

# UNDERSTANDING WHITE

by François-R. Bernier,  
Bajnok Vizslas & GSPs

As with many of the self-coloured breeds, all current Vizsla breed standards penalize the appearance of white in the Vizsla coat. The importance attached to this issue is evidenced by the fact that many of these standards also include a “disqualification” for what are considered unacceptable white markings. This article is intended to provide some of the background that can assist breeders and fanciers in assessing the importance of this trait in our breed. Before dealing with the genetic aspect of this question, it may be of interest to provide a summary overview of the biochemical process by which colour is formed in the coat of the dog.

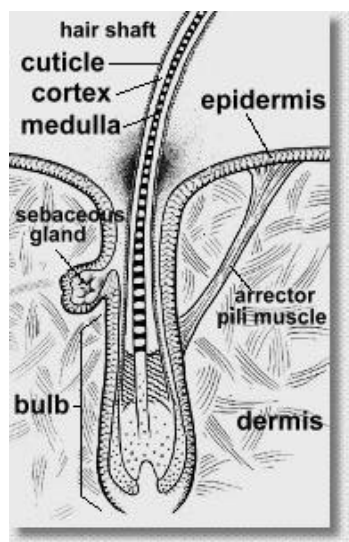
## PIGMENT AND PIGMENTATION PROCESS

The seemingly infinite variety of colour in dog coats is based in reality on two kinds of pigments: eumelanin (black pigment) and phaeomelanin (yellow pigment). Pigment is present in the hairs of the coat as pigment granules and factors such as granule size and granule shape will determine the colour of

the hairs and that of the coat. These pigment granules will be present in the medulla as well as the cortex of each hair (*Figure 1*). For example, both black and brown require the presence of eumelanin (black pigment). In black coats, the eumelanin granules are oval and intensely brown so that the hair appears black to us. By contrast, phaeomelanin granules will be smaller, round and yellowish. In a liver dog, the recessive *b* gene acts on the black pigment granules and lightens their colour so that the coat is changed from black to chocolate or liver to our eye. But the *b* recessive does not act on yellow pigment granules and areas such as the tan points on a “red” Doberman will be unaffected and be the same colour as they would on a black Doberman. The important point to keep in mind is that it is those areas of the coat that lack either black or yellow pigment that appear white to us. White, then, is a result of the *absence* of pigment in the individual hairs that make up the coat.

It may also be of interest to briefly describe the ‘mechanics’ of pigmentation or the process that leads to individual hair becoming pigmented. At the embryonic stage, undifferentiated cells that migrate from the neural crest to various parts of the dog become melanocytes or melanin producing cells (The neural crest of the embryo is located in areas which correspond to the top of the head and the spine in the adult dog). Pigmentation cells are distributed along established migration routes or pathways and eventually are found at the root of each hair. The distribution process of melanocytes over the body of the dog is regulated by various genes and influenced by non-genetic factors as well. Researchers and breeders have observed that: “The distribution of white markings follows a relatively orderly progression as the amount of white increases. The first appearance of white is on the chest, feet, muzzle and end of the tail.” (Robinson,1990). It will be noted that the areas that most often remain unpigmented (i.e. white) in self-coloured dogs are the extremities that are the most remote from the source of distribution of pigmentation cells. It might help to visualize this process to imagine someone slowly pouring a gallon of

*Fig. 1*



thick paint along the back of the dog and then watching it slowly cover the dog's body.

The pigmentation process is not always completed at the foetal stage but often continues after birth. In a Vizsla litter, for example, one puppy had a white marking an inch long and nearly one quarter of an inch wide in the middle of the skull. Months later, the white had completely gone and the whole of the previously white marking was now fully pigmented. While perhaps not always as dramatic an example as the previous one, every breeder has probably had a puppy with a white or roan marking on toes or elsewhere that disappeared or "filled in" as the puppy grew. These are two examples of the pigmentation process continuing past the foetal stage.

#### GENETIC ASPECTS

The appearance of white on any dog is primarily under genetic control and the particular genes that determine the extent and location of white markings form the S series. The genes of that series regulate the distribution of pigment in the coat and indirectly, the amount and placement of white, keeping in mind that white is not a colour but the absence of colour (i.e. black or yellow pigment).

The Spotting or S series includes the following alleles: the S gene (or Self gene) is responsible for the distribution of pigment over the whole dog. Next in the series, we find  $s^i$  (Irish spotting) followed by  $s^p$  (piebald spotting) and finally  $s^w$  (extreme white piebald). These are listed in order of dominance and each of the recessive genes in that series ( $s^i$ ,  $s^p$  and  $s^w$ ) is responsible for a progressively greater restriction of the distribution of colour pigment in the coat resulting in a progressively greater appearance of white or non-pigmented areas. It should be mentioned that considerable variation exists for each genotype due to the action of plus or minus modifiers. For example, Basenjis are reportedly homozygous for  $s^i$  and will exhibit a wide range in the expression of white as seen in **Figure 2**. All of the dogs in this illustration are  $s^i s^i$  but plus modifiers act to increase the

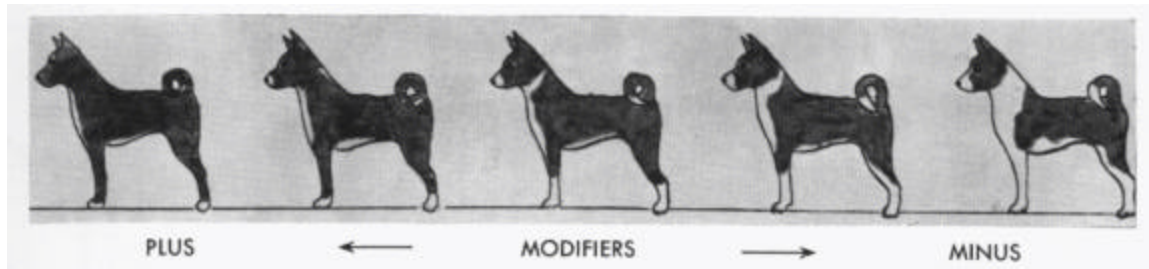
distribution of pigmentation (decreasing the amount of white) and minus modifiers act to reduce that distribution (increasing the amount of white).

Alleles of the S series are incompletely dominant to one another. The S allele for example will be dominant to  $s^i$  but not to  $s^p$  or  $s^w$  and dogs that are  $Ss^p$  or  $Ss^w$  will display an amount of white typical of Irish spotting ( $s^i s^i$ ). The Irish allele  $s^i$  is dominant to  $s^p$  but not to  $s^w$  and a heterozygote  $s^i s^w$  will exhibit an amount of white typical of piebald spotting ( $s^p s^p$ ). These facts are mentioned here for the sake of completeness, as they do not concern us given that Vizslas are part of a group of breeds known as self-coloured breeds which are of the SS genotype, meaning that they carry two copies of the S allele and therefore, the colour pigment in the coat is always distributed over the whole dog. But if this is the case, why do white markings appear on some of them?

The explanation lies in the action of plus or minus modifier genes. Plus modifiers will increase pigment distribution while minus modifiers may operate to limit the distribution of colour pigment even in a dog of the SS genotype with the result that white markings appear. The research of Little (1957) established that this is a fairly common occurrence in self-coloured breeds and that extreme minus modifiers will produce an effect similar to the appearance of Irish spotting with plus modifiers. In terms of phenotype, these two categories will overlap.

What this means in practical terms is that a Vizsla may be homozygous for the self gene (i.e. SS) and yet exhibit a considerable amount of white due to the action of minus modifier genes. Being aware of the overlap in the amount of white appearing on an SS dog with minus modifiers and on some of the low grades of Irish spotting might assist in avoiding hasty conclusions about the parentage of some Vizslas, often from field-bred strains, that exhibit a considerable amount of white. In those cases, casual observers are sometimes quick to conclude that a dog is of impure breeding and whispered accusations of crossbreeding to Pointers follow suit.

**Fig. 2**



Reproduced from *The Inheritance of Coat Color in Dogs* (1957)

Such breedings, accidental or deliberate, may indeed have taken place but we should always keep in mind that dogs with considerable amounts of white might also be produced from the breeding of two purebred Vizslas. If both Vizslas carry extreme minus modifiers, their mating may produce dogs that are self-coloured but that will exhibit as much white as some dogs that are low grade  $s^i s^i$  (low grade Irish spotting is represented in the two Basenji drawings on the left in *Figure 2*). Breeders whose primary selection criterion is field performance may not select against white or minus modifiers as rigorously as breeders whose primary focus is the show bench or may not select against it at all. If selection against the minus modifiers that decrease areas of pigmentation in self-coloured dogs is not a priority with a particular breeder, it can be expected that this breeder may produce more dogs with white markings and with larger white markings than a breeder who makes selection against white markings a priority. It would be my suggestion that not a few cases of rumoured crossbreeding simply involve a purebred Vizsla that exhibits low grade Irish spotting but that is in fact of the correct  $SS$  genotype with extreme minus modifiers.

#### WHITE AND ROAN

It is often the case that a Vizsla exhibits roan markings rather than white marking. A roan marking refers to an area in which there is a mixture of pigmented and unpigmented hairs as opposed to the white marking in which only unpigmented hair will be found.

A variant is the white mottling, which is really closer to ticking. There is no agreement amongst geneticists as to whether there exists a distinct roaning gene or whether roaning is simply a variant of ticking. In his *Inheritance of Coat Color in Dogs* (1957), Clarence Little mentions the possibility of a separate gene responsible for roaning ( $R$ ) but concludes that: "Because of the lack of definitive data and the clear evidence of genetic complexity in the case of roan, it is probably wise to adopt a conservative but open-minded attitude toward the acceptance of a definitely dominant roan gene at a single locus. Present information makes it seem more likely that ticking and roan grade into one another." Winge (1950) accepted the existence of a separate roan gene, as did Burns and Fraser (1966). Willis (1989) is in agreement with Little and also suggests that in the absence of further evidence, "it may be wisest to assume that ticking and roaning are just two versions of the same thing". Robinson (1990) also seems to take the view that ticking and roaning are simply variant expressions of the ticking gene.

It is probably best to assume that a Vizsla that exhibits roaning or mottling carries the dominant  $T$  gene. Ticking ( $T$ ) is dominant to non-ticking ( $t$ ) and a Vizsla with roan markings or ticking could be either  $TT$  or  $Tt$  in genotype. Of course, unless white markings are present on a dog, roaning and ticking cannot be expressed phenotypically and it is not possible to say whether a dog that is completely solid in colour is  $TT$ ,  $Tt$  or  $tt$ . In such a case, the colour ticking against a

background of the same colour as the ticking will not be apparent. Given that ticking is dominant to its absence, if two fully coloured Vizslas produce a Vizsla with a roan marking, it must be assumed that at least one of the parents is ticked.

#### NON-GENETIC FACTORS

When discussing white, it is important to keep in mind that the presence and extent of white markings are not entirely under genetic control. As early as 1957, referring to experiments done by Sewall Wright nearly four decades earlier, Clarence Little cautioned that: "an appreciable amount of variation in the extent of body-surface pigmentation is usually nongenetic in nature." Without discounting the primary role of genes, it seems reasonable to think that various factors other than genetic can influence the distribution of pigmentation cells or perhaps, even interrupt it at times. We know that some factors may delay the normal pigmentation process as happened with the puppy mentioned earlier and there will no doubt be cases where the process is not only delayed but interrupted altogether. These factors could be anything from environmental to nutritional.

#### SOME CONCLUSIONS

Although Vizsla fanciers have decided that their breed should not exhibit any white, this choice should not obscure the fact that white markings are a normal occurrence in self-coloured breeds. To paraphrase a remark of Willis (1989), white in Vizslas is selected against because it is aesthetically disliked rather than because it is associated with a particular biological defect. White markings occur primarily for genetic reasons but variability in their expression can also be caused by non-genetic factors. As with other self-coloured breeds, white is part and parcel of the genetic heritage of the Vizsla and something that will always be with us. Through selection, we can reduce the incidence of white markings and their extent when they occur, but we will never eliminate them entirely. The occurrence of white markings on Vizslas is not proof of impure breeding and even in those cases where the amount of white is quite in excess of that

usually encountered, one should not be too quick to reach the conclusion that the dog involved is not a purebred Vizsla.

That the amount of white that appears on a Vizsla may also be influenced by non-genetic factors should also serve as a further reason to exercise caution before reaching hasty conclusions about the parentage of some Vizslas. Applying the warning about not judging a book by its cover to the vexed issue of white in the Vizsla, one should be careful not to impugn a Vizsla's parentage based on the presence and extent of white markings without careful consideration of the genetic and non-genetic factors involved. The reality is that not every Vizsla that exhibits massive white markings is the result of "impure" breeding. In many cases, the simple explanation is that the Vizsla has pseudo-Irish markings due to extreme minus modifiers in its genetic make-up and in a few cases, the size of the resulting white markings could be further increased by non-genetic factors.

Some of the things to remember are that:

1. Vizslas are a self-coloured breed of the SS genotype.
2. Minus modifiers may restrict the distribution of pigment in the coat and areas that remain unpigmented will appear white.
3. The presence and extent of white markings (i.e. unpigmented areas) is primarily determined by genetic factors but can likely be affected by non-genetic factors as well.
4. There is overlap between the SS with extreme minus modifiers phenotype and low grade Irish spotting phenotype.
5. Significant white markings on a self-coloured dog are not necessarily an indication of impure breeding.
6. The only reason we select against white markings is that breed fanciers have chosen to fault such markings in the breed standard.

## GLOSSARY

**Allele** Alternative forms of a given gene, such as B and b. Also used to refer to the versions of a gene present on each chromosome of a chromosome pair. One allele is inherited from the mother, the other from the father.

**Genotype** The genetic makeup of a given physical trait; also, the total genetic makeup of an individual organism.

**Heterozygous** Having inherited a different allele from each parent, at a given locus on a chromosome; contrasted with homozygous, in which the same allele for a given trait is inherited from both parents

**Homozygous** Having inherited the same allele for a particular trait from both parents.

**Locus** (plural: **loci**) The site on a chromosome where a specific gene is located.

**Phenotype** The visible, physical expression of a genetic trait

## SOURCES AND FURTHER READING

Winge, O., *Inheritance in Dogs* (1950)

Burns, M. and Fraser, M.N., *Genetics of the Dog. The basis of successful breeding* (1966)

Little, C., *The Inheritance of Coat Color in Dogs* (1957)

Robinson, R., *Genetics for Dog Breeders* (1990)

Whitney, L.F., *How to Breed Dogs* (1971)

Willis, M.B., *Genetics of the Dog* (1989)

*UC Davis School of Veterinary Medicine Book of Dogs* (1995) (for the Glossary)

*Questions or comments on this article are welcome and may be sent to*

*vizslas@bajnok.com*

*© Copyright reserved*