# White markings

## – the cause and inheritance with special reference to the Boxer

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**Current fashion dictates that the British Boxer must have near-maximum permissible amounts of white coat markings, strategically-placed, in order to be successful in the showring. Such "flashy" specimens are considered more eye-catching than their "plainer" brethren and, in some circles, it is even held that the indefinable character, quality, is only found in animals with relatively large amounts of white. Aside from the fact that these views, rational or otherwise, render the plain Boxer rather useless for show purposes, the requirement for white markings poses for the breeder a number of unwanted problems. Most people I'm sure recognise that the presence of white markings is associated in some way with the occurrence of all-white or check Boxers. There is also the problem of the mis-marked or over-marked animal and I think it is also generally suspected that there is a connection between white hair in the coat and the lack of pigmentation of the third eyelid, a fault which is now extremely common in the breed. In order to understand how all these characters are inter-related and to what extent they are controllable, it is necessary to know something of the developmental processes concerned to coat and skin pigmentation, how genetic changes alter them and how these genetic changes are inherited. What follows, therefore, are the facts as they are known in the dog, heavily subsidised by current hypotheses developed from studies upon experimental animals, such as the laboratory mouse.**

*The key to the whole process is that pigment, or colour is not produced by the skin or hair itself; it is produced by specialised pigment-producing cells which migrate into the skin and hair follicles during embryonic development. Their migratory pathway in the skin of the mouse is well-documented. Originating in the neural crest of the embryo, single cells initially colonize a limited number of specific sites in the skin along either side of the dorsal areas of the body (including head, neck and tail). These cells then multiply rapidly and begin migrating laterally down each side of the animal. In the mouse, the migration continues until the time of birth, at which point any areas not colonized by the pigment cells never become pigmented, i.e., they remain white. As expected those areas would lie furthest away from the source of the migrating cells, e.g., the belly, legs, feet, tailtip. In the dog, as everyone who has bred Boxers will recognise, the migration continues for many days, if not weeks, after birth, the white areas gradually yielding to the advance of the pigment-producing cells. At some point, however, the progress stops and the final pattern of coloured and white areas is permanently established.*

Another factor that predisposes to the appearance of white areas has been recognised in the mouse. At some point near the end of the migratory phase, the host tissue temporarily becomes hostile to the invading pigment cells such that many fail to produce pigment and die. In regions where the concentration of pigment cells is still low, i.e., the extremities of the migratory pathways, the loss may be virtually complete, but these areas later again become liable to invasion and it is probably this that one observes in the dog. The survival of some isolated cells may be the cause of the small pigmented patches that are often seen to grow and enlarge in the white areas, primarily on the belly. It might be noted that the spread of pigment cells in the skin is invariably more extensive than in the hair. Either the movement into the hair follicles stops earlier or higher concentrations of pigment cells are needed before the follicles are invaded.

The migration of pigment cells is not limited to the outer surfaces of the body. They can and do migrate through internal tissues. Here, their function is now known but their shortage or absence from the inner ear, which is normally pigmented, might be the cause of the deafness often found in white Boxers, Bull terriers, etc. There is little doubt that the third eyelids also become pigmented as a result of invasion by the migratory pigment cells. However, since the route of this migration will bear no relation to that in the surrounding skin, pigmentation in the two tissues

need not be closely correlated. The blue eye sometimes seen in the white Boxer similarly results from the shortage or absence of pigment cells. The region affected in this case is the iris.

● **Genetic changes**

The discussion so far has centered around the "normal" animal and the main conclusion is that all animals may be considered as being predisposed to incomplete pigmentation. Many genetic changes or mutations which enhance this risk are known in the mouse. These operate in one of at least two ways. One causes some defect in the pigment cells such that they multiply less frequently or migrate less. Certainly, there are fewer of them. The other modifies the host tissue in specific regions of the body such that the pigment cells are unable either to enter the hair follicles or produce pigment. Since the end effect, decreased pigmentation, is very similar the mode of action of any new mutation cannot readily be distinguished. Certainly it is not known which of the two mechanisms is responsible for the white markings in the dog. One might only note that the reduction in the pigmented areas aids the recognition of the migratory pathways. Thus, with a limited reduction it can be seen that the pigment cells migrate down the legs primarily on the outer sides. Movement around the legs to the inner areas is accomplished more slowly and is liable to be incomplete. With still less pigment migration, evidence of the pathways down each side of the body can often be seen, e.g., note the shapes of the coloured areas in English Pointers or Spaniels, and, with minimal pigmented areas, some indication of the location of the original sites from which migration begins may be seen. For example, it is fairly obvious that there is a pair of pigment sites on the head in the vicinity of the ears. Other sites in the body can similarly be detected in some animals.

● **The Inheritance**

We can now turn to the inheritance of white markings. Fortunately, this is much better understood in dogs than the developmental processes. Basically it is quite simple but the imprecise manner by which pigmentation is brought about raises many complications.

The presence of white markings is described as *spotting* and the spotting gene has been given the symbol **s**. Four distinguishable forms, or alleles, of this gene have been recognised, these causing different amounts

and distributions of white spotting. These are:

**S**: this is the "normal" allele and specifies full colour, i.e., *the absence of white*. Full pigmentation implies that a full complement of pigment cells are multiplying, migrating and producing pigment. The **S** allele is found in all fully pigmented breeds such as the Alsatian, Dobermann, etc.

**$s^i$**; this allele causes the pattern of white markings known as *Irish spotting*. White may be found on the face, around the neck, forechest, belly, legs and feet. Either the pigment cells tend not to reach these areas, or are killed or do not produce pigment in these regions. Basenjis and Boston terriers owe their white spotting patterns to $s^i$, although it seems that other *s* alleles may be present in some animals.

**$s^p$**; the $s^p$ allele produces what is known as *piebald spotting*. Generally, $s^p$ causes much more white than $s^i$. The distribution of the pigmented areas is less regular and may be confined to the dorsal areas, the back, the rib-cage, around the ears, etc. The various Spaniel breeds, English Pointers, etc., carry $s^p$ but, again, other *s* alleles may be found in some animals.

**$s^w$**; this allele gives and effect which is known as *extreme-white piebald*. As the name suggests, $s^w$ animals are liable to show only some limited degree of pigmentation and may be completely white. The $s^w$ allele is found in most white breeds e.g., the Sealyham and the White Bull Terrier.

## ● Difficult to follow

The inheritance of the *s* alleles is often difficult to follow. There are three reasons for this.

**1)** The dominance of one allele over another is very often incomplete. Generally, the order of dominance is *S*, $s^i$, $s^p$, $s^w$ but both alleles in any combination are liable to produce an effect. For example, the progeny of a Dobermann (*SS*) x White Bull Terrier ($s^w s^w$) cross would all be $S\ s^w$ and might be expected to be fully pigmented. In fact, they might well show significant amounts of white. The foreface, chest and feet could be marked and, without knowledge of the ancestry, such animals might be thought to be $s^i s^i$, i.e., they would show what is known as pseudo-*Irish spotting*. Similarly, $s^i s^w$ animals might show *Irish*, *Piebald* or even *extreme-white piebald* spotting. Perhaps this type of effect is not too surprising when one considers how pigmentation is accomplished and how it is liable to be faulty. One dose of

the dominant gene (as in *S* $s^w$ dogs) is just not as good as two (as in *S S* dogs) and the various factors which contribute to the appearance of spotting enhance the chance that this will be detected.

**2)** The expression of the *s* alleles is highly variable. Thus, the effect of $s^w$ may range from all-white to *piebald*, that of $s^p$ from near all-white through to the *Irish* pattern, that of $s^i$ from something near *piebald* to almost full colour, and white markings can even be found in animals carrying two *S* alleles. This variability most probably results from the interaction with the other secondary factors which play a part in the appearance of spotting, e.g., the cell death phenomenon which occurs when the host tissue temporarily becomes hostile to the invading pigment cells.

**3)** The amount of white spotting attributable to any allele or combination of alleles is due partly to non-genetic causes but is also partly under genetic control and the genetic component could well operate by way of the above-mentioned secondary factors. The extent of white in the coat can thus be increased or decreased by selection without substituting one *s* allele for another. In breeds where white markings represent a fault, not only will they carry two *S* alleles, but the genetic background will have been selected to aid full pigmentation. In Basenjis and Boston terriers there would appear to have been selection for near-maximum effects obtainable with $s^i$. Selection may also operate to modify the extent of white markings in dogs carrying two different *s* alleles, e.g., *S* $s^w$, and this brings us to the Boxer.

● **Three kinds of spotting**

To conform with present-day fashion it would be convenient if Boxers carried only the $s^i$ allele. Most could then have the Boston terrier level of white spotting which most closely approximates that apparently required for the showring. Although it has in fact been suggested that some Boxers carry $s^i$, there is no satisfactory evidence for this. Rather the breeding records indicate that the white spotting is attributable to the presence of $s^w$ Within the breed there are animals of three kinds; **SS S $s^w$** and **$s^w$ $s^w$**. The $s^w$ $s^w$ animals are the unwanted whites or checks, the *SS* animals are the plain ones and the *S* $s^w$ combination gives the desired flashily marked specimen. A first thought might be that this cannot be correct — very few of our plain Boxers are anywhere near totally devoid of white and the flashily marked dog has

levels of spotting which take it into the upper levels of expression of the *Irish spotting* pattern. The answer seems to be that there has been considerable selection for high levels of white markings and here a glance back at "recent" Boxer history in Britain might be of use.

● **Strong selection**

Those familiar with the early post-war Boxer will remember that flashily marked animals were then a rarity. Most dogs were either completely or almost completely devoid of white or only had a small white blaze, some white on the chest and perhaps a few white toes (see photograph p.46, Boxerama No. 3). White puppies were still produced and so the basic situation was the same (see also Frau Stockman's discussion, Boxerama No. 2 pp. 7 and 8). There then followed the importation of more flashily marked dogs from Holland and the United States and the fad for prettily marked dogs really started. Since then there has been strong selection for the favoured white markings. In the main, only well-marked dogs have been shown successfully in top-class competition and have thus formed the principal breeding nucleus, the source of stud dogs etc. Thus, with a little help from specific imports, the amount of white expressed in *S sw* Boxers has increased tremendously. Likewise, the white spotting in the plain animals has increased to almost "acceptable" levels.

● **Problems created?**

We can now return to the opening remarks of this article and evaluate the problems created for the Boxer breeder by the need for white markings.

**1)** The undesirable plain Boxer and the unwanted white: As already discussed, these are genetically *SS* and *sw sw*, respectively, and they are likely to appear in all matings of the desired *S sw* dogs. One should note that crosses of *S sw* x *S sw* will on average produce 25% plain and 25% white puppies. Half the litter must therefore be regarded as useless for show purposes in Britain and this purely because of a single gene. Breeding the plain *SS* animal to the flashily-marked *S sw* does not really help; whites are not produced, of course, but still only half the progeny are likely to be suitably marked (*S sw*), the rest would be plain (*SS*). A 50% loss would also be expected if we were to breed whites to flashily marked dogs. The only cross that would avoid this wastage is that of the plain (*SS*) x white (*sw sw*). Then all the pups could be expected to be suitably marked. This procedure means keeping dogs for breeding that will not be shown, however. It is indeed unfortunate that Boxers do not carry the *s* allele for then not only would most have the desired markings but they would for the most part breed true. No whites and few plains would occur.

**2)** The mis-marked or over-marked animal: These represent the logical outcome of the strong selection for heavily-marked dogs. The amounts of white shown in *S sw* dogs is now liable to exceed the maximum laid down by the standard and mis-marking becomes more prevalent. This constitutes a further loss of show prospects.

**3)** The unpigmented third eyelid: Although not necessarily associated with the amount or distribution of white markings in any given dog, this fault is liable to be correlated with the level of white spotting in the breeding. Thus, the unpigmented third eyelid is again an undesirable product of the selection for high levels of white markings.

*The conclusions from all this are quite obvious. So long as we require white markings we must accept the occurrence of plain and white Boxers. So long as we seek maximum amounts of white, and breed for this we must accept the increasing risk of producing the*

*over-marked dog and unpigmented third eyelids. It follows that we should ask ourselves if there is a real need for white markings. Is this just a fad or could there be any association with "quality" as sometimes claimed?*

● **Only a fad**

While recognising that it is always dangerous to ignore the views of those with long experience in dogdom, I would submit that the most the spotting gene can do is to add contrast to the background colour and somewhat modify it. As we have seen, the white areas do not have pigment cells, but it is likely that the spotting gene has some related effect in the pigmented areas also. This may convert the drab fawn into the sparkling fawn, the deer-red into that lustrous red and the dull brindle into that sleek, glossly brindle with well-defined stripes. My conclusion is that the requirement for white markings represents only a demand for pretty, pretty showdogs. It bears no relationship to the standard of the breed. It is only a fad and one that seems to be found in its most extreme form in Britain.

● **Reducing the selection**

The unfortunate aspect of this is that this fad ensures that many potentially great, but plain, showdogs find their way to pet homes, where they are unlikely to be bred from, or are sold abroad to a country which does not hold to our fetish. Together with the white and over-marked animals, the effect is to reduce our opportunity for the selection of high quality animals and this is surely an unwanted handicap. All this is especially frustrating when it is recognised that many of our "plain" Boxers of today have quite appreciable amounts of white on them. If we chose to breed from them, we could have quite attractively marked animals which did not produce whites, and unpigmented third eyelids would soon be a forgotten fault.