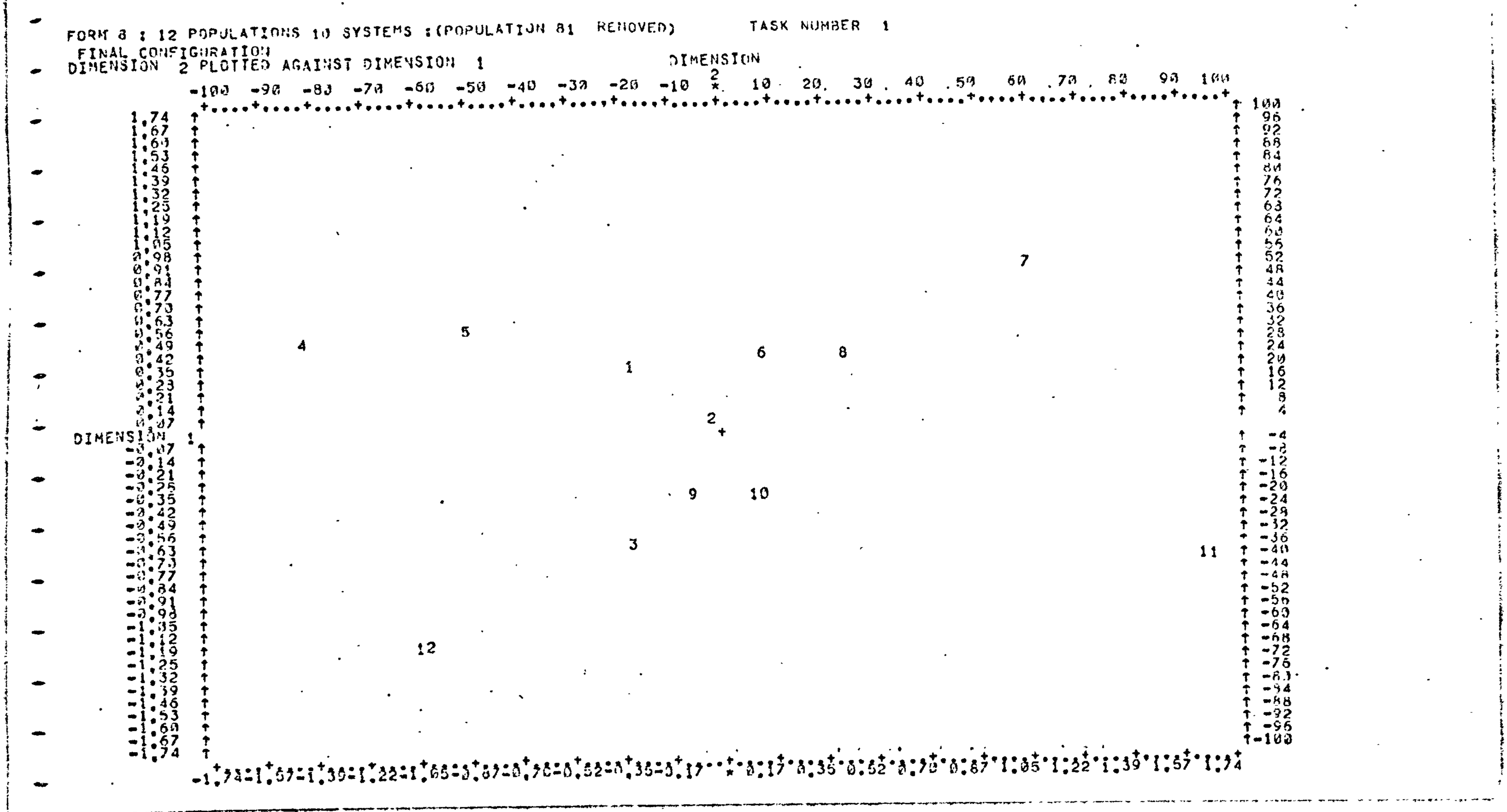


Fig. 5.4.3. 8a. Two-dimensional map of genetic distance for 12 Iranian and neighbouring populations.

Based on gene frequency data for 10 polymorphic loci.

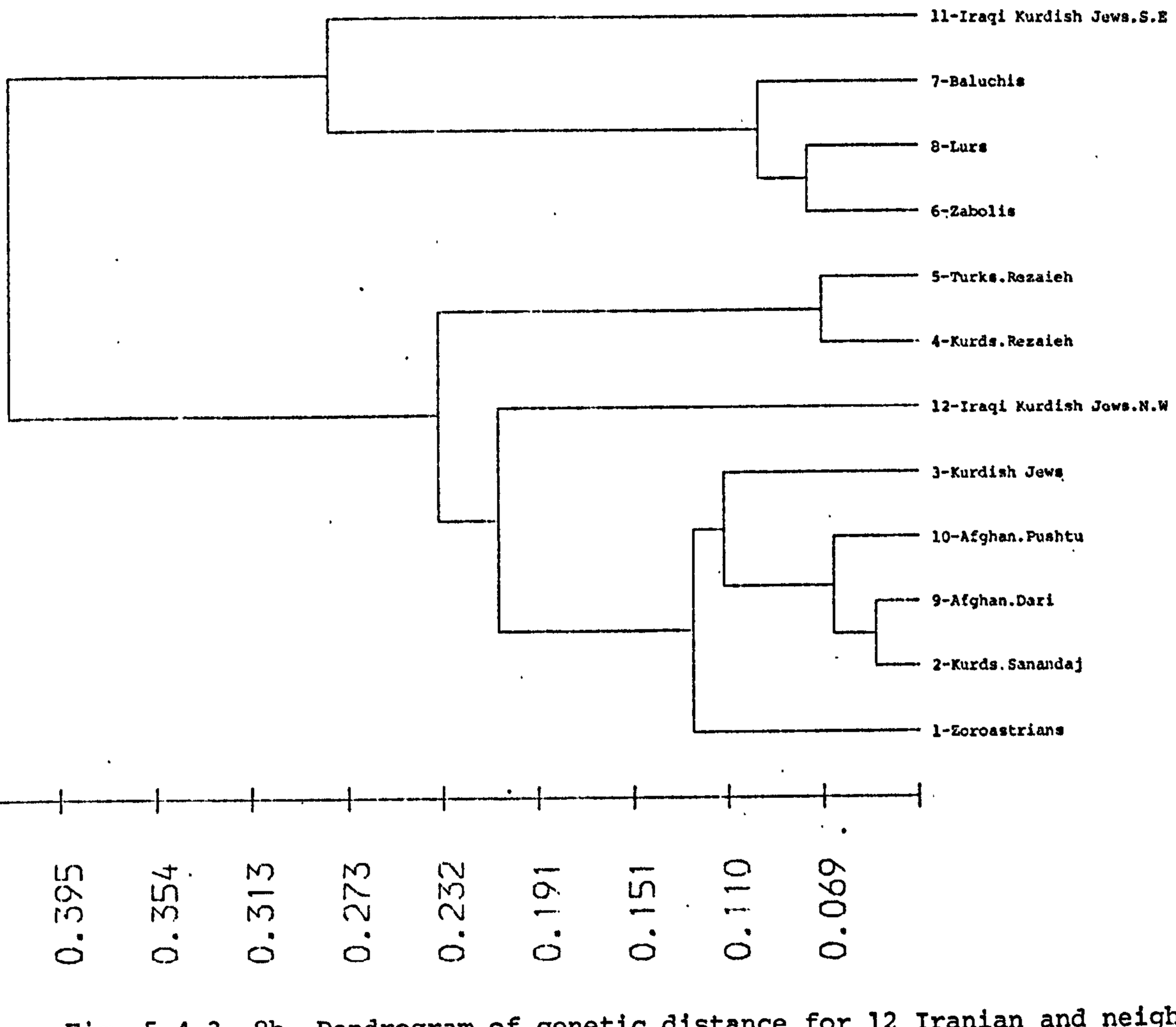


Fig. 5.4.3. 8b. Dendrogram of genetic distance for 12 Iranian and neighbouring populations. Based on gene frequency data for 10 polymorphic loci.

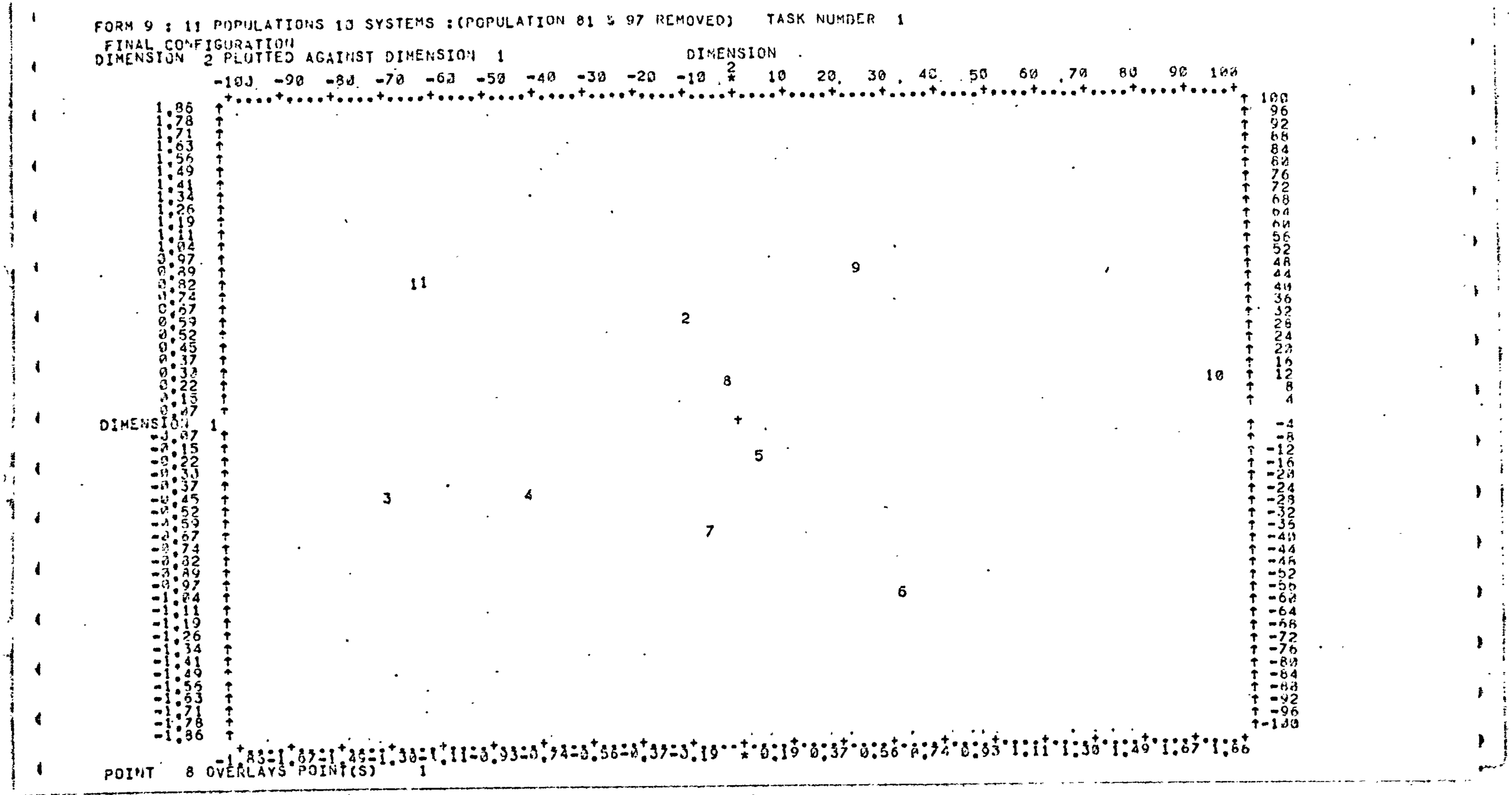


Fig. 5.4.3. 9a. Two- dimensional map of genetic distance for 11 Iranian and neighbouring populations.

Based on gene frequency data for 10 polymorphic loci.

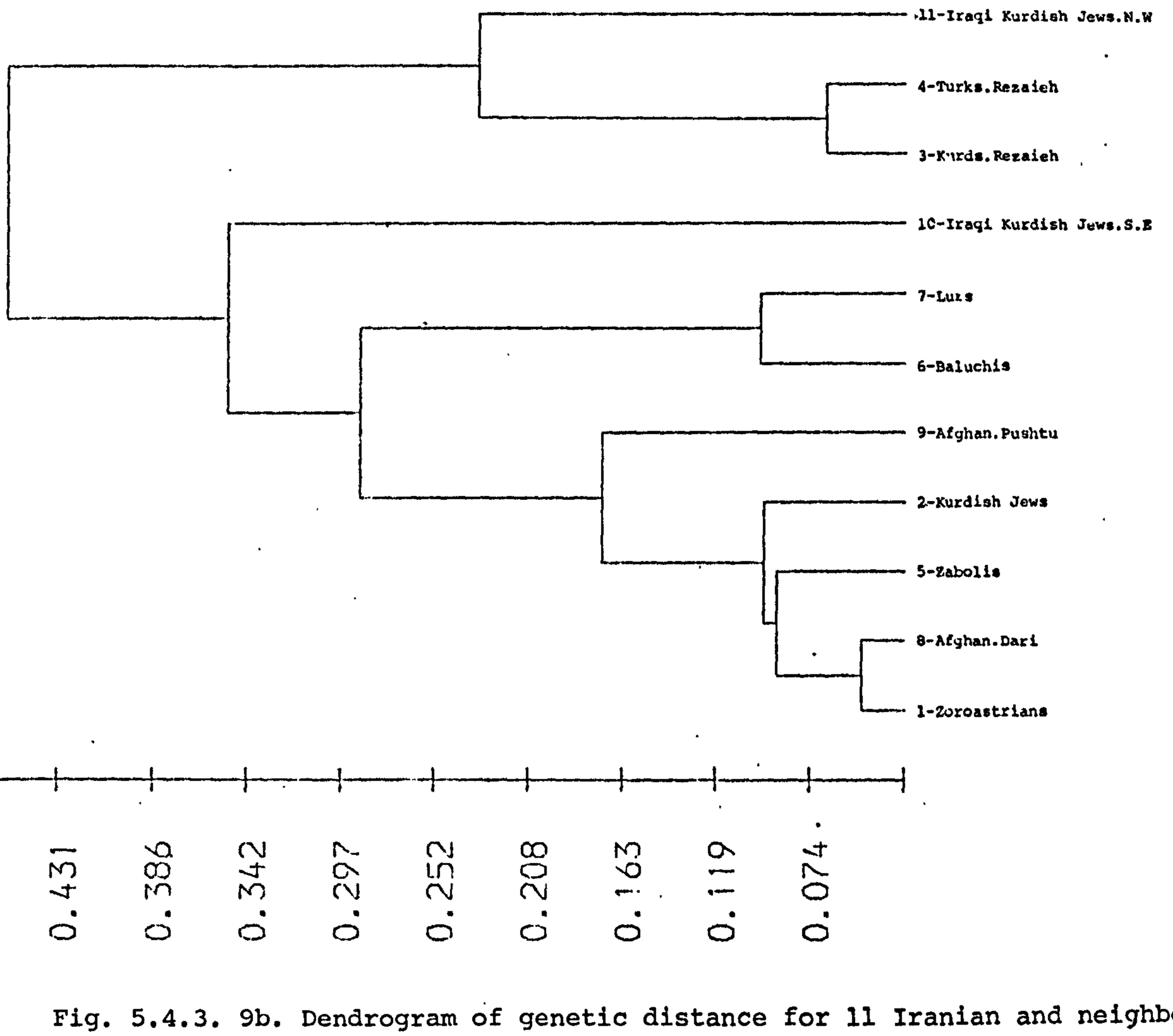


Fig. 5.4.3. 9b. Dendrogram of genetic distance for 11 Iranian and neighbouring populations. Based on gene frequency data for 10 polymorphic loci.

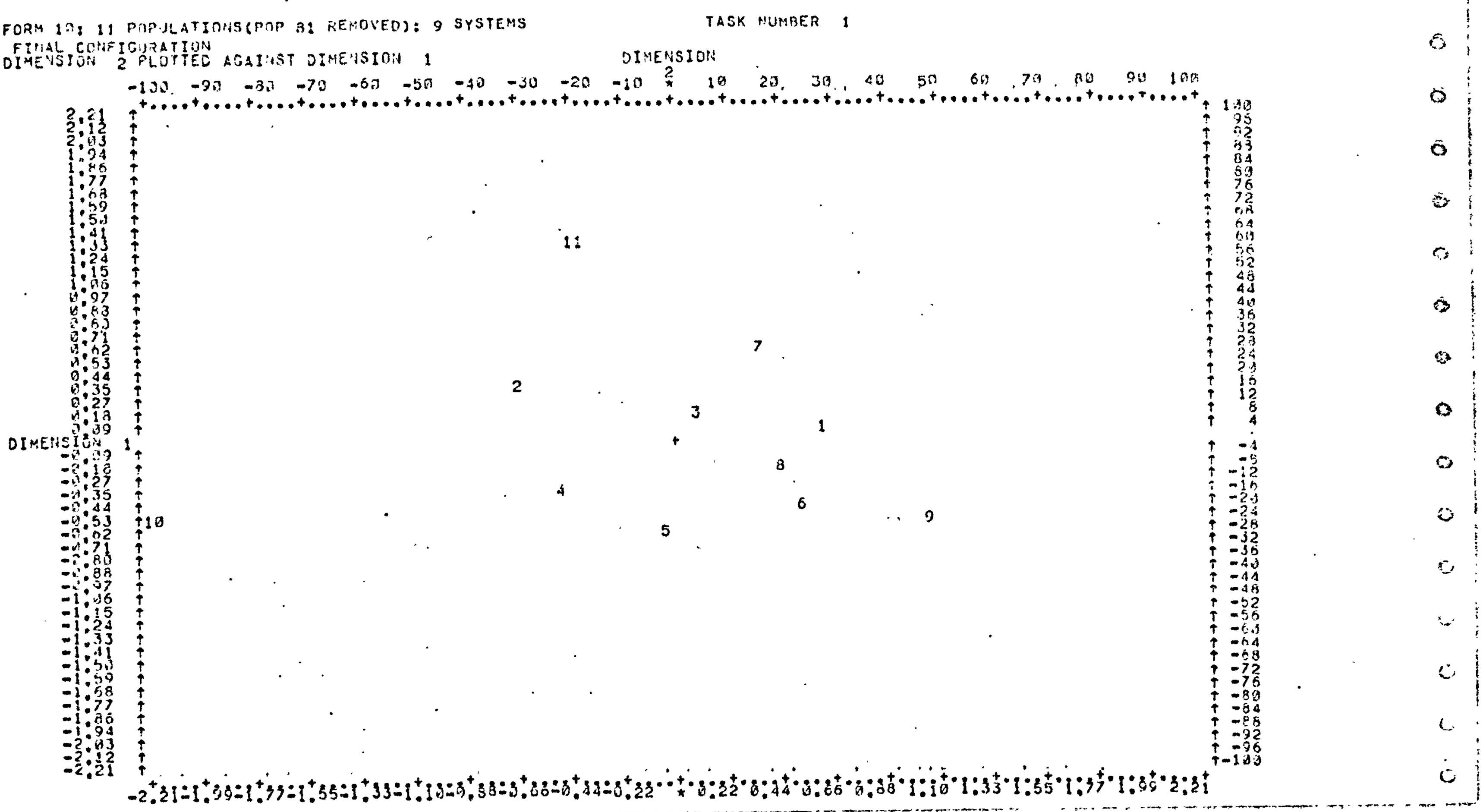


Fig. 5.4.3. 10a. Two- dimensional map of genetic distance for 11 Iranian and neighbouring populations.

Based on gene frequency data for 9 polymorphic loci.

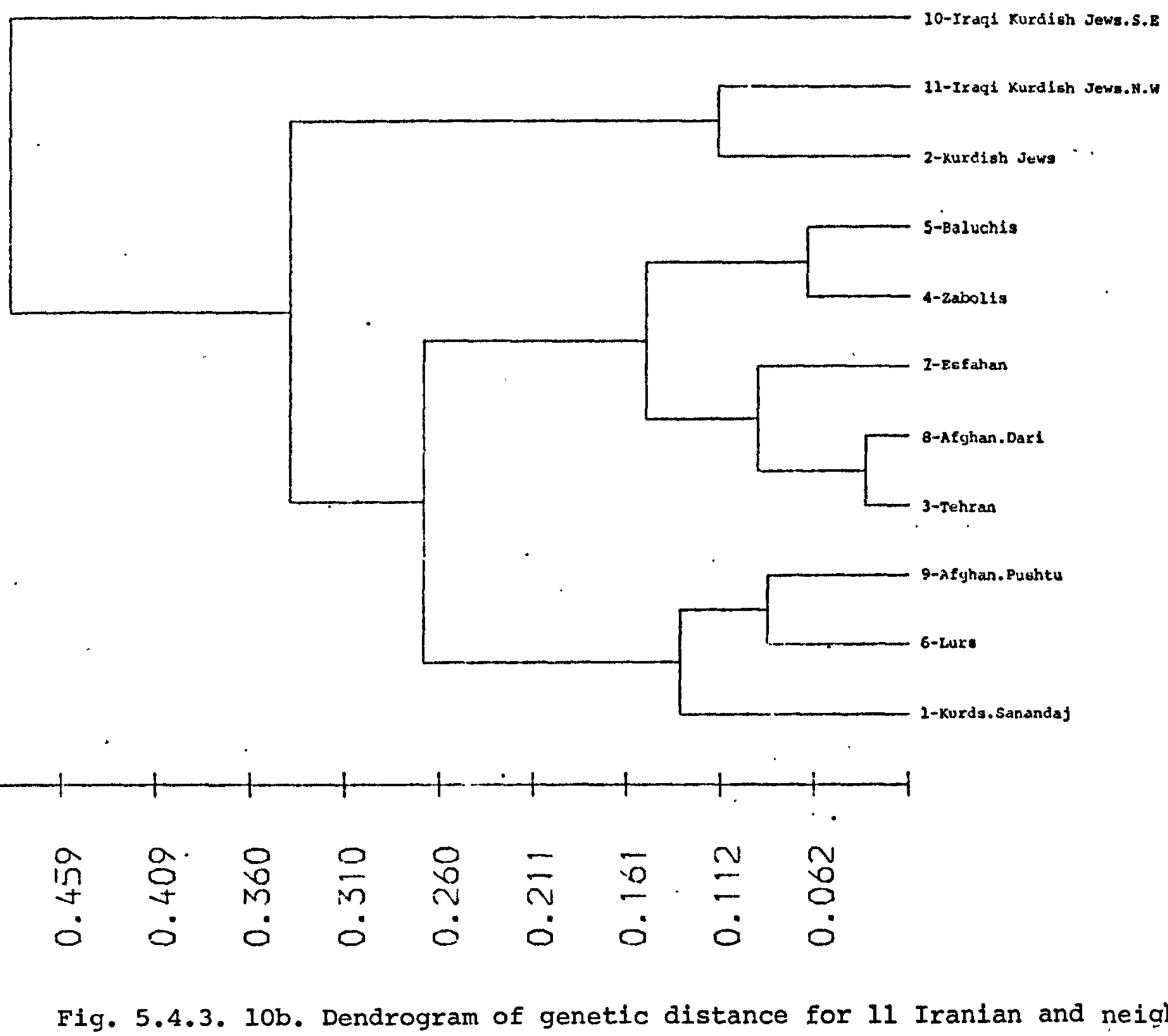


Fig. 5.4.3. 10b. Dendrogram of genetic distance for 11 Iranian and neighbouring populations. Based on gene frequency data for 9 polymorphic loci.

COEFFICIENT NUMBER 1 CALCULATED

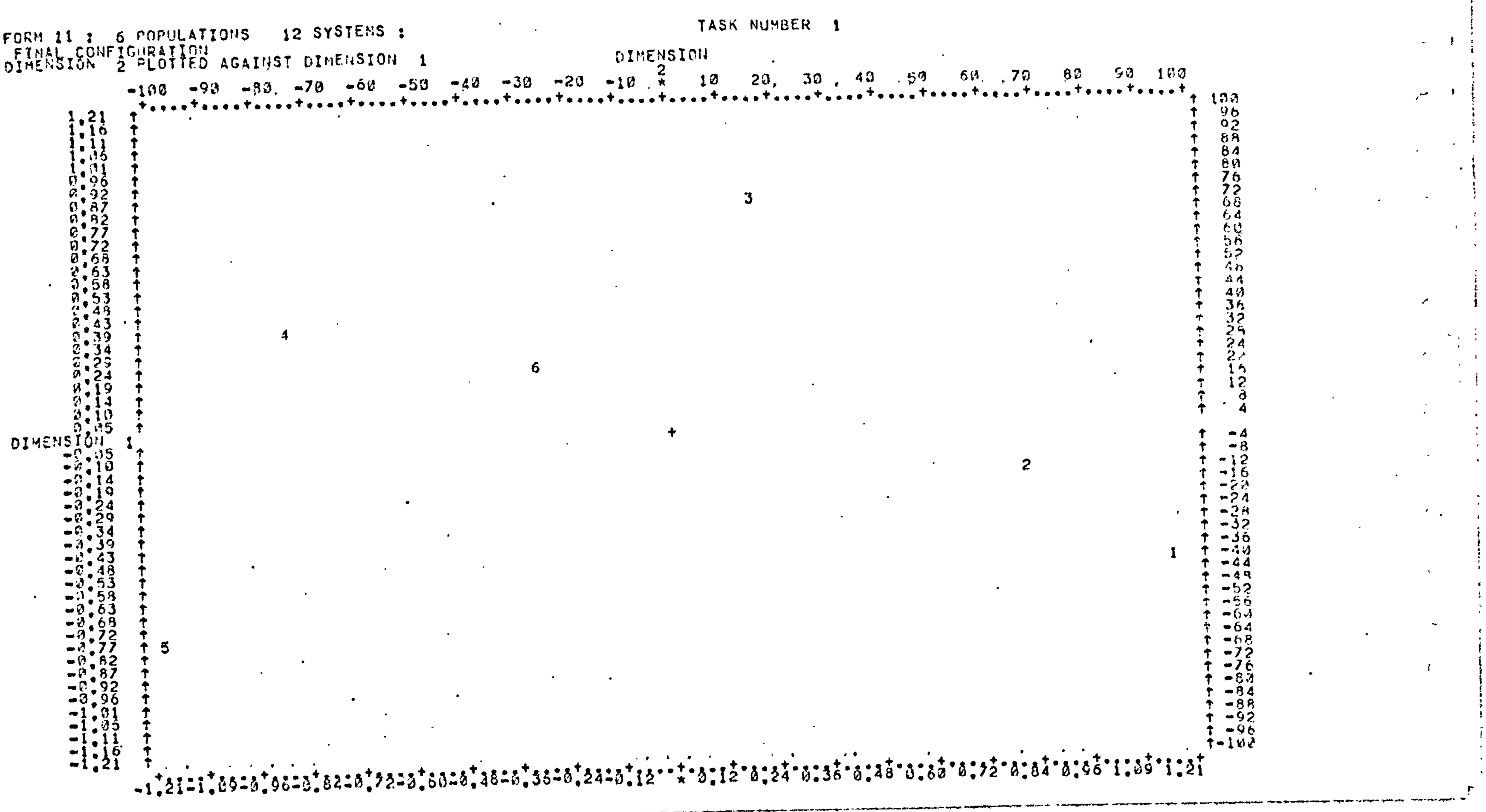


Fig. 5.4.3. 11a. Two- dimensional map of genetic distance for 6 Iranian populations, Based on gene frequency data for 12 polymorphic loci.

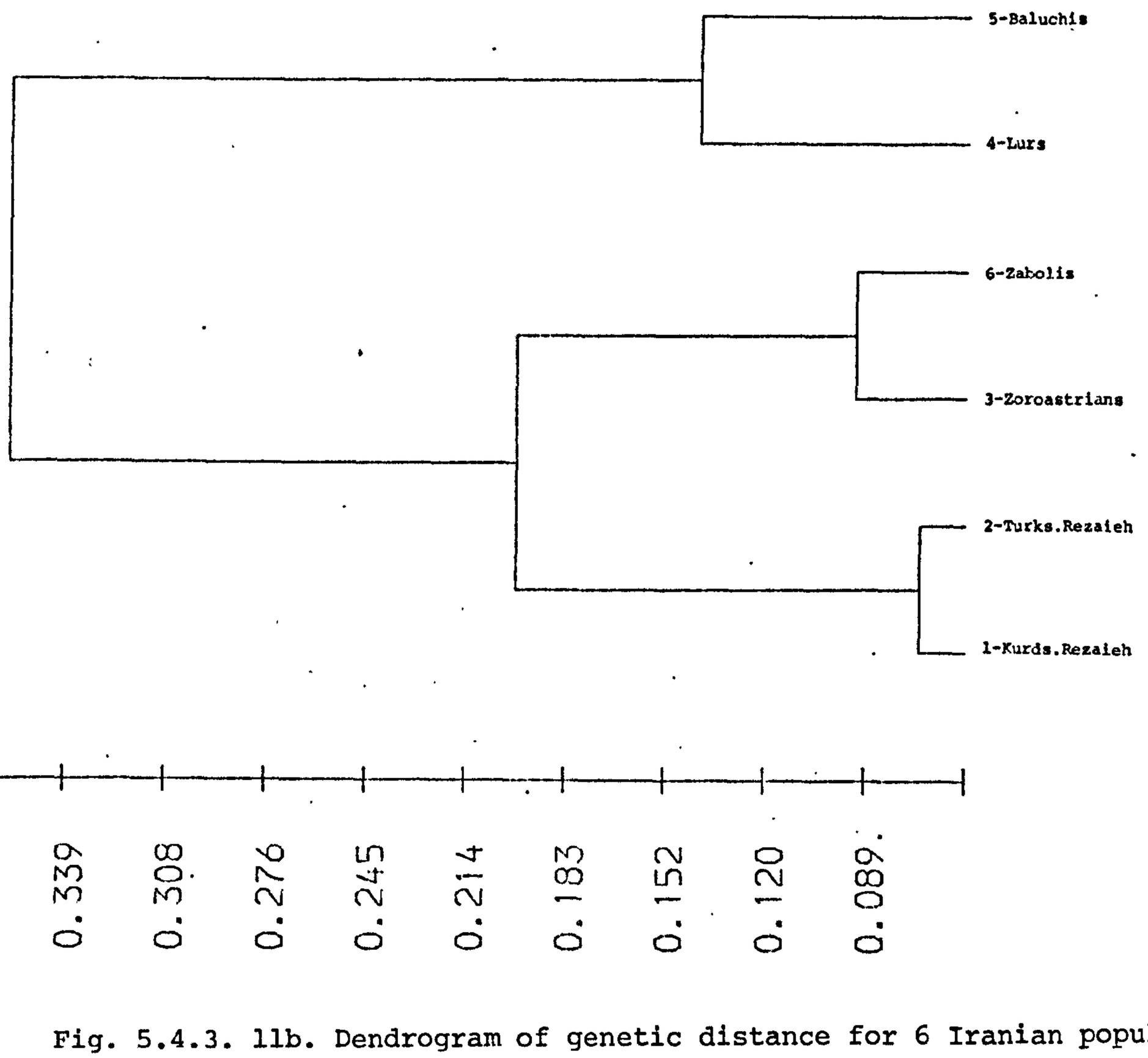


Fig. 5.4.3. 11b. Dendrogram of genetic distance for 6 Iranian populations.

Based on gene frequency data for 12 polymorphic loci.

5.4.4. Discussion

The various population groups from Iran and neighbouring countries have been compared with each other. The neighbouring groups consisted of the Kurdish Jews of the N.W and S.E Iraq, the Daris and Pushtus of Afghanistan and the Saudi Arabian Arabs.

No data, on the serological systems considered in the present analysis, was available for the other neighbouring populations.

Figures 1-11 a and b present the Non-Metric two dimensional scaling and the cluster analysis outputs, respectively, for the eleven sets of analysis produced using Edwards E^2 statistics.

As mentioned before, in the present study two types of analysis were used for the eleven possible combinations of the investigated populations and serological systems.It should be pointed out that on the one hand, the Non-Metric two dimensional scaling analysis and the cluster analysis may give slightly different relationships, so they must be used together and the outlying populations show relationships which agree in both types of analysis. On the other hand, the actual relationships generated by the eleven sets of analysis differ depending on the loci considered, thus, strict relationships should not be emphasised too much unless they appear in more than one set of analysis. Therefore, there will be no clearcut answer from any single analysis, and they have to be taken together.

In this sense, regarding all the eleven sets of analysis, those relationships which are confirmed in more than

one set of analysis will be discussed and the remaining doubtful ones will not be considered.

The results of the present analysis show that the Kurds and Turks of Rezaieh(north western Iran) are closely related to each other and both populations are distantly related to the Turks of Astara living in the same region. These findings seem to indicate that the populations of north western Iran are genetically homogeneous. All these three populations are related at a higher level to the Iranian Kurdish Jews and the Iraqi Kurdish Jews of the north west.

The Kurdish Jews of western Iran and the Iraqi Kurdish Jews of the N.W. are closer to each other than they are to the non-Jewish Kurds of western Iran, they show a moderate genetic resemblance to the indigenous Kurds, while those from south eastern Iraq differ considerably.

As mentioned above, while there is not a close resemblance between the Kurdish Jews and the Kurds, there is a recognizable similarity. This is more particularly so for the Kurdish Jews from western Iran and north western Iraq than for those of south eastern Iraq. The latter indeed differ so widely from any other populations in or near Iraq that is must be assumed that they are an extremely high inbred isolate(Tills et al, 1977).

The Arabs of Saudi Arabia seem to be related only to the Iraqi Kurdish Jews of the south east and unrelated at all to the remaining large number of populations included in the eleven sets of analysis. Generaly, the people of Saudi Arabia have some typical genetic characteristics which suggest a considerable amount of African admixture.

The Zoroastrians are closely related to the Esfahanis, living in nearly the same geographical location, and both populations seem to be distantly related to the Tehranis and Lurs.

The Zabolis and the Baluchis of Sistan and Baluchistan are closely related to each other and both populations seem to be distantly related to the Kermanis and at a higher level to the Zoroastrians and the Lurs.

Again, as in Rezaieh (north western Iran), the Kurds and Turks of Shirvan, Khorasan(north eastern Iran) are closely related to each other and both populations are distantly related to the Gonbadis living in the same location. It should be noted here that the Kurds and Turks of Rezaieh (north west) show no resemblance to the originally same two population groups of Shirvan, Khorasan (north east), the latter two groups being more closely related to the Gonbadis living in the same geographical location. These findings clearly indicate the strong influence of environmental factors on the genetic structure of populations.

The Afghan Daris and Pushtus, in general, both fit into Iranian clusters and are most closely related to the populations of Khorasan (north eastern Iran). The two Afghan groups are considered to be ethnically allied with Indo-Europeans, they are descendents of an Iranian-Aryan tribe.

Finally, the populations of the Caspian Littoral are not homogeneous, either in the geographical Zones they occupy or in their ethnic affiliations. The results of the present analysis seem to indicate they are also genetically heterogeneous.

5.5.Overall conclusion

The Iranian population is among the most heterogeneous in the world. Iran has been invaded by many peoples(i.e., Assyrians, Greeks, Arabs, Turks and Mongols), and each has left its imprint on the gene pool of the pre-existing population and together have contributed to the genetic diversity of present-day Iranian populations. Physical, religious and linguistic barriers have helped to maintain the genetic distinction of many ethnic groups of Iran.

The average gene frequencies for the whole country,of about 22 percent A; 17 percent B; 48 percent CDe(R_1); 28 percent cde(r); 63 percent M; 44 percent Fy^a; 27 percent HP^1; 13 percent $C3^F$; 66 percent AcP^B; 2 percent AcP^C; 6 percent AK^2; 31 percent PGM_1^2; 13 percent ADA^2; and 19 percent EsD^2, all show a departure from the values found in the countries to the west and an approach to those of the Indian region.

The connections of the dominant Iranians are to be sought largely among the relatively little-studied peoples to the north of them, but we might expect connections with the Arab peoples to the west and south-west across the Persian Gulf. The Arab population of south-western Iran, however, show little resemblance to the Arabs of Arabia. The Arabic speakers of Iran have the very high B gene frequency of average about 22 percent, relating them rather to the Arabs of Iraq, but suggesting possible affinities with India. The Iranian Arabs also have average about 14 percent of cDe(Ro), showing African admixture to be slight, though rather more than in most other Iranian populations

in whom cDe(Ro) is only a little above European levels. Of the other Arab characteristics, the Iranian Arabs have rather more K genes than other Iranian populations. Their M gene frequency is average about 61 percent, near the average for Iran. This is much lower than the average for Arabia. On the whole the serological evidence suggests that over a long period there has been little migration into Iran across the Persian Gulf. If this is so it implies that the high frequencies of M in Peninsular Arabia and those in the rest of southern Asia are distinct phenomena, since they are separated on the land connection between the two regions by populations with lower M levels.

The Iranian Armenians, who are recent immigrants,have, as everywhere else, high A and cde(r) frequencies.

The small community of Zoroastrians has attracted considerable attention since its member must be descended from the original Iranians whose religion before the Moslem invasions and conversions was Zoroastrianism. However, at that time and subsequently, many Zoroastrians have migrated to India to give rise to the Parsi community. In general, a high incidence of the gene B exists in all the Zoroastrian populations studied to date. It is known historically that the Zoroastrians of Iran have practiced endogamy since the Arab conquest in the 7th century. The high value of the gene B in the Zoroastrians, while possibly due to genetic drift, is thus more probably the result of the introduction of genes from India or due to the fact that the Zoroastrians were reservoirs of the B gene. On the whole, the pattern of the gene frequencies among Zoroastrians would not be very dissimilar from that of the Iranian Moslems, regarding a

number of serological systems. These may be attributed to the fact that the Zoroastrians of Iran once have been part of a large population in Iran which was later converted to Islam. However, the Iranian Moslems suffered from incursions of various peoples, leading to the introduction of new genetic characteristics.

The Kurds form a numerous and distinct ethnic group in the north west and west of Iran. Serologically they differ little from the Iranians proper and it has been suggested on serological grounds that the latter are the results of mixing of populations similar respectively to the present Kurds and to the Turkish speaking Ghashghais.

One reason for the present interest in the Kurds is the fact that the Kurdish Jews, who lived among them but have now mostly migrated to Israel, have the highest frequencies known of genetically determined glucose-6-phosphate dehydrogenase deficiency. There are substantial frequencies of this condition in most countries of the Near East, both in the indigenous populations (especially the Shia Arabs of Saudi Arabia) and in Jews, but in general the condition is commoner in Jews.

Quite high frequencies of G6PD deficiency are found in the indigenous populations in many parts of Iran, but since one of the main determining conditions is probably a selective advantage of carriers of the gene, in the presence of endemic falciparum malaria, the very much higher frequency in the Kurdish Jews than in Kurds poses a special problem.

The Jews of Iran show considerable genetic heterogeneity, but nearly all the communities have two features in common,

high frequencies of B and Glucose-6-phosphate dehydrogenase deficiency.

Regarding neighbouring populations, the average gene frequencies for the inhabitant of the Caucasus, of about 24 percent A; 11 percent B; 41 percent CDe(R_1); 35 percent cde (r); 50 percent P_1; and 32 percent HP^1, all show relationships rather with the west than with the east.

The average gene frequencies for the population of Turkey, of about 29 percent A; 13 percent B; 32 percent cde(r); 56 percent M; 4 percent AK^2; 10 percent ADA^2; and 2.5 percent PGD^C, all suggest western rather than eastern connections.

The average gene frequencies for the inhabitant of Iraq, of about 24 percent A; 19 percent B; 48 percent CDe(R_1); 27 percent cde(r); 60 percent M; 68 percent AcP^B; 3 percent AcP^C; 17 percent ADA^2; and 22 percent EsD^2, all show relationships rather with the east than with the west.

The Arab populations of Kuwait, Saudi Arabia and the United Arab Emirates with the average gene frequencies, of about 16 percent A; 11 percent B; 71 percent O; 65 percent M; 6 percent K; 21 percent Fy^a; 27 percent Fy null; 6 percent JK^a; 39 percent HP^1; 9 percent $C3^F$; 15 percent Gc^2; 20 percent AcP^A; 78 percent AcP^B; 1.5 percent AcP^C; 3 percent AK^2; and 7 percent PGD^C, in general, resemble North Africans. The factors so far mentioned can be regarded as original features of the desert populations, and the isogenic lines for them form closed Curves around Arabia. There are, however, a number of other blood group genes which reach considerably higher frequencies in Arabia than anywhere in Europe, but which reach much higher frequencies still in Africa south of

Sahara. These are Henshaw (He) in the MNSs system, cDe(Ro) and V in the Rh system, JS^a in the Kell system. For some of these, the data are still inadequate but in every case where the data are sufficient the isogenic lines show an area of high frequencies in Africa extending into Arabia.

The high frequencies of O,M,S, and K are clearly original characteristics of the Arabs, maintained by the isolation of the desert dwellers. They may to some extent have been maintained also by natural selection in the desert environment. The high frequencies of the typical Negroid genes must certainly be attributed in the main to incorporation of Negro slaves into the population, a process which seems to have gone on for thousands of years. Raised frequencies of these genes extend into all the Arab peoples, well beyond the limits of the Peninsular isolate, and obviously for the same reason. The long-settled Shia Moslems of the eastern Oases of Saudi Arabia, which were until recently highly malarious also have very high frequencies of glucose-6-phosphate dehydrogenase deficiency, a condition which, like sickling, due to Haemoglobin s,is known to protect against falciparum malaria. In some villages the frequency of the enzyme deficiency is almost as high as the extreme values found in the Kurdish Jews. The frequency of G6PD deficiency in the members of Sunni sect who entered the area more recently is almost certainly considerably lower than the Shia.

The average gene frequencies for the population of Pakistan, of about 19 percent A; 25 percent B; 55 percent 0;6@ percent $CDe(R_1)$; 24 percent cde(r); 63 percent M; 58 percent Fy^a; 25 percent HP^1; and 13 percent AK^2, all suggest connections with India rather than with neighbouring countries to the

west.

In Pakistan, there is a rapid increase of B gene frequency from below 23 percent to above 29 percent, and the latter level is maintained throughout almost the whole of west Pakistan, northern and Central India. Changes in A and 0 gene frequencies are less striking but there is a gradual fall in the frequency of the A gene from above 28 percent in Pakistan to values below 15 percent in India.

The 0 gene fluctuates mostly around 55 percent in Pakistan rising above 65 percent in India. The frequency of the M gene is high(average 63%) in Pakistan and this is one of the main ways in which its peoples differ serologically from the Mediterranean peoples in the strict sence. However, the presence of a relatively high frequency of the MS combination is typically Caucasoid. Rh frequencies scarcely differ from those of typical Mediterranean populations, with about 60 percent CDe(R_1), 24 percent of cde(r) and other combinations mostly below 10 percent. Both CDE(R_Z) and Cde(r') are present to the extent of about 2 percent each, and such frequencies are typical of the Indian region. Southward and eastward CDe rises and cde falls in frequency.

Generally, as in the Indian region, the frequency of B is high in Pakistan. The high B frequency in Pakistanis and Indians is in some way a response to the environment, and a result of natural selection acting over the last 4000 years.

The average gene frequencies for the Afghan population, of about 20 percent A; 25 percent B; 53 percent CDe(R_1); 23 percent cde(r); 64 percent M; 57 percent FY^a; 26 percent HP^1; 12 percent $C3^F$; 69 percent AcP^B; 1 percent AcP^C;

8.5 percent AK^2; 24 percent PGM_1^2 ; and 13 percent ADA^2, all show both Caucasoid and Mongoloid characteristics. They have average 20 percent of A genes, including 5 percent of A2 which is within the European range, they have also 25 percent of B which is far from being so. Their M gene frequency of 64 percent is typically Asiatic, but the presence of 24 percent of MS shows Caucasoid associations. In the Rh system the cde frequency of 23 percent is very low for a Caucasoid population, whilst cDE level of 19 percent suggests central Asiatic relationships, and the presence of Di^a positive is clearly a Mongoloid feature. They thus resemble many of the peoples living among the mountains on the northern boundary of the Indian region, in showing characteristics of both major races.

CHAPTER 6

The ability to taste phenylthiocarbomide (PTC)

6.1.Introduction

A chance observation by Fox in 1932 was the first to demonstrate the polymorphism with respect to taste sensitivity to the simple chemical compound phenylthiocarbamide (phenylthiourea or PTC). He showed that some people are unable to taste this substance, which others describe as very bitter. Being a chemist he also showed that a considerable number of other closely related compounds, all containing the chemical grouping H-N-C= S, were tasted by the PTC tasters but not by the non-tasters.

It has been shown that this dimorphism in sensitivity is inherited as a simple Mendelian character, the ability to sense the bitterness of PTC being predominant. (Blackeslee and Salmon, 1931; Snyder, 1932).Thus, the inability to taste high concentrations of this group of substances caused by the presence of an autosomal recessive gene in homozygous form. There is however, incomplete penetrance of the taster gene. However, the simple genetic basis of this trait is that non-tasters are homozygous recessive(tt), and tasters either homozygous or heterozygous dominants (TT, or Tt).

Since at this time very few genetical polymorphisms were known in man, this pair of characters was rapidly and extensively applied by geneticists to the study of families and of populations. This is one of the few commonly investigated polymorphisms in man which does not depend upon blood tests.

Unfortunately the early workers, as well as some more recent ones, treated the distinction between tasters and non-tasters as absolute.Most early workers used a rather strong solution, whilst others used the pure crystals, or pieces of filter paper soaked in the solution and dried.Results tended to be erratic, with apparent frequencies of tasters varying widely in the same or closely related populations, or between males

and females in one population.Harris and Kalmus(1950)showed that the distinction was by no means an absolute one, and that reliable results could be obtained only by the use of solutions of known concentration. They devised a method for ascertaining the lowest concentration which could be tasted by each person. They prepared a saturated solution of PTC in tap water, and from this a series of twofold dilutions. Starting with the weakest solution, the various dilutions are successively presented to the subject until he claims to be able to taste one. Two glasses of this dilution and two of tap water are then presented, and he is asked to say which is which. If he answers correctly the dilution is taken to mark his threshold, but if he gives the wrong answer the experiment is repeated with the next strongest solution, and so on.

Nearly every population shows a bimodal distribution of thresholds with a clear-cut intermediate dilution level at which few or no thresholds fall. Those who can taste solutions more dilute than this critical value are classed as tasters, and those thresholds fall at higher concentrations, are non-tasters.

Thus, purely as a taxonomic character the taster phenomenon has not so far proved to be of great value. It is, nevertheless, of great potential importance in population studies because of its relation to thyroid function and its possible involvement in detectable natural selective effects.

The history of this line of investigation goes back to a date before Fox discovered the taster character in man. In 1928 Chesney et al studied goitres in rabbits fed exclusively

on cabbage. Subsequent work showed that the disturbance of thyroid function must have been due to the presence of 1-5-vinyl-2-thio-oxazolidone(goitrin) in these plants,and that a number of other related compounds had a similar effect on the thyroid: this led to the adoption of thiouracil as an antithyroid drug for treating cases of human hyperthyroidism. It later became clear that it was precisely those compounds which showed the taster effect which had antithyroid activity and that, there is a considerable group of substances which are repressors of thyroid activity and to which individuals respond similarly either as tasters of all or as non-tasters of all.(Harris and Kalmus,1949).They mention the importance of the occurence of such substances in food plants and conclude, 'Differences in taste sensivity may reflect differences between individuals with respect to these substances. Such metabolic differences could presumably affect the biological fitness of the three genotypes (TT, Tt, tt) and hence lead to a balanced polymorphism.

Work on the association between chemical constitution and physiological activity was summarized by Harris and Kalmus (1949). This work led to the investigations by Harris and Kalmus, and Trotter (1949) and subsequently by Kitchin et al (1959) of the distribution of taste sensitivity among patients suffering from thyroid disease. Both groups of workers agreed in finding a lower frequency of tasters in cases of nodular-non-toxic goitre, and Kitchin et al also found a significantly raised frequency of tasters in patients with diffuse toxic goitre. Several subsequent investigations in different countries have confirmed these results, but in the

Japanese who normally have a low frequency of non-tasters and in south American Indians who are nearly all tasters, no significant association between goitre and tasting could be found.

Most of the early series of non-toxic goitres were sporadic rather than endemic, but in the Netherlands and in Israel persons with endemic goitre have shown a lowered frequency of tasters.

Moreover, in persons without thyroid disease, Widstrom and Henschen(1963) have shown that there is a statistical correlation between the level of protein-bound iodine in the serum and the ability to taste PTC, persons with a high iodine level (indicating high thyroid activity) being more frequently tasters than those with a low level. This work appears convincing and is consistent with observations on persons with goitres, suggesting that the goitrous cases represent the clinically abnormal extremes of the normal thyroid function range. Unfortunately this work has never been repeated on adequate numbers of subjects, but the results of Becker et al (1966) who investigated relatively small numbers of patients for a possible association between PTC tasting and glaucoma, are in agreement with those of Widstrom and Henschen. But an extensive unpublished survey by Tills and Lehmann(1975), shows no significant differences in thyroxine levels between tasters and non-tasters.

While the situation demands much further work in order to clarify it, many of the above mentioned facts would accord with the suggestion by Harris and Kalmus(1949) of a balanced polymorphism related to the amount of iodine and of thyroid-

inhibiting substances in diet, the frequency of tasters, who fix iodine more efficiently, becoming raised by natural selection to cope at a population level with a low environmental iodine supply or an abundance of thyroid inhibitors,and lowered to meet the opposite conditions. To revert to the hypothesis of a balanced polymorphism, one set of observations which gives some support to it is that of Cartwright and Sunderland(1967) who have shown that the higher incidence of endemic goitre is accompained by a higher incidence of non-tasters.

The implication of the above hypothesis is that the products of the taster and non-taster genes affect not only the ability to taste phenylthiocarbamide and related substances,but also in some way regulate thyroid function. They must therefore be active both in the tongue (for a direct action on the nerves of taste is most unlikely) but also in the thyroid gland(or, rather improbably, the pituitary). The combination of effects on the tongue and the thyroid is not surprising in view of their embryological association.

An indication that the taster phenomenon is of importance in relation to the endocrine balance of healthy persons is the finding by Johnston et al (1966) of a tendency of earlier pubertal development in tasters than in non-tasters. This is perhaps related to a higher level of thyroid activity.The lower incidence of dental caries in tasters than in non-tasters found by Chung et al (1964) is perhaps a related phenomenon.

Ever since the discovery of thyroxine, if not before, it has been realized that goitre distribution is inversely re-

lated to the amount of iodine in the diet (especially the drinking water). Other inorganic (and possibly organic) substances may be involved. A considerable number of the above themes are brought together in a comprehensive review paper by Fischer(1967).

Taking all the evidence into account it may be suggested as a working hypothesis that the occurence of goitre depends, probably among other factors, on the amount of iodine and of antithyroid substances in the diet, and on the PTC taster status of each individual.

It is already clear that taster status is a biochemical character the effects of which extend far beyond the taste papillae of the tongue. It would be most satisfactory if the cumbersome taste test could be replaced by a biochemical test on blood, or perhaps a metabolic test with ingestion of a thiocarbamide derivative and measurement of a metabolic product in the urine.

As a long-term result one might hope to prove the existence of a balanced polymorphism, with an equilibrium betwe-en the taster and non-taster genes depending upon the amounts of iodine and of thiocarbamide derivatives in the diet, and also probably upon the demands of the physical environment for a high or a low level of general metabolism and hence of thyroid activity.

While the presence of a relation between the taster phenomenon and thyroid function is beyond doubt, whatever may be its explanation, the same cannot be said of the suggested relation of tasting to glaucoma. There are a number of papers, mostly of rather early date, suggesting an association between goitre and glaucoma. Becker and Morton (1964 a, b) have found a raised frequency of non-tasters in primary open-angle glaucoma,and of tasters in angle-closure glaucoma, both in Caucasoids and Negroids.

Other conditions which, on adequate numbers, show a strong association with tasting are tuberculosis and leprosy while, on rather small numbers, there are very strong associations with carcinomas of the breast, cervix and corpus uteri, and ovary, and with menopausal metrorrhagia. There exists a highly significant association between diabetes mellitus and non-tasting. Apparent associations of cystic fibrosis and schizophrenia with non-tasting may be due to the patients failing to taste PTC, or to report tasting it, for reasons other than being genetic non-tasters.

6.2.Material and method

Tests for ability to taste PTC were made on six out of the ten Iranian population groups. A total of 1114 individuals was examined.

The method used was a modification of the Harris-Kalmus two stage (a subjective followed by an objective test) technique (Harris and Kalmus 1949).

The test is in two parts, the first giving the approximate and the second the true threshold.

All solution from number 1-11 were used. The dilutions and

controls were made up with local tap water and administered at room temperature. Fresh solutions were made regularly and all the tests were performed in the same way. The strongest solution used,number one, contains 1300 mgs per litre and this is then progressively diluted as follows:

Concentration of PTC solutions

Solution number	PTC mg /litre
1	1300.00
2	650.00
3	325.00
4	162.50
5	81.25
6	40.63
7	20.31
8	10.16
9	5.08
10	2.54
11	1.27

Typically, in populations tested so far, there is a bimodal distribution of tasting acuity.

At whatever solution number the antimodal value falls, let as assume at solution 4, half the frequency of individuals tasting at that solution number are allocated to the taster category with solution numbers 5,6,7,8,9,10 and 11, and the other half to the non-taster category including solutions 1, 2 and 3 as well as complete non-tasters(Sunderland 1966; Mitchell 1972).

The method of determining PTC taste thresholds is very suitable for field conditions.

6.3. Results

Table 6.3. demonstrates the distribution of PTC taster and non-taster phenotypes and respective gene frequencies in the six Iranian samples.

The frequency of the allele t ranges from 23.50 percent in the Zoroastrians to 49.36 percent in the Turks of Shirvan, Khorasan.

6.4. Discussion and conclusion

The distribution of PTC taster and non-taster phenotypes and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 6.4.

The frequency of the allele t ranges from 23.50 to 52.40 percent in Iranians, being lowest in the Zoroastrians (present study) and highest in the Kurds of Sanandaj(Lightman et al, 1970). With an average t gene frequency of 41.14 percent, the Iranian population appears to exhibit a lower t gene frequency than that of around 50 percent found in Europeans(Mourant et al, 1976).

Regarding neighbouring groups, the frequency of the gene t in the populations of the Caucasus varies between 41.32 percent in the Kakhetians, Gurdjaani(Voronov, 1973) and 61.03 percent in the Imeretians of Georgia (Bunak, 1960). With an average t gene frequency of 49.43 percent, the population of the Caucasus seems to show a higher t gene frequency than that found in Iranians but slightly lower than the European frequency.

The t gene frequency of 44.67 percent in the Turks of Turkey (Say,1966) is also higher than the average for Iran but lower than the frequency in Europe.

The frequency of the gene t in the populations of Iraq ranges from 41.55 percent in the Jews to 54.31 percent in the Moslems of Baghdad(Boyd and Boyd, 1941). With an average t gene frequency of 48.77 percent, the Iraqi population also appears to exhibit a higher t gene frequency than that found in Iranians but lower than the European frequency.

The t gene frequency in the populations of Pakistan varies between 48.23 percent in the Chitrali(Graziosi,1964) and 75.59 percent in the East of Indus sample of Bernhard (1967). With an average t gene frequency of 60.55 percent, the Pakistani population seems to show a much higher t gene frequency than that found in Iranians and even much higher than the European frequency. High frequencies of the gene t in Pakistan seem to be more similar to the higher values found in India.

Conclusion

Throughout Europe the frequency of non-tasters is not far from 30 percent, corresponding to a frequency of the non-taster gene of a little above 50 percent. The relatively few results from other regions show that there are significantly fewer non-tasters among Africans than among Europeans and that a very large proportion of American Indians are tasters (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Pakistanis with their much higher frequencies which are more similar to the high values in Indians,frequencies of the gene t appear to be a little under European levels.

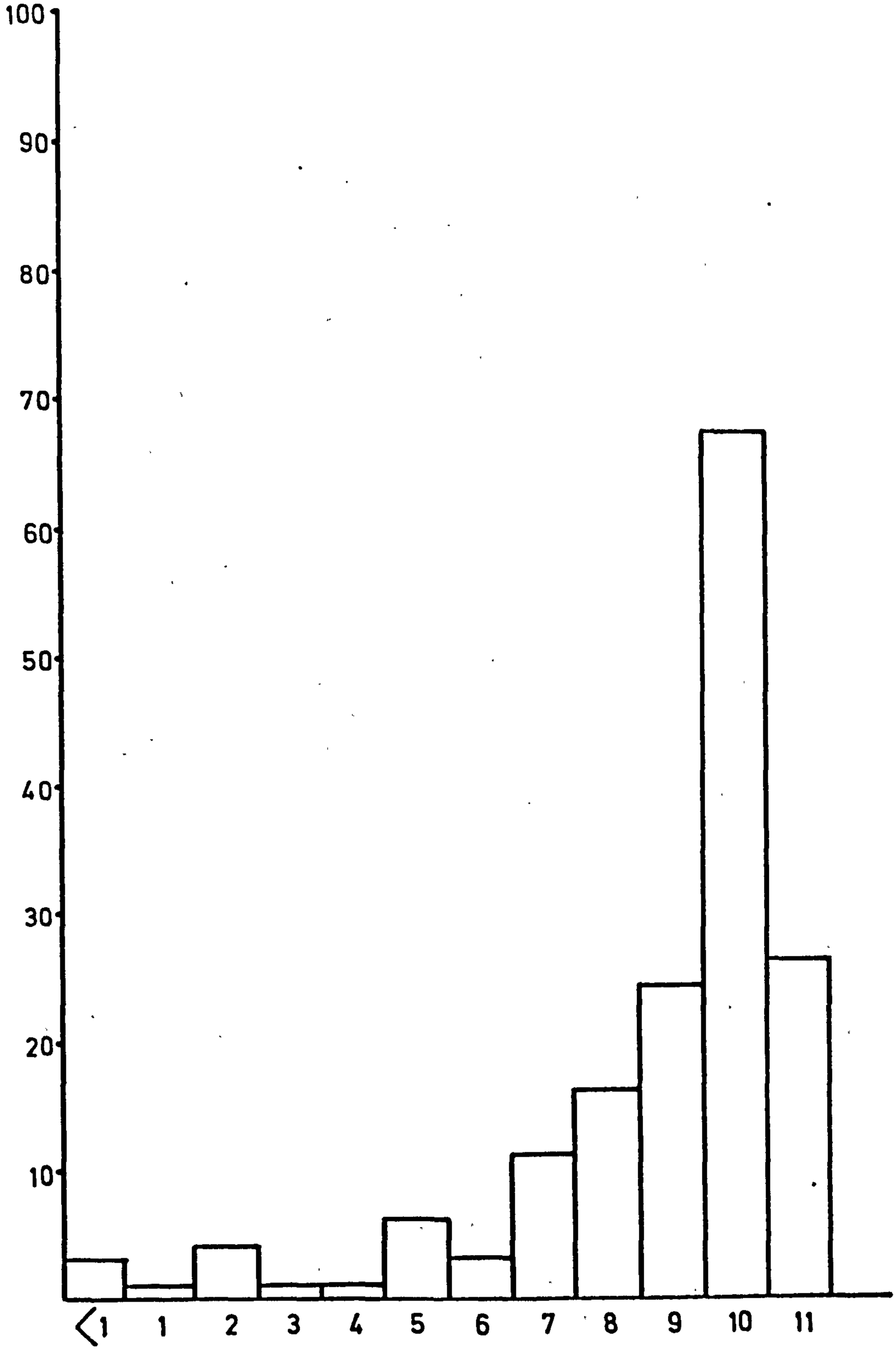


Fig. 6.3.1. The distribution of taste thresholds for P.T.C of the Zoroastrians.

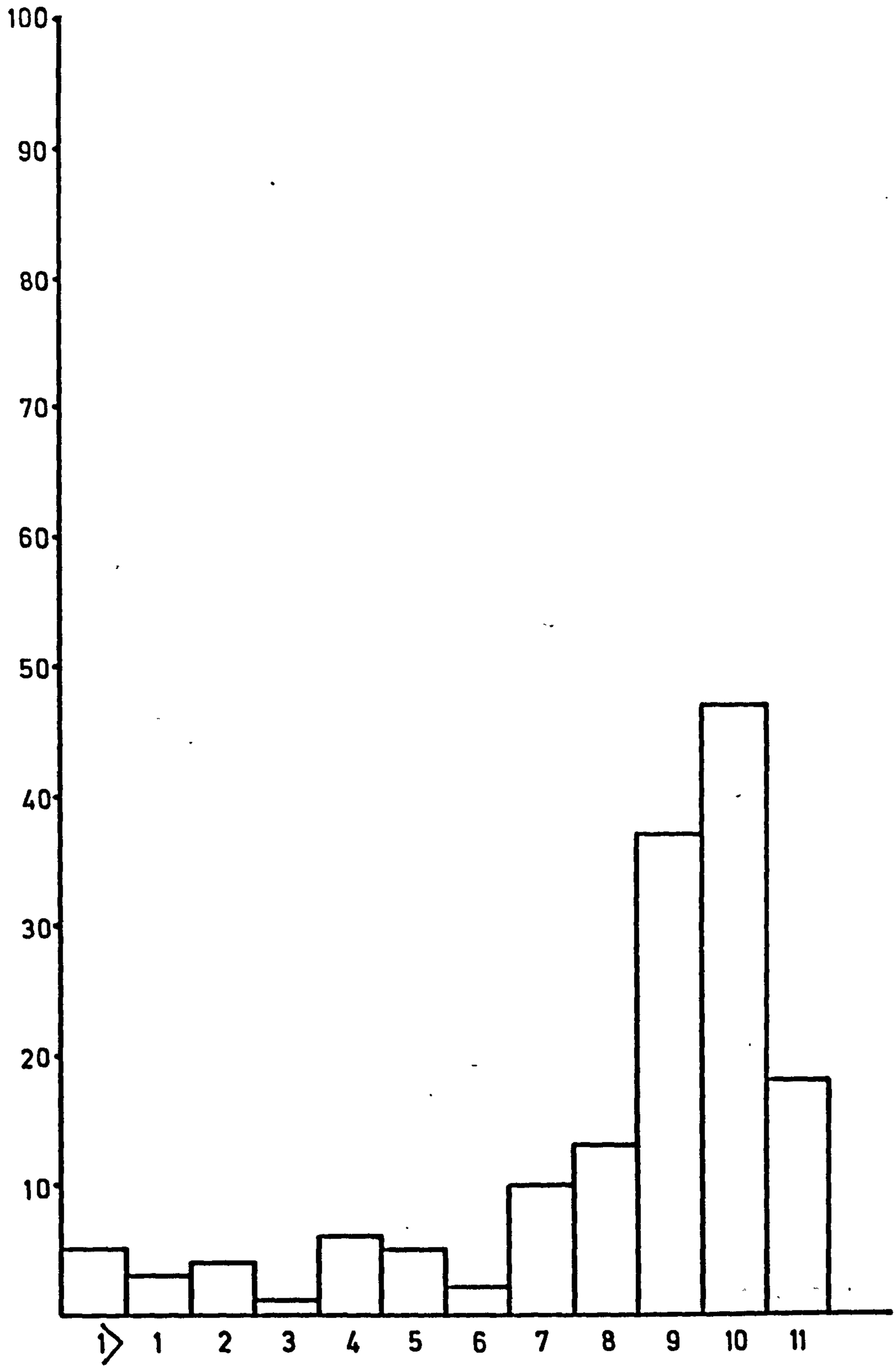


Fig. 6.3.2. The distribution of taste thresholds for P.T.C of the Kurds of Rezaieh.

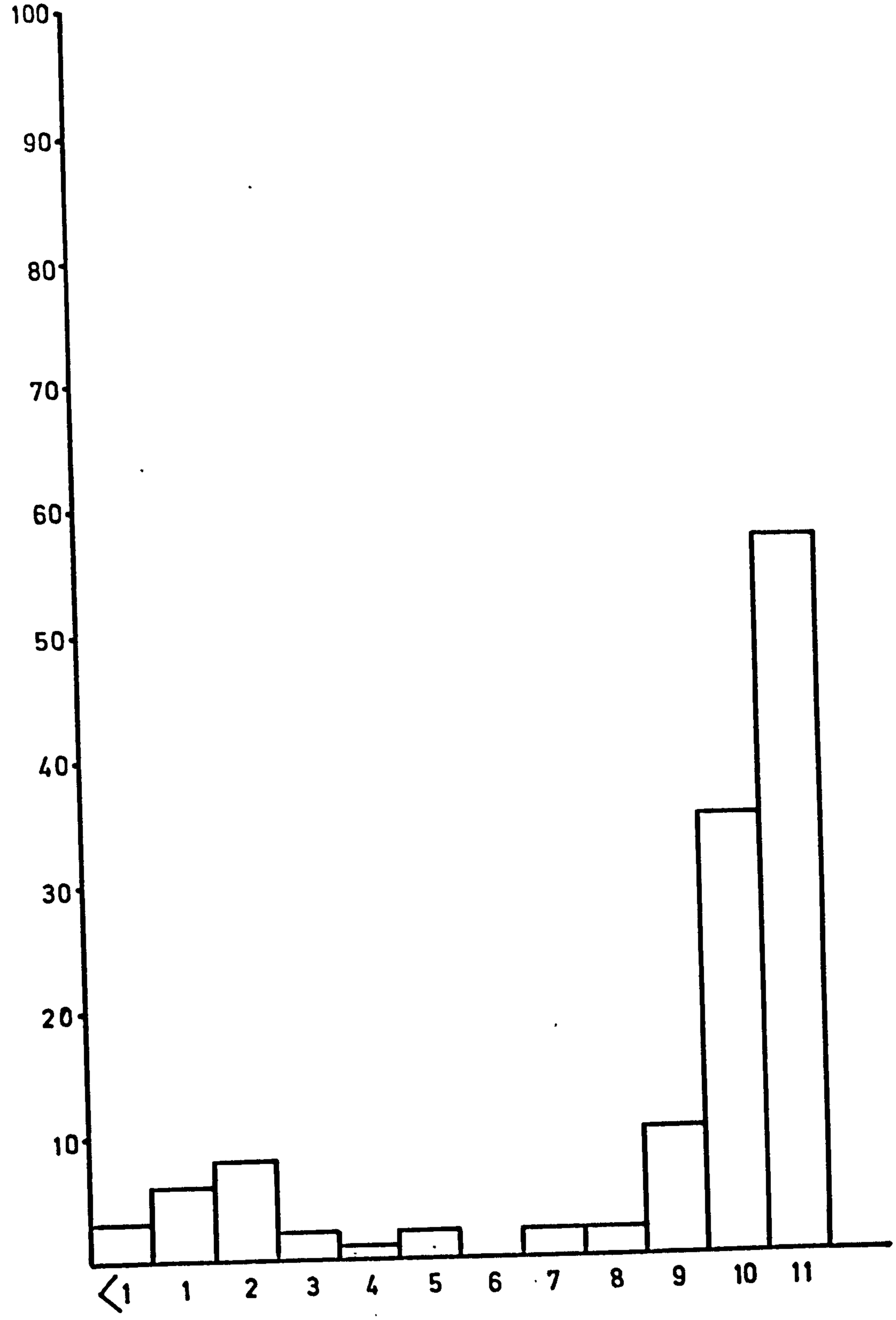


Fig. 6.3.3. The distribution of taste thresholds for P.T.C of the Kermanis.

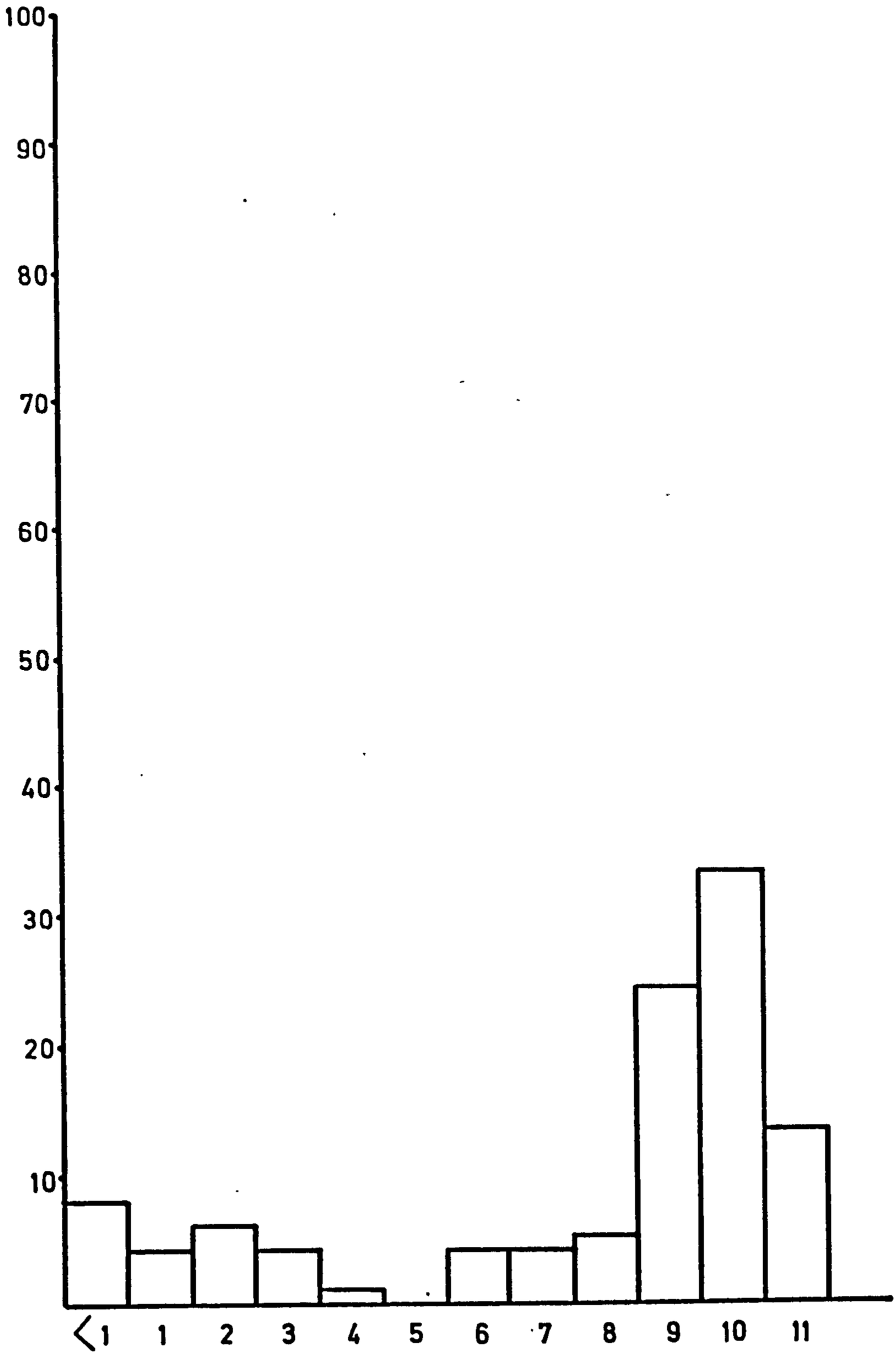


Fig. 6.3.4. The distribution of taste thresholds for P.T.C of the Kurds of Shirvan, Khorasan.

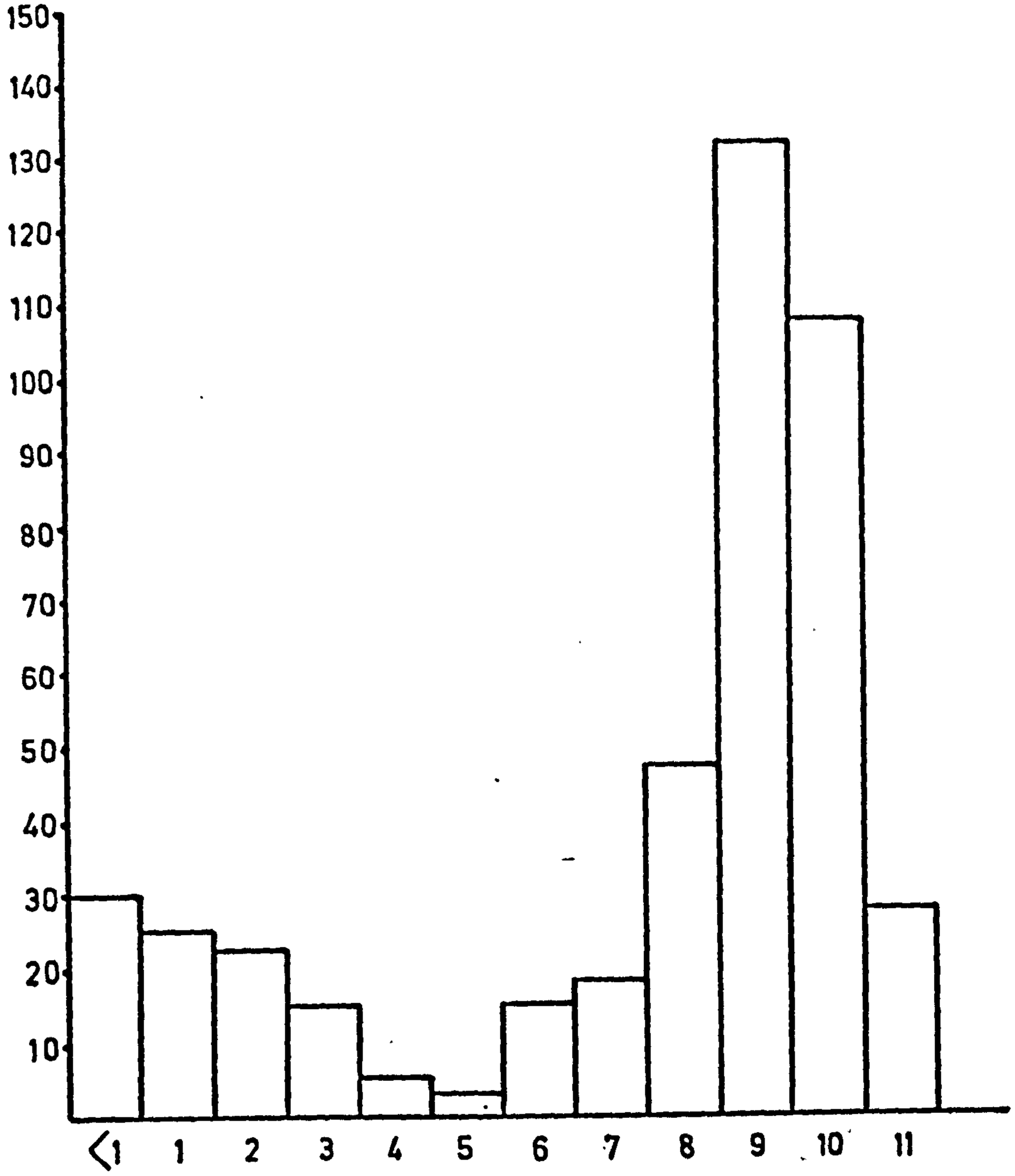


Fig. 6.3.5. The distribution of taste thresholds for P.T.C,of the Tehranis.

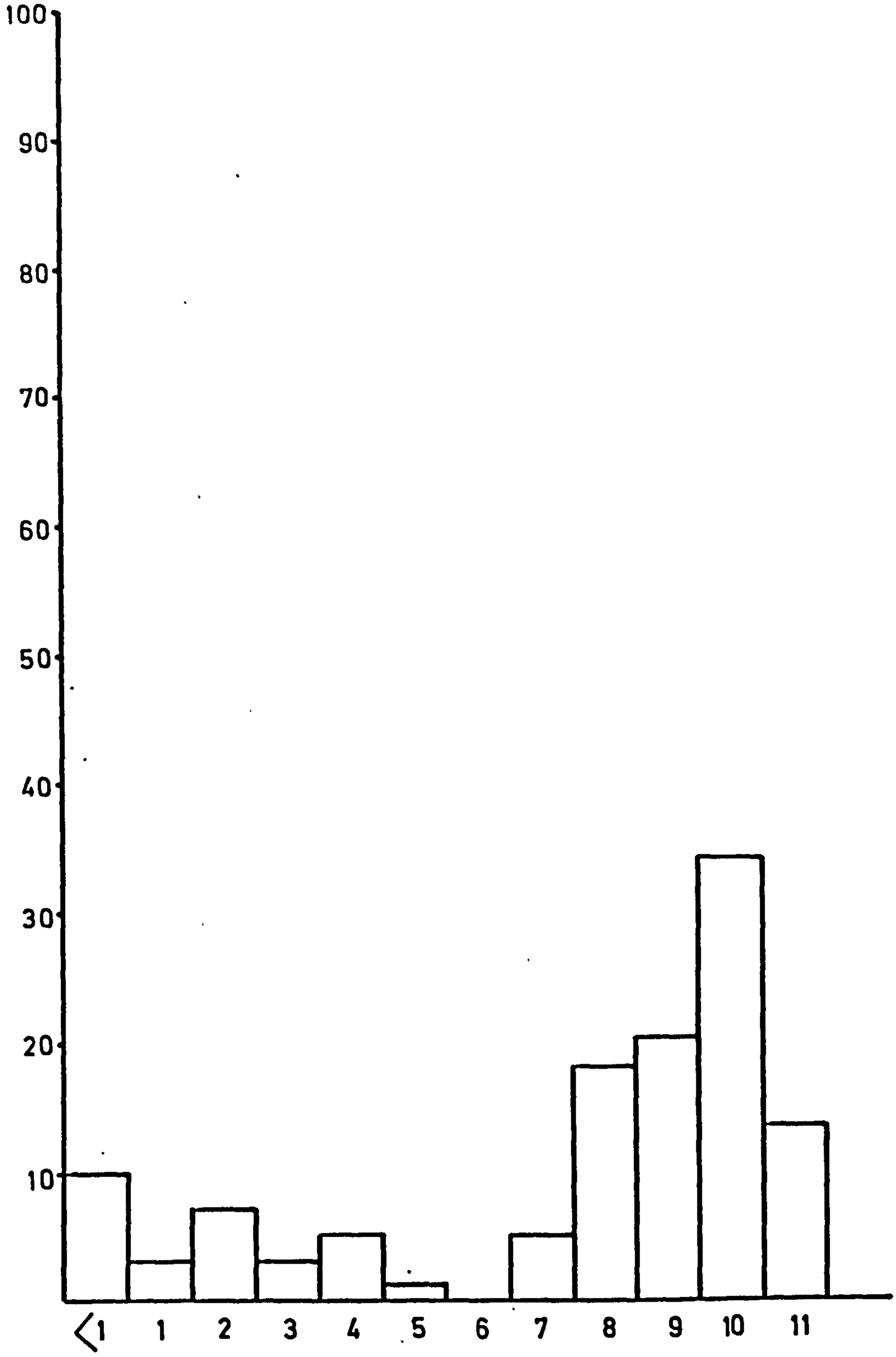


Fig. 6.3.6. The distribution of taste thresholds for P.T.C of the Turks of Shirvan, Khorasan.

Table 6.3. Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes		Gene frequencies	
		Tasters	non-tasters	T	t
1-Zoroastrians	163	154	9	76.50	23.50
2-Kurds.Rezaieh	151	138.50	12.50	71.23	28.77
3-Kerman	128	106	22	58.54	41.46
4-Kurds.Shirvan,Khorasan	106	83	23	53.42	46.58
5-Tehran	447	348.50	98.50	53.06	46.94
6-Turks.Shirvan,Khorasan	119	90	29	50.64	49.36

Table 6.4.I Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in Iran

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Tasters	Non-tasters	T	t	
1-Zoroastrians	163	154	9	76.50	23.50	Present study.
2-Kurds.Rezaieh	151	138.50	12.50	71.23	28.77	Present study.
3-Jews	336	282	54	59.91	40.09	Sheba,C.,et al. 1962
4-Kerman	128	106	22	58.54	41.46	Present study.
5-Kurds.Shirvan,Khorasan	106	83	23	53.42	46.58	Present study.
6-Tehran	447	348.50	98.50	53.06	46.94	Present study.
7-Turks.Shirvan,Khorasan	119	90	29	50.64	49.36	Present study.
8-Kurds.Sanandaj	346	251	95	47.60	52.40	Lightman,S.L.,et al.1970

Table 6.4.II Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in the Caucasus

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		Tasters	Non-tasters	T	t		
1-Kakhetians.Gurdjaani	205			58.68	41.32	Voronov,A.A.	1973
2-Azerbaijanians.Nukha	196			56.55	43.45	Voronov,A.A.	1973
3-Kakhetians.Georgia	124	100	24	56.01	43.99	Bunak,V.V.	1960
4-Azerbaijanians. Shemakha	180			52.86	47.14	Voronov,A.A.	1973
5-Western Georgia	455	349	106	51.73	48.27	Boyd,W.C.,& Boyd,L.G.	1937
6-Kartalins.Georgia	121	91	30	50.21	49.79	Bunak,V.V.	1960
7-Azerbaijanians.Barda	158			46.05	53.95	Voronov,A.A.	1973
8-Gurians and Mingrelians Georgia	32	22	10	44.10	55.90	Bunak,V.V.	1960
9-Imeretians.Georgia	204	128	76	38.97	61.03	Bunak,V.V.	1960

Table 6.4. III Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in Turkey

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Tasters	Non-tasters	T	t	
1-Ankara. Asia Minor	2000	1601	399	55.33	44.67	Say,B.,et al. 1966

Table 6.4. IV Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in Iraq

Population	Number Tested	Phenotypes		Gene frequencies		Authors	
		Tasters	Non-tasters	T	t		
1-Jews.Baghdad	168	139	29	58.45	41.55	Boyd,W.C.,& Boyd,L.G.	1941
2-Baghdadi Jews	200	159	41	54.72	45.28	Sirsat,Satyava-ti M.	1956
3-Kurds.Baghdad	23	17	6	48.92	51.08	Boyd,W.C.,& Boyd,L.G.	1941
4-Cristians.Baghdad	60	44	16	48.36	51.64	Boyd,W.C.,& Boyd,L.G.	1941
5-Moslems.Baghdad	322	227	95	45.69	54.31	Boyd,W.C.,& Boyd,L.G.	1941

Table 6.4. VIII Phenylthiocarbamide(PTC) taster and non-taster phenotypes and gene frequencies distribution in Pakistan

Population	Number Tested	Phenotypes		Gene frequencies		Authors
		Tasters	Non-tasters	T	t	
1-Chitrali.Chitral	43	33	10	51.77	48.23	Graziosi.1964:edited by Bernhard,W. 1967
2-Calash.Chitral	60	45	15	50.00	50.00	Graziosi.1964:edited by Bernhard,W. 1967
3-Kati.Chitral	33	22	11	42.27	57.73	Graziosi.1964:edited by Bernhard,W. 1967
4-Pathan.Dir,Malakand, Mohmands,Swat	111	67	44	37.04	62.96	Bernhard,W. 1967
5-Pathan.Kohat,Mardan, Peshawar	148	78	70	31.22	68.78	Bernhard,W. 1967
6-East of Indus	49	21	28	24.41	75.59	Bernhard,W. 1967

CHAPTER 7.

Colour-blindness

7.1.Introduction

One of the recessive sex-linked characters known in human beings is the defect in vision called colour blindness. The gene basic to this defect is carried in the x chromosome. (Pickford,1956). Various types of this abnormality which have been studied genetically are red, green, red-green, blue-yellow, and complete colour-blindness. In the latter the afflicted person has difficulty distinguishing any colour. Total colour-blindness is very rare. The four kinds of partial colour blindness are conditioned by recessive genes carried at distinct loci in the sex chromosomes. The genes for red,green, and blue-yellow are located in the non-homologous portionoof the x chromosome. These genes are x-linked and are transmitted from an afflicted man to his daughter, who is generally normal, to a grandson who again shows the defect. Total colour blindness is conditioned by a recessive gene at a locus in the homologous parts of the x and y chromosome. Total colour-blindness may be inherited through both sexes. Women may transmit it in the x chromosome through the eggs. Men may transmit it in either the x or the y chromosome through the two types of sperms. Total colour-blindness is another of the conditions probably associated with inbreeding, as occurs in cousin marriages.

There are commonly assumed to be three sets of colour-sensitive eye cells, or cones, sensitive to red, green, and blue(or violet). Blue sensitivity is very rarely affected for genetic reasons defects of colour-vision which are inherited are almost without exception defects of the red/gre-

en system. At the outset it should be pointed out that colour blindness, in which a person can perceive only shades of grey, is a rare phenomenon (with a frequency of less than one per 10 million). Colour-blindness affecting the perception of blue is also an extremely uncommon deficiency, often brought about by injury or disease rather than genetic factors. Instead, what is commonly referred to as " colour-blindness" is a difficulty in distinguishing certain colours in the range from red to green as the rest of the population does. This is not actually a blindness, despite its name, and might be better thought of as colour-confusion. It is a fairly common defect of vision in man. The character expresses itself in an inability to distinguish accurately between red and green colours. In females, with two X chromosomes, the recessive genes must be in the homozygous state to manifest themselves. Males have only a single X chromosome, and in them a single recessive gene can determine colour-blindness. Because of the relationship of the hereditary factor to the X chromosomes, more men than women show colour-blindness.

Actually, there are several types or degrees of red and green colour-blindness, each presumably determined by its own gene, perhaps at a different locus in the X chromosome. Possibly multiple alleles are involved in the different degrees of defect in each case.

There are cases of red-blindness and others of green-blindness, the sense for both colours, however, being somewhat impaired in the two types. It is clear that both types of colour-blindness are similar to some extent; they can readily be distinguished, however, because persons with the red defect will show a greatly reduced sensitivity to pure red, but

not to pure green, whereas those with the green defect have reversed characteristics.

In about sixty percent of the cases, however, the reduction in pigment is much less extreme so that discrimination between red and green is more easily made, but sometimes with difficulty.A common question is: how do the moderately red/green colour-deficient see red and green? They will, in fact, see red and green at the proper points in the spectrum,but the limited response of one of the two types of cones tends to obscure the sharp differences between the two colours of which normal people are quite aware.

This does not, however, impair their ability to distinquish red from green, as in traffic lights, provided that the lights are bright and not too distant.

As mentioned above colour vision deficiency of congenital origin is the commonest form of colour vision disturbances. Most cases of congenital colour vision deficiency are characterized by a red-green deficiency which may be of two types; first, a protan type which may be absolute (protanopia) or partial(protanomalia), and secondly, a deutan type which may be absolute(deuteranopia) or partial(deuteranomalia).

In protanopia, the visible range of the spectrum is shorter at the red end compared with that of the normal, and that part of the spectrum which appears to the normal as blue-green, appears to those with protanopia as grey.

The whole visible range of the spectrum in protanopia consists of two areas which are separated from each other by this grey part. Each area appears to those with protanopia as one system of colour with different brightness and saturation

within each area, the colour in one area being different from that of the other. The red with a slight tinge of purple which is the complementary colour of blue-green appears also as grey.

In deuteranopia, that part of the spectrum which appears to the normal as green, appears as grey, and the visible range of the spectrum is divided by this zone into two areas,each of which appears to be one system of colour. The visible range of the spectrum is not contracted, in contrast to protanopia. Purple-red which is the complementary colour of green appears also as grey.

In protanomalia and deuteranomalia, there is no part of the spectrum which appears as grey, but the part of spectrum which appears to those with protanopia as grey, appears to those with protanomalia as a greyish indistinct colour,and likewise, the grey part of the spectrum seen by the person with deuteranopia appears to those with deuteranomalia as indistinct colour close to grey. Consequently, one of the peculiarities of red-green deficiencies is that blue and yellow colours appear to be remarkably clear compared with red and green colours.

In the congenital colour vision deficiencies, although very rare, there is total colour-blindness which may be typical or atypical. The subject who suffers from typical total colour-blindness shows a complete failure to discriminate any colour variations, usually with an associated impairment of central vision with photophobia and nystagmus. In the atypical total colour-blindness, the colour sensitivity to red and green, as well as to yellow and blue is so low that only very clear colours can be perceived, but, except for the

clour sensitivity, there is no abnormality in the visual functions.

Furthermore, a failure in the appreciation of blue and yellow may be termed tritanomalia if partial,and tritanopia if absolute, but, even if such cases do exist,they ar extremely rare.

In most populations there are about three times as many deutan males as protan males. There are very significant differences in the total frequency of colour-blindness between populations.In general,the more "Primitive"the population,the lower the frequency.Thus,in hunting and gathering communities the frequency of colour-blindness is about 1-2 percent whereas in an industrial society it is around 7 percent.It has been suggested by Neel and Post(1963)and by Post (1962)that colour-blindness may be disadvantage to primitive people because it impairs their ability to distinguish the flora and fauna that are essential for their livelihood. The negative selection against colour-blindness has most likely been relaxed in more modern societies,which would explain its higher frequency in such societies. Whatever the mechanism, colour-blindness presumably had a selective advantage at one time in man's evolution.

According to Post(1962), it is expected that there will be high prevalence of colour-blindness incidence among the populations belonging to pastoral-agricultural economies or settled habitat and low incidence rate in the hunter and food gatherer groups due to the effect of relaxed selection. Virtually all the tribals practise agriculture and secondarily, hunting. Nevertheless, in order to study the selective mechanisms, populations belonging to "culturally advanced "

societies and " Primitive tribals" can be taken into account (Dutta, 1966).

The " primitive tribal" category people having their primitive cultural background have the lowest mean prevalence rates of defective colour-vision. It may by thought that life would be handicapped with such a condition, and the low prevalence may, for one reason, be due to the result of the high degree of selection pressure, whereas the culturally advanced people, the " advanced non-tribal", have exhibited the higher overall prevalence rates of defective colour-vision, which may, at least in part, be due to the low degree of selection pressure. If these two divisions are compared, it appears that the higher prevalence rates of the advanced non-tribals might have been caused by the effect of relaxed selection against colour-blindness.

If the present polymorphism is transient rather than stable, this evolutionary trend may, perhaps, have been reversed by the relaxation of selection against colour-blindness in modern society. If colour-blindness were a stable polymorphism in primitive societies and also now in modern society, it would seem most likely that it is maintained by female heterozygote advantage, since it is hard to believe that selection with respect to colour-blindness in the two sexes could be in opposite directions. It should be noted, however,that for mutation alone to cause the increase from 1 to 6 percent would require a time that is much greater than the length of time separating primitive and modern societies.

The best known of tests for colour-blindness are those of Professor Shinobu Ishihara, who constructed a series of

plates with numbers made up of coloured dots. The normal person associates all reds together and all greens together, regardless of shade or mixture, and sees one number. The colour-blind, being less sensitive to the distinctions between red and green, will associate the lighter shades with each other, regardless of whether they are yellow, pink, or light green, and so see an entirely different number.Different plates can be designed to determine whether it is the red or green cone that is deficient, and whether the red/green colour-blindness is partial or complete.

Such tests are now routinely included in the series of eye tests given to driver's license applicants and it is not unusual for a male in his late teens or early twenties to discover for the first time that he has colour vision deficiency, but has quite successfully and unconsciously compensated for it in his early years. Generally speaking, such colour-deficient persons will be able to discriminate among red, green, and amber sufficiently to qualify for a driver's license.

7.2. Material and method

Tests for colour-blindness were made on six out of the ten Iranian samples.A total of 1370 individuals including 795 males and 575 females was tested.The tests were performed by means of the Ishihara book of 24 colour plates, edition 1974. The plates were read in well illuminated rooms during daylight, shaded from strong sun.The answers to the plates were recorded and colour defect assessed according to the method prescribed by Ishihara(1974). In accordance with Ishihara's statement on the analysis of the results, if, of the first 17 cards, 13, or more

were read correctly, the vision was regarded as normal. If nine or fewer were read correctly, then the preception of colour was regarded as defective. Individuals reading 10, 11 or 12 cards correctly were not encountered.

However, the use of Ishihara plates is not without strong limitations and pitfalls. Krill et al (1966) suggested that the Ishihara tests strength " lies in its ability to differentiate the normal from the abnormal and no more". Salzano (1972) supports this view, commenting that the Ishihara plates detect only a fraction of the colour-blinds present in a group, and ideally therefore,surveys should be conducted with portable anomaloscopes. The problem is that their use involve complex time consuming procedures and this was the case with the present study. So, in the present study no attempt was made to separate protanopes from deuteranopes or to classify the subject according to the degree of the defect.

7.3. Results

The frequencies of red-green colour-blindness for the six Iranian samples(795 males and 575 females) are shown in Table 7.3. Of all the six male series,the Turks of Shirvan, Khorasan record the maximum frequency of colour-blindness as 7.81 percent and the Tehranis the minimum of 2.53 percent. No colour-blind individual was found in any female series in the present investigation.

7.4. Discussion

The frequencies of red-green colour-blindness in Iranian and neighbouring populations are presented in Tables 7.4. As can be seen from the tables, there is little information about the frequency of colour-blindness in Iran and neigh-

bouring countries and what there is has been obtained with the Ishihara plates which are known to be somewhat unreliable in discriminating anomalous trichromats (Kalmus , 1965).

The frequency of defective red-green vision ranges from 2.53 to 8.1 percent in Iranian males, being lowest in the Tehrani sample (present study) and highest in the Kurds of Sandaj (Lightman et al, 1970). With an overall male frequency of 4.88 percent, the population of Iran appears to exhibit a lower frequency of the trait than that of around 8 percent found in Europeans (Klein and Franceshetti, 1964).

Regarding neighbouring populations, the only reported frequency of 8.19 percent of red-green defect is that of Al-Amood et al (1981) for the Arabs of Iraq which is higher than the average for Iran and very similar to the European frequency.

There is no information about the frequency of colour vision defects in other neighbouring populations.

Table 7.3. Frequency of colour-blindness in Iran

Population	Number Tested		Sex	Normal	Colour-blind	
					No.	%
1-Tehran	646	316	M	308	8	2.53
		330	F	330	-	0.00
2-Kerman	140	97	M	94	3	3.09
		43	F	43	-	0.00
3-Kurds.Rezaieh	154	61	M	59	2	3.28
		93	F	93	-	0.00
4-Kurds.Shirvan,Khorasan	141	108	M	103	5	4.63
		33	F	33	-	0.00
5-Zoroastrians	133	85	M	81	4	4.70
		48	F	48	-	0.00
6-Turks.Shirvan,Khorasan	156	128	M	118	10	7.81
		28	F	28	-	0.00

Tale 7.4. I Frequency of colour-blindness in Iranian males

Population	Number Tested	Normal	Colour-blind		Authors
			No.	%	
1-Tehran	316	308	8	2.53	Present study.
2-Kerman	97	94	3	3.09	Present study.
3-Kurds.Rezaieh	61	59	2	3.28	Present study.
4-Kurds.Shirvan,Khorasan	108	103	5	4.63	Present study.
5-Zoroastrians	85	81	4	4.70	Present study.
6-Turks.Shirvan,Khorasan	128	118	10	7.81	Present study.
7-Kurds.Sanandaj	504	463	41	8.1	Lightman,S.L.,et al. 1970

Table 7.4. IV Frequency of colour-blindness in Iraq

Population	Number Tested	Sex	Normal	Colour - blind		Authors
				No.	%	
1-Arabs.Basrah	2066 1221	M	1121	100	8.19	Al-Amood,Wathib.S.,
	845	F	818	27	3.02	et al. 1981

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