

Genetics of Colour Variation

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Introduction

Variation in coat colour and spotting patterns of cattle have been of interest for many centuries, as indicated by the Lascaux cave drawings of cattle, which depict cattle with white-spotting patterns. In the 1800s, when some of the English breeds were being developed, breeders attempted to produce a reasonably uniform coat colour and spotting pattern within most breeds, as an aid to breed identity. The red colour and white spotting pattern of the Hereford breed is an example. In addition to the primarily aesthetic aspects of colour,

there is evidence that, under tropical conditions with high levels of solar radiation, animals with a light-coloured hair coat and darkly pigmented skin are better adapted (Finch and Western, 1977; Finch *et al.*, 1984). Most zebu breeds, which are well adapted to tropical conditions, have such a colour. The Italian breeds that have been imported into the USA, the Chianina, Marchigiana, Romagnola and Piemontese, also show this colour.

Several studies have indicated that the percentage of white on Holstein cows can have an impact on milk yield and reproductive traits in regions of high solar radiation. King *et al.* (1988) reported that cows with over 60% white that had calved in February and March required fewer services per conception and had fewer days open than cows with a higher percentage of black in their coats. Cows with a higher percentage of white showed a lowered depression in milk yield following exposure to intense solar radiation without shade (Hansen, 1990). The white cows also showed smaller alterations in physiological variables than did cows with less white. Becerril *et al.* (1994) reported a modest advantage in productivity of Holstein cows with greater quantities of white during the summer months in Florida. They also observed an advantage (not significant) for reproductive traits in these 'whiter' cows. Any advantage of the white hair coat is due to its lowered absorption of incident solar radiation (Stewart, 1953), which results in reduced heat stress.

Another association of a colour-related trait and an economically important trait is the relationship between eyelid pigmentation and the susceptibility to eye lesions, leading to 'cancer eye' in Hereford and other cattle breeds (Anderson, 1991). It was found that increasing eyelid pigmentation in Hereford cattle resulted in a decreased incidence of lesion development. In addition, several studies were cited by Brown *et al.* (1994) that reported an association between colour and horn fly counts on cattle, with greater numbers of flies being observed on darker than lighter cattle.

Changes in the breeding programmes of the US cattle industry in recent years have led to additional interest in the genetic control of coat colour in cattle. For example, the development of a composite breed of cattle in which it may be desired to fix a certain coat colour, such as solid red, from a foundation population that includes both black and spotted animals will require a knowledge of the inheritance patterns involved if it is to be easily achieved. Such information is also useful in the establishment of new colour and spotting patterns within established breeds, which allow upgrading (e.g. black, non-spotted Simmental). The existence of price discounts or premiums for feeder calves of various colours also leads to greater interest in colour genetics. For example, black or black, white-faced calves may receive a premium, regardless of actual breed composition; on the other hand, calves with zebu breeding that express grey or dark brown colours may receive a price dock, whereas black or red calves with the same zebu breeding may not suffer one.

The study of the genetic control of coat colour in cattle dates back over 90 years to the study of the inheritance of coat colour in Shorthorns and their crosses by Barrington and Pearson (1906). Reviews of the subject have been published periodically since then, including those of Ibsen (1933), Lauvergne

(1966), Searle (1968) and Olson and Willham (1982). This chapter will incorporate the results of recent observations and research to update the conclusions of Olson and Willham.

The Basis of Pigmentation in Cattle

The basis of coat colour in cattle and all mammals is the presence or absence of melanins in the hair (Searle, 1968). The melanin is found in the melanosomes of the cytoplasm of the melanocytes. These melanosomes are transferred to the hair as it grows through a process of exocytosis. The melanocytes migrate from the neural crest during embryonic development and only areas of the body in which they are found are pigmented. White spotting occurs in areas where the skin or hair lacks melanocytes. Pigmentation in all or part of the body may also be diminished by reduced activity of the melanocytes. Melanins of two types, eumelanin and pheomelanin, are found in mammals. Eumelanin is responsible for black and brown colours and pheomelanin for reddish brown, reds, tans and yellows. A more detailed discussion of the production of coat pigmentation can be found in Searle (1968) and the review of sheep colour genetics by Sponenberg (1997).

The Wild Type

To discuss most effectively the variation in coat colour and spotting patterns in any species, it is useful to explain the effects of mutants relative to the wild type. For spotting patterns in cattle, the wild type is simply a solidly pigmented animal or simply the lack of any spotting. Choice of a wild type for pigmentation is somewhat more difficult, but the colour of the aurochs of Europe, the wild ancestor of most (or all) *Bos taurus* breeds, seems appropriate (Olson, 1980). Aurochs were essentially reddish brown to brownish black with a tan muzzle ring. There was apparently some variation in the proportion of the body that was black and bulls were darker than cows. This coat colour or a similar one is occasionally observed in certain breeds today. Some Jersey, Brown Swiss and Longhorn purebreds, as well as crosses of these breeds with red breeds and some crosses of the Brahman with red breeds, produce the wild-type pigmentation pattern. Animals with wild-type coat colour tend to be darker at their extremities (head and neck, feet and hindquarters), similar to bay colour in the horse. Cattle with this type of brownish-black colour at maturity are born a reddish brown and darken when the calves shed out for the first time.

Adult bulls of several wild relatives of cattle, namely bison and banteng, have a similar dark brown coat colour. In banteng, adult cows are much lighter than bulls, having more of a tan colour, whereas, in the bison, cows are coloured like bulls.

Variations from the Wild-type Coat Colour

The most commonly observed variants from wild-type coat colour in cattle are red and solid black. Other coat colours of cattle are simply modifications of three basic colours: black, wild type (brown-black) and red. Most variations from these basic colours involve lightening or removal of pigmentation. Good examples are the light red colour of Limousin, the tan colour of many Jerseys and the almost complete removal of pigmentation of Chianina, some zebu and some Brown Swiss. Other mutant genes are responsible for the dilute colours of the Charolais and Simmental dilute pigmentation uniformly over the entire body, and for those found in Limousin, Jersey, Brown Swiss, Chianina (and the other white Italian breeds) and Brahman (and other zebu breeds), which tend to have differential effects on different parts of the body, especially the underline, the poll and along the back. Mutants thought to influence coat colour of cattle are shown in Table 3.1.

The Extension locus (black/red)

The *Extension* (*E*) locus is responsible for most of the variation in cattle coat colour. Three alleles present at this locus include: E^D , dominant black, E^+ , the wild-type allele responsible for most combinations of red or reddish brown and black; and e , recessive red. The order of dominance of these alleles is $E^D > E^+ > e$ and is complete. This locus has been identified as the *melanocyte-stimulating hormone (MSH) receptor* locus (Robbins *et al.*, 1993) and commercial companies have deoxyribonucleic acid (DNA) probes to identify whether phenotypically black animals carry E^+ or e . A point mutation of a single base substitution (thymine (T) for cytosine (C)) was found in the E^D allele, which resulted in an amino acid change from leucine to proline (Klungland *et al.*, 1995). A base deletion resulting in a frame shift has been found in the e allele (Klungland *et al.*, 1995; Joerg *et al.*, 1996).

Microsatellite analysis by Klungland *et al.* (1995) indicated that the *Extension* locus is located on chromosome 18. The *E* locus alleles regulate the level of tyrosinase production, which, in turn, determines whether eumelanin (black) or phaeomelanin (red) pigment is produced. Low levels of tyrosinase lead to the production of phaeomelanin, while high levels lead to eumelanin production. Tyrosinase levels are determined by the activity of the MSH receptor. If the E^D allele is present, an MSH receptor is produced that is active regardless of the activity of any alleles of the *Agouti* (*A*) locus, resulting in increased tyrosinase within the melanocytes and eumelanin production (Robbins *et al.*, 1993). The colour of animals with E^+ has been shown to be influenced by genes at the *Agouti* locus in other species. When the *wild-type* allele, A^+ , is present at the *Agouti* locus together with E^+ at the *Extension* locus, the combination of areas of black along with red pigmentation of the wild-type colour is produced. A low level of tyrosinase (resulting in red pigment

Table 3.1. Mutants influencing the colour of cattle.

Locus symbol	Locus name	Allele symbol	Allele name/description	Mode of inheritance relative to wild type	Breed distribution
<i>E</i>	<i>Extension</i>	<i>E^D</i>	<i>Dominant black</i> /uniformly black at birth	Dominant	Holstein, Angus, etc.
<i>E</i>	<i>Extension</i>	<i>E⁺</i>	Brown-black with darker extremities, bulls are darker than cows and calves are born a reddish brown (wild type)	—	Jersey, Brown Swiss, Brahman
<i>E</i>	<i>Extension</i>	<i>e</i>	<i>Red/red</i> without any dark pigmentation	Recessive to <i>E^D</i> and <i>E⁺</i>	Hereford, Red Angus, Guernsey, Simmental and other red breeds
<i>Br</i>	<i>Brindle</i>	<i>Br</i>	<i>Brindle</i> /alternating stripes black and red pigmentation	Dominant to lack of brindling	Most solid red and black breeds
<i>A</i>	<i>Agouti</i>	<i>A^{hp}</i>	<i>Patterned blackish</i> /a modifier of wild type that is similar to the wild-type pattern, but nearly entirely black and not influenced by sex	Dominant in the presence of <i>E⁺</i> , hypostatic to <i>E^D</i>	Holstein, Jersey, Brown Swiss, Brahman
<i>A</i>	<i>Agouti</i>	<i>a^w</i>	<i>White-bellied agouti</i> /removal of red pigment and a part of the black pigment while causing more uniform distribution of black pigmentation, especially across the sides of the animal	Recessive	Brown Swiss, Grey Steppe
<i>A</i>	<i>Agouti</i>	<i>a'</i>	<i>Fawn</i> /removal of red and black pigmentation, particularly red along the underline, along the back (dorsal strip) resulting in tan to fawn colour	Recessive	Limousin, Jersey, Brahman, Chianina, Grey Steppe
<i>D_c</i>	<i>Charolais dilution</i>	<i>Dc</i>	<i>Charolais dilution</i> /heterozygotes: strong dilution of black to light grey, red to light cream; homozygotes are white or nearly white	Nearly completely dominant	Charolais
<i>D_s</i>	<i>Simmental dilution</i>	<i>Ds</i>	<i>Simmental dilution</i> /heterozygotes: moderate dilution of black to light grey, red to light red; homozygotes are lighter	Incompletely dominant	Simmental, Scottish Highland, Murray Grey, some Gelbvieh
<i>D_n</i>	<i>Dun</i>	<i>Dn</i>	<i>Dun</i> /removal of red pigmentation with a reduced effect upon black pigment	Incompletely dominant	Brown Swiss, Brahman, Chianina

production only) is present in the melanocytes of individuals homozygous for e , probably as a result of non-functional MSH receptors in such animals (Klungland *et al.*, 1995).

The E^D allele is found in animals born solid black (with or without white spotting) and is responsible for the black colour of Angus and Holstein cattle. Some Texas Longhorns and a number of other breeds worldwide carry E^D . Animals with E^D do not change colour with age (at least not until advanced age, when greying about the face occurs in a small proportion of animals).

The E^+ , or *wild-type* allele, at the *Extension* locus produces a reddish brown with varying amounts of black. The black pigmentation may be restricted to the head and neck, feet, hindquarters and tail or may cover nearly the entire body, with only an area of reddish brown over the ribs, a tan dorsal stripe and a tan muzzle ring and poll. Bulls with wild-type colour are generally darker than cows. Breeds that possess E^+ are Jersey, Brown Swiss, Texas Longhorn and Brahman and other zebu breeds. It is also found in Holstein cattle, where the colour may be referred to as red-black to differentiate it from 'true' red, that produced by e/e . The E^+ gene was increased in frequency within the Holstein breed through use of a popular sire, Hanover-Hill Triple Threat-Red, which was E^+/e in genotype. Calves that are E^+ - (see below) in genotype are born red or reddish brown, perhaps with minimal black pigmentation on the feet and switch. There is complete dominance of E^D over both E^+ and e . Thus, it may be necessary to use DNA probes to differentiate if phenotypically black animals are heterozygous for either E^+ or e .

The red colour of Hereford, Simmental, Red Angus and other red breeds is due to homozygosity for the recessive gene, e . There is considerable variation in the intensity of red in cattle, from the dark red of Red Danish, Shorthorn and Maine-Anjou cattle to the lighter shades of some Herefords and Guernseys. While there may be a major (single) gene influencing the darker red colour and certainly there are single genes that lighten red colour, intensity of red is, in general, quantitative (Koger and Mankin, 1952). The desirable aspect of the red coat colour, from a genetic standpoint relative to composite breed development, is that, as a recessive, it will always 'breed true'. Exceptions would be the segregation of very light red or cream-coloured animals from some light red parents or wild-type animals from cattle that may appear red but on closer examination are brindles with minimal expression of black pigmentation.

The brindle coat colour is characterized by narrow alternating stripes of black and red arranged vertically on the entire body or confined to the head, neck and rear quarters. As mentioned above, some cattle may express the brindle colour only minimally, for example, only on the face. The base colour (the colour of the areas between the black stripes) may range from light red or fawn to dark brown or even nearly white, depending on the effects of alleles at other loci. Brindle coat colour is observed in the Texas Longhorn and Normande breeds, and is often produced in crossbreeding programmes, especially those including zebu breeds. The symbol Br will be used here to identify

the gene responsible for producing the brindle coat colour. The gene responsible for brindling, however, requires the wild-type coat colour (as produced by animals that are E^+/E^+ or E^+/e in genotype) to be expressed (Bjarnadottir, 1993, as cited by Adalsteinsson *et al.*, 1995). Therefore, brindle animals are $E^+ Br-$ in genotype, where the $-$ following the E^+ and Br indicates that the other allele at each locus may be either another copy of the dominant gene or its recessive counterpart.

The Br gene is apparently hypostatic to both the E^D and the e alleles. Since e/e animals have no black pigment, they cannot express the black stripes of the brindle pattern. Also, it appears that the brindle gene is unable to modify the expression of the black coat colour produced by E^D . As a result, $E^D Br-$ animals are black and $e/e Br-$ animals, red. The interaction of the *wild-type* coloration and Br is seen even in interspecific crosses, as bison \times Hereford crosses are often brindle in colour. An explanation for the variation in the quantity of brindling expressed on animals is that brindled areas are apparently restricted to areas that would have been dark brown to black had the animal not possessed Br . Certainly it is true that $E^+ br^+/br^+$ (wild-type or brown) animals show great variation in the amount of black pigmentation they express, from limited amounts on the head or head and neck to animals that may appear to possess an E^D allele as they are essentially solid black. In some adult animals, the only indication that wild-type animals do not possess E^D , that is that they are not true black, is that they may have a tan muzzle ring. Thus, it appears likely that brindling is only expressed in areas that would have been black in $E^+ br^+/br^+$ animals.

The brindle coat colour is produced in a high percentage of the offspring of the cross of Hereford or Red Angus with Brahman (or other white zebu breeds). The Jersey \times Hereford cross also nearly always produces brindle progeny. An explanation for these results appears to be that both the Hereford and Angus breeds carry the Br gene in quite high frequency, but do not express it as they are $E^D e/e$ in genotype. The reason that e/e animals do not express brindling is that they lack the black pigment, which is, in part, removed to produce the striped pattern of brindling. The other piece of this puzzle is that most grey/white zebu breeds, as well as the Jersey and Brown Swiss, carry the E^+ allele at a high frequency. The reason that the zebu breeds do not appear to express the wild-type (brown/black) pattern is that alleles at other loci (see discussions of the *Agouti* and *dun* loci) generally remove all of the red pigment present and most of the black pigment. The alleles that remove pigment in zebu breeds tend to be recessive and therefore are usually not expressed in the zebu \times Hereford or zebu \times Angus crosses. Thus, the F_1 animals from this cross ($E^+/e Br/br^+$ in genotype) express the wild-type coat colour and it is modified by Br to produce the brindle coat colour. Because of this epistatic relationship between the E and Br loci, the F_2 generation of a zebu \times Red Angus cross would be expected to produce a 9 : 3 : 4 ratio of brindle, brown (wild type) and red.

If it were desired to produce a composite breed of cattle based on a zebu \times Hereford/Angus base that was solid red (non-brindle), the E^+ gene

would need to be eliminated from the population. This could be done in one generation of selection if only red F_2 animals were used as sires and dams of the F_3 generation. Care would need to be taken in the evaluation of these cattle, as some brindle animals show only a very limited expression of the pattern, being essentially red, and yet these animals would carry E^+ . The heads, necks and rear quarters of all apparently red animals would need to be carefully examined for brindle striping to ensure that all animals carrying E^+ were eliminated.

Influence of Agouti locus mutants

While various authors have discussed possible mutants at the A locus in cattle that are homologous to those found at the A locus of other species (Lauvergne, 1966; Searle, 1968; Olson and Willham, 1982; Adalsteinsson *et al.*, 1995), the A locus mutants in cattle remain incompletely understood. Mutants at the *Agouti* locus in other species have an impact on the expression of the wild-type pattern. It has been speculated that the lighter belly of the Limousin and Jersey cattle is due to a mutant at this locus (a^i) and that the modifications of the wild-type pattern observed in Brown Swiss, Brahman and Chianina breeds may be due to another allele (a^w). Adalsteinsson *et al.* (1995) have presented data that support the existence of a *recessive black* allele at the A locus, symbolized by a , which, when homozygous, modifies the wild-type coat colour to solid black. The wild-type allele of the A locus, A^+ , allows the expression of the combination of black and red pigmentation of the wild-type coat colour. Homozygosity for a does not have an impact on animals that are e/e or E^D- in genotype. Alleles at the *Agouti* locus with the same type of effect are found in mice, dogs and horses. The cattle that Adalsteinsson *et al.* studied were Icelandic cattle and there is little evidence to date that the a allele exists in other, more populous breeds. The a gene seems to be found only at low frequency even in the Icelandic cattle.

A modification of the wild-type pattern, extending black pigment and resulting in a nearly black animal, has been described in the literature and named 'patterned blackish' (Majeskie, 1970). This pattern of black coat colour does not seem to be affected by the sex of the animal and is sometimes seen in progeny of crosses of Jersey, Brown Swiss and Brahman with red breeds. The *patterned blackish* gene is probably responsible for the occasional nearly black Jersey cows and Brown Swiss cows with greater proportions of black pigmentation. Perhaps the proper symbol to use to describe the mutant responsible for patterned blackish is A^{pb} , given its apparent modifying of the wild-type pattern.

Red cattle with black ear tips have been observed in zebu cattle in the USA (Olson, 1975) and southern China (Chen *et al.*, 1994). It would seem plausible that such animals are produced by modifying the distribution of black pigment in E^+ animals, but no confirmation of this is possible at this time.

The dun locus

The genes that are responsible for the coat colours of Jersey, Brown Swiss, Brahman (and other grey or white zebu breeds), Grey Steppe and Chianina (and similar-coloured Italian breeds) are not well understood, due in part to the great variation in coat colour within these breeds. The coat colours of these breeds are related, as all involve a lightening of the basic coat pigmentation to a greater or lesser degree. In general, the pigmentation tends to be removed to a greater degree on the underline and red pigmentation is removed to a greater degree than black. The removal of red pigmentation is essentially complete in many Brown Swiss and most Brahman and Chianina cattle.

It is clear that these 'lightened' breeds carry similar colour mutants, based on the coat colours of crosses between them. Crosses between Jersey and Brahman resemble Jerseys and crosses between Brown Swiss and both Brahman and Chianina resemble Brown Swiss. Also, in Brown Swiss, grey Brahman, Chianina and crosses among these breeds, there is little to no red pigmentation expressed, indicating the presence of a gene that acts similarly to the 'Chinchilla' mutant, c^{cb} , in other mammalian species. The *dun* gene, which has been studied by S. Adalsteinsson (personal communication) and Berge (1949, 1961) seems to produce the effects produced by this proposed 'chinchilla-like' gene. Perhaps the symbol *dn*, for *dun*, is appropriate to identify this mutant. It seems to be incompletely recessive to its normal allele, Dn^+ . When heterozygous, there is some effect of *dn* on red pigment, but its effect on black pigmentation is minimal. The *A* locus (a^i and a^w) and *dun* mutants, at least when heterozygous, have little or no effect on animals carrying E^D , except for a slight lightening to brown on the poll and along the back. Homozygosity for both a^w and *dn* acting upon black produced by E^D may produce a dilute coat colour, similar to the dilute-coloured animals from crosses of Simmental with Angus or Holstein. The *dn* mutant is probably present in the Brown Swiss, Chianina, Brahman and similarly coloured breeds, in addition to the Scandinavian cattle discussed by Berge.

A hypothesis regarding these colours is that a recessive allele, which could be called *fawn* and be symbolized as a^f , is responsible for the lightened underline and overall lightening resulting in tan to light red coat colours in Limousin, Guernsey and Jersey cattle. The darker extremities of Jersey are due to E^+ , whereas Limousin is e/e , as are most Guernseys. Grey Brahman and Chianina also carry a^f in its homozygous state but, in addition, are homozygous for *dn*, which removes the rest of the red pigment. This results in silver-grey in the case of Brahman, which carries E^+ , and white in Chianina, which is e/e . Since the Chianina and some zebu breeds may not carry E^+ and as a result produce only red pigment, they may also often be nearly white. Jersey and Limousin, which retain some red pigmentation, are probably Dn^+/Dn^+ at the *dun* locus.

The usual coat colour of Brown Swiss differs from that of Brahman (grey) in that its grey pigmentation is more uniformly distributed across the body, except for the underline, and is not usually confined to the extremities. Whether this difference is caused by a different allele at the *A* locus (a^w) or an

independent dominant mutant is not known. For the purposes of this chapter, the coat colour of the Brown Swiss breed will be assumed to be due to homozygosity for a^w . Most US Brown Swiss also seem to be homozygous for dn acting together with a^w on a wild-type background coat colour. Cream-coloured animals, occasionally seen in crossbreeding programmes involving Brahman and Brown Swiss with Red Angus, may be the result of heterozygosity for both a^i or a^w and dn . Nearly white animals are sometimes produced by backcrosses of Brown Swiss or Brahman with their F₁ crosses with Angus. Such 3/4 animals are 'whiter' than their Brown Swiss or Brahman parents. They resemble Chianina cattle in coat colour. This is somewhat confusing, unless one considers the effect of Br on E^+ . The effect of the brindle gene is to remove black pigment and sometimes it removes nearly all the black pigment on an animal, with the brindle colour being shown only on the face. With the eumelanin restricted by Br and the red pigmentation removed by dn acting together with a^i or a^w , perhaps a nearly white animal can be produced.

The Albino locus

Recessive albinism has occurred periodically in cattle of a number of breeds, including the Brown Swiss (Petersen *et al.*, 1944). In some cases, the animals reported had no pigment in either the eyes or skin and so could be considered as being 'true albinos' and would appear to be c/c in genotype, the allelic symbolism for true albinos in rodents and other species. In other cases, such as the recent case of a Florida Cracker calf born of a dam-son mating, the calf involved seemed to have some residual pigmentation. This may have been the same type of albinism that was discussed by Cole *et al.* (1934) and referred to as a 'ghost pattern' in white cattle of Holstein origin. Lauvergne (1966) also described a similar coat colour in a black and white spotted German breed.

Partial albinism, which, in some cases, was associated with dwarfism, was reported in Hereford cattle by Hafez *et al.* (1958). Since dwarfism has not been associated with C locus mutants in other species, another locus may have been involved in the dwarf individuals. It appears likely that none of the C locus mutants involved in these cases have been maintained and certainly none are established in any current breeds. Thus, the C locus mutants are not indicated in Table 3.1.

Dilution mutants of the Charolais, Simmental, Gelbvieh and other breeds

There are at least two additional loci in cattle that result in a diluted coat colour. Nearly all Charolais cattle are homozygous for a dilution mutation that is nearly completely dominant in mode of inheritance. The Simmental,

Gelbvieh and some other breeds carry a second, less dominant dilution mutation at varying frequencies. Both of these mutations uniformly dilute pigment over the entire body. These dilutions affect black at least as much as they do red pigmentation.

The dilution mutation carried by the Charolais breed that is responsible for the white coat colour of Charolais is symbolized by *Dc*, for *Charolais dilution*. The mutant approaches complete dominance over its *wild-type* (non-diluting) allele, *dc*⁺. Most Charolais are homozygous for both *e* and *Dc*, with the red coat that would have been produced by the *e/e* genotype being modified to an essentially white one by *Dc/Dc*. Some US Charolais that have been upgraded from Angus may, however, carry *E*^D. Crosses (F₁) of Angus and Charolais are generally light grey, due to the effect of heterozygosity for *Dc* (*Dc/dc*⁺) acting upon black (*E*^D/*e*). Similarly, crosses of red cattle with Charolais are cream to light yellow. In recent years, there has been an interest in producing red Charolais which would not carry *Dc* and thus be *dc*⁺/*dc*⁺ in genotype. It is not surprising that such animals should be produced occasionally, as all animals are not homozygous for *Dc*. It is not clear what other breeds of cattle possess *Dc*; the Cacerreño breed of Spain, however, does appear to possess an identical coat colour to that of the Charolais.

A number of other breeds (Simmental, Gelbvieh, Scottish Highland, Texas Longhorn and others) carry a different dilution mutation, *Ds*, for *Simmental dilution*. This mutation is incompletely dominant to its wild-type allele, *ds*⁺. Thus, red animals heterozygous for *Ds* (i.e. *e/e Ds/ds*⁺) are light red and black animals (i.e. *E*^D- *Ds/ds*⁺) exhibit varying intensities of grey colour. The Australian breed, the Murrey Grey, appears to be homozygous for *Ds* acting upon a black background. Red (*e/e*) animals homozygous for *Ds* are light yellow and black (*E*^D-) animals homozygous for *Ds* are light grey, similar to black cattle heterozygous for *Dc*. None of the breeds listed above carry *Ds* at a high frequency like that of *Dc* in Charolais. It seems likely that its frequency has decreased in the Simmental breed in the USA, as it is now desirable to have Simmental sires that will not produce dilute calves in crosses with black cows. On the other hand, the frequency of *Ds* in Gelbvieh cattle seems to have increased in the USA, due to the influence of certain popular sires.

The presence of *Ds* in the heterozygous state in animals with black hair coats is always obvious. This is not the case with red animals and this fact may have increased the popularity of the black animals now found in Simmental, Gelbvieh and other European breeds that utilized upgrading programmes in their development in the USA. Through the use of a black Simmental bull on black cows, for example, a commercial cattle producer can be assured that no dilute progeny will be born.

The desire to avoid the production of dilute black cattle can be traced, at least in part, to the phenomenon of the so-called 'rat-tailed' calves. Occasionally, the cross of a Simmental (and some other breeds) with a black mate (often Angus, or Angus crossbred) will result in a very dark, charcoal-coloured calf. Such calves are generally darker than the dilute colour produced by *Ds*,

and apparently the rat-tailed condition is not related to the *Ds* dilution gene. Rat-tailed animals frequently show hair coats that are very dark, but still slightly diluted, and are also abnormal (short and curly) with the hair on the switch being reduced or completely absent. Because of the lack of switch, such animals are referred to as rat-tailed and are often severely discriminated against when such calves are sold as feeder calves. The price discounts are due to the perception that such animals have reduced cold tolerance, and there are reports of death losses in rat-tailed calves in Nebraska, when severe early winter results in extremely rapid temperature drops. Also, rat-tailed calves gain weight more slowly during the winter than calves of comparable genetic make-up with normal hair coats (R.R. Schalles and L.V. Cundiff, personal communication). The bizarre situation with regard to rat-tailed animals is that, when such animals show white spotting, the hair in the white areas is completely normal. It should also be emphasized that the abnormal hair coat is never observed in red animals.

While the mechanism of inheritance of the rat-tailed condition is not completely clear, a procedure to reduce its incidence is available. Since the condition is apparently always expressed in animals with black hair coats, black Simmental do not carry the gene/genes responsible for the condition. Therefore, the use of only black Simmental, or red Simmental that are the progeny of black parents, should eliminate the possibility of producing rat-tailed (or normal-haired dilute) progeny when subsequent crosses are made with black mates. Another approach would be the use of test matings of red sires to homozygous black cows. Bulls that produce seven or more black calves (no rat-tails or dilutes) have less than a 1% chance of carrying the gene/genes responsible for the rat-tailed condition or *Ds*. After several generations of use of bulls tested in this fashion, the gene frequencies of *Ds* and of whatever genes are responsible for rat-tailed calves would be reduced substantially.

In addition to the colours that have been mentioned, other colour patterns caused by additional mutants surely exist in cattle but have not been well documented to date. A partial list of such colours includes black ear rims and black on the feet of basically red zebu and zebu crossbred cattle, irregular spots of light red pigment on a basically dark red or brown zebu (generally found in animals with Gyr breeding), and a type of red in Holsteins that appears not to be recessive in mode of inheritance.

White-spotting Mutants

Since the wild type for white spotting is a lack of spotting, any white spotting on cattle is due to a mutant or combination of several mutants (Olson, 1980). In general, the understanding of the genetic control of white spotting is complete, except for a few, infrequently observed patterns to be discussed later. Major mutant genes affecting spotting patterns in cattle are listed in Table 3.2.

Table 3.2. Common white-spotting mutants in cattle.

Locus symbol	Locus name	Allele symbol	Mutant name/description	Inheritance relative to wild type	Breed distribution
S	<i>Spotting</i>	S^H	<i>Hereford pattern</i> /white face, belly, feet and tail, often with white stripe over shoulders when homozygous. Only white face is present in S^H/S^+	Incompletely dominant	Hereford, Braford, Beefmaster
S	<i>Spotting</i>	S^P	<i>Pinzgauer pattern</i> /sides of body pigmented; variable amounts of white appear along dorsal and ventral areas extending forward from tail and rump	Incompletely dominant	Pinzgauer, Charolais, Longhorn, Florida Cracker
S	<i>Spotting</i>	s	<i>Recessive spotting</i> /piebald: irregular areas of pigmented and white; feet, belly and tail usually white	Recessive	Holstein, Guernsey, Jersey, Simmental, Ayrshire, Maine-Anjou, Belgian Blue and others
R	<i>Roan</i>	R	<i>Roan</i> /homozygote: nearly white except for small amounts of pigmentation on the edges of the ears; heterozygote: a combination of pigmented and white hairs	Incompletely dominant	Shorthorn, Belgian Blue
Bt	<i>Belted</i>	Bt	<i>Belted</i> /belt of white of various widths around paunch	Dominant	Dutch Belted, Galloway
Bl	<i>Blaze</i>	Bl	<i>Blaze</i> /white head, often a blaze when heterozygous, without associated white areas on other parts of body produced by Hereford pattern	Incompletely dominant	Simmental, Holstein(?), Groningen
Bc	<i>Brockling</i>	Bc	<i>Brockling</i> /areas of pigmentation within areas of white spotting produced by other mutants	Dominant	Nearly all solid-coloured breeds plus the Shorthorn, Ayrshire and Normande
Cs	<i>Colour-sided</i>	Cs	<i>Colour-sided</i> / homozygote: white body with pigmented ears, muzzle and feet (white park pattern); heterozygote: colour-sided pattern, white dorsal stripe with irregular edges (roaned) and white roaning on head; roaning may be confined to head, rump and tail	Incompletely dominant	Texas Longhorn, White Park, British White, Florida Cracker, English Longhorn and Belgian Blue

The S locus mutants

Much of the variation in white spotting among US breeds of cattle is due to a multiple allelic series at the *S* locus (Olson, 1981). In mice, this locus is referred to as *piebald* and has been documented to affect the differentiation of melanocytes at the neural crest, as well as their migration from the neural crest to the rest of the body (Jackson, 1994). The action of the *S* locus mutants is to produce mice with various types of white spotting patterns in combination with any pigmentation. The *S* locus in cattle contains at least three mutants, in addition to the wild-type, non-spotting allele, S^+ . These mutants are S^H , which is responsible for the Hereford pattern when homozygous, S^P , which is responsible for the Pinzgauer-type line-backed pattern (sometimes referred to as the Gloucester pattern, after the rare English breed), and s , *recessive spotting*, responsible for the irregular white spotting of the Holstein, Guernsey, Ayrshire, Jersey and Simmental breeds.

The order of dominance at the *S* locus is $S^H = S^P > S^+ > s$. The mutants, S^H and S^P , could be considered as codominant, as animals of genotype S^H/S^P , such as Pinzgauer \times Hereford crossbreds, express both white face due to S^H and a white dorsal stripe and white across the underline due to S^P . Both S^H and S^P are incompletely dominant to S^+ . Animals that are S^H/S^+ , such as Angus \times Hereford crossbreds, express a restricted Hereford pattern, in that they have less white on the head than S^H/S^H and generally have little or no white on other parts of the body, except on the switch and underline. Likewise, animals of genotype S^P/S^+ , such as Pinzgauer \times Angus crossbreds, generally have much less white than animals with genotype S^P/S^P . The white on S^P/S^+ animals resulting from Angus crosses can be restricted to a small amount on the tail or on the tail head extending along the spine across the rump. In other cattle, heterozygous animals may possess the full Pinzgauer pattern. The Charolais breed also possesses S^P in low frequency. Spotting patterns produced by S^P can be seen in animals with Charolais breeding but lacking the *Dc* (dilution) gene. Texas Longhorns and the related Florida Cracker cattle also possess S^P (Fig. 3.1).

Recessive spotting, s , is completely recessive to S^H , S^P and S^+ . Matings between animals with perfect Hereford markings have produced spotted (s/s) progeny (Franke *et al.*, 1975). Similarly, Angus (non-spotted) bred to Holstein have produced spotted calves, indicating that some Angus were S^+/s but did not show excessive white, due to dominance of S^+ over s . The amount of white on animals that are s/s varies considerably. Some Holstein cattle are 90–95% white, whereas others are 90–95% black. Such differences are due to highly heritable (~ 0.9), quantitative, modifying factors (Briquet and Lush, 1947; Becerril *et al.*, 1993). Modifying genes that are quantitative also influence the degree of expression of all other white-spotting patterns. For example, the amount of white on S^P/S^P or S^P/S^+ animals may be increased from that usually observed in the Pinzgauer to cover nearly the entire posterior and part of the anterior half of the body, resulting in pigmentation only on the feet, head, sides of the neck and shoulders. Some Texas Longhorn and Florida Cracker



Fig. 3.1. A Florida Cracker cow with the white-spotting pattern produced by the $S^P/-$ genotype and her nearly white calf, which demonstrates the pattern often produced by the genotype $S^P/-Cs/cs^+$.

cattle display such a spotting pattern. Other Longhorn and Cracker cattle that are S^P/S^+ in genotype may only express a limited amount of white speckling on the rear quarters. Apparently, there has been selection within the Pinzgauer breed for the degree of expression of S^P seen in most Pinzgauer cattle. Similarly, the amount of white could be increased or decreased on Herefords if breed standards of acceptable amounts and locations of white would allow them. The s gene may also exist in yaks, as some yak \times cattle crosses show a spotting pattern similar to that produced by s/s animals.

Blaze mutants

The Simmental breed and perhaps a few Holsteins carry a gene that produces a white spotting pattern on the face that is distinct from S^H . The symbol used for this gene is Bl , for the blaze pattern it usually produces when heterozygous and in combination with S^+ . Since full-blood (of 100% European origin) Simmental cattle are all spotted and must be s/s in genotype, the white facial spotting of the breed must be due to a gene at a locus independent of S . The genotype (for white spotting) of many Simmental \times Angus crosses is $Bl/bl^+ S^+/s$. Such animals are solid-coloured with a white blaze on their face that usually does not include the eyes. In combination with s/s , both Bl/Bl and Bl/bl^+ will usually have a solid white face and head. Photographs of the Gronigen breed of Holland indicate that the white facial spotting of this breed may also be due to Bl .

Roan

Shorthorns, Belgian Blues, Texas Longhorns and Florida Crackers carry a gene, *R*, responsible, when heterozygous, for roan colour. The allele to *R* is *r*⁺, a normal or wild-type gene that does not restrict pigmentation. Roan colour is a mixture of pigmented and white hairs and is produced in animals that are heterozygous, *R/r*⁺. When homozygous for *R*, an almost white animal is produced, with the exception of pigmentation of the hair within the ears. While the most often observed roan is a red roan, as found in the Shorthorn breed, the *Roan* gene acts equally effectively in the removal of any pigment. The roan colour is frequently used as an example of incomplete dominance in genetic textbooks, with the three phenotypes involved being identified as white, roan and red. This leads to the misconception that there are red and 'white' genes that are allelic. Obviously, what is ignored in these texts is that all Shorthorn cattle are *e/e* in genotype and, in this genetic background, *R/R*, *R/r*⁺ and *r*⁺/*r*⁺ animals are white, roan and red, respectively. When an animal is heterozygous for both the *E* and *R* loci, as in the cross of a white Shorthorn with an Angus, a 'blue roan', *E*^D/*e R/r*⁺ is produced. Ibsen (1933) cited the results of Evvard *et al.* (1930), who reported the segregation of colours and roaning patterns from F₂ progeny of blue roan F₁ parents that produced 11 blue roans, one black, three whites (with black points, i.e. black hairs in the ears), one red roan and five reds. These results demonstrate the independence of the *roan* and *E* loci. Just as roaning can occur in both black and red animals, brindle roans, dilute black roans and many other colours in combination with roan could be produced. The expression of the *roan* gene when heterozygous is highly variable, with some animals being roan over the entire body, while, in others, roaning may be restricted to just the centre of the forehead. Recent studies have confirmed the independence of the *roan* and *Extension* (located on chromosome 18 (Klungland *et al.*, 1995)) loci, with the *roan* locus having been determined to be located on chromosome 5 (Charlier *et al.*, 1996).

The white colour caused by homozygosity for *R* has been associated with 'White heifer disease' in the Shorthorn and Belgian Blue breeds. Hanset (1969) has determined that the reproductive problems associated with the white coat colour have both multifactorial genetic and environmental causes. The *R* allele appears to act in conjunction with auxiliary genes to produce the reproductive problems associated with white heifer disease. Systematic selection against the auxiliary genes in the Belgian Blue breed seems to have been effective in lowering the incidence of reproductive problems in these cattle (Hanset, 1969).

Colour-sided

Texas Longhorn, Florida Cracker, English Longhorn, some Scandinavian cattle and apparently some African breeds possess what has been called the colour-sided pattern, the gene for which is symbolized as *Cs* (Wriedt, 1925). What appears to be the colour-sided pattern is also found in yaks. The *Cs* gene is

partially dominant and continues to be expressed in Florida commercial cattle after many generations of crossing with non-spotted breeds. Animals carrying Cs in the heterozygous state show extreme variation in its expression. A pattern commonly seen in animals heterozygous for Cs includes a very irregular white strip along the dorsal and ventral parts of the animal, with roan areas along the edges and a roan or 'dappled' pattern of white on the head (Fig. 3.2). In other heterozygotes, the white stripe may be restricted to the rump and tail along with a little roaning on the head. The spotting patterns produced by animals heterozygous for Cs may be differentiated from those produced by S^p in that the spotting produced by Cs generally has a ragged or roan-like edge, whereas the edges of spots produced by S^p are clearly defined. The nearly white colour of other breeds, such as the British White and American White Park, is also apparently due to the action of Cs . Homozygotes for Cs generally exhibit the 'white park' pattern, that is, a nearly solid white animal with pigmented ears and a pigmented muzzle and often with some pigmentation just above the feet. It appears that the Blanco Orejinegro (translates as white with black ears) breed of Colombia owes its pattern to homozygosity for Cs . It also appears, based on limited data, that a 'white park' pattern may be able to be produced in cattle that are heterozygous for Cs in some breeds, such as the White Galloway and American White Park cattle. Other heterozygotes for Cs may appear similar to 'blue roans' in colour if they also are E^{D-} . Charlier *et al.* (1996) report that the *colour-sided* allele also appears to be present in low frequency in the Belgian Blue breed.



Fig. 3.2. A Florida Cracker cow with the colour-sided pattern produced in animals with the Cs/cs^+ genotype.

It has been observed in Florida Cracker cattle that animals which carry both R and C_s but cannot be homozygous for either are white park in phenotype. Allelism between R and C_s has been suggested; however, Florida Cracker cows that are white park in phenotype (due to carrying both R and C_s) have produced calves without any white spotting. This would indicate a lack of allelism of these two genes. Animals with both C_s and S^p may also show a predominantly white coat. The Florida Cracker calf show in Fig. 3.1 is of the type frequently observed in animals that possess both C_s and S^p . Similarly, Shorthorn \times Hereford crosses that possess both S^h and R may be predominantly white. Thus, it appears that there is an additive effect involved in the reduction of pigmentation in animals heterozygous for several spotting loci.

Belted

One of the most striking white-spotting mutants, Bt , produces the belted pattern of the Dutch Belted and Belted Galloway breeds. Belting is dominant and expresses itself with a white belt of varying widths around the midsection. Crosses of belted cattle with Holsteins (s/s) produce belted animals resembling the belted parent. As is the case in most spotting patterns, modifying genes are responsible for influencing the width and uniformity of this belt. Allelism with other spotting mutants has not been documented.

Brockling/pigmented legs

A major gene, referred to by previous authors as the *brockling* gene (Olson, 1975) or 'pigmented legs' gene (Ibsen, 1933), Bc , interacts with apparently any white-spotting mutant, producing areas of pigmentation within areas that would be white had the Bc gene not been present. The most commonly observed expression of the *brockling* gene is in Hereford \times Angus crossbreds, where Bc from the Angus produces pigmented spots on the face (the brockled-face pattern), which otherwise would be white, due to S^h . In s/s animals, legs are usually white, but when an s/s animal carries Bc as well, legs are pigmented to varying degrees. Ayrshire cattle with white-spotted sides and legs which are pigmented are $s/s Bc-$ in genotype. Only a few spotted breeds appear to carry Bc . These include the Ayrshire, Jersey, Norwegian Red and Normande. In addition to the Angus, most other non-spotted breeds also carry Bc at a reasonably high frequency. The exception to this rule is the Brown Swiss (Olson, 1975). A desirable function of Bc in Hereford crossbreds carrying S^h is that it usually results in pigmented areas surrounding the eyes, which has been shown to reduce the likelihood of cancer eye (Anderson, 1991). The so-called red-eyed condition in Hereford and Simmental cattle is very probably due to a different gene(s), which may be dominant, but this has not been well documented.

Minimal white spotting

In some solid-coloured breeds, white spotting along the underline, especially in front of the navel, can disqualify an animal from registration. Such spotting may be due to the presence of *s*. In many cases, however, such spotting is not caused by *s* and it is unclear as to the genetic mechanism involved. Selection against animals with white areas on the underline should reduce the incidence of such spotting, but this reduces the selection intensity possible for traits related to productivity.

White-spotting patterns in African and zebu breeds

A number of striking spotting patterns can be observed in several of the African Sanga and zebu breeds. These include the Pakistani zebu breed, the Dhanni, which is predominantly white but with small black body spotting and with black ears and a black muzzle. The Landim cattle of Mozambique possess a similar pattern. It is possible that this pattern is produced by modification of the line-backed pattern produced by *S^P*. Many Nguni cattle of South Africa show similar spotting. The Bapedi cattle, which were developed from Nguni cattle, often show a type of mottled grey-black, which is not frequently observed in other breeds. The Lohani cattle of Pakistan have pronounced speckling throughout a black body. This spotting pattern is also observed in some American Red Brahman cattle and has been observed in Texas Longhorn cattle. The mode of inheritance of this pattern is somewhat unclear although it does occur in Red Brahman × Angus crosses. Another Pakistani breed, the Rojhan, appears to have the recessive spotting pattern (*s/s*). The American Brahman does carry *s* at a modest frequency. The cattle referred to as West African Shorthorns and some other African breeds also appear to possess recessive spotting but modified by *Bc*, resulting in pigmentation on the legs and other modifications to recessive spotting.

References

- Adalsteinsson, S., Bjarnadottir, S., Vage, D.I. and Jonmundsson, J.V. (1995) Brown coat colour produced by the loci Extension and Agouti. *Journal of Heredity* 86, 395–398.
- Anderson, D.E. (1991) Genetic study of eye cancer in cattle. *Journal of Heredity* 82, 21–26.
- Barrington, A. and Pearson, K. (1906) On the inheritance of coat color in cattle. I. Shorthorn crosses and pure Shorthorn. *Biometrika* 4, 427–437.
- Becerril, C.M., Wilcox, C.J., Wiggins, G.R. and Sigmon, K.N. (1994) Transformation of measurements of percentage of white coat colour for Holsteins and estimation of heritability. *Journal of Dairy Science* 77, 2651–2657.

- Becerril, C.M., Wilcox, C.J., Lawlor, T.J., Wiggins, G.R. and Webb, D.W. (1993) Effects of percentage of white coat colour on Holstein production and reproduction in a subtropical environment. *Journal of Dairy Science* 76, 2286–2291.
- Berge, S. (1949) Inheritance of dun, brown and brindle colour in cattle. *Heredity* 3, 195.
- Berge, S. (1961) Influence of dun on brown and brindle cattle. *Zeitschrift für Tierzüchtung und Züchtungsbiologie* 75, 298.
- Briquet, R., Jr and Lush, J.L. (1947) Heritability of amount of white spotting in Holstein-Friesian cattle. *Journal of Heredity* 38, 98.
- Brown, A.H., Jr, Johnson, Z.B., Simpson, R.B., Brown, M.A., Steelman, C.D. and Rosenkrans, C.F., Jr (1994) Relationship of horn fly to face fly infestation in beef cattle. *Journal of Animal Science* 72, 2264–2269.
- Charlier, C., Denys, B., Belanche, J.I., Coppeters, W., Grobet, L., Mni, M., Womack, J., Hanset, R. and Georges, M. (1996) Microsatellite mapping of the bovine roan locus: a major determinant of White Heifer Disease. *Mammalian Genome* 7, 138–142.
- Chen, Y., Wang, Y., Cao, H., Pang, Z. and Yang, G. (1994) Black-ear gene and blood polymorphism in four southern Chinese cattle groups. *Animal Genetics* 25 (Suppl. 1), 89–90.
- Cole, L.J., Van Lone, E.E. and Johannsson, I. (1934) Albinotic dilution of colour in cattle. *Journal of Heredity* 25, 145–156.
- Evvard, J.M., Shearer, P.S., Lindstrom, E.W. and Smith, A.D.B. (1930) The inheritance of colour and horns in blue gray cattle. II. *Iowa Agricultural Experiment Station Research Bulletin* 133, 1–16.
- Finch, V.A. and Western, D. (1977) Cattle colours in pastoral herds: natural selection or social preference. *Ecology* 58, 1384.
- Finch, V.A., Bennett, I.L. and Holmes, C.R. (1984) Coat colour in cattle: effect on thermal balance, behaviour and growth, and relationship with coat type. *Journal of Agricultural Science (Cambridge)* 102, 141.
- Franke, D.E., Burns, W.C. and Koger, M. (1975) Variation in the coat colour pattern of Hereford cattle. *Journal of Heredity* 66, 147–150.
- Hafez, E.S.E., O'Mary, C.C. and Ensminger, M.E. (1958) Albino-dwarfism in Hereford cattle. *Journal of Heredity* 49, 111–115.
- Hansen, P.J. (1990) Effects of coat colour on physiological responses to solar radiation in Holsteins. *Veterinary Record* 127, 333.
- Hanset, R. (1969) La White Heifer Disease dans la race bovine de Moyenne et Haute Belgique: un bilan de dix années. *Annales de Médecine Vétérinaire* 113, 12–21.
- Ibsen, H.L. (1933) Cattle inheritance. I. Colour. *Genetics* 18, 441–480.
- Jackson, I.J. (1994) Molecular and developmental genetics of mouse coat colour. *Annual Review of Genetics* 28, 189–217.
- Joerg, H., Fries, H.R., Meijerink, E. and Stranzinger, G.F. (1996) Red coat colour in Holstein cattle is associated with a deletion in the MSHR gene. *Mammalian Genome* 7, 317–318.
- King, V.L., Denise, S.K., Armstrong, D.V., Torabi, M. and Weirsmas, F. (1988) Effects of a hot climate on the performance of first lactation cows grouped by coat colour. *Journal of Dairy Science* 71, 1093–1096.
- Koger, M. and Mankin, J.D. (1952) Heritability of intensity of red colour in Hereford cattle. *Journal of Heredity* 43, 15.
- Klungland, H., Våge, D.I., Gomez-Raya, L., Adalsteinsson, S. and Lien, S. (1995) The role of melanocyte-stimulating hormone (MSH) receptor in bovine coat colour determination. *Mammalian Genome* 6, 636–639.

- Lauvergne, J.J. (1966) Génétique de la couleur de pelage des bovins domestiques. *Bibliographia Genetica* 20, 1–168.
- Majeskie, J.L. (1970) Characteristics and inheritance of certain coat colours and patterns in cattle. PhD thesis, Kansas State University, Manhattan, Kansas.
- Olson, T.A. (1975) An analysis of the inheritance of coat colour in cattle. MS thesis, Iowa State University, Ames.
- Olson, T.A. (1980) Choice of a wild-type standard in colour genetics of domestic cattle. *Journal of Heredity* 71, 442–444.
- Olson, T.A. (1981) The genetic basis for piebald patterns in cattle. *Journal of Heredity* 72, 113–116.
- Olson, T.A. and Willham, R.L. (1982) *Inheritance of Coat Colour in Cattle: a Review*. Iowa State University and Agricultural Experiment Station Bulletin 595, Ames, Iowa.
- Petersen, W.E., Gilmore, L.O., Fitch, J.B. and Winters, L.M. (1944) Albinism in cattle. *Journal of Heredity* 35, 135–144.
- Robbins, L.S., Nadeau, J.H., Johnson, K.R., Kelly, M.A., Roselli-Rehfuss, L., Baack, E., Mountjoy, K.G. and Cone, R.D. (1993) Pigmentation phenotypes of variant extension locus alleles result from point mutations that alter MSH receptor function. *Cell* 72, 827–834.
- Searle, A.G. (1968) *Comparative Genetics of Coat Colour in Mammals*. Logos Press, London.
- Sponenberg, D.P. (1997) The genetics of colour and hair texture. In: Piper, L. and Ruvinsky, A. (eds) *The Genetics of Sheep*. CAB International, Wallingford, pp. 51–86.
- Stewart, R.E. (1953) Absorption of solar radiation by the hair of cattle. *Agricultural Engineering* 34, 235.
- Wriedt, C. (1925) Colour-sided cattle. *Journal of Heredity* 16, 51–56.

