

# COAT COLOR INHERITANCE IN HORSES AND IN OTHER MAMMALS

W. E. CASTLE

*University of California, Berkeley*

Received April 30, 1953

THE colors of horses have long been a subject of interest to owners and breeders of horses as well as to scientists and much information about them is found in the stud-book records of particular breeds. Attempts to use this information in framing explanations of the observed results of matings within or between different color varieties have been made repeatedly since the rise of genetics at the beginning of this century.

Such explanations assume the existence in the primitive undomesticated horse of certain genes for color production which have persisted unchanged in certain domesticated color varieties and undergone mutation in others to produce the genetic combinations recognizable in present-day horses.

Most of the genetic explanations of horse colors have been based on a study solely of horses, ignoring the much larger and more exact knowledge of color inheritance which has been derived from experimental studies made on other mammals, rodents in particular.

A comparative study utilizing this experimental evidence in discussing the genetics of color in horses was made by WRIGHT (1917) and more recently by ODRIOZOLA (1951). Believing this to be a correct method of procedure, I shall attempt in what follows to present a genetic frame-work for explaining horse colors in harmony with that derived from the experimental study of other mammals.

## THE COLOR GENE AND ITS ALBINO MUTATIONS

The colors of mammals in general are derived from the presence in the coat of two different groups of pigments which we may call black-brown and red-yellow respectively. Both are end-products of a process of oxidation of a substance or substances capable of producing color when so oxidized.

The initial step in this process is controlled by a hypothetical color gene (symbol  $C$ ), which may by mutation become inoperative, completely or partially, resulting in albinism. In a complete albino (symbol  $c$ ), no pigment whatever is formed, so that the hair is white and skin and eyes are pink. Familiar examples of complete albinism are found in white rabbits, rats and mice. Historically the albinism found in white mice was the first mutation in an animal shown to be inherited as a Mendelian recessive character.

A form of incomplete albinism results from a different mutation in the color gene  $C$  to an allele designated  $c^H$  which when homozygous produces an albino of the sort seen in the Himalayan rabbit. Its body is white and its eyes pink, but the fur on the extremities (ears, nose, tail and feet) is feebly pigmented.

Even the white body coat is capable of developing pigment if kept at subnormal temperatures. This type of albinism, ( $c^H$ ) as well as complete albinism ( $c$ ) occurs in domestic rabbits. It occurs also in guinea-pigs and in mink as the only known type of albino in those species. The Siamese cat represents an albino of the Himalayan type, in which considerable sootiness is general in the whitish coat, although the pigmentation is much heavier at the extremities.

A third type of albino mutation, which like the other two is found in rabbits, occurs in the so-called chinchilla rabbit, its gene being designated  $c^{ch}$ . It permits pigment to develop in the entire coat, but in a much reduced amount. Experimental crosses show it to be an allele of  $C$ ,  $c^H$  and  $c$ , recessive to  $C$  but dominant over  $c^H$  and  $c$ . A zygote may contain any two alleles of the four, but not more. Thus  $Cc$  individuals bred together may produce only fully colored and completely albino young;  $Cc^H$  individuals may produce only fully colored and Himalayan albino young;  $Cc^{ch}$  parents may produce only full colored and chinchilla young;  $c^Hc$  parents produce only Himalayan and complete albinos;  $c^{ch}c^H$  parents may produce only chinchilla and Himalayan young; and  $c^{ch}c$  parents may produce only chinchilla and complete albino young.

There is in horses no certainly demonstrated albino mutation of the color gene, although white horses of at least two different genotypes are commonly known as albinos. These will be discussed later. ODRIOZOLA alone has suggested that one of these is a true albino mutation.

#### THE BLACK GENE

The coat of most mammals in the wild state is predominantly black pigmented. In the production of black pigment, which is found in the eyes and in the skin of the extremities as well as in the coat, a hypothetical gene  $B$  (black) acts in conjunction with gene  $C$ . In the absence of any genes which regulate the distribution of black pigment, it will be found throughout the coat.

As a matter of fact very few wild mammals are uniform black in color. This is because of the action of other genes which restrict the distribution of black to particular areas of the coat. When under such gene action black ceases to be produced in a particular area, it is automatically replaced there by red-yellow, a different end product of the oxidation process which occurs in cooperation with gene  $C$ . As a result the coat becomes a mosaic of black and yellow pigments which has concealing value in the struggle for existence, allowing a predator to steal upon its victims unobserved, or the prospective victim to escape the notice of the predator.

#### THE WILD PATTERN GENE

A concealing coat pattern of this sort is the agouti pattern of rabbits and other rodents, which has its counterpart in wolves and many other groups of mammals. Its production is controlled by a gene  $A$  (agouti), acting in conjunction with  $C$  and  $B$ . We may regard this as a gene for wild-coat pattern.

It limits the production of black pigment (1) to particular parts of the coat, chiefly dorsal and peripheral, and (2) also to particular parts of the individual hairs. The result is a generally gray coat dorsally with individual hairs having

an intensely black tip, a sub-apical band of yellow, and below that a dull black base. The ventral areas of the coat are lighter, often lacking the black hair tips, resulting in a whitish yellow belly color and tail white underneath as in the cotton-tail rabbit. This is a very effective concealing pattern in a natural environment. It results entirely from the action of the dominant gene  $A$  in cooperation with genes  $C$  and  $B$ .

Loss of the wild pattern of the coat may result from a recessive mutation of  $A$  to  $a$ , leaving the coat a uniform black in the genotype  $aaBC$ , which is known as non-agouti black or recessive black.

In rabbits and many other rodents, as well as in dogs, the wild-pattern gene  $A$  has undergone a mutation (designated  $a^t$ ) which results in the black-and-tan coat pattern. In this mutant, when homozygous ( $a^t a^t$ ), the individual hairs of the dorsal areas lack the yellow band and so the animal has a black back, a light belly, with banded hairs along the sides between back and belly. This mutation is dominant to black but recessive to ordinary agouti ( $A$ ).

Is there a wild-coat pattern gene in horses comparable with the  $A$  gene of rodents and other mammals? There certainly is. For the Prejvalski horse, supposedly sole surviving remnant of the wild ancestral stock from which domestic horses arose, is not uniform black in color but has a distinctly concealing pattern. The general body color is a neutral gray probably of mixed black and yellow pigments, but peripheral areas are of black alone including black mane and tail, black dorsal stripe, and black legs.

This pattern is completely lost in the uniform black variety of domestic horses, which we may assume to be of genotype  $aaBC$ , the ancestral genotype being  $ABC$ . A domestic variety which certainly retains the wild gene  $A$  is the bay. All investigators are in agreement that black ( $aaBC$ ) in horses is recessive to bay ( $ABC$ ) just as black in rodents is recessive to agouti.

ODRIZOLA thinks that the domestic bay retains the wild-pattern gene, not in its original form, but in an allele less effective in producing the wild pattern, just as in rodents  $a^t$  is less effective than  $A$  in producing the agouti pattern.

This may or may not be so. We have at present no experimental evidence upon it, such as a cross of a Prejvalski horse with a domestic bay might yield.

In rodents and many other mammals, a recessive mutation has occurred in gene  $B$  to an allele  $b$ , which when homozygous ( $bb$ ) replaces black pigment with brown throughout the body (eyes, skin and coat). When this happens, the black agouti phenotype becomes brown agouti known as cinnamon (genotype  $AbbC$ ). And the non-agouti or uniform black phenotype (genotype  $aaBC$ ) becomes chocolate in genotype  $aabbC$ .

A comparable recessive mutation in gene  $B$  has occurred in horses, giving rise to the chestnut color variety, which was the first demonstrated mendelizing character of horses, as shown by BATESON et al. All investigators are agreed that chestnut is recessive to bay. Their respective genotypes are, in the terminology here adopted,  $AbbC$ , chestnut; and  $ABC$ , bay. If we replace  $A$  in these formulae with its recessive allele  $a$ , we obtain genotypes  $aabbC$ , uniform brown, "liver"; and  $aaBC$ , uniform black, recessive black.

The wild-pattern  $A$  has been described as one which restricts the distribu-

tion of black pigment in certain portions of the coat or certain parts of individual hairs, where it is replaced by yellow. The result in rodents is the agouti phenotype with its demonstrated concealing character.

The Prejvalski horse also has a concealing coat pattern, as already stated. This pattern, which we assume to be due to the dominant gene *A*, is completely lost in the recessive mutation uniform black, *aaBC*.

A feature of this ancestral pattern is the occurrence, on a prevailing dull ("dun") colored background, of a dark spinal stripe and bars on legs or withers. This feature, as stated by DARWIN, is often found in ponies. I have recently seen two examples of it among Shetland ponies on a California ranch. Its occurrence among feral horses on the pampas of Argentina is also a well-authenticated historical fact.

#### THE EXTENSION-RESTRICTION GENE PAIR, *E*, *e*

In the study of rodents another gene was discovered which further restricts the distribution of black pigment beyond the degree of restriction effected by gene *A*. This gene is designated *e*. It is the supposed recessive allele of a hypothetical dominant gene *E* governing *extension* of black pigment throughout the coat, except in so far as it may be limited by gene *A*.

This new gene when homozygous (*ee*) replaces black or brown pigment with red or yellow in the coat generally, although the eyes, skin of the extremities, and in a minor degree the fur of the extremities may remain black or brown. One investigator has suggested that this be called a *red* mutation, *e* standing for erythros, Greek for red, rather than for restricted distribution of black.

At any rate this mutation results in a generally red or yellow coat, in rabbits and guinea pigs, with the following genetic permutations and phenotypes being clearly distinguishable in rabbits, though in guinea-pigs the agouti and non-agouti yellow phenotypes are less distinct.

<i>ABE</i> , agouti	<i>AbbE</i> , cinnamon
<i>ABee</i> , yellow	<i>Abbee</i> , yellow (brown eyes)
<i>aaBE</i> , black	<i>aabbE</i> , chocolate
<i>aaBee</i> , sooty yellow	<i>aabbee</i> , cream (brown eyes)

The question now arises, has a similar recessive red mutation occurred in horses, restricting the distribution of black pigment in the coat beyond the degree of restriction effected by the *A* gene as seen in the Prejvalski horse, and *replacing* black with red in the coat generally?

I would answer this question with yes, pointing to the typical red bay horse as an example. Its general body color is red, not a mixture of black and yellow pigments as in the Prejvalski horse, but it retains the primitive *A* pattern gene as seen in its black mane, tail, and legs; its genotype then may be expressed thus, *ABCee*.

If we adopt this hypothesis, the genotypes of horses corresponding to those of rodents already tabulated will be:

<i>ABE</i> , Prejvalski and ordinary bay	<i>AbbE</i> , chestnut
<i>ABee</i> , red-bodied bay	<i>Abbee</i> , sorrel (light mane)
<i>aaBE</i> , black	<i>aabbE</i> , liver
<i>aaBee</i> , sooty black, "mouse"	<i>abbee</i> , sorrel (uniform color)

## DOMINANT BLACK

In several mammals a dominant mutation of gene *E* to an allele  $E^D$  results in a greatly increased production of black pigment and its extended distribution throughout the coat so that it obscures the wild pattern even when heterozygous ( $EE^D$ ) and completely hides it when homozygous ( $E^DE^D$ ). This mutation is known as *dominant black*. By contrast *recessive black* lacks the wild-pattern allele *A* and so is of genotype *aaBE*.

This mutation was first observed and has been most carefully studied in rabbits by PUNNETT but has since been shown to occur also in dogs, cats and in the black rat (*Rattus rattus*).

A cross between dominant and recessive black varieties, neither of which shows wild-coat pattern, though one of them carries it hidden by the  $E^D$  allele, may result in genotypes:

- aaBEE*, recessive black
- ABEE*, agouti
- ABEE<sup>D</sup>*, agouti-black, showing traces only of agouti
- ABE<sup>D</sup>E<sup>D</sup>*, dominant black, agouti pattern wholly concealed

There is reason to think that in horses, as well as in some other mammals, both dominant and recessive varieties of black occur, and that crosses between them may result occasionally in the unexpected production of a bay colt, an apparent reversal of dominance, but really due to an  $E^D$  allele which conceals the *A* pattern more or less completely. Dominant black is probably the commonest type of black found among Shetland ponies, and this may account for the increasing scarcity of the bay color variety among Shetlands, but demonstrative evidence of this interpretation is as yet lacking.

## WHITE IN HORSE COLORS

There are horses of many different genotypes which have white or whitish hair on part or all of their bodies.

Among mammals in general the commonest sort of white individual is the albino, completely devoid of pigment in its coat, or nearly so. It arises by a recessive mutation in the color gene *C*, and like all recessives is true-breeding when homozygous.

No true albino mutation has been found among horses, although ODRIOZOLA considers the dilution gene found in Palomino and buckskin horses to belong in this category. Against this view it may be urged that Palomino dilution is not due to a recessive allele of the color gene, but to a dominant modifier of its action, which leaves its latent power unchanged. For when Palomino is mated with Palomino, 25 percent of the colts produced are fully colored chestnut.

*Dominant white*

The best known type of white horse is one which has a completely white coat but colored eyes. It may be called dominant white, gene *W*. Recent studies of white horses of this sort indicate that gene *W* is probably lethal when homozygous, as is a similar dominant white gene in mice. For no white stallion of this type has failed to produce colored colts when bred to any considerable number of mares either colored or white.

A white stallion, when bred to colored mares, commonly produces 50 percent of colored colts, 50 percent of white colts. When bred to white mares, the expectation is that, if *W* is as suggested lethal when homozygous, the ratio of white to colored colts will be 2:1, instead of 3:1 expected when the parents are heterozygous for a non-lethal dominant gene. This expectation has as yet not been put to an experimental or statistical test.

The physiological action of the *W* gene must be regarded as complete inhibition of color production in the coat, though not in the eye or the skin of certain body regions, which are pigmented.

*Dominant dilution*

Another type of white horse, not a pure white but ivory white or pale cream in color and with blue eyes, is the so-called cremello, which is homozygous for the dominant dilution gene *D* already described as present in heterozygous combination in Palomino and buckskin horses.

The investigations of WRIEDT (1925, 1928), SALISBURY (1941) and CASTLE and KING (1951) lead to the conclusion that a Palomino horse is heterozygous for a dominant gene for dilution (*D*) and is of genotype *AbbCDd*. Consequently Palominos are not true-breeding. From the mating of Palomino to Palomino there result colts in the ratio of 1dd:2Dd:1DD as regards genotype. The first is chestnut and full-colored, the second Palomino and the third "albino." The "albino" colt has an almost white, ivory or pale cream colored coat, pink skin and blue eyes. It is true-breeding because homozygous, its genotype being *bbDD*. If bred to a chestnut or sorrel mate, it will produce Palomino colts exclusively, all being heterozygotes (*Dd*).

The most desired type of Palomino is one which has a "golden yellow body color with white mane and tail." This suggests that it must have gene *A* in order to reduce the brown pigment in the coat and concentrate it in mane and tail, where it is diluted by gene *D* to a near-white. It suggests also that body color will be rendered clearer, more free from brown pigment, if the genotype is *ee* as in a sorrel rather than *E* as in a chestnut.

The practical recipe for production of the most desired type of Palomino would then be, (1) Secure a Palomino stallion which approaches as nearly as possible the desired type, clear golden yellow body, white mane and tail, genotype *AbbCDdee*. (2) Breed him to sorrel mares with light or flaxen mane and tail, genotype *AbbCee*. Sorrel mates will be preferable to chestnut, because the latter will be *EE* or *Ee* in genotype, whereas the former are all *ee*. All colts will then be *ee*, half of them Palomino, half of them sorrel. Palomino parents

of this type when interbred will produce colts in the ratio 1 sorrel : 2 Palomino : 1 albino, and all the Palominos should be of the desired body color free from sootiness.

If  $B$  is substituted for  $b$  in a heterozygous dilute ( $Dd$ ) horse, of the same constitution otherwise as a Palomino, there results a buckskin or "dun" of genotype  $ABCDd$ . The homozygous ( $DD$ ) genotype produced by interbreeding buckskin horses will be an "albino" with darker pigment in its mane and tail (diluted black) than is found in an "albino" produced by Palominos, which has a diluted brown mane and tail.

The albino produced by interbreeding buckskins may be designated type  $B$  albino to distinguish it from the albino produced by interbreeding Palominos, called type  $A$  albino. They differ in genotype only as regards gene  $B$ , the former carrying the dominant allele, the latter only the recessive.

A dilution gene even more effective in action than gene  $D$  of Palomino horses has recently been described as occurring in Shetland ponies. This gene is responsible for the production of the silver dapple variety of Shetland ponies and has been designated gene  $S$ .

Gene  $D$  modifies brown pigment in Palomino horses more than it modifies yellow-red pigment, which predominates in its body color, modified merely to a "golden-yellow," whereas the color of its mane and tail, in which brown pigment predominates, is modified to such an extent that it is described as "white" by breeders.

Gene  $S$  like gene  $D$ , reduces the intensity of black-brown pigment more strongly than red-yellow pigment, but its action is so much more energetic than that of  $D$  as to reduce black to a shade of "dark cream or light chocolate," while red is reduced in intensity only moderately. So in Shetland ponies of the silver dapple variety mane and tail, in which black pigment would normally predominate, are nearly white, and the body color is a moderately modified red-yellow, which resembles the chestnut body color of horses, for which reason this color variety is also called "dappled chestnut."

Gene  $S$  of Shetland ponies modifies black to as great an extent as gene  $D$  modifies brown in Palomino horses. What gene  $S$  would do to brown pigment in a genuine chestnut pony ( $bb$ ) has not been determined.

In view of the similarity in action of gene  $D$  in a background of a chestnut or sorrel genotype ( $bb$ ), and that of gene  $S$  in a background of a black or bay genotype ( $B$ ), it seems possible that the two genes may be alleles. The determination of this point awaits further investigation.

*Roan* is a coat variation of horses in which white hairs are intermingled with colored ones. The white hairs are present in the coat from birth on and their number remains substantially unchanged. This may be called a non-progressive form of silvering. It is inherited through the agency of a dominant gene  $R$  for roan. The body color in a roan horse is more often red than black, though both sorts occur, one being called red roan, the other blue roan.

There is reason to think that gene  $R$ , like  $W$  for dominant white, is lethal when homozygous. MR. FRANK R. SMITH finds (personal communication)

that in mating of roan with roan, a majority of the colts are roan, but the ratio of roan to non-roan is nearer to 2:1 than to 3:1 and no roan stallion has been found to sire only roan colts, as would be expected of an  $RR$  individual.

A type of congenital or non-progressive silvering in which the white hairs are localized more or less completely in large patches on the rump is known in the United States as Appaloosa. What its genotype is has not been determined. Non-progressive silvering occurs in cattle and guinea-pigs, comparable with the roan of horses.

*Gray* is the name given to a progressive type of silvering found in Percherons and in certain other breeds of horses, it results from a dominant gene  $G$  (gray). The coat of a gray horse, like that of a roan, consists of a mixture of white with colored hairs (mostly black) but the white hairs are not present in the first coat, as they are in roans, but only make their appearance in a later coat and subsequently increase until the coat may become practically all-white at an age of 4-12 years. So this type of silvering is not congenital but progressive, in which two respects it differs from roan.

Unlike  $R$ , gene  $G$  is not lethal when homozygous, as gray stallions have been definitely identified which produce gray colts exclusively, whatever the colors of their mates.

Gray horses are usually dappled, but WRIEDT has shown that the character of the dappling (distribution of dark spots in the coat) may be modified by an independent mutation, so it seems probable that dappling is a non-essential and independently determined feature of a gray coat.

Progressive non-congenital silvering occurs in rats as well as in horses but it is recessive in inheritance and so not strictly comparable with the gray of horses.

Many mammals have white-spotted varieties, in which definite areas of the coat are unpigmented. Their occurrence and heritability have been given much attention by students of genetics.

#### *White spotting*

In horses minor degrees of white spotting seem to be inherited as recessive characters, such as a star or blaze in the forehead, or one or more white feet. But extensive white spotting, taking the form of large irregular white areas running across the body, is clearly a dominant mutation the gene for which may be called  $P$  (piebald, or Pinto). Most piebald horses are heterozygous ( $Pp$ ) and when bred to normally colored mates produce 50 percent only of piebald colts.

There is no reason to think that  $P$  like  $W$  is lethal when homozygous, but it seems probable that a homozygote would be whiter than a heterozygote, as is certainly the case in dominant white-spotting in rabbits and dogs. Fanciers in such cases prefer the heterozygotes because their pattern is more striking.

#### SUMMARY

The primitive coat color of most mammals results from the activity of four basic color genes,  $A$ ,  $B$ ,  $C$  and  $E$ .

1. A black pigment in the eyes, skin and hair results from the interaction of gene *B* (black) with gene *C* (color). The distribution of black pigment in the coat is influenced by a wild-coat-pattern gene *A* (agouti) which in certain regions of the body or in particular parts of individual hairs replaces black pigment with yellow. The result is a concealing or protective coat pattern.

2. The primitive type of coat color dependent upon four dominant genes has become modified in the course of time by recessive mutations in each of them. These form the basis of domestic color varieties many of which would be unable to survive in a wild population because of their lack of concealing value.

3. The first such mutation to be observed in most mammals is albinism resulting from mutation in gene *C* to a recessive allele *c*, which when homozygous (*cc*) results in a completely unpigmented body.

4. The next mutation to be observed is usually a recessive mutation of *A* to *a*, resulting in complete loss of the wild-coat-pattern in a recessive uniform black of genotype *aaBCE*.

5. A recessive mutation of gene *B* to *b* in many mammals results in a qualitative change in the dark pigment from black to brown. There is thus produced a genotype *AbbCE* which in rodents is called cinnamon, in horses chestnut. Combination of this mutation with the foregoing one results in a double recessive genotype (*aabbCE*), a non-agouti uniform brown known as chocolate in rodents, liver in horses.

6. A recessive mutation of gene *E* to *e* effects simultaneously two changes in the coat pigmentation. (a) Extended (*E*) distribution of black or brown pigment becomes restricted (*ee*) distribution and (b) in areas of restricted distribution, red-yellow pigment replaces black or brown. It has been suggested that this secondary effect be emphasized by calling *e* a red mutation (erythros, Greek for red). This mutation when homozygous (*ee*) results in rabbits and guinea-pigs in all-yellow or red body color, black or brown being restricted to the eyes and skin of the extremities. Such *ee* genotypes in horses are represented by the red bay, in which black pigment is restricted principally to mane and tail and legs, and by the sorrel in which brown pigment is similarly restricted.

7. A dominant mutation of *E* to *E<sup>D</sup>* results in a new genotype *ABCE<sup>D</sup>*, known as *dominant black*. It is found in rodents, dogs, cats and probably also in horses.

8. Mutations resulting in white hairs or white areas in the coat are found in practically all mammals. In some cases they are recessive, and have little effect on the general coat coloration. In other cases they are dominant and affect the general coat coloration profoundly. In horses the following white hair mutations are known.

- (a) Dominant white, coat entirely white, eyes colored. Probably lethal when homozygous, gene *W*.
- (b) Dominant dilution, gene *D*, less effective in heterozygotes than in homozygotes. Palomino and buckskin horse are heterozygotes (*Dd*); the corresponding homozygotes (*DD*) are so-called albinos of types A and B respectively.

- (c) Dominant dilution, gene *S*, found in silver dapple Shetland ponies reduces the intensity of black pigment as much as gene *D* reduces the intensity of brown pigment. In both cases, black or brown concentrated in mane and tail become almost white.
- (d) Roan, gene *R*. A type of congenital, non-progressive silvering, probably lethal when homozygous like gene *W*. A type of congenital, non-progressive silvering in which white hairs are localized on the rump is known in the United States as Appaloosa, genetic basis undetermined as yet.
- (e) Gray, gene *G*. A type of dominant non-congenital but progressive silvering usually dappled due to an independent modifier.
- (f) Recessive white markings, white feet, blaze, genes undetermined.
- (g) Dominant white-spotting, white areas extensive. Gene *P* (Piebald, Pinto). Homozygotes probably whiter than heterozygotes as in similar varieties of white-spotted rabbits and dogs.

## LITERATURE CITED

- CASTLE, W. E., 1951 Dominant and recessive black in mammals. *J. Hered.* **42**: 48-59.  
 1953 Silver, a new mutation of the rat. *J. Hered.* **44**: (in press).
- CASTLE, W. E., and F. L. KING, 1951 New evidence on the genetics of the Palomino horse. *J. Hered.* **42**: 60-64.
- CASTLE, W. E., and FRANK H. SMITH, 1953 Silver Dapple, a unique color variety among Shetland ponies. *J. Hered.* **44**: 139-145.
- ODRIOZOLA, M., 1951 A los colores del caballo. 435 p., illustr. Madrid.
- PUNNETT, R. C., 1912 Inheritance of coat colour in rabbits. *J. Genet.* **2**: 221-238.  
 1915 Further experiments on inheritance of coat colour in rabbits. *J. Genet.* **5**: 37-50.
- SALISBURY, G. W., 1914 The basic colors and patterns (of the horse). *J. Hered.* **32**: 235-240; 255-260.
- WRIEDT, C., 1925 Vererbungs-untersuchungen beim Pferd. *Zeit. ind. Abst. Ver.* **38**: 88-101.  
 1928 Ein neuer Vererbungs-faktor beim Pferd. *Hereditas* **10**: 274-276.
- WRIGHT, S., 1917 Color inheritance in mammals. VII. Horse. *J. Hered.* **8**: 561-564.