

Equine Color Genetics and Deoxyribonucleic Acid Testing

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Abstract

Horses appear in a variety of coat colors and patterns, varying from solid red, black, or white to dapples and pearls, and in patterns like appaloosas and paints. These colors and patterns are managed by genes that may be associated with undesirable flaws or defects that can be fatal, such as the Lethal White Syndrome. Because deoxyribonucleic acid testing is now available to horse breeders, owners, and veterinarians, it is important to understand the fundamental concepts of dominant and recessive genes, genetics, and the potential for flaws to be expressed. Genetic testing can be performed to satisfy curiosity or to implement life saving measures to prevent the death of an offspring.

Key Facts

Color genes determine the distribution of red and black color pigmentation. Melanin is produced by pigment cells known as melanocytes.

Equine base or foundation colors are varied in three manners. These are shade, sooty, and mealy. The allele sequence, *WW*, is lethal resulting in embryo reabsorption or fetal death in the uterus of a mare. The homozygous dominant, or *OO* gene, is linked or associated with the Lethal White Syndrome. This anomaly of foals is characterized by congenital closure or complete absence of their colon with foals living only a small number of days.

Keywords: Equine; Color genetics; Foal anomalies; Genetic testing; Deoxyribonucleic acid

Introduction

Equine color genetics is an important factor when equine veterinarians and horse owners discuss personal color preference, registry guidelines, breeding outcomes, and medical aspects of equine practice. During a period of 6-7 years, approximately 1,500 markers were discovered with a minimum of at least 750 markers designated as genes [1]. Chowdhary and Bailey's [1] work in relation to coat color identified genes linked with coat color and athletic performance, and found genes linked with inherited disorders.

Many believe that the coat color of a horse has little to do with performance; however, coat color is a way to identify the horse [2]. A study by Stachurska et al. (2007) [3] examined whether racing performance was influenced by coat color, particularly in basic and gray colors. They found no relation between coat colors and racing performance [3]. Specifically, racing performance was not linked with the gray locus [3]. Conversely, physiological attributes linked with the luster of the hair and the action of the extension gene (*MC1R*) may contribute to the association between basic colors and racing performance [3]. Coat color is also a useful indication of questionable parentage [2]. Because of the array of existing coat colors and patterns and the possible devastating breeding outcomes that could result genetically, an understanding of the science behind equine color genetics is helpful to the breeder. This article will briefly discuss and summarize fundamental genetics and explain the genetic basis for the development of different coat colors of horses. This is a way for veterinarians to convey this information to breeders.

Discussion of basic color genetics

The coloring of a horse's coat is due to the sulfur-containing pigment,

melanin, which is detailed or mapped by color alleles, or other forms of a gene. One of two or more alternative forms of a gene at the same site or location in each pair of chromosomes determines alternative characteristics in color inheritance [4]. Genes have two or more alleles and some alleles may be dominant while others are recessive. Homozygous means an individual's gene set contains two of the same allele [4]. Heterozygous means an individual's gene set contains two different alleles [4]. Color genes determine the distribution of red and black color pigmentation. Melanin is produced by pigment cells known as melanocytes. One form is pheomelanin which is yellow to red in color whereas, eumelanin exhibits brown to black. Alterations of these two pigments by the appearance of specific genes result in a variety of horse coat colors seen today.

Science behind equine coat colors

Horse base colors initiate color genetics with either red, also known as chestnut, or the base color black [2]. Coat color genes assemble upon chestnut, black, and bay which is a mixture of the base colors red and black. In addition to base color, dilution properties come into the mix. For example, Fornarelli et al. [5] found that some horse phenotypes were described as a polymorphism genotype at the *MATP* gene and a shade of coat color dilution.

Equine base or foundation colors are also varied in three manners. These are shade, sooty, and mealy. Shade describes base colors varying from light to dark [6]. Different factors of genetic control result in variations [6]. According to Thiruvenkadan et al. [2] the modification of base colors by the presence or absence of black hair amongst the coat is termed sooty [2]. Thiruvenkadan et al. [2] also claimed that the genetic control of "sootiness" is not well understood and is also subject to modifications by environmental influences. Some breeders understand sootiness to be a specific gene effect rather than a method

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of alteration [2]. Finally, the third way equine base or foundation colors are varied. This modification results in horses having yellowish or pale red areas. These areas may include and are not limited to the ventral abdomen, the caudal aspect of the elbows, the flanks, and the medial aspect of their limbs, the eyelids, and the muzzle. The mealy effect is a single gene effect, which is dominant [2].

Modifiers can change the base color to produce the variety of colors and patterns appreciated in horses. The genes discussed are the extension gene or the gene allocating black pigmentation or color, the agouti gene or the gene limiting black pigmentation, the white gene or the gene managing whether horses will be fully pigmented or non-pigmented, the gray gene or the gene managing whether a horse will gray with age, the cream gene directing the amount of red pigmentation, and the dun gene managing the intensity of pigment in the coat. The silver dapple gene fades black pigment. Pattern genes discussed are the roan gene with coats appearing covered with white dust or lightly snowed, and the tobiano and overo genes, two of the paint or pinto patterns. The sabino gene appears as paint or with roan-like markings while the appaloosa gene appears as a spotting pattern. Also discussed are the newly discovered dilution genes champagne and pearl.

Extension

In the hair coat, true black pigment can be formed. The extension gene shows as an evenly distributed horse in a black coat or one with black only at the points, like a bay with a red body while the ears, muzzle, mane, tail, and lower limbs are black. Genetically, horses may or may not have the ability to produce black color, forming genotypes of *EE* (homozygous dominant), *Ee* (heterozygous), or *ee* (homozygous recessive), respectively. The latter is most often red haired with black pigmented skin showing colors of chestnut, cremello, gold champagne, gray, palomino or red dun [7]. The *e* allele is also known as the red factor [8]. The look of *EE* or *Ee* horses with black pigment in their skin and hair are bay, black, buckskin, grullo, perlino, seal brown, silver bay, or silver black. Rieder et al. [9] indicate that lighter bay shades are heterozygous for the *Ee* mutation while darker bay shades are homozygous dominant. Fortunately, health problems are not known to be related to the *e* allele.

Agouti

Horses with the agouti gene have the genes *AA* (homozygous dominant) or *Aa* (heterozygous) accountable for the limitation of black color. This limitation usually permits darker pigments to show on the points of the horse, while red coat color is evident on the body. *A'* is another allele that limits black pigment to a seal brown coloring [10]. *A'* is dominant to *a* and recessive to *A*. Rieder et al. [11] researched methods of genotype testing of basic colors [11]. They studied genes at the molecular level and found that the agouti gene, in its mutated structure, is the gene resulting in recessive black coats. While studying and using cross-species techniques on various equine genetic receptors and proteins, it was found that the chestnut allele has no dominant black mutation [9]. Seal brown horses with the occurrence of an *E* allele have the genes *A'A'* and *A'a* and look like a bay horse with the genotype *AA'*. *AA*, *AA'* or *Aa* horses are amber champagne, bay, bay dun, buckskin, or gray with black hair confined to the points. The *aa* (homozygous recessive) horse does not have the agouti pattern. For example, if this horse had an *E* allele, then the horse would be black or a variation of black such as grullo or smoky cream. The agouti gene has no visible change on *ee* horses because there are no black hair to limit on these horses.

White

The white gene is the determining factor of whether a horse will

appear fully pigmented or non-pigmented. The white gene is inherited with unpredictable expressions of depigmentation of coat color and is known to be a monogenic autosomal dominant trait [12] as well as one of several known depigmentation phenotypes in horses [13]. The allelic heterogeneity is elevated in equine white phenotypes, although this does not necessarily suggest a high mutation rate [12]. Although the term "albino" is used to describe non-pigmented horses, true albinos have pink eyes without pigment, impaired vision, and white skin [4]. At times, albino is used incorrectly to describe white horses, even though the horse may have pigmentation at the eyes, eyelids, and at various mucosal junctions such as the sheath. These horses should be called pseudo-albinos or false-albinos. Figure 1 shows an example of a false albino. Albinism means the congenital absence of normal pigmentation in the body often manifesting in the hair, skin, and eyes [4]. Inherited or true albinism is very rare in horses. A true white horse can be produced by the breeding of two overo horses, but the foals have congenital closure or absence of the colon [4]. Unlike horses, true albino Icelandic sheep exist without apparent congenital defects [4]. It is important to note that the alleles *WW* is lethal. According to Mau et al. [14], the result of this allele sequence is embryo reabsorption or fetal death in the uterus of a mare. Homozygosity for dominant white in relation to embryo reabsorption, however, has never been scientifically demonstrated. Genetic testing of the potential sire and dam is recommended to reveal if the horses' genotypes have the *W* allele. A horse with the gene *Ww* has white hair from birth and pink skin, and its eyes are blue or light in color. A *ww* horse is full with pigment. For over 2,000 years, white coat color has been a highly desirable trait in horses with different phenotypes spanning from approximately half of the horse depigmented or white to a horse with an entirely white coat [13].

Unlike coat color, markings of white on a horse's head like a blaze or star or on its legs like a stocking or coronet are a result of both genetic and non-genetic factors [2]. Likewise, numerous genes are responsible for creating white markings [2].

Gray

Gray horses are an anomaly in nature because they are born a non-gray color, but show progressive graying overtime. With age, as with human aging, the horse's coat will gradually become grayer and the graying is thought to be inherited and managed by a dominant allele at the gray locus [15]. The *GG* or *Gg* gene is noted as the gray gene. Horses with this genotype will have pigmentation at the skin and the eyes, and is not to be confused with a white horse. Gray horses will be white to dark gray based on the age of the horse and the amount of coat white hair. If a horse genetically has *gg* as its gray gene, then it will not gray with age.



Figure 1: A pseudo-albino horse; notice the pigmentation at the eyes, eyelids, muzzle, and at the vulva.

A study by Locke et al. [15] found that equine tyrosine related protein 1 (*TYRP1*) and melanocyte protein 17 (*Pmel17*) are not responsible for the gray phenotype. Studies have been done using microsatellite markers to determine whether various horse chromosomes have genes for coat color and melanoma propensities [16]. A study using the DNA of 80 Camargue horses found no relation between a microsatellite and melanoma or gray color [17]. Unfortunately, the gray color has been shown to be associated with melanoma, a tumor arising from pigment cells or melanocytes which is usually malignant or cancerous [4]. Principally, aging gray horses are most vulnerable to melanoma [18]. This tumor can occur at the rectum of a horse and be so devastating to cause interferences with rectal tonicity and fecal elimination.

Cream

The cream gene directs the amount of red pigmentation in the horse's coat similar to that of the agouti gene which limits the amount of black pigmentation; however, the cream gene shows incomplete dominance and is a dilution gene. Horses with the gene *CC* will be completely colored because of the lack of the dilution factor, *Cr*. "Single dilutes" or *CCr* gene horses show as buckskin, palomino [19], or smoky black. Notably, red pigment is visibly weakened to gold with an ivory to white mane and tail, as in a palomino horse, and bays are diluted to buckskins with their red to black coat diluted to a light tan and the points unchanged thus staying black (Figures 2 and 3). Some horses may be diluted further to a cream or virtually white color with pale skin [19]. The palomino and the buckskin colors are the most recognized amongst the production of the dilution genes [2]. Black horses and black pointed horses are not diluted. To determine this invisible dilution factor in potential black horse carriers, genetic testing is necessary to disclose smoky black coloration. "Double dilutes" or



Figure 2: Palomino horse with gold hair coat and an ivory to white mane and tail.



Figure 3: Buckskin horse; points unchanged by the cream dilution gene.



Figure 4: Dorsal stripe; line of darker pigmentation running along the dorsum of a dun horse.

CrCr gene horses result in perlino or cremello horses, also known as "double diluted creams". These horses most likely will have skin diluted to pink and eyes lightened to blue. This gene usually fades black coats to reddish or mouse gray color and red coats to cream. Through the results of a causal mutation study, Mariat et al. [19] identified a molecular test to identify horses carrying the cream allele which lead from an aspartic acid encoded protein to an asparagine substitution. This molecular test is helpful in improving breeding management.

Dun

Another dilution gene is the dun gene or *DD* gene (homozygous dominant) or *Dd* gene (heterozygous). It is a simple dominant gene that affects black pigment and red pigment evenly without affecting the eyes or skin [20]. The dominant *D* allele shows light pigmentation of the body. These horses have faded body colors. The color possibilities are pinkish-red, yellow-red, yellow, or mouse gray and have dark points. Examples of these color possibilities are bay dun, red dun, grullo, amber dun, palomino dun, or gray. *DD* or *Dd* horses also have primitive markings such as a dorsal stripe, shoulder stripe, and leg bars. Figure 4 is an example of a dorsal stripe. Dun offspring will always possess the *D* allele from a *DD* parent. According to Stachurska and Ussing (2012) [21] blue dun foals are the most sought-after concerning the minute markings present and the highly desirable genotype (*aaEEDD*) of the Polish Konik horse. In a study analyzing color variation at the beginning of horse domestication, Ludwig et al. [22] found that European Pleistocene and Siberian horses were bay or bay-dun. Horses that are homozygous recessive, or *dd* horses, have no dilution to their coat colors and no primitive markings. Instead, they have intense color. Examples are rich palominos, grays, chestnuts, bays, or blacks.

Roan

Roan horses are colored with a base color like red or black mixed with white hair. This pattern resembles a base color that is covered with white dust or lightly snowed. Some describe this pattern as if the horse's color is covered with confectioner's sugar. Figure 5 demonstrates a roan pattern. Horses that possess this color arrangement are genetically *RnRn* (homozygous dominant) or *RnRn* (heterozygous). Examples of roans are the blue roan and red roan, also known as the strawberry roan. Figure 6 is an example of a strawberry roan. Note the lack of white dust on the face and distal limbs. An early breed example is the Murgese which can appear black and sometimes roan [23]. Murgese are mesomorph or compact and muscular horses from Italy used for saddle with a registry dating back to 1926 [23]. A study by Marklund et al. [24] comparing equine *KIT* (mast cell growth factor receptor) cDNA



Figure 5: Roan pattern; red base color covered with white dust.



Figure 6: Strawberry roan horse.

sequences, representing three alleles (2 *rn* and 1 *Rn*), uncovered five sequence polymorphisms and a few mRNA splice variants [24]. None, however, were connected with *Rn*. Current studies show no evidence of a “Lethal Roan Syndrome” in *RnRn* roans. Nonetheless, further research studies should be conducted to determine whether the “Lethal Roan Syndrome” exists. Horses genetically *rnrn* (homozygous recessive) have no roan pattern in their coats.

Tobiano

The four types of paints or pintos are tobiano, overo, tovero, and the classically marked. Discussed here is the commonly known tobiano and overo. Tobianos are homozygous dominant, *TOTO* and heterozygous, *TOto* with vertical, defined ovals of white color. White coloration extends over the dorsal midline and down the limbs. Generally, the horse’s face and tail are dark in color. According to Haase et al. [25], there was no noticeable visual difference between horses that are tobiano homozygous or tobiano heterozygous. They also found supplementary data to confirm what was previously documented that the tobiano spotting pattern resulting from the inversion of *ECA3* is the causative mutation [25]. A study using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) with hair follicle DNA to isolate homozygotes for the tobiano gene by Brooks et al. [26] found it to be more effective than present biochemical methods testing with albumin and vitamin D binding protein haplotypes. Horses possessing the alleles *toto* are homozygous recessive and do not have a tobiano pattern. Figure 7 illustrates a paint horse in red and white.

Overo

The overo horse, the other of the four paint or pinto patterns, is

also known as the “frame overo”. This pattern shows as a casing of color surrounding specks of white. The *OO* (homozygous dominant) or *Oo* (heterozygous) horse has white coloration positioned straight across the length of it with imperfect margins. The horse’s face is usually white unlike the tobiano horse which has white coloration extending over the back and down the limbs. The overo horse’s color crosses the back and legs; although some may disagree. The *O* allele is sometimes referred to as *Fr* or *FrO*. Several important facts exist in regard to the overo color pattern. First, the homozygous dominant, or *OO* gene, is linked or associated with the Lethal White Syndrome, also known as Overo Lethal White Syndrome, which is believed to be linked to a protein marker [27]. It is an inherited condition of American Paint horse offspring with overo pattern lineage [28]. This anomaly of foals occurs in white horses or horses that are almost entirely white [28] and is characterized by congenital closure [29] or complete absence of their colon with foals living only a small number of days, if that. A study by Jones found foals to live 2-4 days [29]. Metallinos et al. [30] characterized the syndrome as aganglionosis depicted as a deficiency of submucosal and mesenteric ganglia from the distal small intestine to the large intestine. Associated syndromes have been found in rats, mice, and human beings [28] which are analogous to Hirschsprung’s disease [30]. Anatomically, the colon is underdeveloped resulting in the foal’s failure to eliminate fecal matter. The outcome of the syndrome is fatal. Euthanasia following a few days of birth is justified. Horse enthusiasts sometime refer to this gene as the “double-O 7 gene”. To prevent this syndrome from occurring, DNA testing is recommended to determine horses’ genetics prior to breeding. Second, the overo registration and allele are different because the registration also comprises the sabino and splash overo alleles. Horses possessing the homozygous recessive, or *oo* gene, have no overo pattern to their coats and thus not at risk for Lethal White Syndrome. Although, studies have shown that a number of tobiano horses are carriers which is a suggestion that tobiano is epistatic to frame overo (inhibiting gene) [30]. Interestingly, carriers were also found in horses without any overo coat pattern which indicates inconsistent penetrance of the mutation [30].

Champagne

Champagne is a recently discovered color allele and is sometimes considered a single, autosomal-dominant dilution gene [31]. The champagne dilution factor is apparent for horses genetically mapped as *ChCh* (homozygous dominant) or *Chch* (heterozygous). This phenotype is highly esteemed by horse breeders; however, champagne horses can easily be confused with cream colored horses [31]. Champagne horses possess hair coats with a bronze shade along with characteristic carropy



Figure 7: Red and white paint horse; notice the white coloration extends across the dorsal midline and down the extremities.



Figure 8: Silver dapple horse; the Z allele is diluting eumelanin.

spots on their skin. Their eyes range in color from green to blue, with hazel in between. Adult champagne horses have spotted pink skin around their eyes. Variations of the champagne color include amber, classic, gold, and sable. Homozygous recessive horses, or *chch* horses, have no champagne dilution [32].

Silver dapple

The ZZ or Zz, with some referring to it as *SILV*, horse is known as the silver dapple horse. These horses are altered from black to brown or reddish with a white or silver mane and tail [33] (Figure 8). According to Reissmann et al. [33], this is a pigment dilution gene that takes effect on black eumelanin with the possibility that the *SILV* protein restricts the formation of premature melanosomes [33]. This *SILV* appears to be a structural element. They also found that eumelanin horses, in particular black and bay horses that had an uncommon white mane and tail possessed the haplotype. Although Reissmann et al. [33] concluded in their study that there is relationship with the equine *SILV* haplotype and the silver phenotype, the mutations may actually be markers for the causative mutation. Horses having zz in their genetic makeup are not silver dapples.

Pearl

Another recently discovered dilution gene is the recessive pearl gene. Unlike the others, evidence of this dilution is homozygous recessive (*prlprl*) rather than heterozygous or homozygous dominant, and is often referred to as the “barlink factor”. Horses with the latter, *PrlPrl* or *PrlPrl* respectively, have no pearl dilution. Some consider this gene to be like the champagne gene. Horses with one mutation do not change the coat color of black, chestnut horses, or bay; however, apricot horses may appear when two alleles of the dilution gene are on a chestnut background. The skin of the horse is fair. Horses possessing light skin and teal eyes may be the result of the pearl dilution factor interrelating with the cream dilution factor which is sometimes referred to as pseudo-double cream dilutes. Unfortunately, there is a dearth of information regarding the pearl dilution gene.

Sabino

Sabino horses, *SbSb* (homozygous dominant) or *Sbsb* (heterozygous) are complex with a variety of paint-like or roan-like markings. Sabino may be caused by many different genes. These horses may have large areas of white coloration on their abdomen and extremities, roaned white spots on their body, chin groove, and ventral muzzle, along with interspersed white hair on their midsection [34]. According to Mau et al. [14] the sabino spotting pattern results from incomplete action of the dominant white allele. Sabinos usually have bold markings on their

face. Brooks et al. [34] proposed that the sabino and white phenotypes are heterogeneous. More precisely, the phenotypes are not the exclusive method to generate the sabino pattern [34]. Brooks et al. [34] also proposed that homozygosity at the *SB1* gene will produce an absolute or virtually absolute white phenotype. What can be confusing is that Sabinos are sometimes registered as overo because they closely resemble overo horses; however, Sabinos do not possess the overo genes. “Fully expressed” sabinos may appear evenly white in color and fortunately the foals have no risk of the Lethal White Syndrome. Horses with a genotype of *sbsb* will not possess any sabino markings.

Appaloosa

The appaloosa gene is also called the “leopard spotting” gene or commonly referred to as the “leopard complex” [35]. The location responsible for this pattern is *Lp*, although the causative gene is unknown [35]. Horses with these genetics have coats with symmetrical spotting or speckling patterns over the entire body or confined to certain areas like the loin, hindquarters, and croup [4]. These horses can also possess stippling over dark skin, plumb or upright striped hooves in black and white, and white bordering the eyes [4], as well as spotting in the area of the genitals, rectum, and muzzle [35]. Figures 9 and 10 illustrate spotting over the entire body and hindquarters, respectively. Terry et al. [35] suggested that transient receptor potential cation channel subfamily M, member 1 (*TRPM1*) and the pink eyed dilution (*p*) genes are positional candidate genes for *Lp*. Congenital Stationary Night Blindness (CSNB), as well as other eye problems, have been reported in association with the *Lp* gene in appaloosa horses.

Genetic testing

Genes comprising the makeup of a horse’s coat color may be



Figure 9: Appaloosa horse; observe the diffuse spotting pattern on the body.

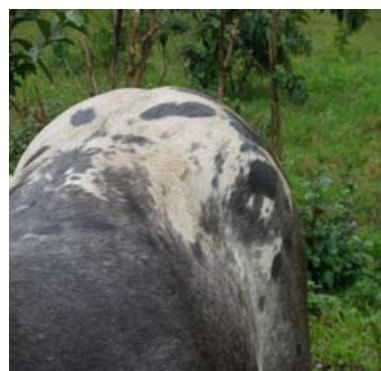


Figure 10: Appaloosa pattern isolated to the hindquarters of the horse.

identified through DNA testing, decreasing the guesswork of breeding outcomes [36]. Other studies have used bacterial artificial chromosome libraries screening for microsatellite markers and Fluorescent *In-situ* Hybridization (FISH) mapping to identify physical anchorage and orientation of equine linkage groups [37]. The gathering of genetic and physical appearance information previously took years of breeding to determine [38]. Currently, a foal's information can be established within months [38]. Bannasch [36] suggested that when faulty mutations are discovered and tests developed to investigate whether an animal is a carrier for a faulty mutation, animals that could develop a faulty mutation should not be created. He also stated that this may appear simple, although complex diseases can make breeding decisions complicated [36]. Study results support color rules such as the gray rule and the chestnut rule; however, there are potential exceptions to them [39]. This is why testing is necessary to validate or prove the exceptions when acceptance to a registry is questioned [39]. Laboratories are able to provide this service to breeders, owners, and veterinarians. These labs can provide tests including DNA typing and also carry out parentage tests. Although a horse's color can be determined simply based on physical appearance, genetic testing is available to identify physical appearances that are visually vague [8]. Possible offspring color genetic combinations can be determined by studying the dam's and sire's DNA. Studies of molecular genetics can provide identification of genes and mutations accountable for the differences in color [2]. Laboratories should be carefully scrutinized. Reputable laboratories, for example those available at university settings, are recommended to decrease lab error [38]. In addition to identifying color genes, labs can also test for genetic or inherited diseases. One disease that can be identified through genetic testing is Hyperkalemic Periodic Paralysis Disease (HYPP), an autosomal dominant trait affecting American Paints, Appaloosas, Quarter Horses, and crosses thereof potentially causing symptoms of muscle fasciculations, weakness, and recumbency [40]. Other diseases include Ileocolonic Agangiosis or Overo Lethal White Syndrome as previously mentioned is an autosomal recessive trait affecting foals of American Paint, Quarter Horses, and at times Thoroughbreds and Junctional Epidermolysis Bullosa (JEB), an autosomal recessive trait of foals seen in Belgians, American Saddlebreds, and other drafts resulting in ulcers on the skin, mucocutaneous junctions of the mouth, anus, genitals, coronary bands, and areas of pressure [40]. In addition, Severe Combined Immunodeficiency (SCID), an autosomal recessive trait of Arabians where foals are more prone to infection resulting in death and Hereditary Equine Regional Dermal Asthenia (HERDA) also known as hyperelastosis cutis, an autosomal recessive disease affecting Quarter Horses and crosses thereof resulting in sloughing of skin, open wounds, hematomas, and seromas can be identified [40]. Others include Glycogen Branching Enzyme Deficiency (GBED), Malignant Hyperthermia, and Polysaccharide Storage Myopathy (PSSM) [40]. Information on various websites is available recommending genetic testing laboratories, genetic books, and websites for the novice and professional. Additional studies are being done and advances are continually made in equine genetics with the hope that identification of more loci for single and polygenic traits are found [40].

Conclusion

Equine color genetics has been and is an important factor considered when based on breeders' personal color preference, registry guidelines, breeding outcomes, and medical aspects of equine practice. Many gene combinations and modifications can result in the array of colors and patterns seen today in equine hair coats. New gene discoveries are made, whether dominant or recessive, restriction or dilution including pattern

effects, adding to the color possibilities. Currently and most importantly in regards to equine color genetics and regardless of coat colors, the goal is not to acquire the allele combination *WW* by breeding because it is lethal resulting in embryo reabsorption or fetal death in the uterus of a mare [14]. Also, another dangerous genetic combination is the *OO* gene which is linked to Lethal White Syndrome. Through research investigation, no evidence of a Lethal Roan Syndrome in *RnRn* roans has been found at this time. Although not proven to be 100% accurate, DNA genetic testing may be recommended for owners and breeders to narrow the desirable coat color possibilities and more importantly to avoid the genetic risk of undesirable, deadly genes of offspring.

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