
The “Syndrome”: *Not Just for Fell Ponies Anymore*

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A Dales pony mare and a very healthy foal (Photo courtesy of Anna Hill)

Introduction

A rare and lethal “syndrome” has been observed in Fell pony foals since at least 1993, when it was first reported by Paul May, a veterinarian in Cumbria. Worldwide approximately 10-12 cases are reported each year out of 300-350 foals that are born, and the disease has been seen in all major bloodlines. To date in North America, less than five confirmed cases (from several different lines) have occurred over the last 15 years, so the disease is indeed very rare. Affected foals appear healthy until about 4-6 weeks, when they begin to lose condition and eventually die or are euthanized before weaning. The cause of death

is usually severe anemia as well as multiple infections. The underlying cause of disease is unknown, but is believed to be due to a genetic fault. Foals that remain healthy at 2 months of age are very unlikely to develop the disease, while those that reach 3 months of age are no longer considered at risk. The “Fell Foal Syndrome” has always been somewhat of an ambiguous term for this disease. What it has referred to is a *syndrome*, or a group of characteristic signs and symptoms, seen exclusively in sick, newborn Fell pony foals. Unfortunately, in 2008, the latter restriction is no longer accurate, as the first confirmed case of the “Syndrome” occurred in the *Dales pony* breed in the UK.

What Exactly Is “The Syndrome”?

For those who might not be familiar with the “Syndrome” that affects these ponies, it may first be helpful to review some details about this disease, especially since the name given to it does not provide such information. Like several human diseases which share similar features, it is a type of **inherited bone marrow failure syndrome**. If one were to give the disease a more descriptive, scientific name, it might be called something like “*Congenital Dyserythropoietic Anemia and Immunodeficiency*”, or perhaps more specifically, “*Congenital Dyserythropoietic Anemia and B Lymphopenia*”. Ok, granted at first glance that may sound like nothing more than a mouthful! But with a few definitions, a better understanding of the disease itself is possible, rather than just memorizing a laundry list of symptoms. *Congenital* means “present at birth”. *Dyserythropoietic* literally means “ill” or “bad” (*dys-*) “red blood cell” (*erythro-*) “production” (*-poietic*). *Anemia* refers to low hemoglobin levels in the blood. (Hemoglobin is the protein that carries oxygen within red blood cells.) Affected foals eventually become **profoundly anemic** (usually by 4-6 weeks of age), with PCV (packed red cell volume) of <10%. The latter finding is almost diagnostic of the Syndrome in a foal of this age, since not much else could cause a PCV to be that low other than obvious hemorrhage or hemolysis (red cell destruction within the blood), both of which are usually easily excluded. The profound anemia develops because affected foals lose the ability to make red blood cells, the cells responsible for carrying hemoglobin. Without hemoglobin, they cannot get oxygen to their tissues. Thus, the anemia, with resultant oxygen deprivation, causes **some of the symptoms of the “Syndrome”, including lethargy, exercise intolerance, poor growth and/or “failure to thrive”**. The foal may simply appear less active than other foals his or her age, and may not grow as well. Note again that foals *become* profoundly anemic – that is, aside from rare exceptions, they are *not born* profoundly anemic. For example, some Syndrome foals which had blood work done early have been shown to have *normal red blood cell counts at birth*¹. Furthermore, in at least one Syndrome foal, a bone marrow sample done very

early in life showed *destruction* of the cells responsible for making red blood cells in the bone marrow¹. Most Syndrome foals do not undergo bone marrow biopsy until they are at the end-stage of disease, by which time an absence of these cells – a condition known as a “red cell aplasia” -- is characteristic. No one knows why these cells disappear. What we do know is that something happens around the time of birth that adversely affects these cells and thus their ability to produce new red blood cells. Meanwhile, red blood cells already in circulation have a fairly long half life of about 120 days. During this time, the foal is also growing and needs to expand its blood volume similar to body growth. It is not unusual for a Fell pony foal to double its birth weight in only 3 or 4 weeks, which normally requires a proportional increase in blood volume. In fact, many normal foals will develop a mild, “physiologic” anemia during the first few weeks of life because of the rapid blood volume expansion, when even their “normal” marrow cannot keep up with demand. This anemia is mild and self-limited. In Syndrome foals, however, their bone marrow cannot even begin to keep up with demand for new red blood cells, so the foal becomes rapidly and progressively anemic. The rate of progression of anemia may vary with the growth of the foal – small foals with failure to thrive early on may develop the anemia more slowly since they are not growing as rapidly, whereas those that are more robust and larger early on may “crash” relatively sooner. By 4-6 weeks most syndrome foals are profoundly anemic.

The “*B Lymphopenia*” is also a key component of the “syndrome”. Lymphocytes are white blood cells, which are cells involved in the immune system. The “*B*” refers to the *B cell*, a specific type of white blood cell which is responsible for producing disease-fighting antibodies. The *-penia* in “lymphopenia” refers to a deficiency. Thus, affected foals have an immunodeficiency, or deficient immune system, due to having a **deficiency of B lymphocytes**. This is the cause for a variety of other symptoms typically seen in the syndrome, which are all related to this **immunodeficiency: nasal discharge, “scours” (diarrhea), pneumonia, or other signs of infection**. Furthermore, it has been shown in at least one Syndrome foal that was tested early that initial *B lymphocyte levels*,



Fortunately "the Syndrome" is very rare. So like the vast majority of Fell pony foals, this one is also robust and healthy!

as well as red blood cell levels, were normal at birth². In fact, research has shown that B lymphocyte numbers may be quite variable in very young, normal Fell foals less than 1 month of age². Therefore in foals of this age group, the B lymphocyte assay may neither confirm nor exclude the diagnosis of the Syndrome, i.e., this test cannot be used as a "screening test" in very young foals. However, very low B lymphocytes (**not** total lymphocytes) in foals approximately 4 weeks of age or older, as shown in other studies, may provide presumptive evidence for the Syndrome in the appropriate clinical setting. B lymphocyte testing requires a **fresh blood sample and a specialized lab**,³ since this cannot be measured with the standard "CBC" (or complete blood count, which only measures total lymphocytes). By the time a Syndrome foal is 4-6 weeks of age, the B lymphocyte level - and thus the ability to produce antibodies - is severely deficient. Even though affected foals initially receive antibodies from the mother via the "first milk" or colostrum, they are not enough to protect these foals from infections. What causes the B lymphocyte levels to plummet? Again, at this point no one knows, but it is apparent that for some reason -- right around the time

of birth -- Syndrome foals lose their ability not only to make red blood cells, but also B lymphocytes, and it is the lack of both which ultimately cause "the Syndrome".

Thus, a presumptive diagnosis of the Foal Syndrome can be made in a foal of the appropriate breed and age which exhibits the classic signs and symptoms of the Syndrome, including profound anemia (PCV <10%) and B lymphopenia. **However, the single most reliable test for confirmation of the diagnosis, providing an adequate sample can be obtained, is a bone marrow aspirate.** An **important caveat** to this statement, however, is that this test must be **interpreted by veterinary clinical pathologist experienced in making and excluding this diagnosis.**³ Furthermore, if there is any problem getting a fresh blood sample tested for B cells in a timely fashion, or if the foal is very small and/or younger than 4 weeks (in which case he/she may not yet be severely anemic), the bone marrow aspirate may be the only way to confirm or exclude the diagnosis. Lymph node evaluations (and spleen evaluations if post-mortem) are also helpful. Bone marrow aspirate is often obtained from the sternum but can also be obtained from the pelvis at the point of the hip. This procedure involves a simple needle aspiration at the point of the hip, where the bone in a young foal is still rather soft and a sample can be obtained with a needle almost as easily as a blood draw. A local anesthetic is helpful so the procedure is painless, and, since it is easiest to do this with the foal lying down, a tranquilizer is usually necessary. ***A bone marrow aspirate may be the only way to reliably confirm or exclude the diagnosis of the syndrome in foals less than 1 month of age, again allowing for the caveat above.*** This is particularly important if the foal has an atypical or early presentation of illness, which could be due to something other than the Syndrome. If the foal dies or is euthanized, bone marrow rapidly deteriorates so the sample *must* be obtained within 30 minutes of death. Since it can be technically difficult to aspirate marrow through a needle post-mortem, pathologists usually recommend cracking open a bone and retrieving marrow directly within 30 minutes of euthanasia.

Foals of any breed can succumb to problems such as anemia and infections from a variety of causes, so just because it is a sick Fell or a Dales foal it does not necessarily mean the cause of its illness is the “Syndrome”. Furthermore, if an effort is made to reliably confirm the diagnosis, it may be a consolation to know that a foal that is lost can at least help with the research and thus may help prevent the disease from affecting other foals in the future.



Most cases of the Syndrome in Fell ponies — and the only case confirmed so far in Dales pony breed — have been in the UK, where most of these rare ponies reside.

Syndrome Research and the Elusive “Marker”

In 2003, FPSNA, Inc. helped to organize a multidisciplinary research team to study this problem, which grew to include scientists from Harvard, Cornell, University of Kentucky, and Texas A&M, as well as scientists and veterinarians abroad who have submitted samples from affected foals for this research effort. Our team also applied for an NIH grant, because research on the syndrome may end up helping children with certain rare forms of inherited bone marrow failure, such as Dyskeratosis Congenita, Diamond-Blackfan Anemia,

and Congenital Dyserythropoietic Anemia Type II. The major stumbling block in this research has been finding the genetic cause for this disease, which the NIH has required prior to further consideration of a grant proposal. The genetic research is very costly, and while the affected foal sample numbers are probably adequate for study using state-of-the-art technology in genetics, the cost of using this technology has been prohibitive. Fortunately, in 2008 Dr. June Swinburne of the Animal Health Trust in the UK received a large grant from The Horse Trust to identify the genetic defect in the Fell Pony Syndrome. They have the genetic samples, and they have the funds, so there should be no stopping them now to finally find the elusive faulty gene!

Although the terms “genetic marker” and “genetic defect” (or “faulty gene”) are often used interchangeably, these are not necessarily the same things. A **genetic marker** is a gene or DNA sequence with a known location on a chromosome that is associated with a particular abnormal gene or trait. Because pieces of DNA that lie near each other on a chromosome tend to be inherited together, a “marker” can be used to determine the precise inheritance pattern of the associated defective gene and predict carrier status, even if the identity of the defective gene itself may not be known. That is, a genetic marker can be valuable for understanding inheritance patterns, even if the precise identity of the associated **genetic defect** — the particular abnormal gene causing disease — is unknown. However, identification of the genetic defect itself is the ultimate goal of research because it identifies the actual cause of the disease at the molecular level.

What kind of genetic defect are scientists expecting to find? Most scientists are assuming that it follows a **simple recessive pattern of inheritance**, and the disease is 100% lethal if a foal is *homozygous recessive*, thus having 2 copies of the faulty gene (i.e., one from each parent). Note that an animal is *homozygous* for a particular gene if both alleles, or alternate forms of a gene, are identical, and *heterozygous* for that gene if the alleles are different. The expressed allele in a heterozygous pair is known as the *dominant* allele, the unexpressed one as the *recessive* allele. A recessive allele is “hidden” (or not expressed in the “phenotype”

or outward appearance) if the corresponding dominant allele is also present, and thus the animal may be considered a hidden “carrier” for that trait. Simple modes of inheritance can be illustrated with a tool called a “Punnett Square”. As an example, let us assume that “N” refers to a “normal” allele, and “s” refers to an alternate abnormal allele for the same gene which in this case can cause the Syndrome. The projected possible outcomes for breeding 2 heterozygous carriers (*Ns*) with a simple inheritance pattern are shown below in a Punnett square. The genotype of each parent is shown in the top row and left column in non-shaded boxes. The shaded boxes show the possible combinations of these genes in the offspring. In this type of pattern, if 2 carrier ponies (each *Ns*) are bred to each other, there is a 25% (1 in 4) chance of producing a homozygous recessive, affected foal (*ss*), a 50% chance of producing a heterozygous unaffected carrier foal (*Ns*), and a 25% chance of producing a homozygous dominant, unaffected, non-carrier foal (*NN*). Notice that, in this situation, there is thus a 75% chance of producing an unaffected foal (*NN* or *Ns*) if 2 carriers are bred, and just as much chance (25%) of producing an affected (Syndrome) foal (*ss*) as a perfectly normal, unaffected, non-carrier foal (*NN*).

		Parent A	
		N	s
Parent B	N	NN	Ns
	s	Ns	ss

On the other hand, if a carrier (*Ns*) is bred to a non-carrier (*NN*), one will produce 50% non-carrier foals (*NN*), 50% carrier foals (*Ns*), and 0% affected foals (*NN* or *Ns*), again assuming a simple recessive mode of inheritance, as depicted below.

		Parent A	
		N	N
Parent B	N	NN	NN
	s	Ns	Ns

However, the above discussion of breeding outcomes is based on the **assumption** that the gene responsible for the syndrome follows a simple recessive

pattern of inheritance. This still has not been proven! This is really important to understand as a breeder. **The assumption could be wrong.** Other possible inheritance patterns suggest that we could be either over- or underestimating the significance of suspected carriers. For example, it is possible that the disease is indeed **recessive but not “simple” recessive**, but rather **“complex” recessive with incomplete penetrance**, requiring not only 2 copies of the gene but also some environmental “trigger” or other unknown factor or modifying gene to express the disease. Local environmental differences could thus explain why some breeders have a greater incidence of the syndrome relative to other breeders, even though the bloodlines of the ponies they are breeding are similar. If the disease is recessive with incomplete penetrance, this could mean the homozygous recessive (*ss*) phenotype is not necessarily 100% lethal nor associated with any expression of the disease (i.e., the syndrome phenotype). The latter has very important implications, even if rare, because it could mean that some presumed “carriers” might actually be “double carriers”, i.e., carrying 2 copies of the gene rather than one, and thus have the potential to pass on one copy of the faulty gene *every time!* Would this then explain why a few presumed carrier mares and stallions have seemed to produce syndrome foals in greater frequency than expected, i.e., greater than “1 in 4”? Or is it simply due to an unlucky roll of the dice each time? Until we find the marker and study this further, we do not know. Similarly, there are other possible modes of inheritance, including **dominant with incomplete penetrance**. This is actually the mode of inheritance in a somewhat similar human disease. In this type of inheritance, one parent, rather than both, is a carrier, but neither parent shows signs of disease. Once again, some other “trigger” is needed to cause disease expression in the offspring if the gene is inherited. This pattern obviously has very different implications for breeding management, but it might explain the sometimes confusing breeding outcomes and statistics. Nonetheless, barring any easily-identified and controlled “environmental factor” at play, the least complicated, easiest-to-manage mode of inheritance is the simple recessive mode, so we should all probably hope the original assumption is correct! Still, at this point, it is important to remember that whether this definitely follows a simple recessive mode of inheritance remains

hypothetical and to date is unproven. Regardless of the mode of inheritance, however, please also remember that the *disease itself only appears to affect young foals prior to weaning -- not older ponies (including suspected carriers)* -- so management of this problem is strictly limited to breeders.

From all the above speculation about the mode of inheritance, one can see why it is imperative that the marker is identified, so that breeders can make breeding decisions based on fact rather than hypothetical information. Until then, breeders must be cautious and avoid jumping to conclusions that might harm the breed and its gene pool in the long term. Fell ponies are known for exercising “sensible caution” when venturing into unfamiliar territory, and we can learn a lot from our four-legged friends! If we are to show the same sensible caution here, we would be wise to generally continue breeding as before, other than to simply avoid breeding a suspected carrier to a suspected carrier. Although not fool-proof, the latter is our best bet right now to avoid producing an affected foal. On the other hand, to eliminate a number of “suspected” carriers from the gene pool of a rare breed, whose carrier status at this point can only be on the basis of speculation, will mark the end of the breed as we know it. What this does is send our precious gene pool down a slippery slope of destruction. Indeed, there were 3 major genetic bottlenecks in the Fell Pony breed during the last century, the 3rd of which is probably responsible for propagating the syndrome fault throughout the Fell gene pool. A 4th genetic bottleneck could effectively be lethal by inbreeding the remaining few ponies. Thus, presumed carriers should NOT be eliminated from the gene pool solely because of a presumed or even proven carrier status, especially when we are dealing with a rare breed. As with any breeding program, **all** the strengths and faults of any pony should be considered in deciding whether a pony, potential carrier or not, has good things to offer to the gene pool. Some of the best overall quality stallions and “Supreme Champions” of the last decade are known to have produced Syndrome foals. Imagine what the breed quality would be like without the contributions of these ponies! Likewise, the same holds true for the critically-endangered Dales ponies, whose gene pool will be devastated by any further restriction. The bottom line to remember is an FPS motto, “Breed

and we will succeed!”

The Dales Connection ⁴

Anyone who has studied Fell pony pedigrees should not be surprised by the occurrence of the Foal Syndrome in the Dales breed. Indeed, one may have wondered why this took so long to happen. Dales ponies originated “next door” to Fell ponies in northern England and most definitely share common ancestry. Remember that the first stud book for Fell ponies was the Fell section of the Polo Pony Society Stud Book in the late 1800’s. There was no Dales stud book at the time, and Dales ponies were actually registered in the Fell section of the Stud Book. The Polo Pony Society Stud Book eventually became the National Pony Society Stud Book, and even after the Dales were granted their own section, the Fell and Dales sections were not completely “closed” to each other until 1969/1970. These facts alone are more than enough to explain their close kinship with frequently overlapping pedigrees.

Up until now, with a relatively large number of common ancestors from different lines, it has been difficult to try to “crack the case” regarding the origin of the Fell Foal Syndrome. Although the genetic bottleneck caused by the Enclosure system is often blamed as the source of the alleged faulty gene, more likely this system helped to propagate the gene in the small population but the original source may well have come before that. Some of the more recent common ancestors like **Waverhead Rambler 4101**, f. 1959, and **Glenwelt 4546**, f. 1964, from the bottleneck of the 1960’s do not appear in the pedigrees of all stallions which are known to have sired Syndrome foals. More remote common ancestors such as **Teasdale Comet 904** (Dales), f. 1898, and **Blooming Heather 325** (Fell), f. 1880, are also common to all Dales pedigrees, which is no surprise since the Fells and Dales shared a stud book at that time. However, if these were the source of the presumed faulty gene, the Dales ponies should have a similar incidence of the syndrome and – at least to date -- they have not. Thus, most likely the origin of a faulty gene was somewhere in between – i.e., after the Dales Society established its own stud book but during the genetic bottleneck that occurred during the World War and post-war

years, and likely involving the prevalent “travelling stallions” of this time period.

Not surprisingly then, the pedigree of the affected Dales foal supports the latter conclusion. As expected, it includes those stallions more remote in time that are ubiquitous in both Fell and Dales pony pedigrees – i.e., Teasdale Comet and Blooming Heather, so these ponies still cannot be eliminated as a possible common, albeit remote, link to the source of the faulty gene. Similarly, **Mountain Ranger 598**, f. 1906, also (remotely) appears many times on both sides of the affected Dales’ foal pedigree. Furthermore, he appears in all currently-licensed Fell stallion pedigrees as well, so he still cannot be excluded as a common remote link which also continues further back via his grandsires, Teasdale Comet and Blooming Heather. On the other hand, barring inaccurate pedigrees, this case does effectively exclude some other possible sources, including more recent prevalent Fell stallions like Waverhead Rambler and Glenwelt, in addition to Storm Boy, Bob Silvertail, and also Heltondale Victor from the earlier part of the war-time era. One may also note, however, that the pedigree of the affected Dales foal includes another pony on both sides of the pedigree – **Hazelgill Darkie 11887**, foaled in 1947 and bred by A. Armstrong. She was an inspected mare of unknown breeding. For her to be the source, since her line does not continue in Fell pedigrees, one would have to assume that her ancestry was common in the Fell breed. Overall she seems an unlikely source.

However, what the pedigrees of both parents of the Dales foal also include in more recent ancestry is the Dales stallion **Master John 2883**, foaled in 1946, and bred by James Wilson of Litz Garth in Stainmore. In fact, in the case of one parent of the affected Dales’ foal, Master John appears more than once in the pedigree. Master John was a 14.0 hand Dales stallion that was sired by **Black Jock II 2321** (Dales) (a great-grandson of Teasdale Comet), out of Dainty Molly (unregistered) by Seldom Seen. (The latter has no number listed in Master John’s pedigree in the Stud Book but is probably **Seldom Seen 1628** (Fell), a grandson of Teasdale Comet.) Master John was registered in the Fell Stud Book in 1952. He actually just made it in – being up-to-height at 14.0 hands.



At the back o' Skidda': Joe Baxter travelling his stallion, Master John. They had walked from Threlkeld through Skiddaw Forest to Caldbeck. Photo by Miss Peggy Crossland, circa 1952 (Courtesy of the Fell Pony Society UK)

It has been said that there was a push at that time to use Dales stallions in the Fell breed because of the small remaining registered Fell population after World War II. He was black with 2 white hind heels, and was well known as the last of the travelling stallions when he was owned by Joseph Baxter of Guardhouse in Threlkeld. He was also the last Dales stallion to be registered in the Fell Stud Book. Since the Fell and Dales Stud Books were not yet completely closed to each other, Master John also sired registered Dales pony stallions, including **Stainton John 4522** (f. 1962), **Kexwith John 4645** (f. 1965), as well as other registered Dales mares. His Dales-registered ancestors in his tail male line, including **Black Jock II 2321** (f. 1937), **Black Prince II 1809** (1932), and **Black Diamond 1635** (f. 1925), also sired other registered Dales progeny. Whether Master John himself, or one of his direct ancestors, was the original source of the faulty gene, we may never know for sure, but this may be as close as we have come to solving the pedigree puzzle of the Syndrome. *Master John was a travelling stallion during a period of genetic bottleneck in the Fell breed, and thus appears not only in the tail male line of approximately 30% of currently-licensed Fell stallions, but also – through maternal lines – in the pedigrees of all the remaining currently-licensed Fell stal-*

lions. Thus, the fact that he appears in 100% of current Fell stallion pedigrees and now also appears several times relatively recently and on both sides of the pedigree of an affected Dales foal provides compelling evidence that either he – or one of his direct ancestors – could have been the original source of a faulty gene. Since Master John’s line—while still very common—is not as prevalent in current Dales stallion pedigrees⁵ compared to that of Fells, this might also explain why the disease is more common in Fell ponies. Furthermore, as previously noted, Master John was a great-great grandson of Teasdale Comet. Mountain Ranger was also a grandson of both Teasdale Comet and Blooming Heather. While Mountain Ranger appears in all currently-licensed Fell stallion pedigrees, neither he nor Blooming Heather appears in the pedigree of Master John. Since *all four* of these stallions appear in *all* current Fell stallion pedigrees, but Master John *does not* appear in *all* current Dales stallion pedigrees, affected Dales’ foal pedigrees in the future may be the only way to eliminate Master John as a possible source. However, with overlapping ancestry and at least three other remote, ubiquitous ancestors in both the Fell and Dales breeds —Teasdale Comet, Mountain Ranger, and Blooming Heather — it will likely never be possible to narrow the possible sources down to only one, at least not on the basis of pedigree analysis alone.

Thus, although it would be sadly ironic and unfortunate if the genetic fault responsible for the “Fell Foal Syndrome” was introduced by a Dales, we may never know for sure. More importantly, the fact that this disease now affects the critically-endangered Dales breed, too, is truly devastating. Hopefully something good will come of this and the Fell and Dales Pony Societies will join forces and make progress in both re-

search and management of this disease. Perhaps also the name of the disease should rightly be referred to simply as the “Foal Syndrome”, or better yet something more descriptive of the disease itself, since it has definitely occurred in more than just the Fell breed. Finally, one may also wonder -- with both Fell and Dales ponies commonly used to cross with “coloured” ponies and cobs, which themselves often have part-Fell and/or part-Dales ancestry -- if the colorful Gypsy horses will be the next affected by the Foal Syndrome? Time will tell. . . MJG

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¹Data courtesy of Dr. Tracy Stokol, Dept. of Population Medicine and Diagnostic Sciences, College of Veterinary Medicine, Cornell University.

²Data courtesy of Dr. Julia Flaminio, Dept. Clinical Sciences, College of Veterinary Medicine, Cornell University

³Recommended Lab in North America (for affected NA foals) for bone marrow evaluation and B-lymphocyte testing is Cornell University College of Veterinary Medicine

⁴Information adapted from the author’s upcoming publication, *The Fell Pony Family Album*, © 1998-2008.

⁵A preliminary study, which included pedigrees of 65* currently-licensed Dales stallions (2008) in the UK (45) and NA (20), showed 62% have Master John at least once in their pedigrees. *(Complete stallion list not available for study.) All text and photos in this article © MJ Gould-Earley (1998-2008), unless otherwise noted herein, and may not be reproduced in any form without written permission of the author.

DNA Testing of Fell Ponies

Many owners have already submitted samples on all Fell ponies, including all foals and imports, as part of Dr. Gus Cothran's ongoing multi-breed equine research program which began in 2000 at the University of Kentucky and is continuing now at Texas A&M. DNA-testing with parentage verification is also required for FPS registration if foals are conceived by artificial insemination, as well as for stallion licensing. If you have additional ponies that have not yet been tested, you are encouraged to do so. DNA samples on as many Fell ponies as possible are needed to help determine the genetic cause for the

syndrome. Your cooperation is appreciated!

The cost for testing (in 2008) is \$27. Please contact Dr. Cothran to verify current price if you plan to send a sample.

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