2018 FALL ICHO GAZETTE

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Hello ICHO Members and Guests! This is a "musing from my armchair," a thoughtful, thankful (isn't it the season?!) reflection of the past year with Curlies...

This has been the most information revealing year that I've experienced in my time with Curlies! The amount of genetic information has been exciting, surprising, enlightening, forward thinking, and scientific. I think I just defined what ICHO is all about! I'm so very proud of this registry and its mission, goals, and everything Curly. When I learned about ICHO many years ago, the defining goal that reached me was it's scientific direction of defining a Curly. Many years went by, curly friends and horses passed….then BOOM! The first curl genes were isolated recently! And then another, and there must be more! How exciting is this?! To me, this is an "EUREKA!" moment! Scientists around the world, Curly friends and supporters have all contributed to knowing Curlies scientifically better. Aren't they more than we ever thought, in a delightful, pleasantly surprising way?!....I think so. Curlies have so much to give!

And there is another Curly gene article inside this issue. Happy Holidays to all!

Joan Henning, ICHO President.

Don't forget to order your 2019 ICHO calendar! This calendar features many aspects of the Curly gene project. See ordering information inside!

GAZETTE AD DEADLINE

We now have a new Gazette ad deadline schedule for submitting ads as follows:

WINTER: Dec 15

SPRING: Mar 15

SUMMER: June 15

FALL: Sept 15

Ads that are received after the deadline will be run in the next following issue. Payments must also be made before the deadline. All ads and payment must be sent to the office. Thanks!

ARTICLES- We are always looking for interesting articles for the Gazette. The article could be about training, health tips, an interesting story about a Curly or anything of interest horse related. Please send all articles to the office before the deadlines. Awaken the writer within you!

INSIDE THIS ISSUE

ICHO now offers DNA testing for:

Ancestral only TX A&M		*$25.00*
Parentage Only U of KY		*$30.00*
CA- (Cerebellar Abiotrophy)VetGen		*$35.00*
Genetic DNA Panel Testing		*$ 99.00*
KRT25 Curly Gene	*$35.00*	
SP6 Curly Gene	*$35.00*	
KRT25 & SP6 Curly Genes	*$65.00*	

Visit the website for more info or contact the office.

Gazette Ad Specials!

Stallion Station Ad– *only* $10.00 per issue, $40.00 for the year, color ad.

Business Card Ad– *only* $10.00 per issue, $40.00 for the year, color ad

All color Ads at Black and White Prices! All Gaz Color Ad fees reduced!!

Full Page Color $40.00, 1/2 Pg Color $20.00, 1/4 Pg Color $10.00, Classified $10.00

Submitting ads: *Pay for ads online on our webpage– http://ichocurlyhorses.org

All ads must be copy ready in word doc format or jpg, png. Since we have reduced our prices we need the ads in ready to print format. For photos png is preferred since it has the best resolution. We can no longer accept pdf format because it does not insert clearly for the Gazette editing process.

All Ads must be sent to the office with payment. Ads also appear on the ICHO webpage.

Thanks! office@curlyhorses.org Gazette editor– Joan Henning

https://www.cafepress.com/icho.1082655410#

2019 ICHO Calendars are available now!

The 2019 ICHO Calendar is now available at- www.cafepress.com/icho

IMPORTANT- You must choose Jan 2019 when choosing the 2019 calendar!

Be sure to go to the calendar month links at top of the ICHO CafePress page to get items with the month page on them. These make great Xmas gifts!

Thank you to all whole participated in making our Calendar and also a huge thanks for Sheryl for getting the pages all together!

ICHO Gift center at Cafepress!

Need gifting ideas? Cafepress has an assortment of ICHO Curly horse items featuring any of the calendar pages and the ICHO logo. Visit the website for the calendar and go shopping!

https://www.cafepress.com/icho.1082655410#

Shirts, totes, hoodies, mugs, hats, magnets, stationary, clocks, children items, and more, and of course.....the 2019 Calendar!

ICHO 2018 AGM

MACKINAC ISLAND, MICHIGAN

Left, ICHO Members and friends gathered at the Grand Hotel with a taste testing of the world famous Mackinac Island fudge, a sweet tooth lovers dream! Right—front row L-R, Terry Schmidt and Dr. Mitch Wilkinson, back row-Joan Henning and Paul Dennis.

Grand Hotel accommodations were simply that, Grand!

Attendees engrossed with Dr. Mitch Wilkinson's presentation on Curly gene inheritance.

Left clockwise, Paul Dennis, Joan Henning, Terry Schmidt, and Dr. Wilkinson. Right, Joan Henning, ICHO President, awarding Dr. Wilkinson with the first ever Sandra Hendrickson research award.

Above, discussion with the genetics presentation. Left, the mode of transportation on Mackinac Island, first class to the Grand Hotel!

Horses everywhere, even with a little rain, no one recalls getting wet. Right, Joan Henning, Julia Hoffman, and Jill Simpson taking in the sights. Below, Ann and Paul Dennis on the beautiful Victorian streets, overhead is even sweet with cotton-candy skies!

Time to grab a horse drawn taxi and head home. Above left, Julia Hoffman and Jill Simpson on the ferry heading to the mainland, Mackinaw City,

Good bye to the beautiful sunrises on Lake Huron, left, a view from the pier on Mackinac Island looking at Mackinaw City.

Happy Holidays from ICHO!

Curly Horses

A Mysterious Tale of Two Transcription Factor Mutations

By Dr. Mitch Wilkinson

Curly Jim

In 1956, two young, curly-coated stallions were bought by a man from Mountain View, Missouri, named Vic Clemens. Clemens bought the horses at an auction in Tennessee and had them shipped by train to Mountain View. The horses resembled Missouri Fox Trotter horses in build; they also were gaited. At the auction, Clemens could not find any information about where the horses came from and who were their previous owners. The background of these horses is still unknown today. (11,12)

One of the two curly stallions was subsequently killed after becoming tangled in a barbed wire fence, but the remaining stallion was named Curly Jim. Curly Jim was about three years old at the time, and his training was begun by a 16-year-old local teenager named Johnny Brooks. (11)

The chestnut colored stallion had several owners throughout his life.

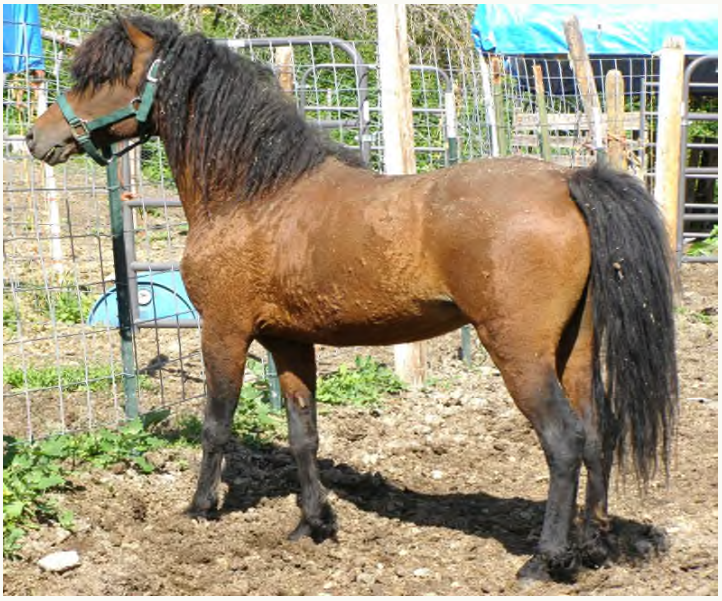
“Bill” Ed Tune and subsequently Gurn Hodge also owned Curly Jim. (11 ,12)

Two curly horses arrive by train to Mountain View, Missouri

Curly Jim was pasture bred to many mares. Recent genetic tests have confirmed that a line of curly-coated ponies known as the McKay ponies owe their curly coats to Curly Jim. Due to lack of records, the McKay pony line and its connection with Curly Jim

had been questioned, but tests have confirmed that Curly Jim was the founding sire. (8, 20)

McKay Ponies



Gentleman Jim

Kopper Kid

Besides founding a line of curly-coated ponies, Curly Jim was responsible for a line of riding horses. Curly Jim's line of gaited curly-coated saddle horses became one of the most popular lines of curly-coated horses. In the 1950's and 1960's, Missouri Fox Trotter horses were bred intensively in the region where Curly Jim resided. Since Curly Jim was gaited and had similar conformation to Missouri Fox Trotters, it was only natural that his line became mixed with Missouri Fox Trotters. Due to extensive breeding with Missouri Fox Trotters, the Curly Jim line will forever be associated with the Missouri Fox Trotter breed.

The connection of the Curly Jim line to the Fox Trotter breed occurred through his daughter Blaze. Curly Jim was bred to a grade mare known as the Bradford Mare. The resulting foal was Blaze. Blaze inherited the curly gene from her father and was curly coated.

Blaze was subsequently bred to Walker's Merry Lad, a renowned, straight-haired Missouri Fox Trotter stallion who was standing at stud in the Mountain View, Missouri, area at that time. The breeding resulted in a gaited, curly-coated stallion named **Walker's Prince T**. Walker's Prince T was a curly stallion that was used extensively and bred not only to other curly horses but also to many Missouri Fox Trotter mares. Eventually, there were three stallions who carried the Walker's Prince T name. These stallions not only passed on the unique curly coat of Curly Jim, but also the gait mutation from the Missouri Fox Trotter mares that were bred into the line. (9, 10, 12)

Walker's Prince T

The Pedigree of Walker's Prince T

The original Walker's Prince T was owned by Lester Tunes. Johnny Brooks owned Walker's Prince T (II). Walker's Prince T (II) was a direct descendent of the horse Brooks trained when he was 16 years old, the great-grandson of Curly Jim. (11)

The combination of gait and the unique curly coat which will not shed mane and tail hair during the summer months makes the Curly Jim line of curly horses extremely popular. Interestingly, both traits are due to mutations within genes that produce **transcription factors**. In order to understand the mechanisms involved in producing these two traits, gait and coat curl, we need to investigate what transcription factors are and how they affect organisms.

Transcription Factors

All the information needed for any living thing to live, grow, and reproduce is stored in its DNA. A fundamental question is **how** and **when** the information coded within the DNA molecule is utilized by the organism.

Basically, the information coded on the DNA molecule is transferred to a messenger RNA molecule (**mRNA**) which in turn

transfers the information to transfer RNA (**tRNA**) molecules. The transfer RNA molecules work within a cell organelle called a **ribosome** which produces proteins. Within the ribosome, tRNA acts as an adaptor to convert DNA information to proteins by recognizing the mRNA codon (three base RNA code) at one end and binding to a single, specific amino acid at the other end. Transfer RNA molecules assure the proper order and kinds of amino acids which when bound together in long chains will become **proteins**.

All the physical characteristics of an organism (known as the **phenotype**) are made up of thousands of proteins. There are also water, minerals, and fats, but all the living functions and the basic framework of all living things are made of proteins.

The reading and transfer of the DNA information to mRNA is known as **transcription;** whereas, the assembly of proteins in the ribosome is known as **translation**. (15)

DNA → mRNA → ribosome + tRNA → Protein

Transcription **Translation**

What Transcription Factors Do

Transcription factors help to regulate the timing of the information that is being transcribed from the DNA molecule to the mRNA molecule. Transcription factors are themselves proteins that are produced by **genes** (discrete segments of DNA at specific locations called **loci**). In order for any organism to live, essential proteins must be produced in the correct amounts, in the proper sequence, and at the correct time. This complex interaction of one group of essential proteins regulating another group of essential proteins is where transcription factors take a critical role.

Transcription factors control the rate of transcription of genetic material from DNA to mRNA.

Without regulation the vital processes of the cells would be random chemical reactions which would produce uncontrolled amounts of proteins that would be incompatible with life processes.

Transcription factors bind to the DNA molecule to regulate the rate of transcription, but they do not regulate transcription by themselves. Other proteins like coactivators (speed up transcription) and corepressors (slow down transcription) work with transcription factors to help regulate transcription. This complex relationship is especially important during fetal development when the timing and amounts of proteins help form the developing animal.

As we will see, mutations within two genes that produce two critical transcription factors produced the iconic traits of coat and gait associated with the Curly Jim line. (6, 15)

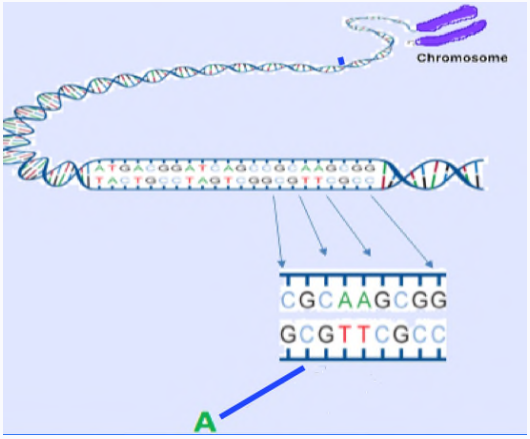
SP6 and DMRT3 Genes

In **August of 2012**, the prestigious science journal, *Nature*, published an article by an international equine genomics group which included Dr. Leif Andersson from Uppsala University in Sweden and Dr. Gus Cothran from Texas A&M University in the U.S. This article showed that in part, gait in horses is due to a **nonsense mutation** in a gene known as **DMRT3** which is located on the equine 23rd chromosome. The DMRT family of transcription factor proteins made from the DMRT3 gene have a unique and distinct binding mechanism for DNA to help regulate the timing of information transferred to mRNA. In the case of the mutation that causes gait, the DMRT3 gene had a misspelling of the DNA code. This

misspelling of the DNA sequence involved a single base pair which is known as a **SNP**. (14)

Misspellings happen on a regular basis as DNA replicates. On average, a misspelling occurs in one out of every **300** base pairs. This is one mechanism that allows **mutations** to occur in nature. Most of the time, the misspellings occur in areas of the DNA that are not being used, but every so often, the misspelling occurs in an area of the DNA chain that codes for a protein. This area of the DNA chain is known as a **gene**. Even then, the DNA can compensate due to multiple spellings of amino acids. Remember, DNA uses a three-letter base code for each amino acid within the protein sequence. Usually, when the altered spelling is translated to mRNA, it will still produce the same or slightly different protein. (15)

In this case, the normal base cytosine or C was substituted with an adenine or A. This produced a triplet code that was by chance a codon for the mRNA to stop reading the DNA molecule. This is known as a **stop codon**. When the mRNA encounters a nonsense mutation, an unreadable triplet base code**,** or in this case, a code to stop reading the DNA chain prematurely, it stops transcription of the DNA chain which results in a defective and truncated protein.



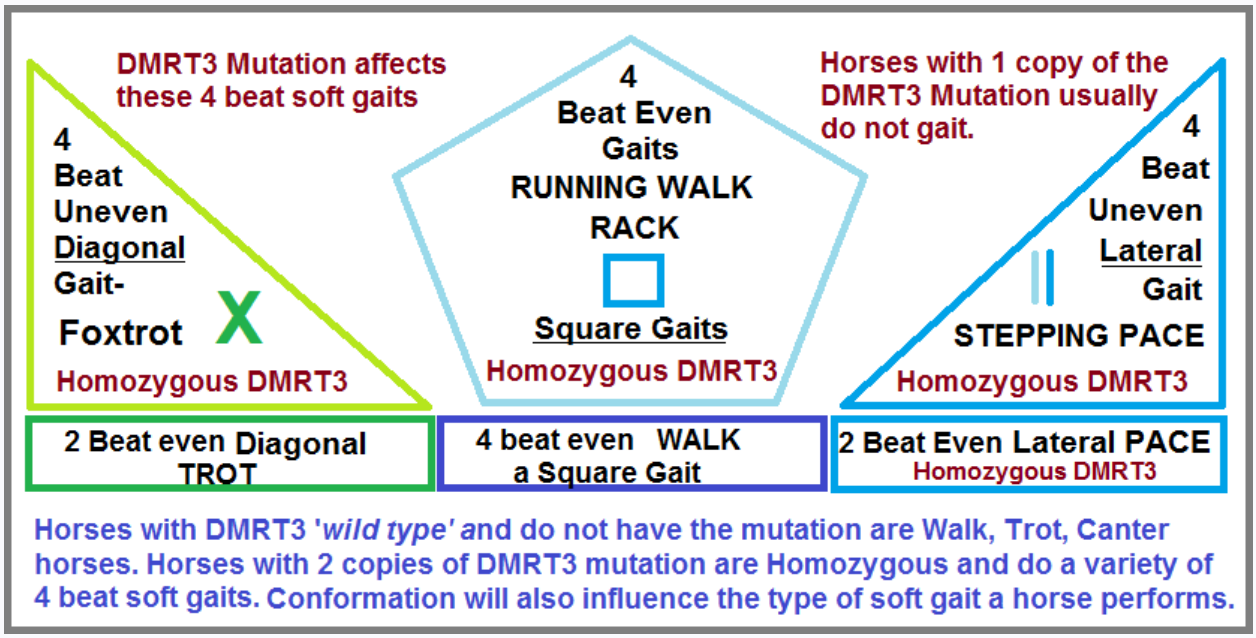
23rd equine chromosome with A substituted for a C resulting in a premature Stop Codon

The misspelling or mutation within the DMRT3 gene caused 174 amino acids to be left off the protein which is an essential transcription factor needed in the development of spinal neuron circuitry during fetal development. The shortened DMRT3 transcription factor causes a defect in the horse's spinal nerves which allows the horse to gait. (14)

This discovery of the mutation within the DMRT3 gene is the first step in understanding gait in horses. There is a large variety of gaits in equines. Other mutations which are thought to combine with the DMRT3 mutation to produce the different gaits are yet to be found.

In the shorthand notation of genetics which shows the base substitutes at specific spots or **loci** where the two different spellings of the gene occur, the DMRT3 mutation is shown with an **A** for the mutated gene and **C** for the wild type or non-mutated version. Each version of the gene is known as an **allele**. (15)

Around 140 curly coated horses have been tested for gait. Most have been from the Curly Jim line. Horses that have tested AA and have received the mutation from both parents show gaited characteristics. Horses that test AC or have only one copy of the DMRT3 mutation can have a softer ride, but that trait is inconsistent. These horses generally do not gait in tested curly horses. Earlene "Bunny" Reveglia has made a very useful chart describing the DMRT3 gene's influence on gait in horses.



DMRT3 Gait Chart- Diagonal Gaits*- the diagonal 2 beat trot on the left (2 diagonal hooves hit the ground at same time) and the DMRT3 mutation soft uneven gait 4 beat diagonal Foxtrot top left (each hoof hits independent of the other diagonally).*

Square Gaits*- Center is the 4-beat square gait walk (each hoof hits the ground independently with equal spaced timing). Center top is the DMRT3 mutation soft 4 beat gait the Running Walk and Rack; they are also square gaits. (Each hoof hits the ground at separate evenly spaced internals).*

Lateral Gaits*- On the right, the 2 beat Pace is also the result of the DMRT3 mutation (Two hooves hit the ground at the same time on one side of the horse, right side, left side with a suspension in the middle). The right top is the result of the DMRT3 mutation and is a 4-beat uneven lateral Stepping Pace gait. (Each hoof on the lateral side hits independent of the other on one side, then other side.)*

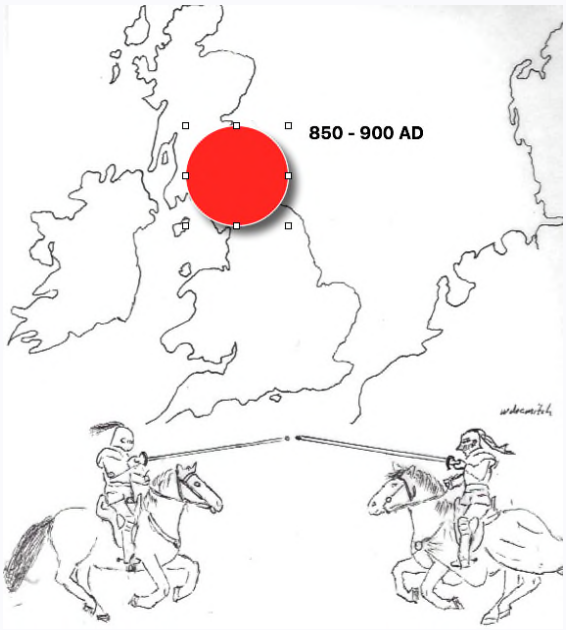
Horses with Wild Type DMRT 3 do the walk, trot, canter, gallop. Horses with two copies of DMRT3 mutation do a variety of soft gaits that is also dependent on their own conformation and ability. Horses with only 1 copy of the DMRT3 mutation generally do not do the soft 4 beat gaits. (7)

It is important to note that “gait” in the Curly Jim line is entirely independent from curly coat producing genes. Gait and curl are different inheritances on different chromosomes.

In 2016, a scientific study was published about the origin of gaited horses. The paper concluded that the DMRT3 mutation occurred with the birth of a single horse that lived somewhere

between 850 AD and 900 AD in England. One of that horse's parents had a misspelling of the DMRT3 gene during the division of the cells that would become sperm or egg cells for reproduction. Having a gait would probably not be an advantage in nature; that is why gaited horses are very rare in the wild horse populations around the world. The horse that was born with gait in England during the middle ages did not come from elsewhere, it was born due to a chance genetic mutation in medieval England. The preference of riders for gaited horses guaranteed the mutation's continuance through the ages. (16,18)

The Curly Jim line like all gaited horses has a direct link with a unique horse born long ago in medieval England.



The DMRT3 gene mutation originated in medieval England.

In the future, the complete genetic picture of gait in equines will be known, but for now, the mutation in the DMRT3 gene on

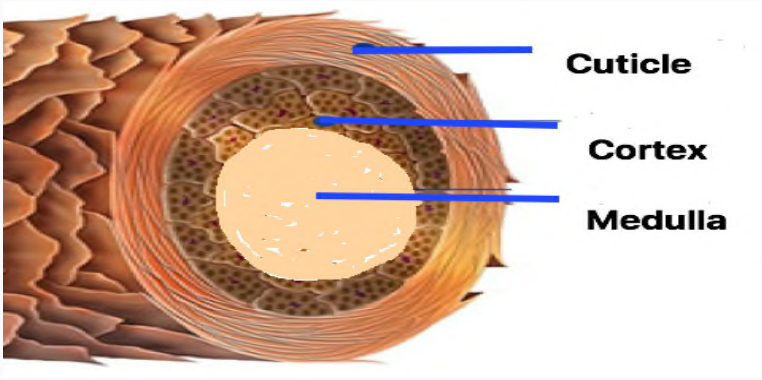
the 23rd equine chromosome is the first step in our understanding all forms of gait.

Isolation of the KRT25 Mutation

In **November 2017**, the scientific journal, ***Genetics Selection Evolution*** published the results of a scientific study that was conducted four years prior to publication. Dr. Laurent Schibler and his team in France at the University of Paris in coordination with Dr. Gus Cothran at Texas A&M University in the U.S. had isolated the first curly gene. This gene mutation that produced curly coats in horses was a mutation within a keratin gene.

The missense mutation found in KRT25 is a mutation of a specific hair producing keratin gene in the **11th equine chromosome** which produces a type 1, inner root sheath specific keratin protein that is essential in the assembly of alpha keratin protein complexes required for the proper construction of the hair shaft. A **missense mutation** is a DNA misspelling which is still readable by the mRNA, but which produces an alternate amino acid within the protein chain. This produces a slightly different protein. (1)

The hair curl that is produced by KRT25 mutation is a consequence of the abnormal structure of the hair shaft itself. Along with curl, brittleness is a characteristic of the hair shaft's abnormal structure. The brittleness is thought to increase with UV light which is at its peak during the summer months. (8)

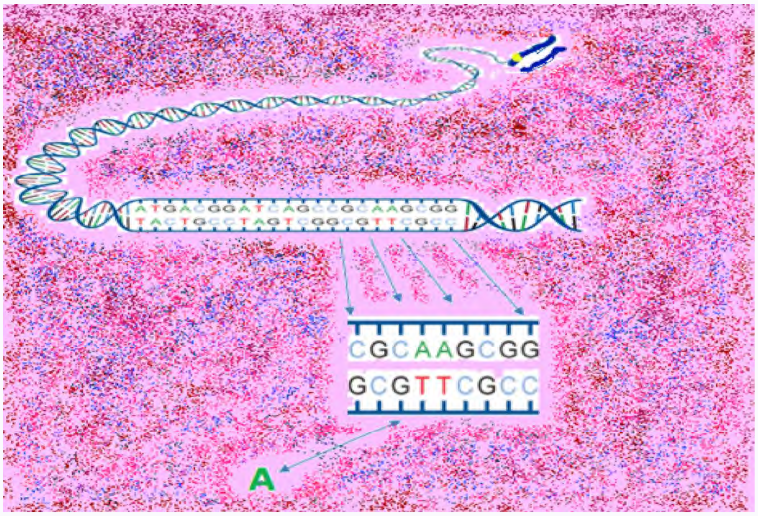


Cross section of hair shaft

The strength of any hair shaft is due to the sulfur bonds present in the middle layer called the **cortex**. The keratin within the cortex contains large amounts of the amino acid cysteine, which allows the keratin molecules to bind together in a helix shape when forming sulfur bonds. In the case of the KRT25 mutation, the hair shaft is bent or curled, but at the expense of strength. (13)

A recent study by German researchers found that hair produced by the KRT25 mutation in many cases lacked a medulla. The exact function of the medulla is not known, but the lack of a medulla shows the unusual structure associated with KRT25 mutation produced hair. (19)

This **missense mutation** found within the KRT25 gene is a consequence of the misspelling of a single base pair of the DNA chain or SNP. Whereas the normal KRT25 gene at the locus(location) of the mutation normally has a guanine or G, the mutated version has an adenine or A. (1)



Substitution of an A for a G in the 11th equine chromosome within the KRT25 gene

When a mutated version of the KRT25 mutation is passed on to the foal by both parents, the foal is said to be **homozygous** for the mutation. This is shown in the genetic shorthand as **AA**. The AA combination produces horses with sparse brittle manes and tails.

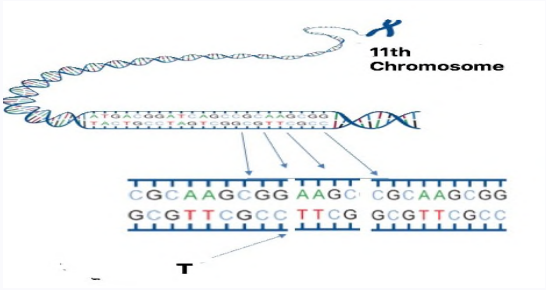
Subsequent testing of a large number of American curly horses demonstrated that the KRT25 mutation was found the majority of horses tested but testing of the Curly Jim line of horses revealed that the KRT25 mutation was not present in this population. (20)

Discovery of the SP6 Mutation

In **April 2018**, Dr. Ottmar Distl and his team in Germany published an extensive study which included not only histological studies of curly horse hair, but also the isolation of a second curly horse gene mutation in a gene which produces a transcription factor protein. The gene is known as **SP6**. The SP6

gene is found on equine chromosome #11 like KRT25, but this mutation is within a gene which codes for critical proteins during fetal development. The paper was published in the scientific journal ***Scientific Reports*****.** (19)

It was found that the Curly Jim line's coat was produced by a mutation in the SP6 gene in which a misspelling of the DNA occurred. This was again a **missense mutation** where a thymine base or **T** was substituted for a cytosine base or **C**. This type of mutation was also a SNP which is the misspelling of a single base in the DNA chain.



*11^{th} equine chromosome showing substitution of a thymine(T) for a cytosine(**C**) in the SP6 gene*

Unlike the KRT25 mutation which happened within a keratin producing hair gene, the SP6 gene mutation happened within a **transcription factor** producing gene which then produced a slightly altered transcription factor protein. The slight variation in the protein produced by the mutated version of the SP6 gene was thought to affect the shape of the forming hair follicles during fetal development. Instead of being round, the **inner root sheath** or hair molding component of the follicle was oval in shape. Oval shaped hairs curl as they grow. Brittleness was lacking in hairs produced with the SP6 mutation; the result was a strong, curly

coat. This curly coat was distinctly different from the one formed by the KRT25 mutation. (19)

The SP6 gene produces two similar transcription factor proteins, epiprofin and **SP6 transcription factor**. These two transcription factor proteins are critical during fetal formation of teeth, limbs, lungs, and hair follicles. A large change in these important SP6 proteins can have devastating effects for the animal, but a very small change resulted in a horse with a curly coat. (6)

Gaited horse with heterozygous SP6 mutation

Gaited horse with homozygous SP6 mutation

Horses of the Curly Jim line that are homozygous are associated with an unusual winter, body coat which has been described as micro-curl or brillo-pad. The curls are extremely tight and curl upon themselves. (8)

Micro curls or "Brillo Pad" curls are tight, short, and coarse. They make up the body coat in horses with Curly Jim homozygous inheritance.

Although KRT25 and SP6 are both curly producing genes on the 11th horse chromosome, they are distinctly separate genes that are inherited separately and independently. They can be **inherited simultaneously** in the same individual.

It was found by Dr. Distl's research group that the effects of KRT25 mutation do affect the characteristics of the SP6 mutation when both are found on the same individual. Because KRT25 affects the basic structure of the hair shaft itself and SP6 is more involved with the shape of the hair as determined by the inner

root sheath of the follicle, an oval shaped hair shaft can be brittle if KRT25 is also present. This effect is termed **epistasis**. (6)

An example often sited to illustrated epistasis is shown below:

Epistatic Gene Relationships

In classical genetics, if genes A and B are mutated, and each mutation by itself produces a unique phenotype but the two mutations together in the same individual show the same phenotype as the gene A mutation, then gene A is **epistatic** and gene B is **hypostatic**.

A classic example of the epistatic effect of one gene over another is found in albinism. The system of genes that determines skin color, hair color, and eye color in humans and animals is independent of the gene responsible for **albinism** (lack of pigment). The gene for albinism is an **epistatic** gene, and the genes for brown hair and other color traits are hypostatic to the albinism gene. The individual still inherits the genes for brown hair and blue eyes, but these genes can't be expressed due to the presence of the gene for albinism.

Epistasis

The genes for brown hair and blue eyes are still present but cannot be expressed due to the presence of the gene for albinism.

Epistatic genes and hypostatic genes are two separate and independent gene mutations that interact on one aspect of the organism by chance. Epistasis is not **dominance**.

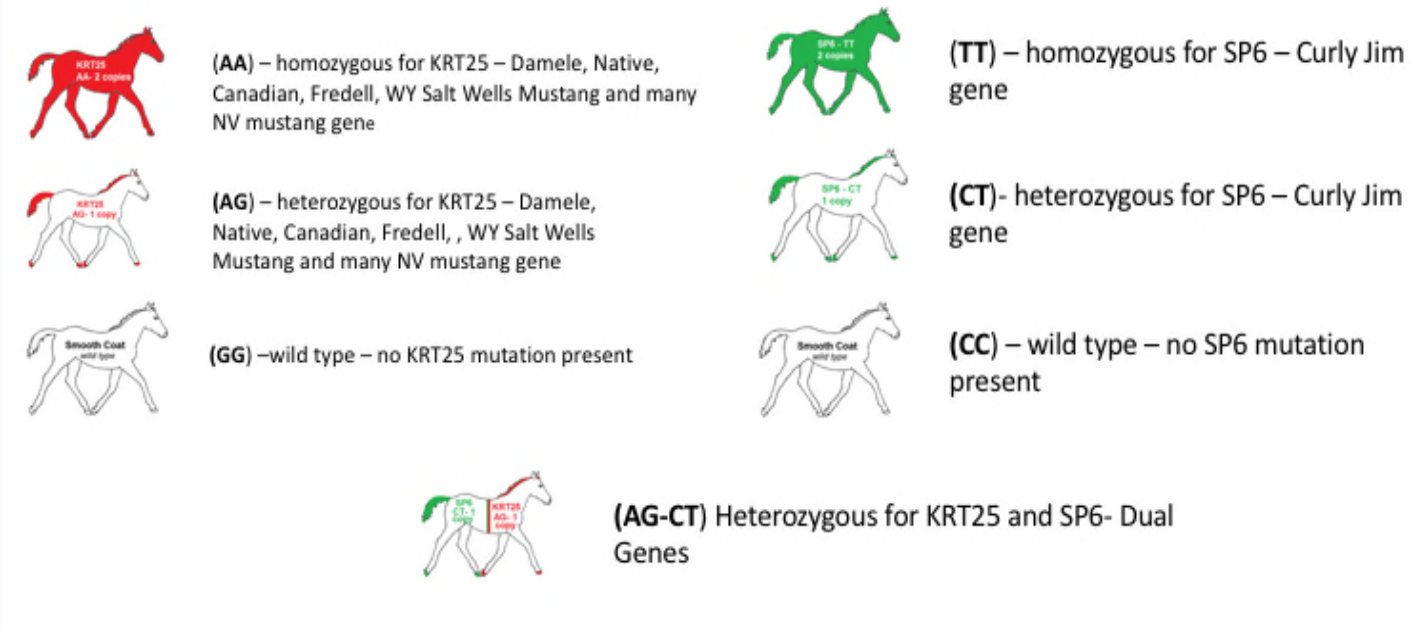
Genetic dominance is an interaction between alleles (two different versions of a gene) at the same gene locus (a specific gene location on a chromosome). Examples: The A allele is dominant over the G allele at the KRT25 locus. Likewise, the T allele is dominant over the C allele at the SP6 locus.

------ so, KRT25 and SP6 are two independent genes at different locations, but the effects of KRT25 mask the effects of SP6 like the gene for albinism masks the effects of a brown hair gene.

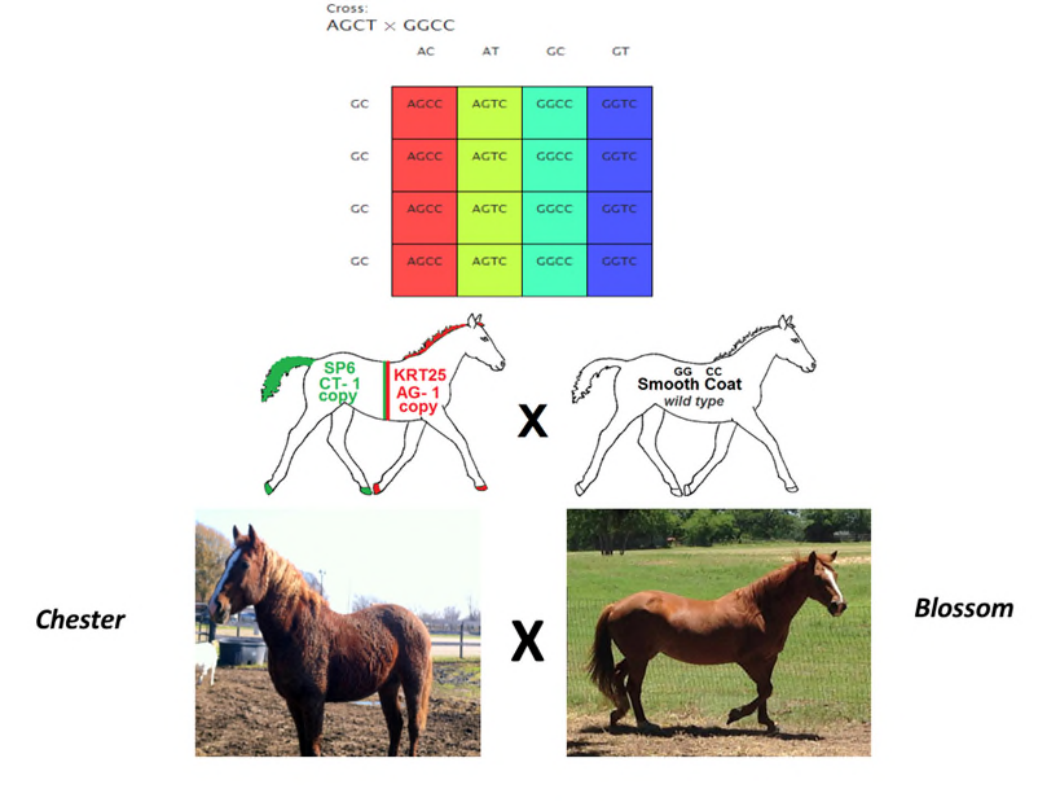
In the case of the two curly genes isolated to date, KRT25 is epistatic or masks to some extent the effects of SP6. (6, 15, 19)

Breeding Considerations with Two Independent Genes

If we look at a horse that carries one copy of the **KRT25** mutation simultaneously with one copy of the **SP6** mutation (**A**GC**T**), and that horse is crossed with a horse that does not have a copy of either the KRT25 nor the SP6 mutation (GGCC), we can see the odds or possibilities for the foal produced. An actual example is shown below with three horses owned by Angie Gaines in Texas.



Legend: Combinations of KRT25 and SP6 mutations



The cross of a dual gene stallion with a smooth coat mare

Each color represents a different genotype

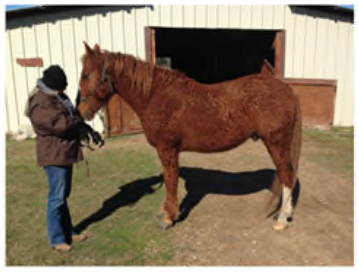
So — — **3/4** chance of having a curly and 1/4 chance of having a straight

1/4 odds of having a foal that is heterozygous for KRT25 but did not inherit SP6 – full mane and tail, but brittle hair

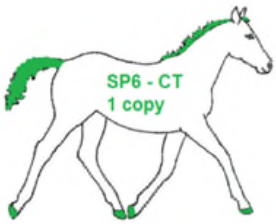
1/4 odds of having a foal that is heterozygous for both SP6 and KRT25 – **dual genes** – full mane and tail, but brittle hair

1/4 odds of having a foal that is heterozygous for SP6 and did not inherit KRT25 – full mane and tail with no brittle hair

0% odds of having a foal with the AA combination which produces sparse mane and tail



Bucky



The foal produced from the mating had one copy of the SP6 mutation

There was a 1/4 chance of this happening

Dr. Distl used the term “hypotrichotic” to describe the scant mane and tail characteristics associated with AA or homozygous horses that inherit the KRT25 mutation. **Hypotrichosis** (scant mane and tail) is a condition characterized by sparse hair or a coat that is not as thick or long as normally expected. It is also used to describe hair coats that have defects in the hair shaft or follicles (dysplastic) that lead to a sparser hair distribution compared to normal hair distribution for the species. (19)

Horses having only one copy of the KRT25 gene mutation or AG are described as having partial hypotrichosis. Dr. D. W. Scott in his 2004 paper on the histology of curly horse hair described the appearance of hair produced by KRT25 mutation as “dysplastic” meaning different from normal horse hair. Both terms can be used to describe the characteristics of hair shafts and coats produced by the KRT25 mutation. (3, 19)

Speculation on the Origins of the SP6 Mutation

In recent years, we have begun to understand that curly coats in horses can be produced by a variety of different and distinct gene mutations. We have also learned that curly coated horses can be found in many places throughout the world. The coats of these horses have distinct phenotypes or properties. As to date, none of the distinct populations have had gene mutations in common. So where did all the curly coated horses originate that we now find in such diverse locations around the world? In the future, we may find that some were imported from different locations and seeded a new population, but we will probably find

the majority resulted from simple and random mutations in local populations of horses.

Somatic Mutations vs Germinal Mutations

Somatic cells produce all non-reproductive tissues in living animals. Mutations in somatic cells are called **somatic mutations**.

Somatic mutations are not passed along to the next generation by sexual reproduction; they die with the animal. Cancer tumors are a unique class of somatic mutations.

In contrast, reproductive cells produce mutations which will be passed on to future generations. These mutations are termed **germinal mutations**. Germinal mutations are not expressed in the parent but are expressed in the offspring and the offspring's descendants. In most cases, only a single offspring will be born with the mutation and other siblings will not have the germinal mutation. (6, 15)

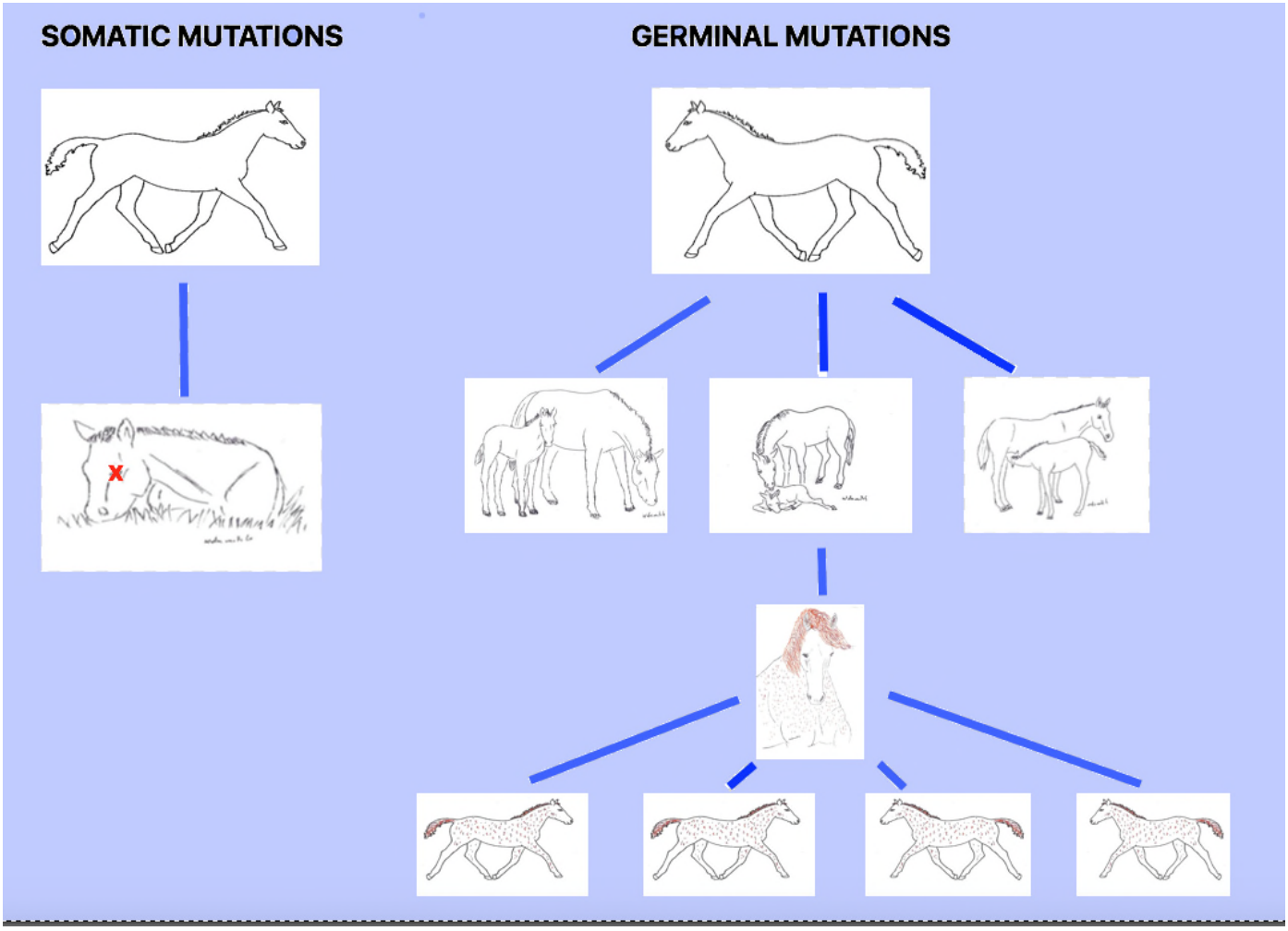


Chart illustrating the difference between somatic and germinal mutations

In the case of Curly Jim, a simple, single DNA base misspelling probably occurred during formation of a reproductive cell which became egg or sperm in a process termed **meiosis**. These reproductive cells or **gametes** have one-half the number of chromosomes found in somatic cells. Gametes are said to be **haploid,** meaning one-half of the animal's genetic material is present. Because there were originally two horses with this curly coat phenotype, the SP6 mutation probably occurred at least two generations before the birth of Curly Jim either in the stallion or mare that were his grandparents.

The SNP that resulted in the SP6 mutation did not affect Curly Jim's grandparents, but was passed as a dominant genetic trait to an offspring that would become one of Curly Jim's parents.

When such things happen, such as two straight haired parents producing an unexpected curly offspring, it is common to deduce that a recessive gene was involved and that both parents were carrying the recessive gene. That can be true, but not always. In this case, it was not.

An unexpected breeding outcome can also happen due to a mutation occurring in either one of the parents' reproductive cells during meiosis. The resulting mutation can be either dominant or recessive. If recessive, it will be passed on and not expressed until a future mating allows the combination of two recessive genes for the same trait to occur in the same individual revealing the dangers of "line breeding". (4, 5)

But if the resulting mutation is dominant, the mutation will increase within the population if it is favorable for survival. In the case of domestic horses like Curly Jim, the selection is due to human favoritism.

Thus, there is a good chance that Curly Jim's coat and the SP6 mutation happened due to a random mutation of a domestic horse in a nearby state. It should be remembered that the SP6 mutation has **not been found** in wild curly horse populations. (20)

Much time and energy have been spent wondering where curly horses originated, and some may have come from other locations, but most will be germinal, dominant mutations that happen spontaneously in local horse populations around the world. No distant curly horse populations to date have matched either KRT25 or Sp6. There is at least one other undiscovered curly producing gene mutation in the domestic curly horse population in the U.S. called the **Cook gene**. A second curly coat gene mutation has been found in **Sulphur Mustangs**. Its outward manifestations are very much like the Curly Jim SP6 mutation. Like the SP6 mutation that has not entered the feral horse

population, the Sulphur Mustang mutation has not yet entered the domestic curly horse population.

The very popular Curly Jim line of horses owes its popularity to mutations in two transcription factor-producing genes, DMRT3 and SP6. The combination of full and non-shedding curly mane and tail with comfortable gait will continue to make this line of curly horse desirable for many owners.

As a group, curly horse enthusiasts should embrace the fact that the curly horses found in the U.S. and Canada may be part of our unique equestrian heritage and that most will surly prove to be horses that were created by nature in North America.

About the Author:

Dr. Mitch Wilkinson has been a lifelong horse enthusiast. After receiving a bachelor's degree in chemistry and professional dental degrees, he earned a post-doctoral master's degree from Baylor University in biology. Currently, Dr. Wilkinson is Chairman of the Curly Mustang Association and Vice- Chair of the ICHO Research Department. His articles have been published in the United States, New Zealand/Australia, Russia, and Austria.

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Dr. Wilkinson and Lucky

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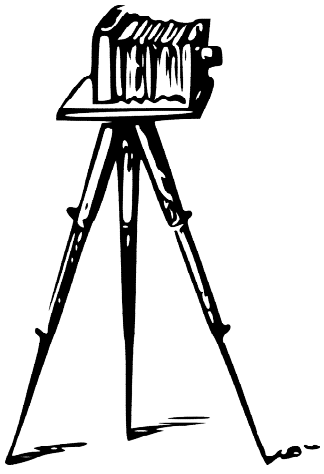

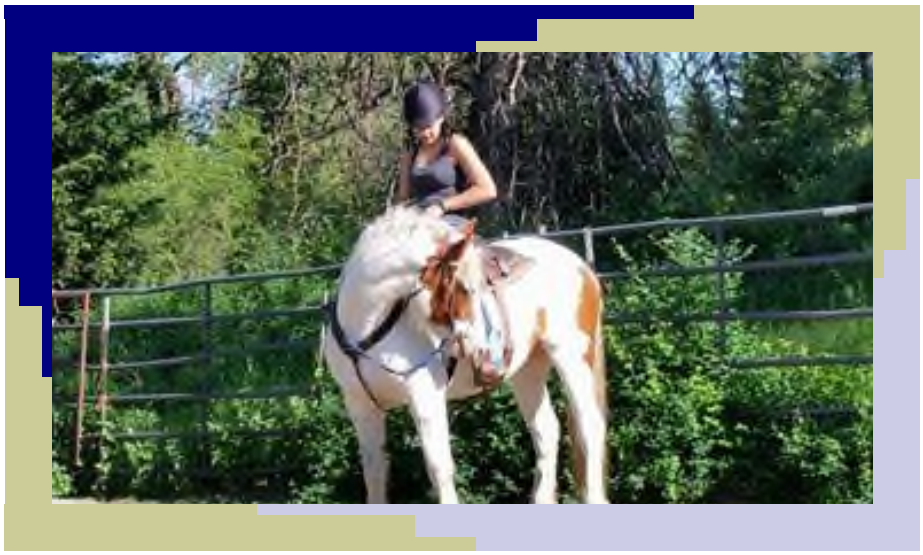

Congratulations to Kendahl D'Uva for earning the Sunny Martin Scholarship. Above left, RRR Black Rose, ICHO #402-D and right, CNC Warriors Angel Xpress, ICHO #1789-D.

Congratulations!

ICHO 2018 Awards

Above left, Dr. Mitch Wilkinson and UBMB Angel Leggs, ICHO# 1263-D, awarded the first Sandra Hendrickson Research Award, and right, Sheryl D'Uva and Warrior's Spiced Dunzule, ICHO# 1509-S, earning the 2018 Andrea Schaap award. Congratulations to all!

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