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Foreword

Those of us that have a Volpino Italiano are passionate about the Volpino!! We love our dogs and we are proud of them. We are also very proud of the absolute beauty they exhibit. Those of us that know the Volpino are also proud of their breed's history. We consider the Volpino a 'special' breed.

The Volpino has a character unlike many others. Of course each individual is different but they all seem to share the same basic traits which I just like to call 'happy'!! They also have a sensitive side which too many may often seem like 'insecurity'. Their attachment to their masters and their immediate family may cause problems, but it is the core to their being. These are the friendliest and most loving dogs you can find.

Unfortunately there is not a great deal of information on the Volpino Italiano. At one time they were 'misrepresented' in the past as being an animal with very few defects or health problems (i.e. only the strongest surviving specimens were used). While this may have been honestly thought by some of the individual breeders in some respects it is not totally accurate. The Volpino, though, when compared to many other breeds will have far fewer health concerns!!

I have written the following articles (summaries actually) that should help you understand the Volpino Italiano, its history and a few of the major genetic problems it faces. In most cases the individual articles will also apply to other breeds. As time allows I will try to add to the story.

If you are so inclined there are many websites on the internet that can offer more in depth information for study.

I would like to hear back from anybody that reads my articles. If you have any criticisms, suggestions, or would simply like to send me a note then please does so.

Send any correspondence or suggestions to

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Michel B. Rubini

Introduction

My Volpino Italiano

For many of us there will always be that one special dog that enters our lives at a particular time and grows with and becomes part of the family. This dog becomes close to all (or most of) the family members. He/she grows up with our kids and greets them all at the door happily. They grow up with us at the family table, lying beside us as the family watches TV and often sleep in a bed with us. That dog will be the one that enters our heart at a fast-paced time of our lives with our growing family. You will know this dog if you ever had one. He will be in the grad photos, the beach photos, the Christmas pictures and yes, even the wedding photos. It will be the dog that deeply affected your family when his or her short life ended. That dog will be the one you will always seem to think of and are reminded of.

Our beloved buddy was that dog. He entered our lives in Feb of 1989 and left us in June of 2004. He grew up with our kids and he was there when they grew up and left. His passing left emptiness in Karen and me, and our hearts hung deep and heavy in our stomachs.

Even though Buddy lived a long life, in truth he was a frail dog, as most Pomeranian's tend to be. He had too many health issues. And so, when we began searching for another pet, we decided against another Pomeranian. Getting another Pom would also have seemed a betrayal of sorts to our Buddy.

And so we began our search for another dog. Our criterion was that the animal would be bigger and would not have the number of health issues that Poms have.

We first looked at the American Eskimo! While this animal is beautiful, we quickly learned that the breed at that time was being devastated with eye problems. We were also warned-- and saw-- that the animal's disposition on average was not quite a friendly one. Later we almost bought a Shiba Inu, which we saw at an exhibition. We turned away from this beautiful animal because of its aggressive and independent nature.

Then we discovered the Volpino Italiano. The breed was like a Pomeranian we thought, but somewhat larger and stronger so we were intrigued and studied it more. Back then, the advertising on the Volpino

was that they were long lived and without any major health concerns - because of their selection from strong health farm animals. This is what we were looking for.

In 2005 during a trip to Italy we wanted to see some Volpino, and so we visited the beautiful home of Roberto Francini in Tuscany. There we met Roberto, his lovely wife, and his daughter Elisa. Elisa spoke good English and translated for us. It is quite an experience to walk into a location with over 20 wonderful barking dogs warning of our presence and then to be happily mobbed by them. After a few minutes we noticed an absolutely beautiful Volpino. He was confident and headed towards another male which he immediately challenged. This was Geo. Geo was one of the most beautiful dogs I had ever seen. I told Roberto that I wanted a puppy that was sired by him. And so a deal was struck between us that I would receive a male puppy from Geo and a female puppy to breed.

In May of 2006 I traveled to Italy to pick up our puppies, which Karen had already named Dezi and Bella. True to form they were beautiful. I was so pleased. Roberto was kind enough to give me tips on grooming and taking care of them. I then left Italy with my dogs and headed back to Canada.

House training was a breeze and both settled in with us wonderfully. It was good to have two happy puppies to play with. There was no danger of the house being too quiet. Dezi was inquisitive but showed a timid nature indoors, while Bella was seemingly fearless. Outdoors though Dezi would run up to any dog or person while Bella would unleash a chorus of warning howls.

In May of 2007 we entered Dezi into his first dog show, a rare breed competition in Tillsonburg, Ontario. We were so inept at showing that the judges actually took us aside and gave us instructions on how to handle and walk the dogs. Several judges stated they were very impressed with Dezi's gait and how he looked. He won BOB, BOG and Best in Show.

After several other shows, Dezi kept winning BOB over poor Bella and also won many group awards. Later we entered them into the UKC Kalamazoo show and it was here we met Kevin Joiner and his beautiful Volpino's. Kevin's dogs were the first Volpino to ever gain champion status at UKC. These were also the first Volpino's we ever met outside of Italy. For those of you interested in showing your dogs, I will say the show experience is rewarding, and I recommend it to anyone who has the time, and money (it can get expensive) to invest in their dogs.

In 2008 we received a note from Terralea Collins in southern Tennessee. She had imported four Volpino puppies from Roberto Francini and had started breeding her dogs in 2006. We kept in contact and struck up a relationship that exist to this day. She started the North American Volpino Club in the hope of receiving AKC recognition for the Volpino Italiano. Although we tried, on several occasions tried to cross breed our dogs the attempts were never successful.

Dezi and Bella had 3 litters with 13 puppies. Having a growing litter of puppies is wonderful!! Bella was a wonderful mother and even Dezi took a roll at being a parent. Although the puppies at times were highly energetic and playful, both parents took their turns in playing with them. The puppies grew up fast. Most are healthy but one female did develop Atkinson's disease and another was killed by a Bobcat in California.

Now, our dogs are healthy. Dezi likes to eat too much and try as we might he has always been 'chubby'. Bella is slim and dainty. There are no major health concerns with our dogs, but Bella (since she was a puppy) always had a tendency to throw up.

In late 2009 we received an email from Raija Kokkonen of Finland asking us about our dogs. In her note she stated that her dog's brother, mother and grandmother all developed the eye disease Primary Lens Luxation (PLL). Apparently four other Volpino's had also developed the eye condition and went blind. At the time the genetic test to isolate the mutated gene causing PLL had not yet been developed. To be truthful we really didn't understand the situation or the meaning of the disease at first. We had our dogs checked and they were fine.

Karen and I started to research the problem, but the hereditary information was missing since the gene causing the disease was not yet found. By late 2009 the mutated gene that caused PLL was finally isolated and a genetic test was available. We contacted Liz Hansen at the OFFA lab at the University of Missouri in 2010 to learn more about the disease and the test. To our amazement she already knew about the PLL infliction in the Volpino race but did not know that any Volpino existed in North America. She then set up a program for us where we could send blood samples from 20 dogs, free of charge. They would analyze it for the defective gene that causes PLL and also for the genetic disease called canine Degenerative Myelopathy (DM). I sent blood samples to the university and then contacted all the other owners and breeders of Volpino's to share the information with them. Dezi tested positive as a carrier while Bella tested normal (clear). Eventually blood samples from 18 other dogs were sent to the university and the results were 4 PLL carriers and 16 PLL normal/clear (see section on PLL). All the DM results returned were negative, so it seems that Volpino's are spared from this disease.

Now, the thing with PLL is that it can devastate a family. Unlike other eye diseases, PLL can become unbearably painful for the animal and is very, very expensive to treat. Treating the disease can cost the family thousands of dollars. So the family must choose between the money and the dog, and for most families the money will understandably win. They will be forced to euthanize their pet or watch it suffer from this painful and devastating disease.

After some more research, we ran across an Italian veterinarian report written in 1995 about PLL in Volpino's. Unfortunately, at that time the only way to know if your dog carried the mutant gene was if the animal actually developed the disease.. So the obvious conclusion is that the breed must have suffered from this condition for many, many years prior to 1995. It is surprising, therefore, that more than ten years later when I bought my Volpino's, there seemed to be a complete silence or denial about the condition, (and other eye conditions) amongst all the breeders.

In early 2010, I wrote letters to Roberto Francini and Antonio Crepaldi about the condition. Antonio did know about the PLL situation in Scandinavia and did understand the condition among the Volpino population. I then sent out letters in early 2011 explaining the genetic mutation PLL to all the breeders and the ATAVI. We also started a campaign to inform everyone interested to check and ask about the disease. There is now enough information published on the internet about PLL that anyone doing a quick check on Volpino's should be able to learn and read about it.

In Sept 2011, Karen Brennan, Terralea Collins and I traveled to Italy to attend a meeting and dog exhibition in Sassuolo, Italy! Here we heard many views and worries about PLL and future plans, many

of which have taken hold. Some did not understand the nature of the disease and gave arguments that those Volpino that had Primary Lens Luxation were not real Volpino's . Some others tried to minimize the effects of the disease by stating that 'in their situation' they had not seen it. In some cases it came down to simple pettiness and that they would not listen to 'Americans' telling them about their Volpino's.

I do understand that before genetic testing and before the meetings in Sassuolo that the breeders may have been bewildered about what to do about the disease. A disease that doesn't manifest itself until later in life is hard to control. I can also understand that some breeders had a large investment in their breeding stock! At any rate, what happened before 2011 is in the past and should remain in the past! It is unrealistic to place blame and place accusations on those before that could not have known there was a problem with their dogs until several generations had passed. However, in the present situation I

would expect that anyone selling a Volpino would reveal the condition of illnesses that the Volpino may have. It is no longer ethical to state the breed has no major illness.

The PLL mutation has reached well over 35% of the Volpino population (according to testing of adults). I believe it has stopped spreading but now needs to be controlled and minimized. I suppose nothing can be done to stop small home breeders spreading the disease but there should be an understanding that breeding practices need to be changed. Genetic testing needs to be performed on any dog with untested parents or any breeding pair in which one may have been a carrier or affected.

At this time I believe that the major breeders are co-operating and trying to eliminate the disease. It is totally possible to wipe out this disease. We can improve the health of the Volpino and eliminate not only Primary Lens Luxation but also Progressive Retinal Atrophy (PRA) in which genetic testing is may also become available.

I would recommend the following.

Test all bloodlines of Volpino Italiano to find the disease 'Primary Lens Luxation' (PLL). Sterilize all dogs that are sold to families that carry the mutant gene.

Enter the eye certification program!

Write up a pamphlet with information about the Volpino Italiano authorized by the ATAVI and FCI about the Volpino Italiano and given to all persons purchasing a Volpino.

Make it mandatory that all Volpino's are sold with a pedigree (if it is not).

Rewrite the standards on size of the Volpino to insure the Volpino doesn't breed down too small. We do not want a 'tiny' Volpino and all the problems associated by it.

Do not award a LIR registration number to any 'rustic' Volpino unless it has been tested for PLL and PRA!

Rewrite the standards allowing for a black Volpino and loosen the standards on color.

The Volpino Italiano is truly a beautiful animal. It is perhaps one of the most loyal and loving breeds of all the Spitz dogs. In truth, it is a breed worth keeping and protecting. It is not, however, a dog for everyone. I would not generally recommend this vocal breed for those living in apartments or those that may move a lot. The Volpino needs stability, and perhaps another Volpino or another dog in the same household. The breeder should look at the family's entire household and should not be afraid of telling the purchaser he should not have a Volpino.

I have written up the following summaries as a small guide explaining the major problems possible in a Volpino Italiano. I hope it will be useful to those wishing to obtain a Volpino and for breeders raising Volpino.

Feel free to email us with your comments

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SPITZ DOGS AND THE VOLPINO ITALIANO

EVOLUTION OF THE EARLY SPITZ

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Edited by Laura Fox

June 2014

The Volpino Italiano is a member of the Spitz group of dogs and so shares the same characteristics as most members of the Spitz Group.

The origins of the Spitz dogs are lost in unrecorded history, but there are several assumptions that knowledgeable persons agree with. The Spitz group originated in the northern parts of Europe and Asia and may have been introduced to the rest of Europe with migrating humans. (Remains of Spitz-like dogs, along with human remains have been found in Northern European bogs dating back to 5000 BC.) While the Spitz is believed to be the oldest domesticated dog with direct ancestry to the Wolf, this is not totally certain. It may be that later humans from about 3000 BC or so, allowed their Spitz-like dogs to mate with wolves. At any rate, genetic testing shows the Spitz with the shortest direct line to the wolf. The Spitz dogs of Northern Europe also show the closest genetic similarity with the wolf.

This wolf's association with Humans probably began casually. Some wolves having less fear of humans remained close to the human tribes for easily acquired scraps of food. It might also be that food was given to individual wolves that were preferred by the human tribe and so this relation progressed. Puppies born close to humans lost fear in them. Over time the wolves evolved by natural selection into a semi-domesticated wolf simply by their association and proximity to human settlements.

It has been shown that even the simple attempt to breed dogs for a particular non-physical trait (i.e. tameness, viciousness etc.) will also change the dog physically. An experiment in Russia in breeding foxes for domestication showed that as the animals became more docile and friendlier then physical changes will also occurred in the animals. Lighter coloring and an upright tail are some of the physical changes brought about by reinforcing behavioral traits and are shown in the Spitz Group.

Humans later would have realized that this evolved wolf-like dog could benefit the settlement with its natural ability to herd animals and to warn the settlement of strangers or predators.

It would be this natural aggression towards non-members of its pact (in which the human tribe now was) and its social behavior that has benefited the dog breeds and their human companions since. It also would have become obvious to the human tribe that puppies born with sheep or other animals would have a natural tendency to herd and protect them. In order to strengthen these natural traits, dogs with a natural tendency to herd would have been bred with other dogs with the same ability to herd. Likewise, those dogs that exhibited a natural aggressive (wariness) trait to protect their pact would have been bred with other dogs that showed this trait. And so, breeding and the early formation of the separate dog breeds from its wolf heritage began. It was likely the wolf evolved into the Spitz in this fashion. The breeding of other dog breeds would have slowly evolved from the Spitz/wolf in this manner over thousands of years. It would not be until the 18th century though, that breeders would start to radically alter the Spitz/dogs into the many varieties of dog races shown today.

It should be noted that the color white is often found with individual Spitz races. It can be assumed that during the early days of the association between humans and the early Spitz dogs that the color white was preferred. Lighter dogs with an ability to protect and herd were bred with other lighter dogs and this continued over the generations of dogs to produce a white or markedly lighter dog than a predator naturally was. This would have had huge advantages for the human tribes because it made it easier to distinguish their dogs from predators when attempting to kill the predator.

Unfortunately, the color white may also have been one of dog's first inbred genetic defects.

THE SPITZ DOG

Most European registries and the America UKC list the Spitz breed dogs in Group 5, or the Primitive or Northern breed group. In this context, primitive means there are fewer changes in these dogs when compared to the Wolf than with other breeds. Other 'national registries' list the individual dog breeds in other groups such as the working or companion groups.

The Spitz group of dogs generally share the following characteristics

- A thick and harsh fur coat with a second soft downy-like undercoat.
- Triangular pointed ears set high on the head and perfectly erect.
- A tail that generally curves over their back but is held 'up' in some fashion.
- A rather 'square' and stocky body structure.

Each individual dog breed will also have its own standard for definition. In most cases, even a cross breed from another breed that exhibits most of the above characteristics can be assumed to be a 'Spitz' dog.

A Spitz is usually a very intelligent animal with his own strong will! Because of this they can be stubborn and sometimes hard to train. Socializing is usually very important for the animal's interaction with other humans and the immediate family. They will, in most cases bond tightly with the individuals of the immediate family and may be wary of other people and animals. They are very loyal but can be 'morbidly' bonded to a family or a particular individual. This can make interaction with other people entering the home or acceptance of a second owner difficult. This trait is strong in the Spitz breeds but it can vary and is dependent not only on the individual breed but also on the individual animal.

These dogs are also considered to be 'Working Dogs' and so can also be highly energetic. Exercise is often required for them to spend energy.

Because of their ancestry, many races of the Spitz breed can be very vocal, for play, warning, and perhaps just communications.

These dogs also can suffer from 'separation anxiety' and may become destructive when left alone.

Therefor a Spitz dog may not be suitable or acceptable for many families. The Volpino Italiano shares many of these temperaments.

THE VOLPINO ITALIANO

At some point in history, the migrating humans brought their Spitz-like dogs in the more southern areas of Europe, including the Italian peninsula. It is also possible that the Volpino Italiano was bred down from the larger Spitz-like dogs which were present from the Roman Era. At any rate, there is Archaeological evidence of Spitz dogs on the Italian Peninsula from before 1000 BC. The actual method of introduction is lost to history, but it is unlikely that the Spitz dogs crossed the Alps on their own.

Evidence of a small whitish Volpino-like Spitz dog in Italy starts about the 16th century. Vittorio Carpaccio in his 1502-5108 'Vision of St Augustine' painting depicts a Volpino-like dog with a short muzzle, erect ears and black nose (The painting's dog is usually referred to as a Pomeranian (see note

below). Michelangelo also kept with him a small Volpino-like Spitz dog (Also often referred to as a Pomeranian) as he painted the ceiling of the Sistine Chapel (1508 – 1512). It is about this time period when these dogs are being referred to as 'Volpino'. The name Volpino translates into English meaning 'little fox' or 'foxy'. The obvious reason being the resemblance to the fox because of the Volpino's Foxy head. However, the fox is not related to the Volpino (or any dog) because of their extensive genetic differences.

The Volpino may also be known by other names given to them from the local areas in which they were found. They may have been referred to with other names such 'Florentine Spitz', 'Italian Spitz' and 'Cane del Quirinale'.

The Volpino was a favorite pet of the Italian royal families and were often found in houses of the Italian Lords. Because of their vocal disposition, they were also often used by cart owning salesman to 'announce' the approach of strangers and summon the cart owner. In local farms, they were kept for centuries by farmers as watch dogs, again because of their sensitive hearing and piercing bark. The Volpino could sense the approach of a predator and with their barking awake the Mastiffs, which would rally and attack or chase the predator away. Indeed, they are still kept in isolated Italian farms often for the same reason.

Note - It should be noted that in the 18th, and 19th centuries The British, and later the established Kennel Club referred to all Spitz- like dogs as 'Pomeranians', including the white Italian Spitz dogs. However, we are not interested at this point with the white Italian Spitz dogs that were sent to England or the use of them in other breeds. Although these were definitely Italian Spitz dogs, they may or may not have been the same as the present day Volpino Italiano. Remember, the Volpino standard did not exist at that time. And while there is a similar, but different Italian Pomeranian, the focus here is on the Italian Volpino Spitz dogs found on the Italian peninsula and defined by the 1902 standard (and the recovered Nov 29 1989 standard).

The popularity of the Volpino in Italy increased during the last couple of centuries, but we cannot say how popular the animal was among the peasant population. But by the 19th century, it was a common

enough pet, if not among the town folk, at least among the mid- and upper classes and at rural farms and livestock operations.

The KKI (Italian Kennel Club now the ENCI) organized the first registration of the Volpino Italiano in 1901. In 1903 the K.K.I. established the first standard, written by Giuseppe Solaro, for the Volpino Italiano. After the Second World War, the popularity of the Volpino Italiano decreased dramatically. This loss in popularity was probably due to the vast number of other breeds now available. It may also have been due to the Volpino's link to the houses of royalty and government, which were now out of favor.

The Italian countryside and towns were now a much quieter place, (which may have been another reason for the Volpino's demise).

Between 1952 and 1962 only nine Volpino births were registered. After that no Volpino Italiano births were registered until 1972 when Enrico Franceschetti attended the dog exhibition in Monza (MI) with two Volpino Italiano dogs (Jojo and Jaja) and successfully obtained registration numbers (LIR) for them

In 1968 Enrico Franceschetti, along with Tonino Casadei, the president of the Kennel Club of Forli, began searching for Volpino-like dogs around the country. In 1984 the ENCI launched a recovery effort for the Volpino Italiano. Unfortunately, probably because of individual preferences, it seems only the white race of Volpino Italiano was selected. Nearly all the Volpino Italiano dogs registered to this day are due to the efforts of these two people.

The recovery of the Volpino Italiano continued successfully as Enrico Franceschetti established the Kennel 'Allevamento della Genzianella' and was helped greatly by Francesco Giuntini. They cooperated with Ezia Valentini and her kennel 'Allevamento della Volpe Candida' to re-establish the various Volpino Italiano Bloodlines.

In the original 1903 standard, the Volpino Italiano was defined as a small Spitz dog in three colors, white, sable and black. The 1989 standard, unfortunately defines the animal in only two colors, white and sable. Although many breeders claimed that the 'sable' race was extinct, red individuals are still being recovered in the countryside and its recovery appears to be well! The situation with the black Volpino may be dire. If no standard exists for it, then interest will be lost in the race. That would be unfortunate. The black Volpino will be lost unless a concerted effort is made to save it.

The similarity of the Volpino Italiano with the German Mittel and Klein Spitz is apparent and expected, considering they share the same ancestry (and probable cross breeding). Note that the colors of the German Spitz are also black, white, sable, and almost everything in-between. But most German Spitz are usually black, black/sable, or a lighter color. Black Volpino's may not have been desirable because of their resemblance to the Klein Spitz. Since there is no recognized black Volpino, it does make separating the two breeds easier.

Certifying a particular individual as a true member of the Volpino Italiano line involves entering the particular animal in a dog show (exhibition) and having the judges' rule that it is a true representative of the Volpino Italiano line. If the judges agree that it 'looks' like a typical Volpino as defined by the breed

standard, then the individual animal is awarded a registration number (LIR). There does not appear to be any oversight or appeal on the final judgment. Of course this process will have a few faults; e.g., competency of the judges, politics between the judges and the breeders; even the relationship between them all.

A controversy was generated in the late 80s when a breeder, Mr. Panciroli, knowingly entered 'Bianca' a white female that was not a Volpino but a separate Kleinspitz breed into an exhibition and obtained a Volpino Italiano registration number. That animal was mated with Italian champion Dario della Genzianella and produced a son Iuri (also an Italian Champion). They produced sons of William di San Tommaso and Willy di San Tommaso. This particular bloodline (in which Billj and Geo are members) produced many, many champions with other blood lines. It seems most of the champions produced shared Iuri's genes. This particular bloodline seems to have a strong stockier body and head and a heftier, blockier muzzle. Some of these are amazingly beautiful animals.

Are members of this bloodline 'True Volpino' ?? Of course they are. It would be silly and unbelievable that others that obtained registration numbers in the same manner were all 'pure Volpino's. By definition obtaining a registration number shows that the animal appears to be a typical Volpino. This is how all present day Volpino's were obtained. The only difference with Bianca is that her history was known. At any rate, so many generations have passed through the gene pool, the genes she passed on to her offspring have been diluted to the point they are dominated by other Volpino genes.

Personality

The Volpino, if nothing else, certainly has its own distinct personality. Depending on the individual animal

a) They are gregarious and totally devoted to their master(s). They absolutely do not care for being alone. A family member that remains at home or a second pet is advisable.

b) They are inquisitive, intelligent and lively. They should be socialized as a puppy.

c) They can be fearless and they make an excellent watchdog. Because of their keen hearing (and smell) they will often alert the household of visitors well before they approach the door. On the street they can be fearless and vocal at approaching animals (often from behind their Master's leg).

d) They are vocal on many levels. They tend to have different barks, growls, moans and groans for all different reasons. They will/could announce their presence when entering a room, warn of passing squirrels at the window, growl at the fire alarm or a newly acquired piece of furniture, and moan for attention or a belly rub etc. Vocal training is required or your home will no longer be the quiet place it may once have been.

e) They are friendly and very playful!!!! They will demand your attention! The Volpino Italiano is absolutely a wonderful dog!! While they may warn of strangers and other animals, they will also demand their attention. This may well be the one dog you will never forget!!

f) Some individuals, probably because of inbreeding, may be very timid.

Choosing a Volpino Puppy

Choosing a Volpino Italiano puppy can be a wonderful and rewarding experience for yourself and your family. There are several suggestions and advice I would like to give you!

- Research the breed you are interested in and know what you are buying. Puppies grow up!

- Consider rescue and adoption instead of buying a puppy. Although finding a smaller size puppy like a Volpino is sometimes difficult keep trying and keep searching.

- Never buy a Volpino puppy from a pet store. Indeed; if a pet supply store sells puppies or kittens go elsewhere.

- Do not take a puppy, Volpino or otherwise, away from their mother before 8 weeks of age. They need this time after weaning at 6 weeks then to socialize with mother and siblings and humans. Find another breeder if they suggest 5 or 6 weeks is OK!

- Try to find the actual breeder and see how the parents are housed and treated. Is the breeder humane? Do they care for their dogs properly? If it looks like a puppy mill, walk away. Puppy mills are a great source of suffering for dogs.

- Always demand a pedigree with your Volpino puppy. Obtain a warranty from the breeder to guard against at least the following conditions Primary Lens Luxation (PLL), Progressive Retinal Atrophy (PRA) and Luxating Patella.

- Do the parents look good??? Were they both healthy. What is the history of diseases or satisfaction of other purchases with their puppies? Try to find out!!

- See how the Volpino puppy behaves when they first meet you. Does he come straight to you and wants your attention, do they shy away and hide, or does he/she unleash a chorus of howls? Choose one that is fairly calm even though they will all be excited by the attention they get. Pick the one that either wants attention or simply looks and ignores you but is not frightened by you. Ensure that the puppy is healthy and active! Small runts may have underlying problems. If the parents are present gauge how they react to you. Though not always the case it is often that the puppies take after

their parents. If the parents are both timid or both hyper and overly aggressive then expect that trait in the puppy!

- If you intend to breed a male puppy then insure it is not Cryptorchidism, which is a genetically inherited condition in which one or both testicles have failed to descend into the scrotum. While it is usually OK to buy a puppy in this condition, the puppy should have its undescended testicle removed as soon as possible to avoid the possibility of developing testicular cancer. (In some Volpino's the testicles drop at 6 months old, some only drop one in the sack the other can be found along side the penis under the skin, Terralea Collins)

- Finally, and importantly, be sure you can afford to support your Volpino over its lifetime. Buying a puppy on impulse is ill advised and leads to a situation where the puppy is no longer wanted. Injuries and diseases of old age can be expensive.

Enjoy your Volpino Italiano

Please send any comments or suggestion to

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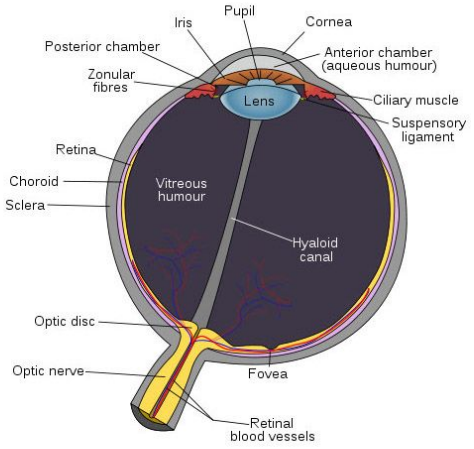
Eye Diseases and the Volpino Italiano

The Canine eye and Lens Luxation

There are many genetic diseases that have / can develop in any particular breed or species by natural aging, genetically inherited or caused by injury or trauma to the eye. The major eye disease that affect canine breeds can include genetically inherited blindness, forms of canine progressive retinal atrophy (often abbreviated to PRA or PRD), suddenly acquired retinal degeneration (abbreviated to SARDS), primarily lens luxation (Abbreviated to PLL), glaucoma and cataracts. There are others.

Because our main interest and the focus of this article is the Volpino Italiano, then, for the purpose of this discussion, we will centre our attention on the main diseases affecting the Volpino. We will focus our attention on progressive retinal atrophy (PRA) primary lens luxation (PLL), and cataracts. However, it does appear that the Volpino Italiano is troubled with its share of most of the known eye problems



The Canine Eye (doghealthdoc.com)

The main effect of the genetic version of PLL is a genetic weakness in the zonular cords and breakage or disintegration of the zonular cords at a genetically predetermined time. The disease usually (80%) manifests itself between 3 and 7 years old.

Primary lens luxation is, quite simply, the movement, or luxation of the lens. The lens structure of the eye is meant to focus light on the retina and that allows us (and most animals) to see an image clearly. The lens is held in place by zonal ligaments (cords) and is not allowed to move from its position between the iris and the vitreous humour in the eye. However, if some of the Zonal Ligaments (cords) disintegrate or break it will allow the lens to 'loosen' and change position (sub-luxation). This can occur in the early stages of PLL. At some point the lens may become completely detached (luxated) and is now free to 'be pushed' into the anterior chamber (this is anterior lens luxation). Primary lens luxation has then occurred! The dislocated lens is now free to move about in the anterior chamber, damaging the Iris and pupil opening and blocking the drainage of the aqueous fluid - raising the pressure in the eye!! This is Glaucoma. With this type of Glaucoma the pressure rises rapidly and blindness quickly follows. This can be agonizingly painful

As the animal ages the composition of the vitreous humour degrades from a clear, thick gelatinous type of substance into a more liquid form. If lens luxation occurs later in life it is possible for the lens to move or fall into the vitreous cavity (this is posterior lens luxation). This scenario may not produce the dramatic damage and pain that moving into the anterior chamber would. PLL in this case may not be detected for some time.

Glaucoma can cause lens luxation as a secondary infliction. However, if PLL is caused by glaucoma then that animal would have been blind far before the onset of PLL. One of the resulting injuries of glaucoma is that it stretches the sclera (globe of the eye) and thus stretches to Zonal ligaments to the point where they tear and break. This will not happen until very late in the disease.

It is also possible that PLL can be caused be trauma to the eye or indirectly by trauma to head. In many cases it will be obvious that the trauma has occurred.

PLL is a condition that can cause great pain to the animal and must be rectified as soon as possible. If the eye specialist notices a lens movement before excessive damage is caused he may be able to remove the lenses and allow the eyes to stay intact. Unfortunately if the lens becomes luxated and excessive damage is caused to the eye then the eyes will then need to be removed. This will likely be the condition that the veterinarian eye specialist finds himself in unless the animal's eyes have been checked regularly. It is often the situation that the owner of the animal will not realize the severity of this condition until the disease and damage to the eye has progressed to this late condition. Unfortunately the expense in helping the animal will be very high and euthanizing the animal will probably be the route chosen by most owners.

Although the attention caused by PLL has been high past few years it is a condition that has plagued the Volpino population since well before 1995 (An Italian veterinarians report was released in 1995 outlining the condition of PLL in Volpino Italiano dogs at that time). We can assume that most, perhaps all of the large breeders after that must have known of the condition from that point until the late 2010. Indeed, since the PLL mutation had spread so wide it would not have been possible for most breeders not to have heard of the condition or of complaints from their customers. However, it is also understandable that tracking the disease in the past would have been difficult. One way or the other, the claim that the Volpino Italiano was free of major health concerns was a very untrue statement!

THE GENETICS OF PLL

In late 2009 the mutated gene that causes PLL was discovered by the OFFA at the University of Missouri, USA (1a). Shortly thereafter it was also co-discovered by the AHT in Cambridge, England. It is here that the location of the mutation (described as G1473+1A ADAMTS17) was first identified and a DNA test developed to identify the mutation. Although this is mainly a disease which has plagued the Terrier Breeds (and those breeds that share the same ancestry) it somehow developed in the Volpino breed. Indeed, if you look through the list of PLL affected breeds it seems that the Volpino Italiano 'is out of place'. It is only recently that the Volpino Italiano is included in most lists of PLL affected (or prone) animals.

It should be noted though that this is not necessarily the only mutation that can cause PLL. Some breeds do suffer from PLL that are separate and caused by a separate mutation unknown at this time but not caused by the G1473+1A ADAMTS17 mutation. At this time that exact mutation has not been discovered.

The PLL mutation is essentially a simple recessive trait and follows the binary rules of Mendelian inheritance.

Normal – The individual has 2 normal (clear or non-mutated) genes inherited from his parents. This individual cannot develop the genetic version of PLL. (found at G1473+1A ADAMS17). This individual can only pass on normal or clear genes to his offspring.

Affected - This individual has 2 mutated (PLL defective) genes inherited from his parents. This individual will, if he lives long enough develop the genetic variation of PLL. This individual can only pass on a mutated (PLL defective) gene to his offspring.

Carrier – This individual inherited 1 each of the clear and mutated genes from his parents. He will, more than likely, not develop the genetic variation of PLL. He can pass on either a defective or clear gene to his offspring. There is a small chance, above the normal population average that the individual dog may develop the genetic form of Primary Lens Luxation.

BREEDING INHERITANCE

These would be the (average) results of inheritance for puppies of breeding parents.

N – (00) NORMAL **A** – (11) AFFECTED **C** - (01 or 10) CARRIER

Parents	Male	Female	Possible results (combinations)	
Genes	**N**	**N**	**N, N, N, N**	100% clear All offspring will be clear
	N	**C**	**N, C, N, C**	50% Normal, 50% Carrier
	N	**A**	**C, C, C, C**	100% carrier - All offspring will be Carriers
	C	**N**	**N,C, N,C**	50% Normal, 50% Carrier
	C	**C**	**N, C, C, A**	25% normal 50% Carrier 25% Affected
	C	**A**	**C, A, C, A**	50% Carriers, 50% Affected
	A	**N**	**C, C, C, C**	100% Carrier - All offspring will be Carriers
	A	**C**	**C, A, C, A**	50% Carriers, 50% Affected
	A	**A**	**A, A, A, A**	100% Affected All offspring will be affected

This average should theoretically be maintained over the population average, although the individual results may vary widely.

As noted above, in a true recessive trait both mutated genes must be inherited from individuals parents for the disease to develop. However, there exists a low risk that a carrier will develop PLL probably because of heterozygosity at a different location on ADAMTS17. Although the actual influence is unknown it may be that 2 to 20% of PLL Carriers may develop PLL. For the Volpino population it appears that the risk is closer to 2%. More test data gathered in future incidences of PLL will be needed to solidify the actual risk!

Because of the possibility of producing PLL Affected animals, PLL Affected animals should never be bred with PLL carriers, PLL Carriers should not be bred with another PLL Carrier. This combination will simply produce too many PLL Affected animals. Indeed; PLL Affected dogs should never be bred!! Although some would suggest that breeding the PLL affected dog with a PLL normal dog would result in PLL Carriers which would then be bred with PLL Normal/ Clear animals and so, should produce a few PLL Clear dogs. That assumed end result is not entirely certain. It is my suspicion and it does appear so that more PLL Carriers than PLL Clear/Normal will be produced. In my view though, it is unethical to breed an individual animal that could only produce Carriers. Breeding a carrier with a normal animal should be considered only if there are certain exceptional traits that the animal exhibits. This is a pairing that should produce more PLL Normal offspring than PLL Carriers and these can be used to continue the blood lines. This is important because of the past excessive inbreeding in the Volpino Italiano. It is more that possible that eliminating the Carrier blood lines may cause damage to the entire breed by introducing even more genetic problems.

The breeding of carriers will need to soon come to a stop at some point, and that point in time should be soon. This step should be considered only as a transitory one towards the goal of eliminating PLL. The breeding of Carriers cannot continue indefinitely!!

Unfortunately there is much more to learn about PLL. While it may be possible to breed PLL out of the Volpino Italiano breed it is also possible that a resurgence of the PLL mutation that is not linked to the present genetic testing may also establish itself in the future. This may be a very old, established canine disease that may manifest itself from different sources. Think of it this way, the Volpino Italiano and some other canine breeds may just be prone to PLL (and other eye diseases).

How did Primary Lens Luxation develop in the Volpino Italiano? The actual facts will never be known but here are some facts. PLL is relatively rare in the Spitz breeds in Europe. Remember that the Volpino breed was resurrected by a few individuals recovered from remote farms and bred to stock the present populations.

- It appears that only one particular bloodline has spread the mutated PLL virus (William di. s.Tommaso/Iury/Dario/Pallino)

- Looking at the early breeding records (see genealogy data) it appears that inbreeding was excessive in the early days of the Volpino recovery. It is possible the gene may have mutated here.

- It is possible that another breed or race that had the PLL mutated gene bred with a Volpino. After a few generations the differences in the introduced genes were lost to breeding, but the PLL mutated gene stayed with the breed

-The Volpino has been kept on Italian farms for centuries. It is here that the present day Volpino (as defined in the 1902 Standard) was essentially developed. On most farms breeding may have / would have been left to the Volpino's themselves. The puppies in some farms would then have been the result of hundreds of generations of inbreeding. It may be here that the gene causing PLL mutated into its present form.

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PROGRESSIVE RETINAL ATROPHY

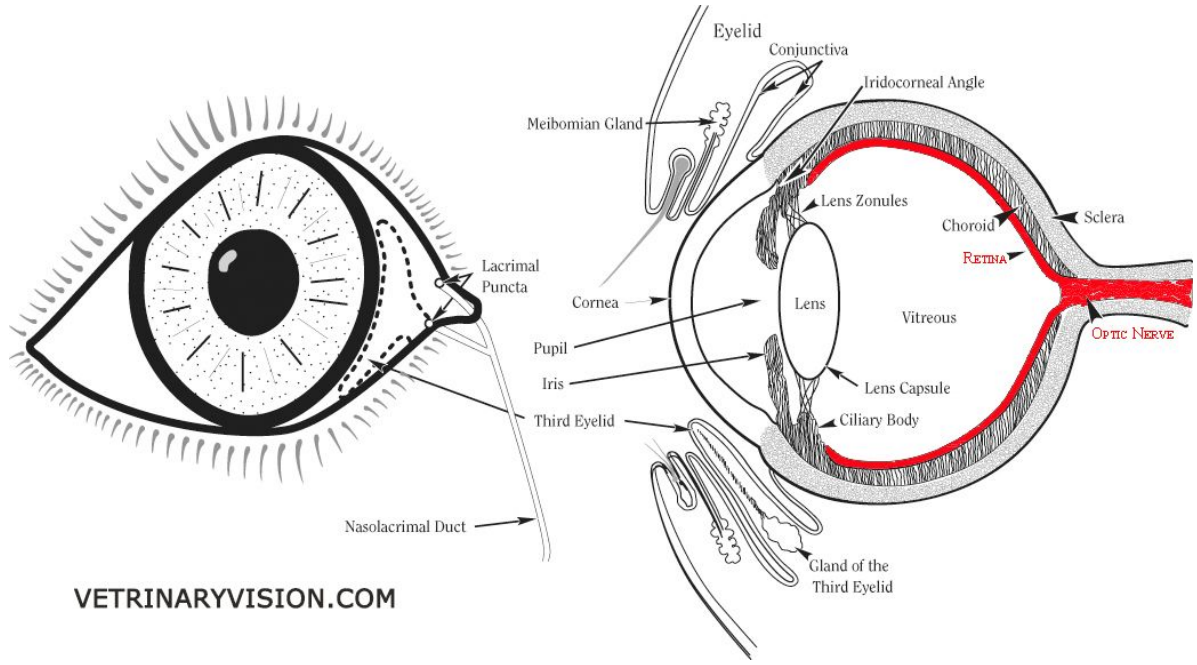
Michel (Mike) B. Rubini

Edited by Laura Fox

June 2014

Progressive Retinal Degeneration, better known as Progressive Retinal Atrophy (PRA) is a genetically autosomal recessive inherited group of diseases which causes irreversible degeneration of the Retina. General inheritance follows the same rules as PLL; each parent must have passed on the effected gene for the disease to develop in their offspring but inheritance rules can be different for some breeds.

There is also another form of PRA that does not appear to be inherited but seems random. This is called Sudden Acquired Retinal Degeneration (SARD)



The Retina, which is part of the central nervous system, contains specialized cells (photoreceptors) that convert light into electrical impulses. These electrical signals are then transmitted by the Optic nerve to the brain for interpretation into images. There are two types of photoreceptors found in the retina;

rods which interpret Black and white images and are more sensitive to low light and cones that interpret color. Dogs depend mostly on rods for vision, Color vision in dogs is poor compared to human vision.

An eye exam by veterinarian can detect changes in the eye that are characteristic of PRA. The level of function in the animal's retina can also be determined by an electroretinography, which measures the amount of electrical activity of the retina in response to light shining on it.

When PRA asserts itself retina degeneration generally begins with the rods. Thus the earliest problems exhibited by the animals will be a loss of night vision. Although the diseases progression can depend on the type of PRA, the breed and even the individual animal the disease generally leads to total blindness within a couple of years. As the disease progresses a 'shine' may be noticed in the animals eyes. This is due to the pupils being dilated and not responding the amount of light as it normally should. The animal will not experience any pain with PRA!

There are several genetically different variations of PRA all caused by a different mutation in a specific gene causing PRA. Although some mutations are specific to a breed, many breeds can share the same PRA gene mutation. Each individual case can be different and initial age of affliction will often determine the progression of the disease. Genetic testing should be performed to verify if the disease is genetic in nature.

New DNA tests for various forms of PRA for specific breeds and genes are constantly being developed.

GENERAL PRA – The bilateral general degradation or degeneration of the retina causing blindness.

LATE ONSET PRA - Onset of the disease usually begins about the ages of 3 to 5 years old but sometimes as late as 10 years. It always results in complete blindness usually within a couple of years.

EARLY ONSET PRA -. Onset of this type of PRA can start as early as 6 weeks old. With early onset PRA In this disease rods and cones do not develop normally in the puppies and will start to degenerate shortly after, usually leading to total blindness in 2 to 3 years.

CENTRAL PROGRESSIVE RETINAL ATROPHY– A separate disease also known as RETINAL PIGMENT EPITHELIAL DYSTROPHY and is a gradual degeneration of the coating of the Retina (pigmented Epithelium) so losing its ability to process light. However, peripheral vision is unaffected for a considerably long time. This is not a common disease.

There is no cure for Progressive Retinal Atrophy. Once the animal is afflicted it will go blind. Since it takes some time for the disease to progress the animal does have time to adjust to the vision loss. Stability in its habitat is essential at this point.

Cataracts can often form in the later stages of PRA because of chemicals released into the eye by the degenerating retina. While complicating the diagnosis it will not help the animal's vision to remove the

cataracts. But because Cataracts can cause other problems in the eye including possibly PLL and Glaucoma the situation should be monitored and perhaps treated.

It may be that 'anti-oxidants' and the consumption of Lutein may slow the progress of the disease if taken early enough. However, it is unknown if they can be of benefit at this time. In most cases the disease will have progressed to a more advanced stage where Vitamins and Lutein may be of little help.

Note – Note that all types of the known inherited PRA is a autosomal recessive gene except for these two exceptions. The Bull Mastiff PRA inheritance is an autosomal dominant inheritance. The Siberian Husky is a sex (X chromosome) linked recessive inheritance trait.

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CANINE CATARACTS

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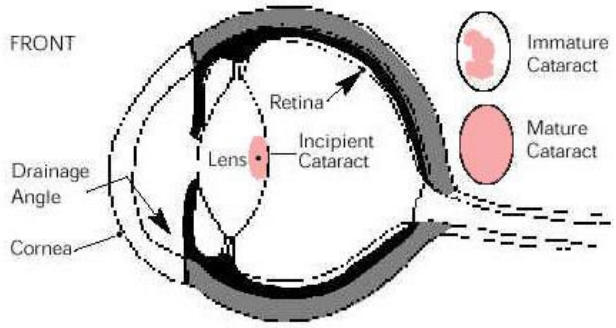
The most common cause of blindness in dog breeds, including the Volpino Italiano is an opaque clouding of the lens called cataracts. Clinically there are several variants and / or stages of cataract development.

- Incipient cataract - The opacity in the lens is very small the cataract may have no or minimal effect on the dogs vision.
- Immature cataract – The opacity of the lens will be larger and cause a blurring of the vision.
- Mature cataract - The opacity of the lens has developed to the point where all functional vision is lost.
- Hypermature cataract – this type of cataract starts to reduce in size due to a loss of water and nutrients

In most cases incipient and immature cataracts will develop into a mature cataract.

It should be noted that as any dog ages they develop a hardening of the lens that appears bluish-greyish. As the dog ages the blue color can intensify. This hardening of the lens, called nuclear sclerosis (also known as Presbyopia) is normal in ageing dogs. It will usually take an ophthalmologist to distinguish between nuclear sclerosis and cataracts or to determine if both are present.



canine cataracts (animaleyecare.net)

The lens is a soft, clear crystalline structure that allows light to pass through it and be focused on the retina. It can be thought of as being functionally equivalent to a camera lens. When the lens loses its transparency and becomes cloudy it then lessens the amount of light that can pass through it. Eventually most light is 'blocked' and the animal

becomes blind.

Cataracts are formed when the fibres of the lens breakdown from their usual arrangements. If the biomechanical exchange system that forces sodium water mixture through the eye is disrupted or breaks down it will then allow extra water to be pumped into the lens. This changes the normal composition in the lens (water 66% to protein 33%) and so increases the amount of water insoluble proteins. Cataract formation starts and the crystalline lens become less transparent.

There are many different types of cataracts and they affect all breeds of dogs. They can be inherited, caused by aging, injury, poisoning, radiation or electric shock. It can also be caused by toxicity in the eye or a simple lack of nutrition. Cataract formation can vary in ages of onset and the speed of development in the canine eye. However there are breeds that are more susceptible to cataract formation than others. Although cataracts are mechanically well understood a great deal of information is still not known about its onset, development and the genetic formation of the cataract.

Inherited cataracts - Most cataracts are inherited! In many breeds the cataract is often caused by a mutation of the HSF4 gene, which is autosomal recessive. In this case of HSF4 the opacity starts small in the back of the lens, growing until a mature cataract is formed. The growth of the cataract is variable though; some dogs go blind quickly while a few others may outlive the growth with fairly clear vision. The cataract may occur between the age of 9 and 15 months and is generally mature in 2 to 4 years. Genetic testing is available for a number of breeds to ascertain if the cataract is a genetic formation or not.

Diabetic cataracts -The second most likely cause of cataracts in dogs is diabetes mellitus. Seventy five percent of diabetic dogs will develop cataracts with a year of diagnosis. With diabetes the excess sugar in the lens cannot be consumed and is transformed into sorbitol. The sorbitol draws excessive water into the eye leading to the cataract formations. Sometimes diabetic cataracts can develop in days. This may be the first sign of diabetes in dogs and an assessment should be done quickly for diabetes. Unfortunately cataracts are virtually assured in any dog that is diabetic, regardless of insulin treatment.

Toxic Cataracts - Cataracts can also be the side products of toxicity or illness in the eye, such as progressive retinal atrophy (PRA), glaucoma and uveitis. In many cases the illness will be known in advance so the cataract may be expected. In PRA, the most common form of toxically formed cataracts the 'death' of the rods and cones in the retina release a toxicity as a by-product that is absorbed by the lens, causing the development of the cataract. Toxic cataracts can also form from ingested lead and inadvertent poisonings from various herbicides (especially Glyphosate exposure which is commonly found in 'Roundup'). It may also be caused by commonly available medications for flea, tick and heartworm prevention.

While cataracts are often not painful they can cause inflammation as the modified liquid in the lens leaks into the eye. In a hyper-mature cataract the lens capsule begins to shrivel because of autolysis of the lens fibers. It is also possible at this stage for the cataract/lens to become luxated which may cause damage and inflammation to the rest of the eye including secondary Glaucoma. At this stage the animal can feel discomfort and/or extreme pain.

The only effective treatment for Cataracts is lens replacement surgery. While supplements are advertised as an effective way to stop or reverse the cataract (some advertisements claim it can stop PRA) there is simply no evidence of it. Various antioxidants, vitamins and zinc are needed for a healthy eye and it is possible it may slow the growth of a cataract. If the cataract has not progressed to a later stage it will probably do no harm yet may simply be a waste of money.

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LUXATING PATELLA

August 2014

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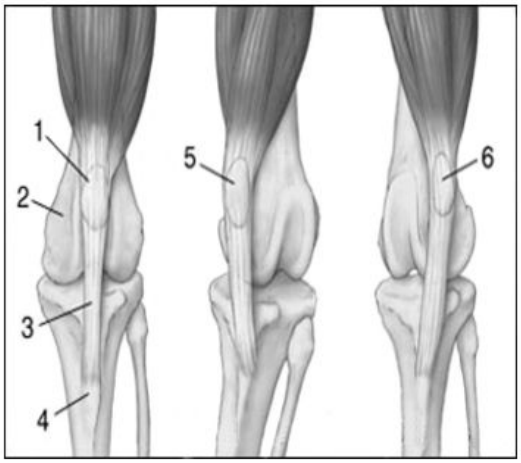
A luxating patella means a moving, or dislocated knee cap.

The patella, or knee cap, normally sits in a formed groove of the lower femur (thigh bone). A luxated patella occurs when the patella is dislocated from the groove of the femur. This is the most common knee joint injury for the canine breeds. It is more common in the miniature or 'toy' breeds (such as Pomeranians, Pekinese, Boston Terrier, Chihuahua etc.). While the condition still applies in a great majority to small breeds a troubling increase of luxated patellas is occurring in larger breeds.



1 - Patella
2 - Femur
3 - Patellar ligament
4 - Tibial Tuberosity
5 - Medial Luxation of Patella
6 - Lateral Luxation of Patella

The Canine Knee (from www.vetmedclinic.com)

The patella slides up and down the trochlear groove of the lower femur bone. The Trochlear groove is actually formed by two trochlear ridges (medial – toward the centre of the animal and lateral away from the animal) on either side of the groove. The patella is imbedded in the quadriceps muscle and is attached to it with the quadriceps tendon. On its lower side it is directly attached to the tibia with the patellar ligament. As the quadriceps muscle contracts it pulls the tibia forward and so the patella guides the movement of the quadriceps muscles during leg motion. The patella, with its tendons and the quadriceps muscles in the rear legs form the 'extensor mechanism'. When operating correctly the Patella increases the efficiency and stability of the canine stifle joint. Any failure of the extensor mechanism or malformation of the tibial and femur bone can eventually lead to a luxating patella.

There can be several reasons for a luxating patella but nearly all are congenital / genetic in nature and the disposition to the condition is likely inherited.

Genetic

A trochlear groove that is too shallow to contain the patella during leg motion.

There can be a weakness of the connective tissues or ligaments that causes instability of the movement of the patella.

The tibial tuberosity that the patellar ligaments attach to on the tibia is misaligned.

The rear skeletal anatomy is misaligned / malformed causing instability and stress in the stifle joint movement. The structure of the leg and hips in some animals are inherited as a breed trait but the resulting stress and bone angles can lead to luxating patella as well as other problems.

Disease – Joint diseases such as arthritis can damage the cartilage leading to the Patella luxation.

Trauma - A defect in bone angle or ligament damage can be caused by trauma to the knee and leg causing luxating patella. This is uncommon and rarely occurs.

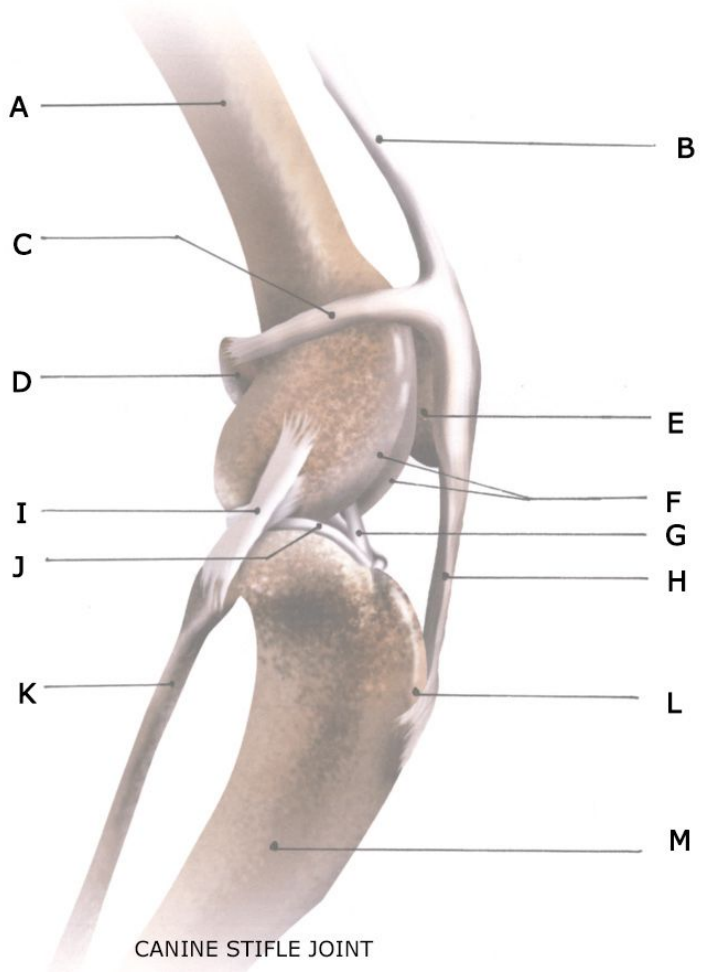
In many, if not most cases, a luxating patella will be a result of a combination of the above problems.

Many animals will develop signs of a luxating patella early in life. The first signs of a luxated patella may occur during normal running or walking. The animal may 'yelp' at the pain as the patella luxates out of position and hold his leg straight out. Once the patella is luxated the animal will not usually feel pain. After a short time the quadriceps muscles will relax and the patella may return to its normal positon.

The condition is easily diagnosed by a veterinarian while the animal is still a puppy. Unfortunately, the earlier the condition develops the greater the severity of the condition and the resulting disability - including deformation of the rear skeleton. In some cases the condition may not appear until later in life. Although this would not be strictly classed as 'congenital' the underlying causes were probably developed over the animal's lifespan from certain defects that were present from a younger age.

Logically (as a general case) the smaller the animal then the smaller the bone structure will be. In many cases the underlying tendency to a luxated patella is often a normal consequence of having an insufficient bone structure to properly develop a strong enough extensor mechanism that can contain and centre the patella while the puppy is growing. There may be insufficient bone to properly develop the trochlear groove and ridges and tendon placement and still allow for stable mechanical containment of the patella against the twisting forces of leg motion.

If the two trochlear ridges are insufficient in height then the groove that the patella slides in will be too shallow to guide the patella. The patella may then slide sideways out of its intended position in the trochlear groove. This is the type of patellar luxation that generally occurs (and is very common) in toy breeds. Female dogs have a 50% greater probability of developing Patellar luxation than do male dogs. Because of normal motion, the mechanics and construction of the rear hind skeleton and extensor mechanism the dislocation of the patella is generally MEDIAL, that is it will luxate toward the 'centre' of the animal. When (for whatever reason) luxation occurs while the animal is still a puppy and the bone



A - FEMUR BONE
B - QUADRICEPS TENDON
C - LATERAL PATELLAR LIGAMENT
D - FABELLA
E - PATELLA
F - TROCHLEAR RIDGES
G - CRANIAL CRUCIATE LIGAMENTS
H - PATELLAR LIGAMENT
I - LATERAL COLLATERAL LIGAMENT
J - MINISCUS
K - FIBULA BONE
L - TIBIAL TUBEROSITY
M - TIBIA BONE

Structure is still growing the patella rubs over the medial trochlear ridge and quickly wears it down. This causes the patella to luxate more frequently and further damages the trochlear ridge. Arthritis will then set in and cause further joint damage. As the animal grows he will try to compensate for this abnormality during normal walking and running and so the entire rear skeleton may develop abnormally. It may also be the case that improper hind skeletal formation, either as a genetic defect or the results of a breed trait will cause excessive stress on the trochlear ridges causing a luxating patella.

It should be noted that normal leg motion is required for the patella to properly 'set' in its required position as the puppy grows. During growth the normal movement of the patella increases the depth and strength of the trochlear groove and ridges allowing the muscles and tendons to properly develop around the patella. There is surgery available to deepen the trochlear groove (or replace it with and artificial one),

For the extensor mechanism to operate properly it must be properly positioned and centered in the stifle joint. Any weakness in the muscle or tendons associated with the patella can cause the patella to luxate during movement. A cause of patellar luxation is that the patellar tendon which connects the patella to the tibular tuberosity (or tibular crest) on the tibia is not placed properly. This causes a 'pull' towards the non-centered direction of the position of the tuberosity. This type of congenital anomaly can be repaired by repositioning the tibular tuberosity.

Weak or misplaced attachments of the patellar lateral tendons in the stifle joint area may also result in a luxated patella. Surgery in these cases to repair the tendons/muscle can be successful.

Larger dogs are less prone to luxated patella than are smaller dogs. Generally they will have larger, stronger leg bones with plenty of room for a deep trochlear groove to contain the patella. However, larger dogs can develop luxating patella's as a secondary condition caused by hip problems (i.e. Dysplasia). The Luxation in these cases can be lateral as well as medial.

A veterinarian can assess the dogs luxating patella and grade the severity of the condition during a routine examination

Grade 1 – the patella will luxate out of its normal position in the trochlear groove but returns to its normal position right away during examination. This would be a recurrent problem.

Grade 2 – the patella will luxate out of its normal position in the trochlear groove but will not always return to its normal position right away. This would be a recurrent problem.

Grade 3 – a luxated patella is normally luxated out of the groove, but can be manually placed back to its normal position. This would probably be a permanent situation.

Grad e 4 – the patella will not stay in the groove. When forcing the patella into its normal position in the groove the patella luxates out of the groove after pressure is released. This is a permanent condition.

Not all Volpinos (or small dogs) will develop a luxated patella but a seemingly growing percentage will. The reasons are varied as outlined above. In most cases it should be considered 'genetic' in nature and inherited even though the cause can be considered simply to be the size of the dog (which is, of course, hereditary). In many cases where normally angled and developed rear hind skeleton is present then a larger dog may dramatically reduce the instance of luxated patellas. A heavier and more substantial bone structure in a puppy may allow for a stronger, more normal development of the canine stifle joint and deeper groove with less instances of the luxated patella.

At any rate it is important NOT to breed a Volpino that have developed or has a family tendency towards a luxated patella. It should also be recorded how many of their offspring actually developed the anomaly. In many cases this will reveal a line that is troubled with the disease.

In many cases a luxated patella can be found during a veterinarian exam while the puppy is still growing. Therefor the breeder should provide some form of warranty against the luxated patella for the first year of the puppies' life!

Please feel free to send any correction, comments or suggestion to me

Aug 2014

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NORTH AMERICAN VOLPINO CLUB 2014

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Progressive Retinal Atrophy (PRA)

University of Prince Edward Island

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Note – Note all Types of the known inherited PRA is a autosomal recessive gene except these two exceptions. The Bull Mastiff PRA inheritance is an autosomal dominant inheritance. The Siberian Husky is a sex (X chromosome) linked recessive inheritance trait.

Living with Blind Dogs by Carol Lewis

Canine Cataracts

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