

A possible dominant white gene in Jersey cattle

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Abstract – A white heifer (“Snow”) was born in 1991 from coloured registered Jersey parents. She produced six calves sired by coloured Jersey bulls: three white bull calves, two white heifer calves, and one coloured bull calf. One of the white bull calves was mated with 40 Hereford × Friesian yearling heifers (white face, predominantly black body with some white patches). The 38 resulting calves included 16 white and 22 coloured calves. Twelve of the 16 white calves were heifers and four were bulls. Red or black spotting was recorded on some white calves. The results are consistent with an autosomal dominant mutant causing the white phenotype. The mutation appears to have arisen spontaneously in Snow, then passing to her white progeny and white grand-progeny. The white individuals varied from entirely white in a few cases, to most having some residual small areas of red or black pigmentation in patterns not typical of other reported white spotting patterns of cattle.

coat colour / cattle / genetics

1. INTRODUCTION

White coat colour can arise from two basically different mechanisms [8]. One of these causes dilution of pigment, in which melanocytes are present but inactive or ineffective in producing pigmented hair. An example of a white phenotype arising from dilution is albinism, where tyrosinase is specifically affected. A second general mechanism for producing white hair is white spotting, where the white phenotype results from an absence of melanocytes in affected white areas. When extensive, white spotting can result in a white or nearly white phenotype. Dilution and white spotting mechanisms have both been documented in cattle.

One mechanism leading to white coat colour in cattle is the *Roan* locus in the Shorthorn and Belgian Blue breeds [1,7,9]. Heterozygotes at this locus have mixtures of coloured and white hairs, and homozygotes are white. The

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Roan locus has recently been shown to be identical to the *Mast Cell Growth Factor* locus. Other white spotting patterns resulting in nearly white cattle include the *white park* allele [5].

Two dominant white mutations have been reported previously in cattle [2, 4]. In neither case was the mode of inheritance clearly demonstrated. Detlefsen reported a curious case in which a normally coloured Holstein bull, mated to normally coloured Holstein cows, produced a white bull and a white heifer calf out of about 20 calves born (suggesting recessive inheritance), but about 20 subsequent progeny from the same bull mated to the same cows were all said to be white. The white spotting that is present in the Holstein breed could have confounded these results.

Leipold and Huston [4] reported a case of albinism in Herefords (both sexes), thought to be caused by a dominant allele with incomplete expression of associated pigmented hair spots and "glass-eyed", or blue, irides. The first white calf was a heifer (purchased *in utero*), which was subsequently used in matings planned to increase the numbers of white animals. The herd of 90 cattle finally contained about 60 white cattle. All the white cattle traced back to the one foundation animal. The only formal testing was carried out by mating a white bull, and subsequently recording 2 calves as white and 2 as normally coloured. This is consistent with a dominant gene but is scant evidence. Leipold and Huston [4] discuss 10 other references in the literature on albinism in cattle, all said to describe recessive inheritance.

The present study reports observations on a white coat-colour mutant which appeared *de novo* in a pedigree Jersey herd near Te Awamutu, North Island, New Zealand, in 1991. The foundation white animal was a heifer calf named "Snow", and the facts were first brought to our attention by the herd-owner (Mr. Jack Willis) in 1993, when Snow produced a white bull calf. This paper reports on the possible mode of inheritance of the white mutant allele, based on the coat colours of her 6 calves and on 38 progeny of a white son of Snow.

2. MATERIALS AND METHODS

Complete calving records were obtained from the herd owner for the foundation cow, "Snow", born in 1991. No previous white-coated Jersey calves had been born in this herd of about 120 cows since the herd owner began dairy farming in the late 1940s. Unfortunately he died in 1998, but his family kindly donated Snow's 1997-born white bull calf to AgResearch for experimental studies on the segregation of the mutant allele. The bull was mated as a yearling in 1998 to 40 Hereford × Friesian yearling heifers (white face, predominantly black body with some white patches). A total of 38 singleton calves (F1) were born in 1999, 37 live and one dead calf (which was black-coated). Chi-square tests were used to evaluate the coat-colour segregation data for Snow's calves and her 38 grand-progeny.

3. RESULTS

Snow's phenotype was white, but she had residual black-pigmented areas on the left side of her head below and including a small amount around her eye,

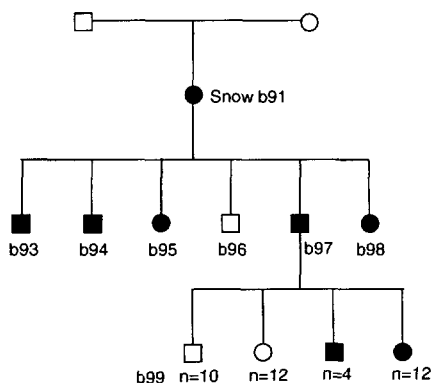


Figure 1. Pedigree showing mutant white calves (■ or ●) in three generations.

and one small area on the lateral neck. Snow produced six calves following mating to coloured Jersey bulls. Of these six calves, five were white and one (a bull) was coloured. Figure 1 shows the detailed pedigree. The five white calves included three males and two females. The 1997-born white bull sired 16 white calves; he also sired 22 red or black coloured calves, carrying typical Hereford or Holstein white markings (Chi-square for 16:22 = 0.95, 1 d.f., $p > 0.3$). The white calves included 12 heifers and four bulls. The total record of calves was 21 white and 23 coloured (Chi-square = 0.09, 1 d.f., $p > 0.7$).

The F1 calves varied from entirely white, to white with varying degrees of pigmented hair spots. Figure 2 shows Snow and 2 of her white calves, including the 1997-born son used to sire 38 experimental calves. The progeny of Snow's son included two entirely white calves (both heifers). Eight other calves had patches of red or black colour on the face, neck, ears, or head, or combinations of these areas. These patches were small, not symmetrical and consisted of a mixture of coloured and white hair fibres. Six of these calves had minimal areas of pigmentation (less than approximately 5%), and two had larger areas of pigmentation. The remaining six white calves had minor red or black speckling over the ribs and hips, including two with minimal areas (less than approximately 5%), and the remaining four with larger areas covered. Thirteen of the 16 white calves born in 1999 had white eyelashes (some with and some without pigment surrounding the ocular region), and the remaining three had some white and some coloured eyelashes. These 3 were all in the spotted class with less than 5% coloured area on the face/ neck/ ears/ head. Figure 3 shows four of the 16 white progeny of Snow's son, displaying a range of pigmented hair.

No glass-eyed phenotype was observed.

At an average of 173 days of age, the sex-adjusted difference in mean live weight of 11 1999-born calves which were weighed differed by only 0.3 kg between coloured ($n = 5$) and white calves ($n = 6$). All other calves were suckled on mothers that went to an embryo-transplant programme as recipients, where calf weights were not recorded. At the time of writing (August 2000), there are 10 surviving white heifers (out of 12 born in 1999). Nine have already



(a)



(b)



(c)

Figure 2. Original cow, Snow (a), with two of her calves born in 1993 (b) and 1997 (c).

reached puberty (displaying at least one behavioural oestrus each), with a mean age of 297 days (range 246 to 342 days). The current age of the remaining heifer is 339 days.

4. DISCUSSION

The segregation of white coat colour in this family of Jersey cattle is consistent with the action of a dominant autosomal gene. Because the white phenotype appeared in Snow, and not in her parents, then it must have been

a mutation *de novo*, produced in the formation of her zygote. She passed on the mutant allele to five of her six calves. One of these five was the bull which was subsequently progeny-tested, also giving a 1:1 ratio in the coat colour data from his 38 progeny.

It is important to eliminate any previously described patterns of white spotting as candidates for causing the patterns in this family of cattle. Several candidate alleles are documented in cattle, but none produces phenotypes typical of this family [3,5].

The most common type of white spotting in the Jersey breed is conditioned by the *spotting* allele at the *Spotted* locus. This allele generally causes white patterns of varying extent on marked cattle [5]. The extent of white in the Jersey breed, caused by this genetic mechanism, is generally modest and includes lower legs, tail tip, blaze on face, and varying amounts of body white. This pattern in the Holstein breed is generally more variable, and the whitest individuals still generally retain pigment over the ocular regions, as well as frequently having coloured ears. In this family of Jerseys, pigment was absent from the ear-tips and around the eyes in 3 of 10 surviving 1999-born heifers inspected after weaning, making the *spotting* allele an unlikely candidate for this phenomenon. Other genetic effects on white spotting in cattle are described [6].

The *Roan* locus (now documented as the *Mast Cell Growth Factor* locus) is another candidate [1,9]. Heterozygotes at this locus have coats that have intermixed white hairs and coloured hairs. In no animal did a pattern segregate that was similar to either roan or spotting. The relative pallor of the obligate heterozygotes produced in this study suggests that it is a new mutant, although the mode of inheritance of this new mutant may well be similar to *Roan*.

A few other patterns are generally more heavily pigmented than any of the animals in this study [3,5]. These patterns include belted, Hereford white head, Simmental white head, and lateral depigmentation. Two patterns that can cause nearly white phenotypes include the *colour-sided* allele as well as the *white park* allele.

The *Roan* genotype is also purported by some authors to result in segmental aplasia of the paramesonephric duct system in homozygous animals (so called "white heifer disease") [7]. However, from the live weights of F1s in this trial at 173 days, and the preliminary evidence on age at puberty in white heifers, it seems unlikely that white heifer disease is a feature of the present phenotype. Full confirmation of this point will require pregnancy records at a later age. The conclusion that homozygous animals at the *Roan* locus have reproductive abnormalities must also be tempered by the finding that not all such homozygotes are so afflicted, and roan and non-roan cattle of similar breeding are likewise afflicted.

Due to phenotypic differences between this and other documented spotting patterns in cattle we conclude that this is probably a new pattern. Its mode of inheritance is consistent with a dominant gene, although some degree of incomplete dominance cannot be completely ruled out by the data available.

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