A Genetic Analysis of the Kaimanawa Horses and Comparisons with Other Equine Types.

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Abstract.

Recorded sightings of feral horses in the Kaimanawa ranges and the surrounding area date back to 1876. Since then, the Kaimanawa horse herd has received many different influxes and during the mid-1900s it's population size increased to the thousands. However, two decades ago the herd went through a substantial reduction in size, numbering only 179 individuals. Hence they were granted legal protection within a defined geographic area. This protection served its' purpose more than adequately, for by January 1994 the population size had increased 10-fold to c.1700. Concurrently, the horse population appeared to be trampling and grazing native endangered flora. Therefore, it has been proposed that some form of population management be enforced on the feral herd. This has evoked opposition in the form of emperically unsubstantiated claims that the Kaimanawa feral horses "may constitute a unique gene pool." (Wright, 1989) This research was executed so as to determine the genetic distance of the Kaimanawa horses from other, more populous equine breeds in New Zealand.

Blood samples were taken from four hundred and eight Kaimanawa horses, and their blood types for the inherited variants of sixteen polymorphic red blood cell and plasma proteins (Al, PGD, PHI, Gc, Tf, α-1 β, Pi, Es, Hb, A, C, D, K, P, Q and U) were determined. From these blood types, the frequency of each of the ninety four alleles within the aforementioned genetic systems was calculated. These frequencies within the Kaimanawa horse sample were then compared with those of Thoroughbred, Arabian, Standardbred, Station Hack and Shire horses. This comparison took the form of genetic distances, calculated via both Roger's and Nei's genetic distance measures.

Both models demonstrated the same trends; the genetic distance is smallest between Kaimanawa horses and Station Hacks (D_R=0.211, D_N=0.070), and greatest between Kaimanawa horses and Shires (D_R=0.337, D_N=0.413). These genetic distance values were then diagrammatically represented in phylogenetic trees.

Aside from genetic distance calculations, a qualitative comparison between the Kaimanawa horses and the other equine types was conducted, based on whether each of the ninety four previously mentioned alleles are shared amongst each of the populations. It was found that all but nine alleles were shared with Thoroughbreds and Station Hacks, with the remaining alleles being found in other equine types existing in New Zealand.

All of these analyses indicated that, a) the two equine types with the greatest amount of genetic similarity to the Kaimanawa horses are the Station Hacks and Thoroughbred horses; and b) every allele present in the Kaimanawa horses can be found in another of the equine types studied. Based on this evidence, the Kaimanawa horses should not be classified as genetically unique and their preservation can not be justified based on an argument of genetic insurance. However, it must be recognised that they have aesthetic and historical value to some New Zealanders.

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1. Introduction.

1.1 Overview.

Since 1876 there have been recorded sightings of "wild" horses in the Kaimanawa Ranges, only sixty two years after the species first arrived in New Zealand. In the years to follow, large numbers of these wild horses roamed over a huge range which encompassed much of the central North Island. However, once such land began to be developed, there was an obvious decline in both the range and size of the horse population. Concern that the herd had reached a population bottleneck prompted a recommendation for the Wildlife Act (1953) to be amended in 1979 so as to grant the Kaimanawa wild horses legal protection. Seventeen years have now passed since such security was bestowed upon the horses. Over this time their population size has increased by tenfold. Rather ironically, the trampling and grazing executed by the preserved horse population appears to have jeopardised the preservation of many already vulnerable plant species peculiar to the area. Hence, it is now apparent that some form of control is required to maintain the horse population at a size which is harmonious with it's botanical environment. Various methods of control have been suggested, some of which have already been practised. This has become somewhat of a controversial issue, with several groups claiming that the Kaimanawa horse herd is a genetically unique population which should remain free-breeding and unmanaged. The genetic analysis required to verify such claims forms the basis of this research.

1.2 History

Domestic horses (*Equus caballus*) were first introduced into New Zealand on December 23, 1814 when Reverend Samuel Marsden shipped them across from New South Wales and landed in the Bay of Islands. (Fleury, 1991). The species wasn't to be found in the central North Island until 1844 when Tamati Waka Nei Nei brought a horse from Hokianga down

to Taupo. In 1850, the first horse to inhabit the Ruapehu region was brought across from Napier. (Wright, 1989). Further horses would have come into the area with travellers, settlers, explorers, the mounted rifle cavalry and there were those which were owned and traded by the Maori people. Horses that escaped, or were liberated from such sources, especially during the land wars, would have constituted the beginnings of a feral herd in the Kaimanawa Ranges.

One of the earliest and probably most significant influxes of horses into the inaugural Kaimanawa herd appear to have been the products of a breeding programme which was initiated by Major George Gwavas Carlyon. (Fleury, 1991 & Wright, 1989) In 1858 he imported, from England, Thoroughbred Exmoor ponies into the Hawke's Bay, and crossed them with local horses to produce the "Carlyon" pony. After Major Carlyon's death, Sir Donald McLean continued the breeding programme. McLean imported two Welsh stallions in 1875 and crossed these with the Carlyon pony, resulting in the "Comet" breed. (Named after one of the stallions used.) Two years later, he released a stallion and some mares of this breed on the Kaingaroa plains. These horses undoubtedly joined and proliferated throughout the feral herd. The introduction of the Comet breed to the feral herd may well have brought in characteristics typical of the former's "semi-wild" British ancestry, namely those which facilitate the successful adaption to harsh conditions. (Batley, 1977). e.g., pony type stature. Morphological observations alone confirm the major influence which the Comet breed had on the feral herd. In 1883, Kerry Nichols, an explorer, described a horse that had been bred and captured from a band of wild horses caught on the Kaingaroa plains as exhibiting Exmoor characteristics. (Batley, 1977 & Wright, 1989). Furthermore, a large proportion of the contemporary herd still resemble their pony forebears.

In 1941 a strangles (infectious, sometimes fatal, respiratory condition) epidemic threatened the horses at the Waiouru Mounted Cavalry Stables. (Batley, 1977 & Fleury, 1991). Hence the horses were released into the Kaimanawas. Some of these horses would have died of the infection, whilst others would have joined the feral herd.

An Arab stallion was released by Nicholas Koreneff at some stage in the 1960's into the area. (Fleury, 1991).

During the filming of a Marlboro Cigarettes commercial in 1984, a Palomino stallion that had been escorted into the area for the purpose of the advertisement, broke away from the set and was unable to be recaptured. For a few years to follow, he was occasionally seen, alone, not far from the Argo Valley. This stallion must have finally formed his own harem as there have been sightings of a band of horses, all with Palomino features, roaming that same area.

In addition, throughout the years, various station hacks would have escaped or been liberated into the region from stations neighbouring the periphery of the Kaimanawa Ranges.

Figure 1.01: Kaimanawa horses being mustered in June 1994.

Such a diverse range of origins is reflected in the lack of morphological uniformity amongst the contemporary Kaimanawa Horses.

Despite all these influxes into the feral herd, since its' origin, the herd has undergone several dramatic fluctuations in both population size and range.

Since the 1930's feral horses occupying land east of the Desert Road, within the boundaries of the army training area, have been passively protected by the army as such land is prohibited to the general public. (Rogers, 1991). However, up until the 1950's such an area did not define the boundaries of the Kaimanawa horse herd range. Instead "wild horses roamed in their thousands across the North Island, from Putaruru in the north to Karioi in the south, and out towards the Kaingaroa Plains in the east." (Wright, 1989). However, such an extensive range wasn't to last, especially with the onset of schemes to develop the central region of the North Island. Increased farmland, forestry, the Tongariro Hydro Scheme, and the sole reservation of the Tongariro National Park for indigenous flora and fauna, facilitated the abatement of the horse's range. In response to this, there would have been greater competition amongst the horses for the available food resources and so their numbers would have decreased. However, this decrease in population size would have been catalysed by amateur hunters and poachers who were encouraged to eliminated these "pests." Rodeos also served to reduce the number of Kaimanawa horses. In 1976 a few remained in the National Park area, however in 1977 the last horse was removed from Karioi. A census conducted in 1979 concluded that there were only one hundred and seventy four Kaimanawa horses remaining. (Aitken *et al*, 1979). They were roaming approximately twenty four thousand hectares localised around the Moawhango River headwaters, close to Waiouru, on the eastern side of the Desert Road. Such a dramatic reduction in population size brought about concern that the herd faced extinction and so a recommendation for their protection was lodged.

Within such a small breeding population, many genetic problems often arise, some of which could be compounded by the social structure of feral horse herds. Feral horses of the Kaimanawa's have a social organisation typical of their international counterparts.

(Kirkpatrick & Turner, 1986). They organise themselves into one of two group types:- harems, consisting of a dominant, polygamous stallion, mares and their offspring; or, bachelor groups of mature males. Within the former, family type group, it is very rare for any male apart from the dominant stallion, to mate with the mares. As a result, all of the offspring within such a group, will be "half-sibs", with a greater likelihood of being genetically similar to each other than to those from other groups. (Berg, 1986). Hence, if the number of individuals within a population decreases, and the choice of mate becomes increasingly limited, the incidence of consanguineous matings will rise. Such inbreeding decreases the amount of genetic variation, or heterozygosity within a genepool. Concurrent to this, is an increased frequency of homozygous genotypes, some of which are often undesirable. (Berg, 1986).

The decrease in reproductive efficiency amongst Thoroughbreds and the increased incidence of ataxia in Przewalski's Horse, are both attributed to high levels of inbreeding within their populations.

In general, traits related to physical fitness and reproductive success are negatively affected by inbreeding, commonly referred to as inbreeding depression. This is best illustrated by the plight of the cheetah, *Acinonyx jubatus.* (O'Brien, 1984). Due to an extreme population bottleneck approximately ten thousand years ago, and a subsequent high level of inbreeding, the comtemporary cheetah population is basically devoid of any genetic variation. The small remaining population suffers from low breeding success (a high incidence of abnormal sperm and an infant mortality rate of up to seventy percent), and hypervulnerability to disease.

However, a high occurrence of inbreeding does not always result in inbreeding depression. The Northern Sea Elephants also experienced a population bottleneck and it has been estimated that in 1884 there were only twenty remaining worldwide. (Bonnell & Selander, 1974). "They now number in the tens of thousands." Yet despite the high levels of inbreeding required to achieve such a recovery in population size, the sea elephants are exhibiting no overt signs of inbreeding depression. As with feral horses, these sea elephants are polygamous, with few males contributing to the genepools of successive generations.

Although few studies of inbreeding within feral horse populations have been conducted, Duncan *et al* (1994) found that amongst a small feral horse herd, the "observed (levels of) inbreeding never exceeded half of the expected value." (Duncan *et al*, 1984). This was mainly due to the complete avoidance of matings between stallions and their dams. Regardless of whether this holds true for all feral horse populations, the herd must be of a size large enough to maintain genetic variability.

To date, the Kaimanawa horses haven't exhibited any overt symptoms of inbreeding depression. Although, if one is to consider the population decrease which the herd has undergone as being a population bottleneck, there is the possibility that they are inbred. However, as will be considered later in this research, it must be recognised that there have probably been c.10 generations of the Kaimanawa horses which does not allow for inbreeding to be as marked as it is in the cheetah population.

Nevertheless, with such considerations in mind, in 1981 the Kaimanawa horses were granted legal protection under the Wildlife Act (1953), as long as they remained within a particular zone. (Fleury, 1991). See figure 1.02 .Horses which roamed beyond this zone were no longer protected. Such protection resulted in a rapid increase in the herd's size. A census of the horses was lead by the Army in 1988 and it identified approximately 760 horses. (Wright, 1989). In 1990 it was estimated that there were 1100 horses in the herd, roaming approximately seventy thousand hectares.

Recent concerns for certain endangered plant species of the area prompted the need for another census. This consisted of both land and aerial counts and was conducted late in April 1994. It was estimated that the herd has grown to a size of approximately 1700.

1.3 The Issue.

Although not apparent at the time of the 1979 census, (Aitken *et al*, 1979) it seems that now the herd is trampling and grazing many threatened plant species. (Rogers, 1991). Ten

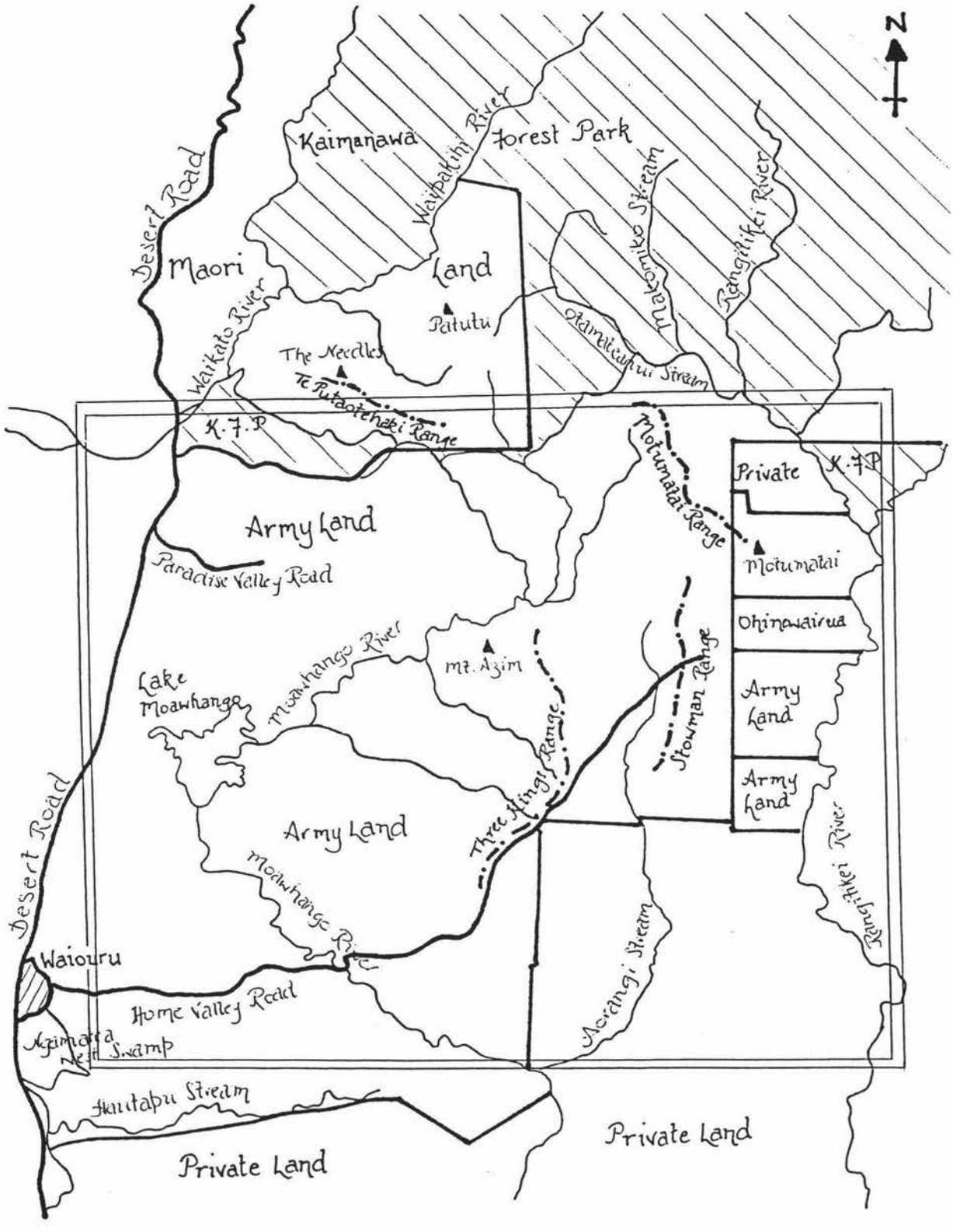


Figure 1.02: Protection zone.

0 10km

Taken from : Fleury 1951

of these are not only recognised as having a "vulnerable" status, but also, within the North Island, their only habitat is that which they share with the horses. Prior to the 1930's much of the land in the Southern Kaimanawa Ranges was covered in hard and red tussock, *Chionochloa pallens* and *C. rubra* respectively. However, with the advent of pasturalisation this land was extensively burned and grazed upon, heavily reducing the occurrence of such plants within the area. In addition, the horses appear to impose a particularly heavy selective grazing pressure upon *C. pallens* over *C. rubra*, possibly due to the higher content of soluble sugars, protein and sodium in the former. Furthermore, there is considerable evidence that many of the areas frequently roamed by the horses are internationally unique in that they are only just beginning to support forestation and vegetative growth, for the first time since the last glaciation. (Rogers, 1991). Hence such areas warrant preservation from the destructive effects of their equine inhabitants. Similar situations have been identified in the United States where it is also necessary to "obtain a balance between preserving the horse herds and maintaining the ... delicate ecosystems in which the horses live." (Cothran, 1992).

Resultantly, the Department of Conservation has suggested that some form of management strategy be imposed to control the size of the Kaimanawa horse population. (Fleury, 1991). Possible forms of control that have been proposed include culling, removal and immunocontraception, or combinations of each. (Kirkpatrick, 1994).

Recently, there has been a substantial amount of opposition to any such form of management. Several groups contend that the Kaimanawa horse herd is genetically unique and that it "may be of future value as a source of traits associated with hardiness as well as agility." (Aitken *et al*, 1979). Hence they believe the herd should remain free from human intervention. Currently, such claims of genetic uniqueness are empirically unsubstantiated.

1.4 This research.

In an attempt to rectify this situation, certain genetically controlled blood markers will be analyzed from blood samples of Kaimanawa horses mustered in 1993 and 1994, and compared with those from other known equine breeding types in New Zealand. This genetic comparson will primarily be in the form of a genetic distance study, based upon allele frequencies.

The blood markers to be analyzed are identified via blood typing which in turn refers to the determination of, a) traditional blood groups and b) the polymorphic proteins present in serum or red blood cells.

1.4.1 Traditional Blood Groups.

On the surface of red blood cell membranes lie a variety of heritable antigens or blood factors. Each of these blood factors belongs to a specific system or blood group, all of which were characterised amongst the equids by Stormont and Suzuki (1964). There are seven internationally recognized equine blood groups, namely A, C, D, K, P, Q and U. The presence of certain factors belonging to each of these blood groups is determined by either agglutination or haemolysis reactions.

1.4.2 Polymorphic Proteins.

Blood contains several polymorphic protein systems. Ashton, 1958, was the first to demonstrate, by starch gel electrophoresis, (following the technique of starch gel electrophoresis that was developed by Smithies, 1955) individual variation in serum proteins in horses. Using techniques commonly used and internationally recognised for parentage verification in the Thoroughbred industry, the variable forms of eleven protein systems can be readily distinguished as they migrate at different velocities towards the anode of an electrophoretic gel. (Anderson *et al*, 1993).. The genetic control for such protein systems was later studied and is still far from being completely understood. No physiological differences appear to result from each of the different heritable variants. (Most of the difference appears to lie solely in the inclusion of single amino acids or insignificantly

different polypeptide conformations of no consequence.) They depend on codominant, autosomal alleles. These polymorphisms appear to be the result of either multiple gene loci or, multiple allelism at a single genetic locus. (Harris & Hopkinson, 1976). In addition, these proteins have a codominant mode of inheritance. Resultantly, heterozygotes for a certain protein system will exhibit equal expression of both forms of such a trait.

The discrete phenotypic differences in the expression of these proteins are a direct reflection of the variable genotypes possible for each protein system. See table 2.01 for a description of the aforementioned eleven protein systems, all of which shall be examined in this study.

1.4.2.1 Albumin, (Al).

Albumin is the most abundant protein in mammalian blood plasma and is primarily a transport protein. (Putnam, 1984). Within this role, it reversibly binds fatty acids and a variety of poorly soluble organic materials, including bile pigments and some steroid hormones. It also binds a wide variety of anions. The circulating molecule is a single polypeptide chain containing approximately 580 amino acids. It is synthesized within and secreted from the liver.

The classically accepted role is that of maintaining colloid osmotic pressure in the vascular system. It also has a general transport function because of it's affinity for fatty acids, vitamins, hormones and metal ions. It can act as a depoisoning agent because of it's affinity for pharmaca and heavy metals. Stormont and Suzuki (1963) first demonstrated the existance of two alleles to control albumin phenotypes. This was confirmed by Braend and Efremov 1965.

1.4.2.2 6-Phosphogluco Dehydrogenase, (PGD) & Phosphohexose Isomerase, (PHI).

The PGD and PHI systems were first described by Bengtsson, and Sandberg, (1973). During the natural maturation (course of development), a red blood cell loses its nucleus and the ribosomes, mitachondria and golgi apparatus disappear from the cytoplasm. Hence, the ability to synthesize proteins or to obain energy through the Krebs cycle is lost. By utilizing some of it's remaining enzymes, the cell can make simple compounds such as glutathione by

going through the process of glycolysis. In this pathway, PGD catalyzes the conversion of 6-phosphogluconate to pentose-phosphate. (Giblett, 1969). The enzyme itself varies in both electrophoretic pattern and catalytic activity. It has been concluded (Kazazian, 1966) that the enzyme is a dimer containing two subunits each with a molecular weight of c.40, 000. Whilst the genetic locus for PGD in Drosophila is on the X chromosome, in mammals the locus is autosomal. (Davidson, 1967).

Phosphohexose isomerase catalyzes the reversible conversion of glucose-6-phosphate to fructose-6-phosphate. The triplet pattern of the variants suggests a dimer structure for this enzyme. Thus, it is assumed that with heterozygosity at the PHI locus, two subunits are produced. (Giblett, 1969).

1.4.2.3 Vitamin D binding protein, (Gc).

Variants of equine vitamin D binding protein were identified by Gahne and Juneja, in 1978. This was done by radioactively labelling horse plasma samples of known post-albumin types with ^{14}C-vitamin D3. The samples were then analyzed by polyacrylamide gel electrophoresis, followed by autoradiography. The patterns observed were identical to those of post albumin variants. Hence, that particular polymorphic post-albumin protein was identified as being the vitamin D binding protein. It is also homologous to the vitamin D binding protein found in human plasma known as the Gc-globulin. (Gc = Group-specific Complement). Hence, Gc has been adopted as the abbreviated locus symbol for the equine vitamin D binding protein. Synthesis of Vitamin D binding protein occurs in the liver. (Putnam, 1984)

1.4.2.4 Transferrin, (Tf).

This protein can have a bacteriostatic effect and may thus contribute to the individuals' resistance to particular bacteria. Braend and Stormont, 1964, demonstrated the existance of six transferrin alleles. These researchers demonstrated, autoradiographically, that the fractions controlled by those alleles could bind iron, and thus indeed belonged to the transferrins. Gahne, 1966, confirmed this variation. The major role of transferrin is to transport iron to the bone marrow. (Putnam, 1984).

The liver is also the major site of Transferrin synthesis. (Giblett,1969). Tf is a true carrier protein in that a single Tf molecule picks up and delivers Fe atoms to specific receptor sites many times before itself being catabolized. The bone marrow of humans is able to utilize c.25mg of Tf daily for haemoglobin synthesis. Since only c.1mg of iron is normally absorbed and excreted per day, iron from catabolized haemoglobin is taken up by Tf and transported to the marrow for re-incorporation into haemoglobin. When excessive Fe is presented to the bone marrow, Tf deposits a load in the liver parenchymal cells. However, under normal circumstances, the principal iron recipient is the red blood cell precursors in the marrow.

1.4.2.5 Alpha 1 Beta-Glycoprotein, (α-1 β).

The research which identified the polymorphic vitamin D binding protein lead on to also reveal polymorphism of yet another post-albumin protein, Pa. (Juneja, Gahne & Sandberg, 1978). This is sometimes referred to as Xk (Trommershausen-Smith and Suzuki, 1978) and has more recently been accurately identified as the α-1 β glycoprotein (Patterson, Bell & Shaw, 1991). It has been suggested that α-1 β glycoprotein is part of a homeostatic system, which controls remodelling and physiological cell death during development. (von Bulow et al, 1993).

1.4.2.6 Protease Inhibitors, (Pi).

Protease inhibitors are present in both plants and animals. In plants they inhibit gut proteases of insects. They are found in storage organs such as tubers and are currently being cloned in plants so as to deter insects from feeding off the plants. (Jameson, 1995).

Blood plasma contains a number of very important protease inhibitors, among which are α_1-antitrypsin, α_1-antichymotrypsin, α_2-macroglobulin, antithrombin III and antiplasmin. The α_1-antichymotrypsin and α_2-macroglobulin are the more abundant of these (3 & 2mg/ml plasma respectively). Humans with genetically defective α_1-antitrypsin are extremely prone to diseases such as emphysema. The apparent cause of the disease is proteolytic degradation of elastin fibres. (Putnam, 1984). Polymorphic forms of the equine protease inhibitor (Pi)

system were characterized by Pollitt and Bell, 1983. In this system, there are at least eighteen different, internationally recognized alleles which are thought to be controlled by at least three loci. However, these loci are believed to be extremely close and they behave as a single locus. Hence the system is regarded as being controlled by a single multiallelic locus. Protease inhibitors may in fact be the products of "multi-gene" families. There is evidence that in the genomes of plants and animals, families of structurally related genes exist, perhaps numbering up to 100 individuals.

1.4.2.7 Esterases, (Es).

Unspecific carboxylesterases which hydrolyze a large number of carboxylic esters, are widely distributed in serum, vertebrate tissues, insects, plants, citrus fruits, mycobacteria and fungi. (Kuby, 1991). Their action is generally restricted to short chain fatty acid esters, or water soluble substrates. Gahne, 1966 first demonstrated the existance of the serum esterase system.

1.4.2.8 Haemoglobin, (Hb).

Haemoglobin is the haem-containing protein, consisting of four globin subunits, two alpha-type and two beta-type, which associate noncovalently to form tetramers. It is an allosteric protein, in that it undergoes configurational alterations which are essential to it's function of cooperatively taking up and releasing oxygen. In the lungs, haemoglobin tightly binds oxygen and then transports it to areas of low oxygen concentration such as the tissues, in response to changing physiological conditions. Genetic polymorphism for haemoglobin was initially proven by Cabannes and Serain, 1955 via paper-electrophoretical analysis. This was confirmed using gel-electrophoresis by both Braend & Stormont, 1964 and Braend & Efremov, 1965.

1.4.3 Electrophoresis of these proteins.

Phosphohexose isomerase (PHI) and 6-phosophgluconate dehydrogenase (PGD) are detected on one horizontal electrophoretic starch gel which is latitudinally sliced for differential staining. Albumins are also run on a separate starch gel. Protease inhibitors and sterases can also be stained for on the one acid polyacrylamide gel, as can vitamin D binding

protein, α-1 β glycoprotein and transferrin on an alkaline polyacrylamide gel. Isoelectric focusing of an agarose gel is used to detect the haemoglobins.

Each of these procedures exploit the fact that there is a one to one correspondence between phenotype and genotype. The banding pattern on a certain electrophoretic gel equates to the individual's phenotype for the particular protein system being stained for. Hence, by observing the phenotype (number and migration pattern of bands), it is possible to immediately note the genotype (the alleles responsible) of an individual, for a particular protein system. e.g., for an albumin gel, phenotypically, if a sample separates into two bands, an A and a B band, the genotype is recorded to be AB. i.e.,

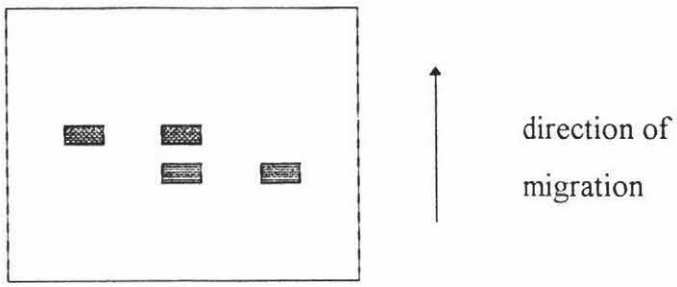


Figure 1.03 :A schematic diagram of the three possible electrophoretic patterns of albumin proteins on a starch gel.

By determining each horses genotype for all nine protein systems and seven blood groups, an individual blood profile can be created for every horse sampled. Unless the population is severely inbred, the probability of two horses having identical profiles is extremely low. For example, about one in twenty thousand for Thoroughbreds, and about one in two hundred thousand for Standardbreds. Blood types were collected from four hundred and eight Kaimanawa horses, a sample size believed to be large enough to represent the entire population.

It is unlikely that nascent electrophoretic banding patterns or blood groups will be revealed. Instead it is possible that this research may elucidate banding patterns rarely observed in blood samples from other New Zealand horses.

Once a blood profile (with respect to each of the sixteen genetic markers examined) for each horse has been obtained, the frequency of each allele within the Kaimanawa horse population will be calculated. The resultant data will then be compared with the frequencies of such alleles amongst known equine breeds within New Zealand, namely Thoroughbreds, Arabs and Standardbreds. Blood samples will be taken from Station Hacks of the Kaimanawa periphery and they too were tested for their genotypes with regard to the sixteen genetic markers. By comparing the allele frequencies of the protein and blood group polymorphisms between the Kaimanawa and the Station Hack sample groups, the influence that Station Hacks have had on the blood lines of the feral herd should be determined. Blood profiles for other New Zealand equine breeding types are readily available on databases from which sample populations will be selected.

1.4.4 The Equine Types Studied.

Before going any further with a study of the genetic differences between various equine types, some discussion of the holistic differences is required. In turn, this necessitates a basic understanding of the equine history.

Numerous forms of *Equus* existed in the glacial and post glacial periods, with their differences being accentuated by the effects of varying environments. It is now believed that the foundations of the domestic horse can be attributed to a) the heavy, slow moving Forest horse (*Equus caballus silvaticus*) of the northern European marshlands, b) Przewalski's Asiatic Wild Horse (*Equus caballus przewalskii przewalskii*), and c) the tarpan (*Equus caballus gmelini*) of Eastern Europe and the Ukrainian Steppes. (Edwards, 1993).

Initially, horse breeds and types developed gradually, by adapting to their enviroment, and through the natural kinship that existed between groups of horses occupying particular regions. Once the horse was domesticated (in Eurasia about five to six thousand years ago, at the end of the Neolithic period), however, human intervention accelerated and altered the development of specific breeds and types. The practice of gelding male horses, allowing breeding to be carried on by a selective process from only the best stock, increased the

quality and accentuated the characteristics most suited to the purposes to which the animals were put.

There are now approximately one hundred and sixty distinctive breeds and types of horse throughout the world. The modern light horse population (hot-bloods and warm-bloods) are thought to descend from the Tarpan, Asian Wild Horses and crosses of the two. (Edwards, 1993). More recently, these light horses have the Arabian horse as their common ancestor.

1.4.4.1 Arabian Horses.

The Arabian horse is unique in that it is the purest and oldest of all breeds, having been carefully bred for thousands of years. Exact origins of the Arabian horses (Arabs) are unclear, although evidence from artwork does show that they existed on the Arabian Peninsula in at least 2500 BC. The spread of Arabs throughout the world was made possible by the Muslim conquests in the seventh century when desert horses swept through Iberia into Christian Europe. However, it wasn't until the 1920's that Arabian horses were first introduced into New Zealand, from India. (Allfrey, 1980).

1.4.4.2 Thoroughbreds.

From the Arab was developed the bigger, faster Thoroughbred, which is now the essential element in the modern competition horse. The Thoroughbred evolved in England in the seventeenth and eighteenth centuries as a result of the crossing of imported Arab sires with a native stock of "running horses." These latter horses were created by mixing Spanish and Italian imports with the Irish Hobby and the Scottish Galloway, subsequently reinforcing with oriental blood. As a breed the Thoroughbreds are extremely well defined, in that their ancestry can be traced back to three foundation stallions, the Byerley Turk, the Darley Arabian and the Godolphin Arabian. See figure 1.04 for a typical Thoroughbred horse. With the exception of its progenitor, the Arab, the Thoroughbred has had more influence on any other breeds than any other horse, and has been used to improve horse and pony breeds throughout the world.

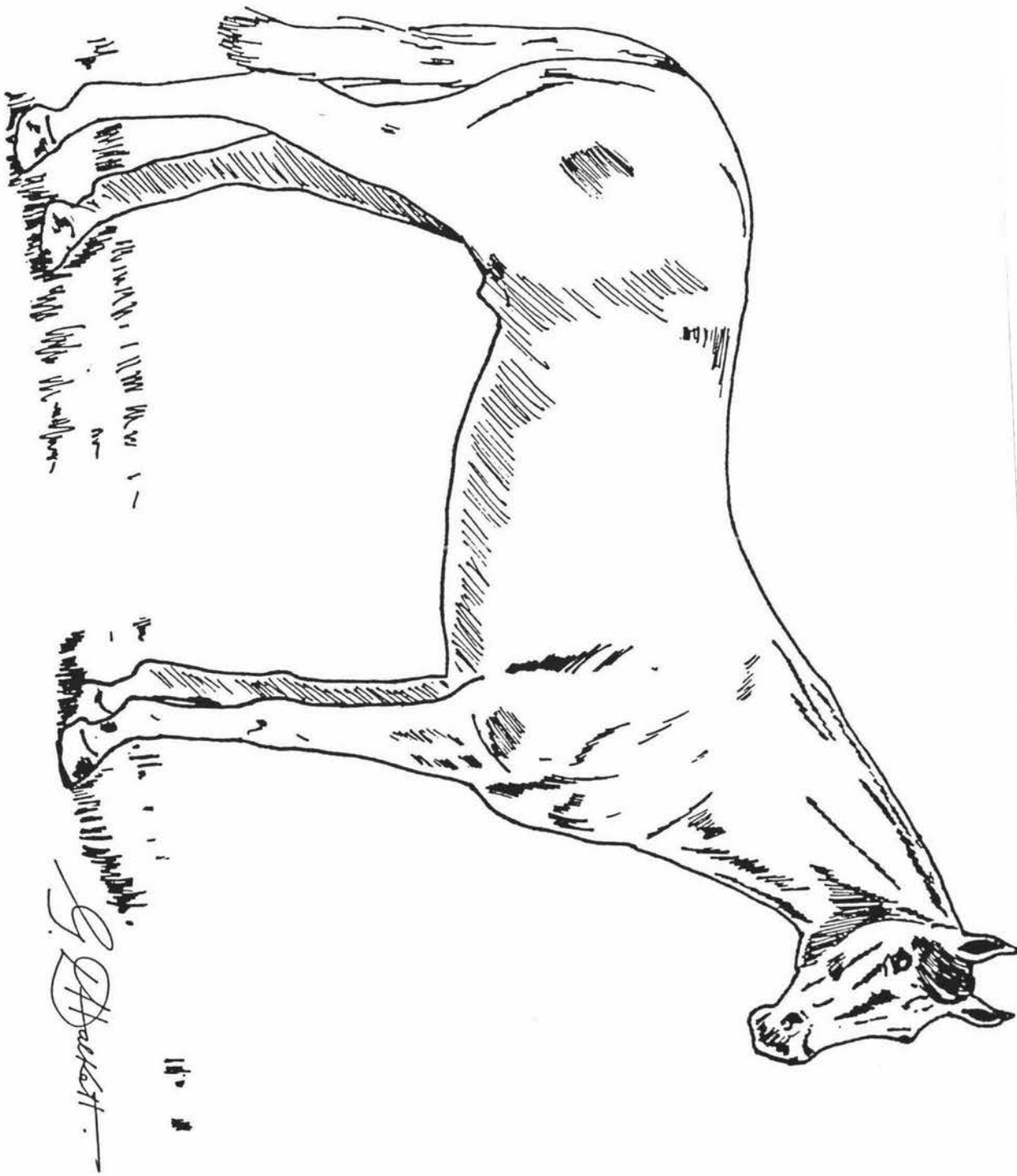


Figure 1.04: A typical Thoroughbred horse.

1.4.4.3 Standardbred horses.

Standardbred horses are referred to as "warm-bloods." In general terms this refers to the fact that they are half-breed or part-bred horses, the result of Thoroughbred or Arab crosses with other blood.

1.4.4.4 Station Hacks.

Dotted around the periphery of the Kaimanawa horses range are various sheep and cattle stations. Historically, a lot of the mustering around these stations was performed with the help of Station Hacks - work horses of no particular breed. Throughout the years the role of these horses has diminished greatly as the use of farm-bikes has increased. However, many of the stations still make use of Station Hacks. Generally, they have Arabian or Thoroughbred blood in them. (J. Roberts, pers comm.)

As these horses are in close proximity with the Kaimanawa horses, and both of the equine types are crossbreds, a sample of Station Hacks will be included in this comparative study.

1.4.4.5 Shire Horses.

At this stage, the only equine types to be incorporated in this study (and hence described above) are either pure-bred hotbloods or mixtures thereof (warmbloods). Therefore, in order to incorporate some form of outgroup[1] in this research, it has been decided to include a representative breed which has derived from the other main line of descent, the heavy, coldblooded horses which have descended from the Forest Horse.

---

[1] Having identified the genetic relationships existing between various populations of closely related breeds, it is often useful to incorporate an "outgroup" in the analysis. This is a population which is thought (from other biological and historical cues) to be less closely related to these populations as they are to each other. The purpose of this, is to obtain an indication as to the significance of the relationships initially studied. (Much in the same way that a domestic cat was used in studies to determine genetic relatedness amongst the modern, non-domestic felines, O'Brien *et al* (1984).)

Allowing for some crossing with the Asian Wild Horse and the latter effects of human intervention, it is agreed upon that the present day heavy horse breeds (cold-bloods) descend from the Forest Horse. (Edwards, 1993). These heavy horse breeds include any large draught horses such as Clydesdales and Shires, the latter of which allele frequency data is available.

Shires are named as such because they were bred in the Midland Shires of Lincoln, Leicester, Stafford and Derby and most probably descend from England's medieval war horse, the Great Horse. In medieval England, these English Great Horses were developed to be strong enough to carry a knight in plate armour, bearing heavy weapons, and still be agile in combat. The principal influence in the evolution of the massive, modern Shire was the heavy Flemish or Flanders Horse. During the sixteenth and early seventeenth centuries, Dutch contractors draining the English Fenlands brought with them these strong horses. These crossed with the aforementioned stock of lighter hot bloods, producing the modern day Shire (figure 1.05) which remains one of the largest horses in the world.

1.4.5 Genetic Distance Measures to be used.

The term "genetic distance" refers to the extent of gene differences (or genomic differences) between populations or species that is measured by some numerical quantity. This quantity can be the number of codon substitutions per locus (between two populations) or, as in the case of this research, the frequency of particular alleles at certain loci.

Two of the most commonly used measures of transforming allele frequency data into genetic distances are to be employed in this investigation. They are Roger's Genetic Distance (Rogers (1972) and Nei's Standard Genetic Distance (Nei (1972)).

1.4.5.1 Roger's Genetic Distance Measure.

The basic, Rogers' equation is:

$$Dp = [\sum_{i=1}^{m} (x_i - y_i)^2]^{1/2}$$

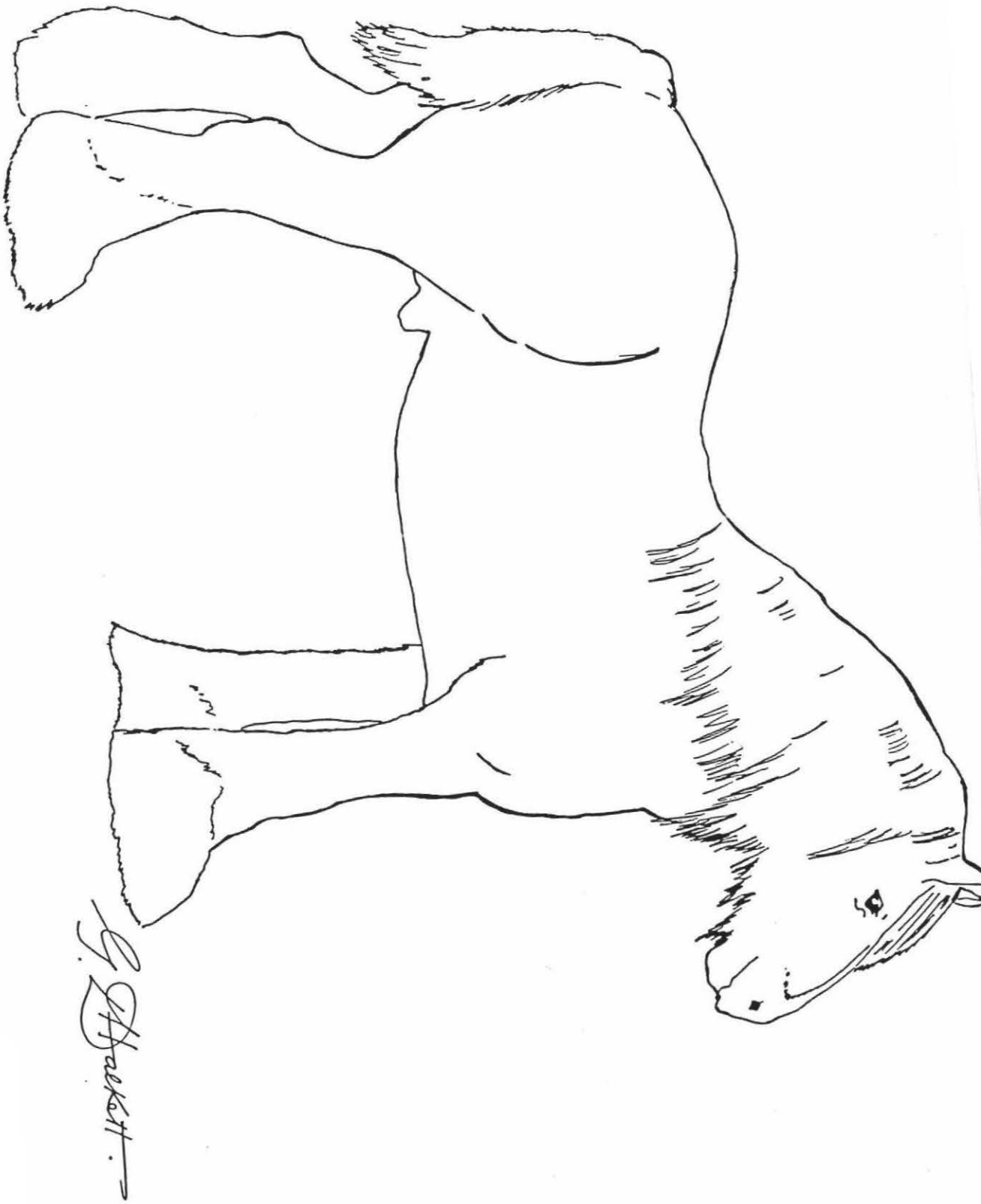


Figure 1.05: The modern Shire horse.

This gives the sum of all the gene frequency differences between two populations, with respect to a single protein system.

m = the number of alleles at a particular locus or within a protein system.

x_i and y_i = the frequencies of the i th allele in populations X and Y respectively.

Each allele frequency can take a value between 0 and 1. Consequently, Dp can range between 0 and $\sqrt{2}$. (Nei, 1987). This latter value is obtained when the two populations are fixed for different alleles, i.e., the two populations are completely different with respect to the frequency of alleles for one protein system. For example, consider the following hypothetical situation. Two populations (I and II) are compared on the basis of the frequency of gene frequencies at the Albumin locus. This locus has two alleles (A & B) and hence three possible genotypes (AA, AB & BB). Every individual in population I has the genotype AA whilst every individual in population II has the genotype BB, i.e., both populations are fixed for the different alleles. Hence, the gene frequencies for each population are as follows:

	Gene frequencies	
	Allele A	Allele B
population I	1.0	0
population II	0	1.0

$$Dp = \left[(1.0-0)^2 + (0-1.0)^2\right]^{1/2}$$
$$= [1+1]^{1/2}$$
$$= \sqrt{2}$$

Oishi (1976) modified Rogers' equation so that it could be applied for dealing with more than one loci.

Once D_p is calculated for every protein system, the following "D" equation is implemented.

$$D = \frac{1}{l}\sum_{m=1}^{l} Dp$$

This gives a genetic distance between two populations, based on the sum of their gene frequency differences for l number of protein systems studied. So as to avoid any confusion throughout this investigation, "D" will be referred to as D_R.

1.4.5.2 Nei's Standard Genetic Distance.

This method was originally designed to measure the number of nucleotide, or codon differences per unit length of DNA. (Nei, 1972). However, at the time of it's design, sequencing of nucleotides was expensive and time consuming even for a short length of DNA. The DNA hybridisation techniques available were too crude to be used for detecting a small number of nucleotide differences that would occur among local populations within a species. Hence Nei proposed to use allele frequency data in this method and subsequently estimate the number of codon differences per locus. Hence, this method is highly applicable for calculating genetic distances with reference purely to allele frequency data.

Consider two populations, X and Y in which multiple alleles are segregating at a locus. Let x_i & y_i be the frequencies of the ith alleles in X and Y respectively. The probability of identitiy of two randomly chosen genes is:

$j_x = \sum x_i^2$ in population X, while it is,

$j_y = \sum y_i^2$ in population Y.

The probability of identity of a gene from X and a gene from Y is $j_{xy} = \sum x_i y_i$

The normalized identity of genes between X and Y is defined as:

$$I = \frac{J_{XY}}{\sqrt{J_X J_Y}}$$

J_X, J_Y and J_{XY} are represent the arithmetic means of j_x, j_y and j_{xy} respectively, over all loci including monomorphic loci. I is equal to one when the two populations have the same alleles with the same frequencies at all loci studied, whereas it is equal to zero when they have no common alleles at any locus. The genetic distance between X and Y is then measured by $-\ln I$.

$$D_N = -\ln I$$

In contrast to I, D_N ranges from zero to infinity, whereby a value of zero indicates no difference between the two populations, with respect to allele frequency.

1.4.6 Representing the Data.

In order to gain some form of visual appreciation of the genetic distances calculated, these values are to be represented in both phylogenetic trees and split decomposition networks.

1.4.6.1 Neighbour Joining.

Devised by Masatoshi Nei (1972), this is a constructive method of phylogenetic tree reconstruction in which the optimal relationships between the populations are displayed. The resultant trees are referred to as being "unrooted," in that they have no starting point and do not make any evolutionary inferences. Instead, they only describe relationships. Trees are made up of paths and edges, the latter of which can be further subdivided into internal and external edges. A path is simply a connection between two taxa and is actually the sum or collection of all edges that join two taxa. See figure 1.05.

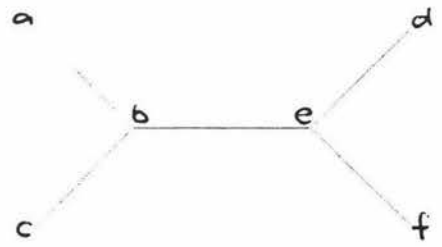


Figure 1.06: The components of a phylogenetic tree resulting from a Neighbour Joining construction. An example of an internal edge is length 'b' to 'e.' An external edge is length 'a' to 'b.' One of the paths on this tree is the length from 'c' to 'd.'

Phylogenetic trees represent hypotheses, diagrammatically of the relationships between populations. Such hypotheses can lead to predictions, which in turn can be independently tested against via information other than that from which the tree was initially built, such as historical and morphological characteristics. In this particular study, the relationships suggested by the phylogenetic trees will be discussed in light of other such information.

1.4.6.2 Split Decomposition Networks.

Rather than building phylogenetic trees, the Split Decomposition Analysis (Bandelt & Dress, 1992) constructs networks between taxa or phylogeny groups. In terms of the relationship between two such groups, these networks show several possible relationships which are supported by the genetic distance data, not just the most optimal relationship.

1.5 General Aims.

The overall objective of this investigation is to obtain an understanding of the genetic similarities and differences between the Kaimanawa Horses and other equine types, in terms of their alleles at sixteen genetic markers which are found in blood.

This will be achieved by comparing allele occurrances and frequencies within the Kaimanawa Horses with those of the five other equine types. Such comparisons will be both qualitative and quantitative.

Ultimately, the genetic distance between the Kaimanawa horses and the aforementioned equine types, based upon allele frequency differences and similarities among all of the populations should be deduced. These genetic distances will subequently be illustrated in a phylogenetic tree.

2. Methods and Materials.

2.1 Raw Data.

Blood types of the Kaimanawa horses, the Arabs, the Thoroughbreds and the Station Hacks were obtained from laboratory work performed at the Equine Blood Typing and Research Centre at Massey University, whereas information about the Standardbreds was obtained from the equivalent laboratory at Lincoln University. Information about the Shire horses was obtained from the equine drug testing laboratory at the University of Kentucky.

2.1.1 Obtaining Raw Material.

Each horse to be included in the sample population had approximately 10mL of blood drawn via venupuncture from its' jugular vein into an anticoagulating ACD (acidulated citrate dextrose) vacutainer. This was centrifuged at 3000 rpm for ten minutes so as to separate the blood into its three constitutive layers.

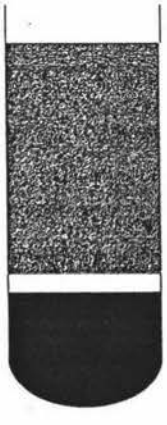


Figure 2.01: A schematic diagram illustrating the three main constituents of blood.

One millilitre of plasma was then pipetted into an eppendorf tube and frozen until required for electrophoretic testing. The remaining plasma and white blood cells (buffy coat layer) help to preserve the red blood cells and so were left on top of the red blood cells until the latter were able to be processed. At that time, any remaining serum and white blood cells were then pipetted off the red blood cell layer and discarded. The red blood cells were then washed twice in saline, prior to testing for the presence of specific red blood cell factors.

In order to obtain raw data (i.e., allele frequencies amongst each of the five equine populations sampled) the "blood type" of each horse included in the sample was ascertained. The term "blood type" is used to include both the determination of traditional blood groups (via serological means) plus the profile of polymorphic proteins (via electrophoretic means) present in plasma and red blood cells. See table 2.01 for a comprehensive listing of all the blood groups and polymorphic proteins tested for in this project.

2.1.2 Electrophoresis

2.1.2.1 Acid Polyacrylamide Gels.

The polymorphic forms of the esterases and protease inhibitors were detected on acid polyacrylamide gels. These were made and run according to the method described by Pollitt and Bell, 1983, with the exception that that only 27.5 grams of Cacodylic acid was used per ten litres in the cathode buffer, rather than the stipulated 110g/10L. This was due to the short supply of cacodylic acid in recent years, however, using only one quarter of the specified amount had no detrimental effects on the running of the gels. (pers. comm: J. Kenny, 1995).

2.1.2.2 Alkaline Polyacrylamide Gels.

The presence of the inherited variants of vitamin D binding protein, esterase, α-1-β glycoprotein and transferrin were all determined by running alkaline polyacrylamide gels, following the method of Juneja, Gahne and Sandberg, 1978.

Blood Group Systems.

System	Factors	Recognized Alleles
A	*a b c* d e *f g*	A^a A^{adf} A^{adg} A^{abdf} A^{abdg} A^b A^{bc} A^{bce} A^c A^{ce} A^e A^-
C	*a*	C^a C^-
D	*a b c d e f g h i k l m n* o p q r	D^{adl} D^{adlnr} D^{adlr} D^{bcmq} D^{cefgmq} $D^{cegimnq}$ D^{cfgkm} D^{cfmqr} D^{cgm} D^{cgmp} D^{cgmq} D^{cgmqr} D^{cgmr} D^{deklr} D^{deloq} D^{delq} D^{dfklr} D^{dghmp} D^{dghmq} D^{dghmqr} D^{dkl} D^{dlnq} D^{dlnqr} D^{dlqr} D^q
K	*a*	K^a K^-
P	*a b* c d	P^a P^{ac} P^{acd} P^{ad} P^b P^{bd} P^d P^-
Q	*a b c*	Q^{abc} Q^{ac} Q^a Q^b Q^c Q^-
U	*a*	U^a U^-

Electrophoretic Systems.

System	Locus Symbol	Recognized Alleles
A1B glycoprotein	A1B	F K S
Albumin	Al	A B I
Carboxylesterase	Es	F G H I L O R S
Vitamin D binding protein	Gc	F S
Phosphohexose isomerase	PHI	F I L S
Hemoglobin	Hb	AI AII BI BII N V
6-phosphogluconate dehydrogenase	PGD	D F S
Protease inhibitor	PI	F G H I K L L_2 N O P Q R S T U V W Z
Transferrin	Tf	D D_2 E F_1 F_2 F_3 G H_1 H_2 J M O R

Blood factors in italics are those tested for at the EBTRC.

Table 2.01: Blood Group Systems and Electrophoretic Systems Utilized in this Study.

2.1.2.3 Starch Gels.

To find out which variants of 6-phospho-gluco dehydrogenase, and phosphohexose isomerase were present, haemolysates were electrophoresced on starch gels in accordance with the method described by Bengtsson & Sandberg, 1973.

The variants of albumin were also determined by starch gel electrophoresis, using Gahne's method from 1966.

2.1.2.4 Agarose Isoelectric Focusing Gels.

Haemolysates were also electrophoresced on agarose isoelectric focusing gels so as to determine which polymorphic form of haemoglobin-αwas present. For this procedure, the method described in the instruction manual for the electrofocusing LKB1802-EF gel apparatus was implemented.

2.1.3 Serology.

2.1.3.1 General Reactions.

The blood of each horse sampled was tested for the presence of the red cell factors listed in table 2.02. This testing was performed in accordance with the method described by Suzuki & Stormont, 1964. Note that this procedure relies on typical antibody-antigen reactions whereby each reagent/antibody will react with only one specific red cell factor/antigen. Positive reactions were usually identified by agglutination. (See figure 2.02)

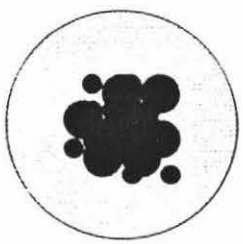


Figure 2.02: The agglutination reaction consists of the binding of antibody with specific cell surface antigens of cells that are in suspension. Such binding results in the formation of multicellular aggregates that cannot remain in suspension and instead rapidly settle on the bottom of the well in the microtitre plate.

Red Blood Cell Factor	Positively Identified By:	
	Agglutination	Haemolysis
A^a	*	
A^b		*
A^c		*
A^f	*	
C^a		*
D^a	*	
D^b	*	
D^c	*	
D^d	*	
D^e	*	
D^f	*	
D^g	*	
D^h	*	
D^k	*	
D^m	*	
D^n	*	
K^a	*	
P^a		*
P^b		*
Q^a		*
Q^b		*
Q^c		*
U^a		*

Table 2.02: Identification of positive reactions to antisera.

The antisera used to test for the presence of red blood cell factors A^a, A^f, D^a, D^b, D^c, D^d, D^e, D^f, D^g, D^h, D^k, D^m, D^n and K^a consist of IgM type antibodies. Whereas, red blood cell factors A^b, A^c, C^a, P^a, P^b, Q^a, Q^b, Q^c, and U^a are tested for by antisera consisting of IgG type antibodies. Generally, antibodies of the IgM class cause relatively strong agglutination, whereas antibodies of the IgG class often produce only a weak reaction or no reaction at all. Such weak reactions can be enhanced by the addition of high molecular weight compounds (e.g., complement) to the reacting mixture of cells and antibodies. Addition of this immunoglobulin makes the reaction more visible. This is referred to as complement fixation. Complement is the thermolabile group of proteins in normal blood serum. (Webster, 1986). It is activated by antibodies and consequently it damages the outer cell membrane of red blood cells. Lysozymes are then able to pass through the hole made by the complement and hence the cell is ruptured. (See figure 2.04). The resultant effect is haemolysis. (See figure 2.03).

If an animal registered a positive reaction to a particular reagent, say D^a, that animal was then said to contain factor Da as part of its range of red cell antigenic determinants. A comprehensive panel of twenty two monospecific antisera reagents representing all the recognised blood groups were used, facilitating the identification of the red cell blood group status of each horse sampled.

The polymorphisms identified by the occurrence of agglutination are shown in table 2.03:

Blood System	Reagents
A	A^a A^f
D	D^a D^b D^c D^d D^e D^f D^g D^h D^k D^m D^n
K	K^a

Table 2.03: Reactions in which agglutination indicates a positive reaction.

Polymorphisms identified by haemolysis are listed in table 2.04. For these reactions, complement was added because each of these antisera were of the IgG type.

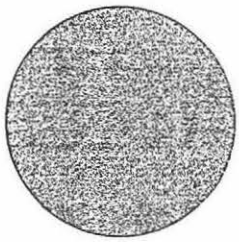


Figure 2.03: Haemolysis, as seen in a microtitre well containing complement, red blood cells and antibodies specific to the red blood cell antigens. This signal provides visual confirmation that the antisera have bound to their specific antigens.

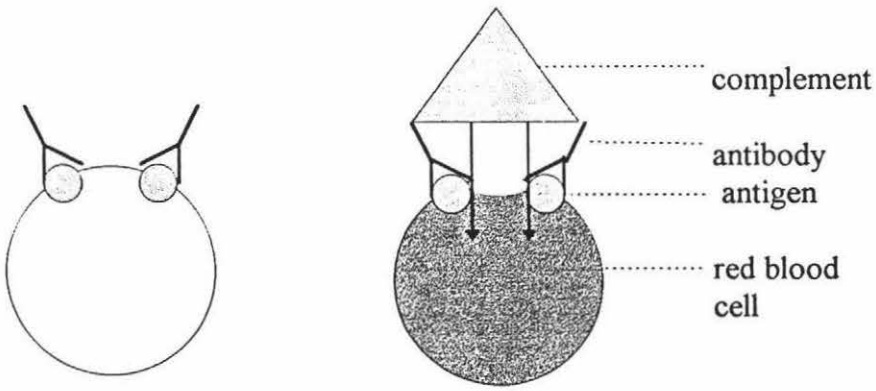


Fig 2.04: The action of complement upon a singular red blood cell.

Blood System	Reagents
A	A^b A^c
P	P^a P^b
Q	Q^a Q^b Q^c
U	U^a
C	C^a

Table 2.04: Reactions in which haemolysis indicates a positive reaction. Note that the reagents are actually antisera which were given the same label as the blood factors which they tested for.

The C, K and U systems, each contained a single factor determined by two recognised alleles. Within the other systems, especially the two major systems (A & D) certain factors occurred in well defined specific combinations which were determined by allelic genes. These combinations of factors e.g., D^{cegmn} were referred to as phenogroups.

All serological results were subsequently noted on record sheets.

2.1.3.2 Making Complement.

Complement was made following the method described by Stormont and Suzuki, 1964 and used rabbit blood supplied by the Small Animal Production Unit at Massey University.

2.2 Data already available.

Allelic frequencies for Thoroughbred, Arabian and Standardbred populations, were obtained from existing databases for these breeds at the equine blood typing laboratories at Massey University (Thoroughbred and Arab) and Lincoln University (Standardbred).

Since its establishment, the Equine Blood Typing and Research Centre at Massey University has blood typed over 60, 000 thoroughbred horses. Hence, in order to obtain a random sample of the Thoroughbreds, a small computer programme was created (by Lim-Computers, Palmerston North), which allowed a random sample of 120 entries to be chosen.

(A single entry details the full bloodtype for an individual horse, with respect to blood group factors and polymorphic protein systems.) The sample was checked to ensure no one particular sire or dam was over-represented. i.e., there are approximately 500 thoroughbred stallions active at any one time in New Zealand. Each of the 120 individuals chosen for inclusion in my sample had a different dam, but were sired by 95 different stallions. Hence, it was decided that the sample population of thoroughbreds was not overly biased by parentage as it is not unusual for one Thoroughbred stallion to breed with more than one mare, (e.g., Kingdom Bay, 102; Centaine, 99; and Imperial Seal, 60; 1993 figures.) Hence it is reasonable to expect a sample population of Thoroughbreds to have fewer sires than there are individuals in the sample.

Arabian horses are bloodtyped far less frequently than Thoroughbreds. Hence, every third record of these horses was chosen for inclusion in a sample population of 129 individuals.

Standardbred information was provided by the equine blood typing laboratory at Lincoln University. One hundred and fifty seven bloodtypes were included in this sample population.

Allele frequency data (based on 276 horses) for Shire horses was provided by Dr. G. Cothran of Lexington University, Kentucky.

2.3 Processing Data.

Every horse included in this study had its entire bloodtype initially recorded on a laboratory worksheet. These were then transcribed into a spreadsheet using Microsoft Excel. A separate spreadsheet was used per sample population. For example, see table I.i.

2.3.1 Calculating allele frequencies from the electrophoretic results.

As can be seen in table I.i, each of the nine protein systems studied were represented in individual columns. Each of these columns underwent a "sort" command from within the "data" menu which grouped all of the same genotypes together in ascending alphabetical

order (table I.ii). Each sorted protein column was then copied and pasted into it's own spreadsheet. Using these spreadsheets, the frequency of each allele was determined within each sample population. (I.iii).

All allele frequencies within each protein system were checked to ensure they summed to one.

2.3.2 Serological Gene Frequencies.

Calculating the gene frequencies of alleles in blood groups A, P and Q is not simply a matter of directly counting from the phenotypes. For example, the Aaf phenotype may be either of the Aaf/af, Aaf/a or Aaf/- genotypes. Parentage studies would possibly give an indication as to what the other allele may be although, independent assortment would not guarantee this. Hence it was necessary to use the extended Hardy Weinburg equation for solving gene frequencies for multi-allelic series. By finding the frequency of the null allele (-), it was then possible to iteratively deduce the frequency of all other alleles in the series. Consider as an example the Thoroughbred Blood Group A.

Phenotype	Possible Genotypes	Frequency (n=120)
-/-	-/-	0.017
af/	af/af or af/-	0.941
af/b	af/b	0.025
b/	b/b or b/-	0.017

Whereby, alleles -, b and af occur in the population with a frequency of p,q and r, respectively.

It is assumed that each of the populations are in Hardy Weinburg equilibrium. This in turn assumes that a) mating is random within the genotypes being considered, and b) no external forces act on the genotypes to change their frequencies.

The following matrix demonstrates the frequency (in italics) of each possible genotype (in regular type) within a population which is assumed to be in Hardy Weinburg equilibrium:

	-	b	af
	p	*q*	*r*
-	-/-	b/-	af/-
p	p^2	*pq*	*pr*
b	b/-	b/b	af/b
q	*pq*	q^2	*qr*
af	af/-	af/b	af/af
r	*pr*	*qr*	r^2

Given these conditions, genotypic frequencies are expressed by the formula:

$$p^2 + 2pq + 2pr + q^2 + 2qr + r^2 = 1$$

Hence, as the frequency (p^2) of the -/- genotype is known, the frequency (p) of the - allele can be readily determined:

$$p^2 = 0.017$$

$$\therefore\ p = 0.017^{1/2}$$

$$\therefore\ p = 0.130$$

This can then be used to find the frequency (q) of allele b. The phenotype b/ has two possible genotypes, the homozygous b/b and the heterozygous b/-, each with frequencies of q^2 and 2pq respectively.

$$\therefore\ q^2 + 2pq = 0.017$$

By taking the quadratic, $p^2 + 2pq + q^2$ out of the main Hardy Weinburg equation, and using the previously determined p value, a frequency value (q) for allele b can be resolved.

$$p^2 + 2pq + q^2 = 0.017 + 0.017$$

$$\therefore\ = 0.034$$

$$\therefore\ (p + q)^2 = 0.034$$

$$\therefore\ (p + q) = 0.184$$

$$\therefore\ q = 0.184 - p$$

$$\therefore\ = 0.184 - 0.130$$

$$\therefore\ = 0.054$$

Since the frequency of all alleles in this system must sum to one [$(p^2 + 2pq + 2pr + q^2 + 2qr + r^2)=1$], this can be used to determine a frequency value (r) for allele af.

i.e., $p^2 + 2pq + 2pr + q^2 + 2qr + r^2 = 1.000$

$\therefore \quad (p + q + r) = 1.000$

$\therefore \quad 0.130 + 0.054 + r = 1.000$

$\therefore \quad r = 1.000 - (0.130 + 0.054)$

$\therefore \quad = 0.816$

Determining gene frequencies of alleles in the D system is straightforward. Since this system has no null alleles, it is possible to simply count the number of times each phenotype occurs. i.e., for this system each phenotype maps directly to only one genotype.

Assuming Hardy-Weinburg equilibrium, the gene frequency of the (recessive) null allele in the C, K and U systems was found by taking the square root of the recessive genotype, as these systems are simply comprised of two alleles, a and (-).

For example, the Thoroughbred C system. Assume the frequencies of genes (-) and a are represented by p and q, respectively. The sum of the two frequencies must be equal to one.

the frequency of the null allele (-) was:

-/- $= 14/120$

$= 0.117$

$= p^2$

$\therefore \quad p = 0.342$

to find the frequency of the 'a' allele:

$p + q = 1$

$\therefore \quad q = 1 - 0.342$

$\therefore \quad = 0.658$

Protein System	Allele	$(Xi_j - Xi_k)$	$(Xi_j - Xi_k)^2$	$[\sum_{i=1}^{n}(Xi - Xi)^2]$	$[\sum_{i=1}^{n}(Xi - Xi)^2]^{1/2}$
Al	A	0.360	0.129		
	B	-0.360	0.129	0.259	0.509
PGD	D	0.154	0.024		
	F	0.205	0.042		
	S	-0.359	0.129	0.195	0.441
PHI	F	0.054	0.003		
	I	-0.054	0.003	0.006	0.076
Gc	F	-0.030	0.001		
	S	0.030	0.001	0.002	0.042
Q	-	0.118	0.014		
	b	0.000	0.000		
	c	0.323	0.104		
	bc	0.000	0.000		
	a	0.000	0.000		
	abc	-0.442	0.195	0.313	0.560
U	-	-0.010	0.000		
	a	0.010	0.000	0.000	0.014
					3.730
			Genetic Distance:		0.233

Table 2.05: Calculating Rogers' Genetic Distance Between the Kaimanawa and Thoroughbred Horses.

2.4 Genetic Comparisons of Populations.

As a basis for calculating genetic distances via either Roger's (Roger, 1972) or Nei's (Nei, 1972) methods, a master spreadsheet entitled "All Allele Frequencies" was created. See table 3.01. This spreadsheet had every sample population labelled across the first row, with the protein systems and frequency of each allele tested listed down the page.

2.4.1 Rogers' comparative measure of genetic distance.

A single spreadsheet was used to calculate Roger's distances amongst all populations. Within this spreadsheet, single columns performed part of the total formula. See table 2.05. N.b., Roger's extended formula:

$$D_R = \frac{\sum_{m=1}^{l} D_P}{l}$$

where

$$D_P = \left[\sum_{i=1}^{m} (X_{ij} - X_{ik})^2\right]^{1/2}$$

Table 2.05 shows six of the sixteen protein systems used to calculate Rogers' genetic distance between the Kaimanawa horses and Thoroughbreds. The first and second columns contain the protein system names and ninety two allele names, respectively. The third column contains the difference in the frequency of each allele between Kaimanawa Horses and Thoroughbred horses. (i.e., $X_{ij} - X_{ik}$).

The difference values in the third column were squared to give the fourth column, so as to remove any negative differences. (i.e., $(X_{ij} - X_{ik})^2$

The fifth column contains the sum of the squared difference values from the fourth column for each of the sixteen protein systems. (i.e, $\left[\sum_{i=1}^{m} (X_{ij} - X_{ik})^2\right]$). The final column contains the square root of column five. (i.e., $\left[\sum_{i=1}^{m} (X_{ij} - X_{ik})^2\right]^{1/2}$). Thus, the final column contains D_p for each protein system studied. All occupied cells of the final column were then

summed to arrive at a total of all the D_ps for that particular population comparison. (i.e., $\sum_{m=1}^{l} D_P$ =3.73 for the Kaimanawa-Thoroughbred population comparison).

Finally, this figure was divided by the number of protein systems which were compared between the two populations, $\frac{\sum_{m=1}^{l} D_P}{l}$. (0.233 for the Kaimanawa-Thoroughbred comparison).

This value is the genetic distance, using Roger's extended calculations, between the two horse populations, based upon a) sixteen certain protein systems, and, b) the horses included in these particular sample populations.

2.4.2 Neis' Standard Genetic Distance.

As with Rogers' distance calculations, a single spreadsheet was used to calculate Neis' Standard Genetic Distances between the six horse populations. Neis's Standard Genetic Distance formula:

$$D_N = -\ln I$$

$$I = \frac{J_{XY}}{\sqrt{J_X . J_Y}}$$

$$I = \frac{\frac{\sum x_i y_i}{n}}{\sqrt{\frac{\sum x_i^2}{n} . \frac{\sum y_i^2}{n}}}$$

Table 2.06 is a condensed version of the spreadsheet used to calculate D_N between the Kaimanawa and Thoroughbred horses, i.e., it only shows five of the sixteen protein systems included in the calculations.

The first and second columns contain the names of the proteins and alleles tested. Allele frequencies (x_i) for the Kaimanawa horses within each of the protein systems are listed in the third column. Each of these values is squared in the following column, to give x_i^2 values. The fourth column contains allele frequencies for the Thoroughbred horses (y_i) and these

Protein System	Alleles	x_i values	x_i^2 values	y_i values	y_i^2 values	$x_i y_i$ values
Albumin	A	0.556	0.309	0.196	0.038	0.109
	B	0.444	0.197	0.804	0.646	0.357
PGD	D	0.154	0.024	0.000	0.000	0.000
	F	0.788	0.621	0.583	0.340	0.459
	S	0.058	0.003	0.417	0.174	0.024
PHI	F	0.054	0.003	0.000	0.000	0.000
	I	0.946	0.895	1.000	1.000	0.946
Gc	F	0.953	0.908	0.983	0.966	0.937
	S	0.047	0.002	0.017	0.000	0.001
U	-	0.841	0.707	0.851	0.724	0.715691
	a	0.159	0.025	0.149	0.022	0.023691
Σ			9.722		10.200	9.170
Σ/n			0.608		0.638	0.573
Calculating I:		0.921				
Calculating D :		0.082				

Table 2.06: Calculating Neis' Standard Genetic Distance Between the Kaimanawa and Thoroughbred Horses.

Protein System	Alleles	x_i values	x_i^2 values	y_i values	y_i^2 values	$x_i y_i$ values
Albumin	A	0.556	0.309	0.196	0.038	0.109
	B	0.444	0.197	0.804	0.646	0.357
PGD	D	0.154	0.024	0.000	0.000	0.000
	F	0.788	0.621	0.583	0.340	0.459
	S	0.058	0.003	0.417	0.174	0.024
PHI	F	0.054	0.003	0.000	0.000	0.000
	I	0.946	0.895	1.000	1.000	0.946
Gc	F	0.953	0.908	0.983	0.966	0.937
	S	0.047	0.002	0.017	0.000	0.001
U	-	0.841	0.707	0.851	0.724	0.715691
	a	0.159	0.025	0.149	0.022	0.023691
Σ			9.722		10.200	9.170
Σ/n			0.608		0.638	0.573
Calculating I:		0.921				
Calculating D :		0.082				

2.06: Calculating Neis' Standard Genetic Distance Between the Kaimanawa and Thoroughbred Horses.

too are squared (y_i^2) in the following column. Columns three and five were then multiplied together, resulting in x_iy_i values which are presented in the seventh column. The fourth, sixth and seventh columns were subsequently summed and divided by the number of protein systems studied so as to obtain $\frac{\sum x_i^2}{n}$, $\frac{\sum y_i^2}{n}$ and $\frac{\sum x_iy_i}{n}$ values. These values equate to the values of J_X, J_Y and J_{XY} which are used to calculate I at the base of the spreadsheet. In order to obtain standard a genetic distance value this normalised identity value is multiplied by a negative natural log function (-ln), resulting in D_N.

Once genetic distance values were obtained from both calculation methods (Rogers' and Neis'), they were entered into a 6x6 matrix. See table 3.03.

2.4.3 Reconstructing a Phylogenetic Tree.

Once genetic distance values were obtained from either of the two calculation methods (Roger's and Nei's) a matrix of the distances was entered into the PHYLIP computer programme (PHYLogenetic Inference Package) version 3.5, (Felsenstein, 1993). PHYLIP was based on the Neighbour Joining method for reconstructing phylogenetic trees. This programme produces a reconstruction of the relationships which were originally constructed as a result of evolution.

The Neighbour Joining method was devised in 1987 by N. Saitou and M. Nei (Saitou & Nei, 1987) whilst the computer package was designed by Joseph Felsenstein (Felsenstein, 1993). This programme gave the internal and external branch lengths of the best possible resultant tree using the given data. The lengths given were in the order of 10^{-2} and so were multiplied by 10^3 and regarded as being in centimetre lengths (see table 3.04) so as to form a scaled representation on the page. See figures 3.26 and 3.27.

2.4.4 Split Decomposition Analysis.

Within any set of genetic distance data, there are various signals for the strength of relationships amongst the populations or taxa. For example, in a hypothetical comparison, the genetic distance between population A and population B may be 5, whilst the genetic distance between populations A and C may be 3. Hence, the relationship between populations A and C is stronger than that between A and B. However, the relationship between A and C is both supported and contradicted in that there is a signal (albeit weaker) in the data for population A to be related to another population, B.

Hence, in order to visualise both support and opposition within the distances data for particular equine relationships, the distance matrices constructed from the genetic distance calculations were run through the Splits Tree computer programme, version 1.0, copyright 1994, Daniel Hudson and Rainer Wetzel, Germany. The resultant networks can be seen in figures 3.28 and 3.29.

2.4.5 Venn Diagrams.

Venn diagrams were created as a means of visualising the ninety four alleles of the sixteen protein systems tested, which are commonly shared amongst each of the equine types studied. Conversely, they also depict mutually exclusive zones.

Two venn diagrams were constructed, one encompassing all of the equine types studied (figure 3.31), and another illustrating the intersecting regions between the Kaimanawa horses and the two equine types with the closest genetic distance to the Kaimanawa horses, the Station Hacks and the Thoroughbreds, (figure 3.30).

3. Results.

3.1 Aspects of Obtaining Raw Data.

Figures 3.01 to 3.09 show the sequence of events from collecting blood to analysing for various traits, thereby providing the raw data required for this genetic distance analysis.

Figure 3.01 displays a group of drafted colts. After each herd of Kaimanawa horses had been mustered into a large holding pen, they were drafted according to age and sex. Each horse was then individually led into a restraining device so that a blood sample (figure 3.02) could be taken.

Subsequent to spending approximately sixteen hours in destain (figure 3.03), acid polyacrylamide gels were packaged in vacuum sealed plastic (figures 3.04). Such packaged gels can remain legible for up to two years depending on conditions such as light and humidity.

Figure 3.05 illustrates a starch gel which had been stained for the presence of albumins and was consequently ready for packaging. Albumin gels such as the above are sliced in half producing two mirror images; a helpful technique in the event of one half being blurred. This particular gel clearly shows examples of each possible genotype for this system. From the left; 3rd run is AB, 4th run is BB & the 7th run is AA.

Figure 3.01: Drafted Kaimanawa horses.

Figure 3.02: Collecting blood from Kaimanawa horses in 1994

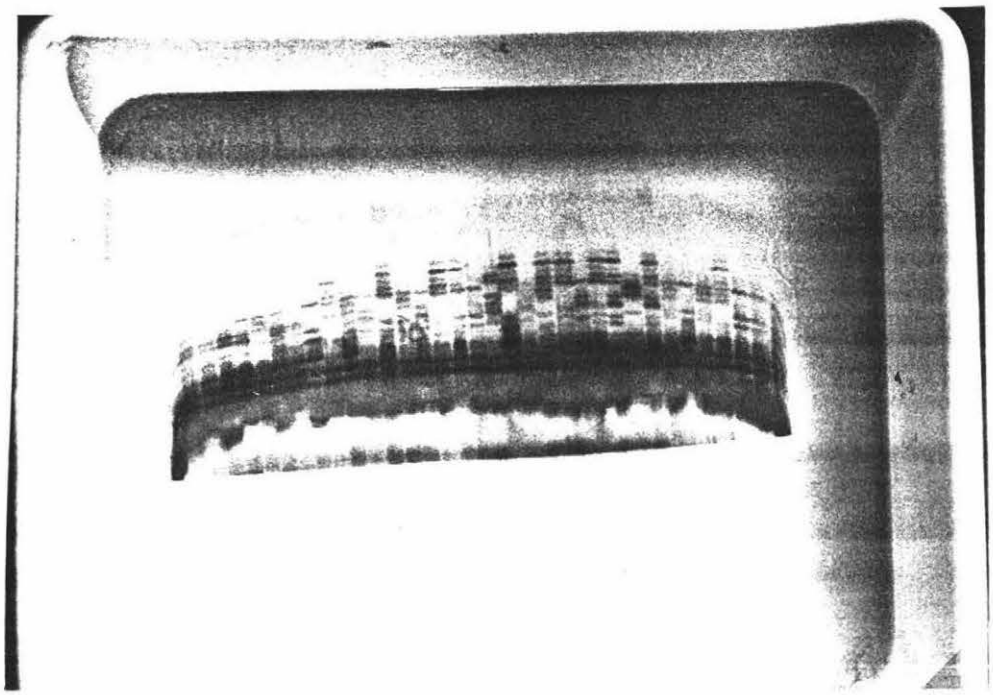


Figure 3.03: An acid polyacrylamide gel in destain.

Figure 3.04: Acid polyacrylamide gels that have been vacuum packed in plastic will remain intact for several years.

A PGD gel with gluconate-6-phosphate stain setting on top of it is shown in figure 3.06. Although faint, it is possible to discern between the different genotypes of the proteins (stained grey) present in the gel. i.e., from the right; 1st run is FF, 2nd is FS & 6th is SS.

In order to distinguish between the various PHI phenotypes, fructose-6-phosphate stain was poured over PHI gels such as that in figure 3.07. As with the PGD gels, the red areas indicate where each blood sample was loaded, whilst the polymorphic proteins manifest as grey banding patterns. All runs in this particular gel represent II genotypes.

Figure 3.08 shows a microtitre plate used to determine the presence of the following blood group alleles; A^a, A^f, D^a, D^b & D^c. Note that the rows of wells are labelled A - H, whereas the columns are labelled 1 - 12. Well A1 contains an example of agglutination (note the fuzzy presence around the periphery of the blood sample), which indicates a positive reaction, whereas D1 contains an example of a negative reaction (the blood sample remains in the bottom of the well and its' periphery has remained uniform.).

Reactions to the antisera A^b, A^c, P^a & P^b are shown in figure 3.09. A positive reaction to P^a is indicated by haemolysis, as can be seen in well E1. A negative reaction to P^a can be seen in well E3.

3.2 Blood types of horses sampled.

Appendix II contains the blood types of each horse included in this study. Where possible, every endeavour was made to obtain a complete bloodtype for each horse. However, this was occasionally not possible and hence there are a few individuals (e.g., K91) for whom not all sixteen genotypes are known. In such cases that particular cell has simply been left blank.

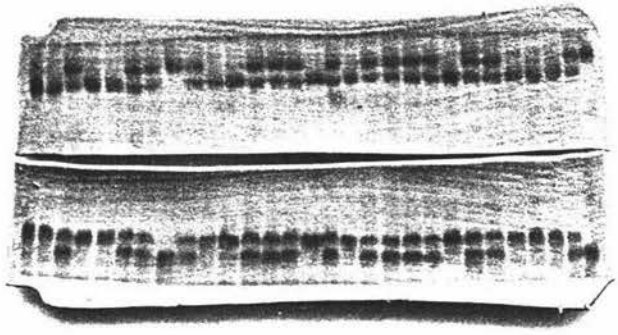


Figure 3.05: A starch gel stained for Albumin proteins, ready for packaging.

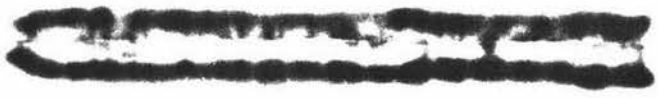


Figure 3.06: A starch gel being stained for the presence of migrating PGD proteins.

Figure 3.07: A starch gel being stained for the presence of migrating PHI proteins.

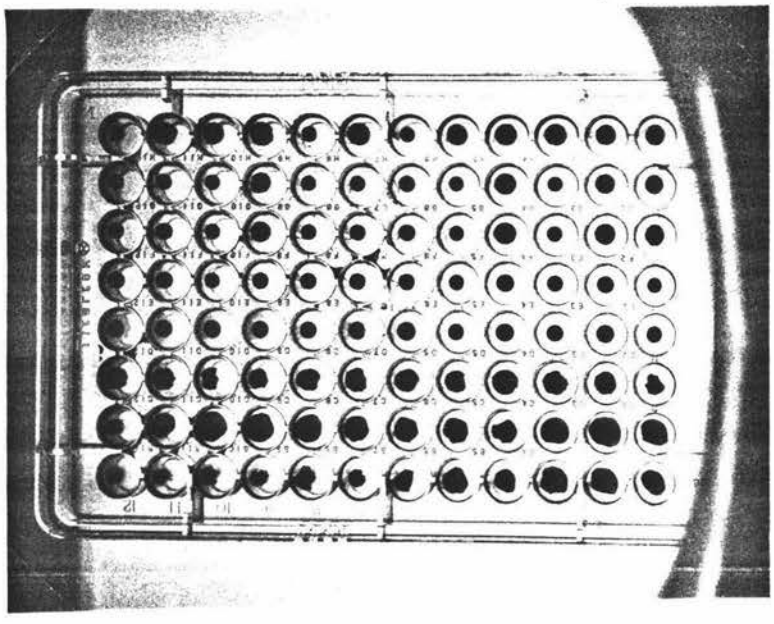


Figure 3.08: A microtitre plate used to test for the presence of agglutinating antibody-antigen complexes.

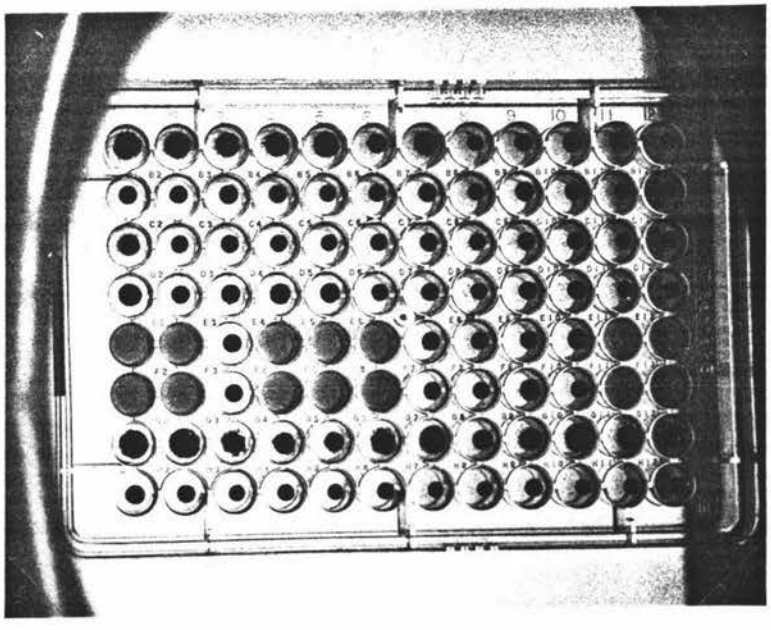


Figure 3.09: A microtitre plate used to test for the presence of haemolysing antibody-antigen complexes.

The entire Standardbred sample (table II.iv) was not tested for Blood Group C and resultantly, the blood type of each Standardbred horse included genotypes for only fifteen protein systems.

3.3 Allele Frequencies.

Allele frequencies which were calculated via the methods described in 2.3.1 and 2.3.2 have been presented in table 3.01. The frequency data for the Shire horses was provided by Gus Cothran (pers comm.) and has been incorporated without modification into this table.

3.4 Allele Frequency Bar Charts and the Chi-square analysis.

Bar graphs for each protein system were created (and presented in figures 3.10 - 3.25) in order to visualise a) whether the equine types studied have the same alleles, and b) whether the frequencies of each allele differ amongst the various equine types. To determine the significance of any differences between such allele frequencies in the Kaimanawa horses and the other New Zealand equine types studied, a chi-square analysis was performed. The null hypothesis of the significance test was that there is no difference between the allele frequencies of these equine types. Comparisons were regarded as being significantly different if they had a probability value (ρ) of less than or equal to 0.05. The results of this analysis are presented in table 3.02.

GENE FREQUENCIES

Protein System	Alleles	Kaimanawa	Thoroughbred	Arab	Standardbred	Stationhack	Shires
ELECTROPHORESIS							
Albumin	A	0.556	0.196	0.566	0.570	0.568	0.391
	B	0.444	0.804	0.434	0.430	0.432	0.609
PGD	D	0.154	0.000	0.000	0.003	0.023	0.115
	F	0.788	0.583	0.543	0.879	0.795	0.885
	S	0.058	0.417	0.457	0.118	0.182	0.000
PHI	F	0.054	0.000	0.004	0.147	0.000	0.000
	I	0.946	1.000	0.996	0.853	1.000	1.000
Gc	F	0.953	0.983	0.996	0.812	0.955	0.986
	S	0.047	0.017	0.004	0.188	0.045	0.014
Transferrin	D	0.233	0.279	0.217	0.236	0.455	0.308
	D2	0.000	0.000	0.000	0.000	0.000	0.069
	F1	0.000	0.250	0.004	0.003	0.045	0.000
	F2	0.372	0.179	0.434	0.541	0.273	0.467
	F3	0.000	0.000	0.000	0.000	0.000	0.000
	H2	0.208	0.058	0.178	0.003	0.114	0.000
	O	0.005	0.113	0.167	0.045	0.114	0.127
	R	0.182	0.121	0.000	0.172	0.000	0.029
Xk	F	0.000	0.000	0.000	0.019	0.023	0.007
	K	0.983	1.000	0.930	0.978	0.977	0.975
	S	0.017	0.000	0.070	0.003	0.000	0.018
Total		1.000	1.000	1.000	1.000	1.000	1.000
Pi	+	0.001	0.000	0.016	0.000	0.000	0.000
	F	0.147	0.071	0.114	0.016	0.068	0.000
	G	0.027	0.033	0.024	0.003	0.000	0.000
	H	0.000	0.000	0.000	0.013	0.023	0.520
	I	0.015	0.063	0.000	0.048	0.000	0.010
	J	0.157	0.000	0.000	0.000	0.023	0.000
	K	0.000	0.000	0.000	0.003	0.045	0.000
	L'	0.000	0.000	0.000	0.003	0.000	0.000
	L	0.207	0.454	0.382	0.143	0.227	0.092
	L2	0.001	0.004	0.012	0.137	0.045	0.041
	N	0.000	0.196	0.043	0.172	0.227	0.097
	O	0.000	0.000	0.000	0.022	0.000	0.015
	P	0.000	0.000	0.016	0.000	0.000	0.000
	R	0.000	0.000	0.000	0.000	0.000	0.046
	S1	0.153	0.033	0.165	0.003	0.091	0.123
	S2	0.291	0.025	0.004	0.003	0.091	0.000
	U	0.000	0.121	0.138	0.417	0.114	0.056
	V	0.000	0.000	0.000	0.003	0.000	0.000
	W	0.000	0.000	0.000	0.013	0.000	0.000
	Z	0.000	0.000	0.087	0.000	0.045	0.000
Es	R	0.000	0.000	0.000	0.000	0.000	0.076
	F	0.121	0.088	0.008	0.041	0.364	0.044
	G	0.000	0.000	0.000	0.102	0.000	0.083
	H	0.000	0.000	0.000	0.003	0.000	0.000
	I	0.842	0.858	0.988	0.736	0.591	0.728

Table 3.01: Allele frequencies.

	S	0.037	0.054	0.004	0.118	0.045	0.069
Total		1.000	1.000	1.000	1.000	1.000	1.000
Hb	A1	0.000	0.000	0.000	0.003	0.045	0.005
	A2	0.000	0.000	0.000	0.000	0.000	0.000
	B1	0.178	0.158	0.646	0.503	0.432	0.577
	B2	0.822	0.842	0.354	0.494	0.523	0.418
A	-	0.206	0.130	0.089	0.077	0.213	0.207
	b	0.143	0.054	0.037	0.497	0.156	0.070
	af	0.644	0.816	0.788	0.408	0.613	0.272
	c	0.007	0.000	0.014	0.000	0.009	0.062
	bc	0.000	0.000	0.000	0.018	0.009	0.058
	a	0.000	0.000	0.072	0.000	0.000	0.000
	adg	0.000	0.000	0.000	0.000	0.000	0.091
	ce	0.000	0.000	0.000	0.000	0.000	0.015
	bce	0.000	0.000	0.000	0.000	0.000	0.225
C	-	0.715	0.342	0.088	n/t	0.212	0.665
	a	0.286	0.658	0.912	n/t	0.788	0.335
D	bcm	0.101	0.244	0.180	0.047	0.092	0.054
	cegmn	0.213	0.178	0.049	0.000	0.136	0.000
	cgm	0.104	0.207	0.098	0.378	0.295	0.080
	de	0.155	0.041	0.144	0.082	0.136	0.037
	dk	0.068	0.274	0.529	0.196	0.227	0.007
	cefgm	0.000	0.000	0.000	0.000	0.046	0.167
	dn	0.090	0.016	0.000	0.000	0.023	0.000
	dghm	0.054	0.012	0.000	0.061	0.045	0.065
	cfmr	0.000	0.000	0.000	0.089	0.000	0.000
	cgmp	0.000	0.000	0.000	0.029	0.000	0.000
	dr	0.000	0.000	0.000	0.031	0.000	0.000
	d	0.000	0.000	0.000	0.003	0.000	0.431
	dfk	0.064	0.028	0.000	0.066	0.000	0.000
	adn	0.083	0.000	0.000	0.018	0.000	0.000
	ad	0.003	0.000	0.000	0.000	0.000	0.159
	dek	0.066	0.000	0.000	0.000	0.000	0.000
K	a	0.007	0.038	0.000	0.226	0.023	0.000
	-	0.993	0.962	1.000	0.774	0.977	1.000
P	-	0.698	0.658	0.628	0.863	0.570	0.087
	a	0.217	0.217	0.359	0.137	0.396	0.000
	b	0.085	0.126	0.013	0.000	0.034	0.000
	ac	0.000	0.000	0.000	0.000	0.000	0.565
	ad	0.000	0.000	0.000	0.000	0.000	0.094
	bd	0.000	0.000	0.000	0.000	0.000	0.152
	d	0.000	0.000	0.000	0.000	0.000	0.102
Q	-	0.646	0.428	0.352	0.977	0.664	0.833
	b	0.000	0.000	0.176	0.010	0.076	0.018
	o	0.369	0.046	0.325	0.000	0.205	0.146
	bc	0.000	0.000	0.000	0.000	0.026	0.000
	a	0.000	0.000	0.000	0.013	0.000	0.000
	abc	0.085	0.526	0.147	0.000	0.129	0.004
U	-	0.841	0.851	0.858	0.749	0.723	0.269
	a	0.159	0.149	0.142	0.251	0.277	0.731

Table 3.01 continued: Allele frequencies.

Protein System	Degrees of freedom	ρ value			
		Kaimanawa-Thoroughbred	Kaimanawa-Arabian	Kaimanawa-Standardbred	Kaimanawa-Station Hack
Albumin	1	0.000	0.842	0.764	0.912
PGD	2	0.000	0.000	0.000	0.024
PHI	1	0.009	0.014	0.000	0.263
Gc	1	0.141	0.024	0.000	0.966
Esterases	4	0.799	0.001	0.000	0.029
α-1 β	2	0.356	0.008	0.009	0.008
Protease inhibitors	18	0.000	0.000	0.000	0.000
Transferrin	6	0.000	0.000	0.000	0.000
Haemoglobin	2	0.879	0.000	0.000	0.000
A	5	0.018	0.000	0.000	0.000
C	1	0.000	0.000	0.000	0.267
D	15	0.000	0.000	0.000	0.000
K	1	0.011	0.341	0.012	0.000
P	2	0.408	0.000	0.000	0.128
Q	5	0.000	0.000	0.000	0.000
U	1	0.791	0.642	0.000	0.146

Table 3.02: Chi-square analysis.

The same frequencies of the alleles in the Albumin protein system, appear to be shared by Kaimanawa horses, Arabian horses, Standardbred horses and Station Hacks. See figure 3.10. These similarities are statistically supported in that, with respect to this system, the only New Zealand equine type to be significantly different to the Kaimanawa horses is the Thoroughbred horses.

Although the frequencies differ significantly, the Kaimanawa horses share the same alleles as Standardbreds and Station Hacks in the 6-Phosphogluco-dehydrogenase protein system. See Figure 3.11. It is interesting to note that the three equine types on this graph which are known mixtures (Kaimanawa, Standardbreds and Hacks), are all constituted of the same alleles with respect to this one particular system.

As can be seen in Figure 3.12, allele F is not commonly seen in the Phosphohexose isomerase protein system. However, it does manifest in the Kaimanawa horses, the Standardbreds and, to a small degree in the Arabs. However, according to the chi-square analysis, the only New Zealand equine type which is not significantly different to the Kaimanawa horses, within this protein system, are the Station Hacks.

The composition of the Kaimanawa sample, with respect to the alleles of the Vitamin D binding protein system (Gc), is similar to that of the Station Hacks. Allele F dominates in all types. See Figure 3.13. This is strongly supported by the chi-square analysis which also implies that, within this protein system, the Thoroughbreds do not differ significantly from the Kaimanawa horses.

The most distinct feature of Figure 3.14 is a parallel between the Kaimanawa horses and the Thoroughbred horses, both in the presence and frequency of each allele in the Esterase protein sytem. The chi-square analysis also shows that the Thoroughbred horses are the only New Zealand equine type which does not differ significantly from the Kaimanawa horses with respect to this protein system.

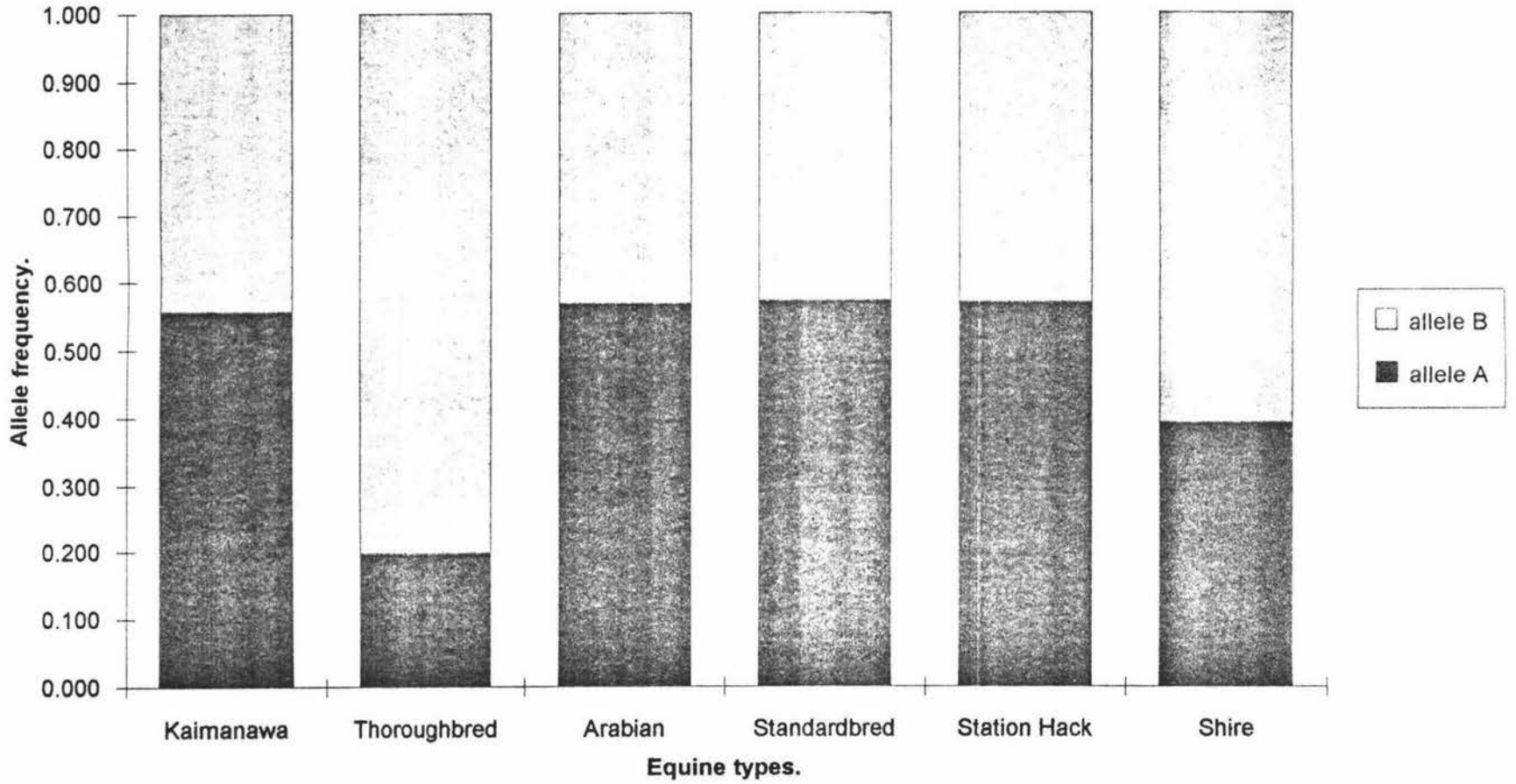


Figure 3.10: Alleles of the Albumin protein system present in the equine types studied.

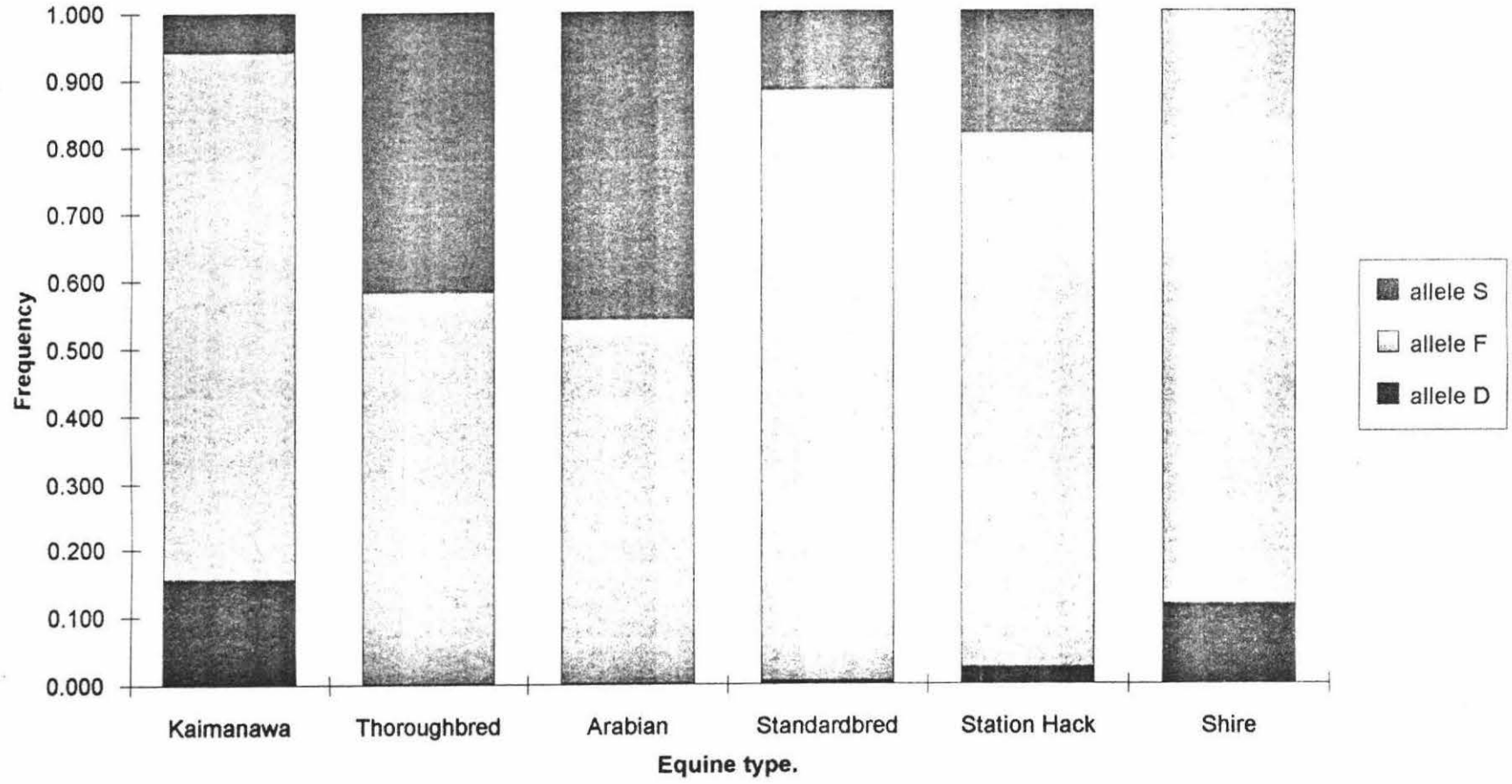


Figure 3.11: Alleles of the PGD protein system present in the equine types studied.

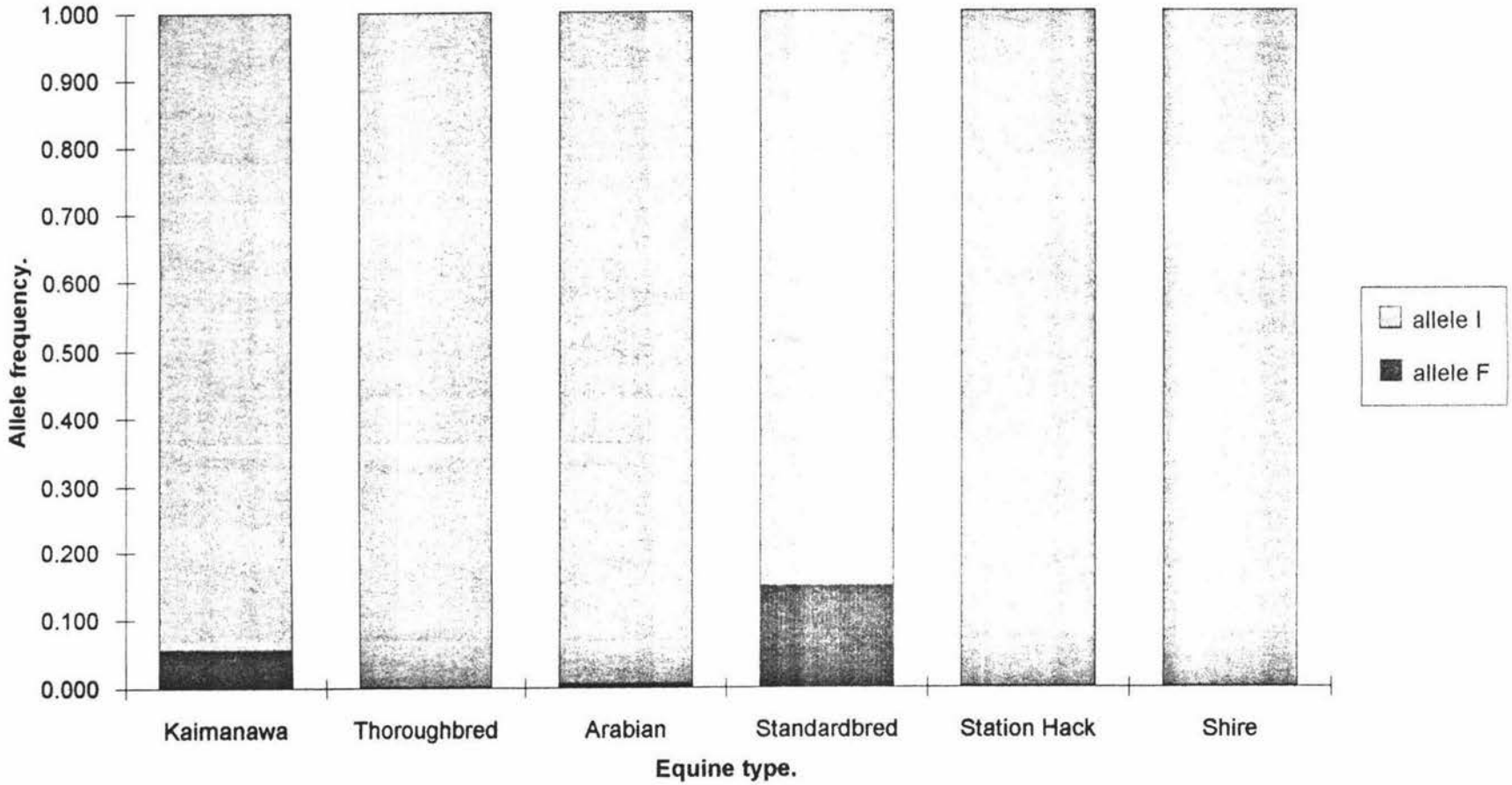


Figure 3.12: Alleles of the PHI protein system present in the equine types studied.

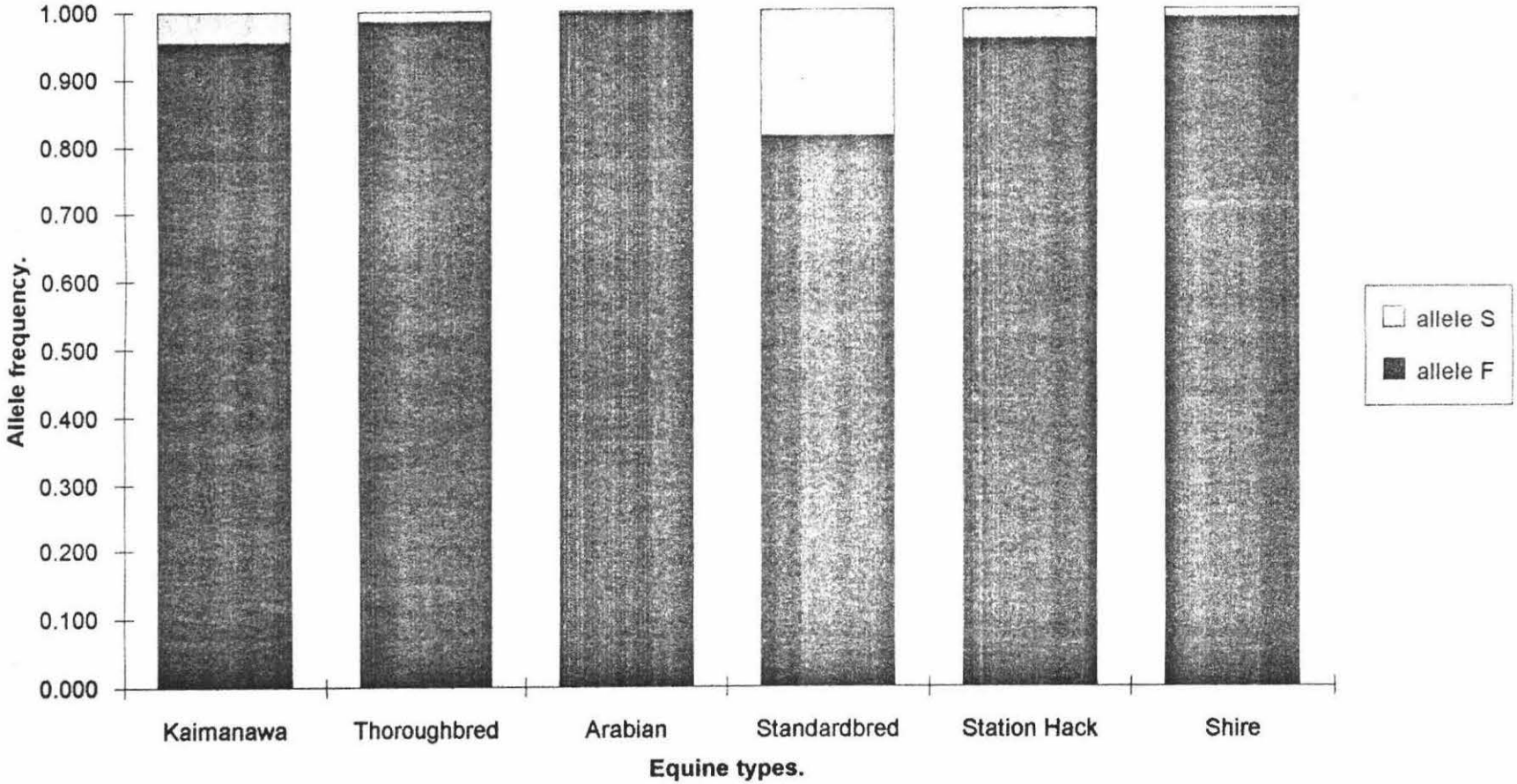


Figure 3.13: Alleles of the Vitamin D binding protein system present in the equine types studied.

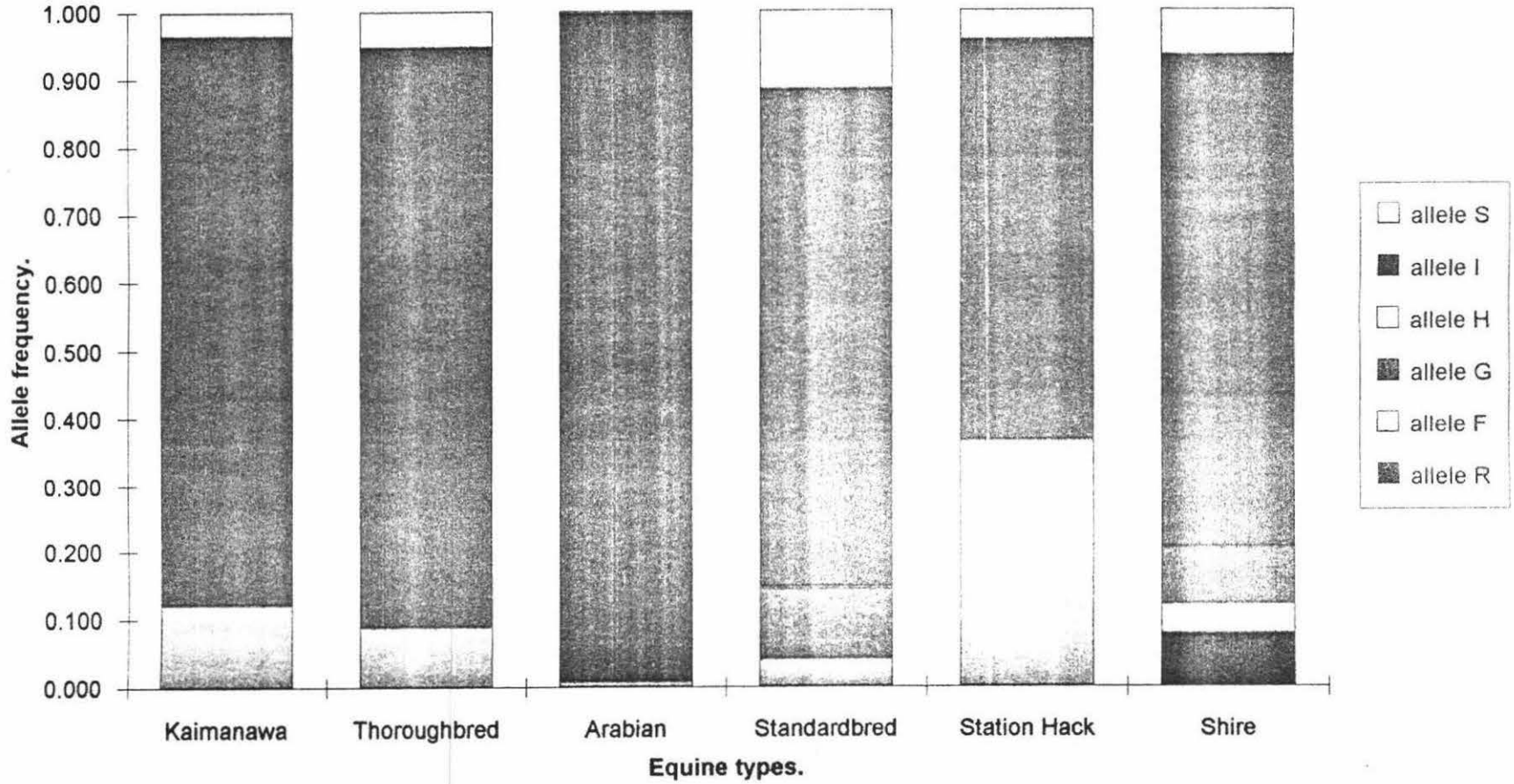


Figure 3.14: Alleles of the Esterase protein system present in the equine types studied.

Figure 3.15 illustrates the frequencies of the Alpha-1 Beta glycoprotein alleles within each of the equine types studied. The Kaimanawa horses have a frequency for allele S which is very similar to that in the Shire horses. The high incidence of allele K is prevalent amongst all equine types. However, in terms of allele frequency, within the α 1-β system, only the Thoroughbred horses are not significantly different compared with the Kaimanawa horses.

The alleles of the Protease Inhibitor system which are present in each equine type included in this study are shown in Figure 3.16. One particularly prominent feature of this graph is the presence of J alleles, occurring with a moderate frequency amongst the Kaimanawa horses. Throughout this entire study the J allele was only seen in one other horse, a Station Hack. In the 408 Kaimanawa horses studied to date, none have carried the U or N alleles, both of which are present in the other five equine types examined. This system is extremely multi-allelic and consequently, the chi-square analysis suggests that every New Zealand equine type studied is significantly different to the Kaimanawa horses.

With respect to the Transferrin protein system, alleles D, F_2 and H_2 are present in the same frequencies in both the Kaimanawa horses and the Arabian horses. The frequency of the R allele is the same for both the Kaimanawa horses and the Standardbreds. See Figure 3.17. Once again, this is also a highly multi-allelic protein system which differs significantly between the Kaimanawa horses and every other New Zealand equine type studied.

Of all the equine types studied, the Thoroughbred horses appear to bear the closest resemblance to the Kaimanawa horses, with respect the the Haemoglobin protein system. See figure 3.18. This is verified by the chi-square analysis which shows that within this protein system, the only equine type not to be significantly different from the Kaimanawa horses is the Thoroughbred horses.

In terms of both allele frequency and presence, in blood group A the Kaimanawa horses appear to bear the closest resemblence to the Station Hacks. This can be seen in figure 3.19. Whilst not as polymorphic as either the Protease inhibitor or Transferrin systems, blood

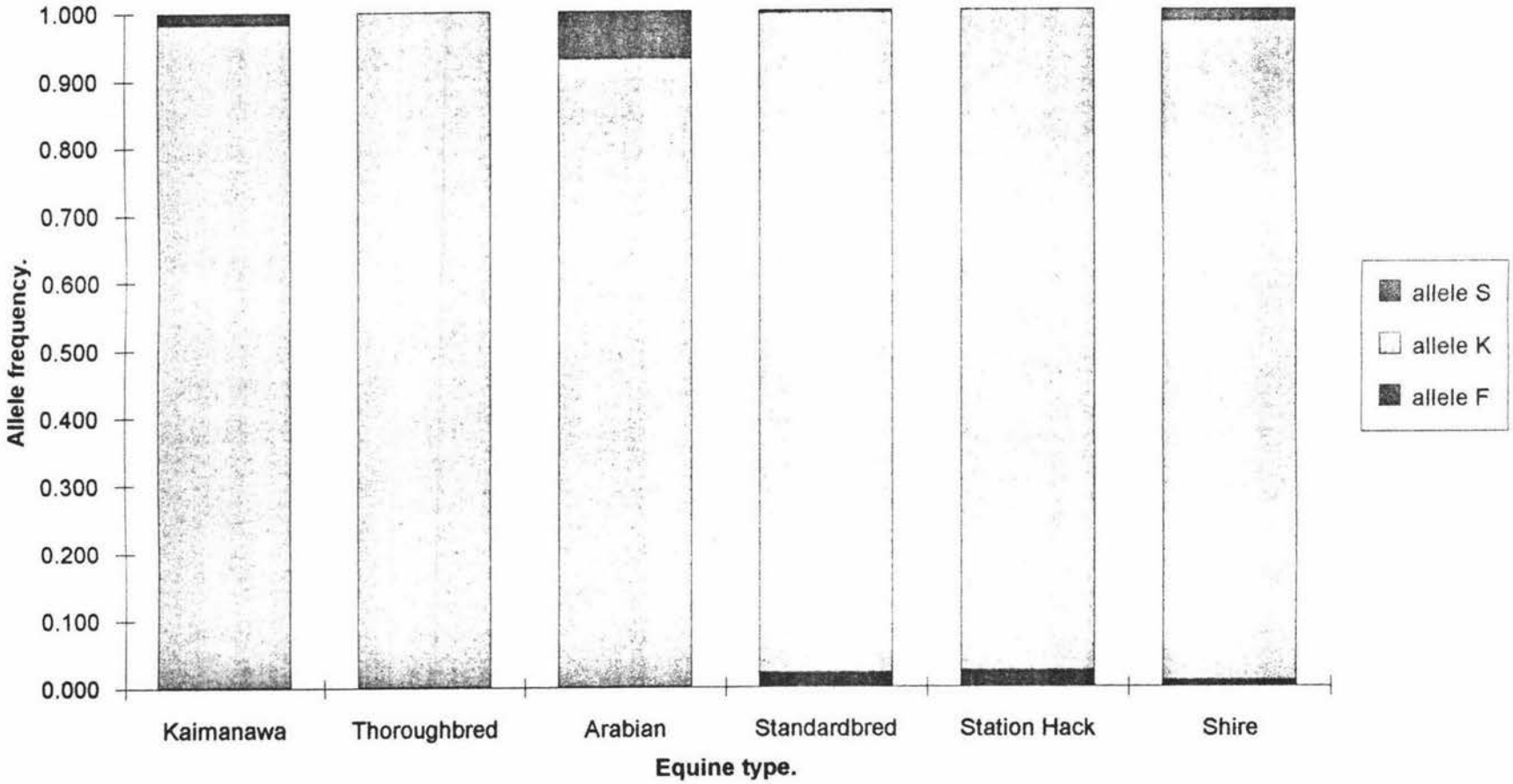


Figure 3.15: Alleles of the Alpha-1 Beta Glycoprotein system present in the equine types studied.

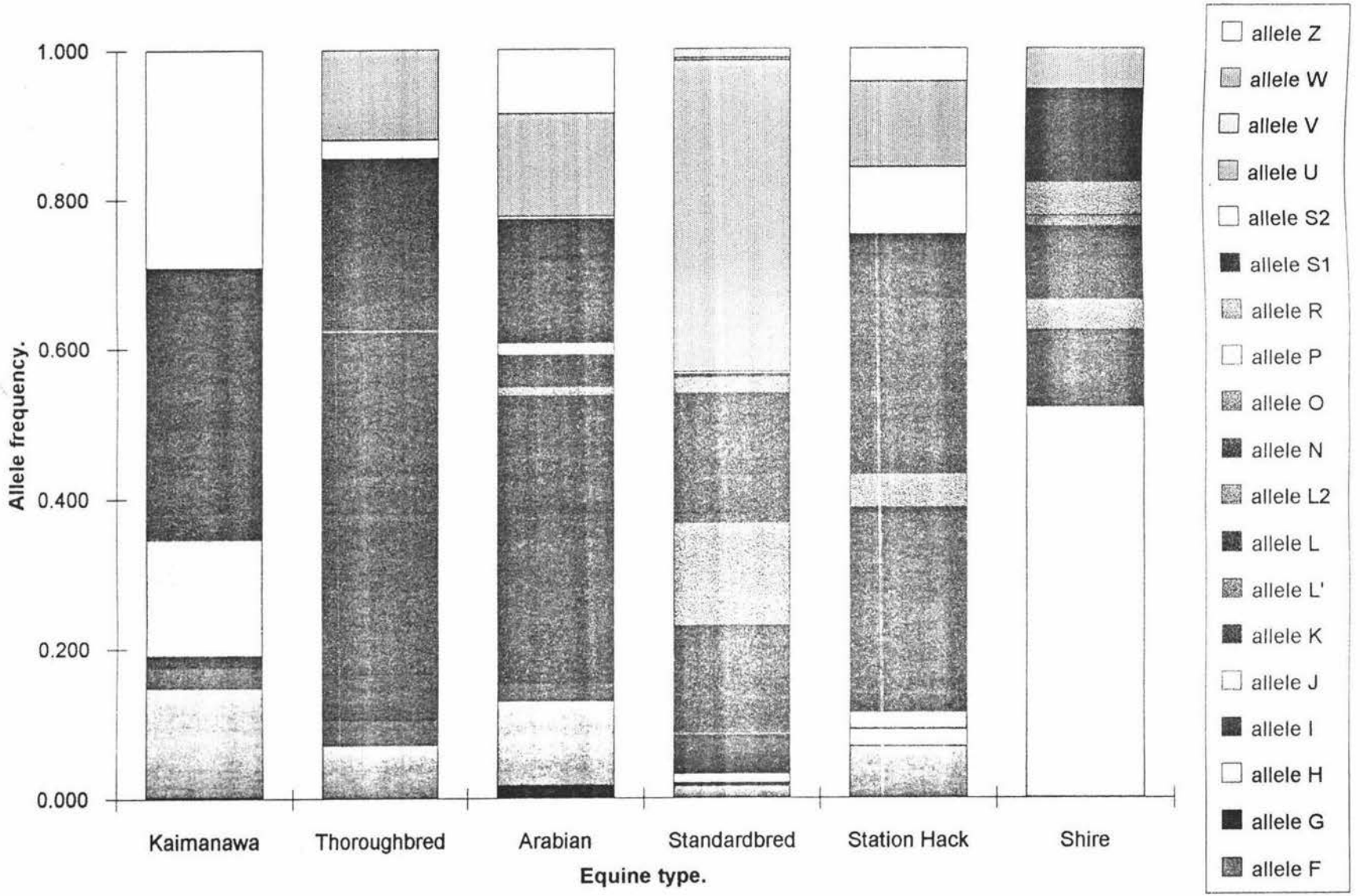


Figure 3.16: Alleles of the Protease inhibitor system present in the equine types studied.

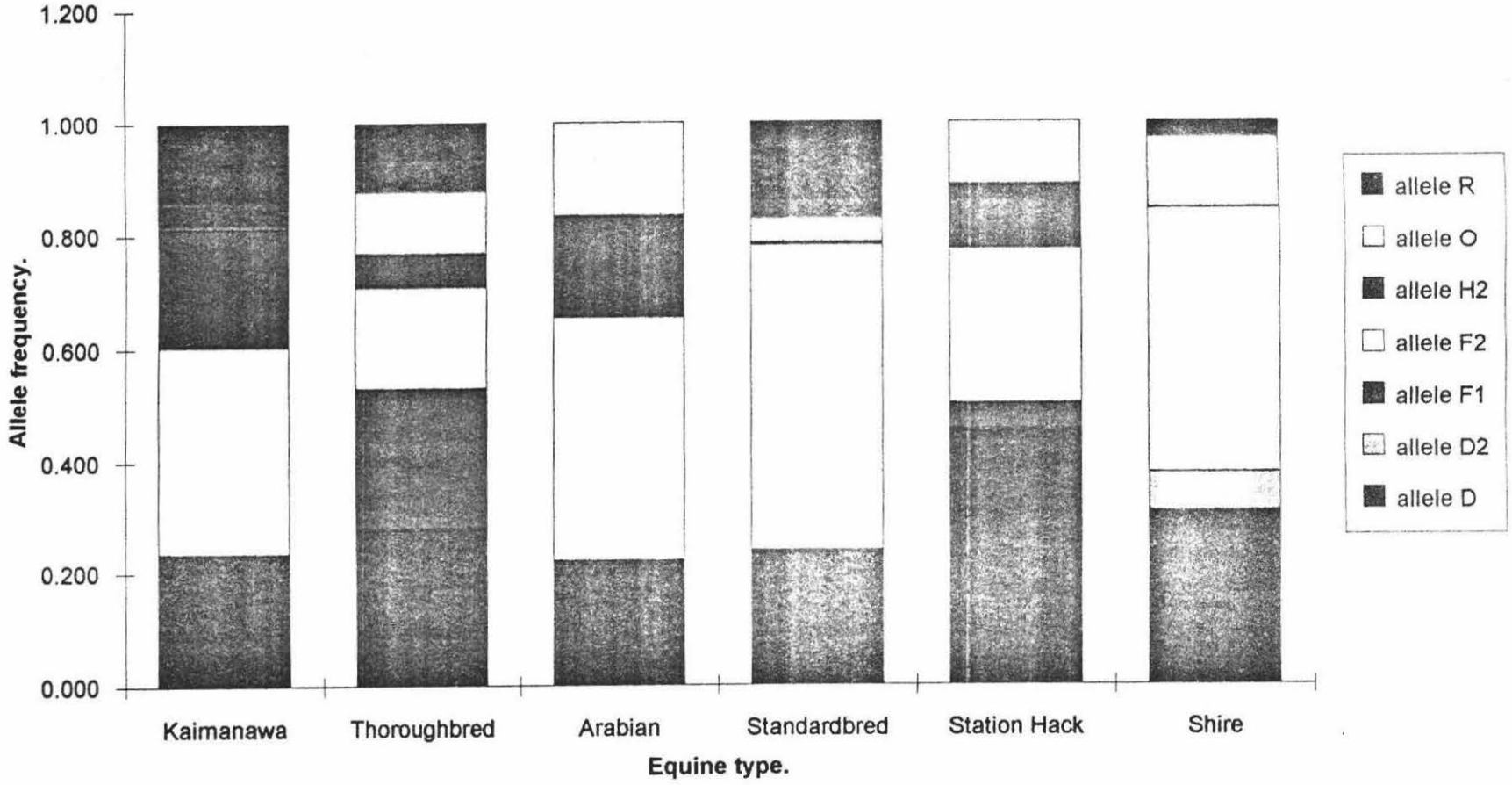


Figure 3.17: Alleles of the Transferrin protein system present in the equine types studied.

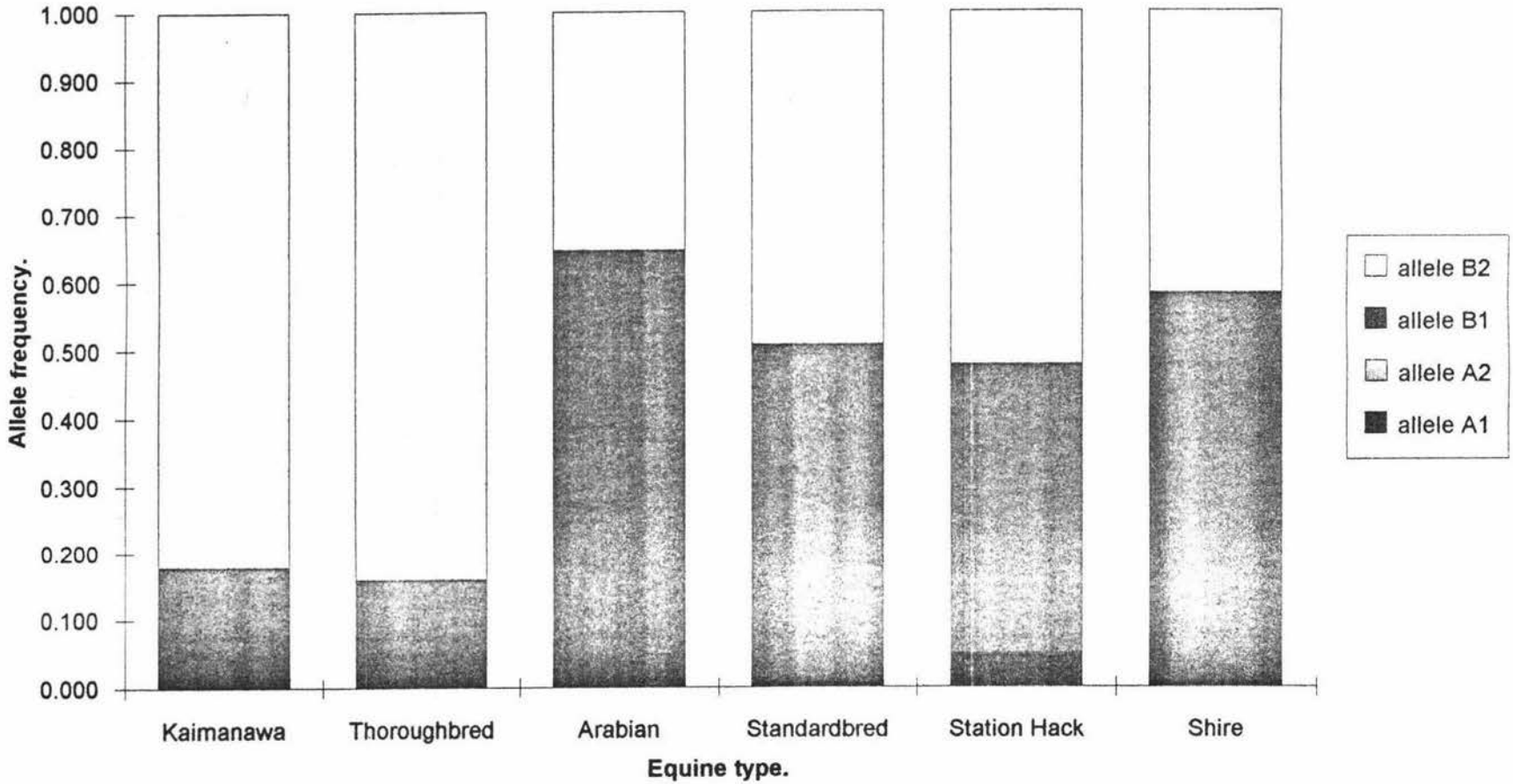


Figure 3.18: Alleles of the Haemoglobin protein system present in the equine types studied.

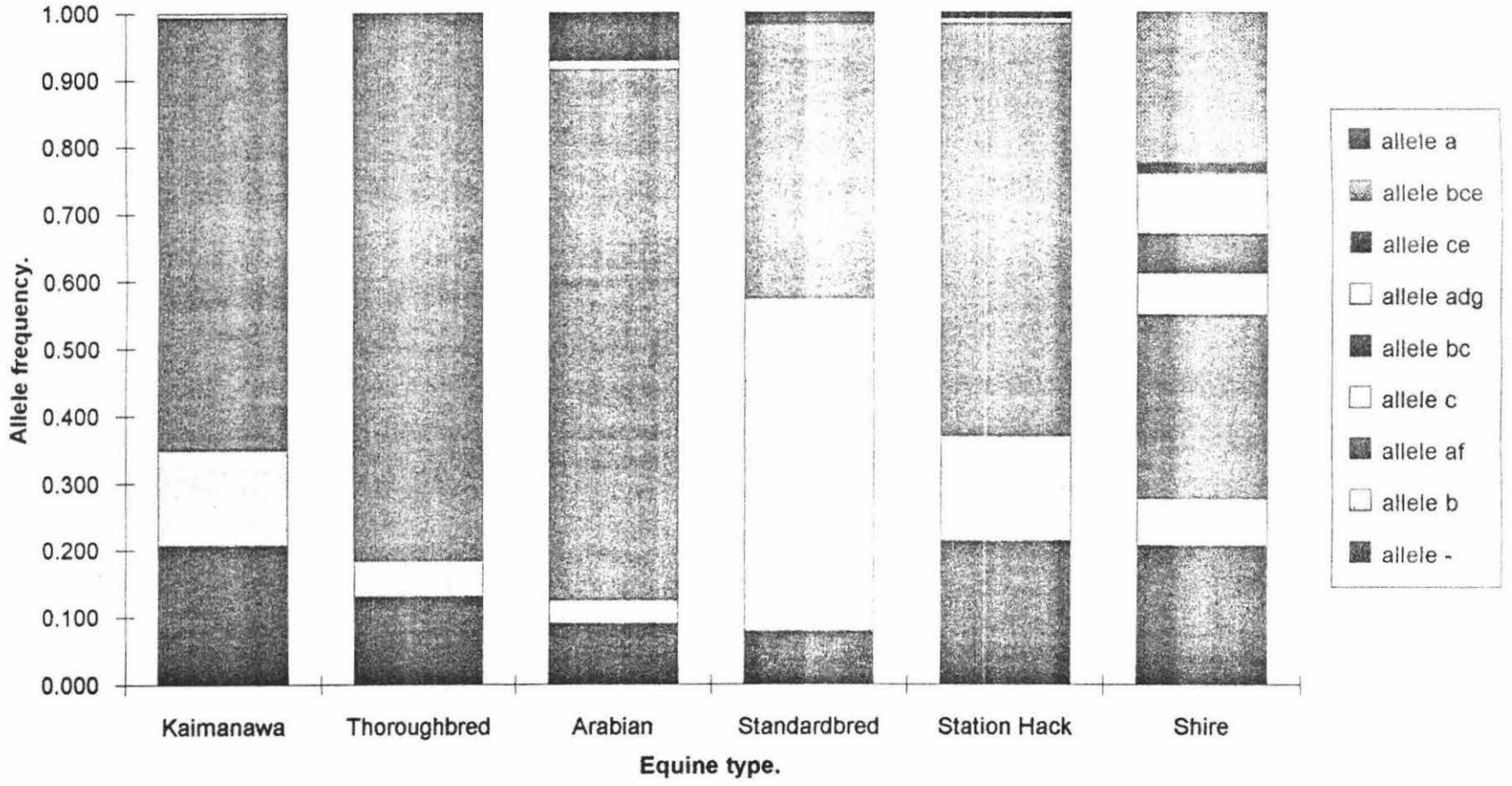


Figure 3.19: Alleles of blood group A present in the equine types studied.

group A is multi-allelic. Hence, the chi-square analysis returns a significant difference between the Kaimanawa horses and all other New Zealand equine types studied.

As can be seen in figure 3.20, within blood group C, the Kaimanawa horses and Station hacks are identical both in allele occurrance and frequency. This is supported by the chi-square analysis in that the Station Hacks are the only New Zealand equine type which is not significantly different, with respect to Blood Group C, to the Kaimanawa horses. Both of these equine types also exhibit a strong similarity (in terms of the alleles in blood group C) to the Shire horses.

The phenogroups of blood group "D" are demonstrated in figure 3.21. Generally, none of the samples seem to resemble each other in either phenogroup occurrence or frequency. However, the frequency of phenotype "cgm" is similar in Kaimanawa horses, Arabian horses and Shire horses. The Kaimanawa horses appear to be the only equine type of those studied with the phenotype "dek." Blood group D is another highly polymorphic system and resultantly, the chi-square analysis performed on it shows that all of the New Zealand non-Kaimanawa equine types differ from the Kaimanawa horses.

From simply looking at the bar chart, the frequencies of the two alleles ("a" and "-") in the equine blood group K, show minimal difference amongst all equine types studied. See figure 3.22. The Kaimanawa horses follow in the trend of there being a low frequency for 'a" and a high frequency for "-". However, the chi-square analysis suggests that the only New Zealand equine type whcih does not have significantly different blood group K allele frequencies to those of the Kaimanawa horses, is the Arabian horses.

Figure 3.23 shows that in Blood Group P the Kaimanawa horses not only strongly resemble the Thoroughbred horses but they are also very similar to both the Arabs and Hacks. Support for a similarity between the Kaimanawa and Thoroughbred horses is given by the

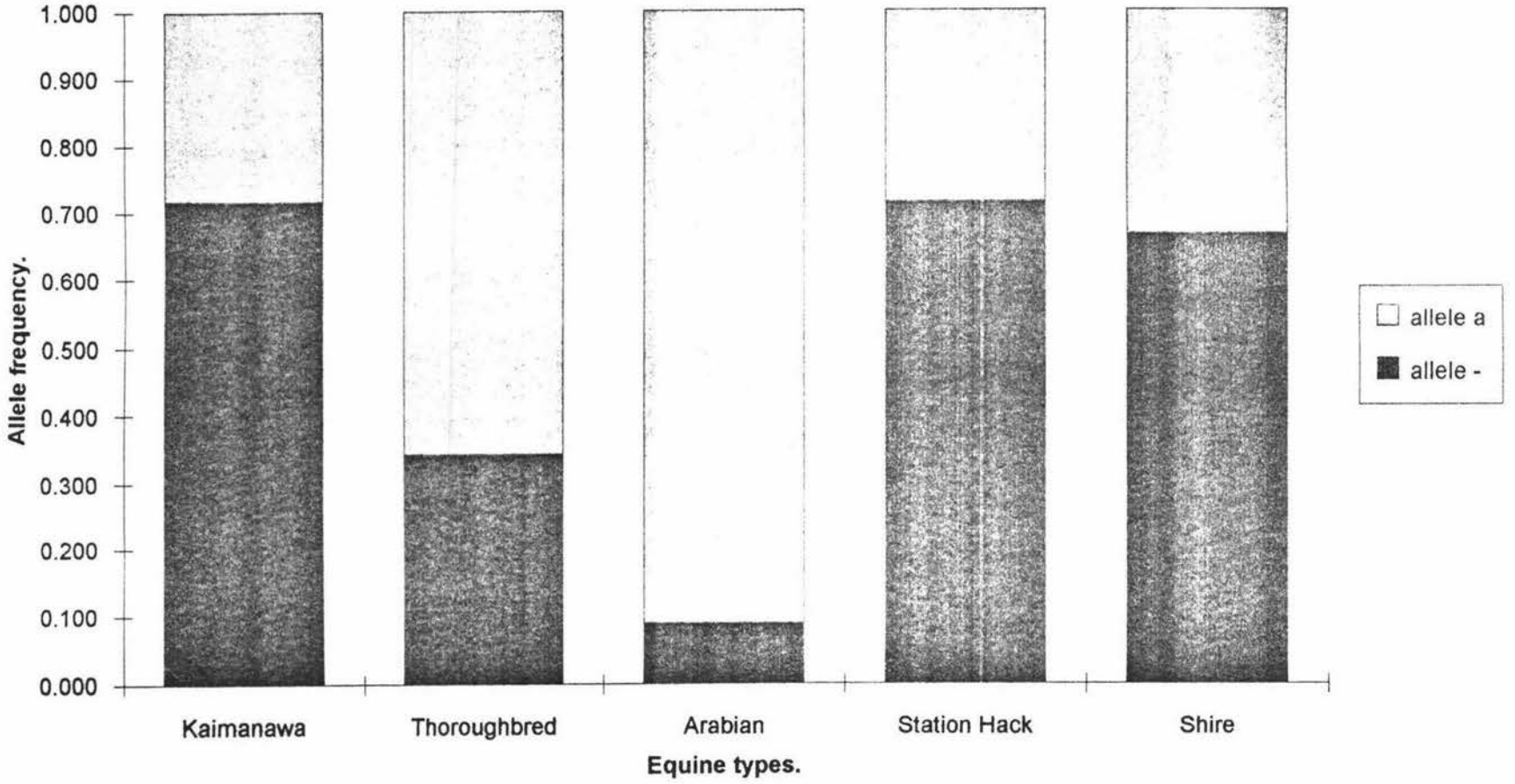


Figure 3.20: Alleles of blood group C present in the equine types studied.

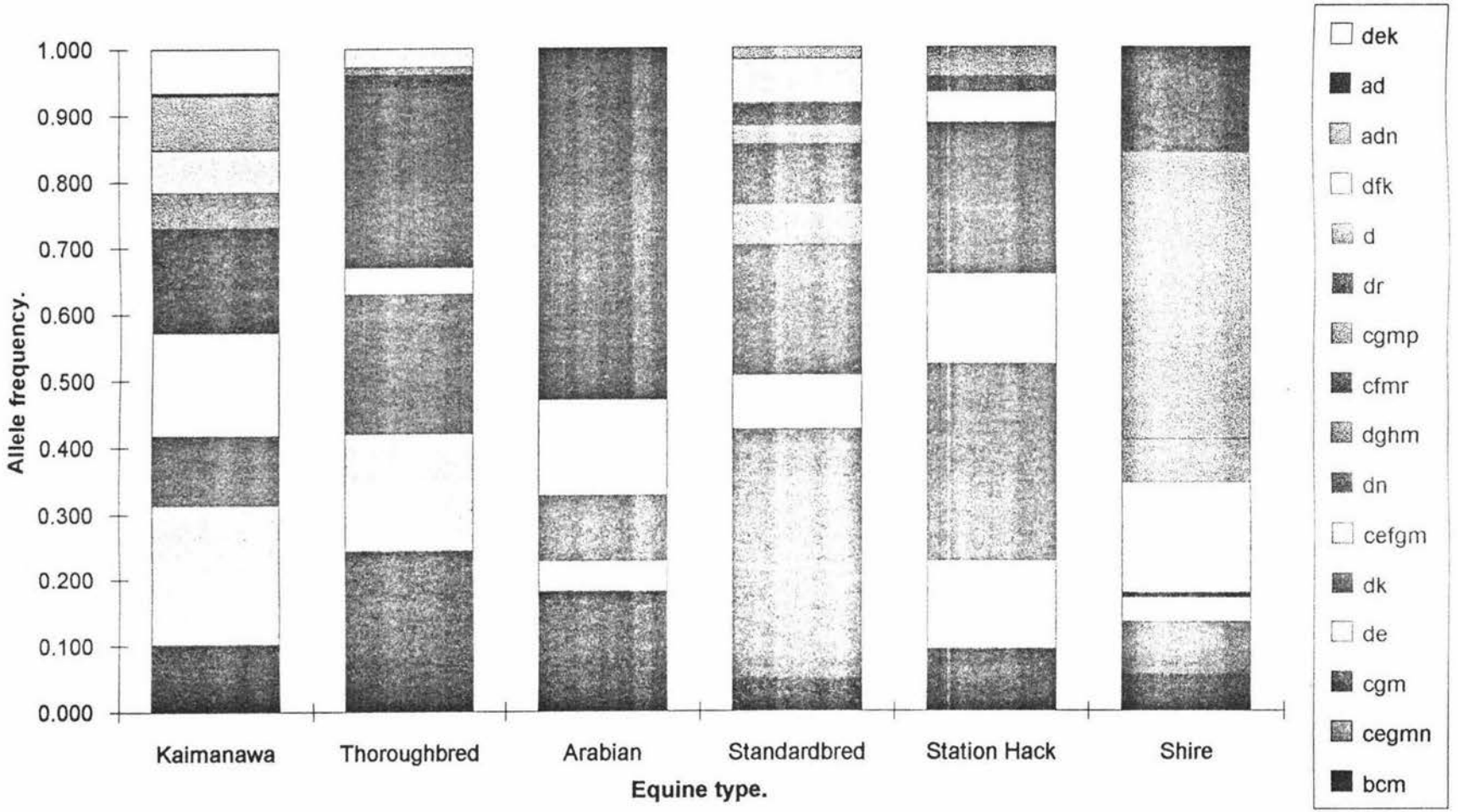


Figure 3.21: Phenogroups of blood group D present in the equine types studied.

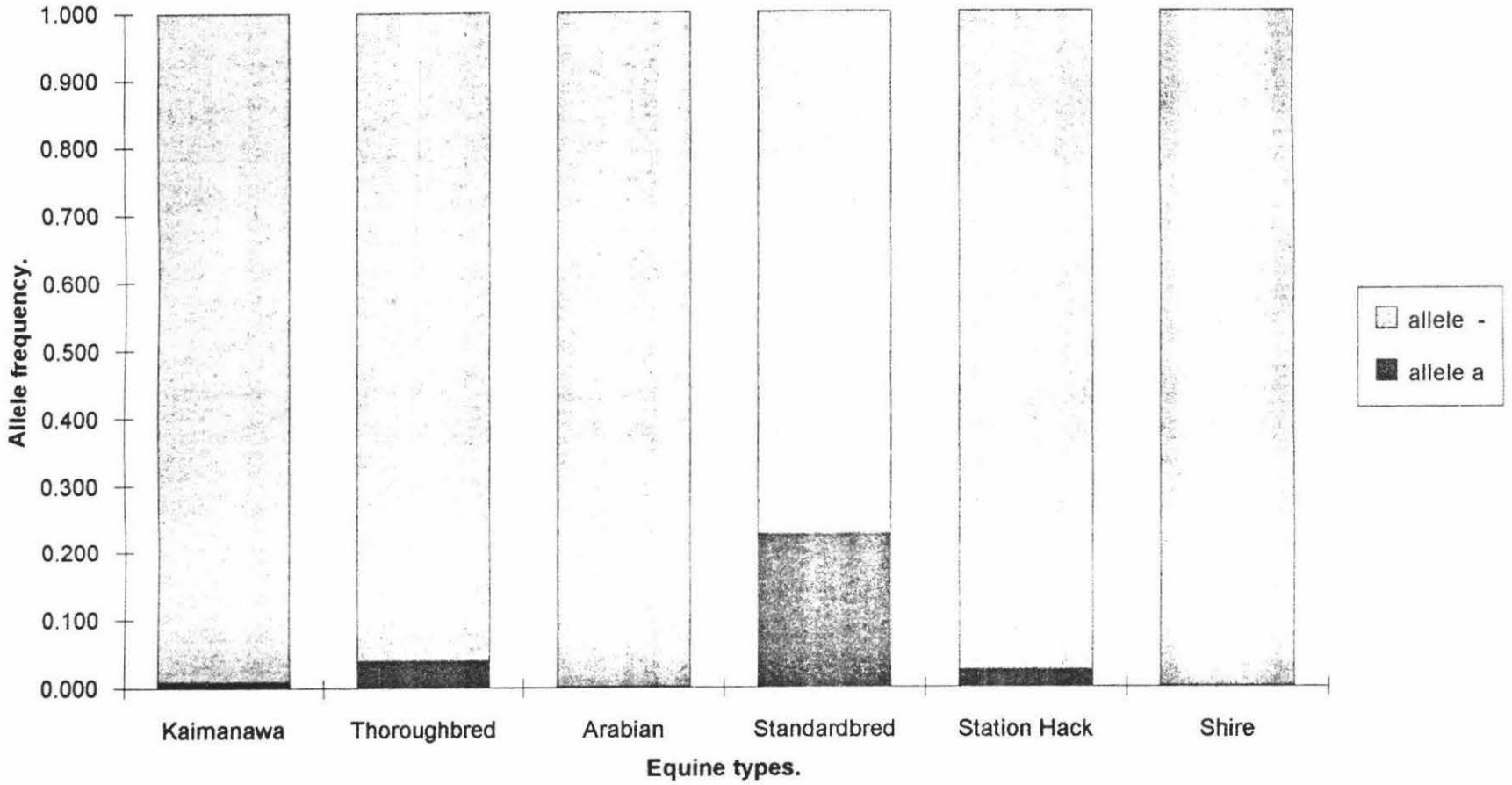


Figure 3.22: Alleles of blood group K present in the equine types studied.

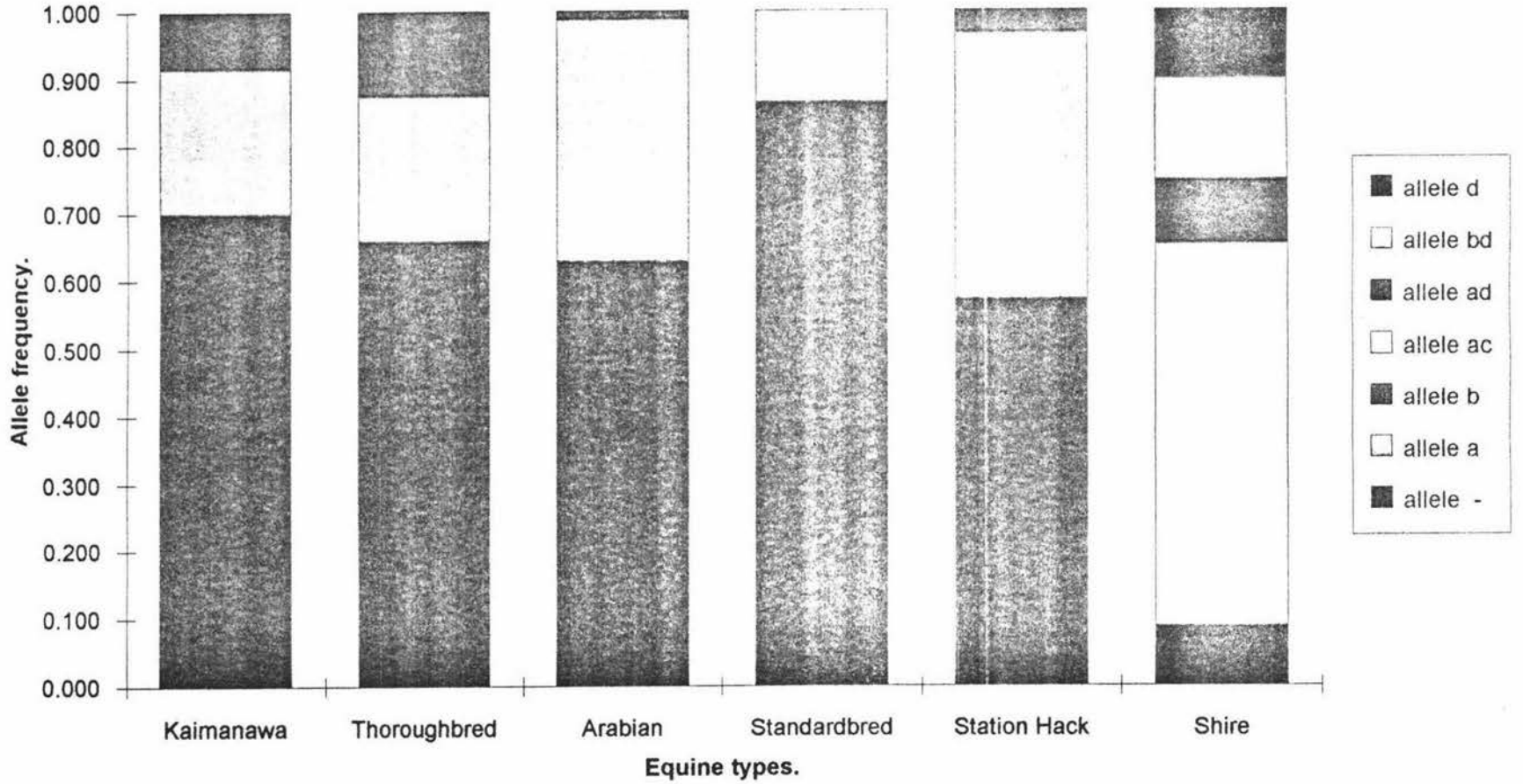


Figure 3.23: Alleles of blood group P present in the equine types studied.

chi-square analysis of this blood group, however there is a significant difference between the Kaimanawa horses and both the Arabian and Standardbred horses.

Within blood group Q (see figure 3.24), the same alleles (-, c and abc) are present in the Kaimanawa horses and the Thoroughbreds, but the frequencies of these alleles differ. Conversely, allele frequencies are closer between the Arabs and the Kaimanawa horses, despite the former also having allele 'b' which the Kaimanawa horses lack. The frequency of the '-' allele in the Kaimanawa horses is similar to that of the Station hacks. Blood group Q is another highly multi-allelic system which significantly differs between the Kaimanawa horses and all other New Zealand equine types studied.

As demonstrated in Figure 3.25 within blood group U, the allele frequencies for the Kaimanawa horses bear the strongest resemblance to those of the Thoroughbreds and the Arabs. Shires are the only markedly different equine type in this particular system in that they demonstrates a high frequency of allele 'a' and a low frequency of allele '-'. According to the chi-square analysis, the only New Zealand equine type which differs significantly to the Kaimanawa horses (within blood group U) is the Standardbred horses.

3.5 Distance Matrices.

Table 3.03, displays both Nei's Standard genetic distance values (top, right of the diagonal) and Rogers' genetic distance values (bottom, left of the diagonal), between all pairwise comparisons of the six equine types included in this study.

One particularly notable feature of both distance value sets, is the much larger genetic distance that the Shire horses have from any of the other equine types.

The equine type with the smallest genetic distance to the Kaimanawa horses, according to both Roger's and Nei's distance values, is the Station Hack. Also supported by both distance measures, is that the Thoroughbred horse is the next equine type with the closest genetic similarity to the Kaimanawa horses.

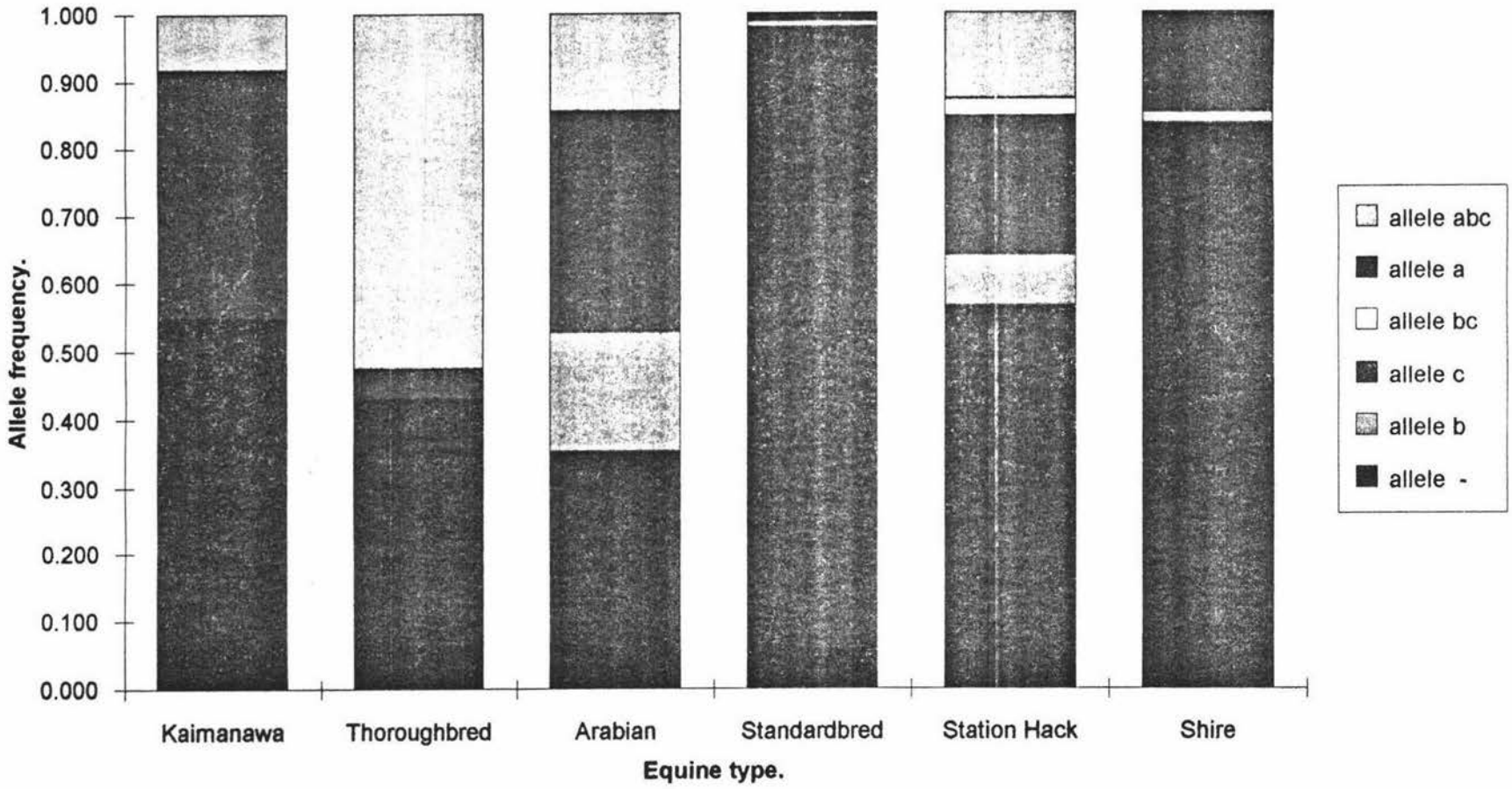


Figure 3.24: Alleles of blood group Q present in the equine types studied.

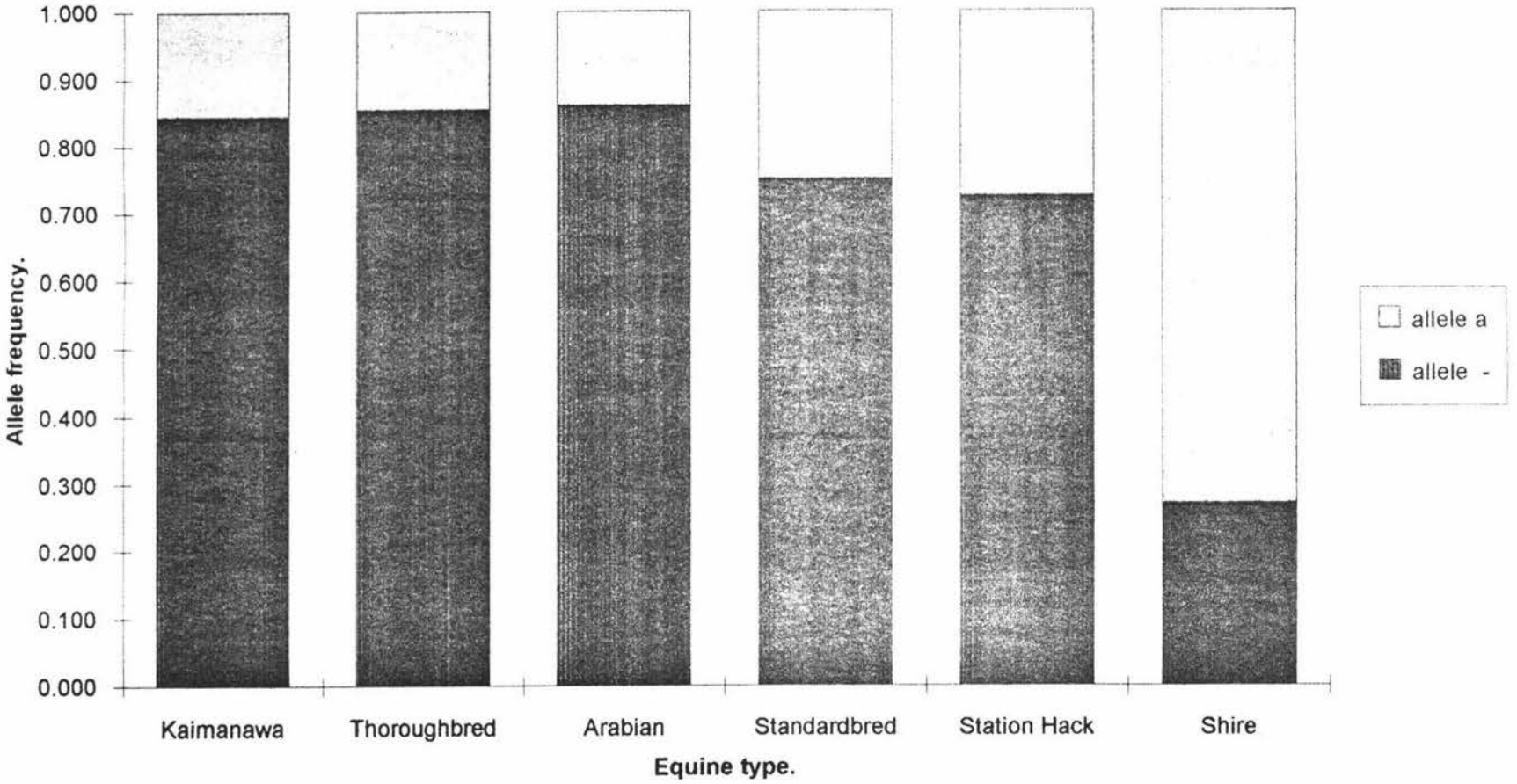


Figure 3.25: Alleles of blood group U present in the equine types studied.

According to Roger's genetic distance values, in relation to the Kaimanawa horses, the Arabian horses follow the Thoroughbred horses and that of the New Zealand horses included in this research, the Standardbred horses are the least proximal to the Kaimanawa horses. However, Nei's distance measure indicates that both the Arabian horses and the Standardbred horses are equally distant from the Kaimanawa horses.

	Kaimanawa	Thoroughbred	Arabian	Standardbred	Station Hack	Shire
Kaimanawa	-	0.233	0.259	0.278	0.211	0.337
Thoroughbred	0.082	-	0.228	0.359	0.239	0.424
Arabian	0.113	0.077	-	0.327	0.198	0.411
Standardbred	0.126	0.169	0.165	-	0.241	0.352
Station Hack	0.070	0.068	0.053	0.111	-	0.340
Shire	0.413	0.457	0.467	0.343	0.414	-

Table 3.03: Genetic distance values. Those above the diagonal are derived from Roger's genetic distance calculation. Those values below the diagonal are derived from Nei's standard genetic distance calculation.

3.6 Neighbour Joining Values.

The lengths given by the Neighbour Joining Programme are presented in table 3.04. These were converted to linear centimetre lengths and utilised to produce the phylogenetic trees mentioned in section 3.7.

3.7 Phylogenetic Trees.

One of the most prominent features of both the phylogenetic trees produced (figures 3.26 and 3.27), is that the Station hacks and the Kaimanawa horses both have very small divergences from the main internal edge link. However, the Thoroughbred horses, Arabian horses, Standardbred horses and Shire horses all sit at the end of comparatively long branches descending from the main internal edge link. In both of the trees the Shire horses have an exceptionally long branch extending from a common node.

Branch	Neighbour Joining Length from Roger's Values
External branches:	
Standardbred horses - Node 4	0.14075
Shire horses - Node 4	0.21125
Kaimanawa horses - Node 2	0.10108
Station Hacks - Node 3	0.07975
Thoroughbred horses - Node 1	0.12037
Arabian horses - Node 1	0.11363
Internal branches:	
Node 1 - Node 2	0.03792
Node 2 - Node 3	0.02250
Node 3 - Node 4	0.01775

Branch	Neighbour Joining Length from Nei's Values
External branches:	
Standardbred horses - Node 4	0.07487
Shire horses - Node 4	0.13113
Kaimanawa horses - Node 3	0.03237
Station Hacks - Node 2	0.00683
Thoroughbred horses - Node 1	0.03550
Arabian horses - Node 1	0.04250
Internal branches:	
Node 1 - Node 2	0.02267
Node 2 - Node 3	0.01963
Node 3 - Node 4	0.01363

Table 3.04: Neighbour Joining Values.

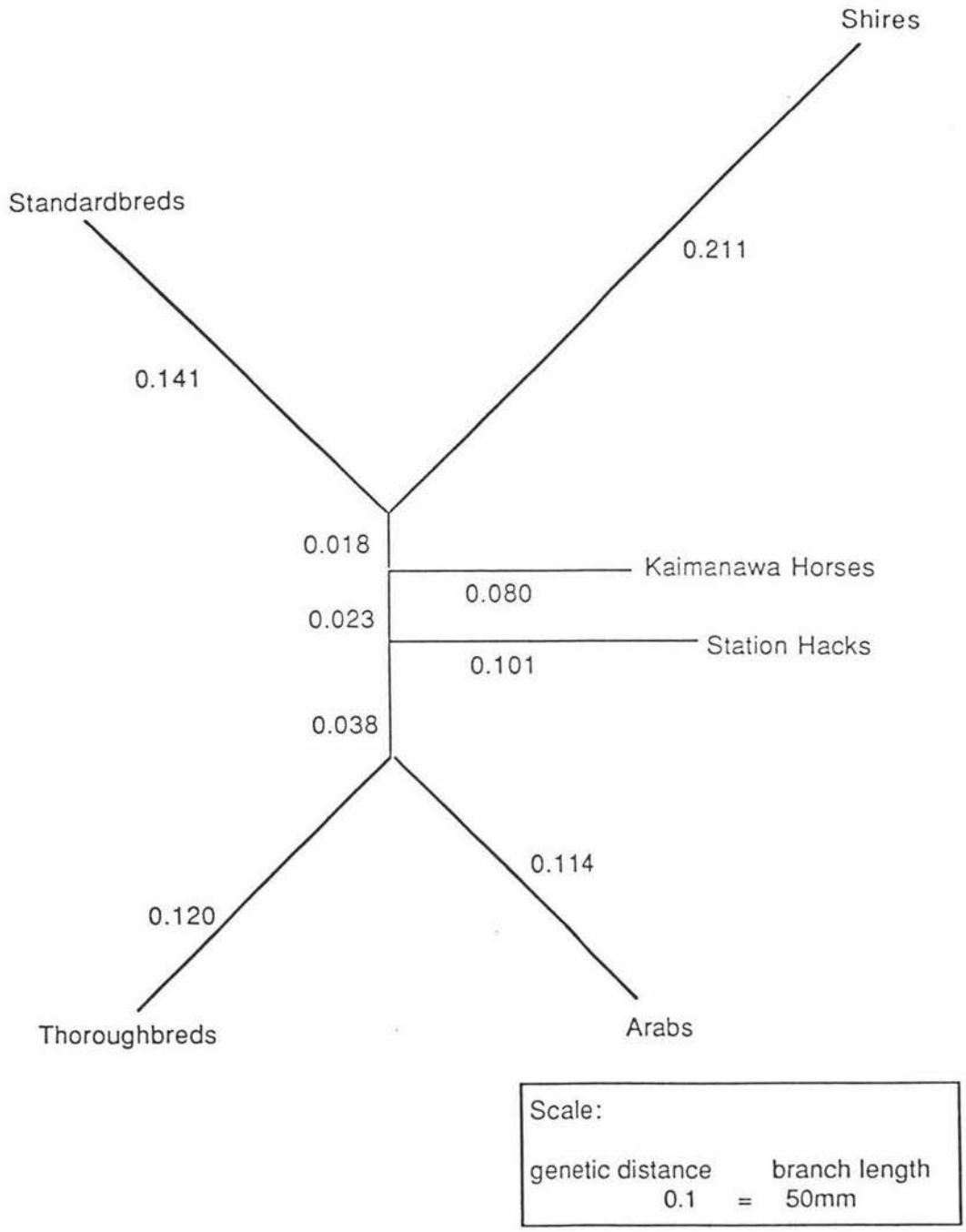


Figure 3.26: Phylogenetic tree based on Roger's genetic distances.

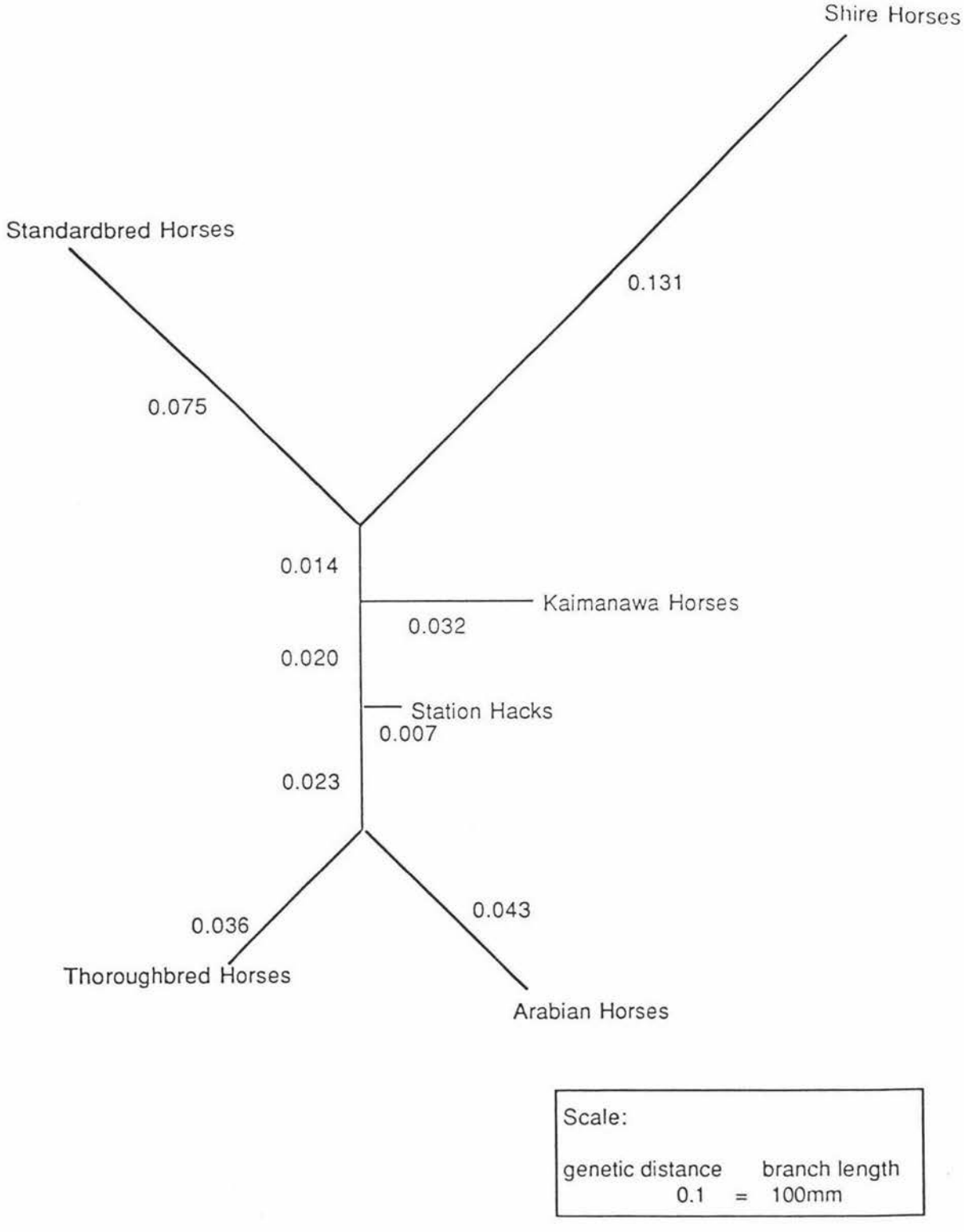


Figure 3.27: Phylogenetic tree based on Nei's standard genetic distance values.

3.8 Split Decomposition Networks.

The most distinct features of the network based on Nei's standard genetic distance values (figure 3.28), are the the short internal edge links or branches, and the occurrence of boxes. Another particularly distinct feature of this network is that the Arabs and the Thoroughbreds are grouped together. Similarily, the Kaimanawa horses and Thoroughbred horses have also been separated from the other equine types. It is also quite important to note that the Station Hacks and the Kaimanawa horses are both centrally positioned within the network rather than branching off in discrete lines.

The Split decomposition network based upon Roger's genetic distance values (figure 3.29) also groups the Kaimanawa horses with Thoroughbred horses and Arabian horses. However, it does not group Station Hacks and Kaimanawa horses together.

Overall there is a lack of clear resolution amongst both the splits networks. However, the network based upon Nei's standard genetic distance data has a fit value of 88.2% and the network based upon Rogers' genetic distance data has a fit value of 97.3%.

3.9 Venn Diagrams.

From figure 3.30 it can be seen that with respect to allele composition, the Kaimanawa Horses almost lie as a subset of a mix between the Thoroughbred horses and Station Hacks. The alleles at the top of the diagram which are not in either the Thoroughbred horses or the Station Hacks, can with one exception be found in Arabs and Standardbreds - see figure 3.31.

On the Venn diagram encompassing all equine types included in this study (figure 3.31), it appears that the Kaimanawa horses share all but one of the alleles studied (the phenogroup D^{dek}) with at least one other equine type. Note that a few alleles appear twice on figure 3.31, e.g., D^{d}. This was a practical limitation and it was not feasible on this venn diagram to

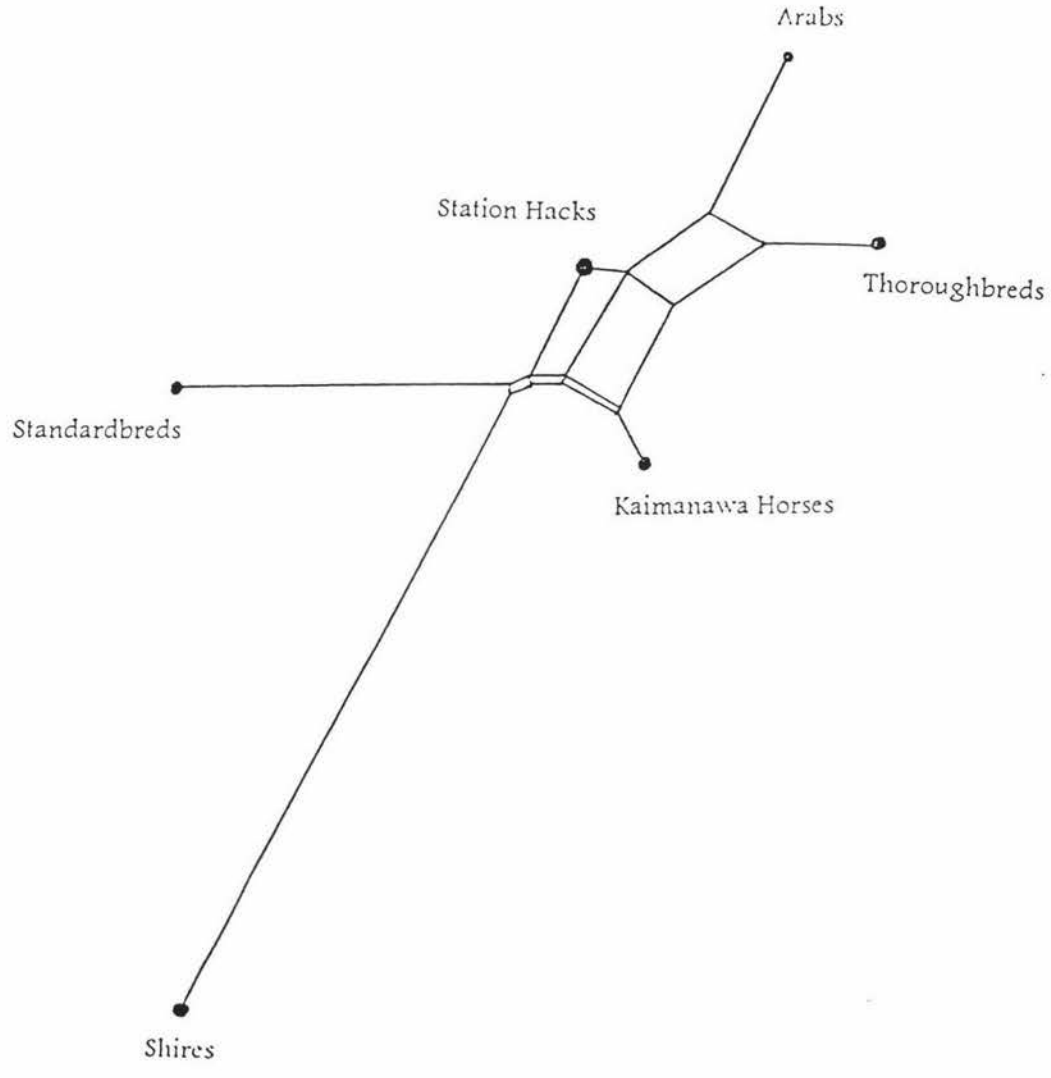


Figure 3.28: Split Decomposition Network Based on Nei's Standard Genetic Distance Values.

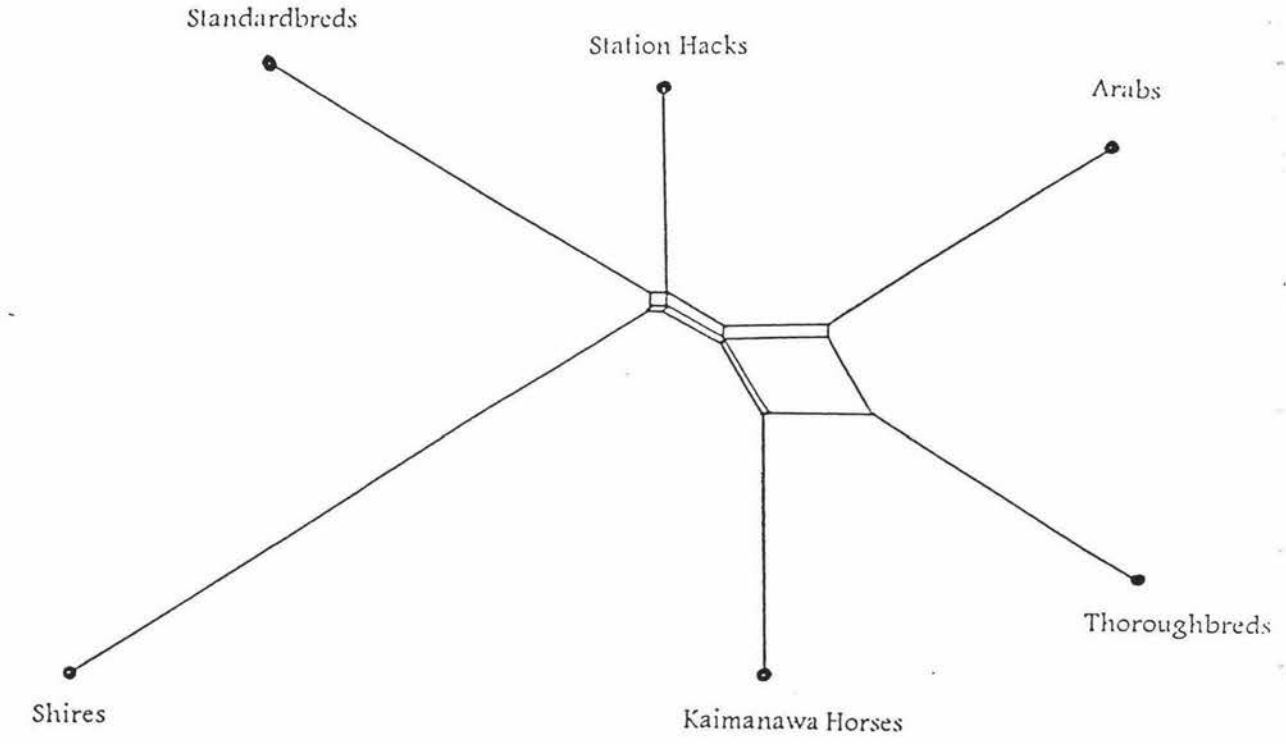


Figure3.29: Split Decomposition Network Based on Roger's Genetic Distance Values.

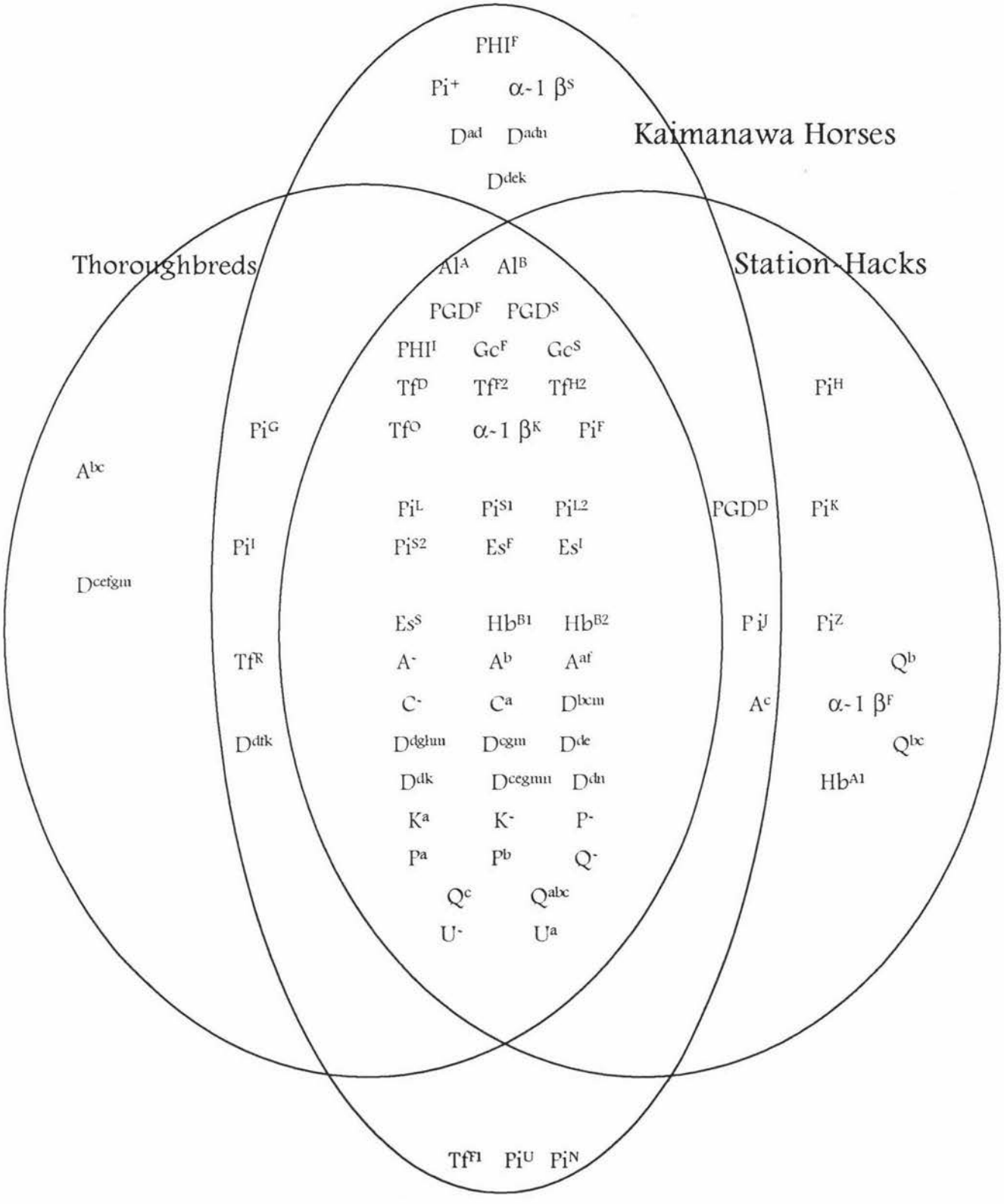


Figure 3.30: A Venn Diagram of the Alleles Present in the Thoroughbred Horses, Kaimanawa Horses and Station Hacks Included in this Investigation.

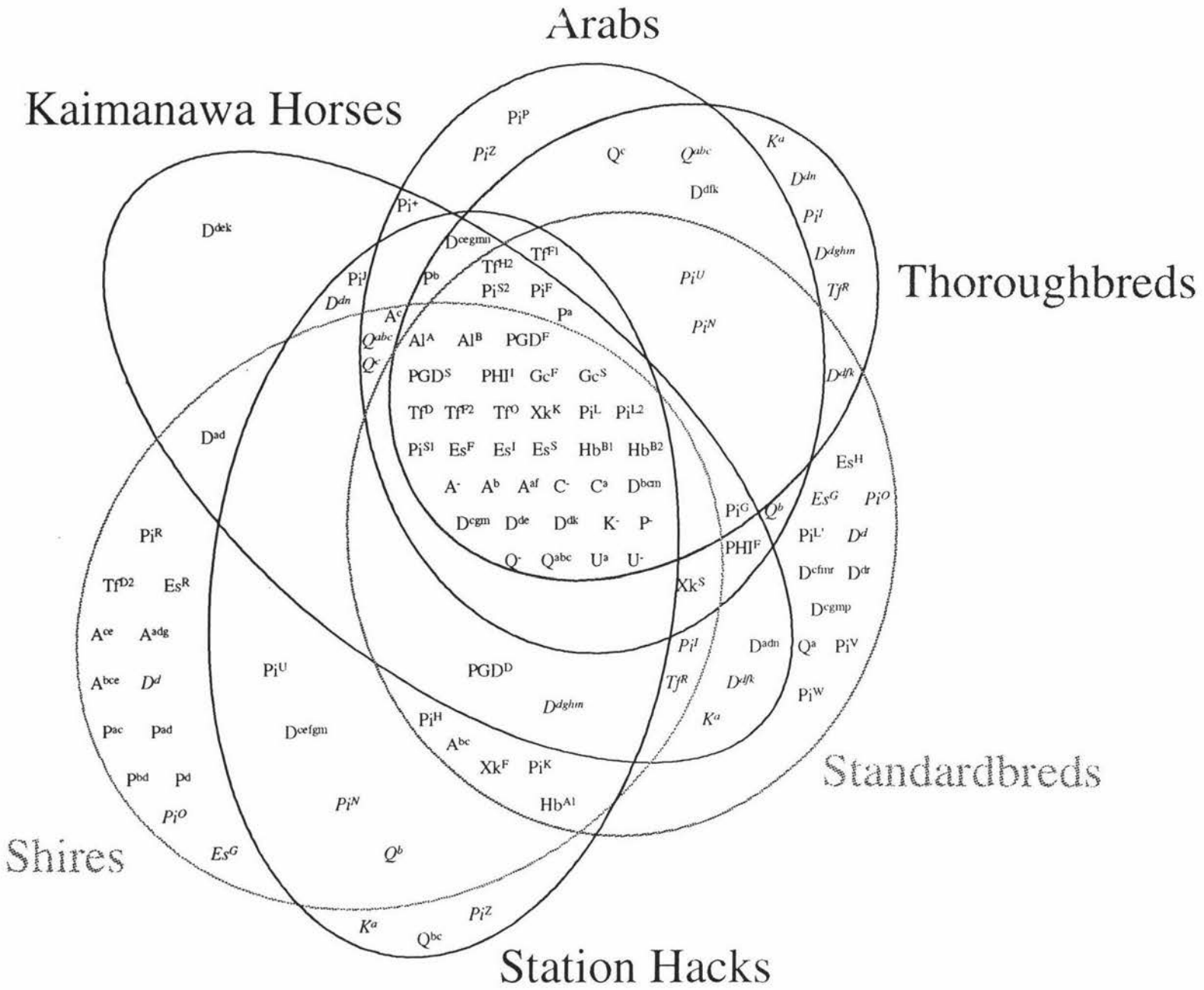


Figure 3.31: A Venn Diagram of the Alleles Present in All of the Equine Types Studied.

obtain intersecting regions between some combinations of sample populations. Any allele which this affected, appears in italics.

3.10 Mixed Populations.

In order to determine which mixture/crossbred of the non-Kaimanawa equine types studied had the closest resemblance to the Kaimanawa horses, their frequency for each allele was calculated and run through the extended Roger's genetic distance calculation. The results of this are presented in table 3.05 and show that a cross bred population of Thoroughbred, Arabian, Standardbred and Station Hack horses have the closest relationship to the Kaimanawa horses.

Crossbred population	Genetic distance to the Kaimanawa Horses
T-A	0.236
T-S	0.195
T-H	0.207
T-Sh	0.240
A-S	0.196
A-H	0.216
A-Sh	0.253
S-H	0.201
S-Sh	0.276
H-Sh	0.249
T-A-S	0.175
T-A-H	0.210
T-A-Sh	0.227
T-S-H	0.173
T-S-Sh	0.215
T-H-Sh	0.219
A-S-H	0.273
A-S-Sh	0.215
S-H-Sh	0.224
A-H-Sh	0.222
T-A-S-H	0.165
T-A-S-Sh	0.193
T-S-H-Sh	0.196
T-A-H-Sh	0.211
A-S-H-Sh	0.193
T-A-S-H-Sh	0.181

Table 3.05: Roger's genetic distance values between the Kaimanawa Horses and crossbred populations of the other equine types studied. Note that T=Thoroughbreds, A=Arabians, S=Standardbreds, H=Station Hacks and Sh=Shires.

4.0 Discussion.

4.1 Common Alleles shared between the Kaimanawa horses and other equine types.

As can be seen in figure 3.30, most of the alleles (in the sixteen protein systems studied) present in the Kaimanawa horses also exist in either Thoroughbred horses or Station hacks. The majority of these alleles are also shared by Station Hacks and Thoroughbreds. Most of the alleles not in either of these two latter breeds can be found in the other equine types studied. For example, PHI^F which is found in both Standardbred and Arabian horses. There was only one allele (of the 94 studied) which was only found present in Kaimanawa horses. This was the D^{dek} allele from the D blood group. However, this phenogroup is not unique to the Kaimanawa horses. According to Bowling & Clark (1985), this phenogroup is present in Quarter horses and Morgan horses. There is also published evidence for this phenogroup being present occasionally (with a low frequency) in Arabian horses and Arabian type crossed breeds (Ouragh *et al*, 1994). The D^{dek} allele is also occasionally seen in the routine blood typing of non-thoroughbred horses such as Shetland ponies and Miniature horses, and it is occurs often in Clydesdale horses (Dr. I. L. Anderson; pers comm.) This latter breed and the Arabian horses are probably the most likely source of this allele into the Kaimanawa horses.

Another possible explanation for the apparent uniqueness of the D^{dek} allele is that it's expression is sometimes masked in particular blood types. For example, an Arabian horse with the genotype $D^{cegmn/dk}$ may actually be $D^{cegmn/dek}$. This is because the signal for a positive reaction to D^e would not be any stronger if it was present in both phenogroups, rather than just one. Secondly, if a horse registers a positive reaction to D^c, D^g, D^m and D^n, then its blood must also contain D^e, to form the D^{cegmn} phenogroup. (See table 2.01 of

internationally recognised phenogroups). Hence, with both of these points in mind, whilst such a horse may actually be $D^{cegmn\,dck}$, it can only be guaranteed as being $D^{cegmn\,dk}$. Therefore, some of the horses included in the Arabian sample may actually be D^{dek} yet part of that phenogroup is simply masked by the constituents of their other phenogroup for the D blood group.

Alternatively, the apparent uniqueness of the D^{dek} allele to the Kaimanawa horses could simply be an artefact of sampling. There may be some unmasked D^{dek} alleles present in Thoroughbred, Arabian, Standardbred and Station Hack horses whose bloodtypes were not included in this study.

Allele J of the Protease inhibitor system was only found in the Kaimanawa horses and one of the Station Hacks sampled. However, although it is very rarely seen in the New Zealand equine types, it often occurs in American saddlebreeds (Dr. I. L. Anderson; pers comm.) It is important to remember that only a small number of Station Hacks were sampled (n=22) and hence, the J allele may in fact be present in many more of this equine type.

4.2 Quantitatively assessing similarities and differences between Kaimanawa and Non-kaimanawa horses.

4.2.1 Allele frequencies and Chi-square analyses.

Similarities or differences between the Kaimanawa horses and each of the other equine types (with respect to allele presence and frequency) were initially assessed qualitatively by chi-squared analysis of the allele frequencies. Table 3.02 displays the probability values for there being a significant difference in the allele frequencies between the Kaimanawa horses and each of the other New Zealand equine types tested. A probability value which was less than or equal to 0.05 was taken as an indication of a significant difference between the two populations.

The protein systems in which the Kaimanawa and Thoroughbred horses are not significantly different are Vitamin D binding protein, α 1-β glycoprotein, Haemoglobin, Esterase, Blood group P and Blood group U.

Albumin, Blood group K and blood group U are the only protein systems in which the Kaimanawa and Arabian horses are not significantly different.

The Kaimanawa and Standardbred horses share very little in common in terms of the protein systems studied. The only system in which the two equine types are not significantly different, is Albumin.

As with the Thoroughbreds, the Kaimanawa horses also share similar gene frequencies in six protein systems with the Station Hacks. These are Albumin, PHI, Vitamin D binding protein, Blood Group C, Blood Group P and Blood Group U.

It was not possible to perform a chi-square analysis on the Shire horses because the only data available about them was allele frequency data whereas the chi-square analysis required numbers of individuals. However, the protein graphs (figures 3.10 - 3.25) show the Kaimanawa and Shire horses as having similar genetic constitutions in the Vitamin D binding protein system, Blood Group C and Blood Group K.

Hence, it can be seen that amongst each of the four comparisons (Kaimanawa - Thoroughbred, Kaimanawa - Arabian, Kaimanawa - Standardbred and Kaimanawa - Station Hack) there are a lot of protein systems which are significantly different. However, they are not all similar for the same protein systems. Instead, as shown in table 4.01, each of the non-Kaimanawa equine types combined basically complement each other with respect to the Kaimanawa Horses. Thus, whereby only the Thoroughbred horses have allele frequencies within the Esterase protein system which are comparable with those in the Kaimanawa horses, only the Station Hacks have allele frequencies within the blood group C which are similar to those in the Kaimanawa horses.

Protein System	Equine populations which are **not** significantly different to the Kaimanawa horses.			
	Thoroughbred	Arabian	Standardbred	Station Hack
Albumin		*	*	*
PGD				
PHI				*
Gc	*			*
Transferrin				
α 1-β	*			
Haemoglobin	*			
C				*
K		*		
U	*	*		*
Q				
P	*			*
Esterases	*			
Protease Inhibitors				
D				
A				

Table 4.01: Populations with gene frequencies which are similar to that of the Kaimanawa horses.

The systems not complemented by any other population are highly multi-allelic and each population contains a different combination of alleles within such systems, e.g., Pi and Tf. Multi-allelic systems result in large variations in gene frequencies between populations. It would hence be very difficult to find two equine types with similar multi-allelic protein systems (in terms of both allele presence and frequency). PGD is only tri-allelic yet the Kaimanawa horses appear significantly different to the other four New Zealand equine types with respect to this protein system. However, this system is present with similar allele frequencies in the Shire horses.

Overall, the chi-square analysis demonstrates that the Thoroughbred horses and Station Hacks bear the closest similarity, in terms of allele frequency, to the Kaimanawa horses.

4.3 Genetic distance between Kaimanawa horses and other equine types.

4.3.1 Relationships inferred from genetic distance values.

According to both Rogers' and Neis' genetic distance values, the Kaimanawa horses seem to have a close genetic relationship with the Station Hacks and the Thoroughbred horses. Circumstantial and historical evidence would suggest that this is to be expected.

Firstly, the Station Hacks sampled were from stations located on the periphery of the Kaimanawa Ranges. Many of the station owners reported seeing the occasional "strange" horse in the back paddock or alternately some of their horses would stray into the ranges for a few days. There have also been reports of unwanted station hacks being released into the Kaimanawa region. Hence, the two populations are not reproductively isolated and so an exchange of genetic material would be expected between the two.

Secondly, one of the known, major influxes of horses into the Kaimanawa herd was the Cavalry horses from Waiouru in 1941. All of the Cavalry horses were either Thoroughbreds

or of Thoroughbred descent. This would provide an explanation for the strong genetic link between the Thoroughbred horses and the Kaimanawa horses.

Conversely, of the equine types studied, the Shire horses appear to be the most distant from the Kaimanawa horses, i.e., when considering the relationship between the Kaimanawa horses and each of the other four equine types studied, the equine type with the largest genetic distance from the Kaimanawa horses is the Shire horses. Consequently, the external branch leading to the Shire horses is by far the longest on both phylogenetic trees.

This can be explained by the fact that the Shire horses are one of the most distinct breeds historically, morphologically and genetically from any other equine type. Each of the other sample populations used in this analysis were either pure-bred hotbloods (Thoroughbreds and Arabs) or mixtures thereof, i.e., warmbloods (Standardbreds, Stationhacks, and, according to historical evidence, the Kaimanawa horses). Whereas, the Shire horses are an equine type known as "cold bloods" in that they do not have any Thoroughbred blood in them. Infact, whilst the hot and warm-blooded horses all descended from the Asiatic wild horse, the cold-blooded horses descended from another primitive horse, *Equus silvaticus*, from which it is thought all modern heavy horse breeds derive.
In other words, the relationships depicted in both the genetic distance values and the phylogenetic trees support historical evidence for such associations.

4.3.2 The optimal relationships present within the data.

One of the most prominent features of both of the phylogenetic trees presented, is that the Station Hacks and the Kaimanawa horses both have very small divergences from the main internal edge link, whereas the Standardbreds, Thoroughbreds, Arabs and Shire horses all sit at the end of comparatively long branches descending from the main internal edge link. This most probably reflects the fact that the latter four equine breeding types are distinct breeds, each of which has been purposefully bred for a particular function, and the latter three now have closed breeding registers.

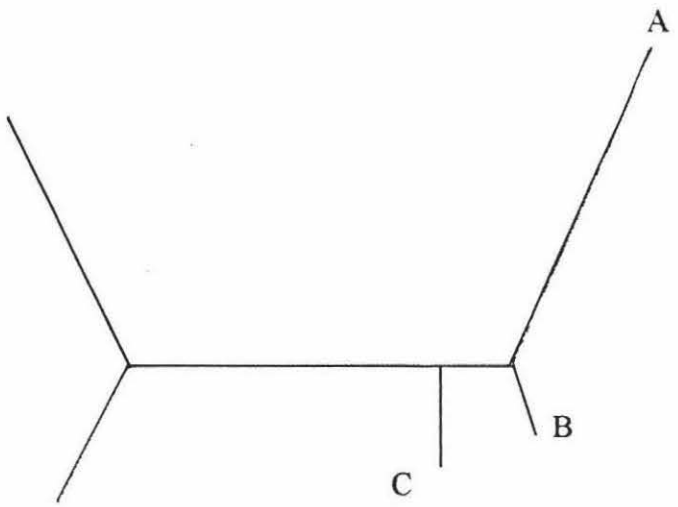
As mentioned earlier in section 4.3.1, the Shire horses have an exceptionally long branch from a common node most likely because of their longstanding separation from the other equine types. Conversely, neither the Stationhacks nor the Kaimanawa horses have undergone any intentional selective breeding since they shared a common ancestor. The Kaimanawa horses and the Station Hacks have remained as a mixture with no specific features attributable to them, i.e., they are simply regarded as an equine "type" or "group."

The internal branch lengths of both trees are particularly small. Such a feature is generally indicative of poor resolution within the data which in turn suggests that there is very little difference between each of the sample populations other than the Shire horses.

In order to judge the magnitude of the distances obtained in this research, another genetic distance investigation involving production animals has been considered. A study performed by Zanotti Casati *et al* (1990) found that the genetic distances among five Italian native sheep breeds, ranged from 0.012 to 0.060 when calculated via Nei's standard genetic distance measure. Whilst these values are less than those obtained for the Kaimanawa horses and other equine types, it should be recognised that the sheep breeds in the afore mentioned study are local breeds, all with a common ancestor.

Unlike the distance values alone, which indicate a close relationship between the Kaimanawa horses and the Station Hacks, the phylogenetic trees do not strongly support this relationship. If the Kaimanawa horses were most closely related to Station Hacks they should both diverge from a common node in the tree. This apparent contradiction in results is because two populations may not be the closest of relatives, yet still have the shortest distance between them, (See figure 4.01).

Trees/dendograms are not an ideal way of illustrating relationships between the populations studied here. This study deals with interbred populations not bifurcating, mutually exclusive, species. Hence, clearly resolved branches are unlikely to be obtained as the lineages of the different breeds are recombining. However with species, they cannot interbreed and so the branches are distinct from each other.



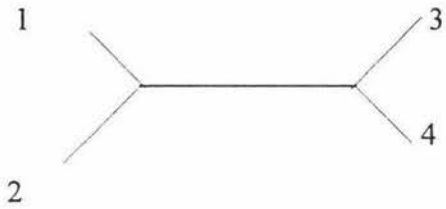
The smallest genetic distance between two populations is between B and C. However, A and B are closer relatives as they share a common node on the phylogenetic tree. Populations B and C simply have not changed or evolved greatly whereas population A has and hence is located at the end of a much longer external branch.

Figure 4.01 : A hypothetical phylogenetic tree illustrating short genetic distances between non-relatives.

4.3.2.1 Suitability of genetic distance measures for particular data.

Phylogenetic trees also provide a good means of deciding whether or not a particular genetic distance measure is to be deemed suitable for the data they summarise. If the distance measure is suitable, then the resultant distances on the tree must exhibit "additivity." See figure 4.02.

If the distances are additive:



$(d_{12} + d_{34}) < (d_{13} + d_{24})$

$(d_{12} + d_{34}) < (d_{14} + d_{23})$

Note; d_{12} represents the genetic distance between population one and population two.

Figure 4.02: Understanding "additivity."

Each of the phylogenetic trees constructed from the genetic distance data were checked for additivity. Results of these checks can be found in Appendix II. This investigation showed that additivity is definitely exhibited in each tree and hence, the distance measures chosen for this study were suited to the data.

4.3.3 Support and Contradictions for Relationships Within the Data.

Whilst phylogenetic trees display the optimal estimate of relationships between the equine types studied, split decomposition networks highlight any conflicts in the data and show the level of support or contradiction present within the data for other, not so optimal, relationships.

Each split decomposition network has a percentage value of fit associated with it. This percentage value is a measure of how well the network represents the data from which it was derived. Values over 70% are regarded as indicators of a good fit. Hence, as the splits graphs based on Rogers' and Neis' genetic distance matrices have fit values of 97.3% and 88.2% respectively, the networks should be considered as being accurate portrayals of the data from which they were constructed.

The most notable observations to be made from both the networks, are the short internal links and the occurrence of "boxes". Both of these features indicate that, once again, there is poor resolution (a lot of contradictions) within the data, and the many different possible pathways could be used to represent the relationships between the New Zealand equine types. The lack of clear resolution amongst both of the networks may also be a result of small samples, both of protein systems and populations. Greater resolution may be obtained using allele frequencies from more protein systems or larger population samples. DNA-based methods could prove usedful here as they provide many more characters for analysis.

Boxes in the split decomposition network show contradictory support for various relationships. This is most likely to be due to the breeds actually being mixtures of each other. Thus, there is no clear cut definition between breeds that would occur between species, where boxes are less likely to occur.

An obvious feature of both split decomposition networks, is that the Thoroughbred horses, Arabian horses and the Kaimanawa horses are grouped together. This is to be expected between the Arabian horses and the Thoroughbred horses as Thoroughbreds were bred from the former.

Similarly, a relationship between the Kaimanawa horses and Thoroughbred horses is also historically supported.

The network based on Neis' standard genetic distances highlights a conflict in the data which groups the Station Hacks with the Thoroughbred, Arabian, and Kaimanawa group. This also is not unexpected considering that many of the Station Hacks sampled, had Arabian dams (pers comm; Station Hack owners).

In particular, the network derived from Neis' standard genetic distances illustrates quite clearly, how the Kaimanawa horses, like the Station Hacks, are not a distinct, separate breed. Both of these equine types occupy nodes positioned in the middle of the other distinct breeds, which is indicative of how they are both crossbreds of all other breeds. If this were not the situation, a distinct line generating from the central clump would be expected, rather than the short links and rather centralised nodes. Rather, they are both poorly defined crossbred types.

4.3.4 Recreating a Kaimanawa-like horse herd.

In the event of reconstituting a Kaimanawa-like herd, the above chi-square analysis could prove helpful in determining which equine types have similar allele frequencies to the those of the existing herd. Similarily, the analysis described in section 3.10 demonstrated that of all the combinations, a population of Thoroughbred-Arabian-Standardbred-Station Hack crossbred horses have the closest genetic distance to the existing Kaimanawa horses.

4.4 Limitations of this research.

One of the most prominent features of this study has been the lack of clarity in the various assessments of relationship, i.e., short internal edges on the phylogenetic trees and the lack of resolution in the split decomposition networks. The most probable cause of this situation is that there are few genomic differences amongst all of the equine types studied.

Whilst the entire horse species has only been in New Zealand for just over two hundred years, a Kaimanawa herd has really only been in existance for the last one hundred years. With this in mind and considering that the average horse generation ranges between six to nine years (Department of Conservation, 1995), this means that there have been only approximately ten generations of Kaimanawa horses. In evolutionary terms, twenty generations does not allow for a great deal of evolutionary change. Hence, markedly different amounts of variation should not be expected between the Kaimanawa horses and the breeds from which they developed. Furthermore, the Kaimanawa horses have not been a reproductively isolated herd. Throughout the c.100 years since the first reported sighting of a "Kaimanawa horse" there have been many influxes of different breeds into the herd.

The poor resolution within the data and consequent relationships, may also be due to small sample sizes. This refers to both the number of comparable markers studied, and the number of horses tested. In order to rectify this, further research could include either DNA marker analyses (allowing more patterns to be observed), or a study of a larger number of protein systems; and finally, larger populations of horses.

5. Conclusion.

The most prevalent conclusion to be drawn from this research is that the Kaimanawa Horses are by no means genetically unique. When compared with other equine types in New Zealand they show little difference in terms of genetic composition as assessed by allele presence and allele frequency. All of the investigations included in this study have shown that the two equine types which bear the closest resemblance to the Kaimanawa horses are the Thoroughbred horses and Station Hacks.

However, although the Kaimanawa horses demonstrate no important genetic significance, their place in the history of New Zealand must still be acknowledged, i.e., they have greater aesthetic value than genetic worth. Plus, as with any population of organisms, genetic viability must be maintained so as to minimise the occurrence of inbreeding amongst the herd. For these reasons, it has been recommended to the Department of Conservation that a herd of three hundred individuals be maintained, (equivalent to an effective population of fifty) so that Kaimanawa horses can exist in harmony with the other organisms sharing the same range.

Appendix I.

Tables I.i to I.iii illustrate the an example of the calculation of electrophoretic allele frequencies.

	SEROLOGICAL RESULTS							ELECTROPHORETIC RESULTS								
horse ID #	A	C	D	K	P	Q	U	Al	PGD	PHI	Pi	Es	Tf	Gc	AlB	Hb
H1	af/	a/	cefgm/de	_/_	a/	b/	a/	AB	FF	II	L S2	II	H2 H2	FF	KK	A1 B1
H2	af/	a/	cegmn/	_/_	a/	abc/	a/	BB	FF	II	L L	FI	F1 H2	FF	KK	A1 B1
H3	af/	a/	cefgm/de	_/_	_/	_/	a/	AB	FF	II	L L	IS	H2 O	FF	KK	B2 B1
H4	af/	a/	cgm/dk	_/_	_/	abc/	_/	BB	FF	II	N S2	II	D F2	FF	KK	B2 B2
H5	af/	a/	bcm/cgm	_/_	a/	abc/	a/	AB	FS	II	L L	FI	D F2	FF	KK	B2 B2
H6	af/	a/	cgm/	_/_	a/	abc/	_/_	AA	FF	II	L L	II	H2 O	FF	KK	B2 B1
H7	_/	a/	bcm/	_/_	_/	_/	_/_	BB	FF	II	L S1	II	D O	FF	FK	B2 B2
H8	af/	a/	cgm/dk	_/_	_/	c/	a/	BB	FS	II	N U	FI	F2 F2	FF	KK	B2 B1
H9	af/	a/	cgm/dn	_/_	_/	abc/	a/	AB	FF	II	S1 S2	II	D F2	FF	KK	B2 B1
H10	af/	a/	dghm/dk	_/_	_/	_/	a/	AA	FF	II	JU	IS	F2 F2	FF	KK	B2 B2
H11	af/	a/	cgm/de	_/_	a/	bc/	_/_	AA	FF	II	U Z	FF	D D	FF	KK	B2 B2
H12	af/	a/	cgm/de	a/	a/	b/	_/_	AB	FF	II	U Z	FF	D D	FF	KK	B1 B1
H13	af/c	a/	cgm/dk	_/_	a/b	_/	_/_	AA	FF	II	N N	FI	D D	FS	KK	B1 B1
H14	bc/	a/	cegmn/dk	_/_	a/	c/	_/_	AB	FS	II	N N	FI	D F2	FF	KK	B1 B1
H15	af/b	_/_	cgm/dk	_/_	_/	c/	_/_	AA	FF	II	F K	II	F2 O	FF	KK	B2 B1
H16	af/	a/	cegmn/de	_/_	a/	abc/	_/_	AA	FS	II	F K	II	D O	FF	KK	B2 B2
H17	af/	a/	cgm/dghm	_/_	a/	_/	_/_	AA	FS	II	H S1	FI	F2 F2	FS	KK	B2 B2
H18	b/	a/	dk/	_/_	b/	_/	_/_	BB	DF	II	N S2	II	D F1	FF	KK	B1 B1
H19	af/b	a/	cgm/de	_/_	a/	c/	a/	AB	FS	II	L2 N	FI	D F2	FF	KK	B2 B2
H20	b/	a/	bcm/cgm	_/_	a/	c/	a/	AA	FF	II	N S1	FF	D D	FF	KK	B2 B1
H21	af/b	a/	cegmn/dk	_/_	a/	_/	_/_	AB	FS	II	L2 N	FF	D D	FF	KK	B1 B1
H22	af/	a/	cegmn/dk	_/_	a/	c/	a/	AB	FS	II	F U	FI	D D	FF	KK	B2 B1

Table Li: Blood types of the Station Hack sample population.

SEROLOGICAL RESULTS							ELECTROPHORETIC RESULTS								
A	D	K	P	Q	U	C	PGD	PHI	Al	Gc	Es	Tf	A1B	Pi	Hb
_/	bcm/	_/_	_/	_/	_/	_/_	DF	II	AA	FF	FF	D D	FK	F K	A1 B1
af/	bcm/cgm	_/_	_/	_/	_/_	a/	FF	II	AA	FF	FF	D D	KK	F K	A1 B1
af/	bcm/cgm	_/_	_/	_/	_/_	a/	FF	II	AA	FF	FF	D D	KK	F U	B1 B1
af/	cefgm/de	_/_	_/	_/	_/_	a/	FF	II	AA	FF	FF	D D	KK	H S1	B1 B1
af/	cefgm/de	_/_	_/	_/	_/_	a/	FF	II	AA	FF	FI	D D	KK	JU	B1 B1
af/	cegmn/	_/_	_/	_/	_/_	a/	FF	II	AA	FF	FI	D D	KK	L L	B1 B1
af/	cegmn/de	_/_	_/	_/	_/_	a/	FF	II	AA	FF	FI	D F1	KK	L L	B1 B1
af/	cegmn/dk	_/_	a/	abc/	_/_	a/	FF	II	AA	FF	FI	D F2	KK	L L	B2 B1
af/	cegmn/dk	_/_	a/	abc/	_/_	a/	FF	II	AB	FF	FI	D F2	KK	L L	B2 B1
af/	cegmn/dk	_/_	a/	abc/	_/_	a/	FF	II	AB	FF	FI	D F2	KK	L S1	B2 B1
af/	cgm/	_/_	a/	abc/	_/_	a/	FF	II	AB	FF	FI	D F2	KK	L S2	B2 B1
af/	cgm/de	_/_	a/	abc/	_/_	a/	FF	II	AB	FF	FI	D F2	KK	L2 N	B2 B1
af/	cgm/de	_/_	a/	abc/	a/	a/	FF	II	AB	FF	II	D O	KK	L2 N	B2 B1
af/	cgm/de	_/_	a/	b/	a/	a/	FF	II	AB	FF	II	D O	KK	N N	B2 B1
af/	cgm/dghm	_/_	a/	b/	a/	a/	FS	II	AB	FF	II	F1 H2	KK	N N	B2 B2
af/b	cgm/dk	_/_	a/	bc/	a/	a/	FS	II	AB	FF	II	F2 F2	KK	N S1	B2 B2
af/b	cgm/dk	_/_	a/	c/	a/	a/	FS	II	AB	FF	II	F2 F2	KK	N S2	B2 B2
af/b	cgm/dk	_/_	a/	c/	a/	a/	FS	II	BB	FF	II	F2 F2	KK	N S2	B2 B2
af/c	cgm/dk	_/_	a/	c/	a/	a/	FS	II	BB	FF	II	F2 O	KK	N U	B2 B2
b/	cgm/dn	_/_	a/	c/	a/	a/	FS	II	BB	FF	II	H2 H2	KK	S1 S2	B2 B2
b/	dghm/dk	_/_	a/b	c/	a/	a/	FS	II	BB	FS	IS	H2 O	KK	U Z	B2 B2
bc/	dk/	a/	b/	c/	a/	a/	FS	II	BB	FS	IS	H2 O	KK	U Z	B2 B2

Table I.ii: Sorted blood types of the Station Hack sample population.

Sample Population:	Station Hacks	
Protein System:	PGD	
Genotypes:		
DF	**Genotype totals**	
FF	DF	1
FF	FF	13
FF	FS	8
FF	Total	22
FF		
FF	**Genotype frequencies**	
FF	DF	0.045
FF	FF	0.591
FF	FS	0.364
FF	Total	1.000
FF		
FF	**Allele frequencies**	
FF	D	0.023
FS	F	0.795
FS	S	0.182
FS	Total	1.000
FS		
FS		
FS		
FS		
FS		

Table I.iii: Calculation of allele frequencies for the PGD protein system of Station Hacks sampled.

Appendix II.

Tables II.i, II.ii, II.iii, II.vi and II.v give the blood types of each horse sampled in each of the equine types included in this study.

SAMPLE POPULATION: Kaimanawa Horses

	Electrophoretic Results									Serological Results						
horse ID #	Al	PGD	PHI	Pi	Es	Tf	Gc	A1B	Hb	A	C	D	K	P	Q	U
K1	AB	DF	II	S1 S2	II	F2 F2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	_/	_/	a/
K2	AB	DS	FI	S1 S1	II	D F2	FF	KK	B2 B1	af/	_/_	bcm/cgm	_/_	a/	c/	a/
K3	AB	FF	II	F L	II	F2 F2	FF	KK	B2 B2	af/	a/	cgm/dghm	_/_	a/	c/	_/_
K4	AB	FF	FI	L S2	FI	D F2	FF	KK	B2 B1	af/	_/_	cegmn/dk	_/_	_/	c/	_/_
K5	AB	FF	II	L L	II	H2 R	FF	KK	B2 B2	af/	_/_	dghm/dn	_/_	a/	abc/	_/_
K6	AA	FF	II	S2 J	II	H2 H2	FF	KK	B2 B2	af/	a/	bcm/dn	_/_	_/	abc/	_/_
K7	AA	FF	II	S1 S2	II	H2 H2	FF	KK	B2 B2	af/	_/_	cgm/dn	_/_	b/	abc/	_/_
K8	AA	FF	II	F F	FI	D H2	FF	KK	B2 B1	af/	a/	de/	_/_	_/	_/	_/_
K9	AB	DF	II	L S2	II	D F2	FF	KK	B2 B2	af/	a/	adn/de	_/_	_/	c/	a/
K10	AA	FF	II	L S2	FI	D F2	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	_/	_/	a/
K11	AA	FF	II	F S1	II	F2 F2	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	_/	c/	_/_
K12	AB	FF	II	L S2	II	F2 F2	FF	KK	B2 B1	af/	_/_	cegmn/de	_/_	_/	c/	_/_
K13	AB	FS	II	S1 S2	II	D F2	FF	KK	B2 B2	af/	_/_	bcm/de	_/_	a/	_/	_/_
K14	BB	DD	FI	S2 J	II	F2 F2	FF	KK	B2 B1	af/	a/	bcm/cgm	_/_	b/	c/	_/_
K15	AB	DF	II	L J	II	D F2	FF	KK	B2 B1	af/	_/_	cgm/	_/_	_/	_/	_/_
K16	BB	DD	II	J J	II	D R	FF	KK	B1 B1	af/	_/_	bcm/cegmn	_/_	b/	c/	_/_
K17	BB	FF	II	S2 S2	FI	F2 R	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	_/	abc/	_/_
K18	AB	FF	II	L J	II	F2 R	FF	KK	B1 B1	af/	_/_	cegmn/dfk	_/_	a/	c/	_/_
K19	AB	DF	II	S2 S2	FF	D F2	FF	KK	B2 B2	af/	_/_	adn/	_/_	_/	c/	a/
K20	BB	FF	II	G J	II	F2 R	FF	KK	B2 B1	_/	a/	cegmn/dk	_/_	_/	c/	_/_
K21	AB	DS	II	L S2	II	F2 F2	FF	KK	B2 B2	af/	a/	adn/bcm	_/_	a/	_/	a/
K22	AB	FF	II	L J	FI	H2 R	FF	KK	B2 B2	af/	_/_	de/dk	_/_	_/	_/	_/_
K23	BB	FF	II	J J	II	F2 F2	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	_/	c/	_/_
K24	AB	FF	II	S2 S2	II	F2 R	FF	KK	B2 B1	af/	a/	dfk/dn	_/_	a/	c/	_/_
K25	BB	FF	II	G J	II	F2 R	FF	KK	B2 B1	_/	a/	cegmn/dk	_/_	a/	c/	_/_
K26	AB	FF	II	L S1	II	F2 H2	FF	KK	B2 B2	b/	a/	cegmn/	_/_	_/	c/	a/
K27	AA	DF	II	L S2	FI	D F2	FF	KK	B2 B2	af/	_/_	bcm/cgm	_/_	_/	c/	_/_
K28	AB	FS	II	S1 S2	II	F2 R	FF	KK	B2 B2	af/b	a/	cegmn/adn	_/_	_/	abc/	a/
K29	AB	FS	II	L S2	II	F2 H2	FF	KK	B2 B2	af/	a/	bcm/dghm	_/_	a/	c/	_/_
K30	AA	FF	II		FI	F2 F2	FS	KK	B2 B2	af/	_/_	bcm/dfk	_/_	a/	_/	a/
K31	AB	FS	II	L S1	II	F2 R	FF	KK	B2 B2	af/	_/_	dk/dn	_/_	a/	abc/	_/_
K32	AB	FS	II	S1 J	FI	R R	FF	KK	B2 B2	af/	_/_	de/dghm	_/_	a/b	c/	_/_
K33	AA	FF	II	G S1	II	D F2	FF	KK	B2 B2	af/	_/_	adn/cgm	_/_	a/	_/	_/_
K34	AB	SS	II	L J	II	D F2	FF	KK	B2 B2	af/	a/	de/dn	_/_	_/	c/	_/_
K35	AB	FF	II	G S1	FI	F2 H2	FF	KK	B2 B2	af/b	_/_	de/dk	_/_	a/	_/	a/
K36	AB	FF	II	L S2	II	D F2	FS	KK	B2 B2	af/	a/	cegmn/	_/_	b/	c/	_/_
K37	BB	FF	II	L S1	II	D H2	FF	KK	B2 B2	af/	a/	dfk/dn	_/_	a/b	c/	a/
K38	AA	FS	II	I S1	II	F2 F2	FS	KK	B2 B2	af/b	a/	bcm/de	_/_	b/	_/	a/
K39	BB	FF	II	S1 S1	II	H2 R	FF	KK	B2 B2	af/b	_/_	de/dfk	_/_	a/	abc/	_/_
K40	AB	FF	II	F S2	II	D D	FF	KK	B2 B2	af/	_/_	no result	a/	a/	_/	_/_

continued overleaf

Table II.i: Blood types of the Kaimanawa horses sampled.

K41	AB	DF	II	F S2	FI	F2 R	FF	KK	B2 B2	af/	_/_	cgm/adn	_/_	_/	_/	a/
K42	AB	FS	II	F S2	II	F2 R	FF	KK	B2 B2	af/	_/_	cgm/dek	_/_	_/	c/	_/_
K43	BB	FF	II	S1 S1	II	H2 R	FF	KK	B2 B2	a/b	_/_	de/dfk	_/_	a/	abc/	_/_
K44	AA	FF	II	S1 S2	II	F2 F2	FF	KK	B2 B2	afb/	_/_	adn/dghm	_/_	a/	c/	_/_
K45	AA	FF	II	L J	II	F2 R	FS	KK	B2 B2	af/b	a/	de/dfk	_/_	_/	c/	_/_
K46	BB	FF	II	S2 S2	II	H2 H2	FF	KK	B2 B1	af/	a/	adn/de	_/_	_/	_/	_/_
K47	AB	DF	II	L L	II	F2 F2	FF	KK	B2 B2	af/b	_/_	bcm/cgm	_/_	a/	c/	_/_
K48	AB	FS	II	L S1	II	D D	FS	KK	#REF!	af/	_/_	bcm/dghm	_/_	a/	_/	_/_
K49	AA	FF	II	L L	IS	H2 R	FF	KK	#REF!	af/b	a/	adn/de	_/_	_/	c/	_/_
K50	BB	DD	II	L S2	II	F2 F2	FF	KK	#REF!	af/	_/_	adn/	_/_	_/	c/	a/
K51	AB	DD	II	L J	II	D F2	FF	KK	#REF!	af/	_/_	cgm/dek	_/_	_/	c/	_/_
K52	AA	FF	II	L S2	II	F2 F2	FF	KK	B2 B1	af/	_/_	cegmn/	_/_	_/	abc/	_/_
K53	AB	FF	FI	F L	II	D F2	FF	KK	B2 B2	af/	_/_	bcm/cgm	_/_	a/	c/	a/
K54	AA	DF	II	L S2	II	D F2	FF	KK	B2 B2	af/	_/_	dk/dn	_/_	_/	_/	a/
K55	AB	FF	II	L J	II	F2 R	FF	KK	B2 B1	af/	_/_	cegmn/dk	_/_	a/	_/	_/_
K56	AB	DF	FI	L S1	II	D R	FF	KK	B2 B2	af/	a/	adn/cegmn	_/_	a/	_/	a/
K57	AA	FF	FI	S2 S2	II	D H2	FF	KK	B2 B2	_/	_/_	bcm/cgm	_/_	a/	_/	_/_
K58	AB	DF	FI	S2 J	FI	F2 H2	FF	KK	B2 B1	af/	a/	adn/	_/_	_/	c/	_/_
K59	AB	DF	II	S2 J	II	F2 H2	FS	KK	B2 B2	af/b	_/_	cgm/dfk	_/_	_/	_/	_/_
K60	AA	FF	II	L2 S2	FI	H2 H2	FF	KK	B2 B2	af/b	_/_	bcm/dfk	_/_	_/	c/	_/_
K61	AA	DF	II	S1 S2	II	F2 H2	FS	KK	B2 B1	af/b	a/	adn/dfk	_/_	a/	c/	a/
K62	BB	FS	II	F S2	II	D F2	FF	KK	B2 B1	af/	_/_	adn/cgm	_/_	b/	c/	_/_
K63	AB	DF	II	L J	II	F2 H2	FS	KK	B2 B1	af/b	_/_	bcm/dek	_/_	_/	c/	_/_
K64	AB	FF	II	L S2	II	D R	FF	KK	B2 B2	af/b	_/_	bcm/dk	_/_	_/	_/	_/_
K65	AA	FF	II	S2 J	FI	D H2	FF	KK	B2 B2	af/	a/	adn/dk	_/_	a/	c/	_/_
K66	BB	FS	II	F J	IS	D D	FF	KK	B2 B1	af/	a/	bcm/dn	_/_	a/	c/	_/_
K67	BB	DF	II	J J	II	F2 F2	FF	KK	B2 B1	b/	a/	bcm/cegmn	_/_	_/	c/	_/_
K68	BB	FF	II	S1 S2	II	F2 H2	FF	KK	B2 B2	af/	_/_	de/dk	_/_	_/	c/	_/_
K69	AA	FF	II	F L	FI	D F2	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	_/	_/	_/_
K70	AA	DF	II	L S1	II	D D	FF	KK	B2 B2	af/	_/_	cegmn/de	_/_	a/b	_/	_/_
K71	AA	DF	II	S2 S2	II	R R	FF	KK	B2 B2	af/	a/	de/	_/_	a/	abc/	_/_
K72	AB	FF	II	F S2	II	F2 H2	FF	KK	B2 B1	af/	a/	de/	_/_	_/	_/	_/_
K73	AB	FF	II	F S1	II	H2 R	FF	KK	B2 B2	af/b	_/_	de/dfk	_/_	b/	c/	a/
K74	BB	FF	II	L L	FI	F2 H2	FF	KK	B2 B2	_/	a/	bcm/	_/_	a/	c/	_/_
K75	AB	FS	II	L S2	II	D H2	FF	KK	B2 B2	af/	_/_	dek/dghm	_/_	_/	c/	_/_
K76	AB	DF	II	S1 S2	FI	F2 H2	FF	KK	B2 B2	af/	_/_	de/dn	_/_	a/	_/	a/
K77	BB	FS	II	L S2	II	F2 H2	FF	KK	B2 B2	af/	a/	degmn/de	_/_	b/	c/	_/_
K78	BB	FF	II	L S1	FI	H2 R	FF	KK	B2 B2	_/	_/_	degmn/dghm	_/_	b/	c/	a/
K79	AB	FF	II	L S2	II	D R	FF	KK	B2 B1	af/	_/_	ad/dn	_/_	b/	c/	_/_
K80	BB	DF	II	J J	II	D F2	FF	KK	B2 B1	af/	_/_	cgm/dek	_/_	_/	c/	_/_
K81	AB	DF	II	S1 S2	II	H2 H2	FF	KK	B2 B2	_/	a/	dek/	_/_	_/	_/	_/_
K82	AB	FF	II	F S1	FI	D H2	FF	KK	B2 B2	af/	a/	de/dghm	_/_	a/	c/	_/_
K83	AB	FF	II	L S1	FI	D H2	FF	KK	B2 B2	af/	_/_	bcm/dghm	_/_	a/	c/	a/
K84	AB	FF	II	F S2	II	H2 H2	FF	KK	B2 B2	b/	_/_	bcm/de	_/_	a/	_/	_/_

continued overleaf

Table II.i: Blood types of the Kaimanawa horses sampled.

K85	AA	FF	II	S1 S2	II	H2 R	FF	KK	B2 B2	af/	_/_	dfk/dek	_/_	_/	abc/	_/_
K86	AA	DF	II	F L	II	D F2	FF	KK	B2 B2	b/	a/	cgm/de	_/_	a/	_/	_/_
K87	AA	DF	II	L S2	II	D F2	FF	KK	B2 B2	af/	_/_	dk/dn	_/_	_/	_/	a/
K88	AB	DF	II	S2 J	FI	H2 H2	FF	KK	B2 B1	af/	_/_	dek/adn	_/_	_/	_/	_/_
K89	AA	DS	II	F S2	II	F2 H2	FF	KK	B2 B1	af/	_/_	dek/	_/_	b/	c/	a/
K90	AA	FF	II	F S1	II	H2 R	FF	KK	B2 B1	af/b	_/_	dfk/dek	_/_	b/	c/	a/
K91	AB	FF	II		II	F2 R	FF	KK	B2 B2	af/	_/_	dghm/dk	_/_	b/	abc/	_/_
K92	AA	FF	II	S1 J	II	H2 H2	FF	KK	B2 B2	b/	a/	bcm/cgm	_/_	b/	abc/	a/
K93	AB	FF	II	S2 S2	FI	H2 R	FS	KK	B2 B2	af/	a/	bcm/dk	_/_	_/	abc/	_/_
K94	AB	FF	II	S2 J	II	F2 H2	FS	KK	B2 B1	af/	a/	bcm/dfk	_/_	a/	c/	_/_
K95	AB	FF	II	F A	II	D H2	FF	KK	B2 B1	af/b	a/	bcm/dek	_/_	b/	_/	a/
K96	AA	FF	II	S2 +	FI	D F2	FS	KK	#REF!	af/	_/_	cegmn/ad	_/_	a/	c/	_/_
K97	BB	FF	II	G J	II	F2 R	FF	KK	#REF!	af/	_/_	cegmn/	_/_	_/	c/	_/_
K98	AA	DF	II	S2 J	II	H2 H2	FF	KK	B1 B1	af/	_/_	bcm/dek	_/_	b/	_/	_/_
K99	AB	FF	II	F J	FI	D F2	FF	KK	B2 B2	af/	_/_	de/dghm	_/_	_/	abc/	_/_
K100	AB	FF	FI	S2 S2	II	D H2	FF	KK	B2 B2	af/	_/_	cgm/de	_/_	_/	abc/	_/_
K101	AA	DF	II	S1 J	II	F2 H2	FF	KK	B2 B2	b/	a/	adn/dek	_/_	a/	c/	a/
K102	BB	FF	II	S2 J	II	F2 H2	FF	KK	B1 B1	af/	a/	dfk/dek	_/_	a/	c/	_/_
K103	AB	DF	II	L S2	II	R R	FF	KK	B2 B1	af/	a/	dfk/dek	_/_	_/	c/	_/_
K104	AB	DF	II	S1 S1	II	H2 H2	FS	KK	B2 B2	b/	a/	bcm/dek	_/_	a/	c/	a/
K105	AB	DF	II	S2 J	II	F2 F2	FS	KK	B1 B1	af/b	a/	de/dfk	_/_	_/	_/	_/_
K106	AA	DS	II	I J	II	F2 F2	FF	KK	B2 B2	af/b	a/	cgm/dfk	_/_	a/	_/	a/
K107	AB	DF	II	J J	II	H2 R	FF	KK	B1 B1	af/b	_/_		_/_	_/	c/	_/_
K108	AB	FF	II	L S1	II	D F2	FF	KK	B2 B2	af/	a/	cgm/dfk	_/_	a/b	c/	a/
K109	BB	FF	II	L J	II	F2 H2	FF	KK	B2 B1	af/	_/_	cegmn/ad	_/_	a/	c/	_/_
K110	AB	FF	II	F F	FI	H2 R	FF	KK	B2 B2	af/b	a/	dek/	_/_	b/	c/	a/
K111	BB	DS	II	S2 J	II	D F2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	a/	c/	_/_
K112	AB	FF	II	G L	II	F2 R	FF	KK	B2 B1	c/	a/	cegmn/dghm	_/_	_/	_/	_/_
K113	AA	FF	II	F S1	II	D R	FF	KK	B2 B2	af/	_/_	bcm/de	_/_	a/	_/	_/_
K114	BB	DF	FI	S1 J	II	F2 R	FF	KK	B2 B1	af/	a/	cegmn/	_/_	b/	_/	a/
K115	AB	FF	II	S2 J	II	D R	FS	KK	B2 B1	af/	_/_	dek/	_/_	_/	_/	_/_
K116	AA	DD	FI	F S2	FI	F2 F2	FF	KK	B2 B2	af/	_/_	adn/	_/_	_/	_/	_/_
K117	AB	FF	FI	S1 J	II	D F2	FF	KK	B2 B2	af/	a/	dfk/	_/_	_/	_/	_/_
K118	BB	FF	II	S2 J	II	F2 F2	FF	KK	B2 B2	af/	_/_	cegmn/dk	_/_	a/	c/	_/_
K119	BB	FF	II	L L	II	F2 F2	FF	KK	B2 B2	afb/	a/	cegmn/de	_/_	_/	c/	_/_
K120	AA	FF	II	L J	FI	D H2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	a/	abc/	_/_
K121	AA	FF	II	S1 S2	FI	D H2	FS	KK	B2 B2	af/	a/	adn/dghm	_/_	a/	c/	_/_
K122	AB	DF	II	F S2	IS	D R	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	a/	c/	_/_
K123	AA	FF	II	F L	II	D R	FF	KK	B2 B2	af/	a/	cgm/	_/_	a/	abc/	a/
K124	AA	FF	II	G S2	FS	D F2	FF	KK	B2 B2	af/	_/_	dek	_/_	a/	_/	_/_
K125	BB	FF	II	L S2	II	D D	FF	KK	B2 B1	af/	_/_	de/	_/_	_/	c/	a/
K126	AA	FS	II	L S2	II	F2 R	FF	KK	B2 B1	af/	a/	dfk/	_/_	a/	c/	_/_
K127	AA	FF	FI	L S2	FI	F2 R	FF	KK	B2 B1	af/	a/	cgm/de	_/_	a/	c/	a/
K128	AB	DF	II	L L	II	F2 R	FF	KK	B2 B1	af/	_/_	adn/cegmn	_/_	b/	c/	_/_

continued overleaf

Table II.i: Blood types of the Kaimanawa horses sampled.

K129	AA	FS	II	L S2	II	F2 R	FS	KK	B1 B1	af/	a/	cgm/de	_/_	a/b	_/	_/_
K130	AA	DF	II	J S2	II	D H2	FF	KK	B2 B1	b/	_/_	bcm/dfk	_/_	_/	abc/	_/_
K131	AB	FF	FI	L S1 (+)	FI	F2 R	FF	KK	B2 B2	af/	a/	bcm/cgm	_/_	a/	c/	_/_
K132	AA	FF	II	L S1 (+)	FI	R R	FF	KK	B2 B1	af/b	a/	bcm/dek	_/_	a/	c/	a/
K133	BB	FF	II	S2 S2	FI	D F2	FF	KK	B2 B2	af/	_/_	adn/dek	_/_	_/	abc/	_/_
K134	AB	FF	II	F I	II	R R	FF	KK	B2 B1	af/b	_/_	dghm/dn	_/_	_/	c/	a/
K135	AB	FF	II	J S1	FI	F2 H2	FS	KK	B2 B1	af/b	a/	cgm/dek	_/_	_/	c/	_/_
K136	AB	FF	II	S1 L	II	F2 F2	FS	KK	B2 B2	af/	a/	bcm/dghm	_/_	a/	c/	_/_
K137	AB	FF	II	F S2	II	R R	FF	KK	B2 B2	af/c	a/	bcm/dfk	_/_	b/	_/	a/
K138	AB	DD	II	J S2	II	F2 F2	FF	KK	B1 B1	af/b	a/	de/dfk	_/_	a/	c/	_/_
K139	AA	FF	II	L S2	II	F2 F2	FF	KK	B2 B2	af/	_/_	bcm/de	_/_	_/	abc/	_/_
K140	AB	FF	II	F F	II	H2 H2	FF	KS	B2 B1	af/	_/_	dek/dn	_/_	a/	_/	a/
K141	AA	FF	II	F S2	IS	D H2	FF	KS	B2 B2	af/b	a/	cegmn/dghm	_/_	b/	_/	a/
K142	AB	DF	II	S2 S2	II	D R	FF	KK	B2 B2	af/	_/_	dn/	_/_	_/	abc/	a/
K143	AB	FS	II	G L	II	F2 F2	FF	KK	B2 B2	af/	a/	dek/dn	_/_	a/	c/	_/_
K144	AA	FF	II	F F	GI	D H2	FF	KK	B2 B1	af/	_/_	de/dn	_/_	a/	_/	_/_
K145	AB	FS	II	L S2	II	H2 H2	FF	KK	B2 B1	_/	_/_	bcm/de	_/_	a/	c/	_/_
K146	BB	FF	II	F J	IS	D F2	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	_/	c/	a/
K147	BB	FF	II	F S1	II	H2 H2	FF	KS	B2 B1	af/	_/_	dn/	_/_	a/	_/	_/_
K148	AA	DF	II	L S2	II	H2 H2	FF	KK	B2 B1	af/	a/	dn/	_/_	_/	abc/	_/_
K149	AB	FS	II	F S2	II	O O	FF	KK	B2 B2	af/	_/_	cgm/de	_/_	_/	c/	a/
K150	AB	FS	II	F L	IS	F2 H2	FF	KS	B2 B2	af/	_/_	cegmn/dk	_/_	a/	c/	_/_
K151	AA	DF	II	JJ	II	F2 H2	FS	KK	B2 B1	af/	_/_	dfk/dn	_/_	_/	c/	_/_
K152	AA	FF	II	F L	II	D F2	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	_/	_/	_/_
K153	AB	FF	II	J S1	II	H2 R	FF	SS	B2 B1	b/	a/	de/dn	a/	_/	_/	_/_
K154	AA	FF	II	S2 S2	GI	D R	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	_/	_/	_/_
K155	AB	FF	II	J S1	II	F2 R	FF	KK	B2 B2	b/	a/	dek/dn	_/_	a/	c/	_/_
K156	AB	FF	II	F I	II	F2 H2	FF	KS	B2 B1	af/	a/	de/	_/_	_/	c/	a/
K157	AB	DF	II	S2 S2	IS	D F2	FF	KK	B2 B1	af/b	_/_	cegmn/dfk	_/_	a/	c/	a/
K158	AB	FF	II	S2 S2	II	D H2	FF	KK	B2 B2	af/	_/_	cgm/de	_/_	_/	_/	_/_
K159	AB	FF	II	S1 S2	FS	D F2	FF	KK	B2 B2	af/	_/_	cgm/de	_/_	a/b	c/	_/_
K160	AB	FS	FI	I S1	II	R R	FF	KK	B1 B1	af/	_/_	cgm/dghm	_/_	a/	c/	a/
K161	BB	FF	II	F S1	IS	F2 H2	FF	KS	B2 B2	af/	_/_	de/dk	_/_	_/	_/	a/
K162	AA	FF	II	S2 S2	GI	D F2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	_/	_/	a/
K163	AB	FF	II	F S2	II	D R	FF	KK	B2 B2	af/	_/_	dk/dn	_/_	_/	abc/	a/
K164	AB	FF	II	J L (+)	II	D D	FF	KK	B2 B1	af/	a/	cegmn/de	_/_	a/	c/	_/_
K165	AB	FF	II	F F	II	F2 R	FF	KK	B2 B2	af/	a/	de/	_/_	b/	c/	a/
K166	AB	FF	II	J S1	II	H2 R	FF	KK	B2 B2	af/	a/	bcm/dek	_/_	a/	_/	_/_
K167	AA	FF	FI	I S1	II	D F2	FF	KK	B2 B2	af/	a/	cgm/	_/_	a/	c/	_/_
K168	AB	DF	II	F S1	II	D F2	FF	KK	B2 B2	b/	a/	de/dn	_/_	a/	_/	a/
K169	AB	FF	II	S2 S2	II	F2 F2	FF	KK	B2 B1	_/	a/	bcm/cegmn	_/_	b/	c/	_/_
K170	AA	FF	II	F I	II	H2 R	FF	KS	B2 B2	af/	a/	dn/	_/_	a/b	_/	a/
K171	AA	FF	II	J L (+)	II	F2 H2	FF	KK	B2 B2	af/b	a/	adn/dek	_/_	a/	c/	_/_
K172	AB	FF	II	F F	II	H2 O	FF	KK	B2 B1	af/b	a/	dfk/dn	a/	a/	_/	a/

continued overleaf

Table II.i: Blood types of the Kaimanawa horses sampled.

K173	AB	FF	II	S1 S2	IS	D F2	FF	KK	B2 B2	af/b	a/	cegmn/de	_/_	b/	c/	a/
K174	AB	FF	II	F S1	GI	D F2	FS	KK	B2 B2	af/	_/_	de/dn	a/_	a/	c/	a/
K175	AA	FF	II	I L	II	D H2	FF	KK	B2 B1	af/	a/	de/dn	_/_	_/	c/	a/
K176	AB	FF	II	J S2	II	D H2	FF	KK	B2 B1	af/	_/_	cegmn/de	_/_	a/	_/	_/_
K177	AB	FS	II	S1 S1	II	F2	FF	KK	B2 B1	af/b	_/_	dghm/dfk	_/_	b/	c/	_/_
K178	AB	FF	II	S1 S2	GI	D	FF	KK	B2 B2	af/	_/_	bcm/de	_/_	a/	c/	_/_
K179	AA	FF	II	I S2 (+)	FI	H2 H2	FF	KK	B2 B2	af/	_/_	adn/dfk	_/_	a/	c/	_/_
K180	AB	FF	FI	S2 S2	II	F2 F2	FF	KK	B2 B1	af/	_/_	dfk/de	_/_	_/	abc/	_/_
K181	AB	FF	II	L L	GI	F2	FF	KK	B2 B2	af/	a/	adn/	_/_	_/	c/	_/_
K182	AA	DS	II	S1 S1	II		FS	KK	B2 B2	af/	a/	dfk/	_/_	a/	c/	_/_
K183	BB	FF	FI	F L	IS	F2 H2	FF	KK	B2 B2	af/b	a/	cegmn/	_/_	a/	c/	_/_
K184	AA	FS	II	F I	II	D F2	FF	KK	B2 B2	af/	_/_	cgm/de	_/_	a/b	c/	_/_
K185	AA	FF	FI	F S1	GI	F2 F2	FF	KS	B2 B2	af/	a/	cgm/dek	_/_	_/	c/	a/
K186	AB	DF	II	S1 S1	IS	H2	FF	KS	B2 B1	af/	a/	cgm/dn	_/_	a/	abc/	_/_
K187	AB	FF	FI	S2 S2	IS	D H2	FF	KK	B2 B2	af/	a/	cgm/dghm	_/_	_/	abc/	_/_
K188	AB	FF	II	S1 S1	FI	D H2	FF	KS	B2 B2	af/	_/_	cgm/dek	_/_	_/	_/	a/
K189	AA	FF	II	S2 S2	II	D F2	FF	KK	B2 B2	af/	a/	cgm/dn	_/_	_/	c/	a/
K190	AA	FF	II	J J	GI	F2 H2	FF	KS	B2 B2	_/	_/_	dek/	_/_	_/	c/	_/_
K191	AB	FF	II	S1 S2	II	F2 F2	FF	KK	B2 B2	af/b	a/	dn/	_/_	a/	c/	a/
K192	AB	FF	II	F F	II	H2 O	FF	KK	B2 B1	af/	_/_	de/dfk	a/	a/	c/	a/
K193	AB	FF	II	F S1	GI	D F2	FF	KK	B2 B2	af/	a/	cgm/dek	_/_	_/	c/	a/
K194	AA	DF	II	L S2	IS	D H2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	_/	c/	a/
K195	AA	DF	II	J J	GI	H2 R	FF	KK	B2 B1	af/	_/_	adn/dek	_/_	a/b	_/	_/_
K196	AA	DS	II	I S1	II	D R	FF	KK	B2 B1	af/	a/	cgm/dghm	_/_	a/	_/	a/
K197	AB	FF	II	F L	II	R R	FF	KK	B2 B2	af/	a/	cgm/de	_/_	_/	c/	_/_
K198	AA	DF	II	F S1	II	D H2	FF	KK	B2 B2	af/	a/	dk/	_/_	_/	_/	_/_
K199	AB	FF	II	J L	IS	D F2	FF	KK	B2 B2	af/	_/_	cegmn/de	_/_	_/	_/	a/
K200	AB	SS	II	S1 I	FF	F2 R	FF	KK	B2 B1	af/	_/_	cgm/	_/_	b/	c/	_/_
K201	AB	FF	II	L S2	II	H2 R	FF	KK	B2 B1	af/	_/_	de/dghm	_/_	_/	c/	a/
K202	AB	DF	II	F F	II	D R	FF	KK	B2 B2	af/	a/	bcm/adn	_/_	_/	_/	_/_
K203	AA	FS	II	F S2	IS	F2 H2	FF	KS	B2 B2	af/	a/	cgm/dk	_/_	a/	c/	a/
K204	AB	FF	II	L S1	II	F2 F2	FF	KK	B2 B2	af/	_/_	cegmn/de	_/_	_/	c/	a/
K205	AB	FF	FI	F S2	II	F2 F2	FF	KK	B2 B1	af/	a/	cgm/de	_/_	_/	c/	
K206	AA	DF	II	S2 S2	II	F2 F2	FF	KK	B2 B1	af/	_/_	de/dn	_/_	_/	_/	_/_
K207	AA	FF	II	F L	FF	R R	FF	KK	B2 B2	af/	_/_	cegmn/de	_/_	a/	_/	_/_
K208	BB	FS	II	L S2	II	H2 H2	FF	KK	B2	af/	_/_	bcm/cegmn	_/_	a/	c/	_/_
K209	BB	FF	II	L S2	II	F2 R	FF	KK	B2 B2	af/	a/	dghm/de	_/_	_/	_/	_/_
K210	AB	DF	II	S2 S2	FI	F2 F2	FF	KK	B2 B2	af/	a/	cgm/dn	_/_	_/	abc/	_/_
K211	AB	FF	FI	G S2	II	D H2	FF	KK	B2 B2	af/	a/	cgm/dfk	_/_	_/	_/	_/_
K212	AB	DF	FI	J S2	II	D F2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	b/	_/	_/_
K213	AB	FF	II	S1 S2	II	D H2	FF	KK	B2 B1	af/	_/_	bcm/	_/_	_/	c/	_/_
K214	AA	FF	II	L S2	II	F2 F2	FF	KK	B2 B1	af/	a/	de/dn	_/_	_/	_/	a/
K215	AB	FF	II	F L	II	D R	FF	KK	B2 B1	af/	_/_	de/dn	_/_	_/	c/	_/_
K216	AB	FF	FI	F S1	II	D F2	FF	KK	B2 B2	_/	_/_	de/dn	_/_	_/	_/	a/

continued overleaf

Table II.i: Blood types of the Kaimanawa horses sampled.

K217	AB	FF	II	G J	II	D D	FF	KK	B2 B1	af/	a/	bcm/de	_/_	a/	c/	_/_
K218	AA	DF	II	G S1	FI	H2 R	FF	KK	B2 B2	af/	a/	adn/de	_/_	a/	c/	_/_
K219	AA	FF	II	F S2	II	D H2	FF	KK	B2 B1	af/	a/	cegmn/de	_/_	b/	_/	_/_
K220	BB	FF	II	S2 S2	II	F2 H2	FF	KK	B2 B1	_/	a/	bcm/cegmn	_/_	b/	_/	_/_
K221	AA	FF	II	S2 S2	II	H2 H2	FF	KK	B2 B1	af/	_/_	bcm/	_/_	_/	abc/	_/_
K222	AB	FF	II	S1 S2	FS	D F2	FF	KK	B2 B2	af/	a/	bcm/de	_/_	b/	_/	_/_
K223	AA	DF	II	F S2	II	H2 R	FF	KK	B2 B2	af/	a/	de/dn	_/_	a/	_/	_/_
K224	AB	DF	II	L S2	IS	D D	FF	KK	B2 B2	af/	a/	de/dn	_/_	_/	abc/	_/_
K225	AB	FS	II	L S1	II	F2 F2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	a/	c/	a/
K226	AB	FF	II	J S1	IS	F2 H2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	_/	_/	_/_
K227	AA	FF	II	L S2	II	F2 H2	FF	KK	B2 B2	af/b	_/_	de/dn	_/_	_/	c/	_/_
K228	AB	FF	II	J S1	FI	R R	FF	KK	B2 B2	af/b	_/_	cgm/dghm	_/_	a/	c/	_/_
K229	AB	FF	II	F S2	FI	D D	FF	KK	B2 B2	af/	_/_	bcm/cegmn	_/_	_/	_/	_/_
K230	AB	FF	II	S1 S2	II	D R	FF	KK	B2 B1	af/	a/	bcm/de	_/_	_/	_/	_/_
K231	AA	DF	II	S2 S2	II	D F2	FF	KK	B2 B2	af/b	_/_	dghm/dk	_/_	a/	_/	a/
K232	AB	FS	II	L S1	II	D F2	FF	KK	B2 B2	_/	_/_	dk/dn	_/_	a/	abc/	_/_
K233	BB	FF	II	L S1	II	H2 R	FF	KK	B2 B2	af/	_/_	de/dk	_/_	_/	_/	_/_
K234	BB	FF	II	F L	II	F2 R	FF	KK	B2 B2	af/	_/_	dghm/de	_/_	a/	c/	_/_
K235	AA	DF	II	F S2	FI	H2 R	FF	KK	B2 B2	af/b	_/_	bcm/de	_/_	_/	_/	_/_
K236	BB	FF	II	S2 S2	II	H2 R	FF	KK	B2 B2	af/	a/	cegmn/	_/_	_/	abc/	_/_
K237	AB	FF	II	F L	II	H2 R	FF	KK	B2 B1	af/	_/_	de/dn	_/_	_/	c/	a/
K238	AB	FF	II	G S2	IS	H2 R	FF	KK	B2 B2	af/	a/	adn/de	_/_	_/	c/	_/_
K239	BB	FF	II	L L	II	D F2	FF	KK	B2 B2	af/	a/	bcm/cegmn	_/_	a/b	c/	_/_
K240	AA	FS	II	F L	FI	F2 F2	FF	KK	B2 B2	af/	a/	dfk/dn	_/_	a/	_/	_/_
K241	AB	FF	II	J S2	FI	H2 R	FF	KK	B2 B2	af/b	a/	cgm/dghm	_/_	a/	c/	_/_
K242	AA	FF	II	J S2	IS	D H2	FF	KK	B2 B2	af/	_/_	de/dn	_/_	_/	_/	_/_
K243	AB	DF	II	S2 S2	II	F2 F2	FF	KK	B2 B2	af/	_/_	dghm/dn	_/_	_/	_/	a/
K244	AB	FF	II	F L	FI	D F2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	a/	c/	_/_
K245	AA	FF	II	F G	II	D R	FF	KK	B2 B2	af/	a/	cgm/de	_/_	a/	_/	_/_
K246	AB	FF	II	S2 S2	II	D F2	FS	KK	B2 B2	af/	a/	cegmn/	_/_	a/b	c/	_/_
K247	BB	FF	II	J J	II	D F2	FF	KK	B2 B1	af/	_/_	bcm/cegmn	_/_	a/b	c/	_/_
K248	AB	DF	II	S2 S2	II	D H2	FF	KK	B2 B2	b/	_/_	bcm/de	_/_	_/	c/	_/_
K249	BB	FF	II	J S2	II	D R	FF	KK	B2 B1	af/	a/	cegmn/dfk	_/_	a/b	_/	_/_
K250	AA	FF	II	F L	II	D F2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	_/	_/	_/_
K251	BB	FF	II	L S2	II	D R	FF	KK	B2 B2	af/	_/_	bcm/dghm	_/_	_/	c/	a/
K252	AB	DF	FI	S2 S2	FI	F2 H2	FF	KK	#REF!	af/	a/	cegmn/dfk	_/_	_/	_/	a/
K253	AB	DS	II	F S2	II	F2 F2	FF	KK	#REF!	af/	_/_	cgm/dn	_/_	a/	c/	_/_
K254	AA	DF	II	S1 S2	II	F2 F2	FF	KK	#REF!	af/	_/_	cgm/de	_/_	a/b	c/	_/_
K255	AA	FF	FI	S2 S2	II	D H2	FF	KK	#REF!	af/	_/_	bcm/cgm	_/_	a/	abc/	_/_
K256	AB	DF	II	S2 S2	II	F2 R	FF	KK	B1 B1	af/	_/_	dek/dn	_/_	_/	c/	_/_
K257	AB	DF	II	S1 S2	II	F2 H2	FF	KK	B2 B2	af/b	_/_	bcm/dfk	_/_	a/	c/	a/
K258	AB	FF	FI	F S2	II	D D	FF	KK	B2 B1	_/	_/_	cgm/de	_/_	_/	_/	_/_
K259	BB	FF	II	L S2	II	D H2	FF	KK	B2 B2	_/	a/	de/dk	_/_	_/	c/	_/_
K260	AB	FF	II	G S2	FI	D F2	FF	KK	B2 B2	af/	a/	cgm/de	_/_	_/	abc/	_/_

continued overleaf

Table II.i: Blood types of the Kaimanawa horses sampled.

K261	AA	FF	FI	F S2	II	D D	FF	KK	B2 B2	af/	_/_	bcm/cgm	_/_	a/	_/	_/_
K262	AB	FF	II	F J	II	F2 F2	FS	KK	B2 B2	af/	_/_	adn/	_/_	_/	abc/	_/_
K263	AB	FF	II	G S1	FI	D H2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	a/	_/	_/_
K264	AB	FF	II	J S1	II	F2 R	FF	KK	B2 B2	af/	_/_	de/dghm	_/_	a/	c/	_/_
K265	AB	FF	II	F L	II	F2 F2	FF	KK	B2 B1	af/	a/	cegmn/	_/_	_/	_/	a/
K266	BB	FF	FI	S1 S1	FI	F2 R	FF	KK	B2 B2	af/	a/	bcm/cgm	_/_	b/	_/	a/
K267	BB	DF	II	L L	II	H2 R	FF	KK	B2 B1	af/	_/_	adn/dghm	_/_	a/	c/	_/_
K268	BB	FF	II	L S2	II	F2 F2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	a/	c/	_/_
K269	AB	FF	II	F L	II	F2 H2	FF	KK	B2 B2	af/	_/_	adn/cegmn	_/_	_/	abc/	_/_
K270	AB	FF	II	S1 S2	FI	F2 H2	FF	KK	B2 B2	af/	_/_	adn/cegmn	_/_	_/	c/	a/
K271	AB	FF	II	S1 S2	II	D F2	FF	KK	B2 B1	af/	a/	cegmn/de	_/_	a/	_/	_/_
K272	AA	FF	II	J L	II	F2 F2	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	_/	_/	_/_
K273	BB	FF	II	L L	II	F2 H2	FF	KK	B2 B2	af/	_/_	bcm/cegmn	_/_	a/	c/	_/_
K274	AB	FF	II	L S2	FI	F2 F2	FF	KK	B2 B2	af/	_/_	dfk/dn	_/_	a/	c/	_/_
K275	AB	FF	II	L L	FI	D R	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	_/	abc/	_/_
K276	AB	FF	II	L S1	II	D H2	FF	KK	B2 B2	af/	a/	dghm/dn	_/_	a/	_/	_/_
K277	AA	FF	II	J J	II	F2 R	FS	KK	B1 B1	af/	_/_	de/dfk	_/_	a/	c/	_/_
K278	AB	FF	II	J J	II	F2 R	FS	KK	B2 B1	af/b	_/_	de/dk	_/_	a/	_/	_/_
K279	AB	FF	II	F S2	II	H2 R	FF	KK	B2 B2	af/	_/_	cegmn/de	_/_	_/	c/	_/_
K280	AA	FF	II	F L	II	F2 F2	FF	KK	B2 B2	af/b	a/	cgm/	_/_	_/	c/	_/_
K281	AB	FF	II	S1 S2	FI	D F2	FS	KK	B2 B2	af/	a/	cgm/dfk	_/_	_/	abc/	_/_
K282	AA	FF	II	L S1	II		FS	KK	B1 B1	af/	a/	cegmn/de	_/_	_/	abc/	a/
K283	BB	DF	II	J S2	II	D F2	FS	KK	B2 B1	af/	_/_	cegmn/dk	_/_	a/	_/	_/_
K284	AB	FF	FI	J S2	FI	F2 H2	FF	KK	B2 B2	af/	a/	cgm/dek	_/_	_/	abc/	_/_
K285	AA	DF	II	L S2	II	F2 R	FF	KK	B2 B2	af/	a/	bcm/de	_/_	_/	c/	_/_
K286	AA	DF	II	J L	II	F2 R	FF	KK	B2 B1	af/	_/_	dfk/dn	_/_	_/	c/	_/_
K287	AB	FF	II	F J	II	F2 R	FS	KK	B2 B1	af/b	_/_	bcm/cegmn	_/_	a/	c/	_/_
K288	AB	FF	II	L S2	II	D H2	FF	KK	B1 B1	af/	a/	bcm/cgm	_/_	a/	abc/	_/_
K289	AA	DF	II	J S2	II	F2 F2	FF	KK	B2 B1	af/b	a/	dfk/	_/_	_/	_/	a/
K290	BB	FF	II	F S2	II	H2 R	FF	KK	B2 B2	af/	_/_	bcm/de	_/_	_/	_/	_/_
K291	AA	FF	II	L S2	IS	D F2	FF	KK	B2 B1	af/	a/	adn/de	_/_	_/	abc/	_/_
K292	AB	DF	FI	J S2	II	F2 H2	FF	KK	B2 B1	af/b	_/_	adn/bcm	_/_	_/	abc/	a/
K293	AB	DD	II	S2 S2	II	D R	FF	KK	B2 B2	af/	a/	de/dfk	_/_	_/	abc/	_/_
K294	AB	DF	II	J S1	IS	D F2	FF	KK	B2 B2	af/b	_/_	adn/dk	_/_	_/	c/	_/_
K295	AB	DF	II	G L	FI	D H2	FF	KK	B2 B1	af/	a/	bcm/cgm	_/_	a/	abc/	_/_
K296	AA	DD	II	F J	II	D F2	FF	KK	B2 B1	af/	_/_	cgm/dfk	_/_	a/	c/	_/_
K297	AB	DF	FI	F L	II	D H2	FF	KK	B2 B2	af/	_/_	cgm/de	_/_	_/	_/	_/_
K298	BB	FF	II	L L	II	F2 H2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	a/	abc/	_/_
K299	AB	DD	II	J S2	II	F2 R	FF	KK	B1 B1	af/	a/	adn/dek	_/_	_/	c/	_/_
K300	AA	FF	II	L S1	IS	D F2	FF	KK	B2 B2	af/b	a/	adn/	_/_	a/	abc/	a/
K301	BB	DF	II	L S2	II	F2 H2	FF	KK	B2 B2	af/	_/_	cgm/dk	_/_	_/	c/	_/_
K302	BB	FF	II	L L	II	D F2	FF	KK	B2 B2	_/	_/_	bcm/dk	_/_	a/	c/	_/_
K303	AB	FF	II	J L	FI	D H2	FF	KK	B2 B2	af/	a/	dk/dn	_/_	a/	c/	_/_
K304	AB	FF	II	F J	II	D R	FS	KK	B2 B1	af/	a/	bcm/dek	_/_	a/	c/	_/_

continued overleaf

Table II.i: Blood types of the Kaimanawa horses sampled.

K305	AA	DF	II	G S2	FI	D F2	FF	KK	B2 B1	af/	a/	bcm/cegmn	_/_	a/	abc/	_/_
K306	AA	DF	II	J L	II	F2 R	FF	KK	B2 B1	af/	_/_	adn/	_/_	_/	abc/	_/_
K307	AB	FF	II	G S2	II	D F2	FF	KK	B2 B2	af/	a/	adn/cgm	_/_	b/	abc/	_/_
K308	AA	DF	II	J+	II	F2 F2	FS	KK	B2 B1	af/	_/_	adn/cgm	_/_	b/	_/	_/_
K309	AB	DF	II	J S2	FI	F2 H2	FF	KK	B2 B2	af/	_/_	bcm/dghm	_/_	a/	_/	_/_
K310	BB	FF	FI	L S1	II	F2 R	FF	KK	B2 B2	af/	a/	cgm/de	_/_	b/	_/	_/_
K311	AA	FF	II	J S2	II	H2 H2	FF	KK	B2 B1	af/	a/	bcm/dn	_/_	_/	abc/	_/_
K312	BB	DS	II	F L	II	F2 F2	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	a/	c/	_/_
K313	AB	FF	II	F J	FI	D D	FF	KK	B2 B2	af/	a/	cgm/dk	_/_	_/	c/	_/_
K314	BB	DF	FI	G S1	II	F2 F2	FF	KK	B2 B2	af/	_/_	adn/cgm	_/_	a/	c/	a/
K315	AA	FF	II	L S1	FI	F2 F2	FF	KK	B2 B2	af/	_/_	cgm/dghm	_/_	_/	_/	a/
K316	AB	FF	II	F J	II	D D	FF	KK	B2 B2	af/	_/_	cgm/de	_/_	_/	c/	_/_
K317	BB	FS	II	L S2	II	D H2	FF	KK	B2 B2	af/	a/	bcm/dn	_/_	a/	c/	_/_
K318	AB	FF	II	S2 S2	II	H2 H2	FF	KK	B2 B2	af/	_/_	bcm/	_/_	a/	abc/	_/_
K319	AB	FF	II	G L	FS	F2 R	FF	KK	B2 B2	af/	a/	adn/dghm	_/_	_/	abc/	_/_
K320	AB	FS	II	J S2	II	D H2	FF	KK	B2 B2	af/b	a/	cgm/dn	_/_	a/b	c/	_/_
K321	AA	F	II	S2 S2	FS	D F2	FF	KK	B2 B2	af/b	_/_	adn/dfk	_/_	a/	c/	a/
K322	AB	FS	II	F F	FI	F2 R	FF	KK	B2 B2	af/	_/_	dek/	_/_	a/	c/	_/_
K323	AB	FF	FI	F S2	II	D F2	FF	KK	B2 B1	af/	a/	bcm/cegmn	_/_	a/	_/	_/_
K324	AB	FF	II	F S1	II	F2 R	FF	KK	B2 B2	af/b	_/_	cgm/dghm	_/_	a/	c/	_/_
K325	AA	FF	II	S1 S2	II	H2 R	FF	KK	B2 B2	af/	a/	bcm/de	_/_	_/	abc/	a/
K326	AB	DS	II	L S2	II	F2 F2	FF	KK	B2 B2	af/	a/	adn/bcm	_/_	a/	c/	a/
K327	AB	DF	FI	F L	II	F2 H2	FS	KK	B2 B2	af/	a/	cegmn/	_/_	_/	_/	a/
K328	AA	DF	II	J S2	II	H2 R	FF	KK	B1 B1	af/	a/	bcm/dfk	_/_	_/	c/	_/_
K329	BB	FF	II	L L	IS	F2 F2	FF	KK	B2 B2	af/	_/_	cegmn/de	_/_	a/	c/	_/_
K330	AB	DF	II	F J	II	D D	FF	KK	B2 B1	af/	a/	bcm/cgm	_/_	a/	c/	_/_
K331	AA	DF	II	F S1	II	D F2	FF	KK	B2 B1	_/	a/	bcm/de	_/_	a/	_/	a/
K332	AA	DF	II	S2 S2	FI	F2 H2	FF	KK	B2 B2	af/b	a/	dfk/dghm	_/_	a/	c/	a/
K333	AA	FF	FI	J J	II	D H2	FF	KK	B2 B1	af/	a/	cgm/dek	_/_	b/	c/	_/_
K334	AB	DF	FI	S1 S2	II	F2 H2	FF	KK	B2 B2	af/	_/_	adn/dfk	_/_	_/	c/	a/
K335	AB	FF	FI	J S2	FI	D H2	FF	KK	B2 B1	af/	_/_	cgm/dek	_/_	_/	_/	_/_
K336	AB	FF	II	L S2	IS	F2 R	FF	KK	B2 B2	b/	a/	adn/de	_/_	_/	abc/	_/_
K337	AB	FF	II	J S1	FI	D D	FF	KK	B2 B2	af/b	a/	cgm/dghm	_/_	a/	c/	_/_
K338	AA	DF	II	L S1	II	H2 R	FF	KK	B2 B2	_/	_/_	dfk/de	_/_	_/	abc/	_/_
K339	AA	FF	II	L S2	II	F2 R	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	b/	_/	_/_
K340	AB	FF	II	F L	FI	F2 F2	FF	KK	B2 B2	af/b	_/_	dghm/dek	_/_	_/	abc/	_/_
K341	AA	FF	II	F F	II	F2 F2	FF	KK	B2 B2	af/b	a/	cegmn/dk	_/_	_/	c/	_/_
K342	AB	DF	II	F S2	IS	D F2	FF	KK	B2 B2	b/	a/	cegmn/de	_/_	a/	c/	_/_
K343	BB	DF	II	S1 S1	II	D F3	FF	KK	B2 B2	af/b	a/	dghm/dfk	_/_	a/b	_/	a/
K344	AA	DD	II	J J	II	F2 F2	FF	KK	B2 B1	b/	_/_	cegmn/	_/_	_/_	c/	_/_
K345	AB	DF	II	J S1	II	F2 F2	FS	KK	B2 B2	af/	_/_	dghm/dfk	_/_	_/	c/	_/_
K346	AB	FF	II	S1 S2	II	D R	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	_/	c/	_/_
K347	AB	FF	FI	L S1	II	D H2	FF	KK	B2 B2	af/	_/_	de/adn	_/_	b/	abc/	a/
K348	AB	DF	FI	F L	II	F2 H2	FF	KK	B2 B1	af/b	_/_	bcm/cegmn	_/_	_/	c/	_/_

continued overleaf

Table II.i: Blood types of the Kaimanawa horses sampled.

K349	AB	DF	FI	J S1	II	F2 F2	FF	KK	B2 B1	af/	_/_	cegmn/	_/_	_/	c/	_/_
K350	AA	FS	FI	F S2	II	D R	FF	KK	B2 B2	af/	a/	cgm/adn	_/_	b/	c/	_/_
K351	AB	DF	II	J J	FI	F2 H2	FF	KK	B2 B1	af/	_/_	dek/dn	_/_	_/	c/	_/_
K352	AB	FF	II	J L	II	F2 H2	FF	KK	B2 B1	af/	_/_	cegmn/de	_/_	b/	_/	_/_
K353	BB	FS	II	L S2	II	D H2	FF	KK	B2 B2	af/	_/_	bcm/dn	_/_	a/	c/	_/_
K354	AB	FF	II	L S1	FI	F2 R	FF	KK	B2 B2	af/	a/	adn/de	_/_	_/	abc/	a/
K355	AA	FF	II	J S2	II	D D	FF	KK	B2 B1	af/	a/	adn/bcm	_/_	a/	abc/	_/_
K356	AA	FF	II	L S2	II	D F2	FS	KK	B2 B2	af/	a/	cegmn/dk	_/_	_/	_/	_/_
K357	AB	FF	II	J S2	II	F2 R	FF	KK	B2 B1	af/	a/	bcm/cegmn	_/_	a/	c/	_/_
K358	BB	FF	II	S1 S1	FI	F2 R	FF	KK	B2 B2	af/	a/	cegmn/dghm	_/_	b/	c/	_/_
K359	AA	FF	II	F S2	II	F2 F2	FF	KK	B2 B1	af/	_/_	cegmn/dk	_/_	b/	c/	a/
K360	AB	FF	II	J S2	II	F2 R	FS	KK	B2 B2	af/	_/_	cegmn/dk	_/_	_/	c/	_/_
K361	AB	FF	II	G S1	FS	F2 R	FF	KK	B2 B2	af/	a/	de/dk	_/_	a/	_/	a/
K362	AB	FF	II	F S2	II	R R	FS	KK	B2 B1	af/	_/_	cegmn/	_/_	_/	c/	_/_
K363	AA	FF	II	S2 S2	II	H2 H2	FF	KK	B2 B2	af/	a/	cgm/dghm	_/_	_/	abc/	_/_
K364	AB	FF	II	S2 S2	FF	F2 R	FF	KK	B2 B2	af/	_/_	cegmn/de	_/_	_/	c/	_/_
K365	AB	FS	II	L L	II	D R	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	b/	_/	_/_
K366	AB	DF	II	F J	II	F2 R	FF	KK	B2 B1	af/b	_/_	cegmn/	_/_	_/	c/	_/_
K367	AA	FS	II	F S2	II	H2 R	FF	KK	B2 B1	af/	_/_	dn/	_/_	_/	abc/	a/
K368	AB	FF	II	F J	II	D D	FF	KK	B2 B1	af/b	_/_	cgm/adn	_/_	a/	abc/	_/_
K369	AB	FF	II	F L	FI	H2 R	FF	KK	B2 B1	af/	_/_	cegmn/adn	_/_	_/	c/	_/_
K370	BB	FF	II	S2 S2	II	D F2	FF	KK	B2 B2	af/	c/	bcm/cegmn	_/_	a/	c/	_/_
K371	AB	FF	II	F J	FI	H2 R	FF	KK	B2 B1	af/b	_/_	cegmn/dk	_/_	_/	c/	_/_
K372	AA	FF	II	L L	II	D R	FF	KK	B2 B2	af/	c/	cegmn/dk	_/_	b/	_/	_/_
K373	BB	DF	II	F S2	IS	F2 F2	FF	KK	B2 B2	af/b	_/_	dek/dn	_/_	a/	c/	_/_
K374	AB	FF	II	L S2	II	D R	FS	KK	B2 B2	af/	_/_	cegmn/dn	_/_	_/	c/	_/_
K375	AB	DF	II	J L	FI	F2 R	FF	KK	B2 B2	b/	c/	cegmn/	_/_	_/	c/	_/_
K376	AB	FF	II	F S1	II	F2 R	FF	KK	B2 B1	af/	c/	cegmn/dk	_/_	b/	_/	_/_
K377	AA	FF	II	L S2	II	D R	FF	KK	B2 B2	af/	_/_	cegmn/dk	_/_	b/	c/	_/_
K378	BB	DF	II	F S2	II	D F2	FF	KK	B2 B2	af/b	_/_	cegmn/dn	_/_	_/	abc/	a/
K379	AB	FF	II	J L	II	D F2	FF	KK	B2 B2	af/	_/_	adn/dek	_/_	b/	_/	a/
K380	AA	FF	II	J L	II	H2 R	FF	KK	B2 B2	af/	_/_	cegmn/adn	_/_	_/	_/	_/_
K381	AB	FF	II	F S2	II	F2 F2	FF	KK	B2 B2	af/b	_/_	cegmn/dk	_/_	_/	c/	a/
K382	BB	FF	II	L S2	II	D F2	FF	KK	B2 B2	af/	a/	bcm/dn	_/_	_/	_/	_/_
K383	BB	DF	II	F S2	II	D D	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	_/	c/	_/_
K384	AB	FF	II	F J	II	D F2	FF	KK	B2 B2	b/	_/_	cegmn/	_/_	_/	c/	_/_
K385	BB	DF	II	F L	II	D R	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	_/	_/	_/_
K386	AB	DF	II	J S2	II	D F2	FF	KK	B2 B1	af/b	_/_	cgm/dek	_/_	a/	c/	_/_
K387	AB	DF	II	S1 S2	II	H2 H2	FF	KK	B2 B2	af/	_/_	bcm/	_/_	_/	_/	_/_
K388	BB	FF	II	J S1	II	D R	FF	KK	B2 B2	af/	a/	dghm/dn	_/_	_/	_/	a/
K389	BB	FF	II	J L	II	D F2	FF	KK	B2 B2	af/	a/	de/dk	_/_	_/	_/	_/_
K390	AB	FF	II	S1 S2	FI	F2 R	FF	KK	B2 B2	_/	_/_	adn/bcm	_/_	_/	c/	a/
K391	AB	DF	II	L S2	II	F2 F2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	a/	c/	_/_
K392	AB	FF	II	J S1	FI	D F2	FF	KK	B2 B2	af/	_/_	cgm/de	_/_	a/	c/	a/

continued overleaf

Table II.i: Blood types of the Kaimanawa horses sampled.

K393	AA	FF	II	L S2	II	F2 R	FF	KK	B2 B2	af/	a/	cgm/dn	_/_	_/	abc/	_/_
K394	AB	DF	II	J S1	FI	F2 R	FF	KK	B2 B2	af/b	a/	cgmn/adn	_/_	_/	abc/	a/
K395	AB	FF	II	J L	II	D R	FF	KK	B2 B1	af/	a/	cegmn/	_/_	_/	c/	_/_
K396	AB	FF	II	F F	II	R R	FF	KK	B2 B1	af/b	_/_	cegmn/	_/_	b/	c/	_/_
K397	AB	DF	II	S1 S2	II	F2 F2	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	a/	c/	_/_
K398	AB	FF	II		II	F2 R	FF	KK	B2 B2	af/	a/	cegmn/	_/_	_/	_/	_/_
K399	AB	FF	II	S1 S1	II	R R	FF	KK	B2 B2	af/b	_/_	dghm/adn	_/_	_/_	c/	a/
K400	BB	DF	II	G L	II	F2 F2	FF	KK	B2 B2	_/	a/	cegmn/dk	_/_	a/	c/	_/_
K401	AB	FF	II	S1 S2	II	H2 R	FS	KK	B2 B2	af/	_/_	cegmn/de	_/_	_/	abc/	a/
K402	AB	FF	II	L S2	FF	H2 R	FF	KK	B2 B2	af/	_/_	adn/cgm	_/_	b/	c/	_/_
K403	AB	FF	II	J L	II	F2 R	FF	KK	B2 B2	b/	a/	cegmn/	_/_	_/	c/	_/_
K404	AB	FF	II	S1 S2	FI	D H2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	_/	c/	a/
K405	AB	FF	FI	F L	II	D D	FF	KK	B2 B1	b/	_/_	cegmn/dk	_/_	b/	c/	_/_
K406	BB	FF	II	L S2	FI	D F2	FF	KK	B2 B2	af/	_/_	adn/de	_/_	_/	abc/	a/
K407	AA	FF	FI	L L	II	D F2	FF	KK	B2 B2	af/	a/	dek/dn	_/_	_/	_/	_/_
K408	AA	FF	II	L S2	II	D D	FF	KK	B2 B2	af/	_/_	adn/dek	_/_	_/	c/	a/

Table II.i: Blood types of the Kaimanawa horses sampled.

SAMPLE POPULATION: Thoroughbred Horses

	Electrophoretic Results									Serological Results						
horse ID #	Al	PGD	PHI	Pi	Es	Tf	Gc	A1B	Hb	A	C	D	K	P	Q	U
T1	AB	FF	II	L L	II	D F2	FF	KK	B2 B1	af/	a/	cegmn/de	_/_	_/	abc/	a/
T2	BB	SS	II	L U	II	D R	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	b/	abc/	_/_
T3	AB	FF	II	L U	II	D H2	FF	KK	B2 B2	af/	a/	bcm/cegmn	_/_	_/	abc/	_/_
T4	AB	FF	II	F N	II	H2 O	FF	KK	B2 B2	af/	a/	cegmn/de	a/	a/b	abc/	a/
T5	BB	FS	II	L N	IS	D O	FF	KK	B2 B2	af/	_/_	cegmn/	_/_	a/	abc/	_/_
T6	BB	FS	II	F N	FI	D F2	FF	KK	B2 B2	af/	a/	cgm/dk	_/_	_/	abc/	a/
T7	BB	FF	II	F S2	II	F2 R	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	_/	abc/	_/_
T8	BB	FS	II	L L	II	F2 F2	FF	KK	B2 B2	af/	a/	bcm/cegmn	_/_	a/	abc/	_/_
T9	BB	FS	II	L L	IS	D F2	FF	KK	B2 B2	af/	a/	dk/dk	_/_	_/	abc/	_/_
T10	BB	FF	II	L L	II	F1 F2	FF	KK	B2 B1	af/	a/	dk/dk	_/_	a/	_/	a/
T11	BB	FS	II	L L	II	F1 F2	FS	KK	B2 B2	af/	_/_	bcm/dk	_/_	a/	abc/	_/_
T12	BB	FS	II	L L2	FI	D F2	FF	KK	B2 B1	af/	a/	bcm/bcm	_/_	a/	abc/	_/_
T13	BB	FF	II	L U	II	D F2	FS	KK	B2 B1	af/	a/	bcm/cgm	_/_	a/	abc/	_/_
T14	BB	FF	II	I S2	II	D O	FF	KK	B2 B2	af/	a/	cgm/dghm	_/_	_/	abc/	_/_
T15	BB	S_	II	F N	II	D F2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	a/b	abc/	_/_
T16	BB	FS	II	L N	II	F1 O	FF	KK	B2 B1	af/	a/	bcm/bcm	_/_	_/	abc/	a/
T17	BB	FF	II	F N	FI	D F2	FF	KK	B2 B2	af/	_/_	cgm/dfk	_/_	_/	c/	_/_
T18	AB	SS	II	L L	II	D F2	FF	KK	B2 B1	af/	a/	cgm/dfk	_/_	_/	abc/	a/
T19	BB	FS	II	L U	II	D R	FF	KK	B2 B2	af/b	a/	bcm/bcm	_/_	_/	_/	_/_
T20	AB	SS	II	N N	FI	D F1	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	a/	_/	a/
T21	AB	FS	II	L U	FI	O R	FF	KK	B2 B2	_/	a/	bcm/dk	_/_	b/	abc/	_/_
T22	BB	SS	II	L L	II	F1 R	FF	KK	B2 B2	af/	_/_	cgm/cgm	_/_	a/	_/	a/
T23	BB	FS	II	G L	FI	O R	FF	KK	B1 B1	af/	a/	bcm/cgm	_/_	_/	abc/	_/_
T24	BB	FS	II	F N	II	D D	FF	KK	B2 B2	af/	a/	bcm/cgm	_/_	b/	_/	a/
T25	BB	FS	II	L U	II	F1 R	FF	KK	B2 B2	af/b	a/	cgm/dk	_/_	_/	abc/	_/_
T26	BB	FS	II	I L	FI	F1 F1	FF	KK	B2 B2	af/	a/	bcm/cgm	_/_	a/	abc/	_/_
T27	BB	FS	II	N N	FI	D D	FF	KK	B2 B2	af/	a/	bcm/bcm	a/	_/	abc/	_/_
T28	AB	FS	II	L L	FI	H2 O	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	_/	abc/	a/
T29	BB	SS	II	I L	FI	F1 O	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	a/b	abc/	_/_
T30	BB	FS	II	N U	II	F1 F1	FF	KK	B2 B2	af/	a/	bcm/cgm	a/	_/	abc/	_/_
T31	BB	FS	II	L N	II	D F1	FF	KK	B2 B1	af/	a/	cgm/cgm	_/_	b/	abc/	a/
T32	AB	FF	II	F N	FI	O R	FF	KK	B2 B2	af/	a/	cgm/dk	_/_	a/	abc/	a/
T33	BB	FS	II	L S2	II	F1 H2	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	_/	abc/	a/
T34	BB	FS	II	L S1	II	D F2	FF	KK	B2 B2	af/	a/	dk/dk	_/_	a/	abc/	_/_
T35	BB	FF	II	G I	II	D F1	FF	KK	B2 B1	af/	a/	cegmn/dk	_/_	_/	abc/	_/_
T36	BB	SS	II	F L	II	F1 O	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	a/	c/	_/_
T37	BB	FF	II	I L	II	D F1	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	b/	abc/	_/_
T38	BB	FS	II	I S1	II	D D	FF	KK	B2 B2	af/	a/	cgm/dn	_/_	_/	abc/	_/_
T39	AB	FF	II	L L	II	F1 F2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	_/	abc/	_/_
T40	BB	FF	II	L N	II	D F1	FF	KK	B2 B2	af/	a/	bcm/cegmn	_/_	a/	abc/	_/_
T41	AB	SS	II	I L	II	F1 O	FF	KK	B2 B1	af/	_/_	bcm/bcm	_/_	b/	abc/	_/_
T42	BB	FS	II	L L	II	D F1	FF	KK	B2 B2	af/	a/	dk/dk	_/_	_/	abc/	_/_

continued overleaf

Table II ii Bloodtypes of the Thoroughbred sample population.

T43	BB	FF	II	G L	II	F1 F2	FF	KK	B2 B2	af/	a/	bcm/cgm	_/_	_/	abc/	_/_
T44	BB	FF	II	L U	II	F1 R	FF	KK	B2 B2	b/	a/	cegmn/de	_/_	_/	abc/	_/_
T45	BB	SS	II	N U	II	D F2	FF	KK	B2 B1	af/	a/	dk/dn	_/_	a/	abc/	_/_
T46	BB	FS	II	N N	II	F1 F1	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	_/	abc/	_/_
T47	AB	FS	II	F I	II	F1 O	FF	KK	B2 B2	af/	_/_	cgm/dfk	_/_	a/	abc/	_/_
T48	AB	FF	II	L S1	II	F1 F1	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	_/	c/	a/
T49	BB	FS	II	F L	II	F2 R	FF	KK	B2 B2	af/	a/	dk/dk	_/_	b/	_/	_/_
T50	AA	FF	II	I L	II	O R	FF	KK	B2 B2	af/	a/	de/dk	_/_	a/	abc/	_/_
T51	AB	FS	II	N U	IS	F2 O	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	b/	abc/	_/_
T52	BB	FF	II	L L	II	H2 R	FF	KK	B2 B2	af/	a/	cgm/cgm	_/_	_/	abc/	_/_
T53	AA	FS	II	F U	IS	F1 F2	FF	KK	B2 B2	_/	a/	cgm/cgm	_/_	b/	abc/	_/_
T54	BB	FF	II	F I	FS	F1 O	FF	KK	B1 B1	af/	a/	de/dk	_/_	b/	abc/	_/_
T55	BB	FS	II	L N	FI	R R	FF	KK	B2 B2	af/	a/	cgm/dk	_/_	b/	abc/	a/
T56	BB	FF	II	L U	II	D F2	FF	KK	B2 B1	af/	_/_	cegmn/dk	_/_	a/	_/	_/_
T57	BB	FS	II	L S1	FI	D H2	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	_/	abc/	_/_
T58	BB	FF	II	L L	II	D F1	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	_/	_/	_/_
T59	BB	FS	II	I L	II	F1 R	FF	KK	B2 B2	af/	a/	cegmn/	_/_	a/	abc/	_/_
T60	BB	FF	II	L U	II	D F1	FF	KK	B2 B2	af/	a/	cgm/cgm	a/	a/b	abc/	_/_
T61	AA	SS	II	N N	II	D D	FF	KK	B2 B1	af/	a/	dfk/dk	_/_	_/	abc/	_/_
T62	BB	FS	II	L L	II	D F1	FF	KK	B2 B2	af/	_/_	bcm/cgm	_/_	a/	abc/	_/_
T63	AB	SS	II	L N	FI	D F1	FF	KK	B2 B1	af/	a/	bcm/cegmn	_/_	_/	abc/	a/
T64	BB	SS	II	L N	II	D H2	FF	KK	B2 B2	af/	a/	cgm/de	_/_	_/	abc/	_/_
T65	AB	FF	II	G L	FI	D F1	FF	KK	B1 B1	af/	a/	dfk/dk	_/_	_/	abc/	_/_
T66	AB	FF	II	L N	II	D F1	FF	KK	B2 B2	af/	_/_	cegmn/dk	_/_	b/	abc/	_/_
T67	AB	FS	II	G N	II	H2 O	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	b/	abc/	_/_
T68	AB	FS	II	G L	II	H2 R	FF	KK	B2 B1	af/	a/	cegmn/dk	_/_	b/	abc/	a/
T69	AB	FF	II	N N	II	F1 O	FF	KK	B2 B2	af/	a/	bcm/dk	a/	a/	abc/	a/
T70	BB	FF	II	N U	II	D R	FF	KK	B2 B2	af/	a/	cgm/dk	_/_	_/	abc/	_/_
T71	AB	FF	II	L U	IS	F1 O	FF	KK	B2 B2	af/	a/	cgm/cgm	_/_	a/	_/	a/
T72	BB	FF	II	L L	II	D O	FF	KK	B2 B2	af/	a/	dk/dk	_/_	a/	abc/	_/_
T73	AB	FS	II	F U	II	F1 O	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	a/b	abc/	a/
T74	BB	FF	II	F L	II	D F2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	b/	abc/	_/_
T75	AA	FF	II	L N	II	F2 F2	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	_/	abc/	_/_
T76	BB	SS	II	L N	IS	F2 O	FF	KK	B2 B2	af/	a/	bcm/cegmn	_/_	_/	abc/	a/
T77	BB	FS	II	L L	II	D F1	FF	KK	B2 B1	af/	a/	cegmn/dk	_/_	_/	_/	a/
T78	BB	FS	II	L U	II	F1 F2	FF	KK	B2 B1	af/	_/_	bcm/cgm	_/_	_/	abc/	_/_
T79	AB	SS	II	L N	II	F1 H2	FF	KK	B2 B2	af/	a/	cgm/cgm	_/_	a/	abc/	a/
T80	BB	SS	II	L L	II	F2 O	FF	KK	B2 B2	af/	a/	cgm/dk	_/_	_/	abc/	a/
T81	AB	SS	II	L L	II	R R	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	a/	_/	_/_
T82	AB	FS	II	L S1	II	D F1	FF	KK	B2 B1	af/	a/	bcm/cgm	_/_	_/	_/	_/_
T83	AB	SS	II	N S1	II	D F2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	b/	c/	_/_
T84	AB	FS	II	I L	IS	F1 R	FF	KK	B2 B2	af/	a/	dghm/dk	a/	a/	_/	a/
T85	BB	FS	II	L L	IS	F2 F2	FF	KK	B2 B2	af/	a/	bcm/dghm	_/_	a/	abc/	_/_
T86	AB	SS	II	L L	II	F2 F2	FS	KK	B2 B2	af/	a/	cgm/dk	_/_	_/	abc/	_/_
T87	BB	FS	II	L N	II	D D	FF	KK	B2 B1	af/	a/	bcm/bcm	_/_	a/	abc/	_/_
T88	BB	FS	II	F U	II	D H2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	a/	abc/	_/_

continued overleaf

Table II ii: Bloodtypes of the Thoroughbred sample population.

T89	AB	FS	II	L U	II	D O	FF	KK	B2 B1	af/	a/	bcm/cegmn	_/_	b/	abc/	_/_
T90	BB	FF	II	S1 U	II	F1 F2	FF	KK	B2 B2	af/	a/	cegmn/	_/_	_/	abc/	_/_
T91	AA	FF	II	L L	II	D D	FF	KK	B2 B2	af/	_/_	bcm/dk	_/_	a/	_/	a/
T92	AB	FF	II	L L	II	F1 F1	FF	KK	B2 B2	af/	a/	dk/dk	_/_	_/	abc/	_/_
T93	BB	FS	II	L U	II	F1 F1	FF	KK	B2 B2	af/	a/	cgm/dk	_/_	_/	abc/	_/_
T94	BB	FF	II	L N	FI	D D	FF	KK	B2 B2	af/	a/	bcm/bcm	_/_	_/	abc/	_/_
T95	AB	FS	II	L N	FI	D O	FF	KK	B2 B2	b/	a/	bcm/cgm	_/_	a/b	_/	a/
T96	BB	FF	II	L L	SS	F1 F2	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	a/	abc/	_/_
T97	BB	FS	II	F N	II	D D	FF	KK	B2 B2	af/	a/	bcm/dk	a/	a/	_/	_/_
T98	AB	FF	II	N S1	II	F1 F2	FF	KK	B2 B2	af/	_/_	bcm/cgm	_/_	a/	abc/	a/
T99	AB	FS	II	L S2	IS	F2 H2	FF	KK	B2 B2	af/	a/	bcm/dk	a/	b/	abc/	_/_
T100	BB	SS	II	U U	II	F1 R	FF	KK	B2 B2	af/	a/	de/dk	_/_	_/	_/	_/_
T101	AB	FF	II	L N	II	F2 F2	FF	KK	B2 B2	af/b	a/	bcm/cegmn	_/_	a/	abc/	a/
T102	BB	SS	II	G N	FI	D H2	FF	KK	B2 B2	af/	a/	dk/dk	_/_	b/	abc/	a/
T103	BB	FS	II	G S2	II	D F1	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	_/	_/	_/_
T104	BB	FF	II	L L	II	F1 R	FF	KK	B2 B1	af/	a/	bcm/cgm	_/_	a/	_/	_/_
T105	BB	FF	II	L U	II	O R	FF	KK	B2 B1	af/	a/	cegmn/dk	_/_	b/	abc/	_/_
T106	BB	FF	II	N N	II	D D	FF	KK	B2 B2	af/	_/_	de/dk	_/_	b/	abc/	_/_
T107	BB	SS	II	L U	FI	F1 H2	FF	KK	B2 B1	af/	a/	cgm/cgm	_/_	b/	abc/	_/_
T108	BB	FS	II	L U	II	D F2	FF	KK	B2 B1	af/	a/	cegmn/	_/_	a/	abc/	_/_
T109	BB	FS	II	I L	II	D R	FF	KK	B2 B2	af/	a/	cgm/dfk	a/	a/	abc/	_/_
T110	BB	FS	II	L U	II	D R	FF	KK	B2 B2	af/	_/_	cegmn/dk	_/_	_/	_/	a/
T111	AB	FF	II	L U	II	D F2	FF	KK	B2 B2	af/	a/	bcm/cegmn	_/_	a/	abc/	_/_
T112	BB	SS	II	I N	II	F1 R	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	_/	abc/	_/_
T113	BB	FF	II	L L	II	D R	FF	KK	B2 B2	af/	a/	cgm/dfk	_/_	a/	_/	_/_
T114	BB	SS	II	I S2	FI	F2 F2	FF	KK	B2 B2	af/	a/	cgm/dk	_/_	_/	abc/	_/_
T115	BB	SS	II	F L	II	D O	FF	KK	B2 B1	af/	a/	cgm/dk	_/_	_/	abc/	_/_
T116	BB	FS	II	L N	II	F2 R	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	_/	c/	_/_
T117	AA	FF	II	L N	II	D F1	FF	KK	B2 B2	af/	a/	bcm/cgm	_/_	a/	abc/	a/
T118	BB	FF	II	N U	II	F1 F1	FF	KK	B2 B2	af/	a/	bcm/cgm	_/_	a/	abc/	_/_
T119	BB	SS	II	L N	II	D F1	FS	KK	B2 B2	af/	a/	cegmn/	_/_	_/	_/	a/
T120	AB	FF	II	L L	IS	O R	FF	KK	B2 B2	af/	a/	cgm/cgm	_/_	_/	abc/	_/_

Table II.11: Bloodtypes of the Thoroughbred sample population.

SAMPLE POPULATION: Arabian Horses

	Electrophoretic Results								Serological Results						
Horse ID #	Al	PGD	PHI	Pi	Es	Tf	Gc	AlB	Hb	A	C	D	K	P	Q	U
A1	AB	FF	II	L L	II	F2 F2	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	_/_	c/_	_/_
A2	AA	FS	II	F L	II	H2 H2	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	_/_	_/_	_/_
A3	AB	FS	II	U Z	II	D F2	FF	KK	B1 B1	af/	a/	bcm/de	_/_	a/	b/	a/
A4	AB	FF	II	L P	II	D O	FF	KK	B2 B2	af/	a/-	bcm/gcm	_/_	_/_	c/_	_/_
A5	AB	FS	II	L N	II	H2 O	FF	KK	B2 B1	af/	a/-	cgm/dk	_/_	a/_	_/_	a/_
A6	AB	FS	II	L L	II	D O	FF	KS	B1 B1	a/	a/-	de/dk	_/_	a/_	b/_	a/_
A7	AB	SS	II	L S1	II	D O	FF	KK	B2 B1	af/	a/-	dk/dk	_/_	a/_	c/_	_/_
A8	AB	FS	II	L S1	II	D F2	FF	KK	B1 B1	af/	a/-	dk/dk	_/_	_/_	b/_	_/_
A9	AB	FS	II	L Z	II	D H2	FF	KK	B1 B1	af/	a/-	dk/dk	_/_	_/_	b/_	_/_
A10	AB	SS	II	F U	FI	D F2	FF	KK	B2 B2	af/	a/-	bcm/dk	_/_	_/_	ab/_	_/_
A11	AB	FF	II	S1 U	II	D F2	FF	KK	B2 B1	af/	a/-	bcm/de	_/_	_/_	c/_	_/_
A12	AA	FS	II	S1 U	II	D F2	FF	KK	B2 B1	af/	a/-	bcm/bcm	_/_	_/_	c/_	_/_
A13	AA	FS	II	S1 S1	II	F2 O	FF	KK	B2 B1	af/	a/	bcm/bcm	_/_	_/	bc/	_/_
A14	AA	FS	II	L Z	II	D H2	FF	KK	B1 B1	af/	a/	dk/dk	_/_	a/	c/	_/_
A15	AA	FF	II	S1 U	II	F2 F2	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	a/	_/	_/_
A16	AB	SS	II	F S1	II	D O	FF	KK	B2 B2	af/	a/	cegmn/dk	_/_	a/	abc/	a/
A17	AB	SS	II	F L	II	F2 F2	FF	KK	B2 B1	af/	a/	cgm/dk	_/_	a/	abc/	_/
A18	AB	SS	II	L L	II	F2 H2	FF	KK	B2 B1	af/	a/	bcm/de	_/_	a/	abc/	_/_
A19	AB	FS	II	S1 U	II	F2 O	FF	KK	B1 B1	af/	a/	de/dk	_/_	a/b	abc/	a/
A20	AB	FF	II	L U	II	F2 O	FF	KK	B1 B1	af/	a/	cgm/dk	_/_	_/	c/	a/
A21	AB	FF	II	L S1	II	H2 O	FF	KK	B2 B2	af/	a/	bcm/bcm	_/_	a/b	b/c	_/
A22	AA	FS	II	L2 U	II	O O	FF	KK	B1 B1	af/	a/	dk/dk	_/_	a/	_/	_/_
A23	AB	FS	II	L U	II	D H2	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	_/	b/	_/_
A24	BB	SS	II	L2 P	II	O O	FF	KK	B1 B1	af/	a/	bcm/de	_/_	a/	c/	_/_
A25	AB	FF	II	L S1	II	D F2	FF	KK	B2 B2	af/	a/	dk/dk	_/_	a/	c/	_/_
A26	AB	FS	II	F S1	II	F2 F2	FF	KK	B2 B1	af/	a/	de/dk	_/_	a/	c/	a/
A27	AA	FF	II	U Z	II	D H2	FF	KK	B1 B1	af/	a/	cegmn/dk	_/_	_/	c/	_/_
A28	AB	FS	II	L L	II	D O	FF	KS	B1 B1	af/	a/	dk/dk	_/_	_/	b/	a/
A29	AA	SS	II	L S1	II	F2 F2	FF	KK	B1 B1	af/	a/	dk/dk	_/_	a/	c/	_/_
A30	AB	FF	II	L S1	II	F2 O	FF	KK	B2 B1	af/b	a/	dk/dk	_/_	_/	c/	_/_
A31	AB	FS	II	G L	II	F2 H2	FF	KS	B2 B1	af/	a/	bcm/bcm	_/_	_/	c/	_/_
A32	AB	FS	II	L L	II	D O	FF	KS	B1 B1	af/	a/	de/dk	_/_	a/	b/	a/
A33	AB	FS	II	S1 S1	II	D F2	FF	KK	B2 B1	af/c	a/	cegmn/dk	_/_	a/	_/	_/_
A34	AB	FF	II	L L	II	D D	FF	KK	B1 B1	af/	a/	bcm/bcm	_/_	a/b	abc/	_/_
A35	AA	FS	II	F L	II	D F2	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	_/	c/	_/_
A36	BB	FF	II	U Z	IS	F2 H2	FS	KK	B1 B1	af/	a/	dk/dk	_/_	_/	abc/	_/_
A37	AB	FS	II	L U	II	O O	FF	KS	B1 B1	af/	a/	dk/dk	_/_	_/	b/	a/
A38	AB	FS	II	F L	II	D F2	FF	KK	B2 B2	af/	a/	de/dk	_/_	_/	c/	a/
A39	AA	FS	II	L Z	II	F2 H2	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	_/	c/	_/_

continued overleaf

Table III. Bloodtypes of the Arabian horse sample.

A40	BB	FF	II	L L	II	F2 H2	FF	KK	B1 B1	af/	a/	de/dk	_/_	a/b	abc/	a/
A41	AB	FS	II	L U	II	F2 H2	FF	KK	B2 B1	af/b	a/	dk/dk	_/_	a/	abc/	_/_
A42	AA	SS	II	L L	II	F2 O	FF	KK	B1 B1	af/	a/	cegmn/dk	_/_	a/	_/	a/
A43	AB	FS	II	S1 U	II	D F2	FF	KK	B2 B2	af/	a/	de/dk	_/_	a/	b/	_/_
A44	AA	FF	II	F U	II	D O	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	_/	abc/	_/_
A45	AB	FS	II	F L	II	F2 O	FF	KK	B1 B1	af/	a/	de/dk	_/_	a/	abc/	_/_
A46	AA	FF	II	L S1	II	F2 H2	FF	KK	B1 B1	af/	a/	de/dk	_/_	_/	c/	a/
A47	AA	SS	II	L 2	II	F2 O	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	a/	c/	_/_
A48	AA	FS	II	L S1	II	F2 F2	FF	KK	B1 B1	af/	a/	cegmn/dk	_/_	_/	abc/	_/_
A49	AB	FS	II	L L	II	F2 F2	FF	KK	B1 B1	af/	a/	dk/dk	_/_	a/	abc/	_/_
A50	AA	FS	II	S1 U	II	F2 F2	FF	KK	B1 B1	af/	a/	dk/dk	_/_	a/	c/	_/_
A51	AA	FS	II	F L	II	F2 F2	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	a/	abc/	_/_
A52	BB	FF	II	L S1	II	F2 H2	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	_/	c/	_/_
A53	AB	FS	II	L U	II	F2 O	FF	KK	B1 B1	af/b	a/	de/dk	_/_	a/	b/	_/_
A54	AA	FS	II	L U	II	D H2	FF	KS	B1 B1	af/	a/	bcm/bcm	_/_	a/	b/	_/_
A55	AB	FS	FI	F F	II	D F2	FF	KK	B1 B1	af/	a/	dk/dk	_/_	a/	c/	_/_
A56	BB	SS	II	S1 2	II	D F2	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	a/	abc/	_/_
A57	AB	FS	II	F L	II	D F2	FF	KK	B2 B1	af/	a/	dk/dk	_/_	a/	c/	_/_
A58	AB	FS	II	L L	II	H2 H2	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	a/b	abc/	_/_
A59	BB	FF	II	F U	II	D F2	FF	KK	B2 B1	af/	a/	dk/dk	_/_	a/	abc/	_/_
A60	AB	SS	II	L U	II	H2 O	FF	KS	B2 B2	af/	a/	bcm/cegm	_/_	a/	abc/	_/_
A61	BB	SS	II	L S1	II	H2 H2	FF	KK	B2 B1	af/	a/	cgm/dk	_/_	a/	abc/	a/
A62	AA	SS	II	F U	II	D H2	FF	KK	B2 B1	af/	a/	cegmn/dk	_/_	a/	abc/	_/_
A63	AA	FS	II	N S1	II	D H2	FF	KK	B1 B1	af/	a/	de/dk	_/_	a/	abc/	_/_
A64	AB	FF	II	G L	II	D H2	FF	KK	B2 B1	af/	a/	dk/dk	_/_	_/	c/	_/_
A65	AA	FS	II	L L	II	F2 F2	FF	KS	B2 B1	af/	a/	cgm/de	_/_	a/	_/	_/_
A66	BB	FF	II	F L	II	F2 H2	FF	KK	B1 B1	af/	a/	cegmn/	_/_	a/	abc/	_/_
A67	BB	FF	II	U 2	II	F2 F2	FF	KK	B1 B1	af/	a/	bcm/de	_/_	a/b	c/	_/_
A68	AA	SS	II	L F	II	D F2	FF	SS	B1 B1	af/	a/	dk/dk	_/_	a/	abc/	a/
A69	AB	FS	II	N N	II	D F2	FF	KK	B1 B1	af/	a/	bcm/de	_/_	_/	abc/	a/
A70	AB	FS	II	N N	II	D O	FF	KK	A1 B1	af/b	a/	de/dk	_/_	a/	c/	a/
A71	AA	FS	II	F L	II	D F2	FF	KK	B1 B1	af/	a/	cegmn/dk	_/_	a/	abc/	_/_
A72	AA	FS	II	L S1	II	D D	FF	KK	B2 B1	af/	a/	cegmn/dk	_/_	a/	_/	a/
A73	AB	SS	II	L2 S1	II	D H2	FF	KK	B1 B1	af/	a/	cgm/dk	_/_	a/	abc/	_/_
A74	AB	FF	II	F U	II	D H2	FF	KS	B2 B1	af/	a/	bcm/de	_/_	a/	c/	_/_
A75	AB	SS	II	F L	II	F2 F2	FF	KS	B1 B1	af/	a/	dk/dk	_/_	a/	abc/	a/
A76	BB	SS	II	L 2	II	F2 F2	FF	KK	B1 B1	af/	a/	de/dk	_/_	_/	_/	_/_
A77	AB	FS	II	L S1	II	D F2	FF	KK	B2 B1	af/	a/	bcm/cgm	_/_	a/	abc/	a/
A78	AB	FS	II	L 2	II	F2 O	FF	KK	B1 B1	af/	a/	de/dk	_/_	a/	b/	_/_
A79	AA	FS	II	L U	II	F2 O	FF	KS	B1 B1	af/	a/	bcm/dk	_/_	a/	abc/	a/

continued overleaf

A80	AB	FS	II	L S2	II	F2 H2	FF	KK	B2 B1	af/	a/	de/dk	_/_	_/	b/	a/
A81	AB	FF	II	L N	II	D H2	FF	KK	B2 B1	af/	a/	de/dk	_/_	_/	c/	a/
A82	AA	SS	II	L L	II	H2 O	FF	KS	B2 B1	af/	a/	dk/dk	_/_	a/	b/	a/

Table II.iii Bloodtypes of the Arabian horse sample.

A83	AB	FS	II	L Z	II	F2 F2	FF	KK	B2 B1	_/	a/	cgm/dk	_/_	a/	c/	a/
A84	AA	FS	II	F F	II	D F2	FF	KS	B2 B2	af/	a/	cgm/dk	_/_	a/	c/	_/_
A85	AB	FS	II	L N	II	F2 O	FF	KK	B2 B1	af/b	a/	cgm/dk	_/_	a/	_/	a/
A86	AA	FF	II	L L	II	F2 H2	FF	KK	B1 B1	af/	a/	de/dk	_/_	a/	_/	_/_
A87	BB	FF	II	N N	FI	F1 F2	FF	KK	B2 B2	af/	_/_	dk/dk	_/_	_/	_/	_/_
A88	BB	FF	II	G L	II	D F2	FF	KK	B2 B1	af/	a/	dk/dk	_/_	_/	b/	_/_
A89	BB	FS	II	F L	II	F2 F2	FF	KK	B1 B1	af/	a/	dk/dk	_/_	a/	c/	_/_
A90	BB	FS	II	L S1	II	F2 F2	FF	KK	B2 B2	af/	a/	bcm/de	_/_	_/	c/	_/_
A91	AB	FF	II	L L	II	D O	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	a/	abc/	_/_
A92	AB	SS	II	L U	II	F2 H2	FF	KK	B1 B1	a(f)/	a/	bcm/cgm	_/_	_/	abc/	_/_
A93	BB	FF	II	G L	II	F2 O	FF	KK	B1 B1	af/	a/	dk/dk	_/_	_/	b/	_/_
A94	AB	SS	II	L S1	II	D O	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	_/	b/	a/
A95	AB	FF	II	L L	II	D F2	FF	KK	B2 B2	af/b	a/	dk/dk	_/_	_/	_/	a/
A96	AB	SS	II	S1 Z	II	D F2	FF	KK	B2 B1	af/	a/	de/dk	_/_	_/	b/c	a/
A97	BB	FF	II	G P	II	F2 F2	FF	KK	B2 B1	af/b	a/	de/dk	_/_	_/	abc/	_/_
A98	AA	FS	II	L L	II	D F2	FF	KK	A1 B1	af/	a/	bcm/dk	_/_	_/	abc/	_/_
A99	AA	FS	II	S1 Z	II	F2 F2	FF	KK	B2 B1	af/b	a/	de/dk	_/_	a/	abc/	_/_
A100	AB	FF	II	S1 Z	II	F2 H2	FF	KK	B1 B1	af/	a/	bcm/de	_/_	b/	_/	a/
A101	AA	SS	II	L U	II	H2 H2	FF	KK	B2 B1	af/	a/	de/dk	_/_	_/	c/	_/_
A102	AB	FS	II	S1 U	II	H2 H2	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	_/	b/c	_/_
A103	AA	FF	II	S1 S1	II	F2 O	FF	KK	B1 B1	af/	a/	dk/dk	_/_	a/	abc/	_/_
A104	AB	FF	II	L S1	II	D F2	FF	KK	B2 B1	b/	a/	cgm/dk	_/_	a/	abc/	a/
A105	AB	SS	II	L L	II	F2 O	FF	KS	B1 B1	af/	a/	de/dk	_/_	_/	abc/	_/_
A106	AB	SS	II	U Z	II	H2 O	FF	KK	B2 B1	af/	a/	dk/dk	_/_	a/b	b/	_/_
A107	BB	FS	II	U Z	II	F2 O	FF	KK	B2 B2	af/	a/	de/dk	_/_	_/	c/	_/_
A108	AB	FS	II	U U	II	H2 H2	FF	KK	B2 B2	af/	a/	dk/dk	_/_	_/	c/	_/_
A109	AB	SS	II	L L	II	H2 H2	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	_/	c/	_/_
A110	AB	FF	II	F L	II	F2 H2	FF	KK	B2 B1	ag/	a/	cgm/cgm	_/_	_/	b/	a/
A111	AB	FF	II	F U	II	F2 F2	FF	KK	B2 B1	af/	a/	bcm/dk	_/_	a/	abc/	a/
A112	AB	FS	II	S1 Z	II	F2 O	FF	KK	B2 B1	af/	a/	cgm/dk	_/_	a/	b/c	_/_
A113	AB	FS	II	F S1	II	F2 F2	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	_/	b/c	_/_
A114	AB	SS	II	U Z	II	O O	FF	KK	B2 B1	af/	a/	dk/dk	_/_	a/	abc/	_/_
A115	AB	FS	II	Z Z	II	D O	FF	KK	B1 B1	af/	a/	de/dk	_/_	a/	c/	_/_
A116	AB	FF	II	F L	II	F2 F2	FF	KK	B1 B1	af/	a/	cgm/dk	_/_	_/	c/	_/_
A117	AB	FF	II	L Z	II	F2 F2	FF	KK	B1 B1	af/	a/	bcm/dk	_/_	a/	abc/	_/_
A118	AB	FS	II	L S1	II	D F2	FF	KK	B1 B1	af/	a/	cgm/dk	_/_	a/	abc/	_/_
A119	AB	FF	II	S1 U	II	D F2	FF	KK	B1 B1	af/	a/	dk/dk	_/_	a/	_/	_/_

continued overleaf

A120	AA	FS	II	F F	II	D H2	FF	KK	B2 B1	af/	a/	cgm/dk	_/_	a/	c/	_/_
A121	AB	FS	II	L S1	II	D D	FF	KK	B2 B2	af/b	a/	cgm/de	_/_	a/	b/	_/_
A122	AB	FF	II	S1 U	II	D O	FF	KK	B2 B1	a/b	a/	de/dk	_/_	a/	b/c	_/_
A123	AA	FS	II	F L	II	F2 F2	FF	KK	B2 B1	af/	a/	cgm/dk	_/_	a/	abc/	_/_
A124	AB	FF	II	G N	II	F2 F2	FF	KK	B2 B1	af/	a/	cgm/dk	_/_	b/	c/	_/_
A125	AA	FS	II	F L	II	F2 F2	FF	KK	B2 B2	af/	a/	bcm/dk	_/_	_/	b/c	_/_

Table II.iii: Bloodtypes of the Arabian horse sample.

A126	AB	FS	II	II	F2	F2	FF	KK	B2	B1	af/	a/-	de/dk	_/_	a/_	c/_	_/_
A127	AB	SS	II	II	F2	H2	FF	KK	B2	B1	af/	a/	cegm/dk	_/_	a/	_/	_/_
A128	AB	FS	II	II	F2	O	FF	KS	B2	B1	af/	a/	cgm/dk	_/_	_/	c/	_/_
A129	BB	FS	II	II	F2	O	FF	KS	B2	B1	af/	a/	cgm/cgm	_/_	_/	c/	_/_

n.b., a blank cell indicates that a clear result was not obtainable.

Table II iii: Bloodtypes of the Arabian horse sample.

SAMPLE POPULATION: Standardbred Horses.

	Electrophoretic Results									Serological Results					
horse ID =	Al	PGD	PHI	Pi	Es	Tf	Gc	A1B	Hb	A	D	K	P	Q	U
S1	AA	FF	II	L L'	II	D R	FS	KK	B1 B2	ab	cfmr/dghmr				
S2	AB	FF	II	U U	IS	F2 F2	FF	KK	B1 B1	a	cgm/dfkr		a		a
S3	BB	FF	II	N N	IS	F2 O	FF	FK	B2 B2	ab	cgm/dghmr		a		a
S4	AB	FF	II	N U	II	F2 F2	FF	KK	B1 B1	a	cgm/dk	a	a		
S5	AB	FS	II	U U	II	D F2	FS	KK	B1 B2	a	cgm/dfkr				a
S6	AB	FF	II	I L2	II	F2 R	FF	KK	B1 B2	ab	cgm/cgm				
S7	AA	FF	II	N U	IH	F2 F2	FF	KK	B2 B2	b	dghmr/dk	a			
S8	AB	FF	II	U U	FI	F2 R	FF	KK	B1 B1	b	cgm/de		a		a
S9	AA	FF	II	U U	II	F2 F2	FF	KK	B1 B1	a	dghmr/dk	a	a		
S10	AA	FF	II	N U	II	D F2	FF	KK	B1 B2	a	cgm/cgm	a	a		a
S11	AB	FF	FI	L2 U	II	F2 F2	FF	KK	B1 B2	ab	cgm/dk				
S12	AA	FS	FI	L U	II	D F2	FF	KK	B1 B2	a	cgm/cgm	a	a	ab	a
S13	AB	FF	II	U U	GS	F2 R	FF	KK	B1 B2	a	cgm/dghmr	a	a		
S14	AB	FF	FI	L L2	FF	F2 F2	FF	KK	B1 B2	b	cgmp/cgm				
S15	AA	FF	FI	U U	II	F2 F2	FF	KK	B1 B1	ab	cgm/dfkr				a
S16	AB	FF	II	L2 U	IS	D F2	FF	KK	B1 B1	ab	cgm/dghmr				a
S17	AA	FF	FI	N U	II	F2 F2	FS	KK	B1 B1	ab	dk/dk				a
S18	AA	FF	II	N U	IS	F2 F2	FS	KK	B1 B2	ab	cgm/cgm				a
S19	AA	FF	II	U U	IS	F2 F2	FF	KK	B1 B1	a	adr/dghmr		a		a
S20	AA	FS	II	U U	II	F2 O	FS	KK	B2 B2	b	dk/dr	a			a
S21	AB	FF	II	U U	IS	F2 F2	FF	KK	B1 B2	ab	cgm/dk		a		
S22	AB	FS	FI	L L	IS	F2 R	FF	KK	B2 B2	a	cgm/cgm	a	a	ab	a
S23	AA	FF	II	U U	II	F2 F2	FF	KK	B1 B1	b	cfmr/de	a			a
S24	AA	FF	II	L U	IS	F2 F2	FF	KK	B1 B1	a	dghmr/dk		a		
S25	AB	FF	FI	U X	II	F2 R	FF	KK	B1 B2	ab	cfmr/cgm				
S26	AB	FS	FI	L2 U	FI	D F2	FF	KK	B1 B2	ab	cfmr/dfkr	a	a		a
S27	AB	FF	FI	U U	IS	F2 O	FS	KK	B2 B2	abf	cgm/cgm		a		
S28	AB	FF	FI	L N	II	D F2	FS	KK	B1 B2	ab	cgm/de		a		a
S29	AA	FF	II	I O	II	F2 F2	FS	KK	B2 B2	ab	cgm/dfkr		a		
S30	AB	FS	II	L U	II	D F2	FF	KK	B1 B2	a	cgm/cgm	a			
S31	BB	FF	II	L U	II	F2 R	FS	KK	B1 B2	ab	cgm/cgm				a
S32	AA	FF	II	U U	IS	O R	FF	KK	B1 B2	b	cfmr/de	a			a
S33	AB	FF	II	L2 N	II	F2 F2	FS	KK	B1 B1	ab	cfmr/cgm	a			
S34	BB	FF	II	U U	II	R R	FS	KK	B2 B2	ab	cfmr/dr		a		
S35	BB	FF	II	L2 U	IS	F2 F2	FF	KK	B1 B2	ab	dk/dk				
S36	AB	FF	II	L2 U	IS	F2 F2	FS	KK	B2 B2	ab	dk/dk	a			a
S37	AB	FF	II	U U	II	F2 R	FF	KK	B1 B2	ab	bcm/dghmr	a			a
S38	BB	FF	II	U U	II	F2 O	FF	KK	B1 B1	ab	cgm/dk		a		
S39	AB	FF	II	N U	II	D R	FS	KK	B1 B2	b	cfmr/dghmr	a	a		

continued overleaf

Table II.iv: Bloodtypes of the Standardbred Horse Sample Population.

S40	AA	FS	FI	L2 U	II	F2 F2	FF	KK	B2 B2	b	cgm/cgmp				
S41	AB	FF	II	L2 U	II	D F2	FF	KK	B2 B2	ab	cgm/dr				a
S42	AA	FF	II	L2 U	GI	D R	FF	KK	B1 B1	a	bcm/cgm	a			
S43	AB	FF	II	N N	II	D R	FF	KK	B1 B1	ab	cfmr/cgm				a
S44	AB	FS	II	U U	FI	D F2	FF	KK	B1 B2	ab	cgm/cgm				a
S45	AA	FS	II	I N	GI	F2 F2	FF	KK	B1 B2	b	dk/dk		a		
S46	AB	FF	II	N U	II	F2 O	FS	KK	B2 B2	b	cgm/cgm				
S47	AB	DF	II	L U	IS	F2 R	FF	KK	B2 B2	b	bcm/cfmr	a			a
S48	AB	FS	II	U X	II	D D	FF	KK	B1 B2	b	bcm/cgmp				a
S49	AB	FS	II	I L	IS	F2 F2	FF	KK	B1 B1	ab	dk/dk				a
S50	BB	FS	II	F L	II	R R	FS	KK	B2 B2	ab	cgm/dr	a			a
S51	BB	FF	II	N U	II	D O	FS	KK	B1 B2	b	cgm/cgm				
S52	AB	FS	II	N N	II	F2 F2	FS	KK	B1 B1	b	dk/dk		a		
S53	AA	FS	II	I L	GI	D F2	FF	KK	B1 B2	b	cfmr/dk		a		
S54	AB	FF	II	L U	II	F2 R	FF	KK	B2 B2	ab	bcm/dfkr			b	a
S55	AB	FF	FI	U U	GI	F2 R	FF	KK	B2 B2	b	cgm/cgm				a
S56	AB	FS	II	U U	GI	O R	FF	KK	B2 B2	a	dfkr/dk			ab	a
S57	AB	FF	II	N O	II	D R	FS	KK	B1 B1	ab	dk/de				a
S58	AA	FF	II	L U	II	F2 F2	FF	KK	B1 B2	ab	cgm/dk				
S59	AB	FF	II	L U	IS	F2 R	FF	KK	B1 B2	b	cgm/dk	a			a
S60	AB	FF	II	N U	II	F2 F2	FF	KK	B2 B2	b	bcm/de	a			
S61	AB	FF	II	L2 U	IS	D F2	FS	KK	B1 B2	ab	cgm/dk	a			
S62	AB	FS	II	N U	II	D F2	FS	KK	B1 B2	ab	cgm/dfkr	a			
S63	AA	FF	II	I N	II	D O	FF	KK	B1 B1	ab	cgm/dk	a			
S64	AB	FF	II	L2 L2	II	D F2	FS	KK	B1 B1	ab	cgm/dghmr	a			a
S65	AB	FF	II	L2 U	IS	F2 F2	FS	KK	B2 B2	b	bcm/dk	a			
S66	AB	FF	FI	F U	IS	O R	FS	KK	B2 B2	ab	cgm/de	a			
S67	AB	FF	II	I O	GS	D F2	FF	KK	B1 B1	ab	cfmr/cgmp				a
S68	AB	FS	II	I L2	GI	F2 F2	FF	KK	B2 B2	ab	cgm/dk				
S69	AA	FF	II	I U	II	F2 F2	FF	KK	B1 B2	ab	dk/dk				
S70	BB	FF	FI	H U	II	D F2	FF	KK	B1 B2	b	cgm/de	a			a
S71	AB	FS	II	F L2	II	F2 R	FS	KK	A1 B1	b	cgm/cgm	a			
S72	AA	FF	FI	L U	GI	F2 R	FF	KK	B1 B2	b	cgm/de				
S73	AB	FF	FF	L2 N	FI	D F2	FS	KK	B1 B2	ab	bcm/dfkr	a			
S74	AB	FS	FI	I U	SS	D F2	FF	KK	B2 B2	b	adr/cfmr		a		a
S75	AA	FF	FF	U U	IS	F2 R	FF	KK	B1 B2	b	cfmr/dr				
S76	AB	FF	II	L2 N	II	F2 R	FS	KK	B1 B2	b	cgm/dghmr	a			a
S77	AA	FF	II	L2 U	IS	R R	FF	KK	B2 B2	bc	cfmr/de	a			
S78	AB	FF	FI	N N	II	F2 R	FS	KK	B1 B1	ab	dghmr/dghmr	a			
S79	AB	FF	FI	I L2	FI	F2 O	FF	KK	B1 B2	b	cgm/de				
S80	AB	FF	FI	L2 L2	II	D F2	FS	KK	B2 B2	abf	cfmr/dr				
S81	BB	FF	II	N L2	IS	F2 R	FS	KK	B1 B2	a	cfmr/cgm				
S82	AB	FF	II	L U	II	F2 F2	FF	KK	B1 B1	b	cfmr/de	a			a

continued overleaf

Table II.iv: Bloodtypes of the Standardbred Horse Sample Population.

S83	BB	FS	II	L2 L2	GI	D F2	FF	KK	B1 B1	ab	dk/dk	a				
S84	AB	FF	II	U U	FI	R R	FF	KK	B1 B1	a	de/dfkr					a
S85	AB	FS	FI	I L	GG	D R	FF	KK	B1 B2	b	cgmp/dghmr	a				a
S86	AB	FF	II	N U	II	F2 O	FF	KK	B1 B2	ab	cgm/dfkr	a				
S87	BB	FF	FF	I L	FI	F2 R	SS	KK	B2 B2	b	cgm/cgm		a			
S88	AB	FF	II	N U	II	D F2	FS	KK	B2 B2	b	cgm/dk					
S89	AB	FF	II	L2 U	II	D R	FF	KK	B1 B1	a	dk/dk					
S90	AB	FF	II	U U	GI	D D	FF	KK	B1 B2	a	dk/de					a
S91	AB	FS	II	U U	IS	F2 F2	FS	KK	B2 B2	ab	bcm/dk	a		ab		a
S92	AB	FF	II	L2 N	II	D F2	FF	FK	B1 B2	ab	cgm/dk					
S93	AA	FF	II	L U	IS	D F2	FF	KK	B1 B2	ab	dfkr/dk	a				a
S94	AA	FS	FI	N U	IS	D F2	FF	KK	B1 B2	a	cgm/de					
S95	AB	FF	II	L2 U	IS	D R	FS	KK	B1 B2	a	cgm/cgm					
S96	BB	FF	II	N U	II	F2 F2	FF	FK	B2 B2	ab	cgm/dr		a			a
S97	AA	FF	FI	N N	IS	F2 F2	FS	KK	B2 B2	ab	dfkr/dk		a			
S98	BB	FS	FI	F L	II	F1 R	SS	KK	B1 B2	ab	cgm/cgm					
S99	AB	FF	II	L U	II	D R	FS	KK	B2 B2	b	cgm/de			b		
S100	AB	FS	II	F U	FI	F2 F2	FF	KK	B1 B2	a	cgm/cgm	a	a			
S101	AA	FS	FI	L2 U	FG	D F2	FS	KK	B1 B1	abf	cgmp/dghmr	a				
S102	AB	FF	II	L2 U	II	O R	FS	KK	B1 B2	abf	cgm/cgm					a
S103	AB	FF	FI	I U	FI	F2 F2	FF	KK	B1 B1	b	cgm/dk					a
S104	AA	FF	II	L U	II	F2 F2	FF	KK	B1 B1	ab	cfmr/cgm	a				
S105	BB	FF	FI	N U	II	D F2	FF	KK	B1 B2	b	cfmr/dr		a			
S106	AB	FF	II	L U	GI	F2 F2	FF	KK	B1 B1	b	cgm/cgm					a
S107	AB	FF	II	L L2	GG	D F2	FF	KK	B1 B2	ab	cgm/cgm					a
S108	BB	FF	FF	L N	II	F2 R	FS	KK	B2 B2	b	cfmr/cgm		a			
S109	AB	FF	II	L N	II	F2 R	FF	KK	B2 B2	b	cgm/dfkr					
S110	AB	FF	II	L N	II	D F2	FF	KK	B1 B2	ab	de/dk			b		
S111	AA	FF	FI	U U	SS	F2 F2	FS	KK	B1 B2	a	dk/de					
S112	AA	FS	II	L L2	II	D D	FS	KS	B1 B1	a	cgm/dghmr	a				
S113	AB	FF	II	N S	II	F2 F2	FF	KK	B2 B2	a	cgm/dfkr					a
S114	AA	FF	FI	N N	II	D D	FF	KK	B1 B2	a	adr/cgm	a	a			a
S115	AA	FF	II	N O	II	D D	FF	KK	B1 B2	ab	adr/dk	a	a			a
S116	AB	FS	II	U O	II	D F2	FF	KK	B1 B2	a	cfmr/dghmr					a
S117	AB	FF	II	G L	II	F2 F2	FS	KK	B1 B2	ab	cgmp/cgm		a			a
S118	AB	FS	FI	L2 L2	II	D R	FF	KK	B2 B2	a	adr/cgm	a				
S119	AB	FF	II	H L	IS	F2 F2	SS	KK	B1 B2	a	cgm/dfkr					
S120	AB	FF	II	N U	II	F2 F2	FF	KK	B1 B1	b	dk/dk					
S121	AA	FF	II	N N	II	D D	FF	KK	B1 B1	b	cfmr/dk	a				a
S122	AB	FF	II	U U	GI	F2 F2	FF	KK	B1 B1	ab	adr/cgm		a			
S123	AB	FF	II	L2 N	GI	F2 R	FS	KK	B1 B1	b	dk/dr					a
S124	AA	FF	II	L L2	II	D D	FF	KK	B1 B2	ab	cfmr/de					
S125	AB	FF	II	L N	GI	D R	FS	KK	B1 B1	ab	cgm/dk					

continued overleaf

Table II.iv: Bloodtypes of the Standardbred Horse Sample Population.

S126	BB	FF	FI	L S2	IS	D O	FF	KK	B1 B2	ab	cgm/cgmp	a		a
S127	AB	FS	II	L2 U	II	D F2	FF	KK	B1 B2	b	bcm/cgm	a		a
S128	AB	FS	FI	L2 U	GI	D F2	FF	KK	B1 B2	ab	cgm/dk	a		a
S129	AB	FF	II	L U	II	D F2	FF	KK	B1 B2	b	cgm/cgm	a		a
S130	AA	FF	FI	K U	II	F2 R	FS	KK	B2 B2	a	cgm/dghmr		a	
S131	AB	FF	II	N U	GI	D F2	FF	KK	B1 B2	a	cgm/dk		a	
S132	BB	FS	II	L U	GI	F2 R	FF	KK	B1 B1	b	cgm/cgm		a	
S133	AB	FS	II	U U	GI	F2 R	FF	KK	B1 B1	b	cfmr/cgm	a	a	a
S134	AA	FF	FI	L U	GI	F2 F2	FS	KK	B2 B2	b	cgm/cgmp			
S135	AB	FS	II	N U	II	D F2	FS	KK	B1 B2	ab	cgm/de	a		
S136	AA	FF	II	L U	II	D H2	FF	KK	B1 B1	b	cgm/dk			
S137	AB	FF	FI	I U	FI	D R	FS	KK	B1 B2	b	bcm/dfkr			a
S138	BB	FF	II	N U	GI	D F2	FF	KK	B1 B2	ab	cgm/cgm			a
S139	AA	FF	II	L2 N	II	D F2	FF	KK	B1 B1	ab	cfmr/cgm			
S140	AB	FF	II	U U	II	F2 R	FF	KK	B1 B2	ab	cgm/cgmp			a
S141	AA	FF	II	N O	II	D F2	FS	KK	B1 B2	a/-	cfmr/dr			a
S142	AB	FF	II	L L2	GI	F2 R	FS	KK	B1 B2	b	cgmp/de	a		
S143	AB	FF	II	L U	II	F2 F2	FF	KK	B2 B2	b	bcm/cgm	a		
S144	AB	FS	II	H X	II	D F2	FF	KK	B1 B2	b	bcm/de			
S145	BB	FF	II	L X	GI	D F2	FF	KK	B1 B1	a	bcm/dk	a		a
S146	AB	FF	FI	L U	GI	F2 F2	FS	KK	B1 B2	b/-	dk/de	a		a
S147	AB	FS	II	H L	SS	F2 F2	FF	FK	B1 B2	a	dk/dk	a		
S148	AB	FF	II	L2 U	GI	D F2	FF	FK	B2 B2		cgm/d			
S149	AA	FS	FI	L U	II	D F2	FF	KK	B1 B2	ab	dfkr/dk	a		a
S150	AB	FF	II	U U	II	F2 F2	FF	KK	B2 B2	a	cgm/cgm	a	a	
S151	AA	FF	II	N U	IS	D F2	FF	KK	B1 B2	a	dk/dk		a	
S152	BB	FF	II	N O	II	R R	FF	KK	B1 B2	ab	cgm/de			a
S153	AB	FF	II	U U	II	D F2	FF	KK	B2 B2	ab	cgm/de	a		a
S154	BB	FF	FI	U U	II	D F2	SS	KK	B2 B2	a/-	bcm/de	a		a
S155	AA	FF	FI	L2 U	GI	F2 F2	FS	KK	B1 B1	a	bcm/cgm			
S156	AB	FF	II	N U	II	D F2	FF	FK	B1 B2	b	cfmr/cgm	a		
S157	AB			V U	GI	F2 R	FS	KK		abf	bcm/dfkr	a	a	

Table II.iv: Bloodtypes of the Standardbred Horse Sample Population.

SAMPLE POPULATION: Station Hacks

	ELECTROPHORETIC RESULTS									SEROLOGICAL RESULTS						
horse ID #	Al	PGD	PHI	Pi	Es	Tf	Gc	A1B	Hb	A	C	D	K	P	Q	U
H1	AB	FF	II	L S2	II	H2 H2	FF	KK	A1 B1	af/	a/	cefgm/de	_/_	a/	b/	a/
H2	BB	FF	II	L L	FI	F1 H2	FF	KK	A1 B1	af/	a/	cegmn/	_/_	a/	abc/	a/
H3	AB	FF	II	L L	IS	H2 O	FF	KK	B2 B1	af/	a/	cefgm/de	_/_	_/	_/	a/
H4	BB	FF	II	N S2	II	D F2	FF	KK	B2 B2	af/	a/	cgm/dk	_/_	_/	abc/	_/
H5	AB	FS	II	L L	FI	D F2	FF	KK	B2 B2	af/	a/	bcm/cgm	_/_	a/	abc/	a/
H6	AA	FF	II	L L	II	H2 O	FF	KK	B2 B1	af/	a/	cgm/	_/_	a/	abc/	_/_
H7	BB	FF	II	L S1	II	D O	FF	FK	B2 B2	_/	a/	bcm/	_/_	_/	_/	_/_
H8	BB	FS	II	N U	FI	F2 F2	FF	KK	B2 B1	af/	a/	cgm/dk	_/_	_/	c/	a/
H9	AB	FF	II	S1 S2	II	D F2	FF	KK	B2 B1	af/	a/	cgm/dn	_/_	_/	abc/	a/
H10	AA	FF	II	JU	IS	F2 F2	FF	KK	B2 B2	af/	a/	dghm/dk	_/_	_/	_/	a/
H11	AA	FF	II	U Z	FF	D D	FF	KK	B2 B2	af/	a/	cgm/de	_/_	a/	bc/	_/_
H12	AB	FF	II	U Z	FF	D D	FF	KK	B1 B1	af/	a/	cgm/de	a/	a/	b/	_/_
H13	AA	FF	II	N N	FI	D D	FS	KK	B1 B1	af/c	a/	cgm/dk	_/_	a/b	_/	_/_
H14	AB	FS	II	N N	FI	D F2	FF	KK	B1 B1	bc/	a/	cegmn/dk	_/_	a/	c/	_/_
H15	AA	FF	II	F K	II	F2 O	FF	KK	B2 B1	af/b	_/_	cgm/dk	_/_	_/	c/	_/_
H16	AA	FS	II	F K	II	D O	FF	KK	B2 B2	af/	a/	cegmn/de	_/_	a/	abc/	_/_
H17	AA	FS	II	H S1	FI	F2 F2	FS	KK	B2 B2	af/	a/	cgm/dghm	_/_	a/	_/	_/_
H18	BB	DF	II	N S2	II	D F1	FF	KK	B1 B1	b/	a/	dk/	_/_	b/	_/	_/_
H19	AB	FS	II	L2 N	FI	D F2	FF	KK	B2 B2	af/b	a/	cgm/de	_/_	a/	c/	a/
H20	AA	FF	II	N S1	FF	D D	FF	KK	B2 B1	b/	a/	bcm/cgm	_/_	a/	c/	a/
H21	AB	FS	II	L2 N	FF	D D	FF	KK	B1 B1	af/b	a/	cegmn/dk	_/_	a/	_/	_/_
H22	AB	FS	II	F U	FI	D D	FF	KK	B2 B1	af/	a/	cegmn/dk	_/_	a/	c/	a/

Table II v. Blood types of the Station Hack sample population.

Appendix III.

Calculating additivity:

Note that: 1 = Thoroughbred Horses (T)

2 = Arabian Horses (A)

3 = Standardbred Horses (S)

4 = Shire Horses (Sh)

Kaimanawa Horses and Station Hacks are excluded from this because they are on the internal edge.

Checking the tree constructed from Rogers' genetic distance values (D_R):

$(d_{12} + d_{34}) = (d_{T\text{-}A} + d_{S\text{-}Sh})$

$= 0.234 + 0.352$

$= 0.586$

$(d_{13} + d_{24}) = (d_{T\text{-}S} + d_{A\text{-}Sh})$

$= 0.340 + 0.404$

$= 0.744$

$(d_{14} + d_{23}) = (d_{T\text{-}Sh} + d_{A\text{-}S})$

$= 0.410 + 0.334$

$= 0.744$

Since $0.586 < 0.744$, the distances can be considered to be additive, and therefore, Roger's genetic distance values (D_R), are to be considered suitable for the data.

Checking the tree constructed from Nei's standard genetic distance values (D_N):

$$(d_{12} + d_{34}) = (d_{T\text{-}A} + d_{S\text{-}Sh}) = 0.079 + 0.206 = 0.285$$

$$(d_{13} + d_{24}) = (d_{T\text{-}S} + d_{A\text{-}Sh}) = 0.168 + 0.231 = 0.399$$

$$(d_{14} + d_{23}) = (d_{T\text{-}Sh} + d_{A\text{-}S}) = 0.224 + 0.175 = 0.399$$

Since $0.285 < 0.399$, the distances can be considered to be additive, and therefore, Nei's standard genetic distance measure (D_N), is suitable for the data.

Checking the tree constructed from Nei's standard genetic distance values (D_N):

$$(d_{12} + d_{34}) = (d_{T\text{-}A} + d_{S\text{-}Sh}) = 0.079 + 0.206 = 0.285$$

$$(d_{13} + d_{24}) = (d_{T\text{-}S} + d_{A\text{-}Sh}) = 0.168 + 0.231 = 0.399$$

$$(d_{14} + d_{23}) = (d_{T\text{-}Sh} + d_{A\text{-}S}) = 0.224 + 0.175 = 0.399$$

Since $0.285 < 0.399$, the distances can be considered to be additive, and therefore, Nei's standard genetic distance measure (D_N), is suitable for the data.

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