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Lethal Dominant White in Horses

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ALTHOUGH there is an American Albino Horse Club, Castle[2] told us some years ago that "no albino mutation has been found among horses" and the present writers can find no report of one in the 15 years that have elapsed since Castle's statement. Many so-called albinos (also known as "blancos", or cremellos) are genetically colored horses that are homozygous for the incompletely dominant dilution gene, *D*, which makes them almost white or pale cream, with pinkish skin and blue eyes.

From the studies of Tuff[17], Salisbury[12], and Castle and King[3], it is now clear that in the heterozygous state *D* converts horses that would otherwise be chestnut to the yellowish color of the breed known in the United States as Palomino. Other expressions of this dilution gene in various genotypes (and lack of its expression in some) are conveniently summarized by Singleton and Bond[15], who use for it the symbol c^{cr}.

A genetically different white is that seen in older horses that were fully colored at birth but subsequently acquired a progressively increasing number of white hairs and, after several years, became practically all white. Castle[2] aptly refers to this kind of white as "progressive silvering".

This paper deals with neither of these kinds of white, but with a dominant white, fully evident at birth, associated with blue or colored eyes (brown, hazel, etc.) and pink skin.

Earlier Studies

The occurrence of dominant white in horses was apparently first recognized by Sturtevant[16], who assigned the symbol *W* to the causative gene. Although Wriedt[17] referred to Sturtevant's report in his genetic analysis of records of the Frederiksborg white horses, he considered the latter to be recessive whites, with homozygotes white, white with gray spots, or gray white ("weissgraue"). Heterozygotes were believed to vary all the way from dilute gray to full color. In the light of more recent evidence, these conclusions now seem to have been erroneous, but we must give Wriedt full credit for his other conclusion, i.e., that in the Frederiksborg stud some lethal gene was responsible for the poor

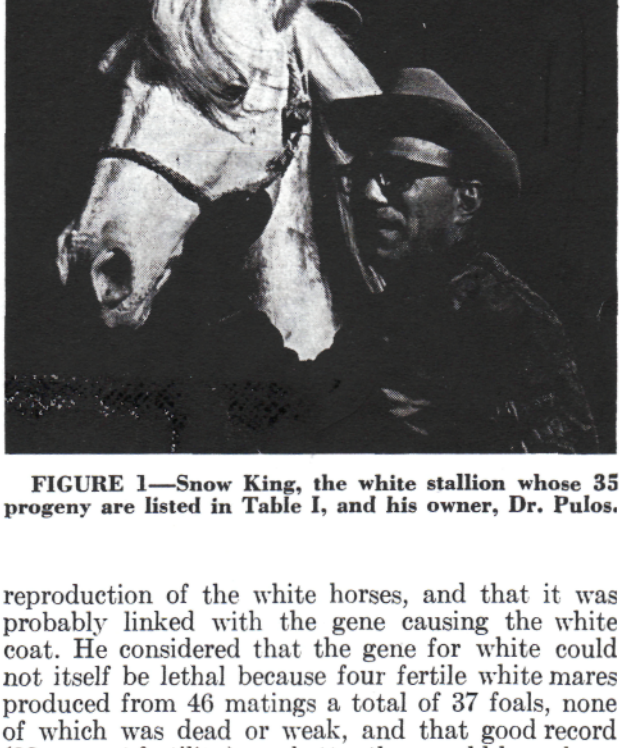

FIGURE 1—Snow King, the white stallion whose 35 progeny are listed in Table I, and his owner, Dr. Pulos.

reproduction of the white horses, and that it was probably linked with the gene causing the white coat. He considered that the gene for white could not itself be lethal because four fertile white mares produced from 46 matings a total of 37 foals, none of which was dead or weak, and that good record (80 percent fertility) was better than could have been expected if the gene for white color were lethal.

Subsequently von Lehmann-Mathildenhöh[6] reported evidence of a dominant white in the Bellschwitz and Ruschof studs, and, from reexamination of Wriedt's data, concluded that dominant white had also been present in the Frederiksborg horses. He did not consider the possibility that it might be associated with any lethal action, but his data indicate that the white horses adequately tested in the studs that he investigated must have been heterozygous.

Similarly, Salisbury[12] considered that the white coat is caused by a single dominant gene and that it is epistatic to all other colors. He made no reference to effects of the gene in homozygotes, but cited the report of one breeder that white horses mated *inter se* produced offspring among which about 75 percent were white.

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In his extensive review of color inheritance in horses, Odriozola[9] added no new data on dominant white, but pointed out that if Wriedt's interpretation of the Frederiksborg data were correct, it should be easy to establish a pure breed of whites, which had not been done. He suggested that different forms of *W* arranged linearly in the chromosome might be responsible for the differing degrees of white (e.g., *W″* for piebald) and that the expression of white is also influenced by modifying genes.

In a more recent review of this subject, Berge[1] lists dominant white horses as heterozygotes, and follows Castle[2] in suggesting that homozygosity for *W* is lethal. If so, then dominant whites mated *inter se* should yield a ratio of 2 white:1 colored, but, as Castle wrote 15 years ago, "this expectation has as yet not been put to an experimental or statistical test". This report provides such evidence.

The Horses

The data to be presented come from a stud started by one of us (W.L.P.) in 1954 with a single white colt and a white pony. The colt produced her first foal in 1957. Other white horses were bought or bred in subsequent years and by 1967 there were 16 white mares (including some immature ones) and one stallion. In the 12 years, 1956 to 1967, four different stallions were used (only one per year) and, as a result, the mares now present are not highly inbred.

The original white pony died without issue, and, from 1955 to 1965, all the ponies, which then numbered eight, were colored or piebald. White stallions were used since 1962; colored mares were gradually eliminated, and in 1968 there remained only four pony mares, all white.

Colors

Coat color, color of eye and sex were recorded at birth for all foals; none changed thereafter. White colts were recorded as "albino", and among 27 others (including foals of the ponies) there were 5 black (or black and white), 12 brown (or brown and white), 3 buckskin, 3 appaloosa, 2 palomino, 1 sorrel, and 1 gray. These limited numbers can contribute nothing to our knowledge of genetic bases for these colors, especially since phenotypes of one or both parents were always obscured by dominant white. Accordingly, to facilitate the study of white coat, all horses utilized in these analyses will be considered simply as either white or colored.

All white horses had pink skin, white manes and tails, and, with the exceptions noted below, white hooves.

Among 15 white mares examined in this stud in June, 1968, three showed small black spots, most of them less than half an inch in diameter. One of the three had only two small spots in the eyelids. Another showed extensive black spotting in the skin on one side of her hind-quarters. She was from a white stallion × black-and-white Shetland mare. Her white daughter also had a few black spots, but far fewer than in the dam. From a distance such small black spots appear bluish. Both the mare with black spotting on her rump and her daughter also showed black streaks in the hooves.

Among the white horses, color of eyes was described as pale blue, dark blue, brown or hazel. One mare had one eye blue, the other brown. Four foals had been described as having at birth eyes that were blue and pink. These probably had the iris blue, with blood vessels of the retina causing the lens to appear pink. It was noted that color of the eyes seems to vary somewhat according to the light. Some white horses show pink when the angle and intensity of light are just right for that, especially when out of direct sunlight.

The white horses were as healthy and hardy as the colored ones, except that the former developed chapped muzzles in the summer, and the colored horses did not.

Genetic Analyses

Coats and eyes

It seemed desirable first of all to determine whether or not blue-eyed white horses differed from brown-eyed whites with respect to transmission of white coat. If they did not, the inevitably small numbers of progeny produced from matings of any one stallion to any one mare could be combined in larger groups to provide numbers adequate for genetic interpretation.

Fortunately, an answer to this question was provided by the progeny of Snow King, a brown-eyed, white stallion used as stud sire in the four years 1964 to 1967. During that period he was mated with eight white mares, four of which had blue eyes and the other four brown. Previously he had sired a foal by one of these mares in 1958.

The record of Snow King's 35 progeny (Table I) shows that the ratio of white to colored foals from

Table I. Results of mating the white stallion, Snow King, to white mares

	Coat color in progeny			
	White		Colored	
White mares	♂♂	♀♀	♂♂	♀♀
A. Blue-eyed				
Snow White	2	—	1	2
Snow Ball	2	3	—	—
Snow Flake	—	2	2	1
Snow Countess	—	3	—	1
Totals	4	8	3	4
	12		7	
B. Brown-eyed				
Snow Scene	1	2	—	1
Snow Crystal	3	—	2	—
Snow Princess	1	1	—	1
Snow Drift	—	2	2	—
Totals	5	5	4	2
	10		6	

blue-eyed mares (12:7) did not differ from that among foals from brown-eyed mares (10:6). It is clear, therefore, that in studying the segregation of *W*, eye color of the dam is immaterial, hence all the white mares can be grouped together in one class.

Autosomal inheritance of W

All of the mares listed in Table I produced at least one colored foal except Snow Ball, but she had had a colored son in 1962 by another white stallion (Snow Rebel). Evidently Snow King and all eight mares were heterozygous for dominant white. If *W* were sex-linked, there should have been no colored daughters, but six such daughters were produced, hence the gene *W* is shown to be autosomal.

Heterozygotes

Each of the five white stallions used in the stud sired one or more colored foals. Similarly, all of the eight white mares that were adequately tested produced at least one colored foal. The fact that these 13 white horses were all proven to be heterozygotes agrees with previous reports that white horses with colored eyes did not breed true to type, but always produced some colored progeny. This, in turn, suggests that the genoytpe *WW* is not viable.

Ratios

If all white horses of this type are heterozygous, and if homozygosity for *W* is lethal before birth, matings of parents white × white should yield in the progeny a ratio of 2 white:1 colored. A corollary is that mating white × colored should produce about 1 white:1 colored.

Table II. Progeny of five white stallions

Stallion	Mares: Number	Mares: Color	Progeny: White ♂♂	Progeny: White ♀♀	Progeny: Colored ♂♂	Progeny: Colored ♀♀
Snow King	8	White	9	13	7	6
Snow Man	4	White	1	3	—	—
Snow Rebel	3	White	1	1	1	—
Snow Storm	1	White	—	—	1	—
		Totals	11	17	9	6
			28		15	
		Expected (2:1):	*28.7*		*14.3*	
Snow King	1*	Colored	—	—	1	—
Snow Man	2*	Colored	—	1	1	—
Snow Storm	1*	Colored	2	—	—	—
Snow Warrior	7*	Colored	4	4	6	4
		Totals	6	5	8	4
			11		12	
		Expected (1:1):	*11.5*		*11.5*	

* All ponies

Both of these expectations were realized in progeny of the five white stallions used in the period covered by our records (Table II).

Fits of observed to expected ratios as good as those shown in Table II are not often found. The ratio of 11 ♂♂:17 ♀♀ in white foals from parents both white suggests a deficiency of white males, but the deviation from the 1:1 ratio expected is not significant ($P = 0.2 - 0.3$).

Time of Lethal Action

Although the data in Table II, coupled with the fact that all 13 white horses tested were found to be heterozygotes, confirm previous indications that the *WW* genotype is not viable, it is not yet clear at what stage it is lethal.

Among six white foals (from parents both white) that died soon after birth, one had been unable to stand and nurse; death of another was attributed to exposure, one was strangled and another killed by the mare. The possibility that any of these might have been homozygotes is refuted by the fact that similar conditions caused death of several foals from the colored pony mares. Some of those foals were white, and some colored, but none could have been *WW*.

It seems probable therefore, that homozygosity for *W* is lethal at some stage during gestation, rather than at birth. As aborted foetuses were not found although a constant watch was maintained for them, it is possible that the homozygotes die early in gestation and are resorbed. The first lethal gene to be recognized as such, that carried by the heterozygous yellow mouse, is lethal to homozygotes at the early stage of development when normal embryos begin implantation[11], and other lethal genes are known to exert their effects at various stages of embryonic growth.

Evidence on this point with respect to the gene *W* in the horse will have to come from controlled matings in which returns to service can be recorded. For the Pulos stud, careful records were kept each year of all mares that were put with the stallion, and of which did or did not produce a foal. Over the 12-year period, 1956 to 1967, there were 56 such "exposures" of white × white, and the number of these that failed to produce a foal was 13, or 23.2 percent.

It would be easy to conclude that those failures correspond to the 25 percent mortality expected from early death of the *WW* zygotes, but there are two good reasons why we should not do so. For one thing, there are no controls to indicate what proportion of such failures is to be expected from other causes among mares not carrying *W*, but running in paddocks, as these were, with the stallion. For another, it seems possible that under such conditions a mare might resorb a *WW* foetus, be remated, and eventually produce a viable foal, either *Ww* or *ww*.

Flexed Forelegs

Among the 66 foals (Table II), there were two born with forelegs so badly crippled that the foals

FIGURE 2—Seven white mares or colts. The dark spots seen on two of the horses came from the muddy environment—not from the genes.

could not stand to nurse, and, therefore, had to be destroyed within two or three days. Both were sired by Snow Man, among whose six progeny the ratio of normal to crippled foals was thus 4:2.

The condition in these two crippled foals was apparently identical with that described and illustrated by Prawochenski[10]. The two lower joints of the forelegs were rigidly flexed with the hooves bent under and smaller than normal. The hind legs were normal, as were joints of the forelegs other than the two lower ones. Among eight such cases reported by Prawochenski among 26 foals sired by the Anglo-Arabian stallion, Menzin, there were at least two (possibly four, as Prawochenski's pedigree chart suggests) in which only one foreleg was thus crippled. In our two crippled foals both forelegs were affected. One was a male, the other a female.

The fact that both of these had a common sire suggests that there must be some genetic basis for the condition. One of their dams, Snow Flake, was a half-sister of Snow Man, but the other (Snow Drift) was entirely unrelated to that sire. Similarly the mares that produced Prawochenski's eight cripples were not closely related, if at all, to Menzin.

Speculation about the possible genetic basis for this lethal defect is unwarranted at this stage. It is to be hoped that this second report of the defect will induce veterinarians, breed associations, owners of horses, and others to be on the watch for its recurrence and that adequate data for genetic interpretations may thus be accumulated.

Discussion

Attention has recently been drawn to several cases in mammals in which genetic dilution or elimination of pigment reduces viability, or fertility, or both[5]. Searle[13] lists 45 cases in 16 mammalian species in which some kind of pathological pleiotropism is associated with coat color, but they include some for which conclusive evidence had yet to be adduced when that list was compiled. One of these is dominant white in the horse.

The remarkable variation in side effects associated with dominant genes diluting color indicates that there must be either several different biochemical processes by which such genes can prevent formation of melanin, or differences among species in the timing of these processes in relation to the stage of embryonic development or postnatal growth. The gene that converts black Karakul sheep to grays apparently has no other effect on heterozygotes, but induces in homozygotes abnormalities of the digestive tract that are usually lethal at four to nine months after birth. In contrast, a gene (S^H) that eliminates much of the color in the mink, is lethal to homozygotes (apparently to most of them before implantation) but induces in heterozygotes that would otherwise be fully colored a type of dilution called Heggedal[7]. Among female Heggedals, about 18 percent are sterile because of abnormalities of the reproductive tract[8].

As in the horse, dominant white is lethal in the canary, apparently at an early stage[4], but in the fowl, dominant white is standard color for the highly fertile, most prolific and currently most popular breed—the White Leghorn. Clearly, generalizations about dominant white are to be avoided.

It is not surprising that white horses have been in demand for centuries, or that those bred at studs maintained for many years should be recognized as special breeds. Among these, perhaps the best known are the Lippizans from Lipizza, near Trieste, and the Kladrubs from a stud near Prague. According to Slagsvold[14], both studs date from the sixteenth century.

While the evidence reviewed earlier indicates that dominant white was present in the Frederiksborg, Bellschwitz and Ruschof studs, that apparently does not apply to the Lippizans. Slagsvold[14] shows a picture of a white mare of that breed with a fully colored foal. According to Berge[1] the Lippizans carry *G*, the gene for gray ("progressive silvering"). His color photograph shows a white Lippizan with dark hooves and a dark muzzle. In contrast the dominant whites of the Pulos stud have white hooves and white muzzles.

The evidence that dominant white is lethal in the horse adds one more case to the list of breeds or varieties of domestic animals in which the breeder has (unwittingly) set up as the distinguishing feature of his breed, or preferred type, a phenotype that is induced by a gene lethal to homozygotes. Like white horses, the creeper fowl, Dexter cattle, platinum fox, and blufrost mink are all heterozygotes and can neither "breed true" nor equal the normal fertility of their species. With gray Karakul sheep, fertility of heterozygotes is normal, but few homozygotes survive to reproduce.

Summary

From data accumulated during several years in a stud of white horses, it is concluded that the autosomal gene *W* inducing dominant white is lethal to homozygotes, and that all horses showing dominant white are heterozygotes. The dominant white horses studied have pink skin, white coats, white manes and tails, and white hooves, but some of them occasionally show small spots of black pigment in the skin or in the hooves.

From matings of four white stallions with white mares, the ratio in the progeny was 28 white:15 colored, a close fit to the 2:1 expected if *WW* be lethal. White stallions × colored mares yielded 11 white to 12 colored. Each of 13 white horses adequately tested produced one or more colored foals.

It is considered probable that the *WW* genotype is lethal early in development.

The ratio of white to colored foals in progeny of blue-eyed, white mares mated with a brown-eyed, white stallion did not differ from that in progeny of brown-eyed white mares mated to the same stallion.

One stallion sired two foals showing flexion of the two lower joints of the forelegs, a lethal condition already on record.

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