Inheritance of Vertical Fiber Hide Defect

Larry V. Cundiff
MARC

Matthew P. Dahms
USDA-ARS

Mary V. Hannigan
USDA-ARS

Alfred L. Everett
USDA-ARS

Peter E. Buechler
USDA-ARS

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Inheritance of Vertical Fiber Hide Defect
Larry V. Cundiff, Matthew P. Dahms, Mary V. Hannigan, Alfred L. Everett, and Peter E. Buechler

Introduction
Cattle leather with vertical fiber hide defect (VFHD) breaks when stretched and, consequently, is not suitable for production of shoe uppers. Typical tensile strength of VFHD leather is only 50% of normal leather. VFHD is caused by a structural defect of collagen fiber orientation in the corium layer of cattle hides. The defect was first described by Amos, an Australian research chemist, in 1958. Economic losses to the leather industry were estimated to exceed $10 million in 1973. The cost can be high because the defect is often not detected until after the expense of tanning has been incurred. The defect does not occur uniformly throughout the hide, but tends to be localized in the upper rear quarter (rump area). Often it may extend forward and downward to involve 75% of the trimmed hide.

Biopsy procedure
Scientists at the Animal Bio-Material Laboratory at the Eastern Regional Research Center (ERRC), ARS, USDA, Philadelphia have been conducting research on this defect since the 1960's. At that time, they developed a biopsy and histological examination procedure which can be used to diagnose VFHD either on hide samples taken by biopsy on live animals or from hide samples taken after slaughter. We have used this procedure in all of our joint experiments on Inheritance of VFHD. Biopsy samples 1 inch in diameter were taken from the rump area of each animal at a point about 10 inches in front of the tail and 10 inches down from the mid-line with an automatic biopsy gun. Each biopsy specimen was put into a tube containing a 10% formalin solution and sent to the ERRC for histological evaluation. The fiber bundle structures are designated as:

- **Normal (N)** = Compact with bundles interweaving at an angle of approximately 50 to 60 degrees;
- **Vertical (V)** = Mostly vertical with little or no interweaving and usually very loose in appearance; and
- **Intermediate (I)** = Loosely interwoven with variable upright angle of weave and vertical appearance in localized areas.

Unfortunately, it has not been practical to adopt this procedure outside of the research laboratory on a routine basis in the tanning industry because the histological examination is time consuming and requires a high level of skill.

Inheritance of VFHD
In early studies, VFHD was diagnosed in the progeny of certain sires but not others, suggesting that VFHD was heritable. In addition to a heritable component, early indications were that the condition was associated with carcass fatness. Thus, biopsy samples were studied from Hereford and Holstein identical and fraternal twin sets fed either high or low energy diets in experiments conducted at the University of Wisconsin in the early 1970's. Thirteen cases of the defect were found in fifteen pairs of Herefords, with matching occurrence in identical pairs but not always in fraternal pairs. The defect was not found in any of the Holstein twin pairs. Diet had no effect on expression of the defect. There was some indication that cows with the condition had lower reproductive performance than those without the condition.

This observation eventually led to a larger experiment at MARC in which 604 biopsies were evaluated from 465 Herefords and 139 Angus ranging from 4 mo to 9 yr in age. Incidence of VFHD was not associated with reproduction rate of females or other performance characters. VFHD was found in 13.3% of the Herefords but in no Angus. The Herefords were progeny of 85 sires. Sire effects were significant, and estimates of heritability were very high (84%).

The high heritability suggested that the condition was primarily, if not completely, under genetic control. The data set on 465 Herefords included 44 offspring-dam pairs. Examination of offspring and parental frequency distributions indicated that inheritance of the condition was not due to a single autosomal additive or dominant gene, but possibly was due to an autosomal recessive.
At about the same time in 1983, Australian workers studied frequency distributions in 365 Herefords grouped according to sires and dams. They also concluded that VFHD was likely due to an autosomal recessive gene. However, this hypothesis could not be confirmed conclusively because no matings were available where known VFHD sires had been mated to known VFHD dams. In 1984 we mated a Hereford bull with a known VFHD phenotype to Hereford cows with known VFHD phenotypes and to Angus cows not showing the defect. Angus were chosen because the defect had never been observed in the breed. All offspring (12) offspring from the VFHD x VFHD matings expressed the defect, while no (12) offspring out of VFHD x non-VFHD (Angus) matings expressed the defect, confirming that VFHD was inherited as an autosomal recessive trait.

Results also indicated that the vertical (V) and intermediate (I) phenotypes represented two degrees of expression for the same genotype, rather than extra intensity of expression associated with one vs two copies of the same allele. Perhaps degree of expression for VFHD varies like that for hornedness in Bos taurus cattle. In Bos taurus breeds, presence or absence of horns is determined by genotype for a simple autosomal recessive gene, but degree of expression varies both within and especially between breeds from short to very long, presumably due to effects of other genes (at other loci) affecting expression of the trait.

Since the condition had only been diagnosed in Herefords and one Hereford x Holstein cross, an additional experiment was conducted to evaluate the frequency of VFHD in other breeds of cattle. Hide biopsies were taken in November of 1985 on 35 Pinzgauer, 55 Red Poll, 47 Brown Swiss, 52 Charolais, 69 Gelbvieh, 55 Simmental, and 45 Limousin heifers produced at MARC. The defect was found in only one Simmental heifer. The sire and dam of this heifer were both 1/16 Hereford. Simmental are registered as purebreds if they are 15/16 or more Simmental. Thus, we do not know whether or not the VFHD gene originated from “full blood” Simmental originally imported from Europe or from Herefords contributing to foundation of the breed in North America. Incidence of the condition in European breeds of cattle has not been studied in Europe. The defect is likely to be present in North American Simmental and other breeds including Pinzgauer, Gelbvieh, and Limousin, at least at a low frequency, if for no other reason than that it was present in Herefords or possibly other breeds used as foundation stock to grade up to purebreds of each breed. However, to date, indications are that the gene, if present, is only present at low frequencies in all breeds evaluated except the Hereford.

Estimates of phenotypic frequency and frequency of the VFHD gene in populations to date are shown in Table 1. In our data, 13.3% of the Herefords were affected, suggesting a gene frequency (q) of 0.37. It was also found in progeny of sires representing diverse lines of the Hereford breed. In Australian data, the phenotypic frequency and gene frequency were even higher than in our data. The Herefords in our study were all horned, but in Australia, both polled and horned Herefords were included in their sample. It is not known why the frequency of the gene is so much higher in Herefords than in other breeds.

To the extent that the VFHD gene is present at a high frequency only in Herefords and either not present or present only at a low frequency in other beef or dairy breeds, it would appear that the problem should be diminishing in importance and costs to the tanning industry with increased use of crossbreeding during the last 20 years. If the defect is present at a frequency V in one breed:

- For n breeds used in rotation, phenotypic frequency of the recessive defect would be reduced to \( (1/n)(1/2n-1)V \). For example, if \( V = 0.133 \) and \( n = 2 \) as in a two breed rotation, (e.g., Hereford and one other breed) 2.2% of the cattle would express VFHD, and in a three breed rotation only .6% of the cattle would express VFHD.

In a composite population, the frequency would be \( Pq(v) \), where \( Pq \) is the fraction contribution of breed v to the composite population. For example, if \( V = 0.133 \) and \( Pq = 1/2 \) (e.g., 1/2 Hereford), 3.3% of the cattle would express VFHD; If \( Pq = 3/8 \), 1.9% of the cattle would express VFHD; and if \( Pq = 1/4 \), only 0.8% of the cattle would express VFHD.

Thus, incidence of the recessive defect will be very low in the total cattle population if systematic crossbreeding programs or composite populations are used for commercial production. It is estimated that about 70% of the calves produced in the U.S. are crossbreds. Considering the expense and technical difficulty of evaluation, intensive selection against VFHD is not justified, especially if the condition is present in only one breed.

Table 1—Estimates of incidence (phenotypic frequency) and gene frequency for vertical fiber hide defect (VFHD) by breed

<table>
<thead>
<tr>
<th>Breed</th>
<th>Number sampled</th>
<th>Number with VFHD</th>
<th>Frequency of phenotype q^2, %</th>
<th>Frequency of gene q, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pinzgauer (MARC)</td>
<td>35</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Red Poll (MARC)</td>
<td>55</td>
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<td>0</td>
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<td>Brown Swiss (MARC)</td>
<td>47</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<td>Charolais (MARC)</td>
<td>52</td>
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<td>0</td>
<td>0</td>
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<tr>
<td>Gelbvieh (MARC)</td>
<td>69</td>
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<td>0</td>
<td>0</td>
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<td>Simmental (MARC)</td>
<td>55</td>
<td>1</td>
<td>1.8</td>
<td>13</td>
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<td>Limousin (MARC)</td>
<td>45</td>
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<td>0</td>
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<td>Angus (MARC)</td>
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<td>Holstein (Wisconsin)</td>
<td>15</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Hereford (MARC)</td>
<td>465</td>
<td>62</td>
<td>13</td>
<td>37</td>
</tr>
<tr>
<td>Hereford (Australia)</td>
<td>362</td>
<td>83</td>
<td>23</td>
<td>48</td>
</tr>
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