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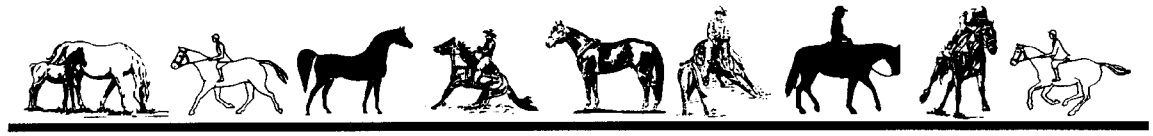

THE GENETICS OF EQUINE COAT COLOR

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INTRODUCTION

History

Great variation is present in horses of today. There are large horses and small horses, riding horses and driving horses, draft horses and race horses. There is, no doubt, a type of horse for almost every possible purpose. Although type and performance ability are quite variable, they are not more variable than the colors of the horses.

The ancestors of the domesticated horse did not vary much in color, but were chiefly of "wild type" coloration. This color was of great value in hiding these wild horses from their natural enemies. This concealing type of coloration can still be seen in the few Przewalkski horses, undomesticated and near extinction in Mongolia in Central Asia. When some of these horses were domesticated some 4,000 years ago, it is probable that some varying colors were preferred in selection and breeding. This breeding by color may have brought together some of the recessive genes, eventually resulting in different colored horses. Also, through the ages, mutations in these basic genes probably played a role in development of different colors.

Ancient paintings and writings suggest that color in horses has been variable for sometime. Spotted horses may have been present as early as 1400-1300 B. C. as Egyptian tomb paintings showed two horses so colored. In the Bible (Rev. 6) symbolic white, red, black and pale colored horses are mentioned.

Today there is disagreement among the experts on many aspects of the genetics of equine coat color. Early research in this area dealt exclusively with analysis of stud books, observation of various matings with subsequent color foals produced, and then hypothesizing which genes were involved. Inaccurate records, foals changing colors, and lack of common color descriptions among horsemen were also problems. Since the early 1940's; however, geneticists have drawn on the more exacting information obtained from planned experimentation with laboratory mammals. The close relationship is now evident and as a result many questions on equine color can now be answered with reasonable certainty.

Genetics

Inside the cells of all animals are found chromosomes and genes. Chromosomes are paired thread-like structures which carry the genes. Each body cell contains a certain number of pairs of chromosomes (eg. horse - 32, man - 23). Genes are the units of inheritance which dictate what an individual will look like. This outward expression, called phenotype, is a result of an animal's genetic makeup, called genotype. As chromosomes occur in pairs, so do genes. Two genes exist side by side, each on one of the chromosomes of a pair. An allele is one member of this pair of genes, or a partner gene. The specific position, where paired genes are found, is called their loci.

The genotype of a chestnut colored horse, in this example, is written as AabbCCddEEggrrssww where each pair of letters represents a pair of genes at a loci. Note within the pairs some genes are written with a capital letter; some with small letters. A capital letter means that particular gene is a strong or dominant gene while a small letter represents a weak or recessive gene. Within the gene pairs within a

genotype, three combinations are possible. There may be two dominant genes (EE), a dominant and a recessive gene, or two recessive genes. Usually when two dominant genes are present the second dominant one adds nothing. Exceptions to this are known though where two dominant genes are twice as potent as one dominant gene. When a dominant and a recessive gene are present within a pair, two expressions are possible. One, the dominant gene is dominant over the recessive gene, called complete dominance; or two, the two express themselves in an intermediate fashion, called incomplete dominance. Lastly, two recessive genes together either add nothing to the phenotype or express themselves differently. Within a given genotype, several pairs of genes work in combination to produce the animals' phenotype. Usually certain genes modify only slightly the expression of other genes; however in some cases, one gene completely dominates all other genes. This is called epistasis.

BASIC COLOR GENES

C Series

The presence of eumelanin (black-brown) and/or phaeomelanin (red-yellow) pigments cause skin, coat and eye color in mammals. When gene C is present, normal pigment formation reactions occur; however, mutants of gene C result in complete or partial albinism. In complete albinism (cc), no pigment is formed. The hair is white, the skin is pink and the eyes are red, due to the visible color of the blood. Some familiar examples of complete albinos are white rabbits, rats, and mice. In some incomplete albinism cases, the skin, coat and eye colors are the result of the actions of such gene C mutants as c^{ch} or c^{H}. Mammals carrying these mutations are the Chinchilla rabbit ($c^{ch}c^{ch}$) and the Himalayan rabbit ($c^{H}c^{H}$).

Horse researchers are in general agreement that a dominant gene C is responsible for presence of pigment in the skin, coat and eyes. Consequently, gene C is usually omitted when writing horse genotypes. Horsemen often refer to horses with light colored skin, hair and partially or totally pigmented eyes as albinos; however, according to Searle (1968), no well authenticated case of complete albinism has ever been reported in the equine. The only gene C mutant proposed in the horse was that of c^{cr} (Odriozola,1951). He postulated this allele was responsible for the color of palominos (Cc^{cr}), buckskins (Cc^{cr}), duns (Cc^{cr}), cremellos ($c^{cr}c^{cr}$), and perlinos ($c^{cr}c^{cr}$) ;however, no conclusive evidence was presented. Singleton and Bond (1966) adapted this c^{cr} mutant theory to explain diluted colors in place of the dominant dilution theory forwarded by Castle (1947). Singleton and Bond (1966) includes this proposed c^{cr} allele in writing horse genotypes.

B Series

In the horse, as other mammals, gene B is responsible for the production of black pigment. Theoretically if no modifier genes affected the intensity or distribution of black pigment, all black horses (BB or Bb) would be the same color. This is not the case. According to Salisbury (1941), some horses are foaled jet black and hold their color, while other horses, are foaled a mouse-brown-gray, are black when kept stalled, but fade either to brown or red in intense sunlight. Some horses have localized black pigmentation in the extremities. Examples are bays, some browns, buckskins, duns, grullas, and perlinos. In both of these cases where black is changed, modifier genes (A, E and/or D) are operating in the genotype.

Gene b, a recessive mutant of gene B, produces a rich brown pigmentation; therefore, horses with genotype bb are chocolate brown colored called chestnut. This rich brown color is different than the straight brown color resulting from the action of the a^{t} allele in the agouti series (Searle, 1968). As is true with gene B, gene b is modified by the action of genes A, E and/or D. Their pigment diluting, restricting and/or extending actions, in conjunction with the bb genes, modifies chestnuts to certain types of browns, sorrels, claybank duns, palominos and cremellos.

A Series

Gene A acts in conjunction with genes C and B to produce a concealing coat pattern known as agouti or "wild type". The characteristics of this coat pattern are black pigment on the dorsal and peripheral parts of the body and to particular parts of the individual hairs. Familiar examples are the cottontail rabbit and the wolf. Loss of the wild pattern results when gene A mutates to recessive gene a. In homozygous form (aa), this gene pair

causes an individual to be uniform in color. In rabbits, rodents and dogs, gene A has also undergone a second mutation to gene a^t. When homozygous (a^ta^t), this genotype causes black and tan coloration. Traces of the agouti are still evident; however, in this pattern.

Gene A, dominant to a, in horses is responsible for bodies of one color and manes and tails of another. This wild type pattern was first noted in the Przewalski horses, the undomesticated ancestor to modern horses. Phenotypically this horse's body color is neutral gray (mixed yellow and black pigments) with a blackish mane and tail, blackish legs, and a dorsal stripe (Castle, 1953). Castle and Singleton (1961) reported seeing horses with similar color on the Argentina Pampas. According to Castle and Singleton (1961), the wild type pattern is seen today in some domestic varieties. Examples are bays, buckskins, duns, grullas, perlinos, palominos and sorrels with light manes and tails. The latter two have lighter manes and tails than bodies due to the action of another gene on dark pigment, after it is concentrated in the manes and tails. All, however, have gene A in their genotypes.

Three alleles of gene A have been reported in the horse. According to Searle (1968), the non-agouti allele, a, leads to absence of yellow pigmentation in the body. Horses of the genotype (aa) are uniformly dark. For example, in a dark bay (ABE), substitution of the non-agouti alleles would render an individual uniform or recessive black (aaBE). Substitution of non-agouti alleles for the agouti allele, in horses with bb in their genotype, produces uniform chestnut or sorrel coloration.

Odriozola (1951) and Castle and Singleton (1961) postulated the presence of an A^+ allele in the horse. They believe the striped agouti pattern (bars on legs, dorsal stripe, etc.) of wild horses and certain domestic breeds is caused by a dominant allele A^+. In addition, Castle and Singleton (1961) postulated an A^+ allele to be responsible for brown, black-brown or seal brown color where the mane, tail, feet and legs are black and the body is brown to black with traces of light areas on the muzzle, neck and flanks. They stated this allele is dominant over non-agouti and recessive to gene A; however, Searle (1968) stated that brown x black matings quite frequently result in bay offspring. Further investigation is needed in this situation.

Generally, geneticists have considered the various colors of duns (ABED) to be the result of the wild pattern A gene in combination with the D and B genes. After the hypothesis of a possible mutant, a^t, Castle and Singleton (1961) stated that the red or claybank dun, possessing a yellowish-brown body color with a dark spiral stripe, bars on the legs and/or both, most likely carries an a^t, not an A gene. The darker extremities in the claybank dun ($a^tbbeeDd$), as compared with the palomino (AbbeeDd), are due to the presence of the a^t gene, according to these authors.

E Series

In the horse, as in many other mammals, genes E, e and E^D are present. Although three alleles are known to be present in this series, an individual can possess only two of them. Gene E permits black-brown pigment to be normally extended uniformly throughout the coat in the absence of A. In the presence of A, the degree of extension is decreased. Examples are the uniform liver chestnut (aabbE) and the chestnut (AbbE), respectively.

Gene e is postulated to be a mutant recessive allele of gene E. In the recessive homozygous form, gene e restricts the distribution of black-brown body pigmentation in a similar manner to that of gene A; however, it does not form the concealing pattern of gene A. A genotype containing genes ee and gene A is characterized by black-brown pigment in the mane, tail and legs while the central body coat remains almost completely devoid of black-brown pigment. In its place, red-yellow pigments form. Castle (1953) points to the typical red or blood bay (ABee) horse as an example of this recessive red mutation. This color bay's general body color is red, not a mixture of black and yellow pigments as in the Przewalski horse (ABE). Both however, have black manes, tails, and legs resulting from the primitive A pattern gene.

If gene A is absent, ee leads to a horse with a more uniform color; however, somewhat more reddish in the body. An example is the recessive black horse (aaBee) with a slightly darker mane and tail than the central body. When exposed to intense sunlight, this color fades to a

redder color.

Salisbury (1941) reported that some black horses were foaled a peculiar mouse-brown-gray color and that they faded to a red color when exposed to the sunlight. Other black horses, he stated, were foaled jet black and remained so even when exposed to intense sunlight. According to Castle (1953), the former is genotypically a recessive black while the latter is a dominant black. Dominant black was first postulated in rabbits (Punnett, 1912) and is common in Shetland ponies. The gene responsible is E^D which increases the amount and distribution of black pigment in any coat. In addition, in some cases, it can even mask the presence of gene A. This explains how a black stallion ($ABCE^DE$) mated to a black mare (aaBEE) will occasionally produce a bay foal.

D Series

Much controversy exists with regard to gene D for dilution. Early researchers referred to it as a dominant dilution gene, but according to Singleton and Bond (1966), gene D is not really completely dominant since the phenotype of the heterozygote (Dd) is halfway between those of the two homozygous forms (DD and dd).

In heterozygous form, gene D results in moderate reduction in the intensity of all pigmentation whether black, brown, red or yellow. For example, gene D dilutes a mahogany bay (ABEdd) to a dun (ABEDd), a red blood bay (ABddee) to at buckskin (ABDee),a recessive black (aaBddee) to a grulla (a^tBDdE), and a seal brown (a^tBddE) to a light seal brown (a^tBDdE). According to Castle and Singleton (1961), the effect of gene D on brown pigment seems greater than on black; therefore, a chestnut (AbbEdd) becomes a palomino (AbbDdE) with a white mane and tail and a yellowish-brown body color. According to Castle and King (1951), Castle and Singleton (1961), and Singleton and Bond (1966), the D gene has little diluting effect in the absence of gene A.

In homozygous form (DD) the dilution gene causes a much greater reduction in the intensity of pigmentation. For example, when mating a palomino with a palomino, the offspring, on the average, will be one full colored chestnut, two palominos and one very dilute individual (AbbEDD). This dilute horse's phenotype is an ivory white coat with a lighter mane and tail and pink skin. This horse is called a cremello or a type A albino. Mating two buckskin horses, the offspring would be one bay, two buckskins and one individual with a cream colored body and a darker mane and tail, resulting from gene B. This individual is called a perlino or type B albino with genotype ABEDD. Note the only difference in the genotypes of these two horses is that the cremello is a diluted chestnut while the perlino is a diluted black.

OTHER COLOR GENES

Gene W

The white horse has pink skin, white coat hair, a white mane and tail, white hooves and always colored eyes, either brown, blue or hazel. Only occasionally are small pigmented areas found in the skin or hooves. Cream, ivory and gray colored horses are often confused with the dominant white horse; however, the former two do not have snow white coats, white mane and tail hairs and the latter does not have pink skin or white hooves.

Wriedt (1925) stated that white in horses was caused by recessive characters; however, most researchers are now in agreement that dominant gene W, epistatic to all coat colors, is responsible for this white phenotype. The ww genotype allows normal expression of the other genes in the genotype. Castle (1953) stated that no true breeding white stallion had ever been reported, suggesting the WW genotype to be a lethal condition. Pulos and Hutt (1969),after 15 years of mating white horses, confirmed this stating that the WW individual dies during early embryonic development. This WW genotype is also lethal in Dexter cattle, platinum foxes, bluefrost minks and certain mammals, according to Searle (1968).

Two additional alleles of the W gene, W^1 and W^2, have been proposed by Miller (1969) to be involved in the complex genetics of Appaloosa coat inheritance. He stated individuals of the W^1 W^1 or W^1w genotypes would exhibit Appaloosa type while those of W^2W^2 or W^2w genotypes would be of ranger type.

Gene G

The graying gene, gene G, prevents melanin granules from passing into the body hairs. Thus horses turn gray with increasing age. Gray horses are born with a solid color coat, but upon loosing this juvenile coat, white hairs begin to appear. At this stage gray horses are often confused with roans. According to Salisbury (1941), the rate at which a foal will gray out depends on his genotype; with homozygous dominant (GG) individuals graying sooner than heterozygotes (Gg). This has not been confirmed by other authors. Castle (1953) stated that some horses are completely gray at four years of age while others require up to 12 years of age before completely graying. He called, this graying process "progressive silvering".

Well authenticated cases have been reported where gray stallions produce all gray offspring; therefore, horses with GG and Gg genotypes are gray (Castle, 1953). Gray is dominant over all colors except white; therefore, only gray horses produce gray offspring. Although, dapples are common among gray horses, not all grays are dappled. According to Wriedt (1925) dappling is caused by another independent gene.

It is interesting to note that gene G has an unfavorable pleiotropic effect, for gray horses are exceptionally prone to skin cancers in the form of melanomas (Searle, 1968). Freckling is common in old white horses and seems to be associated with melanomas in the perianal region.

Gene G is present to some extent in all breeds of horses; however, it is most common among Arabians, Lippizaners and Percherons (Castle and Smith 1953). In some color breeds, gene G is detrimental. Hatley (1962),warned that Appaloosa breeders should avoid the graying gene, "the gene that kills Appaloosa color."

Gene R

Roan color in horses is defined as white hairs intermingled with black, brown, red or yellow hairs in the coat. Individuals of Rr genotype are roan while rr individuals exhibit normal coloration with no roaning. Castle (1953) stated that no true breeding roan horse has been reported, suggesting the RR genotype as was WW, is lethal.

Castle (1953) has referred to roaning as "non-progressive" silvering to distinguish it from graying or progressive silvering. Foals carrying the Rr genotype are born roan and the number of white hairs remain substantially unchanged throughout life. This is in contrast to gray foals who are born a basic color then white hairs appear later and increase in number throughout life. Castle (1953) states also that red roans are more common than blue roans.

Although the genetics of Appaloosa coat color are still unknown, Miller (1969) suggests the possibility that certain Appaloosa color patterns are the result of non-progressive silvering in which white hairs are localized more or less completely in large patches on the rump.

Genes for Appaloosa

Appaloosa coat color inheritance is complex as is suggested by the fact that many different types of Appaloosa patterns occur. Examples are frost, leopard, marble, snowflake, spotted blanket, white blanket patterns, etc. While several authors have referred to Appaloosa patterns, only Miller (1969) has conducted a detailed study and forwarded a possible hypothesis as to the genotypes of these Appaloosa phenotypes. It is as follows:

The basic expression of the Appaloosa color pattern is controlled by a single gene (Ap), which is apparently dominant. When this gene is present the color pattern can be expressed, providing modifying genes are also present. A series of modifying genes control the expression of white and another series control the expression of spots. Horses with the Ap gene will have white distribution if modifying genes are present which prevent pigment formation. Inasmuch as the distribution of white radiates both anteriorly and posteriorly from the sacral (hip) area, the modifying genes work as a series. It may be that if one or two modifying genes are present, the horse will lack pigment in the sacral areas; if three or four modifiers are present a slightly larger area will be white, etc., up to the all white horse. Thus, the modifying genes would be quantitative in nature. A horse with the Ap gene, but with no modifiers, or a horse with modifiers but not the Ap gene, would not show white distribution.

The modifiers cannot express themselves

except ln the presence of the Ap gene. It is possible one pair of recessive genes (ww) must be present to allow expression of the white modifiers. This would explain how a non-blanketed Appaloosa (Ww) would produce blanketed offspring when the other parent was non-Appaloosa.

A series of genes for spotting also served as modifiers of the Ap gene. They are exhibited in horses with or without the white modifiers and they appear to be independent of the white modifiers in their mode of inheritance.

Physiologically, they inhibit the white modifiers and allow the production of pigment in spots. The entire white area may be spotted or only a portion. The expression of these genes tends to begin in the sacral area and radiate both anteriorly and posteriorly. However, this tendency was not nearly so marked as with the expression of white. Some horses had spots in widely scattered areas. Modifying genes for white and spots may be present in solid colored horses.

Blanketed horses with no spots, when mated, can produce spotted offspring. The same is true if one parent is blanketed with no spots and one is solid color. Evidence that the action of spotting genes is not dependent upon the presence of the white modifiers, is furnished by the fact that solid colored horses with the Ap gene can be spotted.

As with the white modifiers, the spotting genes may be controlled by a single pair of recessive genes (ss), which in the presence of the Ap gene allow the expression of a series of modifiers for spots. Thus, horses with blankets and no spots could produce spots.

A model of the color inheritance could be as follows:

Ap - expression of Appaloosa color

ww - expression of white color

w^1w^1, w^2w^2, etc. - modifiers of Ap acting in the presence of ww

ss - expression of spots

s^1s^1, s^2s^2, - modifiers of Ap acting in the presence of ss

Ap - ww ss - blanket with spots

Ap - ww Ss - blanket, no spots could produce spots

Ap - Ww ss - no blanket with spots, could produce blanket

Ap - Ww Ss - no blanket, no spots, could produce blanket and spots

Modifying genes must be present in each of the above four genotypes if blanket or spots are exhibited. Size of the blanket and the number of spots would be determined by the number of modifying genes present.

Studies would indicate that the expression of white and spots are sex influenced. It may be that the genes for white and spots act in following manner:

	Males	Females
WW	Solid	Solid
Ww	White	Solid
ww	White	White
SS	No Spots	No Spots
Ss	Spots	No Spots
ss	Spots	Spots

The roan gene may act in the following manner:

Males	Females
RR Roan	Roan
Rr *	Roan
rr non-roan	non-roan

*This genotype may be expressed in the male as fringe roaning around the blanket

Genes for Spotting

The genetics of white spotting in horses has not been worked out. Several spotting genes have been hypothesized by researchers. In most cases, researchers have; however, recognized that spotted horses are of two distinctly different phenotypes.

Castle (1954) said spotting is caused by gene P (for piebald or pinto) which only expresses itself when in heterozygous (Pp) condition. Lasley (1970) stated that dominant gene S causes white spotting. In addition he added that horses of genotype SS or Ss were spotted while those with ss genotype were full colored. Jones and Bogart (1971) postulated gene T (for tobiano) was responsible for dominant white spotting patterns. These authors also hypothesized that several modifier genes operated in conjunction with the T gene to cause variable white markings on the face, body, feet and legs.

Brown (1970) also referred to a dominant gene T, stating that homozygous recessive (tt) horses were solid colored while heterozygotes (Tt) were spotted. He also stated that since all other pattern genes seem to have harmful effects in the homozygous condition, it would be unusual if animals of TT genotype are unaffected. According to Brown, cases have been reported where two solid colored horses have produced tobiano offspring, suggesting the heterozygote may not always show white spotting.

Concerning recessive spotting, Kremols (1933) coined the term splashed white to describe the ventral lack of pigmentation of this recessive pattern. Castle (1940) postulated that a recessive factor was responsible for minor white spotting on the face, legs and feet and that in its maximum expression, white spotting would be found on the main part of the body. Jones and Bogart (1971) stated that modifier genes caused the variability in splashed white patterns. They called this phenotype overo and stated it was the result of gene O in homozygous recessive (oo) form. According to Brown (1970), gene O in heterozygous form (Oo) allows normal expression of the other genes in the genotype.

A problem of serious nature that the overo breeder faces is the fatal white foal, according to Irving (1971). This foal is produced approximately 10% of the time in overo x overo matings or in matings of two individuals with overo genes in their genotype. The foal from these type matings usually dies within 5 days after birth. The causes of death in this abnormal foal are an atretic color and/or an abnormal blood factor similar to erythroblastosis in humans.

GENOTYPES AND PHENOTYPES

The genotypes listed below were obtained from Castle and Singleton (1961) and Lasley (1968). For genotype simplification, gene C, as well as other non-expressing gene pairs, have been omitted. Also dominant genes alone mean that the allele could be either dominant or recessive without changing the individual's phenotype.

COLOR	GENOTYPE	PHENOTYPE
Black	aaBE	Recessive black, uniform
	aaBee	Recessive (smokey) black, dark mane and tail
	ABE^{D}	Dominant (jet) black
Chestnut	AbbE	Chestnut, dark mane and tail
	aabbE	Liver chestnut, uniform
	aabbEDd	Light chestnut, uniform
Sorrel	aabbee	Sorrel, uniform
	Abbee	Sorrel, light mane and tail
	aabbeeDd	Light sorrel, uniform
Bay	ABE	Ancestral bay
	ABE	Mahogany (dark) bay
	Abee	Blood (red) bay
Brown	$a^{t}BE$	Seal brown, light points
	$a^{t}Bee$	Seal brown, light points inconspicuous
	$a^{t}bbE$	Chestnut "brown"
	$a^{t}bbee$	Sorrel "brown"
	$a^{t}BEDd$	Light seal brown
	$a^{t}BeeDd$	Light seal brown
Dun	ABED	Dun
	$a^{t}bbEDd$	Clay bank dun, yellow-brown body, mane and tail
	$a^{t}bbeeDd$	Clay bank dun, yellow-brown body, dark mane and tail
Buckskin	ABeeDd	Buckskin, clear yellow body color
	aaBbEDd	Crypto-buckskin, dark cream body, mane and tail
	ABEDd	Sooty buckskin, black mane and tail
Dilute Black	aaBEDd	Dilute mouse, black, uniform
	aaBeeDd	Dilute mouse, black, mane and tail

Palomino	abbeeDd	Palomino, clear golden body, white mane and tail
	AbbEDd	Palomino, sooty red body, white mane and tail
	aabbEDd	Palomino, sooty red body, mane and tail
	aabbeeDd	Palomino, clear golden body, mane and tail
Cremello	AbbEDD	Cremello, ivory coat, light mane and tail
Perlino	ABEDD	Perlino, cream body, straw colored mane and tail
Dominant White	Ww	White
	ww	White not expressed
Gray	GG	Gray after first shedding
	Gg	Gray after first shedding
	gg	Gray not expressed
Roan	ABER	Red roan, dark mane and tail
	aaBER	Blue roan
	AbbeeRr	Strawberry roan, light mane and tail

SAMPLE MATINGS

Working crosses with several pairs of coat color genes is not as complicated as it first appears. This is because many times individuals mated are homozygous for the same genes. Each time a pair of genes is homozygous the same way in both parents those parents can produce only that genotype of offspring. On the other hand, when the parents are different in one or more pairs of genes, one to several different offspring genotypes are possible. The more pairs of genes for which parents are different, the more difficult the genetic combinations. For illustration, below are examples of four sample matings:

Example 1. Parents: aabbddeeggrrssww (Sorrel(female), uniform) aabbddeeggrrssww (Sorrel(male), uniform)

Genetic Combinations: Both parents have exactly the same genotype; therefore only one combination is possible.

Offspring: 100% aabbddeeggrrssww sorrels, uniform

In the next 3, more complex examples, examine the genotypes of each parent, noting those specific gene pairs which are not homozygous the same way in both parents. From these, list all possible combinations of genes contributed by the mare to the left of the box. Do likewise for the stallion and list at the top of the box. Fill in offspring squares with the approximate gene combinations. Figure the percentage of each genotype.

Example 2. Parents: $A^1a^2bbddeeggrrssww$ Sorrel(female), light mane and tail — $A^1a^2bbddeeggrrssww$ Sorrel(male), light mane and tail

Genetic Combinations:

(male)

(female)	A^1	a^2
A^1	AA	Aa
a^2	Aa	aa

Offspring: 25% AAbbddeeggrrssww sorrel, light mane and tail
50% Aabbddeeggrrssww sorrel, light mane and tail
25% aabbddeeggrrssww sorrel, uniform

Example 3. Parents: $a^1a^2B^1b^2ddEEggrrssww$ Black(female), uniform — $A^1a^2b^1b^2ddEEggrrssww$ Chestnut (male), dark mane and tail

Genetic Combinations :

(male)

(female)	A^1b^1	A^1b^2	a^2b^1	a^2b^2
a^1B^1	AabB	AabB	aabB	aabB
a^1b^2	Aabb	Aabb	aabb	aabb
a^2B^1	AabB	AabB	aabB	aabB
a^2b^2	Aabb	Aabb	aabb	aabb

Offspring: 25% aabBddEEggrrssww black, uniform
25% AabBddEEggrrssww bay, mahogany
25% AabbddEEggrrssww chestnut, dark mane and tail
25% aabbddEEggrrssww chestnut, uniform

Example 4. Parents: $AAbbD^1D^2E^1E^2ggrrssww$ Cremello (female) — $AAbbd^1d^2e^1e^2ggrrssww$ sorrel (male) light mane and tail

Genetic Combinations:

(male)

(female)	d^1e^1	d^1e^2	d^2e^1	d^2e^2
D^1E^1	dDeE	dDeE	dDeE	dDeE
D^1E^2	dDeE	dDeE	dDee	dDee
D^2E^1	dDeE	dDeE	dDeE	dDeE
D^2E^2	dDeE	dDeE	dDeE	dDeE

Offspring: 100% AAbbDdEeggrrssww palominos

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