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Risk Factors for Congenital Umbilical Hernia in German Fleckvieh

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SUMMARY

Risk factors for congenital umbilical hernias were investigated in German Fleckvieh calves up for sale at livestock markets. Data from 53 105 calves were collected from 77 livestock auctions in 1996 and 1997. The overall incidence of congenital umbilical hernia was 1.8%. A significant influence on incidence was exerted by the sex of the calf, the occurrence of multiple births, the market place/market date, the sire and the sire line. The proportion of Red Holstein blood in the calf, the dam's lactation number, gestation length, 305 day milk performance and the herd milk level were not significant factors. Herdmate averages for calves differed significantly in their incidence. Heritability estimates on the liability scale for congenital umbilical hernia were about 0.4 and progeny groups of sires at risk for congenital umbilical hernia were in the range 0.1% to 14.2%. The segregation pattern could not be explained either by an autosomal recessive or by an autosomal dominant monogenic model. It seems likely that more than one gene locus is involved in the underlying genetic mechanism. Breeders should be aware of the genetic implications of congenital umbilical hernias.

KEYWORDS: Umbilical hernia; cattle; risk factors; inheritance.

INTRODUCTION

Congenital umbilical hernias are common defects in calves, particularly in Holstein Friesian cattle where frequencies between 4 and 15% have been observed (Hondele, 1986; Müller *et al.*, 1988; Virtala *et al.*, 1996). It is generally accepted that a genetic component is involved in congenital umbilical hernias, but the hypotheses on the mode of inheritance are rather conflicting. Outbreaks of umbilical hernias in herds using newly introduced service sires have suggested that an autosomal dominant gene with incomplete penetrance is involved (Angus & Young, 1972; Labik *et al.*, 1997). However, the screening of progeny groups of Holstein Friesian sires has indicated that the segregation of a single recessive gene may be responsible for their development (Gilman & Stringam, 1953; Surborg, 1978; Zhigachev, 1983). Surborg (1978) screened calves hospitalized at the School of Veterinary Medicine, Hannover, and found more common ancestors in animals affected by umbilical hernia compared to control animals. Moreover, increasing the proportion of Holstein Friesian blood was significantly associated with a higher frequency of umbilical hernias in probands. Rieck and Finger (1973) could not confirm either a monogenic recessive inheritance or a polygenic inheritance for umbilical hernias.

The objectives of the present study were to record the incidence of congenital umbilical

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hernias in German Fleckvieh calves using a standardized protocol in order to quantify important influences on occurrence and to analyze progeny of bulls for their incidence of these hernias.

MATERIAL AND METHODS

In total, 53 105 calves of the German Fleckvieh breed were examined for congenital umbilical hernias at 77 auctions held in Miesbach and Traunstein, Upper Bavaria, between January 1996 and June 1997 (Miesbach) and March 1996 and June 1996 (Traunstein). Palpable openings in the umbilical region >1.5 cm were defined as umbilical hernias, even if no hernial sac was developed. Inflammation, abscesses or fistulae were excluded from the diagnosis. Recordings included the animal's identity, the size, form and feel of the hernia and, in the case of hernial content, its consistency, possible adhesions and whether it was easily replacable. Table I shows the distribution of the calves examined by birth, age and sex. The small percentage of female calves is explained by the fact that mainly male calves were brought to the auctions. The age of the calves mostly varied between three and eight weeks, with a mean of five weeks. The diameter of hernial openings was between 1.5 and 9 cm with about 47% of affected calves having a hernia with a diameter of >3 cm. Only a few affected calves (1.8%) had a congenital umbilical opening with a diameter >7 cm. In most cases (76.5%) a hernial sac was present and in 66.5% of affected calves the hernial contents could be squeezed back into the abdomen. In 57.4% of cases the size of the hernial sac reached a diameter of up to 5 cm and it exceeded 5 cm in 19.1% of cases.

Statistical methods

For each calf examined, the identity, market place and date of auction were recorded. Additional data from the breeders' association and from the official milk recording organization in Bavaria (LKV) could be merged with the clinical information using the calf's identity. For the analysis of effects of the dam, only those calves with at least four herd mates in the dataset were used. Thus the original dataset was reduced to 42 853 calves. Inclusions of milk records into the analysis required additional restrictions on the dataset. Heifers and cows without at least one complete 305 day lactation were deleted. In this reduced dataset, 27 023 calves remained.

The statistical data analysis was performed by using generalized linear model procedures of SAS, Version 6.12 (SAS institute Inc., 1996). The generalized linear models shown below were employed to analyze systematic effects on the incidence of congenital umbilical hernia as a binary trait.

Model 1

$$Y_{ijklmn} = \mu + sex_i + birth\ type_j + market_k + age_l + sire_m + e_{ijklmn}$$

Y_{ijklmn}: observed congenital umbilical hernia of the ijklmn-th calf
μ : model dependent constant
e_{ijklmn}: residual variance

Single and twin or triplet births were distinguished using the independent variable birth type. Each auction was regarded as a single factor using the independent variable market. Only sires with more than 100 progeny were regarded in the model; all other sires were combined in a pseudosire effect.

Table I
Distribution of calves examined (n = 53105) by single and twin births, age and sex

Age in weeks	*Single birth*				*Twin birth*			
	Male		*Female*		*Male*		*Female*	
	n	%	*n*	%	*n*	%	*n*	%
≤3	5726	14.3	1443	15.5	102	4.2	36	2.6
4	8434	21.1	1275	13.7	276	11.4	140	10.0
5	10379	26.0	2085	22.5	547	22.5	271	19.4
6	7903	19.8	1836	19.8	622	25.6	325	23.3
7	4258	10.6	1234	13.3	441	18.1	275	19.7
≥8	3295	8.2	1410	15.2	443	18.2	349	25.0
Total	39995	75.3	9283	17.5	2431	4.6	1396	2.6

Model 1 was extended by introducing the proportion of Red Holstein blood in the calf and the effect of sire line (model 2). Sire line was defined by the founder sire traced back through the paternal path up to eight generations. The minimum requirements for a sire line to be regarded in the model were that at least five sires and 50 calves were represented by the sire line. By using the information on the dams of the calves, effects of duration of pregnancy, lactation number and 305 day milk performance for milk yield, fat percentage and protein percentage could be incorporated into model 3. Additionally, the frequency of umbilical hernias for herdmates of calves examined could be tested for significant accumulation in herds.

Model 3

$Y_{ijklmnopqrs} = \mu + F_i + \text{lactation number}_j + b_1 \text{ duration of pregnancy}_k + b_2 \text{ milk yield}_l + b_3 \text{ fat}_m + b_4 \text{ protein}_n + b_5 \text{ HM-hernia}_o + b_6 \text{ HM-Y}_p + b_7 \text{ HM-F}_q + b_8 \text{ HM-P}_r + e_{ijklmnopqrs}$

$Y_{ijklmnopqrs}$: observation of the ijklmnopqrs-th calf

μ : model dependent constant

F_i : fixed effects as in model 1

b_1-b_8 : regression coefficients (linear, quadratic, cubic)

HM-hernia$_o$: frequency of congenital umbilical hernias of herdmates

HM-Y$_p$: 305 day milk yield of maternal herdmates

HM-F$_q$: fat percentage of 305 day milk performance of maternal herdmates

HM-P$_r$: protein percentage of 305 day milk performance of maternal herdmates

$e_{ijklmnopqrs}$: remainder

Milk records related to the mother of the calf. Herdmate frequencies for congenital umbilical hernias included all calves with records for the specific herd except the animal itself. Only these dams whose calves were retained in our material were considered in the dataset. Herdmate averages for 305-day milk records were analogously calculated.

Heritability was estimated using restricted maximum likelihood and the sire variance (s^2_v) and residual variance (s^2_e) component: $h^2 = 4s^2_v / (s^2_v + s^2_e)$. The heritability on the liability scale (h^2_l) was obtained by using following transformation: $h^2 = h_L^2 z^2/(p\ (1–p))$, where z is the ordinate of a standard normal density function corresponding to threshold t, and p is the frequency of affected animals (Gianola, 1982). The threshold t was determined by the frequency of affected animals.

RESULTS

The analyses of variance revealed significant effects for sex of calf, birth type, age of calf at examination, market place and date, sire line, sire, and frequency of affected herdmate calves.

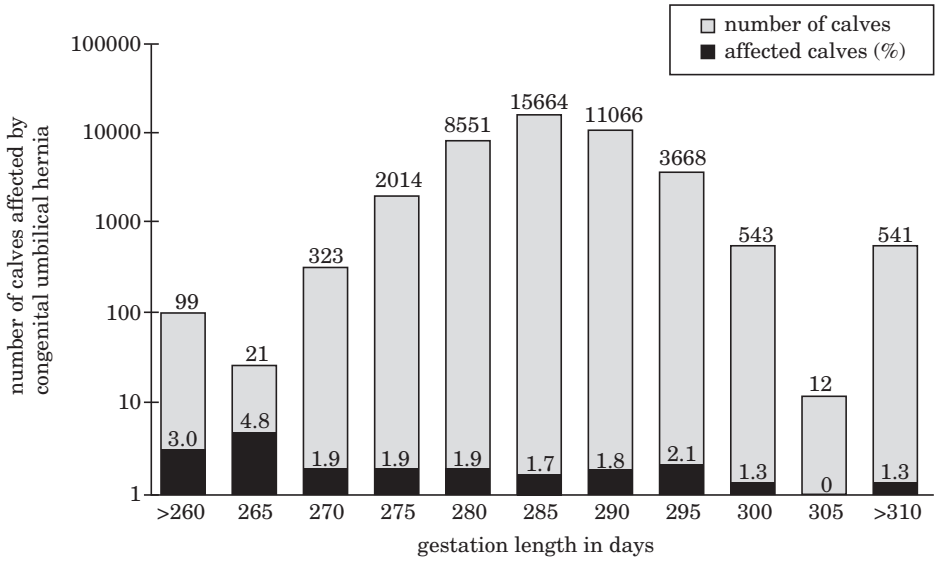

Fig. 1. Risk of congenital umbilical hernia by gestation length (n = 41725).

Table II
Analysis of variance for occurrence of congenital umbilical hernia in German Fleckvieh calves employing models 1, 2 and 3

Source of variation	*df*	*MSR*	*F-value*	P
Model 1				
sex of calf	1	0.4840	28.0	<0.001
birth type of calf	1	0.9087	52.6	<0.001
market place/date	76	0.0493	2.9	<0.001
age at examination	5	0.0445	2.6	0.024
sire of calf	75	0.0755	4.4	<0.001
Model 2				
Red Holstein blood	2	0.9397	2.3	0.101
sire line	21	0.0855	4.9	<0.001
Model 3				
lactation number of mother	5	0.0269	1.8	0.102
gestation length	1	0.0491	3.4	0.067
milk yield (kg) of mother	1	0.0025	0.2	0.677
milk fat (%) of mother	1	0.0173	1.2	0.276
milk protein (%) of mother	1	0.0021	0.1	0.705
herdmate average for:				
– umbilical hernia	1	0.9153	62.7	<0.001
– milk yield (kg)	1	0.0007	0.1	0.823
– milk fat (%)	1	0.0011	0.1	0.781
– milk protein (%)	1	0.0002	0.01	0.909
Total	186	0.2048	14.3	<0.001

Only linear regression coefficients are displayed; df: degrees of freedom; MSR: mean sums of squares

Lactation number, milk performance of the dam, milk level of herd, where the calves originated, and the duration of pregnancy had no significant influence on the incidence of umbilical hernias (Table II). The mean incidence of hernias over all 77 auctions reached 1.8%. Male calves showed a significantly higher incidence (2.2%) compared to female calves (1.5%). Calves of less than five weeks of age exhibited more congenital umbilical hernias than calves aged six, seven or eight weeks. Comparing single to multiple births, a significantly higher incidence was evident in twin or triplet calves. Male calves of single births had an incidence of 1.8%, whereas male calves of multiple births reached an incidence of 3.2%. Similarly, in female calves the corresponding incidences were 1.3% and 3%, respectively. Shorter gestation periods increased the risk for congenital umbilical hernias almost linearly by a factor of 1.3% for 10 days (Fig. 1). The incidences of congential umbilical hernias showed seasonal variations; at the auctions in Miesbach the incidence fluctuated by around 2% between January and June 1996, and in the following period up to the end of the study the incidence was around 1.4%. At the auctions in Traunstein the incidence remained at about 3.5%.

Calves descended from sires with a small percentage of Red Holstein blood exhibited nearly the same incidence as purebred Fleckvieh calves. However, the average proportion of Red Holstein blood was only about 1.5%. All sire lines had progeny with congenital umbilical hernias, but they differed significantly in incidence. Sire lines represented by fewer numbers of progeny showed a tendency to higher incidences (see for example sire lines 2, 16 and 20 in Fig. 2). Sires lines having >3000 progeny reached incidences of between 1.1% (sire line 8) and 2.6% (sire line 3). The highest incidence was observed for sire line 11 with 4.2% affected calves. The effect of the sire on the incidence of congenital umbilical

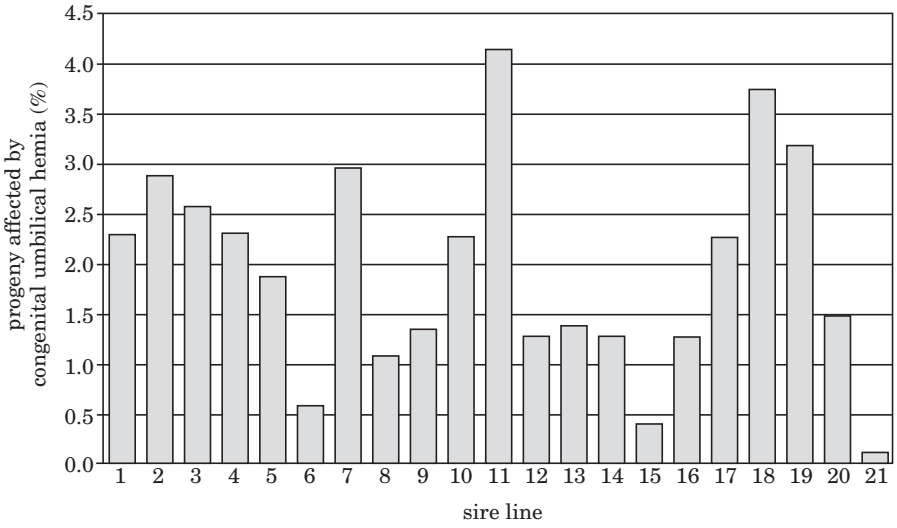

Fig. 2. Incidence of congenital umbilical hernia by sire lines in German Fleckvieh.

hernias was significant. Heritability estimates obtained by restricted maximum likelihood were low. Using 25, 50 or 100 progeny per sire resulted in estimates of $h^2 = 0.04$ (>100 progeny) or $h^2 = 0.05$ (>25 or 50 progeny). Transformation on the liability scale gave estimates of $h^2 = 0.36$ and 0.45 respectively. The incidences for single progeny groups varied between 0.1% and 7.1%. About 75% of the sires showed incidences ranging from 0.1% to 3% (Fig. 3). Among these 76 progeny groups, 14 were at risk up to 1%, 27 at risk from 1% to 2%, 16 at risk from 2% to 3%, and 19 at risk from 3% to 7.1%. When entering progeny groups with 50 progeny into the analysis more progeny groups with higher incidences appeared (Fig. 4). The class with >7% affected progeny included about 5% ($n = 8$) of all sires. Three progeny groups reached incidences >8%. The highest incidence for a progeny group was 14.2%.

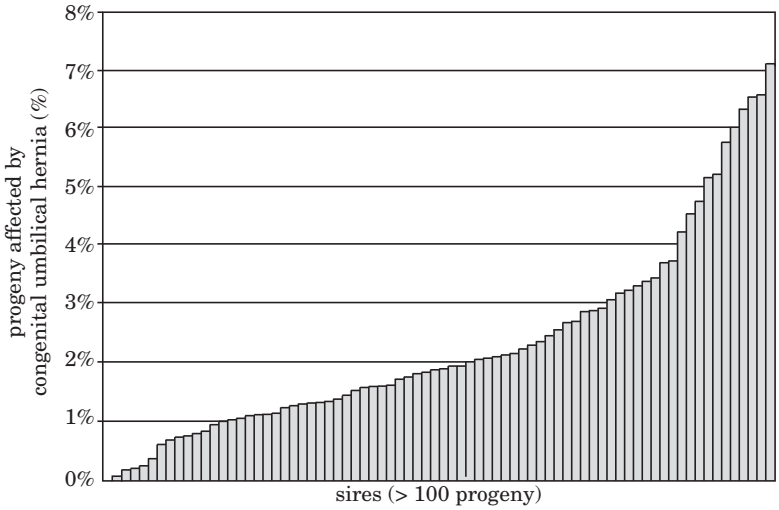

Fig. 3. Distribution of incidences (%) of congenital umbilical hernias by sire progeny groups (>100 progeny per sire; 76 sires total).

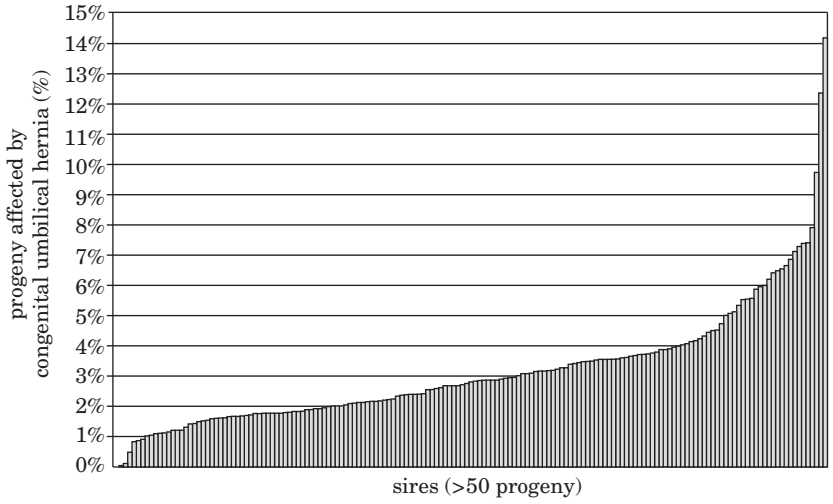

Fig. 4. Distribution of incidences (%) of congenital umbilical hernias by sire progeny groups (>50 progeny per sire; 164 sires total).

We tested inheritance for an autosomal recessive locus using the following assumptions derived from our analyses. Let 'a' equal the recessive defective allele and 'A' the wild-type (normal) allele in a one locus model. Underlying an autosomal recessive allele responsible for congenital umbilical hernias, all sires have to be heterozygous in order to produce affected progeny with heterozygous dams. The segregation of the defective allele must have occurred in all sire lines and sires represented in our material, because in all progeny groups affected calves were observed. Based on the work of Hondele (1986), we assumed that in females the frequency of affected calves is higher by a factor of 1.5. The allele frequency of the dam generation was deduced by assuming the same allele frequency as in non-affected female calves: the frequency of affected female calves in single births = 1.85% × 1.5 = 2.8%; the frequency of genotypes homozygous for aa = 0.028; the frequency of allele 'a' = 0.167 = $\sqrt{0.028}$ and the frequency of allele 'A' = 0.833 = 1–0.167. The frequencies of genotypes AA, Aa and aa equal 0.693, 0.279 and 0.028 respectively. Taking into account selection of non-affected calves as breeding cows, the expected allele frequency of 'a' decreases. After removing 'aa' animals, the frequencies of genotypes AA and Aa in breeding animals are 0.713 and 0.287 respectively, with q_a = 0.1435. Assuming that these dams were mated to heterozygous sires, the expectation for the incidence of affected female calves is equal to the frequency of homozygotes 'aa' = 0.1435 × 0.5 = 0.0718. Using the assumed penetrance for male calves (1/1.5 = 0.67), 4.8% male calves with congenital umbilical hernias are expected under a one locus autosomal recessive gene model. When testing expected frequencies for male calves with single births using this model against observed frequencies, we obtained following χ^2-value with one degree of freedom: $(0.048 \times 39995–0.0185 \times 39995)^2/(0.048 \times 39995) = 725.1$. Thus the model of an autosomal monogenic recessive inheritance had to be rejected ($P < 0.001$).

DISCUSSION

This study comprised a larger dataset in order to analyze the incidence of congenital umbilical hernias in German Fleckvieh calves and to assess the importance of influencing factors. Most epidemiological studies on umbilical hernias in cattle found higher incidences. Hondele (1986) investigated 4108 calves mainly of the German Brown breed and reported a frequency of 3.8 %. His material included 138 German Fleckvieh calves, of which 5.8% were affected. Müller *et al.* (1988) reported an incidence of 11.4% in German Black Pied cattle. The prevalence for an open hernial ring in the first week of life varied between 18% and 24.3% depending on the farm sampled. In a

study of 410 female dairy calves from 18 farms in the USA, a prevalence of umbilical hernias of 15.1% was reported by Virtala *et al.* (1996). Infection played a significant role in the development of hernias in this study. One reason for the lower incidence in our study was that we did not observe the higher incidence in female calves as reported by previous authors (Gilman & Stringam, 1953; Rieck & Finger, 1973; Hayes, 1974; Brem *et al.*, 1985; Hondele, 1986; Müller *et al.*, 1988). Hondele (1986) found a 1.5 fold higher likelihood of female calves being affected compared to male calves. The incidence of umbilical hernias in his study reached 2.4% in male calves which, although higher than in our analysis, is in a similar range. Furthermore, the female calves in our study might not be representative, because usually female calves without any defects are selected for breeding purposes and sold at livestock auctions. Cases of umbilical hernias in female calves may also have been overlooked by farmers. Finally, there can be difficulties when comparing incidences from different studies. For example, in our study we excluded all cases of actual or earlier umbilical infections accompanying umbilical hernias, which may not have been the case in previous work.

Multiple births and shortened gestation lengths are two important risk factors for congenital umbilical hernias. The multiple analysis of variance showed that both factors contribute to the increased risk, even if twins or triplets are born on average six days earlier than single calves. The higher incidence of congenital umbilical hernias in twins or triplets may have additional causes other than a shorter gestation length. It should be noted that in the German Brown study of Hondele (1986), twins were not prone to a higher risk of hernias. The statement of Rieck and Finger (1973) that births with more than one calf are the main cause of umbilical hernias was not supported in our analysis.

The genetic influence on the incidence of congenital umbilical hernias was evident as shown by the significant differences among sire lines and sires – even if in multiple variance analyses other risk factors were seen. Studies in German Black and White cattle as well as in German Black Pied cattle found rather large differences among paternal progeny groups (Surborg, 1978; Müller *et al.*, 1988). In our investigation, we could not find any sire causing an outbreak of congenital umbilical hernias with an incidence >20%. The high incidence in the progeny of eight sires can be explained by the higher genetic disposition of mating partners. An indication of a varying degree of affected calves in different herds and a higher accumulation of defective alleles led to the significant effect of frequency of affected herdmate calves in the analysis using model 3.

We conclude from our analyses that the probability that there is an autosomal monogenic inheritance for congenital umbilical hernia is low. Moreover, a very low frequency for the defective allele in dams has to be assumed in order to achieve an incidence of 1.85% affected male calves. Recessive Mendelian inheritance in our material required all sires to be heterozygous for the defective allele, as affected calves were found in each progeny group. Under this hypothesis the dams must have an allele frequency of $q_a = 0.0552$ ($0.5 \times 0.0552 \times 0.67 = 0.0185$). It is unlikely that the allele frequencies would have undergone such a high drift among parents, because congenital umbilical hernias segregate for a long time in German cattle populations and in the sires of dams (maternal grandsires of calves) we found a similar distribution of affected progeny as in the sires.

The significant effects of birth type, sex and age as well as the clear trend of shorter gestation length to more congenital umbilical hernias indicate the influence of additional genes. It could be that some genes regulate the prenatal opening of the umbilical area and other genes control postnatal ring closure. In our study we were able to show that in some cases the hernial sac was filled with fat and collagenous tissue when a sample of probands was slaughtered at about 15 months of age. This was also the finding of Herrmann (1999). These cases should be distinguished from those where abdominal organs can pass through the umbilical ring into the hernial sac and also indicate the different activity of genes to close an unnatural abdominal opening.

The incidence of congenital umbilical hernias can be reduced if all breeding bulls are examined as calves and a veterinary certificate confirms a closed umbilical ring. Progeny testing of sires should be recommended because most calves are sold via auctions and recording is feasible at fairly low costs. A general progeny test for congenital defects of calves would be more desirable. Veterinary practitioners, AI-technicians and calf handlers would have to cooperate for this data recording. Such a system would however be very helpful to minimize genetic

health problems in calves and to focus more attention on the general health status of calves and important factors influencing it.

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