

Occurrence of Hypotrichosis in Polled Hereford Cattle

T. A. Olson, Ph.D.¹

D. D. Hargrove, Ph.D.¹

H. W. Leipold, Dr. med. vet., Ph.D.²

¹Animal Science Department

University of Florida

Gainesville, Florida 32611

²Department of Pathology

Kansas State University

Manhattan, Kansas 66506

Introduction

The complete or partial absence of hair (hypotrichosis) has been described in most domestic animals and wild-life.^{1 3 5 6 9 18 20 21 24 27 29 31 32} The development of bovine hair was discussed recently.²² Coat color inheritance and inheritance of spotting patterns in cattle also have recently been reviewed.²³

Congenital hypotrichosis in cattle is of concern because of exposure to inclement weather and possibly entry of bacteria or fungi, which can produce skin infection.^{3 14} Reported here is the occurrence of hypotrichosis in Polled Hereford cattle.

Materials and Methods

The occurrence of hypotrichosis in a herd of Polled Hereford cattle on a ranch in Florida was studied. A genealogy was made. Five cattle suspected to be affected with hypotrichosis underwent biopsy examination. The skin sections were fixed in technicon, sectioned at 6 microns, and stained with hematoxylin and eosin (H&E). Skin sections were stained with H&E, periodic acid-Schiff and diastase periodic acid-Schiff, Altmann's acid fuchsin-methyl green, Perl's Prussian blue, toluidine blue, and Masson's trichrome.

Results

In the spring of 1984, three calves, two females and one male, born in a herd of Polled Hereford cattle were observed to have an abnormal type of coat. The hair coat was unusually short and curly in appearance and the color was often lightened or "frosty", particularly on the flanks and ventral portions of the body.

The switch was also much diminished. As the calves developed, the hair pigmentation became closer to normal but the curly coat remained on all or parts of the body. This

curly coat was particularly pronounced on the head and neck of all affected animals. The curly characteristics of the hair coat of the male were also pronounced on the head and underline up to the point of slaughter at about 14 months of age. The switches of affected animals remained sparse as they developed. (figure 1).

The 1985 calf crop from the same herd was closely evaluated and two additional calves, one male and one female, phenotypically appeared to express the same trait. The heifer calf expressed the condition to a greater extent in that her hair coat was extremely light colored and curly as well as being extremely short on her underline and feet at birth, which gave the appearance of hairlessness, although this was not the case. This calf is shown at about 2 months of age in figure 2. By 7 months of age, this heifer calf was much closer to normal in coloration.

Upon close observation of the adults in the herd, one 5-year old cow whose rear quarters are pictured in figure 1, which had been purchased soon after having been closely clipped, was also observed to express the condition. This animal has short, curly hair on the head, neck and legs as well as a short, sparse switch (see figure 1).

The degree of visible expression of the condition varied from animal to animal. Some animals are not easily recognized as being abnormal in hair coat even by breeders of Polled Hereford cattle. Affected animals are most easily determined during the winter months when the longer hair makes the curly condition more readily apparent (see figure 3). The most dependable location for visual detection is on the forehead area where the small tight curls are quite easily observed as abnormal to the experienced observer (figures 3 and 4). Upon observing this condition on the head of an animal, other areas of hair very short curly hair may also be noticed and perhaps small areas of hair loss can be observed on the poll, brisket, and knees. The hair on the feet of affected animals is nearly always so short that they have a pinkish appearance.

Skin samples from the ears of the five affected calves born in 1984 and 1985, and a mature cow, along with another calf

Contribution no. 86-40-J from the Kansas Agricultural Experiment Station.

FIGURE 1. Purebred Polled Hereford with hypotrichosis. Notice hair changes affecting lateral aspects of the body and switch of tail.



FIGURE 2. Purebred Polled Hereford calf affected with hypotrichosis. Notice more severe affliction as compared to figure 1.



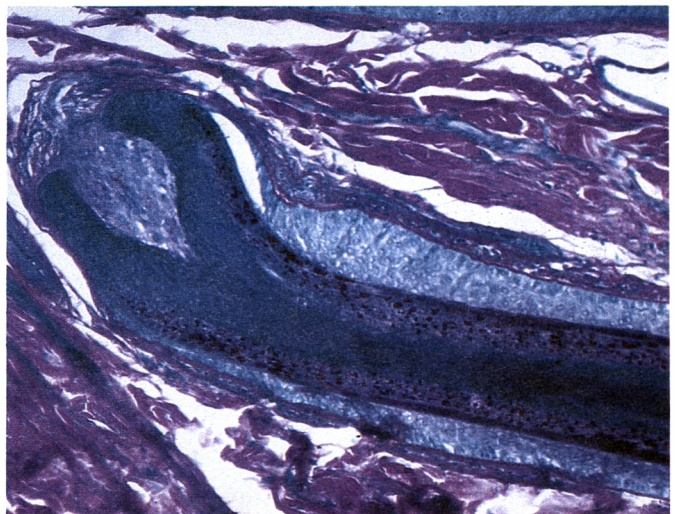
FIGURE 3. Hypotrichosis in a Polled Hereford. Notice curliness of hair which is frequently associated with this condition.



FIGURE 4. Hypotrichosis in a Polled Hereford. Notice short and curly hair on forehead.



FIGURE 5. Photomicrograph of skin of Polled Hereford affected with hypotrichosis. Notice abnormal trichohyalin granules in the Huxley's layer. Altmann's stain, 120X.



thought to be normal but which was slightly more curly than average Polled Herefords, were sent to Kansas State University for evaluation. All but the questioned calf were positive for hypotrichosis.

Five or six calves from which biopsies were obtained had significant skin lesions. Special staining properties are listed in Table I. Sweat glands were dilated. Arrector pili muscles were hypoplastic and not associated with the pilous-sebaceous apparatus. Microscopic lesions were observed in Huxley's layer of cells, which appeared degenerative, and the differentiation of cells in the inner root sheath was arrested. Huxley's layer contained prominent, microdroplets of trichohyalin between the sebaceous gland canal and the hair follicle (figure 5). Altmann's stain was strongly positive; the spheroid, semitranslucent, pleomorphic material stained brilliantly by fuchsinophilia.

The genealogy of affected calves and their relationship is shown in figure 6.

Discussion

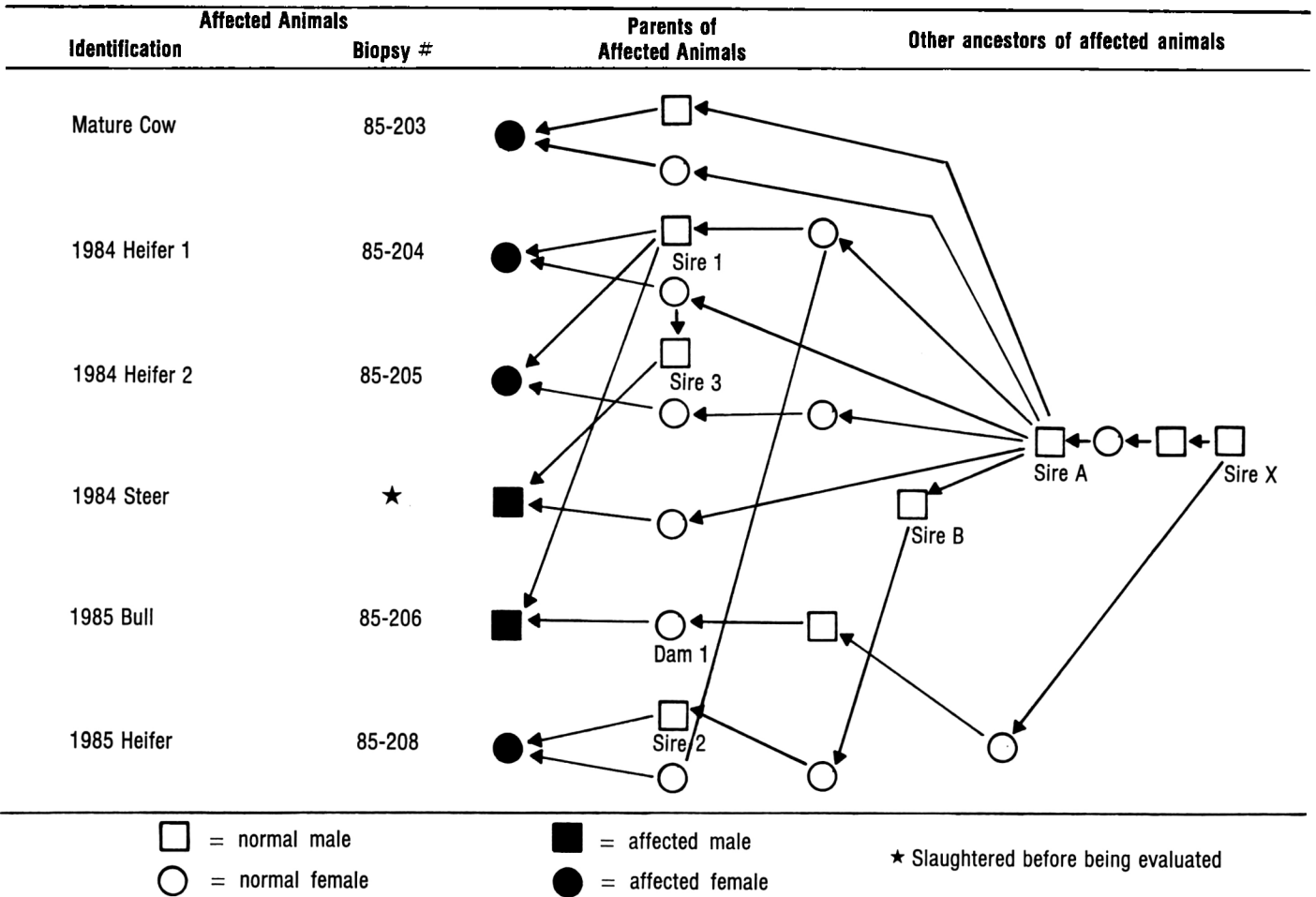
In bovine skin, each hair follicle is associated with an apocrine gland, a sebaceous gland, and an arrector pili

TABLE 1. Staining properties of trichohyaline granules in Huxley's layer of hair follicle in hypotrichotic Hereford cattle.

Stain	Reaction	Remarks
H&E	Pink	± Acidophilic
PAS	Stains the periphery	±
PAS-diastase	Stains the periphery	±
Toluidine blue	Blue	- No metachromasia
Prussian blue	Pink	- No iron
Masson's trichrome	Brown	-
Altmann's acid fuchsin-methyl green	Brilliant red green	± Brilliant fuchsinophilia

muscle.^{4 8 19} There is only one type of follicle in cattle.¹⁹ The normal hair cycle in cattle has been divided into three stages: anagen, catagen, and telogen.²² Embryologic development of hair follicles begins at day 77 of gestation, and a full complement of hair follicles is present at about 200 days gestation.^{8 22} Breed variation has been observed in cattle in the development of hair follicles.⁷ Huxley's layer acts as a depository cell in addition to those present in the follicle bulb for hair differentiation. Huxley's layer and the entire inner root sheath structurally complement the shape and size

FIGURE 6. Pedigree Relationships of Affected Animals.



of the hair and, thus, maintain the core of hardness during hair formation.³⁰

Absence of arginine-converting-enzyme has been suggested to be the cause of hypotrichosis in the Hereford breed of cattle. However, the presence of citrulline in follicular tissue indicates that the enzyme is present in hypotrichotic Hereford cattle.²⁶

Hair from hypotrichotic Hereford cattle is more soluble in tetramethylammonium hydroxide than hair from healthy cattle. The proteins solubilized with β -mercaptoethanol and sodium dodecylsulphate from hypotrichotic Herefords did not reveal any obvious differences, but the "B" proteins were quantitatively greater than in healthy cattle hair as recently reported.²⁵

Degenerative changes in Huxley's layer were reported to be the key to the development of hypotrichotic lesions.¹⁴

An evaluation of the pedigrees of the affected animals (figure 6) indicated that all affected animals, except for calf 85-206, were from both sires and dams that descended from Bull A, a relatively popular Polled Hereford sire, now deceased. No other common ancestor could be implicated. Sire A was the double grandsire of 85-203 and was from one to three generations removed from each of the affected animals's sires and dams again with the exception of the dam of 85-206 (see figure 6). All sires and dams of the affected calves born in this herd were completely normal in hair coat and coloration. The assumption of an autosomal recessive mode of inheritance was tested through an evaluation of the matings of two of the three sires (sires 1 and 2 in figure 6) of affected progeny to females that were progeny of known carriers. Sire 1 has been used for three calf crops and Sire 2 for one calf crop. The sire of the affected 1984 male (Sire 3) only produced one other calf in this herd and the sire of the affected mature cow had never been used in this herd. The females considered were daughters of Sire A, Sire 1, or Sire B (an implicated son of Sire A, see figure 6) or of cows known to have produced an affected calf in this herd. A total of only 10 calves met these criteria. Of these 10, seven were normal and three were affected. The affected 1984 males and the affected progeny of Dam 1 were not included. The expected proportion of affected progeny from matings of known carrier sires with dams that are progeny of a known carrier parent and a homozygous normal parent is one in eight. If some of the mates of the known carrier parents of these dams were themselves carriers, the proportion of affected progeny would be expected to be higher than .125. The observed proportion exceeds the expected proportion of 1 in 8 but the chi-square value, 2.8, is nonsignificant ($.50 > P > .30$). The higher proportion than expected may be explained by the small number of observations or by some carriers among the mates of the known carriers. These data support the conclusion of an autosomal recessive mode of inheritance for the mutant allele responsible for this condition, which is in accordance with other reports. The frequency of the allele appears to be quite low within the Polled Hereford breed but likely has increased because of

the popularity and wide usage of Sire A and his immediate descendants.

Several possible explanations exist for the occurrence of the condition in 85-206 whose dam (Dam 1) is not descended from Sire A in her pedigree of record. One explanation is pedigree error, since a son of Sire A was being used in this herd at the time of the conception of Dam 1. An alternative explanation is that the mutant gene descended through another route from a common great grandsire, Sire X, of both Dam 1 and Sire A (see figure 6). Another possible source of the mutant allele is a cow that was a common ancestor of both Sire A and Dam 1. This cow is five generations removed from Sire A and appears twice in the pedigree of Dam 1, four and five generations removed.

The growth rate of all affected animals has been normal. Both of the 1984 affected heifers became pregnant as yearlings and the affected mature cow also has shown regular reproduction. The weaning weight of the 1985 calf of the affected cow was among the best in the herd. The affected 1985 heifer calf, however, was observed to have rubbed the hair off her knees, and bare areas on the knees, neck, and poll and brisket of other affected animals have been noticed. Under harsher climatic conditions than exist in the southeast, therefore, this condition might possibly result in a reduction in performance. It is also possible that genetic variation in modifying genes may result in more severe levels of hair loss than those of the affected animals in this herd. Cases of hypotrichosis observed in other lines or breeds of cattle have exhibited more severe conditions.^{14 17 18} Whether these more severe conditions are due to the same allele as the one producing the condition in the affected calves in this study, with the more severe manifestation being the result of the presence of the allele in a different genetic and/or environmental milieu, or whether the more severe conditions are due to a different major gene is impossible to determine at this time.

Serious efforts to reduce the frequency of the gene responsible for the condition in this herd do not seem to be warranted because the condition has not resulted in a reduction in performance traits. However, since there is evidence of a more serious condition being caused by this mutant allele or a related one in other herds, it would not be advisable to use affected bulls as herd sires in registered Polled Herefords herds. Heterozygous bulls should be used only when considered very superior for other traits and when care is taken to avoid matings to heterozygous females. Affected females can be used effectively to test potentially heterozygous herd sires. Production of seven or more normal calves by a bull and from homozygous (affected) females without any affected calves indicates that he is free of the responsible allele at the .01 level of probability. Known heterozygous females, those cows that have produced affected calves, can also be used in this manner. At least 16 normal calves with no affected ones are necessary to declare a bull free of the allele with the same degree of accuracy as the production of seven progeny from affected

(homozygous) females.

Summary

Six cases of hypotrichosis were observed in a herd of Polled Hereford cattle. Five cases were from calves born in the herd in the 1984 and 1985 calving seasons and the sixth was a mature cow that had been purchased. Both male and female calves were affected by the condition characterized by curly hair over all or parts of the body, a deficiency of hair in the switch, and detectable amounts of hair loss on the poll, brisket, neck, and legs of some affected animals. In some cases, affected calves were a much lighter than normal color at birth. A positive diagnosis for the hypotrichosis condition was determined on all affected animals currently in the herd (five of the six cases) through evaluation of skin samples at Kansas State University. The microscopic lesion in the hair follicles were characterized by abnormally large trichohyalin granules. Evaluation of the pedigrees of affected animals revealed that five of the six affected animals were from sires and dams that both were descended from a common sire. The sire of the sixth affected animal also descended from the same common sire, whereas the dam of this affected animal descended from a great grandsire of the common sire. These relationships indicate that the common sire was a carrier of the allele responsible for this condition and that the observed hypotrichosis is likely inherited as an autosomal recessive, which is in accord with previous literature. Growth rate of all affected animals has been normal, as has the reproductive performance of the three females of breeding age. While the condition does not result in economic losses from a commercial standpoint, it is somewhat unsightly and could reduce the value of animals as breeding stock. Therefore, efforts should be made to select against hypotrichosis in Polled Hereford cattle.

References

1. Becker, R.B., C.F. Simpson, and C.J. Wilcox. Hairless Guernsey cattle. Hypotrichosis—a nonlethal character. *J. Hered.* 54:1-7. 1963. 2. Blood, D.C. and T.A. Henderson. Inherited congenital hypotrichosis. In: *Veterinary Medicine*, 4th ed. Williams and Wilkins Co., Baltimore. 892-893. 1974. 3. Bracho, G., J. Johnson, K. Beeman, and H.W. Leipold. Further studies of congenital hypotrichosis in Hereford cattle. *Zentralbl. Vet. Med.* 31(A): 72-80. 1984. 4. Carter, H.B. and D.F. Dowling. The hair

follicle and apocrine gland population of cattle skin. *Aust. J. Agric. Res.* 5: 745-756. 1954. 5. Craft, W.A. and W.L. Blizzard. The inheritance of semihairlessness in cattle. *J. Hered.* 25:384-390. 1934. 6. Eldridge, F.E. and F.W. Atkeson. Streaked hairlessness in Holstein-Friesian cattle. *J. Hered.* 44:265-271. 1953. 7. Goldberry, S. and L. Calhoun. The comparative histology of the skin of Hereford and Aberdeen Angus cattle. *Am. J. Vet. Res.* 20:61-68. 1959. 8. Hardy, M.H. and A.G. Lyne. The prenatal development of wool follicles in Merino sheep. *Aust. J. Biol. Sci.* 9:423-441. 1956. 9. Holms, J.R. and S.B. Young. Symmetrical alopecia in cattle. *Vet. Rec.* 66:704-706. 1954. 10. Huston, R., G. Saperstein, and H.W. Leipold. Congenital defects in foals. *Equine J. Med. Surg.* 1:146-161. 1977. 11. Huston, R., G. Saperstein, D. Schoneweis, and H.W. Leipold. Congenital defects in pigs. *Vet. Bull.* 48:645-675. 1978. 12. Hutt, F.B. A note on six kinds of genetic hypotrichoses in cattle. *J. Hered.* 54:186-187. 1963. 13. Hutt, F.B. and L.Z. Saunders. Viable genetic hypotrichosis in Guernsey cattle. *J. Hered.* 44:97-103. 1953. 14. Jayasekara, M.V., H.W. Leipold, and J.E. Cook. Pathological changes in congenital hypotrichosis in Hereford cattle. *Zbl. Vet. Med.* 26(A):744-753. 1979. 15. Kidwell, J.F. and H.R. Guilbert. A recurrence of the semihairless gene in cattle. *J. Hered.* 41:190-192. 1950. 16. Leipold, H.W. and S.M. Dennis. Congenital defects of domestic and feral animals. *Issues and Reviews Teratology*, Vol. II. Plenum Press, New York, New York. 91-136. 1984. 17. Leipold, H.W., S.M. Dennis, and K. Huston. Congenital defects in cattle: Nature, cause and effect. *Adv. Vet. Sci. Comp. Med.* 16:103-150. 1972. 18. Leipold, H.W., K. Huston, and S.M. Dennis. Bovine congenital defects. *Adv. Vet. Sci. Comp. Med.* 27:197-271. 1983. 19. Lyne, A.G. and M.J. Heideman. The pre-natal development of skin and hair in cattle (*Bos taurus* L.). *Aust. J. Biol. Sci.* 12:72-95. 1959. 20. McGavin, M.D. and G.I. Alexander. Viable hypotrichosis in a Jersey calf. *Queensl. J. Agric. Sci.* 18:105-108. 1961. 21. Mohr, O.L. and C. Wriedt. Hairless, a new recessive lethal. *J. Genet.* 19:315-336. 1928. 22. Neurand, K., W. Meyer, and R. Schwarz. Der Haarwechsel der Haussäugetiere. I. Allgemeine Problematik und zeitlicher Ablauf. *Dtsch. tierärztl. Wschr.* 87:27-31. 1980. 23. Olson, T.A. and R.L. Willham. Inheritance of coat coloration and spotting patterns of cattle: A review. *Ag. and Home Ec. Exp. Sta. Ames, Iowa.* 1982. 24. Pirchner, F. and W. Gruneberg. Alopecia in Pinzgauer cattle. *Ann. Genet. Sel. Anim.* 2:129-133. 1970. 25. Rose, R., J.E. Smith, and H.W. Leipold. Increased solubility of hair from hypotrichotic Herefords. *Zbl. Vet. Med.* 30(A):363-368. 1983. 26. Rose, R., J.E. Smith, and H.W. Leipold. Role of arginine-converting enzyme in hypotrichosis of Hereford cattle. *Zbl. Vet. Med.* 30(A):369-372. 1983. 27. Saperstein, G., H.W. Leipold, S.M. Kruckenberger, and N.A. Muckenhirn. Congenital defects of wild and zoo mammals. *ILAR News* 20:A1-A20. 1977. 28. Schleger, A.V., B.J. Thompson, and R.W. Hewetson. Histopathology of hypotrichosis in calves. *Aust. J. Biol. Sci.* 20:661-668. 1967. 29. Shand, A. and G.B. Young. A note on congenital alopecia in a Friesian herd. *Vet. Rec.* 86:907-909. 1964. 30. Straile, W. Root sheath-dermal papilla relationships and the control of hair growth. *American Elsevier Publishing Co. Inc.* 35:37. 1965. 31. Wipprecht, C. and W.R. Horlacher. A lethal gene in Jersey cattle. *J. Hered.* 26:363-368. 1935. 32. Young, J.B. A note on the occurrence of an epithelial defect in a grade Hereford herd. *Aust. Vet. J.* 9:298-300. 1953.

Results.

Why 4 out of 5 veterinarians recommend Ralgro® implants

When something works and gets good results ... the word spreads fast. That's why more veterinarians use and recommend RALGRO cattle implants with confidence.

You and your clients can expect up to 35 extra pounds of gain per head with RALGRO. It's a proven cattle management tool that's safe, easy-to-use and approved for use on both steers and heifers intended for beef — at any stage of growth.

Greater gains and improved feed efficiency are only part of the RALGRO advantage. It also gives you the opportunity to check your clients' herds and offer more services as a consultant on better health care and management programs.

Ask your veterinary distributor for more information on RALGRO growth-promoting implants or write International Minerals and Chemical Corporation, Box 207, Terre Haute, IN 47808. Or call 1-800-457-0871.

Discover the results a RALGRO implant program offers you and your clients today.

 **RALGRO**[®]
Anabolic Agent
The Quality of Life[®]
International Minerals & Chemical Corporation