2018 ICHO GAZETTE

International Curly Horse Organization

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SUMMER GAZETTE 2018

Hello ICHO Members and Guests!

In one month, an exciting ICHO Annual General Meeting will be here! We are honored and pleased to invite our members and public to hear speaker, Dr. Mitch Wilkinson relate the latest Curly genetic news. See inside this gazette edition, a curl gene article written by Dr. Loretta L. Nielsen, PhD, about the recently isolated curl genes and the important implications of this research, it's direction and mysteries yet to be unraveled.

The itinerary for the 2018 ICHO AGM is given inside. With great honor, we invite you to join us on Mackinac Island the first weekend of October, in autumn splendor! Please share this information with your Curly colleagues and friends. Sincerely, Joan Henning, ICHO President

2018 AGM

MACKINAC ISLAND, MI

OCTOBER 5–7, 2018

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*

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

Are you ready?!!!!

We are honored by your presence at the 2018 ICHO AGM, held at historic and beautiful Mackinac Island, Michigan! Please reserve your presence at www.ichocurlyhorses.com. The program is free and open to the public.

Dr. Mitch Wilkinson will speak on the current Curly gene research and more! There is much to learn, ground breaking news, and a great deal of Curly conversation!

Itinerary

Friday, October 5, 2018

Arrive on the island. Horse drawn taxis will take you to your destination or a quick walk will also get you there. Porters will be waiting for you at the docks from various hotels to carry your luggage, they think of everything on Mackinac Island! The main location of the AGM meeting is the Inn on Mackinac, 855-784-3846, mention ICHO and receive a discount on your room!

Saturday, October 6, 2018

Saturday morning—day visitors arrive on the island using one of the several ferries if you haven't arrived already. Sheplers, Star Line, Arnold ferries are available at Mackinaw City or St. Ignace (if you are in the upper peninsula of MI). Ferries leave approx. every half hour, easy to use! Use the morning to sight see, take a horse drawn tour, visit the Manoogian Art Collection, explore downtown, the fort, cemeteries, sugarloaf and other historical attractions!

12-3pm (approx.)—Dr. Mitch Wilkinson will be speaking on Curly Gene research. Location is at the Inn On Mackinac, in the Library Room.

3pm short break

3:15pm—ICHO member meeting

5pm adjourn

7pm—dinner at the Pink Pony and recreation!

Sunday, October 7, 2018

Touring, visiting and leaving the island.

INTERNATIONAL Curly Horse ORGANIZATION

https://www.ichocurlyhorses.com/curly-coat-genes.html

Curly Coat Genes

Two Dominant Curly Coat Genes have been isolated, the **KRT25 mutation** and the **SP6 mutation**. These gene mutations give Curlies their Curly Coats. Horses with *Wild Type* KRT25 and *Wild Type* SP6 do not have have the mutation or the Curly coats and are straight haired. It is the mutations in these two genes that give us our Curly coats in the following lines:

KRT25 mutation is found in the following Curly lines: Damele, Native/Warrior (Eli Bad Warrior, Berndt, Hammrich, NTS), Canadian (Cypress, Sjkonsberg, Naismith), Fredell, WY Salt Wells mustangs and most Nevada mustangs.

SP6 mutation is found in the Curly Jim line- which includes Walker's Prince T, McKay line and Circle B ponies. All horses with the SP6 mutation trace back to Curly Jim, an unknown gaited Curly that showed up in Tennessee.

A huge thank you to Dr Gus Cothran of TX A&M and the French researchers for finding KRT25 and to the German research team for finding SP6. Dr Cothran has confirmed the SP6 gene is found in the Curly Jim line through research testing including the McKay ponies and Circle B ponies.

There are also some unknown Curly coat mutations that have not been identified and that includes some Curly mustangs, the Cook line horses and two curly McKay line ponies out of a SP6 mutation heterozygous sire x straight mares that do not have the SP6 mutation. We have more mysteries to solve and continued research is progressing.

Read the Research papers on our webpage

KRT25 research paper

geneticsselectionevolution-curly_gene_krt25.pdf
Download File

German Research Paper SP6 link- https://www.nature.com/articles/s41598-018-24865-3

Walker's Prince T ABC 90 (f) MFTHBA F-12781

was a Curly Jim grandson that sired many gaited Curlies also registered in the Missouri Foxtrotting Registry MFTHBA. He carried SP6 through Curly Jim.

Curly Jim Line

Walker's Prince T* ABC 90 (f) MFTHBA F-12781

Color:	Sorrel
Markings:	Sabino,
Sex:	Stallion
Date of Birth:	7/1967
Gait:	Fox Trot

Curly Jim line is testing for SP6. Curly Jim is the founding sire of this line and SP6 gene passed on by him.

PEDIGREE

Printer Friendly Version

Grandson of Curly Jim	Walker's Merry Lad MFTHBA 282, AFTHBA F-105, TWHBEA 482752	Collier's Merry Boy TWHBEA 390778	Merry Boy TWHBEA#350189
			Miss Allen Wilson
		Beauty Sweetheart TWHBEA 431944	Noble Knight TWH 400661
Walker's Prince T*			Walker's Beauty TWH 440947
by default SP6 +/-	Blaze* ABC 135(f)	Curly Jim*	unknown
			unknown
	by default SP6 +/-	Bradford Mare	Rock-A-Way MFTHBA F-15256
			unknown

Unraveling the Mystery of Curly Coats in the Horse World

by Loretta L. Nielsen, Ph.D.

Horse enthusiasts have long wondered why some horses are born with curly hair instead of the more common straight hair. At long last, the great mystery of curly horses is starting to be revealed!

Recently, two scientific papers were published proving the existence of at least 2 genes linked to curly hair in horses.[1,2] Both of these curly genes are inherited in a dominant manner. By "dominant", geneticists mean that a curly-coated foal only needs to inherit the curly gene from one parent (sire or dam). This foal is called "heterozygous" for the curly gene and has a 50% chance of passing it on to each of their offspring. A foal who inherits the curly gene from both parents is called "homozygous" and all of their offspring will be curly horses.

To greatly over-simplify the findings, among North American Curly Horses one of the newly identified dominant curly genes (*KRT25 variant*)[1,2] was found primarily in descendants of wild mustangs captured and bred by ranchers in North America. The other dominant curly gene (*SP6 variant*)[2] was found in some of these curly horses plus in descendents of a single stallion of unknown origin who produced curly-haired gaited offspring; some of whom were registered in the Missouri Fox Trotting Horse Breed Association (MBTHA)..

Curly horses display many degrees of hair curliness from soft, relaxed waves all the way to tightly wrapped, brillo-like curls. Various degrees of curliness are also obvious in each individual in their eye lashes, ear hairs, mane, tail, and body coat. Breeders have long postulated the influence of many genes on the primary dominant curly gene to explain why curly horses have such a wide diversity of looks (phenotype). For example, I have two brothers from the same parents. One is black with a brillo-type body coat that gets much shorter during the hot summer months, but remains consistent year-round. His full brother is a cremello with very soft and loose waves of curly hair on his main body. The curls in his body coat get tighter and longer during the winter, but are almost none existent in his very short, summer coat.

Over the past few years, scientists from France, Germany and the U.S.A. set out to find the specific piece(s) of horse DNA responsible for putting the curl into horse hair.[1,2] Their focus was the dominant curly gene, as well as another possible gene that seems to cause some curly horse bloodlines to develop various degrees of hair loss and bald patches; common among American Bashkir Curly horses. They used a genetic technique call genome-wide association to identify pieces of DNA that were present in curly haired horses and absent in straight haired relatives. This then allowed them to sequence those specific pieces of the horse genome. In other words, from all the DNA in a horse, they were able to pull out the pieces they wanted to study and then determine the exact sequence of molecular parts that made each piece unique.

One study used DNA samples from 51 curly- and 19 straight-haired French and North American horses descended from 13 different sires. A single, strong signal associated with curly hair was found on equine chromosome 11, in a region that includes the *type I keratin* gene cluster with its 36 genes. Keratin is one of the primary components of mammalian hair. Therefore, a mutation (change) in the DNA serving as a template for

keratin, and ultimately hair, production makes sense. In order to pinpoint the exact culprit, the scientists did an exhaustive comparison of the DNA in a heterozygous curly-haired stallion and his straight-haired son. Only one gene in the *type I keratin* gene cluster, the *KRT25* gene, was sufficiently different between these two horses to warrant further study. The *KRT25 variant* was then confirmed to be a dominant curly gene using DNA from 353 additional horses. However, there were 5 other horses who had curly hair, but no DNA changes in the *KRT25* gene. Closer examination of the DNA sequence of the *KRT25* gene in two of these horses did not find any missed variations of the *KRT25* gene. These curly horses had wild-type *KRT25*. These data provided strong evidence that some horses have a different dominant curly gene controlling the curliness of their hair. In other words, there is more than one dominant curly gene being passed down in North American curly horse bloodlines. Needless to say, this finding is extremely important for all curly horse breeders to know. Thus, the International Curly Horse Association (ICHO) is currently facilitating testing for the genes *KRT25* and *SP6 (see below)*: wild-type versus variant.

Another study used DNA samples from 216 curly- and straight-haired horses with varying degrees of hair loss or no abnormal hair loss at all. This analysis included horses from a wide variety of breeds, including: American Bashkir Curly, Missouri Fox Trotter, American Quarter Horse, Kentucky Mountain Saddle Horse, Danish Warmblood, Oldenburger, Holsteiner, Hanoverian, Duelmen Horse, Black Forest Coldblood, Norwegian, Lewitzer, Friesian, miniature Donkey, Sorraia, Standardbred, Przewalski, Rhenish German Coldblood, Arabian Thoroughbred, Anglo-Arabian, Austrian Coldblood, Swedish Warmblood, and Trakehner-Barb. Using genetic analysis techniques similar to those described above, this group of scientists identified 2 genes on horse chromosome 11 associated with curly hair. They found genetic variants (areas of gene mutation compared with wild-type) for the *KRT25* and *SP6* genes. Horses who had inherited the *KRT25 variant* from one or both parents had both curly hair and varying degrees of hair loss (from negligible to extensive). In contrast, horses who inherited the normal *KRT25* gene with the *SP6 variant* had curly hair, but no bald patches or hair loss. Horses who inherited the mutated genes for both *KRT25* and *SP6* had both curly hair and areas of hair loss. Thus, the *KRT25 variant* gene "overpowers" the normalizing effects of the *SP6* gene with regards to hair loss. This demonstrates how two genes can influence each other to change how a horse looks. It also appears to be the explanation for why registered curly Missouri Fox Trotters consistently have curly hair but no hair loss. They all inherited the *SP6 variant* from a single founding stallion along with the same wild-type *KRT25* gene found in straight-haired Missouri Fox Trotters. Future research will probably identify more mutations in genes with primary control over the extent of hair curliness in different horse breeds and a multitude of secondary genes influencing their expression. Of note, mutant *KRT25* and *SP6* genes have also been associated with curly hair in humans, rats, mice, and cattle.

For more than a decade, ICHO members have collected DNA samples and photographs from curly-coated horses and their straight-haired relatives in order to create a genetic storehouse that was used by scientists in some of this research. The figures below illustrate how difficult it is to distinguish between the phenotypes (hair appearance) of horses carrying the *KRT25 variant* or the *SP6 variant*. Only genotyping (DNA testing) can separate the types. However, there does seem to be an observable

difference in curl tightness for heterozygotes (1 copy of a variant) versus homozygotes (2 copies of a variant and a tighter curl). Of most interest is the last figure showing a curly horse who carries one copy of each variant. According to his owner, his hair loss has always been minimal and decreased as he got older. However, his general appearance gives no clue as to his actual genotype. Recent testing of DNA from other North American Curly Horses in the ICHO storehouse has tentatively identified some curly horses without either variant (neither *KRT25* nor *SP6*). *[As yet unpublished and exciting data for future studies!]*

Undoubtedly, further exploration of the genetics controlling how horses look and perform will yield other exciting revelations in the coming years. This information is especially important for horse breeders to understand. We now have genetic tests that allow breeding along either the *KRT25 variant* or *SP6 variant* curly bloodlines. No doubt, in the future, geneticists will find other genes that affect the curliness or straightness of horse hair. Some of these genes might also cause health problems that should be eliminated from all bloodlines. For example, some Missouri Fox Trotter foals are reportedly born with extensive health problems, such as ulcers in the lining of the gastrointestinal tract - anywhere from the mouth to the anus, in addition to a curly coat. However, based on available evidence, these horses have inherited a not-yet-identified recessive gene; meaning the health problems only occur when they inherit the disease gene from both parents, never just one. So-called "silent carriers" with only one copy of the disease gene are outwardly healthy. This allows the recessive disease gene to remain hidden for long periods of time until two silent carriers are bred together; with a 25% chance of producing an unhealthy foal with 2 copies of the disease gene. Elimination of this not-yet-identified recessive gene is an important goal for improving the Missouri Fox Trotter breed. However, because these foals are so rare and usually don't reach adulthood, it will require a long-term, serious effort on the part of MFTHBA members to document and collect DNA from affected bloodlines for scientists to analyze. The American Quarter Horse Association (AQHA) has already set a good example of how a breed association, horse owners/ breeders, and scientists can work together to eliminate devastating diseases from bloodlines once genetic tests are available *(see the AQHA website)*.

References

1. Morgenthaler C, Diribarne M, Capitan A, Legendre R, Saintilan R, Gilles M, Esquerré D, Juras R, Khanshour A, Schibler L, Cothran G. A missense variant in the coil1A domain of the *keratin 25* gene is associated with the dominant curly hair coat trait (Crd) in horse. Genet Sel Evol 49:8,2017. DOI 10.1186/s12711-017-0359-5

2. Thomer A, Gottschalk M, Christmann A, Naccache F, Jung K, Hewicker-Trautwein M, Distl O, Metzger J. An epistatic effect of KRT25 on SP6 is involved in curly coat in horses. www.nature.com/scientificreports 8:6374, 2018. DOI:10.1038/s41598-018-24865-3

Figures

Figure 1. Curly hair coat of a Fish Creek, Nevada, wild mustang stallion (Fishy Boy) proven by genetic testing to be homozygous for the *KRT25 variant* dominant curly gene.

Figure 2. Curly hair coat of a gray American Bashkir Curly Horse gelding (Lilly's Moon Man) proven by genetic testing to be heterozygous for the *KRT25 variant* dominant curly gene. The photo shows his soft, wavy coat and was taken in April in southern California.

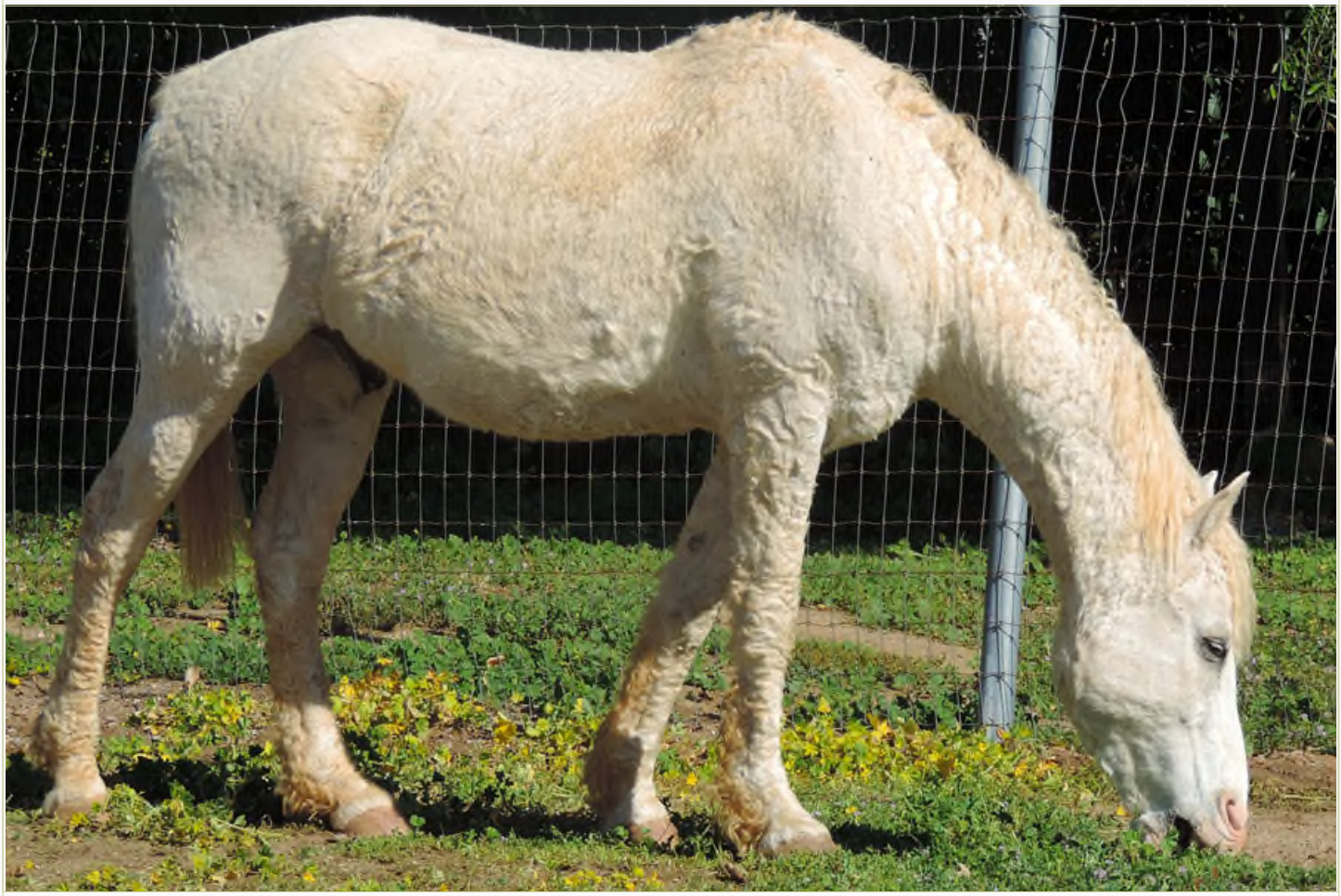


Figure 3. Close-up of the curly coat of a black Missouri Fox Trotter gelding (WDR Diego's Wizard) proven by genetic testing to be homozygous for the *SP6 variant* dominant curly gene. The photo shows the brillo-hair coat in the neck and shoulder area, and was taken in November in southern California.

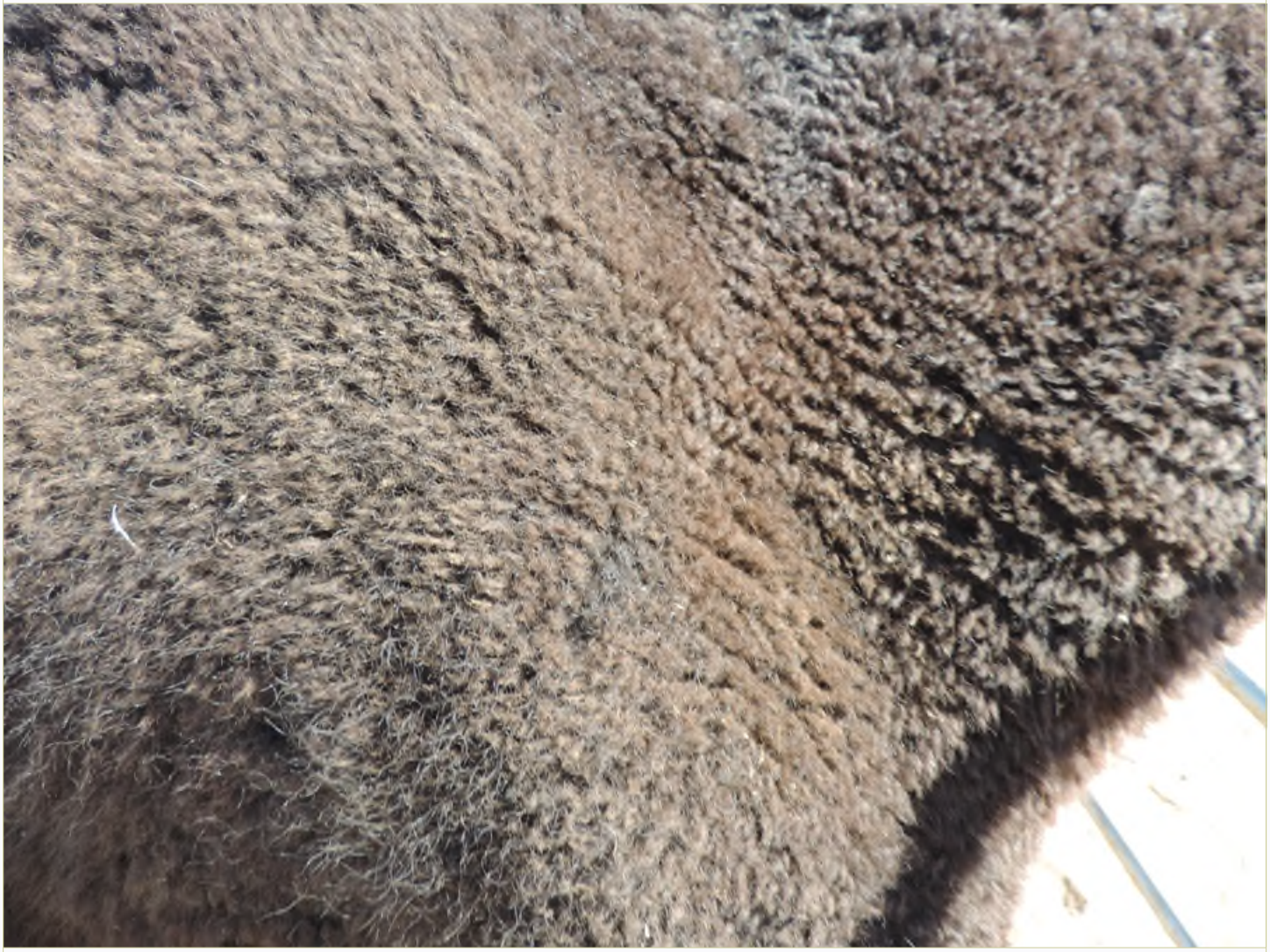
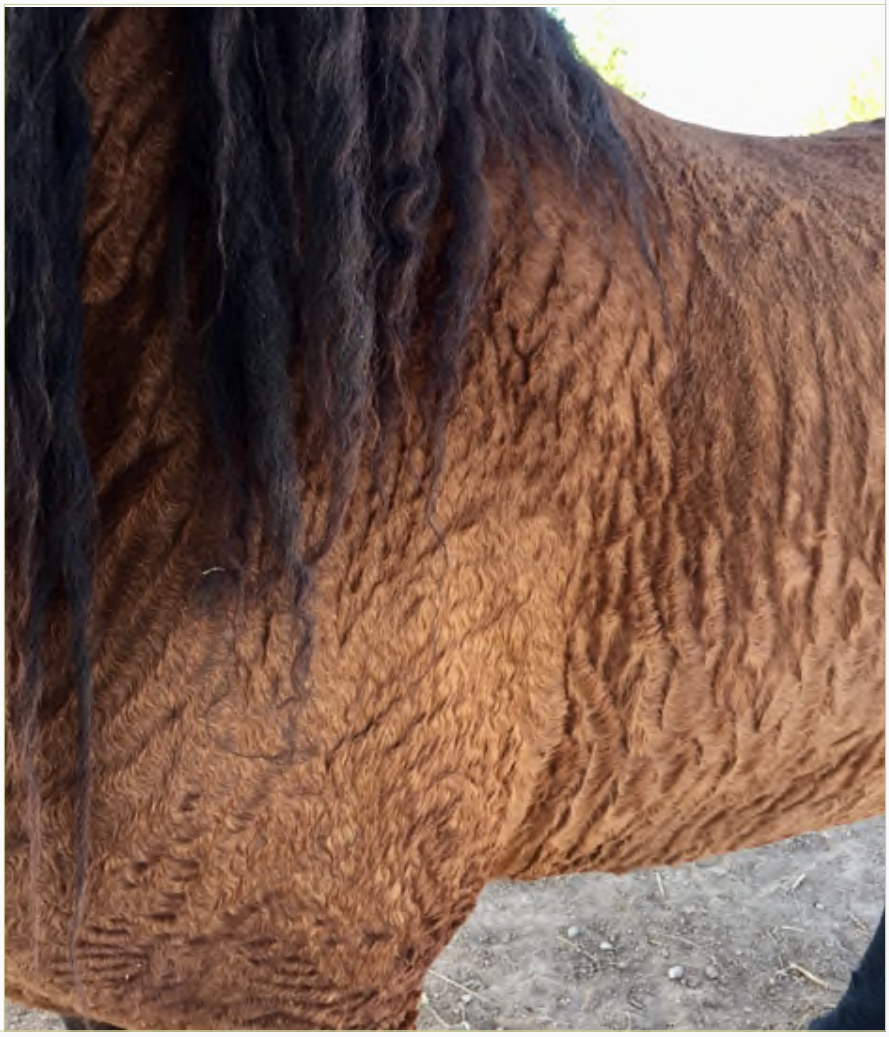


Figure 4. Close-up of the curly coat of a cremello Missouri Fox Trotter gelding (WDR Dragonsmoke) proven by genetic testing to be heterozygous for the *SP6 variant* dominant curly gene. This horse is a full brother of the homozygous *SP6 variant* gelding shown in the previous figure, but has a more relaxed and softer curly coat. The photo shows the hair coat in the neck and shoulder area and was taken in November in southern California.

Figure 5. Curly hair coat of a bay North American Curly Horse stallion (Frostfire's Xeque) proven by genetic testing to be heterozygous for the *KRT25 variant* plus heterozygous for the *SP6 variant* dominant curly genes. This horse is descended from both dominant curly North American mustangs and dominant curly Missouri Fox Trotters.

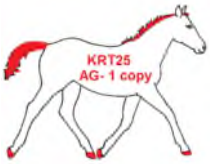


Typical KRT25 and SP6 Crosses

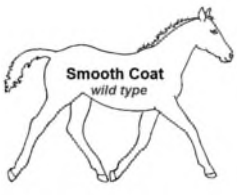
Legend:

Dr Mitch Wilkinson and Bunny Reveglia

(**AA**) – homozygous for KRT25 – Damele, Native, Canadian, Fredell, WY Salt Wells Mustang and many NV mustang gene

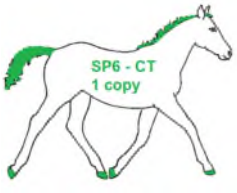


(**AG**) – heterozygous for KRT25 – Damele, Native, Canadian, Fredell, , WY Salt Wells Mustang and many NV mustang gene

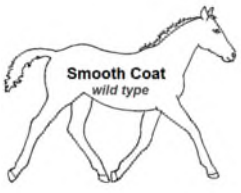


(GG) –wild type – no KRT25 mutation present

(**TT**) – homozygous for SP6 – Curly Jim gene



(**CT**)- heterozygous for SP6 – Curly Jim gene

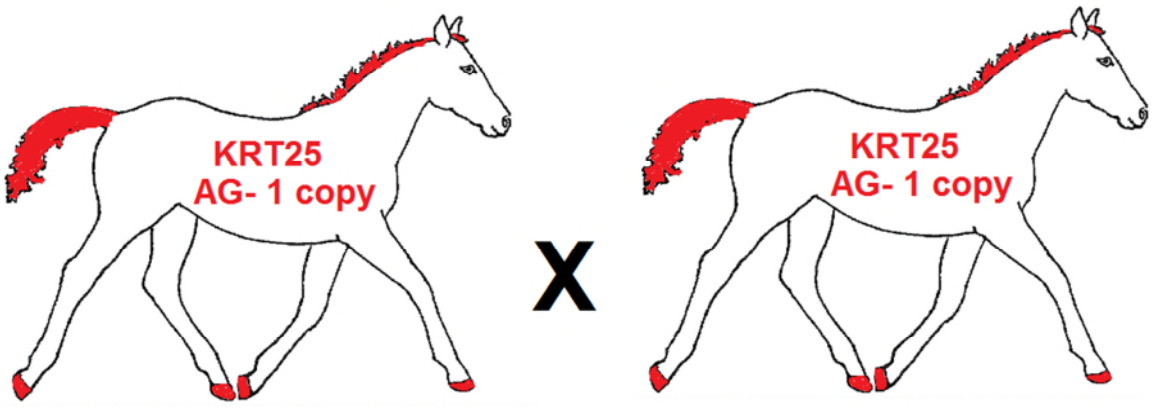


(**CC**) – wild type – no SP6 mutation present

(**AG-CT**) Heterozygous for KRT25 and SP6- Dual Genes

To use this document on typical breeding percentages, the user must know the KRT25 and SP6 status of the potential breeding pair. Hair follicle testing is available through the ICHO office. Once the KRT25 and SP6 status of the breeding pair is known, find the breeding cross that fits. The odds or potential percentages of breeding outcomes are found under each typical breeding scenario.

KRT25 Cross- *heterozygous*



Using a **Punnett Square** to determine the percentages of a mating outcome. A simple cross of two heterozygous horses which have the **KRT25** gene mutation, but **do not** have the SP6.

	A	G
A	AA	AG
G	AG	GG

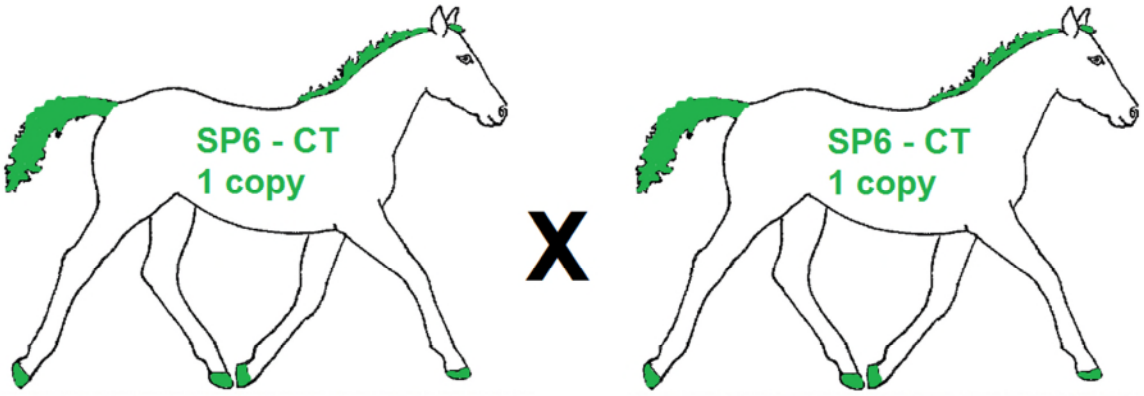
25%	50%	25%
AA	**AG**	**GG**

1/4 odds that the foal will be homozygous for KRT25 with scant mane and tail

1/2 odds that the foal will be heterozygous for KRT25 with mane and tail, but brittle hair

1/4 odds the foal will be born without a curly gene

SP6 Cross- *heterozygous*



Another simple cross of two horses that are heterozygous for **SP6** (Curly Jim), but **do not** have KRT25 in their genetics.

	C	T
C	CC	CT
T	CT	TT

25%	50%	25%
TT	**CT**	**CC**

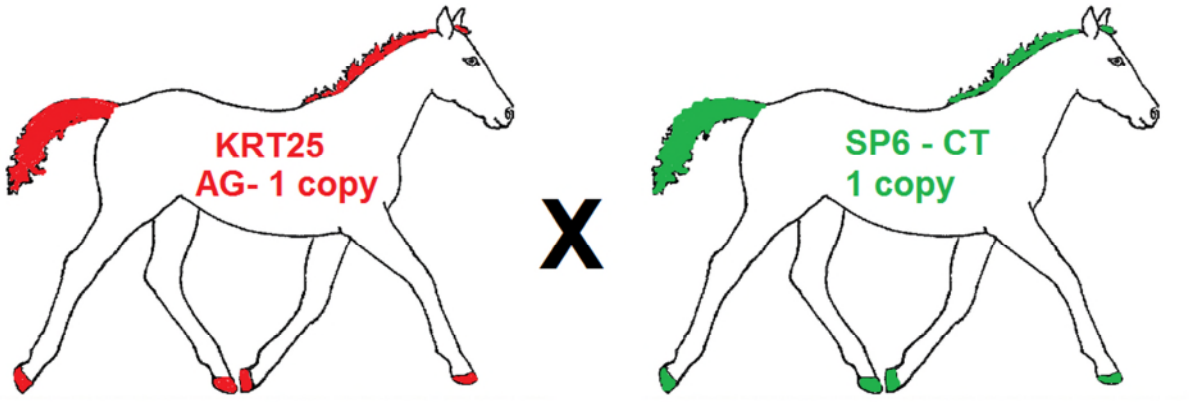
1/4 odds the foal will be homozygous for SP6

1/2 odds the foal will be heterozygous for SP6

1/4 odds the foal will not inherit a curly gene

All results will produce a foal with normal strength hair and full mane and tail

Cross #1



The cross of two heterozygous for each gene: #1 Parent- KRT25 AGCC X #2 Parent- SP6 GGCT- parents only carry KRT25 or SP6 but do not carry both at the same time

Cross:
AGCC × GGCT

	AC	AC	GC	GC
GC	AGCC	AGCC	GGCC	GGCC
GT	AGCT	AGCT	GGCT	GGCT
GC	AGCC	AGCC	GGCC	GGCC
GT	AGCT	AGCT	GGCT	GGCT

25%	25%	25%	25%
AG CC	AG CT	GG CT	GG CC
			Straight

3/4 odds of producing curly foal --- 1/4 odds of producing straight foal

1/4 odds for foal to be heterozygous for KRT25 and curly with no SP6

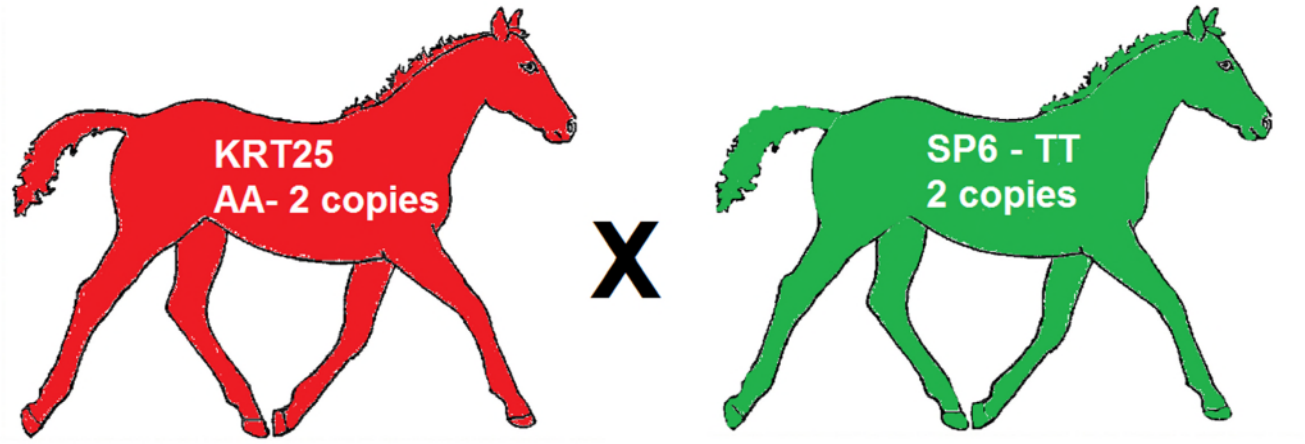
1/4 odds for foal to be heterozygous for SP6 and curly with no KRT25

1/4 odds of foal carrying both KRT25 and SP6 - hetero for both – **dual gene**

1/4 odds of foal not having a curly gene and straight coated

0% odds of producing a foal with sparse mane and tail

Cross #2



#1 Homozygous KRT25 Parent crossed with #2 Homozygous SP6 Parent with each parent carrying 2 copies of a different curly gene

Parent #1 - AA CC (sparse mane and tail)

Parent #2 - GG TT (full mane and tail)

Cross:
AACC × GGTT

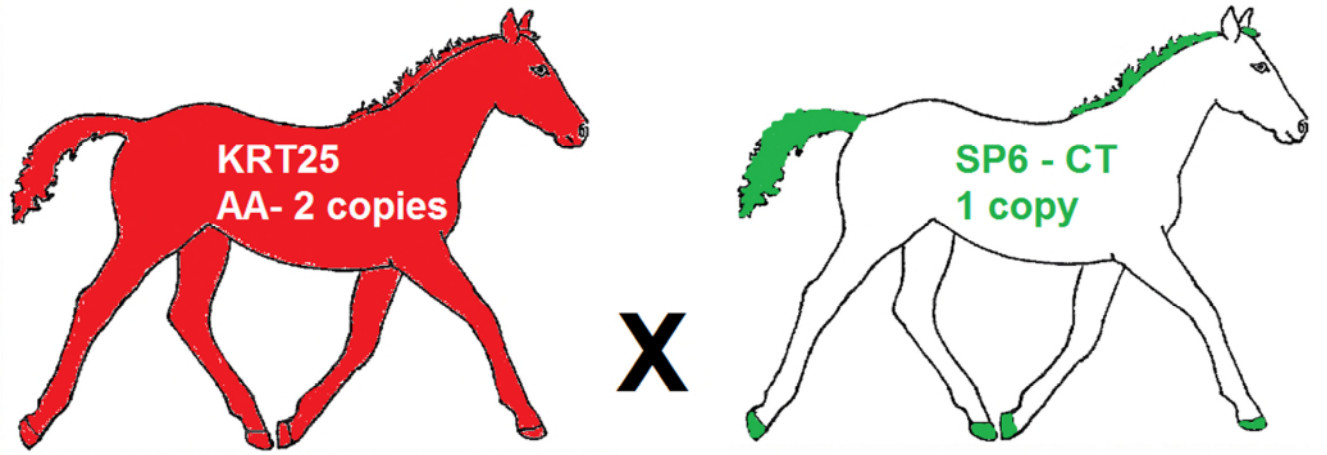
	AC	AC	AC	AC
GT	AGCT	AGCT	AGCT	AGCT
GT	AGCT	AGCT	AGCT	AGCT
GT	AGCT	AGCT	AGCT	AGCT
GT	AGCT	AGCT	AGCT	AGCT

Offspring - 100% - AG CT – Dual Genes- Good mane and tail, but possibly shorter with hair brittle

0 % odds of Straight

0 % odds of foal with sparse mane and tail

Cross #3



#1 Parent Homozygous KRT25 (AACC) x #2 Parent Heterozygous SP6 (GGCT)

Parent#1 AACC (sparse mane and tail) **Parent #2 GGCT (full mane and tail)**

Homozygous KRT25 x heterozygous SP6

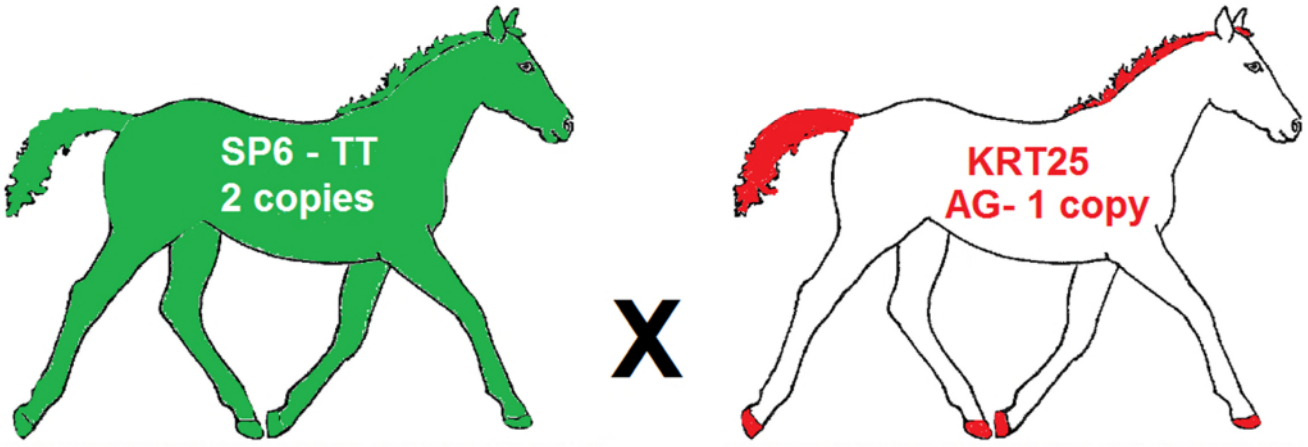
Cross:

AACC × GGCT

	AC	AC	AC	AC
GC	AGCC	AGCC	AGCC	AGCC
GT	AGCT	AGCT	AGCT	AGCT
GC	AGCC	AGCC	AGCC	AGCC
GT	AGCT	AGCT	AGCT	AGCT

50% heterozygous KRT25
50% Dual Gened- heterozygous SP6 & KRT25

Cross #4



#1 Parent Homozygous SP6 (GGTT) x #2 Parent Heterozygous KRT25 (AGCC)

#1 Parent GGTT (Full mane and tail) **#2 Parent (medium mane and tail)**

Homozygous SP6 x Heterozygous KRT25

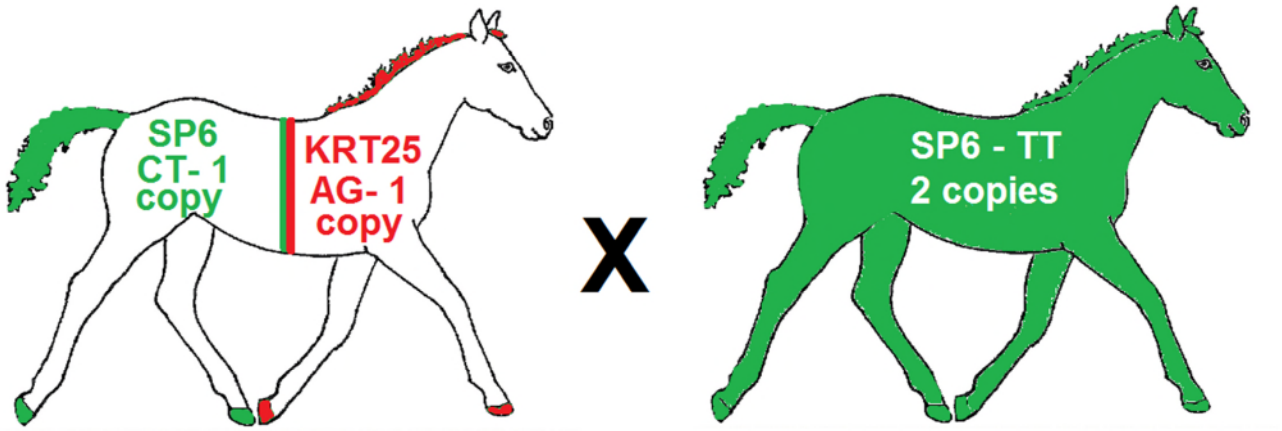
Cross:

AGCC × GGTT

	AC	AC	GC	GC
GT	AGCT	AGCT	GGCT	GGCT
GT	AGCT	AGCT	GGCT	GGCT
GT	AGCT	AGCT	GGCT	GGCT
GT	AGCT	AGCT	GGCT	GGCT

50% Heterozygous SP6
50% Dual Gened- Heterozygous SP6 & KRT25

Cross #5



#1 Parent AG CT- Heterozygous for both genes (KRT25 & SP6) Dual Genes crossed with #2 Parent GG TT- Homozygous for SP6

Cross:
AGCT × GGTT

	AC	AT	GC	GT
GT	AGCT	AGTT	GGCT	GGTT
GT	AGCT	AGTT	GGCT	GGTT
GT	AGCT	AGTT	GGCT	GGTT
GT	AGCT	AGTT	GGCT	GGTT

25% **25%** **25%** **25%** -- or 1/4 odds of each phenotype happening

GG CT **GG TT** **AG CT** **AG TT**

1/4 odds for foal to be heterozygous for SP6 and curly with no KRT25

1/4 odds for foal to be homozygous for SP6 and curly with no KRT25

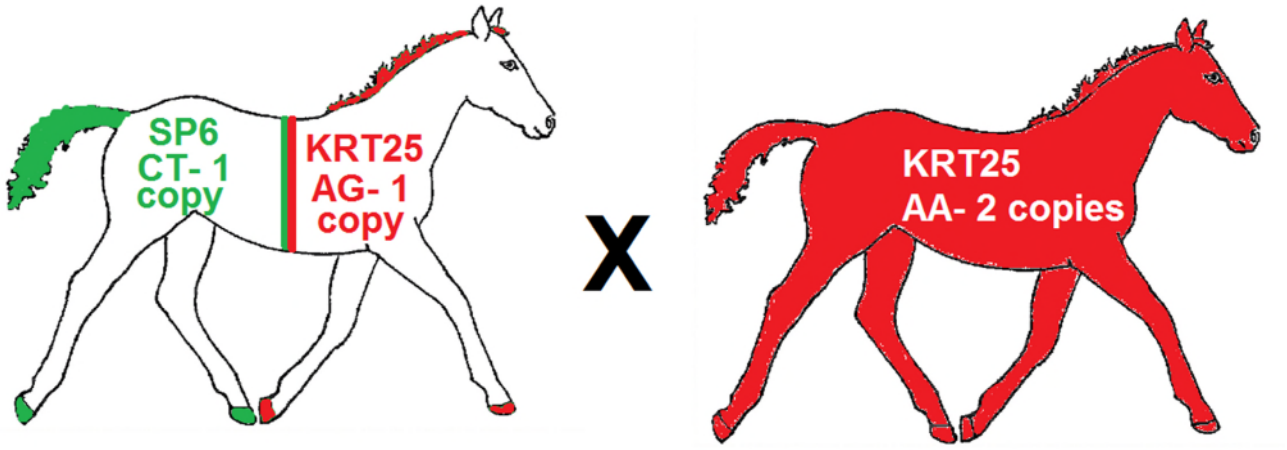
1/4 odds for foal to be heterozygous for both KRT25 and SP6 – **dual gene**

1/4 odds for foal to be heterozygous for Krt25 and homozygous for SP6- **dual gene**

0% - straight offspring

0% - AA or horse with scant mane and tail

Cross #6



The Cross of #1 parent Dual genes AGCT x #2 parent KRT25 AACC homozygous

Cross:
AGCT × AACC

	AC	AT	GC	GT
AC	AACC	AATC	GACC	GATC
AC	AACC	AATC	GACC	GATC
AC	AACC	AATC	GACC	GATC
AC	AACC	AATC	GACC	GATC

25% AA CC **25% AA TC** **25% AG CC** **25% AG TC**

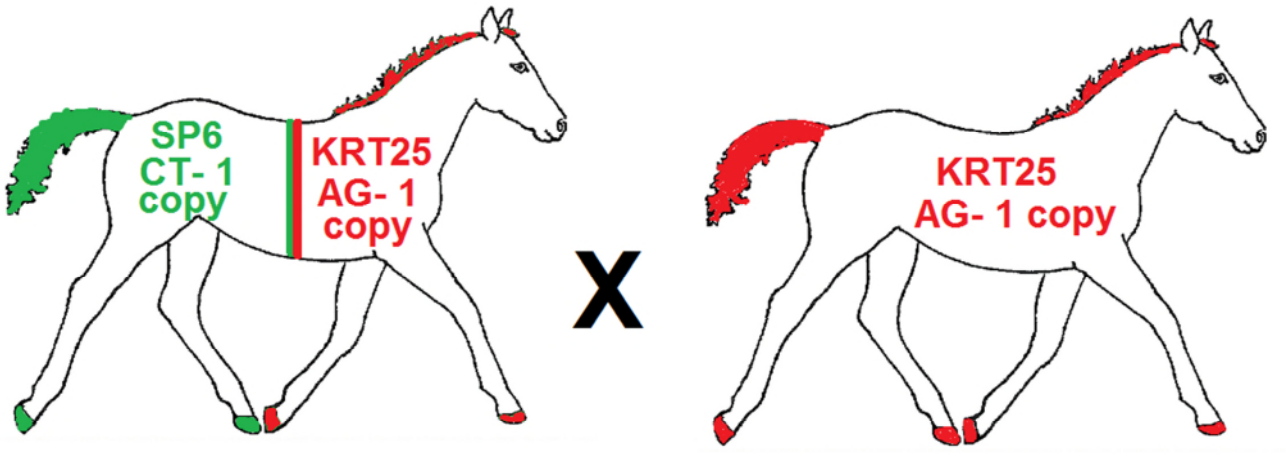
1/4 odds the foal will be homozygous for KRT25 with no SP6 present - scant mane and tail

1/4 odds the foal will be homozygous for KRT25 and heterozygous for SP6 - scant mane and tail

1/4 odds the foal will be heterozygous for KRT25 - mane and tail but brittle hair

1/4 odds the foal will be heterozygous for both KRT25 and SP6 - **dual gene**

Cross #7



The cross of #1 Parent Dual Genes AGCT x #2 Parent Heterozygous KRT25 AGCC

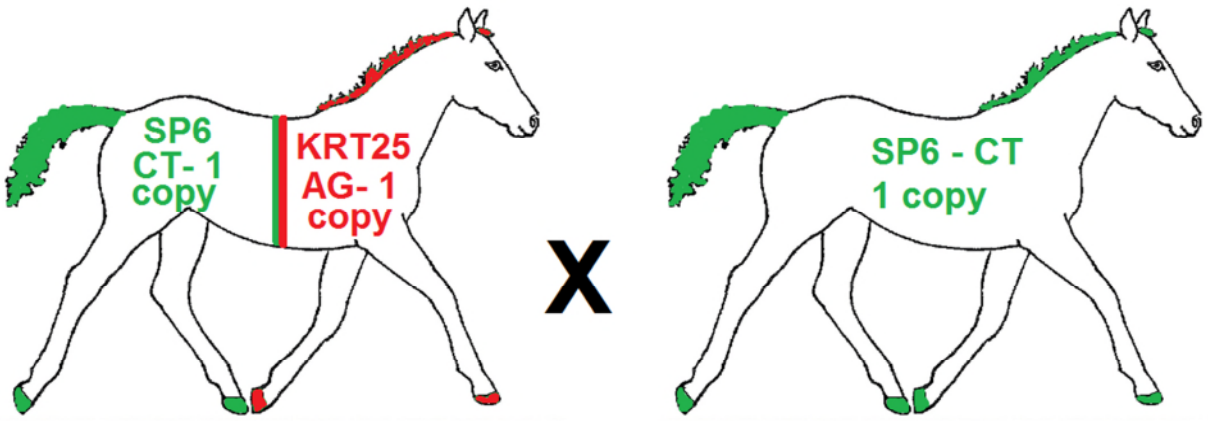
Cross:
AGCT × AGCC

	AC	AT	GC	GT
AC	AACC	AATC	GACC	GATC
AC	AACC	AATC	GACC	GATC
GC	AGCC	AGTC	GGCC	GGTC
GC	AGCC	AGTC	GGCC	GGTC

12.5%	12.5%	25%	12.5 %	25%	12.5%
AA CC	AA TC	AG CC	GG TC	AG TC	GG CC

1/8 odds of AA CC – homozygous KRT25 with no SP6 - scant mane and tail
1/8 odds of AA TC - homozygous KRT25 and heterozygous for SP6 - scant mane and tail - **dual**
1/4 odds of AG CC – heterozygous KRT25 with no SP6 – full mane and tail – brittle hair
1/8 odds of GG TC – heterozygous SP6 with no KRT25
1/4 odds of AG TC - heterozygous for bothKRT25 and SP6 – brittle hair **- dual**
1/8 odds of GG CC - straight

Cross #8



Cross of #1 Parent AGCT Dual genes x #2 Parent Heterozygous SP6 GGCT

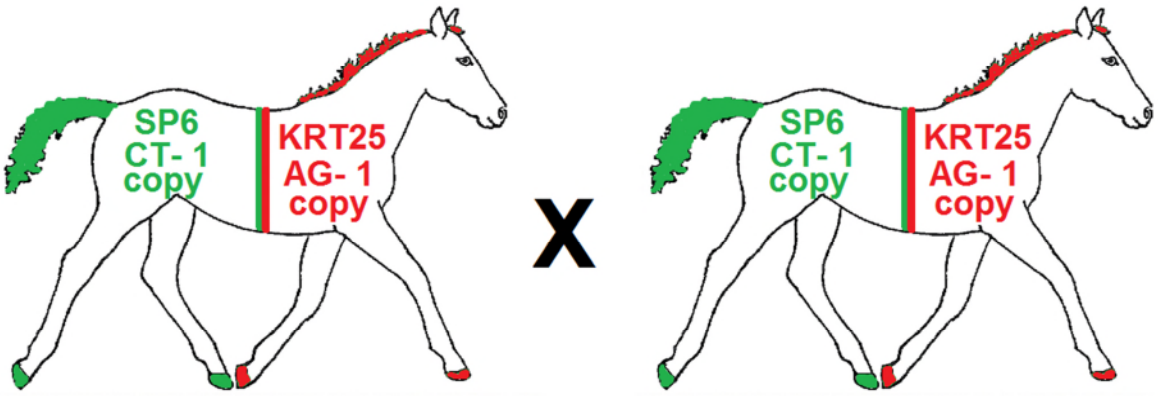
Cross:
AGCT × GGCT

	AC	AT	GC	GT
GC	AGCC	AGTC	GGCC	GGTC
GT	AGCT	AGTT	GGCT	GGTT
GC	AGCC	AGTC	GGCC	GGTC
GT	AGCT	AGTT	GGCT	GGTT

12.5%	25%	12.5%	12.5%	25%	12.5%
AG CC	AG CT	AG TT	GG TT	GG TC	GG CC

1/8 - odds of AG CC – heterozygous for KRT25 but no SP6 present – full mane and tail –brittle hair
1/4 - odds of AG CT – heterozygous for both KRT25 and SP6 – full mane and tail –brittle hair - **dual**
1/8 - odds of AG TT - heterozygous for KRT25 and homozygous for SP6 – full mane and tail
– brittle hair - **dual**
1/8 - odds of GG TT – homozygous for SP6 with no KRT25 present – full mane and tail
1/4 - odds of GG TC – heterozygous for SP6 with no KRT25 present – full mane and tail
1/8 - odds of GG CC - straight

Cross #9



Cross:
AGCT × AGCT

	AC	AT	GC	GT
AC	AACC	AATC	GACC	GATC
AT	AACT	AATT	GACT	GATT
GC	AGCC	AGTC	GGCC	GGTC
GT	AGCT	AGTT	GGCT	GGTT

The cross of two individuals that are heterozygous for both KRT25 and SP6 and carry both genes at the same time. Both Parents Dual Genes- Heterozygous- AGCT

25%	12.5%	12.5%	12.5%	12.5%	6.25%	6.25%	6.25%	6.25%
AG CT	**AG CC**	**AA CT**	**AG TT**	**GG CT**	**AA CC**	**GG TT**	**AA TT**	**GG CC**

1/16 (6.25%) odds of having a straight foal (GG CC)

1/16 (6.25%) odds of having a foal that is homozygous for both KRT25 and SP6 at the same time - **dual gene**

1/4 (25%) odds of having a foal with a scant mane and tail - AA or homozygous for KRT25

1/4 (25%) odds of having a foal that is heterozygous for both Krt25 and SP6 – **dual gene**

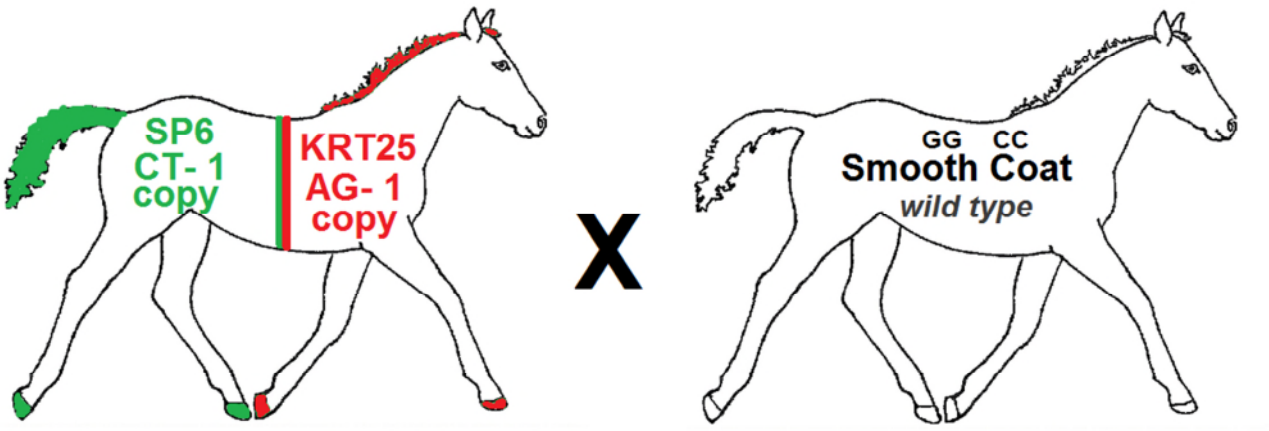
1/8 (12.5%) odds of having a foal that homozygous for KRT25 and heterozygous for SP6 – **dual gene**

1/16 (6.25%) odds of having a homozygous SP6 that does not carry KRT25

1/16 (6.25%) odds of having a homozygous KRT25 foal that does not have SP6

15/16 (93.75%) odds of having a curly foal

Cross #10



The cross of a horse with two genes for curl (heterozygous for both KRT25 and SP6) and a horse with no curly genes- Wild Type- GG CC No mutation (straight) or Smooth Coat.

Parent #1 – AG CT Dual Genes **Parent #2 - GG CC** Straight or Smooth Coat

Cross:
AGCT × GGCC

	AC	AT	GC	GT
GC	AGCC	AGTC	GGCC	GGTC
GC	AGCC	AGTC	GGCC	GGTC
GC	AGCC	AGTC	GGCC	GGTC
GC	AGCC	AGTC	GGCC	GGTC

(AGTC) with a totally straight mare (GGCC) **(AGTC) X (GGCC)** would produce the following genotypes:

25% AG CC **25% AG TC** **25% GG CT** **25% GG CC**

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 with no SP6

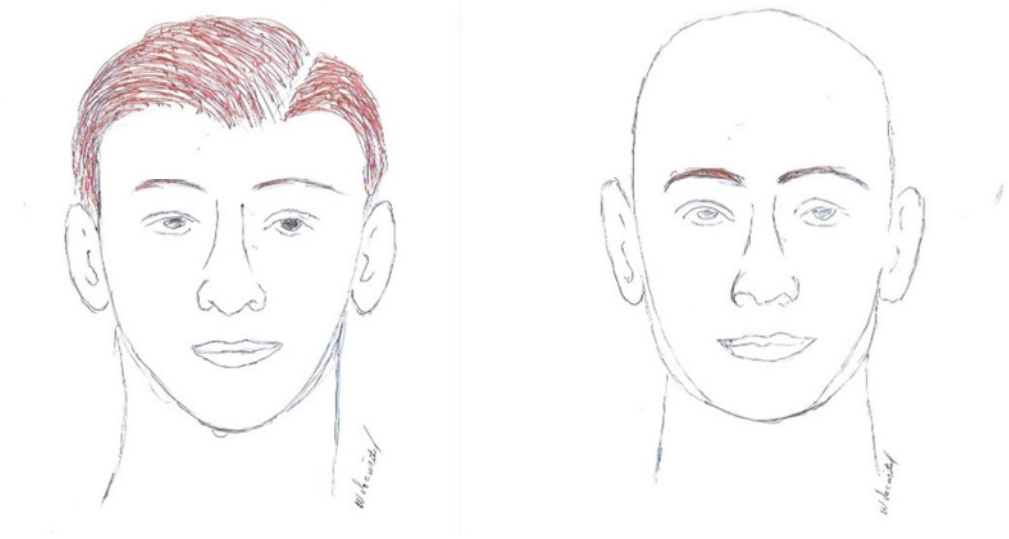
1/4 odds of having a foal that is heterozygous for both SP6 and KRT25 – **dual genes**

1/4 odds of having a foal that is heterozygous for SP6 with no KRT25

Zero chance of having a foal with the AA combination and sparse mane and tail

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**.



For example, the gene for total baldness is epistatic to the gene for red hair. It makes no difference if a person inherits the gene for red hair if he also inherits the gene for baldness. Baldness is epistatic to red hair. Baldness will always "mask" the effects of red hair.

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 baldness masks the effects of a red 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.

Hypotrichosis and Dysplastic Hair Shafts

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.

KRT25

The missense mutation found in KRT25 is a mutation of a keratin gene which produces a type 1, inner root sheath – specific keratin protein that is essential in the assembly of keratin protein complexes required for the proper assembly of the hair shaft. 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 also a consequence of the abnormal structure. The brittleness is thought to increase with UV light which is at its peak during the summer months.

Because KRT25 produces dysplastic hair shafts, it is epistatic to SP6 which produces normal hair shaft morphology.

SP6

The missense mutation found in SP6 is a mutation which affects a transcription factor. SP6 produces a **transcription factor**. The transcription factor's role is to produce a protein which controls the initiation and speed of transcription of genetic information from the DNA molecule to the messenger RNA molecule, by binding to a specific DNA sequence. Much like a capitol letter is used to show the beginning of a sentence, the transcription factor protein shows the messenger RNA when, where, and how fast to begin reading that segment of the DNA chain. The function of transcription factors is to regulate - turn on and off - genes in order to make sure that they are expressed (produce critical proteins) at the right time and in the right amount to produce, in this case, a hair follicle.

The slight variation in SP6 from wild type to SP6 mutation produces an inner root sheath of the hair follicle which is ovoid shaped. The **inner root sheath** acts as a mold to form the shape of the growing hair shaft. Ovoid shaped hair shafts curl. Therefore, the hair shaft produced by the SP6 mutation is **not dysplastic.** It has a normal hair shaft anatomy and is not brittle, but instead has an ovoid shape which produces curl.

Article and illustrations by Dr Mitch Wilkinson

Graphic's by Bunny Reveglia- ICHO

ICHO Testing Available:

Test your horse for the KRT25 Gene
$35.00
Form: KRT25 Test Form

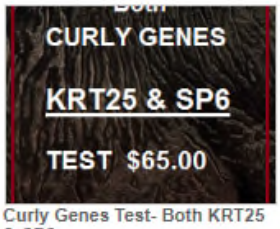
Curly Gene Test- KRT25
$35.00 Add to Cart

Test your horse for the- **SP6 "Curly Jim" Gene.** $35.00
Test form-

sp6_test_form.pdf
Download File

Curly Gene Test- SP6
$35.00 Add to Cart

Test for both **KRT25 & SP6** Genes
$65.00 *form below*



Curly Genes Test- Both KRT25 & SP6
$65.00 Add to Cart

krt25_and_sp6_test_form.pdf
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