

CENTER FOR EQUINE HEALTH

HorseReport

SCHOOL OF VETERINARY MEDICINE • UNIVERSITY OF CALIFORNIA, DAVIS

SUMMER 2020



*Special Issue
Horse Genetics*

DIRECTOR'S Message

As an equine genetics researcher, I am particularly excited to share this special issue of the *Horse Report* with you. Inside, you will find a roadmap to many of the currently available equine genetic tests, including the AQHA “five-panel” test, and more.

The equine genome sequence was published in 2009, the result of a years-long collaborative effort by the international equine research community. This resource drastically changed how researchers approach equine genetics and accelerated the rate of discovery. Increased availability and affordability allowed the application of advanced molecular tools to equine diseases and traits. As a result, genetic tests are available in a variety of breeds.

Most available tests are for simple, Mendelian diseases and traits – those caused by a single gene or locus. Complex diseases and traits likely involve more than one gene and may be influenced by environmental effects. The 2018 release of a new equine genome sequence assembly, coupled with cost reductions that make whole-genome sequencing possible for large numbers of horses, are enabling research in these areas.

As an equine geneticist and veterinarian, I am especially interested in applying whole genome sequencing and advanced diagnostic tools to equine precision medicine. This highly individualized approach will focus on early detection and prevention of disease, taking into account both genetic information and environmental factors. The idea is to target individuals based on their clinical condition as well as their unique body chemistry and genetics.

At CEH, we are fortunate to collaborate with the UC Davis Veterinary Genetics Laboratory (VGL), directed by fellow equine genetics researcher Dr. Rebecca Bellone. A leader in the industry, the VGL offers a wide range of genetic tests for horses and other species and works closely with many breed registries. We are grateful for their contributions to this issue.

We hope you enjoy this glimpse into the fascinating world of equine genetics!

Carrie J. Finno, DVM, Ph.D.
CEH Director



UC Davis Equine Genetic Testing Services

The UC Davis Veterinary Genetics Laboratory (VGL) is a not-for-profit unit of the UC Davis School of Veterinary Medicine that provides animal parentage verification, identification, forensic services, genetic diagnostics and genetic research for a variety of species.

The VGL has been a leader in equine genetic testing and research for decades and has a long history of collaborating with UC Davis researchers, as well as researchers from other institutions, to develop state of the art equine genetic tests, many of which are featured in this issue of the *Horse Report*. The laboratory currently offers diagnostic tests for 52 equine genetic diseases, coat colors, and other traits of interest. The VGL has a very active research and test development program in equine genetics and genomics.

Visit the VGL's website at <https://vgl.ucdavis.edu/> to learn more.

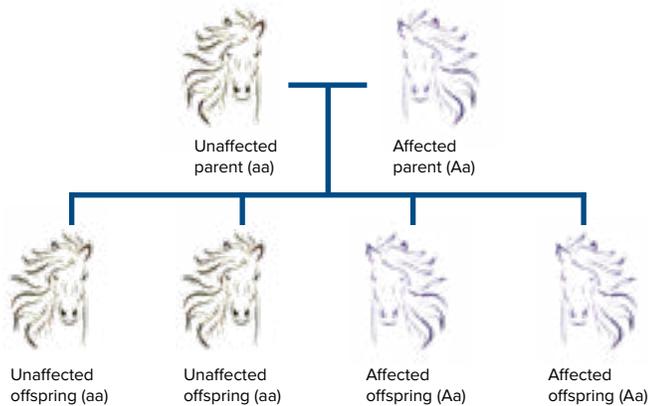


HOW GENETIC DISEASES Are Inherited

Most identified equine genetic diseases have **autosomal dominant** or **recessive** modes of inheritance. “Autosomal” means that males and females are equally affected. Here, “unaffected” and “affected” represent the **phenotype**, or presence of the disease. “Aa” and “aa”, or “BB”, “Bb”, and “bb” represent the **genotype**, or genetic combination of alleles (one from each parent) that causes the phenotype.

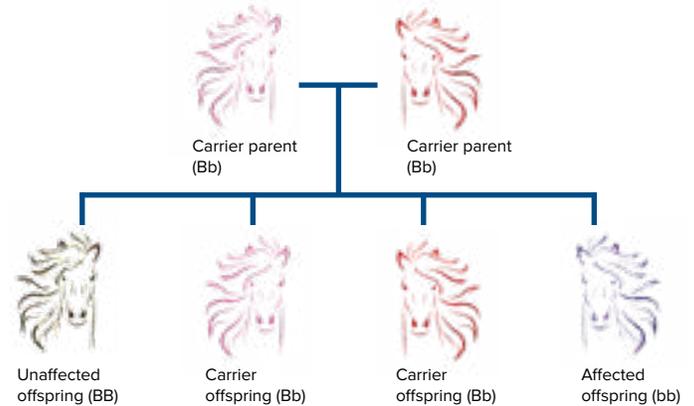
Autosomal Dominant

ONE copy of the gene variant (**allele**) is needed to have the disease/trait. Each offspring of an unaffected parent x affected parent has a 50% chance of inheriting the disease allele (A) and being affected.



Autosomal Recessive

TWO copies of the gene variant (**allele**) are needed to have the disease/trait. Each offspring of parents who carry the condition has a 25% chance of inheriting the disease allele (b) and being affected. Carriers are unaffected, but can pass the disease/trait to their offspring.



STRIKING GOLD – Twice?

When a palomino Quarter Horse colt was born in May of 1994, the Smiths chose the name Sonny's Striking Gold to mark their good fortune. Immediately smitten, Tommy Smith, then 11 years old, fell asleep in the stall with his new best friend.

The pair went on to show in multiple disciplines, winning many awards. Thanks to his calm demeanor, Sonny became a lesson and therapeutic riding horse. At 16 years old, he began to lose weight, despite being healthy otherwise. He passed away at 18 after suffering from a mysterious and debilitating wasting of muscle along his spine and hindquarters.

With Sonny's passing, the Smiths found themselves in a position that many will find relatable. How do you cope with the loss of that “once-in-a-lifetime” horse?

Since Sonny was such an important member of the family, the Smiths became interested in the possibility of cloning. Although future generations would not have the chance to know Sonny, they hoped that they could experience the next best thing. It was cost-prohibitive to pursue cloning at the time, but they worked with a company to preserve some of Sonny's cells in case it became an option in the future.

When information became available about [immune-mediated myositis \(IMM\)](#), an autoimmune disease that causes rapid onset of muscle wasting along the topline and hindquarters, the Smiths recognized signs that Sonny experienced. Sonny's DNA was tested at the UC Davis Veterinary Genetics Laboratory. The

results showed that Sonny did have one copy of the [IMM/MYH1 mutation](#). Although not the results they had hoped for, the information did make the Smiths' decision not to pursue cloning easier, and brought a degree of closure.

“Had we not been able to test for IMM and know that a genetic defect does exist, we would ignorantly have continued our cloning dream,” said Carol Smith. “As difficult as it was to find out, I am so grateful to UC Davis for their hard work to arrive at this discovery – and to develop a test!”

Sonny clearly made a significant impact on those that knew him best. Although his story has ended, it serves as an example of the power of genetic testing and the importance of genetics research to ensure the health of future generations.



HIGH FIVE – AQHA’s Dedication to Research and Success of the “Five-Panel Test”

Genetic diseases can cause significant emotional and financial stress for owners and breeders. The [American Quarter Horse Association \(AQHA\)](#) is committed to educating owners and breeders about inherited conditions so they can make informed breeding decisions. The [American Quarter Horse Foundation \(AQHF\)](#) provides funding for a wide range of research projects, granting more than \$12 million in support of equine research to date. Genetics research accounts for 20% of that total, with 53 projects funded at 12 universities.

As part of this focus on genetics, AQHA supports genetic testing as an important mechanism to ensure the future health of the breed. The “[five-panel test](#)” evaluates a horse for five known inherited diseases, ranging from mild to severe, and for which producing an affected foal may result in unnecessary suffering and financial losses. The AQHA requires testing for breeding stallions, and many breeders test both mares and stallions to make informed breeding decisions.

The “five-panel test” includes the following diseases:

[Glycogen branching enzyme deficiency \(GBED\)](#) – fatal disease of developing fetuses or newborn foals. Affected horses cannot store sugar molecules needed by the heart, skeletal muscle, liver and brain. The disease may result in late-term abortions or stillbirths. Affected foals that are born are weak with decreased muscle tone. They may exhibit low body temperatures, limb deformities, seizures, cardiac arrest, and respiratory failure. All known cases of GBED affected foals have died or were euthanized within five months after birth.

[Hereditary equine regional dermal asthenia \(HERDA\)](#) – degenerative skin disease that causes the skin along the horse’s back and neck to stretch and tear easily. It is present at birth, but commonly noticed when training begins as the friction from tack may cause lesions. Horses with HERDA can develop seromas, hematomas, and ulcerations over the back and sides of the neck. There is no treatment and affected horses are usually euthanized.

[Hyperkalemic periodic paralysis \(HYPP\)](#) – results in an excessive amount of potassium in the blood (hyperkalemia). This causes the muscles to contract more readily than normal. Horses with HYPP can experience unpredictable attacks of muscle tremors or paralysis, which in severe cases can lead to collapse and sudden death due to cardiac arrest or respiratory failure. Stress, dietary changes, fasting, general anesthesia, illness and changes in exercise can cause attacks of muscle weakness. Episodes can be controlled with appropriate nutrition and management. Both heterozygotes (N/H) and homozygotes (H/H) are susceptible to episodes of collapse, although homozygous (H/H) horses have more frequent episodes. It is important to inform your veterinarian of your horse’s HYPP genotype prior to general anesthesia, as this can trigger a paralysis episode.

[Malignant hyperthermia \(MH\)](#) – rare disease that causes a life-threatening condition in susceptible horses triggered by anesthesia drugs. Signs of MH episodes include muscle contracture, elevated body temperature, elevated heart rate, irregular heart rhythm, excessive sweating and shallow breathing. The prognosis for horses that have an MH episode while under anesthesia is poor. For horses that also have PSSM1, clinical signs can be greatly exacerbated.



◀ Examples of clinical signs of HERDA – (left) hyperextensible skin and (right) lesions on the back.

Polysaccharide storage myopathy type 1 (PSSM1) –

causes an abnormal accumulation of glycogen, the form of sugar stored in muscle. This can result in episodes of muscle stiffness and pain after exercise, also known as “tying up,” or exertional rhabdomyolysis. Affected horses may be reluctant to move and display sweating, lameness, and muscle tremors. Diet and exercise play important roles in the onset of clinical signs and some affected horses never display clinical signs. Affected horses can be successfully managed through diet and exercise.

While testing for *immune mediated myositis (IMM)* is not required by the AQHA, some laboratories include IMM/MYH1 testing as part of their Quarter Horse panel test. In addition, the VGL offers IMM/MYH1 testing as a stand-alone test.

Disease	Mode of Inheritance	Gene
GBED	Autosomal recessive	Glycogen branching enzyme (GBE1)
HERDA	Autosomal recessive	Cyclophilin B (PPIB)*
HYPP	Autosomal semidominant	Sodium channel alpha-subunit (SCN4A)*
IMM/MYH1	Autosomal semidominant	Myosin heavy chain 1 (MYH1)*
MH	Autosomal dominant	Ryanodine receptor type 1 (RYR1)*
PSSM1	Autosomal dominant	Glycogen synthase 1 (GYS1)
LWO	Autosomal recessive	Endothelin-B receptor (EDNRB)*

*Causative mutations were identified by UC Davis researchers.

Lethal white overo syndrome (LWO) – disease in American Paint Horses and related breeds in which affected foals are all white and born with an underdeveloped intestinal tract. They cannot move food through their intestinal tract and cannot defecate, leading to colic. Horses with the frame overo white spotting pattern are carriers and can produce affected offspring. There is no treatment or “cure” for LWO. Affected foals die within a few days of birth, or are humanely euthanized. The American Paint Horse Association requires the LWO genetic test for breeding stallions prior to the registration of offspring.



A horse exhibiting the frame overo white spotting pattern.

A GROWING LIST – Research Advances Lead to New Genetic Tests



The first equine genetic tests became available in the 1990's, well before the equine genome sequence was completed in 2009. Technical advances have since led to a rapid expansion in available tests. Here are a few that can be utilized to inform breeding decisions and provide diagnostic information. (For more details, please visit our [Equine Health Topics](#). For testing information, please visit the [UC Davis Veterinary Genetics Laboratory](#).)

Appaloosas

Some Appaloosas are closely related to Quarter Horses, so the five-panel test is recommended (see page 4).

Equine recurrent uveitis (ERU) – characterized by inflammation of the middle layer of one or both eyes. Cumulative damage can lead to cataracts, glaucoma, and blindness. Appaloosas are eight times more likely to develop ERU than other breeds and significantly more likely to

become blind. It is thought to be a complex disorder. However, research identified the leopard complex white spotting pattern (LP) allele as an associated ERU risk factor, with homozygotes (LP/LP) being at highest risk. Genetic testing can be used to identify which horses should be examined more frequently. Infectious organisms, particularly *Leptospira* spp. have also been associated with ERU. There is no cure, and early diagnosis and intervention are associated with the best prognosis.

Congenital stationary night blindness (CSNB) – the inability to see in low to no-light conditions. Horses with two copies of LP have abnormal cell signaling from the rods, or low light detecting cells of the retina, to the next cell in the visual pathway. Affected horses likely have normal vision during daylight, but may exhibit anxiety, apprehension, and confusion in low light. They may be reluctant to move, bump into things, or be prone to injury at night. There is no cure for CSNB, but most horses can be managed successfully. Genetic testing can be used to confirm diagnosis.

A genetic variant associated with [CSNB in Tennessee Walking Horses \(CSNB2\)](#) has recently been identified. The genetic test can be used to inform mating decisions and aid in diagnosis.

An Appaloosa homozygous for leopard complex white spotting (LP/LP). ▶



Arabians

Severe combined immunodeficiency (SCID) –

affected foals are born with severely weakened immune systems. They do not produce functional B and T lymphocytes, and cannot mount appropriate immune responses to challenges, making them highly susceptible to infections. There is no cure, and affected foals die or are euthanized within the first six months of life.

Lavender foal syndrome (LFS) – foals are unable to stand or nurse properly due to neurological impairments including seizures, hyperextension of limbs, neck, and back, and involuntary eye movements. They are born with a characteristic dilute coat color described as lavender or silver. Supportive care can provide temporary relief in some cases, but the condition is ultimately untreatable. Affected foals die or are euthanized shortly after birth. Genetic testing can be used to confirm diagnosis and identify carriers.

Cerebellar abiotrophy (CA) – caused by the progressive death of neurons in the cerebellum. Clinical signs are variable, and can include head tremor, ataxia, exaggerated movement, a wide-based stance, and inability to rise. There is no treatment for CA. Foals that show signs of CA are euthanized or restricted to life as pasture pets, as they are never coordinated enough to be ridden safely. Affected horses are also a danger to themselves because they are predisposed to accidents and injury.

Occipitoatlantoaxial malformation (OAAM) –

results from the malformation of the first two vertebrae of the neck, the atlas (C1) and the axis (C2), and the base of the skull (occipital bone), with the atlas fused to the occipital bone. This causes compression and damage of the upper part of the spinal cord. Clinical signs include abnormal head and neck carriage, reluctance to move the neck, or neck twisting. There is no treatment, and affected foals are usually euthanized. There is extensive variability to this disease, so the term OAAM describes not one disease, but a group of likely inherited malformations. There is currently one genetic test for OAAM1 that can assist in identifying carriers to assist with mating decisions. The genetic basis for other forms of OAAM are under investigation.



An OAAM affected foal (left), with an arrow indicating the asymmetric atlas, and an unaffected foal (right).



Draft Breeds

Junctional epidermolysis bullosa (JEB) – a progressive skin disorder in Belgian Draft Horses and American Saddlebreds. Affected foals develop blistering and skin lesions at pressure points that worsen with time, leaving them susceptible to severe infections. A mutation (JEB1) has been identified in Belgians and related breeds. A different mutation (JEB2) is responsible for the condition in American Saddlebreds. There is no treatment for JEB and most affected foals are euthanized.

Polysaccharide storage myopathy 1 (PSSM1) – Some draft horse breeds are at risk of developing PSSM1. The signs, treatment, and causative mutation are the same as described for PSSM1 as part of the AQHA five-panel test (see page 5).

Ocular squamous cell carcinoma (SCC) – the most common form of cancer to affect the eyes and eyelids of horses. The tumor arises in the outermost layer of skin, conjunctival, or corneal cells, with UV light (sunlight) exposure being a known risk factor. Tumors may grow rapidly and spread to adjacent tissues, causing visual impairment and destruction of the

eye. Treatment options and prognosis are determined by the size and location of the tumor(s). Certain breeds, including Belgians and Haflingers, have a genetic predisposition to ocular SCC development and a major genetic risk factor has been identified. The genetic test is helpful to identify which horses are at highest risk and should be examined more frequently.

Friesians

Hydrocephalus – the excessive accumulation of cerebrospinal fluid in the brain, resulting in severe distension of the head, giving it a large, domed appearance. Hydrocephalus is thought to occur due to an abnormal narrowing of the opening at the base of the skull. Friesian foals affected with hydrocephalus are aborted, stillborn or born with severe neurological issues that warrant euthanasia. A causative genetic mutation has been identified, and testing can assist in identifying carriers.

Dwarfism – disproportionate growth with reduced bone length of limbs and ribs. Affected horses exhibit severely shortened stature, shortened limbs relative to body size, bowed forelegs, a shortened neck, broad chest, and reduced bodyweight. A causative genetic mutation has been identified, and testing can assist in identifying carriers. There is no treatment, but affected foals do grow, albeit at a slower rate than their unaffected counterparts. Most are able to walk, trot, canter, and gallop, and some are even ridden.



◀ A dwarf Friesian



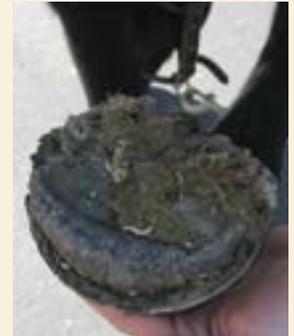
Pony Breeds

Foal immunodeficiency syndrome (FIS or Fell Pony syndrome) – found in rare native U.K. Fell and Dales Pony breeds, and crosses. It causes fatal anemia and a compromised immune system. Affected foals have abnormally low levels of red blood cells and B-lymphocytes (a specific type of white blood cell). They become progressively anemic and lack the ability to produce their own antibodies, which makes them susceptible to infections. The genetic test is helpful to identify carriers or confirm clinical diagnosis. There is no effective treatment for FIS, and affected foals usually die or are euthanized by 4 years of age.

Connemara hoof wall separation disease (HWSD) –

characterized by separation and cracking of the outer hoof wall. This can lead to ponies supporting their weight on the sole of the hoof

instead of the hoof wall, which can result in chronic inflammation, severe lameness, and laminitis. There is no treatment or “cure” for HWSD. Management through hoof care and/or special shoes may be attempted, but these options are expensive and labor-intensive. Affected animals can become severely painful despite careful management. Quality of life may diminish, and euthanasia may be necessary. Even if the condition is initially controllable, ponies may still develop laminitis over time. The genetic test can be used to test mating pairs to avoid producing affected offspring.



The hoof of a pony with HWSD.

Warmbloods and Thoroughbreds

Warmblood fragile foal syndrome type 1 (WFFS) – a defect of connective tissue characterized by hyperextensible, abnormally thin, fragile skin and mucous membranes that are subject to open lesions. Affected horses may also have hyperextensible limb joints, floppy ears, accumulation of fluid (hydrops), subcutaneous emphysema, hematomas, and premature birth. Newborn foals are euthanized shortly after birth due to the poor prognosis. Due to the severity of the disease, there is no treatment or “cure.” Genetic testing prior to mating is advisable to avoid producing affected offspring. There are no known health problems associated with carrier status. This allele was detected in the Thoroughbred at a low frequency, so testing is also recommended for that breed.



Equine familial isolated hypoparathyroidism (EFIH) – An inherited form of hypocalcemia, or low calcium concentrations in the blood, has been identified in Thoroughbred foals. Hypocalcemia

can impair limb movement and weaken bones. In severe cases, it can lead to seizures, muscle fasciculations, intestinal problems, heart issues, and ataxia. Genetic testing is now available through the UC Davis Veterinary Genetics Laboratory.

Table of equine inherited diseases for which genetic tests are available, as discussed. For information about additional tests, please visit the UC Davis Veterinary Genetic Laboratory website (<https://vgl.ucdavis.edu/services/horse.php>).

Disease	Breed(s)	Mode of Inheritance	Gene
SCID	Arabian	Autosomal recessive	<i>DNA-dependent protein kinase (DNA-PK)</i>
LFS	Arabian	Autosomal recessive	<i>Myosin VA (MYO5A)</i>
CA	Arabian	Autosomal recessive	<i>MutY homolog (MUTYH)*</i>
OAAM	Arabian	Autosomal recessive	<i>Homeobox D3 (HOXD3)*</i>
ERU	Appaloosa	Complex	<i>Transient receptor potential cation channel subfamily M member 1 (TRPM1)*</i>
CSNB	Appaloosa, Miniature Horse, Knabstrupper, Noriker	Autosomal recessive	<i>Transient receptor potential cation channel subfamily M member 1 (TRPM1)*</i>
CSNB2	Tennessee Walking Horse	Autosomal recessive	<i>Publication pending*</i>
JEB	Belgian Draft Horse, American Saddlebred	Autosomal recessive	<i>Laminin 5 γ2 chain (LAMC2), laminin 5 α3 chain (LAMA3)</i>
PSSM1	Draft	Autosomal dominant	<i>Glycogen synthase 1 (GYS1)</i>
Ocular SCC	Belgian Draft Horse, Haflinger	Autosomal recessive	<i>Damage-specific DNA binding protein 2 gene (DDB2)*</i>
Hydrocephalus	Friesian	Autosomal recessive	<i>β-1,3-N-acetylgalactosaminyltransferase 2 (B3GALT2)</i>
Dwarfism	Friesian	Autosomal recessive	<i>Beta-1,4-galactosyltransferase 7 (B4GALT7)</i>
FIS	Fell Pony, Dales Pony	Autosomal recessive	<i>Sodium/myo-inositol cotransporter (SLC5A3-P446L)</i>
HWSD	Connemara Pony	Autosomal recessive	<i>Serpin family B member 11 (SERPINB11)*</i>
WFFS	Warmblood, Thoroughbred	Autosomal recessive	<i>Procollagen-lysine, 2-oxoglutarate 5-dioxygenase1 (PLOD1)</i>
EFIH	Thoroughbred	Autosomal recessive	<i>Publication pending*</i>

*Causative mutations were identified by UC Davis researchers.

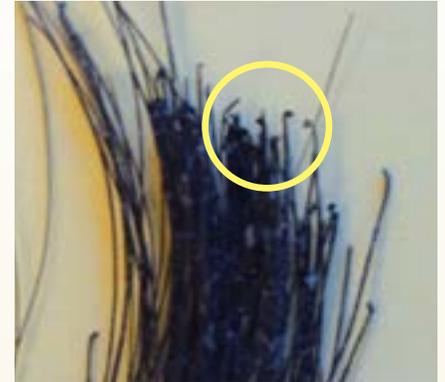


10 THINGS

You Might Not Know About Equine Genetics



1 DNA is found in the nucleus of cells. Samples submitted for genetic testing are usually hair, blood, or tissue. [Hair samples](#) must contain the “bulb”, which looks like a tiny ball at the root of the hair. This is where the DNA is located. When submitting hair samples, make sure to pull, not cut, the hairs so the bulb is present. Blood and tissue samples require additional steps, such as an anticoagulant (for blood) or freezing (for tissues). Always follow the testing laboratory’s instructions for sample submission and contact them with questions.



2 There is no age limit for DNA testing. An animal’s DNA profile does not change over time, so a horse can be reliably tested at any age. Genetic tests can be performed for foals, but it is recommended to submit 30 - 50 tail hairs since the bulbs on foal hair are smaller than adults and more are needed.



3 Parentage testing is based on exclusion. The offspring, dam, and potential sires are tested for a number of markers, and the offspring is compared to the potential parents for each marker. The offspring must have received one allele for each marker from each parent. It is possible to do [parentage testing](#) with a sample from only one parent (~95% accuracy), but results are more accurate (>99%) with both. Accuracy will decrease when the potential parents are part of a large group of closely related animals.



4 [SynchroGait™](#) tests for a genetic variant that facilitates lateral gaits (ambling and pace). Owners can use this test to identify the natural ability of young horses for gait performance, and use the results to make training decisions. Breeders can use the test to select for or against this mutation, depending on what is desirable in their chosen discipline.



5 Horses have 3 base coat colors that are controlled by the interaction of 2 genes.

The [coat colors](#) chestnut, bay, and black are determined by horses’ genotypes at the MC1R gene (extension (E) locus), which controls the production of red and black pigment, and the ASIP gene (agouti (A) locus), which controls the distribution of black pigment to the mane, tail, lower legs, and ear rims (points), or uniformly over the body.



6 Dilution factors modify base coat colors. These include cream, champagne, dun, pearl, silver, and mushroom. The resulting coat color depends on the combination of the base color genotype and the dilution factor genotype. For example, a horse with one copy of the cream allele on a chestnut base color will be palomino whereas on a bay base color it will be buckskin. Modes of inheritance and causative mutations have been identified for these dilution factors, and genetic testing is available. For more on the basics of horse coat color, the Veterinary Genetics Laboratory provides an online [guide to equine coat color genetics](#).



7 Horse size is highly heritable. Height in horses is determined by the interaction of genetic and environmental factors (such as nutrition). Four genetic variants have been identified that account for >80% of horse height. One variant has a particular influence on height in warmbloods and a different variant has a strong influence on height in Shetland ponies and miniature horses. Mutations related to dwarfism have also been identified in some breeds ([dwarfism](#) in Friesians, [skeletal atavism](#) in Shetland ponies and American Miniature horses). Additional unidentified genetic variants that influence height in horses are likely.



8 Equine genetic ancestry tests are available, but there are a few things to keep in mind. Equine ancestry tests, or “breed prediction” tests, compare a horse’s DNA to horses in a reference panel. Results are dependent upon the breeds and number of horses of each breed in that panel (which can vary by test provider). Some horse breeds are not very genetically distinct from one another, and many breeds have influenced the creation of other breeds, which can complicate results. These tests can report the probability that a certain breed is an ancestral breed for a horse, but not the proportion or percent of that breed in a horse’s genetic makeup. The larger the number of breeds involved in a cross, the lower the probability of a clear result.



9 Genetic testing can be performed for embryos. Embryos recovered from uterine flushes as part of embryo transfer procedures can be tested to determine gender and genetic traits prior to implantation in the recipient uterus. This allows for selection of embryos that have the desired sex, coat color variants, or that are free of known genetic diseases.



10 In the future, horses will be able to have their entire genome sequenced. Whole genome sequencing of modern and ancient horses has provided a wealth of information to researchers. As this technology becomes more affordable (currently ~\$1600 per animal), whole genome sequencing of individual horses is likely to become more accessible. Coupled with advanced knowledge in equine health, whole genome sequencing will provide veterinarians, owners, and breeders with a powerful tool to tailor training, management, and health care to the individual animal.



www.vetmed.ucdavis.edu/ceh

Mail ID 1415
Center for Equine Health
School of Veterinary Medicine
University of California
One Shields Avenue
Davis, CA 95616-8589

The *Horse Report* is published by the University of California, Davis, School of Veterinary Medicine: Michael D. Lairmore, dean; Carrie Finno, CEH director; Tom Hinds, director strategic planning and communications; Amy Young, editor; Katie Blakewell, Don Preisler, Carolyn Sawai, Rob Warren, Trina Wood, contributors. The Center for Equine Health is supported with funds provided by the State of California Pari-Mutuel Fund and contributions from private donors.

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Precision Medicine – The Next Frontier

Thanks to advanced molecular tools, researchers are now able to understand the equine genome on a deeper level than ever before. In the future, genetic data and advanced diagnostics could be used to tailor medical treatments to an individual horse's genetics, its environment, and the interactions between the two. The goal is to be able to evaluate, for example, how a particular patient is likely to respond to one drug versus another, making it possible to target treatments for the most successful outcomes.

Dr. Callum Donnelly, a PhD student in [Dr. Finno's laboratory](#), is excited to be partnering with CEH and Platinum Performance on the "Pioneer 100 Horse Project", a first-of-its-kind precision medicine study in horses. This project is gathering a variety of medical and genetic data on 100 CEH teaching herd horses to lay the groundwork for precision medicine approaches in multiple fields.



Horses undergo lameness exams as part of the Pioneer 100 Horse Project.

