Perinatal Mortality in Holsteins

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P. Jeffrey Berger, Professor of Animal Science;
and C. L. Meyer, Graduate Student

Summary and Implications

A comprehensive analysis of perinatal mortality for Holsteins in the upper Midwest was recently completed. Results showed that the incidence of perinatal mortality or calf losses near birth has been increasing since 1985; from 9.5% in 1985 to 13.2% in 1996 for virgin heifers; 5.3 to 6.6% in multiparous cows over the same time period. Replacement value of calves lost to perinatal mortality in the United States is about $125.3 million per year.

Sufficient data exist to permit identification of those sires whose daughters have a higher than average incidence for perinatal mortality with the birth of their calves. Further research is in progress to more completely establish the mode of inheritance for perinatal mortality. Other quantitative traits associated with early calfhood diseases, e.g., gestation length, dystocia, and incidence of twins are being examined.

Introduction

Perinatal mortality (PM), defined as a calf that dies just prior to, during, or within 48 h of parturition, represents a reoccurring concern for breeders of Holstein dairy cattle in the United States. Sufficient data exist from a large number of herds in the upper Midwest to indicate the PM is more than a single aberrant problem in one herd (Table 1). The exact cause of PM is unknown; size of calf, dystocia, and genetic factors have all been proposed as possible explanations.

Results and Discussion

Incidence and spread of early calfhood diseases may be more pronounced now than in the past, particularly because there is more co-mingling of replacement heifers and cows from different herds to meet the demands for expansion and formation of new, large herds.

Table 1. Number of records and the incidence of perinatal mortality (PM) (%) and dystocia (%) from 1985 to 1996.

		PM	Dystocia[3]		
Parity[1]	Records	(%)[2]	1	2	3+
H	167,472	11.1	71.4	9.6	19.0
C	498,869	5.7	89.3	4.7	6.0

[1] H = virgin heifers and C = older cows.
[2] All percentages are simple percent of total number of observations in the given year.
[3] 1 = no assistance, 2 = slight problem, and 3+ = needed assistance.

Dystocia and perinatal mortality are certainly interrelated. It is unclear whether dystocia is the cause of PM or an effect due to PM. They are certainly highly related characters. Perhaps we should no longer think of PM and dystocia as separate characters describing what happens with assistance at parturition. It seems appropriate to move forward with our thinking to design new ways to identify the function of specific genes that cause either PM, dystocia, or both in the sense of interacting biological functions.

Traditional thinking was that dystocia was highly related to birth weight of the calf and that more dystocia was associated with heavier birth weights. Of course, there was never any incentive for dairy producers to weigh calves, so birth weights on dairy cattle have rarely been available, and scores for dystocia were used to determine the expected percentage of difficult births in heifers. This may be partially correct, but other factors are needed to resolve inconsistencies with this line of reasoning. Only 40% of the variation in dystocia is explained by birth weight, leaving sufficient room for other factors. For example the relatively high incidence of PM at 6.1% in virgin heifers and 3.9% in multiparous cows in births requiring no assistance remains unexplained. Compare this with 11.1 and 5.7% PM over all levels of dystocia in Table 1.

Table 2. Incidence of perinatal mortality (PM) among virgin heifers needing assistance with the birth of their calf.

Gestation length [1]	Number	PM (%)
–15 to –13	454	41.6
–12 to –10	865	35.1
–9 to –5	4,625	28.6
–4 to 2	16,344	25.7
3 to 15	9,566	29.2

[1] Expressed as a deviation from mean of 280 d.

Gestation length now appears to be a quantitative character deserving more attention as a way to uncouple the factors causing PM in dairy cattle. Table 2 gives an important discovery. Briefly, a higher incidence of PM, 41.6%, was found at gestation lengths up to two standard deviations (sd = 7.5 d) below the mean gestation length of 280 d for Holsteins. Also, the incidence of PM decreases as gestation lengths approach the mean, e.g., from 41.6% at –15 to –13 d to 25.7% centered about the mean at –4 to 2 d. Similar trends with high incidences of PM for short gestation lengths and lower incidences of PM for longer gestation lengths also were found in multiparous cows at all levels of dystocia, e.g., 1 = no assistance, 2 = slight problem, and 3 = needed assistance.

Holstein-Jersey Crossbreeding

Crosses among widely divergent breeds of dairy cattle have long been discussed as a means of avoiding dystocia in first-calf heifers. A Holstein-Jersey cross is an attractive type of crossbred for several reasons: low dystocia, added value for components, economic pressure, and to decrease inbreeding and increase heterosis (C. Van Tassell, 2000, personal communication, USDA-AIPL Gene Evaluation and Mapping Laboratory, Beltsville, MD). Unfortunately, there is little documented information about the true advantages and disadvantages for this type of crossbred in dairy cattle. Even if these crossbreds exist, it is almost impossible to recover their records from dairy records processing centers for further investigation.

Most dystocia occurs in virgin heifers. We can even identify easy calving sires for use in matings to virgin heifers. Also, dystocia and PM are genetically related. Yet, there seems to be reluctance on the part of breeders to subscribe to a rigid breeding program for heifers to minimize dystocia and subsequent reproductive problems. Perhaps we need to focus more on a bigger problem. That is, getting a heifer to conceive, calve without needing assistance, and conceive again in a reasonable amount of time. Geneticists are discussing Holstein-Jersey crosses more seriously today. More can be learned about the function of specific genes from divergent crosses among breeds than from within-breed matings of elite animals.

On-farm Breeding Trial

Information is beginning to accumulate from on-farm breeding trials. This information has to be viewed with caution because it does not pass the criteria for scientific research, e.g., randomization, replication, and sufficient animals to detect significant differences.

Table 3 gives the incidence of stillbirths, i.e., calves dead at birth, in a 1800-cow Holstein dairy herd. Interestingly, the percentage of stillbirths in this large herd is similar to the incidence of PM in data from many herds (Table 1).

Table 3. Incidence of stillbirths in a large Holstein dairy herd[1].

- 1800 cow dairy
- maternity pens checked every hour, 24 hr per day
- outcome
  - ➔ 12% stillbirths on 629 heifer calvings
  - ➔ 7% stillbirths on 1275 cow calvings

[1] J. Metzger, 4/8/99, unpublished data, Riverview Dairy, Inc., Morris, MN.

Table 4 gives the incidence of stillbirths from an on-farm breeding trial for Holstein heifers. That some reduction in PM was possible by mating Jersey bulls to Holstein heifers cannot be disputed. More on-farm breeding trials are needed to determine if the result can be repeated.

Table 4. Incidence of stillbirths in an on-farm breeding trial for heifers.[1]

Experiment: "We ran natural service Jersey and Holstein bulls with virgin Holstein heifers."

Outcome:	Jersey-sired	Holstein-sired
Stillbirths	1/46 = 2%	18/151 = 12%

[1] J. Metzger, 4/8/99, unpublished data, Riverview Dairy, Inc., Morris, MN.

Mode of Inheritance. Several factors may be responsible for PM. Possible explanations include diseases, environment, improper nutrition and genetics. Autosomal recessively inherited defects or, more broadly, multiple genes may be responsible for PM. An increase in PM has been reported in Sweden. Danish scientists have reported an increasing number of malformed neonatal calves for the Holstein breed since 1999. “Malformations have been observed both in aborted fetuses, prematurely born calves, stillborn calves, and neonatal calves. Cases among older calves have not been observed yet. In general the body weight is reduced. Externally, there are two major findings: in many cases the cervical and/or the thoracic part of the columna seems to be short.” (J. S. Agerholm, unpublished data).

Known today as complex vertebral malformations, or CVM, the affected calves are consistent with an autosomal recessively inherited effect. Further information on the molecular basis of CVM will be released soon. A former elite U.S. Holstein sire, 7H543 Carlin-M Ivanhoe Bell, was found to be a carrier of CVM, therefore the identification of this gene has important implications. Several cases of CVM have been confirmed in the United States (D. Steffen, DVM Ph.D., 2001, personal communication, Veterinary Diagnostic Center, University of Nebraska-Lincoln, NE).

Recommendation. It is no longer acceptable to assist every birth at the first sign of contractions or at visible signs of feet for a new calf. Premature rupture of the umbilical cord can cause substantial harm to the cow and her calf, sometimes death of the calf is even possible. Recent research by Dr. Howard Tyler and graduate student Carrie Hammer at Iowa State University has shown that Holstein cows typically have a two stage parturition; slow dilation that may last as long as an hour, and then a final phase of accelerated contractions. Contractions for some cows may start and stop during the first phase of parturition. No type of assistance should begin before a cow is clearly in the second stage of parturition. Premature rupture of the umbilical cord can increase the likelihood of a dead calf, increased immunological disorders in young calves, and increases the incidence of retained placenta and prolongs rebreeding interval in cows.