Genetical variation in selected populations in the isle of man and neighbouring areas

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ABSTRACT

This study reports upon the investigation of a number of genetic polymorphisms in indigenous population samples of three regions of the British Isles: the Isle of Man, Cumbria and South West Scotland. Sample selection proved to be important because differences were found in the similarly selected indigenous Manx population - between donors and non-donors.

In addition to a study of phenotype distributions and gene frequencies in the three selected populations, a regional analysis of the Manx data, though on a limited level, was effected. Though great difficulties were encountered obtaining indigenous samples, comparisons were performed between the Manx and population samples from selected regions of the mainland of the British Isles as well as certain north-west European populations. Possible explanations of the differences observed between the Manx and surrounding populations were proposed, but it was also suggested that an analysis of the demographic data would be most informative.

GENETICAL VARIATION IN SELECTED POPULATIONS IN THE ISLE OF MAN AND NEIGHBOURING AREAS.

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INTRODUCTION

Anthropology is essentially the study of the human species, the comparative study of man as a physical and cultural being. The physical anthropologist studies man's physical characters, their origin, evolution and present state of development. (Montagu 1960) Striking differences may be present in populations with respect to physical characters such as skin and hair colour, stature and hair form. Accordingly, physical anthropologists have long been interested in the description, comparison and classification of the groups of mankind. For these purposes they once used the measurement and description of various external physical characteristics of the body, such as height, weight, cephalic index and hair form and colour. Though populations could be described on the basis of such traits, assessing a group's exact genetic relationship was exceedingly difficult. First, methodological problems, such as the errors involved in taking body measurements, or the degree of subjectivity involved in classifying hair form and eye colour for example, introduce a certain amount of bias into the data on the populations to be compared. Second, because the characters in question are under the control of many genes, (polygenic traits) and for the most part are extremely sensitive to environmental influences, and because many genotypes cannot be distinguished from each other phenotypically, the statistical methods of studying these characters are very complex.

Today, however, it is realised that differences between populations transcend differences in size and appearance, and extend to biochemical factors and other immunochemical properties. Since the 1900's a new class of physical characters has entered

the field of anthropology; the blood groups. These characters can be more accurately established, are under precise genetic control and are susceptible to statistical analysis and gene counting. Their mode of inheritance is simple, straightforward and usually follows Mendelian laws. The gene frequencies in the population tested can be easily computed from the observed phenotypic frequencies. From the analysis one can hypothesise about the relative influences of natural selection, migration, mutation, population admixture, disease resistance and environmental forces like climate. These biochemical traits are all the more reliable as taxonomic tools as they are genetically determined at conception and remain fixed for life. (Mourant 1954)

Together with the blood group systems, and rapidly increasing in importance, is another set of biochemical markers found also in human blood. These are the blood proteins and cellular enzymes. These components of human blood have been found to exhibit hereditary variation that differs among populations. The variant forms occur too frequently to be due to recurrent mutation and can therefore be considered to be polymorphic in man according to Ford's (1940) definition. Blood proteins, like the blood groups, are a much more immediate consequence of the genetic constitution than are the directly observable morphological characters of the body. (Scozzari et al. 1970) The discovery of these quantitatively and qualitatively different proteins would never have been possible had it not been for the introduction of the technique of starch-gel electrophoresis (Smithies 1955). This sensitive method permits the separation of molecules on the basis of their size as well as electrical charge. Initially it was used for differentiating

serum protein variants, such as haptoglobin and transferrin. However, largely due to the work of Harris and his co - workers, the technique has been extended to the study of the cellular enzymes.

Again, the methods of recording the more traditional anthropometric characters have also been refined. Today, reflectance spectrophotometers are used for objectively measuring skin and hair colour.

The present study reports upon the survey of selected indigenous inhabitants of the Isle of Man in particular, but also of smaller numbers in Cumbria and South West Scotland, to determine whether these groups exhibit variability in many of the above mentioned physical characters.

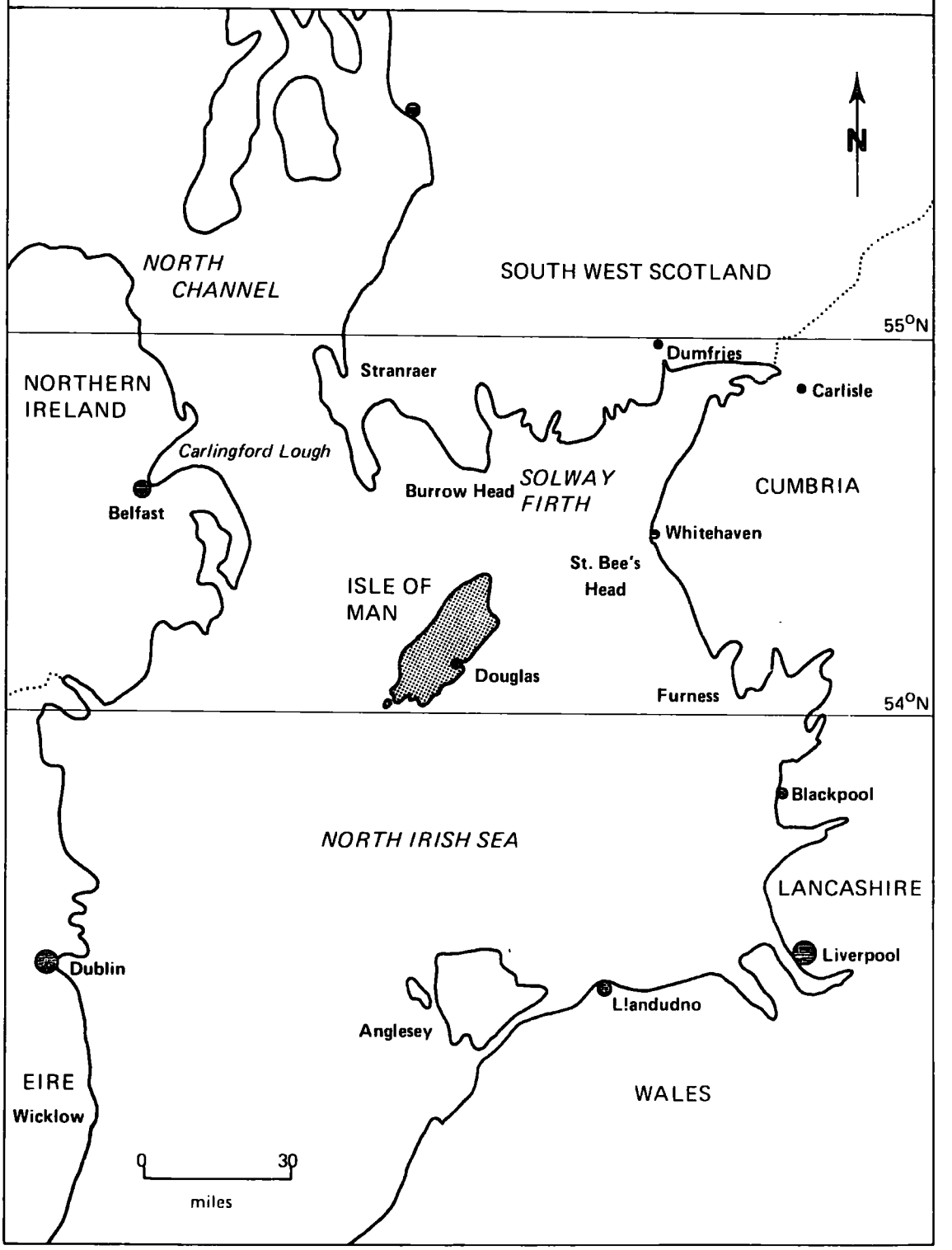
Chapter one is an account of the physical and human background of the Isle of Man, the centre of study. These two aspects are very important to a study of the population of the Isle of Man because the Island's position in the middle of the North Irish Sea basin has produced varying degrees of social isolation, with consequent marked effects on population growth and movements. Chapter two summarizes the selection of the population samples incorporated in the study, as well as the field and laboratory methods employed. It will be shown that the selection of samples has a significant bearing on the nature of the survey of the three population groups. The analysis of the data collected in the Isle of Man, Cumbria and South West Scotland is given in Chapter three which also reports upon inter - population comparisons. An attempt at a regional analysis of the data on the Manx population is included in chapter four. Finally, in chapter five, the indigenous Manx population is compared with other populations, indigenous and resident, of nearby regions such as Ulster and Lancashire, but also as far distant as Scandinavia.

CHAPTER ONE

THE ISLE OF MAN - PHYSICAL AND HUMAN BACKGROUND

Figure 1

THE ISLE OF MAN – POSITION IN THE IRISH SEA



THE ISLE OF MAN - PHYSICAL AND HUMAN BACKGROUND

(a) PHYSICAL BACKGROUND

1. Position and Size. (Fig. 1)

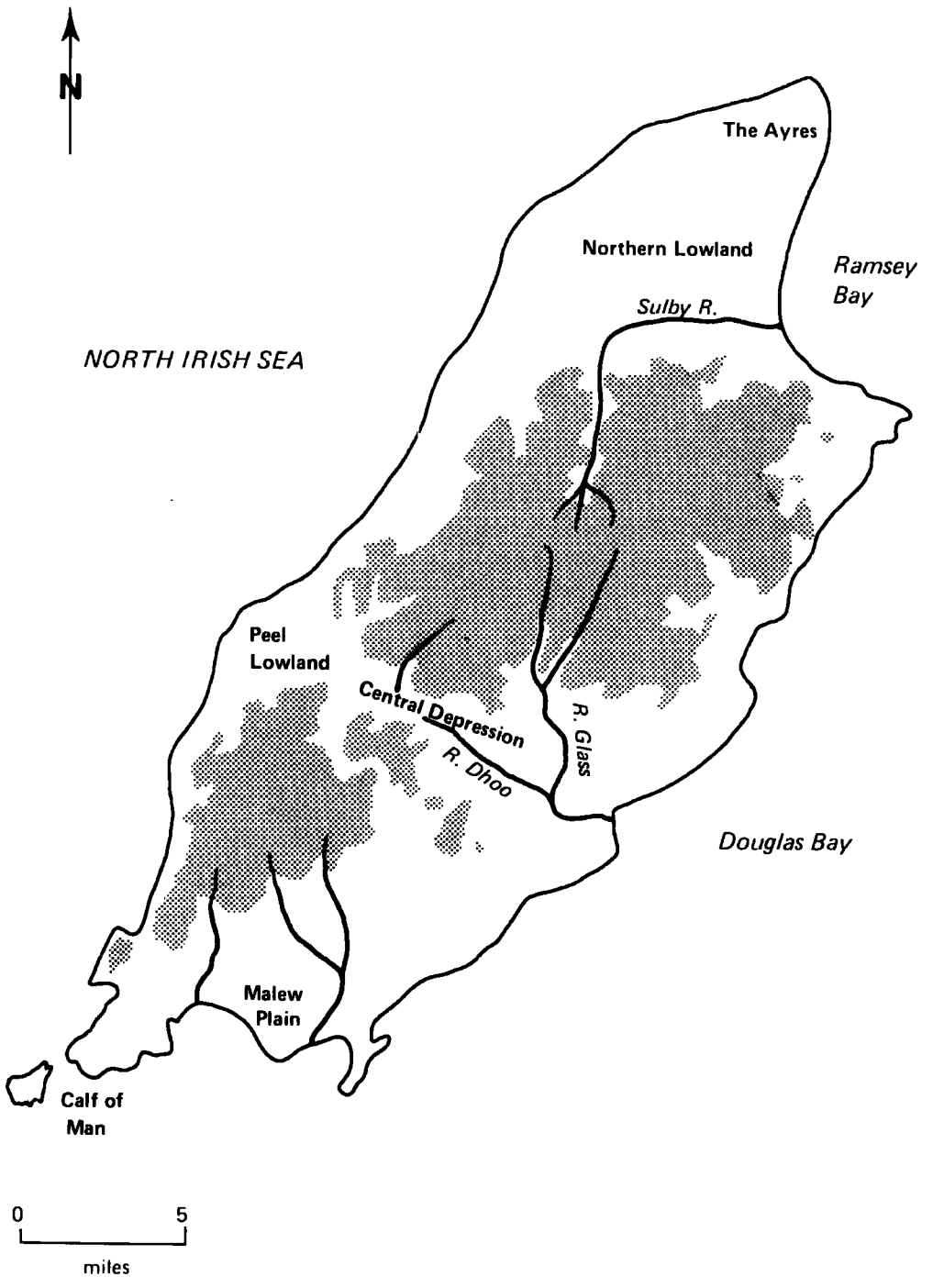
The Isle of Man, with a total area of 220 square miles, lies in the middle of the North Irish Sea Basin, almost equally distant from the coasts of England, Wales, Scotland and Ireland. The Island is 31 . 5 miles long from the Point of Ayre to the Sound of the Calf, with a breadth ranging from 8 miles to 13 miles in its central portion. The nearest point in England, St. Bee's Head, Cumberland, is 28 miles distant, and the entrance of Strangford Lough, Ireland, is over 26 miles away. The nearest coastline is that of Scotland, Burrow Head, Wigtownshire being 16 miles from the Point of Ayre, while the most distant is that of Wales, Anglesey being 45 miles to the South.

The position of the Isle of Man dominates the study of any phenomena to be found on it. (Fig 1). For example, Geikie (1897) discussing Manx geology states that "rising from the middle of the Irish Sea within sight of each of the three kingdoms with a history and associations so distinct, yet so intimately linked with those of the rest of Britain, this Island presents in its geological structures features which connect it alike with England Scotland and Ireland, while at the same time it retains a marked individuality." In similar vein Clark (1935), comments that the prehistory and history of the Isle of Man is of absorbing interest from its geographical position in relation to the larger units of the British Isles. Since the first settlement of the Island

cultural and ethnic influences have approached from all directions, but its size and insular position have been sufficient to ensure vigorous local developments. Bowen (1969) also stressed that the Island's position in the midst of the Irish Sea is basic to an understanding of its cultural life in antiquity, and to the part it played as a focus of ancient seaways in the North Irish Sea Basin.

Figure 2

ISLE OF MAN · RELIEF



Land over 500ft.

2. Relief and Structure. (Fig. 2)

The central mass of the Island consists of a high plateau or moor some 750 feet and more above sea - level, from which a number of peaks of over 1500 feet rise. The dominant N.E. - S.W. trend of the Island is seen in a series of mountain peaks running from North Barrule, 1860 feet, through Snaefell, 2034 feet, to Greeba, 1383 feet. From Greeba the line continues on the other side of the central depression in South Barrule, 1585 feet, to Cronk ny Irree Haa, 1449 feet. In addition to the N.E. - S.W. axis, spurs radiate to the coast across the adjacent coastal plateau and northern plain.

It is this highland belt, along which the line of water parting runs, that subdivides the Isle of Man into two traditionally distinct units, the Northside and Southside (Kinvig 1950). It will be shown that because of the different historical influences impinging upon these two areas they are of prime consideration in this anthropological survey of the Manx population.

On the east and west sides of the central highland mass the structure is one of lower and generally narrow coastal plateaux, with an average height of about 400 feet, but the surface is irregular because rivers break through to the coast cutting deep glens. The plateau along the west coast is generally narrower and also higher than that on the east coast.

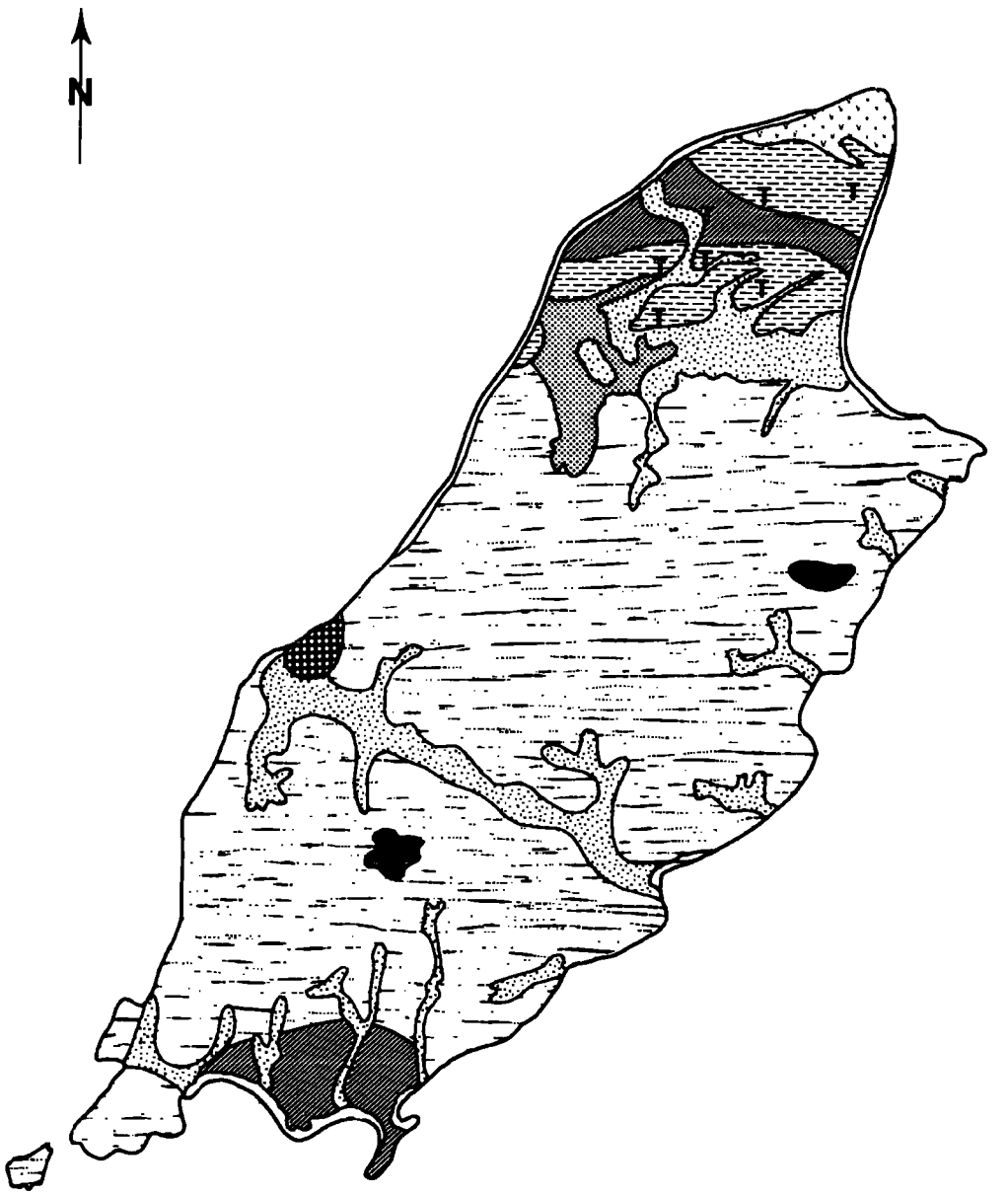
Cutting across the highlands and surrounding plateaux in the middle of the Island is the central depression between Douglas and Peel. This valley is often regarded as the line separating the 'Northside' of the Isle of Man from the 'Southside', but in earlier times the depression was not so important, partly because

of its narrow character and also because its floor was, and to some extent still is, swampy. Communications in a west - east direction formerly tended to keep to the higher ground outside the depression.

There are two relatively low lying areas which are quite distinct from the Manx massif and its plateaux fringes. One is the area extending north of a line between Michael and Ramsey, which is called the Northern Lowland. It is by no means of a uniform level, as it contains a series of morainic hills over 300 feet high in the parish of Bride, and also the Curraghs, formerly a number of small lakes now largely drained. The second area of lowland, the Malew Plain in the south - east, is normally below 100 feet, but also reaches 250 feet in certain points. A third and much smaller lowland is found around Peel.

Figure 3

ISLE OF MAN -- GENERALISED GEOLOGY



Lamplugh G.W. 'The geology of the Isle of Man'

T Overlain till
Granite
Manx slates
Carboniferous limestone
Carboniferous sandstone
Old red sandstone
Post-glacial gravels
Aeolian sands
Alluvium
Beaches

3. Geology. (Fig. 3)

The upland mass and coastal plateaux, which cover more than three-quarters of the Island, constitute a much crumpled boss of very old slaty rock rising above the greatly denuded surrounding belts of newer strata, now mainly submerged by the Irish Sea. The earlier rocks, known as the Manx Slate series, consist of clay slates, grits and flaggy greywackes of possibly Ordovician Age like the Skiddaw Slates of the Lake District. In a few places, large intrusive bodies solidified into massive crystalline rock which are now exposed as granitic bosses, such as the Dhoon granite near Maughold Head (Fig. 3).

Lower Carboniferous Limestone underlies the Malew Plain, and on the west adjoining Peel a narrow strip of Carboniferous Red Sandstone occupies a small triangle of about four square miles. These are the only areas where solid rocks are visible, but Carboniferous, Permian and Triassic deposits lie beneath the Northern Lowland covered by 155 feet plus of glacial drift, with raised beach material and blown sand in the Ayres of the North.

As a result of possibly three successive glaciations, boulder clays together with sands and gravels of varying thicknesses, are now distributed over most of the Island except for the high ground.

There is now general agreement that land bridges between the Isle of Man and the mainland persisted for some time after the Pleistocene period. The Irish Sea is particularly shallow, especially between Cumberland and the Isle of Man which are connected by a submarine ridge only some 15 fathoms deep. The date of separation is now placed about the end of the Boreal period, 6,000 BC, the same time as the Mesolithic hunters settled in Man.

4. Soils.

The glacial period and its aftermath provided many of the soils on which the present day agriculture depends, both on the uplands and lowlands. These soils, especially above 600 feet, are usually thin and relatively infertile, but there are notable exceptions. The soils of the Northern Lowland developed on extra-glacial drift are deep and variable in quality, ranging from heavy clay to light sand and gravel with tracts of alluvium. The boulder clay has usually a sufficient sand content to form a fertile loamy top soil, and the most extensive belt is that lying north of Ballaugh and west of the Curraghs. The Malew Plain is covered with gravel and sand which occur as platforms, which to the north - west are replaced by boulder clay. The soils in this southern lowland are similar to those of the Northern Lowland but more loamy. Finally, the red sandstone of the Peel area is overlain with glacial sands and gravels, producing a light and loamy soil.

5. Climate

The climate is greatly influenced by the Island's small size. Marine influences are everywhere dominant and such climatic variation that does exist is determined principally by the influence of local topography. The Isle of Man experiences characteristically equable, windy, cloudy and humid conditions. The summers are relatively cool but the winters are mild, and the rainfall is generally heavy for most of the year.

(a) Temperature

The equable nature of the Manx climate is best illustrated by the small range of only 17.5° F between the mean sea - level temperatures for the warmest and coldest months at Douglas for the period 1926 - 50 when compared with Blackpool, with a range of 21.1°F.

Douglas		Blackpool	
August	59.0°F	July	60.9°F
February	41.5°F	January	39.8°F
Mean Ann. Temperature	48.7°F	Mean Ann. Temperature	48.0°F

The very slight variation in the mean annual temperature throughout the Island is due primarily to changes in local topography. The mean diurnal range of temperature is very slight throughout the year, at its maximum in July (11.7°F) and at its minimum in December (7.3°F). The prevailing westerly and south-westerly winds exercise a moderating influence on temperature at all seasons of the year.

(b) Precipitation

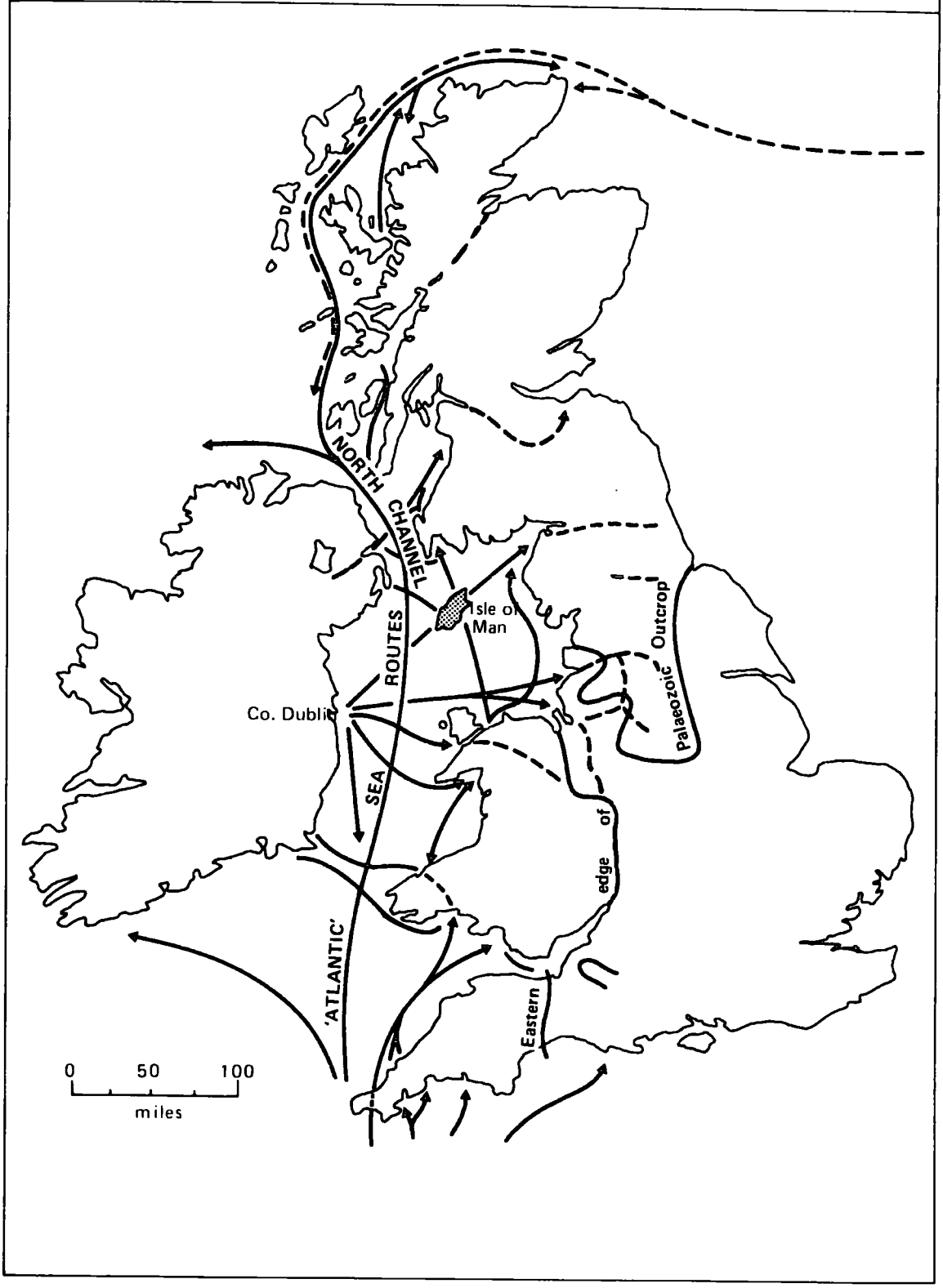
All parts of the Isle of Man are subject to the same rainfall regime modified only by orographic and purely topographic effects.

As the distribution of average annual rainfall is determined by the position of the upland axis in relation to the prevailing moist westerly winds, striking variations do occur in different localities. The Northern Lowland, Malew Plain, the south - west coast and the western coastal fringe north of Dalby are the areas of least precipitation, with a mean of 30" - 40". Around Douglas and most of the central depression the annual average rainfall is between 40" and 45". The remaining area of the plateaux and the Northern and Southern Uplands receive over 45", and in the more elevated parts orographic influences are dominant and the mean figure varies from 50" - 60", with a recorded maximum of 60 . 2" at West Baldwin in Marown.

The seasonal distribution is similar throughout the Island. Precipitation is fairly well distributed throughout the year with an autumn maximum and a spring minimum. Heavy or prolonged snowfalls are not common and snow does not persist over the lowlands and lower coastal plateaux.

Figure 4

ISLE OF MAN – POSITION IN RELATION TO THE WESTERN SEAWAYS



b) Human Background

1. Prehistory and History.

Introduction

By virtue of its insular character and location in the middle of the North Irish Sea, the Isle of Man occupies a unique position, not only in relation to the major sea-routes of prehistoric and early historic times, but also in relation to the larger units of the British Isles which surround it (Fig.4). Two facts result from this, firstly, cultural influences were borne by sea-routes and therefore came from all directions, and secondly, the Island was, and is, sufficiently large and isolated to allow many local cultural developments to occur. (Clark 1935)

Fluctuations in trading conditions along the Western Seaways of Britain have had a great influence on the historical development of the Isle of Man. When trading conditions flourished cultural developments were vigorous, as during the Neolithic Age (before 2000B.C.) and the Early Christian Period (4th - 8th centurys A.D.). However, periods of decline along the western sea - routes resulted in phases of relative backwardness, as during much of the Stanley period (1405 - 1765) when official policy isolated the Island, or during the latter phases of the Bronze Age (650B.C.) when a deterioration in climatic conditions occurred (Kinvig 1966).

Owing to its position, the Isle of Man has frequently had a significance disproportionate to its size. It has often played the role of a pivotal area in the Irish Sea, so that this basin has frequently formed a 'Culture Pool', while it

has certainly helped in the transmission of ideas along the Western Seaway (Kinvig 1958). The map in 'Personality of Britain' (Fox 1943), shows that the Island was in contact with Carlingford Lough, Ulster, the Mull of Galloway, the Solway Firth, Dublin Bay and the Menai Straits by way of the sea routes in prehistoric and later times. (Bowen 1969)

The prehistory and history of the Isle of Man can be subdivided into six periods, namely the:-

Prehistoric Period	before c. 450AD
Early Christian Period or Age of the Celtic Saints	450 - 800 AD.
Scandinavian Period	800 - 1266 AD.
'Age of Strife'	1266- 1405 AD.
The Stanley Period	1405 - 1765
From the Revestment Act to the Present Day	1765 - to date

1. Prehistoric Period

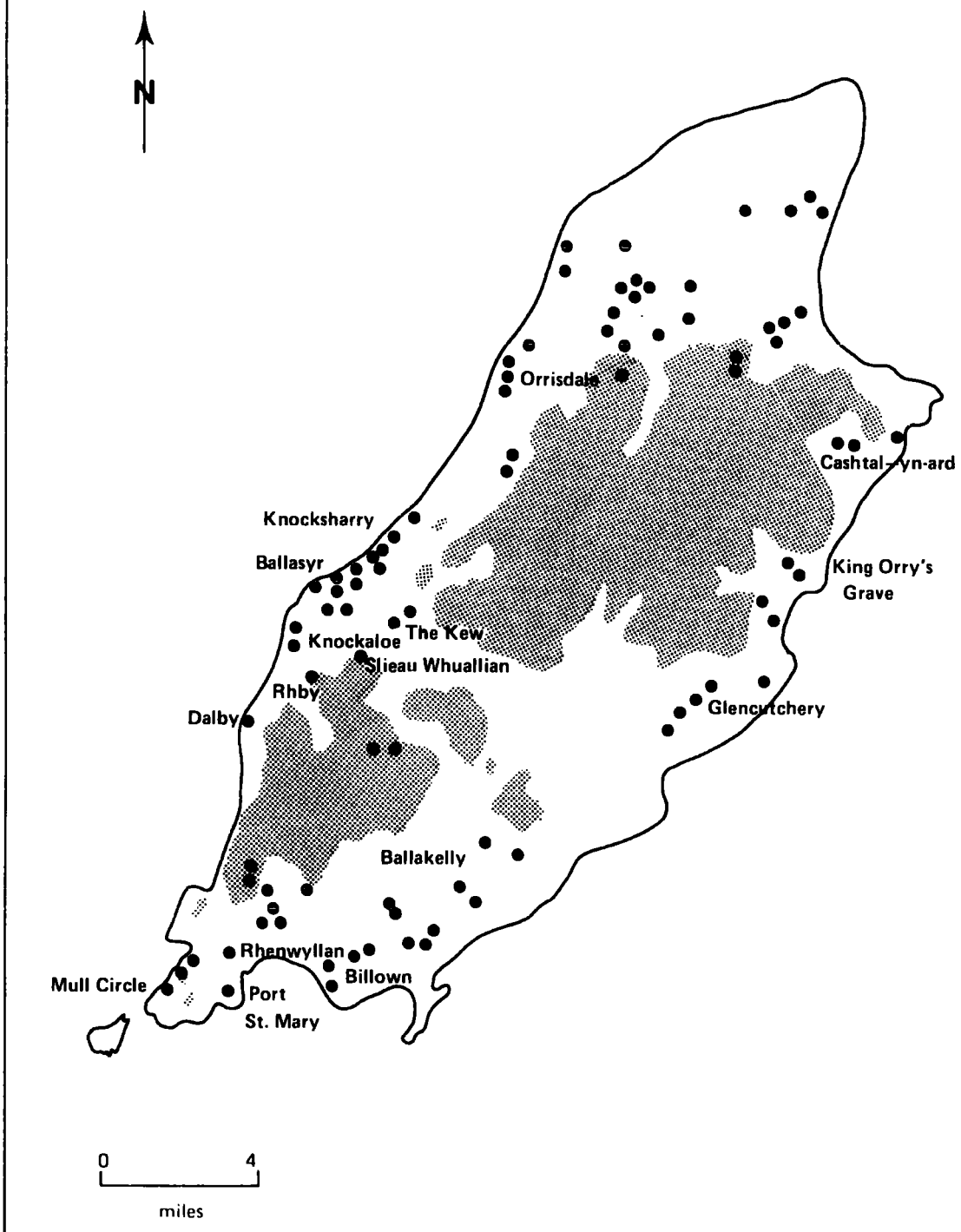
In Bowen's (1969) view the interaction of British and Irish influences impinging on the 'Southside' and 'Northside' of the Island respectively, together with occassional insular developments are the chief features of the pre - and proto - history of the Isle of Man.

(a) Mesolithic c. 3000 B.C.

The earliest trace of human settlement in the Isle of Man dates from the Mesolithic period, with the discovery of microliths of the Sauvet erian culture. The culture is similar to that found in several places in lowland Britain as well as the coasts of Wales and South West England. According to Clark (1935) these people probably reached the Isle of Man from North West England

Figure 5

THE ISLE OF MAN – DISTRIBUTION OF NEOLITHIC FINDS



● Represents one find

Land above 500ft

by means of a land bridge 6000 years ago.

At a slightly later date there is evidence of the tanged flakes of the River Bann culture of Northern Ireland. These finds occur in regions from which the Sauvetterian people were absent as well as in areas common to the two cultures. So in the earliest phase of human settlement one can distinguish influences from the east and west.

b) Neolithic c.2500 - c.2000B.C. (Fig. 5)

By Neolithic times, c2500 - 2000B.C., there is no doubt that Man had really become an Island, so that the new economy, agriculture and stock raising, must have come by sea. In the Isle of Man the megaliths are generally limited to areas between 300 feet and 700 feet, such as Cashtal yn Ard and Gretch Veg, and are very similar to those found in the adjoining countries, especially Cumberland, North East Ireland, the lower Clyde Valley and South West Scotland (Kinvig 1950) (Fig. 5).

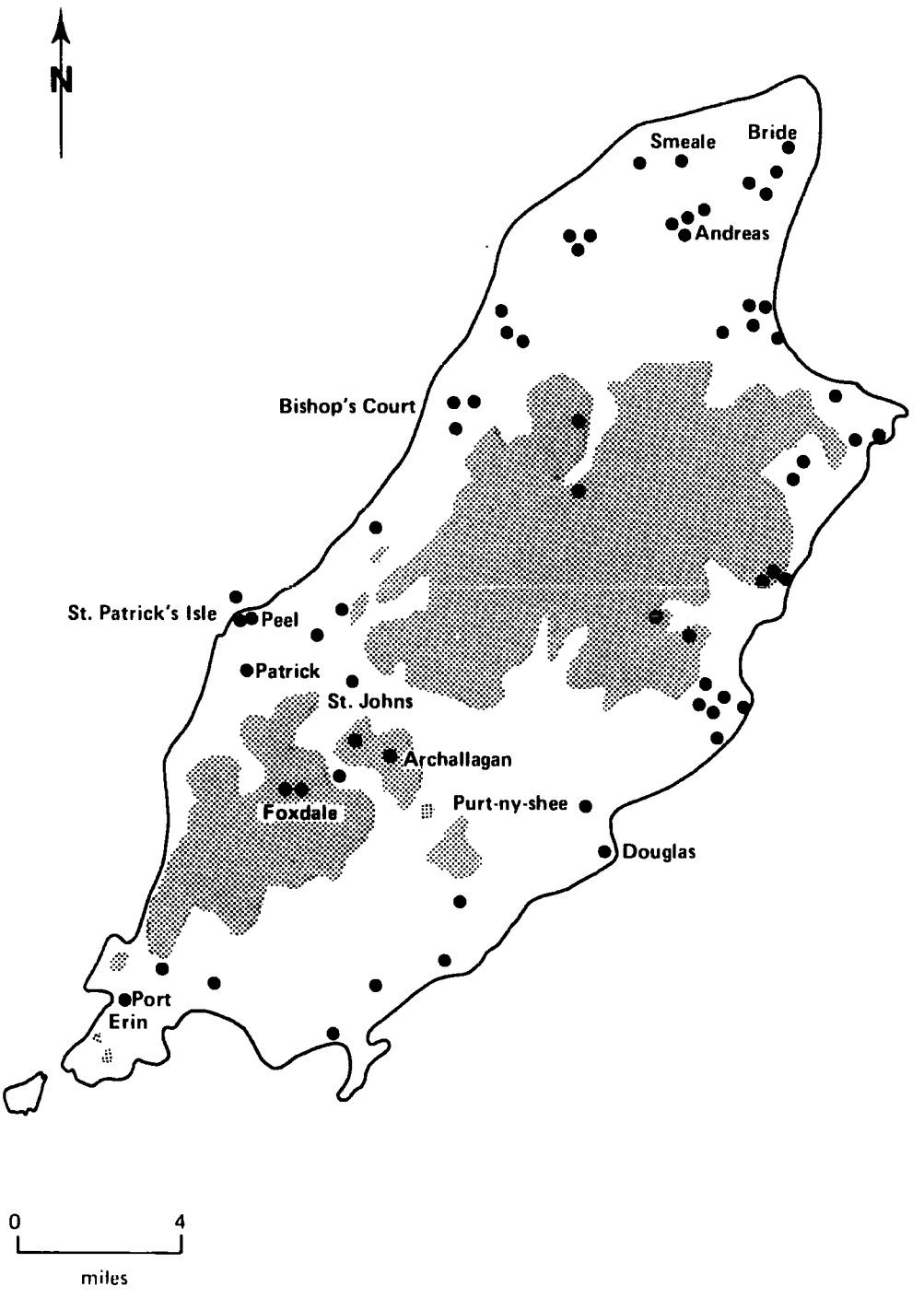
The Island possesses evidence of a distinctive secondary Neolithic culture named after the Ronaldsway type, which represents the assimilation of Neolithic elements by the indigenous hunter - fisher population (Kinvig 1958).

c) Bronze Age. c2000 - 500 B.C. (Fig. 6)

It has been estimated that the Bronze Age in the Isle of Man began about 2000 - 1800 B.C. and lasted until a few centuries before Christ. During this period, Ireland, with its active use of bronze, attracted merchants from many countries, and all this activity must have affected the Isle of Man, it being a convenient stepping stone in the Irish Sea. According to Clark (1935)

Figure 6

THE ISLE OF MAN -- DISTRIBUTION OF BRONZE AGE FINDS



● Represents one find

Land above 500ft.

the Island was one of the most bronze using areas in Britain, as illustrated by the distribution of finds shown in Fig. 6. With one exception the Manx Bronze Age distribution resembled that of Neolithic times, the exception being the Northern Lowland which had become well settled. The entire absence of sites in the mountainous interior is particularly prominent in view of the density of settlement elsewhere on the Island, but this has been a constant factor throughout the history of human settlement in Man. There have been no finds in the central depression, which is understandable as physical conditions would be against settlement. So even at an early stage in Manx history, the Island was separated by the mountain belt into two main divisions, 'Northside' and 'Southside'. From the evidence of three food vessels Clark (1935) concluded that western i.e. Irish influences operated most strongly on the Island during the Bronze Age.

d) Iron Age c500 B.C. - 450 AD

During much of the Iron Age cultural conditions in Lowland Britain and Highland Britain presented very marked contrasts. Whereas in Lowland Britain there is definite evidence of a succession of fresh invasions, in the West, from what little evidence that exists, it gives a picture of stagnation or even of deterioration, especially a diminution of trading movements along the Western Seaway. (Kinvig 1958) In this period the Isle of Man witnessed a period of insular development as was also the case in Ireland.

By Roman Times however, there is evidence of more activity and a period of cultural development. The most distinctive feature of the Manx Iron Age Culture is the large round Celtic

homestead, similar to the 'raths' of Ireland and 'duns' of Scotland. As would be expected therefore the Island showed the major cultural elements characteristic of the surrounding territories at this time.

2. Historic Times

The Early Christian Period or the Age of the Celtic Saints

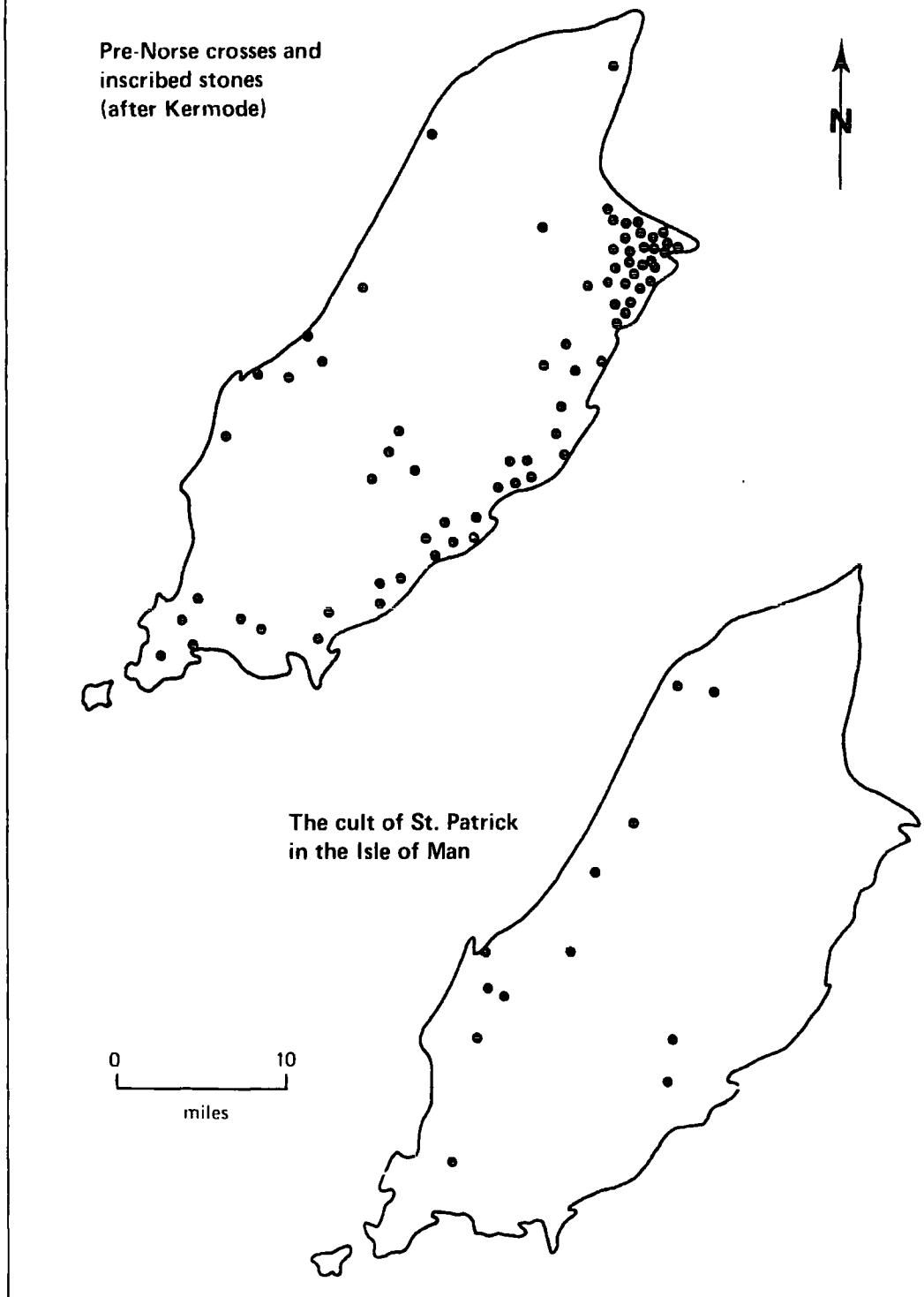
c450 - 800 A.D.

Between the fourth and ninth centuries Christianity first took root and flourished in Western Britain, penetrating along the western sea - routes into the Irish Sea basin. According to Chadwick (1961), the Isle of Man shows a greater concentration of Early Christian remains than can be found in any other area of comparable size in the British Isles. Problems arise from the fact that references to the Celtic Saints in the Isle of Man are of a late date and doubt exists concerning the date of the keeills (early Christian oratory). Megaw's (1937) study indicates that there is little archaeological evidence that they were oratories of the Celtic period in the fifth and sixth centuries. There is nothing to show that these ruins are earlier than the Viking settlement of the ninth century.

However Pre - Norse crosses and inscribed stones have been found in the Isle of Man, including twenty - five cross - slabs from the parish of Maughold. The overall distribution pattern of monuments classified as 'Pre - Norse' shows an unmistakable 'Southside' character, thus reinforcing the cultural dualism between 'Northside' and 'Southside' in the Island (Bowen 1969). (Fig. 7)

Figure 7

ISLE OF MAN – DISTRIBUTION OF EARLY CHRISTIAN PERIOD REMAINS



Jackson (1953) has shown that on linguistic grounds there is evidence of a Brittonic population in the Isle of Man in the Dark Ages, as well as the immigrant Gaelic people from Ireland. Bede even considered the Isle of Man to be British and not Irish territory (Bowen 1969). The only difference in the history of Man being that the Gaelic invaders and their culture were destined to overcome the Brittonic, while in some other areas of Britain, e.g. West Wales, the Gaelic invaders were ultimately absorbed into the local population (Jackson 1953).

The Northside of the Isle of Man is the area where Irish influences are to be expected, yet the direct evidence of such settlement is slight. The inscriptions on six Ogham stones found in the Island show definite evidence of Irish influence but their distribution is not limited to the Northside. In fact three types of script, Gaelic, Latin and Pictish have been found on Manx Oghams. (Kinvig 1950).

According to Bowen (1969) "it might well be that the decisive factor in the Gaelicization of Man was associated with the spread of the cult of St. Patrick." When the Patrician cult appeared in the Island it seemed to have emanated from north - east Ireland. The distribution of keeills bearing St. Patrick's name reveals unmistakably a Northside distribution (Fig. 7) in sharp contrast to the Southside distribution previously mentioned for Pre -Norse monuments. This evidence was sufficient for Bowen to state that he had established the survival of two physical and cultural provinces in the Isle of Man.

Having established the existence of two cultural provinces, Northside and Southside, due to influences from the west and south

respectively between the fifth and eighth centuries, Bowen discussed other ethnic and cultural influences which in consequence of its central position in the North Irish Sea have approached the Island from all directions. Even if the possible influences of the Ninianic Church of Whithorn are excluded, keeills are found dedicated to St. Columba and St. Adamnan in the northern parishes of Andreas and Lezayre. From the east there is both archaeological as well as dedication evidence; in Maughold there isan eighth century cross slab inscribed in runic character with the Saxon name Blackman. At this time the Celtic Church of Galloway had passed under Saxon control and there might well have been overspills into the Isle of Man.

All this evidence adds up to a great deal of movement of people and ideas across the Irish Sea during the fifth and succeeding centuries. It also seems evident that the Isle of Man formed a natural cultural focus in the Irish Sea basin, receiving from north, south, east and west various influences when the sea - routes were dominant.

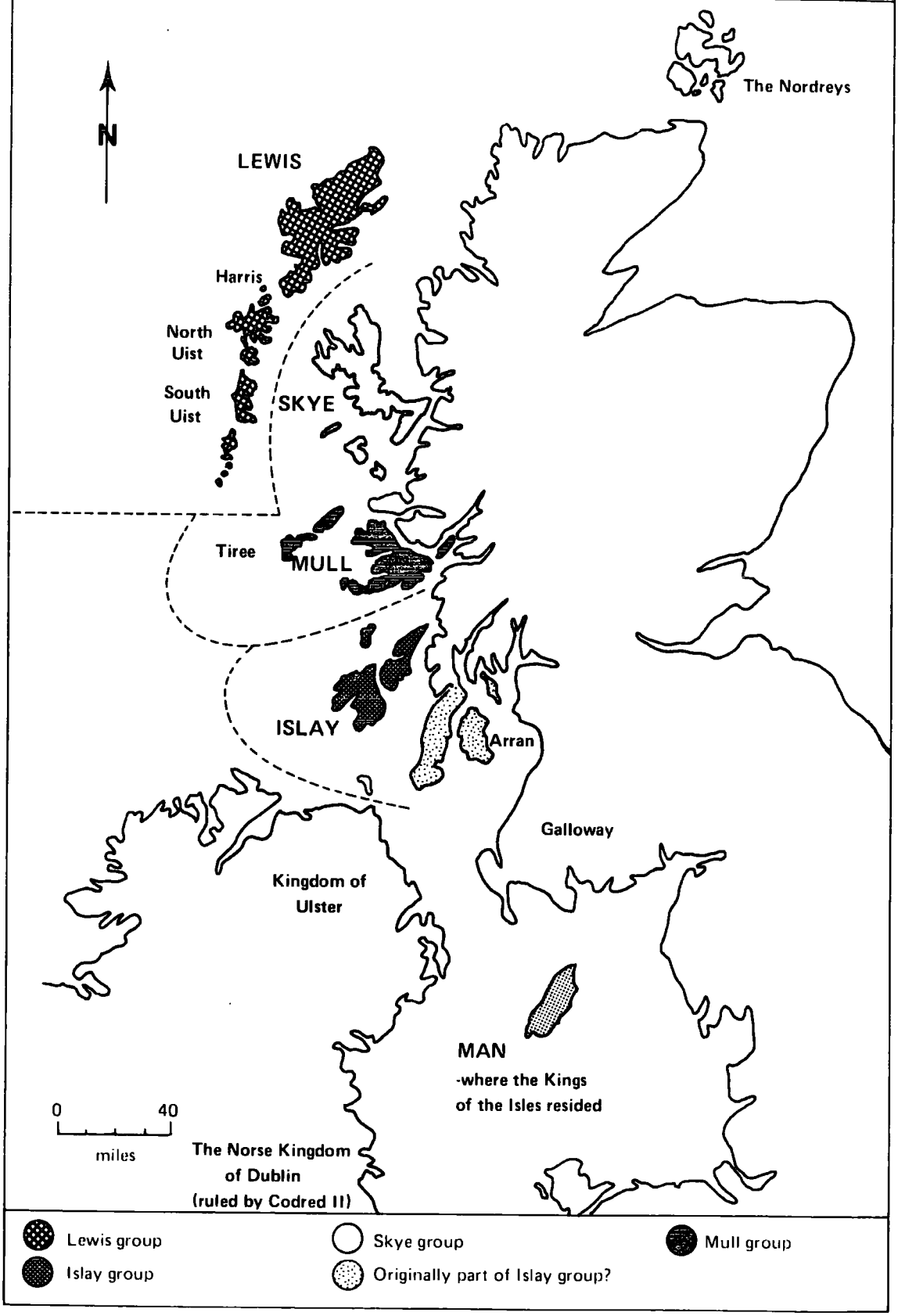
Scandinavian Period. c800 - 1266 A.D.

The Norse Vikings were first recorded in the Irish Sea in the latter part of the eighth century, when they began plundering the coasts of Ireland, Scotland and the Isle of Man. From 835A.D. there was a methodical conquest of the lands surrounding the Irish Sea and in this scheme the strategic position of the Isle of Man was of great importance. Man was deliberately colonized and the effective Scandinavian population dates from the second half of the ninth century, when the newcomers seized the most fertile areas. (Kinvig 1958) In the view of Shetelig (1954) the Island "is the best example of Viking colonization and of the cultural life of the Vikings outside their own homeland."

Though Moore (1900) states that the firstcomers were Norwegians called Fair Strangers who were followed by Danes called Black Strangers by the Celts, the evidence from place - names strongly supports the view that, as in Cumbria, the Norwegians had a decided preponderance in the Isle of Man. As it was young men who came from Norway, from the beginning there must have been consistent intermixture between the Norse and the native Celtic women, so that a new Manx type would be produced by the fusion of these two elements. The mixed population of Man were termed Gael - Galls by the Gaelic population in districts at first unaffected by Scandinavian immigration. (Kinvig 1950). As the Scandinavians were the dominant group in the Manx population they settled chiefly in the fertile Southside with the result that the Celts became predominant in the Northside (Airne 1949). This redistribution of the population naturally would intensify the distinction between the 'Northside' and the 'Southside'.

Figure 8

ISLE OF MAN – THE KINGDOM OF MAN AND THE ISLES



However, as the Scandinavian settlement lasted until the middle of the thirteenth century it is likely that there was a very full settlement of the complete Island.

During much of the Scandinavian period the Isle of Man and the Western Hebrides of Scotland were united constitutionally into one unit, called the Kingdom of Man and the Isles (The Kingdom of the Sudreyjar), with Man as the capital. (Fig. 8)

For administrative purposes all the islands except Man, which was important enough by itself, were divided into four groups based on the main islands of Lewis, Skye, Mull and Islay. Later the two Southern groups, Mull and Islay, were lost to the coastal kingdom of Argyll, and only the northern groups of Lewis and Skye, called the Out Isles, maintained their connection with Man until 1266.

The thirteenth century saw the growth of the power of Scotland under Alexander III who was anxious to possess the Western Isles, and also there was increasing competition between Norway and England for control of the Irish Sea basin. In 1266, following their defeat in the battle of Largs in 1263, for a payment of 4,000 marks the Norse ceded Man and the Western Isles to Scotland, and thus ended their long suzerainty.

Age of Strife. 1266 - 1405

After 1266 there followed more than a century of strife for the Manx population, with the allegiance of the Island often in dispute between England and Scotland. However, since 1344 English suzerainty has been maintained over the Isle of Man, even though Scottish raids continued until as late as 1456.

According to Kneen (1937), it is probable that there were many immigrants from Galloway and Ireland to the Isle of Man after 1266, and as a direct result of this immigration the Norse language and Gael - Gall dialect were eventually superseded by a purer Gaelic idiom. The Manx language had been so strongly influenced by the Scottish form of Gaelic that from this time Manx had a greater resemblance to Scottish Gaelic than Irish.

The Stanley Period. 1405 - 1765

In 1405 Sovereignty was granted to Sir John Stanley, whose descendants as the Earls of Derby, or later as the Dukes of Atholl, ruled the Isle of Man under the title initially of King, but later as Lord, for over 300 years. It was a period of consolidation rather than one of new developments, during which the Island, through official policy, was relatively isolated, so that it could develop and maintain distinctive features as for example its form of government, land system and personal names. Trade was discouraged and strictly regulated.

Conditions began to change rather more radically in the later 17th century with the increasing significance of smuggling or the "running trade" which was carried out along several stretches of the English and Scottish coasts, especially the Solway Firth. The strategic location of the Isle of Man, its political position and its low customs duties introduced in 1577, made it peculiarly well suited for engaging in this traffic. Smuggling was first noticed on any scale in 1697, but then increased rapidly to reach its height early in the 18th century. Glasgow and Liverpool merchants profiting from the tariff differences, made the Island a vast warehouse crammed with goods to be smuggled into Britain, with Douglas flourishing most of all by the activity.

During the 18th century the smuggling trade grew to such proportions that the British Government passed the Revestment Act in 1765, by which the Atholl Lordship was terminated and George III of England became the First Regent, Lord of Man.

From the Revestment Act (1765) to the Present Day.

The drastic changes in the Manx constitution enacted by the legislation in 1765 were initially a disaster for the Island. Although Tynwald, the Manx Parliament, and its branches still survived, no laws costing money could be passed since the customs duties were directed to the British Government. These conditions remained until 1866 when the Manx customs revenue was again transferred to the Island, but with the stipulation that the British Treasury should have the ultimate approval of spending that money. This limitation was repealed only in 1958, so the Island has now more freedom in the conduct of its own affairs. Today, the Lieutenant - Governor is the representative of the British Crown in the Island's Government, while Tynwald comprises the House of Keys and the Legislative Council, akin to the English House of Commons and House of Lords respectively. At the present time there is a small but vocal group of the Manx population pressing for the abolition of all remaining links with the British Government, and for the development of the Isle of Man as an independent nation.

In the economic and social fields conditions gradually became more stable after the abolition of smuggling, and the Island began to benefit by its closer connections with Great Britain in various ways, in particular with the introduction of improved methods of agriculture and more intimate cultural contacts. The second half of the 19th century saw the development of a period of considerable prosperity which has continued, with only small fluctuations, up to recent years.

Place - Names

It is possible that many Celtic place - names are pre - Norse but the only ones for which there is good evidence are Douglas and Rushen (Gelling 1968). The fusion of Gael and Norseman eventually had its influence on the language of the latter people for they spoke a hybrid dialect interspersed with words of Gaelic extraction. The result is that many of the place - names date from this period of the Gall - Gaels. (Kneen 1937)

The majority of Manx place - names are of Gaelic extraction with balla - "a homestead," from the Irish 'baile', as the most common prefix attached to place - names. Study of the Manorial Roll of 1511 - 1515 suggests that many of these date from the fifteenth and sixteenth centuries, but it remains possible that some, especially those in which the second element is not a family name but a topographical term, may have arisen during the period of Norse rule. From the history of the word 'baile' in Ireland, (Price 1967), it is unlikely that the word was used in Gaelic speaking areas at a date anterior to the Norse settlement. Other common Gaelic elements in place - names are:- cooil - 'nook,' cronk or knock - 'hill,' glen - 'valley', kerroo - 'quarterland' and lhergy - 'slope'.

Both Manx and Scottish Gaelic borrowed a large variety of terms from the Scandinavians. Many Manx coastal place - names are of Norse origin, such as vik,- 'creek', berg - 'a rock cliff', borg - 'a small hill', klettr and stakkr.- Marstrander (1932) recorded 28 names of places on the coast which have 'vik' as their final element e.g. Fleshwick. Two other Norse elements, byr which gives the modern suffix - by , Colby, Dalby and Sulby,

Figure 9

ISLE OF MAN – CELTIC AND SCANDINAVIAN PLACE – NAMES

from J.J.Kneen 'Place-names of the Isle of Man' 1925

• Celtic △ Scandinavian ▲ Hybrid

and staðir, both meaning 'farmstead' are found on the Island. Marstrander states that Norse place - names of the Island have no east Scandinavian features but instead point to a south - west Norwegian idiom, closely related to the dialects of the provinces of Jaren and Agder and the Faeroe Islands.

In existing place - names the proportion of Norse to Celtic place - names in the Isle of Man is roughly 1 to 6, whereas in Lewis, the Outer Hebrides, it is nearer to 4 to 1. (Fig. 9)

The majority of English place - names used in Man are probably of very recent date but a few can be traced back to the fifteenth and sixteenth centuries such as Peel, Castletown, Milntown and Fourtowns.

An outline of the distribution of place - names of various types based on the pre - 1796 sheading divisions according to Kneen (1925 - 29) is as follows :-

Rushen. (parishes of Rushen, Arbory and Malew) The greater part of the early place - names date from the eleventh and twelth centuries, and are mostly Norse, such as Fleshwick, Perwick and Spaldrick. Early documents show Norse names which have since been replaced by Gaelic and later by English ones.

Middle. (parishes of Santon, Marown and Braddan)

There are only 24 recorded Norse place - names in the sheading and its toponomy belongs to the later Gaelic period.

Garff. (parishes of Maughold, Lonan and Onchan)

The personal and place - names of this sheading show that it contained a population which was more Norse than Gaelic. Of the

old treen (unit of land division) names five - sixths are Norse and one - sixth Gaelic.

Glenfaba. (parishes of German and Patrick)

Eighteen Norse names still survive in the sheading but none of them contain personal names. Byr is found in Dalby and Rheaby, staðir in Skerestal and dalr in Foxdale.

Michael. (parishes of Michael, Ballaugh and Jurby)

Place - names demonstrate that the early population of the sheading was more Celtic than Scandinavian. Of the ancient treen names, 18 are Gaelic and 10 are of Norse origin.

Ayre. (parishes of Lezayre, Bride and Andreas)

The ancient treen names show that the sheading was well colonized by the Norsemen, 19 bearing Norse names and 15 Gaelic names. The Scandinavian homestead names which still exist are Sulby, Crosby, Rygby, Grest, Aust, Leodest, Bravost and the suffix 'vik' is found in Breryk, Balywarynagh and Baly hamyg.

According to Kneen the evidence of place - names demonstrates that the sheadings of Ayre and Garff constituted a unit which was almost purely Scandinavian. In the other four sheadings Norse names only occur sporadically and they must have contained a population which was largely of Gaelic extraction which in the course of time absorbed the Norse element.

Personal Names (Surnames)

All Manx O' - names come from Ireland but at the beginning of the sixteenth century the prefix had almost disappeared. Some examples of O' - names from the Liber Assedationis (The Manorial Roll) of 1511 - 1515 are O'Fayle and O'Barron; examples from which the prefix had disappeared are Fargher, Gellen, More and Seer.

However, there is evidence that some Manx surnames originated in the Isle of Man, even though the same names are found in Ireland and Scotland. With regard to the Scottish surnames it is very improbable that any great number came to Man, for Scottish surnames did not become common until the sixteenth and seventeenth centuries and Manx surnames were well established at the beginning of the fifteenth century. The following are the surnames which may have originated in the Isle of Man :-

Callin, Callow, Anderson, Cowley, Christian, Cannell, Kerruish, Kinley, Kermode, Cubbon, Mylchreest, Mylvartin now Martin, Mylvoirrey now Morrison, Hudgeon, Kewin, Clucas, Quark, Kneale by translation Nelson, Kneen, Cringle, Quayle, Crennell, Crebbin, Shimmin, Stephen, Stephenson, Comish, Cormode, Kermeen and Quilliam.

About the beginning of the tenth century, the Celts of the upper classes, through intermarriage with the Norse, had become a hybrid race known as the Gall - Gael. In the course of time these Manno - Norsemen added mac - to their own personal names thus forming a series of hybrid names, most of which are still extant. As a consequence of this interchange of names between the two groups, a name, whether Norse or Gaelic, was, at the period when surnames were being formed, no sure indication of nationality,

and for the same reason it is now impossible to say, judging merely from the surname, whether a family is of Gaelic or Norse descent. A Norse eponym, generally speaking, merely indicates a Norse strain in the family (Kneen 1937). The following surnames Kneen allocates to this hybrid class :- Callow, Casement, Castell, Christian, Corkill, Cormode, Cottier, Costain, Cowley, Crennell, Quine, Shimmin, Corlett, Corrin, Corran, Corteen and Scarffe.

Those of Anglo - Norman descent who settled in the Island came from Ireland, and initially resided chiefly in the south, in the parishes of Arbory and Rushen. These settlers had already discarded the Norman prefix Fitz - and adopted the Irish mac -. The most important Norman patronymics resulted in the following extant surnames :- Cubbon, Quilliam, Crebbin, Qualtrough, Watterson, Kinry, Stephen and Stephenson.

According to Kneen (1937) all patronymics derived from scriptural names or names of non - Celtic saints are Norman in origin, such as Mac-Iss-ak (from Fitz Isaac now Kissack) and Mac Querkus (from Fitz Marcus now Corkish).

Translations of Manx surnames into English equivalents are also common, such as Begson for Kinvig, Gibbonson for Cubbon, Nelson for Kneale, Robinson for Crebbin, Watterson for Qualtrough and Wright for Teare.

Excluding imports from other parts of Gaeldom, the majority of Manx exotic surnames come from the northern counties of England, especially Cumbria, Lancashire and Cheshire. With the exception of patronymics ending in - son, these surnames are mostly of the local and occupative type. Of the local types the extant ones include Radcliffe, Skillicorn, Sansbury and Sayle, while the

occupative names include Taubman, Maddrell and Cooper.

Kneen's (1925 - 29) classification of Manx personal names extant on the Island is shown below.

Appendix I

Personal Names of the Isle of Man - based on J.J. Kneen. (1925)

a) Gaelic

Allan

Barron

Boyd

Brew

Cain

Caley

Callin

Callister, Collister

Callow

Campbell

Cannell

Carine, Carran, Karran

Caroon, Carown

Cashin

Clague

Clucas

Cogeen

Colvin

Comish

Condra

Cooil, Coole

Corkan

Corkish

Corris

Cowin

Cowle, Cowell

Craige

Craine

Crebbin

Cregeen

Crellin

Crye

Cubbon

Curghey

Duggan

Farrant

Fargher

Fayle

Gale, Gell, Gill

Garrett

Gawne

Gorry

Hudgeon, Hutchin

Kaighin

Kanneen

Kay, Key, Quay

Keig

Keigan, Keggin

Kelly

Kenna

Kennaugh

Kermeen

Kewin

Kewish

Kewley

Killey

Killip

Kinley

Kinnish

Kissack

Kneale

Kneen

Leece

Lowey

Martin

Moore

Moughtin

Murray

Oates

Quaggan

Qualtrough

Quane

Quark

Quayle

Quiggin

Quill, Quilleash

Quillin

Quine

Quinney

Quirk

Shimmin

Skelly

Taggart

Teare

Vondy

(b) Mixed Norse and Gaelic

Casement

Castell

Christian, Christory

Cleator

Corkill

Corlett

Corrin, Corran

Corteen

Cottier

Costain

Cowley
Crennell
Scarffe

(c) Gaelic Translated and English

Bell

Black

Bridson

Clarke

Creer

Creetch

Crowe

Dawson

Drinkwater

Duke

Farrant

Frowde

Gelling

Gick

Goldsmith

Halsall

Hampton

Harrison

Homes

Hunter

Knight

Maddrell

Morrison

Radcliffe

Sayle

Skinner

Stephenson

Stowell

Taubman

Wattleworth

Woods

The afore mentioned broad sub - divisions of extant Manx personal names based on Kneen's'Place names of the Isle of Man' (1925 - 29), should not be regarded as definitive. In a later volume entitled 'Personal Names of the Isle of Man (1937) by the same author, a different origin is given for at least four surnames. Whereas Callow, Hudgeon, Quine and Shimmin are classified as Gaelic in the above list, in the later volume Kneen attributes to them at least a partly Norse origin.

Fig. 10

Isle of Man - Population 1726 - 1971 and Intercensal Variation 1821 - 1971

Census	Population			Intercensal Increase or Decrease (-)	
	Persons	Males	Females	Amount	% per year
1726*	14,070	Figures not available			
1784*	24,924	" "	"	10,854	1.33
1821	40,081	19,158	20,923	15,157	1.64
1831	41,000	19,560	21,440	919	0.23
1841	47,975	23,011	24,964	6,975	1.70
1851	52,387	24,915	27,472	4,412	0.91
1861	52,469	24,727	27,742	82	0.02
1871	54,042	25,914	28,128	1,573	0.30
1881	53,558	25,760	27,798	-484	-0.09
1891	55,608	26,329	29,279	2,050	0.38
1901	54,752	25,496	29,256	-856	-0.15
1911	52,016	23,937	28,079	-2,736	-0.50
1921	60,284	27,329	32,955	8,268	1.59
1931	49,308	22,443	26,865	-10,976	-1.82
1939+	52,029	23,675	28,354	2,721	0.69
1951	55,253	25,774	29,479	3,224	0.51
1961	48,133	22,034	26,099	-7,120	-1.29
1966	50,243	23,226	27,197	2,290	0.95
1971	56,289	26,461	29,828	5,866	2.33

*Figures based on the Returns made by the Manx Clergy

+Mid-Year Estimate

2. Population (Figs. 10 to Fig 21)

The distribution of population within the Isle of Man has altered considerably over the last 150 years, reflecting changes in the evaluation of the natural resources and geographical position of the Island; an evaluation related in turn to economic, social and technological achievements.

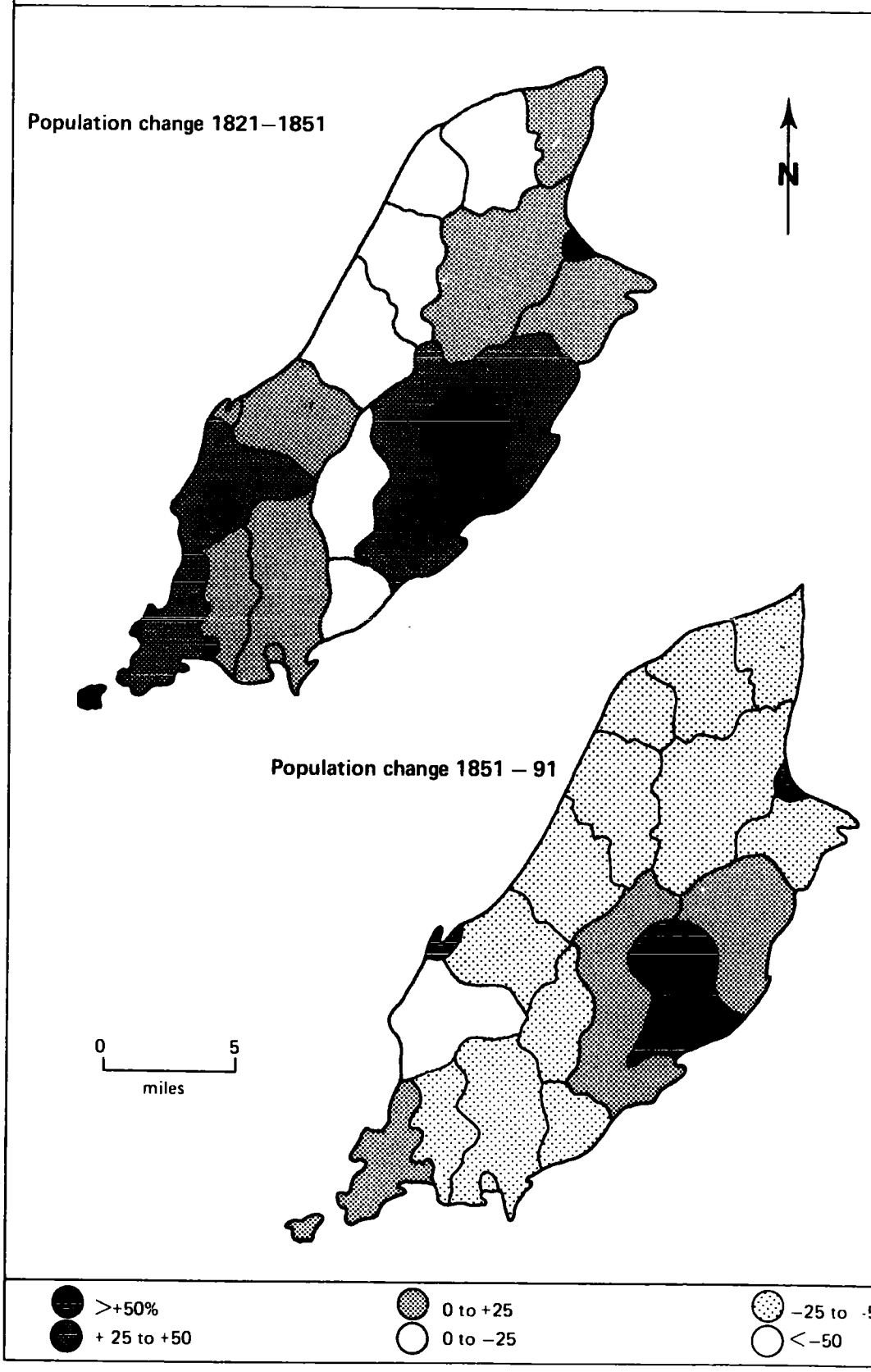
(a) Population about 1821.

Although estimates of the Manx population were made in the eighteenth century based on Clergy Returns, their scope is limited to an account of the "number of souls present", and detailed statistical records are lacking until the first official census of 1821. Moore (1900) estimated that the population in the early seventeenth century was 12,000. It is known that the eighteenth century witnessed an increase in population from over 14,000 in 1727 to 24,924 in 1784 and 40,081 in 1821, a growth rate of significant proportions (Fig. 10). This growth in population was linked to an economic expansion dominated by the lucrative smuggling trade, although such an activity had a deleterious effect on health, as goods and ships also brought vermin. Despite the epidemics of smallpox and cholera, especially in the towns (In 1765, 48 per cent of the population of Peel died of smallpox), and the economic distress with the decline in smuggling after 1765, the population increase was continued by a high, if fluctuating birth - rate.

In 1726 the population of the Four Towns, Douglas, Ramsey, Castletown and Peel had been estimated as 2,530 or 17.3 per cent of the total population. In the first official census of 1821 the urban population had reached 11,512 or 28.5 per cent of the total population of 40,081. At this period growth rates in towns

Figure 11

ISLE OF MAN – A POPULATION WATERSHED



were comparable to those of rural areas, but their natural increase rates were lower under high mortality. The influx of population into the towns, especially Douglas can be attributed to the following :-

1. Between 1737 and 1814 a large number of foreign debtors settled on the Island, attracted by the Manx Act of 1737 by which debts contracted out of the Isle of Man were not recoverable there. This arrival of debtors ceased on repeal of the Act in 1814.

2. The numerous troops stationed on the Island during the Napoleonic Wars, largely confined to urban garrisons.

3. After 1815 the immigration of numerous 'half - pay' officers began, attracted by the comparative lowness of prices and freedom from taxation.

4. Summer visitors had begun to arrive to such an extent that an Island newspaper of 1820 stated that the money received from visitors "more than equalled the returns of an ordinary fishery". (Kinvig 1958) Such an industry stimulated town growth.

b) 1821 - 1861 A Continuation of Rapid Growth

These forty years, 1821 - 1861, comprise a period of continual rapid increase in population, marked by high and fluctuating birth and death rates, tempered by significant emigration, especially in the 1830's. Despite emigration from many areas, especially the northern parishes, the early nineteenth century was a period when the rural increment was retained. Many areas of the Island show a maximum rural population about the end of this period. (Fig. 11) However, with the incorporation of the Island into closer economic activity with England, and the specialisation

of the fishing industry, movement from the rural areas into the larger settlements began. Between 1801 and 1861 the percentage urbanised in the Isle of Man rose from under 25 percent to 39.3 per cent, a rate faster than that in England and Wales during the same period. This increase was systematic of the expansion of Manx economic activity associated with ;

1. The founding in 1830 of the forerunner of the present Isle of Man Steam Packet Co. was linked with a substantial growth in the tourist industry centred on the towns, especially Douglas.

2. The growth in the fishing industry brought a significant prosperity to Peel.

3. The development of manufacturing associated with urbanised living was strongly encouraged by an expanding insular market and by a reduction in the number of articles dutiable in England.

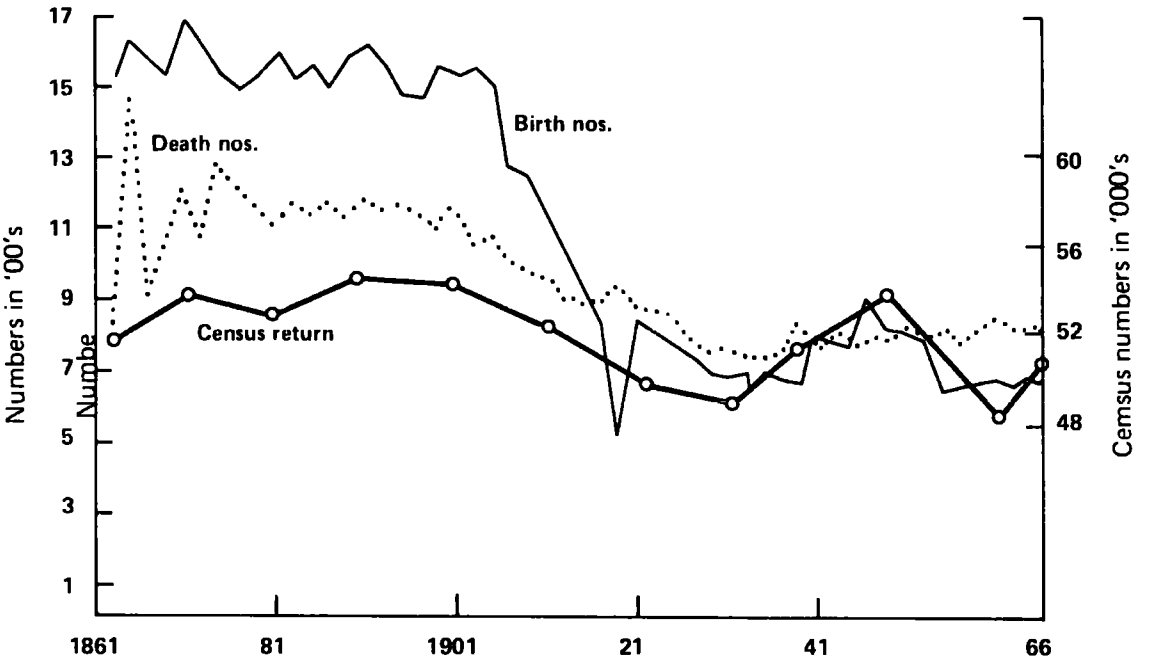
4. The growth of banking and other commercial services which expanded the activities of Douglas, which doubled in population between 1821 and 1861, while the total population increased by 31 per cent.

c) 1861 - 1891. A Slowing Down of Growth.

The mid - nineteenth century marks a watershed in rural migration, with the rural increment failing to be retained. Also, with the continued expansion of the Manx economy until late in the century, urban growth brought with it the need for public services and an urban rationale concerning family size. This, coupled with the considerable emigration between 1851 and 1861, and significant emigration at all times, brought a slowing down of growth. However, even in this period wide fluctuations occurred

Figure 12

ISLE OF MAN – BIRTH AND DEATH STATISTICS 1861–1966



especially in the death rate, when typhoid and smallpox were still prevalent.

By the latter half of the nineteenth century the Manx economy was experiencing a broadly - based expansion using all its natural resources, including location near a populous adjacent mainland. Tourism increased substantially and though this was advantageous to the Island as a whole, the amenities and accommodation were concentrated in the towns, especially Douglas.

The climax of Manx population growth in the nineteenth century is the Census of 1891, when the population of 55,608 indicated a growth of 39 per cent from the 1821 figure. The percentage in the four towns had risen from 28 per cent to 55 per cent during the same period, and the significance of Douglas, with more than one - third of the total population, demonstrated the importance of the economic and social changes that had occurred during the century. Considerable densities of purely agricultural settlements still occurred however, especially where mining and fishing supplemented farm incomes. Moreover, the mining centres of Foxdale and Laxey supported a higher density than their surroundings.

(d) 1891 - 1931 A Continuous Decline.

The contraction of the Manx economy in this period, coupled with the continued emphasis on a seasonal industry, tourism, resulted in further emigration to the English cities or the mining and farming areas of the developing countries such as Australia and Canada. The resultant fall in population was also helped by a decline in the birth - rate. (Fig. 12)

Fig. 13 Isle of Man - Population 1931-1971 Civil Parishes and Four Towns

Parish	1931		1951		1961		1966		1971	
	No	%	No	%	No	%	No	%	No	%
Andreas	952	1.9	1,097	2.0	790	1.6	732	1.5	824	1.5
Arbory	758	1.5	781	1.4	710	1.5	689	1.4	882	1.6
Ballaugh	561	1.1	544	1.0	541	1.1	505	1.0	524	.9
Braddan	3,814	7.7	5,040	9.1	4,150	8.6	4,568	9.1	4,747	8.4
Bride	452	.9	404	.7	344	.7	359	.7	338	.6
German	3,470	7.0	3,691	6.7	3,205	6.7	3,429	6.8	3,846	6.8
Jurby	386	.7	945	1.7	796	1.7	469	.9	549	.9
Lezayre	2,850	5.8	2,708	4.9	2,441	5.1	2,728	5.4	3,655	6.5
Lonan	2,144	4.4	2,408	4.4	2,025	4.2	2,114	4.2	2,267	4.0
Malew	3,185	6.5	4,078	7.4	3,485	7.2	3,825	7.6	4,787	8.5
Marown	816	1.7	919	1.7	858	1.8	908	1.8	1,014	1.8
Maughold	3,274	6.6	4,051	7.3	3,344	7.0	3,450	6.8	3,302	5.9
Michael	662	1.3	794	1.4	657	1.4	705	1.4	804	1.4
Onchan	21,245	43.1	22,474	40.7	20,123	41.8	20,877	41.4	22,885	40.7
Patrick	1,076	2.2	1,130	2.0	978	2.0	946	1.9	1,030	1.8
Rushen	3,267	6.6	3,808	6.9	3,326	6.9	3,716	7.4	4,455	7.9
Santon	396	.8	381	.7	360	.7	403	.8	380	.7
Isle of Man	49,308	99.8	55,253	100.0	48,133	100.0	50,423	100.0	56,289	99.9

Fig. 13 (contd.) Isle of Man - Population 1931-1971 - Civil Parishes and Four Towns

	1931		1951		1961		1966		1971	
Town Districts	No.	%	No.	%	No.	%	No.	%	No.	%
Castletown	1,713		1,755		1,536		2,378		2,820	
Douglas	*		21,648		18,821		19,517		20,389	
Peel	*		2,829		2,483		2,739		3,081	
Ramsey	4,198		4,621		3,789		3,880		5,048	

* 1931 figures not available

Despite a net reduction of over 6 per cent in population between 1891 and 1911 (e.g. Peel's population was reduced by 40 per cent from its peak of 1881), affecting both town and countryside, the dominance of Douglas in the tourist industry ensured its prominence, yet even this centre suffered an overall decline in the inter - war years. As a result of the emigration of the young, especially females, demographic analysis reveals a higher death rate than birth rate.

e) 1931 - Present Day. Post War Recovery. (Fig. 13.)

The recovery of population numbers during the war years was associated with an increase in the birth rate (Fig 12) and the delayed emigration during troubled times. In the immediate years preceding the Second World War, the Isle of Man participated in the general economic revival of the period as the tourist traffic increased. However, after 1949 there was a recession under severe competition from abroad, which resulted in a renewal of heavy emigration; the volume of movement reflecting the accumulation of an emigration potential from the previous decade. Between 1951 and 1961 the population declined by 13 per cent, from 55,253 to 48,123.

However, since 1961 there has been a continual and rapid increase to 50,423 in 1966 and 56,289 in 1971, a growth largely due to immigration. The average yearly percentage increase during the period 1966 - 1971 of 2.33 per cent is the highest ever recorded and the 1971 population is also the highest ever recorded. (Fig. 10) (The 1921 Census reported 60,284 persons but the figure included over 11,000 visitors as the census was taken in June.) In 1971 20,389 persons or 36.22 per cent lived in Douglas,

Fig. 14 Isle of Man - Population by Country of Birth 1951 and 1961

Birthplace	1951 No	1951 %	1961 No	1961 %
Isle of Man	35,521	64.3	32,345	67.2
England and Wales	16,243	29.4	13,069	27.2
Scotland	1,248	2.2	977	2.0
Northern Ireland	305		262	
U.K. (part not stated)	–		81	
Irish Republic	702	1.9	517	1.9
Ireland (part not stated)	10		65	
Channel Islands	18		13	
Total U.K. and Eire	54,047	97.8	47,329	98.3
Commonwealth Countries	399	1.4	386	1.4
Other foreign countries and at sea	354		302	
Birthplace not stated	453	0.8	116	0.2
TOTAL	55,253	100.0	48,133	99.9

No 1966 or 1971 figures available

while 25,196 persons or 44.76 per cent lived in Douglas together with the village district of Onchan, which is now virtually a suburb of Douglas. The Four Towns account for 31,338 persons or 55.67 per cent of the total Manx population.

Areas of once concentrated rural population associated with mining or fishing (such as Foxdale and Laxey) now carry populations more compatible with their agricultural resources, and the new clusters of settlement bear more relation to transport networks, the environment and proximity to towns.

The censuses of 1951 and 1961 recorded the country of birth of all residents in the Isle of Man at that time, and a summary of this information is shown in Fig. 14. Unfortunately similar data for the years 1966 and 1971 were not available. Though the figure may have fallen slightly over the last ten years, it can be seen that over 65 per cent of the Manx population of 1961 were born on the Island. This figure is placed in some perspective when compared with the percentage of the population of the Island in 1970 that had three or four grandparents and both parents born on the Isle of Man. This figure, as will be shown in chapter two, was under 15 per cent. The large difference between these two figures demonstrates the extent of the present mobility of the population off the Island.

Figure 15

ISLE OF MAN – SEX RATIOS 1821, 1851, 1931 and 1966

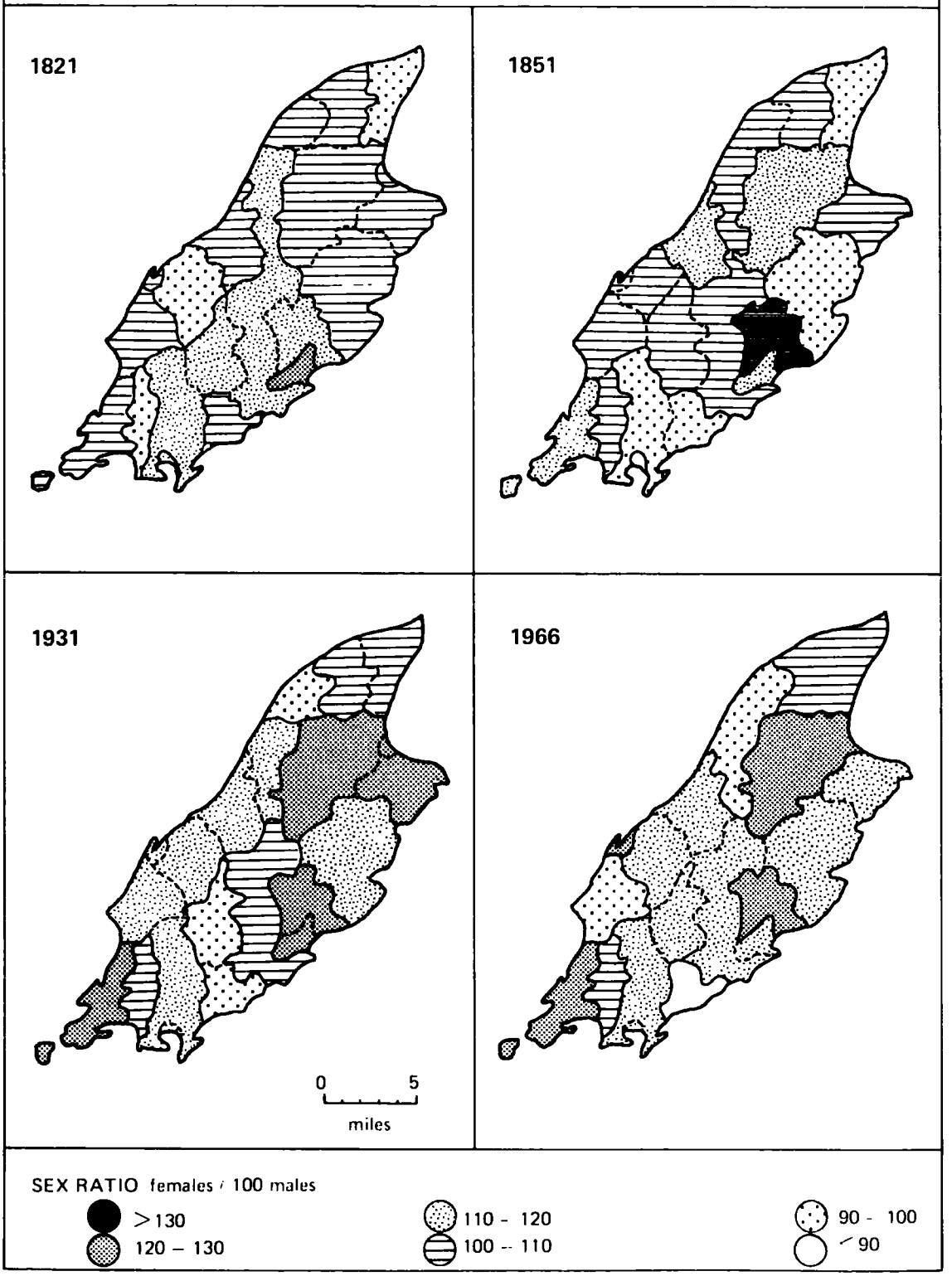


Fig. 16

Isle of Man

Civil Parishes and Four Town Districts

Population Distribution according to sex

1966 and 1971

Parish	1966 Persons	1966 Males		1966 Females	
	No.	No.	%	No.	%
Andreas	732	359	49.0	373	51.0
Arbory	689	333	48.3	356	51.7
Ballaugh	505	261	51.7	244	48.3
Braddan	4,568	2,158	47.2	2,410	52.8
Bride	359	176	49.0	183	51.0
German	3,429	1,593	46.5	1,836	53.5
Jurby	469	237	50.5	232	49.5
Lezayre	2,728	1,213	44.5	1,515	55.5
Lonan	2,114	981	46.4	1,133	53.6
Malew	3,825	1,795	46.9	2,030	53.1
Marown	908	426	46.9	482	53.1
Maughold	3,450	1,572	45.6	1,878	54.4
Michael	705	335	47.5	370	52.5
Onchan	20,877	9,439	45.2	11,438	54.8
Patrick	946	475	50.2	471	49.8
Rushen	3,716	1,659	44.6	2,057	55.4
Santon	403	214	53.1	189	46.9
Town Districts					
Castletown	2,378	1,101	46.3	1,277	53.7
Peel	2,739	1,251	45.7	1,488	54.3
Douglas	19,517	8,890	45.6	10,627	54.5
Ramsey	3,880	1,745	45.0	2,135	55.0
Isle of Man	50,423	23,226	64.1	27,197	53.9

Fig. 16 (contd.)

Isle of Man

Civil Parishes and Four Town Districts

Population Distribution according to sex

1966 and 1971

Parish	1971 Persons	Males		Females	
	No.	No.	%	No.	%
Andreas	824	412	50.0	412	50.0
Arbory	882	423	48.0	459	52.0
Ballaugh	524	258	49.2	266	50.8
Braddan	4,747	2,264	47.7	2,483	52.3
Bride	338	159	47.0	179	53.0
German	3,846	1,814	47.2	2,032	52.8
Jurby	549	282	51.4	267	48.6
Lezayre	3,655	1,695	46.4	1,960	53.6
Lonan	2,267	1,066	47.0	1,201	53.0
Malew	4,787	2,342	48.9	2,445	51.1
Marown	1,014	471	46.4	543	53.6
Maughold	3,302	1,538	46.6	1,764	53.4
Michael	804	385	47.9	419	52.1
Onchan	22,885	10,589	46.3	12,296	53.7
Patrick	1,030	523	50.8	507	49.2
Rushen	4,455	2,042	45.8	2,413	54.2
Santon	380	198	52.1	182	47.9
Town Districts					
Castletown	2,820	1,386	49.2	1,434	50.8
Peel	3,081	1,439	46.7	1,642	53.3
Douglas	20,389	9,497	46.6	10,892	53.4
Ramsey	5,048	2,331	46.2	2,717	53.8
Isle of Man	56,289	26,461	47.0	29,828	53.0

Structure of the Manx Population

The population structure of a society is not only the result of many varied population changes and interdependencies, but also the cause of many population facts. Sex and age structure determine to a large extent the population growth and influence the working capacity of the population.

Sex Structure (Figs. 15 and 16)

In the population history of the Isle of Man a strong link exists between the sex structure of individual parishes and their economic growth. Fig. 15 illustrates the essential economic distinction between the individual areas of the Island over the past 150 years. High sex ratios (i.e. excess of females over males) for the regions around Ramsey and Douglas, and for Rushen since 1851, demonstrate their dependence on tourism and their residential character, both dominated by females.

In more rural areas the dependence on male dominated agricultural and fishing activity, despite a continuous migration of farm labourers, coupled with the preponderance of females in migration, and the lack of attraction in such regions as Santon, the Northern Lowland and Patrick upon the new residents, have kept the sex ratios at roughly even proportions throughout the period 1821 - 1966. However, where the influx of the adventitious population has occurred, in Braddan, Maughold and Onchan, extremely high sex ratios are found. As the adventitious population increases so more parishes are being influenced by their characteristics.

The distribution of males and females in each of the seventeen parishes and also in the Four Town Districts for 1966 and 1971 are shown in Fig. 16. In 1966 only Ballaugh, Jurby, Patrick and

Fig. 17 Isle of Man - Distribution of Population according to selected Age Groupings 1966 and 1971

Year Group	1966						1971					
	Persons		Males		Females		Persons		Males		Females	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0 - under 15 yrs.	9,696	19.2	4,961	21.3	4,755	17.5	11,187	19.9	5,757	21.8	5,430	18.2
Over 15 - under 45 yrs.	16,479	32.7	7,986	34.4	8,493	31.2	18,696	33.2	9,389	35.5	9,307	31.2
Over 45 - under 65 yrs.	14,600	29.0	6,582	28.3	8,018	29.5	15,109	26.8	6,881	26.0	8,228	27.6
Over 65 yrs.	9,648	19.1	3,717	16.0	5,931	21.8	11,297	20.1	4,434	16.8	6,863	23.0
TOTAL	50,423	100.0	23,246	100.0	27,197	100.0	56,289	100.0	26,461	100.1	29,828	100.0

Santon exhibited an excess of males, while by 1971 Ballaugh showed an excess of females, but Andreas had the same number of each sex. The parishes of Arbory, Bride, Michael and Marown fall into the group exhibiting a relatively small excess of females. The remaining parishes, Braddan, German, Lezayre, Lonan, Malew, Maughold, Onchan and Rushen, exhibit high sex ratios in 1966 and 1971. Similarly the Four Town Districts have high sex ratios with Castletown showing the lowest and Ramsey the highest value.

The general imbalance of the population with regard to the sex ratio has had a substantial effect on Manx population growth, especially when linked to age structure, which is perhaps of greater importance.

Age Structure (Fig. 17. Appendix 2)

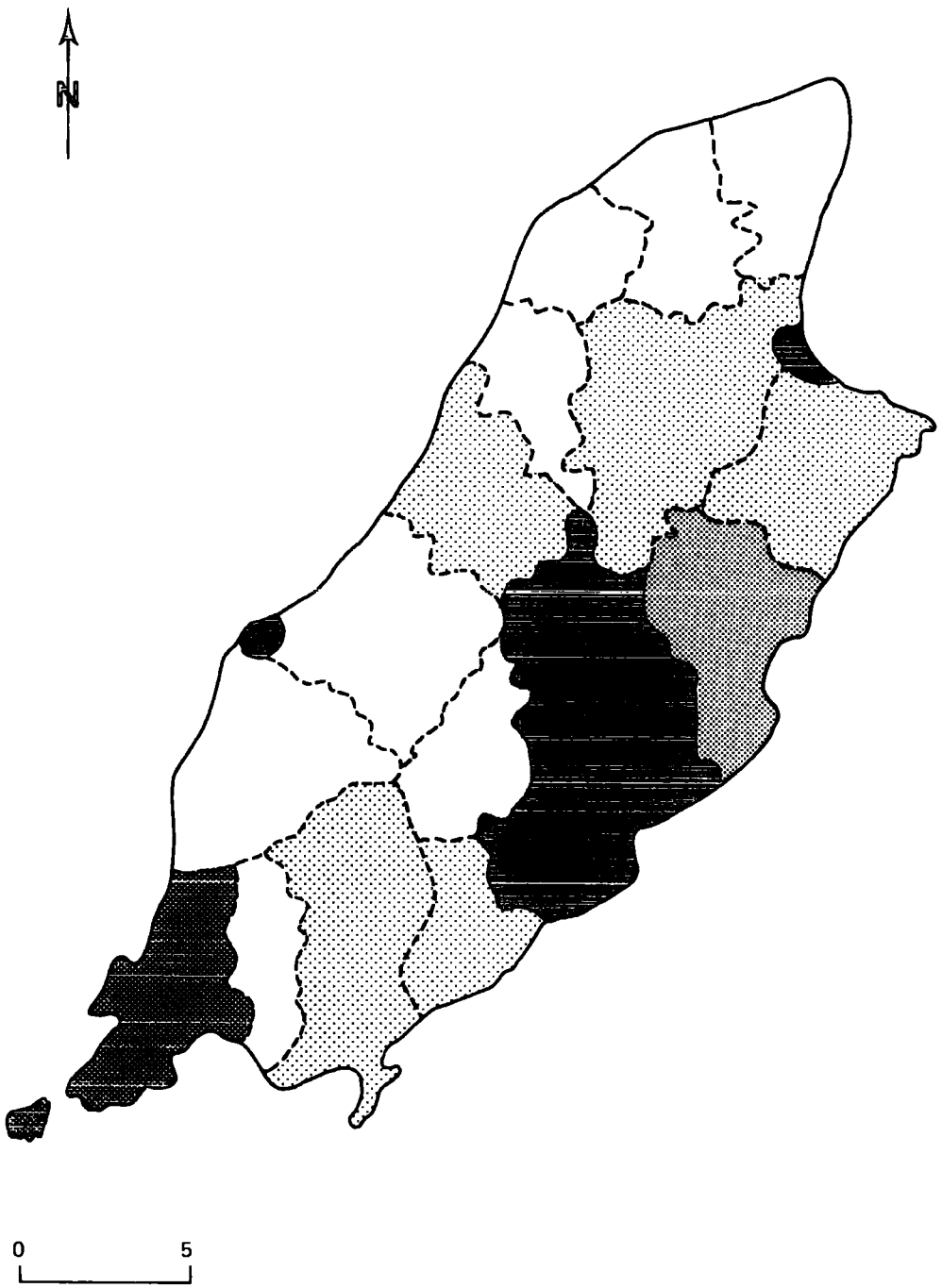
The age structure of the population not only illustrates through time the consequences of Manx economic expansion and contraction, but influences economic activity at the same time. Under the influence of constant emigration of the young in large numbers and the retention and immigration of the old, there has been a marked shift in the balance of population. This shift has accelerated in time as reproduction rates have fallen with continued emigration (Fig. 12) and immigration has risen with the general economic prosperity. The end product is a relatively very low percentage of the population under 45 years of age; (53 per cent in 1971; 57 per cent of the males, and 49 per cent of the females) (Fig. 17) compared to other regions of the British Isles, reflecting the position of the Isle of Man as not only a

depopulated rural area, but also "an extreme case of a resort area" (Dewdney 1968). In 1971 the highest number of persons of any age was 893 at the age of 61 years; the highest number of males is found at the age of under 1 year (415) and the highest number of females is found at 65 years (494). (Appendix 2)

Both emigration from rural areas and immigration to the Town Districts have produced basically the same age pattern over the whole Island, with the distinctive constriction in the age pyramid between 15 and 45 years of age and the sharp gradient in numbers over 65 years of age, reflecting the influx of the retired population.

Figure 18

ISLE OF MAN – POPULATION CHANGE 1821–1966



% Population change

- >+ 50
- +25 to +50
- 0 to +25
- 0 to −25
- −25 to −50
- <−50

Internal Migration Fig. 18

The small size of the Isle of Man meant that few areas were without some contact with cultural patterns associated with an urban society. While the urban centres of the Island, especially Douglas, attracted migrants from rural areas, such movement to these small centres must be related to the attraction exerted by the large cities of northern England. The existence of extra - island transportation rather than insular systems is therefore in some ways of greater importance in Manx migration.

Two distinct trends have accomplished the accelerated decline in rural numbers. Firstly, the movements from the rural areas, has largely been a movement of the young, between 15 and 30 years old. The dearth of young men and women in the rural areas, together with the earlier and relatively greater volume of female migration, combined to produce a fall in the reproductive capacity of the population. Secondly, the decline in the primary population (those exploiting the environment's natural resources) has tended to lessen the secondary population numbers and hence accelerate rural decline.

Fig. 18 illustrates very clearly those parts of the Isle of Man which have suffered an overall net loss of population during the period 1821 - 1966 in contrast with those areas, especially the Four Towns and Village Districts, which have seen a marked increase in their population numbers.

At the present time migration still continues, but it is within United Kingdom internal migration rather than intra - Island movements that are of significance.

Fig. 19

Isle of Man

Annual Number of New Residents and Increase or Decrease (-)

Year	Number	Increase or Decrease (-)	
		No	%
1958	272		
1959	342	70	25.7
1960	471	129	37.7
1961	516	45	9.6
1962	812	296	57.4
1963	968	156	19.2
1964	1,023	55	5.7
1965	1,179	156	15.3
1966	1,171	-8	-0.7
1967	1,072	-99	-8.5
1968	1,527	455	42.4
1969	1,741	214	14.0
1970	2,183	442	25.4
1971 (to April)	639		
TOTAL	13,916		

Immigration. (Fig. 19 - 21 incl.)

Today, as in the past, the relative freedom from taxation and absence of death duties due to the semi - independent status of the Isle of Man, attracts many persons who have private taxable income and wish to benefit more from it. However, today, the situation has changed, since many more people have reached this stage; a significant number returning to Britain in retirement after successful careers overseas. Also, with the use of Ronaldsway Airport the Isle of Man is far less remote than at any time before and has attracted those wishing to continue a business on the mainland but still receive the benefits of Manx taxation law.

Full records of Manx immigrants have been kept only since 1958. Between 1958 and 1971 the Isle of Man received 13,916 new residents, a figure equivalent to approximately one - quarter (24.72 per cent) of the total population in 1971. By far the period of the greatest increase in immigration occurred between 1966 and 1971, when 7,910 persons settled in the Island. The annual number and the yearly increase or decrease of new residents are shown in Fig. 19. It can be seen that the rapid increase of the Manx total population since 1961 is largely attributable to this unprecedented influx of immigrants. Between 1966 and 1971 new residents totalled 7,910 persons whereas the total population of the Island increased by only 5,866 persons during the same period.

The majority of the new residents originate in the United Kingdom ; between 1958 and 1971, 12,524 persons or 90.00 per cent of the total of 13,916 come from there. In the case of new residents from the United Kingdom the females exceed the males, but in those from outside the United Kingdom, the males exceed the

Fig. 20 Isle of Man - Age and sex structure of new residents 1958 - 1971

a AGE STRUCTURE

Age Group	Persons		Males		Females	
	No	%	No	%	No	%
Under 15 yrs	2,525	18.1	1,314	19.7	1,211	16.7
Over 15 - under 45 yrs	5,265	37.8	2,518	37.8	2,747	37.9
Over 45 - under 65 yrs	3,908	28.1	1,742	26.1	2,166	29.9
Over 65 yrs	2,218	15.9	1,096	16.4	1,122	15.5
TOTAL	13,916	100.0	6,670	100.0	7,246	100.0

b SEX STRUCTURE

Persons	Males		Females	
No	No	%	No	%
13,916	6,670	47.9	7,246	52.1

Fig. 21 Isle of Man

Destination of Immigrants

1958 - 1971

District	No	%
Town Districts		
Castletown	763	5.5
Douglas	3,724	26.8
Peel	797	5.7
Ramsey	1,246	9.0
Village Districts		
Laxey	395	2.8
Michael	106	.8
Onchan	1,130	8.1
Port Erin	599	4.3
Port St. Mary	472	3.4
Parish Districts		
Andreas	183	1.3
Arbory	282	2.0
Ballaugh	108	.8
Braddan	373	2.7
Bride	112	.8
German	140	1.0
Jurby	160	1.2
Lezayre	543	3.9
Lonan	381	2.7
Malew	652	4.7
Marown	286	2.1
Maughold	393	2.8
Michael	153	1.1
Onchan	81	.6
Patrick	265	1.9
Rushen	488	3.5
Santon	84	.6
TOTAL	13,916	100.0

females. The greatest intake of new residents between 1966 and 1971 was in the 60 - 64 years age category.

The data on the age and sex structure of the new residents (Fig. 20) illustrate that they are similar to the Manx total population regarding these two characteristics. There is an excess of females in both; females comprise 53 per cent of the total population (1971), 52 per cent of new residents; and there are a greater number of middle - and old - aged persons in both than in a normal British population, 47 per cent of the total population (1971) was over 45 years of age, 44 per cent of new residents.

All areas of the Isle of Man have witnessed some settlement of new residents, but by far the greatest numbers are found in, or near, the established centres of population. Douglas (26.8 per cent) has attracted by far the most new residents with Ramsey (8.95 per cent), Onchan (8.12 per cent), Peel (5.73 per cent) and Castletown (5.48 per cent) the next most popular. (Fig. 21)

Emigration

Manx emigrants before the nineteenth century were mainly those of relatively high social standing, whose numbers were small in comparison with those of the less wealthy middle or lower classes who comprised the main group later. To these latter of especial importance was the desire to escape from the very difficult social and economic conditions in agriculture during the 1820's and 1830's, particularly for many small - holders and labourers in the northern parishes of Jurby, Andreas, Bride, Michael and also around Peel. These conditions were aggravated by the attempt to collect the traditional but discontinued tithe on potatoes and other green crops, the basic crops of the lowlands, which ruined many farmers.

The destination of most of these earlier emigrants was the U.S.A. and the greatest concentration was, and still is, in Cleveland and North - East Ohio. It has been estimated that there were 25,000 - 30,000 people of Manx origin living in Cleveland in the early 1950's. (Kinvig 1955)

The limitations of the size and the economy of the Isle of Man have had a significant force on the migration of its population. As industries exploiting the natural resources declined, so the workers have looked for employment elsewhere. The last decades of the nineteenth century and the first decades of the twentieth century were the major period of economic decline, as the lack of large industrial and commercial concerns, the small immediate market and exhaustion of natural resources took effect. Owing to the increased specialisation on tourism and services, the manual worker was attracted by the greater economic opportunity in the western and southern hemispheres, or more usually in the factories and cities of the United Kingdom.

Recent Trends.

Despite an overall trend in recent years towards immigration (especially 1961 - 1971), emigration, especially by the young, still reaches significant proportions. The skilled and unskilled still seek the greater opportunities outside the Isle of Man.

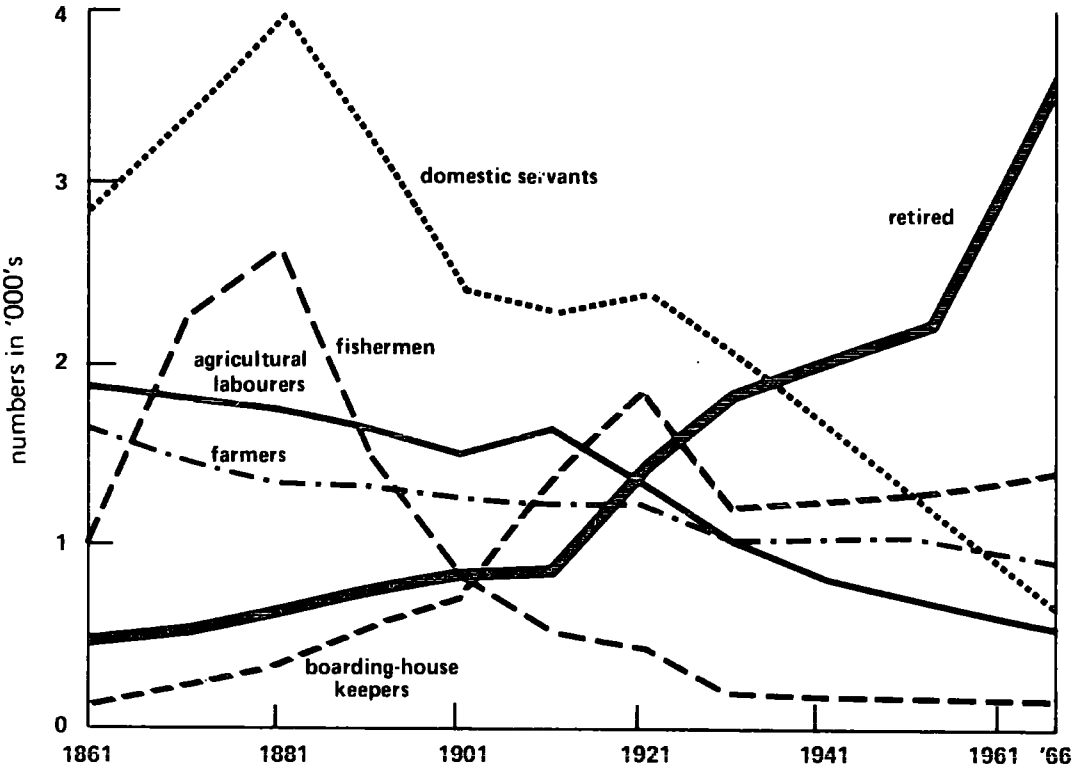
Conclusions.

Manx emigration has caused as many problems as it has solved. Although in the early nineteenth century the emigration of agricultural workers and small - holders to America eased the population pressure on limited resources, the movement of the educated, skilled and the young has had serious economic and social consequences.

These difficulties combined with the immigration in large numbers of those who are generally economically and demographically unproductive, has tended to increase the problem of the population structure of the Isle of Man.

Figure 22

ISLE OF MAN – SELECTED OCCUPATIONS 1861 – 1966



3. Economic Conditions. (Fig. 22)

The present economy is based on four main forms of activity, tourism, farming, sea - fishing and manufacturing. Largely by means of its insularity and position within the Irish Sea, the effective development of the present economy, specially concentrated as it is with tourism, awaited the technical advances which made possible the regular steamer services from the British mainland, the first of which was instituted in 1819. (Birch 1959)

History of Economic Development.

In the early nineteenth century the primary sources of livelihood were farming and fishing, neither of which were very efficient. In agriculture rotation was hardly practised and implements were very primitive. Both industries were severely handicapped by the lack of an adequate, mutual division of labour, notably during the summer herring industry. Most of the persons were said by Quayle in his Agricultural Report of 1807 to be "neither expert at fishing nor skilful cultivators of the earth." Whereas cultivation today is on the lowlands and plateaux up to a height of 600 feet, in the early nineteenth century the limit extended for another 100 feet plus. Manufacturing at this time was mainly domestic in character, meeting only local demands.

After 1830 there began a period of broadly based economic development which extended to about 1895. This involved the full range of the Island's natural resources, including its position in relation to the growing centres of industrial population on the adjacent mainland in which there was a developing demand for seaside holidays. (Kinvig 1958) Within this general period of development, the broadest economic structure existed during the

1870's and 1880's.

While the origin of the Island's tourism can be traced to the third decade of the nineteenth century, its more spectacular growth came after the critical decision in the 1860's to exploit more fully the Island's natural assets. (Fig. 22) (Birch 1959) In 1873 the annual total of visitors reached 90,000, but by 1884 this number had increased to 187,000 and the numbers increased with great rapidity throughout the century.

Manx participation in the local Irish Sea fisheries was at its peak from the 1860's to the 1880's; in 1884 nearly 400 vessels and some 2,600 men and boys were employed, the industry being very largely concentrated upon Peel. From the above date there has been a fairly steady decline so that today the Manx herring fishing fleet, despite Government subsidies, is reduced to less than five vessels. (Kinvig 1966) (Fig. 22)

The most prosperous period for mining of lead and silver deposits on the Isle of Man came after 1840 when Laxey and Foxdale came into the first rank as producers. Mining reached its heyday in the 1870's and 1880's when production figures for lead, silver and zinc reached their highest limits, and well over 1,000 men were absorbed by the mining industry between 1855 - 1880.

However, by 1895 there was clear evidence of a contraction in the Island's industrial structure and of its increasing specialisation in terms of the tourist industry, a process which continued until the mid - 1930's. Thus, while the tourist traffic rose to a record level of 634,512 visitors in 1913, all mining activity had ceased by 1919, due to increasing competition from overseas; the local fishing fleet had shrunk to insignificant

proportions, and many of the manufactures had closed down. There was some revival of manufacturing in the 1920's but this improvement was offset by the ensuing depression of arable farming and even the tourist industry experienced a reduction of activity.

In the years preceding the Second World War, the Isle of Man enjoyed some revival of economic activity, especially in the tourist trade. After the War, the number of visitors also rose rapidly bringing a brief period of prosperity extending to 1949. Since 1949, while agriculture has continued to benefit from its great war - time improvement and sustained Government support, the Island has suffered again from a decline in tourist traffic which is unlikely to be reversed.

Present Day Economic Conditions.

While the characteristic agricultural system prevalent over the whole of the farmland is one of mixed livestock farming based on cereal androot forage crops together with ley pastures, regional variations may be discerned as the result of different conditions. (Kinvig 1966) More specialised dairy - farming areas exist around Douglas and Onchan, due to the large resident population, and also to the fact that most of the visitors stay in this area. Similar smaller belts exist around Ramsey, Peel and Port Erin.

The present seasonal total of visitors to the Island is about 400,000, including about 100,000 day excursionists (Kinvig 1966), which still maintains tourism as the main Manx industry, and the one on which most of the other activities depend. According to Kinvig (1966), tourism's contribution to the Island's

gross income from external sources, apart from investments, can hardly be less than threequarters. However, over the last twenty years the accomodation potential of the Island's hotels and boarding houses has fallen greatly :-

1951	1961	1966	1971	
8,605	8,346	6,288	6,156	single beds
19,357	16,870	13,315	13,321	double beds
47,000	42,000	33,000	32,800	total number

As regards the Manx fishing industry, it is the demersal and shell fisheries which have risen to primary importance with the decline in pelagic fishing. The former now contribute about 80 per cent of the value of all fish landed by local craft. The relative insignificance of the fishing industry today can be judged by the fact that it employs under one per cent of the working male population. However, a number of small fish freezing and processing plants have been established on the Island.

Owing to the decline in all the former basic industries except agriculture, attempts have been made, with the assistance of the Manx Government, to rebroaden and stabilise the economy over the last fifteen years. This has been achieved notably through the introduction of light manufacturing industries. The main obstacles to development are the almost complete lack of raw materials and the expenses of transport, and these emphasize the need for selecting manufactures which require the maximum of skilled labour and yet are light enough to withstand import charges as well as the costs of exporting the finished articles. Successful industries meeting some or all of these conditions

include the traditional woollen mills of St. John's, knitting and cloth garment factories at Douglas, Peel and Laxey, a nylon stocking factory at Ramsey, pipe - making at Laxey and footwear at Ronaldsway. Light engineering industries such as the aircraft components factory at Ronaldsway have been especially introduced with Government assistance. In March 1965, the new factories established since 1955 employed over 1,400 men and women.

CHAPTER TWO

PHYSICAL ANTHROPOLOGICAL STUDIES IN THE ISLE OF MAN.

PHYSICAL ANTHROPOLOGICAL STUDIES IN THE ISLE OF MAN

a) Previous Studies

The first physical anthropological study of the Manx population was that carried out by Beddoe in 1887. He recorded various measurements of thirty - one heads of men "belonging to pure Manx families" and also recorded the eye and hair colour of 265 persons of both sexes of whom "many were certainly native" (Beddoe 1887). Beddoe employs the term "pure Manx descent", but never defines it in specific terms. The measurements of the head taken included the following; maximum length from the glabella, length from the inion to the most prominent part of the frontal arch, the glabella - inial length, the minimum frontal breadth, maximum frontal breadth, maximum zygomatic breadth and auricular breadth.

The most notable features were the large size of the heads, especially in breadth, and the breadth of the cheekbones, which reminded Beddoe of some Norwegian faces. The Norse influence he argued, was also to be seen in the colour of the hair, fair and light brown hair being very common, and Beddoe's Index of Nigrescence was much lower than in most parts of the Highlands of Scotland and Ireland. The distribution and combinations of colour have most resemblance to other Scandio - Gaelic districts such as Wexford, Waterford and the Inner Hebrides. His conclusion was that the physical characters of the Manx agree with their history, that the Norse element is strong, though less strong than the Gaelic. Beddoe also added that "whether there be any decided difference between the Southern and the Northern men, taken en masse, I am not prepared to say." (Beddoe 1887)

The purpose of the second anthropological investigation was "to consider the various races or race - types which have inhabited the Isle of Man, and how they have been distributed in various portions of it." This study was a report on the analysis of the 'Description Book' of the Royal Manx Fencibles by Moore and Beddoe (1898). The Royal Manx Fencibles comprised a series of regiments raised on the Isle of Man between 1779 and 1810 for service in various parts of Great Britain. The 'Description Book' contains the names of about 1,300 men who passed through the ranks between 1803 and 1810. Having subtracted all those under 18 years of age, those not born on the Island, and all those whose names are either not Manx, or are not known in the Island for a generation before 1800, even though they were Manx born, Moore and Beddoe were left with 1,112 men of native origin. The 'Book' describes their complexions, eyes, hair and stature as well as denoting the parish where each man was born and the trade he was brought up to. The proportion to which various parishes contributed to the total number varied from 1.49 per cent of the total population in Lonan, to 5.9 per cent in Malew. The occupations excluded from conscription were farming, fishing and mining.

The authors found the high frequency of fair and light brown hair and comparatively tall stature that Beddoe (1887) had described earlier. In order to determine whether there was any difference in the distribution of these physical traits, they allocated the individuals into various sub divisions of the Island, including north and south, east and west, and finally into each of the seventeen parishes. They found that the eyes of the southern people were darker than the northern, but that the hair of the northern

people was darker than those in the south. The east - west division of the Island, revealed an excess of dark eyes and dark colouring of hair in the east.

An interesting feature of the analysis was that the variations in eye and hair colour rarely corresponded, especially in the Peel district which had the greatest proportion of dark eyes and the smallest of dark hair. The Castletown and Douglas districts exhibited similar findings. However, in the northern division the exact converse was the case, especially in the parishes of Maughold and Lonan which contained in the opinion of the authors, a larger proportion of Gaels than the other parishes. When the north - west parishes of Jurby, Ballaugh and Michael were compared with Maughold and Lonan, it was seen that the former had the smallest proportion of dark eyes and nearly the smallest of dark hair, and the latter nearly the smallest of dark eyes and the largest of dark hair.

The analysis of stature showed that the tallest men were found in districts which, from the colour of hair and eyes, the authors thought contained the largest percentage of men of Scandinavian descent, that is Jurby, Ballaugh, Michael and Andreas, while the shortest men were found in those areas the authors thought were more purely Gaelic, such as Maughold and Lonan. However, it should be recalled that from the evidence of place names provided by Kneen (1925 - 29) it was suggested that Maughold and Lonan were areas well settled by the Norse, rather than Gaels.

Moore and Beddoe (1898) concluded that the native Manx population is Scandio - Gaelic, and that there is a preponderance of people with more Gaelic features in Maughold and Lonan, while

there are distinct traces of alien elements in the towns of Douglas, Castletown and Peel.

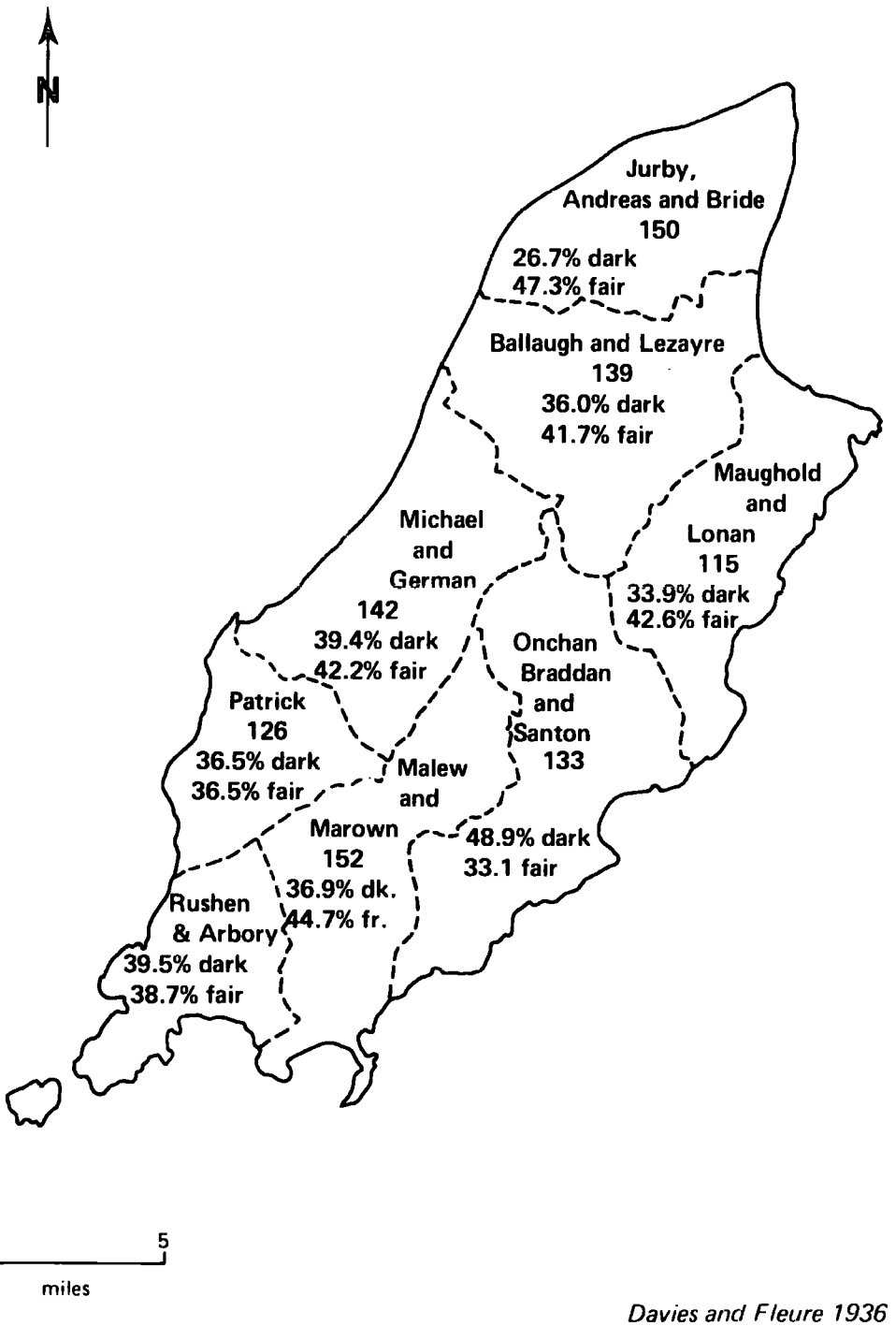
Davies and Fleure (1936) and Fleure and Davies (1937) reported upon an anthropometric survey of the native Manx population in which sixteen measurements and seven observations were recorded for 1,200 males over 21 years of age, "each of whom could give assurances that his four grandparents belonged to the Island by descent." The sample was further limited by the exclusion of any persons with family names not on record for the Island prior to 1800. Of the 213 different surnames in the sample, 172 are on record in 1511 or before, 10 are recorded from the seventeenth century and thirtyone from the eighteenth century. They estimated that their sample formed at least 16 per cent of the total indigeneous adult male population of under 7,000 in 1931.

Davies and Fleure's regional subdivisions of the Isle of Man were very different from those employed by Moore and Beddoe, in that they used seven geographical or 'natural units,' areas within which mixture was more persistent in the days of predominantly local intermarriage. Davies and Fleure also observed the differences between people from different areas of the Island in their physical characteristics including height and various indices of the head, nose and face. Their conclusion was that two physical types predominate in the Isle of Man; the Mediterrarean and the Nordic.

The Mediterrarean type is characterised by dark brown or black hair, brown eyes, long narrow head, moderate to broad nose, medium stature, slim build and short face. The authors thought that the ancestors of this type first came to Britain during the

Figure 23

PERCENTAGE DISTRIBUTION OF DARK AND FAIR COLOURING IN THE ISLE OF MAN



Davies and Fleure 1936

DARK – yellow brown pigment in iris and medium or dark brown hair

FAIR – eyes with no yellow brown pigment in iris with fair hair

Neolithic Age before 2000 B.C., from the Western Mediterranean via France, but are now chiefly found in the western fringes of the British Isles. Though the Mediterranean type was found throughout the Island, it was most numerous in the parishes of Onchan, Braddan and Santon, while they occurred as frequently as the Nordic type in Rushen and Patrick. (Fig.23)

The Nordic type is distinguished by its fair hair, light eyes, usually blue, taller stature, long narrow head, long face, long narrow nose and high forehead. Davies and Fleure linked the presence of this physical type with the period of Scandinavian settlement between the ninth and thirteenth centuries. People of this type were found most frequently in the northern parishes of Bride, Andreas and Jurby, as well as in the southern parish of Malew. (Fig. 23)

Davies and Fleure however, stress the very important fact that much intermixture has taken place over the centuries and that many Manx have blended characteristics, such as dark hair and light eyes, as Moore and Beddoe (1898) had discovered in Peel. In the Isle of Man as a whole, fair colouring (light hair and eyes without brown colouring) has a greater frequency than dark colouring (dark brown or black hair and brown eyes).

(b) The Present Survey.

(i) Scope and Aims

The Isle of Man was chosen for the investigation of local human biological variability because of its unique geographical position in the centre of the North Irish Sea basin. Moreover, the fact that it is an island, and therefore a natural region with a finite boundary,makes such a study more interesting. The degree of social and economic isolation consequent upon its geographical position, as explained in an earlier section, has varied throughout the Islands' history.

The present survey of the Isle of Man has two major aims. Firstly,an investigation of genetically controlled polymorphisms in the indigenous Manx population in order to determine whether there exists any intra - island heterogeneity in the frequency of the respective phenotypes and/or genes. It is of interest to consider whether the genetic traits used in this survey exhibit similar regional variability within the Manx population, as reported for other physical traits in previous studies; that is to determine whether the anthropometric and anthroposcopic differences are paralleled by the distribution of some of the genetic polymorphisms. The second major aim is to compare the frequencies of genetic factors in the indigenous Manx with those found in populations, preferably also indigenous, of other regions of the British Isles.

A very important decision for the survey was to determine the criterion which should be used in selecting individuals who qualify as indigenous Manx. It has been shown earlier that the Isle of Man did, and still does, attract persons with taxable capital

from the mainland of the United Kingdom and Eire, who have no previous association with the Island. It should be noted also, that all the previous surveys included only 'native' Manxmen in their samples. The criterion employed was that only individuals, three or four of whose grandparents and both parents were born on the Isle of Man, should be included in the present sample. This restricted the numbers tested, but it was felt that only in this way could we acquire a truly Manx sample, and one which enabled comparison to some extent with previous anthropological studies of the Island's population.

Another reason for selecting the indigenous Manx population for analysis is that there is good evidence from previous studies of genetic distributions in Britain (usually ABO blood groups) that if selection for a 'native' sample of the population under study is made, one finds that this group may differ, often significantly so, from the total population of which it is a constituent part. Fraser Roberts (1942) in his study of the ABO blood groups distribution in North Wales, found that if he sub-divided his sample into those with and those without recognized Welsh surnames, the former group exhibited a significantly higher frequency of group O. A similar finding was made by Fraser Roberts (1948) in his survey of ABO blood groups in South West England. He found that blood donors bearing names having the prefixes Mac and O' (distinctive to Scottish and Irish surnames) were significantly far lower in group A than the population in which they were living. However, rather than classify the Manx population by employing the list of distinct Manx surnames, which would only provide a very approximate division of the population,

it was decided, for the purposes of greater accuracy, to question each volunteer for this survey about his Manx ancestry back to the grand parental generation, including maiden names of his close female relatives.

This rigorous definition of the indigenous Manx population gave rise to many problems, not least of which were (a) collecting a sufficiently large sample, and (b) the lack of data on regions of the British Isles, based on similarly selected population samples. Very few studies of British regional populations based on samples of the indigenous population have been reported. Those that have been made have tended to concentrate on the ABO and Rh(D) blood group antigens. It was decided therefore, to investigate the native populations of two areas surrounding the North Irish Sea. The two areas, Cumbria (comprising the counties of Cumberland and Westmorland) and South West Scotland (comprising the counties of Dumfries, Kirkcudbright and Wigtown) were selected for a number of reasons. Firstly, geographical location of both Cumbria and South West Scotland place them closest to the Isle of Man. Moreover, a study of the prehistory and history of the Isle of Man, Cumbria and South West Scotland reveals that the three areas have much in common, especially the former two. Each area exhibits evidence of pre - Celtic and Celtic settlements, though it has been suggested that these populations were almost certainly tribally distinct. However, Rollinson (1967) states that the 'Sandhills Culture' spread from Ireland into Galloway, the Isle of Man and Cumbria, so that by 2000 B.C. the Irish Sea had become a single cultural unit.

Only Cumbria, and to a much lesser extent South West Scotland,

experienced Roman colonization, but apart from their buildings the Romans left no lasting evidence of their stay. Cumbria was also the only region to experience effective Anglian domination, from about 550 - 750 AD, as evidenced by place - names, especially prevalent in the coastal plain.

All three regions experienced Norse settlement between the ninth and twelth centuries, but this was especially marked in the Isle of Man and Cumbria. Evidence of the Norse influence is afforded by place names, especially those endings in -by, -dalr, -fjall and -saetr. Rollinson (1967) points out that when the Norse settled in Ireland and the Isle of Man, contact and inter-marriage brought about a fusion of cultures, so that by the time the Vikings reached Cumbria they were Norse - Irish and Norse - Manx rather than true Scandinavians. The mixed origins of these colonists in Cumbria is shown in a number of ways; churches dedicated to Celtic saints, Celtic influenced sculpture with crosses at Gosforth, Gilcrux and Muncaster, similar in style to those found in the Isle of Man. Though authority was transferred to the English King in the eleventh century the Scandinavian influence in Cumbria remained strong and continued long after the Norman Conquest, and the process of place - naming went on until the twelth century.

The formerly active Manx fishing industry of the nineteenth century had many close connections with the ports of the Solway Firth, initiated during the smuggling trade of earlier centuries, and many marriages were transacted between members of these communities. Also, the nineteenth century mining boom in the Isle

of Man attracted miners from the Lake District as well as Cornwall. The first Packet - Boat Service between the Isle of Man and the English mainland, inaugurated in 1767, ran from Douglas to Whitehaven, whereas the major sea and air connections of the present day are with Liverpool and Blackpool respectively.

The most important practical reason for selecting Cumbria and South West Scotland for investigation is that they are nearest geographically to the laboratory in Durham of all lands that border the North Irish Sea.

(ii) Materials and Methods.

In this section the selection of the population samples employed in the survey is described. In addition the methods used in collecting the material in the field and in analysing it, are also reported. That the collection of data was complex and very time - consuming was in large part due to the highly selective nature of the samples.

As mentioned above, an indigenous or native Manx person was defined as one who had three or four grandparents and both parents born on the Isle of Man. The same criterion was also applied to the Cumbrian and South West Scottish samples as far as possible. This criterion was adopted as the one most likely to produce a native sample with most accuracy. To have inquired of the ancestry of individuals beyond the grandparental generation would have taxed the memory and knowledge of most of the participants in the survey, and also would probably have considerably reduced the size and accuracy of the sample obtained.

It was important to determine, however approximately, the proportion of the total population of the Isle of Man which would qualify for the survey. It was felt that a survey of blood donors would provide the quickest answer, and therefore the Director of the Blood Transfusion Service on the Island was informed of the proposed survey. He readily agreed to the survey and it was arranged that Blood Transfusion Service (B.T.S.) staff would ask all donors attending bleeding sessions if they had three or four grandparents born on the Isle of Man, and if so, would they be willing to participate in the survey.

The survey was further publicised by a letter enclosed with a Christmas card sent to all donors by the Blood Transfusion Service. This letter explained that the donor would be asked to permit a blood sample to be taken and that a convenient date would be arranged for Mr. Mitchell to visit him in his own home to perform other tests.

When a number of blood specimens had been received at Durham the author wrote to the donors enclosing a form for completion, requesting details of their family history, including birthplaces and maiden names, (Appendix 3) and arranging a date on which to make a visit. This system had satisfactory results, producing a sample of 219 individuals. However, this number was only approximately 10 per cent of all blood donors registered with the Isle of Man Blood Transfusion Service in 1971.

To acquire a reasonably large sample it was necessary to extend the coverage of the survey to encompass other groups. Secondary schoolchildren are an eminently suitable group for a survey of biological variation in normal populations. (Cartwright and Sunderland 1967. Boyce et al 1973) The Director of Education on the Isle of Man wrote to the Head Teachers of all Secondary Schools under his authority expressing his support of the survey. The author also wrote to each Head Teacher asking permission to test those pupils at their school who qualified on the basis of family history and were willing to participate in the survey. When permission had been obtained from all Head Teachers, forms, which asked for the parents' consent to the tests, as well as details of their family history, were sent some weeks before the actual visit to the school. (Appendix 4) The following four Board of Education

Secondary Schools participated in the survey :-

Castle Rushen High School	-	Castletown
The High School for Boys	-	Douglas
The High School for Girls	-	Douglas
The Grammar School	-	Ramsey

and in addition, one private school;

The Buchan School for Girls	-	Castletown

King William's College, a public school near Castletown, did not take part in the survey because the Headmaster stated that the numbers in the school who qualified were so small that a visit would not be profitable. More than 350 secondary schoolchildren of both sexes were incorporated in the survey, though only 338 supplied finger - prick blood specimens.

The ABO and Rh(D) blood groups of women attending the ante - natal clinic in Douglas were also made available for the survey. With the permission of the Consultant Gynaecologist and the Matron of the Jane Crookall Nursing Home, all women were asked to complete a form similar to that mentioned above, requesting details of their family history. (Appendix 5) The completion of forms was much less satisfactory than in other samples; most women indicating 'Isle of Man' in answer to where each member of the family was born, without indicating the particular parish. Those women who clearly had three or four grandparents born on the Isle of Man were included in the total sample of the indigenous Manx population.

Many surveys of the regional distribution of genetic traits in the population of the British Isles have selected blood donors or the records of a Blood Transfusion Service Centre (Fraser - Roberts 1948, 1953) or schoolchildren (Dodge 1967) for their sample,

acting upon the assumption that each group is a random sample and representative of the population of which it is part. Two workers, Dawson (1964) and Kopeć (1970) looked into the problem of the randomness of donor samples. Dawson (1964) thought he had removed any bias in his survey of the population of Eire "by only including the records of those who did not know their blood groups and who were giving blood for the first time." Kopeć (1970) also thought that consecutive new donors would remove any bias in her sample, and with the exception of Belfast, Transfusion Centres sent her records of only new donors. Kopeć also compared her donors with R.A.F. data for similar areas and no significant differences emerged. It should be pointed out that the Manx survey involved asking <u>all</u> donors to participate, and not just new donors and those who did not know their blood groups. If this had not been the case, the donor sample would have been very much smaller than it is.

Increasing the sample size of the native Manx population was achieved by testing for some of, if not all, the genetic traits, people employed in various services and institutions distributed throughout the Island who qualified for inclusion in the survey. Permission for testing 'volunteers' was given by the following Services and Institutions :-

Police Service - males and females from Douglas, Ramsey and Castletown.

Fire Service - all centres - Douglas, Laxey, Ramsey, Peel, Kirk Michael, Castletown and Port Erin.

Noble's Isle of Man Hospital - Nursing and Ancillary Staff.

College of Further Education, Douglas; College of Domestic Science, Douglas and the Manx Museum and Library, Douglas. Dr. Cartwright of the Department of Anthropology in the University of Durham kindly took venous blood specimens into 5ml. sequestrene tubes which were sent to the M.R.C. Serological Population Genetics Laboratory (S.P.G.L.), London. In each case details of the individual's family history were recorded.

In addition, Sister Corkan bled individuals in the Peel district who volunteered for the survey, and Dr. G. Sabharwal did likewise for some individuals in the north of the Island.

Having collected a relatively large sample of native Manx tested for a variety of genetically controlled factors, it was necessary to obtain a sample of the indigenous population of Cumbria and South - West Scotland. As in the case of the Isle of Man, permission for the survey was requested from the Blood Transfusion Centres in Newcastle-upon-Tyne and Glasgow, which cover the two areas. Permission was given for the author to attend bleeding sessions in the two areas, at which he asked those native donors agreeing to participate in the survey, to complete the details of their family history. (Appendix 6 and Appendix 7) After attending sessions in Carlisle, Cockermouth, Maryport and Whitehaven, further visits in Cumbria became impracticable because the Blood Transfusion Staff have to work to such a tight schedule.

The Glasgow and West of Scotland Blood Transfusion Service visit South West Scotland only twice each year, but the author attended bleeding sessions held in Dumfries and Newton Stewart. However, during this period the Blood Transfusion Service changed over to collecting blood in plastic containers which

effectively prevented the collection of further specimens for the survey. Instead of collecting specimens from natives, arrangements were made for the first 100, or all, if the number was less than 100, of the side tubes collected at a session in South West Scotland to be sent to Durham for serum protein and isoenzyme analysis, after the Transfusion Service had carried out their own tests. Under this arrangement bloods were received from sessions held in Annan, Dalbeattie and New Cumnock.

In Cumbria, as in the Isle of Man, secondary schoolchildren were incorporated in the survey. Procedures were adopted similar to those used in the Isle of Man; permission being obtained from the Directors of Education in the City of Carlisle and the County of Cumberland. Also, the consent form used was similar, the only difference being that no information on family names was requested. (Appendix 8)

The Cumbrian Secondary Schools that participated in the survey were :-

St. Aidan's School	-	Carlisle
Ullswater Road School	-	Penrith
Lairthwaite School	-	Keswick
The Grammar School	-	Workington
Salterbeck School	-	Workington
Netherhall School	-	Maryport
Nelson Thomlinson School	-	Wigton

These schools are distributed widely throughout Cumberland, taking in the industrialised west coast, the market centres of Carlisle and Keswick and the predominantly agricultural areas of the Eden Valley and Solway Plain. It was felt that this coverage

Figure 24

FINAL SAMPLES

Numbers tested in each sample for each genetic factor

Sample	BLOOD GROUP ANTIGENS														SERUM PROTEINS			RED BLOOD CELL ISOENZYMES									NON–SEROLOGICAL TRAITS			
	ABO	Secretor	M+N	S	s	P_1 SPGL	P_1 Durham	Rh.	Rh(D)	Lu^a	KK	Kp^a	Fy^a	Fy^b	Ag	Hp	Tf	AP	PGM	AK	ADA	6–PGD	G–6–PD	LDH	PHI	MDH	TR	CV Males	PTC	Sk. Col.
ISLE OF MAN																													
'Native'																													
Donors	219	143	219	219	219	182	166	219	219	198	217	197	219	188	111	220	220	194	192	195	192	189	187	187	187	153			
Adult Non–Donors	136		134	134	134	154		136	136	134	134	134	134	105		136	136	131	119	131	106	106	106	106	106		270	128	283	
Schoolchildren	338		336	240				270	332																		339	175	392	163
Pregnant Women	116								116																					
TOTAL	809	143	689	593	353	336	166	625	803	332	351	331	353	293	111	356	356	325	311	326	298	295	293	293	293	153	658	303	675	163
'Non-native'																													
Pregnant Women	120								120																				
CUMBRIA																													
'Native'																													
Donors	198		198	198	158		22	198	198	141	102		158			198	199	112	140	57	44	54	30	30	30					
Schoolchildren	341	128	317	294	92			289	289																		247	137	330	252
TOTAL	539	128	515	492	250		22	487	487	141	102		158			198	199	112	140	57	44	54	30	30	30		247	137	330	252
SOUTH WEST SCOTLAND																													
'Native'																													
Donors	72		72	72	72		72	72	72	39	72		58			72	72	66	30	30	30	30	18	18	18	30				
Resident																													
Donors	199		100	100	100			100	298							298	298	269												
TOTAL	271		172	172	172		72	172	270	39	72		58			370	370	335	30	30	30	30	18	18	18	30				

would provide a truly representative sample of the indigenous population of Cumbria.

Samples collected.

(i) Isle of Man.

(a) native blood donors.

(b) native adult non - donors.

(c) native secondary schoolchildren.

(d) native females attending the ante-natal clinic.

(e) non-native females attending the ante-natal clinic.

(ii) Cumbria

(a) native blood donors.

(b) native secondary schoolchildren.

(iii) South - West Scotland

(a) native blood donors.

(b) resident blood donors.

The numbers tested in each of the above samples for the various genetic factors investigated are shown in Fig. 24.

Inter - relationships within the samples.

The problem of inter - relationships, especially primary relationships, in the Manx series was paramount. The Manx schoolchildren sample includes persons with primary and other relationships with other individuals in the sample, deliberately, because this series was drawn from virtually the total Island population aged between 11 and 16 years. In fact the Manx schoolchildren series is not so much a sample, more the total indigenous population of the Island aged between 11 and 16 years.

However, inter - relationships were also a major problem with the other Manx series. The original intention was to exclude primary kin relationships where they were known, but unfortunately there was no way of allowing for unknown relationships within the samples. It has been shown in Chapter I that a relatively small number of distinct surnames predominate on the Isle of Man, so that inevitably there are many instances of individuals bearing the same surname (especially Cain, Caine, Kelly, Quayle and Corkill) in the series; much more so than in most regions of Britain. Some of these persons may be related to each other, but the vast majority of individuals with the same name have no known relationship to each other. Accordingly, all individuals tested were included in the sample, regardless of disclosed relationship to others in the series, rather than give excess weighting to the unknown relationships within the series. One beneficial effect of this decision was that it increased the sample size slightly.

Several advantages derived from the fact that the Manx and Cumbrian series were drawn from a wide range of their respective societies. The samples collected permitted age - comparisons to

be performed for many of the genetic traits investigated. Also, in the Isle of Man, the native blood donor population could be compared with an identically selected non - donor group. In addition regional subdivision of the Manx series was possible because of the relatively large size of the sample.

Sample Size in relation to population.

It would be interesting to discover what proportion each sample collected bears to the total population group of which it is part. In the case of one sample of Manx persons collected, the secondary schoolchildren, this was possible to a relatively high degree of accuracy. Children attending secondary schools are normally aged between eleven and a maximum of eighteen years of age, though the vast majority are between eleven and sixteen years old, with only a relatively small number remaining at school for a further two years in the sixth form.

Most of the sixth form pupils were absent when the survey was being carried out in the schools

Though a small number of all ages did not wish to participate in the survey (this number was less than ten for all the schools on the Island) one can assume that the 338 schoolchildren tested for ABO blood groups approaches closely the total number of the Manx population aged between eleven and sixteen years who have three or four grandparents born on the Island. This is equivalent to 10.33%of the 1966 population and 9.82 % of the 1971 population between the specified years of age. The sample numbers tested for PTC tasting ability and tongue curling are larger, 392 and 388 respectively, because after the major period of testing schoolchildren in July 1970, the author returned to Castle Rushen

School, Castletown in October to test new first year native pupils for these two traits.

The experience of the author in the collection of Manx samples was that the percentage of individuals who qualified for the survey in any group or institution (blood donors, fire service, police service) was usually between 10% and 15%.

Therefore, an estimate of the indigenous population of the Isle of Man at the present day would be between 10% and 15% of the total population of 56,289 (1971), that is between 5,629 and 8,443 persons. In the light of these figures the 809 indigenous Manx persons tested for ABO blood groups in the present survey is seen to correspond to between 9.58 % and 14.37 % of the estimated total indigenous population of the Isle of Man. A sample that corresponds to more than 10 % of the population group that is being investigated is, by any standards, a relatively large one, and one on which the findings can be said to be safe and conclusive. However, it should be borne in mind that for many of the genetic polymorphisms investigated the Manx sample is smaller.

Field Methods.

(a) Blood Collection

The major problem encountered in the field was obtaining blood specimens, because the author is not qualified to take venous samples. Blood donors presented no problem as samples were collected at bleeding sessions into 10ml. heparinized tubes by Blood Transfusion Staff in the Isle of Man, and into 5ml. sequestrene tubes in South West Scotland. In Cumbria clotted specimens were obtained by Transfusion Service Staff and collected by the author from the Regional Transfusion Centre in Newcastle.

However, schoolchildren presented more of a problem regarding the collection of blood specimens. Finger - prick samples were collected by means of a Medi - lab lancet into tubes containing potassium E.D.T.A. The ABO and Rh(D) blood grouping of specimens donated by the women attending the ante - natal clinic was performed by the Pathology Laboratory, Noble's Hospital, Douglas, as part of normal procedure.

A saliva specimen was also taken from as many Manx and Cumbrian individuals as possible. The person was asked to rinse his mouth and spit at least 0.5ml. of saliva into an empty universal container. After collection the salivas were heated in a boiling water bath for 20 minutes to destroy inactivating enzymes, and then the coagulum was removed by centrifugation. The specimens were then stored at - 20°C until testing for secretor status was performed in Durham.

Arrangements were made to despatch the Manx blood specimens to the Anthropology Laboratory, University of Durham and also to the M.R.C. Serological Population Genetics Laboratory (S.P.G.L.)

in postal boxes by first class letter post. Dr. A. E. Mourant, Director of the S.P.G.L. kindly agreed that his laboratory should test all specimens for the red - blood cell isoenzymes and also perform blood grouping and serum protein analysis on those specimens collected while the author was absent from Durham.

The Manx schoolchildren blood specimens were transported to the Pathology Laboratory, Noble's Hospital, and tested there on the evening of the day of collection. Specimens from Cumbrian schoolchildren were despatched by private car to the Anthropology Laboratory, University of Durham at the end of each day's collection for testing the next day.

The Cumbrian clotted blood specimens were collected by the author from the Transfusion Centre in Newcastle-upon-Tyne, on the day the Staff finished their own tests. Miss M. Izatt of the Glasgow, and West of Scotland Blood Transfusion Service arranged the despatch of the selected bloods from South - West Scottish donor sessions.

(b) Phenylthiocarbamide (PTC) Taste Testing

A chance observation by Fox (1932) showed that some people are unable to taste the synthetic compound phenylthiourea (phenylthiocarbamide or PTC.), which others describe as very bitter. Inability to taste PTC is inherited as a recessive trait, but there is some evidence that the threshold is higher in heterozygous tasters(Tt) than in the homozygotes (TT). The method of determining PTC taste thresholds is very suitable for field conditions. In many instances the test was carried out in the individual's own home. The method used was a modification of the Harris - Kalmus

two - stage (a subjective followed by an objective test) technique (Harris and Kalmus, 1949). Solution numbers 2, 8, 10, 12 and 13 were omitted for practical considerations, reducing the number of bottles to be transported and simplifying the testing procedure. The dilutions and controls were made up with local tap water and administered at room temperature. The strongest solution used, number 1, contains 1300 mg. per litre and this is then progressively diluted as follows :-

Concentration of PTC Solutions

Solution Number	PTC mgm/litre
1	1300.00
3	325.00
4	162.50
5	81.25
6	40.63
7	20.31
9	5.08
11	1.27

Typically, in populations tested so far, there is a bimodal distribution of tasting acuity. In British populations the antimode is usually at solution 4. (Sunderland 1966) or between solution 4 and 5 (Harris and Kalmus 1949 ; Kitchin et al. 1959). At whatever solution number the antimodal value falls, let us 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, 9 and 11, and the other half to the non - taster category including solutions 1 and 3 as well as complete non - tasters (Sunderland 1966, Mitchell 1972).

Age differences (Richter and Campbell 1940, Harris and Kalmus 1949 and Mohr 1951) and sex differences (Hartmann 1939, Falconer 1947, Boyd and Boyd 1937 and Harris and Kalmus 1949) have been reported by some workers.

(c) Testing for Colour Vision Deficiency

Screening for colour vision deficiency in the survey was performed by means of the Ishihara Colour Plates, Third Edition, 24 plates. The book of Plates was shown to the person in a room adequately lit by daylight and held at arm's length at right angles to the individual's line of vision. All individuals who could not distinguish the correct number displayed in nine or more cases were recorded as exhibiting deficient colour vision.

However, the use of Ishihara Plates is not without strong limitations and pitfalls. Hamilton et al. (1944) found that over half of those tested and found deficient in the Ishihara Test have normal wavelength discrimination and concluded that "the Ishihara Test seems to evaluate a complex psychic bent rather than a sensory deficiency." Cole (1963) also pointed out that despite the common use of the Ishihara Test the limitations are not always appreciated. "Observers with normal vision do misread some plates due to carelessness or to an inability to make sharp distinctions." The Ishihara Tests strength "lies in its ability to differentiate the normal from the abnormal and no more." Krill et al. (1966) state that the Ishihara Plates should not be used as the only diagnostic test of red - green colour defects in population and linkage studies. 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 survey.

It is generally agreed that all the forms of red - green blindness, excluding minor defects, are inherited by the sex - linked mechanism.(Pickford 1956). Pickford (1958, 1959) suggested that the greater incidence of red - green blindness among European and American Whites (7 % - 8%) in contrast to low frequency among Asia tic Indians (4 % - 5 %) and among American Indians and Australian aborigines (2 % - 3 %) might indicate a relaxation of natural selection. Those groups have more frequent colour blindness who have developed food hygiene to a high degree and who depend less on direct hunting or gathering of food. Post (1962) discussed this relaxation of natural selection hypothesis further in the light of Vernon and Straker's findings that the highest frequency of colour blind males in Britain was found in South West England (9.5 %) and the lowest frequency (5.4 %) in North - East Scotland. (Vernon and Straker 1943)

(d)Reflectance Spectrophotometry of the Skin.

Edwards and Duntley (1939) first investigated systematically the nature of human skin colour by employing reflectance spectrophotometry. They found that variation in melanin concentration is responsible for inter - population differences in skin pigmentation. Skin colour exhibits a wide geographical variation in which a general pattern of decreasing pigmentation with increasing latitude is evident. Compared with the differences between populations, the variation in skin colour within any one population is very small. In consequence the small amount of research that has been carried out on this character has been largely concerned with the inheritance of inter - racial differences.

A reflectance spectrophotometer for measuring skin colour was not used in the field until Weiner (1951) realised the suitability of the Evans Electroselenium Limited (EEL) portable instrument. Reflectance spectrophotometry involves the measurement of the amount of light reflected from a surface illuminated at different wavelengths, relative to the amount reflected from a pure white standard such as is provided by a smooth magnesium carbonate block. The EEL instrument is fitted with nine different Ilford Filters which sample the whole of the visual spectrum. Three filters only were employed in this survey, Filters 601, 605 and 609 which correspond with the dominant wavelengths 425mμ, 545mμ and 685mμ respectively.

Harrison and Owen (1956) demonstrated that in vitro melanin concentration is linearly proportional to the reciprocal of the reflectance value at any one wavelength. Because this relationship exists over a greater range of concentrations at the red end of

the visual spectrum than at the blue, and because variations in the haemoglobin concentration affect the reflectance values least at a wavelength of 685mμ (Jansen 1953), the measurement of the reflectance of red light provides the most reliable method of determining melanin concentration in skin.

Reflectance value readings were obtained on the medial aspect of the right upper arm of each individual. It has been shown that this area is the best site for revealing inherent differences since it has a poorer tanning capacity than other suggested sites, such as the medial aspect of the forearm (Barnicot 1958) and the forehead (Lasker 1954), and also it is less exposed to ultra - violet radiation.

The investigation of skin pigmentation in the populations selected for this survey was restricted to schoolchildren in the Isle of Man and Cumbria, because the EEL instrument was required for use elsewhere much of the time this project was in hand.

(e) Tongue Curling

The survey also involved asking persons to attempt lateral edge curling or rolling of the tongue, for the existence of two fairly distinct classes with respect to the ability to perform this act was first reported by Sturtevant (1940). Sturtevant stated that " it is possible, though not proved, that the ability to turn up the edges of the tongue may be due to a single dominant gene, with the fairly frequent occurrence of additional complications," and concluded that the ability is conditioned at least in part by heredity. From their studies, Urbanowski and Wilson (1947) concluded that tongue - curling is inherited as a simple dominant character with an indication of sex - linked or sex - influenced inheritance, for they noted a greater percentage of females than males could curl their tongues. Liu and Hsu's (1949) work also supported the inherited nature of this dominant trait. Gahres (1952) suggested that the physical expression of the action of the curling gene probably involves the intrinsic muscles of the tongue. The length of the muscle fibres and possibly the pattern of the intrinsic muscles seem to be determining factors in the tongue pattern (Gahres 1952).

However, the examination of monozygotic and dizygotic twin pairs and family studies by Vogel (1957) showed that the ability of "tongue - curling" is to a certain degree, but not exclusively, hereditary. Some persons are able to learn the action and this is especially marked in children. (Sturtevant 1940) Vogel concluded that there is no evidence for the monomere inheritance suggested by some authors.

Laboratory Methods.

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 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.

Before the various methods of determining the genetic polymorphisms found in blood are listed, the background and genetics of the blood group antigens reported upon in the survey are given.

BLOOD GROUP ANTIGENS

1. ABO blood group system.

In 1900 Landsteiner described the agglutination which occurred when red cells of one individual were exposed to the action of serum from another (Landsteiner 1900, 1901). Using the naturally occurring antibodies in the serum he was able to identify three blood groups and later (1902) a fourth was described. The four groups are determined by the presence or absence on the red cells of the antigens A and B, the serum containing naturally occurring antibodies designated anti - A (α) and anti - B (β).

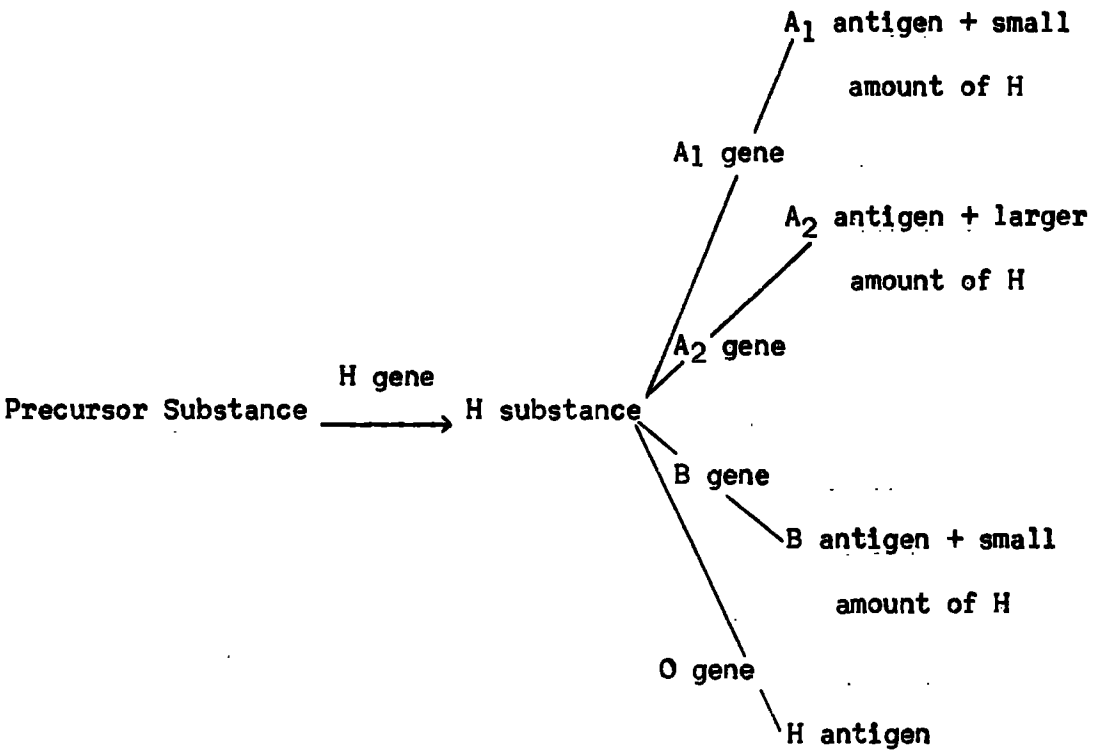
Blood Group	Antigen	Antibody
A	A	anti - B
B	B	anti - A
O	Neither	anti - A + anti - B
AB	A + B	Neither

Dungern and Hirszfeld (1911) first described sub - groups of A, termed A_1 and A_2, which brought the number of groups in the system to six: A_1, A_2, B, A_1B, A_2B and O. The present view of the ABO system is derived from the papers of Thomsen, Friedenreich and Worsaae (1930), Friedenreich and Zacho (1931) and Friedenreich (1931), cited in Race and Sanger (1970). It is generally believed that A_1 has two antigens A and A_1, while A_2 has only one, A, and the A_1 and A_2 antigens can be differentiated by the use of a specific anti - A_1 serum. In the United Kingdom approximately 20 per cent of A and AB bloods possess the weaker antigen A_2.

The method of inheritance of the ABO blood groups was determined by Bernstein (1924). The six genotypes, AA, AO, BB, BO, OO and AB, are the product of the three genes A, B and O. (p, q, and r.) The O gene is really an amorph, therefore groups A and B behave as dominant traits.

It seems there are at least two stages in the formation of the ABO blood group substances, under the control of two different sets of genes situated on different chromosomes. All normal people possess a precursor blood group substance which is acted upon by an H gene and converted to H substance. This in turn is acted upon by the respective A, B and O genes. The A and B genes convert the H substance to A and B substance, leaving a variable amount of H substance unconverted. In group O persons the H substance

is totally unconverted.



During the First World War Hirszfeld and Hirszfeld (1919) discovered that different peoples exhibited different ABO blood group distributions. Since that date hundreds of thousands of individuals have been grouped for anthropological as well as blood transfusion purposes. Collection of results have been made by Boyd (1939), Mourant (1954) and Mourant et al (1958).

Secretor Status.

The antigens A, B and H of the ABO blood group system occur not only as alcohol - soluble substances in erythrocytes and other body cells, but may also be present in body fluids and secretions as water soluble substances. Not all individuals however, secrete their corresponding ABH substances, a proportion are 'non - secretors' - that is their fluids are free from, or contain only trace amounts of, these substances. It is considered that the secretion of the ABH group specific substances is controlled by a pair of allelomorphic genes, Se and se, which are independent of the ABO locus in inheritance. Three types of individual are found; Se Se, Se se and se se. The first two are 'secretors' and the third is a 'non - secretor'.

2. MNSs blood group system.

Landsteiner and Levine (1927a and b) first discovered the MN groups in 1927 and frequencies were reported one year later. (Landsteiner and Levine (1928a and b) In the same year the two allele theory, now universally accepted, was suggested by Landsteiner and Levine (1928a). According to this theory there are two alleles, M and N, either of which determines the presence of the corresponding antigen on the red cells. Thus there are three genotypes MM, MN and NN and three corresponding phenotypes M, MN and N.

In 1947 Walsh and Montgomery (1947) described another antibody related to the MN system. The reactions were investigated by Sanger and Race (1947) and the antibody was termed anti - S. Antigen S was shown not to be an allele of M and N but that it was related to MN as the alleles of the Rh system are related. In 1951 the discovery of the antithetical anti -s was reported by Levine et al (1951b), thus confirming the hypothesis that S and s form another pair of genes closely linked with M and N. The linkage must be very close since crossing over has been shown to occur only very occasionally (Race and Sanger 1970).

3. P blood group system.

The P blood groups were discovered by Landsteiner and Levine (1927b) during the same series of experiments that led to the discovery of the MN groups. The antibody agglutinated approximately 75% of the population and these were termed P+, the remainder being 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 recognised, (Landsteiner and Levine 1927b, 1929) and the P antigen was shown to be inherited as a Mendelian dominant character (Landsteiner and Levine, 1931).

Since then there has been the discovery of antigen Tj^a by Levine et al (1951a), 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.

Phenotype under old system	Modern Phenotype	Genotype	Antibodies present in serum
P+	P_1	P_1P_1, P_1P_2, P_1p approx. 75%	Nil
	P_2	P_2P_2, P_2p approx. 25%	sometimes anti -P
P-	p	pp v. rare	anti P + P_1

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 per cent. Such differences Race and Sanger (1970) stated reflect serological and not anthropological differences.

In the present survey all bloods were tested with anti - P_1 serum only.

4. Rh blood group system.

The discovery of the Rh groups by Landsteiner and Wiener (1940) was the most important discovery in the blood group field since the ABO groups. They showed that antisera produced by injecting red cells of the monkey Macacus rhesus into rabbits and guinea - pigs agglutinated some 85 % of the white population of New York. These 85 % whose red cells were agglutinated by rabbit anti - rhesus serum they called Rh - positive, the remaining 15 % Rh - negative.

It was shown by Levine et al (1941) that erythroblastosis foetalis was the result of Rh blood group incompatibility between mother and foetus. 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, and is now called anti - LW.) 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 recognise sub - types of the Rh groups.

Employing the work of Race and others, Fisher (1944) put forward a synthesis postulating that there were six antigens inherited by closely linked pairs of allelic genes and subsequent work has proved him to be correct. The genes and antigens are designated C, D and E with their respective alleles at the same loci, c, d, and e. As only one of each pair can be carried on each chromosome it gives rise to eight Rh gene complexes.

Fisher - Race Symbols	CDe	cde	cDE	cDe	CDE	Cde	cdE	CdE
Shorthand	R_1	r	R_2	R_o	R_z	r′	r″	r_y

It means that there are 36 possible Rh genotypes, the seven most common in the United Kingdom being in order of usual frequency, R_1r, R_1R_1, rr, R_1R_2, R_2r, R_or and R_2R_2.

Fisher predicted that antisera specific for the antigenic products of the other genes would be found, and at the present time anti -D, -C, -E, -c and -e are known but an anti -d has not yet been identified. The theory of linked genes was not accepted by Wiener, who preferred to regard the Rh system as controlled by a series of alleles at a locus with complex effects.

The above is an account of the Rh system at the level at which they are commonly investigated in population surveys - that is employing five antisera, anti -C, anti -D, anti -E, anti -c and anti -e. In this survey some samples were also tested with anti $-C^w$ which meant that there were 12 possible gene complexes paired in 78 different ways. There are wide racial differences in the frequency of Rh gene complexes, values for numerous populations being reported by Mourant (1954).

D^u variant

Cells are sometimes encountered which react with some anti -D sera and not with others. These are bloods possessing a weaker form of the D antigen, termed D^u, which can be subdivided into high grade and low grade D^u. Most D^u antigens are detected with incomplete anti -D by an indirect globulin technique.

5. Lutheran blood group system.

The antibody which defines the Lutheran blood groups, anti - Lu^a, was briefly reported by Callender, Race and Paykoc (1945) and more fully by Callender and Race (1946). The notation of the blood group system is as follows :-

genes	Lu^a, Lu^b
phenotypes	$Lu(a+b+)$, $Lu(a+b-)$, $Lu(a-b+)$
antibodies	anti-Lu^a, anti -Lu^b

The antigen Lu^a was shown to be inherited as a Mendelian dominant character and to be independent of the other blood group systems. The gene Lu^b was only recognized as the absence of Lu^a until 1956 when anti -Lu^b was described by Cutbush and Chanarin (1956).

6. Kell blood group system.

The original antibody which recognised the Kell antigen was described by Coombs, Mourant and Race (1946). The antigen K was found to be possessed by only 9 % of an English population sample, who were termed Kell - positive. With the discovery of the expected antithetical antibody, anti - k (Cellano) by Levine et al (1949), the inheritance of the Kell groups by means of two allelic genes without dominance was proved.

This simple view of the Kell system lasted until 1957 when Allen and Lewis (1957) described a new antigen, Kp^a, associated with the Kell system, which occurred in about 2 % of the population, who were termed Kp(a+). The antithetical antibody, anti -Kp^b, was described later. Only two Kp(b-) persons were found in 5,500 persons and both were Kp(a+). The Sutter groups were found also to belong to the Kell system (Stroup et al 1965).

7. 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, takes its name from a patient who developed an immune antibody in response to transfusion. The gene giving rise to the recognizable antigen was called Fy^a and its allele Fy^b. The first example of the antithetical antibody, anti - Fy^b, was reported by Ikin et al (1951). They also found that the antigens were inherited by two alleles without dominance and that approximately 65 % of a sample of English adults were Fy(a+).

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°C until required for use. 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 - 1.0ml.) were separated into a tube and washed three times in normal (0.85 per cent) saline and then diluted to a 4 per cent suspension.

3) Haemolysates were prepared by the carbon tetrachloride method of Ager and Lehmann (1961). This involved washing the remaining red cells in normal saline and then packing in 1.2 per cent saline, the buffy coat being removed during this procedure. An equal volume of distilled water was added, a volume of analar carbon tetrachloride, at least equal to twice the volume of cells plus distilled water was added, and the whole throughly mixed. Tubes containing the mixture were spun in a MSE major centrifuge for 1 hour at 3000 r.p.m. The supernatant was placed in tubes and stored at - 20°C until required for use.

Antisera

The sources of antisera used in this survey are shown below, along with a brief description of the method and temperature conditions.

Antiserum	Source	Method	Temperature
anti-A	Blood Group Ref. Lab.	Saline, Tile	Room Temp.
anti-B	Blood Group Ref. Lab.	Saline, Tile	Room Temp.
anti-A+B	Blood Group Ref. Lab.	Saline, Tile	Room Temp.
anti-A_1	Blood Group Ref. Lab.	Saline, Tube	Room Temp.
anti-A_1	Liverpool B. T. S.	Saline, Tube	Room Temp.
anti-H	Hyland Laboratories	Saline, Tube	Room Temp.
anti-M	Blood Group Ref. Lab.	Saline, Tube	Room Temp.
anti-N	Blood Group Ref. Lab.	Saline, Tube	Room Temp.
anti-M	Newcastle B. T. S.	Saline, Tile	Room Temp.
anti-N	Newcastle B. T. S.	Saline, Tile	Room Temp.
anti-S	Ortho Diagnostics	Indirect Coombs Test	37°C
anti-s	Ortho Diagnostics	Indirect Coombs Test	37°C
anti-P_1	Lancaster B. T. S.	Saline, Tile	4°C
anti-D	Ortho Diagnostics	Saline, Tube	37°C
anti-C	Ortho Diagnostics	Saline, Tube	37°C
anti-c	Ortho Diagnostics	Saline, Tube	37°C
anti-E	Ortho Diagnostics	Saline, Tube	37°C
anti-e	Ortho Diagnostics	Saline, Tube	37°C
anti-C^w	Blood Group Ref. Lab.	Albumin, Tube	37°C
anti-Lu^a	Blood Group Ref. Lab.	Saline, Tube	4°C
anti-K	Ortho Diagnostics	Indirect Coombs Test	37°C
anti-k (Cellano)	Ortho Diagnostics	Indirect Coombs Test	37°C
anti-Kp^a	M.R.C. S.P.G.L.	Indirect Coombs Test	37°C
anti-Fy^a	Blood Group Ref. Lab.	Indirect Coombs Test	37°C
anti-Fy^b	M.R.C. S.P.G.L.	Indirect Coombs Test	37°C

Whenever possible the same batch of antisera was used for the whole series collected in the Isle of Man, Cumbria and South West Scotland. Also, all cells had negative and positive controls set up with them.

The serological techniques employed vary with the type of antisera used, specifically depending upon the complete or incomplete nature of the antibodies. Complete antibodies are usually IgM globulins, 950 A^{o} long, while incomplete antibodies are usually Ig G globulins, 250A^{o} long. In saline solution the cells do not come close enough together for the free antigen combining sites on the shorter IgG molecule to reach a free antigen site on adjacent red cells. Complete IgM molecules are capable of bridging the red cells in saline, thus producing agglutination. Incomplete antibodies can be made to agglutinate red cells by adding papain or albumin, the latter affects the field charge between cells so as to allow IgG antibodies to cause agglutination.

Blood Grouping

There are three main methods of blood grouping :-

1. Tile technique. The tile technique involves the use of an equal volume of antiserum and 4 per cent saline suspension of red cells. The cells are mixed with the particular antiserum on a clean white tile and left for a fixed period of time at a certain temperature. 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 - A + B, anti - M, anti - N and anti - P_1 .

2. Tube technique. The tube technique involves placing an equal volume of 4 per cent saline suspension of cells to be tested with antiserum in a precipitin tube, which is left for a specific period of time at a certain temperature. After this time the mixture is examined for agglutination microscopically. The following antisera required this method ; anti - A_1, anti - M, anti - N, anti - D, anti - C, anti - c, anti - E, anti - e and anti - Lu^a. In the case of incomplete antibodies 30 per cent bovine albumin was added as an overlay for the reason described above after one and one - half hours, and left for a further 30 minutes. Only anti - C^w serum required this latter technique.

3. Indirect Coombs Test.

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 5 - 10 per cent saline suspension at 37°C in a precipitin tube for a specific period of time. Afterwards 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 - K, anti - k and anti - Kp^a. Also all Rh(D) negative cells were tested by this method for the presence of the D^u antigen.

Starch - Gel. Electrophoresis.

The protein and enzyme systems all require fairly strict control of electrophoretic methods, pH, temperature, strength of buffer solution, and purity of ingredients used in the buffer and incubation mixtures. Electrophoretic conditions should be designed to give optimum separation of isoenzymes without any loss of activity. All electropheretic 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 paper.

SERUM PROTEINS

1. Haptoglobin (Hp)

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. A simple genetical hypothesis involving two autosomal alleles, Hp^1 and Hp^2, was suggested by Smithies and Walker (1955) to account for the inheritance of the three phenotypes, 1-1, 2-1 and 2-2. Phenotypes 1-1 and 2-2 are the expression of the homozygous forms and 2-1 of the heterozygote of the Hp^1 and Hp^2 genes. Extensive family studies (Smithies and Walker 1955, Galatius - Jensen 1958, Harris et al 1959) confirmed this hypothesis, although some rare exceptions have been noted.

The Hp^1 gene has a single product which yields a fast moving band. The Hp^2 gene gives rise to a series of polymers with different mobilities in starch - gel. The heterozygote of these two genes, Hp 2-1, shows a band in the position of the Hp^1 band, 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.

Though ahaptoglobinaemia or the quantitative variant phenotype Hp 0-0, characterized by a complete lack of detectable haptoglobin, 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) This phenomenon appears to be very uncommon in European populations but is much more frequent among Negroes (Giblett 1969). It could be a product of a modifying gene acting upon the Hp locus.

Walter and Steegmuller (1969) compiling all available data under the various race groupings found a distribution of Hp alleles where Hp^1 allele is more frequent in South America, Africa and Australia than in Europe and Asia. India is marked by a very low frequency of Hp^1.

Factors affecting the maintenance of the Hp^2 gene have been attributed to selective advantage conferred by environment or by malaria (cited in Walter and Steegmuller 1969). It is known that the Hb binding capacity of the 3 Hp types differs Hp1-1 > Hp2-1 > Hp2 -2. From this it has been inferred that Hp2-2 is selected against in an area prevalent with haemolytic disease (Baxi and Camoens 1969). Weitkamp et al (1972) also found a positive correlation between ahaptoglobinaemia and malaria infection in a study of the Yanomama Indians in Venezuela.

2. Transferrin (Tf)

Transferrin (Tf) is a B-globulin that transports iron from the plasma to the receptor cells of the bone marrow and tissue stores. Smithies (1957) demonstrated the existence of inherited variation in its molecular structure. More than twenty variants of Tf have been i dentified, but 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. TfD variants are those having an electrophoretic mobility less than Tf C, while Tf B variants move more rapidly than Tf C. Variants other than C are relatively uncommon, and have generally been found in combination with C, the unusual variant being present in amounts about equal to that of C.

Family studies suggest 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.

So far no specific association between an unusual Tf variant and any particular clinical disorder has been found. However, Walter and Bajatzadeh (1971) have suggested that the relatively high Tf^D gene frequencies in tropical biotopescould indicate a better physiological functioning of these variants in poikilothermic regulation. Ashton (1965) reported a positive association between another slow variant, Tf^E, and a tolerance to hotter climate in cattle.

Method

Hp and Tf typing was performed on the same starch gel because the electrophoretic conditions required are identical. The method is basically that of Smithies (1955) but using the discontinuous system of buffers described by Poulik (1957). One drop of a 4 per cent suspension of fresh haemoglobin is added to three drops of plasma, and the sample so treated is inserted into the gel using a Whatman No. 3 filter paper insert. Horizontal starch gel electrophoresis is carried out at 30mA, 500V. for 3 hours at + 4°C. The buffer system is discontinuous and is made up as follows :-

Tank Buffer	pH 8.5	
0.3M	Boric Acid	46.3g.
0.05M	Sodium Hydroxide	6.0g.
	Distilled Water	1.0L
Dilute for use	1 / 5	
Gel Buffer	pH 8.7	
0.076M	Tris	23.0g.
0.005M	Citric Acid	2.62g.
	Distilled Water	1.0L

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

After electrophoresis the gel is sliced and one - half stained with the solution given below, which detects the presence of the Hb/Hp complex. The benzidine stain used by Smithies (1959), also employed in the Laboratory in Durham, contains 100ml. distilled water, 0.5ml. glacial acetic acid, 0.2g. benzidine and 0.2ml. 30% hydrogen peroxide. The cut surface of the gel is flooded with the stain.

The other half of the sliced gel is stained with a protein stain, 1% Amido - schwarz 10B, for the determination of Tf types. The dye is dissolved in a methanol - water - acetic acid solution, proportions 50 - 50 - 10 respectively and left on the gel for 30 seconds. Then the gel is decolourized in the same solution without the dye.

(3) Beta Lipoprotein Allotypes

Ag^x Antigen

Beta - lipoprotein molecules are the major cholesterol carriers in plasma.

Using the technique of micro - diffusion in agar with human antisera from transfused individuals, Allison and Blumberg (1961) and Blumberg et al. (1962) demonstrated a genetically controlled polymorphism of the human B - lipoproteins. Later, a new isoprecipitin serum was discovered that reacted with approximately 40% of sera from unrelated Swedish individuals. (Hirschfeld 1963, Hirschfeld and Blomback 1964). Family studies showed that the antigen, termed Ag(x), was inherited as a dominant autosomal trait. Consequently the existence of an antithetical allele to the gene Ag^x was predicted and provisionally designated Ag^y. In 1966 Contu (1966) and Okochi (1966) independently discovered two reagents which, on comparison by Hirschfeld, were found to give identical reaction patterns, and seemed to react with a factor controlled by the Ag^y gene. This antibody was called anti - Ag(y) by Hirschfeld et al. (1966). The hypothesis is that the factors Ag(x) and Ag(y) are controlled by a pair of allelic genes Ag^x and Ag^y.

The potential usefulness of the two Ag antigens as genetic markers is indicated by the high frequency of the Ag^x gene in the Asiatic countries (0.70) as opposed to the relatively low frequency in Northern Europe (0.20 to 0.25).

Mr. D. Tills of the M.R.C. S.P.G.L. kindly performed the analysis of the B - lipoproteins of Manx plasma specimens. The technique of double diffusion in agar gel was used for testing the plasma.

RED CELL ISOENZYMES.

The physical anthropologist is not directly interested in the quantitative substrate activity of particular enzymes, but only with their multiple molecular form. These multiple molecular enzymes were termed isoenzymes by Markert and Møller (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. All the isoenzyme systems investigated in this survey have been analysed using red cell erythrocytes because these were most readily available.

Relative to the blood group antigen polymorphisms the red cell enzyme polymorphisms offer a number of advantages; many of them are still to be discovered (Harris 1969b), the reagents required for their study are, at least in principle, unlimited, and the enzymes stand in a closer relation to their structural genes. (Scozzari et al. 1970)

All the red cell isoenzymes reported upon were typed at the M.R.C. S.P.G.L. under the supervision of Dr. A. E. Mourant. However, typing was performed by the author in Durham for acid phosphatase, phosphoglucomutase and adenylate kinase.

The following isoenzymes were examined in some, if not all, specimens.

1. acid phosphatase AP
2. phosphoglucomutase PGM
3. adenylate kinase AK
4. adenosine deaminase ADA
5. 6 - phosphogluconate dehydrogenase 6 - PGD
6. glucose - 6 - phosphate dehydrogenese G - 6 - PD

7. lactate dehydrogenase LDH

8. phospho hexose isomerase PHI

9. malate dehydrogenase MDH

1. Acid phosphatase (AP)

Hopkinson et al (1963) first demonstrated genetically controlled electrophoretic variants of the enzyme AP which catalyses the reaction involving phosphor us transfer. When haemolysates were examined by starch gel electrophoresis they recognised five phenotypes which they called A, BA, B, CA and CB. On the basis of family studies it was suggested that the five phenotypes were controlled by three co - dominant autosomal alleles P^a, P^b and P^c, and the existence of a sixth phenotype C, genotypically CC, was predicted. This rare C phenotype was reported by Lai et al (1964) whose data also confirmed the inheritance hypothesis of Hopkinson et al (1963).

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).

Walter and Bajatzadeh (1968) pointed out that the low P^a and high P^b frequencies in Central and South American Indians are similar to the distribution found in Negroes, the natives of New Guinea and Australian aborigines. Since these populations inhabit a tropical environment they discussed selective factors specific to tropical living conditions whose nature remain unexplained. Wyslouchowa (1970) also suggested that the world distribution of AP alleles indicated some environmentally induced selective pressures are at work. Ananthakrishnan and Walter (1972)

found a marked gradient in the world distribution of the AP alleles. The frequency of the P^b allele 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.

Method.

The method used is that described by Hopkinson et al (1963).

Tank Buffer

0.41 M	Citric Acid	86.1615g.
	Sodium Hydroxide	45.0 g.
	Distilled Water	1.0 L

adjust to pH 6.0 with 4 N Na OH.

Gel Buffer

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

This enzyme is one of the most labile and rapidly forms satellite bands. This can be overcome by the addition of 0.0931 gm. of EDTA and 1ml. of 2 - mercapto - ethanol to 250 ml. of gel buffer.

The haemolysate sample is applied on Whatman No. 17 filter paper inserts and horizontal electrophoresis carried out at 6 volts / cm. for 17 hours at + 4°C. The gels are sliced and the cut surfaces covered with Whatman No. 17 filter paper to which was applied the incubation buffer containing 0.005M phenolphthalein diphosphate pentasodium, 0.2944 g.

Incubation Buffer

Citric Acid	1.05g.
Distilled Water	100 ml.

adjust to pH 6.0.

The phenolphthalein diphosphate is added immediately before use and the pH checked. The gels are then incubated for 3 hours at + 37°C. After this time the filter paper is removed and a few ml. of 0.88 ammonia added which gives a pink colour at alkaline pH. At the sites of enzyme activity phenolphthalein diphosphate is hydrolysed to give free phenolphthalein.

2. Phosphoglucomutase (PGM)

The enzyme PGM catalyses the transfer of a phosphate group between the 1 and 6 positions of glucose. The existence of multiple isozymes of human PGM detectable by starch - gel electrophoresis, and inherited variation in this enzyme were demonstrated by Spencer et al. (1964). They found that normally seven isozymes are possible, 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. PGM_1^1 produces the a and c bands and its homozygote is called PGM 1, while PGM_1^2 gives rise to the band d bands and its homozygote is type PGM 2. The heterozygote PGM 2 - 1 has all four bands; a, b, c, and d. Studies by Hopkinson and Harris (1965 and 1966) indicate that in addition to the common alleles there are at least six rare alleles at this locus, PGM_1^3, PGM_1^4, PGM_1^5, PGM_1^6, PGM_1^7, and PGM_1^8.

Most individuals are homozygous for the commonly occurring allele at the PGM2 locus, PGM_2^1, which determines a set of bands, e, f and g, well separated from PGM_1 components. Variation at the PGM_2 locus has been found only in Negroes with the Atkinson and Palmer phenotypes.

In Europe about 58 % of people are thought to be homozygous for the commonest allele at each locus i.e. PGM_1^1 PGM_1^1 / PGM_2^1 PGM_2^1 and exhibit five isozymes a, c, e, f and g. PGM_1^2 is the less frequent of the two common PGM_1 alleles in most populations, the exceptions so far being the Habbanite Jews

and Lapps in Finland. So far nothing is known of the factors that would 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.

Method

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

Bridge Buffer

0.1M	Tris	12.11 g.
0.1M	Maleic Acid	11.608g.
0.01M	EDTA (Na salt)	3.7225g.
0.01M	$Mg\ Cl_2$	2.0333g.
	Na OH	6.5 g.
	Distilled Water	1.0 L

adjust to pH 7.4 with 4N Na OH.

The S.P.G.L. found the buffers more stable if the concentration of all the ingredients was doubled.

Gel Buffer

Bridge buffer diluted 1:10 with distilled water.

The samples are applied on Whatman No. 3 filter paper inserts and horizontal electrophoresis is carried out at 5.5 volts / cm. for 17 hours at + 4^{o}C. After electrophoresis the gel is sliced and incubated for 3 hours at + 37^{o}C in the incubation buffer which consists of the following ingredients :-

Incubation Buffer

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

These ingredients are dissolved in 100 ml. of 0.03M Tris buffer, pH 8.0. After checking the pH, the mixture is placed over the gel surface.

3. Adenylate kinase (AK)

The enzyme AK catalyses the reversible reaction, 2 - adenosine diphosphate ⇌ adenosine triphosphate + adenosine monophosphate within the red cell and in muscle and other tissues. Electrophoretic variants of this enzyme were detected by Fildes and Harris (1966) and also shown to be determined by two co - dominant autosomal alleles, called AK^1 and AK^2. Individuals homozygous for the alleles are phenotype AK1 and AK2 respectively, and heterozygous persons possessing both AK^1 and AK^2 genes have the AK 2-1 phenotype. AK1 occurs in about 90% of the English population, AK 2-1 in approximately 10% and AK2 is very much rarer. Bowman et al. (1967) described further variants due to the very rare genes, AK^3 and AK^4.

Method

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

Tank Buffer

0.41M	Citric Acid	86.1615g.
	Na OH	45.0 g.
	Distilled Water	1.0 L

adjust to pH 7.0 with 4NNaOH.

Gel Buffer

0.005M	Histidine	1.0482g.
	Distilled Water	1.0 L

adjust to pH 7.0 with 4NNaOH.

After electrophoresis the gels are sliced and both sides of the point of insertion are covered with the incubation mixture, which is applied as an agar overlay.

Incubation Mixture

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

10 .0 M	Glucose	0 . 1802 g.
1 . 0mM	ADP	0 . 0439 g.
0 . 4mM	NADP	0 . 0255 g.
0 . 012%	MTT	0 . 0120 g.
0 . 012%	PMS	0 . 0120 g.
20 . 0mM	$MgCl_2$	0 . 4067 g.
0 . 04units/ml.	G - 6 - PD	4 . 0 units
0 . 08units/ml.	Hexokinase	8 . 0 units
	Agar	0 . 75 g.

Half of the distilled water is heated to achieve complete solution of the agar, which is then allowed to cool to approximately 60°C. The rest of the ingredients are then dissolved in the remaining 50 ml. of distilled water, and the two solutions mixed before being poured on to the cut gel. The agar is allowed to set, and then the gel is incubated at + 37°C for two hours. During this time the isoenzymes become stained in the agar as dark purple bands.

4. Adenosine deaminase (ADA)

In 1968 Spencer et al. (1968) discovered that the enzyme adenosine deaminase (ADA), an aminohydrolase catalysing the deamination of adenosine to inosine, showed electrophoretically different isozyme patterns in human haemolysates. They observed three different patterns, ADA 1, ADA 2-1 and ADA 2, and family studies indicated the patterns to be genetically controlled by two co - dominant alleles at an autosomal locus, termed ADA^1 and ADA^2. The phenotypes ADA 1, ADA 2-1 and ADA 2 according to this hypothesis represent the genotypes ADA^1/ADA^1, ADA^1/ADA^2 and ADA^2/ADA^2 respectively.

In European samples the ADA^2 frequency has been found to vary between 0.05 and 0.07 whereas it is lower, 0.03, in Negroes, and much higher, 0.12, in Asiatic Indians. Two very rare alleles have been reported since 1968. (Hopkinson et al. 1969, Dissing and Knudsen 1970)

Method

Bridge Buffer

0.1 M	Citric Acid	21.0g.
	Distilled Water	1.0L

adjust to pH 5.0 with 4NNaOH.

Gel Buffer

Use the Bridge Buffer diluted 1:20 with distilled water. The haemolysate samples are applied on Whatman No. 3 filter paper inserts and horizontal electrophoresis is carried out for 3 - 4 hours at 250 volts/gel. The initial current should not exceed 60m A. The gels are then sliced and the cut surface stained for ADA types using the agar overlay technique.

Staining Mixture

Mix 60 ml. of 0 . 1 M tris pH 8 . 0, and 40 ml. distilled water.

Add 0 . 75 g. agar to 50 ml. of the above solution and heat to dissolve.

Dissolve 40 mg. adenosine, 150 mg. sodium arsenate, 14 mg. MTT and 14 mg. PMS in the remaining 50 ml. of the above solution and add:

0 . 2 ml. nucleosidepyrophosphorylase

0 . 2 ml. Xanthine oxidase

Mix this and the agar, and pour over the gel and incubate for .5 - 1 hour at + 37°C.

5. 6 - phosphogluconate dehydrogenase (6 - PGD)

Like G - 6 - PD, the enzyme 6 - PGD takes part in the hexose monophosphate shunt, which in man converts hexoses into pentoses for the biosynthesis of nucleic acids. It catalyses the next step in the chain of reactions after that catalysed by G - 6 - PD, causing the conversion of 6 - phosphogluconate to ribulose - 5 - phosphate, the coenzyme NADP being simultaneously reduced to $NADPH_2$.

Fildes and Parr (1963) first demonstrated that this enzyme exhibits genetical variation. They found three electrophoretic patterns and showed that these isozymes represented the two homozygotes and heterozygote of a pair of allelic genes, PGD^A and PGD^C at an autosomal locus. The commonest phenotype, A, has one 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 and PGD^C genes. Individuals homozygous for the PGD^C gene exhibit the 'Canning' variant. Further variants due to alleles at the same locus have been found, including PGD^H and PGD^R (Fildes and Parr 1964, Parr and Fitch 1964 and 1967 and Parr 1966) but all are very rare.

The frequency of the PGD^C gene ranges from 2 to 3 per cent in European populations tested so far, slightly higher in Africans, but reaching 8 - 16 per cent in populations of the Middle East and a world high of over 23 per cent in Bhutan.

6. Glucose -6-phosphate dehydrogenase (G-6-PD)

This enzyme forms part of the hexose-monophosphate shunt, converting G-6 phosphate to 6-phospho-gluconate. Present evidence indicates that the enzyme appears to be controlled by several different alleles at a single locus which is situated on the X chromosome . Starch gel electrophoresis of red cells in normal males usually shows G-6-PD type B, but sometimes type A, the latter being usually restricted to Negroes. As the gene is X-linked the BA phenotype is observed only in females.

7. Lactate dehydrogenase (LDH)

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, 5 molecular types are present, A_4, A_3B, A_2B_2, AB_3 and B_4, giving rise to 5 electrophoretically distinct bands. The two chain types are the products of two sets of alleles at loci which are not closely linked. Variations at either locus are rare, so that only heterozygotes with the normal allele are known. (Vesell 1965) Caucasians show a low frequency of variants with only 7A and 1B variant having been reported. (Vesell 1965, Mourant et al, 1968)

Method

Tests for the above three systems, 6 - PGD, G - 6 - PD and LDH, can be performed by horizontal electrophoresis in the same buffer system and on the same starch - gel. One half of the gel is developed in incubation buffer for LDH and the other half is first developed for 6 - PGD and then for G - 6 - PD by the paint - brush technique of Fitch and Parr (1966).

Tank Buffer

A	0.2M	Mono potassium phosphate (KH_2PO_4)	27.22 g/ltr.
B	0.2M	Disodium hydrogen phosphate ($Na_2PO_42H_2O$)	28.44 g/ltr.

These are mixed in proportions 508 A

492 B

Gel Buffer

The Tank Buffer is diluted 1 : 20 with distilled water

Horizontal electrophoresis is carried out at 12 volts /cm. for 3 hours at + 4°C.

After electrophoresis the gel is sliced and one surface is stained with the undermentioned incubation buffers.

a) 6 - PGD

Incubation Buffer

To 10 ml. of 0 1 M Tris, pH 8 . 0, is added;

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

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

b) G - 6 - PD

Incubation Buffer

To 10 ml. of 0 . 1 M Tris, pH 8 . 0, is added;

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

The gel is then incubated for half an hour at + 37°C.

The other cut surface of the gel is stained with the following incubation buffer for determination of LDH phenotypes.

c) LDH

Incubation Buffer

10 %	Lactic Acid	0 . 25 ml.
	NAD (DPN)	0 . 005 g.
	PMS	0 . 005 g.
	MTT	0 . 005 g.

These are dissolved in 0 1 M Tris buffer, pH 8 . 0, placed on the gel and then incubated for 1 hour at + 37°C.

8. Phospho - Hexose Isomerase (PH1)

PH1 catalyses the reversible conversion of glucose - 6 - phosphate to fructose - 6 - phosphate, and it is widely distributed. Detter et al (1968) examined the haemolysates of nearly 3400 unrelated individuals from several different populations and found in addition to the usual pattern, termed PH1 1, ten distinct variant isozyme patterns called 2 - 1, 3 - 1, 4 - 1 etc. Autosomal co - dominant inheritance has been demonstrated for those variants that have been subjected to family studies, and PH1 1 individuals are homozygous for the common allele at this locus. All of the variant isozyme patterns are rare in the populations reported upon to date.

Method

Buffers

0 . 21 M	Tris	25 . 4 gm.
0 . 15 M	Borate	9 . 3 gm.
0 . 006M	E D T A	1 . 75 gm.
	Distilled Water	1 . 0 L

Gel Buffer

Use above buffer diluted 1 : 10, pH 8 . 6.

Bridge Buffer

Use above buffer undiluted; pH 8 . 0

The samples are applied on Whatman No. 3 filter paper inserts and horizontal electrophoresis is carried out at 12 V / cm. (13mA/ gel) for 20 hours at + 4°C. After electrophoresis the gel is sliced and the lower half is stained for 10 mins. at room temperature, with the following stain:-

Stain

The following ingredients:-

0 . 00032 M	F - 6 - P	0 . 0110 g.
0 . 005 M	$MgCl_2$	0 . 1 g.
0 . 00013 M	NADP	0 . 01 g.
0 . 00024 M	MTT	0 . 01 g.
0 . 00013 M	PMS	0 . 004 g.
	G - 6-PD	0 . 05mg.

are dissolved in 100ml. 0 . 05 M Tris, pH 8 . 0.

9. Malate dehydrogerase (MDH)

MDH catalyses the reversible oxidation of malate to oxaloacetate. Two electrophoretically distinct forms of the enzyme are found in man, one in the cytoplasm and one in the mitochondria. The cytoplasmic form is present in red cell haemolysates and is the one reported upon in this survey.

Genetically controlled polymorphism of the cytoplasmic enzyme was first demonstrated by Davidson and Cortner (1967) in a survey of 1,440 North American Whites and 1,470 North American Negroes. The common MDH phenotype exhibits one main band migrating towards the anode, with two fainter bands moving slightly faster in the same direction. All the Whites and all but one of the Negroes showed the common phenotype, but one Negro exhibited a variant pattern, 2 - 1, consisting of three bands, the fastest of which had the same mobility as the single major normal band. From a family study of the propositus, they were able to show that the variant and the normal types behave as controlled by a pair of co - dominant alleles at an autosomal locus.

Blake et al (1970) described a fast variant, termed 3 - 1, found in a number of persons from New Guinea. This too exhibited a three band pattern, but with the slowest band corresponding to the normal type.

Method

Tank Buffer

A 0.2 M Mono potassium phosphate (KH_2PO_4) 27.22g/ltr.

B 0.2 M Disodium hydrogen phosphate ($Na_2PO_4 2H_2O$) 28.44g/ltr.

These are mixed in proportions:-

508 A

492 B

Gel Buffer

The tank buffer is diluted 1 : 20 with distilled water.

Haemolysate samples are applied on Whatman No. 3 filter paper inserts and the gels are run horizontally at 12 volts / cm. for 3 . 5 - 4 hours at + 4°C.

The mixture described below is added to the cut surface of the gel and incubated at + 37°C for about 45 minutes.

Incubation Buffer

To 100ml. of 0 . 1 M Tris / HCl buffer, pH 8 . 0 add:-

NAD	0 . 01 g.
PMS	0 . 01 g.
MTT	0 . 01 g.
L - malic acid	0 . 1 g.

Statistical Methods

Gene frequencies

Since genotypes could be deduced directly from the results of tests in the case of some of the blood groups, the serum proteins Hp and Tf, and the red cell enzyme systems, gene frequencies were determined by direct gene counting. However, in the case of the ABO, MNSs and Rh blood groups the methods used to calculate the respective gene frequencies are indicated. Where only one antiserum was used in testing for a particular blood group system, e.g. Lutheran system, the gene frequencies were calculated in the following manner. The frequency of Lu^b was obtained by square rooting the frequency of Lu(a -) in the sample. The frequency of Lu^a is therefore found to be 1- frequency of Lu^b. In the Manx sample this works as follows:-

$$Lu^b = \sqrt{0.8886} = 0.9427$$

$$Lu^a = 1 - 0.9427 = 0.0573$$

Internal homogeneity of the samples.

When indicated the phenotype frequencies were tested for Hardy - Weinberg equilibrium and internal goodness of fit by calculating the chi - squared value (X^2), using the equation given below:-

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

In certain cases, where a rare phenotype was found with a very low incidence, the number found was added to that of the next lowest phenotype in order of rarity for the purpose of calculating X^2.

Only X^2 values that exhibited a probability of 5% or less

were classified as statistically significant and the actual X^2 values are given in the text. However, all insignificant X^2 values, i.e. those greater than the 5 % level of probability are not given in the text, only being reported as statistically non - significant.

Contingency Tables

Whenever it was informative to test pairs of population samples for possible relationships, this was done by the standard formulae for 2 x 2, 3 x 2, 4 x 2 etc. contingency tables given below. In most of the plasma proteins and red cell enzyme systems each phenotype included only one genotype, and comparisons could be made in terms of numbers of genes as well as phenotypes.

2 x 2 Contingency Table

Gene	Population 1	Population 2	Total
p	a	b	a + b
Q	c	d	c + d
Total	a + c	b + d	a+ b+ c+ d

The standard formulae for this is :-

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

In the case of the enzyme AP three genes are present at levels in excess of that which would be maintained by mutation alone. In populations which possess all three genes a X^2 value for possible relationship can be obtained using a 2 x 3 contingency table by the method shown below.

2 x 3 Contingency Table. e.g. AP

Gene	Population 1	Population 2	Total
p^a	A^1	A^2	$A^1 + A^2$
pb	B^1	B^2	$B^1 + B^2$
pc	C^1	C^2	$C^1 + C^2$
	$A^1 + B^1 + C^1$	$A^2 + B^2 + C^2$	$A^1 + A^2 + B^1 + B^2 + C^1 + C^2 = N$

Where A^1, A^2, B^1, B^2, C^1 and C^2 are the number of genes of each type in the population and N is the total number of genes. Expected values are then obtained for each of the genes as follows:-

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

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

$$X^2 = \frac{(\text{obs } A^1 - \text{exp } A^1)^2}{\text{exp } A^1}$$

The values of X^2 given by the six cells are then summed to give a total X^2 which has two degrees of freedom.

In other cases such as 6-PGD and PGM enzyme systems, any of the rare alleles can be added to the next nearest allele with confidence, owing to the fact that they have not been found in any populations in very high frequency e.g. PGM 7 - 1, and then a 2 x 2 contingency table is adequate.

However, in the case of some of the blood groups e.g. ABO system, genotypes were not directly obtainable from the phenotypes and statistical comparisons had to be done in terms of phenotype numbers only. Whenever the expected value in any cell was found to be less than 5, the number of that phenotype was added to the phenotype in the same system displaying the next lowest number.

e.g. in the ABO system B and AB were added together and in the AP system CA, CB and C were united. The construction of multiple contingency tables was not performed as this is extremely laborious and would probably only yield information of limited value.

Only those X^2 values with a level of probability of 5 % or less were classified as statistically significant and were included in the text. All other X^2 values were reported as statistically non - significant.

CHAPTER THREE

ANALYSIS OF DATA COLLECTED IN THE PRESENT SURVEY

ANALYSIS OF DATA COLLECTED IN THE PRESENT SURVEY

The primary aim of the present survey was an investigation of genetic variation in the native Manx population, and a comparison of gene and phenotype frequencies with other regional population samples, indigenous if available, surrounding the Irish Sea basin in particular, and with other British and Irish populations more generally. The analysis of the results obtained for the genetic polymorphisms examined in the Manx population sample as well as the series collected in Cumbria and South - West Scotland follows;-

I. Serological Variability.

a. Blood Group Antigens. (Tables 1 - 17)

1. ABO blood group system. (Tables 1 to 7)

a. Isle of Man.

Table I shows the distribution of the ABO blood groups in the various samples collected in the Isle of Man. Two levels of discrimination are shown, depending upon whether anti -A_1 serum was employed in testing the samples. The first five samples shown in Table I comprise individuals, all of whom have three or four grandparents and both parents born on the Isle of Man. No sex difference of significance was found in any of the Manx samples. There was also good agreement between the observed and expected phenotype frequencies in all samples, thus confirming the assumption of Hardy - Weinberg equilibrium. The gene frequencies for the ABO system were calculated using the methods described in Mourant (1954).

Phenotype A_2 is most frequent in Europe where the gene (p^2) frequency ranges around 10% and about 25% of all gene p. (Harrison et al 1964). In the Manx samples the gene frequency rises from

0.0542 in the blood donors to a maximum of 0.0832 in the non - blood donors, with a frequency of 0.0723 in the total series.

Table I also reveals the relatively low frequency of gene B(q) found in the Manx population and also its small range of variation in the samples. The frequency of q rises from 0.0396 in blood donors to a high of 0.0621 in the native women attending the ante - natal clinic, and has a frequency of 0.0490 in the total Manx series.

The most striking feature of Table I is the considerable variation in the frequencies of phenotypes O and A and genes p and r respectively. These differences are reaffirmed by an inspection of the A : A + O indices of the various samples. The reciprocal relationship between groups O and A in the British population has been reported upon. (Fisher and Taylor 1940, Mourant 1954, Mourant et al 1958) The frequency of O rises from 0.4320 in schoolchildren to a maximum of 0.5434 in blood donors, with a value of 0.4668 in the total sample; while the respective gene, r, rises from 0.6561 in schoolchildren to a maximum of 0.7446 in donors, with a frequency of 0.6832 in the total series. Phenotype A shows similar variation, rising from 0.3607 to 0.4704, with a frequency of 0.4326 in the total sample. The frequency of gene p ranges from 0.2158 in the blood donors to 0.2921 in schoolchildren, with a frequency of 0.2660 in the total Manx series. The A : A + O indices show a variation of 12%, rising from 39.9 to a maximum of 52.1.

The blood donor sample was significantly different from the schoolchildren series, $X_2^2 = 7.2209$ $.05 < P > .025$. However, the former series was not significantly different from the smaller non - donor series, or the even smaller sample of women attending the ante -

natal clinic. The blood donors resemble most closely the sample of non - native women attending the ante - natal clinic with respect to ABO blood groups. It is clear from Table 1 that the blood donors are at one extreme of the range of the ABO blood group distribution, and the native samples appear to fall into two groups; blood donors and non - blood donors; the latter comprising samples 2, 3 and 4 of Table 1. (Table 2)

The frequency of gene p^2 is fairly similar in both final Manx native samples, 0.0542 and 0.0812 respectively, and the proportion of p^2 : p is exactly 1:4 in the donors and slightly greater in the non - donor series. Similarly, phenotypes B + AB exhibit little variation between the two series, with frequencies of 0.0959 and 0.1051 respectively.

However, there are considerable differences in the frequency of genes p and r between the two series. The gene p shows a range of nearly 7%, from 0.2158 in the donors, to 0.2856 in the non - donors, while gene r exhibits an even greater variability, over 8%, from 0.6590 in non - donors to 0.7446 in donors.

After statistical analysis the native donors and native non - donors were found to be significantly different, $X_2^2 = 7.6949$ $.025 < P > .01$, the relatively large X^2 value being produced by the discrepancy in the proportion of groups O and A in the two series. How can this difference be explained when the selection procedure for all individuals included in Table 2 was identical; that is all individuals have three or four grandparents and both parents born on the Isle of Man? Any suggested explanations to account for this difference may be important, because many population surveys of genetic variability in the United Kingdom have employed Blood Transfusion Service data for blood group frequencies, the

authors assuming the donors to be representative of the population being investigated. It was mentioned in chapter 2, that certain authors (Dawson 1964, Kopeć 1970) felt that their donors constituted random samples because they included only those who did not know their blood groups and who were giving blood for the first time. It could be important that the Manx donor sample was obtained by asking ALL donors for their participation in the survey and not only new donors.

In connection with the above, and also another possible reason for the observed heterogeneity between the samples in Table 2, it may be that the Blood Transfusion Service (B.T.S.) on the Isle of Man recalls donors to bleeding sessions on a selective basis and this could explain the preponderance of O and Rh (D) negative persons in this series. The Director of the B.T.S. on the Island since its inception, Dr. C. G. Pantin, stated that "during the time that the blood donor service was being established the families of those found to be group O and Rhesus (D) negative had been encouraged to join the donor service" (Pantin 1950). This selection of O,Rh (D) negatives helps explain the difference between the samples, but as the necessity for O,Rh(D) negative blood type is removed, one would expect the frequency of group O to fall in the blood donor panel. It is the author's intention to investigate this phenomenon in the total Manx blood donor panel in the future.

Another possible reason for the observed difference between the two native series may be that because those persons requiring blood and blood products are drawn randomly from the resident Manx population, the frequency of the ABO groups in the native donors reflects the ABO group distribution in the total resident population of the Island (i.e. those with and without Manx ancestry)

(Mitchell 1973). It would be very worthwhile to follow up this study with a similar one of the non - indigenous population of the Island. The only data on the non - indigenous population available are the relatively small sample of women attending the ante - natal clinic in Douglas who did not qualify for the indigenous series (sample 6 in Table 1). On inspection they are found to show fairly similar ABO group frequencies to the native donors, especially as regards group A. If these conditions were found with a larger sample of non - natives or the general resident population, they would strongly support the above hypothesis.

Yet another possible explanation for the difference between the donor and non - donor series is found in Mourant's foreword to Kopeć's 'The Distribution of the Blood Groups in the United Kingdom' (1970). Though the Isle of Man was one of the few areas not covered by Kopeć's research, Mourant points out that "even in proportion to their lower total number, rural populations are likely to be less well represented among donors than the inhabitants of towns. It may happen that some important rural groups have been missed or represented so sparingly as to be swamped statistically by their urban neighbours" (Kopeć 1970). The majority of bleeding sessions on the Isle of Man are held in Douglas, the only truly urban centre. In fact in winter months sessions are confined to Douglas. It will be of interest to see if the frequencies of ABO phenotypes in the Douglas sample are different from other areas of the Island when regional comparisons are effected. In contrast to the donors, the schoolchildren sample in particular, but also the samples of adult non - donors and women attending the ante - natal clinic, had the whole Island

as their catchment area. In fact as mentioned earlier, the school-children series comprise almost the total native Manx population between the ages of eleven and sixteen years.

The problem to be solved is which of the two 'representative' samples in Table 2 illustrates the 'correct' distribution of ABO groups in the indigenous Manx population. To answer this question a full investigation of the ABO blood group distribution in the total Manx population, immigrants as well as indigenes, is ultimately required. However, evidence supporting the view that the native Manx population is characterised by a higher frequency of the p gene than the non - indigenous residents, comes from the unpublished results of an earlier survey carried out by Pantin (1950), shown in Table 4.

Pantin's total sample consists of the blood groups of 2,056 residents on the Isle of Man, determined routinely in the Clegg Laboratory, Noble's Hospital, from May 1946 to the end of 1950. During this time the blood donor panel was being built up, and the families of those found to be group O and Rh(D) negative had been encouraged to join the donor service. To overcome this bias only those who appeared to be unrelated were selected, this number being 1,467. To this pool were added the ABO groups of 33 nurses, thus bringing the Unrelated Sample to 1,500. Of these 1,500 persons, 545 had names of typically Manx origin, but since two thirds (361) of this group were female, many of them married and therefore possibly not native to the Island, the ABO groups of 200 men and unmarried women are listed separately (Table 4).

There is good agreement between the observed and expected frequencies of the ABO phenotypes in the samples, thus confirming the assumption of a Hardy - Weinberg equilibrium and that it is

a random mating population. No significant differences were found among the four samples included in Table 4. However, it is interesting to note that as the samples were selected for 'Manxness', the frequency of gene r falls and that of the p gene rises;

r falls from 0.6919 to 0.6673

p rises from 0.2579 to 0.2742

and the A:A+O Index rose from 46.9 to 49.6.

Again, the distribution of the ABO blood groups in Pantin's native series (samples 3 and 4 in Table 4) corresponds more closely to that found in the non - donor than the blood donor series of the present survey (Table 2). The largest difference found was that between Pantin's Manx Names Sample and the blood donors, $X_2^2 = 5.4765$ $.10 < P > .05$, a value not quite statistically significant.

Pantin's total sample and total Unrelated sample (1 and 2 in Table 4) exhibit a similar distribution of the ABO groups to that found in the sample of all women attending the ante - natal clinic shown in Table 1. This could be expected as selection of individuals for both samples was based on residence in the Isle of Man only. In the author's view, it is most probable that Pantin's total Unrelated sample represents the true ABO blood group distribution in the resident Manx population.

However, for the indigenous Manx population there are three possible 'representative' samples,

1. Blood Donors
2. Non - Blood Donors

which combined produce

3. Total Native Series

Each of these three samples was assumed to be representative when comparisons with other population samples for ABO blood group

distributions were performed.

Table 4 also provides further evidence for the view expressed in Chapter 2, that only a small proportion of the present Manx population is indigenous as defined in the present survey. Only between 200 and 545 persons possessed Manx names or Manx maiden names out of a total of 1,500 unrelated individuals. This is equivalent to between 13.3% and 36.3% of the total, and this reduction occurs without any of the detailed investigation of birthplaces of the family employed in this survey, which presumably would have diminished the number further. In addition Pantin's survey was carried out in the late 1940's when there was far less mobility of the population than there is today.

Another possible distinction between the Manx series collected in the present survey is based on age; the schoolchildren versus the adult series. (Table 3) No significant difference was discerned between these two groupings with respect to ABO groups.

b) Cumbria

Table 5 shows the ABO blood group distributions in the Cumbrian blood donor and schoolchildren series, together with the respective gene frequencies. Again two levels of discrimination are shown, depending upon whether the specimens were tested with anti - A_1 serum. No sex differences were found in any sample. There was also good agreement between the observed and expected phenotype frequencies, thus confirming the assumption of Hardy - Weinberg equilibrium, and that it is a random mating population.

The two samples of native Cumbrians exhibited overall similarity with respect to ABO blood groups, and were pooled to give a total sample of 539. Unlike the Manx, no significant differences were found between the Cumbrian donor and non - donor groups and also no age differences were observed.

The allele p^2 exhibits very little variation, having a frequency of 0.0869 in the total sample; a figure higher than that found in the Manx. Also the proportion p^2:p is much greater than 1:4 in both Cumbrian series. The q gene also exhibits a strikingly similar value in both series, with a frequency of 0.0640 in the total, again more than 1% higher than in the Manx population. This similarity between the Cumbrian samples is also marked with the p and r allele frequencies. The total Cumbrian sample shows a similar frequency of p, 0.2790, to that found in the Manx, while it exhibits a slightly lower frequency of r, 0.6570.

The published studies on the ABO blood group distributions in the population of Cumbria are shown in Table 6. The present sample of native Cumbrians was found to be very different from Fraser Roberts' (1953) total sample, $X^2_2 = 15.0657$ $.001 < P > .0001$. However, Fraser Roberts selected the blood donors for his sample

solely on the criterion of residence in Cumbria. His sample was sub - divided into two units, North and West Cumbria and South Cumbria, on the basis of their distinctive ABO blood group distributions. The present sample was found to be even more different from the North and West Cumbrians, $X^2_2 = 20.1519$ $.001 < P > .0001$, due chiefly to the high frequency of A in the former; but similar overall to the South Cumbria series.

The issue was further confused when it was discovered that the family history of the majority of individuals comprising the present Cumbrian sample lies in North and West Cumbria. Why should there be this difference between the present sample and Fraser - Roberts' North and West Cumbria series? It could be a product of the relatively small size of the present series. However, an alternative explanation is that the native population is distinctive from the non - native residents of Cumbria with respect to ABO blood groups.

Interestingly, Fraser Roberts' sub - samples exhibit very similar frequencies of groups B and AB, demonstrating that the heterogeneity observed between them is a product of variation in groups O and A, just as the present series does not differ from Fraser Roberts' samples in the proportion of B and AB; all the heterogeneity being accounted for by variation in the O and A groups.

Kopeč's (1970) region of Cumbria was defined by the present author as comprising her unit - areas 59 - 72 inclusive, Map Vl, Newcastle - upon - Tyne B.T.S. Centre and unit - area 1, Map Vll, Liverpool B.T.S. Centre (Table 6). This resident sample was also found to be significantly different from the present indigenous series, $X^2_2 = 7.8512$ $.025 < P > .01$.

Once again there was a marked similarity between the two samples in the frequency of groups B and AB, illustrating that the heterogeneity was a product of the variations in the A and O phenotypes.

The present Cumbrian data were also significantly different from the ABO distribution shown for Kopeć's (1970) Final Region 3 (Map 1, p. 87) which comprises the west and central part of the extreme of northern England, $X_2^2 = 7.0719$ $.05 < P > .025$. (Table 6).

c) South West Scotland

Table 5 shows the ABO blood group distribution in the series collected in South West Scotland, comprising native blood donors, resident blood donors and the two combined. Results of testing with and without anti - A_1 serum are given. No sex differences were found in either sample, and there was also close agreement between the observed and expected phenotype values.

The frequency of allele p^2 rises from 0.0513 in the residents to more than double in the natives, 0.1033; and the proportion p^2:p in the natives is in excess of 1:3 whereas in the residents it is less than 1:4. However, the total sample exhibits a frequency of 0.0740, similar to that found in the Manx and Cumbrian series. Alleles p and r show a higher frequency in the native sample, 0.2849 and 0.7051 respectively, than in the residents, 0.2315 and 0.6523.

After statistical analysis the two Scottish series were found to differ significantly from each other, $X_2^2 = 9.1644$ $.025 < P > .01$, the variation in B and AB phenotypes contributing chiefly to the X^2 value. The frequency of allele q exhibits the greatest variability of all the genes, rising from 0.0100 in the natives to 0.1163 in the residents. It is most probable that the exceptionally low frequency of q in the natives is a product of the very small sample size, because in no British population reported is there such a low frequency. Therefore, the author felt justified in combining the two samples for comparative purposes, in spite of statistical conclusions. However, it should be considered that the allele q may be less prevalent among the long established indigenous population, and that its present high frequency in the residents is a result of immigration. Another

possible explanation could be that there exist local pockets of high B gene frequencies. This view gains some support from the fact that 22% of donors attending the New Cumnock bleeding session were group B.

Kopeć (1970) reported the ABO blood group distribution in resident blood donors of South West Scotland, which comprises unit areas 52, 53, 54, 56 and 57 of Map IV, Glasgow and West of Scotland B.T.S. Centre. The present resident series as expected, and the total Scottish sample, exhibited overall similarity to Kopeć's data. However, the small native series was significantly different from Kopeć's series, $X^2_2 = 7.4811$ $.025 < P > .01$, with the variation in group B making the greatest contribution to the X^2 value.

d) Inter - Regional Comparisons (Table 7)

Comparisons between the data collected during the present survey for each genetic factor are presented in this section, rather than later, for two reasons. Firstly, all the samples collected, with the exception of one, comprise individuals indigenous to the area specified. This rigorous selection of samples is rarely fulfilled by British or Irish population data previously reported. Therefore, the author felt it appropriate to compare the Manx data with similarly selected material, distinct from the remainder of the comparative data.

Secondly, the author preferred to analyse the results of his own survey as a unit, before comparing any findings with other available information on British populations.

As mentioned above, three samples were found as possible representatives of the indigenous Manx population, and accordingly each was employed in statistical comparisons. When the total Manx sample was compared with the mainland samples shown in Table 7, it was found that it was significantly different from only one, the total South West Scottish series, $X_2^2 = 8.8301$ $.025 < P > .01$. It is the high incidence of phenotypes B and AB in the Scots (nearly 17%) compared with the Manx (10%) that accounts for this heterogeneity. As the Scottish series comprises in large part persons selected solely on their residence within the area, this finding is perhaps not surprising.

The Manx non - donors, like the total Manx, were also found to differ significantly from the total South West Scottish series; $X_2^2 = 8.3836$ $.025 < P > .01$.

However, the Manx donors exhibited significant differences, many highly significant, from all the mainland series shown in Table 7, with the exception of the native South - West Scots. All the other X^2 values were significant at least at the 5% level of probability.

Manx donors v Cumbrians $X_2^2 = 7.2085$ $.05 < P > .025$

Manx donors v native S.W. Scots Non - significant

Manx donors v Total S.W. Scots $X_2^2 = 7.6339$ $.025 < P > .01$

Whereas the heterogeneity between the Manx and Cumbrian samples is a product of the differing proportions of groups O and A, that found between the Manx and the Scots is largely a product of the different proportions of phenotypes B and O in each. In accordance with this heterogeneity, the A:A + O indices of the samples shown in Table 7 also exhibit considerable variation, ranging from 39.9 in the Manx donors, to 50.7 in Cumbrians, and a maximum of 51.3 in Manx non - donors. When it is seen that the closest A:A + O Index to that found in the Manx donors is 46.7 in the total South West Scottish sample, it is further evidence that the Manx donors display an aberrant distribution of ABO groups compared with the distribution found in the other samples.

Secretor Status (Table 8)

Table 8 shows the distribution of secretors and non - secretors in native Manx and Cumbrian population samples. The Manx sample consists of adults, mostly blood donors, whereas the Cumbrian series comprises schoolchildren. The two populations exhibit a striking similarity with respect to secretor types with a frequency of 29% non - secretors.

ii) MNSs blood group system Tables 9 - 11

(a) Isle of Man

MN blood groups

Tables 9a and 9b present the distribution of MN blood groups and respective gene frequencies in the native Manx population samples. The gene frequencies were obtained by applying the formulae first given by Wiener and Vaisberg (1931) which amounts merely to a direct counting of the two genes. Fairly good agreement was found between the observed and expected phenotype values in all samples, thus confirming the assumption of Hardy - Weinberg equilibrium. In all the Manx samples there was an excess of phenotype MN, but this was especially marked in the schoolchildren series.

No statistically significant heterogeneity was observed between any of the samples shown in Tables 9a and 9b, including the donor and total non - donor samples, and schoolchildren and the total adult samples, with respect to either phenotypes or genes. This homogeneity of MN groups is reflected in the M gene frequency, which exhibits variability of just over 1% in the three Manx series.

An interesting though unexplained finding was that while there was overall similarity between males and females, there was significant heterogeneity between the sexes when phenotype M was compared with the other phenotypes, $X_1^2 = 4.4382$ $.05 < P > .025$, and when phenotype MN was compared with the other groups, $X_1^2 = 4.3268$ $.05 < P > .025$. (Table 10). The existence of a sex difference in the MN blood groups has not been reported previously and the author is unable to suggest an explanation of this finding in the native

Manx population. However, no significant difference was found in the distribution of the two genes between the samples. Whereas very close agreement was found between the observed and expected phenotype values in the females, significant heterogeneity was discerned in the males, $X^2_2 = 9.0546$ $.025 < P > .01$, due largely to the excess of MN in the sample. (Table 10)

MNSs blood groups

Table 9a exhibits the distribution of the MNSs phenotypes and respective gene frequencies in the donor and adult non - donor series as a result of testing with four antisera. Agreement was found between the observed and expected phenotype values in both series included in Table 9a. All sex differences were statistically insignificant. The gene frequencies were calculated according to the method described in Mourant (1954).

Table 9b presents the distribution of MNSs phenotypes in the schoolchildren series and the larger total Manx series, after testing with three antisera only - anti - M, anti - N and anti - S. Both series exhibited highly significant sample heterogeneity with chi - squared values of $X^2_5 = 16.3968$ $.01 < P > .005$ in the schoolchildren and $X^2_5 = 20.7115$ $P < .001$ in the total Manx series. In both samples there was a deficit of MMS and NNS and an excess of MNS groups. All sex differences were statistically non - significant. The gene frequencies were calculated according to the method described in Mourant (1954).

No significant heterogeneity was found among the samples shown in Tables 9a and 9b, including donor and total non - donor samples, and schoolchildren and total adult samples. Accordingly, the native Manx population can be regarded as a homogeneous group

with respect to MNSs groups.

In the samples shown in Table 9a the gene complex MS rises from a frequency of 0.2320 in the donors to 0.2687 in the non - donors, with an incidence of 0.2457 in the total Manx series. Ms has its lowest frequency, 0.2866, in the non - donors rising to 0.2932 in the donors, with an incidence of 0.2840 in the total sample. The NS and Ns genes exhibit the least variability of all, with frequencies of 0.0447 and 0.4256 respectively in the total sample.

Regarding the Manx samples shown in Table 9b, the gene MS rises in incidence from 0.1609 in the schoolchildren to 0.1930 in the total Manx series. Otherwise the three gene complexes in the schoolchildren series exhibit strikingly similar frequencies to those found in the total sample.

b) Cumbria

MN blood groups

Table 11c presents the distribution of MN blood groups and respective gene frequencies in native Cumbrian population samples. All samples exhibited fairly good agreement between observed and expected phenotype values. Whereas the donors exhibited a slight excess of MN the schoolchildren displayed a deficit of this phenotype. Unlike the Manx no significant sex differences were found in any sample.

As no statistically significant heterogeneity could be demonstrated between the two Cumbrian samples with respect to MN phenotypes and genes, they were pooled into one sample exhibiting a frequency of 0.5398 for the M gene.

MNSs blood groups

Table 11a exhibits the frequency of MNSs blood groups and respective gene frequencies in the Cumbrian population samples, expressed in terms of nine phenotypes, after testing with four antisera. Good agreement was found between the observed and expected phenotype values in the Cumbrian donor and total Cumbrian samples, so confirming the assumption of Hardy - Weinberg equilibrium. The sample of schoolchildren was too small in number to permit investigation of sample homogeneity. All sex differences were statistically insignificant.

Table 11c demonstrates the frequency of MNSs blood groups in the same, but slightly larger, samples tested with three antisera only, anti - M, anti - N and anti - S. Internal homogeneity was found in all samples, even though the schoolchildren series

exhibited a deficit of MNss and an excess of MMss and NNss. All sex differences were insignificant.

No significant heterogeneity was discerned between the two Cumbrian series shown in either Table 11a or Table 11c. Therefore the samples can be amalgamated into one relatively large representative Cumbrian sample. The gene complexes in the samples included in Table 11a show remarkable similar frequencies, with MS displaying greatest variability, (as found in the Manx) rising from 0.2393 to 0.2604. The other three genes show a variability of less than 1% between the two samples. The samples in Table 11c also show very similar frequencies for the four gene complexes of the MNSs system.

c) South West Scotland

MN blood groups

Table 11b shows the distribution of the MN blood groups and respective calculated gene frequencies in South West Scottish population samples. Internal homogeneity was found in both samples and the slight sex differences were statistically insignificant. As the Scottish samples exhibited an overall similar distribution of MN phenotypes and genes, they were pooled into one larger sample with a frequency of 0.5582 for the M gene.

MNSs blood groups

Table 11b also exhibits the frequency of MNSs groups and respective gene complex frequencies in the Scottish samples tested with four antisera. Internal homogeneity was found in both samples and all sex differences were insignificant. The two samples exhibited a similar overall distribution of MNSs phenotypes, so they were amalgamated into one sample of 172 persons.

d) Inter - Regional Comparisons

MN blood groups

Statistical comparisons of the Manx sample with those collected from Cumbria and South West Scotland revealed no significant heterogeneity with respect to phenotypes and genes of the MN system. The indigenous populations of the three areas constitute a homogeneous group exhibiting a frequency of 0.53 - 0.54 for the M gene.

MNSs blood groups

When the distributions of the MNSs blood groups in the three regional population samples were compared with each other, by means of 6 x 2 contingency tables, the Manx were found to differ significantly from the Cumbrians, $X^2_5 = 14.6219$ $.025 < P > .01$, due chiefly to the differing proportions of MNS, MNss and NNss groups. However, the Manx exhibited a similar overall distribution to that found in the Southern Scots. The Cumbrians also display a similarity in the distribution of MNSs phenotypes to the Scots. In the only 8 x 2 contingency table (NNSS and NNss added together) used, for comparing the Manx and Cumbrian populations tested with four antisera, the Pvalue was found to be statistically insignificant.

The gene frequencies shown in Tables 9b and 11c reflect the differences reported in MNSs phenotypes between the Cumbrian and Manx populations. Gene complex MS exhibits a low frequency in the Manx, 0.1930, rising to 0.2370 in Cumbrians, while the other three gene complexes exhibit a lower frequency in Cumbrians than in the Manx.

iii. P blood group system (Table 12)

Table 12 shows the distribution of the P blood groups in the native Manx, Cumbrian and South West Scottish series, together with the calculated gene frequencies after testing with anti - P_1 serum. No sex differences were observed in any sample. Some of the Manx specimens were tested in two laboratories, each with different anti - P_1 serum, yet the series show a very similar frequency of P groups. However, there is a significant difference between the Manx donors and non - donors tested with the same S.P.G.L. antiserum, $X_1^2 = 6.9502$ $.01 < P > .005$. The only explanation of this difference that the author can suggest is that as the majority of donors reside in Douglas, this observation could signify an urban - rural difference in the native Manx population.

The minute samples for Cumbria and South West Scotland were pooled and compared with the two Manx series, but no differences were observed.. If the two Manx series tested at the S.P.G.L. are combined, despite the statistical evidence, the frequency of allele P_1 is found to be 0.4796, which is very similar to that found in the Manx (0.4622) and mainland samples (0.4641) tested in Durham. It would appear that the indigenous populations of the Isle of Man, Cumbria and South West Scotland are homogeneous with respect to common P blood groups.

iv Rh blood group system (Tables 13 and 14)

a) Isle of Man

Table 13a presents the distribution of Rh types and gene complex frequencies in the indigenous Manx population samples. The specimens were tested with some or all of the following antisera;- anti - D, -C, -E, -c, - e and - C^w. No significant sex differences were observed in any sample. The gene complex frequencies were calculated according to the methods described in Mourant (1954).

To enable sample comparisons to be performed various Rh types were combined to provide sufficiently large numbers in each category. The following groupings of Rh types were employed in 6 x 2 contingency tables.

1. R_1r, $R_1{}^ur$, $R_1{}^wr$
2. R_1R_1, $R_1{}^uR_1$, $R_1{}^wR_1$
3. R_1R_2, $R_1{}^uR_2$, $R_1{}^wR_2$
4. R_2r, $R_2{}^ur$
5. rr
6. R_2R_2, R_0r, R_1R_z, rr', rr ''

No statistically significant heterogeneity could be demonstrated between the donor and adult non - donor series, the donors and schoolchildren, or between the donor and total non - donor samples. However, significant heterogeneity exists between the adult non - donors and schoolchildren, $X_5^2 = 16.1500$ $.01 < P > .005$ and also between the total adult series and the schoolchildren, $X_5^2 = 12.7015$ $.05 < P > .025$. It is the higher frequency of Rh types R_1r and R_2R_2 and a lower incidence of R_1R_2, and rr in the schoolchildren that contributes to the observed heterogeneity. One explanation

of this finding may be the possible higher number of related persons in the schoolchildren series compared to the other samples.

Table 13b shows the distribution of Rh (D) negative persons in the Manx population samples, including Pantin's (1950) data. Pantin's sample referred to in the section on ABO blood groups was analysed for Rh (D) groups "when all women who sought blood grouping because of pregnancy or the immediate effects thereof, such as abortion, were excluded. Also, only those with typically Manx names were included."

No heterogeneity was demonstrated among any of the samples collected in the present survey and so they could be pooled into one relatively large sample of 803 persons, exhibiting a frequency of 0.1980, Rh(D) negative. The higher frequency of Rh(D) negatives found in the donors, 2% higher than in schoolchildren, is the

finding one would have expected if there was, or still is, selection for O, Rh negative blood donors. Variation that was just statistically significant was found between the donors and Pantin's (1950) series, $X_1^2 = 3.9588$ $.05 < P > .025$, but the latter was similar to the present total Manx sample in the proportion of Rh negative individuals.

b) Cumbria

Table 14a presents the distribution of Rh types and calculated gene complex frequencies in the two samples of the indigenous Cumbrian population. The same Rh type groupings were employed in statistical computations as used in the analysis of the Manx results. No sex differences of significance were found in either sample.

Both Cumbrian samples exhibited an overall similar distribution of the Rh types, and therefore the two were combined into one large sample. This similarity was reflected in the distribution of gene complexes, the largest variability being only just over 4% in the frequency of R_2 . Both the R_1 and r genes exhibited a range of less than 1% between samples.

Table 14b shows the distribution of Rh(D) negative individuals in the Cumbrian population. The two samples exhibit a strikingly similar frequency of Rh(D) negatives, and so again the samples were combined to produce a sample with a frequency of 0.1951 Rh(D) negative. Whereas the Manx donors displayed a higher frequency of Rh negatives than non - donor samples, this does not occur with the respective Cumbrian series.

c) South West Scotland

Table 14a also illustrates the observed number and frequency of Rh types and calculated gene complex frequencies found in the two Scottish population samples. No sex differences of significance were found. The two samples were found to exhibit overall homogeneity in the distribution of Rh types and accordingly were pooled into one large sample. The similarity of the samples was reflected in the gene complex frequency distributions, and, as in Cumbria, the greatest variability, just under 5%, was found in the frequency of the R_2 gene.

Table 14b includes data on the distribution of Rh(D) negative persons in the South West Scottish samples. Again, the similar distribution of Rh types permitted the amalgamation of the two samples into one with a frequency of 0.2459 Rh(D) negative.

d) Inter - Regional Comparisons

Though some Manx samples shown in Table 13a exhibit significant heterogeneity with respect to the proportion of Rh types, the total Manx sample was the one employed in statistical comparisons with Rh data from Cumbrian and Scottish populations. The Manx exhibited significant heterogeneity from the Cumbrians, $X_5^2 = 11.8171$ $.05 < P > .025$, and the South West Scots, $X_5^2 = 11.9965$ $.05 < P > .025$, with respect to Rh types. The greatest variation between the Manx and Cumbrians lies in the proportions of Rh types R_1r, R_1R_2, R_2r and R_1R_1. However, it is the differing proportions of R_1r in particular, rr and R_2r that account for the differences observed between the Manx and the Scots. The Cumbrians and South West Scots also just differ significantly from each other with respect to Rh types, $X_5^2 = 11.8957$ $.05 < P > .025$ The greatest difference between these two populations was in the proportions of Rh types R_1r, rr and R_1R_2.

The frequency distribution of the Rh gene complexes mirror some of this variability among the three samples. The gene complex R_1 rises from just under 0.37 in the Manx and Scots to 0.41 in the Cumbrians, whereas the R_2 gene rises from 0.1140 in the Cumbrians to 0.1446 in the South West Scots, and a maximum of 0.1617 in the Manx. The frequency of the r gene shows very little variability among the three population samples.

Regarding the distribution of Rh(D) negatives, the indigenous populations of the three regions exhibited overall homogeneity. The frequency of Rh(D) negative individuals ranges from 0.15 in Pantin's Manx series, through 0.20 in the present Cumbrian and Manx samples to a maximum of 0.26 in the small Scottish series.

v. Lutheran blood group system (Table 15)

Table 15 shows the distribution of Lutheran blood groups in indigenous Manx, Cumbrian and South West Scottish population samples tested with anti - Lu^a serum. No sex differences were observed. The two Manx samples being similar were pooled to produce a relatively large series having a frequency of Lu(a+) of over 11%. The Manx sample was similar to the Cumbrian and South West Scottish series with respect to common Lutheran groups. Therefore, as with the P blood groups, the indigenous populations of these areas in the North Irish Sea basin are homogeneous.

The frequency of allele Lu^a rises from 0.0392 in South West Scotland, through 0.0435 in Cumbria to a maximum of 0.0573 in the Isle of Man. This latter value is one of the highest frequencies for Lu^a yet reported in a British Isles population. However, Cartwright (1973a) reported a frequency of 0.067 in the population of Holy Island, Northumberland.

vi. Kell blood group system (Table 16)

Table 16a presents the distribution of Kell groups and respective gene frequencies in the Manx, Cumbrian and South West Scottish native population samples. Internal homogeneity was found in all samples and there were no significant sex differences.

Even though the Manx non - donors exhibited a higher frequency of K+ individuals than the donors, the difference was not statistically significant, so the two samples were pooled into one relatively large sample. The Manx series exhibited a similar distribution of Kell groups to that found in both the Cumbrian and Scottish samples. This overall similarity is reflected in the small range in frequency of the K allele, which only rises from 0.0417 in South West Scotland to a maximum of 0.0490 in Cumbria.

Table 16b shows the number and frequency of Kp(a+) individuals in the Manx and South West Scottish samples, together with the calculated gene frequencies. The number of Kp(a+) persons in all samples is too small to permit worthwhile comparisons, but it should be noticed that the frequency of Kp(a+) in the Manx, just under 4%, is the highest yet reported for Caucasoid populations in which the frequency is usually nearer 1%.

vii. Duffy blood group system (Table 17)

Table 17a shows the distribution of Duffy blood groups and respective calculated gene frequencies in two Manx series tested with anti - Fy^a and anti - Fy^b sera. There was good agreement between the observed and expected phenotype values, thus confirming the assumption of Hardy - Weinberg equilibrium. Moreover the sex differences were insignificant.

The gene frequencies shown in Table 17a have been calculated on the assumption that the allele Fy does not exist in the indigenous Manx population. Race and Sanger (1970) pointed out that heterozygotes of an Fy(a - b -) condition must be present in Whites, but there is no satisfactory way of measuring its frequency in a Caucasoid population. Various workers have expressed different ideas on how much of a percentage to allow for gene Fy, ranging from 0.02 by Chown et al (1965) to 0.03 by Race and Sanger (1970). Therefore, the author discarded the Fy allele which also facilitates comparisons with published data on British populations.

Even though the Manx non - donors exhibit a higher frequency of allele Fy^a than the Manx donors, the difference between the two groups was insignificant and so they were pooled, giving a frequency of 0.4283 for the Fy^a allele.

Table 17b shows the distribution of Duffy blood groups in the Manx, Cumbrian and South West Scottish population samples tested with anti - Fy^a serum only. (All individuals in Table 17a are included in the Manx samples shown in Table 17b) As no significant differences existed between them, the two Manx series were pooled into one sample with 64% Fy(a+) and a frequency of Fy^a of

of 0.4002. This Manx sample was found to show a similar distribution of Duffy groups to that found in the Cumbrian and South West Scottish series. The allele Fy^a rises only very slightly in frequency, from 0.3702 in Scotland, through 0.3838 in Cumbria to a maximum of 0.4002 in the Isle of Man. As for the P_1, Lutheran and Kell blood groups, the three indigenous population samples are found to exhibit homogeneity with respect to the Duffy system.

Ib) Serum Proteins (Tables 18 - 20)

i. Haptoglobin (Hp) Table 18

Table 18 shows the distribution of Hp groups and respective gene frequencies in the Manx, Cumbrian and South West Scottish population samples. The gene frequencies shown in Table 18 were calculated excluding phenotype Hp 0 - 0 from all samples. In all samples good agreement was found between the observed and expected phenotypic values, so confirming the assumption of Hardy - Weinberg equilibrium. Also, no significant sex differences were found in any sample.

The two Manx samples, being similar, were pooled. The difference between the two Scottish series was also insignificant, even though there is nearly a 6% difference in the frequency of Hp^1, and it is possible that a larger indigenous sample may produce a real difference. As discovered for a number of the blood group antigens, the native populations of the three areas were found to exhibit overall homogeneity with regard to Hp groups. The frequency of Hp^1 shows very slight variation, rising from 0.3403 in the South West Scottish and 0.3485 in Cumbrians to 0.3503 in the Manx. The resident Scottish donors with a frequency of 0.3862 lie at the extreme end of the range of variation.

ii. Transferrin (Tf) Table 19

Table 19 shows the distribution of Tf groups and respective gene frequencies in the Manx, Cumbrian and South West Scottish population samples. No sex differences of significance were observed. Though contingency tests were impossible, it can be seen that the samples show similarity in serum Tf groups. In all samples shown in Table 19 the frequency of variant alleles other than Tf^{C}, does not exceed 1%. As expected in Caucasoid populations, the most frequent variant, other than C, was the faster moving BC type. It was not possible to sub - type the B variants, but when run collectively on starch - gel all BC variants exhibited similar mobility.

The one slow moving variant, CD type, found in the Manx, was distinguished by its very slow migration in starch - gel. No determination of sub - type was made.

iii. Beta - lipoprotein allotype Ag (Table 20)

Table 20 shows the distribution of Ag groups and the calculated allele frequencies in the small Manx population sample. No significant sex difference was found in the sample.

Ic) Red blood cell isoenzymes (Tables 21 - 27)

i. Acid phosphatase (AP) (Table 21)

Table 21 reveals the distribution of AP groups and respective allele frequencies in the Manx, Cumbrian and Scottish population samples. Close agreement was found between the observed and expected phenotype values confirming the assumption of Hardy - Weinberg equilibrium, and that they are random mating populations. No sex differences were observed.

No heterogeneity was found between the two Manx series with respect to common alleles, so they were combined into one sample. The same was also found with the two Scottish samples, so they were also pooled. The native Cumbrians were found to have a similar distribution of AP alleles to that found in the Manx and South West Scottish series. However, the Manx were significantly different from the total Scottish series, $X_2^2 = 6.5944$ $.05 < P > .025$ and also from the combined mainland sample, $X_2^2 = 7.0846$ $.05 < P > .025$.

The frequency of the P^a allele rises from 0.2500 in the small native Scottish sample, and 0.2866 in the total Scottish series, to 0.3393 in the Cumbrian series and 0.3385 in the Manx, a range of less than 10%. The P^b allele exhibits a range of over 10%, rising from 0.5969 in the Manx to 0.7045 in the native South West Scots. The Manx native population exhibits the highest frequency of P^c (0.0646) in the three populations tested, declining to 0.0493 in South West Scotland and 0.0268 in Cumbria. The Manx frequency of the P^c gene is the highest figure recorded in a British population.

ii. Phosphoglucomutase locus I (PGM_1) Table 22

Table 22 shows the distribution of PGM_1 groups and respective gene frequencies in the Manx, Cumbrian and South West Scottish population samples. There was close 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. All sex differences were statistically insignificant.

No significant differences were demonstrated between any of the samples shown in Table 22 with respect to either common PGM_1 phenotypes or genes, so the indigenous populationsof the three areas comprise a homogeneous unit. All specimens tested exhibited phenotype 1 at the PGM_2 locus.

The frequency of the PGM_1^1 allele has a range of only 4%, rising from 0.7000 in South West Scotland to 0.7412 in the Isle of Man. It is of interest to note that phenotype 7 - 1 was found in all three native populations with a frequency of more than 1%. This phenotype is usually found, if at all, with frequencies much less than reported for these populations.

iii. Adenylate kinase (AK) Table 23.

Table 23 shows the distribution of AK phenotypes and respective allele frequencies in the native Manx, Cumbrian and South West Scottish population samples. Close agreement was found between the observed and expected phenotype values which confirms the assumption of Hardy - Weinberg equilibrium. All sex differences were statistically insignificant.

Just as was found with the PGM_1 system, no significant differences were demonstrated among the samples included in Table 23, and so the three regional populations are also homogeneous with respect to common AK phenotypes and genes. The variation in the frequency of the AK^2 allele is less than 2%, rising from 0.0333 in South West Scotland and 0.0338 in the Isle of Man to 0.0526 in Cumbria. No rare AK variants were discovered in any of the samples examined.

iv. Adenosine deaminase (ADA) Table 24

Table 24 shows the distribution of ADA groups and respective calculated gene frequencies in the native Manx, Cumbrian and Scottish population samples. All samples displayed internal homogeneity and all sex differences were insignificant.

The Manx donors and non - donors were found to differ significantly from each other in the proportion of common ADA alleles, $X_1^2 = 5.1402$ $.05 < P > .025$. A similar finding was noted when the ABO and P blood group distributions were examined in the Manx samples. However, the combined Manx sample was found to exhibit a similar distribution of ADA genes and phenotypes as found in the very small Cumbrian and Scottish series.

Even though not statistically significant, the ADA^2 gene exhibits a variability of over 7% within the three populations. It rises from a frequency of 0.0455 in Cumbrians through 0.0755 in the Manx to 0.1167 in the very small Scottish sample. While bearing in mind the very small size of the samples, except the Manx, it should be noted that the frequencies of ADA^2 in the Isle of Man and South West Scotland are the highest yet reported in the British Isles population .

v. 6 - phosphogluconate dehydrogenase (6 - PGD) Table 25

Table 25 shows the distribution of 6 - PGD phenotypes and respective gene frequencies in the native Manx, Cumbrian and Scottish population samples. Internal homogeneity was found in all samples and all sex differences were statistically insignificant. No variants other than the 'common' CA type were found.

The only significant sample heterogeneity found in Table 25 was the same as that found for the isoenzyme ADA; that between the Manx donor and Manx non - donor samples, $X_1^2 = 6.2825$ $.025 < P > .01$. This difference between Manx donors and non - donors observed for some genetic systems requires further detailed investigation. Collection of greater numbers in each category would certainly make any statistical conclusions more reliable. The heterogeneity demonstrated in the ABO and P blood groups, and the isoenzymes ADA and 6-PGD, could be hiding regional variability, but unfortunately it cannot be determined if this is so for all these systems because the number observed of the least common phenotype, such as ADA 2-1 and 2-2 and 6-PGD CA is insufficient to permit regional subdivision.

If the two Manx samples were pooled (despite the statistical evidence), it was found to exhibit a similar distribution of 6-PGD alleles and phenotypes, as found in the still very small combined Cumbrian and South West Scottish sample.

vi. Glucose-6-phospho-dehydrogenase G-6-PD (Table 26)

Table 26 illustrates the distribution of G-6-PD phenotypes and respective gene frequencies in the indigenous Manx, Cumbrian and Scottish population samples. Only one variant phenotype other than B was found in the Manx, and that was the female variant BA. No variants other than B were discovered in the small mainland series.

vii. Lactate dehydrogenase (LDH) (Table 27)

All 293 specimens from the Isle of Man exhibited the normal phenotype, as also did the 30 Cumbrian and 18 Scottish specimens.

viii. Phospho-hexose isomerase (PHI) (Table 27)

All 293 Manx specimens analysed were found to exhibit phenotype 1. Similar results were obtained for the 30 Cumbrian and 18 Scottish specimens.

ix. Malate dehydrogenase (MDH) (Table 27)

Of the 153 Manx specimens tested for soluble MDH phenotypes all were found to be type 1. (Leakey et al 1972)

II. NON-SEROLOGICAL VARIABILITY.

(a) Tongue Curling (Table 28)

Table 28 exhibits the observed number and frequency of tongue-curlers in Manx adults, Manx children and Cumbrian children. All sex differences were statistically insignificant.

The heterogeneity observed between the two age classified Manx samples was statistically significant, $X_1^2 = 5.4538$ $.025 < P > .01$. However, the two age similar series, the Manx and Cumbrian school-children, exhibit similar proportions of tongue - curlers, and together they comprise a homogeneous population with a frequency of tongue - curlers of 70%. The Manx adults and Cumbrian school-children also exhibit similarity in the proportions of tongue - curlers. The author was unable to discover any report of an age - difference in the ability to curl the tongue in Caucasoid populations. If, in spite of the statistical evidence, the two Manx series are combined into one sample, it is found to have a similar proportion of tongue - curlers as the Cumbrian sample.

(b) Colour vision deficiency. (Table 29)

Table 29 illustrates the observed number and frequency of males and females exhibiting anomalous colour vision in the native Manx and Cumbrian population samples, determined by the use of the Ishihara pseudoisochromatic plates. Being a sex - linked condition the frequency of colour vision defectives is presented in males and females separately.

(i) Males (Table 29a)

Though no significant heterogeneity could be demonstrated

between the Manx adults and Manx schoolchildren it should be noticed that the former exhibit almost double the frequency of colour defectives than the latter. The frequency found in the Manx adults, 0.0781, is much closer to those previously reported in North British populations. (Vernon and Straker 1943, Post 1962) The total Manx sample (0.0561) was found to have a similar proportion of defectives as the Cumbrian sample, (0.0292) so the population samples of the two areas form a homogeneous group, with a frequency of 0.0477 colour - blind.

(ii) Females (Table 29b)

Only occasionally in population surveys of this nature is the frequency of colour defectives in females reported. The results obtained in the present survey are shown in Table 29b. The frequency of colour - blind Manx females (0.0121) is higher than that usually reported in British populations, and also high in relation to the frequency of the abnormal allele in the Manx males. This high frequency may be a result of possibly a larger number of inter - related persons in the female sample. Similar to the Manx, the frequency of colour vision deficiency in Cumbrian females (0.0097) is quite high, especially when set against the relatively low frequency (0.0292) in Cumbrian males.

(c) Phenylthiocarbamide (PTC) tasting ability (Table 30)

The distribution of PTC taste thresholds and the frequency of non - tasters of PTC in native Manx and Cumbrian population samples are shown in Table 30. To enable statistical comparisons the antimodal value was taken at solution 4, and in all samples, with the exception of the Cumbrian schoolchildren, the antimode fell at solution 4.

No statistically significant heterogeneity could be demonstrated between the two Manx series, or between the total Manx and the Cumbrian population with respect to the proportion of non - tasters of PTC. An interesting feature of Table 30 is the lower incidence of non - tasters in the Manx adults, 24.9%, than in the Manx schoolchildren, 30.0%, which is the reverse of the usual finding (Harris and Kalmus 1949). A possible explanation of this non - significant difference may be that one of the samples contains a higher proportion of related individuals than the other. An alternative explanation could be that as the majority of the adults tested are blood donors, most of whom reside in Douglas, there is regional variation in frequency of taster and non - taster phenotypes. The frequency of non - tasters is 28% in the total Manx, 24% in the Cumbrians and 27% in the two samples combined.

Conclusion

The major finding revealed by the analysis of the results of the present study is the overall similarity of the frequency distributions of the genetic polymorphisms investigated in the indigenous population of the Isle of Man, Cumbria and South West Scotland. While the finding was not totally unexpected, the degree of similarity, expressed in genetic terms, is quite striking. Homogeneity was found among the three populations with respect to common phenotypes and/or alleles of the following polymorphic traits; MN, P_1, Lutheran, Kell and Duffy blood group antigens, secretor groups, the serum proteins Hp and Tf, the red cell enzymes PGM locusl, AK, ADA, 6-PGD, G-6-PD, LDH, PH1 and MDH, and the non - serological variables, tongue curling, colour vision deficiency and PTC tasting ability. The Manx and Cumbrian populations are also found to be similar with respect to reflectance spectrophotometry of the skin (see Chapter Five).

In the instances of those polymorphisms where heterogeneity among the three indigenous populations are found, the differences are often only just significant (5% level of probability) e.g. Manx v South West Scots, Manx v Cumbrians, and Cumbrians v South West Scots, with respect to common Rh Types, and the Manx v South West Scots and Manx v mainland samples, with regard to common AP alleles. However, the difference noted between the Manx and Cumbrians in the distribution of MNSs groups is more definite. In the case of the ABO system most of the heterogeneity is found to lie between the Manx donor series and the samples from Cumbria and South West Scotland. Regardless of which of the three samples one employs as representative of the ABO distributions in the Isle of Man, it is found to be significantly different from the South

West Scottish sample which, it should be remembered, largely comprises residents rather than known indigenous inhabitants.

An overall pattern of gene distribution in the three populations is apparent; that of a raised incidence of particular alleles in the Manx, compared with their frequency in the two mainland groups. Those alleles displaying the highest frequency in the Isle of Man comprise the following:- O, N, Ms, NS, Ns, R_2 (cDE), Lu^a, Kp^a, Fy^a, Hp^1, Tf^B, PGM_1^1, G-6-PD^A, colour vision deficiency in males and females and non - tasters of PTC.

The other major finding of the present study, restricted to the Manx, is the heterogeneity, often quite large, between the donor and non - donor samples with respect to phenotypes and/or alleles of certain polymorphic traits, including the ABO and P blood group antigens and the cellular enzymes, ADA and 6-PGD. This finding was totally unexpected. Firstly, because the individuals comprising the donor and non - donor samples were selected by the same criterion, that is all have three or four grandparents and both parents born on the Isle of Man, and secondly, from the work of Kopeć (1970) blood donors were shown to be representative of the populations of which they are a constituent part. It was mentioned that the present survey involved asking <u>all</u> blood donors on the Isle of Man to participate in the survey and not just new donors or those who did not know their blood groups. On comparison with other ABO data for the Manx population (Pantin 1950), it was suggested that the present Manx donors exhibit aberrant ABO frequencies, and reasons were suggested for this phenomenon, principally selection for group O persons, 'the universal donor', in the early days of the establishment of the B.T.S. on the Isle of Man. However, as to why there

should be differences between Manx donors and non - donors with respect to other genetic systems mentioned, the author has no answer at the present time. Regional analysis of these polymorphic systems, which may have determined whether geographical variability lies behind these sample differences, was not possible because of the observed small numbers of each phenotype other than the common one. Further investigation of these differences between Manx donors and non - donors is required, particularly the collection of larger samples in each category to permit regional subdivision.

Significant age differences were reported in the Manx data for common Rh Types and tongue - curling ability. Also, there was a sex difference in the distribution of groups M and MN of the MN system in the Manx data, but not in group N.

Just as selection of group O donors was suggested to account for the higher frequency of O in Manx donors, so selection for Rh(D) negative individuals by the Manx B.T.S. would account for the higher incidence of Rh(D) negative in the donor sample, compared with the non - donor samples. This phenomenon is not reported for the Cumbrian Rh data.

It has also been shown that the indigenous Manx population exhibits amongst the highest incidences reported in the British Isles for the Lu^a and Kp^a alleles.

CHAPTER FOUR

ISLE OF MAN - REGIONAL ANALYSIS

ISLE OF MAN - REGIONAL ANALYSIS

Introduction.

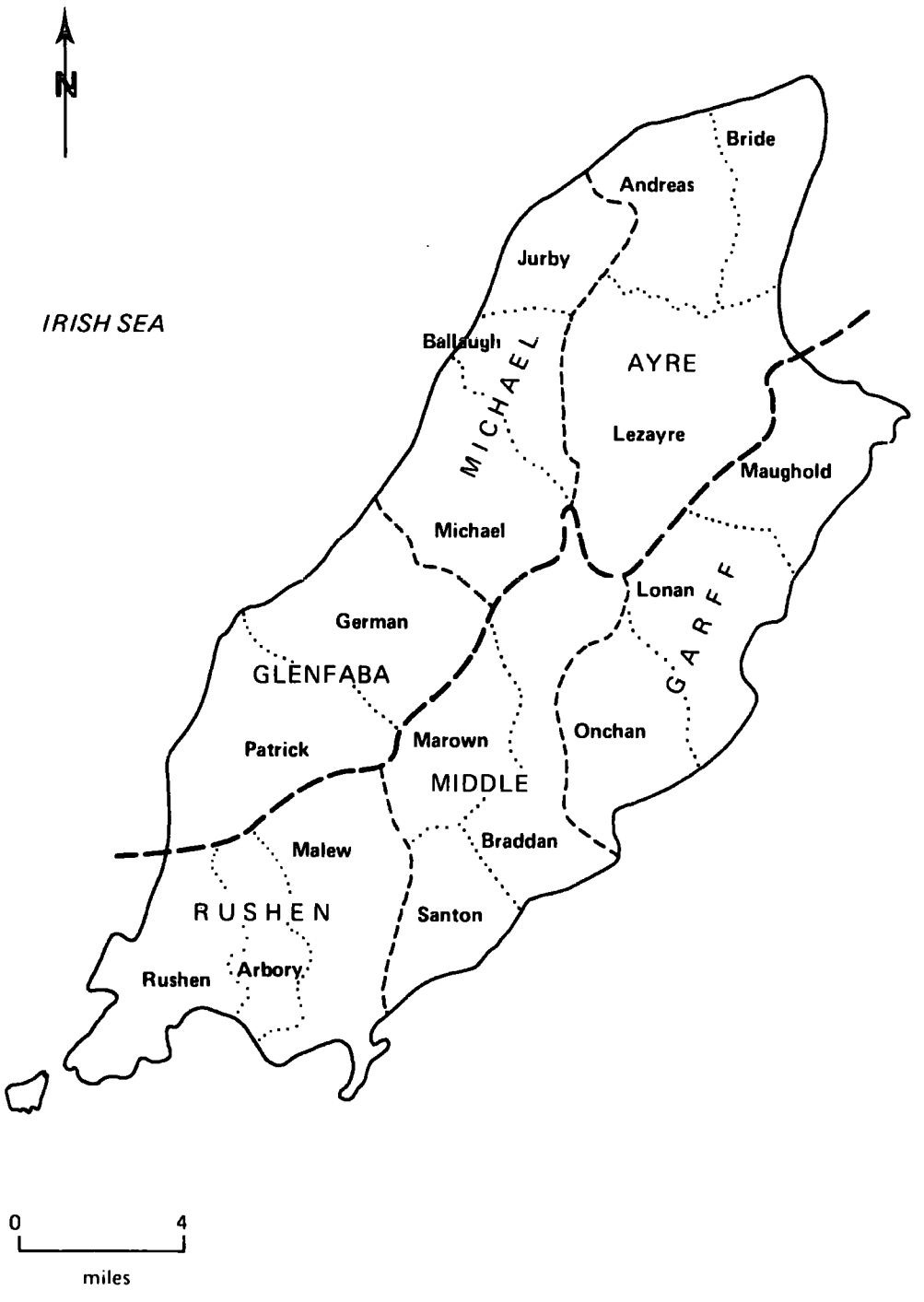
One major aim of the present survey is an investigation of how the indigenous Manx population compares with neighbouring peoples surrounding the Irish Sea basin in particular, and British and Irish populations generally, with respect to the distribution of selected genetic polymorphisms. Another major purpose of the work is to determine whether any intra-island genetic heterogeneity is discernible in the native Manx population, and if so, to suggest possible explanations for the genetic distributions observed.

The Isle of Man today has a population of under 60,000 of which number as explained earlier, perhaps only between 10% and 15% can be called 'native' as the word is employed in this study. Among these 5,500 - 8,500 indigenous inhabitants there exist blood relationships of all complexities. Therefore, to collect a reasonably large random sample of this particular population group would be extremely difficult and time - consuming. The problems are increased when one notes the predominance of a few distinctive surnames (Appendix 1) on the Island and that possession of an identical surname does not necessarily imply any known blood relationship between the bearers. Stenning (1958) states that there is in fact less likelihood of people of the same name being closely related than persons with different names. In support of this he cites a common quip in the Island "Same name, no relation. Different name, probably a cousin." (p. 109)

Accordingly, all regional samples referred to in this section, as also with the total Manx series itself, lay no claim to be random samples of a particular population group of the Island, but rather a collection of individuals, related and unrelated, whose

Figure 25

ISLE OF MAN – ADMINISTRATIVE DIVISIONS (PRE 1796) – ALSO SHOWING DIVISION INTO NORTHSIDE AND SOUTHSIDE



Boundary between Northside and Southside

Sheading Boundary

Parish Boundary

ancestry clearly places them in one of the selected geographical sub-units of the Island.

The Isle of Man is divided for the purposes of government into six units called sheadings which are further subdivided into seventeen parishes. (Fig.25) There is some dispute among scholars concerning the derivation of the the term 'sheading' and its date of origin. Some think that it means 'a sixth part' and is of Celtic or Norse origin, others think that it means 'a ship division' a term introduced by the Norse. More recently it has been suggested that the word is derived from the Middle English 'scheding' meaning a 'division', possibly introduced by the Stanley's in the fifteenth century. (Kinvig 1950)

The organisation of parishes on the Island was probably carried out during the reign of Olaf I (1113 - 1153). Apart from those in the northern lowland, parishes follow a regular pattern in relation to physiography; each having a frontage along the coast and thence running to the main line of water parting (Fig.25). The one exception to this rule is Marown which is entirely inland, but it seems clear that this parish was originally united to Santon, so that initially there were sixteen parishes. (Kinvig 1958) Today the parishes are primarily concerned with ecclesiastical and civil matters but at first their function was more of a military character.

The six sheadings and their constituent parishes before and after the administrative changes made in 1796 are shown below:-

Pre - 1796.

Major Division	Sheading	Parishes
	Glenfaba	Patrick, German
NORTH	Michael	Michael, Ballaugh, Jurby
	Ayre	Bride, Andreas, Lezayre
	Garff	Maughold, Lonan, Onchan
SOUTH	Middle	Marown, Braddan, Santon
	Rushen	Malew, Rushen, Arbory

Post - 1796

	Michael	Michael, Ballaugh, Jurby
NORTH	Ayre	Bride, Andreas, Lezayre
	Garff	Maughold, Lonan
	Glenfaba	Patrick, German, Marown
SOUTH	Middle	Onchan, Braddan, Santon
	Rushen	Malew, Rushen, Arbory

The natural boundary between the sheadings of the North or 'Northside' (Glenfaba, Michael and Ayre) and those of the South or 'Southside' (Garff, Middle and Rushen) is the line of water - parting which closely follows the main highland belt. (Fig.2) The administrative division of the Island prior to 1796 corresponded to this natural division of the Isle of Man. These two areas of the Island were called 'Northside' and 'Southside' by Bowen (1969). The changes made in 1796 blurred the original pattern, since the North was decreed to include Michael, Ayre and Garff sheadings, and the South the other three. Marown parish was also transferred to Glenfaba sheading and Onchan to Middle.

Regional divisions of the Island employed in previous anthropological studies.

The subdivisions of the Isle of Man employed by previous anthropologists in their studies of the Manx population are of interest to the present study. Moore and Beddoe (1898) employed the post - 1796 administrative division of the Island into North and South, which has little or no basis in the historical tradition of the Island. This relatively modern division was a product of the rise in importance of the east - west line of communication between Douglas and Peel during the eighteenth and nineteenth centuries, which served to reduce the formerly more important division into Northside and Southside.

Davies and Fleure (1936) selected seven natural units of the Island for their analysis of anthropometric data on the native Manx population. (Fig.23) The units were merely further subdivisions of the historical Northside and Southside regions, the only difference being that Patrick parish was placed in the Southside instead of the Northside. Davies and Fleure's subdivisions are shown below :-

Parishes/Natural Units	Major Division
1. Bride, Andreas and Jurby	
2. Ballaugh and Lezayre	NORTHSIDE
3. Michael and German	
4. Patrick, Rushen and Arbory	
5. Maughold and Lonan	SOUTHSIDE
6. Onchan, Braddan and Santon	
7. Malew and Marown	

Regional Divisions of the Island used in the Present Study.

It would have been most valuable if analysis of the present data on the level of individual parishes was possible, as Davies and Fleure (1936) had effected. However, the fact that the seventeen parishes exhibit very different population densities excluded this possibility when the size of the present sample is considered. Most of the individuals tested could be allocated into a few of the parishes, there being no even distribution of the birthplaces of parents and grandparents of those incorporated in the survey.

Another suitable level of analysis of the present data is the six sheading divisions. However, the problem of uneven distribution of birthplaces of parents and grandparents meant that the numbers allocated to each sheading were also very unequal, with the largest numbers found in Middle and the lowest in Michael. This finding is not an unexpected one when the present distribution of population is examined.

It was mentioned above that the natural boundary between the Northside and Southside was the line of water - parting which closely follows the highland belt. This division existed not only on the map but in the minds of the Manx people, and there were many points of difference between the Northside and Southside dialects and customs. In early days the two areas were like independent countries occasionally in conflict, as recorded for example in 1098, and the Norse ruled the two areas as semi - independent kingdoms (Kinvig 1950). This feeling of separateness is well expressed in the words of the Manx poet T. E. Brown in ' Braddan Vicarage.'

'I wonder if the hills are long and lonely
That North from South divide;
I wonder if he thinks that it is only
The hither slope where men abide
Unto all mortal homes refused the other side.'

Each region had its own capital, Castletown in the Southside and Peel in the Northside, its own chief and various other officers connected with laws and defence. Until 1918 there was a Northern and Southern Deemster or judge, but since then they have been known as the First and Second Deemsters.

It was felt that the most suitable analysis of the present data would be effected by employing the traditional and historical Northside and Southside regions of the Isle of Man. This division corresponds to the administrative division of the Island previous to 1796, in which Marown was placed in the Southside. Thus the Northside comprises the parishes of Partick, German, Michael, Ballaugh, Jurby, Bride, Andreas and Lezayre, while the Southside, as defined for this study, constitutes the following nine parishes; Maughold, Lonan, Onchan, Marown, Braddan, Santon, Malew, Rushen and Arbory. Individuals were only allocated to the Northside or Southside if they had both of their parents and three or four of their grandparents born in one of these regions. All individuals who did not qualify for either of these two regions were excluded from regional analysis. In view of such rigorous selection, the sample sizes in the two units tend to be small.

In addition to the Northside - Southside division of the Island it was felt appropriate to investigate the possibility of genetic heterogeneity between the urban and rural populations.

Only Douglas, the Island's capital since 1869, qualifies as a truly urban centre. However, throughout modern historical times, Peel, Castletown and Ramsey, together with Douglas have been known as the Four Towns of the Isle of Man, and the former three still provide some of the functions associated with an urban centre.

In a previous chapter the phenomenally rapid growth, by Manx standards at least, of Douglas has been explained and reference made to the variety of immigrants the town attracted in contrast to the remainder of the Island. Owing to these differences in development, Douglas was selected as a single unit for intra-Island comparisons. Individuals were allocated to the Douglas or Total Urban series only if they had three or four grandparents and both parents born in Douglas or the Four Towns respectively. Individuals with two grandparents or less born in one of these respective units were excluded.

One effect of employing the urban - rural distinction was that the Northside and Southside series themselves were further subdivided by removing from their number all those individuals who did not have three grandparents born in the rural area of the respective division.

This four - fold division of the indigenous Manx population sample, Northside - Southside - Urban - Rural, produced the following seven regional units:-

Regional Units of the Isle of Man

1. DOUGLAS - all individuals who have three or four grandparents and both parents born in the urban district of Douglas.
2. TOTAL URBAN - all individuals who have three or four grandparents and both parents born in the Four Towns (Douglas, Ramsey, Castletown and Peel). i.e. includes 1.

3. NORTHSIDE - all individuals who have three or four grandparents and both parents born in the area known as the 'Northside' comprising the parishes of Patrick, German, Michael, Ballaugh, Jurby, Bride, Andreas and Lezayre.
4. SOUTHSIDE - all individuals who have three or four grandparents and both parents born in the area known as the 'Southside', comprising the parishes of Maughold, Lonan, Onchan, Marown, Braddan, Santon, Malew, Rushen and Arbory.
5. RURAL NORTHSIDE - all individuals who have three or four grandparents and both parents born in the rural area of the Northside. (i.e. not in Ramsey or Peel)
6. RURAL SOUTHSIDE - all individuals who have three or four grandparents and both parents born in the rural area of the Southside. (i.e. not in Douglas or Castletown)
7. TOTAL RURAL - all individuals who have three or four grandparents and both parents born in the rural area of the Isle of Man. (i.e. sum of 5 and 6)

Not all the genetic polymorphisms investigated in the Manx population proved to be suitable for regional analysis for one reason or another. Owing to the relatively small size of the samples in the case of some genetic factors (such as the Ag system and secretor status) they were excluded from regional comparisons. Also, even where there was a relatively large sample in each of the regional units, the frequency of the least common phenotype of a particular system such as AK2-1 or ADA 2-1 may be so small that it precluded valid statistical comparisons. Owing to the above restrictions the regional distributions of the following genetically controlled traits only were investigated.

A. Serological

1. Blood Group Antigens - ABO, MNSs, Rh, Lu^a, K,k,Kp^a and Fy^a
2. Serum Protein Groups - Hp
3. Red Cell Isoenzymes - AP and PGM_1

B. Non - Serological

1. PTC Tasting Ability
2. Tongue Curling

A. Serological Traits

1. Blood Group Antigens

(a) ABO system. (Table 31)

Table 31 presents the observed number and frequencies of ABO phenotypes in the seven regional units, tested with and without anti - A_1 serum respectively. The gene frequencies are also included in Table 31. Statistical analysis demonstrated that all seven regions were similar with respect to ABO groups. However, it is noticeable that the Douglas sample (1) lies at the extreme end of variation, exhibiting the highest incidence of group O. In fact the Douglas population is different from regions 3, 5, 6 and 7 at the 10% level of probability but not at the significant (5%) level.

However, when the proportion of phenotypes O and A respectively is compared in each of the regions, Douglas is found to differ significantly from the other regions with the exception of the Rural Northside, A v non A.

Douglas v Northside	(3)	A v. non A	$X_1^2 = 4.5590$	$.05 < P > .025$
Douglas v Rural Northside	(5)	A v. non A	$X_1^2 = 3.6393$	N.S.
Douglas v Rural Southside	(6)	A v. non A	$X_1^2 = 4.0317$	$.05 < P > .025$
Douglas v Total Rural	(7)	A v. non A	$X_1^2 = 4.7280$	$.05 < P > .025$
Douglas v Northside	(3)	O v. non O	$X_1^2 = 5.1501$	$.025 < P > .01$
Douglas v Rural Northside	(5)	O v. non O	$X_1^2 = 5.0932$	$.025 < P > .01$
Douglas v Rural Southside	(6)	O v. non O	$X_1^2 = 4.3218$	$.05 < P > .025$
Douglas v Total Rural	(7)	O v. non O	$X_1^2 = 5.4667$	$.025 < P > .01$

Douglas was not compared with regions 2 and 4 because the individuals comprising the Douglas sample also form part of each of these

two series. With larger sample sizes it may well be found that the populations of some of these regions, especially Douglas, display significant differences with respect to the proportion of ABO phenotypes.

It is in the frequency of groups O and A that the heterogeneity among the seven regional samples is found, with O rising from 0.4314 in the Rural Northside to 0.5900 in Douglas, a range of 16%; while A rises from 0.3200 in Douglas to 0.4586 in the Northside, a range of nearly 14%. This wide fluctuation in these two groups is also expressed in the A:A+O indices, which vary from 35.16 in the Douglas sample to 51.11 in the Rural Northside. This variability is remarkable considering the small size of the Island.

The frequency of A_2 increases from 0.0612 in Douglas to a maximum of 0.1212 in the Rural Northside. In six of the regional samples the p^2:p ratio lies between 1:4 and 1:3, whereas in the Douglas series it is less than 1:4.

There is no significant variation in the proportion of B and AB phenotypes among the seven units, group B rising from 0.0526 in the Rural Southside to 0.0882 in the Rural Northside. This consistency in the frequency distribution of B and AB in the Isle of Man is very different from the situation reported in Northern England by Fraser - Roberts (1953).

The ABO gene frequencies very much reflect the phenotype distributions. Gene p has its lowest frequency, 0.1850, in Douglas and its highest 0.2826, in the Northside, while r exhibits its highest incidence in Douglas, 0.7648, and its lowest 0.6546, in the rural Northside. Gene q shows considerably less variation, ranging from 0.0371 in Douglas to 0.0638 in the Rural Northside.

The observed differences with regard to groups O and A between Douglas and the other Manx regions are very interesting yet not so easy to explain satisfactorily. It was mentioned in chapter one that Douglas experienced a distinct and very rapid development compared with other population centres on the Island. It was shown that the population influxes of the late eighteenth and nineteenth centuries could be attributed to various sources, such as the settlement of foreign debtors and 'half - pay' officers, troop garrisons, the growth of banking and other commercial services and most important of all, the phenomenal growth of tourism which was centred on Douglas. As a result of these changes the population of Douglas doubled between 1821 and 1861, and in 1891 it contained more than one - third of the Island's total population.

There is little doubt that in the nineteenth century the Isle of Man attracted many persons who considered their stay on the Island was only temporary, such as the half - pay officers, troops and debtors. However, many of these remained, most, if not all, of their lives and thus contributed to the genetic pool of the Douglas population. It is possible then that the effects of this group are still being seen today in the distribution of the ABO blood groups in the indigenous Douglas inhabitants.

If there were to be regional differences in the distribution of the ABO blood groups one might have expected to find them associated with the traditional division of the Island into Northside and Southside, rather than with the relatively recent urban - rural distinction. Though the Northside has a higher frequency of group A than the Southside, the difference does not approach the level of significance. The internal and external movements

of the Manx population during the past 200 years could well have removed any significant genetic heterogeneity that may have existed between these two areas prior to the vast social and economic changes of the nineteenth and twentieth centuries.

In conclusion the regional analysis has shown that the indigenous Manx, with the possible exception of the Douglas sample, comprises a homogeneous group with respect to ABO groups.

(b) MNSs system. (Table 32 and 33)

1. MN groups.

The distribution of MN groups and the respective calculated gene frequencies are shown in Table 32 for the seven Manx regions employed in this study. The indigenous Manx population exhibits overall homogeneity with regard to MN phenotypes and genes, but their distribution is of some interest. The frequency of MM rises from just over 0.24 in Douglas and the Southside to a maximum of 0.29 in the Northside, while MN increases from 0.49 in the Rural Northside to over 0.55 in the Rural Southside and NN increases in incidence from 0.20 in the Rural Southside to 0.23 in the Rural Northside and 0.26 in Douglas.

The largest difference in the frequency of the M gene is that found between the Douglas and Total Urban series. The former sample is incorporated in the latter one and this finding strengthens the suggestion of the distinctive genetic structure of the present day indigenous Douglas population, first noticed with respect to ABO groups. The distribution of MN groups in the Urban sample minus the Douglas series is shown along with the Douglas sample below;

	Douglas		Urban Series (3 Towns only)	
Group	No.	Freq.	No.	Freq.
MM	21	.2442	30	.3125
MN	43	.5000	52	.5417
NN	22	.2558	14	.1458
Total	86	1.0000	96	1.0000
M	0.4942		0.5833	
N	0.5058		0.4167	

The disparity between the two samples is a product of the excess of MN and the deficit of MM groups in the Douglas population. However, the relatively small size of the samples should be borne in mind when interpreting these results.

(ii) MNSs groups (Table 33)

Table 33a and 33b present the distribution of MNSs blood groups and respective gene complex frequencies, after testing with four and three antisera respectively, in the seven Manx regional samples. Statistical analysis was only performed on those samples shown in Table 33b tested with anti - M, anti - N and anti - S. Using the chi - squared test, the Manx population is found to exhibit overall homogeneity with respect to common MNS phenotypes. Interestingly, the largest X^2 value, though not significant, is that found between the Northside and Southside populations, $X^2_4 = 8.9556$, $.10 < P > .05$. The second largest value is that found between the Rural Northside and Rural Southside samples. It is the higher frequency of MMS, MNS and NNS in the Southside compared with the Northside that largely contributes to the chi - squared value. In fact there is a 10% higher frequency of S+ persons in the Southside (0.48) compared with the Northside (0.38).

Regarding the distribution of gene complexes in the seven regions shown in Table 33a, two of them, MS and NS, exhibit their greatest difference between the urban and rural samples while the greatest divergences in the frequency of the other two, Ms and Ns, are associated with the Northside - Southside division. Whereas MS exhibits its lowest frequency in the Total Rural sample (0.2207) and its highest (0.2615) in the Total Urban series, NS shows the

lowest incidence in the Total Urban (0.0169) and the highest (0.0561) in the Total Rural samples. Ms increases in frequency from 0.2758 in the Rural Southside to a maximum of 0.3226 in the Rural Northside while Ns increases in frequency from 0.3997 in the Rural Northside to 0.4479 in the Rural Southside.

The frequencies of the gene complexes included in Table 33b are based on larger sample numbers tested with three antisera, anti - M, anti - N and anti - S only. MS rises in frequency from 0.1417 in the Rural Northside to 0.1969 in the Rural Southside and 0.1995 in the Total Urban series and Ms increases in incidence from 0.2984 in the small Douglas sample and 0.3078 in the Southside to a maximum of 0.3912 in the Northside. The other two genes, NS and Ns, show greatest differences between urban and rural samples; NS rising from 0.0499 in the Urban sample to 0.0937 in the Rural series and Ns increasing from 0.3951 in the Rural population to a maximum of 0.4591 in Douglas.

(c) Rh system. (Table 34)

The distribution of Rh types and the frequency of the respective Rh gene complexes found in the seven Manx regional series are shown in Table 34a. The seven regions are found to exhibit overall similarity with respect to common Rh types. For statistical purposes similar Rh groupings as used in chapter three were employed in 6x2 contingency tables. Though no significant heterogeneity was demonstrated, the incidence of R_1r exhibits variability of 10%, rising from 0.2727 in Douglas to 0.3852 in the Northside. Rh types R_1R_1 and R_1R_2 show greatest variability between the Rural Northside and Rural Southside populations while rr exhibits the greatest difference between the Northside and the Rural Southside.

The frequency of gene complex r rises from 0.3897 in Douglas to a maximum of 0.4576 in the Total Urban series. This finding again highlights the distinctiveness of the indigenous population of Douglas compared with the other Manx regions first mentioned in connection with the ABO and MN blood-group systems. The differences in the frequency of gene complexes r and R_2 are most marked between Douglas and the Total Urban series. As the Douglas sample is incorporated within the Total Urban series, it demonstrates how different Douglas is from the three other Manx towns in the distribution of Rh gene complexes. The gene complex R_1 increases in frequency from 0.3338 in the Total Urban sample to 0.3921 in the Rural Southside.

The distribution of Rh(D) negative persons in the seven Manx regional samples is shown in Table 34b. Statistical analysis revealed that the indigenous Manx population is similar

with respect to this genetic trait. No local geographical fluctuations in the frequency of the d gene is observed in the Manx such as has been reported in some British populations. (Brown 1965).

(d) Lutheran system (Table 35)

Table 35 presents the observed number and frequency of Lu(a+) individuals in each of the seven Manx regions, together with the calculated gene frequencies. Owing to the small number of Lu(a+) persons in the Douglas sample not all the usual statistical comparisons could be performed.

Statistical analysis demonstrated that the six regions exhibit similar proportions of Lutheran groups and that the Manx population can be regarded as a homogeneous group. The frequency of Lu^a rises from 0.04 in Douglas, the Southside and Rural Southside to 0.07 in the Northside and Rural Northside. It is of interest to note that the Douglas sample again lies at the extreme end of variability. While a rise in gene frequency of 3% is small in absolute terms, its effect here is to almost double the frequency of the gene in one region compared with the other. Though a rise in frequency of a gene from 0.50 to 0.65 between one population and another is far greater in absolute terms, a rise from 0.04 to 0.07 may have a greater effect on the distinctiveness in genetic terms of the populations concerned.

(e) Kell system (Table 36)

Table 36 shows the distribution of the Kell blood groups and calculated gene frequencies in the seven Manx regional samples. Owing to the very low frequency of phenotype KK found in any population (none were found in the present Manx series) statistical analysis is based upon the distribution of group Kk. As with the Lutheran blood groups, the Douglas series was excluded from statistical procedures because the expected number of group Kk in this sample was found to be less than five. The six remaining samples exhibit no significant differences and therefore the indigenous Manx constitute a homogeneous group with respect to Kell groups.

The variation in the frequency of K is over 2%, ranging from 0.0250 in Douglas to 0.0490 in the Rural Northside. There is very little difference in gene frequency between the Total Urban and Total Rural series, but both Northside series exhibit a higher frequency than the two Southside samples. Once again the Douglas sample lies at one end of the range of variability.

The observed number and frequency of Kp(a+) persons in each of the seven regions, together with the estimated gene frequencies are shown in Table 36b. However, insufficient numbers of Kp(a+) individuals were found in all of the seven regional samples to permit statistical comparisons. The highest frequency of the allele Kp^a is found in the very small Douglas series (0.0426) while the lowest incidence is found in the Rural Southside (0.0048). As with the Kk groups the two Northside regions exhibit a higher frequency of Kp^a than the two Southside regions. The Rural Northside series has a frequency of Kp^a five times greater than that found in the Rural Southside.

(f) Duffy blood groups. (Table 37)

Data on the distribution of Duffy blood groups in the seven regional samples are presented in two parts as a result of some specimens being tested with anti - Fy^a and anti - Fy^b sera, whereas others were tested with anti - Fy^a serum only. Table 37a presents the observed number and frequency of the three Duffy phenotypes and calculated gene frequencies in the seven regional samples. Gene frequencies were again calculated excluding the existence of the allele Fy.

Statistical analysis demonstrated that there are no statistically significant differences either in phenotype or gene distribution among the seven regions, and that the native Manx are homogeneous with respect to Duffy blood groups. The frequency of the Fy^a allele rises from 0.3788 in the Rural Southside to a maximum of 0.4531 in the Northside and there is a similar range of variation between the Total Rural series (0.3894) and the Total Urban series (0.4500). The collection of larger numbers in each regional sample would confirm the statistical insignificance or otherwise of the differences observed.

Table 37b presents the observed number and frequency of Fy(a+) individuals in each of the seven slightly larger regional samples, together with the estimated gene frequencies. Statistical analysis again confirmed that no significant heterogeneity exists among the regions with respect to common Duffy phenotypes. The frequency of Fy^a rises from 0.3355 in the Rural Southside to 0.4374 in the Northside, and from 0.3521 in the Total Rural to 0.4256 in the Total Urban series.

2. Serum Proteins.

(a) Haptoglobin (Hp) (Table 38)

Table 38 presents the distribution of Hp groups and the calculated gene frequencies in each of the seven regional population samples. No statistically significant heterogeneity is found amongst them with respect to the three common Hp phenotypes or the two alleles, Hp^1 and Hp^2. Therefore the Island's native population can be regarded as a homogeneous group but exhibiting a higher frequency of the Hp^2 allele and phenotype 2 - 2 than most other populations of the British Isles.

The frequency of Hp^1 rises from 0.3170 in the Rural Southside to a maximum of 0.3636 in the Rural Northside. Douglas also exhibits a high frequency of this allele in contrast to the neighbouring Rural Southside. It is of interest to note that the largest difference in gene frequency follows the traditional Northside - Southside division.

Phenotype 1 - 1 ranges in frequency from 0.0909 in the Rural Northside and 0.0958 in the Total Rural series to 0.1443 in the Total Urban series and a maximum of 0.1750 in the small Douglas series. Phenotype 2 - 1 increases in frequency from 0.3750 in Douglas and 0.4124 in the Total Urban series to 0.5455 in the Rural Northside. The variability in the frequency of 2 - 2 is nearly 10%, rising from 0.3636 in the Rural Northside to 0.4643 in the Rural Southside.

3. Red Blood Cell Isoenzymes.

(a) Acid phosphatase (AP) (Table 39)

Table 39 presents the observed number and frequency of the six common AP phenotypes together with the respective gene frequencies found in the seven Manx regional samples. All seven regions displayed overall homogeneity with respect to common genes and phenotypes. However, one difference of statistical significance was observed, that between Douglas and the Total Rural series, p^a v. p^b, $X_1^2 = 4.3784$ $.05 < P > .01$. This difference is most probably a product of the small size of the Douglas sample (N = 34). However, it should be recalled that there was a significant difference between these two population samples with respect to groups O and A of the ABO system. Before any firm conclusions can be drawn concerning these differences, larger samples are required. On the basis of the present small numbers the indigenous Manx exhibit overall similarity with respect to AP groups.

The frequency of P^a rises in incidence from 0.2941 in the Rural Northside to 0.3677 in Douglas, while P^b has its lowest frequency, 0.58882, in Douglas and its peak, 0.6373 and 0.6400, in the two Northside population samples. The rarest allele, P^c, shows variability of less than 3%, rising from 0.0441 in Douglas to 0.0686 in the Rural Northside.

The distribution of the AP phenotypes is somewhat different from that found for the three alleles. Phenotype A exhibits its lowest frequency, 0.0882, in Douglas and the highest frequency 0.1250, in the Total Urban series. Phenotype B also has its lowest frequency in Douglas, 0.2941, increasing by more than 13%

to 0.4400 in the Northside. Douglas has by far the highest incidence of BA, 0.5294, while the lowest frequency, 0.3333, is found in the two Northside regions. Douglas also exhibits the lowest frequency of CA, 0.0294, and CB, 0.0588, whereas the highest frequencies of these two groups, 0.0588 and 0.0784 respectively, are found in the Rural Northside.

(b) Phosphoglucomutase (PGM) Locus 1. (Table 40)

Table 40 presents the distribution of phenotypes observed at PGM locus 1 together with the respective calculated gene frequencies found in the seven Manx regional samples. The sample sizes in each case are much smaller than the author would have liked. Statistical analysis demonstrated that there is no statistically significant heterogeneity among the seven regions with respect to either phenotypes or genes, and therefore the Manx indigenes can be regarded as a homogeneous population. In addition all specimens tested were phenotype 1 - 1 at PGM locus 2.

The frequency of PGM_1^1 shows only slight variability, rising from 0.7099 in the Total Urban series to a maximum of 0.7667 in the Rural Northside. Two persons with the rare phenotype 7 - 1 were found, one in an urban and the other in a rural sample.

Just as was found with the isoenzyme AP, the phenotypic variation of PGM is greater than found in the distribution of alleles. The incidence of PGM 1 - 1 rises from 0.4848 in the Northside to 0.5659 in the Southside, with the corollary that PGM 2 - 1 exhibits greatest variability between these two regions also. Owing to the small numbers in the Douglas sample it was excluded from the above comparisons. However, this small series exhibits aberrant phenotype frequencies, having the highest incidence of PGM 1 and PGM 2, 0.6061 and 0.0909 respectively, and the lowest value, 0.2727, for PGM 2 - 1, found on the Island.

(B) Non - Serological Variability.

1. Tongue - curling (Table 41)

The observed number and frequency of tongue - curlers in each of the seven Manx regional samples are shown in Table 41. Statistical analysis demonstrated that the seven regions exhibit overall homogeneity with respect to the proportion of tongue - curlers.

The variability in the frequency distribution of tongue - curlers is very small, less than 5%, with the lowest incidence in the Rural Northside (0.6316) and the highest incidence in the Rural Southside (0.6834). No urban - rural differences exist with respect to this trait.

(2) Phenylthiocarbamide (PTC) Tasting Ability (Table 42)

The distribution of PTC taste thresholds and the number and frequency of non - tasters in the seven Manx regional samples are shown in Table 42. The antimodal value was taken at solution 4 for reasons explained in an earlier section, and fortunately in all seven series the antimodal value clearly falls at this solution, so removing any difficulties in the way of valid comparisons.

No statistically significant differences were found among the seven regions so the indigenous Manx population can be regarded as a homogeneous group with respect to the proportion of non - tasters of PTC. The greatest variability in the frequency of non - tasters is that between the urban and rural populations. The highest incidences are found in Douglas, 31.1%, and the Total Urban series, 29.9%, while the lowest frequencies are reported in the rural areas, with a value of 27.2% in the Total Rural series. Perhaps with the collection of larger numbers in each of these samples the differences noted between the urban and rural populations might approach the level of significance. Though other workers (Cartwright and Sunderland 1967, Mitchell and Swarbrick 1972) have reported urban - rural differences in the frequency of non - tasters of PTC in other British populations, they have found a lower frequency in the urban centres, such as Lancaster and Barrow - in - Furness, than in the surrounding rural areas.

Conclusions

It was mentioned earlier that previous studies of the anthropology of the Manx population reported, in varying detail, significant differences among various regions of the Island. These differences were recorded for a number of anthropometric and anthroposcopic traits, some of which are known to be under total or largely genetic control, such as hair and eye colour and skin pigmentation, and other characteristics known to be only partly genetically determined, such as stature and cephalic index. Because these earlier studies recorded consistent patterns in the distribution of many of these traits within the indigenous Manx population, it could not be totally unexpected to find somewhat similar variation in the distribution of the genetic polymorphisms examined in the present survey. However, no pattern of significant regional heterogeneity is reported for any of the traits investigated. In fact the dominant feature of the analysis is the overall similarity of the selected regions of the Island for most of the genetic factors. However, an overriding consideration is the small size, extremely so in some cases, of the regional samples collected for some polymorphisms. Until further data are collected the evidence supplied by the present study supports the view that the native Manx constitute a homogeneous population.

The two statistically significant differences that did occur were, variation in the distribution of phenotypes O and A of the ABO blood group system between Douglas and most other regions of the Island, and a difference in the AP genes, P^a and P^b, between the Douglas and the Total Rural series. It also appears that Douglas lies at one extreme of the variation exhibited by many of

the genetic systems investigated. Possible reasons for the distinctiveness of the indigenous Douglas population as compared with other regions of the Isle of Man, providing it is not merely a product of small sample size, have been mentioned.

The variation in the frequency of non - tasters of PTC between urban and rural Manx populations is of interest. The highest frequency of non - tasters is found in the urban populations of the Isle of Man, a finding which contrasts with other studies, in which the urban populations exhibited a lower frequency of non - tasters than the surrounding rural populations.

Though none of the differences between Northside and Southside populations reached the level of significance, it is noteworthy that quite often the largest difference in the frequency of phenotypes and/or genes of a particular system lay between these two regions. The author feels that it is necessary to collect further data for many of the genetic traits reported upon in this study to confirm or otherwise the genetic homogeneity of the indigenous Manx population.

However, the evidence supplied by the present study leads to the conclusion that the indigenous Manx population comprises a genetically homogeneous group. Any heterogeneity that may have existed within this population in earlier times has been severely diminished by the influences of hybridization, internal migration patterns and possible emigration of specific groups of the population in more recent centuries. Those physical differences, such as stature and weight, which are more dependent on environmental than genetic influences, may still perhaps exhibit similar variability among the Manx regions as that reported in the earlier studies of 1898 and 1936.

CHAPTER FIVE

COMPARISON OF THE DATA COLLECTED IN THE PRESENT STUDY WITH MATERIAL FROM SELECTED BRITISH, IRISH AND EUROPEAN POPULATIONS

Comparison of the data collected in the Present Study with material from selected British, Irish and European Populations.

The availability of data on genetic systems from the regions of the British Isles depends, as one might expect, very much upon which trait is under consideration. The long established and well investigated genetic factors such as the ABO and Rh(D) blood group antigens, have been reported for numerous British and Irish regional populations (Mourant 1954, Mourant et al. 1958, Dawson 1964 and Kopeć 1970). However, information on the regional distribution of some of the red blood cell isoenzyme and serum protein polymorphisms, which have been discovered since 1955, were not available. Therefore, according to availability, comparative data on genetic factors have been employed drawn from populations as near to the Isle of Man as Ulster and North West England, and as distant as Norway and Iceland. A recent publication "Genetic Variation in Britain" (ed. Roberts and Sunderland 1973) is a valuable source of data, although it is still true that there is a marked shortage of data generally in this region with the exception of ABO and Rh(D) blood groups.

1. Serological Traits

a. Blood Group Antigens

1. ABO blood group system. Table 43.

a. Northern England

Table 43 includes the distribution of the ABO blood groups in selected population samples from Northern England. In chapter three it was shown that there are three possible representative samples of the ABO blood group distribution in the indigenous

Manx population, and all have been employed in the regional comparisons. They are the Manx donors, the Manx non - donors and the total Manx series.

Some of the results of Fraser Roberts' (1953) investigation into possible regional differences in the frequency of ABO blood groups in Northern England are shown in Table 43. The Manx donors are found to exhibit an ABO distribution similar to that found in all his three series, North - West Cumbria, South Cumbria and Total Cumbria. However, the Manx non - donor series showed significant differences from all three series, but markedly so from the North - West Cumbrians.

Manx non-donors v N - W. Cumbria $X_2^2 = 25.4676$, $P < .001$

Manx non-donors v S. Cumbria $X_2^2 = 7.1500$, $.05 < P > .025$

Manx non-donors v Total Cumbria $X_2^2 = 19.6048$, $P < .001$

The total Manx series was found to differ significantly in the distribution of ABO groups from two of the Cumbrian samples, the North West Cumbrians, $X_2^2 = 18.5546$, $P < .001$

and the Total Cumbrians, $X_2^2 = 13.2622$, $.005 < P > .001$,

but not from the South Cumbrians.

The distribution of ABO groups reported by Kopeć (1970) for the same region, but based upon an independent set of data is also shown in Table 43. Kopeć's region incorporates unit - areas 59 - 72 inclusive of Map VI, Newcastle - upon - Tyne B.T.S. and unit area 1 of Map VII, Liverpool B.T.S. (Kopeć 1970).

The Manx donor and Total Manx samples exhibit no significant variation from this series, but the Manx non - donors show a significantly different distribution, $X_2^2 = 7.2313$ $.05 < P > .025$.

Depending upon which of the Manx samples one takes to be representative of the distribution of ABO groups in the indigenous Manx population, it is seen to be similar or different to samples of the Cumbrian resident population. It should be remembered that the indigenous Cumbrian sample collected during this study was found to exhibit a similar ABO distribution to that found in the Manx samples with the exception of the Manx donors.

The frequencies of ABO phenotypes in resident blood donors of the area known as Furness, Final Area 6 in Kopeć (1970), are also included in Table 43. The proportions of the ABO groups are found to be similar between the Furness and Manx donor series, and between the Furness and Total Manx sample. However, the Furness sample exhibits an ABO blood group distribution that is just significantly different from the Manx non - donors, $X_2^2 = 6.1081$, $.05 < P > .025$.

The distribution of the ABO blood groups in another of Kopeć's 39 Final Areas, Area 3, comprising Cumbria, West Durham, South West Northumberland and North West Yorkshire is shown in Table 43. The only observed heterogeneity when the Manx samples were compared with these data was between the Manx non - donors and the Kopeć series, $X_2^2 = 9.2631$, $.01 < P > .005$.

All three Manx samples exhibited an overall similar distribution of ABO groups to that found in Kopeć's Final Area 9 sample which comprises most of Lancashire, except Merseyside and Preston, and parts of Cheshire.

(b) Scotland

Table 43 also includes the ABO group distributions found in selected Scottish population samples. To the author's knowledge the only previous information on the ABO groups based on samples selected on the basis of grandparental birthplaces is that provided by Brown's (1965) study in the north of Scotland. Her samples included only those blood donors whose four grandparents were born in the five northernmost counties of Scotland. In the case of the Orkney and Shetland Isles samples, no data on the ancestry of the individuals were obtained, but she felt that a high proportion of these people would be native to the Islands.

Prior to Brown's (1965) study, Kirkpatrick (1952) had investigated the ABO frequencies in a large sample of the residents of North Scotland which are also included in Table 43. Northern Scotland, like the Isle of Man, experienced a strong and lasting Scandinavian influence but was under Norse sovereignty longer than the Isle of Man. The Total Manx sample differs significantly from all the northern Scottish samples mentioned above.

Total Manx v N. Scotland	(Kirkpatrick 1952)	$X^2_2 = 24.9819$	$P < .001$
Total Manx v N. Scotland	(Brown 1965)	$X^2_2 = 33.4619$	$P < .001$
Total Manx v Shetland Isles	(Brown 1965)	$X^2_2 = 8.4472$	$.025 < P > .01$
Total Manx v Orkney Isles	(Brown 1965)	$X^2_2 = 16.4922$	$P < .001$
Total Manx v Total N.Scotland	(Brown 1965)	$X^2_2 = 34.3140$	$P < .001$
Total Manx v Total N.Scotland	(Kirkpatrick 1952) (Brown 1965)	$X^2_2 = 30.5000$	$P < .001$

However, statistically significant variation between these northern Scottish series and the Manx donors is limited to that between the latter and the Orkney Isles, $X^2_2 = 10.3742$, $.01 < P > .005$.

The Manx non - donor series, like the Total Manx, exhibits very different ABO frequencies from those found in all the northern Scottish samples.

Manx non-donors v N.Scotland	(1952)	$X_2^2 = 28.4816$, $P < .001$
Manx non-donors v N.Scotland	(1965)	$X_2^2 = 40.9489$, $P < .001$
Manx non-donors v Shetland Isles	(1965)	$X_2^2 = 10.1678$, $.01 < P > .005$
Manx non-donors v Orkney Isles	(1965)	$X_2^2 = 16.0878$, $P < .001$
Manx non-donors v Total N.Scotland	(1965)	$X_2^2 = 40.7624$, $P < .001$
Manx non-donors v Total N.Scotland	(1952+1965)	$X_2^2 = 34.7624$, $P < .001$

The relatively high frequency of phenotypes B and AB in the Orkney and Shetland Isles accounts in some measure for the large chi - squared values when these Islands were compared with the Manx data. However the large differences between the mainland Scottish series and two of the Manx samples (Manx non - donors and Total Manx) were due to the differing proportions of phenotypes A and O in each sample.

The distribution of ABO blood groups in a sample of blood donors resident in South West Scotland reported by Kopeć (1970) are also included in Table 43. Kopeć's region incorporates unit areas 52, 53, 54, 56 and 57 of Map IV, Glasgow and West of Scotland B.T.S. (Kopeć 1970). The Manx donors exhibit a similar distribution of ABO groups to this sample, but the Total Manx, $X_2^2 = 10.8985$, $.005 < P > .001$ and the Manx non - donors, $X_2^2 = 12.8180$, $.005 < P > .001$ are found to be very different, especially with respect to the lower frequency of B and higher frequency of A in the latter two series.

The ABO group frequencies reported by Struthers (1951) in a sample of 6,000 donors drawn randomly from the Glasgow and West of

Scotland B.T.S. area are shown in Table 43. As found in Kopeć's series, the Manx donors exhibit a similar ABO distribution; however the other two Manx series are significantly different from the above sample.

Total Manx v Struthers (1951) $X_2^2 = 22.5986$ $P < .001$

Manx non-donors v Struthers (1951) $X_2^2 = 27.4203$ $P < .001$

(c) North Wales

In his survey of the distribution of ABO blood groups in blood donors resident in North Wales (i.e. the counties of Caernarvonshire, Denbighshire and Flintshire), Fraser Roberts (1942) subdivided his sample into those with and without Welsh surnames, in an attempt to obtain a more 'indigenous' sample. Phenotype O was found to have a greater incidence in the Welsh surname sample than in the total sample, coupled with a reciprocal fall in the frequency of A.

The Manx donor and Total Manx series exhibit overall similarity in ABO group distribution to the total North Welsh sample. However, the Manx non - donors exhibit significant heterogeneity from the same sample, $X_2^2 = 8.2341$, $.025 < P > .01$. When the Welsh surname sample is compared with the three Manx series only the Manx donors are found to have a similar distribution of ABO groups. The Total Manx, $X_2^2 = 12.2821$, $.005 < P > .001$, and the Manx non - donors, $X_2^2 = 17.1902$, $P < .001$, exhibit highly significant differences from the Welsh surnames sample. The large chi - squared values are a product of the discrepancies in the proportions of O and A in the respective samples.

Kopeć (1970) reported the ABO blood group distributions in blood donors resident in a similar area of North Wales as that covered by Fraser Roberts' (1942) survey. Kopeć's region incorporated unit areas 51 - 65 inclusive of Map VII, Liverpool B.T.S. (Kopeć 1970). The Total Manx, $X_2^2 = 9.9616$, $.01 < P > .005$, and the Manx non - donors, $X_2^2 = 14.1020$, $P < .001$, were again found to differ significantly from the Welsh with respect to ABO groups, while the Manx donors are again seen to show no differences

of significance. Again the large chi - squared values are in large part accounted for by the fluctuations in groups O and A.

The distribution of the ABO phenotypes in Kopeć's Final Area 13 (Kopeć 1970) which comprises all of north and central Wales is also shown in Table 43. Not surprisingly, similar results to those found with Kopeć's other Welsh series are observed. The Total Manx, $X_2^2 = 13.6659$, $.005 < P > .001$, and the Manx non - donors, $X_2^2 = 17.1718$, $P < .001$, again exhibit significant variation in the proportion of ABO groups, while the Manx donors show overall similarity to this Welsh sample.

(d) Ireland

(i) Ulster

The ABO blood group distributions in three Ulster population samples reported upon by Hart (1944), Hackett and Dawson (1958) and Kopeć (1970) respectively, are shown in Table 43. Hart (1944) claimed that his analysis of the ABO blood groups "reflected the complex origin of the modern population of Northern Ireland." However the area from which his sample was taken was very restricted, with even Belfast being excluded from his survey.

Hackett and Dawson's (1958) Ulster or 'Six - Counties' series referred to individuals born in Northern Ireland who volunteered as blood donors in the Republic of Ireland (Eire) and is, therefore, biased towards inclusion of those northerners who have connections with the Republic. Therefore this sample also cannot be taken as truly representative of the whole Ulster population.

Kopeć's (1970) data were based on all donors in Ulster which of course results in a very large series indeed. No account was taken of the religion of the donor, even though it is perhaps true that Protestants are more preponderant as blood donors in Ulster than as members of the community as a whole.

The Manx donors exhibit a similar distribution of ABO groups to those found in all three Ulster series, but the differences, as measured by the chi - squared test, between the same Ulster series and the Manx non - donor and Total Manx series are highly significant.

Manx non - donors v Hart (1944) $X^2_2=30.9956$ $P < .001$

Manx non - donors v Hackett and Dawson (1958) $X^2_2=48.0049$ $P < .001$

Manx non - donors v Kopeć (1970) $X^2_2=41.5699$ $P < .001$

Total Manx v Hart (1944) $X_2^2 = 25.3270$ $P < .001$

Total Manx v Hackett and Dawson (1958) $X_2^2 = 40.9611$ $P < .001$

Total Manx v Kopeć (1970) $X_2^2 = 34.8728$ $P < .001$

The large differences observed are due chiefly to the low frequency of A and higher incidence of O in the Ulster samples. It is of interest to note that the sample of Ulster born individuals (Hackett and Dawson 1958), bearing in mind the other reservations mentioned in connection with this series, is more different from the two Manx series than the residence only selected series.

(ii) Dublin

The ABO blood group distributions in three Dublin population samples are also shown in Table 43, two samples based upon selection by birthplace of the individual donor (Dawson and Hackett 1958 and Dawson 1964) and one based upon selection by residence only (Dawson 1952).

Whereas the Manx donors exhibit overall similarity in ABO groups to the Dublin samples, the Manx non - donors and Total Manx samples show very significant differences from the Dublin population.

Manx non - donors v Dawson (1952) $X_2^2 = 43.8340$, $P < .001$

Manx non - donors v Dawson and Hackett (1958) $X_2^2 = 37.4293$, $P < .001$

Manx non - donors v Dawson (1964) $X_2^2 = 49.1363$, $P < .001$

Total Manx v Dawson (1952) $X_2^2 = 38.9082$, $P < .001$

Total Manx v Dawson and Hackett (1958) $X_2^2 = 31.9382$, $P < .001$

Total Manx v Dawson (1964) $X_2^2 = 43.9654$, $P < .001$

The explanation of the large chi - squared values is the same as that given when the Ulster series were compared with the

Manx; a higher incidence of phenotype O and lower frequency of A in the Dublin series. Again it is of interest to note that one of the 'native' Dublin samples (Dawson 1964) exhibits the greatest divergence from the Manx series in the distribution of ABO groups.

(iii) Eire

The distributions of ABO blood groups found in Leinster, as well as three seaboard counties within this province, Louth, Wicklow and Wexford, are summarized in Table 43. (Dawson 1964) All donors included in these samples were born in the specified county or province. Hooper (1947) stated that "Leinster being the bridgehead for nearly seven centuries of British colonization shows a lower percentage of O and a higher percentage of A than the other provinces of Eire, and therefore a significantly higher A:A+O index." Dawson's data exhibits similar findings.

The Manx donors are once again found to exhibit ABO phenotype frequencies consistent with those found in Leinster. However, the Total Manx sample differs highly significantly from all the Eire samples.

Total Manx v Louth $X_2^2 = 52.1451$, $P < .001$

Total Manx v Wicklow $X_2^2 = 18.5292$, $P < .001$

Total Manx v Wexford $X_2^2 = 22.9051$, $P < .001$

Total Manx v Leinster $X_2^2 = 48.6767$, $P < .001$

The Manx non - donors are found to exhibit even greater divergence from the Eire samples shown in Table 43.

Manx non - donors v Louth $X_2^2 = 58.4577$, $P < .001$

Manx non - donors v Wicklow $X_2^2 = 23.4783$, $P < .001$

Manx non - donors v Wexford $X_2^2 = 29.2168$, $P < .001$

Manx non - donors v Leinster $X_2^2 = 53.4876$, $P < .001$

The higher frequencies of phenotypes O and B, coupled with the lower incidence of A in the Eire series, compared with the two Manx series produce the large chi - squared values.

Sunderland et al. (1973) reported the ABO gene frequencies in a sample of the indigenous population of Carnew, a village in Co. Wicklow. The frequency of the r gene was 0.734, very similar to that found in the Manx donors. (Table 1)

Secretor Groups. Table 44.

Table 44 presents the distribution of secretor groups and respective allele frequencies in selected British, Irish and Icelandic population samples. The most striking feature of the distribution is the higher incidence, more than 0.5, of gene se in the populations in the north of the British Isles (i.e. the samples from Belfast I 0.5153, Isle of Man 0.5370, Cumbria 0.5376, Aberdeen 0.5459, Belfast II 0.5484 and Dublin 0.5673) compared with its lower frequency in samples from the rest of Britain (Liverpool 0.4767, London 0.4929 and the general English series 0.4922). When these two groups of population samples are compared with respect to the frequency of secretor groups, the heterogeneity between them is found to be statistically highly significant, $X_1^2 = 23.9489$, $P < .001$. Could it be that a higher incidence of the se gene, and therefore a lower frequency of secretors is a characteristic of the 'Celtic' populations of Britain in contrast to a lower frequency of the gene in southern Britain? This hypothesis gains further support when the high frequency (0.6421) of the se gene in the Icelandic sample of Bjarnason et al. (1973) is noted.

In the British and Irish population samples included in Table 44, the frequency of se allele exhibits a variability of 9%, rising from 0.4767 in Liverpool (M^C Connell in Race and Sanger 1970) to 0.5673 in Dublin (Lincoln and Dodd 1973).

2. MNSs blood group system. Tables 45 and 46.

(a) MN groups.

Data on the distribution of the MN blood groups and respective allele frequencies in selected British and Irish population samples are summarized in Table 45. The indigenous Manx sample is found to exhibit MN phenotype frequencies consistent with those found in the three English series (Ikin et al 1952, Cleghorn 1960 and Race and Sanger 1970), the Scottish, Welsh and Northern Irish resident samples of Ikin et al. (1952) as well as the present indigenous series from Cumbria and South West Scotland. The overall similarity of these samples is seen to be even closer when the distribution of the two genes is compared. The frequency of the M gene varies from 0.5290 in the Manx to 0.5582 in the Scots.

Whereas the Manx exhibit no variation of statistical significance in MN phenotype frequencies from one of the Eire samples, (Hackett and Dawson 1958) they do from the Eire series investigated by Palsson et al. (1970), $X_2^2 = 25.6247$ $P < .001$. The difference is due in large part to the lower frequency of MN and higher incidence of M groups in the latter sample. The difference between the two series is also significant with respect to genes, $X_1^2 = 12.9171$ $P < .001$.

It appears that Ireland generally exhibits a slightly higher frequency of the M gene (around 0.60) than other parts of the British Isles. Sunderland et al. (1973) reported widely fluctuating frequencies for the M gene, 0.501 in Co. Cork, 0.621 in Co. Wicklow and a frequency of 0.561 in their total Irish sample. The small Ulster sample exhibits a frequency of 0.6037 for gene M and

Palsson's Eire series shows an incidence of 0.6169. The smaller Eire series of Hackett and Dawson (1958) has a similar frequency for the M gene (0.6169). All three Irish samples (Ikin et al. 1952, Hackett and Dawson 1958 and Palsson et al. 1970) combined are found to differ significantly from the indigenous Manx, $X_1^2 = 15.8729$ $P < .001$ with respect to the proportion of genes.

(b) MNSs groups.

Table 46 summarizes the distribution of the MNSs blood groups and respective gene complex frequencies in selected British and Irish populations. To the author's knowledge there has been no detailed regional investigation of the MNSs blood group distributions in the British Isles, with the exception of the Black Mountain, Carmarthenshire survey of Garlick and Pantin (1957) which unfortunately has little relevance to this particular study.

After statistical analysis the indigenous Manx are found to exhibit significant variation from the three general resident English series, as well as the present indigenous Cumbrian sample.

Manx v English (Race and Sanger 1970) $X^2_5 = 28.2191$ $P < .001$

Manx v English (Ikin et al 1952) $X^2_5 = 13.3124$ $.025 < P > .01$

Manx v English (Cleghorn 1960) $X^2_5 = 14.7710$ $.025 < P > .01$

Manx v Cumbrians (Present Study) $X^2_5 = 14.6219$ $.025 < P > .01$

It is the lower incidence of MMS and the higher frequency of MNS and MNss in the Manx compared to the other four samples that contribute largely to the differences noted between them.

The Manx are also found to be very different from the two series reported for Eire in the proportion of MNSs phenotypes, but especially so from the sample of Palsson et al. (1970).

Manx v Eire (Hackett and Dawson 1958) $X^2_5 = 20.2958$ $.005 < P > .001$

Manx v Eire (Palsson et al. 1970) $X^2_5 = 40.1274$ $P < .001$

Once again it is the excess of phenotypes MNS and MNss and NNss and a marked deficit of MMS in the Manx compared to the Eire series that accounts for the large chi - squared values. Whereas the Manx exhibit MNSs phenotype frequencies consistent with those found in the Welsh series of Ikin et al. (1952) they are significantly

different from the Scots, $X^2_5 = 12.9159$ $.025 < P > .01$ and even more distinct from the Northern Irish, $X^2_5 = 15.9762$ $.01 < P > .005$, reported by the same authors. These differences are accounted for by the generally lower incidence of phenotypes MMS and NNS and the higher frequency of MNS and MNss in the Manx, compared with the other populations.

The differences noted in the phenotype distributions between the Manx and some of the other populations included in Table 46 are reflected in the frequencies of the gene complexes. With the exception of the Northern Irish, the Manx exhibit the lowest frequency of MS and gene S, 0.2712, found in any British Isles' population. It would be very interesting to determine whether the total Manx population exhibits this relatively low frequency of gene S, or whether it is purely a characteristic of the indigenous population. Whereas the Manx exhibit a slight excess of the Ms gene compared with the English and Scottish populations, they show a lower frequency than found in Ulster and most of Eire. The NS gene has a similar incidence in the Manx, English and Welsh populations, but a lower value in the Scottish and Irish samples included in Table 46. The Manx have a similar frequency of the Ns gene to that found in the English and Scots but higher than that found in the Welsh and Scots.

3. P blood group system. Table 47.

The distribution of P blood groups and respective gene frequencies in selected British and Irish populations are summarized in Table 47. The indigenous Manx sample is found to be similar, using the chi - squared test, to all the English series and the Welsh, Scottish and Northern Irish populations.

The larger Eire sample (Palsson et al. 1970) is found to differ significantly from the other Irish series, including Ulster, in the frequency of P groups. Whereas the Manx have P blood group frequencies consistent with those found in the Irish series of Hackett and Dawson (1958) they are very different from those in Palsson's sample, $X_1^2 = 50.6145$, $P < .001$, which exhibits a very low frequency of P_1+ individuals. Evidence supporting the view that the former, smaller of the two samples exhibits a more likely P group distribution in Eire, comes from data reported by Sunderland et al. (1973) on indigenous Irish populations. They reported frequencies for the P_1 gene of 0.493 in Carnew, Co. Wicklow and 0.406 in Rossmore, Co. Cork. In no part of Eire did they find a P_1 allele frequency of less than 0.30, and in a total Irish sample of over 2,000 persons the gene frequency was 0.481.

It should be remembered that variation in P blood group distributions can often be a result of delay in testing specimens as well as varying quality of antisera.

The frequency of the P_1 gene in the populations shown in Table 47 rises from 0.2589 in the Eire series of Palsson et al. (1970), an exceptionally low frequency, to a maximum of 0.5342 in neighbouring Ulster. (Ikin et al. 1952). That Palsson's Eire sample lies very much at one end of the range of P_1 gene variation, is shown by the fact that the Manx sample, closest to the Eire series, displays a frequency of 0.4796 for the P_1 gene.

4. Rh blood group system. (Tables 48 - 50)

The distribution of Rh. blood groups in selected populations of the British Isles is summarized in Tables 48 - 50. The indigenous Manx are found to exhibit significant variation from the general English series cited by Race and Sanger (1954), $X_6^2 = 16.5581$ $.025 < P > .01$, with respect to common Rh. types (Table 49a). In the 7 x 2 contingency table the Rh type groupings employed were as follows ;

R_1r, R_1^wr ;

R_1R_1, $R_1^wR_1$;

R_1R_2, $R_1^wR_2$;

R_2r ;

R_2R_2 ;

rr ;

R_1R_Z, R_or, rr', rr'';

The lower incidence of R_1R_1 and higher frequency of R_2R_2 and rr in the Manx largely accounts for the high chi - squared value.

Table 48b shows the observed number and frequency of Rh types in Manx and selected British and Irish population samples tested with four antisera, anti - D, -C, -E and c. Using the chi - squared test the indigenous Manx exhibit significant variation from each of the four series, but especially from the two English samples. 6 x 2 contingency tables, identical to those employed in chapters 3 and 4, were used in this analysis. The Manx natives are most different from Murray's (1946) general English series, $X_5^2 = 19.6330$ $.005 < P > .001$, but are also highly significantly different from Fisher and Race's (1946) English sample, $X_5^2 = 18.4114$ $.005 < P > .001$. The largest contributing

factor to the observed heterogeneity is the deficiency of CCDee and the excess of ccDE and ccddee phenotypes in the Manx.

The Manx also exhibit a distribution of Rh. types that is just statistically significantly different from the Irish reported by Huth (1953), $X^2_5 = 11.4603$ $.05 < P > .025$. The deficiency of CCDee and excess of ccDE and CcDE types in the Manx produces the largest contribution to the differences noted between the two populations. The Manx natives are even more distinct from the Irish series of Palsson et al. (1970), $X^2_5 = 18.1033$ $.005 < P > .001$. The differing proportions of Rh. Types, CCDee, CcDee and ccDee provide the largest component in the chi - squared value.

The frequency distributions of the Rh. gene complexes reflect some of the variation exhibited by the distribution of Rh types in the populations of the British Isles. In those populations tested with at least five antisera, anti - D, -C, -E, -c and e, included in Table 49, gene complex R_1 (CDe) is found to rise in incidence from around 0.36 in the Manx and south - west Scots, to 0.39 in the general Irish population, and a maximum of over 0.40 in the Cumbrians and English generally. It should be noted that in certain parts of Eire, such as Co. Wicklow and Co. Cork, there is a higher frequency than 0.39 for the R_1 gene.

With the exception of some small Irish population samples, such as those of Co. Cork and Co. Wicklow, gene complex r (cde) exhibits its lowest incidence (0.39) in the general English sample of Race and Sanger (1950), while the other samples show frequencies lying between 0.42 and 0.44. It is interesting to note that only the English sample exhibits a greater frequency of R_1 than r; all the other samples tested with five antisera show a frequency of r exceeding that of R_1.

The R_2 (cDE) gene complex increases in frequency from 0.12 in Cumbria, through 0.14 - 0.15 in the English and Irish populations to a maximum of over 0.16 in the indigenous Manx. However, two relatively small samples, Co. Wicklow and Co. Cork in Eire, exhibit an even higher frequency, 0.172 and 0.204 respectively, for this gene complex.

Owing to the work of the National Blood Transfusion Service (N.B.T.S.) centres throughout the United Kingdom, there are far more data available on the frequency distributions of Rh(D) groups than on the full Rh types mentioned above. This store of data has been used by many authors, but most thoroughly of all by Kopeć (1970). Table 50 summarizes the distribution of Rh(D) groups in selected populations of the British Isles.

The indigenous Manx exhibit a frequency of Rh(D) negatives consistent with those found in the Cumbrian, Furness and South West Scottish resident samples,(Kopeć 1970), and in the indigenous populations of North Scotland, and the Orkney and Shetland Isles (Brown 1965). The Manx are also found to be similar to the North Welsh and Pembrokeshire populations (Kopeć 1970), but different from the general Welsh series reported by Hoare (1943), $X_1^2 = 6.2911$ $.025 < P > .01$. The two Dublin series, with lower frequencies of Rh(D) negative, significantly differ from the Manx.

Manx v Dublin (Stewart 1947) $X_1^2 = 6.0888$ $.025 < P > .01$

Manx v Dublin (Dawson and Hackett 1958) $X_1^2 = 4.3433$ $.05 < P > .025$

Also, because of the lower incidence of Rh(D) negative, the very large Ulster series (Kopeć 1970) shows variation of statistical significance from the Manx, $X_1^2 = 4.5201$ $.05 < P > .025$. However, the Manx exhibit a similar frequency of Rh negatives as found in the Leinster population (Dawson 1964).

5. Lutheran blood group system. Table 51.

The distribution of Lutheran phenotypes and respective allele frequencies in selected British and Irish populations are summarized in Table 51. The indigenous Manx exhibit the highest frequency of the Lu^a gene, 0.0573, yet reported in a British population. The variation in Lutheran groups between the Manx and the two English series included in Table 51 is statistically significant;

Manx v Race and Sanger (1970) $X_1^2 = 4.2320$, $.05 < P > .025$

Manx v Ikin et al. (1952) $X_1^2 = 9.9364$ $.005 < P > .001$

The Manx are found to be even more different from the Welsh (Ikin et al. 1952) $X_1^2 = 11.6322$, $P < .001$, and also the Scots (Ikin et al. 1952) $X_1^2 = 9.1550$, $.005 < P > .001$, with respect to Lutheran groups. Whereas the small Ulster series of Ikin et al. (1952) exhibits Lutheran phenotypes consistent with those found in the Manx, the even smaller sample for Eire (Hackett and Dawson 1958), with its very low frequency for the Lu^a gene of 0.0106, is very different $X_1^2 = 7.3103$, $.01 < P > .005$. The low incidence of the Lu^a gene in Ireland is confirmed by the data of Sunderland et al (1973) on selected populations in Eire. They found a frequency for the Lu^a gene of 0.012 in Carnew, Co. Wicklow, 0.019 in Rossmore, Co. Cork and 0.019 in a total Eire sample of over 2,000 persons.

6. Kell blood group system. Table 52

The distribution of Kell blood groups and the respective gene frequencies in selected British and Irish populations are summarized in Table 52. No significant heterogeneity was found between the Manx and any of the five English samples with respect to Kell groups. The Manx also displayed a similar distribution of Kell phenotypes to that found in the Scottish, Welsh and Ulster samples reported by Ikin et al. (1952).

As with the P blood groups the two Eire samples also differ markedly in the frequency of Kell groups; the Manx exhibiting similarity to the larger sample of Palsson et al. (1970) but differing just significantly from Hackett and Dawson's (1958) series, $X_1^2 = 3.9342$, $.05 < P > .025$, owing to the higher incidence of Kell positives in the latter. A high frequency of Kell positives in Eire populations is not unknown for Casey et al. (1963) reported a frequency of 24% in the native inhabitants of the Slieve Lougher district of South West Ireland. In a later survey of the same area Casey et al. (1969) reported frequencies of Kell positives ranging between 9.2% and 31.0%. These values are among the highest reported in the world. Palsson's sample, being larger in size, perhaps more reliability can be placed upon these figures as being representative of the Eire population. Evidence supporting this view is provided by the data of Sunderland et al. (1973). They found frequencies for the K gene of 0.051 in Co. Wicklow, 0.067 in Co. Cork and 0.044 in the total Irish sample. None of their figures approach 0.089 reported by Hackett and Dawson (1958).

Therefore, with the exception of some Irish groups, the frequency of the K gene lies between 0.035 and 0.060 in populations of the British Isles shown in Table 52.

Penney (Kp^a) blood groups. Table 53

The distribution of Penney groups and calculated allele frequencies in selected populations are shown in Table 53. Apart from the present series, the only other British figures on Kp^a groups are those reported by Cleghorn (1961) cited in Race and Sanger (1970). Though the Manx natives exhibit a higher frequency of Kp(a+) than her series, the difference does not approach the level of statistical significance.

The distribution of Kp^a groups in other Caucasoid populations is also included in Table 53. (Race and Sanger 1970). No significant heterogeneity can be demonstrated between the Manx and any of these samples with the exception of the French, $X_1^2 = 8.8191$, $.005 < P > .001$. The indigenous Manx are seen to have the highest frequency of the Kp^a gene in the world.

7. Duffy blood group system. Table 54.

Table 54 summarizes the distribution of Duffy blood groups and respective gene frequencies in selected British and Irish populations. No significant heterogeneity with respect to Duffy phenotypes or genes could be demonstrated between the Manx and the general English resident sample of Race and Sanger (1970), tested with both anti - Fy^a and anti - Fy^b sera. The Fy^a allele is found to have a similar frequency in both samples, 0.43.

The other population samples included in Table 54 have been analysed only using anti - Fy^a serum, but once again no differences of significance are found between the Manx and either of the English resident series, and the Manx also exhibit Duffy phenotype frequencies consistent with those found in the Scottish, Welsh and Northern Irish samples reported by Ikin et al. (1952). The distribution of Duffy phenotypes is reported in two Eire samples, but, as found with the P and Kell blood groups, they are also significantly different from each other. Whereas the Manx exhibit a distribution of Duffy phenotypes and genes consistent with that found in the smaller Eire sample of Hackett and Dawson (1958) they show significant heterogeneity from the series reported by Palsson et al. (1970), $X_1^2 = 10.3435$, $.005 < P > .001$, which displays a frequency of Fy(a+) of only 51%. The frequencies of gene Fy^a reported by Sunderland et al. (1973) in selected Irish populations, including 0.363 in Carnew, Co. Wicklow, 0.397 in Co. Wicklow, 0.400 in Co. Cork and 0.416 in the total sample of over 2,000 persons, agree much more closely with the frequency found in Hackett and Dawson's sample (0.3845) than in Palsson's series (0.304).

The frequency of the Fy^a gene exhibits a range of variability or nearly 15% among the samples included in Table 54, rising from 0.3038 in Eire to 0.4507 in Wales. However, the majority of samples have a frequency of the gene somewhere between 0.37 and 0.43.

(b) Serum Proteins.

(i) Haptoglobin (Hp) Table 55.

Owing to their relatively recent discovery compared with some of the blood group antigens, there have been fewer regional as well as national studies of the distribution of the serum protein and red cell isoenzyme polymorphisms in the British Isles. Accordingly data for many of these genetic factors from selected European as well as British and Irish populations have been included in the comparisons.

Table 55 summarizes the distribution of Hp groups and respective allele frequencies in selected British, Irish and North European population samples. The Manx sample exhibits Hp phenotype and gene distributions consistent with those found in the North - East English sample of Papiha (1974) which comprises blood donors born in the region. Cartwright's (1973b)sample consists of students attending the University of Durham who have both parents born in the north of England, defined generally as that area of England north of the River Trent. The Manx are similar to this group with respect to Hp. phenotypes but are just significantly different from them in the proportion of the two genes, the Manx exhibiting a lower frequency of the Hp^1 gene, $X_1^2 = 4.3074$ $.05 < P > .025$.

The Manx in fact are found to exhibit closest similarity to the Southern Scottish series of Kamel et al. (1963), the Irish series of Palsson et al. (1970) and the present Cumbrian sample. The frequency distributions of the Hp genes reported by Sunderland et al. (1973) in Eire show considerable variation, with Hp^1 having an incidence of 0.311 in Co. Roscommon, 0.335 in Co. Cork, 0.424 in Co. Wicklow and 0.380 in the total sample comprising more than

2,000 persons.

Could it be, as suggested earlier for the distribution of secretor groups, that two distinct population groups, North and South Britain, also exist with respect to the distribution of Hp groups? Based on data included in Table 55 it is suggested that a lower frequency of the Hp^1 gene (0.34 - 0.38) is characteristic of the areas known geographically and historically as the 'Celtic fringe' of Britain, as distinct from its higher incidence (0.38 - 0.41) in the largely non - Celtic population of Britain south - east of a line drawn from the River Tweed to the River Severn. When the amalgamated population samples for the Isle of Man, Cumbria, South - West Scotland, Central and South - West Scotland and Eire are compared with the large sample resulting from the pooling of the series from North - East England, North England and England, the heterogeneity existing between them is found to be significant with respect to Hp genes, $X_1^2 = 4.4258$ $.05 < P > .025$ and common phenotypes $X_2^2 = 12.3395$ $.005 < P > .001$.

Another possible explanation of the differences observed in the Hp gene distribution is that there may exist a number of gradients throughout the length of the British Isles, subdividing the population into distinct groups, similar to those reported for the ABO blood group distributions in the United Kingdom (Fraser - Roberts 1952 and 1953).

Regarding the continental European samples included in Table 55, the Manx are found to exhibit significant differences compared with the Icelanders of Beckman and Johannsson (1967) with respect to phenotypes $X_2^2=6.6517$ $.05 < P > .025$ and more so in the proportion of common genes, $X_1^2 = 6.9139$ $.01 < P > .005$.

The Manx are also different from the Danish sample of Galatius - Jensen (1958) in the proportion of phenotypes, $X_2^2 = 7.3389$ $.05 < P > .025$. However the Manx natives exhibit Hp phenotype and allele frequencies consistent with those reported for Norwegians (Fleischer and Lundevall 1957) and Swedes (Höglund et al. 1970).

(ii) Transferrin (Tf) Table 56.

Owing to the fact that the frequency of Tf variants, other than type Tf C, found in any Caucasoid population is low, usually only 1% or 2%, little work has been carried out on the distribution of Tf groups on a regional or national scale. The distribution of Tf groups and the respective allele frequencies in selected British, Irish and European populations are shown in Table 56.

Though the Manx natives exhibit the highest frequency of Tf BC, there is a striking homogeneity in Tf groups among all the samples. Sunderland et al. (1973) investigating the frequency of the Tf^B allele in selected Irish groups found that in a total sample of over 2,000 it had an incidence of 0.01.

(iii) Beta - lipoprotein allotype - Ag system. Table 57.

Table 57 summarizes data on the distribution of Ag(x) groups and the respective allele frequencies in selected British and European populations. Whenever a study reported results for antigens in addition to Ag^x, these were modified accordingly for inclusion in Table 57. Though the frequency of Ag (x+) is higher in the English series of Bradbrook et al. (1971) than in the Manx, the difference is not statistically significant. There is also no discernible heterogeneity between the Manx and any of the European series included in Table 57. The indigenous Manx population in fact exhibits the lowest frequency (0.1725) of the Ag^x allele yet reported in a European population. If the very small Finnish series of Hirschfeld and Okochi (1967) is excluded, the frequency of the Ag^x allele in the population samples shown in Table 57 fluctuates between 0.1725 in the Manx and 0.2500 in the Icelandic series reported by Persson and Swan (1971).

(c) Red Blood Cell Isoenzymes.

(i) Acid phosphatase (AP) Table 58.

Table 58 summarizes data on the distribution of AP groups and respective allele frequencies in selected British, Irish and north - west European populations. The indigenous Manx are similar to the Northumbrian series of Papiha (1973) which comprises donors born in this region, with respect to both allele and phenotype frequencies. The frequencies of the three alleles in the Scottish sample of Renwick (1972) are consistent with those found in the Manx series. Whereas the Manx exhibit AP gene frequencies similar to those found in the general English population (Hopkinson et al. 1964) they are different from the same sample in the proportion of common phenotypes, $X^2_3 = 9.4160$ $.025 < P > .01$, (phenotypes C, CA and CB were amalgamated for statistical purposes). The greatest difference between the two samples was found in the case of phenotype BA which exhibited variability of nearly 10%.

When the Manx are compared with the Eire series of Palsson et al. (1970) they are found to show significant heterogeneity in the distribution of genes, $X^2_2 = 10.7393$ $.005 < P > .001$, and phenotypes $X^2_3 = 11.7270$ $.01 < P > .005$. It is the raised incidence of P^b and phenotype B and the lower frequency of P^a and phenotype BA in the Irish that accounts for the high chi - squared value. However Sunderland et al. (1973) report much lower P^b frequencies in selected Irish groups, 0.615 in Co. Wicklow, 0.618 in Co. Cork, 0.659 in Co. Roscommon and 0.618 in their total Irish sample. These frequencies are more similar to those found in the Manx and English populations. Once again it appears that the Irish sample

of Palsson et al. (1970) exhibits frequencies for a genetic polymorphism which lie at one extreme of the range of variation.

With respect to the continental European populations included in Table 58, the Manx are found to exhibit both AP phenotype and allele frequencies consistent with those found in the Icelandic and Danish population samples. The allele frequencies shown for the Norwegian, Swedish and French population samples also fall within the values found in other European populations, including the Isle of Man. It is of interest to note that with the exception of Cumbria, the frequency of P^C rises with increasing latitude to reach a maximum value of 0.0829 in Iceland (Bjarnason et al. 1973).

(ii) Phosphoglucomutase locus 1 (PGM_1) Table 59

The distribution of PGM_1 groups and respective allele frequencies in selected British, Irish and north European populations are summarized in Table 59. The frequencies of PGM_1 alleles and phenotypes in the indigenous Manx sample are consistent with those found in Papiha's (1973) series of locally born Northumbrians, and the two resident English series of Spencer et al. (1964) and Hopkinson and Harris (1966). The gene frequencies in the Manx are also consistent with those found in the Scots investigated by Renwick (1972).

However the Manx are very different from the Irish series of Palsson et al. (1970) both with respect to common PGM_1 genes, $X_1^2 = 12.4707$ $P < .001$, and phenotypes $X_1^2 = 16.9536$ $P < .001$. These differences are accounted for in large measure by the deficiency of PGM_1^2 (0.1368) and PGM 2 - 1 and the excess of PGM 1 in the Irish sample. Sunderland et al. (1973) reported much higher frequencies of PGM_1^2 in Irish populations, with incidences of 0.233 in Carnew, Co. Wicklow, 0.327 in Co. Wicklow generally, 0.278 in Co.Cork and 0.250 in their total Ireland sample. These frequencies are much more consistent with those found in the Manx sample, and perhaps reflect the general distribution of PGM_1 genes in Ireland.

Of the European population samples included in Table 59, the Manx show significant variation from the Danes (Lamm 1970a) in the proportion of genes $X_1^2 = 11.4388$ $P < .001$ and phenotypes $X_1^2 = 11.9793$ $P < .001$, and the Icelandic series of Mourant and Tills (1967) similarly, $X_1^2 = 5.9539$ $.025 < P > .01$ when the genes are compared and $X_1^2 = 6.9519$ $.01 < P > .005$ with respect to phenotypes.

However, the Manx population exhibits both genes and phenotypes in proportions consistent with those found in the Norwegians (Monn 1969) and the Swedes (Hansson 1971).

From Table 59 it is seen that England exhibits the highest frequency of PGM_1^2, 0.2618, while neighbouring Eire, if the series of Palsson et al. (1970) is taken as representative, shows the lowest incidence of the allele, 0.1368. All other north European samples shown in Table 59 exhibit a frequency of PGM_1^2 between these two figures.

(iii) Adenylate kinase (AK) Table 60.

Data on the distribution of AK types and the respective alleles in selected populations of the British Isles and north west Europe are summarized in Table 60. The indigenous Manx are found to be similar to the locally born Northumbrian series of Papiha (1973) and the English sample reported by Rapley et al. (1967) with respect to common phenotypes and genes.

Two samples are shown in Table 60 for the distribution of AK types in Eire, but as found for some blood groups, they are very different from each other. The difference in the frequency of the AK^2 allele between the two series approaches 10%. If the frequencies reported by Palsson et al. (1970) are taken as representative of the Eire population, then this population has the highest recorded incidence of AK^2 in Europe. However, partly because of its much larger size, it is more likely that the series reported by Tills et al. (1970) represents more correctly the general distribution of AK types in Ireland. Moreover, this series comprises specimens obtained from the Irish B.T.S. which covers most of Eire, whereas the smaller sample of Palsson et al. was drawn from selected areas of Eire. Furthermore, figures quoted by Sunderland et al. (1973) for AK gene frequencies in selected Eire groups support this viewpoint. The highest frequency of the AK^2 allele they found was 0.050 in Co. Wicklow, while the incidence was 0.035 in the total Irish sample. The Manx exhibit close similarity to the Irish series of Tills et al. (1970) and the gene frequencies reported by Sunderland et al. (1973), but they are very different from the Irish series of Palsson et al. (1970) with respect to genes, $X_1^2 = 27.0849$ $P < .001$ and phenotypes,

$X_1^2 = 15.9867$ $P < .001$.

Regarding the other European series included in Table 60, the Manx are found to exhibit gene and phenotype frequencies consistent with those found in all samples except the Icelanders (Tills 1970), $X_1^2 = 5.4309$ $.025 < P > .01$ (genes) and $X_1^2 = 5.9841$ $.025 < P > .01$(phenotypes), who show a higher incidence of AK^2 and phenotype 2-1.

If the exceptional figures reported for Eire by Palsson et al. (1970) are excluded, it is seen that the variation in AK^2 shown in Table 60 is in absolute terms very small, from 0.023 in Northumberland to 0.057 in Iceland, but in fact the frequency has more than doubled.

(iv) Adenosine deaminase (ADA) Table 61.

Table 61 presents data on the distribution of ADA groups and respective gene frequencies in selected British, Irish and north west European populations. The indigenous Manx show a striking similarity to the Northumberland sample of Papiha (1973) but exhibit significant heterogeneity from the relatively large English resident series of Hopkinson et al. (1969) in the proportion of genes, $X_1^2 = 6.6848$ $.01 < P > .005$ and common phenotypes, $X_1^2 = 6.8961$ $.01 < P > .005$.

The Manx also show no significant variation from the Irish series of Van den Branden et al. (1971), though the chi - squared value approaches the level of significance. Sunderland et al. (1973) reported frequencies of ADA^2 ranging between 0.056 in Co. Roscommon, 0.110 in Co. Cork and a value of 0.056 in the total Irish sample. It appears that there is considerable variation in the distribution of ADA genes in Ireland. The frequency of ADA^2 in an area close to the Isle of Man, Co. Wicklow, is 0.070, very similar to the frequency in the Manx.

There is no demonstrable significant heterogeneity between the Manx population and any European population group shown in Table 61, with respect to either genes or phenotypes.

Just as there was a suggestion of a division of the population of Britain into two distinct groups, North and South, with respect to secretor groups and Hp groups, or at least of gradients for the distribution of genes and phenotypes of these polymorphisms, it is also possible that a subdivision of the population occurs in the distribution of the ADA groups. When the population of North Britain shown in Table 61, defined as including

the Isle of Man, Cumbria, Northumberland, South West Scotland and Eire are compared with the large English series, drawn chiefly from Southern England, of Hopkinson et al. (1969), the difference is found to be statistically significant, both with respect to genes, $X_1^2 = 5.7752$ $.025 < P > .01$ and phenotypes, $X_1^2 = 4.974$ $.05 < P > .025$. If the Eire sample is excluded from the analysis, the difference between the two regions is found to be even more significant.

$$X_1^2 = 8.9362 \quad .005 < P > .001 \quad \text{(genes)}$$
$$X_1^2 = 9.5969 \quad .005 < P > .001 \quad \text{(phenotypes)}$$

(v) 6 - phosphogluconate dehydrogenase (6-PGD) Table 62.

Owing to the fact that electrophoretic variants of 6-PGD other than A are relatively rare, usually around 4%, it is unlikely that many studies of variation on a regional level will be carried out. However, data on the distribution of PGD types and the respective alleles in selected populations of the British Isles and Europe are summarized in Table 62.

Using the chi - squared test the Manx are found to exhibit PGD phenotypes and genes consistent with the proportions found in the locally born Northumberland series of Papiha (1973) and the two general English resident series of Fildes and Parr (1963) and Parr (1966). The Manx are also similar in PGD groups to the Irish series of Tills et al (1970), even though the frequency of PGD^C of 0.0139 is one of the lowest figures reported for a European population. Sunderland et al. (1973) also reported low frequencies, 0.006 in Rossmore, Co. Cork, 0.010 in Co. Cork generally and 0.015 in their total Irish sample. Interestingly the highest frequency of PGD^C, 0.044, is found in Co. Wicklow, that part of Eire close to the Isle of Man and England.

(2) NON - SEROLOGICAL TRAITS.

(i) Tongue Curling. Table 63.

Previous to the present survey the author was unable to find any data on the frequency of tongue - curlers in a population of the British Isles. However, the available data on the frequency of tongue - curlers in selected world populations are summarized in Table 63. In chapter 3 it was shown that there was a significant age difference in tongue curling in the Manx population, with the adults exhibiting a frequency of 63%, whereas the juveniles showed a much higher incidence, 72%. Accordingly each of the Manx series is compared with the samples included in Table 63.

The Manx juveniles are found to be just significantly different from the U.S.A. series of Sturtevant (1940) which comprises a 'wide varietyof races but mostly Americans of mixed European ancestry,' $X_1^2 = 4.0539$ $.05 < P > .025$, more so from the Chinese series of Liu and Hsu (1949), $X_1^2 = 11.6458$ $P < .001$, and even more different from the U.S.A. Negro sample of Lee (1955), $X_1^2 = 20.5791$ $P < .001$. However, the Manx juveniles are similar to the U.S.A. Whites (Urbanowski and Wilson 1947) and an 'Eastern U.S.A. population comprising Caucasoids of mixed European descent.' (Gahres 1952) with respect to the proportion of tongue - curlers.

The Manx adults exhibit significant heterogeneity when compared with the Eastern U.S.A. series (Gahres 1952), $X_1^2 = 10.6846$ $.005 < P > .001$ and are very different from the U.S. Negro sample of Lee, $X_1^2 = 50.5197$ $P < .001$, but are similar to the other three series in Table 63 with respect to the proportion of tongue - curlers.

(ii) Colour Vision Deficiency. Table 64.

Table 64 presents the regional distribution of colour defectives in selected male population samples of the British Isles, including the large study of Vernon and Straker (1943). The most convenient way of dealing with the comparisons is according to the method of selection of individuals. The samples can be divided into those in which the individuals tested had at least two parents born in the area specified, Table 64a, and those in which the individuals tested were chosen according to their place of residence, Table 64b.

No significant heterogeneity was demonstrated among any of the series in Table 64a, even though the frequency of colour - vision deficiency rises from 2.3% in Scotland to 9.6% in North Wales. It can be seen that the frequency of Manx colour - blind males fits in very nicely with the incidence recorded in the samples of British residents shown in Table 64b.

(iii) Phenylthiocarbamide (PTC) Tasting Ability. Table 65.

Fortunately for the genetic polymorphism PTC tasting there is a relative abundance of data on the regional distribution of taster phenotypes within the population of the British Isles. In addition many of the data are based upon the testing of native individuals, selected on the basis of grandparental and/or parental birthplaces. As mentioned before, the antimodal value in the Manx and Cumbrian series tested by the author was taken at solution number 4 (82.5 mgm/litre), and various papers by Sunderland and Cartwright (1966, 1967 and 1968) also employed the same antimode. In other studies, whenever the antimode has not been taken at solution 4, the frequency of non - tasters has been kept as determined by the respective author(s), such as Harris and Kalmus (1949) and Kitchin et al. (1959).

The most convenient method of handling the comparisons is according to the method of selection of persons for testing. Persons constituting the samples included in Table 65 include those with:-

(1) three or four grandparents born in the area specified.

(2) both parents born in the area specified

(3) residence in the area specified.

The number and frequency of non - tasters of PTC in those British and Irish samples based upon selection of persons having three or four grandparents born in the specified area are shown in Table 65a. The indigenous Manx sample exhibits non - tasting frequencies consistent with those found in the rural North Lancashire, Lancaster City and Derbyshire series reported by Cartwright and Sunderland (1967), as well as the three Irish samples drawn from the rural areas of Co. Wicklow, Co. Cork and

Co. Roscommon (Sunderland et al. 1973). However significant heterogeneity is observed between the Manx and the Barrow series of Mitchell and Swarbrick (1972), $X_1^2 = 5.7329$ $.025 < P > .01$, and between the Manx and Ulster populations (Maybin 1972), $X_1^2 = 3.9377$ $.05 < P > .025$. Both the Barrow and Ulster samples are small in size and exhibit an exceptionally low frequency of non - tasters of PTC for a British Isles population. It is possible that the differences noted, which are not large, are a product of small sample sizes.

However, the Manx population is very different from the relatively large North Welsh series of Fraser - Smith and Sunderland (1969), $X_1^2 = 9.4041$ $.005 < P > .001$, with respect to PTC tasting phenotypes. The Welsh series exhibits a frequency of non - tasters 7% lower than that found in the Manx population. In the samples included in Table 65a the frequency of non - tasters varies by some 24%, rising from 14% to 38%. However it should be borne in mind that many of the samples comprise fewer than 100 persons.

The observed number and frequency of non - tasters of PTC in those British population samples selected upon the basis of individuals having both parents born in the specified area are shown in Table 65b. The Manx population exhibits a similar proportion of non - tasters to that found in all the series except the Orcadians tested by Sunderland (1966) $X_1^2 = 11.4388$, $.001 < P > .0001$, who have an exceptionally high incidence of non - tasters. This is somewhat surprising considering that the two islands have experienced some similar historical influences. However the ABO blood group distributions also showed marked differences between the two populations.

The number and frequency of non - tasters of PTC in the British population samples, selected only on the basis of the person's residence in the specified region, are shown in Table 65c. All these investigations, including those mentioned above, employed the Harris - Kalmus (1949) testing procedure with its two - stage sorting technique. All five samples in Table 65c display overall homogeneity and the Manx exhibit a non - taster frequency consistent with those found in all series, but are most similar to the Liverpool series reported by Kitchin et al. (1959).

(IV) Reflectance Spectrophotometry of the Skin. Table 66.

The mean reflectance values at each of the three wavelengths and the standard deviations (S.D.) are set out for selected British, Irish and European populations according to sex, in Table 66. These mean values of reflectance constitute the basic data from which indices of overall lightness and darkness can be derived. The Manx, Cumbrian, Northumberland and Merthyr Tydfil population samples comprise schoolchildren whose ages vary between 11 and 18 years (The Northumberland series consists of 15 and 16 year olds only), whereas the other series represent older age groups. The extent to which the findings of the present survey should be questioned because these samples comprise individuals between 11 and 18 years of age, during which time there are probably great changes occurring that affect skin pigmentation, cannot be determined by the author. Differentiating the individuals according to individual years or age groups was not appropriate because of the relatively small size of the Manx and Cumbrian series. However it should be noted that differences of statistical significance are found between similarly aged samples, e.g. between the Merthyr Tydfil and Manx children, and between the Merthyr Tydfil and Cumbrian children (Smith and Mitchell 1973).

It is apparent from Table 66 that fair colouring appears to be a characteristic of the northern English, as evidenced by the Manx, Cumbrians and Northumbrians. Interestingly, all three areas lie on a similar latitude, 54°N - 55°N. The British population sample situated at the highest latitude, Northumberland (55°N), also exhibits the highest reflectance values at wavelengths 545mμ (605) and 685mμ (609) found in any population of the British Isles.

Also, the above three series comprise native inhabitants of the area specified, except the Northumbrian sample of Hulse (1973) in which only 60% of the individuals have four grandparents born in the English border counties.

The Manx and Cumbrian series of Smith and Mitchell (1973) in particular, exhibit very similar reflectance values at all three dominant wavelengths, and at two, 545mμ and 685mμ in males, and at all three in females, they show striking similarity to the Northumbrians. The higher reflectance values in these three populations are apparent in particular at wavelength 685mμ in males and females, while there is much less difference from the other British and Irish series at wavelength 425mμ (601), especially in the females.

The Manx and Cumbrian males and females exhibit the greatest difference from the Merthyr Tydfil series (Smith and Mitchell 1973), at all three wavelengths, with the three Irish populations of Sunderland et al. (1973), exhibiting mean reflectance values between these two groups. It is of interest that the fairest Irish population, that of Ballinlough, Co. Roscommon, lies on a similar latitude (54°N) to the Isle of Man and Cumbria. The Manx and Cumbrian figures are very similar to the Liverpool (53°50'N) series of Harrison and Owen (1964) at wavelengths 425mμ and 545mμ; but very different at wavelengths 685mμ. The same two population samples are also different at all wavelengths from Barnicot's (1958) series collected in London (51°50'N) which exhibit reflectance values most similar to those found in the Merthyr Tydfil population, which interestingly is also situated on a similar latitude, 52°N.

Table 66 also summarizes the data on skin colour available for north west European populations. At wavelength 425mμ the Manx and Cumbrian male mean reflectance values approximate most closely to those Belgians tested by Leguebe (1961) but are different from the Belgians reported by Rijn - Tournel (1965), and even more so from the Europeans investigated by Ojikutu (1965). At wavelength 545mμ the same two English series exhibit greatest divergence, in that they are darker, from Ojikutu's sample, but at the longest wavelength, 685mμ, they exhibit mean reflectance values similar to all three male European populations.

The Manx and Cumbrian females exhibit mean reflectance values at wavelength 425mμ similar to one Belgian series (Leguebe 1961) and lower than another series (Rijn - Tournel 1965), at 545mμ the reflectance values are again lower in the English series, while at 685mμ the northern English populations exhibited higher mean reflectance values.

Further analysis of the data in Table 66 reveals that in all the British Isles' samples, with three exceptions, the females are noticeably fairer than the males at all three wavelengths and irrespective of age. The three exceptions are, the Manx and Merthyr Tydfil series at wavelength 545mμ and the Ballinlough, Eire, population at wavelength 685mμ. However, in each of these three exceptions the difference found between the sexes is minute, less than 0.2%. The sex differences in the Manx, Cumbrian and Merthyr Tydfil samples were found to be statistically insignificant at all three wavelengths (Smith and Mitchell 1973). In the continental European populations for which data have been collected, the reverse finding is true, males are lighter than females at all three wavelengths.

Reviewing the literature on human skin colour, one discovers conflicting evidence of sex differences in this genetic trait. The following authors found that the males were darker than females (statistically significant or not) in their respective sample, with respect to one or more wavelength ;-

Author(s)		Population Sample
Lasker	(1954)	Mexican Mestizo
Barnicot	(1958)	Nigerian, Yoruba
		European, English
Tobias	(1961)	Bushmen
Harrison	(1961)	English, Liverpool
Harrison and Owen	(1964)	English, Liverpool
Harrison and Salzano	(1966)	Caingang Indians
Harrison et al.	(1967)	Brazilian "Whites"
		Brazilian Negroes
Hulse	(1973)	English, Northumberland
Smith and Mitchell	(1973)	English, Isle of Man
		English, Cumbria
		Welsh, Merthyr Tydfil
Sunderland et al.	(1973)	Selected Irish groups

However the following authors reported the males in their samples lighter than the females:-

Author(s)		Population Sample
Leguebe	(1961)	Belgium - Brussels
Rijn - Tournel	(1965)	Belgium
Ignazi	(1966)	France

Because of these very different findings regarding sex differences in skin pigmentation, it is difficult to ascertain any single, simple genetic factor operating to cause the differences in human skin colour. Therefore, the importance of sex and age differences, especially during adolescence, on the results of the present investigation cannot be measured. The author feels however that they should be borne in mind when looking at the results shown in Table 66. There can be no doubt that a great deal more work needs to be performed in this aspect of skin colour studies.

Conclusion

It has been shown that the samples of the resident population of Cumbria, and to a lesser extent Furness, exhibit a very different ABO distribution from that reported in two of the Manx series, the Non - Donors and Total Manx, but a similar one to that found in the Donors. However, for the reasons given in chapter 3 the author considers that the indigenous Manx population generally has an ABO distribution more akin to that found in the Non - Donors. It should also be remembered that the indigenous Cumbrian series was similar to the Manx Non - Donors and Total Manx in the proportion of ABO groups, but significantly different from Manx Donors.

Whereas the Manx display a frequency of allele Se similar to that found in the native Cumbrians, they are different from the other reported English resident samples. The Manx exhibit significant variation from the three English resident samples, as well as the indigenous Cumbrians, in the proportions of MNSs phenotypes. That it is generally the lower frequency of S in the Manx that accounts for the differences is shown by the fact that the same samples are similar in MN group distribution. With respect to P, Kell, Penney and Duffy blood groups the Manx exhibit overall homogeneity with the selected English samples. However, the high incidence of Lu^a in the Manx distinguishes this population from the English resident series. In addition the Manx display a different distribution of Rh Types from the English populations, including that of Cumbria.

Analysis of the serum protein polymorphisms of Hp, Tf and the B- lipoprotein allotype Ag, revealed that the Manx exhibit

frequencies of the alleles consistent with those reported in English populations. The red cell isoenzymes AP, PGM, AK and 6 - PGD, like the serum proteins, display phenotype and allele frequencies similar to those reported in the English population. However, the difference observed between the Manx and Southern English with respect to ADA is large and requires further investigation.

Examination of non - serological variability revealed that the Manx have a similar proportion of tongue - curlers and colour vision deficients as Northern England. Also, skin colour analysis showed that their pigmentation is very similar to that reported for Cumbrians and Northumbrians, but lighter than that found in inhabitants of Liverpool.

When the distribution of ABO phenotypes in the Manx was compared with that in selected Scottish populations, similar results were found as when the Northern English and Manx were compared. The Manx Non - Donors and Total Manx series are very different from the Northern Scots and Island series, but the Manx Donors are only different from the Orcadians. The same finding occurs when the Manx and the resident South West Scottish samples are compared. However, evidence that the Manx are not unique in their ABO distributions comes from data collected on the Isle of Bute. Izatt (1974) has found a high frequency of A (44.6%) in the Bute donor panel, compared with a frequency of 32.0% for A reported in Glasgow, Final Area 22 in Kopeć (1970). Further analysis revealed that a much higher frequency of A is found among the native Bute population or Brandanes, (53.5%) and a lower incidence in the incomer group (42.3%). It could well be

that if indigenous samples for many regions of the less industrialised parts of the British Isles were collected, they would be found to exhibit frequencies of ABO and other polymorphic genes different from those recorded for resident only samples and/or incomer groups.

The frequency distributions of the allelic genes controlling the M, N, P_1, Rh(D), K, k_1 Kp^a and Fy^a antigens as well as secretor status are found to exhibit a similar frequency in both Manx and Scottish populations. Also, the frequency distributions of the alleles and phenotypes of the serum proteins, Hp and Tf and red cell enzyme systems, AP and PGM, as well as of colour vision deficients and non - tasters of PTC, are similar in the Manx and Scottish samples. However, the Scots display a significantly lower frequency of Lu^a than the Manx, and the two groups are also distinct in MNSs groups.

The differences found when the Manx are compared with North Welsh samples for ABO group distributions are similar to those reported between the Manx and English. The Manx Donors are similar to, but the Non - Donors and Total Manx are very different from, the selected Welsh series. The Manx diverged most of all from the Welsh surnames sample, suggesting that there is a long standing difference between these two regions. However, areas in Wales exhibiting a higher frequency of A are known, such as part of Pembrokeshire, (Watkin 1960) which could be a product of Viking settlement.

Though the differences between the Manx and Welsh for most blood groups are insignificant, that for Lutheran groups is very marked. Also, analysis of data on PTC tasting ability and reflecance spectrophotometry of the skin revealed marked divergences

between the two groups.

Whereas the Manx Donors display an ABO distribution consistent with those reported in the Ulster series, there are great differences between the other Manx samples and the Ulster population. Heterogeneity between the Manx and Northern Irish is also found in the MNSs and Rh(D) groups, as well as in the frequency of non - tasters of PTC. Otherwise, the Manx are similar to the Ulster population for those polymorphisms where samples exist.

While the Manx Non - Donors and Total Manx are found to differ from the selected Dublin and Eire samples with respect to ABO groups, the Manx Donors are similar. It is of interest to note that in some instances, if there is an 'indigenous' in addition to a resident sample for a region, the two Manx samples are found to exhibit greatest divergence from the 'native' one. e.g. Wales, Ulster and Eire; but this was not the case with Cumbrian samples. For a number of polymorphisms investigated there was found a very real difference between the Manx and Irish samples, in particular the sample of Palsson et al. (1970). Not only does this series exhibit significant differences from the Manx in the distribution of many systems, but it diverges from other Irish series. This sample comprises only 295 young adult males from numerous locations, chiefly in central and western Eire, and therefore sampling might well be the cause. Those polymorphisms where the Manx are similar to all Irish series except that of Palsson et al (1970) include the MN, P and Duffy blood groups and the isoenzymes AP, PGM and AK. In addition the Manx, though different from all Irish series, are very much more different from the sample of Palsson et al. in MNS and Rh

Types. The Irish are also markedly divergent from the Manx in the distribution of Lutheran groups.

The final result of all these comparisons is that though the indigenous Manx exhibit some similarity to the surrounding mainland populations in the distribution of allelic genes at selected loci, it is not easy to demonstrate that the Manx are significantly more akin to the English, Scottish or Irish population samples. On the basis of those polymorphic systems for which comparison of frequency distributions was possible, the Manx seemed to be most different from the Welsh. Perhaps the Manx would be found to be more closely allied to one of the mainland groups if indigenous population samples were available. The present indigenous Cumbrian sample exhibits very close similarity to the Manx in the distribution of alleles at many loci.

However, it is important to stress that the indigenous Manx exhibit frequencies of some alleles that collectively distinguishes them as a unique population in the Irish Sea basin. The allelic genes that exhibit differences from mainland groups include; A(p) and B(q) of the ABO system, S, R_2 (cDE), R_1(CDe), Lu^a, Kp^a, Ag^x, P^c and ADA^2. Though the differences between the Manx and mainland populations for these allelewere not always statistically significant, in total the effect is to indicate a population to some extent genetically different from surrounding groups.

CHAPTER SIX

CONCLUSION

CONCLUSION

The present study was undertaken primarily to obtain data on the frequencies of various allelic genes at some of the blood group, serum protein and red cell isoenzyme loci known to exhibit genetic polymorphism, in the population of three regions; the Isle of Man, Cumbria and South West Scotland. Over the last few years there have been a large number of studies of population genetic variability, ranging from the investigation of differences between the major racial groups (Gordon et al. 1966), to studies of intra - group variability (Arends et al. 1967, Weitkamp et al. 1972). All these studies have as a common purpose the reporting of the frequency distributions of allelic genes at selected loci, and an attempt to explain the variability observed. The core of the present study is an examination of intra - population and inter - population genetic variability in the Isle of Man. Owing to its geographical position and political status throughout historical times, the Isle of Man was considered to be a most suitable region for such a study. In addition the Island's population was previously uninvestigated for its genetical characteristics.

Since many studies of the biology, including genetics, of the world's populations have been and are being performed, a recent work, the International Biological Programme (I.B.P.) Handbook, No. 9, (Weiner and Lourie, 1969), has been published as a guide to the standardising of techniques and methods so as to readily enable comparisons between setsof data to be effected. Where relevant the methods indicated in this book were followed as closely as possible, but in certain instances this proved impossible; for example employing all nine filters of the E.E.L.

reflectance spectrophotometer or the use of a portable anomaloscope for testing colour vision deficients.

In the author's view inter - and intra - population variability in British Isles' populations will be most suitably investigated by the collection of indigenous samples of the regions under study. The author's admittedly arbitrary definition of a 'native' person is not perfect, but it was found to be very efficient in sample collection. To have asked further details of the ancestry of a person would have reduced the completion of questionnaires considerably. Regarding the accuracy of the information given on the forms, the author feels that most, if not all, is correct. Each Manx blood donor was asked details of the birthplaces of his family on two separate occasions, and the changes made were found to be small in number and also very rarely affected the placement of the individual in one of the regions employed in the study of intra - Island variability. Typically, the changes made were a closer definition of the birthplace of an individual in a particular parish or sheading. On this evidence the author feels satisfied that the data on ancestry of individuals are correct.

A major problem with the selected samples, particularly the Manx, was the number of interrelationships within them. It has been shown that a relatively small number of surnames predominate on the Isle of Man, but that possession of the same name does not imply any known relationship between the persons. Sample numbers could have been increased by defining as 'native' all those individuals both of whose parents only were born on the Island. However, the question became one of quantity or quality of the

indigenous Manx sample; the author chose the latter. The author however, considers that a study should be carried out on other groups residing on the Isle of Man. Of particular interest would be the immigrants and also the offspring of incomer - native Manx marriages.

In order to avoid any suggestion that choice of samples may have influenced the results, the survey attempted to include individuals from as wide a spectrum of each society as possible; achieving more success in this in the Isle of Man than in Cumbria and South West Scotland.

The largest possible number of genetic polymorphisms were investigated in each population, including most of the blood groups, three serum proteins and nine red cell isoenzyme systems. Non - serological variability was examined by investigating the frequency of colour vision defectives, tongue curlers and non - tasters of PTC, as well as reflectance spectrophotometry of the skin.

In statistical comparisons based on data from two or more samples on those systems where gene numbers as well as phenotype numbers are determinable, there was the occasional difficulty of deciding whether to compare gene numbers or phenotype frequencies. Owing to the fact that sometimes significant differences were observed in the distribution of genes but not of phenotypes or vice versa, computations were usually performed employing both sets of data. The distribution of phenotypes of a particular system is a product of the mating system in that society, and is more readily subject to change than the distribution of the individual alleles in the same group. Occasionally two populations can be found similar with respect to allele distribution

yet very different in phenotype distribution.

Any study of heterogeneity is related to the numbers available for study. With relatively small numbers gradients can only be worked out very broadly and even wide local fluctuations over small areas may not be discerned at all. Each addition to numbers permits analysis at a new level and it is always possible that at some further stage a hitherto unsuspected pattern of variability may emerge (Fraser - Roberts 1953). For this reason it is hoped to continue this investigation of genetic variability in the Manx.

The major findings of the study are :-

(1) The relatively large difference between two similarly selected Manx samples, Blood Donors and Non - Donors, in the distribution of certain genetic traits, most especially ABO groups. Though the author has suggested possible reasons for the observed heterogeneity, he considers that this phenomenon requires further investigation.

(2) The relatively wide similarity among the indigenous populations from the three regions studied, but especially between the Manx and Cumbrians.

(3) The overall homogeneity of the indigenous Manx population when the distributions of selected polymorphic systems were subjected to a broad regional analysis. Only the ABO groups exhibited a tendency towards significant geographical heterogeneity in their frequency distribution.

(4) The indigenous Manx exhibit some marked similarities to mainland population samples, the majority of the latter comprising persons selected solely on the basis of their residence in

a particular area. However, the Manx also diverge significantly in the frequency distribution of alleles at certain loci, especially those of the ABO, MNS and Lutheran blood groups and the isoenzyme ADA.

(5) Though the Manx show close similarity to the northern English (especially Cumbrians) and some Irish samples in particular, for some polymorphic traits, they exhibit frequencies different from all the surrounding populations for a sufficient number of alleles to merit classification as a distinct population group in the Irish Sea basin.

(6) When the present data from all three populations studied for certain genetic traits are compared with those from other British Isles populations, a distinctive distribution pattern becomes apparent - that of a division between the North British or 'Celtic' populations and the South British or non - 'Celtic' groups. This pattern is found for the Hp and ADA systems as well as for the secretor groups.

However, as to which factors acting upon the alleles and/or phenotypes of the genetic polymorphisms have caused the variability observed, the author at this stage can only surmise. It has been shown that in terms of population history the present Manx population is thought to be originally of Celtic stock modified by the Norse settlement between the ninth and thirteenth centuries, and Anglo - Irish immigration in the thirteenth and fourteenth centuries. However, what numbers were involved and the sex ratios of these immigrants are very much open to conjecture. Certainly the distribution of Norse remains and place - names, which are very widespread, would lead one to think that their numbers were

large. The Anglo - Irish colonists are thought to have settled chiefly in the Southside, especially in the parishes of Rushen, Arbory and Malew. Since the fourteenth century immigration to the Isle of Man, though still important, has been on the individual rather than mass - movement level. As indicated by their surnames many persons have come from the northern English counties.

In an earlier chapter it was mentioned that anthropologists had determined that the indigenous Manx were a mixture of Scandio - Celtic elements, but that the proportions of these two major elements had a different geographical distribution. This study, however, has revealed that the Manx are a genetically homogeneous group.

'Celtic' populations are usually thought of as exhibiting a high frequency of blood group O (Mourant 1954), while Norse Vikings are thought to have possessed a relatively high frequency of A. (Watkin, 1960) If these simple statements were the case, then in the Manx population one might have expected finding differing regional frequencies of O and A, correlating broadly with the distribution of the Mediterrarean and Nordic physical types (Davies and Fleure, 1936). This is not so, though it should be mentioned that the highest frequency of O is found in Douglas, a centre of the Mediterranean type, and also the highest A is found in the Northside, a stronghold of the Nordic type. To add to the problem it is known that other regions of the British Isles with a history of Scandinavian settlement, such as the Orkney and Shetland Isles and the Western Isles of Scotland, generally reveal a low frequency of group A. This situation is also true of the present population of Iceland which is thought

to have been uninhabited prior to the Norse conquest. A suggested answer to this problem is that though the Vikings did indeed colonise these regions, their actual numbers were small relative to the Celtic population there. In the case of Iceland, it has been postulated that the island was settled by a small Viking aristocracy supported by a very much larger number of Celtic serfs (Donegani et al. 1950), and that the latter group necessarily would have made the largest contribution to the Icelandic gene pool.

The question arises though as to why the Manx should exhibit this relatively high frequency of A compared with surrounding populations, and also compared with other groups exhibiting a similar Norse - Celtic admixture. Is it not plausible to suggest that the Vikings, coming from a relatively inhospitable climate and infertile soils, would prefer to settle in greatest numbers in those parts they visited which were most fertile and occupied strategic locations. Such a place would be the Isle of Man, and so important did they regard it that they made it the centre of the Kingdom of Man and the Isles. Perhaps the Island was selected as the capital of this large dispersed unit because the majority of Vikings within it resided here, and that many of the Western Isles, especially the most desolate were regarded by the Norse as mere colonial territories, to be ruled by a small number of officials supervising a much larger Celtic population. What has been said about the Isle of Man could have perhaps also obtained in the Isle of Bute and in that part of Pembrokeshire (Watkin, 1960), where a high frequency of A has been found in constrast to its frequency in the surrounding populations, and

where the Norse are known to have settled. In fact the ABO distributions of Bute and Man show a close similarity especially with respect to the high frequency of A and low incidence of B.

However, it is perhaps just as possible that the relatively high frequency of A and those frequencies of alleles that characterise the Manx, such as S, Lu^a and ADA^2, could have been produced by Scandio - Celtic admixture during the period between the ninth and twelth centuries, followed by a number of generations of random genetic drift. Such a theoretical model has been applied employing the genetic studies of the Icelandic population. (Thompson, 1973)

It was mentioned in chapter one that during much of the Stanley Period the Isle of Man was isolated from the rest of the British Isles as a result of trade and communication being positively discouraged by the Manx administration. This naturally provided suitable conditions for the action of random drift. Historical records reveal evidence of a high incidence of parish endogamy throughout this period, and the strong distinction between Northside and Southside of the Island also restricted the distance from which a person would select his or her spouse. However, any regional heterogeneity in genetic polymorphisms that may have been produced by these conditions seems to have disappeared today; yet differences in body and head measurement remain. Could it be that the present sample, because of its small size, is not representative of the present indigenous Manx? Since it has been estimated that the series incorporates approximately 10% of the total group being investigated, this is most unlikely.

The explanation of the distribution of the allelic genes of

the polymorphisms investigated in this study requires much more detailed investigation. Increased light would be shed on this problem if the Manx parish records and other demographic data (full records do exist) were examined, with a view to determining such information as the degree of inbreeding, frequency of parish endogamy and also of intra - Northside and intra - Southside matings as well as inter - regional marriages over the last 400 years. Such a study, when related to the findings of the present survey, would be most illuminating.

In conclusion, it is suggested that while the indigenous Manx population exhibits overall homogeneity with respect to some genetic polymorphisms, when compared with selected mainland groups, it still displays a distinctiveness in the frequency of some genes. The Manx appear to be most similar to the indigenous Cumbrians, but as similarly selected native samples are very rare for other British regions, it is not possible to say that this would still hold if such samples were available from other regions surrounding the Irish Sea. Finally, it is suggested that a prerequisite for the satisfactory explanation of the genetic distributions observed on the Isle of Man is a detailed analysis of the demographic data.

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TABLE 1. DISTRIBUTION OF ABO BLOOD GROUPS - ISLE OF MAN

a) Tested with anti-A, anti-B, anti-A+B, and anti-A_1 sera

	1 Native Blood Donors				2 Native Non-Blood Donors				3 Native Schoolchildren			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A_1	61	.2785	62.22	.2841	45	.3309	44.85	.3298	121	.3580	120.53	.3566
A_2	18	.0822	18.31	.0836	16	.1176	15.94	.1172	38	.1124	37.86	.1120
B	13	.0594	13.27	.0606	11	.0809	10.98	.0807	24	.0710	23.89	.0707
O	119	.5434	121.46	.5546	60	.4412	59.80	.4397	146	.4320	145.50	.4305
A_1B	6	.0274	2.80	.0128	4	.0294	3.10	.0228	8	.0236	7.41	.0219
A_2B	2	.0091	0.94	.0043	-	-	1.32	.0097	1	.0030	2.82	.0083
Total:	219	1.0000	219.00	1.0000	136	1.0000	135.99	.9999	338	1.0000	338.01	1.0000
Gene Frequencies												
P_1			.1615				.1954				.2117	
P_2			.0542				.0832				.0804	
q			.0396				.0583				.0518	
r			.7447				.6631				.6561	

b) Tested with anti-A, anti-B and anti-AB sera only

	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	79	.3607	80.53	.3677	61	.4485	60.79	.4470	159	.4704	158.39	.4686
B	13	.0594	13.27	.0606	11	.0809	10.98	.0807	24	.0710	23.89	.0707
O	119	.5434	121.46	.5546	60	.4412	59.80	.4397	146	.4320	145.50	.4305
AB	8	.0365	3.74	.0171	4	.0294	4.42	.0325	9	.0266	10.23	.0302
Total:	219	1.0000	219.00	1.0000	136	1.0000	135.99	.9999	338	1.0000	338.01	1.0000
Gene Frequencies												
p			.2158				.2786				.2921	
q			.0396				.0583				.0518	
r			.7446				.6631				.6561	
A: A+O Index.			39.90				50.41				52.13	

TABLE 1 (Cont). DISTRIBUTION OF ABO BLOOD GROUPS - ISLE OF MAN

a) Tested with anti-A, anti-B, anti-A+B, and antit-A_1 sera

	4 Native Women attending Ante-Natal Clinic				5 Total Native Manx			
Group	Obs. No.	Obs. Freq.	Exp. No.	Exp. Freq.	Obs No.	Obs Freq.	Exp. No.	Exp. Freq.
A_1	Not tested with anti-A_1 serum				227	.3276	227.94	.3289
A_2					72	.1039	72.38	.1044
B					48	.0693	48.30	.0697
O					325	.4690	326.41	.4710
A_1B					18	.0260	13.07	.0189
A_1B					3	.0043	4.91	.0071
Total:					693	1.0001	693.01	1.0000
Gene Frequencies								
p^1						.1924		
p^2						.0723		
q						.0490		
r						.6863		

b) Tested with anti-A, anti-B, and anti-AB sera only

	Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	51	.4397	51.02	.4398	350	.4326	351.32	.4343
B	10	.0862	10.01	.0863	58	.0717	58.26	.0720
O	51	.4397	51.02	.4398	376	.4648	377.61	.4668
AB	4	.0345	3.96	.0341	25	.0309	21.86	.0270
Total:	116	1.0001	116.01	1.000	809	1.0000	809.05	1.0001
Gene Frequencies								
p		.2747				.2660		
q		.0621				.0508		
r		.6632				.6832		
A: A+O Index			50.00				48.21	

TABLE 1 (cont.) DISTRIBUTION OF ABO BLOOD GROUPS - ISLE OF MAN

a) Tested with anit-A, anti-B, anti-A+B, and anti-A_1 sera

	6				7			
	Non-Native women attending Ante-Natal Clinic				All Women attending Ante-Natal clinic			
	Obs		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A_1								
A_2								
B	Not tested with anti-A_1 serum				Not tested with anti-A_1 serum			
O								
A_1B								
A_2B								
Total:								

b) Tested with anti-A, anti-B, and anti-AB sera only

	Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	44	.3667	44.28	.3690	95	.4025	95.29	.4038
B	11	.0917	11.06	.0922	21	.0890	21.06	.0892
O	61	.5083	61.35	.5112	112	.4746	112.39	.4762
AB	4	.0333	3.31	.0276	8	.0339	7.25	.0307
Total:	120	1.0000	120.00	1.0000	236	1.0000	235.99	.9999

Gene Frequencies		
p	.2232	.2480
q	.0618	.0619
r	.7150	.6901
A: A+O Index	41.90	45.89

TABLE 2. DISTRIBUTION OF ABO BLOOD GROUPS - MANX NATIVE POPULATION

a) Tested with anti-A, anti-B, anti-A+B, and anti-A_1 sera

	Manx Blood Donors				Manx Non-Blood Donors			
	obs.		exp.		obs.		exp.	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A_1	61	.2785	62.22	.2841	166	.3502	165.39	.3489
A_2	18	.0822	18.31	.0836	54	.1139	53.79	.1135
B	13	.0594	13.27	.0606	35	.0738	34.87	.0736
O	119	.5434	121.46	.5546	206	.4345	205.29	.4331
A_1B	6	.0274	2.80	.0128	12	.0253	10.54	.0222
A_2B	2	.0091	0.94	.0043	1	.0021	4.13	.0087
Total:	219	1.0000	219.00	1.0000	474	.9998	474.01	1.0000
Gene Frequencies								
p_1			.1615				.2070	
p_2			.0542				.0812	
q			.0396				.0537	
r			.7447				.6581	

b) Tested with anti-A, anti-B, and anti-A+B sera only

Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	79	.3607	80.53	.3677	271	.4593	270.23	.4580
B	13	.0594	13.27	.0606	45	.0763	44.91	.0761
O	119	.5434	121.46	.5546	257	.4356	256.23	.4343
AB	8	.0365	3.74	.0171	17	.0288	18.67	.0316
Total:	219	1.0000	219.00	1.0000	590	1.0000	590.04	1.0000
Gene Frequencies								
p			.2158				.2856	
q			.0396				.0554	
r			.7446				.6590	
A: A+O Index				.39.90				51.33

TABLE 3. DISTRIBUTION OF ABO BLOOD GROUPS-MANX NATIVE POPULATION
AGE COMPARISON

a) Tested with anti-A, anti-B, anti-A+B and anti-A_1 sera

Group.	Juveniles (under 18 years old)				Adults (over 18 years old)			
	Obs.		Exp.		Obs.		Exp.	
	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A_1	121	.3580	120.53	.3566	106	.2986	107 23	.3021
A_2	38	.1124	37.86	.1120	34	.0958	34.41	.0969
B	24	.0711	23.89	.0707	24	.0676	24.30	.0684
O	146	.4320	145.50	.4305	179	.5042	181.18	.5104
A_1B	8	.0236	7.41	.0219	10	.0282	5.74	.0162
A_2B	1	.0030	2.82	.0083	2	.0056	2.14	.0060
Total:	338	1.0001	338.01	1.0000	355	1.0000	355.00	1.0000
Gene Frequencies								
p_1			.2117				.1743	
p_2			.0804				.0649	
q			.0518				.0464	
r			.6561				.7144	

b) Tested with anti-A, anti-B and anti-A+B sera only

Group								
	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	159	.4704	158.39	.4686	191	.4055	192.81	.4094
B	24	.0710	23.89	.0707	34	.0722	34.31	.0728
O	146	.4320	145.50	.4305	230	.4883	232.18	.4930
AB	9	.0266	10.23	.0302	16	.0340	11.69	.0248
Total:	338	1.0000	338.01	1.0000	471	1.0000	470.99	1.0000
Gene Frequencies								
p			.2921				.2478	
q			.0518				.0501	
r			.6561				.7021	
A: A+O Index		52.13				45.37		

TABLE 4. DISTRIBUTION OF ABO BLOOD GROUPS - SUMMARY OF PANTIN'S (1950) MANX SERIES

	(1) Total Sample - Related O Rh (D) Neg.bias				(2) Total Sample - Unrelated O Rh (D) Neg.bias			
	Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	865	.4207	870.47	.4234	654	.4360	655.59	.4371
B	147	.0715	147.96	.0720	111	.0740	111.47	.0743
O	978	.4757	984.26	.4787	689	.4593	690.54	.4604
AB	66	.0321	53.24	.0259	46	.0307	42.50	.0283
Total:	2056	1.0000	2055.93	1.0000	1500	1.0000	1500.10	1.0000
Gene Frequencies								
p			.2579				.2688	
q			.0502				.0527	
r			.6919				.6785	
A: A+O Index			46.93				48.70	

	(3) Unrelated Individuals with Manx names				(4) Males and Unmarried Females with Manx names (unrelated)			
	Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	242	.4440	243.26	.4463	86	.4300	86.04	.4302
B	37	.0679	37.53	.0689	18	.0900	17.98	.0899
O	246	.4514	249.64	.4581	89	.4450	89.06	.4453
AB	20	.0367	14.67	.0269	7	.0350	6.90	.0345
Total:	545	1.0000	545.10	1.0002	200	1.0000	199.98	.9999
Gene Frequencies								
p			.2742				.2684	
q			.0491				.0643	
r			.6768				.6673	
A: A+O Index			49.59				49.14	

TABLE 5. DISTRIBUTION OF ABO BLOOD GROUPS - CUMBRIA and SOUTH WEST SCOTLAND

a) Tested with anti-A anti-B anti-A+B and anti-A_1 sera

	Native Cumbrian Blood Donors				Native Cumbrian School-Children				Total Cumbrian Natives			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A_1	63	.3182	62.72	.3168	97	.3003	95.73	.2964	160	.3071	158.41	.3040
A_2	23	.1162	22.90	.1157	42	.1300	41.43	.1283	65	.1248	64.44	.1237
B	18	.0909	17.90	.0904	30	.0929	29.64	.0918	48	.0921	47.54	.0912
O	88	.4444	87.56	.4422	147	.4551	145.04	.4490	235	.4511	232.62	.4465
A_1B	5	.0253	4.83	.0244	7	.0217	7.38	.0228	12	.0230	12.20	.0234
A_2B	1	.0051	2.10	.0106	-	-	3.78	.0117	1	.0019	5.89	.0113
Total:	198	1.0001	198.01	1.0001	323	1.0000	323.00	1.0000	521	1.0000	521.10	1.0001
Gene Frequencies												
p^1		.1883				.1749				.1799		
p^2		.0819				.0897				.0869		
q		.0648				.0653				.0651		
r		.6650				.6701				.6682		

b) Tested with anti-A, anti-B, and anti-AB sera only

Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	86	.4343	85.62	.4325	156	.4575	153.98	.4516	242	.4490	239.5	3.4444
B	18	.0909	17.90	.0904	30	.0880	29.61	.0868	48	.0891	47.5	4.0882
O	88	.4444	87.56	.4422	147	.4311	145.09	.4255	235	.4360	232.6	6.4317
AB	6	.0303	6.93	.0350	8	.0235	12.31	.0361	14	.0260	19	25.0357
Total:	198	.9999	198.01	1.0001	341	1.0001	340.99	1.0000	539	1.0001	538.98	1.0000
Gene Frequencies												
p		.2702				.2842				.2790		
q		.0648				.0635				.0640		
r		.6650				.6523				.6570		
A : A+O Index			.49.43				51.49				50.73	

TABLE 5 (Cont.) DISTRIBUTION OF ABO BLOOD GROUPS - CUMBRIA AND SOUTH WEST SCOTLAND

	Native South West Scottish Donors				Resident South West Scottish Donors				Total South West Scottish Series			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A_1	23	.3194	23.51	.3265	27	.2700	28.62	.2862	50	.2907	52.08	.3028
A_2	11	,1528	11.26	.1564	7	.0700	7.43	.0743	18	.1047	18.77	.1091
B	1	.0139	1.02	.0142	11	.1100	11.67	.1167	12	.0698	12.50	.0727
O	35	.4861	35.80	.4972	46	.4600	48.76	.4876	81	.4709	84.35	.4904
A_1B	2	.0278	0.26	.0036	7	.0700	2.71	.0271	9	.0523	3.03	.0176
A_2B	-	-	0.15	.0021	2	.0200	0.81	.0081	2	.0116	1.28	.0074
Total:	72	1.0000	72.00	1.0000	100	1.0000	100.00	1.0000	172	1.0000	172.01	1.0000
Gene Frequencies												
p_2^1		.1816				.1713				.1756		
p		.1033				.0513				.0740		
q		.0100				.0791				.0501		
r		.7051				.6983				.7003		
	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	34	.4722	34.77	.4829	71	.3568	70.77	.3556	105	.3875	105.11	.3879
B	1	.0139	1.02	.0142	33	.1658	32.88	.1652	34	.1255	34.04	.1256
O	35	.4861	35.80	.4972	85	.4271	84.67	.4255	120	.4428	120.06	.4430
AB	2	.0278	.41	.0057	10	.0503	10.72	.0539	12	.0443	11.79	.0435
Total	72	1.0000	72.00	1.0000	199	1.0000	199.04	1.0002	271	1.0001	271.00	1.0000
Gene Frequencies												
p		.2849				.2315				.2459		
q		.0100				.1163				.0885		
r		.7051				.6523				.6656		
A: A+O Index			49.28				45.51				46.67	

TABLE 6. DISTRIBUTION OF ABO BLOOD GROUPS -- CUMBRIA AND SOUTH WEST SCOTLAND 'PROSPECT' SERIES.

	NORTH and WEST CUMBRIA. Fraser Roberts 1953.		SOUTH CUMBRIA Fraser Roberts 1953.		TOTAL CUMBRIA Fraser Roberts 1953		CUMBRIA Kopeć 1970		FINAL REGION(3) Kopeć 1970		SOUTH WEST SCOTLAND Kopeć 1970	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	975	.3546	934	.3988	2909	.3677	1746	.3927	7943	.3984	224	.3660
B	309	.0914	214	.0914	723	.0914	364	.0819	1594	.0800	77	.1258
O	1342	.5283	1128	.4816	4070	.5145	2218	.4989	9834	.4932	295	.4820
AB	143	.0257	66	.0282	209	.0264	118	.0365	566	.0284	16	.0262
Total	2569	1.0000	2342	1.0000	7911	1.0000	4446	1.0000	19937	1.0000	612	1.0000
Gene Frequencies												
p	.2128		.2440		.2219		.2379		.2421		.2252	
q	.0604		.0629		.0611		.0558		.0549		.0848	
r	.7268		.6931		.7170		.7063		.7030		.6900	
A: AO Index	40.17		45.30		41.68		44.05		44.68		43.16	

TABLE 7 DISTRIBUTION OF ABO BLOOD GROUPS - ISLE OF MAN, DOUGLAS AND AREA AND TOTAL SERIES

a) Tested with anti-A, anti-B, anti-AB and anti-A_1 sera

Group.	Native Manx Blood Donors No.	Native Manx Blood Donors Freq.	Native Manx Non-Donors No.	Native Manx Non-Donors Freq.	Total Native Manx No.	Total Native Manx Freq.	Total Native Cumbrians No.	Total Native Cumbrians Freq.	Native South West Scots No.	Native South West Scots Freq.	Total South West Scotland No.	Total South West Scotland Freq.
A_1	91	.2785	166	.3502	227	.3276	160	.3071	23	.3194	50	.2907
A_2	18	.0322	54	.1139	72	.1039	65	.1248	11	.1528	13	.1047
B	13	.0594	35	.0738	48	.0693	48	.0921	1	.0139	12	.0698
O	119	.5434	206	.4346	325	.4690	235	.4511	35	.4861	81	.4709
A_1B	6	.0274	12	.0253	18	.0260	12	.0230	2	.0278	9	.0523
A_2B	2	.0091	1	.0021	3	.0043	1	.0019	-	-	2	.0116
Total:	219	1.0000	474	.9999	693	1.0001	521	1.0000	72	1.0000	172	1.0000
Gene Frequencies												
p	.1615		.2070		.1924		.1799		.1816		.1756	
p_1	.0542		.0812		.0723		.0868		.1033		.0740	
q	.0396		.0537		.0490		.0551		.0100		.0501	
r	.7447		.6581		.6863		.6682		.7051		.7003	

b) Tested with anti-A anti-B and anti-AB sera only

Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	79	.3607	271	.4593	350	.4326	242	.4490	34	.4722	105	.3875
B	13	.0594	45	.0763	58	.0717	48	.0891	1	.0139	34	.1255
O	119	.5434	257	.4356	376	.4648	235	.4360	35	.4861	120	.4428
AB	8	.0365	17	.0288	25	.0309	14	.0260	2	.0278	12	.0443
Total:	219	1.0000	590	1.0000	809	1.0000	539	1.0001	72	1.0000	271	1.0001
Gene Frequencies												
p	.2158		.2856		.2560		.2720		.2849		.2469	
q	.0396		.0554		.0508		.0640		.0100		.0685	
r	.7446		.6590		.6832		.6570		.7051		.6846	
A: A+O Index	39.90		51.33		48.21		50.73		49.28		46.67	

TABLE 8. DISTRIBUTION OF SECRETOR GROUPS - ISLE OF MAN AND CUMBRIA

	Native Manx		Native Cumbrians	
Group.	No.	Freq.	No.	Freq.
Se(-)	47	.2883	37	.2891
Se(+)	116	.7117	91	.7109
Total:	143	1.0000	128	1.0000
	Gene Frequencies			
Se	.4631		.4623	
se	.5369		.5377	

TABLE 9 (a) DISTRIBUTION OF MNSs BLOOD GROUPS - MANX NATIVE POPULATION

Tested with 4 anti-sera

	DONORS				ADULT NON-DONORS				TOTAL MANX			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq..
MMSS	12	.0548	11.78	.0538	12	.0896	9.67	.0722	24	.0680	21.32	.0604
MMSs	24	.1096	29.78	.1360	10	.0746	19.34	.1443	34	.0963	49.26	.1396
MMss	20	.0913	18.83	.0860	15	.1119	9.67	.0721	35	.0991	28.47	.0807
MNSS	7	.0320	4.95	.0226	2	.0149	2.69	.0200	9	.0255	7.75	.0220
MNSs	52	.2374	49.56	.2263	40	.2985	33.32	.2486	92	.2606	82.79	.2345
MNss	59	.2694	54.71	.2498	28	.2090	30.62	.2285	87	.2465	85.33	.2417
NNSS	2	.0091	.53	.0024	-	-	.19	.0014	2	.0057	.71	.0020
NNSs	5	.0228	9.11	.0416	4	.0299	4.25	.0317	9	.0255	13.43	.0380
NNss	38	.1735	39.75	.1815	23	.1716	24.25	.1810	61	.1728	63.94	.1811
Total:	219	.9999	219.00	1.0000	134	1.0000	134.00	0.9998	353	1.0000	353.00	1.0000

Gene Frequencies

MS	.2320	.2687	.2457
Ms	.2932	.2686	.2840
NS	.0488	.0373	.0447
Ns	.4260	.4254	.4256

MN Groups

Group	Obs.		Exp.		Obs.		Exp.	
	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MM	56	.2557	60.38	.2757	37	.2761	38.69	.2887
MN	118	.5388	109.22	.4987	70	.5224	66.62	.4972
NN	45	.2055	49.39	.2255	27	.2015	28.69	.2141
Total:	219	1.0000	218.99	.9999	134	1.0000	134.00	1.0000

Gene Frequencies

M	.5251	.5373
N	.4749	.4627

TABLE 9 (b) DISTRIBUTION OF MNSs BLOOD GROUPS - MANX NATIVE POPULATION

Tested with 3 antisera

	Schoolchildren				Total Manx			
	Obs.		Exp.		Obs.		Exp.	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MMS	22	.0917	32.25	.1344	80	.1349	96.24	.1623
MMss	27	.1125	27.27	.1136	62	.1046	62.21	.1049
MNS	75	.3125	54.55	.2273	176	.2968	140.60	.2371
MNss	66	.2750	65.45	.2727	153	.2580	155.54	.2623
NNS	11	.0458	21.21	.0884	22	.0371	41.21	.0695
NNss	39	.1625	39.27	.1636	100	.1686	97.19	.1639
Total:	240	1.0000	240.00	1.0000	593	1.0000	592.99	1.0000

Gene Frequencies

MS	.1609	.1930
Ms	.3371	.3239
NS	.0975	.0782
Ns	.4045	.4049

MN Groups	Obs.		Exp.		Obs.		Exp.	
	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MM	85	.2530	93.78	.2791	178	.2583	192.81	.2798
MN	185	.5506	167.46	.4984	373	.5414	343.34	.4983
NN	66	.1964	74.76	.2225	138	.2003	152.85	.2219
Total:	336	1.0000	336.00	1.0000	689	1.0000	689.00	1.0000

Gene Frequencies

M	.5283	.5290
N	.4717	.4710

TABLE 10 DISTRIBUTION OF MN BLOOD GROUPS - MANX MALES AND FEMALES

Group.	Males				Females			
	Obs.		Exp.		Obs.		Exp.	
	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MM	75	.2226	88.83	.2636	103	.2926	104.17	.2959
MN	196	.5816	168.38	.4996	177	.5028	174.64	.4961
NN	66	.1958	79.79	.2368	72	.2046	73.19	.2079
Total:	337	1.0000	337.00	1.0000	352	1.0000	352.00	.9999

Gene Frequencies		
M	.5134	.5440
N	.4866	.4560

TABLE 11a

DISTRIBUTION OF MNSs BLOOD GROUPS - CUMBRIAN NATIVE POPULATION

Tested with 4 antisera

	Cumbrian Donors				Cumbrian Schoolchildren				Total Cumbria			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MMS S	11	.0696	10.71	.0678	5	.0543	5.27	.0573	16	.0640	15.91	.0637
MMSs	23	.1456	23.62	.1495	17	.1848	12.43	.1352	40	.1600	36.04	.1442
MMss	12	.0759	13.02	.0824	10	.1087	7.34	.0798	22	.0880	20.41	.0816
MNSS	7	.0443	4.09	.0259	3	.0326	2.86	.0311	10	.0400	7.03	.0281
MNSs	32	.2025	37.65	.2383	16	.1739	21.57	.2345	48	.1920	59.21	.2368
MN_{ss}	42	.2658	36.54	.2313	13	.1413	21.48	.2334	55	.2200	58.04	.2322
NNSS	-	-	.39	.0025	-	-	.39	.0042	-	-	.78	.0031
NNSs	7	.0443	6.33	.0400	7	.0761	4.94	.0537	14	.0560	11.32	.0453
NNss	24	.1519	25.54	.1622	21	.2283	15.72	.1708	45	.1800	41.27	.1651
Total:	158	.9999	157.99	.9999	92	1.0000	92.00	1.0000	250	1.0000	250.01	1.0001

Gene Frequencies

MS	.2604	.2393	.2523
Ms	.2871	.2824	.2857
NS	.0497	.0650	.0557
Ns	.4028	.4133	.4063

TABLE 11b

DISTRIBUTION OF MNSs BLOOD GROUPS - SOUTH WEST SCOTLAND

a) Tested with 4 antisera

	S.W.SCOTTISH NATIVE DONORS				S.W.SCOTTISH RESIDENT DONORS				TOTAL S.W.SCOTLAND			
	Observed		Expected		Observed		Expected		Observed		Expected	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MMS	5	.0694	6.13	.0851	9	.0900	7.51	.0751	14	.0814	13.57	.0789
Ss	12	.1667	10.79	.1499	13	.1300	15.95	.1595	25	.1453	26.79	.1557
ss	5	.0694	4.75	.0660	11	.1100	8.47	.0847	16	.0930	13.22	.0768
MNSS	-	-	-	-	2	.0200	3.07	.0307	2	.0116	3.19	.0185
Ss	20	.2778	18.96	.2634	26	.2600	24.03	.2403	46	.2674	42.66	.2480
ss	15	.2083	16.70	.2319	19	.1900	22.06	.2206	34	.1977	38.99	.2267
NNSS	-	-	-	-	-	-	.31	.0031	-	-	.18	.0011
Ss	-	-	-	-	5	.0500	4.24	.0424	5	.0291	4.64	.0270
ss	15	.2083	14.67	.2038	15	.1500	14.36	.1436	30	.1744	28.76	.1672
Total:	72	.9999	72.00	1.0001	100	1.0000	100.00	1.0000	172	.9999	172.00	.9999

Gene Frequencies

MS	.2917	.2740	.2809
Ms	.2569	.2910	.2772
NS	.0000	.0560	.0330
Ns	.4514	.3790	.4089

MN Groups

	Observed		Expected		Observed		Expected		Observed		Expected	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MM	22	.3056	21.67	.3010	33	.3300	31.92	.3192	55	.3198	53.58	.3115
MN	35	.4861	35.66	.4953	47	.4700	49.16	.4916	82	.4767	84.83	.4932
NN	15	.2083	14.67	.2037	20	.2000	18.92	.1892	35	.2035	33.59	.1953
Total:	72	1.0000	72.00	1.0000	100	1.0000	100.00	1.0000	172	1.0000	172.00	1.0000

Gene Frequencies

M	.5486	.5650	.5582
N	.4514	.4350	.4418

TABLE 11c

DISTRIBUTION OF MNSs BLOOD GROUPS - CUMBRIAN NATIVE POPULATION

Tested with 3 antisera

	Cumbrian Donors				Cumbrian School-children				Total Cumbria			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MMS	36	.1818	38.26	.1933	66	.2245	60.97	.2074	102	.2073	99.16	.2015
MMss	18	.0909	19.00	.0960	33	.1122	27.19	.0925	51	.1037	46.28	.0941
MNS	52	.2626	48.82	.2466	74	.2517	78.43	.2668	126	.2561	127.24	.2586
MNss	53	.2677	49.61	.2506	50	.1701	67.23	.2287	103	.2093	116.88	.2376
NNS	9	.0455	9.92	.0501	18	.0612	18.61	.0633	27	.0549	28.64	.0582
NNss	30	.1515	32.38	.1635	53	.1803	41.56	.1414	83	.1687	73.80	.1500
Total:	198	1.0000	197.99	1.0001	294	1.0000	293.99	1.0001	492	1.0000	492.00	1.0000
	Gene Frequencies											
MS		.2280				.2435				.2370		
Ms		.3098				.3041				.3067		
NS		.0578				.0764				.0690		
Ns		.4044				.3760				.3873		
MN Groups	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MM	54	.2727	57.28	.2893	102	.3218	92.79	.2927	156	.3029	150.07	.2914
MN	105	.5303	98.43	.4971	139	.4385	157.42	.4966	244	.4738	255.85	.4968
NN	39	.1970	42.29	.2136	76	.2397	66.79	.2107	115	.2233	109.08	.2118
Total:	198	1.0000	198.00	1.0000	317	1.0000	317.00	1.0000	515	1.0000	515.00	1.0000
	Gene Frequencies											
M		.5378				.5410				.5398		
N		.4622				.4590				.4602		

TABLE 12. DISTRIBUTION OF P BLOOD GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

Sample	No. tested	Phenotype				Genes	
		P_1+		P_1-		P_1	P_2
		No.	Freq.	No.	Freq.		
Manx Donors *	182	122	.6703	60	.3297	.4258	.5742
Manx Non Donors*	154	123	.7987	31	.2013	.5513	.4487
Total Manx*	336	245	.7292	91	.2708	.4796	.5204
Manx Donors	166	118	.7108	48	.2892	.4622	.5378
Cumbrian Donors	22	11	.5000	11	.5000	.2929	.7071
South West Scottish Donors	72	56	.7778	16	.2222	.5286	.4714
Total Cumbria & S.W.Scotland	94	67	.7128	27	.2872	.4641	.5359

* These samples tested with MRC. SPGL Anti-P_1 serum

Other samples tested with anti-P_1 serum obtained from Lancaster Blood Transfusion Service.

TABLE 13a. DISTRIBUTION OF Rh. BLOOD GROUPS - NATIVE MANX POPULATION

Tested with anti-D,C,c,E,e, and C^w sera and for D^u Tested with anti -D,C,c,E,eonly.

	Blood Donors		Adult Non-Donors		Schoolchildren		Total Manx	
Rh.Type	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
R_1r	67	.3059	45	.3309	105	.3889	218	.3488
rr	46	.2100	26	.1912	47	.1741	119	.1904
R_1R_2	32	.1461	24	.1765	29	.1074	87	.1392
R_2r	25	.1142	25	.1838	30	.1111	80	.1280
R_1R_1	32	.1461	12	.0882	34	.1259	80	.1280
R_2R_2	6	.0274			16	.0593	22	.0352
R_or	5	.0228	2	.0147	3	.0111	10	.0160
rr"	1	.0046	1	.0074	2	.0074	4	.0064
rr'					3	.0111	3	.0048
$R^w{}_1R_1$	2	.0091						
$R^w{}_1r$								
$R^w{}_1R_2$	1	.0046	1	.0074				
R_1R_z	1	.0046			1	.0037	2	.0032
$R_1{}^ur$	1	.0046						
$R_2{}^ur$								
Total:	219	1.0000	136	1.0001	270	1.0000	625	1.0000

Gene Complex	frequencies			
r	.4283	.4499	.4346	.4353
R_1	.3724	.3419	.3636	.3675
R_2	.1533	.1793	.1624	.1617
R_o	.0226	.0170	.0135	.0180
r"	.0054	.0082	.0062	.0074
r'			.0141	.0056
R_z	.0058		.0055	.0045
$R_1{}^w$	.0068	.0037		
$R_1{}^u$	.0054			

TABLE 13b DISTRIBUTION OF RH (D) NEGATIVES - MANX NATIVE POPULATION

Sample	No.tested	Rh.(D) Negatives.		Genes	
		No.	Freq.	D	d
Blood Donors	219	47	.2146	.5368	.4632
Adult Non Donors	136	27	.1985	.5545	.4455
Schoolchildren	332	64	.1928	.5609	.4391
Women attending Ante-natal clinic	116	21	.1810	.5746	.4254
Total Manx	803	159	.1980	.5550	.4450
Pantin (1950)	410	62	.1512	.6112	.3888

TABLE 14a DISTRIBUTION OF Rh. BLOOD GROUPS - CUMBRIA AND SOUTH WEST SCOTLAND

	Tested with anti D,C,c,E,e and C^w sera and for D^u		Tested with anti D,C,c,E, and e and for D^u		Total Native Cumbria	
	Native Cumbrian Donors		Native Cumbrian Schoolchildren			
Rh.Type	No.	Freq.	No.	Freq.	No.	Freq.
R_1r	71	.3586	116	.4014	187	.3840
rr	38	.1919	51	.1765	89	.1828
R_1R_2	22	.1111	30	.1038	52	.1068
R_2r	17	.0859	25	.0865	42	.0862
R_1R_1	32	.1616	49	.1696	81	.1663
R_2R_2	9	.0455	4	.0138	13	.0267
R_or	-	-	7	.0242	7	.0144
rr"	-	-	4	.0138	4	.0082
rr'	1	.0050	1	.0035	2	.0041
$R_1{}^wR_1$	2	.0101	-	-	2	.0041
$R_1{}^wr$	3	.0152	-	-	3	.0062
$R_1{}^wR_2$	-	-	-	-	-	-
R_1R_z	-	-	1	.0035	1	.0021
$R_1{}^ur$	2	.0101	1	.0035	3	.0062
$R_2{}^ur$	1	.0050	-	-	1	.0021
Total:	198	1.0000	289	1.0001	487	1.0002

Gene Complex Frequencies

r	.4318	.4289	.4313
R_1	.3915	.4170	.4066
R_2	.1407	.0973	.1140
R_o	-	.0285	.0167
r"	-	.0161	.0095
r'	.0126	.0063	.0088
R_z	-	.0042	.0025
$R_1{}^w$	.0126	-	.0051
$R_1{}^u$	.0050	.0017	.0031
$R_2{}^u$	.0058	-	.0024

TABLE 14a (cont.) DISTRIBUTION OF Rh. BLOOD GROUPS - CUMBRIA AND SOUTH WEST SCOTLAND

Tested with anti-D,C,c,E,e, and C^w and for D^u

Rh. Type	S.W. Scottish Native Donors		S.W. Scottish Resident Donors		Total S.W. Scottish	
	No.	Freq.	No.	Freq.	No.	Freq.
R_1r	21	.2917	25	.2500	46	.2674
rr	18	.2500	26	.2600	44	.2558
R_1R_2	8	.1111	16	.1600	24	.1395
R_2r	2	.0278	10	.1000	12	.0698
R_1R_1	13	.1806	15	.1500	28	.1628
R_2R_2	3	.0417	2	.0200	5	.0291
R_or	1	.0139	1	.0100	2	.0116
rr"	1	.0139			1	.0058
rr'						
$R_1{}^wR_1$	2	.0278	1	.0100	3	.0174
$R_1{}^wr$	1	.0139	1	.0100	2	.0116
$R_1{}^wR_2$	1	.0139	3	.0300	4	.0232
R_1R_z						
$R_1{}^ur$						
$R_2{}^ur$	1	.0139			1	.0058
Total:	72	1.0002	100	1.0000	172	.9998

Gene Complex Frequencies

r	.4325	.4416	.4378
R_1	.3797	.3600	.3684
R_2	.1159	.1650	.1446
R_o	.0119	.0084	.0098
r"	.0161		.0066
r'			
R_z			
$R_1{}^w$	.0278	.0250	.0262
$R_1{}^u$			
$R_2{}^u$	.0161		.0066

TABLE 14b

DISTRIBUTION OF Rh.(D) NEGATIVES - CUMBRIA and SOUTH WEST SCOTLAND

Sample	No. tested	Rh (D) Negatives		Genes	
		No.	Freq.	D	d
Native Cumbrian Donors	198	39	.1970	.5562	.4438
Native Cumbrian Schoolchildren	289	56	.1938	.5598	.4402
Total Native Cumbria	487	95	.1951	.5583	.4417
Native S.W. Scottish Donors	72	19	.2639	.4863	.5137
Resident S.W. Scottish Donors	298	72	.2416	.5085	.4915
Total S.W. Scotland	370	91	.2459	.5041	.4959

TABLE 15 DISTRIBUTION OF LUTHERAN BLOOD GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

Sample	No. Tested	Phenotype				Genes	
		Lu(a+)		Lu(a-)		Lu^a	Lu^b
		No.	Freq.	No.	Freq.		
Manx Donors	198	21	.1061	177	.8939	.0545	.9455
Manx Non Donors	134	16	.1194	118	.8806	.0616	.9384
Total Manx	332	37	.1114	295	.8886	.0573	.9427
Cumbrian Donors	141	12	.0851	129	.9149	.0435	.9565
S.W.Scottish Donors	39	3	.0769	36	.9231	.0392	.9608

TABLE 16 DISTRIBUTION OF KELL BLOOD GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

a) Antigens K and k

	MANX DONORS				MANX NON DONORS				TOTAL MANX			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
KK	-	-	0.39	.0018	-	-	0.54	.0040	-	-	0.81	.0023
Kk	17	.0850	17.66	.0814	17	.1269	15.91	.1187	34	.0969	32.36	.0922
kk	200	.9150	198.95	.9168	117	.8731	117.52	.8770	317	.9031	317.83	.9055
Total:	217	1.0000	217.00	1.0000	134	1.0000	133.97	.9997	351	1.0000	351.00	1.0000
Gene Frequencies												
K		.0425				.0635				.0485		
k		.9575				.9365				.9515		

b) Penney (Kp^a) Antigen

Phenotype	No.	Freq.	No.	Freq.	No.	Freq.
Kp(a+)	10	.0508	3	.0224	13	.0393
Kp(a-)	187	.9492	131	.9776	318	.9607
Total:	197	1.0000	134	1.0000	331	1.0000
Gene Frequencies						
Kp^a		.0257		.0113		.0198
Kp^b		.9743		.9887		.9802

TABLE 16 (cont.) DISTRIBUTION OF KELL BLOOD GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

a) Antigens K and k

	CUMBRIAN DONORS				S.W.SCOTTISH DONORS				TOTAL CUMBRIA & S.W.SCOTLAND			
	Observed		Expected		Observed		Expected		Observed		Expected	
Genotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
KK	–	–	0.24	.0024	–	–	0.12	.0017	–	–	0.37	.0021
Kk	10	.0980	9.51	.0932	6	.0833	5.75	.0799	16	.0920	15.27	.0878
kk	92	.9020	92.25	.9044	66	.9167	66.12	.9183	158	.9080	158.36	.9101
Total	102	1.0000	102.00	1.0000	72	1.0000	71.99	.9999	174	1.0000	174.00	1.0000

Gene Frequencies

K	.0490	.0417	.0460
k	.9510	.9583	.9540

b) Penney (Kp^a) Antigen

No.	Freq.
1	.0139
71	.9861
72	1.0000

Kp^a	.0070
Kp^b	.9930

TABLE 17

DISTRIBUTION OF DUFFY BLOOD GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

a) Tested with anti-Fy^a and anti-Fy^b sera

	Manx Donors				Manx Non-Donors				Total Manx			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Fy^aFy^a	36	.1915	31.13	.1656	27	.2571	22.87	.2178	63	.2150	53.75	.1835
Fy^aFy^b	81	.4309	90.74	.4827	44	.4191	52.27	.4978	125	.4266	143.49	.4897
Fy^bFy^b	71	.3777	66.13	.3518	34	.3238	29.86	.2844	105	.3584	95.76	.3268
Total:	188	1.0001	188.00	1.0001	105	1.0000	105.00	1.0000	293	1.0000	293.00	1.0000
Gene Frequencies												
Fy^a			.4069				.4667				.4283	
Fy^b			.5931				.5333				.5717	

b) Tested with anti-Fy^a serum only

Group	Manx Donors		Manx Non-Donors		Total Manx		Cumbrian Donors		S.W.Scottish Donors		Cumbrian & S.W. Scottish Donors	
	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Fy(a+)	136	.6210	90	.6716	226	.6402	98	.6203	35	.6034	133	.6157
Fy(a-)	83	.3790	44	.3284	127	.3598	60	.3797	23	.3966	83	.3843
Total:	219	1.0000	134	1.0000	353	1.0000	158	1.0000	58	1.0000	216	1.0000
Gene Frequencies												
Fy^a		.3844		.4269		.4002		.3838		.3702		.3801
Fy^b		.6156		.5731		.5998		.6162		.6298		.6199

TABLE 18 DISTRIBUTION OF SERUM HAPTOGLOBIN GROUPS - ISLE OF MAN, CUMBRIA and SOUTH WEST SCOTLAND

	Native Manx Donors				Native Manx Non-Donors				Total Native Manx				Native Cumbrian Donors			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	26	.1182	28.01	.1273	20	.1471	15.45	.1153	46	.1292	43.44	.1227	28	.1414	24.06	.1215
2-1	105	.4773	100.98	.4590	51	.3750	60.10	.4485	156	.4382	161.14	.4552	82	.4141	89.91	.4541
2-2	89	.4045	91.01	.4137	63	.4632	58.44	.4361	152	.4270	149.42	.4221	88	.4444	84.05	.4245
0-0	-	-	-	-	2	.0147	-	-	2	.0056	-	-	-	-	-	-
Total:	220	1.0000	220.00	1.0000	136	1.0000	133.99	.9999	356	1.0000	354.00	1.0000	198	.9999	198.02	1.0001
Gene Frequencies					n= 134				n= 354							
Hp^1		.3568				.3396				.3503				.3485		
Hp^2		.6432				.6604				.6497				.6515		

	Native South West Scottish Donors				Resident S.W.Scottish Donors				Total S.W.Scotland Donors			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	6	.0833	8.34	.1158	58	.1946	46.87	.1578	64	.1730	55.05	.1492
2-1	37	.5139	32.33	.4490	120	.4027	142.23	.4789	157	.4243	174.95	.4741
2-2	29	.4028	31.33	.4351	119	.3993	107.87	.3632	148	.4000	139.00	.3767
0-0	-	-	-	-	1	.0034	-	-	1	.0027	-	-
Total:	72	1.0000	72.00	.9999	298	1.0000	296.97	.9999	370	1.0000	369.00	1.0000
Gene Frequencies					n= 297				n= 369			
Hp^1		.3403				.3973				.3862		
Hp^2		.6597				.6027				.6138		

TABLE 19. DISTRIBUTION OF SERUM TRANSFERRIN GROUPS - ISLE OF MAN, CUMBRIA and SOUTH WEST SCOTLAND

Group	Native Manx Donors		Native Manx Non-Donors		Total Native Manx		Native Cumbrian Donors	
	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
CC	217	.9864	132	.9706	349	.9803	197	.9899
BC	3	.0136	3	.0221	6	.0169	2	.0101
CD	-	-	1	.0074	1	.0028	-	-
Total:	220	1.0000	136	1.0001	356	1.0000	199	1.0000
	Gene Frequencies							
	Tf^B	.0068		.0110		.0084		.0050
	Tf^C	.9932		.9853		.9902		.9950
	Tf^D	-		.0037		.0014		-

Group	Native S.W.Scottish Donors		Resident S.W.Scottish Donors		Total S.W.Scotland		Total Cumbria and S.W. Scotland	
	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
CC	72	1.0000	297	.9966	369	.9973	566	.9947
BC	-	-	1	.0034	1	.0027	3	.0053
CD	-	-	-	-	-	-	-	-
Total:	72	1.0000	298	1.0000	370	1.0000	569	1.0000
	Gene Frequencies							
	Tf^B	-		.0017		.0014		.0026
	Tf^C	1.0000		.9983		.9986		.9974

TABLE 20.

DISTRIBUTION OF BETA-LIPOPROTEIN ALLOTYPE Ag IN THE MANX NATIVE POPULATION

Allotype	No.	Freq.
Ag (x+)	35	.3153
Ag (x-)	76	.6847
Total	111	1.0000

Gene Frequencies

Ag^x	.1725
Ag^y	.8275

TABLE 21. DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS - ISLE OF MAN, CUMBRIA and SOUTH WEST SCOTLAND

	Native Manx Donors				Native Manx Non Donors				Total Native Manx				Native Cumbrian Donors			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	20	.1031	19.50	.1005	20	.1527	17.96	.1371	40	.1231	37.25	.1146	13	.1161	12.89	.1151
B	75	.3866	73.61	.3794	45	.3435	42.37	.3234	120	.3692	115.80	.3563	45	.4018	45.00	.4018
BA	73	.3763	75.77	.3906	50	.3817	55.16	.4211	123	.3785	131.33	.4041	48	.4286	48.18	.4302
CA	10	.0515	8.24	.0425	7	.0534	5.92	.0452	17	.0523	14.20	.0437	2	.0179	2.04	.0182
CB	16	.0825	16.01	.0825	9	.0687	9.10	.0695	25	.0769	25.06	.0771	4	.0357	3.81	.0340
C	-	-	0.87	.0045	-	-	0.48	.0037	-	-	1.36	.0042	-	-	.08	.0007
Total:	194	1.0000	194.00	1.0000	131	1.0000	130.99	1.0000	325	1.0000	325.00	1.0000	112	1.0001	112.00	1.0000
Gene Frequencies																
p^a			.3170				.3702				.3385				.3393	
p^b			.6160				.5687				.5969				.6339	
p^c			.0670				.0611				.0646				.0268	

	Native S.W.Scottish Donors				Resident S.W.Scottish Donors				Total S.W.Scottish				Total Cumbria and S.W.Scotland			
	Obs		Exp.		Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	7	.1061	4.13	.0626	15	.0558	23.48	.0873	22	.0657	27.50	.0821	35	.0783	40.18	.0899
B	36	.5455	32.76	.4964	107	.3978	115.16	.4281	143	.4269	147.80	.4412	188	.4206	192.71	.4311
BA	17	.2576	23.25	.3523	120	.4461	104.02	.3867	137	.4090	127.53	.3807	185	.4139	175.98	.3937
CA	2	.0303	1.50	.0227	9	.0335	7.99	.0297	11	.0328	9.48	.0283	13	.0291	11.69	.0262
CB	4	.0606	4.23	.0641	18	.0669	17.67	.0657	22	.0657	21.94	.0655	26	.0582	25.59	.0572
C	-	-	0.14	.0021	-	-	0.68	.0025	-	-	0.80	.0023	-	-	0.85	.0019
Total:	66	1.0001	66.01	1.0002	269	1.0001	269.00	1.0000	335	1.0001	335.05	1.0001	447	1.0001	447.00	1.0000
Gene Frequencies																
p^a			.2500				.2955				.2866				.2988	
p^b			.7045				.6543				.6642				.6566	
p^c			.0455				.0502				.0493				.0436	

TABLE 22. DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

	Manx Donors				Manx Non-Donors				Total Manx			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	105	.5469	105.77	.5509	65	.5462	65.08	.5469	170	.5466	170.86	.5494
2-1	71	.3698	70.51	.3672	46	.3866	45.85	.3853	117	.3762	116.38	.3742
2-2	12	.0625	11.75	.0612	8	.0672	8.08	.0679	20	.0643	19.81	.0637
7-1	4	.0208	2.96	.0154	–	–	–	–	4	.0129	2.96	.0095
7-2	–	–	.99	.0052	–	–	–	–	–	–	1.00	.0032
7-7	–	–	.02	.0001	–	–	–	–	–	–	–	–
Total:	192	1.0000	192.00	1.0000	119	1.0000	119.01	1.0001	311	1.0000	311.01	1.0000

Gene Frequencies

PGM^1	.7422	.7395	.7412
PGM^2	.2474	.2605	.2524
PGM^7	.0104		.0064

	Cumbrian Donors				S.W. Scottish Donors				Total Cumbria and S.W. Scotland			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	78	.5571	74.32	.5309	16	.5333	14.70	.4900	94	.5529	88.99	.5235
2-1	46	.3286	53.92	.3851	9	.3000	11.90	.3967	55	.3235	65.85	.3874
2-2	14	.1000	9.78	.0699	4	.1333	2.41	.0803	18	.1059	12.18	.0716
7-1	2	.0143	1.45	.0104	1	.0333	0.70	.0233	3	.0177	2.17	.0128
7-2	–	–	0.53	.0038	–	–	0.30	.0100	–	–	0.80	.0047
7-7	–	–	0.01	–	–	–	–	–	–	–	0.01	–
Total:	140	1.0000	140.01	1.0001	30	.9999	30.01	1.0003	170	1.0000	170.00	1.0000

Gene Frequencies

PGM^1	.7286	.7000	.7235
PGM^2	.2643	.2833	.2677
PGM^7	.0071	.0167	.0088

TABLE 23

DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

	Manx Donors				Manx Non-Donors				Total Manx			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	185	.9487	185.14	.9494	120	.9160	119.28	.9105	305	.9356	304.35	.9336
2-1	10	.0513	9.73	.0499	10	.0763	11.45	.0874	20	.0613	21.26	.0652
2-2	-	-	0.13	.0007	1	.0076	0.28	.0021	1	.0031	0.36	.0011
Total:	195	1.0000	195.00	1.0000	131	.9999	131.01	1.0000	326	1.0000	325.97	.9999

Gene Frequencies

AK^1	.9744	.9542	.9662
AK^2	.0256	.0458	.0338

	Cumbrian Donors				S.W.Scottish Donors				Total Cumbrian and S.W.Scotland			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	51	.8947	51.16	.8975	28	.9333	28.04	.9347	79	.9080	79.18	.9101
2-1	6	.1053	5.68	.0997	2	.0667	1.93	.0643	8	.0920	7.64	.0878
2-2	-	-	0.16	.0028	-	-	0.03	.0010	-	-	0.18	.0021
Total:	57	1.0000	57.00	1.0000	30	1.0000	30.00	1.0000	87	1.0000	87.00	1.0000

Gene Frequencies

AK^1	.9474	.9667	.9540
AK^2	.0526	.0333	.0460

TABLE 24. DISTRIBUTION OF RED CELL ADENOSINE DEAMINASE GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

	Manx Donors				Manx Non-Donors				Total Manx			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	171	.8906	170.63	.8887	83	.7830	84.25	.7948	254	.8523	254.70	.8547
2-1	20	.1042	20.74	.1080	23	.2170	20.51	.1935	43	.1443	41.60	.1396
2-2	1	.0052	0.63	.0033	-	-	1.25	.0118	1	.0034	1.70	.0057
Total:	192	1.0000	192.00	1.0000	106	1.0000	106.01	1.0001	298	1.0000	298.00	1.0000
Gene Frequencies												
ADA^1			.9427				.8915				.9245	
ADA^2			.0573				.1085				.0755	

	Cumbrian Donors.				S.W.Scottish Donors				Total Cumbrian and S.W.Scotland			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	40	.9091	40.09	.9111	23	.7667	23.41	.7803	63	.8514	63.41	.8569
2-1	4	.0909	3.82	.0868	7	.2333	6.19	.2063	11	.1486	10.18	.1376
2-2	-	-	0.09	.0020	-	-	0.41	.0137	-	-	0.41	.0055
Total:	44	1.0000	44.00	.9999	30	1.0000	30.01	1.0003	74	1.0000	74.00	1.0000
Gene Frequencies												
ADA^1			.9545				.8833				.9257	
ADA^2			.0455				.1167				.0743	

TABLE 25. DISTRIBUTION OF RED CELL 6 -PHOSPHOGLUCONATE DEHYDROGENASE GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

	Manx Donors				Manx Adult Non Donors				Total Manx			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	184	.9735	184.04	.9738	96	.9057	96.23	.9078	280	.9492	280.19	.9498
CA	5	.0265	4.92	.0260	10	.0943	9.53	.0899	15	.0508	14.60	.0495
C	-	-	0.03	.0002	-	-	0.23	.0022	-	-	0.18	.0006
Total:	189	1.0000	188.99	1.0000	106	1.0000	105.99	.9999	295	1.0000	294.97	.9999
Gene Frequencies												
$6PGD^C$			.0132				.0472				.0254	
$6PGD^A$			.9868				.9528				.9746	

	Cumbrian Donors				S.W.Scottish Donors				Total Cumbrian and S.W.Scotland			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	51	.9444	51.04	.9452	30	1.0000	30	1.0000	81	.9643	81.02	.9645
CA	3	.0556	2.92	.0541	-	-	-	-	3	.0357	2.96	.0352
C	-	-	0.04	.0007	-	-	-	-	-	-	0.03	.0004
	54	1.0000	54.00	1.0000	30	1.0000	30	1.0000	84	1.0000	84.01	1.0001
Gene Frequencies												
$6PGD^C$			.0278				.0000				.0179	
$6PGD^A$			.9722				1.0000				.9821	

TABLE 26

DISTRIBUTION OF RED CELL GLUCOSE-6-PHOSPHO-DEHYDROGENASE (G-6-PD) GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

	Manx Donors				Manx Adult Non-Donors				Total Manx				Cumbrian and S.W. Scottish Donors			
	Obs.		Exp.		Obs.		Exp.		Obs.		Exp.		Obs.		Exp.	
Group.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
B	186	.9947	185.99	.9946	106	1.0000	106	1.0000	292	.9966	292.00	.9966	48	1.0000	48	1.0000
BA	1	.0053	1.01	.0054	-	-	-	-	1	.0034	1.00	.0034	-	-	-	-
A	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total:	187	1.000	187.00	1.0000	106	1.0000	106	1.0000	293	1.0000	293.00	1.0000	48	1.0000	48	1.0000

Gene Frequencies

G-6-PD^{A}	.0027	.0000	.0017	.0000
G-6-PD^{B}	.9973	1.0000	.9983	1.0000

TABLE 27. DISTRIBUTION OF RED CELL LACTIC DEHYDROGENASE, PHOSPHOHEXOSE ISOMERASE and MALATE DEHYDROGENASE GROUPS - MANX, CUMBRIAN and SOUTH WEST SCOTTISH NATIVE POPULATIONS

	Isle of Man		Cumbria		South West Scotland	
Isoenzyme	No.tested	Normal Phenotype	No.tested	Normal Phenotype	No.tested	Normal Phenotype
Lactic Dehydrogenase (LDH)	293	293	30	30	18	18
Phosphohexose Isomerase (PH1)	293	293	30	30	18	18
Malate Dehydrogenase (MDH)	153	153	29	29	30	30

TABLE 28. DISTRIBUTION OF TONGUE CURLERS - MANX and CUMBRIAN NATIVE POPULATIONS

	Manx Adults		Manx Schoolchildren		Total Manx	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.
Curler	171	.6333	279	.7191	450	.6839
Non-Curler	99	.3667	109	.2809	208	.3161
Total:	270	1.0000	388	1.0000	658	1.0000

	Cumbrian Schools		Manx and Cumbrian Schools	
Phenotype	No.	Freq.	No.	Freq.
Curler	169	.6842	448	.7055
Non-Curler	78	.3158	187	.2945
Total:	247	1.0000	635	1.0000

TABLE 29. a) DISTRIBUTION OF COLOUR BLIND MALES - MANX and CUMBRIAN NATIVES

Sample	No.Tested	% Colour Blind.
Manx Adults	128	7.81
Manx Schoolchildren	175	4.00
Total Manx	303	5.61
Cumbrian Schoolchildren	137	2.92
Manx and Cumbrian Schoolchildren	312	3.53
Total Manx and Cumbrian Males	440	4.77

b) DISTRIBUTION OF COLOUR BLIND FEMALES - MANX and CUMBRIAN NATIVES

Sample	No.Tested	% Colour Blind
Manx Adults	130	1.54
Manx Schoolchildren	118	0.85
Total Manx	248	1.21
Cumbrian Schoolchildren	207	0.97
Manx/Cumbrian Schoolchildren	325	0.92
Total Manx and Cumbrian Females	455	1.10

TABLE 30. DISTRIBUTION OF PHENYLTHIOCARBAMIDE (PTC) TASTE THRESHOLDS

MANX and CUMBRIAN NATIVE POPULATIONS

	Manx Adults		Manx Schoolchildren		Total Manx	
Solution Threshold	No.	Freq.	No.	Freq.	No.	Freq.
N.T.	16	.0565	18	.0459	34	.0504
1	45	.1590	71	.1811	116	.1719
3	7	.0247	24	.0612	31	.0459
4	5	.0177	9	.0230	14	.0207
5	7	.0247	13	.0332	20	.0296
6	10	.0353	21	.0536	31	.0459
7	65	.2297	67	.1709	132	.1956
9	117	.4134	128	.3265	245	.3630
11	11	.0389	41	.1046	52	.0770
Total:	283	.9999	392	1.0000	675	1.0000
Phenotype	No.	%	No.	%	No.	%
Non-Tasters	70.5	24.91	117.5	29.97	188	27.85
Tasters	212.5	75.09	274.5	70.03	487	72.15
Total:	283.0	100.00	392.0	100.00	675	100.00
Gene t	.4991		.5474		.5277	

TABLE 30 (cont.) DISTRIBUTION OF PHENYLTHIOCARBAMIDE (PTC) TASTE THRESHOLDS

MANX and CUMBRIAN NATIVE POPULATIONS

	Cumbrian Schoolchildren		Manx and Cumbrian Schoolchildren		Total Isle of Man and Cumbria	
Solution Threshold	No.	Freq.	No.	Freq.	No.	Freq.
N.T.	7	.0212	25	.0346	41	.0408
1	47	.1424	118	.1634	163	.1622
3	22	.0667	46	.0637	53	.0527
4	8	.0242	17	.0236	22	.0219
5	5	.0152	18	.0249	25	.0249
6	6	.0182	27	.0374	37	.0368
7	87	.2636	154	.2133	219	.2179
9	127	.3849	255	.3532	372	.3701
11	21	.0636	62	.0859	73	.0726
Total:	330	1.0000	722	1.0000	1005	.9999
Phenotype	No.	%	No.	%	No.	%
Non-Tasters	80	24.24	197.5	27.35	268	26.67
Tasters	250	75.76	524.5	72.65	737	73.33
Total:	330	100.00	722.0	100.00	1005	100.00
Gene t	.4923		.5230		.5164	

TABLE 31. REGIONS OF THE ISLE MAN - DISTRIBUTION OF ABO BLOOD GROUPS

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A_1	24	.2449	56	.2872	42	.3256	103	.2901
A_2	6	.0612	17	.0872	15	.1163	35	.0986
B	8	.0816	11	.0564	10	.0775	22	.0620
O	59	.6020	106	.5436	59	.4574	185	.5211
A_1B	1	.0102	5	.0256	3	.0232	9	.0254
A_2B	-	-	-	-	-	-	1	.0028
Total	98	.9999	195	1.0000	129	1.0000	355	1.0000
Gene Frequencies								
p_1		.1381		.1647		.1903		.1674
p_2		.0384		.0572		.0808		.0656
q		.0507		.0375		.0549		.0419
r		.7729		.7407		.6740		.7252
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	32	.3200	77	.3869	61	.4586	149	.4060
B	8	.0800	11	.0553	10	.0752	22	.0599
O	59	.5900	106	.5327	59	.4436	185	.5041
AB	1	.0100	5	.0251	3	.0294	10	.0376
Total	100	1.0000	199	1.0000	133	1.0000	367	1.0000
Gene Frequencies								
p		.1850		.2300		.2826		.2452
q		.0502		.0371		.0541		.0412
r		.7648		.7329		.6633		.7136
A: A+O Index.		35.16		42.08		50.83		44.61

TABLE 31 (Cont.) REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF ABO BLOOD GROUPS

	RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.
A_1	31	.3131	79	.3074	110	.3090
A_2	12	.1212	29	.1128	41	.1152
B	9	.0909	14	.0545	23	.0646
O	44	.4444	126	.4903	170	.4775
A_1B	3	.0303	8	.0311	11	.0309
A_2B	-		1	.0039	1	.0028
Total	99	.9999	257	1.0000	356	1.0000
Gene Frequencies						
p^1		.1849		.1790		.1806
p^2		.0853		.0770		.0793
q		.0648		.0382		.0455
r		.6650		.7058		.6945
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.
A	46	.4510	116	.4361	162	.4402
B	9	.0882	14	.0526	23	.0625
O	44	.4314	126	.4737	170	.4620
AB	3	.0294	10	.0376	13	.0353
Total	102	1.0000	266	1.0000	368	1.0000
Gene Frequencies						
p		.2816		.2679		.2716
q		.0638		.0375		.0448
r		.6546		.6946		.6836
A: A+O Index		51.11		47.93		48.80

TABLE 32. REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF MN BLOOD GROUPS

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MM	21	.2442	51	.2802	37	.2913	83	.2470
MN	43	.5000	95	.5220	62	.4882	182	.5417
NN	22	.2558	36	.1978	28	.2205	71	.2113
Total:	86	1.0000	182	1.0000	127	1.0000	336	1.0000
Gene Frequencies								
M		.4942		.5412		.5354		.5179
N		.5058		.4588		.4646		.4821

	RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.
MM	27	.2755	62	.2480	89	.2557
MN	48	.4898	139	.5560	187	.5374
NN	23	.2347	49	.1960	72	.2069
Total:	98	1.0000	250	1.0000	348	1.0000
Gene Frequencies						
M		.5204		.5260		.5244
N		.4796		.4740		.4756

TABLE 33 REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF THE MNSs BLOOD GROUPS

(I) Tested with 4 antisera

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MMSS	-	-	5	.0515	6	.0759	7	.0454
MMSs	7	.1750	14	.1443	6	.0759	16	.1039
MMss	3	.0750	8	.0825	12	.1519	15	.0974
MNSS	2	.0500	3	.0309	1	.0127	4	.0260
MNSs	10	.2500	23	.2371	19	.2405	40	.2597
MNss	12	.3000	31	.3196	19	.2405	37	.2403
NNSS	-	-	-	-	1	.0127	1	.0065
NNSs	1	.0250	1	.0103	-	-	5	.0325
NNss	5	.1250	12	.1237	15	.1899	29	.1883
Total	40	1.0000	97	.9999	79	1.0000	154	1.0000
Gene Frequencies								
MS	.2302		.2615		.2283		.2218	
Ms	.3198		.3107		.3223		.2879	
NS	.0448		.0169		.0312		.0542	
Ns	.4052		.4109		.4182		.4361	

(II) Tested with 3 antisera

Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
MMS	8	.1311	21	.1479	13	.1066	36	.1286
MMss	5	.0820	15	.1056	23	.1885	26	.0929
MNS	15	.2459	37	.2606	30	.2459	84	.3000
MNss	18	.2951	42	.2958	29	.2377	73	.2607
NNS	3	.0492	4	.0282	3	.0246	14	.0500
NNss	12	.1967	23	.1620	24	.1967	47	.1679
Total	61	.9999	142	1.0001	122	1.0000	280	1.0001
Gene Frequencies								
MS	.1852		.1995		.1457		.1940	
Ms	.2984		.3322		.3912		.3078	
NS	.0573		.0499		.0651		.0839	
Ns	.4591		.4184		.3980		.4143	

TABLE 33 (Cont.) REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF THE MNSs BLOOD GROUPS

(I) Tested with 4 antisera

	RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.
MMSS	3	.0556	7	.0614	10	.0595
MMSs	4	.0741	9	.0789	13	.0774
MMss	9	.1667	12	.1053	21	.1250
MNSS	1	.0185	2	.0175	3	.0179
MNSs	16	.2963	30	.2632	46	.2738
MNss	10	.1852	25	.2193	35	.2083
NNSS	1	.0185	1	.0088	2	.0119
NNSs	-	-	4	.0351	4	.0238
NNss	10	.1852	24	.2105	34	.2024
Total	54	1.0001	114	1.0000	168	1.0000
Gene Frequencies						
MS		.2237		.2198		.2207
Ms		.3226		.2758		.2912
NS		.0540		.0565		.0561
Ns		.3997		.4479		.4320

(II) Tested with 3 antisera

Phenotype	No.	Freq.	No.	Freq.	No.	Freq.
MMS	8	.0860	28	.1279	36	.1154
MMss	18	.1935	21	.0959	39	.1250
MNS	27	.2903	69	.3151	96	.3077
MNss	18	.1935	55	.2511	73	.2340
NNS	3	.0323	11	.0502	14	.0449
NNss	19	.2043	35	.1598	54	.1731
Total	93	.9999	219	1.0000	312	1.0001
Gene Frequencies						
MS		.1417		.1969		.1768
Ms		.3798		.3100		.3344
NS		.0893		.0912		.0937
Ns		.3892		.4019		.3951

TABLE 34a REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF Rh BLOOD GROUPS

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE	
Rh Type	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
R_1r	18	.2727	51	.3355	47	.3852	96	.3276
rr	12	.1818	29	.1908	21	.1721	61	.2082
R_1R_2	11	.1667	19	.1250	21	.1721	39	.1331
R_2r	8	.1212	17	.1118	13	.1066	36	.1229
R_1R_1	8	.1212	21	.1382	12	.0984	44	.1502
R_2R_2	4	.0606	6	.0395	4	.0328	9	.0307
R_or	2	.0303	3	.0197	2	.0164	5	.0171
rr″			1	.0066	1	.0082		
rr′			2	.0132	1	.0082		
R^WR_1	2	.0303	2	.0132			2	.0068
R_1R_z	1	.0152	1	.0066			1	.0034
Total:	66	1.0000	152	1.0001	122	1.0000	293	1.0000
Gene Complex Frequencies								
r		.3897		.4576		.4227		.4355
R_1		.3517		.3338		.3715		.3831
R_2		.1927		.1173		.1666		.1561
R_o		.0312		.0231		.0198		.0176
r″				.0072		.0097		-
r′				.0144		.0097		-
R_z		.0195		.0400				.0043
R_1^W		.0152		.0066				.0034

TABLE 34a (cont.) REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF Rh BLOOD GROUPS

	RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Rh Type	No.	Freq.	No.	Freq.	No.	Freq.
R_1r	32	.3441	78	.3436	110	.3438
rr	17	.1828	49	.2159	66	.2063
R_1R_2	17	.1828	28	.1233	45	.1406
R_2r	12	.1290	28	.1233	40	.1250
R_1R_1	9	.0968	36	.1586	45	.1406
R_2R_2	4	.0430	5	.0220	9	.0281
R_or	1	.0108	3	.0132	4	.0125
rr″	-	-	-	-	-	-
rr′	1	.0108	-	-	1	.0031
$R_1{}^WR_1$	-	-	-	-	-	-
R_1R_z	-	-	-	-	-	-
Total:	93	1.0001	227	.9999	320	1.0000

Gene Complex Frequencies

r	.4232	.4491	.4415
R_1	.3528	.3921	.3809
R_2	.1989	.1453	.1609
R_o	.0123	.0135	.0132
r″	-	-	-
r′	.0128	-	.0035
R_z	-	-	-
$R_1{}^W$	-	-	-

TABLE 34b. REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF Rh (D) NEGATIVES

Region	No. tested	Rh (D) Negative No.	Rh (D) Negative Freq.
Douglas	66	12	.1818
Total Urban	179	38	.2123
Northside	131	25	.1908
Southside	365	74	.2027
Rural Northside	100	20	.2000
Rural Southside	265	59	.2226
Total Rural	365	79	.2164

TABLE 35. REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF LUTHERAN BLOOD GROUPS

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE		RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Lu(a+)	3	.0833	9	.1000	10	.1351	12	.0844	10	.1408	9	.0849	19	.1073
Lu(a-)	33	.9167	81	.9000	64	.8649	130	.9156	61	.8592	97	.9151	158	.8927
Total:	36	1.0000	90	1.0000	74	1.0000	142	1.0000	71	1.0000	106	1.0000	177	1.0000
Gene Frequencies														
Lu^a		.0426		.0513		.0700		.0431		.0731		.0434		.0552
Lu^b		.9574		.9487		.9300		.9569		.9269		.9566		.9448

TABLE 36 REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF KELL BLOOD GROUP SYSTEM

a) K and k antigens

Phenotype	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE		RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
	No.	Freq.	No.	Freq.	No	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
KK	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Kk	2	.0500	6	.0618	7	.0921	9	.0584	5	.0980	7	.0614	12	.0727
kk	38	.9500	91	.9382	69	.9079	145	.9416	46	.9020	107	.9386	153	.9273
Total:	40	1.0000	97	1.0000	76	1.0000	154	1.0000	51	1.0000	114	1.0000	165	1.0000
Gene Frequencies														
K	.0250		.0309		.0461		.0292		.0490		.0307		.0363	
k	.9750		.9691		.9539		.9708		.9510		.9693		.9637	

b) Kp^a antigen

Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Kp(a+)	3	.0833	3	.0333	4	.0533	4	.0284	4	.0556	1	.0095	5	.0282
Kp(a-)	33	.9167	87	.9667	71	.9467	137	.9716	68	.9444	104	.9905	172	.9718
Total:	36	1.0000	90	1.0000	75	1.0000	141	1.0000	72	1.0000	105	1.0000	177	1.0000
Gene Frequencies														
Kp^a	.0426		.0168		.0270		.0143		.0282		.0048		.0142	
Kp^b	.9574		.9832		.9730		.9857		.9718		.9952		.9858	

TABLE 37 REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF DUFFY BLOOD GROUPS

a) Tested with anti-Fy^a and anti-Fy^b

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE		RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Fy^aFy^a	7	.2059	20	.2500	15	.2344	21	.2100	10	.2128	14	.2121	24	.2124
Fy^aFy^b	15	.4412	32	.4000	28	.4375	37	.3700	18	.3830	22	.3333	40	.3540
Fy^bFy^b	12	.3529	28	.3500	21	.3281	42	.4200	19	.4042	30	.4545	49	.4336
Total:	34	1.0000	80	1.0000	64	1.0000	100	1.0000	47	1.0000	66	.9999	113	1.0000
Gene Frequencies														
Fy^a		.4265		.4500		.4531		.3950		.4043		.3788		.3894
Fy^b		.5735		.5500		.5469		.6050		.5957		.6212		.6106

b) Tested with anti-Fy^a only

Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Fy(a+)	26	.6500	65	.6701	54	.6835	69	.5897	33	.6111	43	.5584	76	.5802
Fy(a-)	14	.3500	32	.3299	25	.3165	48	.4103	21	.3889	34	.4416	55	.4198
Total:	40	1.0000	97	1.0000	79	1.0000	117	1.0000	54	1.0000	77	1.0000	131	1.0000
Gene Frequencies														
Fy^a		.4084		.4256		.4374		.3595		.3764		.3355		.3521
Fy^b		.5916		.5744		.5626		.6405		.6236		.6645		.6479

TABLE 38 REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF SERUM HAPTOGLOBIN GROUPS

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE		RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	7	.1750	14	.1443	9	.1125	18	.1184	5	.0909	11	.0982	16	.0958.
2-1	15	.3750	40	.4124	40	.5000	64	.4211	30	.5455	49	.4375	79	.4731
2-2	18	.4500	43	.4443	31	.3875	70	.4605	20	.3636	52	.4643	72	.4311
0-0	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total:	40	1.0000	97	1.0000	80	1.0000	152	1. 0000	55	1.0000	112	1.0000	167	1.0000

Gene Frequencies

Hp^1	.3625	.3505	.3625	.3289	.3636	.3170	.3323
Hp^2	.6375	.6495	.6375	.6711	.6364	.6830	.6677

TABLE 39 REGIONS OF THE ISLE OF MAN – DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE		RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
A	3	.0882	11	.1250	9	.1200	15	.1128	5	.0980	12	.1212	17	.1133
B	10	.2941	32	.3636	33	.4400	46	.3459	22	.4314	36	.3636	58	.3867
BA	18	.5294	36	.4090	25	.3333	57	.4286	17	.3333	39	.3939	56	.3733
CA	1	.0294	3	.0341	3	.0400	6	.0451	3	.0588	5	.0505	8	.0533
CB	2	.0588	6	.0682	5	.0667	9	.0677	4	.0784	7	.0707	11	.0733
C	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Total:	34	.9999	88	.9999	75	1.0000	133	1.0001	51	.9999	99	.9999	150	.9999

Gene Frequencies

pa	.3677	.3466	.3067	.3496	.2941	.3434	.3267
pb	.5882	.6023	.6400	.5940	.6373	.5960	.6100
p^C	.0441	.0511	.0533	.0564	.0686	.0606	.0633

TABLE 40 REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 GROUPS

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE		RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Phenotype	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
1-1	20	.6061	41	.5062	32	.4848	73	.5659	25	.5556	53	.5521	78	.5532
2-1	9	.2727	32	.3951	31	.4697	44	.3411	19	.4222	35	.3646	54	.3830
2-2	3	.0909	7	.0864	3	.0455	10	.0755	1	.0222	7	.0729	8	.0567
7-1	1	.0303	1	.0123	-	-	2	.0155	-	-	1	.0104	1	.0071
Total:	33	1.0000	81	1.0000	66	1.0000	129	1.0000	45	1.0000	96	1.0000	141	1.0000

Gene Frequencies

PGM_1^1	.7576	.7099	.7197	.7423	.7667	.7396	.7482
PGM_1^2	.2273	.2840	.2803	.2500	.2333	.2552	.2482
PGM_1^7	.0151	.0062		.0077		.0052	.0036

TABLE 41. REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF TONGUE CURLERS

	DOUGLAS		TOTAL URBAN		NORTHSIDE		SOUTHSIDE		RURAL NORTHSIDE		RURAL SOUTHSIDE		TOTAL RURAL	
Phenotype	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Curler	61	64.89	121	67.22	66	67.35	238	67.42	48	63.16	177	68.34	225	67.16
Non-Curler	33	35.11	59	32.78	32	32.65	115	32.58	28	36.84	82	31.66	110	32.84
Total:	94	100.00	180	100.00	98	100.00	353	100.00	76	100.00	259	100.00	335	100.00

TABLE 42. REGIONS OF THE ISLE OF MAN - DISTRIBUTION OF PHENYLTHIOCARBAMIDE (PTC)TASTE THRESHOLDS

Solution No.	DOUGLAS No.	%	TOTAL URBAN No.	%	NORTHSIDE No.	%	SOUTHSIDE No.	%	RURAL NORTHSIDE No.	%	RURAL SOUTHSIDE No.	%	TOTAL RURAL No.	%
N.T.	6	6.32	12	6.52	7	6.80	19	5.28	5	6.25	13	4.90	18	5.22
1	19	20.00	35	19.02	20	19.42	60	16.67	15	18.75	41	15.47	56	16.23
3	4	4.21	7	3.80	3	2.91	20	5.56	2	2.50	16	6.04	18	5.22
4	1	1.05	2	1.09	1	0.97	5	1.39	1	1.25	4	1.51	5	1.45
5	2	2.11	5	2.72	5	4.85	10	2.78	5	6.25	8	3.02	13.	3.77
6	2	2.11	7	3.80	5	4.85	12	3.33	4	5.00	10	3.77	14.	4.06
7	23	24.21	39	21.20	17	16.50	81	22.50	10	12.50	58	21.89	68	19.71
9	31	32.63	61	33.15	34	33.01	126	35.00	29	36.25	95	35.85	124	35.94
11	7	7.37	16	8.70	11	10.68	27	7.50	9	11.25	20	7.55	29.	8.41
Total	95	100.01	184	100.00	103	99.99	360.	100.01	80	100.00	265	100.00	345	100.01
Group	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Non-Tasters	29.5	31.05	55	29.89	30.5	29.61	101.5	28.19	22.5	28.13	72	27.17	94.5	27.39
Tasters	65.5	68.95	129	70.11	72.5	70.39	258.5	71.81	57.5	71.87	193.	72.83	250.5	72.61
Total	95.0	100.00	184	100.00	103.0	100.00	360.0	100.00	80.0	100.00	265	100.00	345.0	100.00

TABLE 43. DISTRIBUTION OF ABO BLOOD GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES

Sample	Number Tested	Phenotypes A		B		O		AB		A:A+O Index	Author(s)
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.		
Isle of Man											
Manx Donors	219	79	.3607	13	.0594	119	.5434	8	.0365	39.90	Present Study
Manx Non-Donors	590	271	.4593	45	.0763	257	.4356	17	.0288	51.33	Present Study
Total Manx	809	350	.4326	58	.0717	376	.4648	25	.0309	48.21	Present Study
Northern England											
Cumbria	539	242	.4490	48	.0891	235	.4360	14	.0260	50.73	Present Study
N.W.Cumbria	5569	1975	.3546	509	.0914	2942	.5283	143	.0257	40.17	Fraser Roberts (1953)
S.Cumbria	2342	934	.3988	214	.0914	1128	.4816	66	.0282	45.30	Fraser Roberts (1953)
Cumbria	7911	2909	.3677	723	.0914	4070	.5145	209	.0264	41.68	Fraser Roberts (1953)
Cumbria	4446	1746	.3927	364	.0819	2218	.4989	118	.0265	44.05	Kopeć (1970)
Furness	2587	1054	.4074	237	.0916	1200	.4639	96	.0371	46.76	Kopeć (1970)
Final Area 3	19937	7943	.3984	1595	.0800	9833	.4932	566	.0284	44.68	Kopeć (1970)
Final Area 9	18533	7730	.4171	1377	.0743	8937	.4822	489	.0264	46.38	Kopeć (1970)
N.England	8716	3516	.4034	744	.0854	4236	.4860	220	.0252	45.36	Fisher & Taylor (1940)
Scotland											
N.Scotland	961	289	.3007	89	.0926	560	.5827	23	.0239	34.04	Brown (1965)
Shetland Isles	146	47	.3219	21	.1438	75	.5137	3	.0205	38.52	Brown (1965)
Orkney Isles	154	51	.3312	21	.1364	70	.4545	12	.0779	42.15	Brown (1965)
N.Scotland & Islands	1261	387	.3069	131	.1039	705	.5591	38	.0301	35.44	Brown (1965)
N.Scotland & Stornoway	3787	1323	.3494	463	.1223	1894	.5001	107	.0283	41.13	Kirkpatrick (1952)
N.Scotland & Islands	5048	1710	.3387	594	.1177	2599	.5149	145	.0287	39.68	Kirkpatrick(1952) Brown(1965)
S.W.Scotland	271	105	.3875	34	.1255	120	.4428	12	.0443	46.67	Present Study
S.W.Scotland	612	224	.3660	77	.1258	295	.4820	16	.0262	43.16	Kopeć (1970)
C. & S.W.Scotland	6011	2115	.3511	654	.1087	3063	.5096	179	.0298	40.85	Struthers (1951)

TABLE 43.(contd)

Sample	Number Tested	Phenotypes A		B		O		AB		A:A+O Index	Author(s)
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.		
Wales											
N.Wales	2550	1009	.3957	234	.0918	1224	.4800	83	.0325	45.19	Fraser Roberts (1942)
N.Wales (Welsh Names)	1132	404	.3569	115	.1016	580	.5124	33	.0291	41.06	Fraser Roberts (1942)
N.Wales	3447	1306	.3789	353	.1024	1689	.4900	99	.0287	43.61	Kopeć (1970)
Final Area 13	7062	2655	.3760	737	.1044	3430	.4857	240	.0339	43.63	Kopeć (1970)
Ireland											
Ulster	10784	3742	.3469	1116	.1035	5612	.5204	314	.0291	40.00	Hart (1944)
Ulster	1473	441	.2994	151	.1025	846	.5743	35	.0238	34.27	Hackett & Dawson (1958)
Ulster	29143	9702	.3329	2838	.0974	15826	.5430	777	.0267	38.01	Kopeć (1970)
Dublin	16865	5549	.3290	1916	.1136	8969	.5318	431	.0256	38.22	Dawson(1952)
Dublin	9867	3320	.3365	1045	.1059	5209	.5279	293	.0297	38.93	Dawson & Hackett (1958)
Dublin	36878	11919	.3232	3926	.1065	19981	.5418	1052	.0285	37.36	Dawson (1964)
Co.Louth	6176	1896	.3070	594	.0962	3557	.5759	129	.0209	34.77	Dawson (1964)
Co.Wicklow	3198	1136	.3552	339	.1060	1627	.5088	96	.0300	41.11	Dawson (1964)
Co.Wexford	4928	1707	.3464	494	.1002	2593	.5262	134	.0272	39.70	Dawson (1964)
Leinster Province	76057	24359	.3184	8237	.1077	41830	.5467	2081	.0272	36.80	Dawson (1964)
Eire	295	70	.238	37	.125	182	.617	6	.020	27.78	Palsson et al (1970)

TABLE 44. DISTRIBUTION OF SECRETOR GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES AND ICELAND

Sample	Number Tested	Phenotypes				Gene Frequencies		Author(s)
		Se(-)		Se(+)		Se	se	
		No.	Freq.	No.	Freq.			
Isle of Man	163	47	.2883	116	.7117	.4630	.5370	Present Study
England								
Cumbria	128	37	.2891	91	.7109	.4624	.5376	Present Study
Liverpool	1118	254	.2272	864	.7728	.5233	.4767	McConnell unpublished in Race & Sanger(1970)
London	284	69	.2430	215	.7570	.5071	.4929	Lincoln & Dodd (1973)
England	2435	590	.2423	1845	.7577	.5078	.4922	Horwich et al (1966)
Scotland								
Aberdeen	510	152	.2980	358	.7020	.4541	.5459	Lincoln & Dodd (1973)
Ireland								
Belfast I	531	141	.2655	390	.7345	.4847	.5153	Dodge(1967)
Belfast II	532	160	.3008	372	.6992	.4516	.5484	Lincoln and Dodd(1973)
Dublin	435	140	.3218	295	.6782	.4327	.5673	Lincoln and Dodd(1973)
Europe								
Iceland	228	94	.4123	134	.5877	.3579	.6421	Bjarnason et al (1973)

TABLE 45. DISTRIBUTION OF MN BLOOD GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES

Sample	Number Tested	Phenotypes M No.	M Freq.	MN No.	MN Freq.	N No.	N Freq.	Gene Frequencies M	N	Author(s)
Isle of Man	689	178	.2583	373	.5414	138	.2003	.5290	.4710	Present Study
England.										
Cumbria	515	156	.3029	244	.4738	115	.2233	.5398	.4602	Present Study
England	1419	402	.2833	701	.4940	316	.2227	.5303	.4697	Race and Sanger (1970)
England	1166	343	.2942	567	.4863	256	.2196	.5373	.4627	Ikin et al (1952)
England	1000	298	.2980	489	.4890	213	.2130	.5425	.4575	Cleghorn (1960)
Scotland										
S.W.Scotland	172	55	.3198	82	.4767	35	.2035	.5582	.4418	Present Study
Scotland	527	142	.2694	284	.5389	101	.1917	.5389	.4611	Ikin et al (1952)
Wales										
Wales	116	36	.3103	54	.4655	26	.2241	.5431	.4569	Ikin et al (1952)
Ireland										
Ulster	106	37	.3491	54	.5094	15	.1415	.6037	.3963	Ikin et al (1952)
Carnew,Co.Wicklow	175	figures unavailable						.529	.471	Sunderland et al (1973)
Co.Wicklow	58	"		"				.621	.379	Sunderland et al (1973)
Rossmore,Co.Cork	157	"		"				.637	.363	Sunderland et al (1973)
Co.Cork	76	"		"				.501	.499	Sunderland et al (1973)
Ireland	>2000	"		"				.561	.439	Sunderland et al (1973)
Eire	104	38	.3654	49	.4712	17	.1635	.6010	.3990	Hackett and Dawson (1958)
Eire	295	123	.4169	118	.4000	54	.1831	.6169	.3831	Palsson et al (1970)

TABLE 46. DISTRIBUTION OF MNSs BLOOD GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES

Sample	Number Tested	Phenotypes MMS		MMss		MNS		MNss		NNS		NNss	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
Isle of Man	593	80	.1349	62	.1046	176	.2968	153	.2580	22	.0371	100	.1686
England													
Cumbria	492	102	.2073	51	.1037	126	.2561	103	.2093	27	.0549	83	.1687
England I	1419	295	.2079	107	.0754	379	.2671	322	.2269	102	.0719	214	.1508
England II	1166	230	.1973	113	.0969	303	.2599	264	.2264	56	.0480	200	.1715
England III	1000	197	.1970	101	.1010	263	.2630	226	.2260	57	.0570	156	.1560
Scotland													
S.W.Scotland	172	39	.2267	16	.0930	48	.2791	34	.1977	5	.0291	30	.1744
Scotland	527	105	.1992	37	.0702	139	.2638	145	.2751	22	.0417	79	.1499
Wales													
Wales	116	20	.1724	16	.1379	36	.3103	18	.1551	9	.0776	17	.1466
Ireland													
Ulster	106	23	.2170	14	.1321	17	.1604	37	.3491	4	.0377	11	.1038
Carnew,Co.Wicklow	175			- figures unavailable -									
Co.Wicklow	58			"		"							
Rossmore,Co.Cork	157			"		"							
Co.Cork	76			"		"							
Ireland	>2000			"		"							
Eire I	295	85	.2881	38	.1288	58	.1966	60	.2034	17	.0576	37	.1254
Eire II	104	31	.2981	7	.0673	25	.2404	24	.2308	6	.0577	11	.1058

TABLE 46 (contd) DISTRIBUTION OF MNSs BLOOD GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES

Sample	Chromosomes				Author(s)
	MS	Ms	NS	Ns	
Isle of Man	.1930	.3239	.0782	.4049	Present Study
England					
Cumbria	.2370	.3067	.0690	.3873	Present Study
England I	.2472	.2831	.0802	.3895	Race and Sanger (1970)
England II	.2402	.2971	.0564	.4063	Ikin et al (1952)
England III	.2371	.3054	.0709	.3866	Cleghorn (1960) in Race and Sanger(1970)
Scotland					
S.W.Scotland	.2809	.2772	.0330	.4089	Present Study
Scotland	.2465	.2924	.0498	.4113	Ikin et al (1952)
Wales					
Wales	.2272	.3159	.1097	.3472	Ikin et al (1952)
Ireland					
Ulster	.1889	.4148	.0463	.3500	Ikin et al (1952)
Carnew, Co.Wicklow	.193	.336	.055	.416	Sunderland et al (1973)
Co.Wicklow	.275	.346	.027	.352	Sunderland et al (1973)
Rossmore,Co.Cork	.259	.378	.024	.339	Sunderland et al (1973)
Co.Cork	.262	.239	.064	.435	Sunderland et al (1973)
Ireland	.268	.293	.058	.381	Sunderland et al (1973)
Eire I	.277	.340	.047	.336	Palsson et al (1970)
Eire II	figures not available				Hackett and Dawson (1958)

TABLE 47 DISTRIBUTION OF P BLOOD GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES

Sample	Number Tested	Phenotypes $P_1(+)$ No.	$P_1(+)$ Freq.	$P_1(-)$ No.	$P_1(-)$ Freq.	Gene Frequency P_1	Author(s)
Isle of Man	336	245	.7292	91	.2708	.4796	Present Study
England							
Cumbria	22	11	.5000	11	.5000	.2929	Present Study
England	500	370	.7400	130	.2600	.4901	Sanger et al (1949)
England	475	347	.7305	128	.2695	.4809	Bertinshaw et al (1950)
England	1166	893	.7659	273	.2341	.5162	Ikin et al (1952)
England	484	374	.7727	110	.2273	.5232	Stratton (1953)
Scotland							
S.W.Scotland	72	56	.7778	16	.2222	.5286	Present Study.
Scotland	527	398	.7552	129	.2448	.5052	Ikin et al (1952)
Wales							
Wales	116	85	.7328	31	.2672	.4830	Ikin et al (1952)
Ireland							
Ulster	106	83	.7830	23	.2170	.5342	Ikin et al (1952)
Carnew,Co.Wicklow	175	figures not available				.493	Sunderland et al (1973)
Co.Wicklow	58	"	"	"		.475	Sunderland et al (1973)
Rossmore,Co.Cork	157	"	"	"		.406	Sunderland et al (1973)
Co.Cork	76	"	"	"		.467	Sunderland et al (1973)
Ireland	>2000	"	"	"		.481	Sunderland et al (1973)
Eire	95	68	.7158	27	.2842	.4669	Hackett and Dawson (1958)
Eire	295	133	.4508	162	.5492	.2589	Palsson et al (1970)

TABLE 48a DISTRIBUTION OF Rh TYPES IN THE MANX AND ENGLISH POPULATIONS

Tested with anti -D-C-E-c-e and $-C^W$ sera

	Manx Present Data		English Race & Sanger (1954)	
Rh.Type	No.	Freq.	No.	Freq.
R_1r	218	.3488	589	.3276
R_1^Wr	-	-	17	.0095
R_1R_1	78	.1248	309	.1719
$R_1^WR_1$	2	.0032	18	.0100
R_1R_2	85	.1360	246	.1368
$R^W_1R_2$	2	.0032	11	.0061
R_2^r	80	.1280	217	.1207
R_2R_2	22	.0352	38	.0211
R_1R_Z	2	.0032	4	.0022
R_or	10	.0160	39	.0217
rr	119	.1904	281	.1563
rr'	3	.0048	17	.0095
rr"	4	.0064	12	.0067
Total:	625	1.0000	1798	1.0000

TABLE 48b DISTRIBUTION OF Rh TYPES IN SELECTED POPULATIONS OF THE BRITISH ISLES

Tested with anti-D-C-E, and -c sera

	Manx Present Study		English Murray (1946)		English Fisher & Race (1946)		Irish Huth (1953)		Irish Palsson et al (1970)	
Rh. Type	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.
CCDE	2	.0032	1	.0010	1	.0011	1	.0050		
CCDee	80	.1280	215	.2071	183	.1974	37	.1850	54	.183
CcDE	87	.1392	119	.1146	126	.1359	21	.1050	31	.105
CcDee	218	.3488	354	.3410	326	.3516	68	.3400	81	.275
Ccddee	3	.0048	6	.0058	6	.0065	1	.0050	5	.017
ccDE	102	.1632	153	.1474	113	.1219	21	.1050	56	.189
ccDee	10	.0160	24	.0231	23	.0248	7	.0350	14	.048
ccddE	4	.0064	7	.0067	12	.0129	1	.0050	2	.007
ccddee	119	.1904	159	.1532	137	.1478	43	.2150	52	.176
Total:	625	1.0000	1038	.9999	927	.9999	200	1.0000	295	1.000

TABLE 49 FREQUENCY DISTRIBUTION OF Rh GENE COMPLEXES IN SELECTED POPULATIONS OF THE BRITISH ISLES

Gene Complex		Tested with anti-D,-C,-E; not -C^w sera							Not tested with anti. -C^w		
		Isle of Man Present Study	Cumbria Present Study	England Race & Sanger (1954)	S.W. Scotland Present Study	Carnew, Co. Wicklow Eire	Co. Wicklow Eire	Rosmore Co. Cork Eire	Co. Cork Eire	Eire	Eire Huth et al (1953)
		Sunderland et al (1973)									
R_1	CDe	.3643	.4097	.4015	.3684	.463	.483	.429	.421	.3862	.3913
r	cde	.4353	.4313	.3903	.4378	.352	.345	.408	.347	.4236	.4255
R_2	cDE	.1617	.1164	.1450	.1512	.146	.172	.127	.204	.1577	.1436
R_o	cDe	.0180	.0167	.0262	.0098	.028	–	.016	.028	.0138	.0108
R_1^w	C^wDe	.0032	.0051	.0128	.0262	.011	–	.006	–	.0111	–
r''	cdE	.0074	.0095	.0097	.0066	–	–	.014	–	.0036	.0230
r'	Cde	.0056	.0088	.0119	–	–	–	–	–	.0038	.0058
R_z	CDE	.0045	.0025	.0027	–	–	–	–	–	.0002	–
Total:		1.0000	1.0000	1.0001	1.0001	1.000	1.000	1.000	1.000	1.0000	1.0000

TABLE 49 (cont)

FREQUENCY DISTRIBUTION OF Rh GENE COMPLEXES IN SELECTED POPULATIONS OF THE BRITISH ISLES.

Tested with anti -D, -C_x, -E_x and -c only

Gene Complex		England Murray (1946)	England Fisher & Race (1946)	Eire Huth (1953)	Eire Palsson et al (1970)
R_1	CDe	.4307	.4361	.4044	.364
r	cde	.3885	.3790	.4393	.402
R_2	cDE	.1365	.1280	.1054	.156
R_o	cDe	.0283	.0305	.0344	.052
R_1^w	C^wDe	-	-	-	-
r''	cdE	.0079	.0170	.0060	.008
r'	Cde	.0071	.0081	.0051	.018
R^z	CDE	.0010	.0013	.0055	-
Total:		1.0000	1.0000	1.0000	1.0000

TABLE 50. DISTRIBUTION OF Rh (D) NEGATIVES IN SELECTED POPULATIONS OF THE BRITISH ISLES

Population	No. Tested	Rh(D) Negative		Gene Frequencies		Author(s)
		No.	Freq.	D	d	
Isle of Man	803	159	.1980	.5550	.4450	Present Study
England						
Cumbria	487	95	.1938	.5583	.4417	Present Study
Cumbria	4446	843	.1896	.5646	.4354	Kopeć (1970)
Furness	2587	516	.1995	.5533	.4467	Kopeć (1970)
England	10000	1722	.1722	.5850	.4150	Discombe(1952) cited in Mourant (1954)
Scotland						
South West Scotland	72	19	.2639	.4863	.5137	Present Study
South West Scotland	370	91	.2459	.5041	.4959	Present Study
South West Scotland	612	121	.1977	.5554	.4446	Kopeć (1970)
North Scotland	961	188	.1956	.5577	.4423	Brown (1965)
Shetland Isles	146	25	.1712	.5862	.4138	Brown (1965)
Orkney Isles	154	33	.2143	.5371	.4629	Brown (1965)
Scotland(Aberdeen)	3601	619	.1719	.5854	.4146	Allan (1949)
Wales						
North Wales	2658	498	.1874	.5671	.4329	Kopeć (1970)
South West Wales	1310	239	.1824	.5729	.4271	Kopeć (1970)
Wales	1122	173	.1542	.6073	.3927	Hoare (1943)
Ireland						
Ulster	25257	4285	.1697	.5881	.4119	Kopeć (1970)
Dublin	4058	659	.1624	.5970	.4030	Stewart (1947)
Dublin	9867	1669	.1691	.5888	.4112	Dawson and Hackett (1958)
Leinster	76507	13261	.1733	.5837	.4163	Dawson (1964)

TABLE 51. DISTRIBUTION OF LUTHERAN BLOOD GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES

Sample	Number Tested	Phenotype				Gene Frequency	Author(s)
		Lu (a+)		Lu (a-)		Lu^a	
		No.	Freq.	No.	Freq.		
Isle of Man	332	37	.1114	295	.8886	.0573	Present Study
England							
Cumbria	141	12	.0851	129	.9149	.0435	Present Study
England	1373	105	.0765	1268	.9235	.0390	cited by Race & Sanger (1970)
England	1166	71	.0609	1095	.9391	.0309	Ikin et al (1952)
Scotland							
South West Scotland	39	3	.0769	36	.9231	.0392	Present Study
Scotland	527	29	.0550	498	.9450	.0279	Ikin et al (1952)
Wales							
Wales	116	1	.0086	115	.9914	.0043	Ikin et al (1952)
Ireland							
Ulster	106	9	.0849	97	.9151	.0434	Ikin et al (1952)
Carnew,Co.Wicklow	175	figures not available				.012	Sunderland et al (1973)
Co.Wicklow	58	" " "				.035	Sunderland et al (1973)
Rossmore Co.Cork	157	" " "				.019	Sunderland et al (1973)
Co.Cork	76	" " "				.034	Sunderland et al (1973)
Ireland	>2000	" " "				.019	Sunderland et al (1973)
Eire	95	2	.0211	93	.9789	.0106	Hackett & Dawson (1958)

TABLE 52. DISTRIBUTION OF KELL BLOOD GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES

Sample	Number Tested	Genotype Kk No.	Kk Freq.	kk No.	kk Freq.	Gene Frequencies K	k	Author(s)
Isle of Man	351	34	.0969	317	.9031	.0485	.9515	Present Study
England								
Cumbria	102	10	.0980	92	.9020	.0490	.9510	Present Study
England	1108	99	.0894	1009	.9106	.0457	.9543	cited by Race & Sanger (1970)
England	566	41	.0726	525	.9274	.0370	.9630	Dunsford (1949)
England	475	33	.0695	442	.9305	.0354	.9646	Bertinshaw et al (1950)
England	1166	90	.0772	1076	.9228	.0394	.9606	Ikin et al (1952)
England	8767	792	.0903	7985	.9097	.0462	.9538	Cleghorn (1961)in R & S (1970)
Scotland								
S.West Scotland	72	6	.0833	66	.9167	.0417	.9583	Present Study
Scotland	527	47	.0892	480	.9108	.0456	.9544	Ikin et al (1952)
Wales								
Wales	116	10	.0862	106	.9138	.0441	.9559	Ikin et al (1952)
United Kingdom	935	64	.0684	871	.9316	.0348	.9652	Parkin (1952)
Ireland								
Ulster	106	8	.0755	98	.9245	.0385	.9615	Ikin et al (1952)
Carnew,Co.Wicklow	175		figures unavailable			.051	.949	Sunderland et al (1973)
Co.Wicklow	58		"	"		.017	.983	Sunderland et al (1973)
Rossmore Co.Cork	157		"	"		.038	.962	Sunderland et al (1973)
Co.Cork	76		"	"		.067	.933	Sunderland et al (1973)
Ireland	>2000		"	"		.044	.956	Sunderland et al (1973)
Eire	94	16	.1702	78	.8298	.0891	.9109	Hackett and Dawson (1958)
Eire	295	17	.0576	278	.9424	.0292	.9708	Palsson et al (1970)

TABLE 53. DISTRIBUTION OF PENNEY (Kp^a) BLOOD GROUPS IN SELECTED POPULATIONS

Sample	Number tested	Phenotypes				Gene Frequency	Author(s)
		Kp(a+)		Kp(a-)		Kp^a	
		No.	Freq.	No.	Freq.		
Isle of Man	331	13	.0393	318	.9607	.0198	Present Study
London, England	1021	25	.0216	996	.9784	.0109	Cleghorn(1961) cited in Race & Sanger(1970)
South West Scotland	72	1	.0139	71	.9861	.0070	Present Study
Paris, France	3034	49	.0162	2985	.9838	.0081	Salmon(1961) cited in Race & Sanger (1970)
Boston, U.S.A.	2363	51	.0216	2312	.9784	.0109	Allen and Lewis (1957)cited in Race & Sanger (1970)
Winnipeg, Canada	1277	32	.0251	1245	.9749	.0126	Chown et al (1963)cited in Race & Sanger (1970)
Winnipeg, Canada	11239	274	.0244	10965	.9756	.0123	Dichupa et al (1969) cited in Race & Sanger (1970)

TABLE 54 DISTRIBUTION OF DUFFY BLOOD GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES

a) Tested with anti-Fy^a and Fy^b sera

Sample	Number Tested	Genotypes Fy^aFy^a No.	Fy^aFy^a Freq.	Fy^aFy^b No.	Fy^aFy^b Freq.	Fy^bFy^b No.	Fy^bFy^b Freq.	Gene Frequencies Fy^a	Fy^b	Author(s)
Isle of Man	293	63	.2150	125	.4266	105	.3584	.4283	.5717	Present Study
England	909	178	.1958	435	.4786	296	.3256	.4356	.5644	cited by Race & Sanger (1970)
Carnew,Co.Wicklow	175	figures unavailable						.363	.637	Sunderland et al(1973)
Co.Wicklow	58	"						.397	.603	Sunderland et al(1973)
Rossmore,Co.Cork	157	"						.376	.624	Sunderland et al(1973)
Co.Cork	76	"						.400	.600	Sunderland et al(1973)
Ireland	>2000	"						.416	.584	Sunderland et al(1973)

b) Tested with anti-Fy^a serum only

Sample	Number Tested	Phenotypes Fy (a+) No.	Fy (a+) Freq.	Fy (a-) No.	Fy (a-) Freq.	Gene Frequencies Fy^a	Fy^b	Author(s)
Isle of Man	353	226	.6402	127	.3598	.4002	.5998	Present Study
Cumbria	158	98	.6203	60	.3797	.3838	.6162	Present Study
England	1166	764	.6552	402	.3448	.4128	.5872	Ikin et al (1952)
England	1944	1293	.6651	651	.3349	.4213	.5787	cited by Race & Sanger (1970)
South West Scotland	58	35	.6034	23	.3966	.3702	.6298	Present Study
Scotland	527	352	.6679	175	.3321	.4237	.5763	Ikin et al (1952)
Wales	116	81	.6983	35	.3017	.4507	.5493	Ikin et al (1952)
Ulster	106	69	.6509	37	.3491	.4092	.5908	Ikin et al (1952)
Eire	95	59	.6211	36	.3789	.3845	.6155	Hackett & Dawson(1958)
Eire	295	152	.5153	143	.4847	.3038	.6962	Palsson et al (1970)

TABLE 55. DISTRIBUTION OF SERUM HAPTOGLOBIN GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES AND NORTHERN EUROPE

Sample	Number Tested	Phenotypes 1-1		2-1		2-2		0-0		Gene Frequencies Hp^1	Hp^2	Author(s)
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.			
Isle of Man	356	46	.1292	156	.4382	152	.4270	2	.0056	.3503	.6497	Present Study
England												
Cumbria	198	28	.1414	82	.4141	88	.4444	-	-	.3485	.6515	Present Study
N.E. England	762	112	.1470	374	.4908	275	.3609	1	.0013	.3929	.6071	Papiha (in press)
Northern England	206	33	.1602	104	.5049	69	.3350	-	-	.4126	.5874	Cartwright (1973b)
England	218	22	.1009	121	.5550	69	.3165	6	.0275	.3892	.6108	Allison et al (1958)
Scotland												
S.W. Scotland	370	64	.1730	157	.4243	148	.4000	1	.0027	.3862	.6138	Present Study
C.&S.W.Scotland	100	10	.1000	49	.4900	38	.3800	3	.0300	.3557	.6443	Kamel et al (1963)
Ireland												
Carnew,Co.Wicklow	175			figures unavailable						.451	.549	Sunderland et al (1973)
Co. Wicklow	58			"	"					.424	.576	Sunderland et al (1973)
Rossmore,Co. Cork	157			"	"					.422	.578	Sunderland et al (1973)
Co. Cork	76			"	"					.335	.665	Sunderland et al (1973)
Ireland	2000			"	"					.380	.620	Sunderland et al (1973)
Eire	295	44	.1492	135	.4576	116	.3932	-	-	.3780	.6220	Palsson et al (1970)
Europe												
Iceland	402	73	.1816	187	.4652	140	.3483	2	.0050	.4163	.5837	Beckman & Johannsson (1967)
Norway	1000	132	.1320	462	.4620	406	.4060	-	-	.3630	.6370	Fleischer & Lundevall (1957)
Sweden	15601	2260	.1449	7367	.4722	5929	.3800	34	.0022	.3820	.6180	Höglund et al (1970)
Denmark	2046	328	.1603	751	.3671	967	.4726	-	-	.3438	.6562	Galatius-Jensen (1958)

TABLE 56 DISTRIBUTION OF SERUM TRANSFERRIN GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES AND NORTHERN EUROPE

Sample	Number Tested	Phenotype						Gene Frequencies			Author(s)
		BC		C		CD		Tf^B	Tf^C	Tf^D	
		No.	Freq.	No.	Freq.	No.	Freq.				
Isle of Man	356	6	.0169	349	.9803	1	.0028	.0084	.9902	.0014	Present Study
England											
Cumbria	198	2	.0101	196	.9899	–	–	.0050	.9950	–	Present Study
England	139	2	.0144	137	.9856	–	–	.0072	.9928	–	Harris (1959)
Scotland											
S.W. Scotland	370	1	.0027	369	.9973	–	–	.0014	.9986	–	Present Study
Ireland											
Carnew, Co. Wicklow	175	figures unavailable						.003	.997	–	Sunderland et al (1973)
Co. Wicklow	58	"		"				–	1.000	–	Sunderland et al (1973)
Rossmore, Co. Cork	157	"		"				.004	.996	–	Sunderland et al (1973)
Co. Cork	76	"		"				.007	.993	–	Sunderland et al (1973)
Ireland	2000	"		"				.010	.990	–	Sunderland et al (1973)
Europe											
Iceland	402	–	–	402	1.0000	–	–	–	1.0000	–	Beckman & Johannsson (1967)
Iceland	2071	2	.0009	2067	.9981	2	.0009	.0005	.9990	.0005	Bjarnason et al (1973)
Norway	950	9	.0095	941	.9905	–	–	.0047	.9953	–	Braend et al (1965)
Norway	2699	31	.0115	2658	.9848	10	.0037	.0058	.9924	.0018	Teisberg (1972)
Sweden	2395	22	.0092	2370	.9896	3	.0013	.0046	.9948	.0006	Beckman et al (1962)

TABLE 57 DISTRIBUTION OF β - LIPOPROTEIN ALLOTYPE -Ag- IN SELECTED POPULATIONS OF THE BRITISH ISLES AND EUROPE

Sample	Number Tested	Phenotypes Ag(x+)		Ag(x-)		Gene Frequency Ag^x	Author(s)
		No.	Freq.	No.	Freq.		
British Isles							
Isle of Man	111	35	.3153	76	.6847	.1725	Present Study
United Kingdom	1222	447	.3674	773	.6326	.2046	Bradbrook et al (1971)
Europe							
Iceland	96	42	.4375	54	.5625	.2500	Persson & Swan (1971)
Norway	3162	1147	.3627	2015	.6373	.2017	Solaas (1970)
Sweden	245	98	.4000	147	.6000	.2327	Hirschfeld & Okochi (1967)
Sweden	216	84	.3889	132	.6111	.2183	Solaas (1970)
Finland	24	11	.4583	13	.5417	.3125	Hirschfeld & Okochi (1967)
North Italy	334	140	.4192	194	.5808	.2379	Morganti et al (1967)
Berne, Switzerland	249	94	.3775	155	.6225	.2110	Morganti et al (1970)

TABLE 58. DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES AND EUROPE

Sample	Number Tested	Phenotypes					
		A		BA		B	
		No.	Freq.	No.	Freq.	No.	Freq.
Isle of Man	325	40	.1231	123	.3785	120	.3692
England							
Cumbria	112	13	.1161	45	.4018	48	.4286
Northumberland	549	64	.1166	221	.4026	208	.3789
England	369	41	.1117	175	.4768	123	.3351
England							
Scotland							
South West Scotland	335	22	.0657	143	.4269	137	.4090
Scotland		figures unavailable					
Ireland							
Carnew, Co.Wicklow	175	figures unavailable					
Co.Wicklow	58	"		"			
Rossmore,Co.Cork	157	"		"			
Co.Cork	76	"		"			
Ireland	> 2000	"		"			
Eire	295	35	.1186	95	.3220	144	.4881
Europe							
Iceland	199	25	.1256	82	.4120	59	.2965
Norway		figures unavailable					
Sweden	517	"		"			
Denmark	679	91	.1340	270	.3976	242	.3564
France	487	figures unavailable					

TABLE 58 (contd.) DISTRIBUTION OF RED CELL ACID PHOSPHATASE GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES AND EUROPE

Sample	Phenotypes						Gene Frequencies			Author(s)
	CA		CB		C					
	No.	Freq.	No.	Freq.	No.	Freq.	p^a	p^b	p^c	
Isle of Man	17	.0523	25	.0769	–	–	.3385	.5969	.0646	Present Study
England										
Cumbria	2	.0179	4	.0357	–	–	.3393	.6339	.0268	" "
Northumberland	16	.0291	40	.0729	–	–	.332	.617	.051	Papiha (1973)
England	9	.0245	19	.0518	–	–	.3624	.5994	.0382	Hopkinson et al (1964)
England							.3595	.6023	.0383	Hopkinson (1966) in Bjarnason et al (1973)
Scotland										
South-West Scotland	11	.0328	22	.0657	–	–	.2866	.6642	.0493	Present Study
Scotland							.3380	.6080	.0540	Renwick (1972) in Bjarnason et al (1973)
Ireland										
Carnew, Co. Wicklow	figures unavailable						.345	.615	.040	Sunderland et al (1973)
Co. Wicklow	"		"				.319	.612	.069	" " "
Rossmore, Co. Cork	"		"				.308	.618	.074	" " "
Co. Cork	"		"				.305	.649	.046	" " "
Ireland	"		"				.337	.618	.045	" " "
Eire	5	.0170	16	.0542	–	–	.2881	.6762	.0356	Palsson et al (1970)
Europe										
Iceland	14	.0704	19	.0955	–	–	.3668	.5503	.0829	Bjarnason et al (1973)
Norway	figures unavailable						.3786	.5548	.0667	Berg in Bjarnason et al (1973)
Sweden	"		"				.372	.558	.070	Broman et al (1971)
Denmark	27	.0398	49	.0707	–	–	.3527	.5914	.0552	Lamm (1970b)
France	figures unavailable						.321	.639	.040	Van Cong & Mollec (1967)

TABLE 59 DISTRIBUTION OF RED CELL PHOSPHOGLUCOMUTASE LOCUS 1 GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES AND NORTH EUROPE

Sample	Number Tested	Phenotypes								Gene Frequencies			Author(s)
		1-1		2-1		2-2		7-1		PGM_1^1	PGM_1^2	PGM_1^7	
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Isle of Man	311	170	.5466	117	.3762	20	.0643	4	.0129	.7412	.2524	.0064	Present Study
England													
Cumbria	140	78	.5571	46	.3286	14	.1000	2	.0143	.7286	.2643	.0071	" "
Northumberland	549	327	.5956	186	.3388	36	.0656	-	-	.7650	.2350	-	Papiha (1973)
England	338	186	.5503	127	.3757	25	.0740	-	-	.7382	.2618	-	Spencer et al(1964)
England	2109	1237	.5865	754	.3575	118	.0560	-	-	.7653	.2347	-	Hopkinson & Harris (1966)
Scotland													
South West Scotland	30	16	.5333	9	.3000	4	.1333	1	.0333	.7000	.2833	.0167	Present Study
Scotland				figures unavailable						.7650	.2350	-	Renwick (1972)in Bjarnason et al(1973
Ireland													
Carnew,Co.Wicklow	175			figures unavailable						.764	.233		Sunderland et al(1973
Co.Wicklow	58			"	"					.673	.327		" " "
Rossmore,Co.Cork	157			"	"					.749	.244		" " "
Co.Cork	76			"	"					.722	.278		" " "
Ireland	>2000			"	"					.750	.250		" " "
Eire	106	82	.7736	19	.1792	5	.0472	-	-	.8632	.1368	-	Palsson et al (1970)
Europe													
Iceland	129	88	.6822	35	.2713	6	.0465	-	-	.8178	.1822	-	Mourant & Tills(1967)
Iceland	199	140	.7035	53	.2663	6	.0302	-	-	.8367	.1633	-	Bjarnason et al(1973
Sweden (South)	180									.781	.219	-	Hansson (1971)
Denmark	1666	1082	.6495	519	.3115	64	.0384	-	-	.8055	.1942	-	Lamm (1970 a)
Norway	2674	1608	.6014	928	.3470	134	.0501	4	.0015	.7757	.2236	.0007	Monn (1969)

TABLE 60. DISTRIBUTION OF RED CELL ADENYLATE KINASE GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES AND NORTHERN EUROPE

Sample	Number Tested	Phenotypes 1-1		2-1		2-2		Gene Frequencies AK^1	AK^2	Author(s)
		No.	Freq.	No.	Freq.	No.	Freq.			
Isle of Man	326	305	.9356	20	.0613	1	.0031	.9662	.0338	Present Study
England.										
Cumbria	57	51	.8947	6	.1053	-	-	.9474	.0526	Present Study
Northumberland	549	524	.9545	25	.0455	-	-	.977	.023	Papiha (1973)
England	1887	1720	.9115	165	.0874	2	.0011	.9552	.0448	Rapley et al (1967)
Scotland										
S.W.Scotland	30	28	.9333	2	.0667	-	-	.9667	.0333	Present Study
Ireland										
Carnew,Co.Wicklow	175	figures unavailable						.974	.026	Sunderland et al (1973)
Co.Wicklow	58	"		"				.950	.050	Sunderland et al (1973)
Rossmore,Co.Cork	157	"		"				.984	.016	Sunderland et al (1973)
Co.Cork	76	"		"				.954	.046	Sunderland et al (1973)
Ireland	> 2000	"		"				.965	.035	Sunderland et al (1973)
Eire	114	92	.8070	15	.1316	7	.0614	.8728	.1272	Palsson et al (1970)
Eire	789	739	.9366	50	.0634	-	-	.9683	.0317	Tills et al (1970)
Europe										
Iceland	1139	1013	.8894	123	.1080	3	.0026	.9434	.0566	Tills (1970)
Norway	377	344	.9125	33	.0875	-	-	.9562	.0438	Berg(1969)
Denmark	239	222	.9289	17	.0711	-	-	.9644	.0356	Lamm (1971b)

TABLE 61 DISTRIBUTION OF RED CELL ADENOSINE DEAMINASE GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES AND NORTHERN EUROPE

Sample	Number Tested	Phenotypes 1-1 No.	1-1 Freq.	2-1 No.	2-1 Freq.	2-2 No.	2-2 Freq.	Gene Frequencies ADA^1	ADA^2	Author(s)
Isle of Man	298	254	.8523	43	.1443	1	.0034	.9245	.0755	Present Study
England										
Cumbria	44	40	.9091	4	.0909	-		.9545	.0455	" "
Northumberland	469	407	.8678	61	.1301	1	.0021	.9328	.0672	Papiha (1973)
England	1353	1223	.9039	127	.0939	3	.0022	.9509	.0491	Hopkinson et al (1969)
Scotland										
South-West Scotland	30	23	.7667	7	.2333	-	-	.8833	.1167	Present Study
Ireland										
Carnew, Co. Wicklow	175			figures not available				.934	.066	Sunderland et al (1973)
Co. Wicklow	58			" " "				.930	.070	" " "
Rossmore, Co. Cork	157			" " "				.942	.058	" " "
Co. Cork	76			" " "				.890	.110	" " "
Ireland	>2000			" " "				.944	.056	" " "
Eire	1215	1084	.8922	122	.1004	9	.0074	.9424	.0576	Van den Branden et al (1971)
Europe										
Denmark	1321	1164	.8812	153	.1158	4	.0030	.9391	.0609	Dissing & Knudsen (1970)
Denmark	247	214	.8664	33	.1336	-	-	.9332	.0668	Lamm (1971a)
Hamburg, W. Germany	861	753	.8746	105	.1220	3	.0035	.9355	.0645	Goedde et al (1970)

TABLE 62 DISTRIBUTION OF RED CELL 6-PHOSPHO-GLUCONATE DEYHYDRONGENASE GROUPS IN SELECTED POPULATIONS OF THE BRITISH ISLES AND ICELAND

Sample	Number Tested	Phenotypes								Gene Frequencies		Author(s)
		A		CA		C		RA,HA,etc.				
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	PGD^A	PGD^C	
British Isles												
Isle of Man	295	280	.9492	15	.0508	–	–	–	–	.9746	.0254	Present Study
Cumbria	54	51	.9444	3	.0556	–	–	–	–	.9722	.0278	" "
Northumberland	549	526	.9581	23	.0419	–	–	–	–	.9791	.0209	Papiha (1973)
England	150	140	.9333	10	.0667	–	–	–	–	.9666	.0334	Fildes & Parr (1963)
England	4557	4362	.9572	188	.0413	3	.0007	4	.0008	.9782	.0214	Parr (1966)
South-West Scotland	30	30	1.0000	–	–	–	–	–	–	1.0000	.0000	Present Study
Carnew, Co. Wicklow	175	figures not available								.983	.017	Sunderland et al (1973)
Co. Wicklow	58	" " "								.956	.044	" " "
Rossmore, Co. Cork	157	" " "								.990	.010	" " "
Co. Cork	76	" " "								.994	.006	" " "
Ireland	2000	" " "								.985	.015	" " "
Ireland	789	767	.9721	22	.0279	–	–	–	–	.9861	.0139	Tills et al (1970)
Europe												
Iceland	832	795	.9555	37	.0455	–	–	–	–	.9772	.0228	Tills et al (1970)

TABLE 63. DISTRIBUTION OF TONGUE-CURLERS IN SELECTED POPULATIONS

Sample	Number Tested	Phenotypes				Author(s)
		Curler		Non-Curler		
		No.	Freq.	No.	Freq.	
Isle of Man - Adults	270	171	.6333	99	.3667	Present Study
Isle of Man - Juveniles	388	279	.7191	109	.2809	Present Study
Isle of Man - Total	658	450	.6839	208	.3161	Present Study
Cumbria - Juveniles	247	169	.6842	78	.3158	Present Study
U.S.A.(mixed European ancestry)	282	183	.6489	99	.3511	Sturtevant (1940)
U.S.A. Whites	1009	694	.6878	315	.3122	Urbanowski and Wilson (1947)
Eastern U.S.A.(mixed European ancestry)	865	637	.7364	228	.2636	Gahres (1952)
U.S.A. Negroes	1890	1549	.8196	349	.1804	Lee (1955)
China	1043	649	.6222	394	.3778	Liu & Hsu (1949)

TABLE 64. DISTRIBUTION OF COLOUR VISION DEFECTIVES IN SELECTED MALE POPULATIONS OF THE BRITISH ISLES

Sample	Number Tested	% Colour Blind	Author(s)
A) Males with at least 2 parents born in area specified			
Isle of Man	303	5.6	Present Study
Cumbria	137	2.9	Present Study
Cumbria	155	7.7	Sunderland (1970)
Northumberland	162	6.8	Sunderland (1970)
Durham	431	8.8	Sunderland (1970)
Yorks. Derbys. and Notts.	72	5.6	Sunderland (1970)
Scotland	132	2.3	Sunderland (1970)
North Wales	458	9.6	Fraser-Smith and Sunderland (1969)
Pembroke, Wales	530	6.98	Pullin and Sunderland(1963)
B) Males resident in area specified			
Carlisle	990	6.9	Post (1962)
Barrow	150	8.7	Post (1962)
N.W.England and Scotland	52797	7.4	Post (1962)
Glasgow	989	7.8	Pickford (1951)
Orkney Isles	404	5.2	Boyce et al (1973)
Scotland	360	7.5	Collins(1937)
Scotland	138	7.2	Gray (1944)
W.Scotland and N.W.England	>6000	7.7	Vernon and Straker (1943)
Industrial N.W.England	>6000	7.7	Vernon and Straker (1943)

TABLE 65 DISTRIBUTION OF PTC TASTING PHENOTYPES IN SELECTED POPULATIONS OF THE BRITISH ISLES

Sample	Number Tested	Phenotype				Gene Frequency t	Author(s)
		Non-Taster		Taster			
		No.	%	No.	%		
a) Individuals with 3 or 4 grandparents born in the area specified							
Isle of Man	675	188.0	27.85	487.0	72.15	.5277	Present Study
Cumbria	330	80.0	24.24	250.0	75.76	.4923	" "
Barrow-in-Furness	64	9.0	14.06	55.0	85.94	.3750	Mitchell and Swarbrick (1972)
North Lancashire	86	25.5	29.65	60.5	70.35	.5445	Cartwright and Sunderland (1967)
Lancaster City	85	20.5	24.12	64.5	75.88	.4911	" " "
Derbyshire 'B'	78	17.5	22.44	60.5	77.56	.4737	" " "
Derbyshire 'C'	72	27.5	38.19	44.5	61.81	.6180	" " "
North Wales	621	127.5	20.53	493.5	79.47	.4531	Fraser-Smith and Sunderland (1969)
Ulster	43	6.0	13.95	37.0	86.05	.3735	Maybin (1972)
Ballinlough, Co. Roscommon	237	73.5	31.01	163.5	68.99	.5569	Sunderland et al (1973)
Rossmore, Co. Cork	193	51.5	26.68	141.5	73.32	.5165	" " "
Carnew, Co. Wicklow	266	80.0	30.08	186.0	69.92	.5485	" " "
Total Eire	694	205.0	29.54	489.0	70.46	.5435	" " "

TABLE 65. (cont.) DISTRIBUTION OF PTC TASTING PHENOTYPES IN SELECTED POPULATIONS OF THE BRITISH ISLES

Sample	Number Tested	Phenotype				Gene Frequency t	Author(s)
		Non-Taster		Taster			
		No.	%	No.	%		
b) Individuals with 2 parents born in the area specified							
Northumberland	383	92.0	24.02	291.0	75.98	.4901	Sunderland (1970)
Co. Durham	762	238.5	31.30	523.5	68.70	.5595	"
North Lancashire	100	32.0	32.00	68.0	68.00	.5657	Cartwright & Sunderland (1967)
Lancaster City	107	25.0	23.36	82.0	76.64	.4833	" "
South Lancashire, Cheshire & Staffordshire.	96	30.0	31.25	66.0	68.75	.5590	" "
Yorks., Notts. and Derbyshire	106	30.5	28.77	75.5	71.23	.5364	" "
Lancashire	818	225.5	27.57	592.5	72.43	.5251	Sunderland & Cartwright (1968)
Derbyshire	800	228.5	28.56	571.5	71.44	.5344	" "
Orkney Isles	420	158.5	37.70	261.5	62.30	.6140	Sunderland (1966)
Scotland	210	66.0	31.43	144.0	68.57	.5606	Sunderland (1970)
Pembroke	1005	277.0	27.56	728.0	72.44	.5250	Pullin and Sunderland (1963)
Carmarthenshire	229	74.5	32.53	154.5	67.47	.5704	Partridge et al (1962)
c) Individuals resident in the area specified							
Liverpool	265	78.0	29.43	187.0	70.57	.5425	Kitchin et al (1959)
Southern England	441	139.0	31.52	302.0	68.48	.5614	Harris and Kalmus (1949)
England	541	169.0	31.24	372.0	68.76	.5589	Harris et al (1949)
Orkney Isles	567	185.0	32.60	382.0	67.40	.5710	Boyce et al (1973)

TABLE 66 MEAN REFLECTANCE VALUES AT 425 mμ, 545 mμ AND 685 mμ FOR SELECTED BRITISH ISLES AND EUROPEAN POPULATIONS

a) Medial Aspect of Upper Arm

MALES

Locality	Sample	Latitude	Number Tested	Wavelength 425 mμ Filter 601		545 mμ Filter 605		685 mμ Filter 609		Author(s)
				mean	SD	mean	SD	mean	SD	
Isle of Man	Native Children	54°N	90	36.62	4.13	41.94	3.76	65.91	2.90	Smith and Mitchell (1973)
Cumberland	" "	54°N	99	35.78	4.03	41.80	3.62	66.46	3.16	" " "
Northumberland	" "	55°N	166	33.55	5.12	42.98	4.12	67.84	4.03	Hulse (1973)
Merthyr Tydfil, S. Wales	" "	52°N	84	32.77	4.91	38.71	5.14	62.80	5.74	Smith and Mitchell (1973)
Carnew, Co. Wicklow	Native - mainly Adults	53°N	105	34.86	4.36	39.37	4.36	64.40	3.52	Sunderland et al(1973)
Rossmore, Co. Cork	" " "	52°N	111	34.63	4.13	40.72	4.26	64.69	3.13	" "
Ballinlough, Co. Roscommon	" " "	54°N	105	35.40	4.30	40.94	4.17	65.31	3.30	" "
Brussels, Belgium	Adults	51°N	143	37.71	4.83	44.77	3.96	67.27	2.90	Leguebe (1961)
Belgium	"	51°N	69	39.12	5.39	43.80	3.95	64.51	3.37	Rijn-Tournel (1965)
Mixed Europeans	"	50°N	74	40.30	1.20	45.20	3.46	66.90	3.02	Ojikutu (1965)

FEMALES

Locality	Sample	Latitude	Number Tested	425 mμ Filter 601		545 mμ Filter 605		685 mμ Filter 609		Author(s)
				mean	SD	mean	SD	mean	SD	
Isle of Man	Native Children	54°N	73	36.75	4.40	41.75	3.58	67.01	2.69	Smith and Mitchell (1973)
Cumberland	" "	54°N	153	36.94	4.79	42.35	4.11	66.96	2.87	" " "
Northumberland	" "	55°N	171	36.10	4.70	44.44	4.33	68.67	3.55	Hulse (1973)
England	Adults	51°50'N	23	34.93		38.58		62.97		Tiwari (1963)
Merthyr Tydfil, S. Wales	Native Children	52°N	98	33.23	3.51	38.66	3.50	63.46	3.97	Smith and Mitchell (1973)
Carnew, Co. Wicklow	Native - mainly Adults	53°N	162	37.23	4.17	42.12	4.01	64.64	2.85	Sunderland etal (1973)
Rossmore, Co. Cork	" " "	52°N	90	35.66	4.05	41.81	4.15	64.75	3.29	" "
Ballinlough, Co. Roscommon	" " "	54°N	127	36.19	3.78	41.90	3.60	65.13	2.58	" "
Brussels, Belgium	Adults	51°N	177	36.50	3.98	44.57	3.62	65.88	2.47	Leguebe (1961)
Belgium	Adults	51°N	46	38.18	5.52	43.15	4.54	63.65	4.16	Rijn-Tournel (1965)

TABLE 66 (cont.) MEAN REFLECTANCE VALUES AT 425 mμ, 545 mμ AND 685 mμ FOR SELECTED BRITISH ISLES AND EUROPEAN POPULATIONS

a) Medial Aspect of Upper Arm

MALES AND FEMALES

Locality	Sample	Latitude	Number Tested	425 mμ Filter 601		545 mμ Filter 605		685 mμ Filter 609		Author(s)
				mean	SD	mean	SD	mean	SD	
Europeans (including mainly Irish Liverpool)	Adults	53°50'N	46-105	36.10	4.61	41.00	4.59	62.30	3.50	Harrison and Owen (1964)

b) Flexor Surface of Right Forearm

MALES

Europeans, London (Mainly English)	Adults	51°50'N		32.80		37.90		61.50		Barnicot (1958)

FEMALES

Europeans, London (Mainly English)	Adults	51°50'N		34.30		40.50		63.10		Barnicot (1958)

Appendix 2.

Isle of Man

Quinary Ages of Population 1971

Age last birthday	Persons		Males		Females	
	No	%	No	%	No	%
0 - 4	3,755	6.7	1,958	7.4	1,797	6.0
5 - 9	3,912	6.9	2,018	7.6	1,894	6.3
10 - 14	3,520	6.2	1,781	6.7	1,739	5.8
15 - 19	3,484	6.2	1,759	6.6	1,725	5.8
20 - 24	3,837	6.8	1,955	7.4	1,882	6.3
25 - 29	3,008	5.3	1,529	5.8	1,479	5.0
30 - 34	2,734	4.9	1,419	5.4	1,315	4.4
35 - 39	2,685	4.8	1,349	5.1	1,336	4.5
40 - 44	2,948	5.2	1,378	5.2	1,570	5.3
45 - 49	3,372	6.0	1,535	5.8	1,837	6.2
50 - 54	3,258	5.8	1,491	5.6	1,767	5.9
55 - 59	4,067	7.2	1,850	7.0	2,217	7.4
60 - 64	4,412	7.8	2,005	7.6	2,407	8.1
65 - 69	4,108	7.3	1,799	6.8	2,309	7.7
70 - 74	3,234	5.7	1,294	4.9	1,940	6.5
75 - 79	2,061	3.7	756	2.9	1,305	4.4
80 - 84	1,198	2.1	380	1.4	818	2.7
85 - 89	499	.9	145	.6	354	1.2
90 - 94	170	.3	57	.2	113	.5
95 - 99	22	.1	3		19	
100 and over	5		-		5	
TOTAL	56,289	99.9	26,461	100.0	29,828	100.0

APPENDIX 3

Isle of Man Project,
Anthropology Department,
Durham University,
DURHAM CITY.

Dear

As you will know on the last occasion you donated blood, Dr. Pantin sent a sample to me for further analysis. This is the first part of the project 'Genetical Variation in the Isle of Man'. I would now like to visit you to carry out the remaining tests at your home, as outlined in my Christmas letter to you.

The tests involve tabulating a selection of population variables such as tasting ability, colour blindness and taking impressions of the palms of the hands. Also I would like to take away with me specimens of saliva and urine. To save time, if it is possible could you have a fresh urine sample ready for me when I arrive.

I hope to visit you at a.m./p.m. on the of . I trust this will be convenient; if so could you acknowledge this on the enclosed card and return it to me. If inconvenient please suggest a time and date between the inclusive; any evening is convenient.

This survey is unique in many respects and for its success requires as many Manx people to help us as possible. Therefore if any of your friends, neighbours or relatives of Manx ancestry would like to take part I shall be very pleased to see them at your house or arrange to see them when mutually convenient.

I hope you will excuse this duplicated sheet - I would like to write to each person individually. Unfortunately owing to the shortage of time and staff this method is most appropriate.

Yours sincerely,

R. J. Mitchell

Would you please fill in the attached form.

APPENDIX 3 cont'd

ISLE OF MAN GENETICS SURVEY NO. _____

Name _____ Sex _____ Date of Birth _____

Maiden Name _____

Place of Birth _____

Place of Birth of both parents

Mother _____

Father _____

Place of Birth of all 4 Grandparents

Mother's Mother _____

Mother's Father _____

Father's Mother _____

Father's Father _____

(Parish if possible)

Mother's Maiden Name _____

Father's Mother's Maiden Name _____

Mother's Mother's Maiden Name _____

APPENDIX 4

Genetical Survey - Isle of Man

As a research worker from the University of Durham I am currently investigating certain inherited features in human beings in various parts of the country. One inherited character is the ability to taste a substance, P.T.C. (phenylthiocarbamide), and the people of the Island may well prove interesting in this respect. The testing procedure in order to find out whether or not someone can taste P.T.C. is quick, harmless and simple.

Are you prepared to allow us to test your child for P.T.C. tasting, colour vision, dermatoglyphics and pigmentation at school? If so, please sign in the space below and return the form to school as soon as possible. This work is done with the co-operation and permission of your child's head-teacher and Director of Education.

It is interesting for us to know the places from which the children, their parents and, if possible, their grandparents also, originate. If you agree to your child being tested would you also fill in the details regarding birth places below.

Also I would like to analyse your child's blood for the various blood group antigens. Allchildren who agree to give a fingerprick sample will receive notification of their ABO and Rhesus blood groups. If you agree to your child giving blood for analysis please sign in the space below.

Thank you very much.

R. J. Mitchell

Full name of child ..... Age .....

I agree to have my child tested .....
(Parent or Guardian)

Place of birth of child .....
Place of birth of both parents

Mother .....

Father .....

Place of Birth of all 4 Grandparents

Mother's Mother .....

Mother's Father .....

Father's Mother .....

Father's Father .....
(Parish if possible)

Mother's maiden name (if possible) .....

Father's mother's maiden name (if possible) .....

Mother's mother's maiden name (if possible) .....

ABO Blood group of child if known .....

I agree to my child giving a small sample of blood for analysis

.....
(Parent or Guardian)

If any answers not known - please state NOT KNOWN

APPENDIX 5

For use in the Jane Crookall Maternity Wing

To all Ladies born on the Isle of Man

Mr.John Mitchell of Durham University is carrying out research to find out whether any of the four blood groups occur particularly often among people of Manx stock.

Would you therefore, be so good as to help him by filling in the form below, so that he may decide how Manx you are. Your blood will be grouped as part of your antenatal examination and if at least three of your grandparents were Manx, your blood group will be included among the other blood groups Mr.Mitchell has found in people of Manx stock.

Manx Genetic Survey

Name.....D.o.B.....Maiden Name.....

Place of birth .....

Place of birth of both parents: Father.....

Mother.....

Place of birth of all 4 grandparents

Father's Father.....

Father's Mother.....

Mother's Mother.....

Mother's Father.....

Mother's Maiden Name.....

Mother's Mother's Maiden Name.....

Father's Mother's Maiden Name.....

FOR LABORATORY USE ONLY

ABO GROUP

Rh.Gp

APPENDIX 6

DURHAM UNIVERSITY - ANTHROPOLOGY DEPARTMENT

Dear Donor,

I am currently engaged in a study of blood group distributions in Cumbria. As a blood donor we know your blood groups, but to make the survey more valuable and accurate I would like to know the birth places of yourself, your parents and your grandparents. Would you therefore fill in the section below to help me in this work. Thank you for your assistance.

R. J. MITCHELL

Your Name ..... Date of birth .....

Your Birthplace .....

Birthplace of mother .....

" " father .....

" " mother's mother .....

" " mother's father .....

" " father's mother .....

" " father's father .....

BE AS ACCURATE AS YOU CAN: IF YOU DO NOT KNOW STATE "NOT KNOWN"

Donor Number:

Date:

APPENDIX 7

DURHAM UNIVERSITY - ANTHROPOLOGY DEPARTMENT

Dear Donor,

I am currently engaged in a study of blood group distributions in South West Scotland. As a blood donor we know your blood groups, but to make the survey more valuable and accurate I would like to know the birthplaces of yourself, your parents and your grandparents. Would you therefore fill in the section below to help me in this work. Thank you for your assistance.

R. J. MITCHELL

Your Name ..... Date of birth .....

Your birthplace .....

Birthplace of mother .....

" " father .....

" " mother's mother .....

" " mother's father .....

" " father's mother .....

" " father's father .....

BE AS ACCURATE AS YOU CAN: IF YOU DO NOT KNOW STATE "NOT KNOWN"

Donor Number:

Date:

APPENDIX 8

Genetical Survey - Cumberland

As a research worker from the University of Durham I am currently investigating certain inherited features in human beings. One inherited character is the ability to taste a substance, P.T.C. (phenylthiocarbamide) and the people of Cumberland may well prove interesting in this respect. The testing procedure is quick, harmless and simple.

Are you prepared to allow us to test yourchild for P.T.C. tasting, colour vision, dermatoglyphics and pigmentation at school? If so, please sign in the space below and return the form to school as soon as possible. This work is done with the co-operation and permission of your child's head-teacher and Director of Education.

It is interesting for us to know the places from which the children, their parents and if possible, their grandparents also originate. If you agree to your child being tested would you also fill in the details regarding birth places below?

Also I would like to analyse your child's blood for various blood group antigens. All children who agree to give a finger-prick sample will receive notification of their ABO and Rhesus blood groups. If you agree to your child giving blood for analysis please sign in the space below.

Thank you very much.

R. J. Mitchell

Full name of child ..... Age .....

I agree to have my child tested .....
(Parent)

Place of birth of child .....
Place of birth of both parents

Mother .....

Father .....

Place of birth of all 4 grandparents

Mother's Mother .....

Mother's Father .....

Father's Mother .....

Father's Father .....
(Parish if possible)

ABO blood group of child if known .....

I agree to my child giving a small sample of blood for analysis

.....
(Parent)

If any answers not known - please state NOT KNOWN